Social Support, Social Functioning, and Psychiatric Symptomatology: Social Selection or Causation?

by

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FACULTY OF GRADUATE STUDIES

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Symptomatology:

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A Thesis/Practicum submitted to the Faculty of Graduate Studies of The University of

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Dedication

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Abstract

Social support and psychiatric illness are reported to be inversely related. The Social Selection and the Social Causation theoretical models explaining the relationship between social support and symptomatology are evaluated. Literature supports both opposing positions and a Reciprocal Causation Model is proposed to explain divergent findings. Social functioning (socioeconomic status [SES], education, income, employment, and financial adequacy perceptions) variables were evaluated for their impact on symptomatology. Significant financial stressors were also evaluated for impact on psychiatric symptomatology. If social support is a malleable environmental characteristic it is important for psychiatric health and program planning. Outcome variables included social support, psychiatric symptomatology, and length of inpatient stay (LOS). A short mail survey, assessing mental health and social support was sent to members of The Mood Disorders Association of Manitoba. Client members (N approximately 600) were asked to complete the surveys and return them by mail (response rate of 36%). Every six months follow-up surveys were mailed until data for four time-points were collected (Time-1, -2, -3, and -4 sample sizes are N = 232, N = 146, N = 123, N = 96 respectively). Path-analytic techniques incorporating cross-lagged panel regression models were used to evaluate the models. Social support and psychiatric symptomatology were inversely related, with small to moderate effect sizes (approximately r = .30) supporting both Social Causation and Selection theoretical models. These findings provide support for some form of Reciprocal Causation Model. SES variables did not impact the models; but financial adequacy perceptions influenced them. The Social Causation Model may be a better explanatory model for these participants. Almost all of the variables, including

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CHAPTER 1:

Introduction

In the 1980s the provision of health care for psychiatrically ill clients changed significantly (Orford, 1986). These changes were partially because of Goffman's (1961) and others' work on the negative effects of institutional treatment on psychiatric patients. However, according to Levine (1981), many other forces played a role in changing the treatment of psychiatric clients. The de-institutionalisation movement resulted in a shift of care and treatment for individuals with serious mental health difficulties from institutions to community based resources (Friedrich et al. 1999). Some see the motivation to downsize psychiatric hospitals and de-institutionalise the mentally ill was due to cost cutting rather than a true desire to mitigate the harmful effects of psychiatric institutions and improve patients' quality of life (Holley, Jeffers, & Hodges, 1997). There was a significant reduction in patient mortality with the de-institutionalisation (Amaddeo et al. 1995). Changes occurred due to improvements in treatment protocols brought about by the advances in pharmacotherapy research. Many mentally ill could be medicated through the worst of their symptoms and ceased to be as great a danger to themselves or others. However, predicting who will be dangerous requires a significant amount of clinical judgment (Beauford, McNiel, & Binder, 1997). Approximately 2-5% of patients display some form of aggressive behaviour and these safety issues have prompted members of the community to call for increased or protected inpatient provisions (Knapp et al. 1997). The fiscal conservatism movement mandated treating individuals in the most cost effective manner. State run institutions were more expensive to run than were

smaller community-based programs. Another movement influencing the shift from institutional treatment to more of a community-based approach involved the civil libertarian doctrine. Mentally ill individuals have a right to live outside of institutions for the mentally ill in the least restrictive environment that would ensure they are not a threat to themselves or others. For an extensive review of the historical and political development of the mental health system from the state hospital model to the more community-based treatment system the reader is referred to Levine (1981). The last of the major changes in the treatment of the psychiatrically impaired involved improvements in alternative community-based programs.

The community-based care movement was born from the beliefs that long-term hospitalizations contributed to isolation, fostered dependency, made it difficult for mentally ill individuals to return to their position in the general (normal) population, and that a person's life quality should improve out of the hospital (Brown & Parrish, 1987). Self-reported life quality has been reported to be negatively related to presence and degree of psychopathology (Angermeyer et al. 2002; Simon, 2003; Suurmeijer, Reuvekamp, & Aldenkamp, 2001; Trompenaars, Masthoff, Van Heck, Hodiamont, & De Vries, 2006). In fact , according to Trompenaars et al. (2006), there is a gradual increase in quality of life when symptoms reduce. Patients with serious pathology (DMS-IV, Axis-I and Axis-II diagnoses) had lower scores on life quality than those without these diagnoses (Suurmeijer et al. 2001; Trompenaars et al 2006). However, there are still important questions remaining about what treatment and social environment are optimal for clients with major psychiatric disorders. Health care services available for psychiatric

clients have improved over the last two decades (Pietzcker & Gaebel, 1987). American hospitals are smaller, better staffed, have more of a treatment orientation, and non-hospital community settings are also more available (Mechanic, 1989).

The de-institutionalisation movement, to replace large psychiatric hospitals with community based services for the seriously mentally ill, became policy in a number of countries including Britain (Knapp et al. 1995), the United States (Levine, 1981), and Canada (D'Arcy, 1978). In Canada, with the advent of the de-institutionalization movement, the policy of providing general hospital beds for the mentally ill began in the 1950s and it was intended to function as a preventative service to those early in the process of developing mental illness (Woogh, 1986). In Canada, the United States, and Britain, there has been a problem with shortage of community beds at the right service level and these shortages result in increased inpatient service consumption and increased mental healthcare costs (Knapp et al. 1997). The care of the seriously mentally ill, who were released from institutional settings, placed pressure on acute psychiatric beds in general hospital facilities, since acute care hospitals were often already running at maximum capacity (Augarde, 1998). According to Harvey et al. (1997), more serious psychiatric disorders also tend to have a chronicity component.

The Canadian system of 'universal health care' is intended to make it possible for the whole population to access an array of psychiatric services. The Canadian system results in the provision of basic universal coverage without costing the consumer directly (Adams, 1994). Direct comparisons between differing countries are not possible because of important differences in each country's legislation (Toews, Prabhu, & El-Guebaly,

1980). Therefore, the likelihood of admission for inpatient treatment should differ when comparing Canada to the United States (Pérez et al. 1985). Yet, differences in health insurance policy between Canada and the United States have been reported not to influence the type of individuals using emergency psychiatric services (Friedman, 1983; Suh & Carlson, 1979). Also, the demographic and clinical characteristics of Canadian samples of emergency patients have been found to be similar to American samples (Blais & Georges, 1969; Harvey & Brown, 1971; Suh & Carlson, 1979).

In Canada, according to D'Arcy, 1978, in general, front line psychiatric care is now delivered by general health practitioners and/or psychiatrists, primarily in physicians' offices on a fee-for-service basis. The majority of inpatient treatment episodes are handled in general hospital psychiatric units rather than psychiatric institutional settings. All of these services are paid for by public funds so there should be no economic barriers to access and early treatment.

The delivery of mental health services in Canada also changed with the arrival of the community mental health movement (D'Arcy, 1978). In Canada, insured medical services are available to everyone without limits on access and with no user fees (Bland, Newman, & Orn, 1990). Within Canada there are also significant differences in the systems of mental health care delivery between the provinces (see Malla, Norman, & Helms, 1987; Adams, 1994, Tataryn et al. 1994). Even with Canada's mental health care delivery system mental heath treatment is not delivered without costs to the patients. Most psychiatrists are located in larger community environments and gaining access to these resources is difficult if you do not reside in a large enough urban area. Rural

persons do not have the same access as urban living patients. Travelling to these larger urban environments results in travel costs. There is a low supply of psychiatrists so accessing this level of help is difficult. Some patients could be helped by psychologists but there is no financial coverage for psychologists' fees. There are also many other costs associated with mental health treatment that serve as barriers to access. Travel time and expense, time lost from employment, and the cost of medications. Some individuals have supplementary coverage (private insurance) to help defray additional costs, while others bear the brunt of all of the additional costs directly.

In Canada and the United States, the move to de-institutionalise mental health patients has been said to be a serious policy failure since many patients released did not receive the necessary community support to integrate them into society (Dear & Wolch, 1987; Nirje, 1976; Wolfensberger, 1972). Community services in Canada were intended to be active and if, future care was necessary, it was intended to occur at general hospital psychiatric units, rather than psychiatric institutions (Holley, Jeffers, & Hodges, 1997). In Canada, there is an active treatment philosophy, with the goal of returning patients to the community at the highest functioning level possible (Binner, 1991; Holley et al. 1997).

In the United States, depressive disorders afflict between 1.5 and 4.4% of the general population (Goodwin & Jamison, 1990). In Australia, depressive disorders are estimated to affect 3% of Australians aged 6-17 years every year (Sawyer et al. 2001). According to Clayton (1981), bipolar disorders (both depressive and manic symptomatology) contribute significantly to the public health problem and influences approximately 1% of the adult population. Adult depression may have had its onset in

adolescents (Fitzpatrick, Piko, Wright, & LaGory, 2005). According to Talbot (1980), patients who are consistently being readmitted create serious difficulties for whatever health care system is in place, since these patients consume huge quantities of the most expensive and limited healthcare resources (Shah & Ganesvaran, 1997). Researchers examining Canadian samples report that they are similar to American samples in demographic and clinical characteristics (Blais & Georges, 1969; O'Regan, 1965; Suh & Carlson, 1979; Watson, 1978). In an adult Province of Manitoba sample, the prevalence of psychotic disorders (or serious mental disorders) was estimated at 1% (Tataryn et al. 1994). According to Douglas & Hart (1996), most research supports an association between violence and psychotic symptomatology (Douglas & Koch, 2001). Canadian results have indicated that just under 1% of adults in the general population have a severe and persistent mental illness (SPMI) and a quarter of those are in need of intensive community support (Wasylenki et al. 2000). "Among adults with psychotic disorders, approximately 30% of hospital admissions are patients being readmitted for the second or third time in the same fiscal year" (Tataryn et al. 1994, p. 3). These disorders have an episodic and chronic course that can lead to severe disruptions in family, social, and occupational realms that may result in a clinically significant increase in mortality (Goodwin & Jamison, 1990).

Psychiatric consultation rates differ from country to country. British researchers found the consultation rate to be as high as 33 visits per year (Goldberg, 1972). Their American counterparts who see psychiatrists see them on average 31.4 times a year (Towery, Sharfstein, & Goldberg, 1980). The rate in Canada of only 5.8 visits a year to

any professional seems low in comparison (Bland, Newman, & Orn, 1990). Canadian patients seeing psychiatrists see them on average 3.6 times a year and these differences may be due to differing availability of these specialty psychiatric services in Canada or access to family physicians (Bland et al. 1990).

Affective disorders, including unipolar and bipolar depression, are considered major psychiatric mood disorders (Goodwin & Jamison, 1990). Depression is both a symptom state and a psychiatric condition (Mechanic, 1989). A person can be suffering depressive symptoms in response to an event or environmental stressor, but not be diagnosed as suffering from either a bipolar or unipolar depressive psychiatric condition (Goodwin & Jamison, 1990). Another person may be diagnosed as suffering from a depressive condition; however, they may not display the visible symptoms of depression (Mechanic, 1989). In simple terms, the difference between someone suffering from depressive symptoms at one point in time and someone diagnosed with a depressive disorder appears to be due to the repetitive nature of the symptoms or how chronic symptomatology has been. Many chronic patients experience high levels of symptoms and have related problematic behaviour (Richards et al. 1997). Depression in normal population members is also physiologically and psychologically distinct from the more severe conditions of those of psychiatric hospital clients (Hall & Johnson, 1988). Depressive symptoms do not necessarily develop into the more chronic conditions that would be considered major psychiatric disorders; however, the clinical treatment of the symptoms may be very similar.

The treatment of clients with major affective disorders often includes

hospitalization, an expensive component of treatment. Both unipolar and bipolar clients are often hospitalized repeatedly (Goodwin & Jamison, 1990). Major affective disorders have been found more likely to have had a longer treatment history and more inpatient hospitalisations (Jordan et al. 1996). Impairments in functioning and emotional well-being are seen in patients with major affective disorders (Cooke, Kruger, & Shugar, 1996). Also, patients with seasonal affective disorders (SAD), who are high on seasonality are likely to be relatively unsociable for a portion of the year, and may develop poorer social support networks (Michalak, Wilkinson, Hood, Dowrick, & Wilkinson, 2003). Of persons hospitalized with a major unipolar depressive episode, between 50% and 85% will have one or more subsequent episodes leading to hospitalization (Papolos & Papolos, 1987). Current results from a sample of Manitoba based hospital treated mood disorder clients indicate a readmission rate of approximately 15% during a six month period (Murray, 2002).

Suicide is a self-directed form of violence, and is a traumatic event of medical and social significance. It causes considerable distress to relatives and professional care-givers and patients with suicidal behaviour have usually been admitted previously for inpatient treatment (Shah & Ganesvaran, 1997). Bipolar patients are often hospitalized because of the high risk of suicide (Goodwin & Jamison, 1990). Approximately 50% of these inpatients had chart references to suicidal ideations, and nearly half had made at least one suicide attempt (Briere et al. 1997). This thesis raises questions about how often inpatient treatment is necessary. According to Hirchfeld and Russell (1997), and Harwitz and Ravizza (2000), "suicide represents the tip of the iceberg

in a population suffering from depression, hopelessness, and added co-morbidity of substance abuse, ninety percent of completed suicides are performed by individuals with a diagnosable psychiatric illness, substance abuse, or both" (Ryb, Sonderstrom, Kufera, & Dischinger, 2006). According to Helliwell (2003), suicidal outcome is based upon behaviour not subjective opinions, which limits the use of subjective well-being.

According to Atkinson, Leim, and Leim, (1986), researchers need to accept, as a fundamental concept, the socially structured interdependence of life circumstances and stressful events. Investigators must take more of a multi-factorial view of the etiology and development of depressive disorders (Eisemann, 1984). "Family situations, personality of the mentally ill person, stressors in the environment, economics, and peers all interact to shape how the chronic patient copes" (Lewis, 1990, p. 925). Chronic stress due to life difficulties in depressed women has been found to be related to elevated cortisol secretion (stress hormone) and these elevations were not found in non-depressed women (Strickland, Deakin, Percival, Dixon, Gater, & Goldberg, 2002).

Personality variables also impact social support availability; for example, hostility may decrease social support availability because the hostile person may increase interpersonal conflict and social stress (O'Neil & Emery, 2002). Hostility is a risk factors for the progression of depressive tendencies (Heponiemi, Elovainio, Kivimaker, Pulkki, Puttoenen, & Keltikangas-Jarvinen, 2006; Vahtera, Kivimaki, Uutela, & Pentti, 2000). The positive impact of social support can be diminished by conflict in one's social network (Okun & Keith, 1998). Social networks may also contribute to stress levels for some and in so doing may create feelings of being 'over protected' and resulting in even

more stress (Boutin-Foster, 2005).

Differing attachment styles also impact the support a patient may need. For example, patients with avoidance attachment styles see others negatively and devalue the importance of others in their lives possibly so they can avoid relying on them for help (Lopez & Brennan, 2000). Those with attachment avoidance would perceive less social support and this perception of lack of support is negatively associated with psychological distress (Vogel & Wei, 2005). On the other hand, patients with attachment anxiety perceive others positively and they often over-emphasize their distress to try to elicit help from others (Cassidy, 2000). These attachment styles contribute to an individual's decision to seek counselling (Komiya, Good, & Sherrod, 2000; Vogel & Wei, 2005).

According to Olsen, Gunner-Svensson, and Waldstrom (1991), research supports the notion that social relationships influence health. Patient adherence may mediate the relationship between health and social support (Dunbar-Jacob & Schlenk, 2001) and treatment outcomes (DiMatteo, Giordani, Lepper, & Croghan, 2002). According to Druley, and Townsend (1998) and Hagedoorn et al. (2000), the pathway from social support to health likely travels through patient adherence to treatment programs (DiMatteo, 2004).

Social exclusion has been found to produce emotional/social pain (Zadro & Williams, 2003) and there is evidence that social and physical pain share a common neuroanatomical basis (Eisenberger, 2003). Isolation is believed to play an etiological role in the development of certain psychiatric disorders, including depression (Wilkinson, 1975; Neziek, Hampton, & Shean, 2000) greater psychological stress negative affect, and

poorer health (Bisschop et al. 2003; Mendes de Leon, Gold, Glass, Kaplan, & George, 2001). According to Uchino (2004), social support acts in a protective manner in the stress-related model as it reduces the effects of stress in a persons life. For patients with renal disease social support was inversely related to depression (Symister & Friend, 2003). Social support was even found to be related inversely to dental difficulties (Merchant, Pitphat, Ahmed, Kawachi, & Joshipura, 2003).

Loneliness has been conceptualized as an unfavourable balance between actual and desired social contact (see Ernst & Cacioppo, 1999; Tomaka, Thompson, & Palacios, 2006). Loneliness by itself has been found to be associated with more depressive symptoms (Cacioppo, Hughes, Waile, Hawkley, & Thisted, 2006). In fact, according to Cacioppo, Hughes Waile, Hawkley, and Thisted (2006), there appears to be a reciprocal relationship between loneliness and depressive symptomatology. The absence of close attachments and recognition of worth causes emotional loneliness and depressiveness (Heikkinen & Kauppinen, 2004; Lyyra & Heikkinen, 2006; Taylor & Lynch, 2004). Loneliness has been found to be related to suicidal behaviour (Goldsmith, Pellmar, Klienman, & Bunney, 2002). Lonely and isolated individuals have higher levels of stress which is related to more negative health outcomes, poorer immune responses (Pressman et al. 2005), and worse mental health (Cacioppo et al. 2003; Mendes et al. 2001). Higher scores on loneliness actually predicted greater depressive symptoms (Heikkinen & Kauppinen, 2004) and this relationship remained after controlling for social support (see Hagerty & Williams, 1999). Poorer mental health has been found to be related to feelings that there was no one to turn to for help (Dunn, 2002). Social isolation was found to be

more of an issue when clients lived in independent living arrangements, and loneliness was found to be greater for single patients (Friedrich et al. 1999).

However, some researchers report that there is a cost associated with being the recipient of social support (Bolger, Zuckerman, & Kessler, 2000). Social functioning variables like social support, education, unemployment, income, and poverty have all been found to influence psychiatric disposition. Individuals with lower incomes tended to experience more depressive symptoms and they have trouble meeting their basic needs (e.g., problems with getting food or transportation), and financial deprivation has been found to have an independent negative impact on psychological well-being (Ennis et al. 2000; Hobfoll et al. 2003) and poor health status (McLeod, Lavis, Mustard, & Stoddart, 2003). For persons in low income groups social supports has been found to be more salutogenic or ameliorating (Vitaiano et al. 2001).

Psychologists, medical doctors, sociologists, and social workers have debated the causes of psychiatric illness. Individuals from different philosophical camps have chosen opposing research results in the literature to support their favoured theoretical position. The data that have become available during the last ten years clearly support a genetic component as at least significantly responsible for the predisposition towards severe mood disorders (i.e., unipolar and bipolar depression)(Goodwin & Jamison, 1990). In fact, prolonged or repeated activation of the sympathetic nervous system and hypothalamic-pituitary-adrenal cortical axis is thought to place a person at risk for the development of psychiatric disorders (Cohen, 2004). Consistent with the nature component of the nature/nurture dichotomy in psychological fields and the social

selection component of the social selection/social causation dichotomy in sociological fields" (Murray, 1996, p. 166).

The literatures clearly indicate that all of genetic, biological, and environmental factors contribute to depressive disorders; so, genetic factors alone cannot account for psychiatric disorders (see Dohrenwend, 1976; Gottesman & Shields, 1976; Merikangas, 1990; Plomin, 1990; Dohrenwend, 1998). According to Spotts et al. (2004), genetic influences (using twin research) did account for some of the covariation between depressive symptoms, social support, and marital quality.

Two diametrically opposed theoretical positions have been proposed to explain the relationship between the social environment, socioeconomic status (SES) and psychiatric pathology. The Social Causation Model, based upon the classical research of Merton (1957), proposes that environmental circumstances create hardship and it is this hardship and distress that leads to mental illness (Bebbington, Christopher, & Hurry, 1981; Brown & Harris, 1978; Faris, 1934; Faris & Dunham, 1939; Hollingshead, 1957; Leighton, Hording, Macklin, MacMillan, & Leighton 1963; Srole, Langner, Michael, Opler, & Rennie, 1962). Social causation, as proposed by environmentally oriented theorists, implicates environmental conditions in explaining the inverse relationship between psychiatric morbidity and social environment and SES, suggesting that lower SES persons are exposed to more environmental adversity and stress (see Bebbington et al. 1981; Brown & Harris, 1978; Dohrenwend, 1998; Faris & Dunham, 1939; Hollingshead & Redlich, 1958; Leighton et al. 1963; Strole et al. 1962). In fact, according to Baum, Garofalo, and Yali (1999), the lower the SES the more chronic stress they

experience. An emphasis on resources in stress research is broadly generalizable across populations and stress levels (e.g., Hobfoll et al. 2003: Holahan et al. 1999, 2000). According to Johnson, Cohen, and Brook (1999), although the two theories originated as competing explanation between SES and the prevalence of psychiatric disorders, empirical evidence increasingly indicated that the process described by social causation and social selection theories are not mutually exclusive (Dohrenwend et al. 1992).

The Social Selection Model, stemming from more genetically oriented theorists, proposes that higher rates of pathology found in lower SES individuals are due to persons with psychiatric difficulties declining in SES and social functioning due to psychiatric morbidity and related characteristics making upward social mobility impossible and downward drift more likely (see Bland & Orn, 1981; Dohrenwend, 1998; Dunham, 1965; Eaton, 1980; Grunefeld & Salveson, 1968; Hafner, Nowotny, & Loffler, 1995; Jablensky, 1986; Jarvis, 1971; Munk-Jorgensen & Mortensen, 1992; Wender et al. 1973; Wiersma, Giel, De Jong, & Slooff, 1983). Individuals of lower SES levels have fewer resources and less instrumental social support to fend off social isolation another risk factor for mental illness (Long, Ickovics, Gill, & Horwitz, 2001). Social inequalities increased exposure to stressful events (Adler, 2001). According to Hobfoll, (2002), key resources may help a person counteract resource loss when confronted with external demands (Gorgievski-Duijvesteijn, Bakker, Schaufeli, & van der Heiden, 2005).

Although these two models have dominated the research on mood disorders there is evidence that the relationships between social support variables and psychiatric symptomatology variables are likely non-recursive in nature, meaning that they probably

work in both directions at the same time (Hanlon, 1981; Murray 1996). A third model has also been proposed to explain the relationship between the social environment and psychiatric morbidity. This third model synthesises both of the previous models and proposes that both processes are working at the same time in a reciprocal relationship, a kind of negative feedback cycle (Murray, 1996). This model has been called the Reciprocal Effects Model (Calsyn & Winter, 2002). This model proposes that as one's mental heath symptoms get worse this aggravates the person's current social functioning; and, as functioning and environment worsen, they have a negative impact on mental health. This model also proposes that a person in a deprived social environment is more likely to develop psychiatric morbidity and then do damage to their already deprived social environment.

According to Dohrenwend et al. (1998), the most significant difficulty related to this research is in creating a viable research paradigm in which the opposing theories (selection and causation) can be evaluated, assessed as reciprocally related, and compared for relative importance. Using a research paradigm involving race as an SES-disadvantaged ethnic group, these authors demonstrated that these processes differed based upon Diagnostically Related Group (DRG). The social selection processes appeared more important in predicting the relationship between mental illness and SES in patients with schizophrenia. However, there was also support for the social causation processes being more important than social selection for individuals with a depressive disorder. Basically, health status improves with increasing social and economic status (Institute of Medicine, 2003; Lynch & Kaplan, 2000). The prime focus of this research

was to evaluate the causation and selection hypotheses. The literature related to these topics uses the theories to explain incident cases of mental illness. This thesis has extended this approach to appraise the impact of the two processes in persons already diagnosed (prevalent cases). This research also attempted to extended research from a kind of generalized stress model approach to focussing only on the financial stress component in mental health clients lives. So only the impact of financial stressors were included when evaluating the relationship between social support, social functioning, and psychiatric symptomatology.

Testing these two models has proven to be extremely difficult due to a multitude of design and methodological limitations. Practical, ethical difficulties arise in much mental health research creating significant design problems (Dohrenwend, 1998). According to Dohrenwend (1998), it is likely that both processes, social selection and causation, are operating at the same time (reciprocal relationship), but specifying the relative importance of the processes has been difficult. Both the social causation and social selection theories predict an inverse relationship between SES and pathology. The relationship between SES and psychiatric symptomatology holds despite all of the differing approaches used to evaluate it. The biggest issue in the relationship between SES and mental illness is whether psychiatric symptomatology causes SES (Social Selection or Social Drift Theory) or SES causes psychiatric symptomatology (Social Causation Theory; Dohrenwend, 1998). According to Kohn, Dohrenwend, and Mirotzinik (1998), "regardless of the definition used for SES, the type of prevalence rate, or the type of case identification instrument, an inverse relationship has been found to exist between

SES and overall psychopathology" (p. 245). In fact, chronic stress associated with lower SES (income, education, and occupation) may also increase morbidity and mortality (Adler & Newman, 2002). Studies evaluating major depression have consistently shown a relationship between depression screening scores and SES status and that the social causation mechanism may be more important for depressive conditions (Kohn, Dohrenwend, & Mirotzinik, 1998). The Social Causation theories were the first to be postulated based upon extreme historical environmental stressful conditions impacting otherwise healthy persons and creating psychiatric morbidity. However, many studies in the literature have not evaluated these relationships in a longitudinal manner allowing for the variables to be placed in a clear time continuum.

The Social Causation and Social Selection theories discussed in the literature to date have focussed more on the SES variables involved. This thesis assessed not only the dominant SES variables for their impact on symptomatology and length of inpatient treatment, but included a measure of patient perceptions of financial adequacy (FAD). This allowed the relationship between symptomatology and financial adequacy perception to be evaluated apart from the more commonly used SES indicators.

This research allowed for the testing of a priori causal relationships between networks of interacting variables to be evaluated. The sample was not restricted to psychiatrically admitted clients; rather, the focus was on a population of individuals with a psychiatric diagnosis who were members of a self-help group. Some group members had been in a psychiatric facility for treatment while others were treated, by private care physicians, or on an outpatient basis. A large population of individuals with depressive symptomatology never receives inpatient treatment (Koenig, George, & Meador, 1997).

Therefore, this study has the opportunity of evaluating the relationship of social functioning variables to psychiatric symptomatology in members of at-risk populations who may not have been admitted to an inpatient psychiatric facility for treatment. Since there are a number of abbreviations used in this thesis providing a table detailing all abbreviations have been provided (see Table.5 below).

Table . 5

All Abbreviations Used in this Thesis

Abbreviation	Abbreviation Meaning
CF	Contact Frequency
EWB	Emotional Well-being
ED	Education
FAD	Financial Adequacy Perception
LOS	Length Of Stay
PROC CALIS(RAM)	Command for the SAS program
SASPA	SAS computer Program for Path Analysis
SBP	Systolic Blood Pressure
SES	Socioeconomic Status
SF1SY2, SF1SY3, and SF1SY4	Social Functioning and Symptomatology
SPMI	Severe and Persistent Mental Illness
SS	Social Support
SVAS	Segmented Visual Analog Scale
SY1SF2, SY1SF3, and SY1SF4	Social Functioning and Symptomatology
SYMP	Symptoms
VAS	Visual Analog Scale
WAS	Winnipeg Area Survey

The subsequent introductory sections of this thesis outline the relationships between social support variables (contact frequency, satisfaction with social support), and SES variables (education, employment, income, and perception of financial adequacy), and how they may influence psychiatric symptomatology and clients' use of psychiatric hospital resources (length of stay; LOS). The impact of the social causation versus social selection theoretical positions is evaluated and expanded to determine how these two diametrically opposed theories explain the relationship between social circumstances and psychiatric symptomatology. Path-analytic models are also presented in an attempt to explain how these variables may be causally interrelated. Data analysis took place in four phases. Phase-I: evaluation of sample selection, control variables, and measurement issues; Phase-II: cross-sectional path models demonstrated the replication of results over a two-year period; Phase-III: social causation and selection models were evaluated for consistency in time-lagged models; and finally Phase-IV: the impact of symptomatology, social support, and socioeconomic status on inpatient treatment was evaluated. Subsequent chapters of this dissertation include: Chapter 2, which provides a literature review of the pertinent variables involved, Chapter 3, which consists of Phase I of this research and details the data analysis issues involved, Chapter 4 consists of Phase II of this research in which the replication of the preliminary Time-1 cross-sectional path diagrams are evaluated, Chapter 5 consists of Phase III of this analysis and provides the longitudinal test of the social causation and social selection theories. Chapter 6 consists of results and discussion of the fourth phase of the research analysis and discussion related to LOS, and finally Chapter 7 consists of a summary of key findings, contributions to the literature, current research limitations, suggestions for future research, policy implications, and a conclusion.

CHAPTER 2:

Review of the Literature on Social Functioning and Socioeconomic Status and Mental Health

This chapter begins by providing the reader with a brief outline of the social support literature as it may be important to the current research questions. Following the section on social support there is a section discussing the importance of socioeconomic indicator variables, including education, employment, income, and perception of financial adequacy. The literature related to inpatient treatment of the mentally ill is then briefly summarized followed by a brief discussion of issues related to causality. This chapter continues with a brief section discussing limitations in the literature and closes with the current research objectives and plan for analyses.

Social Support

The idea that social environment is important in explaining susceptibility or adaptation to disease can be traced to Durkheim's classic research on European suicide rates (Durkheim, 1951). In the mid 1960's, researchers began to study the effects of social structure on well-being at an individual level (Lin & Ensel, 1989). The most basic definition of social support is the 'resources supplied by other persons' (Cohen & Syme, 1985). According to Leavy (1983), social support includes the tangible and intangible assistance that people receive from friends, family members, and others in their social network. "Early researchers conceptualized social support as a generalized resource available from one's network of friends and acquaintances (the social network) that helped one to deal with everyday problems or more serious crises" (Walker, Wasserman,

& Wellman, 1993, p. 71). Social support is a multidimensional construct (Cohen, Gottlieb, & Underwood, 2000; Dean, Kolody, Wood, & Ensel, 1989; House & Kahn, 1985; Tomaka, Thompson, & Palacios, 2006). According to Falk, Hanson, Isacsson, and Ostergren (1992), a more current description of social support includes emotional support, informational support, and material support. Possibly the most encompassing definition of the support concept is summarized by Weiss's (1974) five functions of support. These five functions include: indicating personal value, facilitating belonging as in group membership, providing for attachment and intimacy needs, supplying a needed opportunity for being nurtured, and ensuring the availability of help in the forms of information, emotional, and material aid.

It is generally accepted that individuals need to maintain a basic social support resource for adequate psychiatric and physical well-being (Reis, 1988; Turner, 1981). In fact, negative affect is related inversely with desire to engage in social activity (Watson, 2000). According to Stone et al. (1999) and Vitaliano et al. (2001), exactly how social support contributes to health and the factors that moderate and mediate this relationships are not yet completely understood (DiMatteo, 2004). The literature on the influence of social support on health is extensive. For comprehensive reviews and a discussion of the conceptual and methodological difficulties in this field the reader is referred to Aneshensel and Stone (1983), Cohen and Willis (1985), Leavy (1983), Thoits, (1982) or Cohen, (2004). Strong empirical evidence indicates that a causal relationship exists between an individual's social interactions and health (House, Landis, & Umberson, 1988; Helliwell, & Putnam, 2004). Depressed participants have been found to experience

less enjoyment out of social interactions, they report less intimacy, and they felt that they had less influence over their social interactions (Neziek, Hampton, & Shean, 2000). The perceived social support factors of the Family and Friends component of the MSPSS-12 have been found to have a strong association with psychiatric symptomatology (Clara, Cox, Enns, Murray, & Torgrude, 2003). Perceived availability of social support has been found to reduce the effect of psychological distress and depressive symptomatology (Cohen, 2004). The subjective component of social support has been found to be the most highly correlated with mental health outcomes (Turner, 1999), and support from mother and siblings has been found to strongly predict mental health outcomes (Bassuk, Mickelson, Bisseell, & Perloff, 2002). In fact, younger adolescents report receiving support from their older siblings and this is a significant effect (Branje, van Lieshout, & van Aken, 2004).

There is controversy about exactly what amounts and roles of support are related to the etiology of psychiatric disorders, including depression. However, there are some inconsistencies in the life stress literature. According to McQuaid, Monroe, Roberts, Kupfer, and Frank (2000), methodology may account for some of these inconsistencies. Much of the literature and theory assumes that social supports and stressful life events are independent of one another (Brugha, Bebbington, Sturt, MacCarthy, & Wykes, 1990). Perceived lack of control over life events is associated with an increase in depressive symptoms (Weary & Gannon, 1996). Life events and social support are independent predictors of psychiatric morbidity (Vaananen, Vahtera, Pentti, & Kivimaki, 2004). However, Atkinson et al. (1986) have argued that the assumption of independence

between stressors and supports seems tenuous, considering that social support indices and stressful life events are often correlated (Carveth & Gottlieb, 1979). Social support has been found to be important to mortality (Walter-Ginzburg. Blumstein, Chetrit, & Modan, 2002), morbidity, and emotional health regardless of the particular measure of social support used (Berkman & Syme, 1979; Cohen & Wills, 1985; Seeman & Syme, 1987). Very stressful or negative life events have been found to increase depression (Kendler et al. 1999). Depression and low levels of support have been found to be associated with higher morbidity rates in coronary patients (ENRICHD, 2003). According to Allen, Ciambrome, and Welch (2000), both instrumental and emotional support are important in mediating depressed mood. Social support has been found to be related to schizophrenia (Erickson, Beiser, & Iacono, 1998), bipolar affective disorder (Johnson et al. 1999), and the progression of these diseases (Pennix et al. 1996). Time-lagged impact of social support predicted lower negative mood on the next day (Feldman, Downey, & Schaffer-Neitz, 1999).

Historically, reduced levels of social support have been associated consistently with physical and psychiatric illness, especially depressive disorders (Brown, Bhrolchain, & Harris, 1975). The relationship between social connections and mental well-being has been well documented (Kafetsios, 2006; Putnam, 2000). Social support has been found to protect against stressors (Cohen & Wills, 1985; Dean, Kolody, & Wood, 1990; King et al. 1999; McDonald et al. 1999). Higher levels of social support have been found to have a beneficial effect on psychological well-being (Arling, 1987;.Cohen, Gottlieb, & Underwood, 2000; House, Robbins, & Metzner, 1982; King et al. 1999; Kiecolt-Glaser &

Newton, 2001; McDonald et al. 1999; Vaananen, Vahtera, Pentti, & Kivimaki, 2004). Access to, and interacting with, one's support networks has been found to reduce the effect of stressful life events (Sherkat & Reed, 1992). Weak social support has been found to correlate with more physical and mental health complications (LaRocco, House, & French, 1980). Poor social support, depression severity, and the number of chronic medical conditions, have been found to negatively impact quality of well-being (Mittal, Fortney, Pyne, Edlund, & Wetherell, 2006). Social support has been found to be important for the well-being of people with psychiatric disabilities (Baker, Jodrey, & Intagliata, 1992; Earls & Nelson, 1988; Nelson, Hall, & Walsh-Bowers, 1999). A number of studies have reported an important relationship between larger social networks and better health status or well-being (Nelson et al. 1999). Causes of depressive disorders are not completely clear but are thought to include exposure to stressful childhood events and current psychosocial adversity (Butler et al. 2006). According to Valliant (2002), for decades the literature has reported that childhood affects the well-being of adults, yet more current literature reviews explain that these childhood experiences may not be as important as was previously thought.

Some authors have proposed that lack of social support is a direct cause of depression (Aneshensel & Stone, 1982; Pearson, Wilmot, & Padi, 1986), impacts the onset and severity of depression (Coyne & Downey, 1991), and has been shown to predict relapse (Casey, Meagher, & Butler, 1996; Mann, Jenkins, & Belsey, 1981). However, according to Neziek, Hampton, and Shean (2000), like lonely people, depressed people suffer more from lack of rewarding social contact than from a lack of social contact per

se. For example, people may initiate more interactions with depressed people because they think the individual who is depressed needs more support, and this may partially explain why some depressed people think they have no control over their social interaction with others. The chronically ill are less likely to spend time with friends and more likely to spend time with their immediate family members as a consequence of the illness (O'Brien, 1980).

Stress level and social support have been found to have a reciprocal relationship in depressed individuals (Coyne, Aldwin, & Lazarus, 1981; Schaefer, Coyne, & Lazarus, 1981). Environmental stressors have been found to produce longer recovery times (Brugha, et al. 1997). Social support may bolster functioning by preventing or reducing stress (Gore, 1981; Mitchell, Billings, & Moos, 1982). Individuals with serious depressive conditions usually have social support deficits, fewer friends (Bebbington et al. 1988; Billings & Moos, 1985; Billings, Cronkite, & Moos, 1983; Birtchnell, 1971; Black et al. 1991; Brugha et al. 1990; Brugha et al. 1987; Brugha, et al. 1982; Cornelis, Ameling, & de Jonghe, 1989; Croughan et al. 1988; George et al. 1989), and are less sociable (Barnett & Gotlib, 1988). Stress intensity and perception of social support both predicted well-being, and social support demonstrated a moderate role in mediating the impact of stress (Skok, Harvey, & Reddihough, 2006). Perception of social support is related to the stability of mental health (Gencoz & Astan, 2006; Fujita & Kanaoka, 2003). Social support, whether from within the "home" or outside the "home," is very important for the well-being of people with psychiatric disabilities (Nelson et al. 1999). Patients with a poor social support network may not be encouraged to maintain contact with the

treatment system and these individuals may have difficulty maintaining relationships with care-givers (Tehrani et al. 1996). Depressed individuals often feel alone (Amann, 1991; Eisemann, 1984; Lowenstein & Rosen, 1989). Given these deficits, patients may also be less satisfied with their support system. The number of individuals and number of contacts with one's support system has been found to be more important in predicting recovery than how satisfied the patient is with their support system (Brugha et al. 1997). According to Corrigan, Giffort, Rashid, Leary, and Okeke (1999) and Corrigan and Phelan (2004), emerging evidence indicates that social network supports may act as facilitators to the recovery process from mental illness). In fact, reciprocally supportive relationships with social network members was the most important predictor of mental health recovery (Pernice, 2006). Both helping and receiving help were significant predictors of mental health and giving help was an important predictor of better mental health than was just being on the receiving side of the relationship (Schwartz et al. 2003). However, worse mental health has been found to be related to being overwhelmed by others' demands (Schwartz et al. 2003). Perceived demand may make more impact in psychological well-being than perceived support (Finch et al. 1999). Depressed individuals have also been found to have a negative impact on interaction partners, leading, partners to withdraw and resist future interaction (Vaerum & McCabe, 2001). Interaction between depressive cognition and perceived support may help to account for the increasing sense of isolation experienced by many depressed individuals even when it appears that they have support (Maher, Mora, & Leventhal, 2006). In fact, many mentally ill persons feeling less valued as a friend, romantic partner, or group member than one

desires (Leary & Springer, 2001). According to MacDonald, and Learly (2005), this devaluation is experienced as aversive because it signals and increased probability of ultimate exclusion.

A controversy remains over whether social support and mental health are related through a direct contribution to mental health or an indirect effect through buffering the effects of stressors (Sherbourne, 1988). Some have argued that having few social support resources can, by itself, be stressful and have a direct influence upon psychological symptomatology (Aneshensel & Fredricks, 1982; Cohen, 2004; Thoits, 1983). Reviews by Cohen and Willis (1985) and Kessler and McLeod (1985) suggest that buffering may occur only with perceived or functional support measures, while more structural measures like network membership may influence mental health directly (Vaananen, Vahtera, Pentti, & Kivimaki, 2004). Social support has been found to buffer the impact of negative life events and anxiety and maladaptive behaviours (Carothers, Borkowski, & Whitman, 2006). According to Ullah, Banks, and Warr (1985), neither the direct nor the indirect approaches may be comprehensive enough to explain the entire relationship between social support, life events, and psychological health. Other researchers have found evidence that social support can act in a direct and indirect manner simultaneously (see Bolton & Oatley, 1987; Cohen & Willis, 1985).

A distinction between functional and structural support has been made (Avlund et al. 2004; Uchino, 2004). Various population studies have shown the absence or perceived absence of social supports, infrequent social contact, and changes in a person's network of support to be directly related to higher rates of general pathology (Kessler & McLeod,

1985), emotional distress (Revicki & Mitchell, 1990), and depressive symptomatology (Reis, 1988; Schulz et al. 1987). Social support may come from a variety of sources and support available from all sources should be considered when evaluating an individual's social support resource availability (Dean et al. 1981; Dean et al. 1990; House & Kahn, 1985; Lin et al. 1986).

Williams, Ware, and Donald (1981) have proposed that stressful life events have a negative effect on mental health outcomes, while social support is seen to have a positive effect. According to Patrick et al. (1986), deterioration in well-being was greatest for respondents with a low level of support. In clients dealing with grief, the underutilization of support, the poor quality of support, and reduced levels of social integration or frequency of support all have significant negative effects on depressive symptoms (Sherkat & Reed, 1992) and mortality (Berkman & Glass, 2000, Cohen, 2004; Walter-Ginzbutg, Blumstein, Chetrit, & Modan, 2002). Mortality has been found to have an inverse relationship with received social support (Kaplan et al. 1996). Social support quality has been found to be an important factor in predicting relapse in depressive episodes and future levels of depressive mood and symptoms (Hobfall et al. 2003; Holahan, Moos, Holahan, & Cronkite, 1999, 2000; Joiner & Coyne, 1999). Higher levels of social support, in the presence of stressful events, are associated with a reduced risk of depression (Brown & Harris, 1978). According to Krause (1999), stressful experiences have been shown to increase the risk of developing physical and mental health problems.

Much of the available literature on social support measures attempts to operationally define the global construct of social support. Few available studies have

selectively explored the differential effects of various aspects of social support (Revicki & Mitchell, 1990). In the past, the literature has neglected structural support variables, for example, contact frequency with network members (House & Kahn, 1985). Social contact, the structural measure of support is related to perceived social support (Peirce, Frone, Russell, Cooper, & Mudar, 2000). More recent literature evaluates many components of the social support construct, including the relationship of social support to physical and mental health (e.g., Cohen et al. 2000), the structure of social networks (e.g., Brissette et al. 2000), differing sources of support (e.g., Cohen et al. 2000), the quality of social support (e.g., Kiecolt-Glaser & Newton, 2001), social roles, the interconnectedness of networks, social isolation and the number of social roles (eg., Cohen, 2004).

Dominant approaches for assessing social support include: a) assessing existence of support, type of support, and quantity of available support; b) assessing frequency of interaction, and structure of networks; and c) qualitative assessments measures including components of supportive relationships and satisfaction with support from various sources (House & Kahn, 1985). Both quantitative (structural) and qualitative (functional) measures of social support have been successfully used in the support literature (Revicki & Mitchell, 1990).

Much of psychiatric research has focussed on the role of social support variables (Butler et al. 2006). Researchers studying the effect of social support have taken two basic approaches to measuring the social support construct (Henderson et al. 1978). The first approach concentrates on the quantitative aspects of social support (Fiore et al. 1986) and operationally defines the support variable in structural terms, that is, in terms of the

number or frequency of social ties to network members. Frequency or structural measures assess social support in terms of social integration (House & Kahn, 1985). Structural support indices are also considered the more objective or quantifiable social support measures (Finlayson, 1976; Mancini, 1979), and although they can be discriminated from, they underlie (Hanson & Ostergren, 1987), and correlate well with, the more subjective functional (qualitative) variables (Sarason, Levine, Basham, & Sarason, 1983).

The second approach to the measurement of social support focuses on the quality or function of the available social network (Patrick, Morgan, & Charlton, 1986). According to Allen, Ciambrome, and Welch (2000), network size may also be related to dependability with larger networks providing more opportunities to get help from different network members. Functional measures assess the perceived quality of one's relationships or "what Gottlieb has called the 'psychological sense of support" (Bolger & Eckenrode, 1991, p. 440).

Greater perception of social support has been found to have a positive effect on mental health (Allen, Ciambrome, & Welch, 2000; McKee-Ryan, Song, Wanberg, & Kinicki, 2005). Social contact is often related to subjective well-being but the parameters that affect this relationship are not well understood (Diener, 1984). In the elderly, quality of social contacts shows a stronger association with well-being than the quantity of such contacts (Pinquart & Sorensen, 2000). Perception of social support availability has been found to be associated with better adjustment (Bolger, Zuckerman, & Kessler, 2000) and social support is one of the most important factors perceptions of happiness and success in life (King et al. 2000). The presence of a supportive other is sufficient to convey social

support (Brown, Sheffield, Leary, & Robinson, 2003). Being optimistic has been found to be related to better adjustments to stress and more supportive relationships with friends (Brissette, Scheier, & Carver, 2002) and social support has been found to be positively related to optimism (Cannella, 2006; McNicholas, 2002). However, people's perceptions of social support are often confounded with personality characteristics (Henderson, Byrne, & Duncan-Jones, 1981; O'Neil & Emery, 2002; Vraneanu, Gallo, & Bogart, 2006) including attachment styles (Lopez & Brennan, 2000), and by prior mental health status (Bolger & Eckenrode, 1991; Monroe et al. 1986) than structural or quantitative measures (Bolger & Eckenrode, 1991). According to Brugha et al. (1990), in a depressed cohort, the number of contacts clients made was stable; but the perception of satisfaction with emotional support was less stable over time. Although both structural and functional approaches are used to measure social support and focus on different aspects of the support construct, they are not necessarily mutually exclusive or measuring completely distinct entities (Patrick et al. 1986).

The definitions of social support, theoretical perspectives, measures of adjustment, and results vary across studies. According to Hammer, Gutwirth, and Phillips (1982), both network size and frequency of contact may be important for health. Is it the quantity of support received, the perceived availability or adequacy of support, the size or density of the support network, or satisfaction with the support received that is important for well-being? "Clarification of these issues has both theoretical and clinical implications" (Fiore et al. p. 95). The size of a persons helping network has been found to be a strong mediator in variability in negative mood states (Allen, Ciambrome, & Welch,

2000). According to Bazargan, HammBaugh (1995) and Biegel, Magaziner, and Baum (1991), larger networks is a good indicator of adequate instrumental support, thus mediating negative mood among adults with chronic disabling conditions (Allen, Ciambrome, & Welch, 2000). For more information about the impact of chronic disability see King, Brown, Smith, (2003) and King et al. (2003). Blazer and Williams (1980) believed that certain social network characteristics, such as low contact levels, are associated with poor physical health and mental health outcomes. And experiencing adverse mental health outcomes can reduce a persons capacity to maintain social relationships that can provide future support (Bassuk, Mickelson, Bisseell, & Perloff, 2002).

Being interconnected in a community is necessary for health and well-being (Berkman & Glass, 2000). Larger social networks have been found to be related to better health status or well-being (Anfinson & Kathol, 1992; Baker, Jodrey, & Intagliata, 1992; Berkman & Breslow, 1983; Berkman & Syme, 1979; Cohen, 2004; Earls & Nelson, 1988; Nelson, Hall, & Walsh-Bowers, 1995, 1999; Williams et al. 1981). Social support and social networks have positive effects on well-being (Fratiglioni, Wang, Ericsson, Mayten, & Wimblad, 2000; Sarason, Sarason, & Gurung, 2001). Structural support measures indirectly indicate the availability or adequacy of social support (Sherbourne, 1988). Contact frequency (CF) is an index of the accessibility of individuals in a person's network (Cutrona, 1986), and network contact is a prerequisite for social support to occur (Vega et al. 1991). Those with more frequent contact with their networks have been reported to ask for support more frequently and perceive support to be more available

(Fiore et al. 1986). Also, according to Cutrona (1986), "frequent contact increases the speed with which an individual's needs for support would be communicated to others and would increase the probability that a supportive individual would be present to offer help when needs arise" (p. 349). Contact frequency (CF) indices are more reliable (Hanson & Ostergren, 1987) and stable (Marsden, 1990) than items measuring the number of individuals in the social network. However, a close association exists between items measuring number of individuals in a person's social network and CF, making the latter the quantitative social support variable of choice (Hanson & Ostergren, 1987). Contact frequency was also found to be more important for mood disorder clients who had been recently released from inpatient treatment, even after controlling for satisfaction with support (Murray, 2002).

According to Strauss, and Carpenter (1977), CF has been found to be positively related to most mental health outcome measures. Disabling health conditions have been found to cause a constriction of the social network and reduce the level of contact with friends (Arling, 1987). Improved social networks have been found to be associated with better social functioning (Thornicraft & Breakey, 1991) and better prognoses (Brugha et al. 1990; Swindle et al. 1989), and there appears to be a positive relationship between social support and perceived health status in adolescents (Rew, Fouladi, & Yockey, 2002). Bipolar adolescents have been found to have social skills performance deficits (Goldstein, Miklowitz, & Mullen, 2006). Adolescents reporting more negative life were morel likely to experience depressive pathology, providing support for the belief that life-time negative events increase the risk for developing depressive pathology (Burton, Stice,

& Seeley, 2004). Adolescent boys were found experience higher mental health stigma than adolescent girls and girls are more willing to use mental health services (Chandra & Minkovitz, 2006). Lack of social support during negative life events can reduce self-esteem and can result in patients devaluing their social network, resulting in isolation (Amann, 1991; Cohen, 2004). According to Lewinsohn et al. (1999), negative life events and lacking parental support are correlated with growth in depressive pathology. Being in conflict with members in a persons support network can also lead to adverse mental health outcomes (Bassuk, Mickelson, Bisseell, & Perloff, 2002). Interaction with friends has been found to be important (Arling, 1976; Cohen, Gottlieb, & Underwood, 2000; Wood & Robertson, 1978) and lack of contact may produce distress and symptomatology (Arling, 1987; Cohen, Gottlieb, & Underwood, 2000). Deficits in social support have been found to be inversely related to the development of depressive pathology in youth (Burton, Stice, & Seeley, 2004). Patients who interact less with others have been found to have greater levels of symptomatology (Cohen, 2004; Falloon & Marshall, 1983).

As mentioned above, the depressed individual has a diminished social support network. Depressed patients have also been found to be less satisfied with their smaller social support networks (Billings & Moos, 1985). Some would argue that it is perception of support rather than the veridical "quantity" of support that is actually more important for the person's health (Henderson, 1998). The perception of support has been found to predict a positive relationship with longer life outcome in cardiac patients (Brummett, et al. 2005), decreases in depressive symptoms (Barefoot et al. 2000), and had a unique effect on vascular and myocardial responding (Christian & Stoney, 2006). Depressive

symptoms contribute negatively to cardiovascular diseased (Barth, Schumacher, & Herrmann-Lingen, 2004; Carney & Sheps, 2004). According to Elal, and Krespi (1999), depressed dialysis patients reported less social contact with others, perceived a smaller amount of social support, and were less satisfied from the support they received (Gencoz & Astan, 2006). Adolescents with diabetes who perceive their families as providing more diabetes-specific support also viewed their families as more cohesive and emotionally supportive (La Greca & Bearman, 2002).

Patients have a more positive prognosis if they are satisfied with their social support (Brugha et al. 1990; George et al. 1989), and a patient's perception of his or her support network has been found to become more positive upon enrollment in a community-based program (Junginger, 1990). In fact, some believe that social support could significantly predict all health outcomes (Wang, Wu, & Liu, 2003). According to Park, Fenster, Suresh, and Bliss (2006), greater levels of satisfaction with support and percentage of active coping used were prospectively related to lower levels of depression. However, satisfaction with support from others has also been found not to be predictive of mental health outcome (Brugha et al. 1990).

Social support has been found to have an impact on etiology and symptom expression. Patients who report higher depressive symptomatology also report fewer close friends, fewer positive events, lower levels of family support (Mitchell & Moos, 1984), and smaller support networks (Denoff, 1981). Lack of social support has also been found to be associated with depression onset, especially in a time of crisis (Brown et al.1986). A consistent pattern of results indicates that social support is negatively related

to psychiatric symptomatology for individuals with depressive disorders (Aneshensel & Stone, 1982; Dean et al. 1990; Grant, Patterson, & Yager, 1988; Hafner & Boker, 1973; Ross & Mirowsky, 1989) and lack of support has been found to impair the improvement of depressive symptoms (Aneshensel & Stone, 1982; Brugha et al. 1990; Cornelis, Ameling, & de Jonghe, 1989; Eisemann, 1984; George et al. 1989; Sorensen & Mors, 1993). In congestive heart failure patients, satisfaction with social support and percentage of active coping employed were prospectively related to lower levels of depression (Park, Fenster, Suresh, & Bliss, 2006). Systolic blood pressure (SBP) has also been found to be impacted by social connectedness, positive emotion, and more rapid SBP recovery from daily negative emotional states (Hawkley et al. 2003; Ong & Allaire, 2005), sleep patterns (Cacioppo et al. 2002; Hawkley, Burleson, Berntson, & Cacioppo, 2003), silent coronary problems (Sorkin et al. 2002), diabetes and its complications, and arthritis (Tomaka, Thompson, & Palacios, 2006). In fact according to Tomaka, et al. (2006), diabetes and arthritis may reduce the ability of a person suffering from these disorders to maintain optimal support levels.

A patient's premorbid capacity to maintain an adequate social support system has been found to be related to inpatient treatment (Denoff, 1981). The decision to admit has been found to be influenced by the presence of family support (Henry et al. 1993). Admission decisions are influenced by objective and subjective factors (Hendryx & Rohland, 1997). Repeatedly admitted psychiatric patients tend to have deficient social and personal resources (Holsten & D'Elia, 1985; Lelliot, Wing, & Clifford, 1994), and patients are generally socially isolated on admission (Rud & Noreik, 1982). Often,

patients who return to the community after hospitalization are lonely and isolated, and have other social and psychological difficulties with which to contend (Harris, Brown, & Bifulco, 1987 In fact, according to Cacioppo, Hughes Waile, Hawkley, and Thisted (2006), there appears to be a reciprocal relationship between loneliness and depressive symptomatology). This kind of social isolation has been reported to be positively related to psychiatric symptomatology (Cohen, 2004; Wilkinson, 1975).

LOS has been found to be longer for individuals with inadequate social support (Holsten & D'Elia, 1985; Mai et al. 1993). More established social support networks have been found to reduce inpatient treatment admissions and LOS (Parks & Josef, 1997). Social variables are important when evaluating LOS predictors (Parks & Josef, 1997). Social isolation has been found to contribute to prolonged hospital stays (Rud & Noreik, 1982). Patients have been found to experience breakdowns in their social networks during hospitalisation, and LOS may contribute to this situation (Todd, Bennie, & Carlisle, 1976). However, it is difficult to distinguish whether the symptom onset comes before a decline in social support or whether social functioning causes symptomatology (Turnbull, George, & Landerman, 1990). Patients remain in hospital longer if they have less support to ensure aftercare (Allen et al. 1992; Carpenter & Strauss, 1991; Pottick et al. 1995). New long-stay patients tend to be multiply readmitted despite using or having access to community based services (Korkeila et al. 1995; Saarento et al. 1997).

Reduced LOS has been found to be related to larger social support networks, but this relationship interacts with gender; the relationship between support and appears to be more important for females (Al-Issa, 1982). Young females with diabetes reported higher

levels of friend support than males (Bearman & La Greca, 2002). Females' family support was negatively related to hopelessness, depressive symptoms, and suicidal ideation (Kerrr, Preuss, & King, 2006). Gender has also been found not to be predictive of LOS (Bezold et al. 1996; Boot et al. 1997).Women who appear in emergency room settings have experienced a history of victimization and are in acute distress (Briere et al. 1997). Some patients have multiple trauma histories and have been re-victimized (Briere et al. 1997). Abuse histories are associated with clinical outcomes, ranging from drug addiction and violence against others to depression, non-manic psychosis, and suicidal behaviours (Briere et al. 1997). Trauma is a common experience in persons with severe mental illness and it is related to poor outcomes, increased symptomatology, substance abuse, and homelessness (Goodman et al. 1999; Mueser et al. 2001). Homeless persons demonstrate significantly higher levels of mental health problems than the general population members (Leal, Galanter, Dermatis, & Westreich, 1999; LePage et al. 2006). Persons with severe and persistent mental illness also demonstrate social and interpersonal skills deficits (Iyer, Rothman, Vogler, & Spaulding, 2005). People with severe mental illness usually have significant functional impairments in the activities of daily living (Substance Abuse and Mental Health Services Administration, 2003). Limitations in activity at one point in time predict psychosocial functioning at a later point in time (Eide & Roysamb, 2002).

Psychosis-prone individuals have also been found to report more negative social support experiences (Dangelmaier, Docherty, Akamatsu, 2006). According to Lewis and Rook (1999), efforts to provide support to others may be perceived as controlling and

interfering by the recipient (Taylor, Sherman, Kim, Jarcho, Takagi, & Dunagan, 2004). According to Bolger, Zuckerman, and Kessler (2000), when people actually draw on their social support network for help during stressful times, support seeking often served as an additional cause of distress, because expressing one's needs to others was esteem reducing, and/or drawing on taxing that other person's time and attention (Taylor et al. 2004).

Violent behaviour towards staff results in increase costs of care (Flannery et al. 1997). Female patients have been shown to have higher rates of post-hospitalisation treatment adherence when compared to men (Smith et al. 1997). Other research findings report that gender does not impact hospital admission (Korkeila et al. 1995). Partnered individuals have also been found to have better social support and shorter LOSs (Laessie et al. 1988) and readmissions have been found to be more likely among males (Haywood et al. 1995).

A bipolar or depressed patient's social support has been found to be positively related to his or her outcomes (Billings & Moos, 1985; Brugha et al. 1990; George et al.1989; O'Connell et al. 1991). An absence of family support has been found to be related to worse prognoses in patients with depression (Boyce & Parker, 1985; Moos, 1990; Keitner et al. 1992; Mai et al. 1993; Swindle et al. 1989). The more supportive the parental environment was, the fewer depressive symptoms were experienced (Fitzpatrick, Piko, Wright, & LaGory, 2005). Better clinical outcomes have been found to be related to higher levels of social integration (Klein et al. 1998) and social interconnectedness (Brugha et al. 1987). Better outcomes would therefore be seen in patients with close

networks that are larger and more pro-social (Brugha et al. 1987). However, satisfaction with support from others was found to be less predictive of outcome than other measures of support (Brugha et al. 1987).

It is reasonably clear that despite the differing approaches used to assess social support the results are generally consistent. Social relationships and psychiatric health are inversely related. According to Haughton Mc Neill, Kreuter, and Subramanian (2006), social support and social networks are positively related to emotional health. Poor mental health is related to poorly structured social networks (Brissette et al. 2000), low levels of social support from others (Cohen et al. 2000), poor quality of social support (Kiecolt-Glaser & Newton, 2001), and minimal involvement in a variety of social roles or social isolation (Cohen, 2004). Being isolated and not being connected to a social network is clearly a strong predictor of health (Adler & Newman, 2002). The most socially isolated women have been found to have a reduction in mental health score (Achat et al.1998). Also, those with more social support have been found to be more likely to seek early treatment for psychotic symptomatology (Drake, Haley, Akhtar, & Lewis, 2000; Kalla et al. 2002; Malla et al. 2004).

Socioeconomic Factors

Socioeconomic status (SES) is a construct that can be composed of multiple factors. These factors can include education level, occupation and or employment status, and family income. SES status is related to both income and the person's perception of the adequacy of their financial resources to meet their financial needs. Chronic stress can result from low SES which result in increased mental health problems (Baum, Garofalo, & Yali, 1999). My primary research interests were to evaluate how significant financial stressors impacted the relationship between social support, social functioning, and psychiatric symptomatology.

Education

According to Hoffmann and Mastrianni (1993), the goal of education is to impart the logic of inquiry, discipline, and capacity for observation that make it possible for a person to develop a systematic knowledge of the world and self. Education increases cognitive flexibility and coping and exposes people to different ways of viewing life (Wheaton, 1980). Education is a primary component of SES and it shapes future occupational opportunities and earning potential (Adler & Newman, 2002). Persons who develop a significant psychiatric disorder in their adolescents are left at a distinct disadvantage and these results in a trajectory of poor vocational outcomes and poverty (Collins & Mowbray, 2005). However, with the psychiatric medications and vocational rehabilitation programs, more recently diagnosed persons with significant mental illnesses have better opportunities to pursue higher education goals (Haefner & Maurer,

2000).

Education also symbolizes worth to oneself and to others (Ross & Huber, 1985). Education appears to function not only by supplying a person with information, credentials, and skills, but by reducing feelings of hopelessness and increasing active problem-solving approaches to life's stressors (Wheaton, 1980). Education impacts the experience of unemployment, persons with higher educational levels may have more positive expectations about their chances of finding a satisfactory job (Price & Fang, 2002). In Canada, different levels and experiences of hopelessness have been found in aboriginal groups, with Aboriginals indicating lower levels of hopelessness than Caucasians (Enns et al. 1997).

Individuals with higher levels of education appear to have better social support (i.e., friends who are well educated can better help with information) and larger family incomes (Ross & Huber, 1985). Better educated individuals are also more likely to feel in control of their lives (Ross & Mirowsky, 1989) and experience more support since they have higher levels of friend contact as well (Vega et al. 1991). Education has been found to be positively related to subjective well-being (Clark et al. 2003). Education and income are two of the most important SES indicators for psychological well-being (Ross & Huber, 1985). Virtually all assessments of the relationship between physical and mental health favour persons of higher socioeconomic status (Gottfredson, 2004). Higher income can provide better nutrition, housing, schooling, and recreation (Adler & Newman, 2002). Housing is a concrete manifestation of socioeconomic status, which has an important part to play in the development of explanations of the social production of health inequalities

(Dunn, 2002). The health effects of low SES are particularly difficult for those in the lowest SES group (Adler & Newman, 2002). Negative mental health outcomes negatively impact those that live in resource-deficient social and physical environments (Berkman & Kawachi, 2000). Well-educated persons suffer less from the economic hardship due to low income than individuals who are poorly educated (Ross & Huber, 1985).

The relationship between education and inpatient treatment is not clear. Educational attainment has been found to be only slightly related to psychiatric hospitalization (Anthony, 1980; Shapiro, 1983). Despite the inverse relationship between SES and mental illness (Dohrenwend & Dohrenwend, 1969; Kessler, 1982), persons with higher income and education have been found to make use of psychiatric services more frequently (Leaf et al. 1985). Some researchers reported that those with less education were more likely to be admitted (Levav & Arnon, 1976) and readmitted (Nuehring, Thayer, & Ladner, 1980). Yet, other researchers reported that patients who were readmitted for psychiatric treatment were found to be equivalent in education to those not involved in inpatient treatment (Buell & Anthony, 1975; Hull et al. 1996; Kirk, 1976; Shapiro, 1983; Woogh, 1986; Zohar, Hadas, & Modan, 1987). Higher levels of education have also been reported to be related to reduced readmissions (Geddes et al. 1994; Tsoi & Wong, 1991), lower risk of long-term treatment (Rud & Noreik, 1982), lower admission rates (Zohar, Hadas, & Modan, 1987), and better prognoses (Huber, Cross, Schluttler, & Linz, 1980). However, in a different group of depressed patients, recovery rates were not related to the patient's educational level (Keitner et al. 1992), and education levels have also been found not to be related to the prediction of treatment response (Zubenko et al.

1994).

Education has also been found to impact treatment compliance; those with more education have been found to be more compliant with treatment and to completion of mental health treatment programs (Gillis, Russell, & Busby, 1997). According to Leaf et al. (1984), using the New Haven Epidemiological Catchment Area data, a "significant inverse relationship between 6-month prevalence and years of education, personal income, and employment status" (p. 241). Having less education or belonging to the lower social classes is often related to higher rates of disease, mortality and morbidity (Hanson & Ostergren, 1987). Those in the lowest occupational levels with low decision latitude, and lower social support at work were associated with subsequent higher depressive symptoms (Paterniti, Niedhammer, Lang, & Consoli, 2002). Persons with higher levels of education (Sherkat & Reed, 1992), and larger family incomes (Ross & Mirowsky, 1989) have lower levels of depression. According to Wykes and Holloway (2000), persons with serious mental illness are at a significant disadvantage in the labour marked because of the interruptions in post-secondary education. Yet, Sherkat and Reed's (1992) results indicated that after including support constructs in the model, education's influence became non-significant. However, "persons with low levels of education face a multiplication of disadvantages; they have lower levels of control and of support. Either control or support could compensate for the absence of the other, but poorly educated people lack both" (Ross & Mirowsky, 1989, p. 219). One's educational level also influences employment status (Costello, 1991). According to Kohn, Dohrenwend, and Mirotzinik (1998) employment was related in an inverse manner to prevalence rates.

According to Friedman (1966; cited in Friis & Nanjundappa, 1986), lack of an education and skills are severe employment impediments in competitive employment markets.

In summary, it appears that education has an important role to play in mental health. Education allows better choice of employment opportunities and leads to higher income. Given the impact of education on employment and income it is clear that an education cut short can lead to significant stressors like unemployment and financial hardship. In turn, financial stressors and lack of meaningful activity contribute to negative mental health outcomes. Lower levels of education, and/or low social class force poorer people to live in more stressful environments (Baum, Garofalo, & Yali, 1999).

Employment

Work, according to Szasz (1974), is the best general panacea known to medical science (Lloyd, 1986) and the relationship between employment status and psychological well-being is complex (Roberts et al. 1982). Employment has been found to be positively related to emotional well-being (Clark et al. 2003), and unemployment has been found to be related to increased risk of suicide (Kposowa, 2000). According to Murray-Parkes (1979), when working was necessary just to avoid starvation, work functioned as a means of obtaining food and shelter. However, when social policies provided food for unemployed individuals, it became apparent that work served other functions. Apart from meeting physical needs, work fulfills a number of important social and psychological needs (Jahoda, 1982). Employment seems to offer the individual a means of structuring time purposefully, allows for personal development, and provides opportunities for social

interaction (Lloyd, 1986). Social roles associated with employment allow people to feel worthwhile (Bolton & Oatley, 1987). The status ascribed to particular forms of employment provides the individual with a self-image, self-esteem, and a way of relating to society (Lloyd, 1986). Employment is an important social marker and most individuals experiencing involuntary unemployment are significantly influenced (Brenner & Mooney, 1983). According to Ross and Mirovsky (1993), it is as simple as employed versus not employed, the employed have better health than the unemployed (Adler & Newman, 2002). Increased levels of financial problems, due to job loss, temporarily increase psychological distress Gorgievski-Duijvesteijn, Bakker, Schaufeli, & van der Heiden, 2005).

Unemployment, especially for prolonged periods, has been found to be a severe stressful life event (Pearlin, 1982) that threatens physical security, social identity (Weber, 1946), and self-esteem (Dressler, 1986). Unemployed individuals had lower psychological well-being (Viswesvaran, Sanchez, & Fisher, 1999) and they experience financial strain (Creed & Macintyre, 2001; Vinokur & Schul, 2002). According to Price et al. (1998), job loss is a stressful event that leads to secondary stressors. This cascade of secondary stressors can present a greater risk to mental health as the job loss itself (Howe, Lockshin, & Caplan, 2004). According to Kinicki, Prussia, and McKeen-Ryan (2000), a displaced worker's social resources were depleted during periods of unemployment. According to Ennis, Hobfoll, and Schroder (2000), negative changes in material resources was related to increased depression in inner-city women (Gallo & Mathews, 2003).

Poverty, low SES, and unemployment have been found to be related to higher

stress (Brown et al. 1975; Eaton, 1974; Lindblad-Goldberg et al. 1988; Makosky, 1982; McLanahan, 1983; Miller, 1988). Depression has been reported to be more prevalent among long-time unemployed (Stankunas, Kalediene, Starkuviene, & Kapustinskiene, 2006). Joblessness, by itself, creates stress that can aggravate or cause health problems (B. S. Dohrenwend et al. 1978). Lack of health insurance, unemployment, economic distress, and being forced to live on minimal disability income are stressful for those with health problems (Charlifue & Gerhart, 1991). Having higher education and higher income have been reported to reduce the risk of depression for the unemployed (Stankunas, Kalediene, Starkuviene, & Kapustinskiene, 2006)."Mental disorders on average were 2.6 times more prevalent among individuals in the lowest than in the highest socioeconomic stratum" (Kohn, Dohrenwend, & Mirotzinik, 1998, p. 235). "The effects of an equivalent amount of stress are greater for the lower class than for the middle and upper class" (Eaton, 1974, p. 29). This makes sense when one considers the level of income required to meet basic needs. Those from lower SES have lower absolute levels of economic resources leading to less discretionary funds. This situation leaves those from lower SES groups unable to absorb additional unplanned expenses that are encountered.

In general populations, unemployment has been directly associated with increases in psychiatric admissions (Ahr et al. 1981), self-reported perceived physical illness, anger, paranoia, drinking problems, family conflict (Kirsh, 1983), general psychological distress (Brenner & Starrin, 1988), anxiety (Cobb & Kasl, 1977; cited in Hamilton, 1993), poorer levels and quality of support (Atkinson et al. 1986), diminished network resources (Anfinson & Kathol, 1992; Arling, 1987; Fisher, 1982; Hall, Williams, & Greenberg,

1985; Milburn, Brown, & Gary, 1984; Stephens, 1978), and depressive symptomatology (Friis & Nanjundappa, 1986; Milburn, Brown, & Gary, 1984). Also, in patients with depressive disorders incomplete anger suppression is likely to be associated with increased depression and somatic symptoms (Koh, Kim, Kim, & Park, 2005). According to Madonia (1983), unemployed individuals who are used to the daily structure and psychological gratifications of employment may feel insecure, inadequate, and display a decrease in autonomous behaviour and an increase in dependency. According to Bond and Feather (1988), unemployed individuals who reported higher levels of daily structure and purpose in their use of time had higher self-esteem and fewer depressive symptoms.

Issues around the relationship of employment to health are difficult to disentangle. The concern is whether unemployment inversely influence health or are individuals with lower levels of health more likely to become unemployed (Kasl, Rodriguez, & Lasch, 1998)? According to the above authors, the evidence supports the existence of both processes. Depression, according to Perrucci et al. (1988), can be a long lasting mental health consequence of unemployment (Hamilton et al. 1993). Unemployed individuals, even after controlling for other sociodemographic variables, were found to have significantly elevated rates of depressive symptoms (Dressler, 1986). According to Madonia (1983), debilitating psychiatric symptomatology resulting from unemployment may require crisis intervention and short-term psychotherapy. Yet, an individual's psychological response to unemployment is not uniform (Liem & Rayman, 1982).

Psychiatric patients are often unemployed and, as discussed previously, unemployment and LOS are positively related. There is no ambiguity about this

relationship in the literature. The problem resides in the causal attribution attached to the findings. There are two difficulties related to explanations of this relationship. The first difficulty considers the fact that unemployment and increased LOSs can both be an indication of the severity of pathology. This results in the relationship between employment and hospitalization being confounded by severity of pathology. The second difficulty is related to the direction of causation. Did the unemployment cause the inpatient stay to increase or did the inpatient stay create the deficit in employment? Regardless of direction of causation, many mentally ill patients have large gaps in their employment histories that they need to explain to prospective employers, making it very difficult for them to find employment.

The experience of unemployment can be modified by other depression related variables; however, unemployment also has an independent effect on depressive symptoms (Dressler, 1986). According to Dressler, having a smaller income is related to higher levels of depressive symptomatology. Depressive symptoms are higher among the unemployed, those with less education, those who perceive themselves as beset by more chronic non-economic life changes (Dressler, 1986), and those with fewer financial and social support resources (Brown & Lawrence, 1985). In fact, the impacts of other non-economic stressful life events are exacerbated as the person's ability to adjust is compromised by unemployment (Dressler, 1986). Emotional support from family and friends has been found to help older adults cope more effectively with the deleterious effects of financial strain (Krause, 2005).

The relationship between social support and employment is complex. Being

employed increases one's social support since employed individuals usually have larger social networks (Atkinson et al. 1986) and interact with more people on a daily basis (Hammer et al. 1982). As far back as the Great Depression, unemployment has been found to lead to increases in the social isolation of the non-working individual (Bakke, 1933, cited in Feather, 1989). According to Atkinson et al. (1986), the social networks of unemployed and re-employed individuals tended to be in 'flux' and unemployed individuals report experiencing lower levels of support over time. Social support helps to moderate the effects of unemployment on mental health (Gore, 1978). The lack or loss of employment can cause depression, especially when no alternative source of social interaction and support are available for the unemployed individual (Bolton & Oatley, 1987). According to Cohen and Willis (1985), social support buffers the negative psychological effects of unemployment by providing protection from the total loss of social interaction opportunities that are important for an individual's sense of worth. According to Bolton and Oatley (1987), low levels of social support act both as an independent stressor, and via statistical interaction, to make people vulnerable to adversity (see: Bolton & Oatley, 1987; Cohen & Willis, 1985; Leavy, 1983; Oatley & Bolton, 1985).

The literature on unemployment and its relationship to social support and psychiatric symptomatology is often confounded by the non-recursive relationships between the variables involved, leading to a less powerful test of theories with standard statistical methodologies (Cohen & Willis, 1985). For example, stressors, like unemployment, and potential supports are dependent; "individuals with poor social

support may have higher psychological symptom levels, not because they had limited support for coping with stress, but because unemployment produced both elevated symptoms and lowered support" (Atkinson et al. 1986, p. 327). According to Hall and Johnson (1988), being subjected to a stressful life event and low social support greatly increases the risk of depression, especially for the unemployed. Little literature is available concerning the impact of being unemployed in populations that are most likely at risk of experiencing the event. Much of the literature focuses on the effects of unemployment in general populations. Few studies have dealt with the effects of work status on those with psychiatric impairments (Lindsey & Ozawa, 1979).

Employment, hospitalisation, and psychopathology are interrelated (Allen et al. 1992; Westermeyer & Harrow, 1988). SES and employment are related to one another and both are related to inpatient treatment variables. Unemployment places the majority of mentally ill patients in lower SES groups (Aneshensel et al.1981). The population rate of employment has been found to be negatively related to the rate of hospitalisation (Vetter & Koller, 1996). The relationship between employment and inpatient treatment is difficult to determine, given that both variables are near proxy measures for patient pathology, making causal statements about the impact of employment highly suspect. Direct causal statements are not warranted. Those who are admitted to hospital cannot be at work and those with more pathology are likely not working (Allen et al. 1992). The unemployed have been found to be more likely to be admitted for inpatient treatment (Burgess et al. 1992; Hall & Royse, 1987; Hirsch, 1988; Hobfoll & Jackson, 1991; McCarthy, 1991; Sorensen & Mors, 1993; Woogh, 1986), have greater LOSs (Carpenter

& Strauss, 1991; Rud & Noreik, 1982; Strauss & Carpenter, 1977), and less favourable outcomes or treatment success (Steinhart & Priebe, 1992; Tohen, Waternaux, & Tsuang, 1990). Those with greater LOSs are unlikely to have worked because you cannot spend a significant amount of time in inpatient treatment and be collecting a pay cheque. Utilization of mental health services has been found to increase after unemployment levels increase (Brenner, 1973; Dear, Clark, & Clark, 1979). There is an increase in inpatient admissions months after the unemployment rates increase, and unemployment rates correlate with first admissions and readmissions (Ahr, Gorodezky, & Cho, 1981). Pearlin et al. (1981) believe that the relationship between unemployment and psychiatric distress is mediated by financial resources. Those who are from a higher household income bracket are less influenced by their unemployment since their partners or relatives have resources to help the unemployed mentally ill. Other researchers have reported that employment status was not useful in predicting outcome (Tsoi & Wong, 1991), admission (Castle et al. 1994), or readmission (Buell & Anthony, 1975; Vetter & Koller, 1996). Psychiatric distress and symptomatology have been reported to be negatively related to employment (Gurel & Lorei, 1972; Strauss & Carpenter, 1972). This may be a result of too much unstructured time fewer depressive symptoms are reported when time is purposefully structured (Bond & Feather, 1988; Feather & Bond, 1983; Feather, 1989).

Extended hospitalisations have also been reported to lead to stagnation of social skills, reducing the patient's capacity to fulfill role requirements (Hoffmann & Mastrianni, 1993), and diminishing employment skills (Freeman, 1980). Existing psychopathological impairment can impact psychosocial role functioning making these

patients unable to sustain employment and familial roles which usually results in lower SES (Aneshensel et al. 1981).

Income and Financial Adequacy

Studies using North American populations usually find an inverse relationship between socioeconomic (SES) and psychopathology (Kessler, 1982; Kessler & Cleary, 1980). SES can be related to psychological distress (Dohrenwend, 1969), length of psychiatric hospital stays (Mai et al. 1993), and treatment of psychiatric symptomatology (i.e., whether psychiatric hospitalization is recommended over community-based treatment (Mollica & Milic, 1986). Yet, some have reported that SES does not predict recovery from psychiatric symptomatology (Keitner et al. 1992). Nonmedical factors such as income can explain the level and distribution of health in populations (Benzeval & Judge, 2001; Berkman, 1995; Lavis et al. 2001). Economic disadvantages are important components of any mental health research since these disadvantages impact all facets of a patient's environment (Feather, 1989). The demands of and resources available to positions in the social hierarchy create differences in distress levels (Pearlin et al. 1981) and financial stresses are positively related to psychological distress, including depressive symptomatology (Feather, 1989). "Higher SES communities appear to have fewer hazards or privations, more support, and to be able to afford more options for coping with problems when they occur" (Baum, Garofalo, & Yali, 1999, p. 4). According to Dooley and Catalano (1980), decrements in SES constitute stressful life events and have been reported to be related to the appearance of psychological symptoms.

SES is a broad construct that includes education, income, occupation, and area of residence. SES and the existence of economic hardship are usually determined by an individual's available financial resources. Financial stresses are positively related to psychological distress, including depressive symptomatology (Feather, 1989)."Family income, from whatever source--a spouse's earnings, social security, or public assistance--is important to psychological well-being because it allows one to pay the bills, feed, clothe, and care for the health of one's family" (Ross & Huber, 1985, p. 316). Lack of financial resources reduces an individual's ability to meet basic needs, thereby influencing psychological health and adjustment (Feather, 1989). Those who were exposed to more episodes of economic hardship were at greater risk of depression (Baum & Posluszny, 1999). Economically disadvantaged individuals, below the poverty level, have been found to have significantly higher rates of depressive symptoms and more frequent lifetime diagnoses of major unipolar depression (Narrow et al. 1990). Low-income environments have been found to be particularly 'toxic' for psychiatric patients (McGlashan, 1991).

According to Maffeo, Ford, and Lavin (1990), family and personal income both influence depressive symptomatology, with low family income creating financial stress and low personal income acting negatively on self-esteem. Personal earnings symbolize worth in North American society, making an individual's income important for self-esteem (Ross & Huber, 1985). Although the income from unemployment compensation (Ross & Huber, 1985) and welfare (Serban & Thomas, 1974) is important to pay the most basic of expenses, this kind of income does nothing for an individual's

self-worth. In fact, welfare creates a psychological factor that undermines psychiatric care and promotes further deterioration of the recipient's status and self-concept (Serban & Thomas, 1974). Older individuals who receive public assistance benefits tend to get less social support over time and public policymakers, should consider developing interventions that address the wider psychosocial problems associated with receiving public assistance and recognize that participating in public assistance programs may disrupt the functioning of informal social networks (Krause & Shaw, 2002).

Income, from whatever source, interacts with unemployment, education, and social support to contribute to mental health outcomes. Studies of mental health problems in the unemployed often underestimate the importance of financial concerns (Rodgers, 1991), yet financial problems (e.g., household income) impact on the relationship between unemployment and depressive symptoms (Frese, 1987). According to Feather (1989), economic hardship measures are needed when the psychological effects of unemployment are evaluated since the level of financial problems is much higher in individuals without employment (Frese & Mohr, 1987). Chronically unemployed individuals face economic hardship due to a lack of financial support leading to a loss of control and subsequent feelings of helplessness and depression (Ross & Huber, 1985).

Epidemiological literature has reported a direct relationship between being poor and the need for mental health services (Dohrenwend & Dohrenwend, 1969; Hall & Royse, 1987; Longest et al. 1979; White, 1986; Zautra & Simons, 1978). Neighbourhood income is an ecological variable and is related to household income. Individuals in lower SES neighbourhoods can face stress due to the neighbourhood characteristics. Treatment

prevalence for psychiatric difficulties increases as neighbourhood income levels reduce, especially for psychotic disorders, and as people become more disabled they often are forced to move to even lower SES neighbourhoods (Dohrenwend, 1998; Tataryn, Mustard, & Derksen, 1994). A Canadian study found that lower-SES Canadians used primary care more frequently than those from higher SES groups (Dunlop, Coyte, & McIssac, 2000). Increases in utilization of mental health care resources have been found to be related to low SES neighbourhoods (Tataryn et al. 1994), low family income, lack of education, and unemployment (Hall & Royse, 1987; Srole et al. 1962; Zautra & Simons, 1978).

The incidence of mental illness has been found to be negatively related to income and positively related to demand for services (Jackson et al. 1982; Longest, Kovan, & Tweed, 1979; White, 1986). In the United States individuals from higher SES groups have access to more comprehensive medical insurance coverage (Dohrenwend & Dohrenwend, 1969; Kessler, 1982). Income influences whether a patient is likely to see a psychiatrist since these professionals have been found to work primarily "with middle-to upper-socioeconomic-class adults suffering from schizophrenia, affective disorders, and psychoneuroses" (Redlich & Kellert, 1978, p.26). In Canada there are no differences in public medical coverage. However, there are differences in private insurance coverage. Those with additional (purchased by insurance) medical coverage have greater access to additional treatment options, (for example, seeing a psychologist). Those from higher household incomes are more likely to be able to afford and benefit from additional insurance. In Canada, it seems likely and plausible that income inequality is associated

with health (Statistics Canada, 1997)

As described above, components of SES include education, family income, and employment. A high positive correlation exists between education and income (Brown & Lawrence, 1985) and low levels of both variables are associated with an increased risk for psychiatric symptomatology (Ross & Mirowsky, 1989). Patients who lived in a home that was supported by income from employment were less likely to be readmitted (Franklin et al. 1975), while those living in a home supported by social assistance were more likely to be admitted (Ahr et al. 1981). Poverty synergistically interacts with a lack of education, creating more economic hardship; low education translates into low earnings and low education increases the difficulties of dealing with low income (Ross & Huber, 1985)

SES variables, like income and education have also been found to impact mortality (Berkman & Syme, 1979). According to Haughton Mc Neill, Kreuter, and Subramanian (2006), low SES, and significant income inequality are related to emotional health. Chronic economic difficulties have been found to be extremely important factors in the etiology of depression (Ross & Huber, 1985). Pearlin et al. (1981) proposed that chronic economic strain may help in explaining the relationship between income and depression; as income decreases economic hardship increases and, as a result, depression also worsens. However, the correlation between income and depressive symptomatology is problematic; most of the difficulty arises from the lack of clear distinctions between subjective dissatisfaction and objective financial measures (Carpiniello et al. 1989). According to Marans and Rogers, objective status characteristics, like family income, are insufficient to explain assessments of one's environment, attitudes, or satisfaction with

various dimensions of life, including financial status (as in Ross & Huber, 1985). While objective measures of family income have a large impact on perceived economic hardship, the perceptions are not a one-to-one reflection of income (Ross & Huber, 1985).

According to Carpiniello, Catra, and Rudas (1989), depressive symptomatology can aggravate the extent of a client's dissatisfaction with financial status (financial adequacy perception) and financial adequacy perceptions were more likely to be driving the depressive symptomatology. These two factors are likely to be circular in their effect on the poorer and more afflicted. Poorer individuals are more likely to suffer depressive symptomatology resulting in lost capacity to work, reducing income, creating financial strain, diminishing financial adequacy perceptions, leading to more depressive symptomatology and the incapacity for employment. The findings of a significant review of the literature, Diener, Suh, Lucas, and Smith (1999), concluded that wealthier people were consistently found to be happier than their poorer counterparts and past researchers have also argued that subjective well-being is negatively related to the importance a person attaches to money (Srivastava, Locke, & Bartol, 2001).

Loss of income, resulting from unemployment, causes disruptions in the family and non-familial social relationships (Fryer, 1988), reduces social interaction (Aiken, Ferman, & Sheppard, 1968), and eliminates sources of recreation and other stress-reducing activities (Madonia, 1983). The unemployed spend progressively less time in activities that require spending money, leading to an increase in passive unstructured use of time and lower scores on indicators of psychological health and adjustment (Feather, 1989). Financial difficulties and poverty lead to a reduction of the

scope of one's social life facilitating the development of depression (Frese, 1987). Those who are unemployed tend to have poorer levels and quality of social support (Atkinson et al. 1986), and low income has been found to be related to low network resources (Arling, 1987; Fisher, 1982; Hall, Williams, & Greenberg, 1985; Milburn, Brown, & Gary, 1984; Pini et al. 1995; Stephens, 1978) as well as increased levels of depressive symptomatology (Milburn, Brown, & Gary, 1984). Males and females have been found to both be equally able to access social support at work (Baruch-Feldman, Brondolo, Ben-Dayan, Schwartz, 2002). However, females appear to benefit more from receiving social support than males (Perrewe & Carlson, 2002).

According to Auslander (1988), few existing research studies focus on the health effects of social networks among the poor; yet both poverty and, as cited previously, poor social support have been shown to influence health, and the poor have been shown to have fewer network resources. According to Gurung, Taylor, and Seeman (2003), people with larger networks were more likely to report increases in emotional support. Overall, economically disadvantaged individuals have been found to have smaller social networks (Fisher, 1982), fewer friends (Auslander, 1988), poorer social adjustment (Garvin, Kalter, & Hansell, 1993), and less frequent contact with network members (Rosenberg, 1970). The "poor who had no ties or saw them infrequently were in worse health than those who saw their ties more frequently" (Auslander, 1988, p. 206).

A number of SES, social role, and sociodemographic variables interact to impact one's well-being and treatment by the mental health care system. Many economically related variables may impact directly or interact to impact the likelihood of inpatient

treatment. SES variables, such as median family income and percentage of female-headed households, have been found to be negatively related to admissions (Hall & Royse, 1987). Admissions and poverty rates are positively related (Errera et al. 1963; Hollingshead, 1957; Shader et al. 1969). In some American studies, low-SES patients were reported to be more likely to be admitted for inpatient treatment (Gerson & Bassuk, 1980; Shader et al. 1969). In a Canadian study, those hospitalised with both psychotic and non-psychotic disorders were more likely to live in the poorest 20% of neighbourhoods (Tataryn et al. 1994). This is likely due to the psychiatric patients' lack of economic resources. In Britain, social deprivation of lower classes has also been found to be positively related to high admission levels (Thornicroft, 1991; Thornicroft, Margolius, & Jones, 1992).

Re-hospitalisation has been found to be related to low SES (Schweitzer & Kierszenbaum, 1978), poverty (McCartney, 1991), and crowding/household density, including the density of single resident occupancy (SRO) hotel facilities (Schweitzer & Kierszenbaum, 1978). Low SES patients have been found to be more likely to return to the hospital more often (Ahr et al. 1981). Chronic stressors, such as lack of employment and inadequate income, force the mentally ill into a state of socio-psychological-economic dependency which contributes to readmission and increased LOS (Franklin, Kittredge, & Thrasher, 1975).

According to Fraser, Nelson, and Rivard (1997), much of mental health resources have been targeted for inpatient treatment/substitute care, social control, or confinement of the mentally ill from the healthy. Lengths of stay (LOS) in a psychiatric facility have been reported to be influenced by economic variables (Cyr & Haley 1983; Laessle,

Yassouridis, & Pfister, 1988; Mai, et al. 1993). Patients from higher SES groups have been found to have reduced LOSs (Laessle et al. 1988). In a Canadian study, patients from lower SES groups were also found to have longer LOSs (Mai et al. 1993). Many patients have been maintained in inpatient treatment because of variables related to low SES and limited treatment and discharge options (Laessle et al. 1988; Mai et al. 1993; Mezzick & Coffman, 1985). Social factors beyond the "control of the psychiatrist are major factors prolonging hospital stays in a large minority of patients" (Mai et al. 1993, p. 44).

According to some authors, depression recovery rates have been found not to be impacted by SES (Keitner et al. 1992). Yet, other researchers report that high SES has been found to be related to better outcomes in both mania and depression (Harroe et al. 1990; O'Connell et al. 1991; Strakowski et al. 1998; Tohen et al. 1990). Recovery times have been found to be longer for depressed patients with lower family incomes (Keller et al. 1982). And, lower income levels have been reported to be related to lower well-being for patients (Sylvester & Bean, 1989). Recent Canadian, British, and American literature indicates that unemployment and high population density are related to increased suicide risk (Balon, 1987; Hawton, 1987; Hellon & Solomon, 1980; Holinger, Offer, & Zola, 1988; Hopkins, 1971; Murphy & Wetzel, 1980), and suicide has also been found to be related to economic stressors (Rich et al. 1991). Patients with mania and depression with poor occupational status have also been found to have poorer outcomes (Gitlin et al. 1995; Tohen, Waternaux, & Tsuang, 1990). An increased risk of mortality has been reported from all causes among lower SES populations (Antonovsky, 1967; Dayal et al.

1984; Haan et al. 1987; Morris, 1979). The increased mortality rates among psychiatric patients could be partially due to the poverty conditions they endure (Segal & Kotler, 1991). Poor health habits may contribute to increased mortality (Berkman & Breslow, 1983; House et al. 1982; Wiley & Camacho, 1980), and are common in the chronically mentally ill, especially those with schizophrenia (Farmer, 1987; Harris, 1988; Lieberman & Test, 1987; Materson & O'Shea, 1984).

In summary, although there are a few exceptions, the preponderance of the literature indicates that low SES factors are related to the outcomes for patients with psychiatric disabilities. Lower SES is related to poorer outcomes, higher pathology rates and higher stress rates. Lack of employment in the mentally ill results in below poverty line existence. Low levels of income (insufficient to pay basic expenses of life) place significant stress on patients, resulting in significant financial stressors.

Hospitalization Literature

In developed countries, psychiatric hospitalization has historically been the main form of treatment for the mentally ill with major mental disorders (Reynolds & Hoult, 1984). Following the advent of de-institutionalisation and community care, many believe that there were increases in criminal offending by the seriously mentally ill (Hall 1998; Mullen et al. 2000). However, prolonged psychiatric hospitalization itself can lead to negative consequences, including: dependence and helplessness (Decker, 1972), stagnation of employment and social skills, internalization of the patient role, leading to chronicity (Freeman, 1980), stigmatization leading to community members distancing themselves from the ex-patient (Link, 1982), and difficulty in assuming roles previously held in one's social environment (Hoffmann & Mastrianni, 1993). Historically, in Canada, D'Arcy (1978) proposed that earlier intervention would increase the possibility of retaining individuals with psychiatric disorders in the community. Stigma has been defined as being socially discredited or being perceived as flawed based on a personal characteristic (Blaine, 2000). Self perceived stigma have been found to be one of the most important factors influencing people to avoid or delay seeking treatment for mental illness (Cooper, Corrigan, & Watson, 2003; Barney, Griffiths, Jorm, & Christiansen, 2006; Nolan & Badger, 2006; Schraufnagel, Wagner, Miranda, & Roy-Burne, 2006). Mental illness has been significantly impacted by the stigma of being mentally ill and there is an increased understanding that this situation contributes to the undermining of social connectedness, social support, and opportunities for recovery (Satcher, 1999; Wahl, 1999). In fact, according Klonoff et al. (1999), Sanders et al. (2004), and Utsey et

al. (2000), there is a connection between discrimination on the basis of stigma related to mental illness and increased symptomatology and stress reactions and perceived social rejection (Thompson, Sanders, Noel, & Campbell, 2004). Mental illness, starting at the time of first contact for service has been found to correlate with an increase in the report of discrimination by patients (Sanders, Thompson, Noel, & Campbell 2004).

Disability onset may cause a period of adjustment or disruption, promoting feelings of worthlessness or hopelessness that may fuel depressive symptoms (Boerner, 2004; Bruce, 2001; Taylor & Lynch, 2004). There is a developing literature evaluating the impact of psychosocial factors in relation to disability (see Dunn, 2000). The disabled often display a tendency to social isolation (Simmonds et al. 1996). According to Chwalisz and Vaux (2000), there is evidence that social support is inversely associated with coping, depression, adjustment, self-esteem for individuals with disabling conditions. In fact, according to WHO (2000), unipolar depression is one of the leading causes of disability (Murray & Lopez, 1997). Social support has been purported to mediate the relationship between disability and depressive symptoms (Taylor & Lynch, 2004). According to Willoughby, Brown, King, Specht, and Smith (2003), social support at crucial transitions in life is very important (King, Willoughby, Specht, & Brown, 2006). Specifically, a persons perception of social support has been shown to attenuate the effect of disability on depressive symptoms (Allen, Ciambrone, & Welch 2000; Jang et al. 2000). Increased disability is accompanied by more received social support (Mendes et al. 2001). However, according to Jang et al. (2002), received social support did not ameliorated disability but is did impact the effects of disability on mental health.

According to Taylor & Lynch, 2004, increasing trajectories of depression tend to have increasing trajectories of disability and this is consistent with previous research by Bruce (2001).

With the arrival of the community mental health movement, the delivery of mental health services has changed (D'Arcy, 1978). The goals of this movement were to benefit psychiatric clients by reducing the negative effects of prolonged psychiatric confinement ('institutionalism'), providing psychiatric health services in a more normal environment (Hoffmann & Mastrianni, 1993), and shortening psychiatric hospital stays (D'Arcy, 1978; Mai et al. 1993). This movement has resulted in less restrictive, short-term, and crisis intervention style treatment options (Caton & Gralnick, 1987). Yet, psychiatric hospitalization remains a large part of the appropriate medical treatment of individuals with chronic psychiatric illnesses (Solomon, Davis, & Gordon, 1984). However, inpatient treatment in Canada is costly (Russell et al. 1996). With the decline in the LOS came a reduction in available psychiatric beds, an increase in alternative day treatment programs (Russell et al. 1996), increases in tertiary care programs like Assertive Community Treatment programs (Wasylenki et al. 2000), an increase in admissions, an increase in readmissions, an increase in the number of patients seen as outpatients, and the opening of new decentralized facilities (D'Arcy, 1978). Physicians were under pressure to minimize bed utilization and LOS (Russell et al., 1996). In practice, "partial hospitalization programs have focussed largely on the rehabilitation of patients with chronic psychiatric disorders" (Russell et al. 1996, pp. 629-630). Psychosocial problems have been found to influence rehabilitation (see, e.g., Brewer et al.

2000). It is important that psychosocial programs aimed at providing support services be included in the rehabilitation programs focussing on preventing psychosocial deterioration and thereby reduce activity limitations (Eide & Roysamb, 2002). There is a group of mental health clients that will always need intensive care housing arrangements and if community beds are not available these patients will remain in the acute care inpatient environment, reducing available beds for those in more need (Smyth et al.1997).

According to Mosher (1984), hospital-based treatment is expensive and traditional hospital based treatment strategies are perpetuated despite the existence of more effective, feasible, and less expensive community-based alternatives. Hospitalized psychiatric clients consume large quantities of health care resources. According to Bengelsdorf, Church, Kaye, Orlowski, and Alden (1993), whenever possible, avoiding hospital admissions by substituting prompt, intensive outpatient treatment can save both the human and financial costs of extended hospital stays. Yet, these authors warn against cost-cutting at the expense of humane and safe care for the more seriously ill. Kiesler (1992) goes so far as to predict the failure of American mental health policy because of its continued emphasis on hospital care.

Explaining the use of mental health resources is important for ensuring the availability of appropriate treatment for psychiatric clients and controlling medical expenses. Usage of mental health resources is influenced not only by client status, but many administrative, political, social, and legal factors (Lewis & Hugi, 1981). Since short hospital stays were found neither to increase the likelihood of readmission (Baker & Rochon, 1989) nor to deteriorate the quality of care received (Schwarz, 1988), changes in

social policy and court rulings have aimed at, and resulted in, shorter stays in inpatient psychiatric facilities (Mai et al. 1993).

The majority of literature on the factors influencing the usage of mental health resources focuses on readmission rates. More frequent admissions obviously lead to an increase in total bed days and resource usage; however, admission rates do not, by themselves, portray the entire picture of mental health service utilization. Clients with longer hospital durations are more likely to be rehospitalized in the future (Davis, 1985).

Mental health service usage, measured as: length of psychiatric stay(s), admission rates, and costs, is influenced by numerous variables, including the client's mental status or symptomatology (Ware et al. 1984; cited in Sherbourne, 1988). Depression may impair motivation to recover and to comply with medical treatments, leading to slower recovery and poorer outcomes (Johnson et al.1999; Koenig & Kuchibhatla, 1998; Penninx et al. 1998; Schroevers, Ranchor, & Sanderman, 2003). However, the psychiatric client's symptoms (Keitner et al. 1992) and diagnosis related group (DRG) are poor predictors of resource use (Essock & Norquist, 1988; Mattes, 1987). According to Mai et al. (1993), age, gender, marital status, the presence of concomitant physical illness, previous psychiatric admissions, involuntary admission, and social functioning have all been shown to influence average length of psychiatric hospital stays. The young have been found to return to hospital more frequently (Daniels et al. 1998; Haywood et al. 1995). Younger age at illness onset is often regarded as an indication of more serious pathology (Goodwin & Ghaemi, 1998; Wehr et al. 1998) and better outcomes were found to be related to an older age of onset (Keck et al. 1998). Although gender and age correlate

with length of hospital stays (Taube et al. 1984), by themselves, they do not significantly improve the prediction of resource use (Gordon et al.1985). According to Keitner et al. (1992), a patient's age, education, and SES do not help in predicting recovery rates, either. The literature also indicates that patient age, by itself, does not predict resource consumption, however, other researchers have found that being single predicted increased admission (Korkeila et al. 1995).

Length of stay in a psychiatric facility is influenced by economic, demographic and social characteristics (Laessle et al. 1988; Mai et al. 1993). When economic conditions are poor, patients return to the hospital more frequently (Ahr et al. 1981). According to Allen et al. (1992), longer stays in a psychiatric facility are also associated with poor pre-morbid functioning, a more chronic course, and less outside support. A coexisting substance-related disorder has been found to be one of the best predictors of readmission (Haywood et al. 1995). Unipolar depressive disorders with psychotic symptom expression are readmitted more frequently and readmission for these patients increased with substance abuse, medication non-compliance, and comorbid personality disorders (Haywood et al. 1995). Patients of all ages who are afflicted with medical or psychiatric comorbidities are heavy utilizers of health care services (Draper & Luscombe, 1998). Concurrent medical illness has been previously reported to be associated with poorer response to treatment and greater chronicity (Parks & Josef, 1997). Untreated medical illness comorbidity has been partially blamed for higher mortality rates in the mentally ill (Felker, Yazel, & Short, 1996). Substance abuse was found to exacerbate psychiatric distress and suicidality, impair functioning, and reduce self-control even after

controlling for the patient's depressive and anxiety symptomatology (Jordan et al. 1996). American clients who are repeatedly admitted to psychiatric facilities appear to suffer from both social and economic problems (Munves et al. 1983), substance abuse (Haywood et al. 1995) and medical comorbidity (Felker et al. 1996; Parks & Josef, 1997), and these types of factors, in a Canadian teaching facility, were found to contribute to extended stays (Mai et al. 1993). Comorbid substance abuse morbidity has been found to be related to poor outcome (van Gorp et al. 1998).

Social support factors and life stress events influence psychiatric symptomatology and service usage. However, the relationships among social support, life stress, and mental health resource use depend upon the type of stress assessed and how social support is evaluated (Sherbourne, 1988). For example, social support has many components, including perception of support and/or a number of contacts. Stress has also been operationally defined many different ways. For example, stress has been defined as financial stress and at other times is defined as emotional stress. Therefore, researchers need to evaluate different types of stressors and to determine which components of social support predict mental health service usage. Also, Cohen and Sokolovsky (1978) believed that a stronger focus should be placed on separating out the effects of qualitative versus quantitative dimensions of the support concept.

A positive association between isolation and psychiatric symptomatology exists (Wilkinson, 1975). Stress buffering models have been presented in many different versions (Lakey & Cohen, 2000). According to Sherbourne (1988), regardless of life stress events, the presence of friends and relatives reduces the use of mental health

services. Perceived social support has been claimed to buffer the effects of stressful encounters (Cohen et al. 2000). Perceived social support has been reported to be negatively related to depressive tendencies (Heponiemi, et al. 2006; Maher, Mora, & Leventhal, 2006). Whether social support works directly on mental health service consumption, or indirectly through a buffering mechanism, is not clear. However, recent work by Burton, Stice, and Seely, (2004), Ritter, Hobfoll, Lavin, Cameron, & Hulsizer, (2000), Wade and Kendler (2000), and Wildes et al. (2002), do not support the stress-buffering effect of social support on depression (Heponiemi et al. 2006). Sherbourne (1988) found chronic stressors were important predictors of service use, but acute stressors neither predicted service use, nor were reactions to acute stressors buffered by the available social support. These results indicate that it may be the chronicity of the stressor that is the important factor in the use of mental health services.

Lack of social support, inadequate income, and unemployment are chronic stressors that have been found to contribute to both readmission rates and extended hospital stays. Patients with more severe and persistent difficulties, and higher impairments in self care are at higher risk for poor outcomes resulting in readmission (Lyons et al. 1997). Understanding the relationship between readmission and outcome is important, especially with more popular short LOSs becoming the norm (Lyons et al. 1997). Short LOSs have been found to be effective in reducing acute symptomatology (Lyons et al. 1997)."Those who are rehospitalized are likely to be in a state of socio-psychological-economic dependency, have poor interpersonal relationships with significant others, lack meaningful social outlets, and have poor self-images"(Franklin,

Kittredge, & Thrasher, 1975, p. 751). Clients with inadequate social support upon discharge are admitted more often and stay in the psychiatric facility longer (Holsten & D'Elia, 1985). Readmitted patients also have fewer visitors (Franklin et al., 1975).

According to Allen et al. (1992), the relationship between unemployment and psychiatric hospitalization is hard to disentangle. Patients who have had to spend a lot of time in the hospital are likely to have poor employment records. As a result, they are also likely to have difficulty finding employment (Allen et al. 1992). Clients who receive income from employment (either theirs' or family members') were less likely to be readmitted (Franklin et al. 1975). Subjective well-being in the elderly is related inversely with financial resources (Pinquart & Sorensen, 2000). Individuals receiving financial support from social assistance (Ahr et al. 1981) or who were young (Davis, 1985), or unemployed (Woogh, 1986) were more likely to be readmitted. Females have also been found to have longer hospital stays (Daniels et al. 1998; Dewees et al. 1996). Lower levels of education are associated positively with psychiatric admission rates (Levav & Arnon, 1976). However, contrary results indicated that, overall, hospitalized or readmitted clients are not likely to be from among the less educated (Woogh, 1986).

In summary, inpatient treatment is expensive and there are other options that can deliver necessary treatment in an alternative community based approach. In Canada, inpatient treatment is influenced by many administrative, political, social, and legal factors (Lewis & Hugi, 1981). Clients have the same satisfaction levels whether they are treated as inpatient or outpatient treatment options (Hendryx & Rohland, 1997). In general, inpatient treatment has not been found to provide a better outcome for patients;

outpatient treatment was found to provide equivalent or improved outcomes (Hendryx & Rohland, 1997). However, the literature is reasonably clear that those who are admitted for inpatient treatment tend to suffer from a) poor social support, b) economic difficulties, c) more severe pathology, d) poor premorbid functioning, e) significant stress, and f) unemployment difficulties.

A Little About Causation

Before we can truly comprehend the complexity of this literature a very brief discussion of the subject of causality is necessary. Mackie (1974) proposes that the essential billiard-ball theory of causation is possible but he also believes that in social research practice this 'A causes B' type of causal knowledge is virtually unattainable. Causality is not limited to simple 'billiard-ball' causality with one cause and one effect. True experiments via classical 'billiard-ball' ideas of causation can contribute to social causal-model pictures by using manipulability theory (Collingwood, 1940; Gasking, 1955; Whitbeck, 1977; Shadish, Cook, & Leviton, 1991). However, for the concept of causality to be functional in social research it needs to include "such things as mutual influence, multiple causation, or probabilistic causation" (Shadish, 1995, p. 69). All methods of inquiry are deficient and provide bias and error of one kind or another (Sechrest & Sidani, 1995). The ultimate purpose of any research design is to minimize threats to the validity of research conclusions (Chen, 1993).

All methods or designs used in scientific research have strengths, weaknesses, and errors in assessment (Sechrest & Sidani, 1995). Experimental designs depend upon manipulation combined with philosophical falsification (being able to falsify a null

hypothesis) to determine the probability of the obtained results given that the assumed reality is that there is no or null effect. However, according to Campbell (1969), classical falsification approaches to determining reality are fallible. Experimentally obtained results, being touted as the cornerstone of true research, never prove or confirm any theory or belief; all they demonstrate is a failure to confirm the null hypothesis (Campbell & Stanley, 1963). Experimental design, is in its simplest form, based upon the manipulation of only one variable (Collingwood, 1940; Gasking, 1955; Whitbeck, 1977; Shadish et al. 1991), and random assignment to comparison groups. The only evidence about the experimental hypotheses, that is available to the researcher, is indirect in nature. In other words, if the test is significant all we can say is that the Null Hypothesis is highly unlikely. Therefore there is indirect support for the alternative or research hypotheses.

Experimental manipulation and random assignment are the best research tools for demonstrating control of variables involved and causal relationships. However, the applications of findings collected in a strict experimental design to the real world environment, creating difficulties with external validity and construct validity. There are, for example, significant problems with random assignment in most social research since components of cause and effect relationships are multivariate in nature and random assignment is not sufficient to control for the influence of non-measured variables (Campbell, 1969). There is evidence that the larger the sample the better random assignment works to distribute all other variables. However, even with large samples there is no guarantee. There are also ethical and practical limitations when trying to include random assignment into research. One simply cannot randomly assign

participants to one gender or the other. Also, in most cases, it would be unethical to randomly assign one woman to a new treatment for cancer and assign the next woman to no treatment at all. However, in most instances the woman would be assigned to the current conventional treatment. Correlational designs are often used to assess relationships between variables when manipulation and/or random assignment are not possible.

Observational, non-manipulating, methodologies like correlation designs allow researchers to view the key variables interacting with their environment. Yet, these methodologies are limited if one wants to make causal arguments (Glick, Burti, Suzuki, & Sacks; as in Mirin, Gossett, Grob, 1991). In correlational designs it is important to note that, although variables may be related, it does not mean that there is a causal process limited to the two variables that are being observed (Scott, Mason, & Chapman, 1999). There are two dominant problems with correlational designs. The first, is the issue of direction of causation. This is especially an issue when the data have been collected at one point in time. The second problem is that the relationship between the two variables may be due to a third variable causing both the other variables to covary and, thus, resulting in a spurious relationship or the confounding variable may suppress a real relationship between the two variables of interest. For example, when the relationship between stress and psychiatric decompensation is being evaluated, at the same point in time, it is impossible to tell if the decompensation caused the stress or the stress caused the decompensation (Marken et al. 1992). It is also possible that the two variables were acting in a bi-directional manner. Prospective longitudinal studies are necessary to

decipher this kind of 'chicken-or-egg' problem (Deutsch & Davis, 1983; Fennig, Craig, Koenig, & Kuchibahatla, 1998; Helzer et al. 1978; McCabe, 1976; MacMahon & Pugh, 1970; Tanenberg-Karant & Bromet, 1994). However, the 'third variable problem' is much more difficult to resolve. For example, social support, stressful life events and psychiatric symptomatology have been found to covary systematically with each other. In this case these variables interact with each other and confound study results. If social support is related to life events and symptomatology then researchers may conclude that elevated symptoms are caused by low support levels when, in fact, stressful events actually depleted available support and contributed to symptomatology (Thoits, 1982). Then, of course stressful life events would be confounding the relationship between social support and life events. In a sample of pregnant women stress was found to be related to increased depression and according to Ritter et al. (2000) results suggest that less stress, greater satisfaction with social support, and greater income are important predictors if decreased depressed mood. Future correlational research, at least in these areas, will need to require causal knowledge that is multivariate-explanatory rather than just bivariate and descriptive (Shadish, Cook, & Leviton, 1991). The existing empirical results evaluating the relationships among stress, support, and depression indicate the need for more understanding of the complex causal relationships that are involved in humans' interactions with their environments (Aneshensel & Frerichs, 1982).

Social theories attempt to explain how humans interact with their worlds (Shadish et al. 1991). Factors associated with social problems tend to be multi-causal in nature and simple linear cause-effect relationships are rare (Kettner et al. 1990). Traditional cause-

effect relationships cannot be evaluated in much applied human service research (Chelimsky & Shadish, 1997; Shadish, 1995). Social theories and hypotheses are inundated with complex multivariate issues (Shadish, Cook, & Leviton, 1991) and the theory is a possible explanation about reality that needs to be tested (Wilkins, 1964). Multi-causal models are necessary to understand the complex issues found in human service research (Kettner, Moroney, & Martin, 1990). Epidemiological approaches propose that there are structural causal links connecting the variables involved, and if these links are broken or changed then outcomes can also be changed (Kettner, Moroney, & Martin, 1990). Causal-modelling is an orientation towards answering research questions, not a methodology per se, and is compatible with both non-experimental and experimental designs (Petrosino, 2000). Links among variables of interest form a structural model and each link needs to be tested (Mason, 1991). A causal-model approach with a coherent causal theory is recommended for answering how complex social variables work together (Chen & Rossi, 1980, 1983, 1987, 1992; Chen, 1990). Researchers need, then, to test the causal links specified in the causal model (Petrosino, 2000). With an empirical test of the critical links, one can know if the data fit the predicted causal model (Weiss, 1999). Data must be collected in a manner that allows testing links in the underlying causal model (Petrosino, 2000) and models need to be mutually determinative and responsive to a range of conditions (Mitchell & Moos 1984). Models must explain chains of causation with nodes in this chain of causation including the outcome (Eaton, 1994). A multi-variable problem requires statistical control of related variables and it is very difficult to control for all of the necessary variables (Hodgins,

1993).

Replication is one of the dominant argument tools for proof in social research; whereby a long series or program of research can reduce uncertainty about existing causal relationships by verification of findings through consistent production of similar findings (Shadish et al.1991). Once again, replicated results from various conditions, including partial causes, strengthen causal arguments (Shadish et al. 1991). Although one set of data neither proves to be a necessary nor a sufficient cause on its own, when there are a large number of cases demonstrating the same relationships it indicates "that the cause operates in a large enough proportion of cases (Eaton, 1981). However, "few public health problems have easily identifiable necessary and sufficient conditions for their etiology" (Eaton, 1994, p. 27).

Social science researchers often uses experimental design methodology which tend to favour the methodology first over the question of interest. Cronbach (1957) had argued for selecting the question of interest and using multiple 'albeit experimentally flawed' eclectic methods and designs that complement each other. "This preference transcends the usual notion of research design predicated on a single method providing the best answer for a particular inference, whether about causal connections (the experiment), causal explanation (modelling techniques), or generalization to populations (sampling theory)"(Shadish et al. 1995, p. 72). Cronbach (1957) pays attention to an innate ontological assumption of causal complexity in social systems and the need for replication. Social problems and research rarely fit into 'linear cause-effect relationships'. Multiple causation also tends to be the rule in this kind of research (Kettner et al. 1990);

and appropriate control groups are often difficult to find and/or construct (McGlashan, as in Mirin et al.1991).

Limitations of the Body of Empirical Literature

Many previous studies have evaluated the influence of social support and stressful life events (like unemployment and low income), upon mental health status. However, few have evaluated the effects these factors have on the consumption of mental health services (Sherbourne, 1988). Moreover, most of the studies looking at the influence of these variables evaluate some measure of current mental health or mood instead of diagnosis (Goulding, 1988) or resource usage. Instead of simply using a current measure of mental health, like psychological distress, that may or may not indicate general mental health, Bolton and Oatley (1987) recommended psychiatric inpatient treatment may be a more salient measure of mental health.

Most of the literature focussing on psychiatric symptomatology resulting from poor social networks and life stress has been conducted on samples that are either normal population members or non-psychotic clients (i.e., neurotic) (Romans & McPherson, 1992); and focus upon what Frank (1973) called demoralization or what Finlay-Jones and Burvill (1979) labelled minor psychiatric morbidity. Because of researchers focussing on normal or non-psychotic populations, little research has been conducted on the influence of these variables on symptomatology or hospitalization in the most susceptible populations. The available literature that does evaluate some of these variables in client groups only considers discharged patients. This is likely due to the problem of finding a

sample of clients who have not been admitted to a psychiatric facility. By ignoring diagnosed psychiatric clients who have avoided hospitalization, researchers may not have considered the possible salient characteristics that predict better coping and reduced resource utilization, or may have failed to identify those who were able to access needed resources.

According to Roberts, Roberts, and Stevenson (1982), much of the research has failed to consider demographic variables like age, income, and education. Yet, these demographic variables have been found to influence psychological well-being. Those studies that have addressed the effects of age, sex, social class, and socio-psychological influences on physical or mental health status in chronically ill patients have usually been conducted with severely impaired clients, ignoring better functioning psychiatric clients (Patrick et al. 1986).

According to Tausig (1992), there are many definitional and operational variations in the support literature that are due to a failure to distinguish between the structure of the support network, mobilization, and the effects of support on health outcomes. Depressive symptoms have been suggested to be related to a person's concern/lack of concern about the consequences of unhealthy behaviours (Allgower, Wardle, & Stepto, 2001). Much of the literature fails to consider the multidimensional nature of social support (Barrera, 1986). Earlier longitudinal research has failed to control for base-line psychopathology (Brugha et al. 1997). Many available social support studies use only self-reports of functional qualitative measures of support (i.e., perception of support). Consequently, these measures do not account for distortions in reporting that are possible due to

psychiatric impairment (Goodman & Johnson, 1986) which may result in a contamination effect (Eaton, 1978). Some consider social support more important than others (O'Driscoll & Dewe, 2001). Depressive symptoms place one at risk for functional impairments (Mehta, Yaffe, & Covinsky, 2002). Depressive symptomatology has been found to cause an over estimation of functional impairment assessments by patients (Cooke, Kruger, & Shugar, 1996). Also, and unfortunately, research has often focussed on variables that cannot be manipulated or treated to influence a change in outcomes. For example, it would be much easier to increase a client's contact frequency than change his or her perception of support, especially if she or he is still experiencing psychiatric symptoms. Because of the many limitations in the literature, the full ramifications of these support and stress factors on mental health and health service resource utilization are not well understood.

Current Research Objectives and Data Analysis Plan

Usually, past literature has taken bivariate, correlational, and unidirectional approaches to the variables of interest (Revicki & Mitchell, 1990) and has not evaluated the interrelationships among social contact and unemployment, income, and morale (Jones, 1988). Phase-I of this research describes preliminary data analysis including issues of control variables (impact and possible biases), missing data, response rates, zero order correlations, attrition issues, variable operational definitions, basic descriptive statistics, scale reliability and validity issues, factor analysis results, and global hypotheses. Phase-II of this research was necessary to evaluate the cross-sectional model for replication of previous results from cross-sectional research on the same sample (Murray, 1996). Phase-II of this research, via path analysis, evaluated the combined effects of social support (contact frequency and satisfaction with support), education, employment, income, and perception of financial adequacy on psychiatric symptomatology in a group of depressive and manic-depressive members of a self-help group, The Society for Depression and Manic-Depression of Manitoba. These data were collected over an 18-month time frame (1995-1997), and allowed four cross-sectional models to be produced, two for each year. This stage of the analysis evaluated the hypothesized models and computed model fit indices. Both the direct and indirect influences of each variable were considered in the specification of the final four models.

The goal of Phase-III of this research was to evaluate the existence of two competing theoretical causal approaches that explain the relationship between social support, SES, and mental health. This component of this research evaluated cross-lagged

models using the same data used in Phase-II. The two models were designed to test the Social Causation Theory and the Social Selection Theory. This phase of the research allowed the variables to be placed in a time continuum diminishing direction of causation ambiguities that plague cross-sectional research in this field.

Phase-IV of this research evaluated the relationships among social support, SES, symptoms, and length of inpatient treatment stays. This section is primarily descriptive in nature, given difficulties with sample attrition. There was also an attempt to compare those who had a history of psychiatric hospitalization with individuals without such a history.

CHAPTER 3:

Phase-I Methodology

This chapter begins with an explanation of the global hypotheses. The balance of this chapter deals with methodological/measurement issues and Phase-I of data collection and analyses. Sections include information about sampling strategy, self-help group information, data gathering approach, measurement of variables, and plans for further analysis. Preliminary analysis includes information about self-report issues, response rates, response biases, attrition, missing data, outliers, control variables influences, reliability and validity of measures. Parametric assumptions related to correlation, regression and t-tests are evaluated for suitability. And finally, information about path analysis plans is discussed.

Global Hypotheses

There are numerous hypotheses related to this research and in text format it would be very difficult to follow. To facilitate ease of understanding, the general hypotheses related to the theoretical positions are discussed below. All of the more phase-related, detailed hypotheses are discussed with the appropriate phase of the research to follow. This approach will allow the reader to compare the particular hypotheses figures to the results figures that follow almost immediately thereafter. The hypothesized directionally specific relationships in all of the models are indicated with valence signs. The specified relationships could represent the direct, indirect, or total effects of the variables involved. For cross-sectional models the reader is referred to Chapter 4. For time-lagged models the

reader is referred to Chapter 5. And, for hospitalization models the reader is referred to Chapter 6.

According to Johnson, Cohen, and Brook (1999), the social causation processes may play a more important role than the social selection process in anxiety, depressive, and personality disorders. From the social causation perspective a person's social environment would be proposed to negatively impact her or his psychiatric symptomatology for both cross-sectional and time-lagged path models. However, the impact of support may not become immediately apparent (Sheffield, Christie, Davidson, Manns, Shah, & Reid, 2000). Being exposed to a poor environment would be related to increased levels of psychiatric symptomatology. The primary theory related hypotheses would predict that the social support (CF, SAT, and SS) and social functioning (FAD, ED, EMP, and INC) variables would be inversely related to the pathology variables (SYMP, and LOS see Figures 1 and Figure 2 below).

Figure 1. The preliminary fully specified symptomatology path models for Time-1, -2, -3, and -4. Valence signs indicate the direction of twenty-one hypothesized variable relationships {(+) positive and (-) negative correlation}.

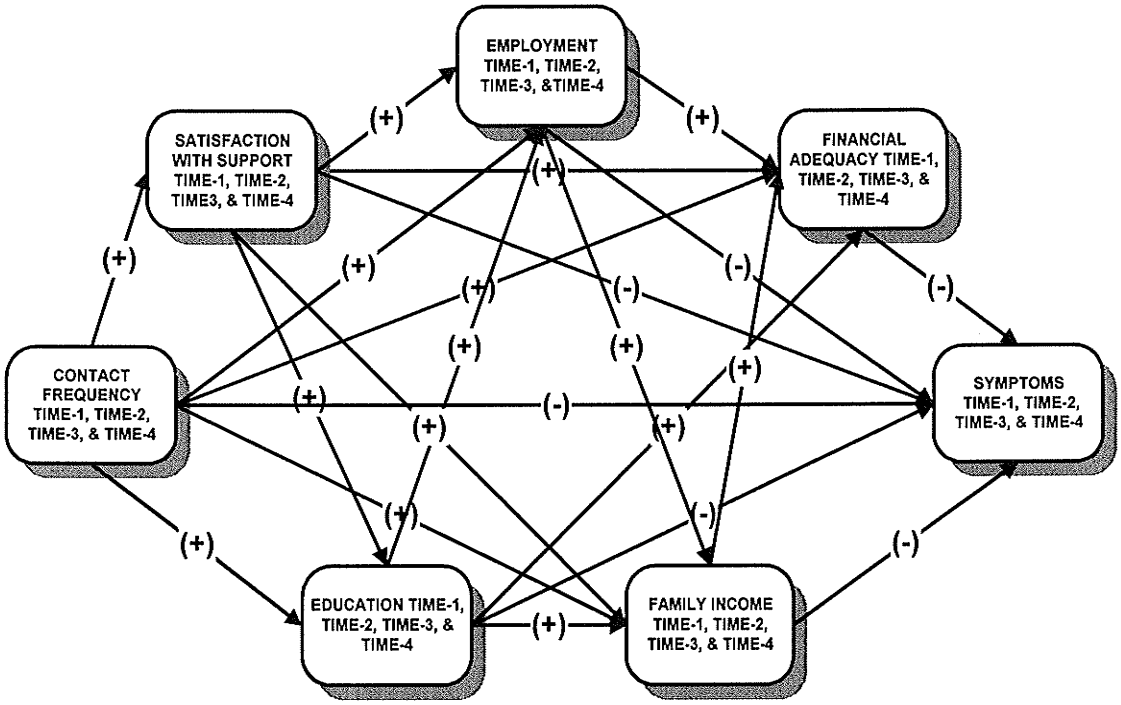
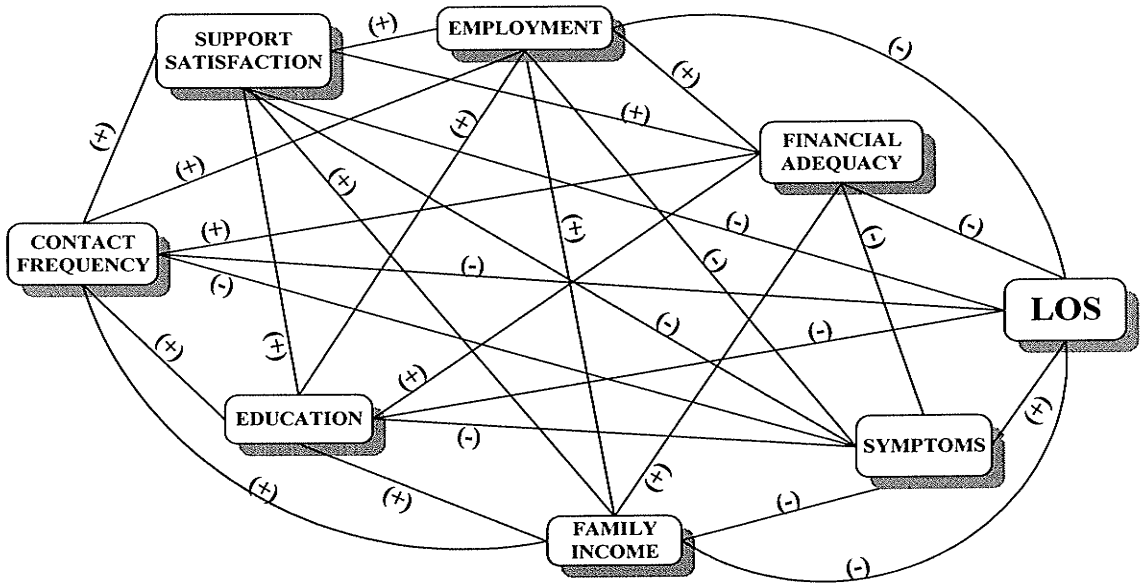


Figure 2. The preliminary fully specified LOS path models for Time-1. Valence signs indicate the direction of twenty-eight hypothesized variable relationships {(+) positive and (-) negative correlation}.



From the social selection theoretical position psychiatric symptomatology would be proposed to impact social functioning in a negative manner in both the cross-sectional and time-lagged models. In other words, a person's high psychiatric symptomatology would be related to poorer social functioning. The primary theory-related hypotheses would predict that the pathology variables (SYMP, and LOS see Figures 1 and 2 above) would be inversely related to the social functioning (FAD, ED, EMP, and INC) and the social support (CF, SAT, and SS) variables. The time lagged models allow differentiation of the hypotheses. Both predict negative cross-sectional correlations. Hospitalization results are discussed more fully in Chapter 6.

Sampling Strategy and Data Gathering Approach

A mail survey approach was selected to carry out this research because mail surveys can be completed within time and financial resource restrictions, while still maintaining the clients' confidentiality, in this clinically sensitive population. A convenience sample (the entire mailing list of the self-help Mood Disorders Association of Manitoba) was used rather than a random sample of the general Winnipeg population for a number of reasons. First, sampling the entire group resulted in a large enough sample; a sample size of more than 100 respondents is needed for the assumption of normality required by the path analytic procedure. Second, the target group of respondents included people with the psychiatric diagnosis of unipolar or bipolar disorder (depression or manic-depression) and these clients are rare in the general population. They would also be expensive to identify in a general representative population sample.

Clients used in this research indicated that they were diagnosed by a psychiatrist, general practitioner, or psychiatric nurse. If the person indicated that they had been admitted to a hospital to be treated for a psychiatric difficulty, but left the diagnosis question blank, they were also included in the sample. Third, because of the sensitive nature of the illness, these clients were less likely to respond to any surveys received from unknown persons or agencies, making contact through an established connection important (i.e., the Mood Disorders Society). Finally, this sample was a better medium to assess the relevant hypotheses than a discharged hospital cohort since the Society membership included individuals who had never been hospitalized and some whose hospitalization was not recent.

According to Katz (1981), self-help groups made up of lay members are the most widely spread system of care for human ills. Self-help groups have been described as patient-led change-oriented organizational (Kurtz, 1997). Self-help groups arise in society when certain needs are not met by the formal health care system (Den Boer, Wiersma, & Van Den Bosch, 2004; Lieberman, 1990). Self-help organizations are distinct from consumer-provided services in which consumers work as paid providers within clinical rehabilitative settings (Davidson et al. 1999; Solomon & Draine, 2001). The belief process behind the self-help movement is that a collective wisdom is born through the shared experience of participants rather than without the problems associated with dealings with professionals (Davison, Pennebaker, & Dickerson, 2000). Providing information may be one of the important functions of self-help groups (Citron, Solomon, & Draine, 1999). For example, cancer aids research found that self help groups provided

a lot of informational support (Adamsen, 2002). Self-help groups provide the opportunity to be exposed to stories of those who moved from poor to better quality of life might help those currently struggling with their illness (Dibb, & Yardley, 2006).

Self help groups are voluntary associations between persons with similar problems and the groups are usually controlled by the members (Humphreys & Rappaport, 1994). Some believe that theses self-help participants may not meet the criteria of diagnosis, duration, and disability that characterized clients of formal treatment services and this is due to person self-referral to the group, and the fact these self-help programs do not have admission criteria, and there are no formal assessments or history taking (Goerring, Durban, Sheldon, Ochocka, Nelson, & Krupe, 2006). According to Ragins and Cotton (1999), non-institutionally arranged self-help relationships, have been characterized as providing greater identification, role modelling, and interpersonal comfort (Sherman, DeVinney, & Sperling, 2004). However, according to Segal et al. (2002), participants in self-help groups reported higher distress levels but fewer acute symptoms, higher levels of functioning, and higher levels of well-being (Goerring, et al. 2006). According to Fraskovaik and Segal (2002), because of this perception self-help organizations have trouble demonstrating that they serve equivalent disabled populations when they are competing for resources. The literature needs research comparing those who are affiliated with self-help organizations and those who are not, with the goal of assessing similarities and differences in the two populations (Segal, Hardiman, & Hodges, 2002). In Canada, Dewa et al. (2002), found self-help users were drawn from a broader population but this included many who are severely mentally ill, who reported

being less stable and higher stress levels, who were more likely to have visited an ER for treatment, and more crisis prone. But once again there was still some evidence of higher functioning.

Someone who has successfully coped with a similar illness, disability, trauma, can be a mentor and can facilitate better adjustment (Hibbard et al. 2002; Thoits, Hohmann, Harvey, & Fletcher, 2000; Whittemore, Rankin, Callahan, Leder, & Carol, 2002). The respondents in this research, unlike the hospitalized clients in the pretest sample, are already members of a self-help group. Clients who participate in such groups are different from other individuals who have the same psychiatric diagnoses (Gidron, Guterman, & Hartman, 1990) and these individuals have been found to report a reduction in disease-related stress, increased social activation, and social competence (Trojan, 1989). Social contact is one of the important assets of self-help organizations. Yet, only 50% of the family members of those clients with chronic mental disorders believed that support/self-help groups were important (Cutler, 1994) in a recent Winnipeg study. It is likely that individuals who belong to self-help groups are more active help-seekers, and are supported in seeking help by their social support networks. Social support is also positively related to support help-seeking (Ben-Zur, 2002). Members of self-help groups have reported reduced symptomatology and reduced use of professional services (Kurtz, 1990). However, assessments of treatment/participation efficacy of self-help is problematic from the outset because of its inherently self-selected nature of these groups; but, results seem to be consistently positive for many participants (Davison, Pennebaker, & Dickerson, 2000). According to Gidron, Guterman, and Hartman (1990), those who

participated in self-help groups for parents of the mentally ill tended to come from a higher socioeconomic background, used more interactive active coping strategies, and were more stressed with the psychosocial burdens of the situation; while those not participating tended to suffer more from basic survival issues (like inadequate income) and used fewer active coping approaches. Usually, the members of these types of societies are highly motivated in endeavours that may help further their cause, creating a volunteer bias. A pilot test of the survey instrument indicated that respondents were well motivated since they took extra time to complete an open-ended question about current society functioning. However, at the same time, any particular group of members may be dealing with a recurrence or exacerbation of psychiatric symptomatology, reducing the likelihood of responding to surveys.

Because the population being evaluated was a psychiatric population and many may not have been functioning at their best, the survey instrument was brief while still covering the necessary variables. A short (four pages, 10 to 15 minutes completion time), primarily closed question format, mail survey was distributed to all members of the Society's 1996 mailing list ($\underline{N}$ = 922). The specificity of closed responses reduces the risk of misinterpretation (Converse & Presser, 1988) and minimizes the amount of cognitive energy required to complete the questionnaire (Converse & Presser, 1988; Dillman, 1978). A more open format (fill in the blank) was used for questions with responses that may have been unintentionally attenuated with closed response categories (for example when requesting feedback about Society services).

According to Bradburn et al. (1987), survey accuracy declines rapidly as the

number of questions increases, especially if the response time-frame is limited. Since this was a mail survey and the respondents may have been dealing with psychiatric symptoms, a fifteen minute time-frame was selected for maximum response and survey completion rates. These restrictions made it impossible to include a standard measure of symptomatology. The current measure of symptomatology was quite different from past measures (e.g., The Rand 36-item health survey's Emotional Well-Being Scale; Hays et al. 1993). The goal in this research was to evaluate an overall measure of psychiatric symptomatology not just a measure of depression. The present measure not only asked respondents how severe their current symptoms were, but it also asked them to reference their symptoms to their own experience over time (i.e., one pole of the measure is "the worst they have ever been"). This approach does not focus exclusively on depressive symptomatology, as manic symptoms, and other psychiatric symptoms may be included in clients' reporting of severity of symptomatology, as the current measure was self-referencing. Future research should validate the very simple measure of symptomatology used in this research against a commonly accepted measure like the Rand 36-Item Health Survey Emotional Well-being Scale (Hays et al. 1993). Possibly in future research larger measures like the Severely and Persistently Mentally Ill scale could be compared to the simple measures employed here (SPMI, Burlingame et al. 2006). The Brief Psychiatric Rating Scale (Overall et al. 1962). Has been found to consist of four factors but it does address assessing a wide variety of psychiatric symptoms (Shafer, 2005)

The survey packages included: a) a cover letter on Society letterhead explaining the goals of the research, promising confidentiality, and asking for voluntary

participation; b) the survey; and c) a stamped self-addressed envelope. Surveys were mailed from the Society office by staff who have access to the mailing list. Each survey was numbered for follow-up purposes (explained to respondents in a letter of introduction). As each survey was mailed, the identification number was recorded on the mailing list so the staff member could record responders' numbers to facilitate follow-up procedures (Dillman, 1978). Surveys were returned to the Society offices to reduce the concerns respondents might have about others having access to their responses. Informed consent was assumed since the participants chose to fill in and return the survey. Each person on the mailing list had personal contact with the self-help group at some time in the past and would either be a client, the friend of a client, a client's family member, or a professional who works in the area. Patients and family members of this group tend to contact the group when psychiatric symptoms have had a major impact on their or their family member's life. Group members are also recruited in psychiatric hospital facilities. These kinds of groups are less likely to attract people who are experiencing less severe or less frequent psychiatric symptomatology, for example, someone who has a reactive depression after the death of a loved one and who is treated by a family practitioner or a clinical psychologist.

Prior to dissemination the survey instrument was fully evaluated. Evaluation sessions with the director of the Society, staff members, and with patients were held to ensure instrument suitability, comprehensibility, and an appropriately short completion time. The survey questions (variables) were pre-tested on a small sample of recently discharged clients, in conjunction with a survey evaluating the usefulness of a new

outreach worker position at the University of Manitoba Health Sciences Centre (Murray, 1995). Only minor changes in layout and variable measurements were necessary in response to feedback from pre-test participants.

Since the Society's mailing list did not identify which of its members were clients and which were friends and/or family members, the entire list of Society members was used as the sampling frame. At last count, the Society membership list had approximately 976 (in 1995) individual names for the entire province of Manitoba. In a survey conducted of the Society's mailing list in the past (MacDonald, 1992) the overall response rate for the entire mailing list was 36%. Based upon Society meetings, approximately 30% to 40% of those who attend are family and friends and the other 60% to 70% are clients. This approximation was estimated from attendance at meetings from the number of persons in the small groups. Of the 365 surveys MacDonald dispatched, approximately 35% went to non-eligible participants (non-clients). Of the remaining 237 surveys, 143 were returned after a follow-up letter was mailed to initial non-responders. Based upon these figures a client response rate of forty percent was expected, especially since no follow-up mailing was planned for non-responders.

A follow-up mailing was decided against because this research was going to continue longitudinally, and, therefore, respondents who chose to continue would be receiving surveys every six months. To send follow-up reminders was going to become not only confusing, but possibly annoying, to respondents. Also, since clients are members of a psychiatric population, a follow-up may have been considered annoying, especially if the client was not well at the time. For example, if a client intended to send

back the survey but forgot for any number of reasons related to being ill, then reminding them may remind them of their inability to function normally. Also, the author did not wish to damage the relationship the membership had with the Society, thereby alienating possible future survey respondents.

Employment (EMP) and income (INC) information are less emotionally charged measures. However, most people are accustomed to reporting yearly income to the Canada Revenue Agency so they should have little difficulty estimating the requested income information or checking their tax returns. Employment measures should be salient for respondents. Estimating either number of weeks they worked during the year or the number of hours they were employed in the last two weeks should not be too difficult for them.

According to Bradburn, Rips, and Shevell (1987), although both omissions (forgetting) and telescoping (bringing events forward in time into the survey time frame) play a role in response bias, the longer the time period respondents are asked to remember over the greater the omissions regardless of the type of information being recalled (also see Sudman & Bradburn, 1973). Omissions lead to under reporting events and reductions in variable variability. Any degradations in variable variability in correlational and regression research result in reductions of the size of both the correlation coefficient and the beta weights of existing relationships resulting in an underestimation of the effect sizes and conservative results (Tabachnick & Fidell, 1989).

In general, depressive and general psychiatric symptomatology appears to be under-reported to health professionals by clients (Steketee et al. 1996; Tondo et al. 1988).

Removing identification of clients facilitates veridical (truthful) reporting of symptomatology by removing the possibility of penalties that may be imposed upon the report of higher symptomatology levels (i.e., more medication, more side-effects, or hospitalization).

Other response bias issues that may influence the results involve response set biases and a nonresponder bias. The questionnaire response scale scores (i.e., adequacy of financial situation and contact frequency) were not counterbalanced (reverse scored) to avoid response set patterns. This kind of adjustment in the response scale scoring was avoided to ensure ease of reading, increased comprehensibility, reduced confusion, and to facilitate a shorter completion time. The author felt that the possible disadvantages of a response set bias would be more than offset by the aforementioned advantages.

Self-report Issues

Self-reported psychiatric symptomatology has been used for many years as an indication of a client's emotional state. Physicians also use these self-reports to make decisions about appropriate medical treatments, and decide whether a client is a danger to themselves or others. The client's perception of difficulty, in many cases, may be used as an appropriate indicator of his or her mental health. Many standard diagnostic tools, even those requiring professional administration, rely on the client to provide the information about how he or she is 'feeling'. There have been various difficulties raised about the veridicality (truthfulness) and denotability (ability to assign a numeric value) of these self-report measures since a client's emotional condition has been said to influence

distortions in a client's self-perception, especially in depressive disorders (Beck, 1976; as cited in Cooke et al. 1996). According to Maughan and Rutter (1997), self reports are seriously flawed but others disagree. According to Bernstein et al. (1994), retrospectively obtained histories of childhood experiences are generally stable over time, show good agreement with reports of other informants (see Goodman et al. 2003; Paivio, 2001; Krause, Shaw, & Cairney, 2004).

Therefore, objective observational measures are often touted as the best approach for assessing psychiatric symptomatology. The advantages and disadvantages of self-report measures will be discussed together with veridicality and denotability issues, as they relate to self-report symptomatology measures, in the paragraphs that follow.

According to Aaronson (1989), a general review of the literature about the advantages and disadvantages in using self-administered versus interview-administered measures tends to find in favour of self-administered tests. Also, "self-administered questionnaires have been shown to be more reliable than those given by a trained interviewer" (Storstein, 1987, p. 210). Self-ratings have obvious advantages since they require little professional time, are inexpensive, can be completed quickly with little difficulty, can be used repeatedly (Goodwin & Jamison, 1990), and have been found to be clinically relevant in case identification (Matt et al. 1992). When you get right down to: "What do I really want to measure?" Then we realize that the patient has an ideal vantage point in assessing their situation. It is not the patients perceptions of their situation that fuels their behaviour? Only patients know how they feel, how a disorder affects their vigour, self-confidence, ability to socialize, obtain work, and function at home and on the

job (Trompenaars, Masthoff, Van Heck, Guss, Hodiamont, & De Vreis, 2006).

Primary disadvantages related to the use of self-report measures include the fact that patients must be literate, cooperative, not too depressed or manic to be able to physically respond and to concentrate, and the findings that clients with depressive symptomatology tend to under-report the true severity of their symptoms (Murphy et al. 1982b). According to Eastwood, Whitton, and Cramer (1994), the self-report approach to the assessment of intensity of psychiatric symptoms is considered the most feasible method for collecting data on quantitative changes in mood and other affective symptoms over long periods of time. Self-report information is readily obtained, can cover a long period of self-observation conveniently, and provides data on inner states that are not readily accessible by other methods (Epstein, 1979). However, according to Helliwell (2003), suicide is assessed on actually observed behaviour not subjective opinions which limits the use of subjective well-being in suicide prediction.

Self-report data are also considered controversial because these measures are, in effect, dealing with subjective experiences. However, according to Irwin, Kamman, and Dixon (1979), there is no guarantee that external observations are any more accurate than the client's self-reported observations. In fact, according to Murphy, Pickar, and Alterman (1982b), the self-report technique is better because patients are less influenced by theoretical biases affecting trained observer ratings and actually have access to the total global experience rather than a specific finite subset of behaviours available to the external observer. According to Goodwin and Jamison (1990), patients are in a unique position to provide information about their feelings and moods that are key indicators in

any assessment of mood disorders. Self-report assessments also provide an opportunity for a holistic perception of an unobservable phenomenon that is only accessible to the client experiencing the symptoms (Cronbach, 1984).

Client self-ratings and clinician ratings have consistently been found to be highly correlated (Tondo et al.1988). A client's total scores on various self-rating symptomatology scales tends to be higher than the corresponding symptomatology scale scores assigned by treatment professionals (Steketee, Frost, & Bogart, 1996; Tondo et al. 1988) and nurse ratings of symptomatology (Zeally & Aitken, 1969). Also, according to Raskin, Schluterbrandt, Reatig, and McKeon (1969), a mood scale completed by patients was one of the best measures of significant treatment effects. Even individuals with manic symptomatology appear to be capable of reporting their symptoms, regardless of their insight into their condition (Braunig, Shugar, & Kruger, 1996).

The client's perception of his or her symptomatology can be more important than what have been called objective measures because it is the clients' perception of symptomatology that guides individual clients' actions ('reality being the sum total of its consequences'). "Since psychiatric treatment is largely directed toward symptom relief, due weight should be given to the validity and immediacy of the patient's record" (Zeally & Aitken, 1969, p.995).

According to Cronbach (1988), "if a thing exists, it exists in some amount. If it exists in some amount then, it can be measured" (p.41). The fact that a construct exists but is not usually directly observable (unlike weight and height variables) means that researchers must make an attempt at assigning the construct a numerical measurement.

The assigning of a numerical value to a construct relates to Aftanas' (1994) issue of denotability (the capacity to assign a numeric value to the construct of choice). The issue of denotability with respect to self-report symptomatology is not as problematic an issue if one is prepared to accept that what is being assessed is the individual's perception of their psychiatric symptoms. Therefore, "the acceptance of this assumption means that the individual's responses can be taken at face value and interpreted accordingly" (Aftanas, 1994, p. 136).

In other words, there could actually be less difficulty isolating what is being measured than commonly believed, especially when we are considering a client's self-reported perception of general psychiatric symptoms. When we ask a client to give his or her perception of his or her psychiatric symptoms, we have the opportunity to evaluate private aspects of his or her experience that are not truly observable to 'objective observers'. However, we cannot be sure if the response we receive from the client is a veridical statement about the individual's situation (Aftanas, 1994). The issue of veridicality in this instance must be evaluated carefully as it depends primarily on why the client may not provide a veridical or true answer (possibly due to inability, or to consequences related to the truth). In situations of this type, according to Aftanas (1994), an independent assessment should be obtained, if possible. However, should veridicality not be focussed upon what is real to the client rather than what is real to objective observers? For example, is it not the client's perception of his or her symptomatology that dictates/predicts whether he or she seeks medical treatment, goes to work, or takes his or her life. Therefore, possibly the best approach under these circumstances is to compare

self-report measures with each other or to preexisting measures available in the literature. If the new measure functions well alongside a preexisting validated measure then the new measure may be useful for clinical evaluation.

Variable Operational Definitions

Symptomatology

According to Aiken (1970), the most practical instrument for assessing change in affective symptoms is a graphic self-rating scale; the most common of these scales being the visual analog scale. These scales have been reported as reliable, valid, and clinically useful for assessing moods over time with individual subjects (Lauria, 1975). According to Miller and Daron (1993), the Visual Analog Scale (VAS) is a technique used to measure subjective phenomena and is a self-reporting device consisting of a line of predetermined length that separates extreme boundaries of the phenomenon being measured (anchored at each end with opposite descriptors; e.g., 'the worst I have ever felt' and 'the best I have ever felt' (Zealley & Aitken, 1969). The classical scale requires a patient to place a mark or line across the line at the point on the scale that best characterizes his or her mental state. Classical scale advantages included the rapidity of assessment, involvement of the patients in their research and treatment protocols, relative lack of cultural and educational bias, and good reliability and validity (Zeally & Aitken, 1969).

According to Jordan et al. (1996), patients experience symptomatology in terms of a global psychological distress rather than in terms of specific identifiable symptoms. The

simplicity of the test makes it ideal for depressed patients who are very ill. Yet, the VAS is not sensitive to low levels of depression and other types of pathology. However, "the most important limitation of these VAS scales is that they are global measures; [therefore, they are] not designed to assess specific psychopathological states or symptoms" (Goodwin & Jamison, 1990, p. 329). An alternative format of the VAS is the segmented visual analog scale (SVAS). The segmented graphic rating scale (SVAS) is identical to the VAS except for vertical lines breaking the horizontal line into clear equal interval segments. The SVAS scoring is simpler (Kellner & Sheffield, 1968). Participants have been found to prefer the segmented visual analog scale (SVAS) to the unsegmented version (VAS) and the two versions produced the same results (Eastwood et al. 1994). Therefore, the segmented visual analog scale format was selected for the single-item measures of psychiatric symptomatology.

With the aforementioned measurement issues in mind an attempt was made to evaluate simple self-report measures of psychiatric symptomatology in a group of individuals with unipolar and bipolar depressive diagnoses who were members of The Society for Depression and Manic-Depression of Manitoba (Murray, 1996). The operational definitions for all salient variables used in this research can be found in Appendix A. A measure of current symptomatology (SYMP, see Appendix A) was assessed with a single SVAS question. This approach was chosen because participants were not mentally healthy, and were likely to have reduced abilities to tolerate completing more extensive clinical measures. The logic behind this approach was to facilitate a more accurate measurement by removing concentration and time pressures, and possibly

facilitate an increase in response rates because of the reduction in time required to complete the survey measures (Dillman, 1978).

Respondents were asked to indicate, on a single-item segmented visual analog scale, how severe their psychiatric symptoms had been during the two weeks preceding the survey date. A score of '0' indicated no psychiatric symptoms while a score of '10' indicated the most severe symptoms the respondents had ever experienced. This measure of symptomatology is not a simple depression assessment but also included manic symptomatology and/or any other symptomatology considered by the client as psychiatric in nature. It was also self-reported as well as self-referenced. One must keep in mind though that only patients really have access to how they are feeling and how felling in this way impacts their, self-confidence, ability to socialize, obtain work, and function at home and on the job (Trompenaars, Masthoff, Van Heck, Guss, Hodiamont, & De Vreis, 2006).

Since this simple SYMP measure has not been used, prior to now, to evaluate psychiatric symptomatology, the five question emotional well-being (EWB) scale from the Rand 36-Item Health Survey (Hays et al.1993; Ware et al. 1992) was administered at Time-2 for validation purposes. Initial reports of well-being were found to be strongly related to well-being later in time (Carveer, Smith, Antoni, Petronis, Weiss, & Derhagopian, 2005). Space for the EWB scale was made by removing questions that were no longer necessary at Time-2. Scores for this measure of EWB range from 0-100 with zero indicating low well-being or pathology and 100 indicating high well-being. For a detailed breakdown of the measures the reader is referred to Appendix A. The segmented visual analog scales was compared to a standard compound 5-item EWB scale, at Time-2,

Time-3, and Time-4.

The EWB was designed to assess general emotional well-being in individuals with various chronic medical conditions and is made up of five questions with a Likert-type response format, including descriptors at each numeric point (see Appendix A). The EWB measure controls for response set bias by having some items keyed in the opposite direction. The MOS health study and the Rand 36-item measure have been used to evaluate global health issues in numerous populations, including emotional well-being in clients with epilepsy, renal disease, heart disease, prostrate and breast cancer, diabetes, multiple sclerosis, and various psychiatric disorders, including mood disordered clients. However, bipolar disorders have not yet been studied using medical outcomes study methodology (Cooke et al. 1996).

Cronbach's alpha for the 5-item EWB measure has been found to range from an alpha of .76 to .90 for most usages of the sub-scale (Hays et al. 1993). Current results indicated inter-item correlations ranged from $\underline{r} = .53$ to $\underline{r} = .73$ ($\underline{p}$-values $< .0001$). Alpha if each specific scale component item is deleted ranged from .85 to .88. Alpha for the scale was .90 for both the standardized and un-standardized reliability measures. Alpha coefficients above .40 are usually considered to demonstrate fair to good internal consistency; while alpha coefficients above .75 demonstrate excellent internal consistency (Barker et al. 1994). Results also indicated that the single-item measure performed well when compared to the EWB scale for differing diagnostically related groups (DRGs). At Time-2 the correlations ranged from a high of $\underline{r} = -.82$ ($\underline{p} < .0001$) for those with a diagnosis of depression, $\underline{r} = -.67$ ($\underline{p} < .0001$) for those with a bipolar diagnosis, and a

score of $r = -.74$ ($p < .0001$) for the total sample.

Social Support

The Winnipeg Area Survey (WAS) is a representative yearly sample of individuals living in the City of Winnipeg. This survey is conducted by the University of Manitoba Sociology Department. Questions measuring current contact frequency consisted of a combination of questions modified from both the Winnipeg Area Study (WAS, 1988) and the Weissman's (1971) Social Adjustment Scale. Portions of the questions and response scales came from both sources; however, the response scales are most similar to Weissman's and the questions are more similar to the WAS. They gave a measure of the frequency of contacts between the respondents and others in their social network in structural terms. This measure is similar in nature to the measure used by Strauss and Carpenter (1977). According to Rushton, Brainerd, and Pressley (1983), the principle of aggregation is an effective means of minimizing error associated with individual measures. The principle states that "the sum of a set of multiple measurements is a more stable and representative estimator than any single measurement" (Rushton et al. 1983, p. 18). Therefore, contact frequency (CF) was computed by summing the responses to four questions about the frequency of the respondents' social interaction during the last two weeks. These questions asked how often the respondent had gone out with, depended upon for help of any sort, invited over, called (or was contacted by), or visited their friends and/or relatives. Each question had five possible responses creating a five point response scale. These questions were summed to arrive at a contact frequency

score. Contact Frequency inter-item correlations ranged between $\underline{r} = .40$ and $\underline{r} = .57$ (all p-values $\underline{p} < .0001$) with an overall reliability scale alpha = .78. There are differing opinions about the preferred level of scale reliability, Ideally one would like overall Cronbach's alpha to fall in the .70s (Barker et al. 1994), .80s, or .90s (Anastasi, 1988). These results indicate that the homogeneity of the items is not as high as it could be; but this consideration should be tempered by the fact that there are only four items in the scale limiting its variability. This variable ranged from the lowest score of 4 to the highest score of 20.

The client's satisfaction with social support (SAT) was also assessed. Respondents were asked to indicate, using a segmented visual analog scale, how satisfied they were with the support they received from others. A score of '0' indicated that they were very dissatisfied while a score of '10' indicated they were very satisfied with the support they received from others. Both measures of social support were used in model specification and hypothesis evaluation. A compound measure of social support was also created by adding the CF score and two times the SAT score. Multiplying the SAT score by two allowed for equal numeric weighting to arrive at a reasonable compound score. This compound variable was then used to evaluate the cross-lagged models in Phase-III.

Since the two social support variables behaved in a like manner, and to facilitate a more parsimonious test of the Social Selection and Social Causation theoretical positions, a factor analysis was run to see if CF and SAT could be combined into one social support (SS) variable. Factor structure diagnostics indicated that the four items in the CF measure and the single SAT measure do indeed act as a single factor. Inter-item correlations

ranged from $r = .27$ ($p < .01$) to .58 ($p < .0001$). Multiple colinearity was not a problem since none of the inter-item correlations were above $r = .80$ (Tabachnick & Fidell, 2001). Communality scores ranged from .410 to .639. A single factor was extracted using Principal Component Analysis and the total variance explained was 54.1%. Factor loadings were from .64 to .80. The single factor had an Eigenvalue of 2.706. These results were confirmed with a single factor appearing on the scree plot indicating that treating CF and SAT as a single combined score is reasonable for further model testing.

Education, Employment, and Income

Education (ED), employment (EMP), and income (INC) are closely related variables and are often used, separately or in combination, as a measure of socioeconomic status (SES) or social functioning (SF). For the purposes of this research, education was operationally defined as the number of years of schooling each client acquired (Winnipeg Area Study, 1988). The respondent's employment score was computed by summing the number of weeks of part-time employment with twice the number of weeks of full-time employment. This weighting of part and full-time employment was necessary to give more weight to employment weeks that were full-time in nature. The respondent's employment score assessed the amount of time the respondents spent in paid employment the year preceding data collection. This measure does not assess the level of unemployment directly; but indirectly through the frequently indicated low levels of the employment score. The employment measure was adjusted for subsequent administrations (i.e., 6 months).

Income was operationally defined as the respondent's gross income score and the family's gross income for the previous tax year. Although Myers and Bean (1968) found similar results with either family or individual income, family income is the more desirable measure when one is considering "the consequences of the debilitating effects of the social treatment of psychiatric patients" (Link, 1982, p. 206). Yet, personal income has been found to influence psychiatric symptomatology by influencing self-esteem (Ross & Huber, 1985). Family income was evaluated with the same question the Winnipeg Area Study (1988) used with slightly modified response scales and provided a measure of the total financial resources available to respondents. The modification in response scales was necessary. Since many people diagnosed with psychiatric disorders are living well below the poverty line; so few respondents were expected in the highest income brackets that two of the highest income brackets were eliminated. Since income is a sensitive issue with survey respondents under the best of circumstances (Dillman, 1978), the lower income brackets were expanded slightly so that respondents were not forced to answer the lowest available alternative. To avoid a reduction in response rates that was evident on the income question in Macdonald's (1992) survey results for this population, personal income was not requested directly. Personal income was computed by asking respondents what percentage of the total family income they contributed. This less direct approach, requiring more cognitive effort, seemed less offensive to clients during pretesting and improved the response rates on the income component over MacDonald's survey. Preliminary correlational analysis indicated that both personal and family income behaved in the same manner with symptomatology. The magnitude and direction of the

relationships between both the income measures with the symptomatology variable were identical. Therefore, since personal income was computed from family income it was excluded from further analysis of models. Subsequent waves of the survey asked for period specific income. For example, respondents were asked to provide the household income (before taxes) during the last year or the last six months.

Financial Adequacy Perception

Perception of financial adequacy (FAD) was evaluated by summing the responses to '6' questions about the respondent's satisfaction with the income they had available to meet their basic financial needs. These questions were, in part, from the 1988 Winnipeg Area Study (1988) with adjustments made for future savings and excluding questions about satisfaction with living accommodations. The questions making up the financial adequacy perception were similar to the measure employed by Feather (1989). Components of financial adequacy included whether the respondent was satisfied with the funds available for clothes, personal expenses, things for the home, money to do things with and to spend on the people they love, saving for the future, funds for leisure, and for their family's financial needs. The responses could range from '1' = very dissatisfied, while a score of '7' indicated very satisfied. The responses were added together to produce the FAD score. Scores could range from 6-28. Inter-item correlations ranged from .67 to .79 (p-values all $< .0001$), alpha if item deleted values were stable at .94, and Chronbach's alpha was .95.

Length of Hospital Stay

The length of psychiatric hospital stays (LOS) is strongly associated with the cost of health care and is also considered as an easy way to assess a quality-of-care indicator (Mai et al. 1993). Yet, often studies use a measure of current mental health or psychological distress during the period of influence by an acute stressor (i.e., death in the family) to measure the influence of other salient variables. Bolton and Oatley (1987) believed psychiatric inpatient treatment would be a more clear-cut measure of psychiatric disturbance. However, LOS measures do not take into consideration the variable influence of various sociodemographic characteristics or symptom severity; but, they do show the amount of mental health resources consumed by those admitted.

For the purposes of this research, length of psychiatric hospital stays (LOS) was assessed as the number of nights the respondent had spent in a psychiatric facility during the last three years. This measure is similar to a measure taken by Glick and Zigler (1986). Considering the entire sample resulted in LOS variables that were not normally distributed. This difficulty is a direct effect of having only 79 participants having a hospital admission during the last three years and only 46 participants reported having a hospital stay during the last year. These measures were adjusted for subsequent waves (i.e, asked only about the last six months). These numbers were lower than expected. Unfortunately, of the Time-1 hospitalization measure (LOS) from the smaller sample proved not to be normally distributed. The decision was made to evaluate the influence of the social functioning variables against the most extensive global measure, three year hospitalization, since it allowed the largest sample and was the best distributed of the

measures. A number of transformations were attempted and are discussed more fully later in this thesis.

Preliminary Analyses

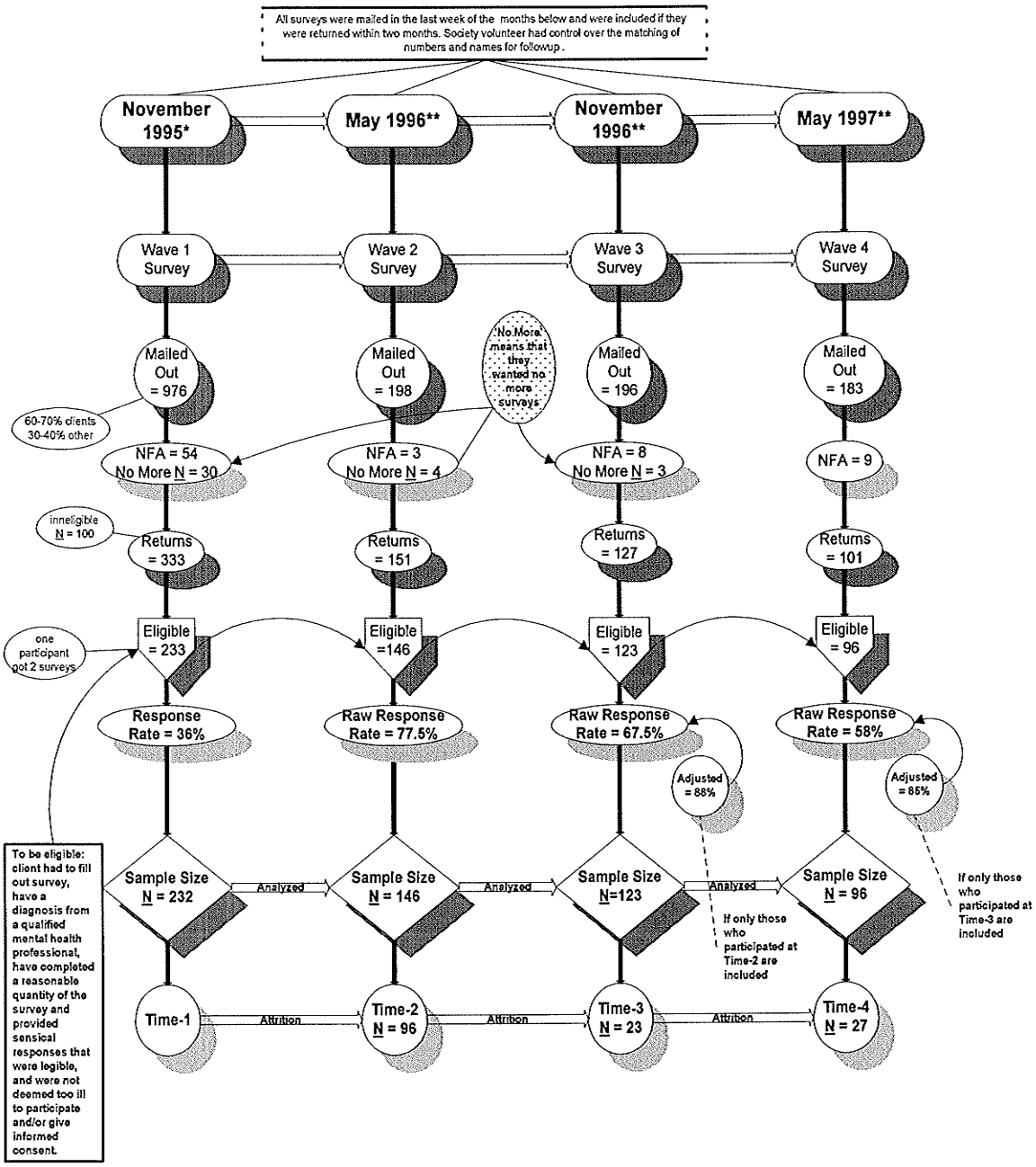
Response Rates, Response Bias, and Attrition

Response Rates

It is very difficult to maintain contact with psychiatric patients (Lyons et al. 1997). It is very difficult to follow released patients to see if they have been readmitted to other facilities (Lyons et al. 1997). Surveys were mailed out to all members on the mailing list at the end of November, 1995 and two months were allowed for the return of the surveys. The largest portion was returned during the first month; however, surveys continued to be returned steadily until the end of the seventh week. According to Narrow, Rae, Moscicki, Locke, and Regier (1990), another important consideration in non-response is that depressed respondents, because of the intrinsic nature of the disorder, are also more likely to be among the nonresponders. To try to eliminate some non-response bias and ensure a high response rate the surveys were not mailed in the peak depression winter months of December, January, and February or the peak manic symptomatology months of June, July, and August. March-May and October-November appear to be the best months for soliciting information from clients in both Depressive and Manic-Depressive diagnostic categories (see Goodwin & Jamison, 1990). If these cyclical issues were not considered an even lower response rate could have been encountered. The current response rate was 36%. Biases may have resulted due to illness. Individuals who were very ill or

hospitalized may be more likely in the ranks of those choosing not to or unable to respond. However, response rate results (40%) from a recent survey of clients just released from the University of Manitoba Psychealth Centre at the Health Science Centre indicated a similar rate to the one obtained in this research (Murray, 2002). To qualify for consideration each survey must have come from a client who a) had a diagnosis from a mental health professional, b) had completed a reasonable quantity of the survey, c) provided survey responses that made sense, d) was legible, e) had been completed by the patient (not family members), and f) was not so ill that they openly discussed suicide (too ill to participate and/or give informed consent). See Figure 2.5 for the total data analysis process.

Figure 2.5. Mood Disorders Association of Manitoba: Self-Help data collection and analysis flow chart.



* LOS variable asked for number of nights during the previous three years
** Note: the LOS variable used was the previous six months only

The response rate was of 36% of the original qualified clients from the mailing list and is in keeping with other response rates in similar client populations. There were surveys returned from the post office as undeliverable due to changed addresses (NFA). Time-1 NFAs were 54, Time-2 were 3, Time-3 were 8, and Time-4 were 8. The individuals (all of the 976 individuals on the self-help society's mailing list.) on the mailing list were asked to complete the survey if they belonged to the client designation, give it to the client they were associated with, or indicate they were not clients and mail back the survey in the envelope provided. For the purposes of this research 'client' referred to a person who had psychiatric symptoms that were severe enough to receive a psychiatric diagnosis from a health professional. Although Dimirsky (1990) reported that chronic mental disorder should be identified by a more multidimensional clinical diagnosis, this study used groupings based upon a traditional medical diagnosis category for the primary identification criterion. Independent clinical diagnoses were not made because to do so would have involved identifying and contacting clients and would violate the Society's basic ideology of appropriate mailing list usage. The mailing list was designed to contact members and keep them informed, not to identify clients for clinical subject pools. Individuals were included in the sample(s) if their response to a question regarding their psychiatric diagnosis showed that they had received a diagnosis of any form of depressive or manic-depressive disorder.

Of the total mailing, 54 Time-1 surveys were returned because of invalid or no forwarding addresses, leaving 922 active surveys. Of the active surveys 333 were returned in the two months following the mailing resulting in an overall response rate of 36%

(333/922). Non-eligible returns (100 surveys) were ones in which the individual receiving the survey indicated that they were not eligible since they did not have a psychiatric diagnosis. Since 100 of 333 responders were ineligible the percentage of members of the mailing list who were clients was computed to be approximately 70% (232/333). The eligible clients' response rate was 36% (922 * .70 = 645; 232/645 = 36%). This response rate is similar in magnitude to both MacDonald's (1992) response rate and the response rate of 40% acquired from the evaluation study in a small hospitalized sample (Murray, 1995). The response rate for the current sample of 36% does not compare well to the 1995 WAS's completion rate of 71.4%. However, the WAS uses a telephone survey approach with 10 repeated call-backs, for one-time assessment of measures, while the design of this research called for a longitudinal design. A single-wave mail survey was used , without call-backs, so not to burden clients and in keeping with the society's wishes. A few surveys could not be used because of inconsistent and inappropriate responses or frequent non-response. For example, clearly confused responses, nonsense responses, or completely blank surveys (N = 3). Two of the surveys had been completed by someone other than the participant. There were two surveys that were only half completed so they were eliminated. Surveys were also eliminated from useable surveys if they were returned minus the title page (N= 15, This did not influence Time-1 but follow up mailings was not possible for these individuals). The title pages are necessary because of the identification numbers that facilitated through time matching. Some individuals clearly indicated that they were dangerous to themselves (N = 2). In these cases there was a discussion with someone at the self-help group and contact was made by one of the

workers at the Mood Disorders Association of Manitoba as required in the approved research ethics protocol. These surveys were not included because the persons were too ill to give informed consent. In total eight surveys were eliminated at Time-1. There were also two suicides (that the researcher knew about) in people who participated at time one but were not alive to participate with all four waves of data collection. Suicide cases are the "tip of the iceberg in a population suffering from depression, hopelessness, and added co-morbidity of substance abuse, ninety percent of completed suicides are performed by individuals with a diagnosable psychiatric illness, substance abuse, or both" (Ryb, Sonderstrom, Kufera, & Dischinger, 2006).

For respondents to qualify to receive the Time-2 survey they must have completed the Time-1 survey (November 1995) and indicated, in the appropriate space, that they were a client and that they wanted to continue to participate with this research. If the respondent said that they would rather not continue to receive surveys they were dropped off the mailing list for subsequent waves. For more detailed explanations of the intricacies of the process the reader is referred to Figure 2.5 above. For respondents to qualify to receive the Time-3 survey they must have completed the Time-1 survey, not have had their Time-2 survey returned by the post office, and did not indicated that they would rather not continue to participate on either the Time-1 or the Time-2 survey instrument.

For respondents to qualify to receive the Time-4 survey they must have completed a Time-1 survey, did not have their Time-2 or Time-3 survey returned by the post office, and did not indicate on any survey that they would rather not continue with this research.

There were a total of 183 Time-4 surveys mailed out; 9 of these surveys were returned by the post office with no forwarding addresses leaving a total of 174 active Time-4 surveys. Of these 101 were returned to the Society office resulting in a Time-4 response rate of 58%. Of these 96 surveys were used in the balance of model testing analyses.

The previously computed response rates for Time-3 and Time-4 are somewhat artificially deflated because of the denominators including individuals who did not mail back either Time-2 or Time-3 surveys yet failed to indicate that they would rather not continue with the research on Time-1 or Time-2 surveys. For example, an individual could have sent the Time-1 survey back and left the question about continuing with the subsequent waves of the survey research blank. This individual would have still qualified and received a Time-2 survey. At this point if the individual failed to return the Time-2 survey they would still qualify to receive the Time-3 survey. Even if this individual did not send back the Time-3 survey they would still qualify to receive the Time-4 survey. This protocol was used to maximize the number of eligible respondents for Time-4 of the research.

The individuals in this population are often sick and may opt not to or forget to respond to a particular wave because their psychiatric symptoms interfere with their ability to respond. However, they may return subsequent waves because they are feeling better or are able to remember to respond. If we only include individuals who have participated at Time-2 then the adjusted response rate for Time-3 becomes 88% (127/144). Of these, 123 were deemed useable by previously stated criteria). If only participants who responded in Time-3 were used to compute the response rate at Time–4

then the adjusted response rate is 85% (101/119). Of these 96 were deemed useable by previously stated criteria). The qualified, useable, sample size resulting from the first mailing (Time-1) was N = 232; the second (Time-2) mailing retained N = 146 clients with non-missing values, Time-3 and Time-4 managed to maintain the sample sizes at N = 123 and N = 96, respectively.

Response Bias

Before discussing the research findings, it is important that the reader be aware of the response bias that may be influencing results. Individuals who are severely ill with depressive and/or manic symptomatology are less likely to have returned the surveys and if they did return them were less likely to be able to complete them at an acceptable level. Those who would return or receive (because they were not in the hospital) the surveys would be able to complete it without any difficulty. However, they would likely be less ill than the balance of this population which would result in reducing the variability of all of the measures, leading to a suppression effect on the size of the correlation coefficients. Reducing the range over which an effect/relationship can be detected because of either under-reporting of symptomatology or limitation to the full range of variable scores, due to sicker individuals not contributing to the data set as much as others, likely served to reduce the variability of all of the variables. As a consequence the correlation coefficients will be under-estimated and the probability of finding significant results will be biassed in a conservative manner (Tabachnick & Fidell, 1989).

The literature explains that certain response biases are expected using survey

methodologies. For example, individuals with higher income and education are more likely to send in surveys in the first place (Dillman, 1978). This bias should be expected to compound through the time frame of the research. Therefore, individuals with higher income and educational attainment are more likely to have responded to all four waves of the research. The effect of this bias on the generalizability of the findings will be discussed below.

Individuals who are more ill should be less likely to respond to all of the waves (less likely to comply with returning all the surveys). Sicker individuals are less likely to be employed and are more likely to have had their employment impacted by their illness. Therefore, individuals with lower employment scores should be less likely to respond to all four waves. A group of dichotomous variables were created for evaluating, via Chi Squared tests, if any biases existed in the likelihood of responding to subsequent waves (Time-2, Time-3, and Time-4) of the survey because of differences in primary variables at Time-1. All results are referenced to Time-1 distributions. A logistic regression was also performed on the total sample to determine if any of the primary variables could predict participating in all four waves of the study. Results indicated a non-significant Chi-squared value ($\chi^2_{(7)} = 11.494$, $\underline{p} = .1185$).

Time-1 data were used to evaluate if there were differences in variables at Time-1 that predicted those who also complied at Time-2. There were no significant differences in the average age, contact frequency scores, financial adequacy perceptions, employment scores, three year hospitalization LOS (for the entire group), or self reported symptoms. There were differences in household income, education, social support satisfaction, and

three year LOS (in the group with a positive hospitalization history during the last three years). Individuals who responded to both Time-1 and Time-2 surveys had marginally significant higher household incomes at Time-1 ($N_{(\text{did not respond to Time-2})} = 84$, $M = 12.54$, $SD = 6.86$; $N_{(\text{did respond to Time-2})} = 149$, $M = 14.20$, $SD = 6.77$; $t = -1.79$, $p = .074$, two-tailed). This failure to reach significance was, in my opinion due to sample size not effect size. Individuals responding to both Time-1 and Time-2 surveys had significantly higher educational scores at Time-1 than those not responding at Time-2 ($N_{(\text{did not respond to Time-2})} = 84$, $M = 13.21$, $SD = 3.59$; $N_{(\text{did respond to Time-2})} = 149$, $M = 14.31$, $SD = 3.34$; $t = -2.34$, $p = .020$, two-tailed). Individuals responding to both Time-1 and Time-2 surveys also indicated that they were more satisfied with the support they have available but this effect was not significant ($N_{(\text{did not respond to Time-2})} = 84$, $M = 4.59$, $SD = 2.18$; $N_{(\text{did respond to Time-2})} = 149$, $M = 5.04$, $SD = 1.81$; $t = -1.71$, $p = .106$, two-tailed; adjusted for unequal variance, $F = 5.506$, $p = .020$). This was probably not significant due to a small effect magnitude. In the group of individuals who indicated a Time-1 non zero three year hospitalization score; individuals who responded to both Time-1 and Time-2 surveys had significantly higher (the opposite of what would be expected) three year LOS score ($N_{(\text{did not respond to Time-2})} = 31$, $M = 37.94$, $SD = 36.8$; $N_{(\text{did respond to Time-2})} = 47$, $M = 60.06$, $SD = 60.06$; $t = -1.91$, $p = .061$, two-tailed; adjusted for unequal variance, $F = 4.64$, $p = .035$). In this case, the effect magnitude appear to be of a salient magnitude so sample size appears to explain why the difference in LOS (approximately 23 nights) is not statistically significant.

Time-1 data were used to evaluate if there were differences in variables at Time-1

based upon whether the respondent who complied at Time-1 also complied at Time-3. There were no significant differences in the average age, contact frequency scores, perceptions of support, financial adequacy perceptions, employment score, three year LOS (for the entire group), or self reported symptoms. Individuals who responded to the Time-1 and Time-3 surveys, unlike those responding to the Time-1 and Time-2 surveys, were not significantly different in their satisfaction of support or three year LOS in individuals with a non-zero three year hospitalization scores. It is not clear why this occurred. Individuals who responded to both Time-1 and Time-3 surveys had significantly (no longer marginally) higher household incomes at Time-1 ($N_{\text{(did not respond to Time-3)}} = 109$, $M = 12.54$, $SD = 6.76$; $N_{\text{(did respond to Time-3)}} = 124$, $M = 14.54$, $SD = 6.79$; $t = -2.26$, $p = .025$, two-tailed). Individuals responding to both Time-1 and Time-3 surveys had significantly higher educational scores at Time-1 than those not responding at Time-3 ($N_{\text{(did not respond to Time-3)}} = 109$, $M = 13.04$, $SD = 3.43$; $N_{\text{(did respond to Time-3)}} = 124$, $M = 14.69$, $SD = 3.32$; $t = -3.72$, $p < .0001$, two-tailed).

Time-1 data were used to evaluate if there were differences in variables at Time-1 based upon whether the respondent who complied at Time-1 also complied at Time-4. There were no significant differences in the average age, contact frequency scores, satisfaction with support, financial adequacy perceptions, employment score, or three year LOS (for the entire group), or self reported symptoms. Individuals who responded to the Time-1 and Time-4 surveys, like those responding to the Time-1 and Time-3 surveys, were not significantly different in their length of hospitalization than individuals with non-zero three year LOS scores. This finding agrees with the results comparing those who

responded in Time-1 and Time-3. Individuals who responded to both Time-1 and Time-4 no longer had significantly higher household incomes ($N_{\text{(did not respond to Time-4)}} = 136$, $M = 13.11$, $SD = 6.995$; $N_{\text{(did respond to Time-4)}} = 97$, $M = 14.31$, $SD = 6.57$; $t = -1.32$, $p = .187$, two-tailed). Individuals responding to both Time-1 and Time-4 surveys still had significantly higher educational scores at Time-1 than those not responding at Time-4 ($N_{\text{(did not respond to Time-4)}} = 136$, $M = 13.52$, $SD = 3.34$; $N_{\text{(did respond to Time-4)}} = 97$, $M = 14.46$, $SD = 3.58$; $t = -2.06$, $p = .041$, two-tailed). It appears that only the education bias appears to be consistent across all the above comparisons and this bias is congruent with the general trends noted in the literature (Dillman, 1978). Interestingly enough, even this bias has reduced in magnitude by the fourth wave of the survey.

Two other approaches were used to evaluate possible response biases. First, Time-1 data were used to evaluate how respondents who responded to 3 or more waves compared with those who responded to fewer than three waves to see if there were any systematic differences in the primary variables. Individuals who responded to three or more waves were different from those who did not respond to at lease three waves. Those who responded to three or more had marginally significant (10 point) higher employment scores ($N_{\text{(three or more waves)}} = 125$, $M = 39.38$, $SD = 44.44$; $N_{\text{(less than three waves)}} = 108$, $M = 29.86$, $SD = 40.09$; $t = -1.70$, $p = .09$, $df = 232$, two-tailed). This non significant difference was probably due to a small effect-magnitude rather than sample size, family incomes ($N_{\text{(three or more waves)}} = 125$, $M = 14.58$, $SD = 6.72$; $N_{\text{(less than three waves)}} = 108$, $M = 12.48$, $SD = 6.82$; $t = -2.37$, $p = .019$, $df = 232$, two-tailed), education ($N_{\text{(three or more waves)}} = 125$, $M = 14.52$, $SD = 3.45$; $N_{\text{(less than three waves)}} = 108$, $M = 13.21$, $SD = 3.37$; $t = -2.91$, $p = .004$,

df = 232, two-tailed), and were more satisfied with their social support ($N_{(three\ or\ more\ waves)}$ = 125, M = 5.13, SD = 2.16; $N_{(less\ than\ three\ waves)}$ = 108, M = 4.60, SD = 2.16; t = -2.10, p = .002, df = 204.57, two-tailed, adjusted for unequal variances). There were no differences in respondents' age, LOS, contact frequency, symptoms, financial adequacy perceptions, or age.

Second, Time-1 data were used to evaluate how respondents who responded to all four waves were compared to those responding to fewer than four waves to see if there were any systematic differences in the primary variables. Individuals who responded to all four waves were different from those who failed to comply at all four points in time. Those who responded to all of the waves had non-significant differences in family incomes ($N_{(all\ four\ waves)}$ = 76, M = 14.68, SD = 6.6; $N_{(less\ than\ four\ waves)}$ = 157, M = 13.08, SD = 6.9; t = -1.68, p = .095, df = 231, two-tailed). This result was probably due to a small effect-magnitude rather than power difficulties.

Those who responded to all of the waves had significantly higher education scores ($N_{(all\ four\ waves)}$ = 76, M = 14.82, SD = 3.26; $N_{(less\ than\ four\ waves)}$ = 157, M = 13.48, SD = 3.49; t = -2.80, p = .006, df = 232, two-tailed). This result is expected given the common response bias towards higher education responding to mail surveys (Dillman, 1978). For those responding to all four waves, there were no differences in respondent's age, LOS, contact frequency, perception of support, symptoms, employment scores, financial adequacy perceptions, or age. In summary, persons who participated with more waves of these surveys have higher education and marginally significant household incomes than those who participated less. These persons also appeared to be similar in symptom level

and had better employment profiles.

Response Bias and Control Variables

The proportion of individuals in the total responding sample (N = 232) with unipolar and bipolar depressive disorders was approximately equal (44.0% unipolar, 39.7% bipolar, and 15.5% not clearly defined or multiple diagnoses). Less than 1.0% of the respondents did not indicate any form of diagnosis. Ninety-three percent of the sample clearly indicated that they had received their diagnosis from a qualified health professional and the other 7.0% either did not indicate who gave them a psychiatric diagnosis or mentioned someone other than a medical doctor, psychologist, psychiatrist, or psychiatric nurse. These individuals were included in the sample since they either indicated that they were taking medications for psychiatric symptomatology, that they had been hospitalized for psychiatric reasons, or had interactions with a psychiatric department social worker and a psychiatric hospitalization history.

Due to a lack of random sampling of the mailing list, it was possible that the acquired sample was not representative of the full membership. In an attempt to evaluate if a response bias was operating in the group of respondents who complied with the survey request, both gender and postal code information were recorded along with the survey identification number. The mailing list included individuals from urban and rural sections of Manitoba. The postal codes were available for 920 of the active surveys. The proportion of urban (82.5%) and rural (17.5%) postal codes in the active survey list was close to the proportion of the aforementioned designations in the total mailing list

(81.7% and 18.3%, respectively). Therefore, the sample used in the study was representative of the mailing lists urban/rural distinction.

A clear determination of gender was made for 929 individuals on the mailing list. Some members of the list could not be clearly identified as belonging to either gender because the mailing list only included either the individual's first initial, the mailing list entry title was Mr. and Mrs. and the client could not be identified, or determining the individual's gender was very difficult due to an ethnic first name. The identified group consisted of 560 (60.3%) females and 369 (39.7%) males. The gender of the sample respondents was 62.5% female and 36.6% male. This is often reported with mail surveys (Dillman, 1976). Therefore, the sample obtained was representative of the gender split on the original mailing list. The gender split in the Winnipeg Area Survey results (1995) also display an over-representation of females (56% females and 44% males).

This research relies on retrospective self-report data. Since memory plays a role in determining accuracy of reporting, this approach can lead to a contamination effect or an availability bias. Self-administered questionnaires usually lead to an under reporting of behavioural items but reduce the under reporting of embarrassing information (Bradburn, 1985). Since the respondents are being asked to recall information for a three year period, an availability bias is expected to influence the responses assessed over longer time frames. Salience of the events being recalled also influences response bias. Recall is more difficult if there are many similar incidents and easier if the events have an emotional component (Bradburn et al. 1987). Hospital stay information may be under-reported to some extent because of the influence of memory for the time frame. However, since

psychiatric hospitalization is a salient emotional event for clients, recall should be reasonably accurate. In most cases pretest information indicated that respondents could answer, to the day, the time they had spent in the hospital. Also, according to Eaton (1978) in a re-analysis of panel data, the "effect of recall distortion on the life events-mental disorder relationship is negligible" (p. 230). However, before measures can be considered completely adequate, their validity needs to be established through independent sources (Birkel & Reppucci, 1983).

In an attempt to verify hospitalization data one of the Society volunteers with a recent hospitalization history contacted some of the respondents reporting a psychiatric hospitalization history. The volunteer discussed the reason for the phone contact with 13 of 76 respondents with a three year hospitalization score. At this time the respondent was asked to answer the LOS hospitalization question again after checking any of the information they had available about their hospitalization history. Respondents were questioned about how many times they had been admitted for inpatient treatment during the last three years and how many nights they had spent in an inpatient facility during the last three years. Although responses at this time did not exactly match the earlier survey responses (results showed a reduction from first to second measure for LOS) a Wilcoxon Matched-Pairs Signed Rank Tests indicated no significant pair-wise differences in hospitalization LOS ($Z_{(\text{three year stay})(12)} = -.18$, $p = .859$; two-tailed tests). The results of the paired signed rank test, while non-significant, probably due to a lack of power from the small sample, do directionally support a general decline in accuracy (under-reporting possibly due to forgetting) of reporting over time as suggested by Bradburn et al. (1987).

This under-reporting will serve to reduce the variability of the hospitalization variable. As a consequence the correlations and beta weights will be under-estimated (Tabachnick & Fidell, 1989).

Time-2 results indicate that the sample is comprised of 88 of 143 (62.0%) individuals who have indicated that they have a psychiatric hospitalization history which is equal to the Time-1 rate of 140 of 226 (62.0%). At Time-2 the distribution of marital/cohabitating status is also equal to Time-1. At Time-2, 84 of 148 (57.0%; Time-1 = 56.5%) individuals indicated that they were either married or cohabitating with a partner. The urban/rural composition of the Time-2 sample also mirrors Time-1. The Time-2 sample is composed of 82.5% urban residents (Time-1 proportion = 82.0% urban respondents). Gender is also comparable between the two points in time. At Time-2 the sample was made up of 36.3% males and 63.7% females (Time-1 was 36.5% and 63.5% respectively). The diagnostic category distribution for the Time-2 sample was also similar to the Time-1 sample. The proportion of respondents indicating that they belonged to the depressive group was 46.0%, the bipolar group was 46.0%, and the undifferentiated/multiple-diagnoses/non-response group was 8.0% (This compares reasonably well to the Time-1 proportions of 45.0%, 41.0%, and 14.0% respectively.). Respondent's ages at Time-1 and Time-2 were not significantly different based upon Time-1 data (Time-1: $\underline{M}_{age} = 46.5$, $\underline{SD} = 14.4$; Time-2: $\underline{M}_{age} = 45.2$, $\underline{SD} = 13.4$, $\underline{N} = 149$; $\underline{t}_{(232)} = 1.3$, $\underline{p} = .21$, two-tailed; This $\underline{t}$-test was a single sample $\underline{t}$-test using the standard error, based upon the larger sample size, of Time-1; therefore, it is a liberal test biassed in favour of finding a significant effect).

Time-3 results indicate that the sample is comprised of 74 of 127 (58.3%) individuals who have indicated that they have a psychiatric hospitalization history which is similar to the Time-1 and Time-2 rate of 62.0%. The Time-3 distribution of marital/cohabitating status is also similar to Time-1 and Time-2. At Time-3, 76 of 127 (Time-3 = 59.8%; Time-2 = 57.0%; Time-1 = 56.5%) individuals indicated that they were either married or cohabitating with a partner. The urban/rural composition of the Time-3 sample is somewhat different than the samples at Time-1 and Time-2. The Time-3 sample is composed of 77.2% urban residents (Time-2 proportion = 82.5%; Time-1 proportion = 82.0% urban respondents). The Time-3 sample appears to be composed of a somewhat greater proportion of individuals from a rural area than either the Time-1 or Time-2 samples.

Gender is also comparable between the first three points in time. The Time-3 ratio of males to females is 34.7%: 62.2% (Time-2 was 36.3%:63.7% and Time-1 was 36.5%:63.5%.). The proportion of respondents indicating that they belonged to the depressive group was 47.0%, the bipolar group was 37.8%, and the undifferentiated/multiple-diagnoses/non-response group was 15.2% (This compares to the Time-2 proportions of 46.0%, 46.0%, 8.0% and Time-1 proportions of 45.0%, 41.0%, and 14.0% respectively.). The diagnostic category distribution for the Time-2 sample was similar to the Time-1 sample; however, it appears as in the Time-3 sample those with bipolar disorder are somewhat under-represented. Respondent's ages at Time-1 and Time-3 were not significantly different (Time-1: $\underline{M}_{age} = 46.5$, $\underline{SD} = 14.4$; Time-3: $\underline{M}_{age} = 47.4$, $\underline{SD} = 13.5$, $\underline{N} = 127$; $\underline{t}_{(232)} = -1.0$, $\underline{p} = .300$, two-tailed; This t-test was a single

sample t-test using the standard error, based upon the larger sample size, of Time-1; therefore, it is a liberal test biassed in favour of finding a significant effect).

Time-4 results indicate that the sample is comprised of 58 of 101 (57.4%) individuals who have indicated that they have a psychiatric hospitalization history, which is similar to the Time-1, Time-2, and Time-3 rates of 62%, 62%, and 58.3% respectively. The trend in the hospitalization rates shows a slight reduction in the proportion of the sample who have been hospitalized for psychiatric reasons. This small bias was expected as individuals who are more ill were expected to have a higher attrition rate. This kind of bias will cause a reduction in the values of the illness variables resulting in reduced variable variability and conservative outcomes for statistical tests (Tabachnick & Fidell, 1989).

At Time-4 the distribution of marital/cohabitating status is also similar to Time-1, Time-2 , and Time-3. At Time-4, 58 of 101 (Time-4 = 57.4%; Time-3 = 59.8%; Time-2 = 57%; Time-1 = 56.5%) individuals indicated that they were either married or cohabitating with a partner. The urban/rural composition of the Time-4 sample is similar to the Time-3. The Time-4 sample is composed of 76.3% urban residents (Time-3 = 77.2%; Time-2 = 82.5%; Time-1 = 82% urban respondents). The Time-4 sample, like the Time-3 sample, appears to be composed of more individuals from a rural area than either the Time-1 or Time-2 samples. Gender is also not directly comparable at all points in time. The Time-4 proportion of males to females is 28.7%: 67.3% (Time-3 was 34.7%:62.2%, Time-2 was 36.3%:63.7%, and Time-1 was 36.5%:63.5%.). It appears that males are somewhat less likely to have responded to the final wave of the

survey.

The proportion of respondents indicating that they belonged to the depressive group was 46.5%, the bipolar group was 39.6%, and the undifferentiated/multiple-diagnoses/non-response group was 13.9% (This compares to the Time-3 proportions of 47.0%, 37.8%, and 15.2%; Time-2 proportions of 46.0%, 46.0%, and 8.0%; and Time-1 proportions of 45.0%, 41.0%, and 14.0% respectively.). The diagnostic category distribution for the Time-2 sample was similar to the Time-1 sample. However, it appears that the Time-3 sample of individuals in the bipolar diagnostic group are under-represented. The Time-4 sample reflects more the distribution of the Time-1 sample;. Therefore, any biases evident in the distributions of the diagnostic categories making up the four samples does not appear to be patterned or systematic in nature. Respondents' ages at Time-1 and Time-4 were only marginally different with the later sample appearing somewhat older (Time-1: $\underline{M}_{age} = 46.5$, $\underline{SD} = 14.4$; Time-4: $\underline{M}_{age} = 48.2$, $\underline{SD} = 13.9$, $\underline{N} = 101$; $\underline{t}_{(232)} = -1.9$, $\underline{p} = .06$, two-tailed). This is expected since participants were expected to age during the study time. This marginally significant difference was due to the nature of the one sampled $\underline{t}$-test used. This was a single sample $\underline{t}$-test using the standard error, based upon the larger sample size, of Time-1. Therefore, it is a liberal test biased in favour of finding a significant effect. Because of the liberal nature of this particular $\underline{t}$-test an alternative comparison was made using the sample size from Time-4 and results indicated a non-significant difference in age of the respondents between the two waves (Time-1 and Time-4; Time-1: $\underline{M}_{age} = 46.5$, $\underline{SD} = 14.4$; Time-4: $\underline{M}_{age} = 48.2$, $\underline{SD} = 13.9$, $\underline{N} = 96$; $\underline{t}_{(96)} = 1.3$, $\underline{p} = .20$, two-tailed). Therefore, the respondent's who

responded to Time-4 are similar to the Time-1 respondents in age (other than considering that participants aged during the study period).

Attrition Issues

Using two-by-two tables and Time-1 results for comparison purposes, a chi squared analysis indicated that individuals with a bipolar diagnosis were somewhat less, although not significantly less, likely to respond to all four waves ($\chi^2_{(2)} = 3.756$, $p = .153$). When one compared the proportion of individuals who reported no symptoms at Time-1 (28%) to those who reported no symptoms at time-2 (9.1%) it appears that those who responded at a later time were less likely to report no symptoms. Although there were no statistically differences in symptoms or LOS reported, it makes sense that the more ill of the sample would be less likely to participate at an adequate level over time. This is likely to have a suppressive impact on most of the social support and pathology variables. Males and females, according to chi squared tests, did not differ in the likelihood that they would participate at all four points in time ($\chi^2_{(1)} = 1.326$, $p = .250$). Cohabitation status did not appear to be related to whether someone participated at all four time points ($\chi^2_{(1)} = .071$, $p = .790$). Whether a participant had a psychiatric hospitalization history also did not predict who would participate with all four data collection points ($\chi^2_{(1)} = .293$, $p = .588$). In addition, the urban/rural distinction did not appear to be related to participation at the four measurement points ($\chi^2_{(1)} = .110$, $p = .740$). Participant employment status/variables also did not relate to whether a participant participated at all four points in time (employed full-time, $\chi^2_{(1)} = .001$, $p = .979$; employed part-time,

$\chi^2_{(1)} < .0001$, $p = .986$; unemployed looking for work, $\chi^2_{(1)} = .578$, $p = .447$; unemployed and not looking for work, $\chi^2_{(1)} = .208$, $p = .648$; retired, $\chi^2_{(1)} = .052$, $p = .819$; in school, $\chi^2_{(1)} = .060$, $p = .807$; maintaining a home, $\chi^2_{(1)} = 1.345$, $p = .246$; volunteering time, $\chi^2_{(1)} = .010$, $p = .921$; unemployed due to illness, $\chi^2_{(1)} = .140$, $p = .708$). This style of data analysis allows Type-I error rate to escalate. However, with this application this results in very conservative outcomes.

Some data were collected to discover what kinds of community/medical supports people relied upon when they were not hospitalized to help them deal with their psychiatric symptoms. Two-by-two chi square analysis were performed to evaluate whether the patient used a psychiatrist to help him or her cope did not differentiate those who did and did not participate at all four points in time ($\chi^2_{(1)} = 1.717$, $p = .190$). Most of the self care variables did not differentiate those individuals who did and did not participate at all four points in time (friends, partner, or relatives, $\chi^2_{(1)} = .130$, $p = .719$; self-help and crisis phone services, $\chi^2_{(1)} = .446$, $p = 504$; outpatient or day programs, $\chi^2_{(1)} = 1.118$, $p = .290$; the McKewan Program, $\chi^2_{(1)} = .983$, $p = .332$; social worker, counsellor, pastor, or paraprofessional, $\chi^2_{(1)} = 1.540$, $p = .215$). People reporting that they used exercise to help them cope with psychiatric symptomatology tended to be somewhat more likely (not significantly) to participate at all four points in time ($\chi^2_{(1)} = 2.005$, $p = .157$). There is one notable exception to all of these null findings. Individuals who reported using 'self-care' (for example, relaxation or getting additional sleep) were more likely to participate at all four points in time ($\chi^2_{(1)} = 6.894$, $p = .009$). This may be due to individuals who see themselves as more responsible for their care being more likely to

attempt all possible approaches to care making them more likely to see themselves as being responsible to participate in research.

When assessing the primary variables it became apparent that there were few differences between those who did and did not participate at all four points in time. Respondents' age did not differentiate those who did and did not participate in all waves ($t_{(231)} = -.400$, $p = .689$). Social support status also was not related to participation (SAT, $t_{(231)} = -.036$, $p = .972/2$; CF, $t_{(231)} = -.171$, $p = .865$). EMP and financial adequacy perceptions (FAD) also did not predict full compliance (EMP, $t_{(231)} = -.019$, $p = .984$; FAD, $t_{(231)} = .349$, $p = .727$). There were some predictable differences in who responded to all four waves. The literature clearly indicates that those individuals with higher income and higher education should be more likely to respond to all four waves (see Dillman, 1978). Given literature findings directional predictions are warranted and one-tailed tests were used. The present t-test results were in agreement with the literature in that those with more education (ED, $t_{(231)} = 2.80$, $p = .006/2$) were more likely to respond to all four waves as expected. Those with greater household incomes were also more likely to respond (INC, $t_{(231)} = -1.67$, $p = .095/2 = .048$). When pathology variables are considered, one would predict that those with less symptomatology, fewer inpatient stays, and lower average LOSs would be less likely to participate in all four waves. Symptomatology was not different for those who did and did not participate in all four waves of the project (SYMP, $t_{(231)} = .722$, $p = .471/2$). When assessing the hospitalization variables, those who failed to participate for all four waves had marginally significant increased number of hospital stays than those who did participate in all four assessment

points (number of stays in three years, $t_{(199.997)} = -1.643$, $p = .102/2$). However, when assessing the average LOSs in the last one and three year time frames, results indicate that there was no LOS difference between those who did and did not participate at all four points in time (average three year stay, $t_{(231)} = -.421$, $p = .674/2$; average one year stay, $t_{(231)} = -.785$, $p = .433/2$). When one compares those with and without any history of inpatient treatment and whether they did and did not participate at all four time points there is also no differences ($\chi^2 = .293$, $p = .588$).

Missing Data and Outliers

Through Time Missing Data and Outliers

For a review on possible missing data analysis techniques, the reader is referred to Roth (1994). According to Roth (1994), missing data cause two primary problems; a) lost data decreases statistical power, and b) missing data can also bias parameter estimates. "In many cases in applied psychology, missing data may bias correlation coefficients downward. The downward bias is most likely as high and/or low scores lost on either predictors or criteria restrict the variance in one variable and attenuate the correlation with another variable" (Roth, 1994, p. 538)."Some researchers may be willing to tolerate conservative parameter estimates to avoid added complexity" (Roth, 1994, p. 552).

One of the simplest approaches to deal with missing data is to delete the respondent from the analysis. Although this approach is easy it also has serious drawbacks. This approach results in large losses of data and correspondingly power for analysis (Gilley & Leon, 1991). List-wise deletion of missing data can also introduce

significant biases in parameter estimation (Little & Rubin, 1987).

Pair-wise deletion of missing data only eliminates data if one of the pair comparisons is missing in computing parameter estimates. This helps avoid throwing out the entire respondent's results just because of a few missing points and preserves data. However, pair-wise deletion can create bias problems when comparisons are made among statistics because different subjects are used in the computation of the statistical measures (Roth, 1994). This approach can also lead to mathematically inconsistent correlations and covariance matrices that are not 'positive definite' (Malhotra, 1987). Therefore, pair-wise deletion of missing data would not be appropriate when using path analysis since these techniques require covariance matrices to compute path coefficients.

According to Roth (1994), most of the research has focussed on cross-sectional designs and the use of the traditional missing data techniques can be less appropriate for repeated measurement designs. List-wise and pair-wise deletion results in losses of data that make analysis much less powerful and biassed. The research comparing missing data techniques for repeated measurement designs is not well developed. Medical researchers have tested several missing data techniques and have ranked the options from least to most biassed. According to the medical research results, the least biassed approach involves an interpolation of the nearest points of the same variable (Roth, 1994). There was also the option of using the average score of the subject (intra subject mean); however, this approach introduces more bias. None of the options tested by the medical researchers included an inter subject mean substitution through time.

There are a number of factors that must be considered in the choice of a missing

data technique. These issues include the amount of missing data, the accuracy and bias involved, the ease of use and time to implement solutions, and finally power (Roth, 1994). If there are few missing data points (less than 10% random or systematic, Malhotra, 1987) then the choice of technique makes little difference (Gilley & Leone, 1991). The choice of technique makes more difference as the proportion of data points that are missing increases. However, once again, this information is pertinent to single measurement designs not to repeated measurement designs.

To date, Missing Data Technique (MDT) research, especially for repeated measurements through time, is not conclusive and "the performance of any MDT depends heavily on the mechanisms that led to the missing values" (Little & Rubin, 1987, p. 39). In this case, deleting subjects with minimal missing data is not a viable option because the loss of power and bias introduced would be prohibitive. Deleting subject information on a pair-wise basis would also be problematic considering the difficulties of comparing different individuals and uneven sample sizes, especially if the statistical analyses depend on covariance and correlation matrices. Therefore, to maximize sample sizes and therefore power, reduce bias, allow for using correlational and covariance matrices, keep the analysis understandable, and maintain a conservative approach (Tabachnick & Fidell, 1989), an 'intra' subject mean substitution was used for missing variables at different points in time. This technique was used to replace missing values across the time periods for 5 data points allowing these participants to remain in the sample for all 4 of the waves.

Wave Specific Missing Data and Outliers

Time-1: All variables were examined for missing values and outliers. According to Tabachnick and Fidell (1989), in correlational and regression research using mean substitution for missing data will lead to more conservative beta weights and correlation coefficients. Attempting to find patterns in missing data is difficult when only a few data points are missing for any one case. Therefore, mean substitutions were used for missing values. Overall, the pattern of missing responses appeared to be random in nature, except that sicker individuals tended to leave more components unanswered. Having sicker individuals leave out more responses results in an under-estimation of variable variability and biases results and has a conservative impact on correlation and regression statistical analyses (Tabachnick & Fidell, 1989). Of the 232 Time-1 surveys considered for data analysis there were approximately 7,200 possible responses. This figure can only be approximated because of the nature of some survey questions (e.g., skip questions). Mean substitutions were made for 159 values, which is equivalent to approximately 2.5% of all the data points. Once again, if there are few missing data points (less than 10%; Malhotra, 1987) then the choice of technique makes little difference (Gilley & Leone, 1991).

There were no missing data for the contact frequency variable. There were 24 missing data points (24/232; 10.0%) for the satisfaction variable. These points were evaluated for patterns using a dummy variable coded as missing or not missing a satisfaction value. Individuals missing a score on the satisfaction variable were significantly higher in symptoms than those who provided satisfaction scores ($t_{(230)} = -2.742$, $p = .007$). Individuals who were missing the satisfaction variable also

reported lower contact frequency scores ($t_{(230)} = -3.028$, $p = .003$). There were only five missing responses in the hospitalization variable so little analysis was possible. Dummy coding (1, 0) for missing hospitalization information indicated that none of the primary variables were different for those leaving out the hospital information. The employment variable had only three missing values so little could be done with a missing values evaluation. The family income measure, as could be expected, was one of the more likely variables to be left blank with a total of 21 missing observations (9.0%). When all of the primary variables were compared between those who did and did not supply their household income some differences became evident. Individuals who failed to report their household income had shorter LOSs ($t_{(77.63)} = 3.049$, $p = .003$), were significantly older ($t_{(230)} = -2.123$, $p = .035$), and had significantly less education ($t_{(230)} = 3.358$, $p = .001$) than those participants who provided household income. However, when zero order correlation tables for the primary variables are considered there are no salient differences in the correlations with and without those missing the household income value. Given there were no salient differences in the zero order correlational relationships, and that mean substitutions are conservative (Tabachnick & Fidell, 2001), each missing data point for the income variable was replaced with the mean of the respective income question.

When the financial adequacy variable was assessed for missing values each component of the summed score were evaluated separately. There are 6 items that are summed to compute the financial adequacy variable they include; a) satisfaction with personal funds, b) satisfaction with funds for home, c) satisfaction with monies for loved

ones, d) satisfaction with funds for future needs, e) satisfaction with funds available for leisure activities, and f) satisfaction with funds for family needs. Item 'a' was missing 10 points (4.3%). Item 'b' was missing 12 points (5.2%). Item 'c' was missing 9 points (3.9%). Item 'd' was missing 6 points (2.6%). Item 'e' was missing 4 points (1.7%). Item 'f' was missing 12 points (5.2%). There were a total of 53 missing data points for the financial adequacy variable. None of the percentages of missing data are even close to the 10% value Malhotra (1987) mentions, so the choice of technique makes little difference (Gilley & Leone, 1991). Therefore, once again, inter-item mean substitutions were used to impute missing values for each of the components of financial adequacy. When the total final financial adequacy score is assessed for missing items results it is indicate that 29 of the 232 scores have at least one missing value. Subsequent analyses, comparing those with and without missing values on the financial adequacy scale, indicated that there were some differences in those who did and did not provide all data points. Those who did not comply with all items were lower in satisfaction with support ($t_{(230)} = 2.023$, $p = .044$), lower in employment score ($t_{(44.387)} = 3.10$, $p = .003$), reported lower household income ($t_{(230)} = 3.043$, $p = .003$), were older ($t_{(31.447)} = -2.801$, $p = .009$), and less educated ($t_{(230)} = 4.67$, $p < .0001$).

The symptomatology variable was missing 16 (6.9%) data points at Time-1. Once again inter-subject mean substitutions were used to impute the mean of the symptom variable. The mean was 3.08. When the primary variables are evaluated for patterns of missing symptomatology variable values it becomes evident that those who left the question blank were older ($t_{(230)} = 3.28$, $p = .001$), less educated ($t_{(230)} = -2.45$, $p = .015$),

and had lower employment scores ($t_{(21.347)} = -3.175$, $p = .004$). Given that these individuals are less educated, less employed and older they may be more incapacitated than the balance of the sample.

There were few outliers (those with values beyond three standard deviations from the mean of the variable) evident in the data; this was in part due to the restricted ranges of most of the variables involved. There were no multivariate outliers once univariate outliers were remedied. There were some extreme ages noted in the sample, but since age was not one of the primary variables and was excluded in the control variable analyses (using three age groupings) these outliers were ignored. Only the education and the three-year hospital stay data displayed any outliers. Two extreme data points were removed from the education variable and replaced with the mean of the education variable. There were extreme outliers in the three-year hospital stay variable. These outliers were not removed, but Spearman Rank Correlations were used to assess total effect rather than Pearson Correlation Coefficients. Since the Spearman Correlation is based on data ranks, it is not as influenced by outliers. This issue is discussed in more detail below.

Time-2: Overall, the pattern of missing responses appeared random in nature, except that more symptomatic individuals tended to leave more components unanswered, as in Time-1 responses. Of the 146 surveys considered for Time-2 analysis there were approximately 7,097 possible responses. Two surveys returned at Time-2 were almost blank and were deleted from further analysis. For the rest of the surveys, Time-2 inter-subject mean substitutions were made for 146 values, which is less than approximately

1.0% of all the data points. The family income measure, as expected, was the most likely variable to be left blank with a total of 9 missing observations (6.0%). Each missing data point was replaced with the Time-2 mean of the respective question or component prior to computing scale variables. There were no additional outliers requiring removal. There were no multivariate outliers at Time-2.

Time-3: Mean substitutions were used for 53 non-patterned missing values. Overall, the pattern of missing responses appeared random in nature, except that more symptomatic individuals, once again, tended to leave more components unanswered. Of the 123 surveys considered for data analysis there were approximately 6,000 possible responses. Mean substitutions were made for 53 values, which is equivalent to less than 1.0% of all the data points. The family income measure had a total of 8 missing observations (6.0%). Each missing data point was replaced with the Time-3 inter-subject mean of the respective question or component prior to computing scale variables. There were no additional outliers that needed removal. There were no multivariate outliers at Time-3.

Time-4: Mean substitutions were used for 58 non-patterned missing values. Overall, the pattern of missing responses appeared random in nature, except that, as in the first three waves, individuals reporting higher levels of symptomatology tended to leave more components unanswered. Of the 96 surveys considered for data analysis there were approximately 4,700 possible responses. Time-4 inter-subject mean substitutions were made for 58 values, which is equivalent to approximately 1.2% of all the data points. Unlike the other three waves, the most likely question to be left blank asked if the

respondent was satisfied with the money they had to 'spend on the people they loved?'. This question was missing in 8 of 96 possible responses (8.0%).The family income measure was the next most likely variable to be left blank with a total of 5 missing observations (5%). Each missing data point was replaced with the mean of the respective question or component prior to computing scale variables. There were no additional outliers requiring removal. There were no multivariate outliers at Time-4.

Distributional Assumptions

According to Tabachnick and Fidell (1989), for results to be valid, correlation and regression require that the variables involved are; a) linearly related, b) independent of one another, c) distributed in a similar manner (homogeneous), and d) normally distributed. Therefore, path analysis, based upon correlation and regression, require that the variables be normally distributed with similar standard deviations. Given these data, the observations over time are not independent of each other observation. Usually, one would want all measures to be independent of each other but these data are specifically designed to be related to data at subsequent points in time. Therefore, the data from a variable at a particular point in time is independent from all the other subjects' data at that point in time as required. However, the data across the time frames are not independent. Plots of all of the interrelated variables were obtained and indicated that the linearity requirement was tenable or a near zero relationship existed.

A sample size of more than 100 respondents is needed for the assumption of normality required by the path analytic procedure (Sobel, 1988). If sample sizes are

greater than 100 the sample is assumed to be taken from a normal population, thus validating the use of the Z-statistics and their corresponding p-values as tests of significance. Sample sizes for this research include: Time-1 (N = 232), Time-2 (N = 146), Time-3 (N = 123), and Time-4 (N = 96) samples. Therefore, we can assume normality. However, visual inspection of the detrended normality plots provided by SPSS indicated that almost all of the variables were distributed in a reasonably normal manner. There were the occasional exceptions.

The employment variable at the four points in time was not normally distributed due to a large percentage of individuals who reported that they had no employment. When one removed the individuals with zero employment the balance of the data were normally distributed. In an attempt to try to normalize the distribution of this variable a number of transformations (LN, SQRT, and EXP functions) were attempted with no successful improvements in normalization. Transforming this variable also does not make sense since it has a meaningful scale which would become less interpretable after a transformation (Tabachnick & Fidell, 2001). The other variable that there was difficulty with was three year length of stay (LOS). There were a few extreme outliers and once they were removed and means were substituted the variable conformed reasonably well to the assumption of normality.

Most of the variables used in this research had limited ranges over which they could vary leading to reduced variability resulting in conservative outcomes in linear regression and correlation. The largest range was for the hospitalization (LOS) variable with an extreme of 365 days. This value was later replaced with the mean of this

particular variable (see outlier treatment). Leaving out the employment variable, the other variables ranged from zero (see SAT, and SYMP) to a high of 100 for the Rand EWB scale. There was no indication of further outliers.

The next variable with a significant range was the employment variable. This value reached 156, which indicated that an individual had 52 weeks of full-time work and 52 weeks with part-time employment, as well. There were other difficulties with the employment variable with only 118 individuals having a non-zero employment score at Time-1. There were also difficulties with the assumption of homogeneity with the EMP variable due to the large number of individuals with a zero EMP score. To evaluate if the primary relationships between the variables would be different if only employed individuals were included; correlational analyses were conducted on the whole Time-1 group and the Time-1 group members with employment scores greater than zero (see Table 1 and Table 2). There appear to be some differences in zero order correlations based upon those who did and did not report whether they were employed. The distributional problem with the employment variable may be responsible for these findings resulting in unstable estimates of the measures involved leading to increased error and resulting in conservative outcomes. Distributional difficulties should be considered keeping in mind the descriptive exploratory nature of much of this literature.

Table 1

Correlation Coefficients Between Original Primary Variables with Employment Variable Intact. N = 232

	SS	ED	EMP	INC	FAD	SYMP
SS	--	+ .04 p = .504	+ .05 p = .451	+ .11 p = .086	+ .28 p < .001	- .45 p < .001
ED		--	**+ .24** **p < .001**	**+ .24** **p < .001**	- .02 p = .411	- .04 p = .560
EMP			--	+ .44 p < .001	**+ .11** **p = .042**	**- .23** **p < .001**
INC				--	+ .43 p < .001	- .17 p = .012
FAD					--	- .32 p < .001

Table 2

Correlation Coefficients Between Original Primary Variables Including Only Those With Non-Zero Employment Scores. N = 118

	SS	ED	EMP	INC	FAD	SYMP
SS	--	+ .00 p = .999	- .10 p = .265	+ .10 p = .285	+ .28 p = .003	- .35 p < .001
ED		--	**+ .02** **p = .984**	**+ .16** **p = .084**	+ .07 p = .432	- .02 p = .820
EMP			--	+ .43 p < .001	**+ .20** **p = .026**	**- .10** **p = .263**
INC				--	+ .42 p < .001	- .18 p = .047
FAD					--	- .41 p < .001

Note. These notes pertain to Tables 1 and 2 above. Abbreviations are as follows: p = two-tailed probabilities, SS = compound measure of social support, ED = years of formal schooling, EMP = employment score, INC = Family income score, FAD = Financial Adequacy Score, and SYMP = Symptomatology measure.

Control Variables

Numerous variables have been found to influence psychiatric symptomatology and hospitalization including: age, gender, marital status, and urban versus rural living. The literature is not clear whether these effects are causal or are due to the influence of related variables. The literature pertaining to the influence of each of the aforementioned variables is discussed below and this section concludes with measures taken to ensure that any possible effects that existed did not interfere with conclusions about the predictor variables from this research.

Age

In general, epidemiological studies of community samples demonstrate a curvilinear association between age and depressive symptomatology, with the rates being higher for seniors and young adults (Schwab et al.1973; Dresler, 1986).The relationship of age with depression has been challenged because other depression-related variables are correlated with age (Blazer et al. 1991). For example, disability has been found to correlate with both age and depression (Berkman et al. 1986). Disability then may be the explanation of the relationship between depression and an advanced age (Blazer et al. 1991). Also, since individuals with lower levels of education (Kovess, Murphy, & Tousignant 1987) and employment (Perrucci et al. 1988; cited in Hamilton, 1993) are at higher risk for depression, and given that younger people are more likely to be both less educated and have lower employment rates, then the correlation of age with employment and education may also explain the higher rates of depressive symptomatology in the

young. Higher risks of violence are also associated with being younger (Beauford, McNiel, & Binder, 1997).

In general, older adults do not lose social support as they age (Lang, 2000). Some researchers have concluded that the participants' present age is not causally influencing depressive symptomatology because, once the influences of other variables are considered, the relationship between depression and age loses its significance (Blazer et al. 1991). In earlier research age apparently has no influence on the effects of social networks (Olsen, 1993) or the relationship between support and well-being (Vaux, 1985). Age has been found to be inversely related to emotional well-being (Clark et al. 2003). Perceived social support were significantly linked with children's self-report psychological symptoms (McLeister, Anant, & Barnett, 1999). Social isolation, loneliness, and lack of social support diminish well-being and physical health among the retired and elderly population (G. D. Cohen, 2000; Sorkin, Rook, & Lu, 2002). However, elderly females showed positive associations with quality of social contacts and subjective well-being (Pinquart & Sorensen, 2000). Elderly living in dilapidated neighbourhoods receive less social support and encounter more negative interactions, however, lack of social support and negative interactions are completely offset for older individuals who possess strong social skills (Krause, 2006). Perceives satisfaction with support was more strongly related with well-being in the older adults (Kafetsios & Sideridis, 2006). Link (1982) also excluded age and gender from a model explaining psychiatric impairment and patient status because their effects were not significant.

Gender

In general women have been found to suffer more depressive symptomatology than men (Radloff & Rae, 1979). Females were more likely to have neurotic depressive symptomatology (Brodaty et al. 1997). The literature indicates that this phenomenon may be due to women being subjected to more 'depressing life circumstances' (Gander & Jorgensen, 1990; Maffeo et al. 1990). Adverse social conditions, in sufficient magnitude or in interaction with predisposing conditions, can lead to depression (Gander & Jorgensen, 1990; Maffeo et al. 1990).Women's friendships outside the home do not change as they age (Field, 1999). According to Field (1999), for men, friendship and nonfamily activities decline in males as they age. Among males, peer support was positively associated with depressive symptoms and suicidal ideations (Kerrr, Preuss, & King, 2006). According to Taylor et al. (2000) and Unger et al. (1999), gender is one of the most robust predictors of the use of social support, females make use of social support more often than males (Gurung, Taylor, & Seeman, 2003). According to Greene (1987), gender differences in depression scores are less likely to be observed if other variables that may modify the situation are controlled. For example, men tend to die earlier than women which can leave females alone and reactive depressions due to loss of a spouse can be an issue. Maffeo et al. (1990) reported that no gender differences in depressive symptoms remained after the effects of salary, age, and education had been accounted for, suggesting that overall gender differences in depression were moderated by social conditions. Goulding (1988) stated that the gender differences found in depressive symptomatology lack robustness and that, when differences occur, they can be explained

indirectly through differences in employment, income, and education.

For example, unemployed people report more depressive symptomatology than employed individuals (Eaton & Kessler, 1981; Gore & Mangione, 1983). According to Brown and Harris (1978), employment (among other factors) protects women from depression. Unemployed females have been found to have higher levels of depression (Kessler & McRae, 1982) and lower levels of social support (Hall & Johnson, 1988) than employed females. Nathonson (1980) and Waldron (1980) have suggested that the positive effects of employment on health (for women) is due to a reduction in isolation. However, men are not immune to the effects of social interaction and support provided by employment. Bolton and Oatley (1987) found unemployed men who developed depressive symptomatology interacted less socially, prior to unemployment, than non-depressed jobless males.

Women today, including mothers, face different role prescriptions than those maturing in the past in that they are expected to be employed (Reading & Amatea, 1986). According to Murphy (1986), historical trends have shown that as men's and women's employment rates have converged (women are now employed out of the home more than in the past) so have depression scores (resulting in reduced depression scores in women).

Low income (Eaton & Kessler, 1981) and less education (Ross & Huber, 1985) are also positively associated with economic hardship and depressive symptomatology. Ross and Huber (1985) found the interactive effect of low income and less education on economic hardship was especially salient for married females. The literature indicates that a significant negative association exists between education and depressive symptoms for

males (Craig & Van Natta, 1979; Radloff & Rae, 1979) and male adolescents are at an even higher risk for suicide since teenage males are also more likely to successfully commit suicide than their female counterparts (Dicker et al. 1997; Hirchfeld & Russell, 1997, Harwitz & Ravizza, 2000; Ryb, Sonderstrom, Kufera, & Dischinger, 2006). Yet, a "man's education is important to his psychological well-being only in that it increases his income and decreases perceived economic hardship. A woman's education plays an independent role in her psychological well-being" (Ross & Huber, 1985, p. 324). Employed men and women were also more likely to feel that their finances were more adequate. However, men were slightly more likely than women to feel their incomes were inadequate (Keith, 1985). According to Goulding (1988), white women and men in a community population tend to be more similar than different in levels of depressive symptomatology, but women are more likely to be demographically disadvantaged. In other words, they are less educated, more likely to be unemployed, and have lower income levels. Women seem to benefit from giving support in intimate relationships, and men seem to benefit from receiving support inn intimate relationships, suggesting that being a provider of support by itself may be positively related to female well-being by increase in self-esteem (Yaananen, Buunk, Kivimaki, Pentii, & Vahtera, 2005). In general, men have been found to reap greater benefits from social ties than do women (Unger et al. 1999). Therefore, women are more likely to have extreme depression scores (Goulding, 1988). These relations may be impacted culturally, the association between social support from others and life satisfaction was observed more among Japanese males than females and Japanese males also get more support from their families (Wang, Iwaya,

Kumano, Suzukamo, Tobimatsu, & Fukudo, 2002). A Japanese males' social support has been found to reduce depressive symptoms under stressful circumstances and reciprocal support (giving and receiving) is important for the mental health of these males (Takizawa, Kondo, Sakihara, Ariizumi, Waranabe, & Oyama, 2006).

Marital Status

The effect of marriage on psychiatric symptomatology is unclear. According to Ross and Huber (1985), the inverse effect of marriage on depression may be partially explained by an increase in social support. Yet, other researchers report no significant differences in social support based upon marital status (Neff & Karney, 2005; Roberts &Greenberg, 2002). Divorce has been found to be related increased suicide rates (Kposowa, 2000). Marital quality rather than status is important for positive mental health (Spotts et al. 2005). Marriage, satisfaction with one of life's most intimate relationships, has been found to have a significant inverse relationship with depression (Whisman, 2001; Whisman, Sheldon, & Goering, 2000). Living alone has been found to be related to lower levels of social support (Williams et al. 2004). Married people have been found to have higher emotional well-being (Clark et al. 2003).

Yet, Hall and Johnson (1988) warned against assuming that marriage necessarily functions in a supportive manner by reducing the impact of stressful life events. Also, Krause, Laing, and Keith (1990) found that the direct, indirect, and total effects of marital status on psychological distress were not statistically significant. Being married has also been found to have protective health effects (Waite & Gallagher, 2001), and being

married was found to predict lower levels of loneliness (Hughes et al. 2004).

However, Melchoir, Berkman, Niedhammer, Chea, & Goldberg (2003), believe that the protective effects of marriage is operating through both socioeconomic as well as psychological variables. According to Pasch and Bradbury (1998), spouses that received higher support levels also reported greater marital satisfaction. Living alone has been found to be related to lower levels of social support (Williams et al. 2004). Those with higher social support also experienced better longitudinal marital outcomes (Conger, Rueter, & Elder, 1999; Neff & Karney, 2005). Unsupportive interactions may be detrimental to well-being and more positive marital interactions were predictive of lower depressive symptomatology (Druley & Townsend, 1998). According to DeLongis, Capreol, Holtzman, O'Brien, and Campbell (2004), positive day-to-day marital interactions are related to greater happiness. According to Kiecolt-Glaser and Newton (2001), marital satisfaction is positively related to happiness. Spousal strain was positively related to negative affect (Okun & Keith, 1998). Spouses reported that wives provided better support on days that their husbands experiences greater stress (Neff & Karney, 2005). However, when wives experienced greater stress, their husbands displayed both support and negativity (Neff & Karney, 2005).

According to Davis (1985), marriage rates have also been found to interact positively with age, with lower rates of marriage among younger subjects. Davis explains these findings as resulting from one of two possibilities. Either, older individuals have had more opportunity to get married simply by virtue of time, or marriage acts as a screening device, excluding those who are more disabled and less socially competent. As

individuals mature, they become more competent socially and financially making them more attractive marriage partners. A disabled person's development (social and economic) may lag behind other non-disabled individuals in the same age grouping making them less desirable partners.

The literature pertaining to the influence of marriage upon psychiatric symptomatology is unclear. Patients who have been admitted for inpatient treatment tend to be less likely to have partners (Murray, 2002). However, the effect of marriage on psychiatric hospital stays is unequivocal. Married individuals, regardless of gender, spend significantly less time in inpatient treatment, possibly indicating an increase in coping ability (Shanks & Atkins, 1985). Married individuals have also been found to be admitted less than the unmarried (Pilisuk, 2001). Does the availability of a spouse may act as a care-giving resource? According to Cochrane and Stopes-Roe (1981), in a British sample, marital status, for both sexes, was also associated with a lower risk of being admitted for inpatient psychiatric treatment. However, according to Roberts and Greenberg (2002), others researchers have reported no significant differences between husbands' and wives' supportive behaviors (Neff & Karney, 2005). According to Williams et al. (2004), marriage appears to provide more benefits for men and males with multiple sclerosis have been found to report higher levels of total support than women.

Urban versus Rural Residence

According to D'Arcy (1976), norms for deviance are better defined in urban areas where a wider variety of individuals may be labelled as mentally ill. This rural lack of

recognition of mental illness may be due to a combination of factors, such as lower education levels and social isolation, resulting in the recognition of only the most deviant behaviour, such as schizophrenia (D'Arcy & Brockman, 1976; Edgerton & Bentz, 1969).

According to Mueller (1981), the literature on differences in depression rates in urban versus rural settings is equivocal. Research focussing on symptoms of depression has not yielded consistent results, while surveys assessing depression as a syndrome appear to find consistently that urban dwellers report higher prevalence rates (Henderson & Pollard, 1992). Urban and rural populations often differ on many demographic variables that are related to the risk of depression (Neff, 1983). Measures of social networks tend to differentiate less in rural areas since friends and relatives are likely to be seen at work or while shopping (Olsen, 1993). A rural birthplace has also been found to correlate with good employment and social outcomes (Munk-Jorgensen & Mortensen, 1992).

In a Quebec metropolitan sample, Kovess et al. (1987) found, "a significantly greater risk of major depressive episodes than either the countryside or the county town samples after allowance had been made for sex, education, employment status, marital status, and experience of negative life events" (p. 461). According to these authors, those living in a metropolitan area had greater risk of depression than either a small town or a rural population. Yet, these researchers did not find a difference in satisfaction with or frequency of social interaction except in church going. Country residents tended to attend church more frequently than urban dwellers. "Consequently, neither frequency of nor satisfaction with social interactions seems likely to explain the urban-rural differences in

rates of depression" (Kovess et al. 1987, p. 462). Ellison and Levine (1998) pointed out, the health-related impact of religion may be explained, in part, by social relationships that flourish in church settings. However, according to Krause, Ellison, and Wulff (1998), that there is often significant interpersonal conflict in churches and this negative interaction may exert a negative effect on well-being. Hostile behaviour may decrease social support availability because the hostile person may increase interpersonal conflict and social stress (O'Neil & Emery, 2002).

There are few mental health services or supports that exist outside major urban centres. In Manitoba, people with psychiatric disorders tend to congregate in the major urban centres and Winnipeg is known to have a better range of psychiatric services than either rural areas or smaller urban settings. According to Mueller (1980), increased rates of depressive symptomatology in urban settings may be due to increased isolation resulting in a lack of social support leading to depression (see Brown & Harris, 1978). Kovess et al. (1987) attributed much of the difference in depression rates in metropolitan areas to the large number of unemployed males. These researchers found that the unemployed experience more depression than the currently employed and that the urban unemployed suffer much more than their rural counterparts.

According to MacDonald, in a 1992 survey of the Mood Disorders Association of Manitoba, there were no differences in symptomatology measures pertaining to manic or depressive symptomatology resulting from age groupings, gender, or urban/rural residence. According to literature reviews by Hartmann (1988) and Moss (1989) (as cited in McDonald, 1992), there was little difference between males and females, rural or urban

dwellers, or between age groupings in the degree of impairment from mood disorders. These findings do not agree with Kerrr et al's. (2006) findings that support was positively associated with depressive symptoms and suicidal ideations in males.

Assessing Control Variables

Age groupings were created to evaluate the impact of age upon the primary variables. Age groupings were decided upon once the range (12-113 years of age) of the variable was determined. The age range covers 100 years; both the 12 and 113 year old are extremes, but there was no logical reason to eliminate them from the total sample. The other information given by these individuals was congruent with their reported ages. A three-way split was performed to ensure approximately equal sample sizes in each age grouping. Group 1 consisted of individuals 12 to 39 years of age ($N = 72$, $M = 31.8$, $SD = 1.90$), Group 2 consisted of individuals 40 to 50 years old ($N = 83$, $M = 44.5$, $SD = 1.75$), and finally Group 3 was made up of individuals 51 to 113 years of age ($N = 77$, $M = 62.4$, $SD = 1.83$). There were no significant differences in either symptomatology, $F_{(2, 75)} = .87$, $p = .423$, or number of nights of inpatient hospitalization treatment during three years, for the three age groupings, $F_{(2, 75)} = 1.499$, $p = .230$. This finding does not support Clark et al's (2003) finding that age was inversely related to emotional well-being. The raw age variable (for the entire sample) did correlate negatively with number of nights in an inpatient treatment facility ($r = -.16$, $p = .015$ two-tailed). This negative correlation could be due to the disease patterns common in both unipolar and bipolar depression. Clients who are diagnosed with these disorders

usually begin to display symptomatology in the late teen years (Fitzpatrick, Piko, Wright, & LaGory, 2005; Goodwin & Jamison, 1990) and it takes time for diagnosis and treatment to have an effect. Many clients spend years finding just the right treatment for their particular symptomatology pattern. However, when one considers only those with a hospitalization history (excluding outliers) during the last three years the negative correlation between age and hospital stay becomes nonsignificant ($r_{(Pearson,\ 76)} = -.148$, $p = .195$).

The sample was then split dichotomously according to respondents' gender, marital status (cohabiting with a partner/not cohabiting with a partner), diagnosis (unipolar/bipolar), and hospitalization history (yes/no). Males and females did differ in symptomatology reported. Females tended to report more symptomatology during the two weeks prior to the survey, $t_{(228)} = 2.78$, $p = .006$ (equal variance two-tailed test). However, there were no gender differences in the length of time spent in an inpatient treatment facility over the last three years, $t_{(219)} = .20$, $p = .839$ (equal variance, two-tailed test). Marital status results indicated that there were marginally significant differences in self-reported symptomatology based upon marital status. The literature is not clear whether married individuals are less symptomatic than their single counterparts. However, present results indicated that single individuals tend to report slightly more symptoms [M(single) = 3.33, SD = 1.86; M(cohabitating) = 2.86, SD = 1.76; $t_{(219)} = 1.96$; $p = .051$ equal variance, two-tailed test]. This test was marginally significant.

The expected marital status difference in length of hospitalization did not hold for the current sample. Married individuals have been found to be admitted less than the

unmarried (Pilisuk, 2001). Married individuals (in the entire sample) spent slightly, but not significantly, less time in an inpatient treatment facility during the last three years [M(single) = 18.87 nights, SD = 45.21; M(married) = 17.19 nights, SD = 38.21; $t_{(232)} = -.30$; $p = .764$, equal variance, two-tailed test]. Married individuals in this sample (who reported an inpatient stay during the last three years) tended to spend a little more time in inpatient treatment [M(single) = 47.9 nights, SD = 62.01; M(married) = 54.64 nights, SD = 51.24; $t_{(76)} = .52$; $p = .602$, equal variance, two-tailed test] although the difference was not significant.

This aberrant finding may be due to the population being considered in the current sample. These individuals are members of a self-help group indicating a propensity for help-seeking or at least an absence of fear in obtaining help. It is possible that members of these groups are more supported by their partners and therefore they are more inclined to seek help, are more effective in seeking treatment, or have partners who are more effective in delaying early discharge. According to Williams et al. (2004), living alone was also related to lower levels of social support. The positive impact of social support can be diminished by conflict in one's social network (Okun & Keith, 1998). It is also likely that this group of clients is better supported than persons who are not affiliated with a self-help group. Single individuals in the group may also receive more support from group membership equating their support with those of married individuals. Many people are afraid to participate in self-help groups because admitting that one is mentally ill could bee seen as a dangerous secret, because of stigma, that they don't want everyone to know (Davison, Pennebaker, & Dickerson, 2000; Griffiths, et al. 2004). Self perceived

stigma have been found to be one of the most important factors influencing people to avoid or delay seeking treatment for mental illness (Cooper, Corrigan, & Watson, 2003; Barney, Griffiths, Jorm, & Christiansen, 2006; Nolan & Badger, 2006; Schraufnagel, Wagner, Miranda, & Roy-Burne, 2006).

The entire sample of 232 respondents consisted of two primary diagnostic groups, unipolar and bipolar depressive disorders. The severity of psychiatric symptomatology is usually more intense in the bipolar group because of the extreme points in the spectrum from mania to depression (Goodwin & Jamison, 1990). There are four classic features defining mania, including euphoric mood, psychomotor pressure, grandiosity, and irritable aggression (Cassidy et al.1998). When considering only the group with a measure of psychiatric hospitalization during the last three years, bipolar individuals spent slightly, not significantly, more nights in inpatient treatment facilitiesthan non-bipolar patients [$\underline{M}$(unipolar) = 43.6, $\underline{SD}$ = 43.2; $\underline{M}$(bipolar) = 57.5, $\underline{SD}$ = 68.9; $t_{(76)}$ = -.95; $\underline{p}$ = .17; equal variance, one-tailed test]. This result likely failed to attain significance due to a lack of power and due to the impact of extreme standard deviations. In examining the entire sample it becomes more evident that clients with bipolar disorders spend significantly more nights in inpatient facilities [$\underline{M}$(unipolar) = 12.8, $\underline{SD}$ = 30.5; $\underline{M}$(bipolar) = 24.5, $\underline{SD}$ = 52.9; $t_{(219)}$ = -1.81; $\underline{p}$ = .04 unequal variance, one-tailed test]. The variances were also not equal. The bipolar group three year hospital stay results were significantly more variable according to Levine's test for homogeneity, $F_{(2, 229)}$ = 6.76; $\underline{p}$ = .01. An analysis of hospitalization history and diagnostic groupings demonstrated that those with a bipolar diagnosis were more likely to have a

hospitalization history ($\chi^2_{(2)} = 10.03$, $p = .007$) than those with a unipolar diagnosis.

The symptomatology measure results were the opposite of the hospitalization findings. It appears that, during the two weeks prior to respondents receiving the surveys, unipolar clients reported experiencing significantly more severe symptomatology [M(unipolar) = 3.62, SD = 1.82; M(bipolar) = 2.76, SD = 1.75; $t_{(192)} = 3.34$; $p = .001$, equal variance, two-tailed test].

The total sample was then split based upon whether the respondent indicated they had a history of psychiatric hospitalization. One would assume that clients with a psychiatric hospitalization history would report more psychiatric symptomatology than individuals who have never been hospitalized for psychiatric treatment. This was not the case for the current sample. For the two weeks prior to receiving the survey, respondents with a psychiatric hospitalization history reported slightly lower measures of recent self-report symptomatology than their non-hospitalized counterparts [M(hospitalized) = 2.92, SD = 1.85; M(never hospitalized) = 3.24, SD = 1.75; $t_{(224)} = -1.30$; $p = .194$; equal variance, two-tailed test].

For the purposes of this research, since there is no clear guideline in the literature (Kovess et al. 1987), postal code designations were used as the criterion for making the urban/rural distinction. Statistics Canada defines rural areas as those lying outside urban areas. An urban area is defined by a population of at least, 1000 people; but with a density of at least 400 persons per square kilometre. All areas lying outside the urban areas are defined as rural. According to Statistics Canada (1991), the postal code is a six-character alphanumeric code defined and maintained by the Canada Post Corporation for the

processing of mail. The second character of the postal code will be a zero if the post office has designated the corresponding area as a rural area. Characters other than a zero indicate urban designations.

Urban/Rural distinctions were made after the surveys were returned based upon the postal code designation on the master mailing list. Results of the independent samples t-test indicated there was no significant mean difference in either symptomatology [M_{urban} = 3.14, SD = 1.79; M_{rural} = 2.66, SD = 1.70; $t_{(213)}$ = -1.57, p = .117; equal variance, two-tailed test] or number of nights hospitalization [M_{urban} = 18.90, SD = 43.54; M_{rural} = 14.37, SD = 34.68; $t_{(221)}$ = -.62, p = .535; equal variance, two-tailed test] based upon the urban versus rural living situation. An evaluation of those who were and were not hospitalized was made, dependant upon urban rural distinctions. Results indicated that there was no difference in the proportions of individuals hospitalizes based upon rural status ($\chi^2_{(1)}$ = .241, p = .886).

The control variables will not be discussed any further at this point. Suffice to say that although Mai et al. (1993) found age, sex, and marital status influenced the length of psychiatric hospital stays, the current results did not indicate a differential pattern of influence when these variables were controlled. The control variables are likely to act upon an individual simultaneously so the contributions of the control variables impact were assessed all at once. The entire sample was split according to the gender, age, marital status, urban/rural residence, diagnosis category, and whether the respondent had ever been treated in an inpatient psychiatric facility. A partial correlation analysis was performed to assess the influence of the control variables. The partial correlation patterns,

when all the control variables were included, were compared to the raw zero order correlations without controls (see Table 3 and Table 4). Partial correlational procedures are related to multiple linear regression and provide a measure of linear association between two variables while adjusting the linear effects of the additional variables. The results indicated that controlling for all the effects of the possible confounding variables did not significantly change the pattern of the zero order correlation coefficients between the variables to be used in the models. Visual analysis of the correlations with and without the control variables indicated that they were of the same magnitude and valence [except for small zero effect correlations; i.e., education and satisfaction with support]. Near zero correlations did vary between positive and negative valences. However their magnitudes did not change. Large positive correlations with control variables were still large positive relationships without the control variables held constant. Moderate positive correlations were still moderate and positive after including the control variables in the partial correlation procedure. There was some vacillation between correlations that were on the edge of the line demarking small and moderate correlations (Cohen & Cohen, 1983).

Table 3

Zero-Order Correlation Coefficients Between Original Time-1 Variables, N = 232

	CF	SAT	ED	EMP	INC	FAD	SYMP	LOS**
CF	- -	+ .46 p < .001	+ .11 p = .052	- .03 p = .314	+ .05 p = .237	+ .19 p = .002	- .32 p < .001	+ .07 p = .139
SAT		- -	- .001 p = .495	+ .10 p = .056	+ .14 p = .018	+ .28 p < .001	- .44 p < .001	+ .09 p = .091
ED			- -	+ .24 p < .001	+ .23 p < .001	- .02 p = .411	- .04 p = .260	- .03 p = .325
EMP				- -	+ .44 p < .001	+ .11 p = .042	- .23 p < .001	- .17 p = .006
INC					- -	+ .43 p < .001	- .17 p = .006	+ .01 p = .435
FAD						- -	- .32 p < .001	+.02 p = .378
SYMP							- -	0.16 p = .007

Note. **This column has only participants who had an inpatient stay during the past three years. The abbreviations are as follows: p = two-tailed probabilities, CF = contact frequency, SAT = satisfaction with support, ED = years of formal schooling, EMP = employment score, INC = Family income score, FAD = Financial Adequacy Score, DRG = diagnostically related groups, SYMP = Symptomatology measure, and LOS = Length of Inpatient Stay. All probabilities are two tailed for the appropriate values related to directional predictions this value needs divided by two.

Table 4

Partial Correlation Coefficients After Controlling for the Effects of Age Grouping, Hospitalization History, Postal Code, Marital Status, DRG, and Gender for Time-1 Variables, N = 232

	CF	SAT	ED	EMP	INC	FAD	SYMP	LOS**
CF	--	+ .46 p <.001	+ .12 p =.039	- .08 p =.130	+ .09 p =.087	+ .23 p <.001	- .33 p <.001	+ .05 p = .245
SAT		--	+ .028 p =.344	+ .06 p =.197	+ .13 p =.036	+ .28 p <.001	- .40 p <.001	+ .05 p = .230
ED			--	+ .18 p =.007	+ .23 p <.001	+ .07 p =.148	- .09 p =.093	- .04 p = .306
EMP				--	+ .35 p<.001	+ .11 p =.063	- .17 p =.012	- .19 p = .003
FIN					--	+ .36 p<.001	- .11 p =.066	+ .07 p = .146
FAD						--	- .28 p<.001	+.08 p = .136
SYMP							--	0.22 p < .001

Note. **This column has only participants who had an inpatient stay during the past three years. The abbreviations are as follows: p = two-tailed probabilities, CF = contact frequency, SAT = satisfaction with support, ED = years of formal schooling, EMP = employment score, INC = Family income score, FAD = Financial Adequacy Score, DRG = diagnostically related groups, SYMP = Symptomatology measure, and LOS = Length of Inpatient Stay. All probabilities are two tailed for the appropriate values related to directional predictions this value needs divided by two.

When the correlations, with and without the impact of the control variables, were compared it became apparent that the control variables did not drastically alter the pattern of correlations among the variables being considered. At Time-1of the 28 zero-order correlational (see Time-1 on Table 3 and Table 4 above for the correlations specific to other points in time the reader is referred to Appendix B, Time-2 on Table B1 and Table B2 Time-3 on Table B3 and Table B4, and Time-4 on Table B5 and Table B6) 16 were significant. This compares to the same 16 significant relationships in the controlled correlations at Time-1. Differences that did exist in the size of the significant correlations between the two correlational patterns were not consistent. So, no clear advantage was seen in including the control variables. At this stage of the analysis the decision was made to ignore the control variables in further analysis. There is an advantage in being able to ignore the influence of the control variables. This simplifies the model testing process and makes program evaluation and planning decisions, based upon these parsimonious models, more definitive.

Reliability and Validity of Measures

Scale Reliability and Validity Issues

Since support measures must be chosen and/or designed based upon the research question of interest (Cohen & Syme, 1985) few data are available on the psychometric qualities of the various measures of social support, social functioning, or symptomatology employed in this research. Because of time and space limitations, and because of the

limits on our participants abilities to respond, the variables used in this research had to be very simple to complete and brief. Also, according to House and Kahn (1985), there is "no criterion validity against which measures of social relationships, networks, and support can be assessed" (p. 86). These authors suggest that the best test of the validity of a social support measure is whether it significantly predicts a health outcome. For example, according to House and Kahn (1985), "the litmus test of the validity of any measure of social support is it's [sic] relationship to the major causes and consequences of social support"(p. 87), in this case, psychiatric symptomatology and the length of psychiatric hospital stays.

Scale Reliability

Although "high reliability, in terms of both internal consistency and test-retest stability, is a desirable feature of any measure and a crucial precondition of validity" (House & Kahn, 1985, p. 85), unfortunately there are few scale reliability measures available for the existing social support or the financial adequacy variables created for this research. However, internal reliability analyses were conducted on scale variables (e.g., Cronbach's Alpha) and the sample was partitioned into two sub-samples (odd survey numbers in one sample and even ones in the other) before scale score split-half reliability measures were evaluated. This procedure should minimize error based on chance and test the replicability of the findings for each scale (Krause et al. 1990; Finifter, 1972).

Contact Frequency. The contact frequency (CF) measure consisted of the following four components: the number of times respondents had gone out with others, the number of times they got together with friends and family, the amount of contact with friends and family (e.g., phone calls), and the number of times they depended on friends and family (requiring contact). The four Time-1 components of the aggregate correlated with each other significantly with moderate to large correlation values ($\underline{r}$ = .42 to .58 probability values < .001 two-tailed, single factor variance accounted for = 60.05%). The Cronbach's Alpha for the four components of the contact frequency variable was $\underline{r}$ = .78. Split-half reliability (using an odd even split based upon the survey numbers) for the two sets of data and the four items ranged between $\underline{r}$ = .71 and .74. Time-2, Time-3, and Time-4 results were similar. Time-2 inter-item correlations ranged from .30 to .56 with item deleted values ranging from .65 to.73. Overall alpha for the Time-2 CF measure was $\underline{r}$ = .74 (single factor variance accounted for = 58.54%). Time-3 inter-item correlations ranged from .33 - .58 with item deleted values ranging from .67 to .76. Overall alpha for the Time-3 CF measure was $\underline{r}$ = .77 (single factor variance accounted for = 73.65%). Time-4 inter-item correlations ranged from .36 to .61 with item deleted values ranging from .69 to .78. Overall alpha for the Time-4 CF measure was $\underline{r}$ = .78 (single factor variance accounted for = 60.85%). Therefore, for Phase-III of this research, the CF variable, after a confirmatory factor analysis results, was combined with the SAT variable to produce a compound measure of social support (SS) and scale reliability was reevaluated.

Time-1 inter-item correlations ranged from .27 to .58 with item deleted values

ranging from .70 to .78. Overall alpha for the Time-1 SS measure was r = .77. Time-2 inter-item correlations ranged from .18 to .56 with item deleted values ranging from .65 to .74. Overall alpha for the Time-2 SS measure was r = .73. Time-3 inter-item correlations ranged from .11 to .58 with item deleted values ranging from .63 to .76. Overall alpha for the Time-3 SS measure was r = .72. Time-4 inter-item correlations ranged from .35 to .60 with item deleted values ranging from .71 to .77. Overall alpha for the Time-4 SS measure was r = .79.

Financial Adequacy. The financial adequacy (FAD) variable was computed by summing the responses from six questions evaluating how satisfied the respondents were with their financial status. The components evaluated how satisfied respondents were with funds available for their personal needs, their home, their loved ones, for the future, for leisure, and for their family's needs. Time-1 correlations among the sub-components of the FAD measure ranged between r = .72 and .86 (probability values were all < .001 two-tailed). All of the six components were related to each other with approximately the same strength so no modification of the variable components was necessary. Overall scale reliability for Time-1 was high, Cronbach's Alpha was equal to r = .93 and scale split-half reliability measures ranged between r = .90 and .94. Time-2 correlations among the sub-components of the FAD measure ranged between r = .67 and .87 with item deleted values ranging from .93 to .95. Overall scale reliability for Time-2 was high, Cronbach's Alpha was equal to r = .95. Time-3 correlations among the sub-components of the FAD measure ranged between r = .68 and .86 with item deleted values ranging from .94 to .95. Overall

scale reliability for Time-3 was high, Cronbach's Alpha was equal to r = .95. Time-4 correlations among the sub-components of the FAD measure ranged between r = .77 and .93 with item deleted values ranging from .95 to .97. Overall scale reliability for Time-4 was high, Cronbach's Alpha was equal to r = .97.

Symptomatology. At Time-2, -3, and -4, to help with assessment of the validity and reliability of a global single-item segmented visual analog symptomatology measure, the emotional well-being component of the Rand 36-item Health Survey (EWB; Hays et al. 1993) was added to the measures sent to clients.

The EWB measure (discussed previously) has been used in numerous populations to assess EWB. The EWB has also been found to be reliably able to discriminate those who are depressed in a group of chronically ill individuals supporting the validity of the measure. There are also standardized scores available for comparison purposes (Hays et al. 1993). Chronbach's Alpha internal consistency measure ranges from alpha = .76 to .90 for most usages of the sub-scale (Hays et al. 1993). These correlations demonstrate that the Rand EWB score functions with well for this data demonstrating both test retest reliability and stability. At Time-2 Time-3, and Time-4 Cronbach's alphas for the EWB variable were .90, .91, and .90 respectively. Means and standard deviation scores on the EWB variable for Times-1, -2, and -3 were M = 56.12, SD = 23.35; M = 56.20, SD = 23.43; and M = 54.35, SD = 23.89, respectively. Males and females also demonstrated equivalent scores at each point in time (see Table 7 below).

Both the EWB and SYMP variables were reasonably normally distributed upon

visual inspection of the normality plots provided by SPSS. It appears that the single-item global symptomatology measure did well when compared with the EWB component from the Rand 36-item Health Survey (Hays et al. 1993). This means that both measures appear to perform similarly at Time-2, Time-3, and Time-4 (see Table 5 below). Standardized scores for the Rand EWB measure can be found on Table 6 (below).

Table 5

Relationships Between the Global Symptomatology Measures and the Rand EWB (Hays et al. 1993) Scores with the Total Samples Available at Time-2, Time-3, and Time-4

	EWB Score Time-2	EWB Score Time-3	EWB Score Time-4
Symptom-Time-1	r = -.46 n = 145 p < .0001	r = -.49 n = 120 p < .0001	r = -.44 n = 95 p < .0001
Symptom-Time-2	**r = -.68** **n = 124** **p < .0001**	r = -.63 n = 93 p < .0001	r = -.41 n = 73 p < .0001
Symptom-Time-3	r = -.54 n = 107 p < .0001	**r = -.81** **n = 121** **p < .0001**	r = -.51 n = 83 p < .0001
Symptom-Time-4	r = -.45 n = 86 p < .0001	r = -.52 n = 85 p < .0001	**r = -.79** **n = 97** **p < .0001**

Note: All p-values presented are two-tailed, abbreviations are as follows: r = Pearson correlation coefficient, EWB = Emotional Well Being, n = sample size for that particular diagnostic group at that time, and p = p-value.

Table 6

The EWB Component of the Rand 36-item Gender T-score results for N = 2471 Individuals With Chronic Health Difficulties (Hays et al. 1993)

Gender	Depression Status	Mean T-Scores	Standard Deviations
Female	Not Depressed	M = 54.30	SD = 7.09
Female	Depressed	M = 42.58	SD = 9.65
Male	Not Depressed	M = 55.35	SD = 6.49
Male	Depressed	M = 43.09	SD = 9.61

Note: EWB = Emotional Well-Being.

Symptomatology is assessed on a scale where higher scores indicate pathology while the EWB scale indicates lower scores indicating pathology. At Time-2 the correlation between SYMP and EWB was $r = -.78$ ($p < .0001$). The same correlation for Time-3 was $r = -.81$ ($p < .0001$), and for Time-4 it was $r = -.79$ ($p < .0001$). Although, almost all of the correlations (validity coefficients; Cronbach, 1984) through time were significant the largest and most consistent effects occurred upon the diagonals of the tables where the correlations between both the variables were assessed at the same point in time. Therefore, it appears that the single item global symptom measure was working reliably for assessing clients global psychiatric symptomatology, and clients seemed capable of filling out the measure easily.

The Chronbach's alpha for the EWB measure ranged from .50 to .70 for Time-2 to Time-4 for the total sample which are moderate to large correlational effects according to Cohen and Cohen (1975). All of the correlations between the EWB and SYMP variables ranged from r = .41 to .81 (see Table 5 above). According to Cronbach (1984), "it is unusual for a validity coefficient to rise above 0.6. because social situations are continually changing and because people change themselves, perfect prediction is a false idea" (p. 140). The majority of the correlations between the global symptom measures in time also appear to be between moderate and large in effect size (Cohen & Cohen, 1975; see Table 5 above).

These correlations indicate that the global symptom measure of psychiatric symptomatology used in the present research correlates highly with the EWB scale often used in the literature to assess well-being as a general concept and these correlations are

relatively stable through time. According to Allen and Yen (1979), "convergent validity is demonstrated by high correlations between scores on tests measuring the same trait [construct] by different methods "(p. 110). According to Cronbach (1984), these findings indicate that the simple one-item SVAS global symptomatology measure has high predictive, convergent, and concurrent validity for these timed samples. Concerns about lack of variability in this single-item measure of symptomatology were not founded since variability was evident in final evaluation through time. Difference scores between the waves clearly indicate enough variability between points in time. For example the range of time-1minus time-2 symptom scores produced a variability from -7.5 to +9.0,

There may be some bias with less well participants being less likely to mail back their surveys. In this case the participants who would return surveys would likely be less symptomatic than the balance of this population; therefore, reducing the variability of the EWB and SYMP measures, leading to a suppression effect on the size of the correlation coefficients. This would reduce the probability of finding significant results so the results may be biassed in a conservative manner (Tabachnick & Fidell, 1989). These findings must be replicated before conclusions may be drawn about single item symptom measures.

Table 7

Rand EWB Mean T-Scores and Standard Deviations for Males and Females in the Total Sample at Time-2, -3, and -4

Time/Gender	Sample Sizes	Means	Standard Deviations
T-2/Female	N = 95	M = 48.85	SD = 9.88
T-3/Female	N = 81	M = 49.33	SD = 9.36
T-4/Female	N = 68	M = 50.05	SD = 10.05
T-2/Male	N = 52	M = 52.10	SD = 10.08
T-3 /Male	N = 42	M = 51.30	SD = 11.13
T-4/Male	N = 29	M = 49.88	SD = 10.04

Note: Abbreviations are as follows: T-2, T-3, and T-4 = Time-2, Time-3, and Time-4 Respectively, N = gender specific total sample size for that point in time, M = mean of the T-scores, and SD = Standard Deviations.

Other Primary Variables

When one considers how the other primary variables behave through time it is clear that the reliability/validity coefficients (Cronbach, 1984) are all significant (see Tables 8, 9, 10, 11, 12, and 13). All of the primary Time-1 to Time-4 variables correlated through time at acceptable levels (r = .43 to r = .94, all p-values <.0001) demonstrating high test-retest reliability (Chronbach, 1984). SYMP correlations ranged from r = .43 to .68, SS correlations ranged from r = .59 to .81, education correlations ranged from r = .89 to .94, FAD correlations were from r = .69 to .78, EMP rates ranged from r = .69 to .85, and finally INC correlations ranged from r = .61 to .85. According to Wiggins (1988), if

the same measure is administered on more than one occasion and the outcomes are significantly correlated (coefficient of stability) then the measure is said to be stable. If two separate administrations of the test are correlated strongly then the test is reliable (Goodwin, 1995).

Table 8

Reliability Correlation Coefficients SYMP Time1-4, N = 96-146

	SYMP Time-1	SYMP Time-2	SYMP Time-3	SYMP Time-4
SYMP Time-1	- -	+ .459 p < .0001	+ .564 p < .0001	+ .433 p < .0001
SYMP Time-2		- -	+ .675 p < .0001	+ .468 p < .0001
SYMP Time-3			- -	+ .580 p < .0001
SYMP Time-4				- -

Note. The abbreviations are as follows: p = one-tailed probabilities, and SYMP = Symptomatology measure. All probabilities are two tailed for the appropriate values related to directional predictions this value needs divided by two.

Table 9

Reliability Correlation Coefficients SUPPORT Time1-4, N = 96-146

	SUPPORT Time-1	SUPPORT Time-2	SUPPORT Time-3	SUPPORT Time-4
SUPPORT Time-1	- -	+ .666 p < .0001	+ .586 p < .0001	+ .625 p < .0001
SUPPORT Time-2		- -	+ .729 p < .0001	+ .811 p < .0001
SUPPORT Time-3			- -	+ .690 p < .0001
SUPPORT Time-4				--

Note. The abbreviations are as follows: p = one-tailed probabilities, and SUPPORT = combined social support measure. All probabilities are two tailed for the appropriate values related to directional predictions this value needs divided by two.

Table 10

Reliability Correlation Coefficients ED Time1-4,. N = 96-146

	ED Time-1	ED Time-2	ED Time-3	ED Time-4
ED Time-1	- -	+ .935 p < .0001	+ .902 p < .0001	+ .937 p < .0001
ED Time-2		- -	+ .881 p < .0001	+ .913 p < .0001
ED Time-3			- -	+ .933 p < .0001
ED Time-4				- -

Note. The abbreviations are as follows: p = one-tailed probabilities, and ED = education measure. All probabilities are two tailed for the appropriate values related to directional predictions this value needs divided by two.

Table 11

Reliability Correlation Coefficients FAD Time1-4, N = 96-146

	FAD Time-1	FAD Time-2	FAD Time-3	FAD Time-4
FAD Time-1	- -	+ .752 p < .0001	+ .693 p < .0001	+ .751 p < .0001
FAD Time-2		- -	+ .727 p < .0001	+ .776 p < .0001
FAD Time-3			- -	+ .784 p < .0001
FAD Time-4				- -

Note. The abbreviations are as follows: p = one-tailed probabilities and FAD = Financial Adequacy measure. All probabilities are two tailed for the appropriate values related to directional predictions this value needs divided by two.

Table 12

Reliability Correlation Coefficients EMP Time1-4, N = 96-146.

	EMP Time-1	EMP Time-2	EMP Time-3	EMP Time-4
EMP Time-1	- -	+ .804 p < .0001	+ .725 p < .0001	+ .687 p < .0001
EMP Time-2		- -	+ .848 p < .0001	+ .781 p < .0001
EMP Time-3			- -	+ .840 p < .0001
EMP Time-4				- -

Note. The abbreviations are as follows: p = one-tailed probabilities, and EMP = Employment measure. All probabilities are two tailed for the appropriate values related to directional predictions this value needs divided by two.

Table 13

Reliability Correlation Coefficients INC Time1-4,. N = 96-146

	INC Time-1	INC Time-2	INC Time-3	INC Time-4
INC Time-1	- -	+ .672 p < .0001	+ .824 p < .0001	+ .668 p < .0001
INC Time-2		- -	+ .668 p< .0001	+ .607 p < .0001
INC Time-3			- -	+ .851 p < .0001
INC Time-4				- -

Note. The abbreviations are as follows: p = one-tailed probabilities, and INC = Household Income measure. All probabilities are two tailed for the appropriate values related to directional predictions this value needs divided by two.

Plans for Path Analysis

Path analysis was originally developed by Sewell Wright (1921, 1934), a genetic statistician, for analysing the causal relationships among measured variables. Burkes (1928) and Peaker (1971) extended path analysis to the field of education while others applied these techniques in the social sciences (Blalock, 1961, 1968; Duncan, 1966). Keves (1988) examined the fundamental principles underlying path analysis for measured variables and Loehlin (1987) discussed the basic analysis of latent variables. Path analysis has developed to date into a complex set of approaches and computer programs that explain the relationships among both measured and latent variables in both recursive and nonrecursive models. In recursive models the flow of causation is unidirectional in nature. Unlike the recursive model, the nonrecursive model allows for reciprocal causal feedback loops. Nonrecursive models are very difficult to interpret and require complex statistical techniques for estimating effects (Cohen & Cohen, 1983).

According to Cohen et al. (1983), standardized coefficients are useful when measures variables are unfamiliar and when comparisons of effect magnitudes across different causes is desirable. Unstandardized coefficients were used in the modelling process since they are based on raw data and they can be more stable when comparing effect sizes over different models. Unstandardised coefficients are also useful when the raw variables are meaningful. The total effect between two variables is equal to the unstandardised correlation coefficient between the variables considered.

Why path analysis? Linear causal modelling, including path analysis procedures, permits a researcher to examine large networks of interrelated variables all at the same

time. Including modelling without reciprocal influences (Pedhauzer, 1982) and allows hierarchical analysis with variables in differing orders. Path analyses models must be based upon theoretical underpinning. In the case of this research, path analysis is used to evaluate the Social Causation and Social Selection theories important in linking social support to SES and mental health. Path analysis also allows for smaller samples than other Structural Equation Modelling procedures. However, if too many models are tested, with different causal orderings, in the hope of finding the best statistical fitting model the researcher moves from a confirmatory approach to an exploratory data analysis approach. Moving from a confirmatory to an exploratory approach results in a possibly significant inflation of Type-I error escalation. Since is a confirmatory approach there is some protection for capitalizing on Type-I error rates.

Path analysis allows a one way causal flow to evaluate the strengths of specific hypothetical interrelations using explanatory regressions applications. A basic assumption underlying path analysis is the specification of the model by temporally ordering the theoretically salient variables. Causal analysis in this instance is different from experimental causation as there is not one cause-effect relationship to be manipulated or considered, but rather a complex network of linkages among all the unmanipulated variables involved. According to Hamilton et al. (1993), causal modelling research is quasi-experimental in nature and models represent 'pictures of possible reality'. Unlike an experimental intervention, tests of the theorized models do not "constitute the construction of that reality" (p. 244). Causal inferences must be more tentative since quasi-experimental designs, including path analysis, cannot prove causality (Spady &

Greenwood, 1971). Path analysis allows one to estimate the magnitude of the relationships among the variables in the specified model and uses these estimates to provide information about the underlying causal processes (Spady & Greenwood, 1971). The match between theoretically driven or expected major pathways and those obtained from the data are generally believed to provide a reasonable estimate of the validity of the model (Berry & Williams, 1987). "While it is possible to test the mathematical accuracy of a simple model, its causal validity (including comprehensiveness) can only be supported on theoretical and substantive grounds" (Spady & Greenwood, 1971, p. 3).

Kendall and O'Muircheartaigh (1977) discussed two basic types of path models, the saturated additive model and the unsaturated model. A saturated model is a recursive model in which each variable is related to each variable preceding it in the causal hierarchy. In contrast, an unsaturated model is one in which all the variables are not necessarily related to all causally prior variables, not all of the variables are ordered hierarchically, or some possible paths are omitted. Some paths may be omitted (set to a zero value) because theory dictates that they do not influence the variables that follow them in the model or their measures of error are too large.

Path analysis methodology forces the researcher to specify explicitly the presumed causal relationships among the variables in the proposed model, and allows for the decomposition of the original zero-order correlation between any two variables into precise direct and indirect effects (Spady & Greenwood, 1971). Direct effects are effects one variable has upon another without the mediating or moderating effect of other variables involved in the model. An indirect effect is one in which a variable influences

another via a third variable. Variables in a proposed path model are hierarchically causally ordered. The order of the variables in the path model depends upon the available literature and theoretically feasible relationships. Variables are considered exogenous, if they can influence, but are not themselves influenced by, other variables in the model. Variables that are not designated as exogenous become either endogenous or outcome variables. An endogenous variable is a variable that is influenced by preceding variables in the model, and in turn influences the variables that follow it in the model. The outcome variable is the final variable in the causal model. It is influenced by any combination of the preceding exogenous and endogenous variables. A variable can function as both an independent and dependent variable depending upon the relationship being assessed. For example, an endogenous variable can function as a dependent variable in relation to the exogenous independent variable that precedes it in the model and it can be considered as an independent variable when one is considering the effect it has on the outcome or dependent variable that follows it in the model.

According to Kendall and O'Muircheartaigh (1977), simple path analysis models require linearity, additivity, and independence of errors. The variables involved in the modelling process need to be linearly related to the other variables. The set of multiple regression equations needs to be additive in nature. The error terms are assumed to be uncorrelated with each other and with other prior variables. This research is assessing complex models and we are automatically violating the assumptions of a simple model. So assumptions of linearlity, additivity, and uncorrelated error terms are violated whenever we are dealing with non-simple models (Kendal & O'Muircheartaigh,1977).

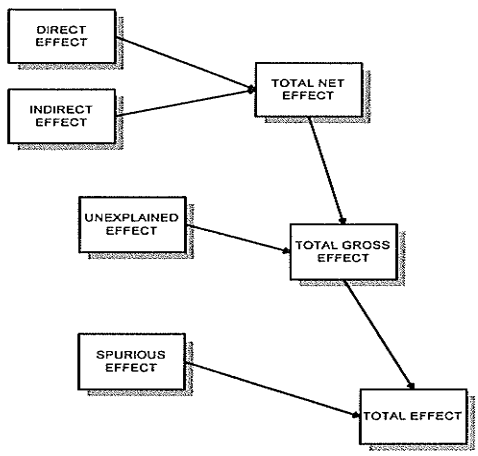
The primary difficulty with these designs are related to using repeated measures over time. This automatically violates the independence of errors requirement of many parametric tests used. However, all raw data was strictly screened for normality, outliers, and tenable linearity before any path modelling was attempted. All of the data went into the path models with no outliers, almost all were normally distributes, the linearity assumption was tenable, and covariance matrices values were all less than two.

An advantage of path analysis is that both the direct and indirect effects can be tested for statistical significance (Sobel, 1988). Many computer programs are available to decompose the path model effects. Each uses a different approach to break down and test the effects. The underlying mathematical framework of path analysis used by most of the available computer programs is regression. Simple and multiple regressions are used to estimate the magnitude of both the direct and indirect effects of variable pairs. SASPA (Huynh, 1992), an extension of a SAS (SAS Institute Inc., 1992) computer program by J. Scott Long (see Sobel, 1987), and SAS's PROC CALIS are two programs available for testing model effects. PROC CALIS (RAM statements) is by far the easier to use with regard to time and programming expertise and provides model fit indices. However, SASPA, unlike CALIS, supplies individual path error measures (for both direct and indirect effects) and test statistics (z-tests with corresponding probabilities) for one- and two-tailed tests. Both CALIS and SASPA provide equivalent direct and indirect effects. CALIS would be the better choice if model fit was the research question. Unlike SASPA, CALIS provides a selection of model fit indices including: the Null Model Chi-square statistic, the Bentler-Bonett normed fit index, and Bentler's comparative fit index. A good

model fit is indicated by values in excess of .90 for the Bentler and Bentler-Bonett indices (Bentler, 1989; Bentler & Bonett, 1980). However, if specific hypotheses are being tested, SASPA would be the better choice because each effect is evaluated against the respective error measures and the program provides the corresponding z-value and probability.

SASPA uses both standardized and unstandardized beta coefficients from simple regression models with an intercept to estimate the total effect one variable has upon another. The total effect is made up of a direct, indirect, unexplained, and spurious effect (see Figure 3) and represents the theorized causal relationship between the two variables being considered. The total net effect is the sum of the direct and indirect effects. The total gross effect is the sum of the direct, indirect, and unexplained effects. Paths in any model can be not applicable because it is physically impossible for an indirect effect to exist. Some paths may turn out to have zero-values and other paths may be excluded due to large error measures related to the them.

Figure 3. The decomposition of the total effect of one variable upon another as broken down by SASPA (Huynh, 1992).



Direct effects are estimated using the partial regression coefficients from the multiple regression model containing all the theorized causes of the dependent variable. Indirect effects exist when three variables are involved. Here, one of the variables is a proposed cause of one variable while it is the effect of another variable. The product of the direct effects, forming a causal pathway between two or more variables, is used as an estimate of the indirect effect. If more than one pathway exists to a particular effect, the indirect effect becomes the sum of the products of the direct effects that form the sequence of causal estimates from a cause to an effect (Cohen & Cohen, 1983). The direct and indirect effects are summed to arrive at the total net effect. Systematic error, error that cannot be explained or a non-causal component of the model (Fox, 1980), is represented by the unexplained effect. The difference between the total effect and the total gross effect is used as an estimate of the spurious effect and represents random or residual error due to sampling fluctuation. If either the unexplained or spurious effects is larger than the direct effect then the path pertaining to the error measurement is deemed invalid. The presence of an invalid path(s) indicates the model has been mis-specified, the path(s) needs to be deleted, and the model analysed again. Accordingly, models were tested and invalid paths were removed and models rerun. Since the intent was to attempting to test theoretical models using a confirmatory approach non-significant paths were not removed from the models. Almost all of the literature on path removal in path analysis clearly states that path deletion should be made upon substantive not statistical grounds. Paths therefore should not be removed on statistical grounds even if removing these paths would result in the 'best' model on statistical grounds. It is obvious that

invalid paths create havoc with path modelling but this does not apply to non significant paths. However, non-significant relationships between variables can't be removed from th participants lives so the decision was made to leave the non-significant paths in the models. Removing non-significant paths may impact/remove or reduce the indirect relationships between variables which could not be done if some of the insignificant paths were removed.

The chapters that follow provide a detailed path analysis of a number of models in an attempt to evaluate whether the cross-sectional model data repeats through time. Phase-III of Data analysis presents time-lagged path analytical models of the data related to the social causation social selection theoretical models. Phase IV of data analysis evaluates the inpatient treatment LOS results in an exploratory manner.

CHAPTER 4:

Phase-II Cross-Sectional Data Analysis

In this chapter cross-sectional results are evaluated for consistency through time frames. Hypothetical path diagrams used in the first stage of the research are presented with valence signs indicating directional hypotheses, including the rationale for all of the hypotheses. The primary objective is a replication of the cross-sectional results from Time-1.

This chapter begins by describing basic descriptive statistics related to the primary variables including employment, education, income, symptomatology, contact frequency, satisfaction with support, and perception of financial adequacy. Subsequent components of the chapter include structural equations, variance explained, Phase II cross-sectional path analysis methodology, fit indices; path diagrams will be discusses in the Appendices and Tables that follow along with the results of this research phase.

Primary Variable Descriptive Information

Sample descriptive statistics for the average variable values for the hospitalized, and total samples are in Table 14. For descriptive information about the primary variables at Times-1, -2, -3, and -4 the reader is referred to Table 15. For descriptive information about inpatient LOSs, hospitalization rates, and comparisons of primary variables for hospitalized versus not hospitalized subjects the reader is referred to Tables in Chapter 6.

Table 14

Descriptive Statistics for Sample of Individuals with a Hospital Stay During the Last Three Years and Total Samples

	N	CF	SAT	ED	EMP	INC	FAD	SYMP	ALOS
Individuals with three year hospital history.	76	M = 14.0 SD = 4.5	M = 4.9 SD = 1.9	M = 13.2 SD = 3.68	M = 25.1 SD = 29.3	M = 12.8 SD = 6.9	M = 22.2 SD = 10.6	M = 3.2 SD = 1.8	M = 33.6 SD = 27.5
Total Sample	232	M = 13.6 SD = 4.6	M = 4.9 SD = 1.9	M = 13.9 SD = 3.5	M = 34.9 SD = 42.7	M = 13.6 SD = 6.8	M = 22.3 SD = 11.1	M = 3.1 SD = 1.8	M = 10.6 SD =21.9

Note. N = Sample Size, CF = Contact Frequency, SAT = Satisfaction with Support, ED = Education, EMP = Employment, INC = Household Income, FAD = Financial Adequacy Perceptions, SYMP = Symptomatology, and ALOS = Average Length of Hospital Stay.

Table 15

Descriptive Statistics for the Total Sample Primary Variables (CF, SAT, FAD, ED, EMP, INC, SYMP) at Time-1, -2, -3, and 4

Variable		Time-1 (N = 232)	Time-2 (N = 146)	Time-3 (N = 123)	Time-4 (N= 96)
CF	M	13.58	12.19	13.17	12.95
	SD	4.63	4.45	4.42	4.47
SAT	M	6.96	6.73	6.78	6.75
	SD	2.80	1.64	2.53	2.50
FAD	M	22.35	22.84	24.52	23.32
	SD	11.07	11.41	10.73	11.60
ED	M	13.95	14.54	14.64	14.63
	SD	3.36	3.17	3.22	3.42
EMP	M	34.99	18.48*	18.73*	16.95*
	SD	42.66	21.50*	22.33*	21.23*
INC	M	13.57	11.17	16.68	15.42
	SD	6.83	6.69	6.81	7.26
SYMP	M	4.37	4.79	4.50	4.99
	SD	2.59	2.80	2.76	2.86

Note. The abbreviations are as follows: CF = contact frequency, SAT = satisfaction with support, ED = years of education, EMP = employment score, INC = family income score, FAD = financial adequacy score, and SYMP = symptomatology measure. '*' Values marked with an asterisk are for only half a year.

Employment

The employment distribution for Time-1 to Time-4 can be found in Table 16. Respondents were asked to indicate all categories that applied to them so the percentages do not sum to 100%. The current sample replicates the results from MacDonald's (1992) sample since 17% of the 1992 sample of the same respondents were unemployed. The WAS (1995) employment distribution clearly demonstrates that a larger percentage of the general population sample was gainfully employed since 52.9% of the WAS sample was employed full-time, 20.7% part-time, 16% were retired, 8.6 % were unemployed looking for work, and 22.1% were in school. There also appears to be a difference in the proportions of the samples who are in school and those looking for employment.

Table 16

Employment Status Distributions over Time-1 Through Time-4

Employment Status	Time-1 %	Time-2 %	Time-3 %	Time-4 %
Employed Full Time	25	22.8	28.3	24.8
Employed Part Time	16.8	15.4	18.9	18.8
Unemployed	16.4	12.1	11.8	14.8
Retired	16.4	18.8	21.3	24.8
School	6.9	5.4	6.3	3
Maintaining a Home	20.3	16.8	25.2	22.8
Unemployed Due to Illness	28	28.9	29.1	23.8

The figures on the table representing those who are unemployed are likely underestimating the true number of individuals in this population who are without desired employment. Results of employment distributions in Table 16 appear to be relatively stable through the two year period. There is a large proportion of individuals in this sample who are reporting that they were unemployed due to illness. Many chronically mentally ill individuals may have long since given up looking for work or considering themselves unemployed. These results are in keeping with the notion that those who are ill are less likely to be employed and that the mentally ill face a negative cyclical pattern of less employment, more symptomatology, and reduced income levels (Matson & Rusch, 1986). When one compares those who were unemployed due to illness to those indicating that they were unemployed it is interesting that at Time-1 only 28.0% of the sample indicated that they were unemployed due to illness. This is roughly twice the proportion of individuals claiming unemployment. In fact, this figure appears to be relatively stable through the four time-frames.

There were some difficulties with the distribution of the employment variable. Employment was calculated by multiplying twice the full-time weeks of employment and adding it to the part-time weeks of employment (Time-1, $\underline{N} = 232$, $\underline{M} = 33.5$, $\underline{SD} = 40.5$; Time-2, $\underline{N} = 146$, $\underline{M} = 18.4$, $\underline{SD} = 21.4$; Time-3, $\underline{N} = 123$, $\underline{M} = 18.7$, $\underline{SD} = 22.4$; Time-4, $\underline{N} = 96$, $\underline{M} = 16.9$, $\underline{SD} = 21.2$). Time-1 values are greater than the other time-frames because they refer to the previous year. Time-2, -3, and -4 refer to the previous six month periods. If one considers those with a non-zero employment there are no difficulties with

the variables distribution (normality is tenable). However, there is a significant group of individuals who participated that had no employment, leaving a number of zeros for the employment variable. For example, 48.9% of the sample indicated a zero employment situation at Time-1, 48.0% at Time-2, 53.0% at Time-3, and 52.5% at Time-4. Transforming (exponential, Base 10, and Natural Logarithm) these values was not productive since the transformations still did not adjust the zero scores or provided huge numbers and did not normalize the data they produced.

Education

The level of education in the current sample is similar to the WAS sample in the number of individuals who had a Junior-high education or less (5.6% and 6.2%, respectively). The next category in the WAS is represented by those individuals with High-school or non-university training (56.3%). The balance of the WAS distribution consisted of 37.5% of the sample with some university training. Unfortunately, the WAS (1995) did not keep a more refined measure of education for comparison to the current sample. Thirty percent of this sample had only a high school education and 64% of the current sample had a post secondary or trade education of some sort.

Family Income

Family income values for the current study were compared to the WAS (1995) and results clearly show that the family income for the sample from the self-help group

population appears different from the general Winnipeg sample. The median income category for the current self-help sample was 28,000-29,999 dollars per year (for the 1994 tax year), while the median category for the WAS in 1994 was $40,000+. The $40,000+ category contained 52.9% of the general WAS sample but only 23.7% of the self-help group sample. The bottom income category on the WAS was less than $10,000 and 4.2% of the general city sample indicated that they fell into this category. The same category in the self-help sample included 12.5% of the respondents. It is clear from these results that the respondents with psychiatric diagnoses affiliated with this self-help group are at an economic disadvantage when compared to a general Winnipeg survey sample.

Financial Adequacy Results

Given the employment status of many persons with psychiatric disabilities, one would expect individuals from the self-help group to have lower financial adequacy scores than those from a normal population. For comparison purposes Winnipeg Area survey (WAS) participants were contacted and asked if they would fill out the same survey measures used for the clinical sample. This small general population sample (gathered during 1997) was collected from participants of the current year's Winnipeg Area Survey (WAS, N = 125) for comparison purposes. Participants of the current WAS who had agreed to be recontacted were mailed a slightly modified survey which included most of the same questions the mood disorder participants answered. This resulting sample is expected to be biassed with individuals more likely to volunteer, having higher

education and income levels. Results for the clinical population indicated a differing distribution of the financial adequacy (FAD) variable. Self-help participants scored lower on financial adequacy ($\underline{M}_{self}$ = 22.39, $\underline{SD}$ = 11.07) than the general population participants ($\underline{M}$ = 27.81, $\underline{SD}$ = 10.18, $\underline{t}_{(334)}$ = 4.64, $\underline{p}$ < .0001/2). Only participants who answered all of the components were considered due to inconsistencies that replacing missing values may create. All of the components making up the FAD variable were also significantly lower for the self-help participants (monies for personal use, $\underline{M}_{self}$ = 4.05, $\underline{SD}$ = 2.10, $\underline{M}_{general}$ = 4.78, $\underline{SD}$ = 1.88, $\underline{t}_{(306.5)}$ = 3.34, $\underline{p}$ =.001/2; monies for one's home, $\underline{M}_{self}$ = 3.93, $\underline{SD}$ = 2.02, $\underline{M}_{general}$ = 4.70, $\underline{SD}$ = 1.83, $\underline{t}_{(305.8)}$ = 3.55, $\underline{p}$ < .0001/2; money for ones loved ones, $\underline{M}_{self}$ = 3.89, $\underline{SD}$ = 2.04, $\underline{M}_{general}$ = 4.84, $\underline{SD}$ = 1.91, $\underline{t}_{(297.7)}$ = 4.316, $\underline{p}$ < .0001/2; monies for leisure, $\underline{M}_{self}$ = 3.65, SD = 2.05, $\underline{M}_{general}$ = 4.35, $\underline{SD}$ = 1.95, $\underline{t}_{(334)}$ = 3.303, $\underline{p}$ = .001/2; families financial needs, $\underline{M}_{self}$= 3.81, $\underline{SD}$ = 2.02, $\underline{M}_{general}$ = 4.88, $\underline{SD}$ = 1.88, $\underline{t}_{(301.07)}$ = 5.212, $\underline{p}$ < .0001/2).

Analyses of categorical distribution frequencies demonstrated significantly differing distributions. In general, for the self-help participants, there were more individuals in the lower satisfaction categories than in the higher satisfaction categories ($\chi 2_{(1)}$ = 22.485, $\underline{p}$ < .0001/2; using a median split of 18). Self-help members were significantly lower in each of the categories (monies for personal use, $\chi 2_{(6)}$ = 14.212, $\underline{p}$ = .027/2; monies for one's home, $\chi 2_{(6)}$ = 18.828, $\underline{p}$ = .004/2; monies for one's loved ones $\chi 2_{(6)}$ = 23.800, $\underline{p}$ = .001/2; monies for leisure, $\chi 2_{(6)}$ = 11.822, $\underline{p}$ = .066/2; families financial needs $\chi 2_{(6)}$ = 27.589, $\underline{p}$ < .0001/2). Clearly, self-help clients' perceptions of

financial adequacy are quite different from individuals making up this general population sample. This indicated that the perception of financial pressures is different from clients with psychiatric disabilities.

Symptomatology

Symptomatology mean scores and standard deviations for the whole samples for Time-1, -2, -3, and -4 can be found in Table 17. It can be noted that 29.3% of subjects has a zero symptomatology score at Time-1. This percentage is larger than the other time frames. For Time-2 the value was 15.4%. The Time-3 percentage of individuals who indicated that they had no current symptoms was 18.4%. And finally, the Time-4 percentage of individuals scoring a zero on the symptom measure was 16.8%. The decrease in proportion of individuals reporting no symptomatology at Time-1 leads one to think that individuals with no symptoms were less likely to respond on subsequent waves. However, subsequent analyses demonstrated that this finding was due to individuals who reported no symptomatology at Time-1 were quite likely to report increased symptomatology at a subsequent time-frame. There were a few individuals who reported zero symptoms at all time-frames. Intercorrelations for Time-1, -2, -3, and -4 symptomatology are clearly related and stable (Time-1 and Time-2, $N = 145$, $r = .407$, $p < .0001$; Time-1 and Time3, $N = 123$, $r = .572$, $p < .0001$; and Time-1 and Time-4, $N = 96$, $r = .450$, $p < .0001$).

Table 17

Descriptive Symptomatology Scores for Individuals with Non-zero Symptom Scores

	Sample Size	Means	Standard Deviations
Symptoms at Time-1	N = 164	M = 5.47	SD = 2.13
Symptoms at Time-2	N = 121	M = 5.02	SD = 2.80
Symptoms at Time-3	N = 101	M = 4.72	SD = 2.73
Symptoms at Time-4	N = 83	M = 5.33	SD = 2.72

Cross-Sectional Models Evaluation Phase-II

Cross-Sectional Hypotheses

The primary hypotheses predicted inverse relationships between the social support variables (contact frequency and satisfaction with support) and the outcome symptomatology and hospitalization variables. Since secondary hypotheses were numerous, in order to facilitate both clarity and readability, the reader is referred to Figure-1 for the cross-sectional symptomatology models. The hypothesized relationships are indicated with valence signs. The specified relationships could represent the direct, indirect, or total effects of the variables involved. The one set of hypothetical relationships are applicable to the four time frames. The research goal here is to demonstrate replication through times (Time-1, -2, -3, and -4).

Rationale for Hypotheses

Although directional predictions were being made, the reader is cautioned that with this type of cross-sectional, quasi-experimental, design the question of causation cannot be answered unequivocally. There are two major limitations in correlational research. The first difficulty is often referred to as the 'direction of causation' problem. In cross-sectional research all of the variable information is collected at one point in time. Then researchers try to attribute causal relationships among the variables. In these situations the issue becomes to determine which variable is the cause and which is the effect. For example, are symptoms causing reductions in social support or are deficits in social support influencing symptomatology. The other difficulty relates to what has been referred to as the 'third variable problem'. This means that a third, possibly unmeasured, variable may be the cause of the relationships between social support and symptomatology. With these limitations in mind, the hypotheses presented, on the path diagrams, are supported by both the literature and on theoretical grounds. Krause, Liang, and Keith (1990) warned that even with theoretical support, several alternative causal orderings could be specified for any model.

The following causal order assumptions were made for this research: 1) As satisfaction with support and the frequency of contacts between respondents and those in their social network reduce, their overall well being will worsen. 2) Reduced perceived support and contact with persons who could help further not only educational goals but opportunity for employment would negatively affect employment and employability.

3) Lower levels of employability would result in reducing available income, thereby negatively influencing perceptions of financial adequacy. The combined effect of these social functioning variables would increase the burden of chronic social and financial stressors in a respondent's life and influence psychiatric health, leading to more psychiatric symptomatology and extended hospitalizations.Psychiatric clients are often socially withdrawn or isolated (Cohen, 2004) and isolation is a strong predictor of health (Adler & Newman, 2002) and emotional health (Haughton Mc Neill, Kreuter, & Subramanian 2006).Yet, according to Cohen and Sokolovsky (1978), frequency or length of psychiatric hospitalization (LOS) seems unlikely to have caused this withdrawal. Yet, the 'Social Breakdown Syndrome' suggests that reduced contact is the result of lost role functioning and identity, resulting in a passive withdrawal of the client diagnosed with a psychiatric illness (Cohen, 2004; Kuypers & Bengtson, 1973) resulting in minimal involvement in a variety of social roles (Cohen, 2004). The positive impact of social support can be diminished by conflict in one's social network (Okun & Keith, 1998). The social support/health relationship could function in both directions or be a result of another unmeasured variable (Hammer et al. 1982). But, according to Berkman and Syme (1979), it is more likely that social networks influence health outcomes. Kaplan, Salonen, Cohen, Brand, Syme, and Pyska (1988) have stated that available research fails to support the theory that illness causes social support inadequacies. Mortality has been found to have an inverse relationship with received social support (Berkman & Glass, 2000; Cohen, 2004; Kaplan et al. 1996; Walter-Ginzbutg, Blumstein, Chetrit, & Modan, 2002).

According to Wellman, social contact leads to better social support, and better social support can result in better employment opportunities that can translate into more stable financial resources (cited in Hobfoll & Jackson, 1991). For example, contact with a network can inform members of available job opportunities and provide members in need with valuable introductions to prospective employers.

According to Bolger, Zuckerman, and Kessler (2000), when people actually draw on their social support network for help during stressful times can result in even more stress and lower self-esteem because they feel that they are taking up too much from another persons time and attention (Taylor et al. 2004). The inverse relationship between socioeconomic status and psychological distress is well documented (Kessler, 1982; Wheaton, 1978). According to Pearlin (1982), longitudinal analysis supports a causal interpretation of the relationship between socioeconomic status variables and health.

Unemployment, especially for prolonged periods, has been found to be a severe stressful life event (Pearlin, 1982) that threatens physical security, social identity (Weber, 1946), self-esteem (Dressler, 1986), well-being (Viswesvaran, Sanchez, & Fisher, 1999), increased financial and strain negative changes in financial resources (Creed & Macintyre, 2001; Gallo, & Mathews, 2003; Vinokur & Schul, 2002); and an increase in secondary stressors (Howe, Lockshin, & Caplan, 2004; Kinicki, Prussia, and McKeen-Ryan, 2000; Price et al.1998). The order of the socioeconomic variables of education, employment, and income is based upon work by Turnbull, George, and Landerman (1990). Education is assumed to be a causal factor in employment status, which is, in turn

a causal factor in income.

The relationship between employment (unemployment) and psychiatric health is harder to disentangle. According to Dressler (1986), "it is much more likely that unemployment preceded and contributed to the reporting of depressive symptoms" (p.644). According to Bolton and Oatley (1987), in a panel study, unemployed depressed individuals became less depressed upon finding employment and those who remained unemployed became more depressed. According to Hamilton et al. (1993), not only can unemployment lead to depression, but, depression can lead to unemployment. The reciprocal relationship, between unemployment and depression, may be due to selection biases in cross-sectional samples. It is just as possible that distressed individuals are more likely to become unemployed as unemployed individuals becoming depressed (Hall & Johnson, 1988; Kessler, House, & Turner, 1987). In a longitudinal design, Kessler, Turner, and House (1989) did not find support for the idea that distress from unemployment reduces the likelihood of future employment. These researchers found that those who became depressed after losing their jobs were somewhat, although not significantly, more rather than less likely to find employment. Frese (1987) also believed that depression is the result of unemployment rather than the inverse and that the effect of unemployment is moderated by hope and one's financial situation.

According to Ross and Huber (1985), the interaction of education and income significantly influences perceptions of economic hardship. Longitudinal analysis supports the notion that economic hardship influences depressive symptomatology rather than vice

versa (Stolsenberg, 1980). Pearlin et al. (1981) found that changes in family income influenced economic strain, leading to changes in depression.

The causal ordering specified in these proposed models are supported by literature. However, several causal orderings are possible based upon the available literature. The present models test one pattern of orderings, but in reality many of the aforementioned variables could be placed in numerous places in this type of social causation model. For example, family income could precede social support, education, and LOS at one point in time and family income could be influenced by social support at a later point in time. There are many different patterns possible for this type of research, especially if one considers the variables in time, and many of these relationships are non-recursive, meaning that they work in both directions. For example, if someone has a poor level of contact with a supportive network at the beginning of a psychiatric illness and is admitted to an inpatient psychiatric facility, this may serve notice to the family that there is a problem. Individuals with bipolar depressive disorders often experience residual symptomatology after discharge; having the patient record moods at home can help in maintenance pharmacotherapy (Cooke, Kruger, & Shugar, 1996). Despite pharmacotherapy many bipolar patients continue to experience persistent symptomatology and continued impairment (Keck et al. 1998). After the individual is released from hospital, individuals in the client's network may now make a point of seeing the client more frequently. This increased contact could result in symptoms being recognized earlier and treatment handled on an outpatient basis or another hospitalization. The hospitalized individual could also alienate network members resulting in a reduction

of contact after the first hospitalization. Either direction of causation is possible and they are both partially supported by the available literature. This is the primary topic of Phase-III of this thesis where social selection and social creation theories are tested against one another in an attempt to determine which model is more veridical.

Cross-Sectional Path Analyses Methodology

The preliminary fully specified models (see hypotheses Figure 1) were run in both SASPA and CALIS(RAM). Cross-sectional models were first subjected to SASPA for the identification of invalid paths. A fully specified model is one in which each variable in the model is connected to all variables that causally follow it. The fully specified models in SASPA were then evaluated for the validity of each path. Paths that were deemed invalid by the previously detailed requirement were dropped and the multiple regressions and the path program rerun until all remaining paths were valid. The multiple regressions and path analysis programs were rerun excluding all of the invalid paths for all of the models (see Table 18 for Time-1 and for Times-2, -3, and -4 see Tables C1, C2, and C3 in Appendix C).

Table 18

Cross-Sectional Time-1 Direct, Indirect, Unexplained, Spurious, and Total Effects for the Symptomatology Path Model, Including Regression Beta Weights, N = 232

Variable Pairs IV/DV	Direct Effect	Indirect Effect	Unexplained Effect	Spurious Effect	Total Effect
CF/SAT	β= .1949 Z= 24.40 p < .0001	not applicable	-1.4 E-17	1.4 E-17	β= .1949 Z= 7.7150 p < .0001
SAT/ED	invalid path	invalid path	invalid path	invalid path	invalid path
CF/ED	β= .0499 Z= 1.09 p= .138	not applicable	-1.7 E-18	1.7 E-18	β= .0499 Z= 1.09 p= .138
SAT/EMP	β= 3.3824 Z= 2.17 p= .015	not applicable	-1.22	-0.0193	β= 2.1464 Z= 1.48 p= .069
ED/EMP	β= 3.692 Z= 4.28 p < .0001	not applicable	-0.069	-0.0132	β= 3.6102 Z= 4.16 p < .0001
CF/EMP	β= -1.3010 Z= -1.95 p= .026	β= .8434 Z= 2.40 p= .008	1.4 E-17	-8.6 E-7	β= -.4576 Z= -.74 p= .230
SAT/FIN	β= .3283 Z= 1.40 p= .081	β= .2114 Z= 2.05 p= .020	-0.022	0.006	β= .5115 Z= 2.22 p= .013
ED/FIN	β= .3271 Z= 2.45 p= .007	β= .2307 Z= 3.54 p < .0001	-0.006	-0.002	β= .5610 Z= 4.03 p < .0001
EMP/FIN	β= .0625 Z= 6.29 p < .0001	not applicable	0.007	5.8 E-4	β= .0702 Z= 7.33 p < .0001
CF/FIN	β= .0330 Z= .33 p= .371	β= .0517 Z= .76 p= .224	1.4 E-17	-5.4 E-7	β= .0847 Z= .853 p= .197

Table 18 (continued)

Variable Pairs IV/DV	Direct Effect	Indirect Effect	Unexplained Effect	Spurious Effect	Total Effect
SAT/FAD	β= 1.0956 **Z**= 2.92 **p**= .002	β= .3355 **Z**= 1.77 **p**= .038	0.187	-0.002	β= 1.6160 **Z**= 4.46 **p** < .0001
ED/FAD	β= -.4257 **Z**= -1.98 **p**= .024	β= +.3429 **Z**= +2.87 **p**= .002	0.0488	-0.007	β= -.0412 **Z**= .18 **p**= .430
EMP/FAD	β= -.0197 **Z**= -1.15 **p**= .125	β= .0466 **Z**= 4.68 **p** < .001	0.001	7.8 E-5	β= .0282 **Z**= 1.64 **p**= .051
FIN/FAD	β= .7451 **Z**= 6.99 **p** < .0001	not applicable	0.0453	-0.00671	β= .6932 **Z**= 7.10 **p** < .0001
CF/FAD	β= .1865 **Z**= 1.17 **p**= .121	β= .2644 **Z**= 2.52 **p**= .006	-1.4 E-17	5.4 E-7	β= .4509 **Z**= 2.8430 **p**= .002235
SAT/SYMP	β= -.2703 **Z**= 4.39 **p** < .0001	β= -.0752 **Z**= -1.78 **p**= .038	-0.064	0.009	β= -.4009 **Z**= -7.22 **p** < .0001
ED/SYMP	β= -.0072 **Z**= -.21 **p**= .417	β= -.0253 **Z**= -1.75 **p**= .040	-0.013	0.002	β= -.0443 **Z**= -1.16 **p**= .122
EMP/SYMP	β= -.00763 **Z**= -2.98 **p**= . 002	β= -9.3E-4 **Z**= -1.01 **p**= .156	-0.001004	0.000048	β= -.0095 **Z**= -3.46 **p** < .0001
INC/SYMP	invalid path	invalid path	invalid path	invalid path	invalid path
FAD/SYMP	β= -.0345 **Z**= -3.53 **p** < .0001	not applicable	-0.017	0.00013	β= -.0514 **Z**= -4.99 **p** < .0001
CF/SYMP	β= -.0610 **Z**= -2.37 **p**= .009	β= -.0651 **Z**= 4.26 **p** < .0001	1.40000e-17	0.002	β= -.1242 **Z**= -4.97 **p** < .0001

Note: Abbreviations are as follows: β = path coefficients, **Z** = Z-scores , **p** = one-tailed probability values, IV/DV = independent/ dependent variable pairs for each path, CF = contact frequency, SAT = satisfaction with support, ED = years of education, EMP = employment score, INC = family income, FAD = financial adequacy score, SYMP = symptomatology score, and not applicable = it was physically impossible for an indirect effect to exist.

For the first of the cross-sectional models (Time-1) the paths connecting SAT with ED and the path connecting INC with SYMP were invalid . For the Time-2 cross-sectional model only the path connecting SAT with ED was deemed invalid. In the third cross-sectional model (Time-3) once again the path connecting SAT and ED was invalid as the two previous models but also the path connecting FAD and SYMP was also invalid. The final cross-sectional model Time-4 had the same SAT and ED path invalid as the other models but also the path connecting EMP and FAD was invalid.

The preliminary fully specified symptomatology models (Time-1 to Time-4) were run in PROC CALIS to estimate goodness of fit of the saturated preliminary model. The RAM component of this program uses the standardized variables and variable covariance matrix as input. The RAM input statement specifies the number of directional causal arrow heads, which variable the path points to and from, and gives the path coefficient a name (SAS, 1992). Error measures associated with each endogenous variable in the model are also specified. They consist of two-headed arrows representing paths going to the variables in the model from the error term.

The PROC CALIS procedure provides a number of model fit indices. The Null Model Chi Square computed by CALIS is used to evaluate whether a model is even required to explain the data. The variable intercorrelations observed in the data are compared first to a Null Model of 'independent or uncorrelated variables' (Bentler, 1989; p. 92). If the resulting chi squared value is large, it indicates that the Null Model does not explain the data well and some model needs to be specified to explain data intercorrelations (Bentler, 1989). The Null Model Chi Square for the current data sets

(Time-1 to Time-4) indicates that the data need to have a model fit to reduce variability (see Table 19).

Table 19

Chi Squared Values and Fit Indices for Null and Reduced Cross-Sectional Models

Model	Chi Squared Statistics (χ^2)						Goodness of Fit Indices		
	Null Model (1)	$df_{(1)}$	$p < 2$	Reduced Model (2)	$df_{(2)}$	$p < 2$	GFI	BBNFI	BOLEN
Time-1	282.14	21	<.0001	1.03	2	0.96	1	1.04	0.996
Time-2	128.09	21	<.0001	0.002	1	0.96	1	1	0.999
Time-3	122.3	21	<.0001	0.158	2	0.92	1	0.999	0.986
Time-4	95.82	21	<.0001	0.007	2	1	1	0.999	0.999

Note: Abbreviations: $df_{(1)}$ = degrees of freedom for the Null Model, $df_{(2)}$ = degrees of freedom for the Reduced Model, p = p-value, df = degrees of freedom, $p < 2$ = the probability of the Null and Reduced Models Chi Squared value being less than two, GFI = Goodness of Fit, BBNFI = Bentler and Bonett's Normed Fit Index, and BOLEN = Bollen's Normed Index.

The other Chi-square model fit index provided by CALIS represents the Chi Square value computed from the proposed model for the data. For the proposed model to fit the data well the chi squared index needs to be small and not significant at the .05 level. When this chi squared value is divided by the appropriate remaining degrees of freedom the resulting value should be less than 2.0 to indicate a good fit according to Joreskog and Sorbom's suggestion (Loehlin, 1987). According to the output from PROC CALIS RAM, the fully specified symptomatology models were all over-specified as most saturated or fully specified models usually are. The fully specified models fit indices chi square values were all zero with a negative degrees of freedom. This kind of result was expected since a fully specified model was input and would use up all of the available degrees of freedom. To improve the fit of the CALIS models the invalid paths, identified via SASPA, were deleted from the fully specified models at this time and PROC CALIS-RAM was run again. The results of the reduced model indicated that the proposed models for the data were good fits (see Table 19). For example, for Time-1 the Null Model χ^2 value 282.14 with a p-value of <.0001 and after the model was fit to the data the χ^2 value was reduced to 1.03 with a p-value of .597. This result clearly indicates that fitting the model to the data explained a lot of variance that existed in the Null Model. The corresponding fit indices for this model were also very close to 1, indicating that the model was a good fit for the data.

PROC CALIS also provides a number of other fit indices that can be used to assess overall model fit. These include The Goodness of Fit Index (GFI), Bentler and Bonett's Normed Fit Index (NFI), and Bollen's fit index. Each of the fit indices has their

own particular advantages and disadvantages. In general, model fit indices are based upon a fit function and a baseline model of uncorrelated (independent) variable measures. The worse the baseline (Null Model) fit and the better the proposed model fit, the better the fit indices. Values of the NFI, GFI, and BOLLEN which are greater than .90 are desirable for adequacy of proposed model fit (Bentler, 1989; Bentler & Bonett, 1980). The model fit indices for the current data all exceeded the .90 rule of thumb for a suitably fit model (see Table 19 above).

Structural Equations for Cross-Sectional Analyses

The structural equations used in the path analysis models are dictated by the theoretically specified causal orderings of the variables involved and their related hypotheses. The actual equations are a result of the ordering of the variables in the theoretical models. Path numbers indicate the sequence of each relationship being evaluated. These tables also indicate the invalid paths discovered by the SASPA program. Since the four cross-sectional tables are identical the Time-1, -2, -3, and -4 can be found in Appendix D (Tables D1, D2, D3, and D4). The reduced models (with invalid paths deleted) can be found on Tables E1, E2, E3, and E4 in see Appendix E for adjusted path numbers). The variance explained by the Time-1 model can be found in Table 20 below. The explained variances for the other models can be found in Appendix F (Tables F1, F2, and F3).

Table 20

Multiple Regression Variance Explained for the Structural Equation System Based Upon Time-1 Reduced Path Model, N = 232

Equation Number	R^2	MS_e	F-ratio value	p-value
EQ #1	0.2085	181.45	59.526	$p < .0001$
EQ #2	0.01	11.9	1.191	$p = .2764$
EQ #3	0.095	13139.4	7.826	$p < .0001$
EQ #4	0.2249	600.1	16.173	$p < .0001$
EQ #5	0.2583	1453.1	15.464	$p < .0001$
EQ #6	0.2742	40.9	16.775	$p < .0001$

Note: Abbreviations: EQ = equation number from the structural equation system, R^2 = proportion of the variance accounted for by the particular equation, p-value = probability of F-value, and MS_e = mean square error.

Cross-Sectional Symptomatology Models Results

Cross-sectional results can be found on Figures 4, 5, 6, and 7 below. The detailed tables with exact probabilities for each effect can be found in Appendix C (Tables C1, C2, C3, and C4). There will be no formal statistical comparison of the effects related to the four within time cross-sectional models above since there is no reason to compare exact magnitudes of all of the actual effects through time. However a visual inspection of repeating patterns is used to compare the four cross-sectional models (Figure 8 below). Each of the symptomatology models was evaluated in the same manner as the Time-1 model (Unpublished Masters Thesis, Murray, 1996) and they were all compared to each other to evaluate whether the symptom model repeated through the four points in time,

creating an intra-study replication of the symptom model. If the original model was a reasonable reflection of what is real it should replicate at Time-2, Time-3, and Time-4. All points in time will be discussed together with specific paths being discussed in the order indicated on the hypothesis model (see Figure 1).

Figure 4. Final reduced path model for the psychiatric symptomatology cross-sectional model for Time-1.

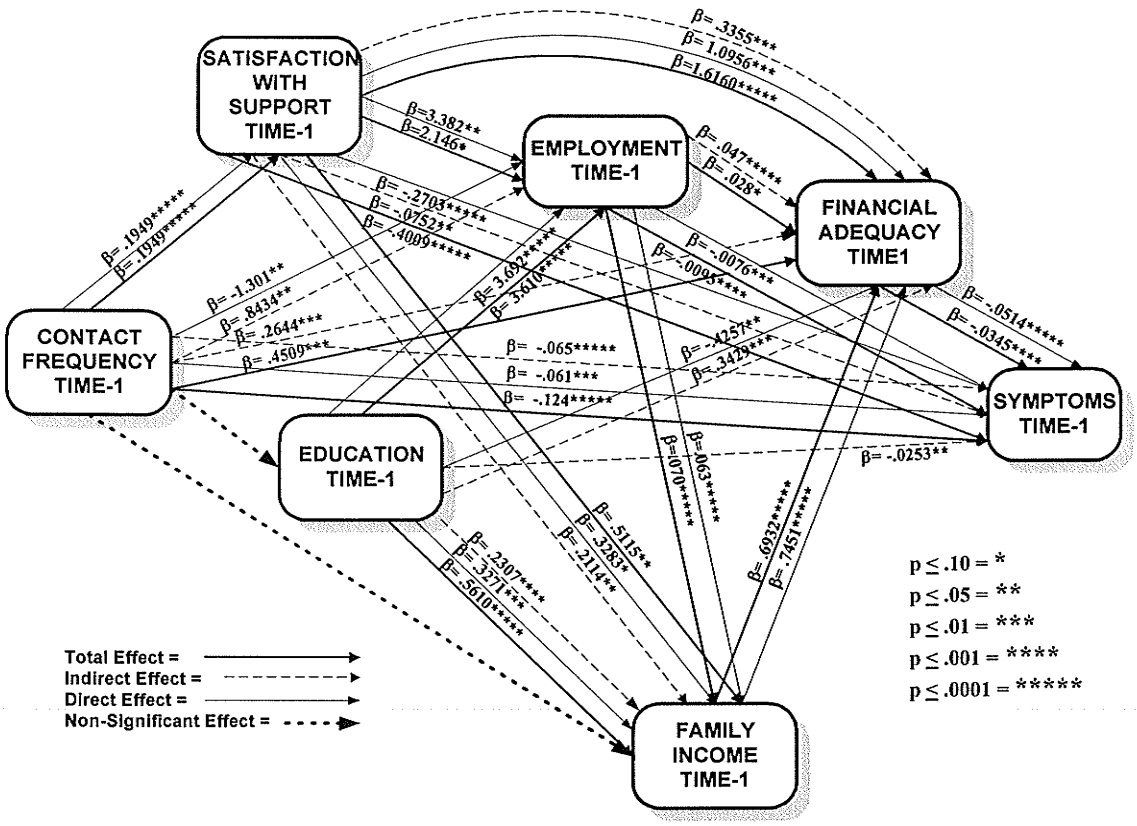


Figure 5. Final reduced path model for the psychiatric symptomatology cross-sectional model for Time-2.

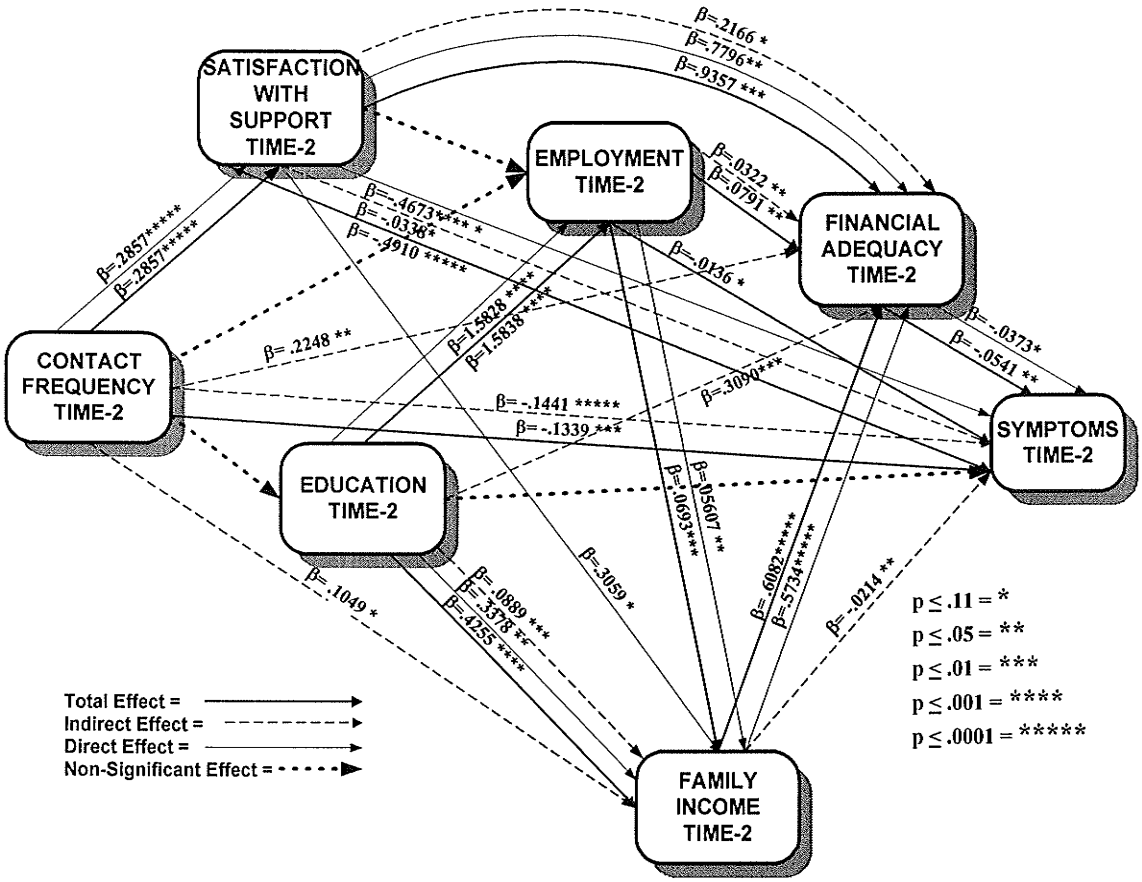


Figure 6. Final reduced path model for the psychiatric symptomatology cross-sectional model for Time-3.

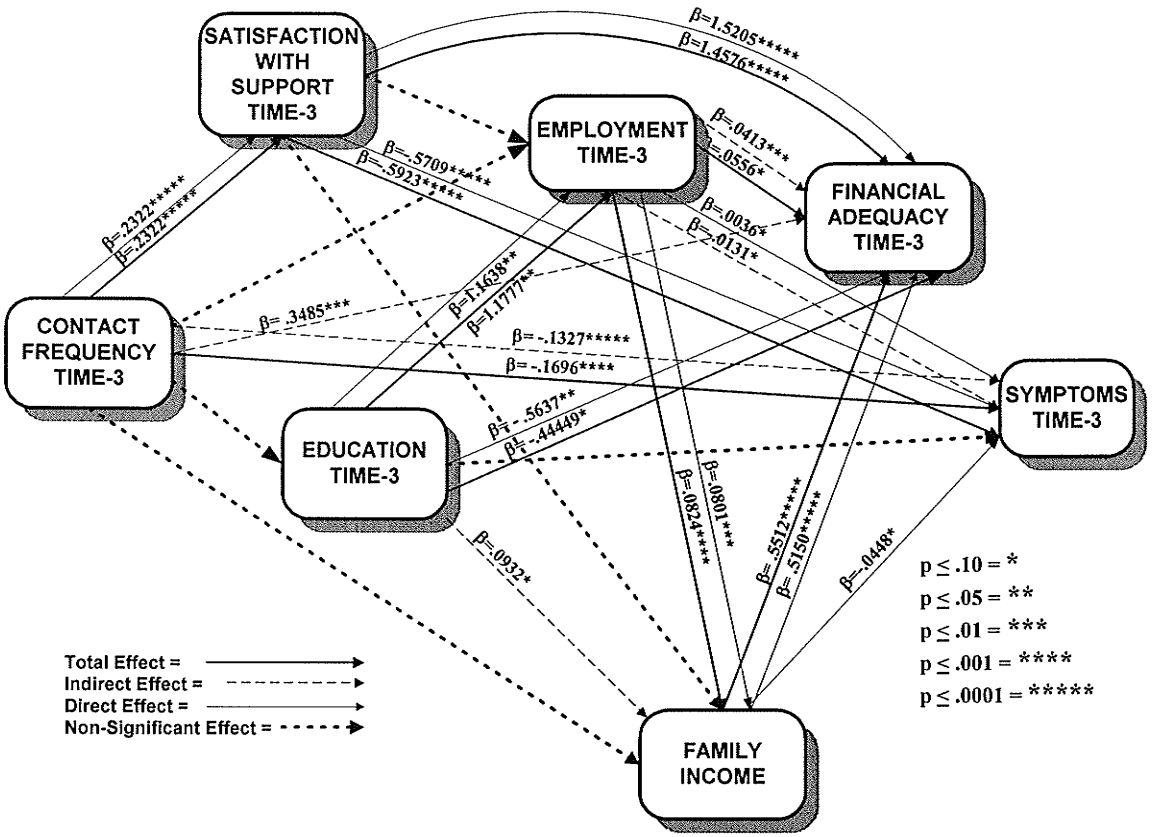
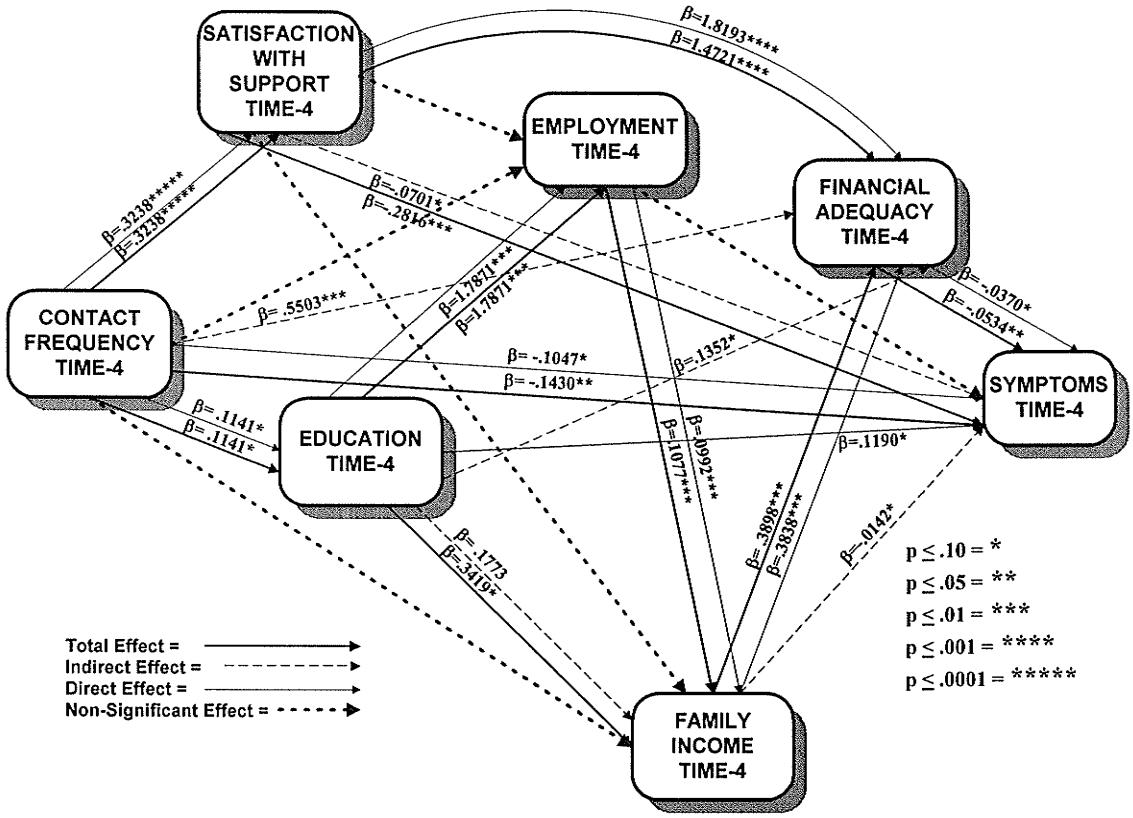


Figure 7. Final reduced path model for the psychiatric symptomatology cross-sectional model for Time-4.



The exact p-values for each effect through time were not compared since the sample sizes at each of the points in time are different and power is diminishing through the study period. However, a similar pattern in magnitude of effects should be evident across all points in time. Although a confirmatory research approach is being used that provides some protection against inflating Type-I error rates, there are disagreements about how to handle Type-I error rates in these research situations since there are so many beta coefficients being evaluated for significance. Rather than continue this issue, the exact p-values for each beta coefficient are provided on these so the readers can make up their own minds (see Tables C1, C2, C3 and C4 in Appendix C). When evaluating fit indices (see Table 19), it becomes clear that each model is clearly an improvement over no model for each time-frame. The models tested demonstrated significant reductions in the χ^2 values when a model was fit to the data. There was difficulty in deciding which paths were going to be assessed as significant, and lack of power through the time points complicated this process. So, paths that appear salient will be discussed in the text that follows. For the purposes of this analysis, salient paths were deemed to have a listed p-value of less than .11. This value was picked to allow paths that appeared to be stable through time to be included in the models and discussion. The choice of selecting paths to be discussed is somewhat arbitrary and was done in an attempt to maintain salient repeating paths in the model for discussion. Also, power was an issue but larger, more appropriate, sample sizes were not possible.

In assessing the Time-1, -2, -3, and -4 cross-sectional symptomatology model there were a total of 21 paths with each path having three possible effects (direct, indirect,

and total effect). As stated previously, all of the models had some invalid paths because of excessive error measures. The Time-1 cross-sectional model had two invalid paths. Of the remaining 19 paths there were a possible remaining 50 effects, (57 effects, less the 7 indirect effects that do not exist because of the ordering of the variables). The Time-2 symptomatology model had only one invalid path leaving 20 remaining paths. The Time-3 and Time-4 models also had two invalid paths leaving a total of 19 paths for each model.

There were instances when some significant, or at least salient, paths had valence signs in the opposite direction to the direction hypothesized. There were other paths that were opposite to the predicted valence. However, they were not of a salient magnitude. For the Time-1 model the paths connecting CF with EMP were both negative and positive. A positive relationship was predicted. In this case there was a significant negative direct effect and a positive indirect effect resulting in a non-salient total effect. The relationship between ED and FAD also demonstrated this pattern. There was a significant negative direct effect and a significant positive indirect effect. In both these instances the opposite valence signs cancelled out a total effect. For the Time-2 model there were no significant or salient paths that demonstrated a valence sign opposite to the hypotheses. The Time-3 symptom model also had two of these conflicting paths. ED and FAD demonstrated a significant negative direct and a non-significant positive indirect effect contrary to predictions also leaving a marginally significant total effect. The path connecting EMP and SYMP also demonstrated a salient positive direct effect and a salient negative indirect effect, leaving no total effect. The Time-4 model had only one

salient beta value that was in the opposite direction to the one predicted. The relationship between ED and SYMP demonstrated a salient positive instead of negative direct effect. This path was not consistent over the four time periods going from an indirect negative significant effect at Time-1, a non significant effect at Time-2 and Time-3, and a salient positive direct effect at Time-4.

The social support variables CF and SAT (path #1) were positively related in all four points in time. Both direct and total paths were significant with beta values ranged from .194 to .324. Clearly these two support variables are related in the cross-sectional path models and demonstrated that they in fact could be considered as one factor for subsequent analyses. This also clearly demonstrated that one's satisfaction with support is partially due to how many contacts an individual has during the last two weeks. However, it is also possible that satisfaction with support could lead to higher contact frequency.

Path #2 connects SAT and ED. This path was invalid in all of the models and there is not much to be said about their relationships since the error coefficients were too large. CF and ED (path #3) were not related in any consistent manner. This path was not significant in the Time-1, Time-2, or Time-3 models. There was a marginally significant direct and total effect in the Time-4 model. The general conclusion here is that contact with one's social network was not related to education for this group of participants. There are some difficulties when considering the effect SAT and CF had on ED. The primary reason for this may be due to the fact that education did not change appreciably during the two year evaluation period making teasing out effects impossible. Measures of ED changed so little that the measures were multi-collinear.

Path #4 connects SAT and EMP. These variables were positively connected with a direct effect at Time-1 and a marginally significant total effect. However, there was no significant effect for the balance of the path models. This indicates that these two variables are likely not related in a consistent manner for these participants. ED and EMP (path # 5) are related in the hypothesized manner displaying salient direct and total effects at all four points in time.

Path # 6 connects CF and EMP (path # 6). The only time that this relationship attains a significant path is for Time-1. At this point it demonstrated both a significant negative direct effect and a significant positive indirect effect. At the other points in time these variables were clearly not related in any consistent manner.

The relationship between SAT and INC (path # 7) was mixed. At Time-1 there are clear positive relationships between the variables. At Time-1 there was a significant positive direct effect. At Time-2 there was a significant positive direct effect. At Time3 and at Time-4 these relationships dissipated completely.

Path # 8 joins ED and INC. in this case there were positive relationships between these variables as expected and this relationship remained $p < .0001$ over the four points in time. Time-1 and Time-2 had positive significant direct, indirect, and total effects. At Time-3 only an indirect effect appeared. And at Time-4 there were an indirect and total effect evident.

The relationship between EMP and INC (path #9) was consistent for all four time frames. In all cases there were a significant positive direct and total effects. This pattern would be expected regardless of the population of interest. The relationship between CF

and INC (path #10) was inconsistent. The majority of the time frames demonstrate a non-significant relationship between the two variables. The only exception was in Time-2 where a salient positive indirect effect existed.

FAD and SAT (path #11) were related in all of the time frames. There were salient positive direct and total effects evident for all if the four points in time. However, the indirect effect only reaches significance at times one and two.

Path # 12 connects ED with FAD. There was a consistent positive indirect effect connecting these variables in Time-1 and Time-2 models. There was also some evidence of a direct effect at Time-1 and Time-3; but in these cases the direct effect was negative, not positive as hypothesized. At Time-3 there was also evidence of a negative total effect which was not evident in any of the other time frames.

EMP and FAD (path #13) were clearly and consistently positively related with the exception of Time-4 when the path was invalid. Time-1, Time-2, and Time-3 models demonstrated positive indirect and total effects. Interestingly, a direct effect was not found.

Path # 14 connects INC and FAD and results were consistent. INC and FAD were positively related in all cases as predicted. All time frames have positive total effects and all time-frames demonstrated positive direct effects. The final FAD relationship is with CF (path # 15). At each time point there was an indirect significant positive relationship between CF and FAD. At Time-1 and Time-4 there were also positive total effects evident.

Path # 16 connected symptomatology with satisfaction. At all time points SAT

and SYMP had a negative significant total effect. Times-1, -2, and -4 also had statistically significant negative indirect effects. Times-1, -2, -3 had statistically significant negative direct effects.

The relationship between ED and SYMP (path #17) was not consistent among the four points in time. Time-1 had only a salient negative indirect effect. Time-2 and Time-3 had non-significant effects and Time-4 showed a direct positive effect.

Path # 18 joins EMP with SYMP and results are mixed for this relationship. Time-1 information indicated that EMP and SYMP were negatively related as expected and had both significant direct and total paths. Time-2 results indicate that there is also a salient negative total relationship between these two variables. Time-3 results indicate that there was a salient negative direct relationship and a salient positive indirect relationship. These two relationships cancel each other out. At Time-4 none of the relationships were significant.

INC and SYMP (path #19) relationships were also not consistent. For Time-1 the path was invalid and was deleted from the model. At Time-2 there is evidence of a significant indirect effect only. Time-3 data indicate that there was a salient positive direct effect between income and symptomatology. Time-4 data indicated that there was a salient negative indirect effect between these two variables.

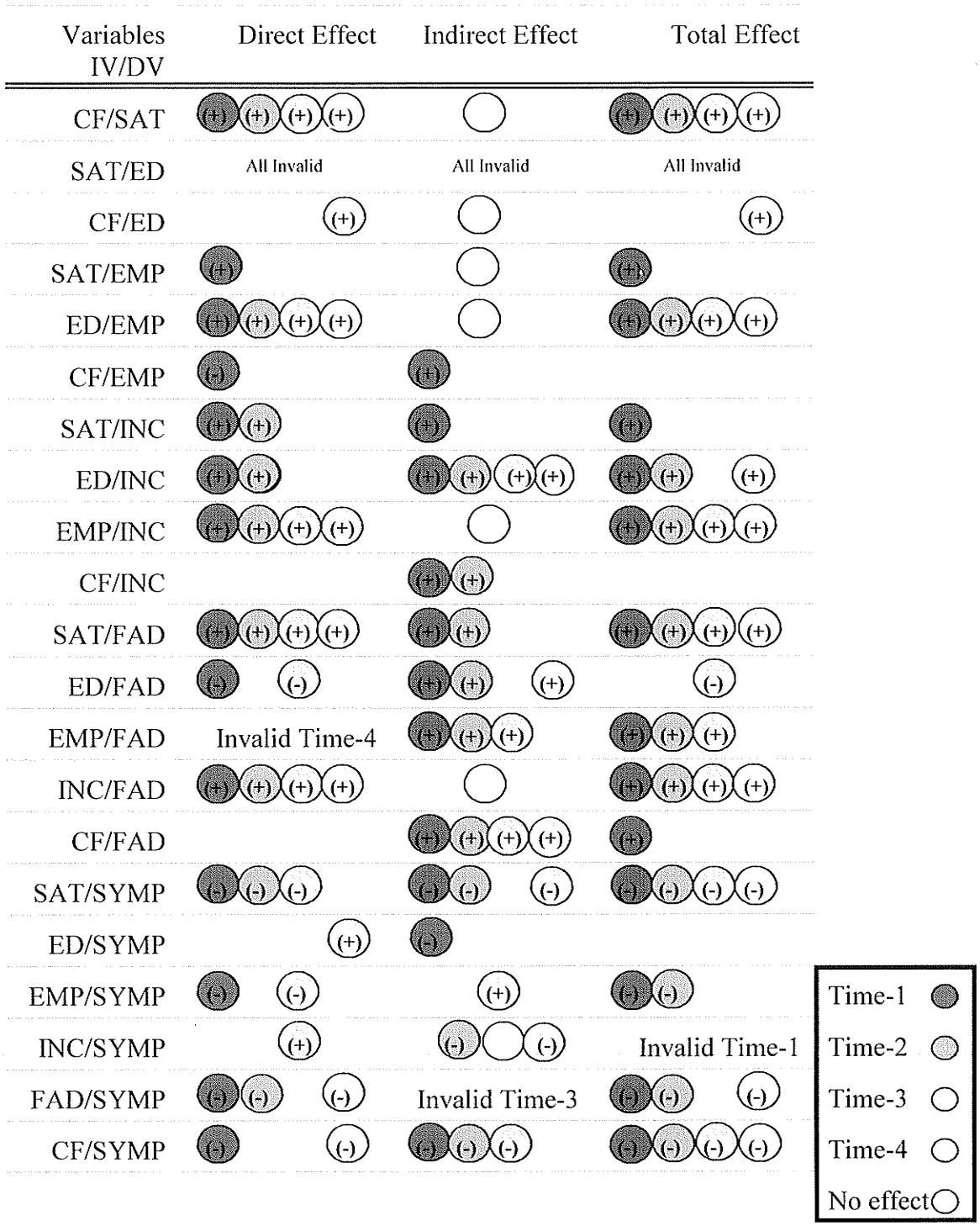
Path #20 connects FAD with SYMP and results were reasonably clear. Excluding Time-3 results indicating an invalid path, the other time-frame results indicate a consistent significant negative total relationship connected these two variables. There were also stable results indicating significant/salient negative direct effects.

And finally, CF and SYMP (path # 21) were related in a consistent manner. All of the time frames indicated that a significant negative total relationship existed between CF and SYMP. Time-1, Time-2, and Time-3 results indicated a significant negative indirect effect, while Time-1 and Time-4 had significant/salient negative direct effects.

Cross-Sectional Model Comparisons

When the cross-sectional path models are compared (see Figures 8 and Figure 9 below) it becomes clear that there was a pattern of consistent or concordant findings between the social support, social functioning, and symptomatology variables. The three replications indicate a number of stable relationships through the four distinct time periods. In total, the cross-sectional models have a total of 84 paths, 21 paths for each cross-sectional model. Only six of these paths were deemed to be invalid. The most consistent invalid path is the one joining SAT with ED. As stated previously, there was very little variability in the education variable and this may be due to the impact of the social functioning variables and education. Also, education was established prior to the support measures. To really be able to evaluate the impact of social support upon education participants would need to be evaluated at index admission or right after symptoms began to be expressed. This issue also impacted cross-lagged results discussed later.

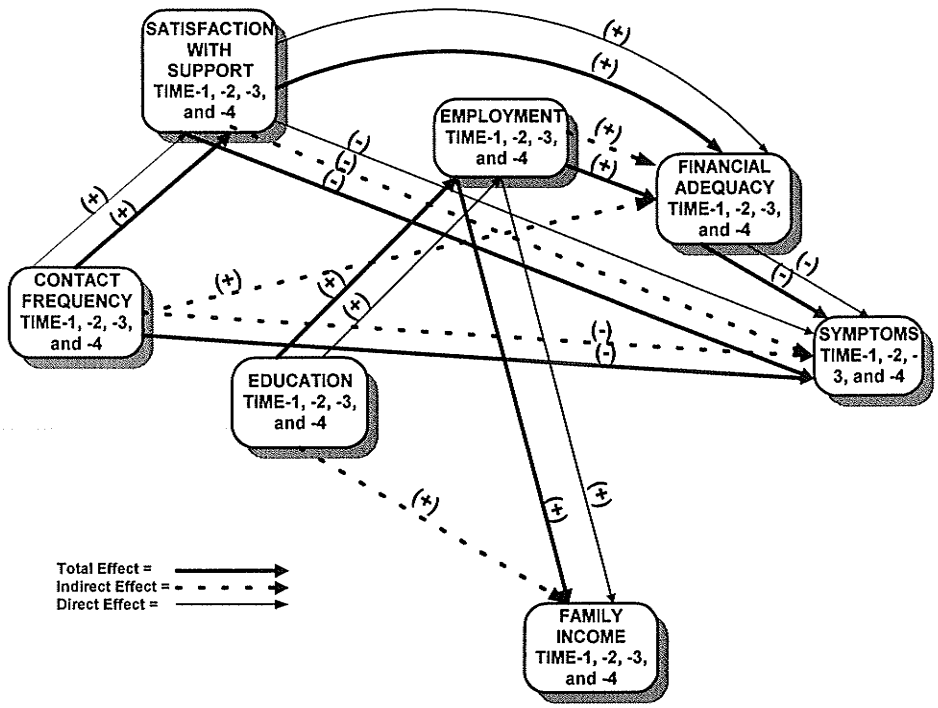
Figure 8.
Symptomatology Cross Sectional Model Comparisons for the Four Points in Time.



Note: Abbreviations are as follows: IV/DV = independent/ dependent variable pairs for each path, CF = contact frequency, SAT = satisfaction with support, ED = years of education, EMP = employment score, INC = family income, FAD = financial adequacy score, SYMP = symptomatology score, (-) = negatively related variables, and (+) = positively related variables.

The paths that were the most stable through the four time points include the path connecting CF and SAT (those effects with at least three similar findings see Figure 9). This relationship was clearly positive with significant direct and total effects. The two social support variables were also consistently related to the symptomatology variable. In most instances the relationship between the two support variables (CF and SAT) demonstrated a negative relationship with the pathology variable (SYMP), including direct, indirect, and total effects. Since these two support measures interacted with the pathology measures in a consistent way, the decision was made to combine these measures for later time-lagged models, in the interest of parsimony (see Chapter 5).

Figure 9. Paths of effects that were concordant through three of four possible cross-sectional results.



Social functioning variables (FAD, ED, EMP, and INC) also demonstrated relatively stable relationships with each other, but they were less consistently related to symptomatology. For example, ED and EMP, ED and INC, EMP and INC, INC and FAD are directly related in a positive manner. EMP and FAD are related in an indirect manner resulting in a total positive effect. ED and INC also have a consistent positive indirect relationship with each other. ED and FAD were related in both a direct negative and an indirect positive manner. EMP and FAD demonstrated reasonably consistent positive indirect and total effects.

Support measures were also related to social functioning. SAT and FAD were positively directly and indirectly related. FAD and CF were indirectly related in a positive manner. Symptoms were negatively related to FAD in a direct manner resulting in a significant total negative effect.

Cross-Sectional Hypothesis Testing and Discussion

Whether one considers the structural (CF) or functional (SAT) measures used in the current research the results are similar. Structural and functional measures of social support are not mutually exclusive and do not measure entirely different entities, but as noted by Patrick et al. (1986), it appears as if they focus on different aspects of the support construct. Social support is accepted to be a multidimensional construct (Cobb, 1979; Dean et al. 1989; House & Kahn, 1985). According to Revicki and Mitchell (1990), much of the available literature attempts to operationally define the global construct of social support and few studies have selectively explored the differential effects of various

aspects of social support.

The current results indicated that these two measures of social support, quantitative (structural, CF) and qualitative (functional, SAT), correlate well with each other (Time-1, $r_{(232)} = .46$, $p < .0001$; Time-2, $r_{(146)} = .48$, $p < .0001$; Time-3, $r_{(123)} = .41$, $p < .0001$; Time-4, $r_{(96)} = .58$, $p < .0001$) supporting work by Sarason et al. (1983) and the work by Allen, Ciambrome, and Welch, (2000) and McKee-Ryan, Song, Wanberg, and Kinicki, 2005) who report that greater perception of support has a positive impact on mental health. These results also support the findings by Kafetsios, (2006), Mittal, Fortney, Pyne, Edlund, and Wetherell (2006); and Putnam (2000) that social connections and mental well-being are related. These results also support the findings of King, et al. (2000) that social support is one of the most important factors perceptions of happiness. Hostility is a risk factors for the progression of depressive tendencies (Heponiemi, Elovainio, Kivimaker, Pulkki, Puttoenen, & Keltikangas-Jarvinen, 2006; Vahtera, Kivimaki, Uutela, & Pentti, 2000). Yet, contact frequency only accounts for 21to 25% of the variability in satisfaction with support. Multicolinearity, in this case, was not a problem and the two measures of social support clearly represent different aspects of the support construct. According to Vaananen, Vahtera, Pentti, & Kivimaki, (2004), more structural measures like network membership may influence mental health directly. According to Fiore et al. (1986), those individuals with more frequent contact with their networks ask for support more frequently and perceive support to be more available. The positive, large to moderate, correlation between contact frequency and satisfaction with support tends to corroborate Fiore et al.'s (1986) results and the findings of Brown et al.

(2003) that just the presence of a supportive other is sufficient to convey social support. These measures of different aspects of the support concept clearly demonstrate that both structural and functional measures of support can be used to assess the relationship between mental health and social support. These results also support the findings of Fujita, and Kanaoka, (2003); and Gencoz and Astan, (2006) that perception of social support is related to the stability of mental health.

When considering the impact of contact frequency on symptomatology, two out of four models indicate that contact frequency demonstrated a direct effect. In fact, the direct and indirect effects of Time-1 contact frequency on symptomatology were approximately equal (Time-1, df = 231, direct effect β = -.0610, p .009; indirect effect β = -.0651, p < .0001, resulting in a total effect of β = -.124, p < .0001). This pattern did not replicate completely. In fact, both Time-2 and Time-3 models indicated that the indirect effect was the more important path (Time-2, df = 145, indirect effect β = -.1441, p < .0001, and a total effect of β = -.1339, p = .0043; Time-3, df = 122, indirect effect β = -.1327, p < .0001, resulting in a total effect of β = -.1696, p < .0001). Yet, the Time-4 results indicated that the direct effect was the most consistent finding (df = 95, direct effect β = -.1047, p = .0864; resulting in a total effect of β = -.1430, p = .0112). The Time-4 statistical test results were definitely less powerful than the previous time frames so it appears that for CF the indirect effect may be the most important or consistent finding. These findings are contrary to those of Henderson (1998), who stated that it is the perception, rather than the veridical quantity, of support that was important for health.

The current results confirm findings of Bolger, Zuckerman, and Kessler (2000);

Kafetsios, (2006); Putnam (2000); Reis (1988), Revicki and Mitchell (1990), and Turner (1981) that individuals need to maintain a basic social support resource for adequate psychiatric and physical well-being. According to Sherkat and Reed (1992), human social relationships modify the negative effects of life stressors on mental and physical health. The current research demonstrates that social support clearly modifies the effect of life stressors, like low income, insufficient levels of employment, and perceptions of poor financial adequacy, on psychiatric symptomatology, supporting the work of Brown, Bhrolchain, and Harris (1975) and Vitaiano et al. (2001). These findings are consistent with the work by Angermeyer et al. (2002); Simon, (2003); Suurmeijer, Reuvekamp, & Aldenkamp (2001); and Trompenaars et al (2006) who found that symptom reduction improves a patients quality of life.

The indirect effects of satisfaction with support and contact frequency in the symptomatology model significantly influence psychiatric symptomatology in a negative manner. The indirect effects are computed from the sum of the cross-products of the direct effects making up the path(s) between the independent and the dependent variables. Social support, whether measured as a functional or structural variable influences psychiatric symptomatology indirectly by influencing the effects of employment, family income, and financial adequacy perceptions.

Satisfaction with support clearly has a significant impact upon symptomatology based upon the four points in time. All of the data sets demonstrated significant negative total effects (Time-1, $\underline{df} = 231$, $\underline{\beta} = -.4009$, $\underline{p} < .0001$; Time-2, $\underline{df} = 145$ $\underline{\beta} = -.4910$, $\underline{p} < .0001$; Time-3, $\underline{df} = 122$, $\underline{\beta} = -.5923$, $\underline{p} < .0001$; Time-4, $\underline{df} = 95$, $\underline{\beta} = -.2816$,

$p = .0047$). Indirect effects occurred in three out of four models (Time-1, $df = 231$, $\beta = -.0752$, $p = .0380$; Time-2, $df = 145$, $\beta = -.0338$, $p = .0879$; Time-4, $df = 95$, $\beta = -.0701$, $p = .0977$). There were also salient direct effects in three of four models (Time-1, $df = 231$, $\beta = -.2703$, $p < .0001$; Time-2, $df = 145$, $\beta = -.4673$, $p < .0001$; Time-3, $df = 122$, $\beta = -.5709$, $p < .0001$). However, given the magnitude and consistency of the effects, it appears that a direct effect is the more reliable finding. These outcomes support the research by Arling (1987), House, Robbins, and Metzner (1982); Olsen, (1993); and LaRocco, House, and French (1980). These findings also are in agreement with Fujita and Kanaoka's (2003), and Gencoz and Astan's (2006) findings that perception of social support is related to the stability of mental health. Contrary to the findings of Brugha et al. (1987) satisfaction with support from others was not found to be less predictive of outcome than other measures of support.

Some theorists believe that social support has its influence through the interaction of social support and environmental stressors (Cohen & Wills, 1985; Dean et al. 1990; Lin & Ensel, 1989; Sherkat & Reed, 1992). This theoretical position is supported with the indirect effects evident in three of the time frames. Others believe that social support impacts health directly. The current results clearly demonstrate that social support impacted symptomatology in a direct and inverse manner which lends support to the theory that social support acts independently of environmental stressors. These results support the findings of Cohen, Gottlieb, and Underwood (2000); King et al. (1999); Kiecolt-Glaser and Newton (2001); McDonald et al. (1999); Vaananen, Vahtera, Pentti, and Kivimaki, 2004) that higher levels of social support have been found to have a

beneficial effect on psychological well-being. Social support is said to have a direct effect on health when it improves health independent of the stressors that may be present (Sherkat & Reed, 1992). The current findings tend to indicate that both indirect and direct theoretical position may be acting for this group of patients.

According to Sherbourne (1988), there has been a controversy over whether social support acts in a direct or indirect manner. Each of the support measures influenced the symptomatology measure in both direct and indirect manners supporting both the direct and indirect theories of the influence of social support on health. These results also support Ullah, Banks, and Warr's (1985) belief that neither direct nor indirect theories are adequate by themselves to explain the impact of social support on health. Social support, whether measured as a structural or functional variable, appears to act in a direct and indirect manner simultaneously on self-reported psychiatric symptomatology, supporting work by Bolton and Oatley (1987), Cohen and Willis (1985), and Sherkat and Reed (1992). According to Vaananen, Vahtera, Pentti, and Kivimaki, (2004), more structural measures like network membership may influence mental health directly. However the current results indicate that the impact is also indirect. The current results are also congruent with the opinion of Ullah et al. (1985), and support the theory that neither direct nor indirect theories are comprehensive enough by themselves to explain the entire relationship between social support and well-being. Both satisfaction with support and contact frequency demonstrated significant relationships with symptomatology in the predicted manner and appear to be important for mental health. These findings support the results reported by Mittal, Fortney, Pyne, Edlund, and Wetherell (2006) that low

levels of social support and depression severity, have been found to negatively impact quality of well-being. Higher contact with one's network and stronger perceptions of support are both directly and indirectly inversely related to psychiatric symptomatology, supporting the findings of Allen, Ciambrome, and Welch, (2000); Gencoz and Astan, (2006); Fujita and Kanaoka, (2003); House and Khan (1985), Helliwell, and Putnam (2004); Kessler and McLeod, (1985), McKee-Ryan, Song, Wanberg, and Kinicki, (2005); Patrick et al. (1986); Revicki and Mitchell, (1990); Reis, (1988), Schulz et al. (1987). Given that both measures of support appear to be related to pathology it appears important to consider all forms and sources of support when evaluating an individual's coping resources (Dean, Lin, & Ensel, 1981; Dean, Kolody, & Wood, 1990; House & Kahn, 1985; Lin, Dean, & Ensel, 1986). Given that good social support is associated with coping, positive psychological and physical health, and quality of life, we consider low social support a risk factor for a globally poor outcome (Williams, Turner, Hatzakis, Chu, Rodriquez, Bowen, & Haselkorn, 2004). According to Kafetsios (2006) and Michalak, Yatham, Kolesar, and Lam (2006), people thought that social support was an important factor in determining quality of life.

Social support appeared to have little relationship to education. Paths connecting CF and SAT with ED were either invalid, not significant, or only slightly related [as in the Time-4 data (Time-4, $\underline{df} = 95$, $\underline{\beta} = -.1141$, $\underline{p} = .0668$)]. This was contrary to expectation. According to Vega et al. (1991), better educated individuals experience more support since they have higher levels of friend contact. This finding was not supported by the current research (at least for this group of clients). The relationship between contact

frequency and education was positive but it did not reach significance. Also, the paths (direct, indirect, and total) connecting satisfaction with support and education were eliminated due to excessive error measures (either the effect of satisfaction with support on education is negligible or there was not enough variability in comparison to error to assess an effect).

It is also possible that these divergent findings are due to the nature of the samples considered. This research used a self-help group of individuals dealing with unipolar or bipolar depressive disorders; Vega et al. (1991) considered only Mexican American immigrant females who had been in the United States for at least two years and were not necessarily experiencing clinical depressive symptoms. Also, the measure of psychiatric symptomatology used for the two pieces of research was significantly different. Vega's study used a modified measure of the Centre for Epidemiological Studies of Depression scale, the CES-D (Ross & Mirowsky, 1989), while the current research used a self-referenced measure of combined manic, depressive, and possibly other symptomatology rather than a simple depression measure. These findings were also contrary to Ross and Mirowsky's (1989) findings that higher levels of education were related to higher levels of social support.

According to Roberts et al. (1982), the relationship between employment and psychological well-being is complex. The results from the current research support this complexity. For the Time-1 data, both measures of social support displayed significant positive relationships to the employment score. Those with higher satisfaction scores were more likely to have higher employment scores. Those with higher social support

assessed as contact frequency displayed an indirect positive effect and a direct negative effect. These two effects cancelled each other out, resulting in a non-significant total effect. Individuals who are employed appeared to have a lower level of contact frequency with friends and family (indicated by the direct effect). Yet indirectly, employment and contact frequency are positively related. The indirect effect on unemployment could have been mediated by the relationship with education. Employment may serve to boost contact frequency scores indirectly (social support), supporting Jahoda (1982) and Lloyd's (1986) findings, or those with higher contact may be more likely to find employment because of networking connections (boosting employment opportunities) or because they are not as ill.

However, For Time-1 individuals, contact frequency also displayed a significant negative direct relationship with employment score; as contact frequency increased employment scores decreased. It is possible individuals who are employed do not have time to socialize to the same extent as individuals who are not employed. These results, when combined with the inverse indirect effect of social support on psychiatric symptomatology (mediated by satisfaction with support), support the findings of Cohen and Willis (1985) that social support buffers the negative psychological effect of unemployment by substituting family and friend social interaction for lost employment interaction. It is also possible that individuals who are more ill with psychiatric symptomatology have more time to spend with their support network since they are too ill to hold a job. Due to the cross-sectional nature of this component of this design it is not possible to determine direction of causation unequivocally.

Time-1 results were not replicated in any format when one considered the employment social support relationships. All of Time-2, Time-3, and Time-4 results indicated unequivocally that employment was not related to either contact frequency or satisfaction with support. This brings into question the original Time-1 results above. Time-1 results were possibly a chance finding. However, not much can be said about these findings since they may have been impacted by the aberrant (non-normal) distribution of the employment variable.

At Time-1, as hypothesized, individuals reporting high satisfaction scores tended to have higher family incomes confirming the findings of Brown and Lawrence (1985). These authors found that depressive symptoms were higher among those with fewer financial resources and less social support. All three effects ($df = 231$, direct, $\beta = .3283$, $p = .081$; indirect, $\beta = .2114$, $p = .0200$, and total effect, $\beta = .5115$, $p = .0130$) were positive and the indirect and total effects were statistically significant. The direct and indirect effects were approximately the same magnitude. Considering Time-1 data, it was thought that the indirect effect of satisfaction with support was through the effects of support on employment. The more satisfied respondents were with support, the larger were their employment scores. Satisfaction with one's support interactions may result in a more stable environment possibly resulting in less psychiatric symptomatology and higher levels of employability, leading to higher family incomes. It may also be that individuals from families with more financial resources are more satisfied with family relationships and individuals with more stable employment may be more satisfied with their social support from fellow workers.

Interestingly, Time-2, Time-3, and Time-4 data do not support the theories above. Satisfactions with satisfaction with support and contact frequency at Time-2 were positively related to family income but at a marginal level. But these findings did not replicate for Time-3 and Time-4. Again, the causal direction of the effects cannot be unequivocally determined with this cross-sectional model. The following time-lagged component of this research indicated that social support (the compound measure) was positively related to income in two of the three social causation models. When considering the social selection models household income was not related to social support since the paths were either

non-significant or invalid.

At Time-1, both support measures were significantly related to financial adequacy in the predicted manner. Individuals who were satisfied with their support and had more frequent contact with support resources clearly perceived their financial situation to be more adequate. Contact frequency indirectly influenced financial adequacy ($\underline{df} = 231$, $\underline{\beta} = .264$, $\underline{p} = .0060$) via satisfaction with support, employment, education, and family income. Contact frequency also displayed a significant total effect upon financial adequacy perceptions ($\underline{df} = 231$, $\underline{\beta} = .4551$, $\underline{p} = .0022$) and a direct effect failed to reach statistical significance ($\underline{df} = 231$, $\underline{\beta} = .187$, $\underline{p} = .1210$). The relationship between contact frequency and financial adequacy perceptions was not straightforward. For the Time-1 sample there were indirect and total effects of CF on FAD. All subsequent time frames demonstrated only indirect effects (Time-2, $\underline{df} = 145$, $\underline{\beta} = .2248$, $\underline{p} = .0520$; Time-3, $\underline{df} = 122$, $\underline{\beta} = .3485$, $\underline{p} = .0070$; Time-4, $\underline{df} = 95$, $\underline{\beta} = .5503$, $\underline{p} = .0040$).

At Time-1, satisfaction with support related to financial adequacy directly (df = 231, β = 1.096, p = .0020) and indirectly (df = 231, β = .3355, p = .0380) via employment and family income, resulting in a total effect of β = 1.616, p < .0001, df = 231. Subsequent time data clearly indicate that SAT is positively related to FAD (Time-2: df = 145, total effect, β = .9357, p = .0040; direct effect, β = .7796, p = .0200; and indirect effect, β = .2166, p = .0760) but the indirect effect was only marginal. Time-3 results indicated that SAT and FAD were related directly and totally only (df = 122; total, β = .1.4576, p < .0001; direct, β = 1.5205, p < .0001) and this result repeated for the Time-4 data (df = 95, total, β = 1.4721, p = .0010; direct, β = 1.8193, p = .0020). It appears that satisfaction with support consistently and directly relates to financial adequacy perceptions, while CF consistently demonstrates an indirect relationship with FAD. The two differing forms of social support interact with FAD in differing ways.

Williams, Ware, and Donald (1981) have proposed that stressful life events have a negative effect on mental health outcomes. Stressful life events have also been found to impact onset of episodes of major depression (Kendler, Karkowski, & Prescott, 1999). Financial adequacy perceptions are related to household income and employment. Individuals who are unemployed due to illness contend with lower household incomes resulting in economic stress and this should result in lower scores of financial adequacy. Social support reduces the impact of stressful life events (Sherkat & Reed, 1992) and various other stressors (Cohen & Wills, 1985; Dean et al. 1990). One would expect that higher social support could reduce the stressors related to lower household incomes and diminish the impact of inadequate financial resources (Vitaiano et al. 2001).

Other social functioning variables (ED, EMP, and INC) were also related to each other and psychiatric symptomatology as predicted. At all four points in time, education exerted a consistent significant positive influence on employment scores (Time-1, $df = 231$, direct effect, $\beta = 3.692$, $p < .0001$, and total effect, $\beta = 3.61$, $p < .0001$; Time-2, $df = 145$, direct effect, $\beta = 1.5828$, $p = .0020$, and total effect, $\beta = 1.5838$, $p = .0020$; Time-3, $df = 122$, direct effect, $\beta = 1.1638$, $p = .0300$, and total effect $\beta = 1.1777$, $p = .028$; and Time-4, $df = 95$, direct effect, $\beta = 1.7871$, $p = .002$, and total effect, $\beta = 1.7871$, $p = .001$). Indirect effects were not applicable due to path orderings. In other words is was physically impossible for an indirect effect to exist. These effects were relatively consistent but the power to detect significance reduced as time frames progressed due to shrinking sample sizes. Small sample sizes are common in the mental health research field (Bothwell & Scott, 1997). Education and employment are positively related in this sample supporting the findings of Wheaton (1980) and Costello (1991).

Education and income were also related as predicted. However, the effects were not as consistent over the four time frames (Time-1, $df = 231$, direct effect, $\beta = .3271$, $p = .007$, indirect effect $\beta = .2307$, $p < .0001$, and total effect, $\beta = .561$, $p < .0001$; Time-2, $df = 145$, direct effect, $\beta = .3378$, $p = .0258$, indirect effect $\beta = .0888$, $p = .0407$, and total effect, $\beta = .4255$, $p = .00642$; Time-3, $df = 122$, indirect effect $\beta = .0932$, $p = .0555$; Time-4, $df = 95$, indirect effect $\beta = .17731$, $p = .0199$, and total effect, $\beta = .3419$, $p = .0521$). Clearly the results become less consistent at later time frames, possibly due to a lack of power. However, the majority of the findings do tend to support the results of Brown and Lawrence (1985) that a high positive correlation exists between

education and income.

Higher educational attainment also appears to have a complicated relationship to financial adequacy perceptions. All four models demonstrated salient relationships between ED and FAD. At Time-1 there were indirect and direct effects but they were in opposite directions (df = 231, direct effect, β = -.4257, p = .024, indirect effect, β = .3429, p = .002). For Time-1 the effects of education on financial adequacy perceptions appeared to cancel each other out, resulting in a neutral total effect. At Time-2 only an indirect effect of any consequence existed (df = 145, β = .30903, p = .010). Time-3, exhibited a direct and total effect (df = 122, direct, β = -.56371, p = .017; total β = -.4445, p = .067). However, these effects were more marginal. At Time-4, once again, there is evidence of a marginal indirect effect existing between ED and FAD (df = 95, β = .1352, p = .0841). When one considers all four points in time results are inconsistent and the effects were marginal at the later points in time. It appears from the Time-1 data that education has an indirect positive effect on financial adequacy, moderated by both employment score and family income. As education increases, employment scores increase, family income increases, and financial adequacy perceptions increase. This pattern is easy to explain. This is the normal pattern for higher educated individuals and requires very little discussion. These findings are congruent with research by Ross and Huber (1985).

The more intriguing finding at Time-1 and Time-3 is the direct negative effect education appears to exert on financial adequacy perceptions. All of the direct effects were negative at all four points in time. This does not appear to be congruent with general

population patterns. One would expect higher educational attainment to be related to greater income and therefore higher adequacy perceptions because of higher incomes that come from higher educational attainments. However, if one considers that many individuals with psychiatric difficulties fail to acquire and maintain employment commensurate with their educational achievement, this result begins to make some sense. Individuals with higher levels of education usually have higher expectations for higher incomes. Therefore, they expect to be more financially secure. When they evaluate their standard of living in comparison with others of a similar educational attainment, they are likely to be disappointed, leading to poorer perceptions of financial adequacy. These results could also be indicative of a reduction in the relationship between education and income leading to poorer financial adequacy perceptions due to the current changes in the economic climate. These results would be better explained if there were current data on financial adequacy perceptions in the general Winnipeg population. Such data are not available. The inconsistency in these results requires further research.

The Time-1 model demonstrated that education appeared to exert its effect on psychiatric symptomatology in an indirect manner ($\underline{df} = 231$, indirect effect, $\underline{\beta} = -.0253$, $\underline{p} = .040$) through its effect on financial adequacy perceptions, employment score, and family income. However, these results do not replicate at the other three points in time. Time-2 and Time-3 models do not have any significant or salient relationships between ED and SYMP. The Time-4 model, in fact, demonstrates a marginal positive direct effect ($\underline{df} = 95$, $\underline{\beta} = .1190$, $\underline{p} = .082$). According to Sherkat and Reed (1992), education is negatively related to depression. Only the Time-4 results demonstrate a direct effect

between ED and SYMP and this relationship is in the opposite direction. The balance of the current results did not support a direct negative relationship between education and symptoms. None of the first three models included a direct effect of ED on SYMP. Therefore, according to the current data, education by itself did not exert a consistent direct effect on psychiatric symptomatology.

These findings partially support the results of Sherkat and Reed (1992) in a recently bereaved population. Education significantly predicted depression in these bereaved individuals until the social support variables were included into the model. The zero order correlation between education and depressive symptomatology (Time-1, $\underline{r}_{(232)} = -.04$, $\underline{p} = .506$; Time-2, $\underline{r}_{(146)} = -.012$, $\underline{p} = .889$; Time-3, $\underline{r}_{(123)} = .033$, $\underline{p} = .714$; Time-4, $\underline{r}_{(96)} = .145$, $\underline{p} = .157$) in the current research did not support that education was significantly related to depressive symptomatology in this group of individuals prior to the support variables being considered in the model. The current results indicate that education does not significantly predict symptomatology directly, when social support variables are included in the model (partial correlation coefficients ED and SYMP controlling for social support; Time-1, $\underline{r}_{(232)} = -.0467$, $\underline{p} = .577$; Time-2, $\underline{r}_{(146)} = -.0028$, $\underline{p} = .974$; Time-3, $\underline{r}_{(123)} = .0030$, $\underline{p} = .975$; Time-4, $r_{(96)} = .1299$, $\underline{p} = .239$), this is contrary to Sherkat and Reed's (1992) and Ross and Mirowsky's (1989) findings.

These divergent findings must also be considered in light of the differences in the measures used to assess psychiatric symptomatology. Sherkat and Reed's (1992) and Ross and Mirowsky's (1989) research used a modified measure of the Center for Epidemiological Studies of Depression (CES-D; Ross & Mirowsky, 1984). The CES-D

measure of depression is specific to a community sample and does not indicate a diagnosis of clinical depression (Ross & Mirowsky, 1989). The symptomatology measure employed with the current research was intended to give an estimate of the severity of clinical symptoms for individuals with both manic and depressive symptomatology so it is not directly comparable to the CES-D.

At Time-1 family income was not related to psychiatric symptomatology since the path had to be deleted due to inflated error measures. At Time-2 there was some evidence of a negative indirect effect (df = 145, β = -.0214, p = .0428). At Time-3 there was a marginally negative direct effect (df = 122, β = -.0448, p =.0809). And at Time-4 there was a marginally significant indirect effect (df = 95, β = -.0142, p = .1080). These results, in general, appear not to support Ross and Mirowsky's (1989) general population sample results that found that family income levels were negatively related to depression levels. These findings must be considered in light of the special nature of this population, the necessity of deleting the path connecting psychiatric symptomatology and family income (due to excessive error), and the fact that the zero order correlation between family income and symptomatology, $r_{(232)} = -.17$ (p = .006), was in the predicted direction.

Considered together education, employment, and family income are common indicators of socioeconomic status. Nonmedical factors like employment, explain some of the level and distribution of health in populations (Benzeval & Judge, 2001; Berkman, 1995; Lavis et al. 2001). Employment is an important social marker (Brenner & Mooney, 1983), unemployment has an independent effect on depressive symptoms and exacerbates a person's ability to adjust to stress (Dressler, 1986), and lack of financial resources

hampers one's ability to meet basic needs, influencing psychological health (Feather, 1989). According to Ross and Huber (1985), unemployed individuals face economic hardship because of a lack of income resulting in depression and feelings of helplessness. Chronic economic difficulties may explain the relationship between income and depressive symptomatology (Pearlin et al. 1981). The majority of the current results support the findings in the literature. Employment was related to family income, financial adequacy, and psychiatric symptomatology in the predicted manner.

Employment was related directly to higher levels of household income (Time-1, df = 231, direct effect, β = .063, p < .0001, total effect, β = .0702, p < .0001; Time-2, df = 145, direct effect, β = .05607, p = .015, total effect, β = .0693, p = .003; Time-3, df = 122, direct effect, β = .0801, p = .001, total effect, β = .0824, p = .001; Time-4, df = 95, direct effect, β = .0992, p = .002, total effect, β = .1077, p = .001) and perceptions of financial adequacy (Time-1, df = 231, indirect effect, β = .046, p < .0001, total effect, β = .0282, p = .051; Time-2, df = 145, indirect effect, β = .03215, p = .026, total effect, β = .0791, p = .034; Time-3, df = 122, indirect effect, β = .0413, p = .008, total effect, β = .0556, p = .096; Time-4, df = 95, invalid path due to high error measures).

Individuals with higher employment scores have higher family incomes, and higher perceptions of financial adequacy as predicted. Employment and perception of financial adequacy are related to psychiatric symptomatology in an inverse manner as hypothesized. Employment was negatively related to symptomatology (Time-1, df = 231, direct effect, β = -.0076, p = .002, total effect, β = -.0095, p < .0001; Time-2, df = 145,

total effect, β = -.0136, p = .103; Time-3, df = 122, direct effect: β = -.01312, p = .091, indirect effect, β = -.0036, p = .102; Time-4, df = 95, non significant/salient path). The pattern of results indicates progressively more marginal results from Time-1 to Time-4.

According to Keith (1985), income exerts its effect on health indirectly through perceived adequacy of income rather than directly. The present results support Keith's (1985) findings. Family income did not exert a consistent direct effect on symptomatology since the path had to be deleted at Time-1, Time-2 demonstrated an inverse indirect effect (df = 145, β = -.0214, p = .043). There was a direct marginal negative effect at Time-3 (df = 122, β = -.0448, p = .081), and an indirect marginal negative effect at Time-4 (df = 95, β = -.0142, p = .1083).

However, family income did exert a consistent effect on perception of financial adequacy (Time-1, df = 231, direct effect, β = .7451, p < .0001, total effect β = .6932, p < .0001; Time-2, df = 145, direct effect, β = .5734, p < .0001, total effect β = .6082, p < .0001; Time-3, df = 122, direct effect, β = .5150, p < .0001, total effect β = .5512, p < .0001; Time-4, df = 95, direct effect, β = .3838, p = .005, total effect β = .3898, p = .006) and financial adequacy influenced symptomatology (Time-1, df = 231, direct effect, β = -.0345, p < .0001, total effect β = -.0514, p < .0001; Time-2, df = 145, direct effect, β = -.0373, p = .0298, total effect β = -.0540, p = .003; Time-3, df = 122, path deleted due to large errors; Time-4, df = 95, direct effect, β = -.0370, p = .079, total effect β = -.0534, p = .014) in a negative manner. These results also support the findings of Laing et al. (1980) and Ross and Huber (1985) that subjective dimensions mediate the objective characteristics of the environment. Also, Pearlin et al. (1981) proposed, that

chronic economic strain may help in explaining the relationship between income and depression; as income decreases economic hardship increases and as a result, depression also worsens. The current results support this explanation of the relationship between income and depression. In the symptomatology model, income was inversely related to financial adequacy and financial adequacy perceptions had an inverse relationship with self-reported psychiatric symptomatology.

In summary, the four time-frame comparisons provide support for a positive (direct and total) relationship between contact frequency (CF) and satisfaction with support (SAT), education (ED) and employment (EMP), education (ED) and income (INC), employment (EMP) and income (INC), satisfaction with support (SAT) and financial adequacy perception (FAD), and income (INC) and financial adequacy perception (FAD). These results also indicate employment (EMP) and financial adequacy perception (FAD) are positively related (indirect and total effect). Contact frequency (CF) and financial adequacy perception (FAD) appear to be consistently related in an indirect positive manner. Satisfaction with support (SAT) and symptomatology (SYMP) are consistently negatively related in direct, indirect, and total manner. The relationships between symptomatology (SYMP) and education (ED), symptomatology (SYMP) and employment (EMP), symptomatology (SYMP) and income (INC), are inconsistent. And finally, the relationship between contact frequency (CF) and symptomatology (SYMP) are consistently negative and include negative direct, indirect, and total effects.

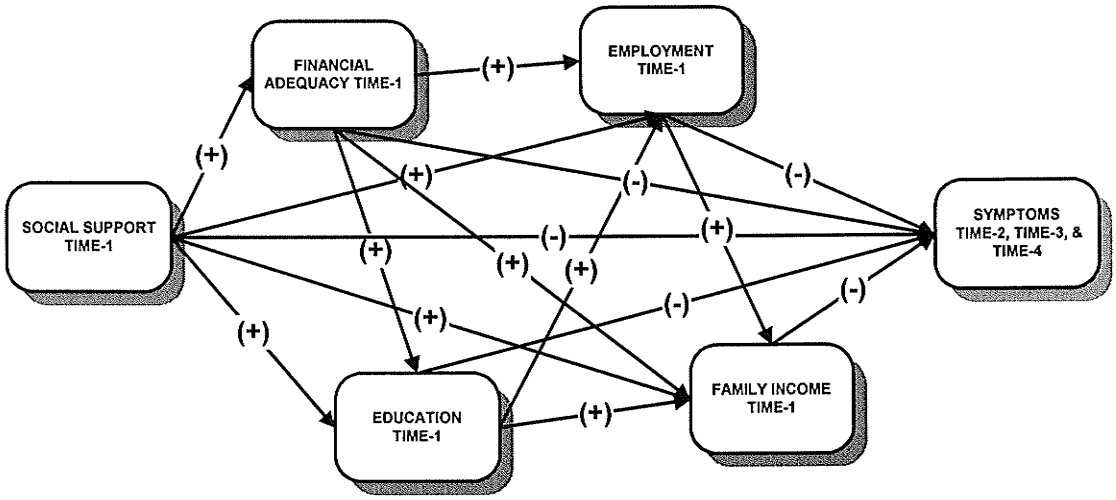
CHAPTER 5:

Phase-III Cross-Lagged Data Analysis

The possible causal relationships between the pertinent social support, social functioning, and psychiatric symptomatology variables are evaluated, more specifically, in Phase-III of the analysis. In this phase of the data analysis the approach will consist of six path analysis models and test indices. These data were collected every six months for a period of two years. However, some researchers believe that a two-year follow-up period may be too short (Bothwell & Scott, 1997). Collecting the data through time and lagging the variables rules out the 'direction of causation' difficulties that plague cross-sectional correlational designs. However, this approach still does not eliminate the 'third variable problem that is always an issue in this type of quasi-experimental design.

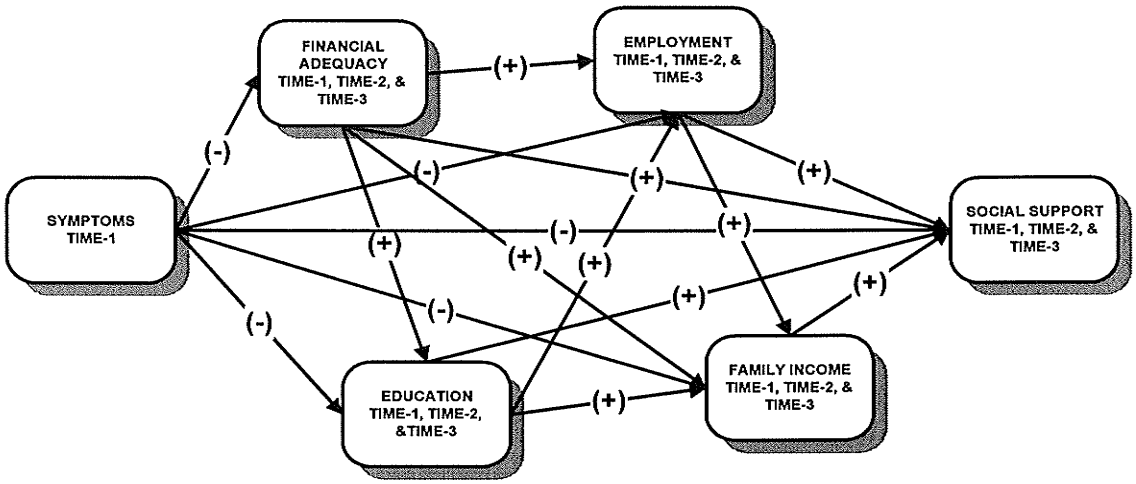
Lagged model hypotheses for the Social Causation Theoretical Model can be found on Figure 10. The first three of these models will evaluate the influence of the social support (SS; social support compound variable combining CF and SAT from the cross-sectional models) and social functioning variables (FAD, ED, EMP, and INC) at Time-1 upon the psychiatric symptomatology (SYMP) variable measured at Time-2, Time-3, and Time-4. This set of path diagrams (SF1SY2, SF1SY3, and SF1SY4) is used to evaluate the Social Causation Theory used to explain relationship between psychiatric symptomatology and SES and social functioning. The lagging of the variables includes using Time-1 SS and Times-2, -3, and -4 SYMP to evaluate the Social Causation Model by eliminating the opposite direction of causation.

Figure 10. The preliminary hypothesised symptomatology lagged path models for the Social Causation theoretical position (SF1SY2, SF1SY3, and SF1SY4). Valence signs indicate the direction of twenty-one hypothesized variable relationships {(+) positive and (-) negative correlation}.



The hypotheses diagram for the Social Selection theoretical position can be found on Figure 11. The second set of three models (SY1SF2, SY1SF3, and SY1SF4) will be used to evaluate the influence of the symptomatology variable at Time-1 on subsequent social support and social functioning at Time-2, Time-3, and Time-4. These models are used to evaluate the validity of the Social Selection theory of the relationship between social functioning and psychiatric symptomatology. The Social Selection Model is evaluated by using Time-1 SYMP and lagging Time-2, -3, and -4 social support and functioning variables; this allows for the elimination of the opposite direction of causation.

Figure 11. The preliminary hypothesised symptomatology lagged path models for the Social Selection theoretical position (SY1SF2, SY1SF3, and SY1SF4). Valence signs indicate the direction of twenty-one hypothesized variable relationships {(+) positive and (-) negative correlation}.



Path models and model diagnostic procedures are used to assess how these models explain the relationships involved. This approach allows all of the pertinent variables to be placed clearly in a causal chain and in time. This was done in an attempt to eliminate some of the causal ambiguities with direction of causation in most of the past literature. Once again the exact p-values for each effect through time were not compared since the sample sizes at each of the points in time were different and power diminished through the study period. However, a similar pattern (concordance) should be evident across all points in time.

Given the literature supporting, independently, both directions of causation (see previous chapters), both directions of causation are expected to be supported. In fact, there may be evidence of a reciprocal negative feedback cycle. In other words, impairments in social functioning will increase psychiatric symptomatology and higher levels of psychiatric symptomatology will influence social functioning in a negative manner. These results were therefore expected to support a reciprocal causation theory. This supported theory allows for synthesis of both the Social Selection and Social Causation theoretical positions.

The primary hypotheses, for both sets of models, predicted inverse relationships between the social support variable (SS) and the outcome symptomatology variables (SYMP). Since secondary hypotheses were numerous, in order to facilitate both clarity and readability, the reader is referred to Figure 10 (above), for the cross-lagged Social Causation hypotheses model and Figure 11 (above) for the Social Selection hypotheses model. The hypothesized relationships are indicated with valence signs. The specified

relationships could represent the direct, indirect, or total effects of the variables involved. The one set of hypothetical relationships are applicable to the three lagged time frames. The research goal here was to demonstrate, for both the Selection and Causation Models, that the effects replicated through all of the time lags and that both models were operating. The two sets of models will also be compared with each other for importance in the theoretical processes.

This quasi-experimental attempt at removing the direction of causation difficulties with correlational research does not contend with the third variable problem with correlational research so caution is warranted and causation questions still cannot be answered unequivocally. However, this approach arguably has improved the quality of causal arguments involved. Once again, the hypotheses presented are supported by both the literature and on theoretical grounds with one set of hypotheses supporting the Social Causation Theory and the other supporting the Social Selection Theory. The relationships predicted between the SES variables and SS are the same for this phase of the research as they were in Phase-II and once again these hypotheses can be found on Figures 10 and 11. Keep in mind, as Krause, Liang, and Keith (1990) warned, that even with theoretical support, several alternative causal orderings could be specified for any model.

The following causal order assumptions were made for the Social Causation Model (see Figure 10) component of this research: As social support (SS) reduces, there will be a negative impact on well being. High levels of SS could help further not only educational goals but opportunity for employment, improving perceptions of financial adequacy (FAD), employment (EMP), and employability, increasing available income

(INC). The combined effect of these social functioning variables, if improved, would reduce the burden of chronic stressors in a mental health client's life and influence psychiatric health, leading to less psychiatric symptomatology.

For the Social Selection Model component, the patient's psychiatric symptomatology was expected to impact social functioning (see Figure 11). When symptoms increase they should exert a negative impact upon perceptions of financial adequacy, education, employment, and family income. Psychiatric symptomatology should interfere with educational attainment, limiting employability in a negative manner and result in lower household incomes. This would, in turn, have a negative impact upon a patient's social support availability (SS).

Both the Social Causation Model and the Social Selection Model are expected to explain the current data relationships. In other words, both models should explain a significant amount of variability among the variables. In fact, with both directions of causation arguments gaining support the Reciprocal Causation Theory will be supported. Given Dohrenwend et al.'s (1998) work, one position is expected to be more compelling than the other. These authors have reported that the Social Causation Model was more important for individuals with depressive disorders.

Psychiatric clients are often socially withdrawn, yet, according to Cohen and Sokolovsky (1978), frequency or length of psychiatric hospitalization seems unlikely to have caused this withdrawal. Yet, the 'Social Breakdown Syndrome' would propose that reduced contact is the result of lost role functioning and identity, resulting in a passive withdrawal of the client diagnosed with a psychiatric illness (Kuypers & Bengtson, 1973).

The social support/health relationship could function in both directions or be a result of another unmeasured variable (Hammer et al. 1982). But, according to Berkman and Syme (1979), it is more likely that social networks influence health outcomes. Kaplan, Salonen, Cohen, Brand, Syme, and Pyska (1988) believe that the available research fails to support the theory that illness causes social support inadequacies. The balance of the literature supporting the hypotheses are the same as for the cross-sectional models (see Chapter 2 and 4). Raw correlations (time-lagged variables) for the Social Causation Model can be found in Table 21. The raw correlations (time lagged variables) for the Social Selection Model can be found in Table 22. The balance of the time lagged correlations can be found on Tables G1, G2, G3, G4 and G5 in Appendix G.

Table 21

Raw Correlations SF TIME-1 SYMP TIME-2 Time-Lagged Model, Social Causation Model N = 146

	SUPPORT TIME-1	FAD TIME-1	ED TIME-1	EMP TIME-1	INC TIME-1	SYMP TIME-2
SUPPORT TIME-1	- -	+ .28 p < .001	+ .05 p = .451	+ .05 p = .451	+ .11 p = .043	- .31 p < .001
FAD TIME-1		- -	- .02 p = .817	+ .11 p = .084	+ .43 p < .001	- .26 p < .001
ED TIME-1			- -	+ .24 p < .001	+ .24 p < .001	- .01 p = .889
EMP TIME-1				- -	+ .44 p < .001	- .09 p = .259
INC TIME-1					- -	- .07 p = .439
SYMP TIME-2						- -

Note. The abbreviations are as follows: p = one-tailed probabilities, SUPPORT= combined social support variable, ED =years of formal schooling, EMP = employment score, INC = Family income score, FAD =Financial Adequacy Score, SYMP = Symptomatology measure, and SF = social support and social functioning. All probabilities are two tailed for the appropriate values related to directional predictions this value needs divided by two.

Table 22

Raw Correlations SYMP TIME-1 SF TIME-2 Time-Lagged Model, Social Selection Model. N = 146

	SYMP TIME-1	FAD TIME-2	ED TIME-2	EMP TIME-2	INC TIME-2	SUPPORT TIME-2
SYMP TIME-1	- -	- .18 p = .032	- .03 p = .703	- .18 p = .027	- .09 p = .284	- .31 p < .001
FAD TIME-2		- -	+ .01 p = .861	+ .15 p = .070	+ .36 p < .001	+ .18 p = .029
ED TIME-2			- -	+ .23 p = .004	+ .20 p = .014	+ .01 p = .965
EMP TIME-2				- -	+ .22 p = .006	+ .08 p = .333
INC TIME-2					- -	+ .06 p = .495
SUPPORT TIME-2						- -

Note. The abbreviations are as follows: p = one-tailed probabilities, SUPPORT= combined social support variable, ED =years of formal schooling, EMP = employment score, INC = Family income score, FAD = Financial Adequacy Score, SYMP = Symptomatology measure and SF = social support and social functioning. All probabilities are two tailed for the appropriate values related to directional predictions this value needs divided by two.

Phase III Path Analysis Methodology

Structural Equations for the time-lagged models can be found on Table 23 and Table 24 below. For the Reduced Equation models the reader is referred to Appendix H Tables H1, H2, H3, H4, H5, and H6.

Table 23

Structural Equations for the Multiple Regressions for All (Time-2 to Time-4) of the Social Selection (SY1SF2, SY1SF3, and SY1SF4) Fully Identified Models (Hypothesized Relationships are Indicated with Valence Signs for Each Path).

Effects	Endogenous Variables						Exogenous Variable
Equation number	DV T-2, -3, & -4	FAD T-2, -3, & 4	ED T-2, -3, & 4	EMP T-2, -3, & 4	INC T-2, -3, & 4	SS T-2, -3, & 4	IV–SYMP T-1
EQ#1	FAD T-2, -3, & 4	------	------	------	------	------	path #1(+)
EQ#2	ED T-2, -3, & 4	path #2(+)	------	------	------	------	path #3(+)
EQ#3	EMP T-2, -3, & 4	path #4(+)	path #5(+)	------	------	------	path #6(+)
EQ#4	INC T-2, -3, & 4	path #7(+)	path #8(+)	path #9(+)	------	------	path #10(+)
EQ#5	SS T-2, -3, & 4	path #11(-)	path #12(-)	path #13(-)	path #14(-)	------	path #15(-)

Note: Abbreviations: independent (IV)/dependent (DV) variable pairs for each path, and structural equation number (EQ); SS = Social Support, FAD = financial adequacy score, ED = years of education, EMP = employment score, INC = household income, SYMP = symptomatology score, T1, 2, 3, and 4 = measurement at Times-1 through 4 respectively, SF = social functioning variables, and SY = symptomatology variables.

Table 24

Structural Equations for the Multiple Regressions for all (Time-2 to Time-4 models) for the Social Causation (SF1SY2, SF1SY3, and SF1SY4) Fully Identified Models (Hypothesized Relationships are Indicated With Valence Signs for Each Path)

Effects		Endogenous Variables					Exogenous Variable
Equation number	DV	FAD T-1	ED T-1	EMP T-1	INC T-1	SYMP T-2, -3, & -4	IV–SS T-1
EQ#1	FAD T-1	------	------	------	------	------	path #1(+)
EQ#2	ED T-1	path #2(+)	------	------	------	------	path #3(+)
EQ#3	EMP T-1	path #4(+)	path #5(+)	------	------	------	path #6(+)
EQ#4	INC T-1	path #7(+)	path #8(+)	path #9(+)	------	------	path #10(+)
EQ#5	SYMP T-2, -3, & -4	path #11(-)	path #12(-)	path #13(-)	path #14(-)	------	path #15(-)

Note: Abbreviations: independent (IV)/dependent (DV) variable pairs for each path, and structural equation number (EQ); SS = Social Support, FAD = financial adequacy score, ED = years of education, EMP = employment score, INC = household income, SYMP = symptomatology score, T-1, -2, -3, and -4 = measurement at Times 1-4 respectively, SF = social functioning variables, and SY = symptomatology variables.

Multiple regression results for the three Social Causation Models, including variance accounted for, can be found in Tables 25, 26, and 27 below. The coefficient of determination for all of the lagged models varied between .0144 and.3126. There is great similarity in pattern for all of the R^2 values for all of the lagged models. The majority of variance was associated with the fourth multiple regression equation. It is interesting to note that the Social Causation models accounted for more variability than the Social Selection models and the Causation models had more statistically significant F-values than the Selection models. These tables include the proportion of variance accounted for by the collection of variables in each equation (R^2), mean squared error, the F-value for the equation, and the F-values associated p-value. Each of the equations in Table 25 demonstrates a significant contribution to variance accounted for except equation number two. It is important to keep in mind that longitudinal cross-lag coefficients analysis are more likely to generate small effect sizes relative to cross-sectional research (Peirce, Frone, Russell, Cooper, & Mudar, 2000).

Table 25

Multiple Regression results for the Structural Equation System Based Upon the Social Causation Theory Reduced Path Model SF1SY2, N = 146

Equation Number	R^2	MS_e	F-ratio	p-value
EQ #1	0.0777	1322.9787	12.13	p = .0007
EQ #2	0.0005	0.35733	0.04	p = .964
EQ #3	0.0721	6432.8223	3.68	p = .0137
EQ #4	0.3943	657.13497	22.95	p < .0001
EQ #5	0.1424	32.27692	4.65	p = .0006

Note: Abbreviations: R^2 = Proportion of Variance Accounted for by the Model, EQ = equation number from the structural equation system, MS_e = mean square error, SF = Social Functioning, and SY = Symptomatology.

Table 26

Multiple Regression Results for the Structural Equation System Based Upon the Social Causation Theory Reduced Path Model SF1SY3, N = 123

Equation Number	R^2	MS_e	F-ratio	p-value
EQ #1	0.074	980.6614	9.67	p = .0023
EQ #2	0.0074	5.0179	0.45	p = .6406
EQ #3	0.0703	5709.3964	3	p = .0335
EQ #4	0.3538	501.02473	16.15	p < .0001
EQ #5	0.1556	27.60474	4.31	p = .0012

Note: Abbreviations: R^2 = Proportion of Variance Accounted for by the Model, EQ = equation number from the structural equation system, MS_e = mean square error, SF = Social Functioning, and SY = Symptomatology.

Table 27

Multiple Regression Results for the Structural Equation System Based Upon the Social Causation Theory Reduced Path Model SF1SY4, N = 96

Equation Number	R^2	MS_e	F-ratio	p-value
EQ #1	0.0776	895.47017	8	$p = .0057$
EQ #2	0.0144	7.45961	0.68	$p = .5069$
EQ #3	0.0929	5054.40541	3.17	$p = .0278$
EQ #4	0.3126	331.52484	10.46	$p < .0001$
EQ #5	0.1223	32.44154	4.32	$p = .0068$

Note: Abbreviations: R^2 = Proportion of Variance Accounted for by the Model, EQ = equation number from the structural equation system, MS_e = mean square error, SF = Social Functioning, and SY = Symptomatology.

Table 28

Multiple Regression Results for the Structural Equation System Based Upon the Social Selection Theory Reduced Path Model SY1SF2, N = 146

Equation Number	R^2	MS_e	F-ratio	p-value
EQ #1	0.032	596.48257	4.76	$p = .0307$
EQ #2	0.0009	0.83775	0.07	$p = .9369$
EQ #3	0.0721	1598.66293	3.68	$p = .0137$
EQ #4	0.171	279.70719	7.27	$p < .0001$
EQ #5	0.1135	236.34308	3.59	$p = .0044$

Note: Abbreviations: R^2 = Proportion of Variance Accounted for by the Model, EQ = equation number from the structural equation system, MS_e = mean square error, SF = Social Functioning, and SY = Symptomatology.

Table 29

Multiple Regression results for the Structural Equation System Based Upon the Social Selection Theory Reduced Path Model SY1SF3, N = 123

Equation Number	R^2	MS_e	F-ratio	p-value
EQ #1	0.0609	845.36778	7.85	p = .0059
EQ #2	0.0186	11.89895	1.14	p = .3236
EQ #3	0.0531	1082.15729	2.23	p = .0888
EQ #4	0.1736	245.87155	6.2	p < .0001
EQ #5	0.128	24.462911	4.33	p = .0026

Note: Abbreviations: R^2 = Proportion of Variance Accounted for by the Model, EQ = equation number from the structural equation system, R^2 = proportion of the variance accounted for, MS_e = mean square error, SF = Social Functioning, and SY = Symptomatology.

Table 30

Multiple Regression Results for the Structural Equation System Based Upon the Social Selection Theory Reduced Path Model SY1SF4, N = 96

Equation Number	R^2	MS_e	F-ratio	p-value
EQ #1	0.0686	867.5568	7	p = .0095
EQ #2	0.0012	0.6739	0.06	p = .9459
EQ #3	0.0849	1221.80835	2.88	p = .0403
EQ #4	0.1698	215.63873	4.7	p = .0017
EQ #5	0.1313	227.94006	3.48	p = .0109

Note: Abbreviations:R^2 = Proportion of Variance Accounted for by the Model, EQ = equation number from the structural equation system, R^2 = proportion of the variance accounted for, MS_e = mean square error, SF = Social Functioning, and SY = Symptomatology.

Cross-Lagged Model Results Phase III

For the time-lagged Tables of the Social Causation theoretical position the reader is referred to Appendix I for the time-lagged Tables I1, I2, and I3. For the time-lagged Social Selection theoretical positions the reader is referred to Tables J1, J2, and J3 in Appendix J. Model fit indices can be found in Table 31 below. The Null Model χ^2 values are all significantly greater than two indicating that the data required models to be fit. After fitting models to the data sets the χ^2 indices were all less than two and all were non-significant. Also, the model fit indices in Table 31.5 are all acceptable with values ranging from .9 to just over 1. The models fit the data at acceptable levels (Bentler & Bonett, 1980; Bentler, 1989). The Akai's Information Criterion (AIC) guideline is "the smaller the better". The Parsimonious Index is also provided and indicates that these are not the most parsimonious models, but this was expected since non-significant paths remain in the models. All of the original multiple regression variance covariance matrices were under two.

Table 31.

Chi Squared Values and Null and Reduced Time-Lagged Models.

Chi Squared Statistics (χ^2)					
Null			Reduced		
Model(1)	$df_{(1)}$	$p < 2$	Model(2)	$df_{(2)}$	$p < 2$
117.62	15	<.0001	1.461	2	0.4809
93.05	15	<.0001	0.017	1	0.9
67.06	15	<.0001	0.0449	2	0.9778
59.94	15	<.0001	1.9361	4	0.4147
56.98	15	<.0706	1.6025	1	0.2055
67.06	15	<.0001	0.0449	2	0.9778

Note: Abbreviations: $df_{(1)}$ = degrees of freedom for the Null Model, $df_{(2)}$ = degrees of freedom for the Reduced Model, SY = Symptoms, SF = Social Functioning, p = p-value, df = degrees of freedom, $p < 2$ = the probability of the Null and Reduced Models Chi Squared value being less than two.

Table 31.5

Fit Indices for Null and Reduced Time-Lagged Models

Model		Goodness of Fit Indices								
Time-Lagged Models	Critical N	GFI	AGFI	CFI	AIC	NNFI	NNID	BBNFI	BOLEN	Parc. GFI
SF1SY2	595	0.997	0.965	1	-2.5	1.04	1.001	0.988	0.907	0.1329
SF1SY3	27447	0.999	0.999	1	-2	1.19	1.011	0.999	0.997	0.0667
SF1SY4	12822	0.999	0.9984	1	-4	1.28	1.03	0.999	0.995	0.1333
SY1SF2	349	0.991	0.9532	1	-4.1	1.01	1.001	0.934	0.954	0.2643
SY1SF3	292	0.908	0.9083	0.987	-0.4	0.985	0.989	0.972	0.989	0.0664
SY1SF4	4432	0.999	0.9939	1	-1.9	1.43	1.02	0.999	0.995	0.0926

Note:, SY = Symptoms, SF = Social Functioning, p = p-value, df = degrees of freedom, p < 2 = the probability of the Null and Reduced Models Chi Squared value being less than two, AIC = Information Criteria GFI = Goodness of Fit, AGFI= Adjusted Goodness of Fit Index, NNFI = Non-normed Fit Index, BBNFI = Bentler and Bonett's Normed Fit Index, BOLEN = Bollen's Normed Index, and Parc.GFI = Parcemonious Goodnes of Fit Index.

According to Tabachnik and Fidell (2001), other modelling tools, like the Lagrange Multiplier Tests, indicate the paths that should be added to the model. The multivariate component of this test eliminates overlapping variances. This was not an issue in the case of these models since they started fully saturated and then only paths with large errors were removed. There are also the multivariate Wald Tests provided with the output that indicate the parameters that could be fixed to zero. In other words, they indicate which parameters are not necessary in the model. Parameters are considered for deletion only if deletion does not cause the associated multivariate Chi Squared with the Wald Test to become significant. Non-significance is desired (see Tables 31.6 and 31.7 below).

Table 31.6

Multivariate Wald Test Results for all of the Social Causation Models

SF1SY2				SF1SY3				SF1SY4			
beta	χ^2	*df*	*p-value*	*beta*	χ^2	*df*	*p-value*	*beta*	χ^2	*df*	*p-value*
3	0	1	0.9622	12	0.148	1	0.7003	10	0	1	0.977
2	0.07	2	0.9634	3	0.369	2	0.8316	3	0.143	2	0.9312
10	0.292	3	0.9615	13	0.932	3	0.8176	2	1.398	3	0.7058
6	1.406	4	0.8432	2	1.623	4	0.8048	12	2.975	4	0.562
12	2.624	5	0.7577	10	2.438	5	0.7958	4	4.844	5	0.4252
4	4.507	6	0.6084	6	4.051	6	0.6698	13	7.885	6	0.2466
				4	6.182	7	0.5187	5	11.32	7	0.125

Note:, SY = Symptoms, SF = Social Functioning, df = degrees of freedom, p-value = the probability of each beta weight, and χ^2 = the chi squared value associated with each particular beta.

Table 31.7

Multivariate Wald Test Results for all of the Social Selection Models

SY1SF2				SY1SF3				SY1SF4			
beta	χ^2	*df*	*p-value*	*beta*	χ^2	*df*	*p-value*	*beta*	χ^2	*df*	*p-value*
2	0	1	0.9851	12	0.6	1	0.8964	12	0.03	1	0.8592
9	0	2	0.9971	10	1.237	2	0.8719	13	0.05	2	0.9738
5	0.04	3	0.9978	6	1.899	3	0.8628	10	0.128	3	0.9883
10	2.461	4	0.6516	8	2.884	4	0.8232	2	0.37	4	0.9848
3	5.488	5	0.3593	2	4.996	5	0.6605	6	0.744	5	0.9805
7	8.52	6	0.2024	4	7.367	6	0.4976	4	1.149	6	0.9793
				5	10.88	7	0.2837	8	2.098	7	0.9542
								3	3.847	8	0.8707

Note:, SY = Symptoms, SF = Social Functioning, df = degrees of freedom, p-value = the probability of each beta weight, and χ^2 = the chi squared value associated with each particular beta.

Social Causation model effects (SF = Social Functioning, SY= Symptomatology; SF1SY2, SF1SY3, and SF1SY4; see Figures 12, 13, and 14) are discussed first followed by the Social Selection model (SY = Symptomatology, SF = Social Functioning; SY1SF2, SY1SF3, and SY1SF4; see Figures 15, 16, and 17) results. The numbers in the models indicate what point in time the variable was assessed. For example, for the Social Causation model 'SF1SY2' the social functioning variables are from Time-1 while the symptomatology variable is from Time-2. The first results discussed are for the final paths in the models, those joining social support and symptomatology for all of the models. The balance of the results is discussed in path numerical order in an attempt to keep things orderly. The reader is cautioned that the path numbers will not match up with the variables being compared. Therefore, one needs to be looking at the path diagrams when reading this section. Not all of the paths discussed bear upon the theoretical comparison of the two competing theoretical positions. Those that bear on the comparisons have their path numbers bolded for ease of comparisons.

Figure 12. Social Causation theoretical position. The final reduced path lagged model for SF1SY2.

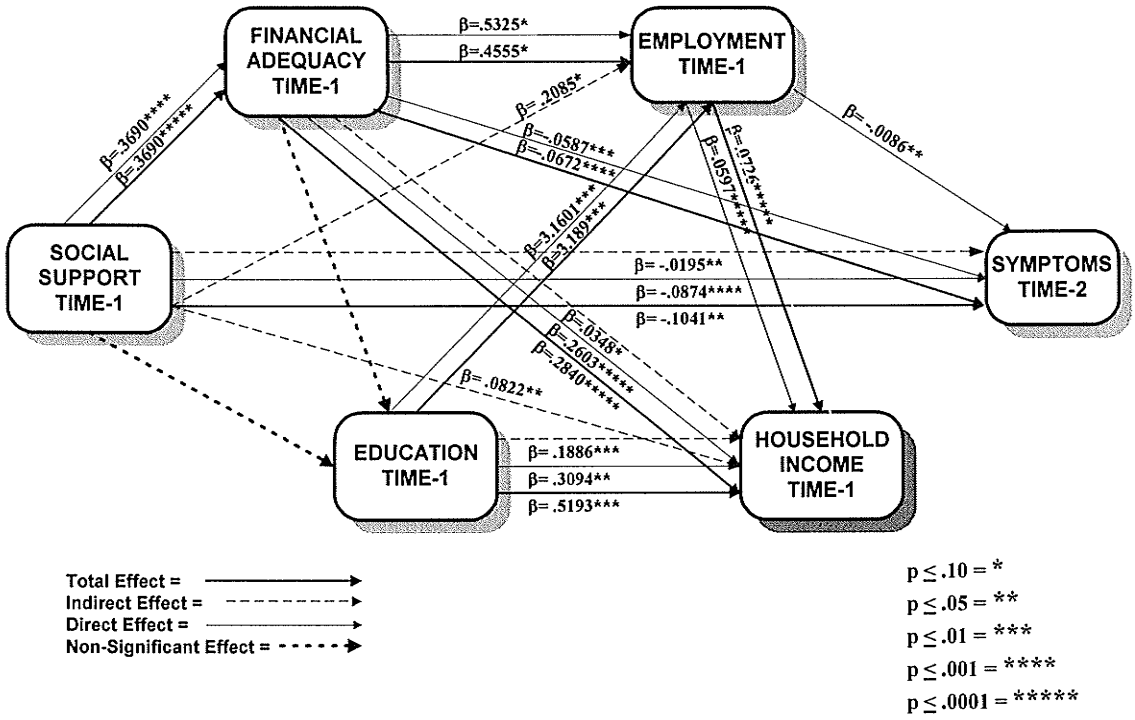


Figure 13. Social Causation theoretical position. The final reduced path lagged model for SF1SY3.

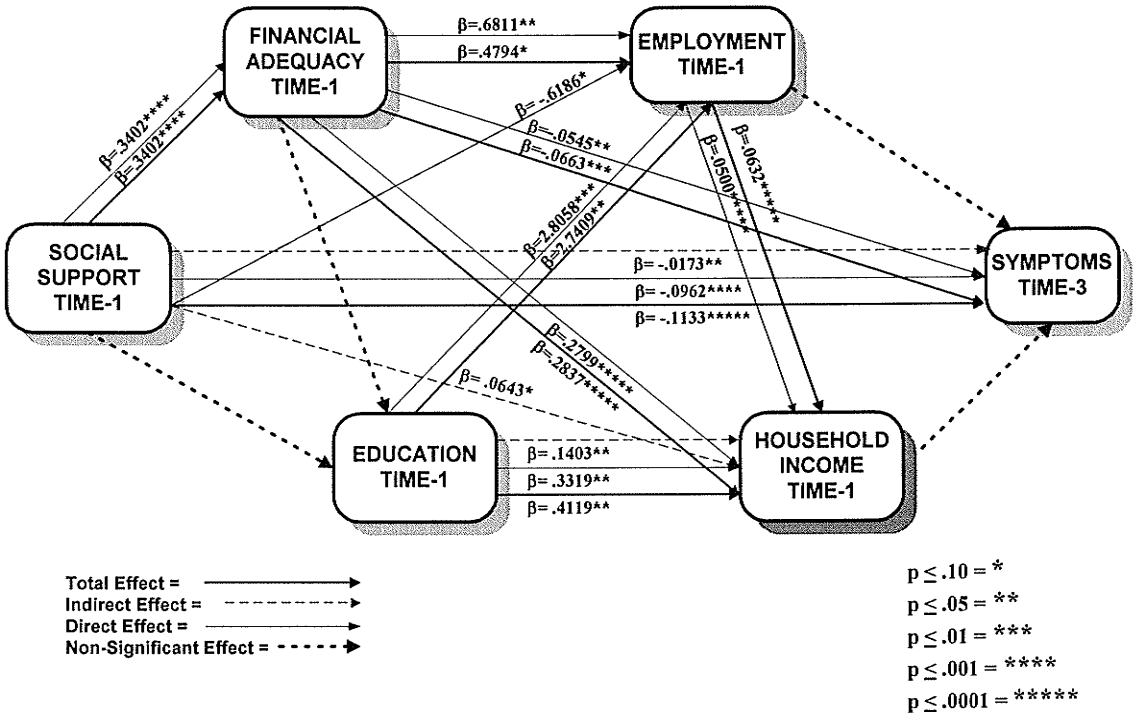


Figure 14. Social Causation theoretical position. The final reduced path lagged model for SF1SY4.

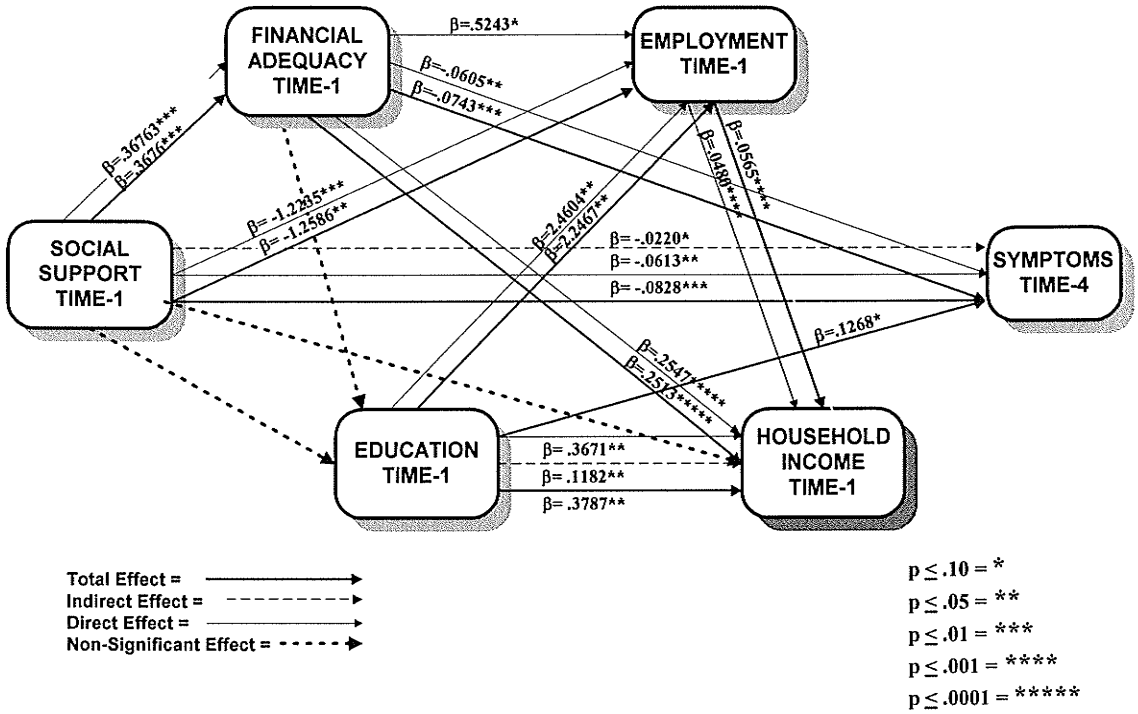


Figure 15. Social Selection theoretical position. The final reduced path lagged model for the SY1SF2 model.

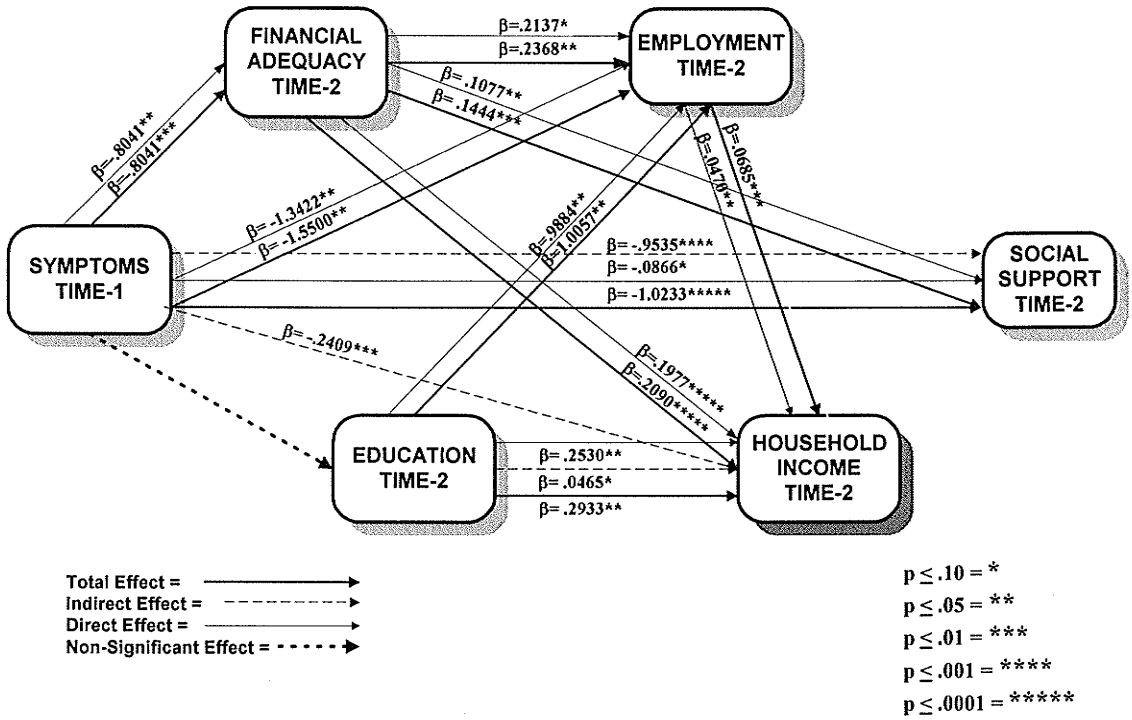


Figure 16. Social Selection theoretical position. The final reduced path lagged model for the SY1SF3 model.

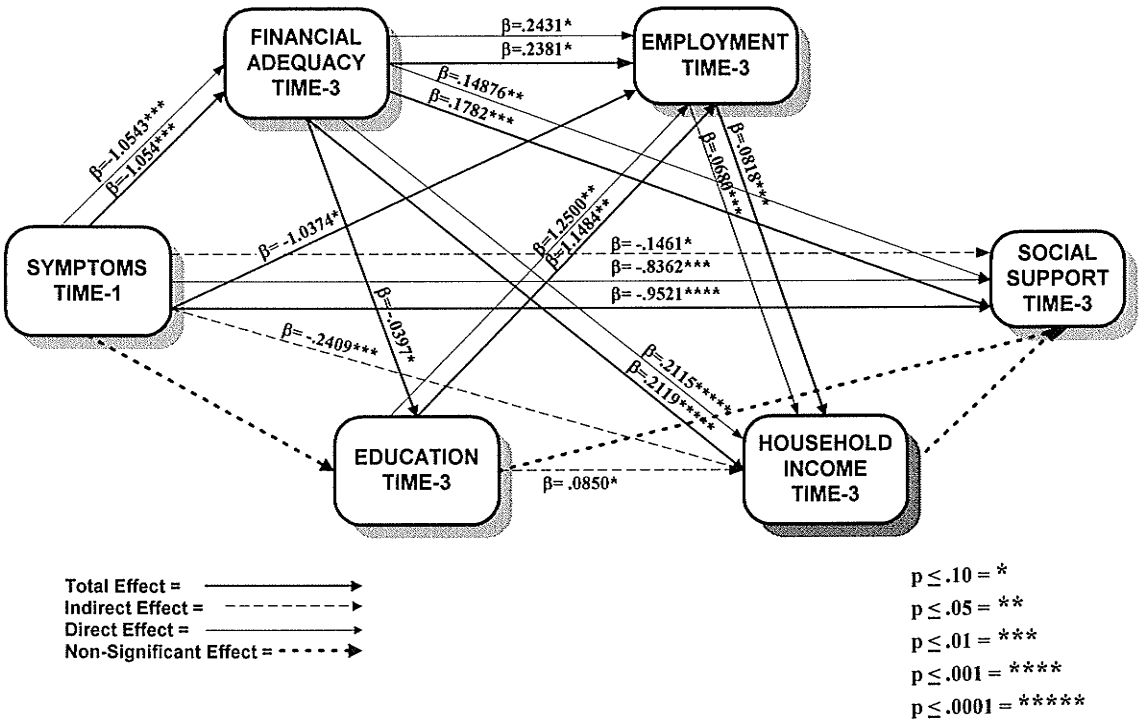
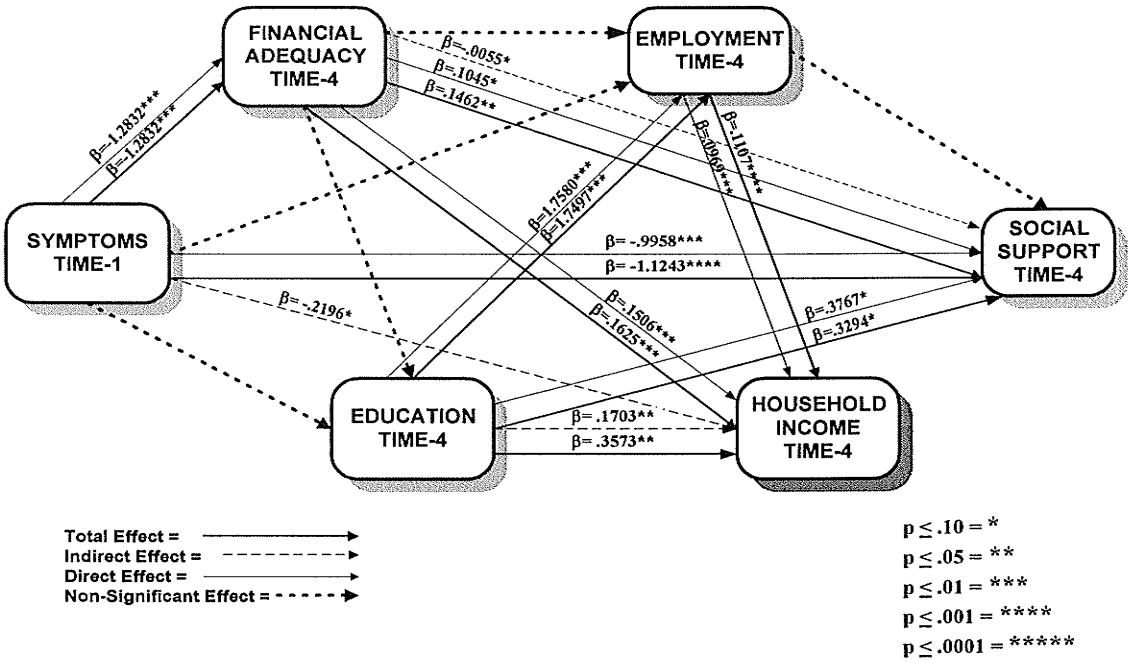


Figure 17. Social Selection theoretical position. The final reduced path lagged model for the SY1SF4 model.



There were a number of invalid paths (error effects greater than the direct effect) that had to be eliminated in the Social Causation model specification. For the SF1SY2 model the path connecting ED Time-1 and SYMP Time2 was invalid due to large errors. The path connecting INC Time-1 and SYMP Time-2 was also invalid and was removed. For the SF1SY3 model the path connecting ED Time-1 and SYMP Time2 was also invalid. For the SF1SY4 model there were two invalid paths. The path connecting EMP at Time-1 and SYMP at Time-4 was invalid. The path connecting INC at Time-1 and SYMP at Time-4 was invalid.

Social Causation Model Results

SS Time-1 is related to SYMP at Time-2 in a negative manner as predicted for the SF1SY2 model (all p-values are one-tailed; **Path # 15**; indirect effect, β = -.0195, p = .0411; direct effect, β = -.0874, p = .0009; total effect, β = -.1041, p < .0001). SS Time-1 is also related to Time-3 symptoms in the hypothesised manner for the SF1SY3 model (Path #15; indirect effect, β = -.0173, p = .0467; direct effect, β = -.0962, p = .0004; total effect, β = -.1133, p < .0001). Time-1 SS is also related to SYMP Time-4 as predicted for the SF1SY4 model (Path # 15; indirect effect, β = -.0220, p = .0521; direct effect, β = -.0613, p = .0468; total effect, β = -.0828, p = .0082). Social Support (SS Time-1) is clearly related to symptomatology at Times-2, -3, and -4 for all of these models (SF1SY2, SF1SY3, and SF1SY4).

SS and FAD also appear to be consistently related to one another as predicted. SS at Time-1 was positively related to FAD at Time-1 for the SF1SY2 model (Path #1; direct

effect, β = + .3690, p = .0003; total effect, β = + .3690, p = .0003). SS at Time-1 was positively related to FAD at Time-1 for the SF1SY3 model (Path #1; direct effect, β = + .3402, p = .0009; total effect, β = + .3402, p = .0009). Time-1 SS is also related to FAD at Time-1 for the SF1SY4 model (Path #1; direct effect, β = + .3676, p = .0023; total effect, β = + .3676, p = .0023). There are no indirect effects in these cases since the two variables follow one another in the causal chain. For this longitudinal effect, social support at one point in time is clearly related to one's perception of financial adequacy in the same point in time. This finding is consistently found for all of the models evaluated (SF1SY2, SF1SY3, and SF1SY4).

The relationship between FAD and ED (Path #2) both Time-1 variables for the SF1SY2, SF1SY3, and the SF1SY4 models are all non-significant for the Social Causation models. It appears that for these participants that there is not a relationship between their perceptions of financial adequacy and their educational level. This path also demonstrated some inconsistencies in the cross-sectional models.

Path number three connects social support (SS, Time-1) and education (ED, Time-1) in the Social Causation theoretical model. In all cases of this model the path was not significant. Therefore it appears that this relationship does not exist in any consistent way for the Social Causation theoretical position. This lack of effect, as discussed previously with the cross-sectional models, can be due to causal ordering difficulties due to education being measured at the time support was assessed. To be able to evaluate this effect measures of education and support would need to be evaluated at either index hospitalization or at first symptom expression.

The relationship between financial adequacy and employment (Path #4) was significant in the Social Causation models. The relationship between FAD and EMP, was positive as expected in all cases. For the SF1SY2 there was a positive direct and total effect (direct effect, β = + .5325, p = .0554; total effect, β = + .4555, p = .0823). For the SF1SY3 model there were also significant direct and total effects (direct effect, β = + .6811, p = .0422; total effect, β = + .4794, p = .1075). For the SF1SY4 model there was only a marginally significant direct effect (direct effect, β = + .5243, p = .0892). Although not perfect, it is reasonable to assume that there is some form of positive relationship between FAD and EMP. And this would make sense in a general population also.

Path # 5 of the Social Causation Model connects education (ED; Time-1) with employment (EMP; Time-1). The results for this path are very consistent. All of the models (SF1SY2, SF1SY3, and SF1SY4) demonstrate consistent positively related direct and total effects as predicted (SF1SY2, direct effect, β = + 3.160, p = .0024; total effect, β = + 3.189, p = .0023; SF1SY3, direct effect, β = + 2.806, p = .0044; total effect, β = + 2.7409, p = .0107; SF1SY4, direct effect, β = + 2.4604, p = .0242; total effect, β = + 2.2467, p = .0375). There are no indirect effects applicable because of the order of these variables in the models. These result indicate that education and employment are positively related in this sample as would be the case in a general population sample.

Social support (SS, Time-1) and employment (EMP, Time-1) are connected with path #6 in the Social Causation models. The results of the path coefficients are not consistent over the three lagged models. For the SF1SY2 model there is a marginally

significantly indirect positive effect, as predicted ($\underline{\beta}$ = + .2085, $\underline{p}$ = .1088). For the SF1SY3 model there was a marginally significant negative direct effect, in the opposite direction of predictions ($\underline{\beta}$ = - .6186, $\underline{p}$ = .1049). And finally, for the SF1SY4 model there appears to be significant negative direct and total effects (direct effect, $\underline{\beta}$ = - 1.2235, $\underline{p}$ = .0083; total effect, $\underline{\beta}$ = - 1.2586, $\underline{p}$ = .0197) that are the opposite of predictions. No clear conclusions about the veridical nature of these relationships can be made at the present time.

Path #7 connects financial adequacy (FAD) at Time-1 with household income (INC) at Time-1 for the Social Causation models. Theoretical considerations and information from the literature would predict that the relationships between these variables would be positive. Results indicate a reasonably consistent positive relationship between these variables, as predicted (SF1SY2, direct effect, $\underline{\beta}$ = + .2603, $\underline{p}$ < .0001; indirect effect, $\underline{\beta}$ = + .0348, $\underline{p}$ = .0749; total effect, $\underline{\beta}$ = + .2840, $\underline{p}$ < .0001; SF1SY3, direct effect, $\underline{\beta}$ = + .2799, $\underline{p}$ < .0001; total effect, $\underline{\beta}$ = + .2837, $\underline{p}$ < .0001; SF1SY4, direct effect, $\underline{\beta}$ = + .2547, $\underline{p}$ < .0001; total effect, $\underline{\beta}$ = + .2513, $\underline{p}$ < .0001). There is little to no evidence that there are indirect relationships, via education, involved for these path coefficients.

The relationship between education (ED) and household income (INC) is evaluated by path #8 in the Social Causation models. For the SF1SY2 model there are significant positive indirect, direct, and total effects as predicted (indirect effect, $\underline{\beta}$ = + .1886, $\underline{p}$ = .0059; direct effect, $\underline{\beta}$ = + .3094, $\underline{p}$ = .0179; total effect, $\underline{\beta}$ = + .5193,

$p = .0017$). For the SF1SY3 model there were significant positive effects, also as predicted (indirect effect, $\beta = +.1403$, $p = .0128$; direct effect, $\beta = +.3319$, $p = .0164$; total effect, $\beta = +.4119$, $p = .0119$). And finally, for the SF1SY4 model there appear to be significant positive effects (direct effect, $\beta = +.3671$, $p = .0204$; indirect effect, $\beta = +.1182$, $p = .0454$; total effect, $\beta = +.3787$, $p = .0314$). Clearly there are consistent significant positive effects between education and household income for the Time-1 (ED and EMP) variables. There is clear indication that education acts directly on income as well as indirectly through the employment variable. These result indicate that education and household income are positively related in this sample as would be the case in a general population sample.

Path # 9 connects Time-1 EMP and Time-1 INC for all of the Social Causation models. The results are clear in the case of these two variables. There are significant positive direct and total effects for all three time frames. For the SF1SY2 model there were significant positive direct and total effects as predicted (direct effect, $\beta = +.0597$, $p < .0001$; total effect, $\beta = +.07258$, $p < .0001$). For the SF1SY3 model there were significant positive direct and total effects, also as predicted (direct effect, $\beta = +.05001$, $p < .0001$; total effect, $\beta = +.0632$, $p < .0001$). And finally, for the SF1SY4 model there appeared to be significant positive direct and total effects (direct effect, $\beta = +.0480$, $p = .0005$; total effect, $\beta = +.0565$, $p = .0013$). In this case there are no indirect effects possible due to the ordering of the variables in the path model. These results indicate that employment and household income are positively related in this sample as would be the

case in a general population sample.

The relationship between social support and household income (path # 10) was inconsistent for the Social Causation models. For the SF1SY2 model there was a positive indirect effect as predicted (indirect effect, $\underline{\beta}$ = + .0822, $\underline{p}$ = .0379). For the SF1SY3 model there was a marginally significant positive indirect effect, also as predicted (indirect effect, $\underline{\beta}$ = + .0643, $\underline{p}$ = .0855). And finally, for the SF1SY4 model there appeared to be no significant relationships between SS and INC. The existence of only an indirect effect demonstrates that SS may only impact INC via its relationship with ED, EMP, and FAD.

Path # 11 connects financial adequacy perceptions (FAD) with psychiatric symptomatology (SYMP) in the Social Causation models. Results clearly indicate significant negative relationships exist between FAD at one point in time and SYMPs at a later point in time. For the SF1SY2 model there are significant negative indirect and total effects as predicted (indirect effect, $\underline{\beta}$ = - .0587, $\underline{p}$ = .0028; total effect, $\underline{\beta}$ = - .0672, $\underline{p}$ = .0059). For the SF1SY3 model there were significant negative direct and total effects, also as predicted (direct effect, $\underline{\beta}$ = - .05447, $\underline{p}$ = .0163; total effect, $\underline{\beta}$ = - .0663, $\underline{p}$ = .0018). And finally, for the SF1SY4 model there appear to be significant negative direct and total effects (direct effect, $\underline{\beta}$ = - .0605, $\underline{p}$ = .0229; total effect, $\underline{\beta}$ = - .0743, $\underline{p}$ = .0002).

The relationship between education (ED) and symptomatology (SYMP, Path # 12) was inconsistent for the Social Causation models. For the SF1SY2 model there were no significant effects. The path had to be eliminated due to large error measures. For the

SF1SY3 model there was no effect reported either since this path also had high error measures and needed to be eliminated from the model. And, finally, for the SF1SY4 model there appear to be marginally significant positive total effects (total effect, $\underline{\beta} = +.1268$, $\underline{p} = .0764$), contrary to expectations. This path may have turned out to be invalid and/or not significant due to limited range in the variables. This outcome also confirms that using Education as the first endogenous variable rather than Financial Adequacy would have created difficulties for the SASPA path analysis program. To have this program work adequately the first path needs to be valid.

Path # 13, for the Social Causation models connects employment (EMP) and symptomatology (SYMP). Results are not consistent across the three lagged models. For the first Social Causation model there is evidence of a marginally significant inverse relationship as expected (SF1SY2, direct effect, $\underline{\beta} = -.00875$, $\underline{p} = .0441$). For the Social Causation model SF1SY3 there are non-significant effects between EMP and SYMP. And for the SF1SY4 model this path had to be eliminated because of error measures that were too large.

The final path (Path # 14) to be discussed connects household income (INC, Time-1) and symptomatology (SYMP, Time-2, -3, and -4). Results are fairly consistent for this path also. For the SF1SY2 model the path had to be eliminated from the model due to large error measures. For the SF1SY3 model the path between INC (Time-1) and SYMP (Time-3) was not statistically significant. Path #14 also had to be removed from this model (SF1SY4) due to large error measures.

In summary, it is clear from the results that social support at Time-1 is inversely

related to symptomatology at later points in time. Social support at one point in time is clearly related to one's perception of financial adequacy in the same point in time. Financial adequacy and social support were found not to be related to education. However, financial adequacy was positively related to employment. Education and employment were positively related. No conclusions can be drawn about the relationship between social support and employment. There is evidence for a reasonably consistent positive relationship between financial adequacy and household income and between education and income. Employment and household income were positively related as expected. The relationship between social support and household income was inconsistent. Results clearly indicate significant negative relationships exist between financial adequacy at one point in time and symptoms at a later point in time. The relationships of education and employment with symptomatology were inconsistent. And finally, the relationship between income and symptomatology was inconsistent due primarily to large measurement errors.

Social Selection Model Results

For the Social Selection models invalid paths also existed at all three lag times. All p-values provided are one-tailed. For the SY1SF2 model the paths connecting FAD at Time-2 and ED at Time-2 was invalid. There was an invalid path between ED at Time-2 and SS at Time-2. The path connecting EMP at Time-2 and social support (SS) at Time-2 was invalid. And finally, the path connecting INC at Time-2 and SS at Time-2 was eliminated due to large error measures. For the SY1SF3 model there were two invalid

paths. The path between EMP at Time-3 and SS at Time-3 was invalid as was the path connecting INC at Time-3 and SS Time-3. And finally, for the SY1SF4 model the path connecting INC at Time-4 and SS at Time-4 was invalid due to large error measures. For model fit indices for the Social Selection Models can be found on Table 31 above.

SYMP Time-1 is related to SS at Time-2 in a negative manner as predicted for the SY1SF2 (**Path # 15**; direct effect, β = -.9535, p = .0002; indirect effect, β = -.0866, p = .0825; total effect, β = -1.0233, p < .0001). SYMP Time-1 is also related to Time-3 social support in the hypothesised manner for the SY1SF3 model (Path # 15; indirect effect, β = -.1461, p = .0529; direct effect, β = -.8632, p = .0015; total effect, β = -.9521, p = .0003). Time-1 SYMP is also related to SS Time-4 as predicted for the SY1SF4 model (Path # 15; direct effect, β = -.9958, p = .0032; total effect, β = -1.1243, p = .0008). Symptoms (SYMP, Time-1) are clearly related to social support at Times-2, -3, and -4 for all of the lagged models (SY1SF2, SY1SF3, and SY1SF4).

SYMP and FAD also appear to be consistently related to one another as predicted. SYMP at Time-1 was negatively related to FAD at Time-2 for the SY1SF2 model (Path #1 direct effect β = - .8041, p = .0145; total effect, β = - .8041, p = .0015). SYMP at Time-1 was positively related to FAD at Time-3 for the SY1SF3 model (Path #1; direct effect, β = - 1.0543, p = .0026; total effect, β = - 1.0543, p = .0023). Time-1 SYMP is also related to FAD at Time-4 for the SY1SF4 model (Path #1; direct effect, β = -1.2831, p = .0041; total effect, β = -1.2832, p = .0040). There are no indirect effects in these cases since the two variables follow one another in the causal chain. For this cross-sectional effect, symptoms at one point in time are clearly related to ones' perception of financial

adequacy at a later point in time. This finding is consistently for all of the models evaluated (SY1SF2, SY1SF3, and SY1SF4).

The relationships between FAD and ED (Path #2) were inconsistent for the Social Selection models (SY1SF2, SY1SF3, and SY1SF4). For the SY1SF2 model the path was invalid due to large error measures so it had to be deleted. For the SY1SF3 model a marginal significant negative relationship between FAD and ED (total effect, $\underline{\beta} = -.0397$, $\underline{p} = .0735$) existed. However, this relationship was opposite to that predicted. For the SY1SF4 model the path connecting FAD and ED was not significant. Therefore, for this sample of individuals it appears that education and perceptions of financial adequacy are not related in any consistent manner. This path also demonstrated some inconsistencies in the cross-sectional models.

Path number three for the Social Selection model connects (SYMP, Time-1) and education (ED, Time-2, -3, and -4). In all cases of this model the path was not significant. Therefore it appears that this relationship does not exist in any consistent way for the Social Selection theoretical position. A likely explanation for these findings may be due to lack of variability through time for the education variable. As expected education at the four points in time are so correlated (Time-1with Time-2, $\underline{r} = .935$, $\underline{p} < .0001$, $\underline{N} = 146$; Time-1 with Time-3, $\underline{r} = .902$, $\underline{p} < .0001$; $\underline{N} = 123$; Time-1 with Time-4, $\underline{r} = .937$, $\underline{p} < .0001$; $\underline{N} = 97$). This finding is not surprising since the education variable is not likely to change dramatically during the two-year time-frame of the research. The actual means and standard deviations were almost identical and were not statistically different from one another at any of the time frames (Time-1, $\underline{M} = 13.95$, $\underline{SD} = 3.4$; Time-2, $\underline{M} = 14.54$,

SD = 3.2; Time-3, M = 14.64, SD = 3.2; Time-4, M = 14.63, SD = 3.4).

The relationship between financial adequacy and employment (Path #4) was significant in the Social Selection models. The relationship between FAD (Times-2, 3, and -4) and EMP (Times-2, -3, and -4), in all cases was positive as expected and in most was significant. For the SY1SF2 there was a positive direct and total effect (direct effect, β = +.2137, p = .0846; total effect, β = +2368, p = .0455). For the SY1SF3 model there were also significant direct and total effects (direct effect, β = +.2431, p = .1062; total effect, β = +.2381, p = .1038). For the SF1SY4 model this path was no longer statistically significant. Although not perfect, it is reasonable to assume that there is some form of positive relationship between FAD and EMP. And this would make sense in a general population also. However, once again, one would expect that this relationship should be much larger in magnitude in a general population and a normal population will also have a much higher proportion of individuals who are employed. One also needs to be mindful of the difficulties with the distribution/nature of the EMP variable when drawing conclusions.

Path # 5 of the Social Selection Models connects education (ED; Time-2, -3, and -4) with employment (EMP; Time-2, -3, and -4). The results for this path are very consistent. All of the models (SY1SF2, SY1SF3, and SY1SF4) demonstrate consistent positively related direct and total effects as predicted (SY1SF2, direct effect, β = + .9884, p = .0211; total effect, β = + 1.0057, p = .0207; SY1SF3, direct effect, β = + 1.2500, p = .0224; total effect, β = + 1.1484, p = .0322; SY1SF4, direct effect, β = + 1.7580, p = .0020; total effect, β = + 1.7497, p = .0019). There are no indirect effects applicable

because of the order of these variables in the models. These results indicate that education and employment are positively related, through all three lagged points in time, in this sample, as would be the case in a general population sample.

Symptoms (SYMP, Time-1) and employment (EMP, Time-2, -3, and -4) are connected with **path #6** in the Social Selection models. The results of the path coefficients are somewhat inconsistent over the three lagged models. For the SY1SF2 model there are significant direct and total negative effects as predicted (direct effect, $\underline{\beta}$ = - 1.3422, $\underline{p}$ = .0272; total effect, $\underline{\beta}$ = - 1.5500, $\underline{p}$ = .0129). For the SY1SF3 model there was a marginally significant negative total effect as expected ($\underline{\beta}$ = - 1.0374, $\underline{p}$ = .1003). And finally, for the SY1SF4 model there appear to be no significant effects for this path. There was no evidence of an indirect effect for any of these paths. The reason for inconsistent results may be due to lower power and the distance of symptoms which may have changed.

Path #7 connects financial adequacy (FAD) at Times-2, -3, and -4 with household income (INC) at Times-2, -3, and -4 for the Social Selection models. Theoretical considerations and literature information would predict that these relationships would be positively related to one another. Results indicate a reasonably consistent positive relationship, between these variables, as predicted (SY1SF2, direct effect, $\underline{\beta}$ = + .1977, $\underline{p}$ < .0001; total effect, $\underline{\beta}$ = + .2090, $\underline{p}$ < .0001; SY1SF3, direct effect, $\underline{\beta}$ = + .2155, $\underline{p}$ < .0001; total effect, $\underline{\beta}$ = + .21187, $\underline{p}$ < .0001; SY1SF4, direct effect, $\underline{\beta}$ = + .1506, $\underline{p}$ = .0080; total effect, $\underline{\beta}$ = + .1625, $\underline{p}$ = .0049). And, once again, like the Social Causation models, there is no indication that an indirect effect, through education, is functioning in

the Social Selection models.

Education (ED, Time-2, -3, and -4) and household income (INC, Time-2, -3, and -4) are evaluated by path #8 in the Social Selection models. For the SY1SF2 model there are significant positive indirect, direct, and total effects as predicted (direct effect, $\underline{\beta}$ = + .2530, $\underline{p}$ = .0424; indirect effect, $\underline{\beta}$ = + .0465, $\underline{p}$ = .0838; total effect, $\underline{\beta}$ = + .2933, $\underline{p}$ = .0294). For the SY1SF3 model there was only a marginally significant positive indirect effect, as expected (indirect effect, $\underline{\beta}$ = + .0850, $\underline{p}$ = .0561). And finally, for the SY1SF4 model there appear to be significant positive indirect and total effects (indirect effect, $\underline{\beta}$ = + .1703, $\underline{p}$ = .0326; total effect, $\underline{\beta}$ = + .3573, $\underline{p}$ = .0455).

Path # 9 connects EMP (Time–2, -3, and -4) and INC (Time–2, -3, and -4) for all of the Social Selection models. The results are also clear for the Social Selection models as in the case of these two variables in the Social Causation model. There are significant positive direct and total effects for all three time frames. For the SY1SF2 model there are significant positive direct and total effects as predicted (direct effect, $\underline{\beta}$ = + .0470, $\underline{p}$ = .0300; total effect, $\underline{\beta}$ = + .0685, $\underline{p}$ = .0036). For the SY1SF3 model there were significant positive direct and total effects, also as predicted (direct effect, $\underline{\beta}$ = + .0680, $\underline{p}$ = .0047; total effect, $\underline{\beta}$ = + .0808, $\underline{p}$ = .0011). And finally, for the SY1SF4 model there appear to be significant positive direct and total effects (direct effect, $\underline{\beta}$ = + .0969, $\underline{p}$ = .0022; total effect, $\underline{\beta}$ = + .1107, $\underline{p}$ = .0004). There are no indirect effects possible due to the ordering of the variables in the path model. The direct effect and indirect effects are consistent with all three time-lagged models. Therefore the Time-1 relationships did

replicate completely through Time-2, -3, and -4. These results indicate that employment and household income are positively related in this sample as would be the case in a general population sample.

The relationship between symptomatology and household income **(path # 10)** was consistent for the Social Selection models. For the SY1SF2 model there was a negative indirect effect as predicted (indirect effect, $\underline{\beta}$ = - .2409, $\underline{p}$ = .0098). For the SY1SF3 model there was a significant negative indirect effect, also as predicted (indirect effect, $\underline{\beta}$ = - .2948, $\underline{p}$ = .0081). And, finally, for the SY1SF4 model there also appears to be a marginally significant negative indirect relationship between SYMP and INC as predicted (indirect effect, $\underline{\beta}$ = - .2196, $\underline{p}$ = .0752).

Path # 11 connects financial adequacy perceptions (FAD) with social support (SS) in the Social Selection models. Results clearly indicate significant positive relationship exist between FAD at one point in time and SS at the same point in time. For the SY1SF2 model there are significant positive direct and total effects as predicted (direct effect, $\underline{\beta}$ = + .1077, $\underline{p}$ = .0359; total effect, $\underline{\beta}$ = + .1444, $\underline{p}$ = .0091). For the SY1SF3 model there were significant positive direct and total effects, also as predicted (direct effect, $\underline{\beta}$ = + .1488, $\underline{p}$ = .0018; total effect, $\underline{\beta}$ = + .1782, $\underline{p}$ = .0033). And, finally, for the SY1SF4 model there appear to be significant positive direct, indirect, and total effects (direct effect, $\underline{\beta}$ = + .1048, $\underline{p}$ = .0852; indirect effect, $\underline{\beta}$ = - .0056, $\underline{p}$ = .0752; total effect, $\underline{\beta}$ = + .1462, $\underline{p}$ = .0250). The only exception to these consistent findings was a marginally significant negative indirect effect at Time-4. Financial adequacy appears to be consistently related to social support at least during the same points in time as in the

cross-sectional models.

The relationship between education (ED) and social support (SS, Path # 12) was inconsistent for the Social Selection models. For the SY1SF2 model there are no significant effects. The path had to be eliminated due to large error measures. For the SY1SF3 model there was no effect reported either since this path was non-significant. And, finally, for the SY1SF4 model there appear to be marginally significant positive total effect (direct effect, $\underline{\beta} = + .3767$, $\underline{p} = .0661$; total effect, $\underline{\beta} = + .3294$, $\underline{p} = .0906$, in agreement with expectations.

Path # 13, for the Social Selection models connects the paths between employment (EMP, Time-2, -3, and -4) and social support (SS, Time-2, -3, and -4). Results are a bit more consistent across the three lagged models. For the first Social Selection model SY1SF2 there is no path connecting EMP and SS. This path was deemed to be invalid because the path had to be eliminated due to excessive error. For the Social Selection model SY1SF3 there is also no path connecting EMP and SS. Once again, this path proved to be invalid due to excessive error. For the final Social Selection model SY1SF4 there is a path in this model; however, it is not significant. And for the SY1SF4 model this path had to be eliminated because of error measures that were too large.

The final path (Path # 14) to be discussed connects household income (INC, Time-2, -3, and -4) and social support (SS, Time-2, -3, and -4). Results are fairly consistent for this path also. For the SF1SY2 and SF1SY4 models the paths had to be removed because they were invalid. Also, for the final Social Causation model (SY1SF3), this path was not statistically significant.

In summary, symptomatology and social support were consistently negatively related. Symptomatology at one point in time was inversely related to perception of financial adequacy at a later point in time. Yet, education was not related to financial adequacy in any consistent manner. There is also no evidence of a relationship between education and symptomatology. There is evidence of a positive relationship between financial adequacy and education with employment. Symptomatology was found not to have a consistent relationship with employment. There is reasonably consistent evidence of a positive relationship between financial adequacy and income, between education and income, and between employment and income. There was also evidence of a consistent negative indirect effect between income and symptomatology. Results also clearly indicate a significant positive relationship exists between financial adequacy and social support. Results for the relationship between education and social support are inconsistent. Paths connecting social support and employment were eliminated due too large errors. Social support and income were inconsistently related due either to invalid paths or non-significant results. This also can mean that there is no evidence of a relationship between the variables.

Phase III Discussion

There are many different causal variable patterns possible for this type of research especially if one considers the variables in time, and many of these relationships are non-recursive. Only results pertinent to the Social Causation and Selection theoretical positions are discussed here. This section begins with a discussion of the final path in the

model (Path # 15), follows with results from the SES variables, and then continues with a discussion of other paths that are pertinent to the Social Causation versus the Social Selection arguments. One must keep in mind that paths that turn out to be invalid eliminate the possibility of evaluating the relationships involved. But, it should be clear that there is significant error variation in these situations.

Given the time-lagged results of the current data it becomes clear that social support impacts symptomatology at a later point in time and that symptoms at an earlier point in time impact support at a later point in time. These findings support results cited by Hobfall et al. (2003); Holahan, Moos, Holahan, and Cronkite, (1999, 2000); and Joiner and Coyne, (1999) that social support quality has been found to be an important factor in predicting relapse in depressive episodes and future levels of depressive mood and symptoms. This clearly supports the work by Feldman, Downey, and Schaffer-Neitz (1999) who found that time-lagged impact of social support predicted lower negative mood on the next day. This is clearly supported with the current data for Path #15 in both the Social Causation and Social Selection models, demonstrating that support and symptomatology are related through time in causal chains. According to Corrigan, Giffort, Rashid, Leary, and Okeke (1999) and Corrigan and Phelan (2004), social network supports may act as facilitators to the recovery process from mental illness. For the Social Causation models, social support at one point in time predicted symptomatology at the three later points in time. As social support increases symptomatology reduces supporting the findings of Corrigan, Giffort, Rashid, Leary, and Okeke (1999) and Corrigan and Phelan (2004). These results also support the findings of Berkman and Syme (1979);

Brown, Bhrolchain, and Harris (1975); Cohen and Wills (1985), Fratiglioni, Wang, Ericsson, Mayten, and Wimblad (2000); Haughton Mc Neill, Kreuter, and Subramanian (2006); Mittal, Fortney, Pyne, Edlund, and Wetherell (2006); Reis (1988), Sarason, Sarason, and Gurung (2001); Seeman and Syme (1987), and Turner (1981). According to Baker, Jodrey, and Intagliata (1992); Earls and Nelson (1988), Nelson, Hall, and Walsh-Bowers (1999); social support has been found to be important for the well-being of people with psychiatric disabilities. These results confirm the importance of social support in predicting symptom expression. These results also support the findings reported by Fratiglioni, Wang, Ericsson, Mayten, and Wimblad, (2000); and Sarason, Sarason, and Gurung, (2001), that social networks have positive effects well-being.

For the Social Selection models it is clear that symptomatology at an earlier point in time impacts social support in an inverse manner at a later point in time. This supports the 'Social Breakdown Syndrome' discussed by Kuypers and Bengtson (1973). This theory proposes that increased psychiatric symptoms can result in a decreased social support and that reduced social contact can result in lost role functioning and identity resulting in a passive withdrawal of the client diagnosed with a psychiatric illness. These results also support the belief that the support/health relationship function in both directions, but does not eliminate the possibility that a third variable may be involved in the equation (Hammer et al. 1982).

According to Berkman and Syme (1979), it is more likely that social networks influence health outcomes. This position was partially supported with the current research. In fact, both directions of causation were reasonably equally supported with

these findings. According to Pernice (2006), the presence of reciprocally supportive relationships with social network supports was the most important predictor of mental health recovery. These findings also support the findings of Haughton Mc Neill, Kreuter, and Subramanian (2006), that good social support and networks were related to better EWB. This research does not support the findings of Kaplan, Salonen, Cohen, Brand, Syme, and Pyska (1988) who have stated that available research fails to support the theory that illness causes social support inadequacies. Although this research is correlational in nature, there appears to be consistent and compelling results indicating that increased psychiatric symptomatology was related to lower social support resources. Some authors have proposed that lack of social support is a direct cause of depression (Aneshensel & Stone, 1982; Pearson, Wilmot, & Padi, 1986), impacts the onset and severity of depression (Coyne & Downey, 1991), and has been shown to predict relapse (Casey, Meagher, & Butler, 1996; Mann, Jenkins, & Belsey, 1981). The current results support this reciprocal relationship between social support and psychiatric pathology.

It was clear from almost all of the models that social support had an indirect effect on psychiatric symptomatology and visa versa. These results confirm the findings of Bolton and Oatley (1987), Cohen and Willis (1985), Coyne, Aldwin, and Lazarus (1981); Haughton Mc Neill, Kreuter, and Subramanian (2006), Schaefer, Coyne, and Lazarus (1981); that stress (assessed by divergent measures) and social support have a reciprocal relationship in depressed individuals. Social support has been found to protect against stressors (Cohen & Wills, 1985; Dean, Kolody, & Wood, 1990). According to Sherbourne (1988), there has been significant debate over whether social support and

mental health are directly or indirectly related. These results support both direct and indirect effects. Indirect effects existed through financial adequacy, education, employment and household income. In fact, these results support the belief of Ullah, Banks, and Warr (1985), that neither the direct nor the indirect approach, by itself, may be comprehensive enough to explain the entire relationship between social support, life events, and psychological health. According to Gore (1981), and Mitchell, Billings, and Moos (1982), social support may enhance a patient's functioning by preventing or reducing the stress they experience.

The inverse relationship between SES and psychological distress is well documented (Kessler, 1982; Wheaton, 1978). According to Pearlin (1982), longitudinal analysis supports a causal interpretation of the relationship between SES and health. For the purposes of this research, SES was represented in this research by Employment, Education, Household Income, and Perception of Financial Adequacy. The relationships among social support, psychiatric symptomatology, and employment are difficult to untangle and can be confounded by their non-recursive nature resulting in less powerful tests of theoretical models (Cohen & Willis, 1985). For example, stressors, like unemployment and potential supports, are dependent; "individuals with poor social support may have higher psychological symptom levels, not because they had limited support for coping with stress, but because unemployment produced both elevated symptoms and lowered support" (Atkinson et al. 1986, p. 327). According to Wellman (1981), individuals with better social support are more likely to have higher levels of employment due to more employment opportunities and more stable financial resources

(Hobfoll & Jackson, 1991). Prolonged periods of unemployment are severe stressors (Pearlin, 1982) that threaten physical security, social identity (Weber, 1946), and self-esteem (Dressler, 1986). Many participants with psychiatric disabilities face significant employment barriers. Of the Time-1 sample 114 of 232 (49.1%) participants were unemployed. According to Hamilton et al. (1993), not only can unemployment lead to depression, but depression can lead to unemployment. The reciprocal relationship between unemployment and depression may be due to selection biases in cross-sectional samples. It is just as possible that distressed individuals are more likely to become unemployed as unemployed individuals becoming depressed (Hall & Johnson, 1988; Kessler, House, & Turner, 1987).

One's employment status has been found to be influenced by ones educational level (Costello, 1991). According to Friedman (1966; cited in Friis & Nanjundappa, 1986), lack of education and skills are severe employment impediments in competitive employment markets. According to Ross and Huber (1985), education and income are two of the most important socioeconomic status indicators for psychological well-being. These authors suggest that well-educated persons suffer less from the economic hardship due to low income than individuals who are poorly educated. According to Hanson and Ostergren (1987), having less education and being lower in SES status is often related to disease and morbidity. For the socioeconomic variables used in this research the results are clear. Education, employment, and household income are related to each other in the predicted manner for all of the Social Causation and Selection models. However, although they are related to each other, in general they are not related to or predictive of

either symptomatology or social support. At least for the education variable, this may be due to education being a long-standing level so accommodation has already occurred. These results could be due to the fact that there was little variability in the education variable and this variable was consistent during the study period (Time-1, $\underline{M}$ = 13.95, $\underline{SD}$ = 3.36; Time-2, $\underline{M}$ = 14.54, $\underline{SD}$ = 3.17; Time-3, $\underline{M}$ = 14.64, $\underline{SD}$ = 3.22; Time-4, $\underline{M}$ = 14.63, $\underline{SD}$ = 3.42). Correlations for the four points in time all exceeded .9 and all $\underline{p}$-values were < .0001). These results do not support the work of Kohen, Dohrenwend, and Mirotzinik (1998). These authors found that there was a significant inverse relationship between prevalence and education, personal income, and employment status. Therefore, there is no support for either the Social Selection or Causation theoretical models when considering only the SES variables.

There is evidence for a positive relationship between ED and INC but these effects are less consistent for the Social Selection models than the Social Causation models. Therefore the Time-1 relationships did not replicate completely through the time-lagged results for this sample. These results fail to support the work of Sherkat and Read (1992) that persons with higher levels of education and larger household incomes have lower levels of depression. However, these authors also indicated that, after considering social support, the impact of education dissipated. The current findings support this alternative finding.

When considering the Social Causation position and the relationship between education and psychiatric symptomatology results were inconsistent. Either paths for the effects were invalid or effects failed to reach significance. In fact one of the effects that

did exist demonstrated a marginally significant opposite valence effect. This path may have turned out to be invalid and/or not significant due to the limited range in the variables. This relationship between ED and SYMP may have been more problematic for the education variable since these disorders tend to erupt during late adolescence when many clients are trying to take further education after high school. The Social Selection model connects education and symptomatology. In all cases of this model the path was not significant. Therefore, it appears that this relationship does not exist in any consistent way for the Social Selection theoretical position either.

Results for the Social Causation models connecting the paths between employment and symptomatology are also not consistent. There was a marginally significant inverse relationship as expected. However, the majority of these paths were non-significant or had error measures that were too large. In fact there appears to be little to no relationship between employment and symptoms. And, there is no evidence of any indirect effects. Given these results there is no support for the relationship between employment and psychiatric symptomatology for the Social Causation model for these participants. These results do not support the current literature findings mentioned above. However, this result needs to be carefully considered in light of the non-normal distribution of the employment variable. When one considers the Social Selection theoretical position, there are no clear conclusions about the relationship between symptoms and employment at a later point in time. However, there is marginal support for an inverse relationship that would support the Social Selection theoretical model.

Considering the Social Causation theoretical position, the current evidence clearly

demonstrated no relationship between household income at Time-1 and later psychiatric symptomatology. These results support the findings of Keitner, Ryan, Miller, and Norman, (1992) that SES did not predict recovery from psychiatric symptomatology rather than the findings of Feather (1989), McGlashan (1991), and Narrow, Rae, Moscicki, Locke, and Regier (1990). Results are fairly consistent for this path. These findings do not support the theoretical position that purports that financial resources at one point in time impact symptomatology at a later point in time. These results again fail to support the current literature findings above. When examining the Social Selection models it is clear that symptomatology and household income may be related in an indirect manner. The existence of only indirect effects demonstrates that symptomatology may only impact income via its relationship with education, employment, and perception of financial adequacy. These results do support the Social Selection theory relating mental illness symptomatology with declining social functioning. These results support the findings reported by Feather (1989), McGlashan (1991), McKenry, Browne, and Kotch (1990); Narrow, Rae, Moscicki, Locke, and Regier (1990).

Given the Social Causation model, one would predict that social support and education would be positively related. In all cases of this model the path was not significant. Therefore, it appears that this relationship does not exist in any consistent way for the Social Causation theoretical position. These results were almost identical to those found with the Social Selection models. All of the paths were not significant so, given these inconsistences, there is not a lot that can be said about this path in the Social Selection model, either. One would expect these variables to be positively related given

the available literature (see Ross & Huber, 1985). However, there may be unique qualities related to this sample. These results do not support the Social Selection or Social Causation theory proposed to explain the relationships between socioeconomic status, social support, and psychiatric symptomatology.

According to Ross and Huber (1985), the interaction of **education and income** significantly influences perceptions of economic hardship. Unemployment is common among these participants. In fact, for the Time-1 sample only 25% of the sample was employed full-time and 17% were employed part-time. This leaves 58% of the Time-1 sample not gainfully employed. Frese (1987) believes that depression is the result of unemployment rather than the inverse and that the effect of unemployment is moderated by hope and one's financial situation. According to Pearlin et al. (1981), changes in family income influenced economic strain leading to changes in depression. Longitudinal analysis supports the notion that economic hardship influences depressive symptomatology rather than vice versa (Stolsenberg, 1980).

The current results, while proving to be ambiguous with respect to employment by itself, were not equivocal about the impact of perceptions of financial adequacy. Regardless of the causal model applied, financial adequacy was related to both psychiatric symptomatology and social support. For the Social Causation model significant negative relationships exist between financial adequacy perceptions at one point in time and symptoms at a later point in time. For the first time-lagged model there was evidence of an indirect effect of financial adequacy (Time-1) on symptomatology (Time-2, -3, and -4). Given this indirect effect it appears that financial adequacy at Time-1 impacts,

through mediation, symptoms at Time-2 via education, employment, and income. However, the other two models indicate only the presence of a direct effect of financial adequacy at Time-1 upon symptomatology at Time-3 and Time-4, suggesting that the other SES variables did not mediate the impact of financial adequacy perceptions at these time frames. These results support the belief that social functioning, via financial adequacy perceptions, at one point in time does appear to consistently impact psychiatric symptomatology at a later point in time, whether the effect is direct or indirect, supporting the Social Causation Theory. This finding also demonstrates that it may be the perception rather than the actual income level that is important in these clients' lives.

From the Social Selection theoretical position, psychiatric symptomatology and financial adequacy perceptions also appear to be consistently related to one another as predicted. For this effect, symptoms at one point in time are clearly related to one's perception of financial adequacy at a later point in time. This finding is consistent for all of the models evaluated. This consistent finding clearly indicates that symptomatology can impact social functioning at a later date supporting the Social Selection theoretical position.

There are difficulties relating financial status with psychiatric symptoms since it is difficult to determine a clear distinction between the subjective and the objective resources (Carpiniello et al. 1989). According to Marans and Rogers, objective status characteristics, like family income, are insufficient to explain assessments of one's environment, attitudes, or satisfaction with various dimensions of life, including financial status (as in Ross & Huber, 1985). This appears to be the case with these findings. The

objective indicators of SES did not consistently relate to either symptomatology (Social Causation) or social support (Social Selection). However, it is unequivocal that the perception of financial adequacy was related in all of the models representing both theoretical positions.

Epidemiological literature has reported a direct relationship between being poor and the increased need for mental health services (Dohrenwend & Dohrenwend, 1969; Hall & Royse, 1987; Jackson et al. 1982; Longest, Kovan, & Tweed, 1979; Srole et al. 1962; White, 1986; Zautra & Simons, 1978). Poverty, low SES, and unemployment have been found to be related to higher stress (Brown, Bhrolchain, & Harris 1975; B. S. Dohrenwend et al.1978; Eaton, 1974; Lindblad-Goldberg, Dukes, & Lasley, 1988; Makosky, 1982; McLanahan, 1983; Miller, 1988). Low-SES environments are stressful and reduce people's capacity to manage stress, thereby increasing negative emotions (Gallo & Mathews, 2003). Chronic economic stress impacts all facets of patients' environments (Feather, 1989). The mentally ill are often unemployed and from lower income categories (Pearlin et al. 1981). Economic distress, and being forced to live on minimal disability income are stressful for those with mental health problems (Charlifue & Gerhart, 1991). As income decreases economic hardship increases and chronic economic difficulties are extremely important factors in the etiology of depression (Ross & Huber, 1985).

According to Pearlin (1982), longitudinal analysis supports a causal interpretation of the relationship between socioeconomic status variables and health. They also state that the relationship between socioeconomic status and psychiatric distress is mediated by

financial resources with chronic economic strain explaining the relationship between income and depression; as income decreases economic hardship increases, and as a result depression worsens and low income leads to perception of poor financial inadequacy.

The current crossed-lagged results fail to support Pearlin's (1981) findings. According to both theoretical positions it was financial adequacy that impacted psychiatric symptomatology not the raw SES variables. In fact, it was financial adequacy that mediated the effects of both social support and symptomatology upon a subsequent support or symptom variable.

Decrements in SES have been found to be related to the appearance of psychological symptoms (Dooley & Catalano, 1980). Being in an economically disadvantaged group can be particularly toxic for psychiatric patients (McGlashan, 1991). Treatment prevalence for psychiatric difficulties increases as neighbourhood income levels reduce, especially for psychotic disorders, and as people become more disabled they often are forced to move to even lower SES neighbourhoods (Dohrenwend, 1998; Tataryn, Mustard, & Derksen, 1994). It appears that having worsening psychiatric symptoms is related not only to worse social support but to worse perceptions of financial adequacy. And it seems that low levels of support are related to higher levels of symptomatology and lower financial adequacy perceptions.

When evaluating the Social Causation Model as a whole it becomes evident that social support at an earlier point in time impacts symptomatology at a later point in time in an inverse manner. Findings also support the inverse relationship between financial adequacy perceptions at an earlier point in time and psychiatric symptomatology at a later

point in time. These findings support the Social Causation Model. The findings about the relationships between education, employment, and income with symptomatology do not support these components of the Social Causation Model. These results were all inconsistent in one manner or another.

When evaluating the Social Selection model as a whole it becomes clearer that symptomatology at one period in time and social support at a later point in time are inversely related supporting the Selection Model. There is also clear evidence of an inverse relationship between symptomatology and financial adequacy perceptions supporting the Social Selection theoretical position. When evaluating the relationships between education, employment, and income with symptomatology, results are inconsistent and do not support these components of the Social Selection Model.

CHAPTER 6:

Phase-IV Hospitalization Data Analysis

The objectives of this component of the research were to explain how the hospitalisation stay variable (LOS) interacted with the other primary variables. However, the longitudinal data available were significantly limited. Given these limitations this portion of this dissertation is descriptive in nature. Rather than repeat the relevant literature here the reader is referred to the hospitalization literature in Chapter 2.

For Phase-IV of this research the total sample was split, based upon recent hospitalization history. There were 86 participants who indicated that they had never been treated as an inpatient for psychiatric difficulties. Of the 232 Time-1 participants 140 reported at least one hospitalization. Of these, only 79 participants reported having had an inpatient experience during the last three years. These participants were selected ($\underline{N} = 79$) to evaluate the influence of the model variables upon hospitalization and the impact inpatient treatment had upon primary variables. Participant number 203 was eliminated due to a large quantity of missing information and extreme or irrational responses. The hospitalization (LOS) and employment variables, in this smaller hospitalized sample, were neither distributed normally (evaluated with normality plots) nor was the sample size large enough to assume the variables came from a normal distribution. To assume normality one requires a sample size of at least 100 (Sobel, 1988). None of the attempts at transforming the employment variable were successful.

The LOS variable was also not normal with some extreme outliers. Transforming this variable was attempted successfully using a natural log transformation. The

transformed variable was normally distributed. Comparisons of Spearman correlational patterns, of the Logged values with non-logged values, resulted in identical correlational values. Evaluating the Pearson correlation patterns using the Natural Log or the raw value for LOS also resulted in near identical correlation values. Hierarchical regressions were also performed, using both LOS and the Log of LOS, to evaluate whether results would be different using the Natural Logged values for LOS. There were virtually no differences between the models based upon using the logged or non-logged LOS variable. According to Tabachnick, and Fidell (2001), transformations can hinder the interpretation when the scale being transformed is meaningful and this meaning is widely used. The LOS variable does have a meaningful quality. Therefore, no further evaluations of transformations were performed.

The sample size was also too small for the requirements of multiple regression. An adequate sample size for seven predictor variables, a moderate effect size, and a dependent variable that is non-skewed, should be at least 111 individuals (Tabachnick & Fidell, 1989). This sample dependent variable was skewed, and a small to moderate effect size was expected. Therefore, the path analysis techniques used on the larger sample were not appropriate here. Neither were many other multivariate regression procedures (Tabachnick & Fidell, 1989). The relationships between the variables in the hospitalized sample were evaluated with Spearman Rank Order Correlations (see Table 32) since non-parametric measures of association do not require a normality assumption to be valid (Levine & Fox, 1994).

Table 32

Spearman Rank Order Correlation Coefficients for the Respondents with a LOS During the Last Three Years, all Time-1 measures, N = 76

	CF	SAT-1	ED-1	EMP-1	INC-1	FAD-1	SYMP-1	LOS-1
CF-1	- -	r_s = .491	r_s = .150	r_s = .144	r_s = .085	r_s = .179	r_s = -.287	r_s = .212
		p< .001/2	p=.628/2	p = .215/2	p=.467/2	p = .123/2	p = .012/2	p = .066/2
SAT-1		- -	r_s = .013	r_s = -.083	r_s = -.019	r_s = .179	r_s = -.269	r_s = .217
			p = .911/2	p = .477/2	p = .872/2	p = .121/2	p = .019/2	p = .060/2
ED-1			- -	r_s=.285	r_s = .281	r_s= .083	r_s = -.016	r_s = .166
				p = .013/2	p = .014/2	p = .474/2	p = .892/2	p = .152/2
EMP-1				- -	r_s = .232	r_s = -.043	r_s= -254	r_s = - .209
					p = .011/2	p = .709/2	p = .027/2	p = .070/2
INC-1					- -	r_s = .487	r_s = - .058	r_s = .180
						p < .001/2	p = .618/2	p = .121/2
FAD-1						- -	r_s = -.251	r_s = .134
							p = .029/2	p = .250/2
SYMP-1							- -	r_s = .205
								p = .075/2
LOS-1								- -

Note: Abbreviations are as follows: r_s = Spearman Correlation Coefficient, p = probability, p-value/2 = adjustment for one-tailed p-values, CF = contact frequency, SAT = satisfaction with support, ED = years of education, EMP = employment score, INC = family income, FAD = financial adequacy score, SYMP = symptomatology score, and LOS = number of nights in an inpatient psychiatric facility during the last three years.

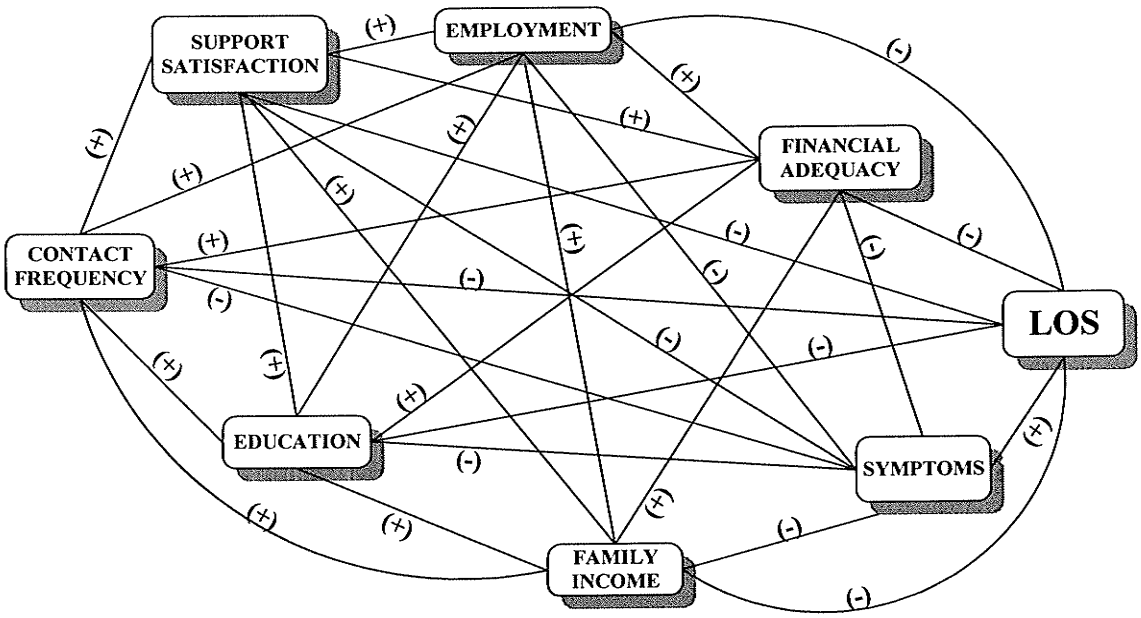
This section begins with a description of individuals in the hospitalized group as they are compared to the group with no hospitalization history. Correlational information is provided demonstrating relationships between primary variables. This is followed by descriptive information including means, standard deviations, and percentages. Although the entire path analysis procedure was not performed on the smaller hospitalized sample ($\underline{N}_{H3} = 78$), because of failure of normality and sample size requirements, a tentative hypothetical hospitalization model is provided in Figure 18 with results in Figure 19 (pages later). Scatter diagrams indicated that the assumption of linearity, necessary for Spearman rank correlations to be valid, was tenable.

Phase-IV Hypotheses and Rationale

According to Cohen and Sokolovsky (1978), psychiatric clients are often socially withdrawn and the length of inpatient treatment stays (LOS) is unlikely to be the reason why. However, others believe that social withdrawal is part of the 'Social Breakdown Syndrome'. Hospital stays facilitate the syndrome by reducing contact with social networks, resulting in diminished role functioning and identity, and passive withdrawal of mentally ill patients (Kuypers & Bengtson, 1973). Syme, and Pyska (1988) have stated that available research fails to support the theory that illness causes social support inadequacies. Some believe that the social support/health relationship could function in both directions or be a result of another unmeasured variable (Hammer et al. 1982). When one is in the hospital one cannot be out socializing or occupied with employment, impacting social functioning in a negative manner. For a more detailed literature review

the reader is referred to Chapter 2. Hospitalization visits are primarily dependent upon the visitor and not the patient. Patients reported that, after the first hospitalization, they received many fewer visitors on subsequent admissions ($\underline{N}$ = 3, personal communication, Murray, 1994). Given the aforementioned literature, inpatient length of stay was expected to impact social functioning and social support in a negative manner, and social support was also expected to impact psychiatric symptomatology in an inverse manner. LOS was also expected to be positively related to psychiatric symptomatology. More symptomatic individuals should be expected to spend more time as inpatients.

Figure 18. The preliminary fully specified hypotheses hospitalization tentative LOS model. Valence signs indicate the direction of twenty-eight hypothesised variable relationships {$\underline{N}_{H3}$ = 78, (+) indicating positive and (-) negative correlation}.



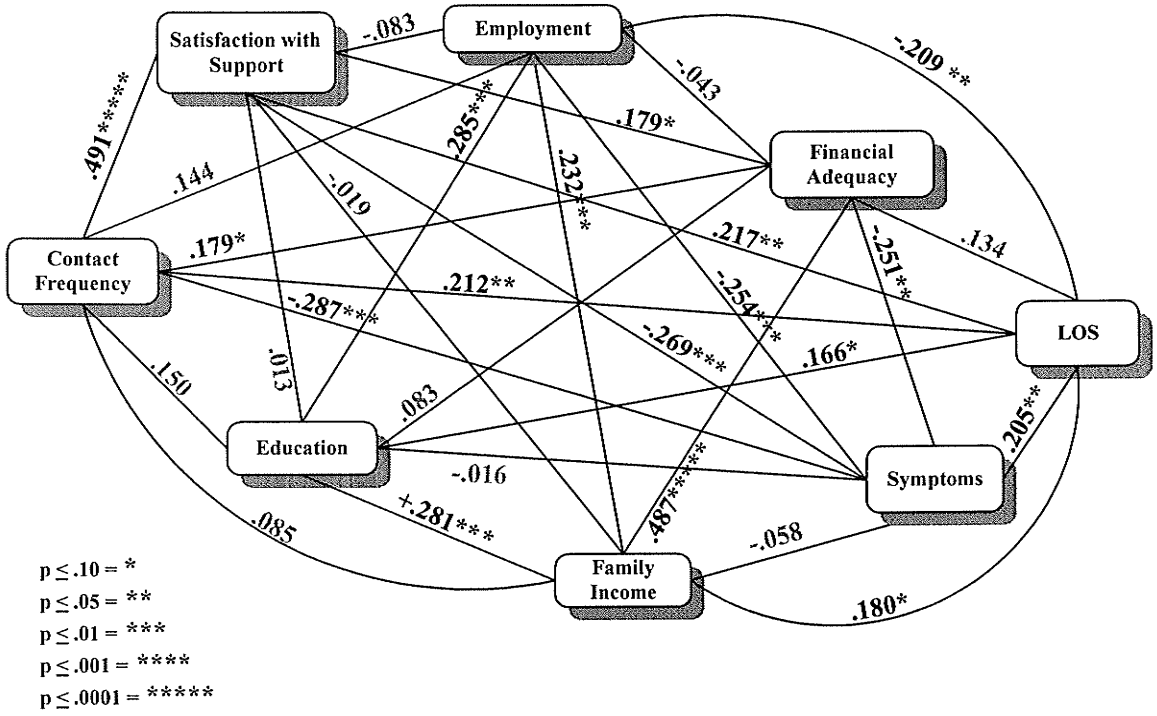
As previously discussed, employment, education, income, and financial adequacy perceptions are closely related and should demonstrate positive relationships. The relationship between employment and inpatient treatment is difficult to disentangle. One cannot be working if one is being treated as an inpatient. Serious depressive disorders tend to appear in late adolescence, interrupting educational opportunities. These disruptions can create cascading difficulties with other social functioning variables like education, employment, and income. These difficulties will all lead to fewer income earning opportunities. The reciprocal relationship between unemployment and depression may be due to selection biases in cross-sectional samples as it is just as possible that distressed individuals are more likely to become unemployed as unemployed individuals becoming depressed (Hall & Johnson, 1988; Kessler, House, & Turner, 1987). In a longitudinal design, Kessler, Turner, and House (1989) did not find support for the idea that distress from unemployment reduces the likelihood of future employment. Given this information, the SES variables (education, employment, and family income) were expected to have a positive relationship to the financial adequacy variable, and negative relationships with psychiatric symptomatology and LOS.

A patient's perception of financial adequacy is impacted by his or her financial situation. The interaction of education and income impacts the perceptions of economic hardship (Ross & Huber, 1985). According to Stolsenberg (1980), longitudinal analysis indicated that economic hardship impacts depressive symptomatology rather than the opposite direction of causation. Pearlman's et al. (1981) results confirmed that reductions in family income negatively impacted economic strain and depression. Given this

information, negative relationships between the financial adequacy variable and the symptomatology variable were expected.

The rationale for these hypotheses (reported previously) is followed by model results using the Spearman Rank Order Correlations (with their corresponding probability levels) between the variables of interest to represent the total effect of variable pairs as hypothesized in Figure 18 with results on Figure 19. These are not path models they are just possible relations representative of the data we do have. These effects were smaller and would require a sample size more than three times than the one obtained for sufficient power to detect the effects of such magnitude. Of the participants indicating a three-year inpatient stay only 8 (10.35%) had been rehospitalized during the first six months of the study. Finally, a **very tentative** time-lagged model with **Spearman Rank Order Correlations** is provided with hypothetical relationships between Time-1 LOS and Time-2 for those participants with Time-1 hospitalization scores who participated at Time–2 ($\underline{N} = 46$). Time-1 LOS is evaluated for its impact upon Time-2 social support and social functioning variables. The opposite direction of causation was not possible to evaluate due to the time/logic flow of the variables involved.

Figure 19. The preliminary fully specified Spearman Rank Order Correlation results of the model, $\underline{N}_{H3} = 78$.



Description of Hospitalized Participants

Participants who indicated that they had a hospitalization history some time during the three years (H3) preceding the Time-1 survey (N_{H3} = 78) were significantly different, on some of the primary variables, (as might be expected) from individuals who indicated that they had never been hospitalized (NH) for psychiatric reasons (N_{NH} = 86, see Table 33). This table provides one-tailed p-values since we expect those with a hospitalization history to be different from those with no hospitalization history. Results indicate that these groups were equivalent on the social support variables (CF, SAT) and symptomatology (SYMP). Expectations were that participants with a three-year hospitalization history would have lower social support scores. These participants were also expected to be lower on all of the social functioning variables (ED, EMP, INC, FAD) than participants without a hospitalization history. They were also equivalent on perception of financial adequacy. Those with a history of hospitalization during the last three years were significantly different from the NH group on income, education, and employment. In the case of these variables the participants in the NH group demonstrated significantly higher performances on all three of these variables than the H3 group. This was expected since inpatient treatment impacts ED, EMP, and INC in a negative manner. When someone is in the hospital they cannot be increasing their educational attainment, employment weeks, and/or their income.

The findings indicate that the participants with a hospitalization history reported the same level of psychiatric symptomatology as those without a hospitalization history. One would expect those with a history of inpatient treatment would report more

psychiatric symptomatology than those without a history of inpatient treatment. However, at the time when these participants were admitted for inpatient treatment they probably were exhibiting more severe pathology and the current level of symptomatology may be indicative of treatment success.

Table 33

Descriptive Statistics for all of the Primary (CF, SAT, FAD, ED, EMP, INC, SYMP) for Those With a Hospitalized History During the Last Three Years (N_{H3} = 78) and Participants with No Hospital History (N_{NH} = 86) at Time-1

Variable		H3	NH	t-value	one-tailed p-value
CF	M	14.04	13.37	$.937_{(df = 162)}$	p = .150
	SD	4.42	4.76		
SAT	M	4.91	4.63	$.919_{(df = 162)}$	p = .155
	SD	1.85	2.03		
FAD	M	22.19	21.64	$.318_{(df = 160.98)}$	p = .375
	SD	10.54	11.75		
ED	M	13.18	14.53	$-2.51_{(df = 162)}$	p = .007
	SD	3.85	3.10		
EMP	M	25.01	45.00	$-3.13_{(df = 156.02)}$	p = .001
	SD	35.40	46.21		
INC	M	12.81	14.87	$-1.92_{(df = 162)}$	p = .028
	SD	7.18	6.64		
SYMP	M	3.18	3.22	$-.131_{(df = 162)}$	p = .448
	SD	1.92	1.76		

Note. The abbreviations are as follows: NH = no history of psychiatric admission, H3 = hospitalization history during the last three years, CF = contact frequency, SAT = satisfaction with support, ED = years of education, EMP = employment score, INC = family income score, FAD = financial adequacy score, and SYMP = symptomatology measure.

Hospitalized Versus Non-Hospitalized Correlational Data

When one compares the relationships between the primary variables for those with a hospitalization history (HH, $\underline{N}_{HH}$= 140) and those who have no psychiatric hospitalization history (NH, $\underline{N}_{NH}$ = 86) some differences begin to emerge. What follows is only descriptive information in nature. The relationship between EMP and SAT, INC and SAT, INC and CF, ED and SYMP, and EMP and FAD are all near zero for the HH group (see Table 34 and 35). The same variables in the NH group were all significantly related and ranged from $\underline{r}$ = - .18 to $\underline{r}$ = + .34. The NH group appears to conform to more of the simple single variable hypotheses (see Figure 18). For example, given the literature, one would predict that the simple social support variables would be positively related to employment and income. This turns out to be the case for SAT with reference to EMP and INC for the NH group but is not supported with the HH group.

Table 34

Pearson Correlations for Social Support and SES Variable Relationships for Individuals with a Psychiatric Hospitalization History, N_{HH} = 140. One-Tailed P-Values

	CF	SAT	EMP	INC	ED	FAD	SYMP
CF	--	+ .45 p < .0001	- .04 p = .3380	**- .02** **p = .3920**	+ .13 p = .0640	+ .21 p = .0075	-.33 p < .0001
SAT		--	**+ .02** **p = .0426**	**+ .04** **p = .3345**	+ .01 p = .455	+ .27 p < .0001	-.39 p < .0001
EMP			- -	+ .44 p < .0001	+ .22 p = .0056	**+ .01** **p = .444**	+ .18 p = .0017
INC				- -	+ .19 p = .0110	+ .45 p < .0001	-.122 p = .0750
ED					- -	- .04 p = .3255	**.025** **p = .3845**
FAD						- -	-.39 p < .0001

Table 35

Pearson Correlations for Social Support and SES Variables Relationships for Individuals with NO Psychiatric Hospitalization History, N_{NH} = 86, One-Tailed P-Values

	CF	SAT	EMP	INC	ED	FAD	SYMP
CF	--	+ .42 p < .0001	- .03 p = .3910	**+ .18** **p = .0405**	+ .09 p = .2055	+ .172 p = .0565	-.28 p = .0050
SAT		--	**+ .24** **p = .0125**	**+ .34** **p = .0005**	+ .03 p = .3865	+ .314 p = .0015	-.49 p < .0001
EMP			- -	+ .41 p < .0001	+ .26 p = .0075	**+ .25** **p = .0115**	- .33 p = .0010
INC				- -	+ .29 p = .0035	+ .421 p < .0001	-.286 p = .0040
ED					- -	.051 p = .3210	**-.21** **p = .0285**
FAD						- -	-.25 p = .0055

Note: Abbreviations for the two tables above are as follows: CF = contact frequency, SAT - satisfaction with support, ED = years of education, EMP = employment score, INC = family income, FAD = financial adequacy score, and SYMP = symptomatology score.

The relationship between CF and INC is non-existent for those with a HH but was marginally significantly negative for those with NH of hospitalization. The relationship between SAT and EMP also was very small for those with a HH and was significantly positive for those in the NH group. The relationship between EMP and FAD was near-zero for participants with a HH yet was significantly and positively related in the NH group. ED and SYMP also demonstrate differences. There was a near zero effect for the HH group and a significant negative effect for the NH group. And finally, the relationship between EMP and INC was different for the two groups. The HH group had a near zero effect again and the NH group demonstrated a moderate positive effect. This last effect was the largest of the five different correlations.

EMP and SYMP are also different when one compares the correlations between the HH and NH groups. The relationship, according to the literature and common sense would be the higher the symptoms the lower the employment. The data demonstrate that for the HH group EMP and SYMP are significantly and positively related (small effect size) rather than inversely related. The same relationship for the NH group demonstrates a moderate significant negative relationship between these variables. At the present there is no clear explanation of this phenomenon. It may be due to the lack of normality and unequal variances of the employment variable.

It is interesting looking at these differences in correlational patterns. It appears that the hypothesized relationship for these variables fits the NH group better that the HH group. It is also interesting, in light of the previous comparisons of the primary variables (t-tests see Table 34 above) that all of the different correlations involve at least one of the

variables with significant differences according to the t-test data (ED, EMP, and INC see Table 33 above). One must also keep in mind that there are significant distribution difficulties with the EMP variable. These findings are tentative, and not predicted, and will need further research to tease out the differences between those with and those without a hospitalization history.

Hospitalization Rates

According to Tataryn et al. (1994), in an adult Canadian sample the prevalence of psychotic disorders was estimated at 1% and was similar for both genders. Also, 10.5 % of Manitobans require acute hospital care for any mental health disorder. These rates vary between 7.8 % of the population of Central region to 11.8 % of the population of Thompson region" (p. 1) and persons with psychotic disorders have an exceptionally high hospitalization rates (Tataryn et al. 1994). In the Mental Health Epidemiological Catchment Area Study mood disorders, major depression, dysthymia, and bipolar disorder had an overall prevalence of 8.3% (Regier et al. 1990). Relapse rates have been found to be between 23.0% and 53.8% for individuals with depression (Colenda, Trinkle, Hamer, & Jones, 1991). Subclinical depressive symptoms are strongly predictive of future major depression (Judd & Akiskal, 2000). Patients with late-onset illness had a significantly higher readmission rate (47.4%) than other patient groups (Colenda et al. 1991). Historical Canadian cohort studies report that "the proportion of first admissions having a non-psychotic disorder increased from 26.0% in 1954 to 48.0% in 1962, to 67.0% in 1969" (Woogh, 1986, p. 214). During a psychiatric emergency assessment the

potential for violence is a significant factor in the decision to admit someone for inpatient treatment (Beauford, McNiel, & Binder, 1997). Increasingly, in Canada, psychiatric difficulties are being treated on an outpatient basis (whenever possible) rather than an inpatient basis. This has led to a reduction in LOS (for those who have been admitted), and an increase in the demand for outpatient services (D'Arcy, 1978).

Initial expectations (based upon a rough estimate of the attendance of regular meetings of the Mood Disorders Association) were that the total sample ($\underline{N}_T = 232$) would consist of two approximately equal sized groups (at least 100 in each group) of clients: those who had a hospitalization history during the three years preceding the survey, and those who had never been admitted to an inpatient treatment facility or had no history of hospitalization during the last three years. Many studies follow patients after they have been identified at an emergency ward; this results in excluding patients who are identified somewhere else in the mental health system (Keck et al. 1998). However, at Time-1, the resulting sample consisted of only 78 (33.0% of the total sample) respondents who had been admitted for inpatient psychiatric treatment during the last three years. Of the total sample, 60.3% ($\underline{N}_{HH} = 140$) indicated that they had a hospitalization history in the past and 37.0% ($\underline{N}_{NH} = 86$) had never been hospitalized for psychiatric reasons. Of those individuals who indicated they had a psychiatric hospitalization history only 54.0% ($\underline{N}_{H3}= 78$) had been admitted to a psychiatric facility during the last three years (H3) and only 32.0% ($\underline{N}_{H1} = 45$) had been admitted for treatment during the year preceding (H1) the survey.

There was missing information on hospitalization for 2.8% of the sample.

Participants in self-help groups have been found to be more likely to seek help, tended to come from a higher socioeconomic background, used more interactive active coping strategies (Gidron, Guterman, & Hartman, 1990). This finding will lead to a biassed sample that will likely not be representative of all individuals with mood disorders.

At Time-2, 9 individuals reported being inpatients in the previous six months. Eight of them provided a positive LOS for the previous six months (subject numbers, 59, 66, 100, 488, 532, 570, 817, 860). Of these, only two had not reported a previous three year LOS. This results in a rehospitalization rate of 7.7% for the persons who participated at both Time-1 and Time-2. There were six individuals who reported a Time-3 six-month LOS (subject numbers, 59, 358, 363, 383, 817, 885). The numbers of participants who reported a six-month stay dropped to 4 for Time-4 (subject numbers, 133, 379, 532, 806). Only two patients were involved in more than one of these inpatient reports. There is little overlap in subjects across the four time-points.

Hospitalization and Primary Variables

When one considers individuals who reported having a hospital stay during the last three years (H3) and compares them to those reporting never having being treated as an inpatient (NH) some less than predictable results occurred (see Table 36). The average SAT scores, CF scores, FAD scores, and SYMP scores were not significantly different (See Table 37). Given the literature and hypotheses, one would have expected those with a three year hospitalization history to have a more serious disorder, to have lower levels of social support, financial adequacy perceptions, and greater symptomatology scores.

There were some predictable differences in education, employment levels, and household income (see Table 37). Those who report having a hospitalization during the last three years had lower scores on ED, EMP score. Since EMP was not distributed normally a MannWhitney 'U' test was performed and was also significantly different between those with and without a history of hospitalization ($\underline{M}_{NH}$ = 45.00, $\underline{SD}$ = 46.212; $\underline{M}_{H3}$ = 25.02, $\underline{SD}$ =35.404; $\underline{U}$ = - 2.297, $\underline{p}$ = .022/2). It appears for the H3 group that having had a recent inpatient stay is indeed related to lower scores on SES variables like education, employment, and household income.

Table 36

Descriptive Information about Hospitalization Histories

	Total Sample	No History	% No History	HH	% HH	LTY	Last 6-12	A	MI
Time-1	$\underline{N}$ = 232	86	37.0%	140	60.0%	78	45*	39.7	3.0%
Time-2	$\underline{N}$ = 146	53	38.0%	81	58.0%	78	8	30.5	4.3%
Time-3	$\underline{N}$ = 123	50	39.0%	78	59.0%	78	6	37.3	1.3%
Time-4	$\underline{N}$ = 96	39	39.0%	60	59.0%	78	6	27.5	2.0%

Note. HH = Hospitalization History, LTY = Hospitalized during the Last Three Years, LAST 6-12 = Hospitalized during the last six to twelve months, A = Average Length of Stay, and MI = % of Missing Data. * = data for a twelve month period other figures are for a six month period.

Table 37

Descriptive Statistics for Sample of Individuals With and Without Hospital Stay During the Last Three Years and Total Samples

	N	CF	SAT	ED	EMP	INC	FAD	SYMP	ALOS
Individuals with three year hospital history (H3)	78	M = 14.0 SD = 4.5	M = 4.9 SD = 1.9	M = 13.2 SD = 3.68	M = 25.1 SD = 29.3	M = 12.8 SD = 6.9	M = 22.2 SD = 10.6	M = 3.2 SD = 1.8	M = 33.6 SD = 27.5
Individuals with NO hospitalization history (NH)	86	M = 13.8 SD = 4.5	M = 4.6 SD = 2.0	M = 14.5 SD = 3.11	M = 45.0 SD = 46.2	M = 14.8 SD = 6.6	M = 21.9 SD = 11.6	M = 3.2 SD = 1.8	N/A
Total Sample	232	M = 13.6 SD = 4.6	M = 4.9 SD = 1.9	M = 13.9 SD = 3.5	M = 34.9 SD = 42.7	M = 13.6 SD = 6.8	M = 22.3 SD = 11.1	M = 3.1 SD = 1.8	M = 10.6 SD =21.9

Note. N = Sample Size, CF = Contact Frequency, SAT = Satisfaction with Support, ED = Education, EMP = Employment, INC = Household Income, FAD = Financial Adequacy Perceptions, SYMP = Symptomatology, and ALOS = Average Length of Hospital Stay.

The likelihood of being hospitalized for this self-help group was not based upon marital status $\chi^2_{(1)} = 1.391$, $\underline{p} = .499$), employment status $\chi^2_{(1)} = 1.934$, $\underline{p} = .380$), or gender ($\chi^2_{(1)} = 1.476$, $\underline{p} = .478$). However, there was a predictable relationship between admission and 'Bipolar' diagnosis. Individuals who indicated that they were bipolar were more likely to be admitted than individuals who indicated that they had only a depressive disorder ($\chi^2_{(1)} = 10.092$, $\underline{df} = 231$, $\underline{p} = .039$).

Given the literature about hospitalization and illness severity one would predict those who have been hospitalized for their mood disorders to report significantly higher levels of symptomatology. Individuals in the group with a hospital stay during the previous three years reported symptomatology that was no different than those individuals who did not have a hospital stay ($\underline{M}_{H3} = 3.18$, $\underline{SD} = 1.94$; $\underline{M}_{\text{no stay}} = 2.99$, $\underline{SD} = 1.75$; $\underline{t}_{(224)} = .754$, $\underline{p} = .452/2$). When the group with no history of hospitalization is compared to those with a hospitalization history there are also no significant differences in recent psychiatric symptomatology ($\underline{M}_{NH} = 3.22$, $\underline{SD} = 1.76$; $\underline{M}_{HH} = 2.92$, $\underline{SD} = 1.83$; $\underline{t}_{(225)} = 1.20$, $\underline{p} = .26/2. .452/2$). For the total sample at Time-1, recent symptomatology was correlated to the last years LOS ($\underline{r} = .240$, $\underline{p} < .0001/2$) and to the number of stays during the last three years ($\underline{r} = .129$, $\underline{p} = .053/2$). However, recent symptomatology does not appear to be related to average three year hospital stay ($\underline{r} = .064$, $\underline{p} = .221/2$). Once again, these symptomatology results might be expected since symptoms may have been exacerbated in acuity just prior to hospitalization.

Ideally, psychiatric treatment should be based on a symptom/diagnosis basis rather than on socioeconomic variables, like class membership. When one considers the severity of self-reported symptomatology one would expect that the individuals reporting a history

of psychiatric hospitalization would report more severe symptomatology than those individuals reporting no history of psychiatric hospitalization. This may also be due to symptoms being ameliorated as a result of (or at least since) hospitalization. The current results indicated that those with a psychiatric hospitalization history did not report more severe psychiatric symptomatology than those with no psychiatric hospitalization history when data was collected. In fact, self-reported psychiatric symptomatology, for the two weeks prior to filling out the survey, was marginally higher in those without a psychiatric hospitalization.

Hospitalization and Other Treatment Resources

A single item open-ended question, about other resources was included with the previously mentioned pertinent variables. The question asked participants, when they were ill with psychiatric symptoms and were not admitted to the hospital, what did they do to care for themselves. They were also prompted for information about community resources that they may have depended upon. Responses were arranged into a number of categories (see Table 38 below) including depending upon friends, family, or partners; professional care (doctor, psychiatrist, psychiatric nurse), non-professional care like self-help groups, outpatient or day services, self care, exercise, para-professional services like pastors, social workers, home care, and counsellors. When distributions of responses of this data were evaluated it became apparent that there were almost no differences in the proportion of response categories endorsed by those with and without hospitalization histories.

Table 38

Categorical Distributions of Community Resources Endorsement

	Total Sample $N_{(total)} = 232$	History of Hosp $N_{HH} = 140$	No History of Hosp $N_{(NH)} = 86$	Yes H3 $N = 78$	No H3 $N = 147$
Self Care	23.6%	19.7%	28.7%	23.1%	24.3%
Professional Care	34.8%	36.1%	39.1%	30.8%	37.8%
Family, Friends and Partners	21.0%	18.0%	23.0%	23.1%	20.3%
Self Help, Salvation Army	21.0%	23.0%	19.5%	23.1%	20.9%
Outpatient or Day Programs	3.0%	100.0%	1.1%	6.4%	1.4%
Para-professional, Social Worker, Counsellor, Pastor, Home Care	11.6%	13.1%	11.5%	10.3%	12.2%
Exercise	8.2%	9.8%	10.3%	5.1%	10.1%

Note. H3 = hospitalization during the three years prior to the survey.

Subsequent analyses were performed on the frequencies above. Results indicate that there were few differences in categorical frequency distributions based upon whether the participants reported a three year hospitalization history or not, and whether the participants had ever been treated in an inpatient facility for their psychiatric disorder. For individuals reporting a hospitalization history during the last three years it is clear from the 2-by2 Chi Squared statistics that there was only one category in which a difference occurred, and that was the use of outpatient services ($\chi^2_{(1)} = 4.311$, $\underline{p} = .038$). It makes sense that there would be more use of outpatient services by those reporting a hospitalization stay during the last three years. This is due to inpatients graduating to outpatient care with treatment intensity higher just after release from inpatient care. Other categories were not different for these two groups (self care, $\chi^2_{(1)} = .056$, $\underline{p} = .813$; professional care, $\chi^2_{(1)} = 1.194$, $\underline{p} = .275$; friend, partner, and family, $\chi^2_{(1)} = .216$, $\underline{p} = .642$; non-professional, self-help, $\chi^2_{(1)} = .118$, $\underline{p} = .731$; para-professional, counsellor, pastor, $\chi^2_{(1)} = .197$, $\underline{p} = .657$; exercise, $\chi^2_{(1)} = 1.698$, $\underline{p} = .193$).

When one compares individuals with and without a hospitalization history then there are no significant differences in the frequency distributions for alternative community resource these participants depended upon (self care, $\chi^2_{(1)} = 1.689$, $\underline{p} = .194$; professional care, $\chi^2_{(1)} = 1.039$, $\underline{p} = .308$; friend, partner, and family, $\chi^2_{(1)} = .203$, $\underline{p} = .653$; non-professional, self-help, $\chi^2_{(1)} = .300$, $\underline{p} = .584$; outpatient services, $\chi^2_{(1)} = 1.196$, $\underline{p} = .274$; para-professional, counsellor, pastor, $\chi^2_{(1)} = .013$, $p = .908$; exercise, $\chi^2_{(1)} = .764$, $\underline{p} = .382$). These descriptive results appear to indicate that

individuals treated as an inpatient some time in their psychiatric history are very similar to those without such a history in their use of non-hospital resources. This is supportive of earlier symptomatology results.

Income and Inpatient Treatment

There have been considerable changes over time in the characteristics of the Canadian population treated for mental illness (D'Arcy, 1978). The median household income for the Winnipeg Area Study (WAS) participants in 1994 was between $40,000-44,999 dollars. The median income for the self-help participants was only $28,000-29,999). There appears to be a difference in median incomes when these participants are compared to the WAS for the same year (Mann-Whitney U-test, $z = -7.603$, $df = 334$, $p < .0001$). When one compares those with a hospitalization history against those without a hospitalization history distinct differences also appear. Those without a hospitalization history report their median household incomes fall between $36,000-37,999 which is much closer to the WAS results. Those with hospitalization histories report their median household income to be $26,000-27,999 when compared to those without a hospitalization history (Mann-Whitney U-test, $z = -1.842$, $df = 224$, $p = .0325$).

These results indicate that families of psychiatric patients have a lower standard of living. The general population sample, given literature findings, should have a higher median income category than those with a psychiatric client in the household. And even the self-help sample should be made up of the less ill and possibly those with higher incomes as in most surveys (Dillman 1978). When one considers those with and without

a hospitalization history the differences become even more evident. Admission for inpatient treatment was related to household income category for these participants.

Phase IV: Cross-Sectional Hospitalization LOS

Results and Discussion

The primary hypothesis of this research predicted that a causal negative relationship existed between social support (contact frequency and satisfaction with support) and psychiatric symptomatology and hospitalization. The results from the symptomatology models supported these hypotheses (see Figure 19 above). As previously discussed, for the symptomatology models, both contact frequency and satisfaction with support were negatively related to symptomatology and positively related to each other. For the hospitalization data (see Table 39) it appears that both contact frequency and satisfaction with support were, again, positively related to each other as in the symptomatology model ($\underline{N} = 78$, $\underline{r}_s = .49$ $\underline{p} < .0001/2$), and are both significantly inversely related to self-report psychiatric symptomatology ($\underline{N} = 78$, SAT, $\underline{r}_s = -.269$, $\underline{p} = .019/2$; CF, $\underline{r}_s = -.287$, $\underline{p} = .012/2$). However, in the hospitalization results, contact frequency and satisfaction with support were both positively related to three year hospital stays ($\underline{N} = 78$, it should be noted that these results are the opposite of valence expected, $\underline{r}_s = .212$, $\underline{p} = .066/2$; $\underline{r}_s = .217$, $\underline{p} = .060/2$, respectively). These relationships are the opposite of the predicted relationships and are incongruent with the pretest sample of hospitalized clients (Murray, 1995).

Table 39

Spearman Rank Order Correlation Coefficients for the Respondents with a LOS During the Last Three Years (H3), N = 78, one-tailed p-values

	CF	SAT-1	ED-1	EMP-1	INC-1	FAD-1	SYMP-1	LOS-1
CF-1	-	$r_s = .491$	$r_s = .150$	$r_s = .144$	$r_s = .085$	$r_s = .179$	$r_s = -.287$	$r_s = .212$
	-	$p < .0001$	$p = .314$	$p = .1075$	$p = .2335$	$p = .0615$	$p = .0060$	$p = .0330$
SAT-1		- -	$r_s = .013$	$r_s = -.083$	$r_s = -.019$	$r_s = .179$	$r_s = -.269$	$r_s = .217$
			$p = .4550$	$p = .2360/2$	$p = .4360$	$p = .0615$	$p = .0095$	$p = .0300$
ED-1			- -	$r_s = .285$	$r_s = .281$	$r_s = .083$	$r_s = -.016$	$r_s = .166$
				$p = .0065$	$p = .0070$	$p = .2370$	$p = .4460$	$p = .0760$
EMP-1				- -	$r_s = .232$	$r_s = -.043$	$r_s = -254$	$r_s = - .209$
					$p = .0055$	$p = .3545$	$p = .0135$	$p = .0350$
INC-1					- -	$r_s = .487$	$r_s = - .058$	$r_s = .180$
						$p < .0001$	$p = .3090$	$p = .0605$
FAD-1						- -	$r_s = -.251$	$r_s = .134$
							$p = .0145$	$p = .1250$
SYMP-1							- -	$r_s = .205$
								$p = .0375$

Note: Abbreviations are as follows: r_s = Spearman Correlation Coefficient, p = one-tailed p-values, CF = contact frequency, SAT = satisfaction with support, ED = years of education, EMP = employment score, INC = family income, FAD = financial adequacy score, SYMP = symptomatology score, and LOS = number of nights in an inpatient psychiatric facility during the last three years.

These findings are possibly due to the nature of the population being assessed. The respondents in this research, unlike the hospitalized clients in the pretest sample, were already members of a self-help group. Clients who participate in such groups are different from other individuals who have the same psychiatric diagnoses but do not participate in these groups (Gidron, Guterman, & Hartman, 1990). It is possible that individuals who belong to self-help groups are more active help-seekers, and are supported in seeking help by their networks. It may also be possible that individuals with hospitalization histories expect and receive more social support from their networks given a recent hospitalization. Contact frequency and satisfaction with support may also be influenced by an increase in contact and support with other patients, possibly including those met in the inpatient environment.

Another explanation for the positive relationship between social support and hospitalization might be that increased contact with a supportive network of friends and family results in an increased likelihood of symptom identification and resource mobilization. An increase in the identification of severe psychiatric symptomatology could result in a strong support system mobilizing to facilitate psychiatric hospitalization. According to Mechanic (1989), there are many variables that influence recognition of psychiatric symptomatology and the initiation of care. Some of these variables include: the visibility of symptoms, and degree of recognition of symptoms, the salience of deviant behaviour; the extent to which a person perceives the symptoms as serious; the extent to which symptoms disrupt primary role functioning and social activities; the persistence or frequency of symptoms; the tolerance of those interacting with the ill individual; the

availability of information; knowledge of and cultural understanding of the evaluator; and the availability of financial and treatment resources (Mechanic, 1989). Numerous mechanisms interact to facilitate symptom identification and resource mobilization. It is possible that more contact with the individual with psychiatric symptomatology helps family and friends become more knowledgeable about psychiatric symptoms so they recognize smaller variations in the client's "normal" behaviour. It is also possible that larger networks of individuals supporting a particular client are also more likely to include at least one individual who is less tolerant of deviations in the client's behaviour. Also, frequent contact with a smaller network of supportive individuals may lead to the "burn-out" of available supports and tolerance for aberrant behaviour.

Social support was related to LOS for the hospitalization H3 model. Both of the support variables appeared to have a positive relationship with LOS ($\underline{N}$ = 78, SAT, $\underline{r}_s$ = .217, $\underline{p}$ = .060/2; CF, $\underline{r}_s$ = .212, $\underline{p}$ = .066/2) rather than an inverse relationship as predicted and contrary to the results obtained by Holsten and D'Elia, (1985), and Mai et al. (1993) who reported that individuals with inadequate support experienced increased LOSs, and results from Rud and Noreik (1982) who found that social isolation was related to increased LOSs. These results seem to support the findings of Cohen and Syme (1985) who reported that "large networks may be stress reducing but, at the same time consist of cosmopolitan members who have pro-medical care orientations" (p. 255). One must keep in mind that it is difficult to distinguish whether the symptom onset comes before a decline in social functioning or whether changes in social functioning cause symptomatology (Turnbull et al. 1990). The present results appear to support Cohen and Syme's (1985)

perspective. Not only are contact frequency and satisfaction with support negatively related to symptomatology, but they positively relate to the hospitalization measure which is also positively related to education, family income, and financial adequacy perceptions. Social support was not statistically related to any of the SES variables considered for the hospitalized H3 model. The lack of a significant relationship between education and contact frequency fails to support the results of Vega et al. (1991) that those with higher educations had more contact with their support networks. However, these results mirror the previously reported symptomatology results. The lack of a relationship between contact and income is also consistent with the symptomatology model results.

According to the hospitalization H3 data, financial adequacy perceptions are marginally related to both measures of social support. The failure to reach significance in these cases is likely due to the small effect size and corresponding lack of power. According to Cohen (1988), correlation coefficients of .10 were small, effects of .30 to .50 were moderate and large effects were greater than .50. According to Richard, Bond, & Stokes-Zoota (2003), .10 is small, .20 is medium, .30 is large. These relationships in the full cross-sectional sample ($\underline{N}$ = 232) results were significant for both CF and SAT with FAD for Time-1, -2, -3, and -4. However, for the H3 model, financial adequacy appears not to be related to either education or employment (ED, $\underline{r}_s$ = .083, $\underline{p}$ = .474/2; EMP, $\underline{r}_s$ = -.043, $\underline{p}$ = .709/2). This result is not congruent with most of the cross-sectional symptomatology model effects. In the symptomatology model there were primarily direct and indirect positive effects between employment and financial adequacy. These effects were relatively stable at least for Time-1, -2, and -3 models. The cross-section models also

demonstrated a mixed effect of education on financial adequacy. There are a number of positive indirect effects and two models with negative direct effects. The failure to obtain a significant effect for the H3 sample is not likely due to a lack of power. The lack of effect in the H3 sample could be due to the limited variability of the employment variable in the hospitalized sample ($\underline{N}$ = 78, $\underline{M}$ = 25.1, $\underline{SD}$ = 29.3). Most of the hospitalized clients had no employment during the year preceding the survey. Family income and financial adequacy were positively related in the H3 sample ($\underline{N}$ = 78, $\underline{r}_s$ = .487, $\underline{p}$ < .0001/2) mirroring the symptomatology models. Clearly, those who perceive their financial situation as more positive are likely to come from families with higher family incomes. Financial adequacy was significantly inversely related to symptoms for both the H3 model and most of the symptomatology models. Financial adequacy was not significantly related to LOS for the H3 model ($\underline{N}$ = 78, $\underline{r}_s$ = .134, $\underline{p}$ = .250/2). The Spearman Rank Order correlation was small in magnitude and was in the opposite direction to the direction hypothesized. This may be due to the positive correlation between family income (which positively influences perception of financial adequacy) and hospitalization.

According to Cohen and Sokolovsky (1978), psychiatric clients are often withdrawn and isolated. However, they do not believe that LOS was likely to have caused this withdrawal. According to Spohn (1992), longer stays in psychiatric facilities are correlated with poor pre-morbid functioning, a more chronic course, and less outside support. Clients who are repeatedly admitted to psychiatric facilities appear to suffer from economic problems (Munves et al. 1983). According to Allen et al. (1992) and Westermeyer, and Harrow (1988) employment, hospitalisation, and psychopathology are

interrelated.

According to Aneshensel, Frerichs, and Clark (1981), employment and SES are related to each other, and they are both related to inpatient treatment with many of the mentally ill in lower SES groups. According to Vetter and Koller (1996), hospitalization is negatively related to employment. Employment is often used as an indication of social functioning and contributes not only to family income but to perceptions of financial adequacy. Economic conditions were also found to contribute to extended stays (Mai et al. 1993). For this component of the research, the relationship between family income and hospitalization was and marginally significant using a one-tailed t-test, but was in the opposite direction hypothesized. This finding needs replication before conclusions can be drawn about the direction of this small effect. Apparently, for these participants, individuals with more financial resources were admitted to inpatient treatment facilities for longer periods. Possibly the trend away from psychiatric hospitalization because of scarce resources is influencing the relationship between family income and psychiatric stays. These results are opposite to results of a recent Manitoba study that found the seriously mentally ill congregated in neighbourhoods with low average household incomes and experienced longer hospital stays than those living in higher income neighbourhoods (Tataryn et al. 1994). The measure of income in this study was each client's family income in 1994 and is not directly comparable to the average neighbourhood income measure used in the Tataryn et al. (1994) findings. Tataryn et al. also evaluated a different population (included schizophrenics with other psychotic disorders) than the present research which focussed only on unipolar and bipolar depressives.

Most research finds an inverse relationship between socioeconomic status and psychopathology (Kessler, 1982) and socioeconomic status has been found to inversely influence length of psychiatric hospital stays (Mai et al. 1993). The current results indicate that employment and LOS are inversely related as expected (see Table 39). For the H3 model education was also positively correlated with length of stays, employment score, and family income as predicted and these findings are congruent with the symptomatology models. It appears that those with more financial stability (at least in this self-help client sample) may now be retained longer in increasingly scarce inpatient treatment beds. According to Anthony (1980) and Shapiro (1983), educational attainment has been found to be only slightly related to hospitalization. Other researchers report that persons with higher income and education have been found to make use of psychiatric services more frequently (Leaf et al. 1985). These results appear to imply that those from lower socioeconomic groups bare the burden of mental illness and receive the least care (Hollingshead & Redlich, 1958).

Hollingshead and Redlich (1958), in their ground-breaking research, discovered that there were distinct differences in social class and the response to mental illness. These differences included: a) a bias in the prevalence of treated mental illness based on class structure, b) a difference in diagnostic categories over differing class structures, and c) treatment variations for different classes. For example, there were clear differences in which class of individuals received which type of treatment. Higher class individuals received most of their care from private care physicians (since they were able to absorb the financial costs) while lower class individuals were more likely to receive care from public

and state run facilities (where the cost to the patient would be minimal). There was also a negative association between class and being a psychiatric patient; the lower the class the greater the proportion of patients. Class and diagnostic category displayed significant relationships. Lower class individuals were more likely to be labelled psychotic while higher class individuals received neurotic diagnoses more frequently. Higher class patients were also more expensive to treat. According to Hollingshead and Redlich (1958), these differences are in part due to class differences between the treating personnel (psychiatrists) and the client. Lower class individuals were seen to benefit less (be less treatable) from intervention that included being seen by a psychiatrist and receiving some form of 'insight therapy'.

The current results could also be due to family and friends with more educational and economic resources being more able to mobilize and make a plea the client's case with admitting personnel. Individuals with more contact with a support network made up of "higher-class" individuals seem to have more educational and economic resources and it may be easier for admitting personnel (psychiatrists and doctors) to identify with the client and family members in keeping with Hollingshead and Redlich's (1958) findings that psychiatrists were more likely to identify higher-class patients as more treatable (better patients).

Molica and Milic (1986), in a follow-up of Hollingshead and Redlich's (1958) work, found that the model proposed by Hollingshead and Redlich needed revisions. Their follow-up study indicated that social class was not the only variable that influenced the treatment of mental illness. Diagnosis, employment history, and gender contributed to the

types of treatment offered lower-class patients. Females from lower classes were more likely to be assigned to units in facilities that offered psychotherapeutic treatments. Psychotherapy was also more available to lower class individuals with psychoneurotic diagnoses. The degree of successful employment also appears to positively predict the likelihood of the client being assigned psychotherapy as treatment. This follow-up considered lower-class patients only for the determination of treatment assignment and found a more symptom/diagnosis based approach to assigning treatment. Drug and alcohol-addicted patients were excluded from psychotherapy and were treated in more categorical unit settings specializing in each illness.

Ideally, psychiatric treatment should be determined on a symptom/diagnosis basis rather than on socioeconomic variables, like class membership (keeping in mind that the symptom measure was taken after their release from hospital). When one considers the severity of self-reported symptomatology one would expect that the individuals reporting a history of psychiatric hospitalization would report more severe symptomatology than those individuals reporting no history of psychiatric hospitalization. This is not the case with these data. The current results indicate that those with a psychiatric hospitalization history did not report more severe psychiatric symptomatology than those with no psychiatric hospitalization history. In fact, self-reported psychiatric symptomatology, for the two weeks prior to the receiving the survey, was marginally higher in those without a psychiatric hospitalization. The apparent reduced level of symptomatology in the hospitalized group could also be due to the treatment effect of the psychiatric hospital stay, bringing symptomatology of the hospitalized group more in line with individuals who are

not hospitalized for psychiatric symptomatology. It may also be possible that the inpatient treatment acted to reduce the stressors that can trigger symptomatology,

Psychiatric symptomatology was found to be predicted by employment, but not education. Financial adequacy was also related to symptomatology in the predicted inverse direction. This result is congruent with the symptomatology model. The relationship between family income and symptomatology failed to reach significance in the hospitalization model (deleted due to a large magnitude error in the symptomatology model). As stated previously, psychiatric patients are often unemployed, and unemployment and LOS are positively related. There is no ambiguity about this relationship in the literature. The problem resides in the causal attribution attached to the findings. There are two difficulties related to explanations of this relationships. The first explanation considers the fact that unemployment and increased LOSs can both be an indication of the severity of pathology. The second explanation is related to direction of causation. Did the unemployment cause the inpatient stay to increase or did the inpatient stay create the deficit in employment? Regardless of direction of causation, many mentally ill patients have large gaps in their employment histories that they need to explain to prospective employers, making it very difficult for them to find future employment.

The relationship between psychiatric symptomatology and three year hospital stays was positive and significant at alpha .05, as predicted but was of a small effect magnitude (Cohen et al. 1983). However, it was of the same magnitude as the effects of other predictor variables considered in this research. Given the small effect magnitude, these results support the research findings of Keitner et al. (1992), Norquist (1988), and Mattes

(1987) that symptomatology and diagnostic related groups are poor predictors of resource consumption. This symptomatology effect on hospitalization is of the same magnitude as satisfaction with support, contact frequency, education, employment, and family income. It appears as though all these social functioning variables play a role in the length of psychiatric hospital stays in this population. Some of these correlations failed to reach significance since the sample size was restricted. However, the magnitudes of the correlations are large enough to warrant further investigation with larger samples. Since age, sex and marital status were dropped from further consideration because of the results of the partial correlations, there is more evidence supporting Gordon et al's. (1985) findings that these variables do not by themselves significantly improve the prediction of resource consumption.

The relationships among social support, social functioning, and LOS are difficult to disentangle. The direction of causation cannot be determined unequivocally with this approach at this time. Longitudinal research with adequate sample sizes will be necessary to answer this question definitively.

Hospitalization Lagged Spearman Correlational

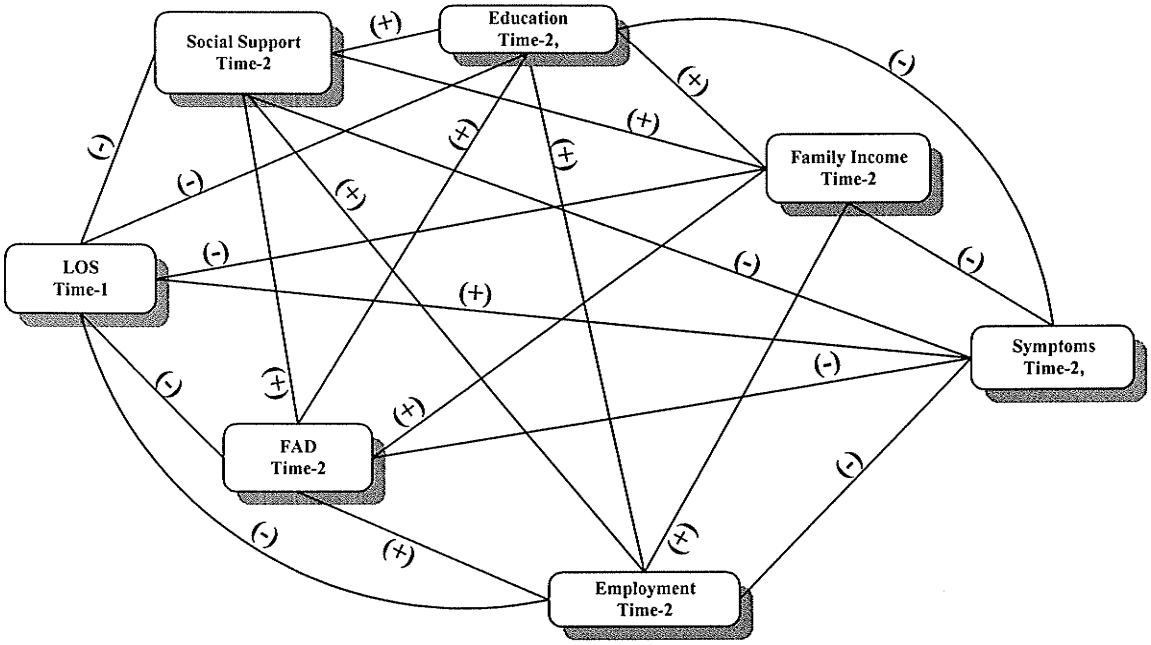
Results and Discussion

Respondents with a non-zero three-year hospitalization history were used to evaluate the influence of the inpatient LOSs at Time-1 upon later symptomatology, social support, and social functioning variables at Time-2. Of the participants at Time-1 there were 43 who indicated that they had a non-zero three year LOS, who continued to participate at Time-2, and whose results were useable. The other direction of causation tested in Phase III of this research could not be carried out in this phase since there were too few participants who indicated that they had a three year stay LOS and there were very few who were admitted during the study time-frame.

All possible pair-wise scatter diagrams indicated that the assumption of linearity was tenable. Like the previous assessment of the inpatient treatment variable, the hospitalization and employment variables in this smaller hospitalized sample were neither distributed normally, nor were the sample size large enough to assume the variables came from a normal distribution. Therefore, the path analysis techniques used on the symptomatology sample were not appropriate and Spearman correlation coefficients were used instead of parametric correlation coefficients (see Table 40). Therefore, what follows is only descriptive in nature.

Although the entire path analysis procedure was not performed on this smaller hospitalized sample ($\underline{N}_{H3} = 43$), a preliminary **hypothetical time-lagged** hospitalization model is provided in Figure 20. The literature supporting the hypothetical valence patterns is the same as for the other hospitalization model. Primary hypotheses for this component of the research would predict that LOS at one point in time would be inversely related to social support and social functioning at a later point in time.

Figure 20. Hospitalization hypotheses cross-lagged Spearman Rank Correlation coefficients Social Selection theoretical position LOS Time-1 and Social Functioning at Time-2. N= 43.



The results for this hypothetical model can be found on Figure 21. This model uses the zero-order Spearman Rank Order Correlations ($\underline{N} = 43$, with their corresponding probability levels) between the variables of interest to represent the total effect of variable pairs as hypothesized in Figure 20. This figure is NOT a path diagram as there are no arrows indicated, nor am I using path analytic procedures. The drawing is an attempt at looking at the only valid data that were available using the zero-order Spearman Correlation coefficients. There was inadequate power to evaluate many of the effects. In an attempt to control for the other pathology variable, symptomatology at Time-1, comparisons were made between Spearman (Table 40) and Pearson correlations (zero-order and partial). Results indicated that none of the effects generated from these correlation approaches vary significantly from one another (see Appendix K Table K1). These results make it clear that the correlational patterns remain stable whether one is looking at the Spearman lagged results, the Pearson lagged results, or the Partial Lagged results controlling for Time-1 symptomatology (see Table K2 in Appendix K). Direction of correlations and magnitudes are similar in all three cases.

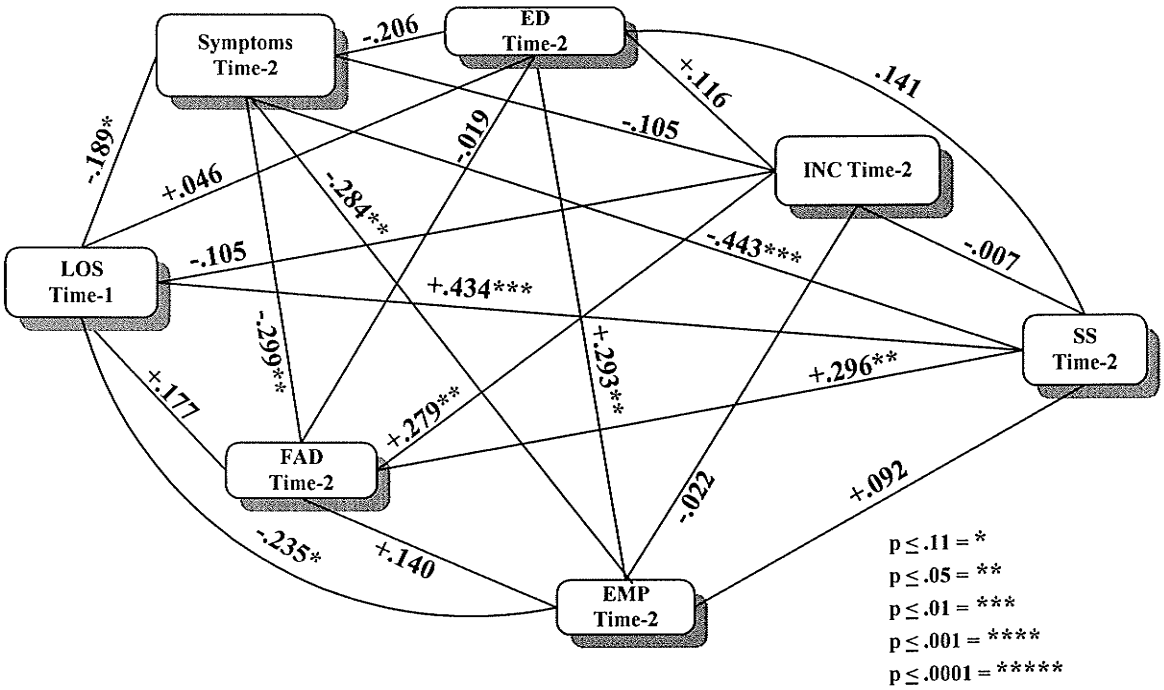
Table 40.

Cross-Lagged Spearman Rank Order Correlation Coefficients (LOS Time-1, SS, and SF Time-2) for the Respondents with a LOS During the Last Three Years and Participated at Time-2, N = 43, one-tailed p-values.

	LOS Time-1	SYMP Time-2	FAD Time-2	ED Time-2	EMP Time-2	INC Time-2	SS Time-2
LOS Time-1	- -	r_s = -.189 p = .1130	r_s = .177 p = .1126	r_s = .046 p = .3860	r_s = -.235 p = .0650	r_s = .065 p = .3405	r_s = .434 p = .0020
SYMP Time-2		- -	r_s = - .299 p = .0251	r_s = -.206 p = .0920	r_s = - .284 p = .0325	r_s = - .105 p = .2510	r_s = -.443 p = .0015
FAD Time-2			- -	r_s = -.019 p = .4530	r_s = .140 p = .2545	r_s = .279 p = .0350	r_s = .296 p = .0270
ED Time-2				- -	r_s = .293 p = .0285	r_s = .116 p = .2300	r_s = .141 p = .1842
EMP Time-2					- -	r_s = -.022 p = .4780	r_s = .092 p = .2780
INC Time-2						- -	r_s = -.007 p = .4825
SS Time-2							- -

Note: Abbreviations are as follows: r_s = Spearman Correlation Coefficient, p = one-tailed p-values, SS = compound social support, ED = years of education, EMP = employment score, INC = family income, FAD = financial adequacy score, SYMP = symptomatology score, and Time-1 LOS = number of nights in an inpatient psychiatric facility during the last three years as at Time-1.

Figure 21. Hospitalized tentative cross-lagged Social Selection theoretical position **Spearman Correlation coefficients** (Time-1 LOS, and SF Time-2) for the Respondents with a LOS During the Last Three Years and Participated at Time-2, N = 43.



Although a path-like diagram has been furnished for these lagged results, the reader is reminded that the coefficients on the path model are Spearman Rank Order Correlations and not regression beta weights as in the symptomatology model. As a result, one-to-one or direct comparisons are not appropriate between the two models. Also, the Spearman Rank Correlation Coefficients in Figure 21 represent the total effect of one variable rank upon another in standardized scores. Therefore, they cannot be decomposed into the direct and indirect effects that are appropriate for the symptomatology model. Some of these correlations failed to reach significance since the sample size was restricted. However, the magnitudes of the correlations are large enough to warrant further investigation with larger samples. Many of the effects in Table 40 are not significant or salient in magnitude and are not discussed below.

Longer stays in psychiatric facilities have been found to correlate with poor pre-morbid functioning, more chronic course, and less outside social support (Spohn, 1992). For this time-lagged model, LOS Time-1 and SS Time-2 are related ($\underline{N} = 43$, $\underline{r}_s = .434$, $\underline{p} = .004/2$). However, they were not related in a negative manner as predicted. It seems that increased LOSs at Time-1 are related to increased social support at a later point in time. These findings may be due to the nature of the population being assessed. The respondents in this research, unlike the hospitalized clients in the pretest sample, were already members of a self-help group. Clients who participate in such groups are different from other individuals who have the same psychiatric diagnoses (Gidron, Guterman, & Hartman, 1990). It is possible that individuals who belong to self-help groups are more active help-seekers, and are supported in seeking help by their networks. For further

explanation the reader is referred to the earlier hospitalization results.

Psychiatric Symptomatology (Time-2) and social support (Time-2) are also negatively related in this group as in the previous hospitalization results ($N = 43$, $r_s = -.443$, $p < .003/2$). This effect appears to be relatively stable in all of the findings and demonstrates a moderate correlation magnitude (Cohen & Cohen, 1983). This effect is of the largest magnitude when one considers all of the correlations involved on Table 40. Social support and education ($N = 43$, $r_s = .141$, $p = .368/2$) were not significantly related to one another unlike in the previous hospitalization results which does not support the Vega's et al. (1991) findings. SS and FAD were marginally related to each other ($N = 43$, $r_s = .296$, $p = .054/2$). Neither employment nor household income were related to Social Support ($N = 43$, employment, $r_s = .092$, $p = .556/2$; income, $r_s = .007$, $p = .965/2$) which mirrors the cross-sectional hospitalization model results.

Education and symptomatology were predicted to be inversely related. These results demonstrate that there is not a significant relationship between ED and SYMP ($N = 43$, $r_s = -.206$, $p = .184/2$). But, this could be due to lack of power. Income and Symptomatology were not significantly related in this model ($N = 43$, $r_s = -.105$, $p = .502/2$) unlike in the hospitalization cross-sectional model. EMP and SYMP were significantly and inversely related to each other as predicted ($N = 43$, $r_s = -.284$, $p = .065/2$). This finding is consistent with the previous hospitalization results. One's capacity for employment, in this very tentative model, appears to be related to current symptomatology. Financial adequacy at Time-2, like Time-1, was marginally negatively correlated with symptomatology ($N = 43$, $r_s = -.299$, $p = .057/2$). This result is also

congruent with the cross-sectional model.

Employment and FAD were not significantly related ($N = 43$, $r_s = .140$, $p = .509/2$). This finding is congruent with the cross-sectional hospitalization results. This effect would probably have been greater if not for the limited variability of the employment variable, since most of the hospitalized clients had no employment during the year preceding the survey. ED and FAD were not significantly related as in the cross-sectional hospitalization model ($N = 43$, $r_s = -.019$, $p = .906/2$). Family income and FAD are positively related in this cross-lagged model ($N = 43$, $r_s = .279$, $p = .070/2$), as would be predicted and this is concurrent with results from the hospitalization cross-sectional model. Those who perceive their financial situation as more positive are likely to come from families with higher family incomes.

According to Munves et al. (1983), clients who are repeatedly admitted to psychiatric facilities appear to suffer from economic problems. For this sample Education and Employment were marginally related, in a predictable manner ($N = 43$, $r_s = .293$, $p = .057/2$), as in the full cross-sectional hospitalization results. LOS Time-1 was not significantly related to Education ($N = 43$, $r_s = -.046$, $p = .722/2$) at Time-2 like the cross-sectional hospitalization results. Education and Income ($N = 43$, $r_s = .116$, $p = .460/2$) also appear not to be significantly related unlike in the cross-sectional hospitalization model. This result should be considered in light of psychiatric symptomatology interrupting academic progress. However, employment and income were not significantly related ($N = 43$, $r_s = -.022$, $p = .889/2$) in this cross-lagged results unlike the full cross-sectional hospitalization model. This effect is not likely due to a loss of

power in this case since the effect is near zero. This may have been impacted by all of the zero scores on the employment variable. It also makes sense that if one is an inpatient one is likely to have lost some weeks of employment when employment is assessed at a later point in time. For this time-lagged model financial adequacy was not significantly related to LOS ($N = 43$, $r_s = .177$, $p = .256/2$). This effect mirrors the same effect in the cross-sectional hospitalization model.

As discussed previously, keeping in mind the point when symptomatology was assessed, the relationship between psychiatric symptomatology at Time-2 and three year LOS at Time-1 was not significant ($N = 43$, $r_s = -.189$, $p = .226/2$). One would expect that those with longer LOSs would have higher symptomatology scores and more serious psychiatric disorders. These results indicate that longer LOSs were inversely related to symptomatology at a later point in time. Time-1 LOS appears to be a poor predictor of subsequent symptomatology at least for this rather small sample.

According to Mai et al. (1993), economic conditions were also found to contribute to extended LOSs. The current results indicate that LOS Time-1 was not significantly related to Time-2 income ($N = 43$, $r_s = .065$, $p = .681/2$), or employment ($N = 43$, $r_s = -.235$, $p = .130/2$), not the same as the non-lagged model). Therefore, it appears that the relationship between LOS and income may be non-existent, which does not agree with previous findings of an inverse relationship between household income and LOS (Tataryn et al. 1994). This effect approached significance in the cross-sectional results so the two variables may be related, but not in a causal manner. This finding is also contrary to results from North American populations. Most research finds an inverse relationship between

socioeconomic status and psychopathology (Kessler, 1982), and socioeconomic status has been found to inversely influence length of psychiatric hospital stays (Mai et al. 1993). Possibly the trend away from psychiatric hospitalization because of scarce resources is influencing the relationship between family income and psychiatric stays.

In summary, according to the time-lagged Spearman Rank Correlations it appears that LOS did not impact the majority of the social functioning variables involved at a later point in time. However, LOS impacted social support in a positive manner, contrary to the hypothesized direction. This result was consistent with the previous finding that support and LOS are positively related. These results fail to support the social Selection Theoretical Model of causation that a pathology variable (LOS) at Time-1 impacts social functioning (FAD, ED, EMP, and INC) and social support (SS) at Time-2. These results must be considered tentatively for a number of reasons. They cannot be replicated at the present. Therefore replication is necessary in the future. Sample sizes are very small and as a result findings can be unstable. The sample needs to be larger for the formal path analytic models to be tested adequately.

CHAPTER 7:

"No one does more damage than the man

that is sure he is right!"

Anonymous

Conclusions, Limitations, and Implications

This chapter opens with a summary of the key findings related to each phase of the research. Subsequent sections pertain to contributions to the field, limitations of the research, and suggestions for future research. Finally, this chapter closes with a summary of possible policy implications.

Key Findings and Implications

Phase II Key Findings. In the Phase II data analysis, the multidimensional nature of the social support construct was confirmed. Both of the social support measures used correlated to most of the endogenous variables and to the symptomatology outcome variable as predicted. These support measures, while different from one another, tend to be significantly related to each other. According to Avlund et al. (2004) and Uchino, (2004), there is a distinction between functional and structural support. The structural and functional measures of social support focus on different aspects of the support construct and the components are not mutually exclusive or assessing completely different entities (Patrick et al. 1986). Factor analysis confirmed that these two measures could be considered as part of the same overall factor. This confirms the appropriateness of combining the two measures into one social support measure for the cross-lagged models.

In almost all cases support was related to social functioning and psychiatric symptomatology as predicted for all four cross-sectional models. Higher contact with one's network and stronger perceptions of support are both directly and indirectly inversely related to psychiatric symptomatology. These findings are congruent with those reported by Cohen, Gottlieb, and Underwood (2000), that lack of contact may produce distress and symptomatology. It appears that both of the measures used to evaluate social support are important for the mental health of these participants with depressive disorders. There was no evidence that larger social networks or more contact were related to increased stress or feelings of being overprotected. These results do not support Boutin-Foster's (2005) findings that some with higher contact feeling overprotected and contributed to the stress they experienced.

Regardless of the measure (structural count, or functional satisfaction) used to assess social support and pathology the results concur. Therefore, empirical results using these various measures tend to produce converging results despite the differing measures employed (Olsen, 1993). Replicating models lends support for the veridical nature of the relationships involved. According to Fiore, Coppel, Becker, and Cox (1986), it is important to know which component of the social support construct influences well-being for theoretical and clinical reasons. The results of this phase of the research clearly indicate that both contact frequency and satisfaction with support are important for emotional health. These results support Haughton Mc Neill, Kreuter, and Subramanian's (2006) findings that emotional health improves with higher social status (Institute of Medicine, 2003; Lynch & Kaplan, 2000).

These results support the findings of a number of theorists. Larger networks have been found to be related to better health status and well-being (Allen, Ciambrome, & Welch, (2000); Anfinson & Kathol, 1992; Baker, Jodrey, & Intagliata, 1992; Berkman & Breslow, 1983; Berkman & Syme, 1979; Earls & Nelson, 1988; Haughton Mc Neill, Kreuter, and Subramanian (2006), Lynch & Kaplan, 2000; Nelson et al. 1999; Nelson, Hall, & Walsh-Bowers, 1995; Williams, Ware, & Donald, 1981). According to Sherbourne (1988), structural social support variables are indicative of the availability or adequacy of social support (Allen, Ciambrome, & Welch, 2000; Peirce, Frone, Russell, Cooper, & Mudar, 2000). Network contact is necessary for social support to occur (Vega et al. 1991) and contact frequency is an indication of the accessibility of social support in a persons' support network (Cutrona, 1986). The structural measure of support has also found to be related to social support perception (Peirce, Frone, Russell, Cooper, & Mudar, 2000). According to Allen, Ciambrome, and Welch (2000); and Fiore et al. (1986), individuals with more contact with their support network tend to ask for support more often and perceive that support is more available. According to Cutrona (1986), "frequent contact increases the speed with which an individual's needs for support would be communicated to others and would increase the probability that a supportive individual would be present to offer help when needs arise" (p. 349). Those with more social support may seek treatment for psychosis more promptly (Drake et al. 2000; Kalla et al. 2002; Malla et al. 2004). According to Hanson and Ostergren (1987), and Marsden (1990), contact frequency measures are more reliable and stable than assessing the number of persons in a person's social network. However, there is a close relationship between the

number of individuals in a persons network and contact frequency (Hanson & Ostergren, 1987).

Various population studies have shown the absence or perceived absence of social supports, infrequent social contact, and changes in a person's network of support are directly related to higher rates of general pathology (Kessler & McLeod, 1985), emotional distress (Revicki & Mitchell, 1990), and depressive symptomatology (Cohen, 2004; Reis, 1988; Schulz, Tompkins, Wood, & Decker, 1987). Individuals with serious depressive conditions usually have social support deficits, fewer friends (Bebbington et al. 1988; Billings & Moos, 1985; Billings et al. 1983; Birtchnell, 1971; Black et al. 1991; Brugha et al. 1990; Brugha et al. 1987; Brugha et al. 1982; Cornelis, Ameling, & de Jonghe, 1989; Croughan et al. 1988; George et al. 1989; Swindle, Cronkite, & Moos, 1989), and are less sociable (Barnett & Gotlib, 1988). Social support may come from a variety of sources and support available from all sources should be considered when evaluating an individual's social support resource availability (Dean et al. 1981; Dean et al.1990; House & Kahn, 1985; Lin et al. 1986).

Depressed individuals often feel alone (Amann, 1991; Eisemann, 1984; Lowenstein & Rosen, 1989). Social isolation has been found to be related to psychiatric symptomatology (Wilkinson, 1975). Social isolation has been found to be another risk factor for mental illness (Long, Ickovics, Gill, & Horwitz, 2001). Individuals with depressive disorders have been found to have smaller social support networks and also tend to be less satisfied with their social support (Billings & Moos, 1985). According to Henderson (1998), some believe that it is the perception rather than the quantity of support

that is more important for emotional health. The current findings demonstrate that it is not an either/or situation. Both components of support were important and related to psychiatric pathology. These results also concurred with the research by Brugha et al. (1990) and George et al. (1989) who report that patients have a more positive prognosis if they are satisfied with their social support. According to Allen, Ciambrome, and Welch, (2000); and McKee-Ryan, Song, Wanberg, and Kinicki, (2005), greater perception of social support has been found to have a positive effect on mental health. The current results support these findings.

What is also clear is that the subjective component of SES (Financial Adequacy Perception) is important for all of the cross-sectional models. In almost all of these models Financial Adequacy Perception was inversely related to Symptomatology. The current results indicate that the objective components of SES (education, employment, and household income) were less consistent in their impact upon Symptomatology at the same point in time. The lagged models moved financial adequacy perception to earlier in the models and the impact of SES variables was assessed after the FAD variable.

Phase III Key Findings. The primary focus of this phase of the research was to determine which of two diametrically opposed theoretical positions (Social Causation verses Social Selection) best explained the relationship between psychiatric pathology, social support, and social functioning (SES). According to Merton (1957), the Social Causation Model or thesis proposes that environmental circumstances create hardships and distress and this financial hardship and related distress leads to increased psychiatric

pathology (Bebbington, Christopher, & Hurry, 1981; Brown & Harris, 1978; Faris, 1934; Faris & Dunham, 1939; Hollingshead, 1957). These environmental theorists propose that environmental stress explains the inverse relationship between psychiatric morbidity and SES.

The Social Selection theory or antithesis, with its roots in genetic theories, proposes that SES and pathology are related because persons with psychiatric disabilities decline in SES as their pathology intensifies, resulting in a patient drifting down (Social Drift Theory) to lower SES levels (see Bland & Orn, 1981; Dohrenwend, 1998; Dunham, 1965; Eaton, 1980; Grunefeld & Salveson, 1968; Hafner, Nowotny, & Loffler, 1995; Jablensky, 1986; Jarvis, 1971; Munk-Jorgensen & Mortensen, 1992; Wender et al. 1973; Wiersma et al. 1983). These two theoretical positions are both supported by the literature, but tests of which theory is 'correct' and/or more important have been difficult. The most significant difficulty related to this research has been designing a research paradigm in which the two theories can be evaluated and compared for relative importance (Dohrenwend et al. 1998). There is also little information about the ideal length of follow-up for a panel design for a prospective outcome study relating social support to outcome (Brugha et al. 1997).

According to the literature, regardless of the operational definition of SES, the type or prevalence rate measure, or the case identification instrument, an inverse relationship between pathology and SES exists and in cases of major depression a near linear relationship exists between SES and pathology (Kohn et al. 1998). What is clear from the current theoretical models is that there are indications that Financial Adequacy Perceptions

do impact the outcome variable whether it is symptomatology or social support in the predicted manner. Financial Adequacy is also related to the SES variables in the predicted manner. What is also clear is that the SES variables (ED, EMP, and INC) do not impact the outcome variables consistently in the predicted manner. This is contrary to much of the published literature (see Leaf et al. 1985; Levav & Arnon, 1976; Nuehring et al. 1980; Ross & Huber, 1985; Zubenko et al. 1994). However there are some conflicting results in the literature (see Anthony 1980; Hull et al. 1996; Buell & Anthony, 1975; Kirk, 1976; Shapiro, 1983; Woogh, 1986; Zohar, Hadas, & Modan, 1987). The reasons for the current findings are not clear at the present time and need further study.

Phase IV Key Findings. Phase IV of this research evaluated how the primary variables related to Length of inpatient stay (LOS). The range of time a patient needs to be ill to qualify as an new long stay patient ranges from several months up to two years (Richards et al.1997). According to Hoffmann and Mastrianni (1993), long hospital stays can lead to stagnation of social skills and leave patients impaired in their capacity to fulfill role requirements. Extended LOSs has also been found to diminish employment skills (Freeman, 1980). These changes can result in lower SES for patients with extended LOSs (Aneshensel et al. 1981). However, according to Turnbull, George, and Landerman (1990), it is impossible to tell whether the symptom onset comes before a decline in social functioning or whether social functioning can be said to have caused symptomatology. Previous phases of this research clearly indicate that psychiatric symptoms were negatively impacted by low levels of social support and that low levels of social support could

negatively impact psychiatric symptomatology.

One of the key findings of this phase of the research demonstrated that Social Support at Time-2 was positively related to LOS (Time-1) which was the opposite of expectations. This outcome does not support the work by Turnbull, George, and Landerman (1990) that it is impossible to determine whether symptoms preceded a decline in social functioning in hospitalized patients or visa versa. The LOS Model that lagged the Social Support to a later point in time clearly demonstrates that Time-1 LOS is positively related to Time-2 Social Support. The longer the patient had been hospitalized the greater their social support. It seems evident that extended LOSs, at least in this sample, tend to martial social support resources. This finding will need to be replicated in a much larger sample since the current model had only 49 participants.

In the Hospitalization Model, the effect of family income on LOS was also the opposite of predictions and is not in agreement with published findings (Mai et al. 1993). Education was also positively related to LOS for this sample contrary to predictions These findings would be interesting once they have been replicated as it appears that those from higher SES, more financially stable, backgrounds may be spending more time in inpatient care that those from lower SES backgrounds. With the present data one can only speculate why this was the case. Are those from more affluent backgrounds gaining access/extended access to the more expensive treatment options under conditions of short supply and rationing? When one considers the lagged LOS Model the relationship between LOS Time-1 and Family Income Time-2 is not significant.

Each of the primary variables in the Hospitalization Model explained about 3.0%

to 4.0% of variability in LOS. This amount of variability is small; but it is important that all of the variables are approximately equally strong in predicting LOS. It is interesting that psychiatric symptomatology did not account for more than approximately 4.0% of inpatient LOS, which is the same variability predicted by the Social Functioning, SES, or social support variables. Why is the contribution of Symptomatology so small in magnitude? All these social support and social functioning variables play a role in LOS. Given these results it would be helpful to examine these relationships in larger and differing samples.

When one is comparing individuals with a hospitalization history with those without such a history, one would expect individuals with an inpatient treatment history to be sicker than those without such a history. This proved not to be the case with this sample. There was no statistical difference in symptom severity. This may be due to either treatment success at ameliorating symptomatology. It is possible that patients who were admitted for treatment had more severe symptomatology at their admission time. If this is a veridical finding, why are some admitted for inpatient care while other patients avoid inpatient treatment?

To summarize, the multidimensional nature of the social support construct was confirmed. Different aspects of the support construct are not mutually exclusive or assessing completely different entities. However, both of the measures used to evaluate social support are important for those with depressive disorders. Higher contact with one's network and stronger perceptions of support are both directly and indirectly inversely related to psychiatric symptomatology. These results are congruent with the findings

reported by Allen, Ciambrome, and Welch, (2000); McKee-Ryan, Song, Wanberg, and Kinicki, (2005). These authors report that perception of social support has been found to have a positive effect on mental health. Replicating models lends support for the converging veridical nature of the relationships involved. Results converged as both perception and quantities of support were important for mental health for these participants. Time-lagged model results support the Social Causation/thesis, the Social Selection/antithesis, and a Reciprocal Causation Model/synthesis. The hospitalization LOS data demonstrated that each of the primary variables explained approximately 3.0% to 4.0% of variability in LOS. There is also some evidence of similarity in symptomatology between those who are and are not admitted for inpatient treatment. This result begs the question 'why are some hospitalized and others not?' A comprehensive needs assessment is necessary if a coordinated continuum of community resources is to be developed to meet the needs of the entire range of patients with chronic psychiatric difficulties (Holley, Jeffers, & Hodges, 1997). According to Swanson et al. (1997), the risk that psychiatric patients will be violent towards others is increased by a lack of community resources and deficient contact with mental health professionals (Douglas, & Koch, 2001)

Contribution to the Field

This research provides an approach to evaluating two diametrically opposed theoretical approaches that seek to explain the relationship between social support and psychiatric morbidity. The Social Causation Model proposes that a patients' social environment impacts a patients' psychiatric morbidity. For example, a poor environment

with little social support acts by increasing psychiatric symptomatology. The Social Selection Model proposes that psychiatric morbidity does damage to a patients' social environment. For example, high levels of psychiatric symptomatology impact social support in a negative manner. Testing these two theories has proven to be difficult due to design and methodological limitations. According to Dohrenwend et al. (1998), both processes are likely occurring in their research. The Social Selection process appeared to be more important for individuals with schizophrenia while the Social Causation process appeared more important for those with depression. This research was designed specifically to test these two theoretical models and to determine if a theoretical reciprocal causation model was supported. These findings help explain the support for the diametrically opposed results that plague the social support literature.

When one considers the Social Causation results it is clear that Social Support at Time-1 and Symptomatology at times -2, -3, and -4 are related in a consistent negative manner. It is also unequivocal that Financial Adequacy at Time-1 is consistently negatively related to Symptomatology at times-2, -3, and -4. The relationships between education, employment, and household income with symptomatology are not consistent for this model. Either these paths were not significant or were eliminated due to large errors. Given these results it is clear that the SES variables, at Time-1, by themselves do not predict Symptomatology at a later point in time. So, this component of the Social Causation Model fails to explain the findings. However, the effects of the Financial Adequacy relationships are tied to the SES variables lending indirect support for the SES variables. These results support the environmentalist theorists like Merton (1957; see also

Berkman & Syme, 1979; Brown, Bhrolchain, & Harris, 1975; Cohen & Wills, 1985; Ennis et al. 2000; Reis, 1988; Seeman & Syme, 1987; Turner, 1981). These results also partially support the findings of Hobfoll et al. (2003), McLeod, Lavis, Mustard, and Stoddart (2003), that financial deprivation had an independent negative impact on psychological well-being.

When considering the Social Selection Model results it is unequivocal that symptoms at Time-1 are related in a consistently negative manner to social support at times -2, -3, and -4. Once again, there is also a consistent significant negative relationship between symptomatology at Time-1 with financial adequacy perceptions at times -2, -3, and -4. The relationships of education and employment with symptomatology were also not consistent or were not significant. The paths connecting symptomatology at Time-1 and Income at times-2, -3, and -4 are consistent. They are negative, but the effect is only an indirect one, which means that it is being mediated by financial adequacy, education, and employment. Once again the impacts of symptomatology on the SES variables are not all as predicted which fails to support most of the SES component of the Social Selection Theory.

Current results confirm that the Social Causation Model process explained more variance for these participants with depressive disorders. Chi Squared indices for the Null Models (Social Causation Model, $\chi^2_{(1)} = 117.62$; Social Selection Model, $\chi^2_{(1)} = 59.94$) indicate that there is more variability to be explained by the Social Causation Model. When explanatory models were fit to the data it became clear that these models reduced the fit indices from the null models and reduced the variability to acceptable non

significant levels (Social Causation Model, $\chi^2_{(2)} = 1.461$, $p_{(\text{less than } 2)} = .4809$; Social Selection Model, $\chi^2_{(4)} = 1.9361$; $p_{(\text{less than } 2)} = .4147$). The Social Causation Model explained more available variability than the Social Selection Model for this group of participants. The change in explained variance was $\chi^2_{\text{delta (1)}} = 116.159$ for the Social Causation Model which is larger than the Social Selection Model $\chi^2_{\text{delta (1)}} = 58.004$. The differences in the delta values is significant (Social Causation Model - Social Selection Model, $\chi^2_{\text{delta(1)}} = 58.155$, $p < .001$). If these data are taken together there is support for a Reciprocal Causation theoretical synthesis of social support, one that combines both the selection and the causation theories, in a form of a Reciprocal Action Model or a Reciprocal Effects Model (Calsyn & Winter, 2002).

Limitations

There are a number of limitations inherent in this kind of research in general and this study in particular. This research is applied in nature. Applied correlation research is not amenable to random assignment and experimental manipulation and these difficulties limit causal inferences. There are likely methodological biases introduced by self report data and availability biases (a type of response bias due to memory problems). This response bias may have served to limit the range of some of the variables (for example, LOS information).According to Maughan and Rutter (1997), self reports are seriously flawed but others disagree. According to Bernstein et al. (1994), retrospectively obtained histories of childhood experiences are generally stable over time, show good agreement with reports of other informants (see Goodman et al. 2003; Paivio, 2001; Krause, Shaw, &

Cairney, 2004).

Since this sample was not a randomly selected sample from the general population, the results are not representative of all clients with Depression and Manic-Depression leading to a generalizability problem. These results are only generalizable to members of a self-help group who are diagnosed with Depression or Manic-Depression because, as stated previously, individuals who affiliate themselves with self-help groups are different from those who choose not to participate. However, assessments of treatment/participation efficacy of self-help are problematic from the outset because of the inherently self-selected nature of these groups; but, results seem to be consistently positive for many participants (Davison, Pennebaker, & Dickerson, 2000). Biases that may arise from lack of random sampling may be offset by a reduction in bias due to a reduced response rate that may have resulted from a mass mailing to a general population sample. Social contact is one of the important advantages or assets of self-help organizations, yet only 50% of the family members of those clients with chronic mental disorders believed that support or self-help groups were important (Cutler, 1994). According to Dibb, and Yardley (2006); Hibbard et al. (2002); Thoits, Hohmann, Harvey, and Fletcher, (2000); and Whittemore, Rankin, Callahan, Leder, and Carol, (2002), self help interactions may provide a client with a positive perception of outcome if they can meet someone who has successfully coped with a similar illness, disability, trauma, can be a mentor and who can facilitate better adjustment. Gidron, Guterman, and Hartman (1990) found that those who participated in self-help groups for parents of the mentally ill tended to come from a higher socioeconomic background, used more interactive active coping strategies, and were more

stressed with the psychosocial burdens of the situations in which they found themselves; while those not participating tended to suffer more from basic survival issues (like finances) and used fewer active coping approaches. Usually, the members of these types of societies are highly motivated in endeavours that may help further their cause, creating a volunteer bias. A pilot test of the survey instrument indicated that respondents were well motivated since they took extra time to complete open-ended questions about current society functioning. However, at the same time, any particular group of members may be dealing with a recurrence or exacerbation of psychiatric symptomatology reducing the likelihood of responding to surveys.

Most studies on the effects of social support on psychiatric symptomatology have been cross-sectional. They are therefore ambiguous as to the direction of causality (Bolton & Oatley, 1987). The majority of this thesis depends upon quasi experimental correlational designs and correlation does not necessarily equal causation. These designs are subject to two dominant limitations, that of directionality and third variable causation ambiguities. Phase II of the research dealt with replicating cross-sectional models pertaining to psychiatric symptomatology. Although the pattern of relationships tended to be replicated, one cannot rule out neither the directionality problem nor the third variable causation difficulties. Phase III of the data analysis used cross-lagged models which deal with directionality difficulties since these causal models clearly laid the variables in an unambiguous time continuum. However, the difficulty related to third variable causation has not been addressed except to control for some possible 3rd variables in the analysis. Phase IV of this research is also limited to descriptive information due to limited cross-

sectional results. However, an attempt was made to provide a tentative cross-lagged model. The findings in all four phases of this research need to be replicated, including in other populations to see if the results generalize.

The LOS variable also had a significant number of zero scores. This created a problem for testing cross-lagged models for the LOS models. There were too few individuals with an inpatient stay during the period of study and too few persons indicating that they had a three year stay score ($\underline{N}$ = 78). In the final stage of this research there were very few individuals who had hospitalization histories and who responded to more than one wave. There were even fewer participants who were hospitalized after they completed the Time-1 survey who were readmitted during the previous six months. This left a small sample of individuals for testing LOS models, resulting in inadequate power for the effect sizes that exist. Larger sample sizes are needed in future research. The symptomatology model had enough power to detect even a small effect; however, the hospitalization sample was too small to have adequate power to detect small to moderate effect sizes (Cohen, 1983). In fact, power was an issue with all of the models tested after the first cross-sectional model tested. Not much could be done to rectify this difficulty for this research. Future research needs increased sample sizes.

Since quantitative contact frequency reports are easy to obtain from respondents, the primary support measure used in this research estimated the quantity of interactions respondents had in the last two weeks and used this as a stable measure of available support to evaluate the relationship between support and the three-year hospitalization variable. There are limitations related to the use of such a limited variable that had not

been used in the past literature. However, according to Brugha et al. (1990), levels of support contact were stable through time, but levels of negative interaction and dissatisfaction with support were less stable. According to House and Kahn (1985), contact frequency measures have been found to have a high 1-year test-retest reliability, be more stable when compared with qualitative or functional measures, and not be as influenced by mental health status. Also, social relationships must "exist in some quantity before they can have a structure and supportive content or function" (p. 89). These authors also stated that these types of quantity measures of social contact have been found "to relate cross-sectionally, retrospectively, and prospectively to lower rates of psychological and physical disorders and mortality" and that there is "substantial evidence for their construct validity in terms of their relationships with health outcomes" (pp 89-90).

There are some limitations related to operational definitions of some of the variables. The Contact Frequency measure had never been used prior to this study. There was a need to find a short measure of social contact, given limited time and cognitive effort so this measure will require further validation upon replication. Both the satisfaction with support variable and the psychiatric symptomatology variable are single item segmented visual analog scales. These measures are rather simplistic, but they were designed for ease of administration to participants experiencing significant psychiatric impairment. Given these limitations, and the fact that a mail survey was used to protect participant confidentiality, measures that could be completed in less than ten minutes and required little cognitive effort were necessary. The measures need further validation, but they appeared to work well for these purposes. The full SSQ-27 measure of social support

results in difficulties for clients with psychiatric symptomatology. Responding or compliance with providing names and relationships diminishes as one proceeds through the instrument, resulting in reliability difficulties for the count component of this scale (Torgrud, Walker, Murray, Cox, Chartier, & Kjernisted, 2004). The SSQ-6 demonstrates excellent psychometric properties while reducing required patient effort (Grivell et al. 2000). The MSPSS-12 has been used as a measure of support in very recent studies (Stanley, Beck, & Zebb, 1998; Wu & Serper, 1999) and the SSQ(short) (Sarason, Levine, Basham, & Sarason, 1983) has also been used in recent studies assessing the support mental health relationship. Both the SSQ-6 and the MSPSS-12 are commonly used in more recent studies to assess social support (Forbes & Roger, 1999). It would be helpful if the new measures were compared to these standard measures for reliability and validity. The single-item symptomatology measure also worked much like the EWB component of the Rand Health Survey (Hays et al. 1993), with correlations ranging from -.68 to -.81 for the samples at differing points in time.

There were also some difficulties related to the employment variable and not much could be done about its distribution. There were a lot of individuals in the sample indicating that they had a zero employment score. This outcome made testing the impact of employment difficult and results should be interpreted with this in mind.

Another significant issue is our ability to model the variables involved in the whole process. Psychiatric illness/health depends upon a number of factors in a persons' life. Psychological stress and negative affect have been found to be related to poorer overall health (Bisschop et al. 2003; Institute of Medicine, 2003; Lynch & Kaplan, 2000; Mendes

de Leon, Gold, Glass, Kaplan, & George, 2001) and negative affect is related to reduced desire to engage in social activity (Watson, 2000). Mental illness results from the influence of a combination of psyhco-bio-social factors. This research attempts to explain only some of this influence. The impact of the biological component of this theoretical model of illness is not and cannot be determined with these data. This biological component is often referred to as the genetic component of mental illness. According to Butler et al. (2006) genetic factors also may be important, indicating that several chromosomal regions may be involved.. Individuals with a genetic predisposition to develop a Bipolar/Unipolar Affective Disorder often exhibit symptoms after they have been exposed to a number of contributing environmental and personal factors that become a cascade of psycho-bio-social factors that trigger the underlying predisposition to psychiatric morbidity (Goodwin & Jamison, 1990). These genetic risk factors for major depression are positively related to the risk factor of stressful life events (Kendler & Karkowski-Shuman, 1997; Kendler et al. 1993; Kendler et al. 1999). This psycho-bio-social phenomenon already accounts for the participants being in our sample, and it impacts every stage of this kind of research. Most of these participants will have encountered such an environmental trigger in addition to a biological predisposition, and the current research cannot account for these issues.

At this point Type-I error rates need to be discussed. Although path analyses used in this research were confirmatory which provided some protection of Type-I error rates, there has been very little done to attempt to control the overall alpha for this research. This type of general quasi-experimental design, and path analysis in particular, have little

control over individual p-values used to test for significance. In model building, like path analysis, the model fit indices are used to determine model significance. Where possible, rather than force artificial p-values at all phases of the research, exact p-values are provided so the reader can make his or her own mind up about what effects are or are not statistically significant or salient. There are some instances, when appropriate and possible, where alpha levels are adjusted for directional predictions and Type-I error is controlled. In situations where multiple correlations are used in diagrams of models the reader should be concerned with effect magnitudes rather than exact p-values.

Future Research

Future research needs to replicate these findings in another self-help group or a group of inpatients with mood disorders not affiliated with a self-help group. This research needs to be extended to other samples of individuals with differing psychiatric disorders to test for generalizability, for example, and to individuals with schizophrenic disorders and other serious disorders. There is some evidence that the Social Selection Model may be more important for individuals with schizophrenia (Munk-Jorgensen & Mortensen, 1992). Future research needs to evaluate revised models that combine both theoretical positions (components from both theoretical models) being evaluated at the same time. This would allow for a clearer evaluation of some form of a Reciprocal Action Model or Reciprocal Effects Model (Calsyn & Winter, 2002). According to Laing, Krause, and Bennett (2001), and Okum and Keith, (1998), social support research needs to focus more on the varying dimensions of social support, the distinct contribution of each support construct, and the

causal ordering of social support constructs and a time-sequence between constructs needs to be addressed (Laing, Krause, & Bennett, 2001). According to Gigliotti (2002, 2004, & 2006), attention to network compositions differences hold great promise for strengthening the link between social support theory and research. Future research may also investigate the impact of support based upon whether the interactions were positive in nature (Okun & Keith, 1998) and did not contribute to the stress patients were experiencing and to feelings of overprotectiveness (Boutin-Foster, 2005).

Research about the variables that predict whether someone has a hospitalization history or not is necessary. The results of this study did not differentiate adequately why some persons were hospitalized while others with the same magnitude of symptomatology were not admitted for inpatient treatment. In fact, there were few differences found between those with and without hospitalization histories. Once again, the issue of symptoms explaining only 4.0% of inpatient LOS needs further evaluation. If this is a veridical finding, then research is needed to determine if programs targeting other areas of a patient's life may be more productive, less demoralizing, and less costly in the long run. For example, according to Dixon et al. (2001), families of the mentally ill who completed a family education program tended to have greater family, community, and service system empowered but this program did not change the burden of caring for a mentally ill family member or the ill family member's self esteem. Engaging families in the treatment of persons with serious mental illness is a complex, multi-determined process (Sherman, Faruque, & Foley, 2005). For example, the SAFE program provides information about services, symptoms, and information gathering for families, to reduce stigma, with the

hope that this communication would allow earlier interventions and provide support for family and patient through community support and medical help (Sherman, 2003). Standard programs for families with members experiencing serious mental illness can reduce the risk of relapse, enhance social support functioning and cut costs (Falloon et al. 2002; Pitschel-Walz et al. 2001). Also, online support cognitive behavioural therapy programs have been found to be successful with more than 50% of participants heavily using the online support and 37.9% preferring online communication to face-to-face counselling (Houston, Cooper, & Ford, 2002).

Future research should consider the apparent reduced rate of hospitalization in self-help group members or will have to treat the hospitalization in a more categorical form to allow for the consideration of the many zero values in this population. If a categorical value was used to assess the relationship between the predictor variables and hospitalization then, a form of logistic regression would be necessary for statistical testing purposes and this would require much larger sample sizes. This categorical approach for the presence of psychiatric hospitalization would also allow for the assessment of which variables predicted whether psychiatric hospitalization occurred.

Further research is also necessary to determine why individuals remain in this type of longitudinal research. It was clear that those with higher incomes and educational backgrounds were more likely to remain in the sample. This finding is common with survey research (Dillman, 1978) and was an expected outcome. This research was also biassed towards individuals who were more likely to participate or volunteer. Those who participate in research studies tend to be less socially disabled than those who do not

participate (Leff & Trieman, 2000). Illness severity was also an issue. Poor outcomes have been reported to be related to high symptom severity (Leff & Trieman, 2000). Full compliance with recommended treatment has been found to be related to better outcomes (Keck et al. 1998). However, self perceived stigma has also been found to impact adherence in a negative manner (Ayalon, Arean, & Alvidrez, 2005; Sirey, Bruce, & Alexopoulos, 2005; Siry et al. 2001), and stigma prevents people from participating in self-help groups (Davison, Pennebaker, & Dickerson, 2000; Griffiths, et al. 2004) and delays treatment seeking (Cooper, Corrigan, & Watson, 2003; Barney, Griffiths, Jorm, & Christiansen, 2006; Nolan & Badger, 2006; Schraufnagel, Wagner, Miranda, & Roy-Burne, 2006). Patient adherence may mediate the relationship between health and social support (Dunbar-Jacob & Schlenk, 2001), feelings that there is no one to turn to for help (Dunn, 2002), and treatment outcomes (DiMatteo, Giordani, Lepper, & Croghan, 2002). According to Druley, and Townsend (1998) and Hagedoorn et al. (2000), the pathway from social support to health likely travels through patient adherence to treatment programs (DiMatteo, 2004).In fact, according Klonoff et al. (1999), Sanders et al. (2004), and Utsey et al. (2000), there is a connection between discrimination on the basis of stigma related to mental illness and increased symptomatology, stress reactions, and perceived social rejection (Thompson Sanders, Noel, & Campbell, 2004)

Although there appeared not to be a significant differences in symptomatology between those who participated at all four points in time and those who did not, there was some evidence of a reduction in the proportion of the sample who had been hospitalized for psychiatric reasons. Once again, this small bias was expected as individuals who are

more ill were expected to have a higher attrition rate. These biases are likely to impact results in a conservative manner.

The relationship between education and financial adequacy needs further evaluation since the effects found were negative instead of positive as predicted. This may be due to persons with higher educational backgrounds expecting higher family incomes, which may not be the case if the person has a psychiatric disability. The relationships between the variables not involved in the social causation/selection debate need to be evaluated further. There is little literature evaluating the impact of being unemployed in populations that are most likely at risk of experiencing the event. What is available focusses on general populations, resulting in few studies looking at the impact of unemployment on psychiatric patients (Lindsey & Ozawa, 1979). The results relating Financial Adequacy Perception and SES variables to outcome measures (symptoms and social support) need further examination. The fact that it is how patients perceive their financial situation and not the actual level of the SES variables (ED, EMP, and INC) that impact the social support or symptom measures is interesting and contrary to some of the findings in the literature. Future research would need to replicate the present findings. Also, it would be interesting if a much larger sample could be grouped into differing income brackets and have the findings of this section tested with persons from different SES strata.

The causal orderings specified in these tested models are supported by theoretical and empirical literature. However, several causal orderings are possible based upon the available literature. The present research attempted to test the differing causal orderings.

For example, family income could precede social support and education, and hospitalization, at one point in time, may influence social support in another. There are many different patterns possible for this type of research, especially if one considers the variables in time.

There are a number of possible causal orderings, between the social functioning and morbidity variables that could be supported by the literature. Many of these relationships are non-recursive, meaning that they work in both directions in time. For example, if someone has a poor level of contact with a supportive network at the beginning of a psychiatric illness and is admitted to an inpatient psychiatric facility, this may serve notice to the family that there is a problem. After the individual is released from hospital, individuals in the client's network may now make a point of seeing the client more frequently. This increased contact could result in symptoms being recognized earlier and treatment provided on an outpatient basis or another hospitalization. The hospitalized individual could also alienate network members resulting in a reduction of contact after the first hospitalization. Either direction of causation is possible and is partially supported by the available literature.

Alternative data analysis strategies can be applied to the data. In particular, SAS PROC MIXED procedures for repeated measures would allow for the data to be correlated through time. This would deal with the independence assumption in path analysis by removing the variability from the dependence of the measures through time. This approach allows for across time random factors including handling missing data better. Also, this approach does not require multivariate sphericity assumptions and allows for the use of

differing variance covariance matrices which may include different patterns among the measurement occasions. It would be interesting to compare the path analysis results to the PROC MIXED outcomes. There are also other SEM procedures available, for example, AMOS.

Future research would benefit from larger sample of patients with hospital stays. The current models pertaining to LOS cannot be considered longitudinally unless there are adequate numbers of individuals with inpatient treatment (to increase power). An effort would have to be made to collect all of the social support and social functioning variables at an earlier point in time to allow for reexamining the relationships in time lagged models to be tested and to maintain an appropriate logical flow of variables in time. Longitudinal research in this area also needs to consider the confounding and possibly non-recursive relationships that exist in these types of social causation models (Hanlon, 1981).

Triangulating different research sources and techniques is important if a clear picture of the problem is going to be obtained. This field of study does not allow for experimental research and experimental control techniques to be applied to these research questions. Also, more work needs to be done on the issue of third variable confounding of causation. Triangulating results of various research paradigms is needed to support policy decisions.

Persons with severe and persistent mental illness also demonstrate social and interpersonal skills deficits (Iyer, Rothman, Vogler, & Spaulding, 2005). These findings could be used to support the development of more comprehensive support programs for individuals with psychiatric conditions. Programs need to help patients with social and

living skills since graduates of these types of programs appear to be more functional and had more reciprocal relationships with other people (Pilisuk, 2001). Current research has demonstrated the effectiveness and efficacy of programs teaching social skills to persons with chronic mental illness (Kopelowicz, Liberman, & Zarate, 2006). Programs that include activities directed toward enhancement of social support are a key component in interventions for young adolescents (Harter, 1999). The effects of psychological treatments in adolescents are modest so there is a pressing need to extend the range of treatments (Jorm et al. 2006). Research on social networks, norms of reciprocity, and trust that encourages people to act together to attain shared goals is required (Kreuter & Lezin, 2002). It is important that patients have some symmetrical relationships that allow them the opportunity to reciprocate to give as well as to receive. Reciprocity in relationships assures the individual of his or her importance to other people (Laing, Krause, & Bennett, 2001; Pilisuk, 2001).

Comprehensive community care for individuals with serious psychiatric disabilities will be expensive because of the array of social and medical factors involved (Holley, Jeffers, & Hodges, 1997). Programs to help the mentally ill who live in poverty should help patients meet their basic needs (Plant & Sachs-Ericsson, 2004). Modifying a personality construct like hostility may be a useful avenue in health-enhancing therapeutic interventions (Kop, 2005; Smith et al 2004; Vraneanu, Gallo, & Bogart, 2006). These programs would also have to be evaluated against the status quo situation to justify possible economic advantages of expanded programs. This research would have to include the costs of extended inpatient treatment against support program costs, including all of

the costs encountered by participants. Merton (1949) made a distinction between studying manifest functions refer to the anticipated and intended consequences of a given social practice, whereas latent functions are unintended consequences of social action. In order for public assistance programs to be more effective, it is crucial that their latent, as well as manifest functions, be explored fully (Krause & Shaw, 2002).

Ultimately, the goal of treatment of the mentally ill is minimal stigmatization, least restrictive environment, and adequate treatment. Whether these goals are met with maximal hospital diversion (access to hospital for only the most disturbed patients) or not remains to be seen. Alternatives to inpatient treatment have been developing for the past thirty years or more. Alternative treatment can include receiving treatment and medications at home. Research has demonstrated that, when family and friends were educated and incorporated into the treatment strategy whenever possible, this resulted in reduced state hospital admissions (Reding & Raphelson, 1995). The additions of mobile crisis teams have also proved to be beneficial in reducing hospital admissions (Reding & Raphelson, 1995). These crisis teams have been found to be effective in several European countries and in Canada (Reding & Raphelson, 1995). According to Mosher and Burti (1989), a 24-hour mobile crisis team should be an integral part of every community mental health program.

The trend has been a shift away from hospitalization as the sole response to psychiatric emergencies (Friedman, 1983). In home treatment of patients has been found to be better at reducing symptomatology, less expensive, to result in a faster return to normal functioning, to decrease the burden felt by family members and patients, and in-home care

has been found to be acceptable to families and patients (Kates et al.1991). Community resources are often overlooked (Zubenko et al. 1994). According to Hatfield, Gearon, and Coursey (1996), there is a failure of coordinating quality community resources with family collaboration. The adequacy of a community care program depends on the adequacy of the services available outside the hospital (Harris et al. 1987). "Success of this intervention argues that the role of relatives should be taken into account, within community care, when planning multidisciplinary psychiatric services. The severity of a patients psychopathology has a negative impact on his or her caregivers' mental health (Lee et al. 2006). Appropriate reallocation of resources and priorities to meet the needs of the chronically mentally ill and their families would be required, if such a policy is to be successful" (Tarrier et al. 1988, p. 541). If non-medical factors such as income and employment explain a substantial part of the variation in the level and distribution of health in populations, then research should begin to focus on the health consequences of tax-and -transfer policies, labour market policies, and social service policies (Lavis, 2002)

According to Sharstein (1991), who studied the differences in outcomes of short- and long-stay hospitalizations, these studies reflect a point of view that length of stay may be decreased with outpatient alternatives and community services substituted for inpatient care (Mirin, Gossett, & Grob, 1991). However, these studies often did not include more severe cases and, in general studies that look at diagnosis and severity of illness are lacking or incomplete (Mirin et al. 1991). Serious mental illnesses are the most costly to treat (Knapp, Almond, & Percudani, 1999). To consistently assess mental health care costs it is important that outcome evaluation indicators be carefully selected (Lehoux & Battista,

2000).

Assertive community treatment programs that are high in fidelity and focus on very specific patient difficulties tend to have high retention rates and reduce hospital stays (McHugo et al. 1999). According to Hollingsworth, and Sweeney (1997), community services can include clinical and rehabilitative outpatient service support, counselling, day treatment, additional medical treatment, case management, medication checks and medication counselling, crisis care, substance abuse treatment, daily living and vocational services, supportive home care, transportation options, other services for clients at high risk of inpatient treatment, and residential services. Residential care services include housing and adult family home care. These types of services are similar to the categories of care recommended by Hu, Snowden, and Jerrell, (1992). However, in a study by Hollingsworth, and Sweeney (1997), which provided the above types of services, expenditures for these community-based outpatient services were greater than expenditures for inpatient care. To be able to provide these types of services to clients, managed care plans would need to use a much broader definition of service and to budget for significant amounts of non-medical services (Mehcanic, Schlesinger, & McAlpine, 1995; Tanenbaum & Hurley, 1995). Managed care environments challenge providers and require meaningful information about services, outcomes, and consumer perceptions (Rakutis, Feidley, & Wood, 1998). Possibly a single point of responsibility for the chronically mentally ill would facilitate the management of these cases in a more cost effective manner (Shah & Ganesvaran, 1997). Although the provision of these community based services may cost more, without services like these "stable community-based lives

may be significantly reduced" (Hollingsworth & Sweeney, 1997, p.489). It is important to understand all of the factors relating to inpatient treatment so the most cost-effective and clinically appropriate decisions can be reached resulting in a better outcome for patients (Hendryx & Rohland, 1997).

Policy Implications

According to D'Arcy (1978), it is important, for therapeutic and social policy implications, to incorporate the social and system factors involved in the process of becoming mentally ill. About 25% of persons with severe and persistent mental illness will need of intensive community support (Wasylenki et al. 2000). According to Formoso, Gonzales, & Aiken (2000), relationships with families, friends, schools, churches, and other service-based organizations all play an important role in stress reduction. It is also important to intervene in a patient's social environment to improve mental health and aid in stress reduction (Cohen, 2004). However; intervening in a person's social environment is not a simple process (Cohen, 2004; Gottlieb, 2000). According to Sherbourne (1988) and Cutrona (1986), structural support measures like contact frequency indicate the availability of support. This result partially supports results from Vaananen, Vahtera, Pentti, and Kivimaki, (2004), who believe that structural measures, like contact frequency and network membership may influence mental health directly. Contact frequency has been found to be inversely related to psychiatric pathology (Cohen et al. 2000; Strauss & Carpenter, 1977). For social support to occur it is important that patients have network contact (Vega et al. 1991). Contact frequency has been found to be related to requests for

support and satisfaction with support (Fiore et al. 1986). Frequent contact with one's support network has been found to increase the probability of support being made available (Cutrona, 1986). Unfortunately, persons with significant depressive symptomatology have lower levels of support (Holsten & D'Elia, 1985; Lelliot, Wing, & Clifford, 1994), lower levels of family support (Mitchell & Moos, 1984), and smaller support networks (Denoff, 1981).

Social exclusion, common in psychiatric clients, produces emotional/social pain (Zadro & Williams, 2003). Patients who interact less with others have been found to have greater levels of symptomatology (Cohen, 2004; Falloon & Marshall, 1983).Mentally ill patients are often lonely and isolated after impatient treatment (Harris, Brown, & Bifulco, 1987; Rud & Noreik, 1982). In fact, according to Cacioppo, Hughes Waile, Hawkley, and Thisted (2006), there appears to be a reciprocal relationship between loneliness, depressive symptomatology, and suicidal behaviour (Goldsmith, Pellmar, Klienman, & Bunney, 2002; Heikkinen & Kauppinen, 2004). Perception of satisfaction with support has been found to improve with enrollment in community-based programs (Junginger, 1990) and network coaching has been found to be productive (Gotlieb, 1981). Patients were still isolated and faced significant stigma and loneliness, and pervasive hard times (Friedrich et al. 1999). This type of stigma prevents many inflicted individuals from participating with self-help groups (Davison, Pennebaker, & Dickerson, 2000; Griffiths, et al. 2004). Stigma has also been found to be one of the most important factors influencing people to avoid or delay seeking treatment for mental illness (Cooper, Corrigan, & Watson, 2003; Barney, Griffiths, Jorm, & Christiansen, 2006; Nolan & Badger, 2006; Schraufnagel, Wagner,

Miranda, & Roy-Burne, 2006). According to Wilkinson (1975), social isolation has been reported to be positively related to psychiatric symptomatology. Isolation is believed to play an etiological role in the development of depression (Wilkinson, 1975; Neziek, Hampton, & Shean, 2000). Mood disorder clients' social support has been found to be related to treatment outcomes (Billings & Moos, 1985; Brugha et al. 1990; George et al. 1989; O'Connell et al. 1991). The current results support the findings of Mittal, Fortney, Pyne, Edlund, and Wetherell (2006) that psychiatric patients need social support for psychiatric well-being. Systematic inclusion of self-help treatment could reduce psychiatric patients' reliance upon the number of visits to mental health professionals (Kessler et al. 1994). Self-help therapy has been found to be effective in the treatment for those with mild to moderate depression (McKendree-Smith, Floyd, & Scogin, 2003) and computerized self-help program/materials was also found to be effective (Marks et al. 2003). Internet depression support groups warrant continued research regarding supplementation of face-to-face depression care (Houston, Cooper, & Ford, 2002). In fact, self-help treatment has been found to provide approximately the same treatment effect as other traditional psychotherapy interventions (McKendree-Smith, Floyd, & Scogin, 2003). Exposure to web-based resources has also been found to reduce the personal stigma of mental illness (Griffiths et al. 2004). There is a need for much more research (controlled studies) about the effectiveness of self-help groups in treatment of emotional disorders. For example, are they cost effective and do they benefit all patients (Den Boer, Wiersma, & Van Den Bosch, 2004)?

Regardless of the model assessed, it is clear that social support and

symptomatology were inversely related. Severe psychiatric disorders and their sequella have significant consequences for patients and community members. According to Rosenblatt and Atkinson (1993), there is a need for the provision of innovative services that can improve the quality of life for patients and minimize the impact on patients who are suffering.

According to Thornicraft and Breakey (1991), improved social networks have been found to be associated with better social functioning. Limitations in activity at one point in time predict psychosocial functioning at a later point in time (Eide & Roysamb, 2002). Improved social functioning and integration have also been found to be related to better prognoses (Brugha et al. 1990; Klein et al. 1998; Swindle et al. 1989). Disabling health conditions, like psychiatric pathology, have been found to cause a constriction of the social network and reduce the level of contact with friends (Arling, 1987), psychological stress, and negative affect have been found to be related to poorer health (Bisschop et al. 2003; Mendes de Leon, Gold, Glass, Kaplan, & George, 2001), and health status has been found to improve with increasing social and economic status (Institute of Medicine, 2003; Lynch & Kaplan, 2000). These factors reduce, not only the patient's satisfaction with support, but contact frequency. Lack of social support during negative life events can reduce self-esteem and can result in patients devaluing their social network, resulting in isolation (Amann, 1991). Those that live in resource-deficient social environments experiences negative mental health outcomes (Berkman & Kawachi, 2000).

The present results indicate that high levels of satisfaction with support and contact frequency are important in reducing symptom severity at a later point in time. These

findings support the results Allen, Ciambrome, and Welch, (2000) and McKee-Ryan, Song, Wanberg, and Kinicki, (2005) have reported, that greater perception of social support has a positive effect on mental health. Although it may be difficult to modify a client's satisfaction with support, contact frequency is much more amenable to modification. Could innovative programs be designed to help psychiatric patients maintain a sufficiently large support network? One of the advantages to online support for depression includes accessibility by individuals from a wide geographic area who may not have easy access to groups in their community (Houston, Cooper, & Ford, 2002). Given that the actual count (CF) is important for these participants, possible programs could be designed to help teach patients how to maintain sufficient contact with their support network (eg. Network Coaching, see Gottlieb, 1981). Cognitive Behavioral Therapy (CBT) has been found to improve overall social functioning exhibit and higher levels of treatment satisfaction (Bauer et al. 2006). In fact, internet-delivered cognitive-behavioural therapy programs have been found to be a good adjunctive/complimentary treatment for those with mild to moderate depression (Anderson, Bergstrom, Carlbring, Kaldo, & Ekselius, 2005). An absence of family support has been found to be related to worse prognoses in patients with depression (Boyce & Parker, 1985; Moos, 1990; Keitner et al. 1992; Mai et al. 1993; Swindle, Cronkite, & Moos, 1989). Family, friends, and significant others can also be educated about the importance of this type of contact and they may be able to facilitate regular and frequent contact with the patient. Since contact frequency is important, independent of satisfaction, these results also indicate that some form of in-hospital and after release visitation programs may be beneficial. Possibly, visitation

programs can involve patients who are stable, visiting those who are less stable as in the current inpatient visitation program run by the Mood Disorders Association of Manitoba. Patients enrolled in this program have positive perceptions of these visits (Murray, 1994).

"Since very little can be done to alter the nature or social selection component of psychiatric illnesses the author believes it is up to researchers to discover if the social creation/nurture component can be modified to bring about a reduction in malleable social stressors that do influence psychiatric disposition in susceptible populations" (Murray, 1996, p. 167). Many other social programs may also impact the patient's perception and frequency of social supportive interactions. Existing psychopathological impairment can impact psychosocial role functioning, making these patients less able to sustain employment and familial roles, which often results in lower SES (Aneshensel et al. 1981). Many persons with psychiatric disorders find their way into the lowest SES groups in society (Aneshensel et al. 1981). These patients subsist on extremely low income levels. This outcome creates a lot of economic distress. Social inequalities, like income inequality, increased exposure to stressful events (Adler, 2001). According to Ross, Wolfson, Dunn, Berthelot, Kaplan, and Lynch (2000), reducing income inequality would be beneficial for population health, especially in the United States since income is a much stronger determinant of life chances and, in turn, "health chances" than in Canada. Poverty, low SES, and unemployment have been found to be related to higher stress (Brown, Bhrolchain, & Harris 1975; Eaton, 1974; Lindblad-Goldberg, Dukes, & Lasley, 1988; Makosky, 1982; McLanahan, 1983; Miller, 1988). This research indirectly and directly supports these findings. Economic stress exacerbates symptomatology. This

situation severely limits patients' leisure activities, which also exerts a negative impact upon their social support networks and leaves them with a lot of unstructured time alone. According to Hobfoll, (2002), key resources may help a person counteract resource loss when confronted with external demands (Gorgievski-Duijvesteijn, Bakker, Schaufeli, & van der Heiden, 2005).

When considering the current research, it became clear that the standard SES variables did not reliably predict psychiatric symptomatology. However, the results did indicate that SES, education, and household income were related to perception of financial adequacy which did play a significant role in symptom magnification. As stated previously, many of the mentally ill are not employed. The current results indicate that those who were not employed were more likely to be admitted. There is a need for more creative and innovative employment programs for patients. This may help mitigate these kinds of difficulties and consequential barriers, and to facilitate employment opportunities for patients who are capable. Current programs tend to train persons with psychiatric disabilities with "menial tasks which are not in keeping with their educational backgrounds" (Ms. M, personal communication, 2005; Ms. M is a client with a bipolar disorder who is currently on a Vocational Rehabilitation Education Program waiting list and has experience with the other vocational programs). These jobs also tend to be primarily low paying, creating further economic stress.

Even though education was not directly related to psychiatric symptomatology it was related to perceptions of financial adequacy, which in turn, impacted the severity of symptomatology. According to Ross and Huber (1985), well-educated persons suffer less

from economic hardships due to low income than individuals who are poorly educated. There are presently programs designed to educate any psychiatric patient via Vocational Rehabilitation but the waiting list is almost two years long (Ms. M., personal communication, 2005). This waiting list tends to discourage patients from even bothering to apply.

To alleviate the stress brought about by lack of gainful employment and low incomes, patients need comprehensive programs that reduce some of the economic pressures and improve their perception of financial adequacy. It is important that patients receive income that provides an adequate living level to reduce financial stressors. Patients should have access to an independent source of income so they are not economic burdens to potential partners.

Almost all patients with psychiatric disorders are treated with medications at some time in their treatment history. Medication compliance in psychiatric patients is difficult and failure to comply is related to increased symptomatology (Hendryx & Rohland, 1997; Kirk & Therrein, 1975; Labbate & Doyle, 1997). Some patients reported not taking the medications they were prescribed due to financial difficulties (Murray, 1996). Programs need to ensure that patients never have to decide whether they take their medications due to financial reasons.

Other programs that provide access to transportation are needed, for example, making public transportation more accessible by providing monthly bus passes even if the patient is not 'looking for work'. Presently, many psychiatric patients have to fight with welfare to gain access to basic transportation and funding for telephones (Dr. Steibart,

personal communication, 2001; Dr Steinbart is a psychiatrist who has treated many persons with mental illness). Programs are needed that provide the patient with access to other activities; for example, museums, zoos, art galleries, YMCA memberships, baseball or football games, and other activities most healthy individuals and those with adequate income take for granted. These activities will facilitate the patient's contact with other non-ill members of society and allow the patient to go out with the friends they do have. Comprehensive programs should educate patients about the importance of social support and teach them how to cope, given the financial resources they have available.

There are also policy implications relating to LOS results from this study. If one considers components of SES to include education, family income, and employment, these are related to LOS. Considering the non-lagged results (see Figure 19), why were those with higher ED and INC more likely to spend more time in inpatient care? Current results are the opposite of predicted results and are not in agreement with the bulk of the literature. In a Canadian study, patients from lower SES groups were found to have longer LOSs (Mai et al. 1993; Laessle, Yassouridis, & Pfister, 1988). Chronic stressors, such as lack of employment and inadequate income force the mentally ill into a state of socio-psychological-economic dependency which contributes to readmission and increased LOS (Franklin, Kittredge, & Thrasher, 1975). Readmitted patients have been found more likely to have impairments in self-care and poor premorbid functioning and more intense clinical pathology (Lyons et al. 1997).

Admission rates have been found to be higher among those with little education (Zohar et al. 1987) and family income has been found to be negatively related to the probability of admissions (Hall & Royse, 1987). Admission and poverty rates are

positively related (Errera et al.1963; Hollingshead, 1957; Shader et al. 1969) and low-SES patients were more likely to be admitted for inpatient treatment (Gerson & Bassuk, 1980). In Canada, social deprivation has been found to be positively related to high admission rates (Thornicroft, 1991; Thornicroft et al. 1992). Yet, other researchers have reported that SES and admission status are not related (O'Regan, 1965; Tischler, 1966). The current results support the findings of Errera, Wyshak, and Jarecki (1963), Gerson & Bassuk (1980), Hall and Royse (1987), Hollingshead (1957), Shader et al. (1969). Those from higher income households were less likely to have been admitted for inpatient treatment. The current research also confirms that those with lower educational attainment were more likely to have been admitted for inpatient treatment, supporting the findings or Zohar, Hadas, and Modan (1987).

Poverty synergistically interacts with a lack of education, creating more economic hardship. Low education translates into low earnings and low education increases the difficulties of dealing with low income (Ross & Huber, 1985). A high positive correlation exists between education and income (Brown & Lawrence, 1985) and low levels of both variables are associated with an increased risk for psychiatric symptomatology (Ross & Mirowsky, 1989). When the lagged results were considered (see Figure 21) these findings supported the findings from the literature that greater LOSs was inversely related to ED and INC. These results will need replication and further investigation into their veridical nature.

Once again, the fact that psychiatric symptomatology only explained 4% of LOS is interesting. Given that the other variables considered in the LOS Hospitalization Model (see Figure 19) all accounted for approximately 4% of the unique variability in LOS, there

must be some policy approach that could facilitate reducing the impact of all of the other factors that explain LOS. There have to be better approaches to reduce inpatient LOS because it is so strongly influenced by irrelevant factors.

According to the current results, in the LOS Hospitalization model (see Figure 19), CF and SAT were positively associated to LOS. This finding is also not congruent with the bulk of findings from the literature. LOS has been found to be longer for individuals with inadequate social support (Holsten & D'Elia, 1985; Mai et al. 1993). Social isolation has been found to contribute to prolonged hospital stays (Rud & Noreik, 1982; Holsten & D'Elia, 1985; Mai et al. 1993). Also, patients have been found to remain in hospital longer if they had less support to ensure adequate aftercare (Allen et al. 1992; Carpenter, Strauss, 1991; Pottick et al. 1995). The current results are clearly not concordant with published findings. And, these findings were replicated when one considers the lagged results (see Figure 21). Higher LOSs at an earlier point in time are related to higher levels of support at a later point in time. The first step to explain these divergent findings would be to attempt to replicate them. If these results replicate, then there are policy ramifications. Could greater LOSs be helpful in allowing the patient access to higher levels of support either through contact with professionals or para-professionals, or with their family and friend support networks? It may also be that greater LOSs allow patients to meet other patients in the same situation and may nurture new friendships, leading to higher social support levels. Patients may also have been contacted by the outreach worker from the Mood Disorders Association, which may also facilitate both increased contact frequency and satisfaction with support at a later point in time.

References

Aaronson, N. K. (1989). Quality of life assessment in clinical trials: Methodological issues. *Controlled Clinical Trials, 10*(4 Suppl.), 195S-208S.

Accordino, M., P., & Herbert, J. T. (2000). Treatment outcome of four rehabilitation interventions for persons with serious mental illness. *Journal of Mental Health Counseling, 22,* 268-282.

Achat, H., Kawachi, I., Levine, S., Berkey, E., Coakley, E., & Coditz, G. (1998). Social networks, stress and health-related quality of life. *Quality of Life Research, 7,* 735-750.

Adamsen, L. (2002). 'From victim to agent': The clinical and social significance of self help group participation for people with life-threatening diseases. *Scandinavian Journal of Caring Sciences, 16*(3), 224-231.

Adler, E., & Newman, K. (2002). Socioeconomic disparities in health: Pathways and policies. *Health Affairs, 21*(2), 60-76.

Adler,N. E. (2001). A consideration of the multiple pathways from sopcioeconmic status to health. In J. A. Auerbach and B. K. Krimgold, (Eds.), *Income, Socioeconomic Status and Health: Exploring the Relationships,* Washington, DC: National Policy Association.

Aftanas, M. S. (1994). *Basic principles in the assessment and measurement of behavior.* (Revised ed.). Winnipeg, MB: University of Manitoba Printing Services.

Ahr, P. R., Gorodezky, M. J., & Cho. D. W. (1981). Measuring the relationship of public psychiatric admissions to rising unemployment. *Hospital and Community*

Psychiatry, 32(6), 398-400.

Aiken, R. C. B. (1970). Communication of symptoms. *Psychotherapy and Psychosomatics, 18,* 74-80.

Aiken, M., Ferman, L., & Sheppard, H. (1968). *Economic failure, alienation and extremism.* Ann Arbor, MI: University of Michigan Press.

Al-Issa, I. (1982). Gender and adult psychopathology. In I. Al-Issa, *Gender and psychotherapy* (pp. 83-101). New York NY: Academic.

Allen M. J., & Yen, W. M. (1979). *Introduction to measurement.* Monterey, CA: Brooks/Cole.

Allen, N. B., & Badcock, P. B. T. (2003), The social risk hypothesis of depressed mood: Evolutionary, psychosocial and neurobiological perspectives. *Psychological Bulletin, 129*(6), 887-913.

Allen, J. G., Coyne, L., Lyle, J., & Spohn, H. E. (1992). Assessing need for extended psychiatric hospitalization. *Comprehensive Psychiatry, 33*(5), 346-352.

Allen, S. M., Ciambrone, D., & Welch, L. C. (2000). Stage of life course and social support as mediator of mood state among persons with disability. *Journal of Aging and Health, 12*(3), 318-341.

Allgower, A., Wardle, J., & Steptoe, A. (2001). Depressive symptoms, social support and personal health behaviors in young men and women. *Health Psychology, 20*(3), 223-227.

Alloy, L. B., Abramson,L. Y., Whitehouse, W. G., Hogan, M. E., Tashman, N. A., Steinberg, D., et al. (1999). Depressogenic cognitive styles: Predictive validity,

information processing and personality characteristics, and developmental origins. *Behaviour Research and Therapy, 37,* 503-531.

Amaddeo, F., Bisoffi, G., Bonizzato, P., et al. (1995). Mortality among patients with psychiatric illness. A ten-year case register study in an area with a community-based system of care. *British Journal of Psychiatry, 166,* 783-788.

Amann, G. (1991). Social network and social support deficits in depressed patients: A result of distorted perception? *European Archives of Psychiatry and Clinical Neuroscience, 241,* 49-56.

Andersson, G., Bergstrom, J., Hollandare, F., Carlbring, P., Kaldo, V., & Ekselius, L. (2005). Internet-based self-help for depression: Randomised controlled trial. *British Journal of Psychiatry, 187,* 456-461.

Aneshensel, C. S., & Frerichs, R. R. (1982). Stress, support, and depression: A longitudinal causal model. *Journal of Community Psychology, 10,* 363-376.

Aneshensel, C. S., & Stone, J. D. (1982). Stress and depression: A test of the buffering model of social support. *Archives of General Psychiatry, 39*(12), 1392-1396.

Aneshensel, C. S., Frerichs, R. R. & Clark, V. A. (1981). Family roles and sex differences in depression. *Journal of Health and Social Behaviour, 22,* 379-393.

Anfinson, T. J., & Kathol, R. G. (1992). Screening laboratory evaluation in psychiatric patients: A review. *General Hospital Psychiatry, 14*(4), 248-257.

Angermeyer, M. C., Holzinger, A., Matschinger, H., & Stengler-Wenzke, K. (2002). Depression and quality of life: Results of a follow-up study. *International Journal of Social Psychology, 48,* 189-199.

Anthony, W. (1980). *The principle of psychiatric rehabilitation.* Baltimore. University Park Press.

Antonovsky, A. (1967). Social class, life expectancy and overall mortality. *Milbank Memorial Fundund Quarterly, 45,* 31-73.

Antonucci, T. C., & Israel, B. A. (1986). Veridicality of social support: A comparison of principle and network members' responses. *Journal of Consulting and Clinical Psychology, 54,* 432-437.

Argoff, C. E. (2007). The coexistence of neuropathic pain, sleep, and psychiatric disorders: A novel treatment approach. *The Clinical Journal of Pain, 23*(1), 15-22.

Arling, G. (1976). The elderly widow and her family: Neighbors and friends. *Journal of Marriage and the Family, 38,* 757-768.

Arling, G. (1987). Strain, social support, and distress in old age. *Journal of Gerontology, 42,* 107-113.

Arnow, B. A., Hunkeler, E. M., Blasey, C. M., Lee, J., Constantino, M. J., Fireman, B., Kraemer, H. C., Dea, R., Robinson, R., & Hayward, C. (2006). Comorbid depression, chronic pain , and disability in primary care. *Psychosomatic Medicine, 68,* 262-268.

Arthur, H. M. (2006). Depression, isolation, social support, and cardiovascular disease in older adults. *Journal of Cardiovascular Nursing, 21*(5), S2-7.

Asipinwall, L. G., Hill, D. L., & Leaf, S. L. (2002). Prospects, pitfalls ans plans: A proactive perspective on social comparison. *European Review of Social Psychology, 12,* 267-298.

Atkinson, T., Liem, R., & Liem, J. (1986). The social costs of unemployment: Implications for social support. *Journal of Health and Social Behaviour, 27*(4), 317-331.

Augarde, K. (1998). Alternatives to hospital admission: An emergency day care pilot scheme. *Mental Health Care, 1(10),* 340-343.

Auslander, G. (1988). Social networks and the functional health status of the poor: A secondary analysis of data from the National Survey of Personal Health Practices and Consequences. *Journal of Community Health, 13*(4), 197-109.

Avlund, K., Lund, R., Holstein, B. E., Due, P., Sakari-Rantala, R., & Heikkinen, R. L. (2004). The impact of structural and functional characteristics of social relations as determinants of functional decline. *Journal of Gerontology: Social Sciences, 59B,* S44-S51.

Ayalon, L., Arean,P. A., & Alvidrez, J. (2005). Adherence toantidepressant medications in black and Latino elderly patients. *American Journal of Geriatric Psychiatry, 16,* 572-580.

Baker, B., & Rochon, J. (1989). Length of stay, short stay units and psychiatric emergency admissions. *Canadian Journal of Psychiatry, 34,* 39-42.

Baker, F., Jodrey, D., & Intagliata, J. (1992). Social support and quality of life of community support clients. *Community Mental Health Journal, 26,* 397-411.

Bakke, E. W. (1933). *The unemployed man: A social study.* London: Nisbet.

Balon, R. (1987). Suicide: Can we predict it? *Comprehensive Psychiatry, 28,* 236-241.

Barefoot, J. C., Brummett, B., Clapp-Channing, N. E., Siegler, I. C., Vitaliano, P. P.,

Williams, R. B., & Mark, D. B (2000). Moderators of the effect of social support on depressive symptoms in cardiac patients. *American Journal of Cardiology, 86,* 438-442.

Barnett, P. A., & Gotlib, I. H. (1988) Psychosocial functioning and depression: Distinguishing among antecedents, concomitants, and consequences. *Psychological Bulletin, 104,* 97-126.

Barney, L. J., Griffiths, K. M., Jorm, A. F., & Christiansen, H. (2006). Stigma about depression and its impact on help-seeking intentions. *Australia and New Zealand Journal of Psychiatry, 40,* 51-54.

Barrera, M. (1981). Social support in the adjustment of pregnant adolescents: Assessment issues. In B. Gottleib, (Ed.), *Social networks and social support* (55-66). Beverly Hills, CA: Sage.

Barth, J., Schumacher, M., & Herrmann-Lingen, C. (2004). Depression as a risk factor for mortality in patients with coronary heart disease: A meta-analysis. *Psychosomatic Medicine, 66,* 802-813.

Bartlett, C. A., & Ghosal, S. (2002). Building competitive advantage through people. *MIT Sloan Management Review, 453,* 34-41.

Baruch-Feldman, C., Brondolo, E., Ben-Dayan, D., & Schwartz, J. (2002). Sources of social support and burnout, job satisfaction and productivity. *Journal of Occupational Health Psychology, 7*(1), 84-93.

Bassuk, E. L., Bissell, H. D., Mickelson, K. D., & Perloff, J. N. (2002). Role of kin and nonkin support in the mental health of low-income women. *American Journal of*

Orthopsychiatry, 72(1), 39-49.

Bauer, M. S., McBride, L., Williford, W. O., Glick, H., Kinosian, B., Altshuler, L., Beresford, T., Kilbourne, A. M., & Sajatovic, M. (2006). Collaborative care for bipolar disorder: Part II. Impact on clinical outcome, function and costs. *Psychiatric Services, 57,* 937-945.

Baum, A., Garofalo, J. P., & Yali, A. M. (1999). Socioeconomic status and chronic stress: Does stress account for SES effects on health? *Annals of the New York Academy of Sciences, 896*(1), 131-144.

Baum, A., & Posluszny, D. (1999). Health psychology: Mapping biobehavioral contributions to health and illness. *Annual Reviews Psychology, 50,* 137-163.

Bearman, K. J., & La Greca, A. M. (2002). Assessing friend support of adolescents' diabetes care: The diabetes social support questionnaire-friends version. *Journal of Pediatric Psychology, 27*(5), 417-428.

Beauford, J. E., McNiel, D. E., & Binder, R. L. (1997). Utility of the initial therapeutic alliance in evaluating psychiatric patients' risk of violence. *American Journal of Psychiatry, 154(9),* 1272-1276.

Bebbington, P., Hurry, J., Tennant, C., Sturt, E., & Wing, J. K. (1981). Epidemiology of mental disorders in Camberwell. *Psychological Medicine, 11,* 561-579.

Bebbington, P., Christopher, T., & Hurry, J. (1981). Adversity and the nature of the psychiatric disorders in the community. *Journal of Affective Disorders, 3,* 345-366.

Bebbington, P., Brugha, T., MacCarthy, B., Potter, J., Stuart, E., Wykes, T., & Katz, R. (1988). The Camberwell collaborative depression study. I. The investigation of

depressed probands. *British Journal of Psychiatry, 152,* 754-765.

Beehr, T. A., Farmer, S. J., Glazer, S., Gudanowski, D. M., & Nair, V. N. (2003). The enigma of social support and occupational stress: Source congruence and gender role effects. *Journal of Occupational Health Psychology, 8*(3), 220-231.

Ben-Zur, H. (2002). Monitoring/blunting and social support: Associations with coping and affect. *International Journal of Stress Management, 9*(4), 357-373.

Bengelsdorf, H., Church, J., Kaye, R., Orlowski, B., & Alden, D. (1993). The cost effectiveness of crisis intervention: Admission diversion savings can offset the high cost of service. *The Journal of Nervous and Mental Disease, 181*(12),757-762.

Bentler, P. M., & Bonett, D. G. (1980). Significance tests and goodness of fit in the analysis of covariance structures. *Psychological Bulletin, 88,* 588-606.

Bentler, P. M. (1989). *EQS Structural equations program manual.* Los Angeles, CA: BMDP Statistical Software.

Benzeval, M., & Judge, K. (2001). Income and health: The time dimension, *Social Science and Medicine, 52,* 1371-1390.

Berkman, L., & Syme, S. L. (1979). Social networks, host resistance, and mortality: A nine-year follow-up study of Alameda County residents. *American Journal of Epidemiology, 109,* 186-204.

Berkman, L & Kawachi, I. (2000). A historical framework for social epidemiology. In L. Berlman, and I. Kawachi, (Eds.), *Social Epidemiology,* New York, NY: Oxford University Press.

Berkman, L. F., & Writing Committee for the ENRICHD Investigators (2003). Effects of treating depression and low perceived social support on clinical events after myocardial infarction. *Journal of the American Medical Association, 289*(23), 3016-3116.

Berkman, L. F. (1995). The role of social relations in health promotion. *Psychosomatic Medicine, 57,* 245-254.

Berkman, L. F. (2000). Social support, social networks, social cohesion, and health. *Social Work in Health Care, 32*(2), 3-14.

Berkman, L. F., & Glass, T. (2000). Social integration, social networks, social support, and health. In L. F. Berkman, & I. Kawachi (Eds.), *Social Epidemiology (pp. 137-173).* New York, NY: Oxford University

Berkman, L., & Breslow, L. (1983). *Health and ways of living: The Alameda County Study.* New York, NY: Oxford University Press.

Berkman, L., Berkman, C., Kasi, S., Freeman, D., Leo, L., & Ostfeld, A. (1986). Depressive symptoms in relation to physical health and functioning in the elderly. *American Journal of Epidemiology, 124,* 372-388.

Berkman, L., & Kawachi, I (2000). *Social Epidemiology,* New York, NY: Oxford University Press.

Bernstein, D. P., Fink, L., Handelsman, L., Lovejoy, M., Wenzel, K., et al. (1994). Initial reliability and validity of a new retrospective measure of child abuse and neglect. *American Journal of Psychiatry, 151,* 1132-1136.

Bernstein, D., & Rubin, D. C. (2002). Emotionally charged autobiographical memories

across the life span: The recall of happy, sad, traumatic, and involuntary memories. *Psychology and Aging, 17,* 636-652.

Berry, R, & Williams, F. (1987). Assessing the relationship between quality of life and marital income satisfaction: A path analytic approach. *Journal of Marriage and the Family, 49,* 107-116.

Bezold, H. S., MacDowell, M., & Kunkel, R. (1996). Predicting psychiatric length of stay. *Administration and Policy in Mental Health, 23(5),* 407-423.

Billings, A. G., & Moos, R. H. (1985). Psychosocial processes of remission in unipolar depression: Comparing depressed patients with matched community controls. *Journal of Consulting and Clinical Psychology, 53,* 314-325.

Billings, A. G., Cronkite, R. C., & Moos, R. H. (1983). Social-environmental factors in unipolar depression: Comparisons of depressed patients and nondepressed controls. *Journal of Abnormal Psychology, 92,* 119-133.

Birkel, R. C., & Reppucci, N. D. (1983). Social networks, information-seeking, and the utilization of services. *American Journal of Community Psychology, 11*(2), 185-205.

Birtchnell, J. (1971). Social class, parental social class, and social mobility in psychiatric patients and general population controls. *Psychological Medicine, 1,* 209-221.

Bisschop, M. I., Kriegsman, D. M., van Tilburg, T. G., Penninx, B. W., van Eijk, J. T., & Deeg, D. J. (2003). The influence of differing social ties on decline in physical functioning among older people with and without chronic disease. *Longitudinal Aging Study Amsterdam. Aging Clinical and Experimental Research, 15,* 164-173.

Black, D. W., Goldstein, R. B., Nasrallah, A., & Winokur, G. (1991). The prediction of recovery using a multivariate model in 1471 depressed inpatients. *European Archives of Psychiatry and Clinical Neuroscience, 241,* 41-45.

Blaine, B. E. (2000). The Psychology of Diversity (pp. 109-120). Mountain View, CA: Mayfield.

Blakely, T. A., & Woodward, A. J. (2000). Ecological effects in multi-level studies. *Community Health, 54,* 367-374.

Blalock, H. M. (1968). Theory building and causal inferences. In H. M. Blalock & A. B. Blalock (Eds.), *Methodology in social research* (pp. 295-315). New York: McGraw-Hill.

Blalock, H. M. (1961). *Causal inferences in non-experimental research.* Chapel Hill, NC: University of North Carolina Press.

Bland, R. C., & Orn, H. (1981). Schizophrenia: Sociocultural factors. *Canadian Journal of Psychiatry, 26,* 186-188.

Blazer, D. G., Burchett, B., Service, C., & George, L. K. (1991). The association of age and depression among the elderly: An epidemiological exploration. *Journals of Gerontology, 46*(6), M210-M215.

Blazer, D., & Williams, C. (1980). Epidemiology of dysphoria and depression in an elderly population. *American Journal of Psychiatry, 137,* 439-444.

Boerner, K. (2004). Adaptation to disability among middle-aged and older adults: The role of assimilative and accommodative coping. *Journal of Gerontology: Social Sciences, 59B,* S35-S42.

Bolger, N., Kessler, R. C., & Zuckerman, A. (2000). Invisible support and adjustment to stress. *Journal of Personality and Social Psychology, 79*(6), 953-961.

Bolger, N., & Eckenrode, J. (1991). Social relationships, personality, and anxiety during a major stressful event. *Journal of Personality and Social Psychology, 61*(3), 440-449.

Bolger, N., Zuckerman, A., and Kessler, R. C. (2000). Invisible support and adjustment to stress. *Journal of Personality and Social Psychology, 79,* 953-961.

Bolton, W., & Oatley, K. (1987). A longitudinal study of social support and depression in unemployed men. *Psychological Medicine. 17*(2), 453-460.

Bond, M., & Feather, N. (1988). Some correlates of structure and purpose in the use of time. *Journal of Personality and Social Psychology, 55,* 321-329.

Boot, B., Hall, W., & Andrews, G. (1997). Disability, outcome and case-mix in acute psychiatric in-patient units. *British Journal of Psychiatry, 171(Sept.),* 242-246.

Bosma, H., Van de Mheen, H. D., & Mackenbach, J. P. (1999). Social class in childhood and general health in adulthood: Questionnaire study of contributions of psychological attributes. *British Medical Journal, 318,* 18-22.

Bothwell, R., & Scott, J. (1997). The influence of cognitive variables on recovery in depressed inpatients. *Journal of Affective Disorders, 43,* 207-212.

Boutin-Foster, C. (2005). In spite of good intentions: patients' perspectives on problematic social support interactions. *Health and Quality of Life Outcomes, 3*(52), 1-11.

Bower, G. H. (1981). Mood and memory. American Psychlogist, 36, 129-148.

Boyce, P., & Parker, G. (1985). Neuroticism as a predictor of outcome in depression.

Journal of Nervous and Mental Disease, 173, 685-688.

Bradburn, N. M., Rips, L. J., & Shevell, S. K. (1987). Answering autobiographical questions: The impact of memory and inference on surveys. *Science, 236,* 157-161.

Branje, S .J. T., van Aken, M. A. G., & van Lieshout, C. F. M. (2004). Relations between big five personality characteristics and perceived support in adolescent's families. *Journal of Personality and Social Psychology, 86*(4), 615-628.

Braunig, P., Shugar, G., & Kruger, S. (1996). An investigation of the Self-Report Manic Inventory as a diagnostic and severity scale for mania. *Comprehensive Psychiatry, 37*(1), 52-55.

Breines, E. B. (2004). Response to American Journal of Occupational Therapy, 58(3), 315-316.

Brenner, M. H., & Mooney, A. (1983). Unemployment and health in the context of economic change. *Social Science and Medicine, 17,* 1125-1138.

Brenner, S., & Starrin, B. (1988). Unemployment and health in Sweden: Public issues and private troubles. *Journal of Social Issues, 44,* 125-140.

Brenner, M. H. (1973). *Mental illness and the economy.* Cambridge, MA: Harvard University Press.

Brewer, B. W., Van Raalte, J. L., Cornelius, A. E., and Petitpas, A. J. (2000). Psychological factors, rehabilitation adherence, and rehabilitation outcome after anterior cruciate ligament reconstruction. *Rehabilitation Psychology, 45,* 20-37.

Briere, J., Woo, R., McRae, B., et al. (1997). Lifetime victimization history, demographics, and clinical status in female psychiatric emergency room patients.

The Journal of Nervous and Mental Disease, 185(2), 95-101.

Brissette, I., Carver, C. S., & Scheier, M. F. (2002). The role of optimism in social network development, coping, and psychological adjustment during a life transition. *Journal of Personality and Social Psychology, 82*(1), 102-111.

Brodaty, H., MacCuspie-Moore, C. M., Tickle, L., et al. (1997). Depression, diagnostic sub-type and death: A 25 year follow-up study. *Journal of Affective Disorders. 46,* 233-242.

Brown, N., & Parrish, J. (1987). Community support and rehabilitation of the mentally disabled in the United States. *International Journal of Mental Health, 15,* 16-25.

Brown, G. W., Andrews, B., Harris, T. O., Adler, Z., & Bridge, L. (1986). Social support, self-esteem, and depression. *Psychological Medicine, 16,* 813-831.

Brown, D., & Lawrence, G. (1985). Predictors of depressive symptoms among unemployed Black adults. *Journal of Sociology and Social Welfare, 12*(4), 736-754.

Brown, G. W., & Harris, T. (1978). *Social origins of depression: A study of psychiatric disorder in women.* New York: Free Press.

Brown, G. W., Bhrolchain, M., & Harris, T. (1975). Social class and psychiatric disturbance among women in an urban population. *Sociology, 9,* 225-254.

Brown, G. W., Harris, T. O., Hepworth, C., & Robinson, R. (1994). Clinical and psychosocial origins of chronic depressive episodes: II: A patient enquiry. *British Journal of Psychiatry, 165*(4), 457-465.

Bruce, M. L. (2001). Depression and disability in later life: Directions for future research.

American Journal of Geriatric Psychiatry, 9, 102-112.

Brugha, T. S., Bebbington, P. E., Sturt, E., MacCarthy, B., & Wykes, T. (1990). The relation between life events and social support networks in a clinically depressed cohort. *Social Psychiatry and Psychiatric Epidemiology, 25*(6), 308-313.

Brugha, T., Bebbington, P. E., MacCarthy, B., Potter, J., Sturt, E., & Wykes, T. (1987). Social networks, social support, and the type of depressive illness. *Acta Psychiatrica Scandinavica, 76,* 664-673.

Brugha, T., Conroy, R., Walsh, N., Delaney, W., O'Hanlon, J., Donodero, E., Daly, L., Hickey, N., & Bourke, G. (1982). Social networks attachments and support in minor affective disorders: A replication. *British Journal of Psychiatry, 141,* 249-255.

Brugha, T. S., Bebbington, P. E., MacCarthy, B., Sturt, E., Wykes, T., & Potter, J. (1990). Gender, social support and recovery from depressive disorders: a prospective clinical study. *Psychological Medicine, 20,* 147-156.

Brugha, T. S., Bebbington, P. E., Stretch, D. D., et al. (1997). Predicting the short-term outcome of first episodes and recurrences of clinical depression: A prospective study of life events, difficulties, and social support networks. *Journal of Clinical Psychiatry, 58,* 298-306.

Brugha, T. S., Bebbington, P. E., MacCarthy, B., Potter, J., Sturt, E., & Wykes, T. (1987). Social networks, social support and the type of depressive illness. *Acta Psychiatrica Scandinavica, 76,* 664-673.

Brummett, B. H., Mark, D. B., Siegler, I. C., Williams, R. B., Babyak, M. A.,

Clapp-Channing, N. E., & Barefoot, J. C. (2005). Perceived social support as a predictor of mortality in coronary patients: Effects of smoking, sedentary behavior, and depressive symptoms. *Psychosomatic Medicine, 67,* 40-45.

Brunner, E. (1997). Socioeconomic determinants of health: Stress and the biology of inequality. *British Medical Journal, 314,* 1472-1476.

Bryk, A. S., & Raudenbush, S. W. (1987). Application of hierarchical linear models to assessing change. *Psychological Bulletin, 101*(1), 147-158.

Buell, G., & Anthony, W. (1975). The relationship between patient demographic characteristics and psychiatric rehabilitation outcome. *Community Mental Health Journal, 12,* 208-214.

Burgess, P. M., Joyce, C. M., Pattison, P. E., & Finch, S. J. (1992). Social indicators and the prediction of psychiatric inpatient service utilisation. *Social Psychiatry and Psychiatric Epidemiology, 25,* 144-148.

Burlingame, G. M., Seaman, S., Johnson, J. E., Whipple, J., Richardson, E., Rees, F., Earnshaw, D., Spencer, R., Payne, M., & O'Neil, B. (2006). Sensitivity to change of the brief psychiatric rating scale-extended (BPRS-E): An item and subscale analysis. *Psychological Services, 3*(2), 77-87.

Burton, E., Stice, E., & Seeley, J. R. (2004). A prospective test of the stress-buffering model of depression in adolescent girls: No support once again. *Journal of Consulting and Clinical Psychology, 72*(4), 689-697.

Butler, R., Carney, S., Cipriani, A., Hatcher, S., Price, J., & von Korff, M. (2006). Clinical evidence concise. A publication of British Medical Journal publishing group.

Depressive disorders. *American Family Physician, 73*(11), 1-8.

Cacioppo, J. T., Hawkley, L. C., & Bernston, G. G. (2003). The anatomy of loneliness. *Current Directions in Psychological Science, 12,* 71-74.

Cacioppo, J. T., Hughes, M. E., Waite, L. J., Hawkley, L. C., & Thisted, R. A. (2006). Loneliness as a specific risk factor for depressive symptoms: Cross-sectional and longitudinal analyses. *Psychology and Aging, 21*(1), 140-151.

Cacioppo, J. T., Earnst, J. M., Burleson, M. H., McClintock, M. K., Malarkey, W. B., Hawkley, L., et al. (2002). Lonely traits and concomitant physiological processes: The MacArthus social neuroscience studies. *International Journal of Psychophysiology, 35,* 143-154.

Campbell, D. T., & Stanley, J. C. (1963). *Experimental and quasi-experimental designs for research.* Chicago: Rand McNally.

Campbell, D. T. (1969). Reforms as experiments. *American Psychologist, 24,* 409-429.

Cannella, B. L. (2006). Mediators of the relationship between social support and positive health practices in pregnant women. *Nursing Research, 55*(6), 437-445.

Carney, R. M., & Sheps, D. S. (2004). Depression is a risk factor for mortality in coronary heart disease. *Psychosomatic Medicine, 66*, 799-801.

Carothers, S. S., Borkowski, J. G., & Whitman, T. L. (2006). Children of adolescent mothers: Exposure to negative life events and the role of social supports on their socioemotional adjustment. *Journal of Youth and Adolescence, 35*(5), 822-832.

Carpenter, W. T., & Strauss, J. S. (1991).The prediction of outcome in schizophrenia: IV. Eleven-year follow-up of the Washington IPSS cohort. *Journal of Nervous and*

Mental Disorders, 179(9), 517-525.

Carpiniello, B., Catra, M., & Rudas, N. (1989). Depression among elderly people: A psychosocial study of urban and rural populations. *Acta Psychiatrica Scandinavica 80,* 445-450.

Carver, C. S., Smith, R. G., Antoni, M. H., Petronis, V. M., Weiss, S., & Derhagopian, R. P. (2005). Optimistic personality and psychosocial well-being during treatment predict psychosocial well-being among long-term survivors of breast cancer. *Health Psychology, 24*(5), 508-516.

Carveth, W., & Gottlieb, B. (1979). The measurement of social support and its relation to stress. *Canadian Journal of Behavioral Science, 11,* 179-86.

Casey, P., Meagher, D., & Butler, E. (1996). Personality, functioning, and recovery from major depression. *Journal of Nervous and Mental Disease, 184*(4), 240-245.

Cassidy, F., Forest, K., Murray, E., et al. (1998). A factor analysis of the signs and symptoms of mania. *Archives of General Psychiatry, 55,* 27-32.

Cassidy, J. (2000). Adult romantic attachments: A development perspective on individual differences. Review of General Psychology, 4, 111-131.

Castle, D. J., Phelan, M., Wessely, S., & Murray, R. M. (1994). Which patients with non-affective functional psychosis are not admitted at first psychiatric contact? *British Journal of Psychiatry, 165,* 101-106.

Caton,C. L., & Gralnick, A. (1987). A review of issues surrounding length of psychiatric hospitalization. *Hospital and Community Psychiatry, 38,* 858-863.

Cattell, V. (2001). Poor people, poor places, and poor health: The mediating role of social

networks and social capital. *Social Science & Medicine, 52*(10), 1501-1516.

Chandra, A., & Minkovitz, C. S. (2006). Stigma starts early: Gender differences in teen willingness to use mental health services. *Journal of Adolescent Health, 38*(6), 754-758.

Charlifue, S. W., & Gerhart, K. A. (1991). Behavioural and demographic predictors of suicide after traumatic spinal cord injury. *Archives of Physiology and Medical Rehabilitation, 72,* 488-492.

Chelimsky, E., & Shadish, W. R. (1997). *Evaluation for the 21st century: A handbook.* London: Sage.

Chen, H. T. (1990). *Theory-driven evaluations.* Newbary Park, CA: Sage.

Chen, H. T. (1993). Emerging perspectives in program evaluation. *Journal of Social Service Research, 17,* 1-17.

Chen, H. T., & Rossi, P. H. (1980). The multi-goal, theory-driven approach to evaluation: A model linking basic and applied social science. *Social Forces, 59,* 106-122.

Chen, H. T., & Rossi, P. H. (1983). Evaluation with sense: The theory-driven approach. *Evaluation Review, 7,* 283-302.

Chen, H. T., & Rossi, P. H. (1987). The theory-driven approach to validity. *Evaluation and Program Planning, 10,* 95-103.

Chen, H. T., & Rossi, P. H. (Eds.). (1992). *Using theory to improve program and policy evaluations.* Westport, CT: Greenwood.

Christian, L. M., & Stoney, C. M. (2006). Social support versus social evaluation: Unique effects on vascular and myocardial response patterns. *Psychosomatic Medicine,*

68(6), 914-921.

Chronbach, L. J. (1980). *Toward reform of evaluation: Aims, methods of institutional arrangements.* San Francisco: Jossey-Bass.

Citron, M., Solomon, P., & Draine, J. (1999). Self-help groups for families of persons with mental illness: Perceived benefits of helpfulness. Community Mental Health Journal, 35(1), 15-30.

Clara, I. P., Cox, B. J., Enns, M. W., Murray, L. T., & Torgrude, L. J. (2003). Confirmatory factor analysis of the Multidimensional Scale of Perceived Social Support in clinically distresses and student samples. *Journal of Personality Assessment, 81*(3), 265-270.

Clark, A. E., Diener, E., Georgellis, Y., & Lucas, R. E. (2003). Lags in leads in life satisfaction: A test of the baseline hypothesis. See http://www.delta.ens.fr/clark/BLINEaug03.pdf.

Clayton, P. J. (1981). The epidemiology of bipolar ive disorder. *Comprehensive Psychiatry, 22,* 31-43.

Cobb, S. (1979). Social support and health through the life course. In M. W. Riley (Ed.), *Aging from birth to death (pp. 93-106).* Boulder, CO: Westview Press.

Cobb, S., & Kasl, S. V. (1977). *Termination: The consequence of job loss.* Cincinnati, OH: Department of Health Education and Welfare (NIOSH).

Cochrane, R., & Stopes-Roe, M. (1981). Women, marriage, employment and mental health. *British Journal of Psychiatry, 139,* 373-381.

Cohen, C., & Sokolovsky, J. (1978). Schizophrenia and social networks: Ex-patients in the

inner city. *Schizophrenia Bulletin, 4*(4), 546-560.

Cohen, G. D. (2000). Loneliness in later life. *American Journal of Geriatric Psychiatry, 8,* 273-275.

Cohen, J. (1988). *Statistical Power Analysis for the Behavioral Sciences (2nd ed.).* New York: NY, Academic Press.

Cohen, J., & Cohen, P. (1983). *Applied multiple regression/correlation analysis for the behavioral sciences* (2nd ed.). Hillsdale, NJ: Lawrence Erlbaum.

Cohen, J., & Cohen, P. (1975). *Applied multiple regression/correlation analysis for the behavioral sciences.* Hillsdale, NJ: Lawerence Earlbaum.

Cohen, S. (2004). Social relationships and health. *American Psychologist, 59,* 676-684.

Cohen, S., Doyle, W. J., Turner, R. B., Alper, C. M., & Skoner, D. P. (2003). Sociability and susceptibility to the common cold. *Psychological Science,14,* 389-395.

Cohen, S., Gottlieb, B. H. & Underwood, L. G. (2000). Social relationships and health. In S. Cohen, L. G. Underwood, & B. H. Gottlieb (Eds.), *Social support measurement and intervention* (pp. 3-25). Oxford: Oxford University Press.

Cohen, S., Kaplan, G. A., & Salonen, J. T. (1999). The role of psychological characteristics in relation between socioeconomic status and perceived health. *Journal of Applied Social Psychology, 29,* 445-468.

Cohen, S., & Rodriguez, M. S. (1995). Pathways linking affective disturbances and physical disorders. *Health Psychology, 14*(5), 374-380.

Cohen, S., & Syme, S. L. (1985) Issues in the study and application of social support. In S. Cohen, & S. L. Syme, (Eds.), *Social support and health* (pp.3-72). New York, NY:

Academic Press.

Cohen, S., & Willis, T. A. (1985). Stress, social support and the buffering hypothesis. *Psychological Bulletin, 98,* 310-357.

Collingwood, R. G. (1940). *An essay on metaphysics.* Oxford: Clarendon.

Collins, M. E., & Mowbray, C.T. (2005). Higher education and psychiatric disabilities: National survey of campus disability services. *American Journal of Orthopsychiatry, 75*(2), 304-315.

Conger, R. D., Rueter, M. A., & Elder,G. H. (1999). Couple resilience to economic pressure. *Journal of Personality and Social Psychology, 76,* 54-71.

Converse, J., & Presser, S. (1988). *Survey questions.* London: Sage.

Cooke, R. G., Kruger, S, & Shugar, G. (1996). Comparative Evaluation two self-report mania rating scales. *Society of Biological Psychiatry 40,* 279-283.

Cooke, R. G., Robb, J. C., Young, L. T., & Joffee, R. T. (1996). Well-being and functioning in patients with bipolar disorder assessed using the MOS 20-Item short form (SF-20). *Journal of Affective Disorders, 39*(2), 93-97.

Cooper, A. E., Corrigan, P. W., & Watson, A. C. (2003). Mental Illness stigma and care seeking. *Journal of Nervous and Mental Disease, 191,* 339-341.

Cornelis, C. M., Ameling, E. H., & de Jonghe, F. (1989). Life events and social network in relation to onset of depression. *Acta Psychiatrica Scandinavica, 80,* 174-179.

Corning, A. F. (2002). Self-esteem as a moderator between perceived discrimination and psychological distress among women. *Journal of Counceling Psychology, 49,* 117-126.

Costello, E. (1991). Married with children: Predictors of mental and physical health in middle-aged women. *Psychiatry, 54*(3), 292-305.

Coyne, J. C., Aldwin, C., & Lazarus, R. S. (1981). Depression and coping in stressful episodes. *Journal of Abnormal Psychology, 90,* 439-447.

Coyne, J. C., & Downey, G. (1991). Social factors in psychopathology. *Annual Review of Psychology, 42,* 401-425.

Craig, T. J., & Van Natta, P. A. (1979). Influence of demographic characteristics on two measurements of depressive symptoms: The relation of prevalence and persistence of symptoms with sex, age, education, and marital status. *Archives of General Psychiatry, 36*(2), 149-154.

Creed, P. A., & Macintyre, S. R. (2001). The relative effects of deprivation of the latent and manifest benefits of employment on the well-being of unemployed people. *Journal of Occupational Health and Psychology, 6,* 324-331.

Cronbach, L. J. (1984). *Essentials of Psychological Testing,* (4th ed.). New York, NY: Harper & Row.

Croughan, J. L., Secunda, S., Katz, M. M., Robins, E., Mendels, J., Swann, A., & Harris-Larkin, B. (1988). Sociodemographic and prior clinical course characteristics associated with treatment response in depressed patients. *Journal of Psychiatry Research, 27,* 227-237.

Crystal, L. P., Fenster, J. R., Suresh, D. P., & Bliss, D. E. (2006). Social support, appraisals, and coping as predictors of depression in congestive heart failure patients. *Psychology and Health, 21*(6), 773-789.

Cutler, J. (1994). *Family members behavioural intentions to advocate for resources for community-based care, including home care, for individuals with a chronic mental disorder.* Unpublished doctoral dissertation. Winnipeg, MB: University of Manitoba.

Cutrona, C. (1986). Objective determinants of perceived social support. *Journal of Personality and Social Psychology, 50*(2), 349-355.

Cyr, I. J., & Haley G. A. (1983). Use of demographic and clinical characteristics in predicting length of psychiatric hospital stay: A final evaluation. *Journal of Consulting Clinical Psychology, 51,* 637-640.

D'Arcy, C. (1978). Changing patterns of psychiatric morbidity in Saskatchewan. Canada's Mental Health, 26(1), 6-12.

D'Arcy, C., & Brockman, J. (1976). Changing public recognition of psychiatric symptoms & Blackfoot revisited. *Journal of Health & Social Behaviour, 17,* 302-310.

Dangelmaier, R. E., Docherty, N. M., & Akamatsu, T. J. (2006). Psychosis proneness, coping, and perceptions of social support. *American Journal of Orthopsychiatry, 76*(1), 13-17.

Daniels, B. A., Kirkby, K. C., Hay, D. A., et al. (1998). Predictability of rehospitalisation over 5 years for schizophrenia, bipolar disorder and depression. *Australian and New Zealand Journal of Psychiatry 32,* 281-286.

Davidson, L., Chinman, M., Kloos, B., Weingarten, R., Stayner, D., & Kraemer Tebes, J. (1999). Peer support among individuals with severe mental illness: A review of the evidence. *Clinical Psychology: Science and Practice, 6,* 165-187.

Davis, S. (1985). The "new young chronic" psychiatric patient: A study in Vancouver. *Social Work in Health Care, 11*(2), 87-100.

Davison, K. P., Pennebaker, J. W., & Dickerson, S. S. (2000). Who talks? The social psychology of illness support groups. *American Psychologist, 55*(2), 205-217.

Dayal, N., Chiu, C. Y., & Sharvar, R. (1984). Ecological correlates of cancer mortality patterns in an industrialized urban population. *Journal of the National Cancer Institute, 73,* 565-574.

De Silva, M. J., McKenzie, K., Harpham, T., & Huttly, S. R. A. (2005). Social capital and mental illness: A systematic review. *Journal of Epidemiology and Community Health, 59,* 619-627.

Dean, A., Kolody, B., Wood, P., & Ensel, W. M. (1989). Measuring the communication of social suppo from adult children. *Journal of Gerontology, 44,* S71-79.

Dean, A., Lin, N., & Ensel, W. M. (1981). The epidemiological significance of social support systems in depression. In R. C. Simmons *Research in community and mental health* (pp. 77-109). Greenwich, CT: JAI Press.

Dean, A., Kolody, B., & Wood, P. (1990). Effects of social support from various sources on depression in elderly persons. *Journal of Health and Social Behavior, 31*(June), 148-161.

Dear, M., Clark, G., & Clark, S. (1979). Economic cycles and mental health care policy: An examination of the macro-context for social service planning. *Social Science and Medicine, 13,* 43-53.

Decker, J. (1972). Crisis intervention and prevention of psychiatric disability: A follow-up

study. *American Journal of Psychiatry, 129*(6), 725-729.

Deestra, J. T., Peeters, M. C. W., Schaufeli, W. B., & Stroebe, W. (2003). Receiving instrumental support at work: When help is not welcome. *Journal of Applied Psychology, 88*(2), 324-331.

DeLongis, A., Capreol, M., Holtzman, S., O'Brien, T., & Campbell, J. (2004). Social support and social strain among husbands and wives: A multilevel analysis. *Journal of Family Psychology, 18*(3), 470-479.

Denoff, M. S. (1981). *Social competence and the supportive network of deinstitutionalized mental patients.* Unpublished doctoral dissertation, University of Pittsburgh, Pennsylvania.

Deutsch, S. I., & Davis, K. L. (1983). Schizophrenia: A review of diagnostic and biological issues. I: diagnosis and prognosis. *Hospital and Community Psychiatry, 34,* 313-322.

Dewa, C., Durbin, J., Wasylenki, D., Eastabrooke, S., Boydell, L., et al. (2002). Considering a multi-site study? Taking the leap and what to look for before you do. *Journal of Community Psychology, 30,* 173-188.

Dewees, M., Pulice, R. T., & McCormick, L. L. (1996). Community integration of former state hospital patients: Outcomes of a policy shift in Vermont. *Psychiatric Service, 47,* 1088-1092.

Dibb, B., & Yardley, L. (2006). How does social comparison within a self-help group influence adjustment to chronic illness? A longitudinal study. *Social Science & Medicine, 63*(6), 1602-1613.

Dicker, R., Morrissey, R. F., Abikoff, H., et al. (1997). Hospitalizing the suicidal adolescent: Decision-making criteria of psychiatric residents. *Journal of American Academy of Child & Adolescent Psychiatry, 36(6)*, 769-776.

Diener, E., Suh, E. M., Lucas, R. E., & Smith, H. L. (1999). Subjective well-being: Three decades of progress. *Psychologica Bulletin, 125,* 276-302.

Diener, E., Suh, E.M., Lucas, R. E., & Smith, H. L. (1999). Subjective well-being: Three decades of progress. *Psychological Bulletin, 125*(2), 276-302.

Diener, E. (1984). Subjective well-being. *Psychological Bulletin, 95*(3), 542-575.

Diez Roux, A. V. (2001). Investigating neighbourhoods and area effects on health. *American Journal of Public Health, 91*(11), 1786-1789.

Dillman, D. (1978). *Mail and telephone surveys: The total design method.* New York: John Wiley & Sons.

DiMatteo, M. R., Giordani, P. J. Lepper,H. S., & Croghan,T. W. (2002). Patient adherence and medical treatment outcomes: A meta-analysis. *Medical Care, 40,* 794-811.

DiMatteo, M. R. (2004). Social support and patient adherence to medical treatment: A meta-analysis. *Health Psychology, 23*(2), 207-218.

Dimirsky, M. A. (1990). *The quality of life of the chronically mentally ill living in the community: An investigation of the Oregon quality of life theory.* Unpublished doctoral dissertation. Winnipeg, MB: University of Manitoba.

Dixon, L., Stewart, B., Burland, J., Delahanty, J., Lucksted, A., & Hoffman, M. (2001) Pilot study of the effectiveness of the family-to-family education program. *Psychiatric Services, 52*(7), 965-967.

Dohrenwend, B. P. (1975). Sociocultural and social-psychological factors in the genesis of mental disorders. *Journal of Health and Social Behaviour, 16,* 365 - 392.

Dohrenwend, B. S., Krasnoff, L., Askenasy, A., & Dohrenwend, B. P. (1978). Exemplification of a method for scaling life events: The PERI life events scale. *Journal of Health and Social Behavior, 19,* 205-229.

Dohrenwend, B. P., & Dohrenwend, B. S. (1969). *Social status and psychological disorder: A causal injury.* New York, NY: Wiley.

Dohrenwend, B. P. (Ed.). (1998). *Adversity, stress and psychopathology.* New York, NY: Oxford University Press.

Dohrenwend, B. P., Levav, I., Shrout, P. E., Schwartz, S., Naveh, G., Link, B. G., Skodol, A. E., & Stueve, A. (1992). Socioeconomic status and psychiatric disorders: The causation-selection issue. *Science, 255,* 946-952.

Dohrenwend, B. P., Levav, I., Shrout, P. E., Schwartz, S., Naveh, G., Link, B. G., Skodol, A., & Stueve, A. (1998). Ethnicity, socioeconomic status, and psychiatric disorders: A test of the social causation-social selection issue. In B. P. Dohrenwend (Ed.), *Adversity, stress and psychopathology* (pp. 285-318). New York, NY: Oxford University Press.

Dooley, D., & Catalano, R. (1980). Economic change as a cause of behavioral disorder. Psychological Bulletin, 78(2), 450-468.

Douglas, K. S., & Koch, W. J. (2001).Civil commitment and civil competence: Psychological issues. In J. R. P. Ogloff (Ed.), Introduction to psychology and law: Canadian Perspectives (pp.353-374).

Drake, R. E., Rosenberg, S. D., Teague, G. B., Bartels, S. J., & Torrey,W. C. (2003). Fundamental principles of evidence-based medicine applied to mental health care. *Psychiatric Clinics of North America, 26,* 811-820.

Draper, B., & Luscombe, G. (1998). Quantification of factors contributing to length of stay in an acute psychogeriatric ward. *International Journal of Geriatric Psychiatry, 13,* 1-7.

Dressler, W. W. (1986). Unemployment and depressive symptoms in a southern Black community. *Journal of Nervous and Mental Disease, 174*(11), 639-645.

Druley, J. A., & Townsend, A. L (1998). Self-esteem as a mediator between spousal support and depressive symptoms: A comparison of healthy individuals and individuals coping with arthritis. *Health Psychology, 17*(3), 255-261.

DuBois, D. L., Burk-Braxton, C., Swenson, L. P., Tevendale, H. D., Lockerd, E. M., & Moran, B. L. (2002). Getting by with a little help from self and others: Self-esteem and social support as resources during early adolescence. *Developmental Psychology, 38*(5), 822-839.

Dunbar-Jacob, J., & Schlenk, E. (2001). Patient adherence to treatment regimen. In A. Baum, T. A. Revenson, and J. E. Singler (Eds.), *Handbook of Health Psychology (pp. 571-580).* Maheah, NJ: Erlbaum.

Duncan, O. D. (1966). Path analysis: Sociological examples. *American Journal of Sociology, 72,* 1-16.

Dunham, H. W. (1965). *Community and schizophrenia an epidemiological analysis.* Detroit: Wayne State University Press.

Dunlop, S., Coyte, P., & McIssac, W. (2000). Socio-economic status and the utilization of physicians' service results from the Canadian National Population Health Survey. *Social Science and Medicine, 51(1)*, 123-133.

Dunn, J. R. (2002). Housing and inequalities in health: A study of socioeconomic dimensions of housing and self reported health from a survey of Vancouver residents. *Journal of Epidemiology and Community Health, 56,* 671-681.

Dunn, D. S. (2000). Social psychological issues in disability. In R. G. Frank and T. R. Elliott (Eds.). *Handbook of Rehabilitation Psychology (pp. 565-584).* Washington, DC: American Psychological Association.

Durkheim, E. (1951). *Suicide.* New York: The Free Press.

Earls, M., & Nelson, G. (1988). The relationship between long-term psychiatric clients' psychological well-being and their perceptions of housing and social support. *American Journal of Community Psychology, 16,* 279-293.

Eastwood, R. E., Whitten, J. L., & Kramer, P. M. (1994). A brief instrument for longitudinal monitoring of mood states. *Psychiatry Research, 11,* 119-125.

Eaton, W. W. (1981). Demographic and social risk factors for mental disorders. In D. A. Reiger, & G. Allen, (Eds.), *Risk factors of research in the major mental disorders.* Proceedings of a Conference (DHHS Pub. No. ADM 83-1068). Washington, DC: U.S. Government Printing Office.

Eaton, W. W. (1980). A formal theory of selection for schizophrenia. *American Journal of Sociology, 86,* 149 - 158.

Eaton, W. W. (1978). Life events, social supports, and psychiatric symptoms: Pre-analyses

of the New Haven data. *Journal of Health and Social Behavior, 19,* 230-234.

Eaton, W. W. (1974). Residence, social class and schizophrenia. *Journal of Health and Social Behaviour, 15,* 289-299.

Eaton, W. W. (1994). Social facts and the sociological imagination: The contributions of sociology to psychiatric epidemiology. *Acta Psychiatrica Scandinavica, 90*(385), 25-38.

Eaton, W., & Kessler, L. (1981). Rates of symptoms of depression in a national sample. American Journal of Epidemiology 114, 528-538.

Edgerton, J. W., & Bentz, W. K. (1969). Attitudes and opinions of rural people about mental illness and program services. *American Journal of Public Health, 59,* 470-477.

Edwards, R. J., & Bagozzi, R.P. (2000). On the nature and direction of relationships between constructs and measures. *Psychological Methods, 5*(2), 155-174.

Ehleres, A., Mayou, R. A., & Bryant, B. (1998). Psychological predictors of chronic posttraumatic stress disorder after motor vehicle accidents. *Journal of Abnormal Psychology, 107*(3), 508-519.

Eide, A. H., & Roysamb, E. (2002). The relationship between level of disability, psychological problems, social activity and social networks. *Rehabilitation Psychology, 47*(2), 165-183.

Eisemann, M. (1984). The relationship of personality to social network aspects and loneliness in depressed patients. *Acta Psychiatrica Scandinavica, 70*(4), 337-341.

Eisenberger, N. I. (2003). Does rejection hurt. An FMRI study of social exclusion.

Science, 302(5643), 290-292.

Elal, G., & Krespi, M. (1999). Life events, social support and depression in hemodialysis patients. *Journal of Community and Applied Social Psychology,9,* 23-33.

Ellison, C. G., & Levine, J. S. (1998). The religion-health connection: Evidence, theory, and future directions. *Health Education & Behavior, 25,* 700-720.

Emmons, K. M. (2000). Health behaviors in a social context. In L. F. Berkman and I. Kawachi (Eds.), *Social Epidemiology.* New York, NY: Oxford University Press.

Ennis, N. E., Hobfoll, S. E., & Schroder, K. E. E. (2000). Money doesn't talk it swears: How economic stress and resistance resources impact inner-city women's depressive mood. *American Journal of Community Psychology, 28,* 149-173.

Enns, M. W., Inayatulla, M., Cox, B., et al. (1997). Prediction of suicide intent in aboriginal and non-aboriginal adolescent inpatients: A research note. *Suicide and Life-Threatening Behaviour, 27(2),* 218-224.

Epstein, S. (1979). The ecological study of emotions in humans. In P. Pliner, K. Blanksstein, & I. Spegel I. (Eds.), *Advances in the study of communication and : Perception of emotion in self and others* (pp.22-33). New York, NY: Plenum Press.

Erickson, D. H., Beiser, M., & Iacono, W. G. (1998). Social support predicts 5-year outcome in first-episode schizophrenia. *Journal of Abnormal Psychology, 107,* 681-685.

Ernst, J. M., & Cacioppo, J. T. (1999). Lonely hearts: Psychological perspectives on loneliness. *Applied and Preventative Psychology, 81,* 1-22.

Errera, P., Wyshak, G., & Jarecki, H. (1963). Psychiatric care in a general hospital

emergency room. *Archives of General Psychiatry, 9,* 105-112.

Essock, S., & Norquist, G. (1988). Towards a fairer prospective payment system. *Archives of General Psychiatry, 45,* 1041-1044.

Ettinger, A. B., Reed, M. L., Goldberg, J. F., & Hirschfeld, R. M. A. (2005). Prevalence of bipolar symptoms in epilepsy vs other chronic health disorders. *Neurology, 65,* 535-540.

Ettner, S. L., & Grzywacs, J. G. (2003). Socioeconomic status and health among Californians: An examination of multiple pathways. *American Journal of Public Health, 93*(3), 441-444.

Faden, V. B., Day, N. L., Windle, M., Windle, R., Grube, J. W., Molina, B. S. G., Pelham, W. E., Gnagy, E. M., Wilson, T. K., Jackson, K. M., & Sher, K. J. (2004). Collecting longitudinal data through childhood, adolescence, and young adulthood: Methodological challenges. *Alcoholism: Clinical and Experimental Research, 28*(2), 330-340. (2005).

Falk, A., Hanson, B., Isacsson, S., & Ostergren, P. (1992). Job strain and mortality in elderly men: Social network, support, and influence as buffers. *American Journal of Public Health, 82*(8), 1136-1139.

Falloon, I. R. H., Roncone, R. Held, T., et al. (2002). An international overview of family interventions: Developing effective treatment strategies and measuring their benefits for patients, carers, and communities. In H. P. Lefly, & D. L. Johnson, (Eds.), *Interventions in Mental Illness: International Perspectives.* Westport, CON: Praeger.

Falloon, I. R., & Marshall, G. N. (1983). Residential care and social behavior: A study of rehabilitation needs. *Psychological Medicine, 13*(2), 341-347.

Faris, R. E. (1934). Cultural isolation and the schizophrenic personality. *American Journal of Sociology, 39,* 155-164.

Faris, R. E., & Dunham, H. W. (1939). *Mental disorders in urban areas.* Chicago: University of Chicago Press.

Farmer, S. (1987). Medical problems of chronic patients in a community support program. *Hospital and Community Psychiatry, 38,* 745-749.

Feather, N. (1989). Reported changes in behavior after job loss in a sample of older unemployed men. *Australian Journal of Psychology, 41*(2), 175-185.

Feather, N. T., & Bond, M. J. (1983). Time structure and purposeful activity among employed and unemployed university graduates. *Journal of Occupational Psychology, 56,* 241-254.

Feldman, S. I., Downey, G., & Schaffer-Neitz, R. (1999). Pain, negative mood, and perceived support in chronic pain patients: A daily diary study of people with reflex sympathetic dystrophy syndrome. *Journal of Consulting and Clinical Psychology, 67,* 776-785.

Felker, B., Yazel, J. J., & Short, D. (1996). Mortality and medical comorbidity among psychiatric patients: A review. *Psychiatric Services, 47,* 1356-1363.

Field, D. (1999). Across-cultural perspective on continuity and change in social relations in old age: Introduction to a special issue. International. *Journal of Aging and Human Development, 48,* 257-261.

Finch, J. F., Okun, M. A., Pool,G. J., & Ruehlman, L. S. (1999). A comparison of the influence of conflictual and supportive social interactions on psychological distress. *Journal of Personality, 67,* 581-622.

Finlay-Jones, R., & Burvill, P. (1979). Women, work and minor psychiatric morbidity. *Social Psychiatry, 14,* 53-57.

Finlayson, A. (1976). Social networks as coping resources. *Social Science and Medicine, 10,* 97-103.

Fiore, J., Coppel, D., Becker, J., & Cox, G. (1986). Social support as a multifaceted concept: Examination of important dimensions for adjustment. *American Journal of Community Psychology, 14*(1), 93-111.

Firzpatrick, R., Newman, S., Lamb, R., & Shipley, M. (1988). Social relationships and psychological well-being in rheumatoid arthritis. *Social Science and Medicine, 27,* 399-403.

Fisher, C. (1982). *To dwell among friends: Personal networks in town and city.* Chicago: University of Chicago Press.

Flannery, R. B., Hanson, M., A., Penk, W. E., et al. (1997). Hospital downsizing and patients' assaults on staff. *Psychiatric Quarterly, 68(1),* 67-76.

Fletcher, A. C., Hunter, A. G., & Eanes, A. Y. (2006) Depressive symptomatology, exposure to violence, and the role of social capital among African American adolescents. *American Journal of Orthopsychiatry, 75*(2), 262-274.

Foa, E. B., Cashman, L., Jaycox, L., & Perry, K. (1997). The Validation of a self-report measure of posttraumatic stress disorder: The posttraumatic diagnostic scale.

Psychological Assessment, 9(4), 445-451.

Forbes, A., & Roger, D. (1999). Stress, social support, and fear of disclosure. *British Journal of Health Psychology, 4,* 165-179.

Formoso, D., Gonzales, N. A., & Aiken, L. S. (2000). Family conflict and children's internalizing and externalizing behavior: Protective factors. *American Journal of Community Psychology, 28,* 175-199.

Frank, J. (1973). *Persuasion and healing.* Baltimore: Johns Hopkins University Press.

Franklin, J., Kittredge, L., & Thrasher, J. (1975). A survey of factors related to mental hospital readmissions. *Hospital and Community Psychiatry, 26*(11), 749-751.

Fraser, M. W., Nelson, K. E., & Rivard, J. C. (1997). Effectiveness of family preservation services. *Social Work Research, 21(3),* 138-153.

Fraskovaik, P., & Segal, S. (2002). Substance use and mental disorder diagnostic profiles in a sample of tong-term self-help agency users. *American Journal of Orthopsychiatry, 72,* 72, 232-240.

Fratiglioni, L., Wang, H. X., Ericsson, K., Mayten, M., & Wimblad, B. (2000). Influence of social network on occurrence of dementia: A community-based longitudinal study. *Lancet, 355,* 1315-1319.

Frazier, P. A., Tix, A. P., & Barron, K. E. (2004). Testing moderator and mediator effects in counseling psychology. *Journal of Counseling Psychology, 51*(1), 115-134.

Freeland, K. E., Skala, J. A., Carney, R. M., Raczynski, J. M., Barr Taylor, C., Mendes de Leon, C. F., Ironson, G., Youngblood, M. E., Krishnan, K. R. R., & Veith, R. C. (2002). The depression interview and structured Hamilton (DISH): rationale,

development, characteristics and clinical validity. *Psychosomatic Medicine, 64,* 897-905.

Freeman, H. (1980). Coping with schizophrenia. *British Journal of Hospital Medicine, 23,* 54-58.

Frese, M. (1987). Alleviating depression in the unemployed: Adequate financial support, hope, and early retirement. Special Issue: Unemployment and health. *Social Science and Medicine, 25*(2), 213-215.

Frese, M., & Mohr, G. (1987). Prolonged unemployment and depression in older workers: A longitudinal study of intervening variables. *Social Science and Medicine, 25,* 173-178.

Freze, M. (1999). Social support as a moderator of the relationship between work stressors and psychological dysfunctioning: A longitudinal study with objective measures. *Journal of Occupational Health Psychology, 4*(3), 179-192.

Friedman, R. S. (1983). Hospital treatment of psychiatric emergencies. *Psychiatric Clinics of North America, 6*(2), 293-303.

Friedrich, R. M., Hollingsworth, B., Hradek, E., et al. (1999). Family and client perspectives on alternative residential settings for persons with severe mental illness. *Psychiatric Services, 50(4),* 509-514.

Friis, R., & Nanjundappa, G. (1986). Diabetes, depression and employment status. *Social Science and Medicine, 23*(5), 471-475.

Fryer, D. (1988). The experience of unemployment in social context. In S. Fisher & J. Reason (Eds.), *Handbook of life stress, cognition and health* (pp. 211-238).

Chichester: Wiley.

Fujita, D., & Kanaoka, M. (2003). Relationship between social support, mental health and health care consciousness in developing the industrial health education of male employees. *Journal of Occupational Health, 45,* 392-399.

Gallo, L. C., Smith, T. W., & Ruiz, J. M. (2003). An interpersonal analysis of adult attachment style: Circumplex descriptions, recalled developmental experiences, self-presentation, and interpersonal functioning in adulthood. *Journal of Personality, 71,* 141-181.

Gallo, L. C., & Matthews, K. A. (2003). Understanding the association between socioeconomic status and physical health: Do negative emotions play a role? *Psychological Bulletin, 129*(1), 10-51.

Gander, A., & Jorgensen, L. (1990). Postdivorce adjustment: Social supports among older divorced persons. *Journal of Divorce, 13*(4), 37-52.

Garvin, V., Kalter, N., & Hansell, J. (1993). Divorced women: Individual differences in stressors, mediating factors, and adjustment outcomes. *American Journal of Orthopsychiatry, 63*(2), 232-240.

Gasking, D. (1955). Causation and recipes. *Mind, 64,* 479-487.

Gatchel, R. J. (2004). Comorbidity of chronic pain and mental health disorders: The biopsychosocial perspective. *American Psychologist, 59,* 795-805.

Geddes, J., Mercer, G., Frith, C. D., MacMillan, F., Owens, D. G. C., & Johnstone, E. C. (1994). Prediction of outcome following a first episode of schizophrenia: A follow-up study of Northwick Park first episode study subjects. *British Journal of*

Psychiatry, 165, 664-668.

Gee, C. B., & Rhodes, J. E. (2003). Adolescent mothers' relationship with their children's biological fathers: Social support, social strain, and relationship continuity. *Journal of Family Psychology, 17*(3), 370-383.

Gen, T., & Astan, G. (2006). Social support, locus of control and depressive symptoms in hemodialysis patients. *Scandinavian Journal of Psychology, 47*(3), 203-208.

George, L. K., Blazer, D. G., Hughes, D. C., & Fowler, N. (1989). Social support and the outcome of major depression. *British Journal of Psychiatry, 154,* 478-485.

Geronimus, A. T. (2000). To mitigate 'resist' or undo: Addressing structural influences on the health of urban populations. *American Journal of Public Health, 90*(6), 601-611.

Gerson, S., & Bassuk, E. (1980). Psychiatric emergencies: An overview. *American Journal of Psychiatry, 137,* 1-11.

Gibbons, F. X. (1999). Social comparison as a mediator of response shift. *Social Science and Medicine, 48*(11), 1517-1530.

Gidron, B., Guterman, N. B., & Hartman, H. (1990). Stress and coping patterns of participants and non-participants in self-help groups for parents of the mentally ill. *Community Mental Health Journal, 26,* 483-495.

Gigliotti, E. (2002). A confirmation of the factor structure of the Norbeck Social Support Questionnaire. *Nursing Research, 51,* 276-284.

Gigliotti, E., (2004). Types and sources of social support and maternal-student role stress in married associate degree nursing students. *Issues in Mental health Nursing, 25,*

415-432.

Gigliottie, E. (2006). A confirmatory factor analysis of situation-specific Norbeck Social Support Questionnaire items. *Nursing Research, 55*(3), 217-222.

Gilley, O. W., & Leon, R. P. (1991). A two-stage imputation procedure for item nonresponse in surveys. *Journal of Business Research, 22,* 281-292.

Gillis, K, Russell, M. B., & Busby, K. (1997). Factors associated with unplanned discharge from psychiatric day treatment programs a multicenter study. *General Hospital Psychiatry, 19,* 355-361.

Gillis, M. E., Lumley, M. A., Mosley-Williams, A., Leisen, J. C. C., & Roehrs, T. (2006). The health effects of at-home written emotional disclosure in fibromyalgia: A randomized trial. *Annals of Behavioral Medicine, 32*(2), 135-146.

Gitlin, M. J., Swendsen, J., Heller, T. L., & Hammen, C. (1995). Relapse and impairment in bipolar disorder. *American Journal of Psychiatry, 152,* 1635-1640.

Glick, M., & Zigler, E. (1986). Premorbid social competence and psychiatric outcome in male and female nonschizophrenic patients. *Journal of Consulting and Clinical Psychology, 54*(3), 402-403.

Glick, I. D., Burti, L., Suzuki, K., & Sacks, M. (1991). Effectiveness of psychiatric care: I. A cross-national study of the process of treatment outcomes of major depressive disorder. In S. M. Mirin, J. T. Gossett, & M. C. Grob (Eds.), *Psychiatric treatment: Advances in outcome research* (pp. 107-126). Washington, DC: American Psychiatric Press.

Goering, P., Durbin, J., Sheldon, T. S., Ochocka, J., Nelson, G., & Krupa, T. (2006). Who

uses consumer-run self-help organizations? *American Journal of Orthopsychiatry, 76*(3), 367-373.

Goffman, E. (1961). *Asylums*. Garden City, NY: Doubleday.

Gogievski-Juijvesteijn, M. J., Bakker, A. B., Schaufeli, W. B., 7 van der Heijden, P. G. M. (2005). Finances and well-being: A dynamic equilibrium model of resources. *Journal of Occupational Health Psychology,* 10(3), 210-224.

Goldsmith, S. K., Pellmar, T. C., Klienman, A. M., & Bunney, W. E. (2002). *Reducing suicide: A national imperative.* Washington, DC: National Academy Press.

Goldstein, T. R., Miklowitz, D. J., & Mullen, K. L. (2006). Social skills knowledge and performance among adolescents with bipolar disorder. *Bipolar Disorder, 8*(4), 350-361.

Goodman, S., & Johnson, M. (1986). Life problems, social supports, and psychological functioning of emotionally disturbed and well low-income women. *Journal of Community Psychology, 14,* 150-158.

Goodman, L., Thompson, K., Weinfurt, K., Corl, S., Acker, P., Meuser, K., & Rosenberg, S. (1999). Reliability of reports of violent victimization and PTSD among men and women with SMI. *Journal of Traumatic Stress, 12,* 587-599.

Gottlieb, B. H. (2000). Selecting and planning support interventions. In S. Cohen, L. Underwood, & Gottlieb (Eds.), *Social Support Measurement and Interventions: A guide for health and social scientists (pp. 195-220).* New York, NY: Oxford University Press.

Goodman, G. S., Ghetti, S., Quas, J. A., Edelstein, R. S., Alexander, K. W., Redlich, A.

D., et al. (2003). A prospective study of memory for child sexual abuse: New findings relevant to the repressed-memory controversy. *Psychological Science, 14,* 113-118.

Goodwin, C. J. (1995). *Research in psychology: Methods and design.* New York, NY: John Wiley & Sons.

Goodwin, F. K., & Ghaemi, S. N. (1998). Understanding manic-depressive illness (commentary). *Archives of General Psychiatry, 55(1),* 23-25.

Goodwin, F., & Jamison, K. (1990). *Manic-Depressive Illness.* New York NY: Oxford University Press.

Goodwin, R. (2006). Age and social support perception in Eastern Europe: Social change and support in four rapidly changing countries. *British Journal of Social Psychology, 45*(4), 799-815.

Gordon, R., Vijay, J., Sloate, S., Burket, R., & Gordon, K. (1985). Aggravating stress and functional level as predictors of length of psychiatric hospitalization. *Hospital and Community Psychiatry, 36*(7), 773-774.

Gore, S. (1978). The effects of social support in moderating the health consequences of unemployment. *Journal of Health and Social Behavior, 19,* 157-165.

Gore, S. (1981). Stress-buffering functions of social supports: An appraisal and clarification of research models. In B. S. Dohrenwend and B. P. Dohrenwend (Eds.), *Stressful life events and their contexts* (pp. 202-222). New York, NY: Oxford University Press.

Gore, S., & Mangione, T. (1983). Social roles, sex roles, and psychological distress:

Additive and interactive models of sex difference. *Journal of Health and Social Behavior, 24,* 300-312.

Gottesman, I. I., & Shields, J. (1982). *Schizophrenia: The epigenetic puzzle.* Cambridge: Cambridge University Press.

Gottfredson, L. S. (2004). Intelligence: Is it the epidemiologists' elusive "fundamental cause" of social class inequalities in health? *Journal of Personality and Social Psychology, 86*(1), 174-199.

Gottleib, B. H. (1981). Preventive interventions involving social networks and social support. In B. H. Gottleib (Ed.), *Social networks and social support*, (pp. 201-232). Beverly Hills: Sage.

Goulding, J. (1988). Gender differences in depressive symptoms. *Psychology of Women Quarterly, 12,* 61-74.

Graney, M. J. (2000). The reciprocal relationship between disability and depression. *Journal of the American Geriatrics Society, 48,* 452-453.

Grant, I, Patterson, T. L., & Yager, J. (1988). Social supports in relation to physical health and symptoms of depression in the elderly. *American Journal of Psychiatry, 145*(10), 1254-1258.

Greene, R. (1987). Ethnicity and MMPI performance: A review. *Journal of Consulting and Clinical Psychology, 55,* 497-512.

Griffiths, K. M., Christensen, H., Jorm, A. F., et al. (2004). Effect of web-based depression literacy and cognitive behavioural therapy interventions on stigmatising attitudes to depression: Randomised controlled trial. *British Journal of Psychiatry,*

185, 342-349.

Grivell, J. E., Murray, L. T., MacPherson, P. S., et al. (2000). Sarasons' Social Support Questionnaire: Do the First Six Questions Suffice? Presented at the Red River Psychology Conference at North Dakota State University in Fargo North Dakota in March 2000.

Grivell, J. E., Murray, L. T., Frankel, S. I., et al. (2000). Sarasons' Social Support Questionnaire: Three Competing Six-item Versions. Presented at the Prairie Undergraduate Conference at the University of Winnipeg in Winnipeg Manitoba in April 2000.

Grunefeld, B., & Salveson, C. (1968). Functional psychoses and social status. *British Journal of Psychiatry, 114,* 733-737.

Guay, S., Billette, T., & Marchand, A. (2006). Exploring the links between posttraumatic stress disorder and social support: Processes and potential research avenues. *Journal of Traumatic Stress, 19*(3), 327-338.

Gurel, L., & Lorei, T. W. (1972). Hospital and community ratings of psychopathology as predictors of employment and readmission. *Journal of Consulting and Clinical Psychology, 39,* 286-291.

Gurung, R. A. R., Taylor, S. E., & Seeman, T. E. (2003). Accounting for changes in social support among married older adults: Insights from the MacArthur Studies of Successful Aging. *Psychology and Aging, 18*(3), 487-496.

Guyll, M., & Madon, S. (2003). Trait hostility: The breadth and specificity of schema effects. *Personality and Individual Differences, 34,* 681-693.

Haan, M., Kaplan, G., & Camacho, T. (1987). Poverty and health: Prospective evidence from the Alameda County study. *American Journal of Epidemiology, 125,* 989-998.

Hadjistavropoulos, T., & Bieling, P. (2001). File review consultation in the adjudication of mental health and chronic pain disability claims. *Consulting Psychology Journal: Practice and Research, 53*(1), 52-63

Haefner, H., & Maurer, K. (2000). The early course of schizophrenia: New concepts for early intervention. In G Andrews, & S. Henderson (Eds.), *Unmet need in psychiatry: Problems Resources, Responses (pp.218-232).* New York, NY: Cambridge University Press.

Hafner, H. O., & Boker, W. (1973). Mentally disordered violent offenders. *Social Psychiatry, 8*(4), 220-229.

Hafner, H., Nowotny, B., & Loffler, W. (1995). When and how does schizophrenia produce social deficits? *European Archives of Psychiatry and Clinical Neuroscience, 246,* 17-28.

Hagedoorn, M., Kuijer, R. G., Buunk, B. P., DeJong, G. M., Wobbes, T., & Sandermand, R. (2000). Marital satisfaction in patients with cancer: Does support from intimate partners benefit those who need it most? *Health Psychology, 19*(3), 274-282.

Hagerty, B. M., & Williams, A. R. (1999). The effects of sense of belonging, social support, conflict, and loneliness on depression. *Nursing Research, 48*(4), 215-219.

Hall, C. (1998). *Mental health's shake-up.* London: Daily Telegraph.

Hall, E., & Johnson, J. (1988). Depression in unemployed Swedish women. *Social Science*

and Medicine, 27(12), 1349-1355.

Hall, L. A., Williams, C. A., & Greenberg, R. S. (1985). Supports, stressors, and depressive symptoms in low-income mothers of young children. *American Journal of Public Health, 75,* 518-522.

Hall, O., & Royse, D. (1987). Mental health needs assessment with social indicators: An empirical case study. *Administration in Mental Health, 15*(1), 36-46.

Hamilton, V., Hoffman, W., Broman, C., & Rauma, D. (1993). Unemployment, distress, and coping: A panel study of autoworkers. *Journal of Personality and Social Psychology, 65*(2), 234-247.

Hammer, M., Gutwirth, L., & Phillips, S. (1982). Parenthood and social networks: A preliminary view. *Social Science and Medicine, 16*(24), 2091-2100.

Hanlon, M. D. (1981). Determinants of primary group assistance during unemployment. *Journal of Sociology and Social Welfare, 8*(3), 623-639.

Hanson, B. S., & Ostergren, P. (1987). Different social networks and social support characteristics, nervous problems and insomnia: Theoretical and methodological aspects on some results from the population study "Men born in 1914," Malmo, Sweden. *Social Science Medicine, 25*(7), 849-859.

Harris, A. (1988). Physical disease and schizophrenia. *Schizophrenia Bulletin, 14,* 85-96.

Harrow, M., Goldberg, J. F., Grossman, L. S., & Meltzer, H. Y. (1990). Outcome in manic disorders: A naturalistic follow-up study. *Archives of General Psychiatry, 47,* 665-671.

Harter, S. (1999). *The Construction of the Self: A Developmental Perspective.* New York,

NY: Guilford Press.

Harvey, P. D., Powchik, P., Parrella, M., et al. (1997). Symptom severity and cognitive impairment in chronically hospitalised geriatric patients with ive disorders. *British Journal of Psychiatry, 170,* 369-374.

Harwitz, D. & Ravizza, L. (2000). Suicide and depression. *Emerging Medical and Clinical Journal of North America, 18,* 263-271.

Hatfield, A. B., Gearon, J. S., & Coursey, R. D. (1996). Family members' ratings of the use and value of mental health services: Results of a national NAMI survey. *Psychiatric Services, 47*(8), 825-831.

Haughton McNeill, L., Kreuter, M. W., & Subramanian, S. V. (2006). Social environment and physical activity: A review of concepts and evidence. *Social Science & Medicine, 63*(4), 1011-1022.

Hawkley, L. C., Burleson, M. H., Berntson, G. G., & Cacioppo, J. T. (2003). Loneliness in everyday life: Cardiovascular activity, psychosocial context, and health behaviors. *Journal of Personality and Social Psychology, 85,* 105-120.

Hawton, K. (1987). Assessment of suicide risk. *British Journal of Psychiatry, 150,* 145-153.

Hays, R. D., Sherbourne, C. D., & Mazel R. M. (1993). The Rand 36-Item Health Survey. *Health Economics, 2,* 217-227.

Haywood, T. W., Kravitz, H. M., Grossman, L. S., et al. (1995). Predicting the'Revolving Door' phenomenon among patients with schizophrenia, schizoaffective disorders. *American Journal of Psychiatry, 152,* 856-861.

Heikkinen, R., & Kauppinen, M. (2004). Depressive symptoms in late life: A 10-year follow-up. *Archives of Gerontology and Geriatrics, 38,* 239-250.

Helgason, V. S., Cohen, S., Schultz, R., & Yasko, J. (2001). Long-term effects of educational and pert discussion group interventions on adjustment to breast cancer. *Health Psychology, 20*(5), 387-392.

Heller, K., Swindle, R. W., & Dusenbury, L. (1986). Components of social support process: Comments and integration. *Journal of Consulting and Clinical Psychology, 54,* 466-470.

Heller, D., Watson, D., & Ilies, R. (2004). The role of person versus situation in life satisfaction: A critical examination. *Psychological Bulletin, 130*(4), 574-600.

Helliwell, J. F. (2003). *Well-being and social capital: Does suicide pose a problem?* (Unpublished manuscript).

Helliwell, J. F., & Putnam, R. D. (2004). The social context of well-being. *Philosophical Transactions of the Royal Society of London, 359,* 1435-1446.

Hellon, C. P., & Solomon, M. I. (1980). Suicide and age in Alberta, Canada, 1951 to 1977: The changing profile. *Archives of General Psychiatry, 37,* 505-510.

Helzer, J. E., Clayton, P. J., Pambakian, R., & Woodruff, J. R. (1978). Concurrent diagnostic validity of a structured psychiatric interview. *Archives of General Psychiatry, 35,* 849-853.

Henderson, S., Byrne, D., & Duncan-Jones, P. (1981). *Neurosis and the Social Environment.* Diego, CA: Academic Press.

Henderson, J., Jr., & Pollard, C. (1992). Prevalence of various depressive symptoms in a

sample of the general population. *Psychological Reports, 71,* 208-210.

Henderson, A. S. (1998). Social support: Its present significance for psychiatric epidemiology. In B. P. Dohrenwend (Ed.), *Adversity stress and psychopathy* (pp 390-397). New York, NY: Oxford University Press.

Henderson, S., Duncan-Jones, P., McAuley, H., & Ritchie, K. (1978). The patient's primary group. *British Journal of Psychiatry, 132,* 74-86.

Hendryx, M. S., & Rohland, B. M. (1997). Psychiatric hospitalization decision making by CMHC staff. *Community Mental Health Journal, 33*(1), 63-72.

Henry, C. S., Stephenson, A. L., Hanson, M. F., et al. (1993). Adolescent suicide and families: An ecological approach. *Adolescence, 28,* 291-308.

Heponiemi, T., Elovaninio, M., Kivimski, M., Pulkki, L., Puttonen, S., & Keltikangas-Jsrvinen, L. (2006). The longitudinal effects of social support and hostility on depressive tendencies. *Social Sciences & Medicine, 63*(5) 1374-1382.

Hibbard, M. R., Cantor, J., Charatz, H., Rosenthal, T., Ashman, T., Gundersen, N., et al. (2002). Peer support in the community: Initial findings of a mentoring program for individuals with traumatic brain injury and their families. *Journal of Head Trauma Rehabilitation, 17,* 112-131.

Hirchfeld, R. M. A. & Russell,J. M. (1997). Assessment and treatment of suicidal patients. *New England Journal of Medicine, 337,* 910-915.

Hirsch, S. (1988). *Psychiatric bed and resources: Factors influencing bed use and service planning.* London: Gaskell.

Hobfoll, S. E., Johnson, R. J., Ennis, N., & Jackson, A. P. (2003). Resource loss, resource

gain, and emotional outcomes among inner city women. *Journal of Personality and Social Psychology, 84,* 632- 643.

Hobfoll, S., & Jackson, A. (1991). Conservation of resources in community intervention. *American Journal of Community Psychology, 19*(1), 111-121.

Hobfoll, S. E. (2002). Social and psychological resources and adaptation. *Review of General Psychology, 6,* 307-324.

Hocking, B. (2003). Reducing mental illness stigma and discrimination - everybody's business. *The Medical Journal of Australia, 178*(9), S47-S48.

Hodgins, S. (1993). *Mental disorder and crime.* Newbury Park, CA: Sage.

Hoffmann, F., & Mastrianni, X. (1993). The role of supported education in the inpatient treatment of young adults: A two-site comparison. Special Issue: Supported education. *Psychosocial Rehabilitation Journal, 17*(1), 109-119.

Holahan, C. J., Moos, R. H., Holahan, C. K., & Cronkite, R. C. (1999). Resource loss, and resource gain, and depressive symptoms: A 10-year model. *Journal of Personality and Social Psychology, 77,* 620-629.

Holahan, C. J., Moos, R. H., Holahan, C. K., & Cronkite, R. C. (2000). Long-term post-treatment functioning among patients with unipolar depression: An integrative model. *Journal of Consulting and Clinical Psychology, 68,* 226-232.

Holinger, P. C., Offer, A., & Zola, M. A. (1988). A prediction model of suicide among youth. *Journal of Nervous and Mental Disorders, 176,* 275-279.

Holley, H. L., Jeffers, B., & Hodges, P. (1997). Potential for community relocation among residents of Alberta's psychiatric facilities: A needs assessment. *Canadian Journal*

of Psychiatry, 42, 750-757.

Hollingshead, A. B. (1957). *Two-factor index of social position (Updated four-factor version available from Department of Sociology).* New Haven, CT: Yale University.

Hollingshead, A. B., & Redlich, F. C. (1958). *Social class and mental illness: A community study.* New York, NY: Wiley.

Hollingsworth, E. J., & Sweeney, J. K. (1997). Mental health expenditures for services for people with severe mental illnesses. *Psychiatric Services, 48*(4), 485-490.

Holsten, F., & D'Elia, G. (1985). Patients with multiple admissions in a community mental health service in Western Norway. *Social Psychiatry, 20*(2), 55-59.

Hopkins, M. T. (1971). Patterns of self-destruction among the orthopedically disabled. *Rehabilitation and Research Practice Review, 3*(1), 5-16.

Hormozdi, S. M., Ward, M. F., Ramos, A., Smith, C., Panabianco, N., Projansky, M., Litroff, A., Kuznewski, S., & Ricardi, D. (2006). A quantitative assessment of the presence of hostility, anger, depression, social support, hazardous alcohol use and comorbidity in the adult trauma population. *Annals of Emergency Medicine, 48*(4), S83.

House, J., Robbins, C., & Metzner, L. (1982). The association of social relationships and activities with mortality: Prospective evidence from the Tecumseh community health study. *American Journal of Epidemiology, 116,* 123-140.

House, J., Landis, K., & Umberson, D. (1988). Social relationships and health. *Science, 241,* 540-544.

House, J., & Kahn, R. (1985). Measures and concepts of social support. In S. Cohen & S. L. Syme (Eds.), *Social support and health* (pp. 83-108). San Diego, CA: Academic Press.

Houston, T. K., Cooper, L. A., & Ford, D. E. (2002). Internet support groups for depression: A 1-year prospective cohort study. *American Journal of Psychiatry, 159,* 2062-2068.

Howe, G. W., Levy, M. L., & Caplan, R. D. (2004). Job loss and depressive symptoms in couples: Common stressors, stress transmission, or relationship disruption? *Journal of Family Psychology, 18*(4), 639-650.

Hu, T. W., Snowden, L. R., & Jerrell, J. J. (1992). Costs and use of public mental health services by ethnicity. *Journal of Mental Health Administration, 19,* 278-287.

Huber, G., Cross, G., Schluttler, R., & Linz, M. (1980). Longitudinal studies of schizophrenic patients. *Schizophrenia Bulletin, 6,* 592-605.

Hughes, J. W., Herwood, A., Blumenthal, J. A., Suarez, E. C., & Hinderliter, A. L. (2003). Hostility, Social support, and adrenergic receptor responsiveness among African-American and white men and women. *Psychosomatic Medicine, 65,* 582-587.

Hughes, M. E., Waite, L. J., Hawkley, L. C., & Cacioppo, J. T. (2004). A short scale for measuring loneliness in large surveys. Results from two population-based studies. *Research on Ageing, 26,* 655-672.

Hull, J. W., Yeomans, F., Clarkin, J., Li, C., & Goodman, G. (1996). Factors associated with multiple hospitalizations of patients with borderline personality disorder.

Psychiatric Services, 47, 638-641.

Huynh, C. L. (1992). *SASPA: A SAS computer program for path analysis.* Unpublished manuscript. Winnipeg, MB: University of Manitoba, Psychology Department.

Institute of Medicine, (2003). *The future of the public's health in the 21st century.* Washington, DC: National Academies of Press.

Irwin, R., Kamman, R., & Dixon, G. (1979). If you want to know how happy I am, you'll have to ask me. *New Zealand Psychologist, 8,* 10-16.

Iyer, S. N., Rothmann, T. L., Vogler, J. E., & Spaulding, W. D. (2005). Evaluating outcomes of rehabilitation for severe mental illness. *Rehabilitation Psychology, 50*(1), 43-55.

Jablensky, A. (1986). Epidemiology of schizophrenia: A European perspective. *Schizophrenia Bulletin, 12,* 52-73.

Jackson, D. J., Tweed, D. L., Goldsmith, H. F., Rosen, B. M., & Babigian, H. M. (1982). Mental health needs assessment using predicted psychiatric episode rates. In R. A. Bell, H. F. Goldsmith, E. Lin, R. K. Hirzel, & S. Sobel, (Eds.), *Social indicators for human service systems* (pp. 1-15). Louisville: University of Louisville.

Jahoda, M. (1982). *Employment and unemployment: A social psychological analysis.* Cambridge: Cambridge University Press.

Jang, Y., Haley, W. E., Small, B. J., & Mortimer, J. A. (2002). The role of mastery and social resources in the association between disability and depression in later life. *The Gerontologist, 42,* 807-813.

Jarvis, E. (1971). *Insanity and idiocy in Massachusetts: Report of the Commission on*

Lunacy, 1855. Cambridge, MA: Harvard University Press.

Johnson, S. L., Winett, C. A., Myer, B., Greenhouse, W. J., & Miller, I. (1999). Social support and the course of bipolar disorder. *Journal of Abnormal Psychology, 108*(4), 558-566.

Johnson, J. G., Cohen, P., Dohrenwend, B. P., Link, B. G., & Brook, J. S. (1999). A longitudinal investigation of social causation and social selection processes involved in the association between socioeconomic status and psychiatric disorders. *Journal of Abnormal Psychology, 108*(3), 490-499.

Johnson, S. L., Winett, C. A., Meyer, B., et al. (1999). Social support and the course of bipolar disorder. *Journal of Abnormal Psychology, 108,* 558-666.

Joiner, T., & Coyne, J. C. (1999). *The Interactive Nature of Depression: Advances in Interpersonal Approaches.* Washington, DC: American Psychological Association.

Jordan, L. C., Luke, D. A., Mowbray, C. T., et al. (1996). Correlates of functioning in a population with dual diagnoses: An examination of diagnosis and problem history. *The Journal of Mental Health Administration, 23,* 260-270.

Jorm, A. F., Allen, N. B., O'Donnell, C. P., Parslow, R. A., Purcell, R., & Morgan, A. J. (2006). Effectiveness of complementary and self-help treatments for depression in children and adolescents. *The Medical Journal of Australia, 185*(7), 368-372.

Judd, L. L., & Akiskal, H. S. (2000). Delineating the longitudinal structure of depressive illness: Beyond clinical subtypes and duration thresholds. *Pharmacopsychiatry, 33,* 3-7.

Junginger, J. (1990). Psychiatric hospitalization and community-based program

attendance. *Psychiatric Quarterly, 61,* 251-259.

Juujsrvi, S. (2006). The ethic of care development: A longitudinal study of moral reasoning among practical-nursing, social-work and law-enforcement students. *Scandinavian Journal of Psychology, 47*(3), 193-202.

Kafestios, K. (2006). Social support and well-being in contemporary Greek society: Examination of multiple indicators at different levels of analysis. *Social Indicators Research, 76*(1), 127-145.

Kafestios, K., & Sideridis, G. D. (2006). Attachment, social support and well-being in young and older adults. *Journal of Health Psychology, 11*(6), 863-876.

Kalla, O., Aaltonen, J., Wahlstrom, J., Lehtinen, V., Garcia Cabeza, I., & Gonzalez de Chavez, M. (2002). Duration of untreated psychosis and its correlates in first episode psychosis in Finland and Spain. *Acta Psychiatrica Scandinavica, 106,* 256-275.

Kaplan, G. A., Pamuk, E. R., Lynch, J. W., Cohen, R. D., & Balfour, J. L. (1996). Inequality in income and mortality in the United States: Analysis of mortality and potential pathways. *British Medical Journal, 312,* 999-1003.

Kaplan, G. A., Salonen, J. T., Cohen, R. D., Brand, R. D., Syme, S. L, & Puska, P. (1988). Social connections and mortality from all causes and cardiovascular disease: Prospective evidence from eastern Finland. *American Journal of Epidemiology, 128,* 370-380.

Kasl, S. V., Rodrigues, E., & Lasch, K. E. (1998). The impact of unemployment on health and well-being. In B. S. Dohrenwend and B. P. Dohrenwend (Eds.), *Stressful life*

events and their contexts (pp. 111-131). New York, NY: Oxford University Press.

Kates, N., Webb, S., & LePage P. (1991). Therapy begins at home: The psychiatric house call. *Canadian Journal of Psychiatry, 36,* 673-676.

Kawachi, I., & Berkman, L. F. (2001). Social ties and mental health. *Journal of Urban Health: Bulletin of the New York Academy of Medicine, 78,* 458-467.

Keck, P. E., McElroy, S. L., Strakowski, S. M., et al. (1998). 12-month outcome of patients with bipolar disorder following hospitalization for a manic or mixed episode. *American Journal of Psychiatry, 155(5),* 646-652.

Keith, P. M. (1985). Financial well-being of older divorced/separated men and women: Findings of a panel study. *Journal of Divorce, 9*(1), 61-72.

Keitner, G., Ryan, C., Miller, I., & Norman, W. (1992). Recovery and major depression: Factors associated with twelve month outcome. *American Journal of Psychiatry, 149*(1), 93-99.

Keller, M. B., Lavori, P. W., Endicott, J., Coryell, W., & Kelerman, G. L. (1983). "Double depression": Two-year follow-up. *American Journal of Psychiatry, 140,* 689-694.

Keller, M. B. (1988). Undertreatment of major depression. *Psychopharmacology Bulletin, 24,* 75-80.

Keller, M. B., Shapiro, R. W., Lavori, P. W., & Wolfe, N. (1982). Recovery in major depressive disorder: Analysis with the life table and regression models. *Archives of General Psychiatry,* 39, 905-910.

Kellner, R., & Sheffield, B. F. (1968). The use of self rating scales in a single-patient multiple cross-over trial. *British Journal of Psychiatry, 114,* 193-199.

Kelly, C. M., & Jorm, A. F. (2007). Stigma and mood disorders. *Current Opinion in Psychiatry, 20*(1), 13-16.

Kendall, M. G., & O'Muircheartaigh, C. A. (1977). *Path analysis and model building.* WFS Technical Bulletin, No. 2, Voorberg, Netherlands: International Statistical Institute.

Kendler, K. S., Kessler, R. C., Neal, M. C., Heath, A. C., & Eaves, L. J. (1993). The prediction of major depression in women: An integrated etiological model. *American Journal of Psychiatry, 150,* 1139-1148.

Kendler, K. S., & Karkowski-Shuman, L. (1997). Stressful life events and genetic liability to major depression: Genetic control of exposure to the environment? *Psychological Medicine, 27,* 539-547.

Kendler, K. S., Karkowski, L. M., & Prescott, C. A. (1999). Causal relationship between stressful life events and the onset of major depression. *American Journal of Psychiatry, 156,* 837-841.

Kerr, D. C. R., Preuss, L. J., & King, C. A. (2006). Suicidal adolescents' social support from family and peers: Gender-specific associations and psychopathology. *Journal of Abnormal Child Psychology, 34*(1), 99-110.

Kessler, R. C, House, J., & Turner, J. (1987). Unemployment and health in a community sample. *Journal of Health and Social Behavior, 28,* 51-59.

Kessler, R. C., & Cleary, P. (1980). Social class and psychological distress. *American Sociological Review, 45,* 463-478.

Kessler, R. C. (1982). A disaggregation of the relationship between socioeconomic status

and psychological distress. *American Sociological Review,* 47, 752-764.

Kessler, R. C., Turner, J., & House, J. (1989). Unemployment, reemployment, and emotional functioning in a community sample. *American Sociological Review, 54,* 648-657.

Kessler, R. C., & McRae, J. (1982). The effect of wives' employment on the mental health of men and women. *American Sociological Review, 47,* 216-231.

Kessler, R. C., & McLeod, J. (1985). Social support and mental health in community samples. In S. Cohen & S. L. Syrne (Eds.), *Social support and health* (pp. 219-240). Orlando, FL: Academic Press.

Kettner, P. M., Moroney; R. M., & Martin, L. L. (1990). Designing and managing programs. An effectiveness-based approach. In A. Lauffer & C. Garvin (Eds.), *Sage source books for the human services series*. Newbury Park, CA: Sage Publications.

Kiecolt-Glaser, J. K., & Newton, T. L. (2001). Marriage and health: his and hers. *Psychological Bulletin, 127,* 472-503.

Kielhofner,G., & Forsyth, K. (2004). Commentary on Cutchin's "Using Deweyan philosophy to rename and reframe adaptation-to-environment." *American Journal of Occupational Therapy, 58*(3), 313-314.

Kiesler, C. A. (1992). U.S. mental health policy-doomed to fail. *American Psychologist, 47,* 1077-1082.

King, G., Cathers, T., Brown, E., Specht, J. A., Willoughby, C., Miller Polgar, J., et al. (2003). Turning points and protective processes in the lives of people with chronic

disabilities. *Qualitative Health Research,10,* 734-749.

King, G., King, S., Rosenbaum, P., & Goffin, R. (1999). Family -centred caregiving and well-being of parents of children with disabilities: Linking processing with outcome. *Journal of Pediatric Psychology, 24,* 41-53.

King, G., Brown, E., & Smith, L. (2003). *Resilience: Learning from People with Disabilities and Turning Points in Their Lives.* Westport, CT: Praeger.

King, G., Willoughby, C., Specth, J. A., & Brown, E. (2006). Social support processes and the adaptation of individuals with chronic disabilities. *Qualitative Health Research, 16*(7), 902-925.

Kinicki, A., J., Prussia, R. E., & McKeen-Ryan, F. M. (2000). A panel study of coping with involuntary job loss. *Academy of Management Journal, 43,* 0-100.

Kirk, S. (1976). Effectiveness of community services for discharged mental hospital patients. *American Journal of Orthopsychiatry, 46,* 646-659.

Kirk, S., & Therrein, M. (1975). Community mental health myths and the fate of former hospitalized patients. *Psychiatry, 38,* 209-217.

Kirsh, S. (1983). *Unemployment: Its impact on body and soul.* Toronto: Canadian Mental Health Association.

Klein, A. R., Cnaan, R. A., & Whitecraft, J. (1998). Significance of peer social support with dually diagnosed clients: Findings from a pilot study. *Research on Social Work Practice, 8,* 529-551.

Klonoff, E. A., Landrine,H., & Ullman, B. J. (1999). Racial discrimination and psychiatric symptoms among Blacks. *Cultural Diversity and Ethnic Minority Psychology, 5,*

329-339.

Knapp, M., Chisholm, D., Astin, J., et al. (1997). The cost consequences of changing the hospital- community balance: The mental health residential care study. *Psychological Medicine, 27,* 681-692.

Knapp, M., Almond, S., & Percudani, M. (1999). Costs of schizophrenia: A review. In M. Maj and N. Sartorius (Eds.), *Schizophrenia (pp. 407-454).*

Knapp, M., Beecham, J., Fenyo, A., et al. (1995). Community mental health care for former hospital in-patients: Predicting costs from needs and diagnoses. *The British Journal of Psychiatry, 166(suppl. 27),* 10–18.

Koenig, H. G., & Kuchibhatla, M. (1998). Use of health services by hospitalized medically ill depressed elderly patients. American Journal of Psychiatry, 155(7), 871-877.

Koenig, H. G., George, L. K., & Meador, K. G. (1997). Use of antidepressants by nonpsychiatrists in the treatment of medically ill hospitalized depressed elderly patients. *American Journal of Psychiatry, 154,* 1369-1375.

Koh, K. B., Kim, D. K., Kim, S. Y., & Park, J. K. (2005). The relation between anger expression, depression and somatic symptoms in depressive disorders and somatoform disorders. *Journal of Clinical Psychiatry, 66*(4), 485-91.

Kohn, R., Dohrenwend, B., & Mirotzinik, J. (1998). Epidemiological findings on selected psychiatric disorders in the general population. In B. P. Dohrenwend (Ed.), *Adversity, Stress, and Psychopathology,* (pp. 235-284). New York, NY: Oxford University Press.

Komiya, N., Good, G. E., & Sherrod, N. B. (2000). Emotional openness sd s predictor of

college students' attitudes toward seeking psychological help. *Journal of Counseling Psychology, 47,* 138-143.

Kop, W. (2005). Psychological interventions in patients with coronary heart disease. In L. C. James and R. A. Folen (Eds.), *The Primary Care Consultant: The Next Frontier for Psychologists in Hospitals and Clinics (pp. 61-81).* Washington, DC: American Psychological Association.

Kopelowicz, A., Liberman, R. P., & Zarate, R. (2006). Recent advances in social skills training for schizophrenia. *Schizophrenia Bulletin, 32*(1), S12-S23.

Korkeila, J. A., Karlsson, H., & Kujari, H. (1995). Factors predicting readmissions in personality disorders and other nonpsychotic illnesses. *Acta Psychiatrica Scandinavica, 92,* 138-144.

Koukouli, S., Vlachonikolis, I. G., & Philalithis, A. (2002). Socio-demographic factors and self-reported functional status: The significance of social support. *BCM Health Services Research, 2*(1), 1-23.

Kovess, V., Murphy, H., & Tousignant, M. (1987). Urban-rural comparisons of depressive disorders in French Canada. *Journal of Nervous and Mental Disease, 175*(8), 457-466.

Kposowa, A. J. (2000). Marital status and suicide in the national longitudinal mortality study. *Epidemiology and Community Health, 54,* 254-261.

Krause, N. (2005). Exploring age differences in the stress-buffering function of social support. *Psychology and Aging, 20*(4), 714-717.

Krause, N., Liang, J., & Keith, V. (1990). Personality, social support, and psychological

distress in later life. *Psychology and Aging, 5*(3), 315-326.

Krause, N. (2006). Neighborhood deterioration, social skills, and social relationships in late life. *International Journal of Aging and Development, 62*(3), 185-207.

Krause, N., Morgan, D. L., Chatters, L., & Meltser, T. (2000). Negative interaction in the church: Insights from focus groups. *Review of Religious Research, 41,* 510-533.

Krause, N., Ellison, C. G., & Wulff, K. M. (1998). Church-based emotional support, negative interaction, and psychological well-being: Findings from a national sample of Presbyterians. *Journal for the Scientific Study of Religion, 41,* 510-533.

Krause, N. (1999).Mental disorders in late life: Exploring the influence of stress and socioeconomic status. In C. S. Aneshensel, & J. C. Phelan, *Handbook of the Sociology of Mental Health, (pp. 183-208).* New York, NY: Plenum.

Krause, N., Ellison, C. G., & Shaw, B. A. (2001). Church-based social support and religious coping. *Journal for the Scientific Study of Religion, 40*(4), 637-656.

Krause, N., Shaw, B. A., & Cairney, J. (2004). A descriptive epidemiology of lifetime trauma and the physical health status of older adults. *Psychology and Aging, 19*(4), 637-648.

Krause, N., & Shaw, B. A. (2002). Welfare participation and social support in late life. *Psychology and Aging, 17*(2), 260-270.

Kreuter, M. W, & Lezin, N. (2002). Social capital theory: Implications for community-based health promotion. In R. J. Clemente, R. A. Crosby, and M. C. Kegler (Eds.), *Emerging Theories in Health Promotion Practice and Research: Strategies for Improving Public Health.* San Fransisco, CA: Jossey-Bass.

Kubany, E. S., Leisen, M. B., Kaplan, A. S., & Kelly, M. P. (2000). Validation of a brief measure of posttraumatic stress disorder: The Distressing Event Questionnaire (DEQ). *Psychological Assessment, 12*(2), 197-209.

Kunitz, S. J. (2004). Social capital and health. *British Medical Bulletin, 69,* 61-73.

Kurtz,L. F. (1997). *Self-help and Support Groups: A Handbook for Practitioners.* California, CA: Sage Publications.

Kuypers, J. A., & Bengtson, V. L. (1973). Social breakdown and competence: A model of normal aging. *Human Development, 16*(3), 181-201.

La Greca, A. M., & Bearman, K. J. (2002). The Diabetes Social Support Questionnaire-Family Versions: Evaluating adolescents' diabetes-specific support from family members. *Journal of Pediatric Psychology, 27*(8), 665-676.

Labbate, L. A., & Doyle, M. E. (1997). Recidivism in major depressive disorder. *Psychotherapy and Psychosomatics, 66*(3), 145-149.

Laessie, R. G., Yassouridis, A., & Pfister, H. (1988). Sociodemographic characteristics and length of psychiatric hospital stay: Application of a proportional hazards model. *Acta Psychiatrica Scandinavica, 77,* 349-351.

Laing, J., Kahana, E., & Doherty, E. (1980). Financial well-being among the aged: A further elaboration. *Journal of Gerontology, 35,* 409-420.

Lakey, B., & Cohen, S. (2000). Social support theory and measurement. In S. Cohen, L. G. Underwood, & B. H. Gottlieb (Eds.), *Social support measurement and intervention (pp. 29-52).* Oxford: Oxford University Press.

Lakey, B., & Cassady, P. B. (1990). Cognitive processes in perceived social support.

Journal of Personality and Social Psychology, 59(2), 337-343.

Lang, F. R. (2000). Endings and continuity of social relationships: Maximizing intrinsic benefits within personal networks when feeling near death. *Journal of Social and Personal Relationships, 17,* 155-182.

Lang, F. R., & Carstensen, L. L. (1998). Social relationships and adaptation in later life. In A. S. Bellack and M. Hersen (Eds.), *Comprehensive Clinical Psychology, (pp. 55-72).* Oxford, England: Peragom Press.

Larkin, K. T., Martin, R. R., & McClain S. E. (2002). Cynical hostility and the accuracy of decoding facial expressions of emotions. *Journal of Behavoral Medicine, 25*(3), 285-292.

LaRocco, J., House, J., & French, J. (1980). Social support, occupational stress and health. *Journal of Health and Social Behavior, 21,* 202-218.

Larsen, R. J., & Buss, D. M. (2005). *Personality Psychology. Domains of Knowledge about human nature (2nd ed., pp. 420-423).* New York, NY: McGraw-Hill.

Lauria, R. E. (1975). The validity and reliability of the visual analogue mood scale. *Journal of Psychiatric Research, 12,* 51-55.

Lavis, J. N. (2002). Ideas at the margin or marginalized ideas? Nonmedical determinants of health in Canada. *Health Affairs, 21*(2), 107-112.

Lavis, J. N., et al. (2001). Work-related population health indicators. *Canadian Journal of Public Health, 92*(1), 72-78.

Leaf, P. J., Bruce, M. C., Tischler G. L., et al. (1985). Contact with health professionals for the treatment of psychiatric and emotional problems. *Medical Care, 23,*

1322-1337.

Leal, D., Galanter, M., Dermatis, H., & Westreich, L. (1999). Correlates of protracted homelessness in a sample of dually diagnosed psychiatric inpatients. *Journal of Substance Abuse and Treatment, 16,* 143-147.

Leary, M. R., & Springer, C. A. (2001). Hurt feelings: The neglected emotion. In R. Kowalski (Ed.), *Aversive Behaviors and Interpersonal Transgressions (pp. 151-175).* Washington, DC: American Psychological Association.

Leavy, R. L. (1983). Social support and psychological disorder: A review. *Journal of Community Psychology, 11,* 3-21.

Lee, R. M., & Robbins, S. B. (2000). Understanding social connectedness in college women and men. *Journal of Counseling and Development, 78,* 484-491.

Lee, R. M., & Lee, S. (2001). Social connectedness, dysfunctional interpersonal behaviors, and psychological distress: Testing a mediator model. *Journal of Counseling Psychology, 48*(3), 310-318.

Lee, T. C., Yang, Y. K., Chen, P. S., Hung, N. C., Lin, S. H., Chang, F. L., & Cheng, S. H. (2006). Different dimensions of social support for the caregivers of patients with schizophrenia: Main effect and stress-buffering models. *Psychiatry & Clinical Neurosciences, 60*(5), 546-550.

Lee, R. M., & Robbins, S. B. (1998). The relationship between social connectedness and anxiety, self-esteem, and social identity. *Journal of Counseling Psychology, 45,* 338-345.

Leff, J., & Trieman, N. (2000). Long-stay patients discharged from psychiatric hospitals:

Social and clinical outcomes after five years in the community. The TAPS Project 46. *British Journal of Psychiatry, 176,* 217-223.

Lehman, A. F.,Buchanan, R. W., Dickerson, F. B., Goldberg, R., Green-Paden, L., et al, (2003). Evidenced-based treatment of schizophrenia. Psychiatric Clinics of North America, 26, 939-954.

Lehoux, P., & Battista, R. N. (2000). Monitoring health technology assessment agencies. *The Canadian Journal of Program Evaluation, 15*(2), 1-33.

Leighton, D. C., Hording, J. S., Macklin, D. B., MacMillan, A. M., & Leighton, A. H. (1963). *The character of danger: Psychiatric symptoms in selected communities.* New York: Basic Books.

Lelliot, P., Wing, J., & Clifford, P. (1994). A national survey of new longstay psychiatric patients. 1: Method and description of the cohort. *British Journal of Psychiatry, 165,* 160-169.

LePage, J. P., Bluitt, M., McAdams, H., Merrell, C., House-Hatfield, T., & Garcia-Rea, E. (2006). Effects of increased social support and lifestyle behaviors in a domiciliary for homeless veterans. *Psychological Services, 3*(1), 16-24.

Lerner, R. M., & Castellino, D. R. (2002). Contemporary development theory and adolescence: Developmental systems and applied developmental science. *Journal of Adolescent Health, 31,* 122-135.

Lerner, R. M. (1998). Theories of human development: Contemporary perspectives. In R. M. Learner (ED.), *Handbook of Child Psychology (5th ed., vol. 1, pp. 1-24*). New York, NY: John Wiley.

Levav, I., & Arnon, A. (1976). Emotional disorders in six Israeli villages. *Acta Psychiatrica Scandinavica 53,* 387-400.

Levine, J., & Fox, J. A. (1994). *Elementary statistics in social research.* New York; NY: Harper Collins.

Levine, M. (1981). *The history and politics of community mental health.* New York, NY: Oxford University Press.

Lewis, D., & Hugi, R. (1981). Therapeutic stations and the chronically treated mentally ill. *Social Service Review 55*(2), 206-220.

Lewis, D. (1990). From programs to lives: A comment. *American Journal of Community Psychology, 18*(6), 923-926.

Lewis, M. A., & Rook, K. S. (1999). Social control in personal relationships: Impact on health behaviors and psychological distress. *Health Psychology, 18,* 63-71.

Lewisnsohn, P. M., Allen, N. B., Seeley, J. R., & Gotlib, I. H. (1999). First onset versus recurrence of depression: Differential processes of psychosocial risk. *Journal of Abnormal Psychology, 108*(3), 483-489.

Liang, J., Krause, N. M., & Bennett, J. M. (2001). Social exchange and well-being: Is giving better than receiving? *Psychology and Aging, 16*(3), 511-523.

Lieberman, A., & Test, M. A. (1987). Health care practices and health status of the mentally ill in the community. *Health and Social Work, 12,* 29-37.

Liem, R., & Rayman, P. (1982). Health and social costs of unemployment. *American Psychologist, 37,* 1116-1123.

Lin, N., & Ensel, W. (1989). Life stress and health: Stressors and resources. *American*

Sociological Review 54, 382-399.

Lin, N., Dean, A., & Ensel, W. (Eds.). (1986). *Social support, life events and depression.* New York, NY: Academic Press.

Lindblad-Goldberg, M., Dukes, J. L., & Lasley, J. H. (1988). Stress in black, low-income, single-parent families: Normative and dysfunctional patterns. *American Journal of Orthopsychiatry, 58,* 104-120.

Lindsey, D., & Ozawa, M. (1979). Schizophrenia and SSI: Implications and problems. *Social Work, 24*(2), 120-126.

Link, B. (1982). Mental patient status, work, and income: An examination of the effects of a psychiatric label. *American Sociological Review, 47*(2), 202-215.

Link, B. G., & Phelan, J. C. (1999). Public conceptions of mental illness: Labels, causes, dangerousness, and social distance. *American Journal of Public Health, 89,* 1328-1333.

Link, B. G., & Phelan, J. C. (1996). Understanding sociodemographic differences in health: the role of fundamental social causes. *American Journal of Public Health, 86*, 471-473.

Link, B. G., Lennon, M. C., & Dohrenwend, B. P. (1993). Socioeconomic status and depression: The role of occupations involving direction control and planning. *American Journal of Sociology, 98*(6), 1353-1387.

Little, R. A. J., & Rubin, D. B. (1987). *Statistical analysis with missing data.* New York, NY: Wiley.

Lloyd, K. (1986). The Bec Enterprises work scheme. *British Journal of Occupational*

Therapy, 49(8), 257-258.

Loehlin, J. C. (1987). *Latent variable models.* Hillsdale, NJ: Lawrence Earlbaum Associates.

Long, J. A., Ickovics, J. R., Gill, T. M., & Horwitz, R. I. (2001). The cumulative effects of social class on mental status decline. *Journal of the American Geriatrics Society, 49*(7), 1005-1007.

Longest, J., Kovan, M., & Tweed, D. (1979). *A study of deficiencies and differentials in the distribution of mental health resources in facilities.* (DHEW Pub. No. ADM70-517). Washington, DC: U. S. Government Printing Office.

Lopez, F. G., & Brennan, K. A. (2000). Dynamic processes underlying adult attachment organization: Towards an attachment theoretical perspective on the healthy and effective self. *Journal of Counseling Psychology, 47,* 283-301.

Lowenstein, A., & Rosen, A. (1989). The relation of widows' needs and resources to perceived health and depression. *Social Science Medicine, 29,* 659-667.

Lundeberg, O. (1991). Childhood living conditions, health status, and social mobility: A contribution to the health selection debate. *European Sociological Review, 7*(2), 149-162.

Lynch, J., & Kaplan, G. (2000). Socioeconomic position. In L. Berknam and I Kawachi (Eds.), *Social Epidemiology,* New York, NY: Oxford University Press.

Lynch, J. W., Smith, G. D., Kaplan, G. A., & House, J. S. (2000). Income inequality and mortality: Importance to health of individual income, psychosocial environment, or material conditions. *British Medical Journal, 320,* 1200-1204.

Lyons, J. S., O'Mahoney, M. T., Miller, S. I., et al. (1997). Predicting readmission to the psychiatric hospital in a managed care environment: Implications for quality indicators. *American Journal of Psychiatry, 154(3),* 337-340.

Lyubomirsky, S. (2001). Why are some people happier than others? The role of cognitive and motivational processes in well-being. *American Psychologist, 56*(3), 239-249.

Lyubomirsky, S., King, L., & Diener, E. (2005). The benefits of frequent positive affect: Does happiness lead to success? *Psychological Bulletin, 131*(6), 803-855.

Lyyra, T. M., & Heikkinen, R. L (2006). Perceived social support and mortality in older people. *The Journals of Gerontology Series B: Psychological Sciences and Social Sciences, 61,* S147-S152.

MacCallum, R. C., Zhang, S., Preacher, K. J., & Rucker, D. D. (2002). On the practice of dichotomization of quantitative variables. *Psychological Methods, 7*(1), 19-40.

MacDonald, G., & Leary, M. R. (2005). Why does social exclusion hurt? The relationship between social and physical pain. *Psychological Bulletin, 131*(2), 202-223.

MacDonald, R. (1992). *Verification of selected needs for patients of mental health services.* Winnipeg, MB: University of Manitoba.

Macintyre, S., Ellaway, A., & Cummings, S. (2002). Place effects on health: How can we conceptualise, operationalise and measure them. *Social Science and Medicine, 55*(1), 125-139.

Mackie, J. L. (1974). *The cement of the universe: A study of causation.* Oxford: Clarendon.

MacMahon, B., & Pugh, T. F. (1970). *Epidemiology principles and methods.* Boston.

Little & Brown.

Madonia, J. (1983). The trauma of unemployment and its consequences. *Social Casework, 64*(8), 482-488.

Maffeo, P., Ford, T., & Lavin, P. (1990). Gender differences in depression in an employment setting. *Journal of Personality Assessment, 55,* 249-262.

Maher, M. J., Mora, P. A., & Leventhal, H. (2006). Depression as a predictor of perceived social support and demand: A componential approach using a prospective sample of older adults. *Emotion, 6*(3), 450-458.

Mai, F., Gosselin, J., Varan, L., Bourgon, L., & Navarro, J. (1993). Effects of treatment and alternative care on the length of stay on a general hospital psychiatric unit: Results of an audit. *Canadian Journal of Psychiatry, 38*(1), 39-45.

Makosky, V. (1982). Sources of stress: Events or conditions: In D. Belle, (Ed.), *Lives in stress: Women and depression.* Beverly Hills: Sage.

Malhotra, N. K. (1987). Analyzing marketing research data with incomplete information on the dependent variable. *Journal of Marketing Research, 24*, 74-84.

Malla, A. K., Norman, R. M. G., McLean,T. S., McDonald, C., McIntosh, E., Dean Lashley, F., Lynch, J., Scholten, D., & Ahmed, R. (2004). Determinants of quality of life in first episode psychosis. *Acta Psychiatrica Scandinavica, 109,* 46-54.

Mallinckrodt, B., & Wei, M. (2005). Attachment, social competencies, social support and psychological distress. *Journal of Counseling Psychology, 52*(3), 358-367.

Mancini, J. (1979). Family relationships and morale among people 65 years of age and older. *American Journal of Orthopsychiatry, 49,* 292-300.

Mann, A. H., Jenkins, R., & Belsey, E. (1981). The twelve-month outcome of patients with neurotic illness in general practice. *Psychological Medicine, 11,* 535-550.

Marken, P. A., Stanislav, S. W., Lacombe, S., Pierce, C., Hornstra, R., & Sommi, R. W. (1992). Profile of a sample of subjects admitted to an acute care psychiatric facility with manic symptoms. *Psychopharmacology Bulletin, 28*(2), 201-205.

Marks, I. M., Mataix-Cols, D., Kenwright, M., et al. (2003). Pragmatic evaluation of computer-aided and self-help for anxiety and depression. *British Journal of Psychiatry, 183,* 57-65.

Marsden, P. (1990). Network data and measurement. *Annual Review of Sociology, 16,*435-463.

Marshall, M., Crowther, R., Almaraz-Serrano, A., Creed, F., Sledge, W., Luiter, H., Roberts, C., Hill, E., Wiersma, D., Bond, G. R., Huxley, P., & Tyrer, P. (2001). Systematic reviews of the effectiveness of day care for people with severe mental disorders: (1) Acute day hospital versus admission; (2) Vocational rehabilitation; (3) Day hospital versus outpatient care. *Health Technology Assessment, 5*(21), 1-75.

Marshall, M., Crowther, R., Almaraz-Serrano, A., Creed, F., Sledge, W., Kluiter, H., Roberts, C., Hill, E., & Wiersma, D. (2003). Day hospital versus admission for acute psychiatric disorders. *Evidence Based Nursing, 6*(4), 108.

Mason, G. (1991). Longitudinal research in program evaluation. In A. J. Love (Ed.), *Evaluation methods sourcebook* (pp. 68-87). Ottawa: Canadian Evaluation Society.

Masthoff, E. D., Trompenaars, F. J., Van Heck, G. L., Hodiamont, P. P. G., & De Vries, J.

(2005). Validation of the WHOQOL-100 in a population of Dutch adult psychiatric outpatients. *European Journal of Psychiatry, 20,* 465-473.

Materson, E., & O'Shea, B. (1984). Smoking and malignancy in schizophrenia. *British Journal of Psychiatry, 145,* 429-432.

Matt, G. E., Dean, A., Wang, B., & Wood, P. (1992). Identifying clinical symptoms in a community sample of elderly persons. *Psychological Assessment, 4*(2), 174-184.

Mattes, J. (1987). The optimal length of hospitalization for psychiatric patients: A review of the literature. *Hospital and Community Psychiatry, 33,* 824-828.

Maughan, B., & Rutter, M. (1997). Retrospective reporting of childhood adversity: Issue of long-term recall. *Journal of Personality Disorders, 11,* 19-33.

McCabe, M. S. (1976). Reactive psychosis and schizophrenia with good prognosis. *Archives of General Psychiatry, 33,* 571-576.

McCarthy, P. L. (1991). Catchment areas demographic characteristics and rates of state psychiatric hospitalization. *Administration and Policy in Mental Health, 18,* 325-332.

McDonald, T. P., Poertner, J., & Pierpont, J. (1999). Predicting caregiver stress: An ecological perspective. *American Journal of Orthopsychiatry, 69*(1), 100-109.

McGlashan, T. H. (1991). Selective review of recent North American long-term follow-up studies of schizophrenia. In S. M. Mirin, J. T. Gossett, & M. C. Grob (Eds.), *Psychiatric treatment: Advances in outcome research* (pp. 61-106). Washington, DC: American Psychiatric Press.

McHugo, G. J, Drake, R. E., Teague, G. B., et al. (1999). Fidelity to Assertive Community

Treatment and Client Outcomes in the New Hampshire Dual Disorders Study. *Psychiatric Services, 50(6),* 818-824.

McKee-Ryan, F. M., Song, Z., Wanberg, C. R., & Kinicki, A. J. (2005). Psychological and physical well-being during unemployment: A meta-analytic study. *Journal of Applied Psychology, 90*(1), 53-76.

McKendree-Smith,N. L., Floyd, M., & Scogin, F. R. (2003). Self-administered treatments for depression: A review. *Journal of Clinical Psychology, 59,* 275-288.

McKenry, P., Browne, D., & Kotch, J. (1990). Mediators of depression among low-income, adolescent mothers of infants: A longitudinal perspective. *Journal of Youth and Adolescence, 19*(4), 327-347.

McLanahan, S. (1983). Family structure and stress: A longitudinal comparison of two-parent and female-headed families. *Journal of Marriage and Family Therapy, 9,* 347-357.

McLeister Anan, R., & Barnett, D. (1999). Perceived social support mediates between prior attachment and subsequent adjustment: A study of urban African American children. *Developmental Psychology, 35*(5), 1210-1222.

McLeod, C. B., Lavis, J. N., Mustard, C. A., & Stoddart, G. L. (2003). Income inequality, household income, and health status in Canada: A prospective cohort study. *American Journal of Public Health, 93*(8), 1287-1293.

McNicholas, S. L. (2002). Social support and positive health practices. *Western Journal of Nursing Research, 24,* 772-787.

McQuaid, J. R., Monroe, S. M., Roberts, J. E., Kupfer, D. J., & Frank, E. (2000). A

comparison of two life stress assessment approaches: Prospective prediction of treatment outcome in recurrent depression. *Journal of Abnormal Psychology, 109*(4), 787-791.

Measelle, J. R., Stice, E., & Springer, D. W. (2006). A prospective test of the negative affect model of substance abuse: Moderating effects of social support. *Psychology of Addictive Behaviors, 20*(3), 225-233.

Mechanic, D., Schlesinger, M., & McAlpine, D. D. (1995). Management of mental health and substance abuse services: State of the art and early results. *Milbank Quarterly, 75,* 19-55.

Mechanic, D. (1989). *Mental health and social policy* (3rd Ed.). Englewood Cliffs, NJ: Prentice-Hall.

Mehta, K. M., Yaffe, K., & Covinsky, K. E. (2002). Cognitive impairment, depressive symptoms, and functional decline in older people. *Journal of the American Geriatrics Society, 50,* 1045-1050.

Melchoir, M., Berkman, L. F., Niedhammer, I., Chea, M., & Goldberg, M. (2003). Social relations and self-reported health: A prospective analysis of the French Gazel cohort. *Social Science and Medicine, 56,* 1817-1830.

Mendes de Leon, C. F., Gold, D. T., Glass, T. A., Kaplan, L., & George, L. K. (2001). Disability as a function of social networks and support in elderly African Americans and Whites: The Duke EPESE 1986-1992. *Journals of Gerontology: Series B: Psychological Sciences and Social Sciences, 56,* S179-S190.

Merchant, A. T., Pitiphat, W., Ahmed, B., Kawachi, I., & Josipura, K. (2003). A

prospective study of social support, anger expression and risk of periodontitis in men. *Journal of the American Dental Association, 134*(12), 1591-1596.

Merton, R. K. (1949). *Social Theory and Social Structure: Toward the Codification of Theory and Research.* Glencoe, IL: Free Press.

Merton, R. K. (1957). *Social theory and social structure.* New York, NY: Free Press.

Mezzick, J., & Coffman, G. (1985). Factors influencing length of hospital stay. *Hospital and Community Psychiatry, 36,* 1262-1264.

Michalak, E. E., Yatham, L. N., Kolesar, S., & Lam, R. W. (2006). Bipolar disorder and quality of life: A patient-centered perspective. *Quality of Life Research, 15*(1), 25-37.

Michalak, E. E., Wilkinson, C., Hood, K., Dowrick, C., & Wilkinson, G. (2003). Seasonality, negative life events and social support in a community sample. *British Journal of Psychiatry, 182,* 434-438.

Miech, R. A., Caspi, A., Moffitt, T. E., Wright, B. E., & Silva, P. A. (1999). Low socio-economic status and mental disorders: A longitudinal study of causation and selection. *American Journal of Sociology, 4,* 1096-1131.

Milburn, N. G., Brown, D. R., & Gary, L. E. (1984). *Depressive symptoms and social network characteristics.* Paper presented at the National Association of Social Workers Health Conference, Washington, D.C.

Miller, G. A., & Chapman, J. P. (2001). Misunderstanding analysis of covariance. *Journal of Abnormal Psychology, 110*(1), 40-48.

Miller, T. W. (1988). Advances in understanding the impact of stressful life events on

health. *Hospital and Community Psychiatry, 39,* 615-622.

Mirin, S. M., Gossett, J. T., & Grob, M. C.(Eds.) (1991). *Psychiatric treatment: Advances in outcome research.* Washington, DC: American Psychiatric Press.

Mitchell, R. E., Billings, A. G., & Moos, R. H. (1982). Social support and well-being: implications for prevention programs. *Journal of Primary Prevention, 3,* 77-98.

Mitchell, R. E., & Moos, R. (1984). Deficiencies in social support among depressed patients: Antecedents or consequences of stress. *Journal of Health and Social Behavior, 25,* 438-452.

Mittal, D., Fortney, J. C., Pyne, J. M., Edlund, M. J., & Wetherell, J. L. (2006). Impact of comorbid anxiety disorders on health-related quality of life among patients with major depressive disorder. *Psychiatric Services, 57*(12), 1731-1737.

Mollica, R. F., & Milic, M. (1986). Social class and psychiatric practice: A revision of the Hollingshead and Redlich model. *American Journal of Psychiatry, 143*(1), 12-17.

Monroe, S., Bromet, E., Connell, M., & Steiner, S. (1986). Social support, life events, and depressive symptoms: A one-year prospective study. *Journal of Consulting and Clinical Psychology, 54,* 424-431.

Moos, R. H. (1990). Depressed outpatients' life contexts, amount of treatment and treatment outcome. *Journal of Nervous and Mental Disorder, 178,* 105-112.

Morlan, K., & Tan, S. (1998). Comparison of the Brief Psychiatric Rating Scale and the Brief symptom Report. *Journal of Clinical Psychology, 54*(7), 885-894.

Morris, J. N. (1979). Social inequality undiminished. *Lancet, 1,* 87-90.

Mosher, L. R., & Burti, L. (1989). *Community mental health: Principles and practice.*

New York, NY: W. W. Norton.

Mosher, L. R.(1982). Italy's revolutionary mental health law: An assessment. *American Journal of Psychiatry, 139,* 199-203.

Mueller, D. (1980). Social network: A promising direction for research on the relationship of the social environment to psychiatric disorder. *Social Science Medicine, 14A,* 147-161.

Mueller, D. (1981). The current status of urban/rural differences in psychosocial disorder: An emerging trend for depression. *Journal of Nervous and Mental Disease, 169,* 18-27.

Mueser, K. T., Salyers, M. P., Rosenberg, S. D., Ford, J. D., Fox, L., & Carty, P. (2001). Psychometric evaluation of trauma and posttraumatic stress disorder assessments in persons with sever mental illness. *Psychological Assessment, 13*(1), 110-117.

Mueser, K. T., Bond, G. R., Drake, R. E., et al. (1998). Models of community care for severe mental illness: A review of research on case management. *Schizophrenia Bulletin, 24,* 37-74.

Mullen, P. E., Burgess, P., Wallace, C., et al. (2000). Community care and criminal offending in schizophrenia. *Lancet, 355,* 614-617.

Munk-Jorgensen, P., & Mortensen, P. (1992). Social outcome in schizophrenia: A 13-year follow-up. *Social Psychiatry and Psychiatric Epidemiology, 27*(3), 129-134.

Munves, P., Trimboli, F., & North, A. (1983). A study of repeat visits to a psychiatric emergency room. *Hospital and Community Psychiatry, 34*(7), 634-638.

Murphy, D. L., Pickar, D., & Alterman, I. S. (1982b). Methods for the quantitative

assessment of depressive and manic behavior. In E. I. Burdock, A. Sudilovsky, and S. Gershon, (Eds.), *The Behavior of Psychiatric Patients: Quantitative Techniques for Evaluation.* New York, NY: Marcel Dekker.

Murphy, J. (1986). Trends in depression and anxiety: Men and women. *Acta Psychiatrica Scandinavica, 73,* 113-127.

Murphy, J. A., & Wetzel, R. D. (1980). Suicide risk by birth cohort in the United States, 1949 to 1974. *Archives of General Psychiatry, 37,* 519-523.

Murray-Parkes, C. (1979). Psychosocial transitions: A field for study. *Social Science and Medicine, 5,* 101-115.

Murray, L. T. (1995). *Outreach program evaluation: Executive Summary.* The Society for Depression and Manic Depression of Manitoba. Unpublished manuscript.

Murray, L. T. (2002; January). *Social supports relationship to well-being and psychiatric symptomatology: What matters most?* Winter Colloquium Series, Department of Psychology, University of Manitoba.

Murray, L. T. (1996). *Influence of social support on psychiatric symptoms and hospitalization: A path analysis delineating the interaction of education, employment, income, and perception of financial adequacy.* Unpublished master's thesis, University of Manitoba, Winnipeg, MB: Canada.

Murray, C. J., & Lopez, A. D. (1997). Alternative projections of mortality and disability by cause 1990-2020: Global burden of disease study. *Lancet, 349,* 1498-1504.

Myers, J., & Bean, L. (1968). *A decade later: A follow-up of social class and mental illness.* New York: Wiley.

Narrow, W., Rae, D., Moscicki, E., Locke, B., & Regier, D. (1990). Depression among Cuban Americans: The Hispanic health and nutrition examination survey. *Social Psychiatry and Psychiatric Epidemiology, 25*(5), 260-268.

Nathonson, C. (1980). Social roles and health status among women: The significance of employment. *Social Science and Medicine, 14A,* 463-471.

Natterlund, B., & Ahlstrom, G. (1999). Experience of social support in rehabilitation: A phenomenological study. *Journal of Advanced Nursing, 30*(6), 13332-1340.

Neff, J. (1983). Urbanicity and depression reconsidered: The evidence regarding depressive symptomatology. *Journal of Nervous and Mental Disease, 171,* 546-552.

Neff, L. A., & Karney, B. R. (2005). Gender differences in social support: A question of skill or responsiveness? *Journal of Personality and Social Psychology, 88*(1), 79-90.

Nelson, G., Hall, G. B., & Walsh-Bowers, R. (1999). Predictors of the adaptation of people with psychiatric disabilities in group homes, supportive apartments, and board-and-care homes. *Psychiatric Rehabilitation Journal, 22,* 381-389.

Nelson, G., Hall, G. B., & Walsh-Bowers, R. (1995). An evaluation of supportive apartments for psychiatric consumers/survivors. *Canada's Mental Health, 43*(2), 9-16.

Nezlek, J. B., Hampton, C. P., & Shean, G. D. (2000). Clinical depression and day-to-day social interaction in a community sample. *Journal of Abnormal Psychology, 109*(1), 11-19.

Nolan, P., & Badger, F. (2006). Aspects on relationship between doctors and depressed patients that enhance satisfaction with primary care. *Journal of Psychiatric Mental Health Nursing, 12,* 146-153.

Nuehring, E. M., Thayer, J. H., & Ladner, R. A. (1980). On the factors predicting rehospitalization among two state mental hospital patient populations. *Administration in Mental Health, 7*(4), 247-270.

O'Brien, M. (1980). Effective social environment and hemodialysis adaptation: A panel analysis. *Journal of Health and Social Behavior, 21,* 360-370.

O'Connell, R. A., Mayo, J. A., Flatlow, L., Cuthbertson, B., & O'Brien, B. E. (1991). Outcome of bipolar disorder on long-term treatment with lithium. *British Journal of Psychiatry, 159,* 123-133.

O'Driscoll, M. P., & Dewe, P. J. (2001). Mediators and moderators of stressor-strain linkages. In P. L. Perrewe & D. C. Ganster (Eds.), *Research in occupational stress and well being: Exploring theoretical mechanisms and perspectives* (pp. 257-287). New York, NY: JAI Press, Elsevier Sciences.

O'Neil, J. N., & Emery, C. F. (2002). Psychosocial vulnerability, hostility, and family history of coronary heart disease among male and female college students. *International Journal of Behavioral Medicine, 9,* 17-36.

O'Regan, J. B., (1965). A psychiatric emergency service: A preliminary report. *Canadian Medical Association Journal, 93,* 691-695.

Oatley, K., & Bolton, W. (1985). A social cognitive theory of depression in reaction to life events. *Psychological Review, 92,* 372-388.

Okum, M. A., & Keith, V. M. (1998). Effects of positive and negative social exchanges with various sources on depressive symptoms in the younger and older adults. *Journal of Gerontology: Psychological Sciences, 53*(B), 4-20.

Olsen, O. (1993). Impact of social network on cardiovascular mortality in middle aged Danish men. *Journal of Epidemiology and Community Health, 47*(3), 176-180.

Olsen, R. B., Olsen, J., Gunner-Svensson, F., & Waldstrom, B. (1991). Social networks and longevity: A 14 year follow-up study among elderly in Denmark. *Social Science and Medicine, 33*(10), 1189-1195.

Ong, A. D., & Allaire, J. C. (2005). Cardiovascular intraindividual variability in later life: The influence of social connectedness and positive emotions. *Psychology and Aging, 20*(3), 476-485.

Orford, J. (1986). Long-term mental health disability: Going to the consumers to assess need in the community. *British Journal of Occupational Therapy, 49*(11), 357-361.

Ormel, J., Rysdyk, F. V., Siullivan, M., van Sonderen, E., & Kempen, G. I. J. M. (2002). Temporal and reciprocal relationships between IADL/ADL disability and depressive symptoms in late life. *Journal of Gerontology: Social Sciences, 59B,* S333-S347.

Orth, U., & Wieland, E. (2006). Anger, hostility, and posttraumatic stress disorder in trauma-exposed adults: A meta-analysis. *Journal of Consulting and Clinical Psychology, 74*(4), 698-706.

Orth,U., Cahill,S. P., Foa, E. B., & Maercker, A. (2006). Anger and posttraumatic stress disorder. A causal analysis with longitudinal data.

Overall, J. E., & Gorham, D. R. (1962). The Brief Psychiatric Rating Scale. *Psychological Reports, 10,* 799-812.

Paivio, S. C. (2001). Stability of retrospective self-reports of child abuse and neglect before and after therapy for child abuse issues. *Child Abuse and Neglect, 25,* 1053-1068.

Panksepp, J. (1998). *Affective Neuroscience.* New York, NY: Oxford University Press.

Papolos, D., & Papolos, J. (1987). *Overcoming depression.* Toronto: Harper and Row.

Pargament, K. I. (1997). *The Psychology of Religion and Coping: Theory, Research, and Practice.* New York, NY: Guilford.

Park, C. L., Fenster, J. R., Suresh, D. P., & Bliss, D. E. (2006). Social support, appraisals, and coping as predictors of depression in congestive heart failure patients. *Psychology and Health, 21*(6), 773-789.

Parks, E. D., & Josef, N. (1997). A retrospective study of determinants of length of stay in a geropsychiatric state hospital. *Psychiatric Quarterly, 68(2),* 91-99.

Pasch, L. A., & Bradbury, T. N. (1998). Social support, conflict, and the development of marital dysfunction. *Journal of Consulting and Clinical Psychology, 66,* 219-230.

Paterniti, S., Niedhammer, I., Lange, T., & Consoli, S. M. (2002). Psychosocial factors at work, personality traits and depressive symptoms: Longitudinal results from the GAZEL study. *British Journal of Psychiatry, 181,* 111-117.

Patrick, D., Morgan, M., & Charlton, J. (1986). Psychosocial support and change in the health status of physically disabled people. *Social Science and Medicine, 22*(12), 1347-1354.

Payne, M. (1997). *Modern social work theory.* Chicago: Lyceum Books.

Pearlin, L. I., Menaghan, E. G., Lieberman, M. A., & Mullan, J. T. (1981). The stress process. *Journal of Health and Social Behavior, 22,* 337-356.

Pearlin, L. I. (1982). The social contexts of stress. In L. Goldberger & S. Breznitz (Eds.), *Handbook stress: Theoretical and clinical aspects* (pp.367-379). New York: Free Press.

Pearson, M., Wilmot, E., & Padi, M. (1986). A study of violent behaviour among in-patients in a psychiatric hospital. *British Journal of Psychiatry, 149,* 232-235.

Peirce, R. S., Frone, M. R., Russell, M., Cooper, M. L., & Mudar, P. (2000). A longitudinal model of social contact, social support, depression, and alcohol use. *Health Psychology, 19*(1), 28-38.

Penninx, B. W. J. H, Leveille, S., Ferrucci, L., van Eijk, J. T. M., & Guralnik, J. M. (1999). Exploring the effects of depression on physical disability: Longitudinal evidence from the Established Populations for Epidemiological Studies of the Elderly. *American Journal of Public Health, 89,* 1346-1352.

Penninx, B. W. J. H., van Tilburg, T., Boeke, A. J. P., Deeg, D. J. H., Kriegsman, D. M. W., & van Eijk, J. T. M. (1998). Effects of social support and personal coping resources on depressive symptoms: Different for various chronic diseases? *Health Psychology, 17*(6), 551-558.

Pennix, B., Kriegsman, D., van Eljk, J., et al. (1996). Differential effect of social support on the course of chronic disease: A criteria-based literature study. *Families, Systems, & Health, 14,* 223-244.

Pernice-Duca, F. M. (2006). The relationship between social network supports and recovery from mental illness. *Dissertation Abstracts International: Section B: The Sciences and Engineering, 67(2-B),* 1161.

Perrewe, P. L., & Carlson, D. S. (2002). Do men and women benefit from social support equally? Results from a field examination within the work family context. In D. L. Nelson & R. J. Burke (Eds.), *Gender, work stress, and health: Current research issues (pp. 101-114).* Washington, DC: American Psychological Association.

Perrucci, R., Perrucci, C. C., Targ, D., & Targ, H. R. (1988). *Plant closings: International context and social costs.* New York, NY: Aldine de Gruyter.

Petrosino, A. (2000). Answering the 'Why' question in evaluation: The causal-model approach. *The Canadian Journal of Program Evaluation, 15*(1), 1-24.

Pietzcker, A., & Gaebel, W. (1987). Prospective study of course of illness in schizophrenia: I. Outcome at 1 year. *Schizophrenia Bulletin, 13*(2), 287-297.

Pilisuk, M. (2001). A job and a home: Social networks and the integration of the mentally disabled in the community. *American Journal of Orthopsychiatry, 71*(1), 49-60.

Pini, S., Piccinelli, M., & Zimmermann-Tansella, C. (1995). Social problems as factors in medical consultation: A comparison between general practice attenders and community probands with emotional distress. *Psychological Medicine, 25,* 33-41.

Pinquart, M., & Sorensen, S. (2000). Influences of socioeconomic status, social network, and competence on subjective well-being in later life: A meta-analysis. *Psychology and Aging, 15*(2), 187-224.

Pitschel-Walz, G., Leucht, S., Bauml, J., et al., (2001). The effect of family interventions

on relapse and rehospitalization in schizophrenia: A meta analysis. *Schizophrenia Bulletin, 27,* 73-92.

Plant, E. A., Sachs-Ericsson, N. (2004). Racial and ethnic differences in depression: The roles of social support and meeting basic needs. *Journal of Consulting and Clinical Psychology, 72*(1), 41-52.

Pottick, K., Hansell, S., Gutterman, E., & Raskin-White, H. (1995). Factors associated with inpatient and outpatient treatment for children and adolescents with serious mental illness. *Journal American Academy of Child Adolescent Psychiatry, 34*(4), 425-433.

Pressman, S. D., Cohen, S., Miller, G. E., Barkin, A., & Rabin, B. (2005). Loneliness, social network size, and immune response to influenza vaccination in college freshmen. *Health Psychology, 24*(3), 297-306.

Price, R. H., & Fang, L. (2002). Unemployed Chinese workers: The survivors, the worried young, and the discouraged old. *International Journal of Human Resource Management,13,* 416-430.

Price, R. H., Friedland, D. S., & Vinokur, A. D. (1998). Job loss: Hard times and eroded identity. In J. H. Harvey (Ed.), *Perspectives on Loss: A Sourcebook. Death, Dying, and Bereavement (pp. 303-316).* Philadelfia, PA: Brunner/Mazel.

Putnam, R. (2000). *Bowling Alone: The Collapse and Revival of American Community.* New York, NY: Simon and Schuster.

Radloff, L., & Rae, D. (1979). Susceptibility and precipitating factors in depression: Sex differences and similarities. *Journal of Abnormal Psychology, 88,* 174-181.

Ragins,B. R., & Cotton, J. L. (1999). Mentor functions and outcomes: A comparison of men and women in formal and informal mentoring relationships. *Journal of Applied Psychology, 84,* 529-550.

Raina, P., O'Donnel, M., Rosenbaum, P., Brehant, J., Walter, S. D., Russell, D., Swinton, M., Zhu, B., & Wood, E. (2005). The health and well-being of caregivers of children with cerebral palsy. *Pediatrics, 115,* 626-636.

Rakutis, M. E., Feidley, K., & Wood, J. (1998). Focus groups, program evaluation and the mentally ill: A case study. *Journal of Health and Social Policy, 10(21),* 75-92.

Raskin, A., Schluterbrandt, J., Reatig, N., & McKeon N. (1969). Replication of factors of psychopathology in interview, ward behavior and self-report ratings of hospitalized depressives. *Journal of Nervous and Mental Disorders, 148,* 87-98.

Reading, J., & Amatea, E. S. (1986). Role deviance or role diversification: Reassessing the psychosocial factors in the parenthood choice of career-oriented women. *Journal of Marriage and Family, 48,* 255-260.

Reding, G. R., & Raphelson, M. (1995). Around-the-clock mobile psychiatric crisis intervention: Another effective alternative to psychiatric hospitalization. *Community Mental Health Journal, 31*(2), 179-187.

Redlich, F., & Kellert, S. R. (1978). Trends in American mental health. *American Journal of Psychiatry, 135,* 22-28.

Reis, J. (1988). A factorial analysis of a compound measure of social support. *Journal of Clinical Psychology, 44*(6), 876-890.

Revicki, D. A., & Mitchell, J. P. (1990). Strain, social support, and mental health in rural

elderly individuals. *Journals of Gerontology, 45*(6), S267-S274.

Rew, L. Fouladi, R. T., & Yockey, R. D. (2002). Sexual health practices of homeless youth. *Journal of Nursing Scholarship, 34,* 139-145.

Reyes-Ortiz, C. A. (2006). Spirituality, disability and chronic illness. *Southern Medical Journal, 99*(10), 1172-1173.

Reynolds, I., & Hoult, J. (1984). The relatives of the mentally ill: A comparative trial of community-oriented and hospital-oriented psychiatric care. *Journal of Nervous and Mental Disease, 172*(8), 480-489.

Rhoades, H., & Overall, J. (1988). The semistructured BPRS interview and rating guide. *Psychopharmacology Bulletin, 24*(1), 101-104.

Rice, C., & Longabaugh, R. (1996). Measuring general social support in alcoholic patients: Short forms for perceived social support. *Psychology of Addictive Behaviours, 10*(2), 104-114.

Rich, C. L., Warsradt, G. M., Nemiroff, R. A., Fowler, R. C., & Young, D. (1991). Suicide, stressors, and the life cycle. *American Journal of Psychiatry, 148*(4), 524-527.

Richard, F. D., Bond, C. F., & Stokes-Zoota, J. J. (2003). One hundred years of social psychology quantitatively described. *Review of General Psychology, 7*(4), 331-363.

Richards, J., Smith, D. J., Harvey, C. A., et al. (1997). Characteristics of the new long-stay population in an inner Melbourne acute psychiatric hospital. *Australian and New Zealand Journal of Psychiatry, 31,* 488-495.

Ritter, C., Hoboll, S. E., Lavin, J., Cameron, R. P., & Hulsizer, M. R. (2000). Stress,

psychosocial resources, and depressive symptomatology during pregnancy in low-income, inner-city women. *Health Psychology, 19*(6), 576-585.

Roberts, L. J., & Greenberg, D. R. (2002). Observational "windows" to intimacy process in marriage. In P. Noller and J. A. Feeney (Eds.), *Understanding Marriage: Developments in the study of couple interaction (pp. 118-149).* Cambridge, UK: Cambridge University Press.

Roberts, C. R., Roberts, R. E., & Stevenson, J. M. (1982). Women, work, social support and psychiatric morbidity. *Social Psychiatry, 17*(4), 167-173.

Rodgers, B. (1991). Socioeconomic status, employment and neurosis. *Social Psychiatry and Psychiatric Epidemiology, 26*(3), 104-114.

Rogosa, D., (1980). A critique of cross-lagged correlation. *Psychological Bulletin, 88*(2), 245-258.

Romans, S. E., & McPherson, H. M. (1992). The social networks of bipolar ive disorder patients. *Journal of Affective Disorders 25,* 221-228.

Rook, K. S. (1984). The negative side of social interaction: Impact on psychological well-being. *Journal of Personality and Social Psychology, 46*(5), 1097-1108.

Rook, K. S. (1984). Promoting social bonding: Strategies for helping the lonely and socially isolated. *American Psychologist, 39,* 1389-1407.

Rook, K. S. (1984). Strategies for helping the lonely and socially isolated. *American Psychologist, 39*(12), 1389-1407.

Rosenberg, G. (1970). *The worker grows old.* San Fransisco, CA: Jossey-Bass.

Rosenblatt, A., & Attkisson, C. C. (1993). Assessing outcomes for sufferers of severe

mental disorders: A conceptual framework and review. *Evaluation and Program Planning, 16*(4), 347-363.

Rosenthal, R. (1984). *Meta-Analytic Procedures for Social Research.* Newbury Park, CA: Sage.

Rosenthal, R., & Rubin, D. B. (2003). A simple effect size indicator. *Psychological Methods, 8*(4), 1-7.

Ross, N. A., Blakely, T., Woodward, A., Razum, O., Wolfson, M., Berthelot, J. M., Dunn, J., Kaplan, G., & Lynch, J. (2000). Income inequality and mortality in Canada and the United States. *British Medical Journal, 321,* 1532.

Ross, N. A., Wolfson, M. C., Dunn, J. R., Berthelot, J. M., Kaplan, G. A., & Lynch, J. W. (2000). Relation between income inequality and mortality in Canada and in the United States: Cross sectional assessment using census data and vital statistics. *British Medical Journal, 320,* 898-902.

Ross, C. E. & Mirowsky, J. (1989). Explaining the social patterns of depression: Control and problem solving - or support and talking? *Journal of Health and Social Behavior, 30*(2), 206-219.

Ross, C., & Huber, J. (1985). Hardship and depression. *Journal of Health and Social Behavior, 26*(4), 312-327

Ross, N. A., Wolfson, M. C., Dunn, J. R., Berthelot, J. M., Kaplan, G. A., & Lynch, J. W. (2000). Relation between income inequality and mortality in Canada and in the United States: Cross sectional assessment using census data and vital statistics. *British Medical Journal,* 320, 898-902.

Roth, P. L. (1994). Missing data: A conceptual review for the Applied Psychologist. *Personnel Psychology, 47*(3), 537-560.

Rud, J., & Noreik, K. (1982). Who becomes long-stay patients in a psychiatric hospital? *Acta Psychiatrical Scandinavica, 65,* 1-4.

Rushton, J., Brainerd, C., & Pressley, M. (1983). Behavioural development and construct validity: The principle of aggregation. *Psychological Bulletin, 94,* 18-38.

Ryb, G. E., Soderstrom, C. A., Kufera, J. A., & Dischinger, P. (2006). Longitudinal study of suicide after traumatic injury. *Journal of Trauma, Injury, Infection and Critical Care, 61*(4), 799-804.

Ryff, C. D., & Singer, B. (2000). Interpersonal flourishing: A positive health agenda for the new millennium. *Personality and Social Psychology Review, 4*(1), 30-44.

Saarento, O., Niemenen, P., Hakko, H., et al. (1997). Utilization of psychiatric in-patient care among new patients in a comprehensive community-care system: A 3-year follow-up study. *Acta Psychiatrica Scandinavica, 95,* 132-139.

Saguaro Seminar. (2000). *Betting together: Report by the Saguaro Seminal on civic engagement in America.* John F. Kennedy School of Government, Cambridge, MA: Harvard University.

Sanders Thompson, V. L., Noel, J. G., & Campbell, J. (2004). Stigmatization, discrimination and mental health: The impact of multiple identity status. *American Journal of Orthopsychiatry, 74*(4), 529-544.

Sarason, B. R., Sarason, I. G., & Gurung, R. A. R. (2001). Close personal relationships and health outcomes: A key to the role of social support. In B. R. Sarason and S.

W. Duck (Eds.), *Personal Relationships: Implications for Clinical and Community Psychology (pp. 15-43).* New York, NY: Wiley.

Sarason, J., Levine, H., Basham, R., & Sarason, B. (1983). Assessing social support: The social support questionnaire. *Journal of Personality and Social Psychology, 44,* 127-139.

SAS Institute Inc. (1992). *SAS user's guide* (Version 6). Cary, NC: SAS Institute Inc.

Satcher, D. (1999). *Mental Health: A Report of the Surgeon General.* Rockville, MD: U. S. Department of Health and Human Services, Substance Abuse and Mental Health Services Administration, Center for Mental Health Services, National Institutes of Health, National Institute of Mental Health.

Sawyer, M. G., Arney, F. M., Baghurst, P. A., et al. (2001). The mental health of young people in Australia: Key findings from the child and adolescent component of the national survey of mental health and well-being. *Australian and New Zealand Journal of Psychiatry, 35,* 806-814.

Schaefer, C., Coyne, J. C., & Lazarus, R. S. (1981). The health-related functions of social support. *Journal of Behavioral Medicine, 4,* 381 - 406.

Schell, T. L., Marshall, G.. N., & Jaycox, L. H. (2004). All symptoms are not created equal: The prominent role of hyperarousal in the natural course of posttraumatic psychological distress. *Journal of Abnormal Psychology, 113*(2), 189-197.

Schneier, F. R. (1997). Quality of life in anxiety disorders. In H Katsching, H Freeman, & N. Sartorius (Eds.), *Quality of life in mental disorder (pp. 149-168).* Chichester: Wiley.

Schraufnagel, T. J., Wagner, A. W., Miranda, J., & Roy-Burne, P. P. (2006). Treating minority patients with depression and anxiety: What does the evidence tell us? *General Hospital Psychiatry, 28,* 17-36.

Schulz, R., Tompkins, C., Wood, D., & Decker, S. (1987). The social psychology of care giving: Physical and psychological costs of providing support to the disabled. *Journal of Applied Social Psychology, 17*(4), 410-428.

Schwab, J. J., Holzer, C. E., & Warheit, G. J. (1973). Depressive symptomatology and age. *Psychosomatics, 14,* 135-141.

Schwartz, C., Meisenhelder, J. B., Ma, Y., & Reed, G. (2003). Altruistic social interest behaviors are associated with better mental health. *Psychosomatic Medicine, 65,* 778-785.

Schwarz, C. (1988). Psychiatry short-stay units across Canada. *Canadian Journal of Psychiatry, 33*(3), 179-182.

Scott, K. G., Mason, C. A., & Chapman, D. A. (1999). The use of epidemiological methodology as a means of influencing public policy. *Child Development, 70,* 1263-1272.

Sechrest, L., & Sidani, S. (1995). Quantitative and qualitative methods: Is there and alternative? *Evaluation and Program Planning, 18*(1), 77-87.

Seeman, T., & Syme, S. L. (1987). Social networks and coronary artery disease: A comparison of the structure and function of social relations as predictors of disease. *Psychosomatic Medicine, 49,* 340-353.

Seeman, T. E., Lusignolo, T. M., Albert, M., & Berkman, L. (2001). Social relationships,

social support, and patterns of cognitive aging in healthy, high-functioning older adults: MacArthur studies of successful aging. *Health Psychology, 20*(4), 243-255.

Seethalakshmi, R., Dhavale, H. S., Gawande, S., & Dewan, M. (2006). Psychiatric morbidity following motor vehicle crashes: A pilot study from India. *Journal of Psychiatric Practice. 12*(6),415-418.

Segal, S. P., & Kotler, P. L. (1991). A ten-year perspective of mortality risk among mentally ill patients in sheltered care. *Hospital and Community Psychiatry, 42*(7), 708-713.

Segal, S. P., Hardiman, E. R., & Hodges, J. Q. (2002). Characteristics of new clients at self-help and community mental health agencies in geographic proximity. *Psychiatric Services, 53,* 1145-1152.

Segal, S. P., & Silverman, C. (2002). Determinants of client outcomes in self-help agencies. *Psychiatric Services, 53,* 304-309.

Serban, G., & Thomas, A. (1974). Attitudes and behaviors of acute and chronic schizophrenic patients regarding ambulatory treatment. *American Journal of Psychiatry 131*(9), 991-995.

Shader, R. I., Binstock, W. A., Ohly, J. I., & Scott, D. (1969). Biasing factors in diagnosis and disposition. *Comprehensive Psychiatry, 10,* 81-89.

Shadish, W. R., Jr., Cook, T. D., & Leviton, L. C. (1991). *Foundations of program evaluation: Theories of practice.* Newbury Park, CA: Sage.

Shadish, W. R. (1995). Philosophy of science and the quantitative-qualitative debates: Thirteen common errors. *Evaluation and Program Planning, 18*(1), 63-75.

Shafer, A. (2005). Meta-analysis of the Brief Psychiatric Rating Scale factor structure. *Psychological Assessment, 17*(3), 324-334.

Shah, A. K., & Ganesvaran, T. (1997). Inpatient suicides in an Australian mental hospital. *Australian and New Zealand Journal of Psychiatry, 31,* 291-298.

Shanks, H., & Atkins, P. (1985). Psychiatric patients who marry each other. *Psychological Medicine,15*(2), 377-382.

Shapiro, J. G. (1983). Patients refused admission to a psychiatric hospital. *Hospital and Community Psychiatry, 34*(8), 733-736.

Sheffield, D., Christie, I., Davidson, G., Manns, S. I., Shah, M., & Reid, C. (2000). Social support and blood pressure response to public speaking. *Annals of Behavioral Medicine, 22,* S112.

Sherbourne, C. D. (1988). The role of social support and life stress events in use of mental health services. *Social Science and Medicine, 27*(12), 1393-1400.

Sherkat, D., & Reed, M. (1992). The effects of religion and social support on self-esteem and depression among the suddenly bereaved. *Social Indicators Research, 26*(3), 259-275.

Sherman, M. D. (2003). The Support and Family Education (SAFE) Program: Mental health facts for families. *Psychiatric Services, 54*(1), 35-37.

Sherman, M. D., Faruque, H. D., & Foley, D. D. (2005). Family participation in the treatment of persons with serious mental illness. *Psychiatric Services, 56,* 1624-1625.

Sherman, J. E., DeVinney, D. J., & Sperling, K. B. (2004). Social support and adjustment

after spinal cord injury: Influence of past peer-mentoring experiences and current life-in partner. Rehabilitation Psychology, 49(2), 140-149.

Shinn, M., Knickman,J. R., & Weitzman, B. C. (1991). Social relationships and vulnerability to becoming homeless among poor families. *American Psychologist, 46*(11), 1180-1187.

Simon, E. S. (2003). Social and economic burden of mood disorders. *Biological Psychiatry, 54,* 208-215.

Sirey, J. A., Bruce, M. L., Alexopoulos, G. S., et al., (2001). Perceived stigma as a predictor of treatment discontinuation in younger and older outpatients with depression. *American Journal of Psychiatry, 158,* 479-481.

Sirey, J. A., Bruce, M. L., & Alexopoulos, G. S. (2005). The treatment initiation program: An intervention to improve depression outcomes in older adults. *American Journal of Psychiatry, 162,* 184-186.

Sirey, J. A., Bruce, M. L., Alexopoulos, G. S., et al., (2001). Stigma as a barrier to recovery: Perceived stigma and patient-rated severity of illness as predictors of antidepressant drug adherence. *Psychiatric Services, 52,* 1615-1620.

Skevington,S. M., & Wright, A. (2001). Changes in the quality of life of patients receiving antidepressant medication in primary care: Validation of the WHOQOL-100. *British Journal of Psychiatry, 178,* 261-267.

Skok, A., Harvey, D., & Reddihough, D. (2006). Perceived stress, perceived social support, and wellbeing among mothers of school-aged children with cerebral palsy. *Journal of Intellectual & Developmental Disability, 31*(1), 53-57.

Smith, T. E., Hull, J. W., Anthony, D. T., et al. (1997). Post-hospitalization treatment adherence of schizophrenic patients: Gender differences in skill acquisition. *Psychiatry Research, 69,* 123-129.

Smith, T. W., Glazer, K., Ruiz, J. M., & Gallo, L. C. (2004). Hostility, anger, aggressiveness, and coronary heart disease: An interpersonal perspective on personality, emotion, and health. *Journal of Personality, 72,* 1217-1270.

Smyth, C., Mohan, A., Buckley, C., et al. (1997). Where to? A survey of the residual population of a district mental hospital and their predicted placement needs. *Irish Journal of Psychological Medicine, 14(3),* 105-108.

Sobel, M. (1988). Direct and Indirect effects in linear structural equation models. In J. S. Long (Ed.), *Common problems/proper solutions: Avoiding error in quantitative research* (pp. 46-64). Beverly Hills, CA: Sage.

Solomon, P., & Draine, J. (2001). The state of knowledge of effectiveness of consumer provided services. *Psychiatric Rehabilitation Journal, 25,* 20-27.

Solomon, P., Davis, J., & Gordon, B. (1984). Discharged state hospital patients' characteristics and use of aftercare: Effects on community tenure. *American Journal of Psychiatry, 141*(12), 1566-1570.

Sorensen, L. V., & Mors, O. (1993). Social conditions and personality of depressed patients at first admittance and 12 months later. *Nordic Journal of Psychiatry, 47,* 101-107.

Sorkin, D., Rook, K. S., & Lu, J. L. (2002). Loneliness, lack of emotional support, lack of companionship, and the likelihood of having a heart condition in an elderly sample.

Annals of Behavioral Medicine, 24, 290-298.

Spady, W., & Greenwood, D. (1971). *Instant path analysis: An elementary cookbook.* Ontario: Ontario Institute for Studies in Education.

Spotts, E. L., Neiderhiser, J. M., Ganiban, J., Reiss, D., Lichtenstein, P., Hansson, K., et al., 2004). Accounting for depressive symptoms in women: A twin study of associations with interpersonal relationships. *Journal of Affective Disorders, 82,* 101-111.

Spotts, E. L., Pedersen, N. L., Neiderhiser, J. M., Reiss, D., Lichtenstein, P., Hansson, K., & Cederblad, M. (2005). Genetic effects on women's positive mental health: Do marital relationships and social support matter? *Journal of Family Psychology, 19*(3), 339-349.

Sprang, G., Clark, J., Kaak, O., & Brenzel, A. (2004). Developing and tailoring mental health technologies for child welfare: The Comprehensive Assessment and Training Services (CATS) Project. *American Journal of Orthopsychiatry, 74*(3), 325-336.

Srivastava, A., Locke, E. A., & Bartol, K. M. (2001). Money and subjective well-being: It's not the money, it's the motives. *Journal of Personality and Social Psychology, 80*(6), 959-971.

Srole, L., Langner, T. S., Michael, S. T., Opler, M. K., & Rennie, T. A. C. (1962). *Mental health in the metropolis: The Midtown Manhattan Study,* New York: McGraw Hill.

Stankunas, M., Kalediene, R., Starkuviene, S., & Kapustinskiene, V. (2006). Duration of unemployment and depression: A cross-sectional survey in Lithuania. *BMC Public*

Health, 6(174), 1-9.

Stanley, M. A., Beck, J. G., & Zebb, B. J. (1998). Psychometric properties of the MSPSS in older adults. *Aging and Mental Health, 2,* 186 - 193.

Statistics Canada, (1997). *1996 Census: Immigration and citizenship.* The Daily Statistics Canada, Nov 4.

Stefan, S. (2001). *Unequal Rights: Discrimination Against People with Mental Disabilities and the Americans With Disabilities Act.* Washington, DC: American Psychological Association.

Steffen,V. (1997). Life stories and shared experience. *Social Science, 45*(1), 99-111.

Steinhart, I., & Priebe, S. (1992) Prediction of hospitalization within a psychiatric community care system: A five year study. *Social Psychiatry and Psychiatric Epidemiology 27,* 270-273.

Stekette, G., Frost, R., & Bogart, K. (1996). The Yale-Brown obsessive compulsive interview versus self -report. *Behaviour Research and Therapy, 34*(8), 675-684.

Stephens, J. H. (1978). Long-term prognosis and follow-up in schizophrenia. *Schizophrenia Bulletin, 4,* 25-47.

Stice, E., Barrera, M., & Chassin, L. (1998). Prospective differential prediction of adolescent alcohol use and problem use: Examining mechanisms of effect. *Journal of Abnormal Psychology, 107,* 616-628.

Stolsenberg, R. (1980). The measurement and decomposition of causal effects in nonlinear and nonadditive models. In K. F. Schuessler (Ed.), *Sociological Methodology* (pp. 459-488). San Fransisco: Jossey-Bass.

Stone, A. A., Mezzacappa, E. S., Donatone, B. A., & Gonder, M. (1999). Psychological stress and social support are associated with prostrate-specific antigen levels in men: Results from a community screening program. *Health Psychology, 18,* 482-486.

Stone-Romero, E. F., & Anderson, L. E. (1994). Relative power of moderated multiple regression and the comparison of subgroup correlation coefficients for detecting moderating effects. *Journal of Applied Psychology, 79*(3), 354-359.

Storstein, L. (1987). How should changes in life-style be measured in cardiovascular disease? *American Heart Journal, 114*(1 Pt 2), 210-212.

Stougaard Nielsen, M. (2003). Prevalence of posttraumatic stress disorder in persons with spinal cord injuries: The mediating effect of social support. *Rehabilitation Psychology, 49*(4), 289-295.

Strakowski, S. M., Keck, P. E. Jr, McElroy, S. L., West, S. A., Sax, K. W., Hawkins, J. M., Kmetz, G. F., Upadhyaha, V. H., Tugrul, K. C., & Bourne, M. L. (1998). Twelve-month outcome following a first hospitalization for depressive psychosis. *Archives of General Psychiatry, 55,* 49-55.

Strauss, J. S., & Carpenter, W. T. (1977). Prediction of outcome in schizophrenia: III. Five-year outcome and its predictors. *Archives of General Psychiatry, 34,* 159-163.

Strauss, J. S., & Carpenter, W. T. (1972). Prediction of outcome in schizophrenia: I. Characteristics of outcome. *Archives of General Psychiatry, 27,* 739-746.

Strickland, P. L., Deakin, J. F. W., Percival, C., Dixon, J., Gater, R. A., & Goldberg, D. P. (2002). Bio-social origins of depression in the community: Interactions between

social adversity, cortisol and serotonin neurotransmission. *British Journal of Psychiatry, 180,* 168-173.

Stuck, A., Walther, J., Nikolaus, T., et al., (1999). Risk factors for functional status decline in community-living elderly people: A systematic literature review. *Social Science and Medicine, 48,* 445-469.

Substance Abuse and Mental Health Services Administration. (2003). *The NSDUH Report: Reasons for Not Receiving Treatment Among Adults with Serious Mental Illness.* Retrieved Agust 13, 2004, from http: //www .oas.samhsa .gov /2k3/ Hmno TX.pdf.

Suchman, N. E., McMahon, T. J., Slade, A., & Luthar, S. S. (2005). How early bonding, depression, illicit drug use, and perceived support work together to influence drug-dependent mothers' caregiving. *American Journal of Orthopsychiatry, 75*(3), 431-445.

Sudman, S., & Bradburn, N. M. (1973). Effects of time and memory factors on response in surveys. *Journal of the American Statistical Association, 68*(344), 805-815.

Sullivan, M. D., LaCroix, A. Z., Russo, J. E., & Walker, E. A. (2001). Depression and self-reported physical health in patients with coronary disease: Mediating and moderating factors. *Psychosomatic Medicine, 63,* 248-256.

Sundin, L, Bildt, C., Lisspers, J., HochwΣlder, J., & Setterlind, S. (2006). Organisational factors, individual characteristics and social support: What determines the level of social support? *Work, 27,* 45-55.

Suurmeijer, T. P. B. M., Reuvekamp, M. F., & Aldenkamp, B. P. (2001). Social

functioning, psychological functioning and the quality of life in epilepsy. *Epilepsia, 42,* 1160-1168.

Swindle, R. W. Jr, Cronkite, R. C., & Moos, R. H. (1989). Life stressors, social resources, coping, and the 4-year course of unipolar depression. *Journal of Abnormal Psychology, 98,* 468-477.

Sylvester, C. T., & Bean, G. J. (1989). Predicting community tenure after psychiatric hospitalization. *Community Mental Health Journal, 25*(2), 109-120.

Symister, P., & Friend, R. (2003). The influence of social support and problematic support on optimism and depression in chronic illness: A prospective study evaluating self-esteem as a mediator. *Health Psychology, 22*(2), 123-129.

Szasz, T. (1974). *The second sin.* London: Routledge & Kegan Paul.

Tabachnick, B. G., & Fidell, L. S. (2001). *Using multivariate statistics* (3rd ed.). New York: Harper & Row.

Tabachnick, B. G., & Fidell, L. S. (1989). *Using multivariate statistics* (2nd ed.). New York: Harper & Row.

Takizawa, T. M., Kondo, T., Sakihara, S., Ariizumi, M., Watanabe, N., & Oyama, H. (2006). Stress buffering effects of social support on depressive symptoms in middle age: Reciprocity and community mental health. *Psychiatry and Clinical Neurosciences, 60*(6), 652-661.

Tanenbaum, S. J., & Hurley, R. E. (1995). Disability and the managed care frenzy: A cautionary note. *Health Affairs, 14*(4), 213-219.

Tataryn, D., Mustard, C., & Derksen, S. (1994). *The utilization of medical services for*

mental health disorders Manitoba: 1991-1992. Manitoba Centre for Health Policy and Evaluation, Department of Community Health Sciences, Faculty of Medicine, University of Manitoba.

Taube, C., Lee, E. S., & Forthofer, R. N. (1984). DRGs in psychiatry. *Medical Care, 22,* 597-610.

Tausig, M. (1992). Caregiver network structure, support, and caregiver distress. *American Journal of Community Psychology, 20,* 81-95.

Taylor, C. E., & Owen, L. E. (2006). Small-scale randomized controlled trials need more powerful methods of mediational analysis than the Baron-Kenny method. *Journal of Clinical Epidemiology, 59*(5), 457-464.

Taylor, S. E., Sherman, D. K., Kim, H. S., Jarcho, J., Takagi, K., & Dunagan, M. S. (2004). Culture and social support: Who seeks it and why? *Journal of Personality and Social Psychology, 87*(3), 354-362.

Taylor, M. G., & Lynch, S. M. (2004). Trajectories of impairment, social support, and depressive symptoms in later life. *Journal of Gerontology: Social Sciences, 59B,* S238-S246.

Taylor, S. E. Klein, L. C., Lewis, B., Gruenewald, T., Gurung, R. A. R., & Updegraff, J. (2000). The female stress response: Tend and befriend not fight or flight. *Psychological Review, 107,* 411-429.

Tehrani, E., Krussel, J., Borg, L., & Munk-Jorgensen, P. (1996). Dropping out of psychiatric treatment: A prospective study of a first-admission cohort. *Acta Psychiatrica Scandinavica, 94*(4), 266-271.

Tennen, H., Affleck, G., Armeli, S., & Carney, M. A. (2000). A daily process approach to coping: Linking theory, research and practice. *American Psychologist, 55*(6), 626-636.

The Enhanced Recovery in Coronary Heart Disease Patients (ENRICHD) (2003). *Journal of American Medical Association, 289*(3), 3106-3116.

Thoits, P. A. (1982). Life stress, social support, and psychological vulnerability: Epidemiological considerations. *Journal of Community Psychology, 10,* 341-362.

Thoits, P. A. (1983). Multiple identities and psychological well-being: A reformulation and test of the social isolation hypothesis. *American Sociological Review, 48,* 174-187.

Thoits, P. A., Hohmann, A. A., Harvey, M. R., & Fletcher, B. (2000). Similar-other support for men undergoing coronary artery bypass surgery. *Health Psychology, 19,* 264-273.

Thornicroft, G., Margolius, O., & Jones, D. (1992). The TAPS project. 6: New long-stay psychiatric patients and social deprivation. *British Journal of Psychiatry, 161,* 621-624.

Thornicroft, G., & Breakey, W. R. (1991). The COSTAR programme. 1: Improving social networks of the long-term mentally ill. *British Journal of Psychiatry, 159,* 245-249.

Thornicroft, G. (1991). Social deprivation and rates of treated mental disorder: Developing statistical models to predict psychiatric service utilisation. *British Journal of Psychiatry, 158,* 465-484.

Tischler, G. (1966). Decision-making processes in the emergency room. *Archives of General Psychiatry, 14,* 69-78.

Todd, N. A., Bennie, E. H., & Carlisle, J. M. (1976). Some features of 'new long-stay' male schizophrenics. *British Journal of Psychiatry, 129,* 424-427.

Tohen, M., Waternaux, C. M., & Tsuang, M. T. (1990). Outcome in mania: A 4-year prospective follow-up of 75 patients using survival analysis. *Archives of General Psychiatry, 47,* 1106-1111.

Tomaka, J., Thompson, S., & Palacios, R. (2006). The relation of social isolation, loneliness, and social support to disease outcomes among the elderly. *Journal of Aging and Health, 18*(3), 359-384.

Tondo, L., Burrai, C., Scamonatti, L., & Weissenberger, J. (1988). Comparison between clinician-rated and self-reported depressive symptoms in Italian psychiatric clients. *Neuropsychobiology, 19*(1), 1-5.

Torgrud, L. J., Walker, J. R., Murray, L., Cox, B. J., Chartier, M., & Kjernisted, K. D. (2004). Deficits in perceived social support associated with generalized social phobia. *Cognitive Behaviour Therapy, 33,* 87-96.

Trompenaars, F. J., Masthoff, E. D., Van Heck, G. L., Hodiamont, P.P., & De Vries, J. (2006). The WHO quality of life assessment instrument (WHOQOL-100): Investigating its discriminant ability for psychiatric outpatients. *European Journal of Psychological Assessment, 22*(3), 207-215.

Trunzo, J. J., & Pinto, B. M. (2003). Social support as a mediator of optimism and distress in breast cancer survivors. *Journal of Consulting and Clinical Psychology, 71*(4),

805-811.

Tsoi, W. F., & Wong, T. W. F. (1991). A 15-year follow-up study of Chinese schizophrenic patients. *Acta Psychiatrica Scandinavica, 84,* 217-220.

Turnbull. J. E., George, L. K., & Landerman, R. (1990). Social outcomes related to age of onset among psychiatric disorders. *Journal of Consulting and Clinical Psychology, 58*(6), 832-839.

Turner, R. J. (1999). Social support and coping. In A. V. Horwitz & T. L. Scheid (Eds.), *A handbook for the study of mental health: Social contexts, theories, and systems* (pp. 198-210). New York: Cambridge University Press.

Turner, R. J. (1981). Social support as a contingency in psychological well-being. *Journal of Health and Social Behaviour, 22,* 357-367.

Turner, R. J. (1981). Social support as a contingency in psychological well-being. *Journal of Health and Social Behavior, 22,* 357-367.

Uchino, B. N. (2004). *Social Support and Physical Health: Understanding the Health Consequences of Relationships.* New Haven, CT: Yale University Press.

Ullah, P., Banks, M., & Warr, P. (1985). Social support, social pressures and psychological distress during unemployment. *Psychological Medicine, 15*(2), 283-295.

Unger, J. B., McAvay, G., Bruce, M. L., Berkman, L., & Seeman, T. (1999). Variations in the impact of social network characteristics of physical functioning in elderly persons: MacArthur studies of successful aging. *Journal of Gerontology, 54,* 245-251.

Unger, J. B., McAvey, G., Bruce, M. L., Berkman, L., & Seeman,T. (1999). Variation in the impact of social network characteristics on physical functioning in elderly persons: MacArther Studies of Successful Aging. *Journal of Gerentology, 54(B),* S1-S7.

Utsey, S. O., Ponterotto,J. G., Reynolds, A, L., & Cancelli, A. A. (2000). Racial discrimination, coping, life satisfaction, and self-esteem among African Americans. *Journal of Counseling and Development, 78,* 72-80.

Vaananen, A., Buunk, B. P., KivimΣki, M., Pentti, J., & Vahtera, J. (2005). When it is better to give than to receive: Long-term health effects of perceived reciprocity in support exchange. *Journal of Personality and Social Psychology, 89*(2), 176-193.

Vaerum,V. N., & McCabe, S. B. (2001). Rejection of dysphoric actors and implications of depressive symptom display. *Journal of Social and Clinical Psychology, 20,* 431-451.

Valliant, G. E. (2002). *Aging Well.* Boston, MA: Little, Brown.

van Gorp, W. G., Altshuler, L., Theberge, D. C., et al. (1998). Cognitive impairment in euthymibic bipolar patients with and without prior alcohol dependence: A preliminary study. *Archives General Psychiatry, 55,* 41-46.

Vaux, A. (1985). Variations in social support associated with gender, ethnicity, and age. *Journal of Social Issues, 41*(1), 89-110.

Vega, W., Kolody, B., Valle, R., & Weir, J. (1991). Social networks, social support, and their relationship to depression among immigrant Mexican women. *Human Organization, 50*(2), 154-162.

Vetter, P., & Koller, O. (1996). Clinical and psychosocial variables in different diagnostic groups: Their interrelationships and value as predictors of course and outcome during a 14-year follow-up. *Psychopathology, 29,* 159-168.

Vinokur, A. D., & Schul, Y. (2002). The web of coping resources and pathways to reemployment following a job loss. *Journal of Occupational Health Psychology, 7,* 68-83.

Vinokur, A., Schul, Y., & Caplan, R. D. (1987). Determinants of perceived social support: Interpersonal transactions, personal outlook, and transient affective states. *Journal of Personality and Social Psychology, 53(6),* 1137-1145.

Viswesvaran, C., Sanchez, J. I., & Fisher, J. (1999). The role of social support in the process of work stress: A meta analysis. *Journal of Vocational Behavior, 54,* 314-334.

Vitaliano, P. P., Scanlan, J. M., Zhang, J., Savage, M. V., Brummett, B., Barefoot, J., & Siegler, I. C. (2001). Are the salutogenic effects of social supports modified by income? A test of an "Added Value Hypothesis". *Health Psychology, 20*(3), 155-165.

Vogel, D. L., & Wei, M. (2005). Adult attachment and help-seeking intent: The mediating roles of psychological distress and perceived social support. *Journal of Counseling Psychology, 52*(3), 347-357.

Vonokur, A. D., & van Ryn, M. (1993). Social support and undermining in close relationships: Their independent effects on the mental health of unemployed persons. *Journal of Personality and Social Psychology, 65,* 350-359.

Vranceanu, A., Gallo, L. C., & Bogart, L. M. (2006). Hostility and perceptions of support in ambiguous social interactions. *Journal of Individual Differences, 27*(2), 108-115.

Wahl, O. F. (1999). Mental health consumers' experience of stigma. *Schizophrenia Bulletin,25,* 467-478.

Waite, L. J., & Gallagher, M. (2001). *Case for Marriage: Why Married People are Happier, Healthier, and Better Off Financially.* New York, NY: Doubleday.

Waldron, I. (1980). Employment and women's health: An analysis of causal relationships. *International Journal of Health Services, 10*(3), 435-454.

Walker, M., Wasserman, S., & Wellman, B. (1993). Statistical models for social support networks. *Sociological Methods and Research, 22*(1), 71-98.

Walter-Ginzbutg, A., Blumstein, T., Chetrit, A., & Morgan, P. (2002). Social factors and mortality in the old-old in Israel: The CALAS study. *Journal of Gerontology: Social Sciences, 59B,* S308-S318.

Wang, H. H., Wu, S. Z., & Liu, Y. Y. (2003). Association between social support and health outcomes: A meta-analysis. *The Kaohsiung Journal of Medical Sciences, 19*(7), 345-351.

Wang, C. W., Iwaya, T., Kumano, H., Suzukamo, Y., Tobimatsu, Y., & Fukudo, S. (2002). Relationship of health status and social support to the life satisfaction of older adults. *The Tohoku Journal of Experimental Medicine, 198,* 141-149.

Ware, J. E. Jr., Manning, W. G. Jr., Duan, N., Wells, K., & Newhouse, J. P. (1984). Health status and the use of outpatient mental health services. *American Psychologist, 39,*

1090-1100.

Ware, J. E., & Sherbourne, C. D. (1992). Mental Health Index (MHI-5): The MOS 36-Item Short Form Health Survey [SF(36)]. *Medical Care, 30,* 473-481.

Wasylenki, D., Georing, P., Cochrane, J., et al. (2000). Tertiary mental health services: I. Key concepts. *Canadian Journal of Psychiatry, 45,* 179-184.

Watanabe, M., Irie, M., & Kobayashi, F. (2004). Relationship between effort-reward imbalance, low social support and depressive state among Japanese male workers. *Journal of Occupational Health, 46,* 78-81.

Waterston, T., Alperstein, G., & Brown, S. S. (2004). Social capital: A key factor in child health inequalities. *Archives of Disease in Childhood, 89,* 456-459.

Watson, C. G. (1990). Psychometric posttraumatic stress disorder measurement techniques: A review. *Psychological Assessment: A Journal of Consulting and Clinical Psychology, 2*(4), 460-469.

Watson, D. (2000). *Mood and Temperament.* New York: NY, Guilford Press.

Weary, G., & Gannon, K. (1996). Depression, control motivation, and person perception. In P. M. Gollwitzer, and J. A. Bargh (Eds.), *The Psychology of action (pp. 146-167).* New York, NY: Guilford Press.

Weber, M. (1946). Class, status, party. In H. H. Gerth & C. W. Mills (Eds.), *From Max Weber: Essays in sociology* (pp.180-195). New York: Oxford University Press.

Wehr, T. A, Turner, E. H., Shimada, J. M., et al. (1998). Treatment of a rapidly cycling bipolar patient by using extended bedrest and darkness to stabilize the timing and duration of sleep. *Biological Psychiatry, 43(11),* 822-828.

Wei, M., Mallinckrodt, B., Russell, D, W., & Abraham, W. T. (2004).maladaptive perfectionism as a mediator and moderator between adult attachment and psychological distress. *Journal of Counceling Psychology, 51,* 201-212.

Wei, M., Heppner, P. P., & Mallinckrodt, B. (2003). Perceived coping as a mediator between attachment and psychological distress: A structural equation modeling approach. *Journal of Counseling Psychology, 50,* 438-447.

Weiss, R. S. (1973). *Loneliness: The Experience of Emotional and Social Isolation.* Cambridge, MA: MIT Press.

Weiss, R. S. (1974). The provisions of social relationships. In Z. Rubin (Ed.), *Doing unto others* (pp. 17-26). Englewood Cliffs, NJ: Prentice Hall.

Weiss, C. H. (1998). *Evaluation: Methods for studying programs and policies* (2nd ed.). Upper Saddle River, NJ: Prentice Hall.

Weissman, M. M. (1971). The social adjustment scale. In I. McDowell & C. Newell (Eds.), *Measuring health: A guide to rating scales and questionnaires* (pp.192-203). New York: Oxford University Press.

Wender, P. H., Rosenthal, D., Kety, S. S., Schulsinger, F., & Welner, J. (1973). Social class and psychopathology in adoptees. *Archives of General Psychiatry, 28,* 318-325.

Westermeyer, J. F., & Harrow, M. (1988). Course and outcome in schizophrenia. In M. T. Tsuang & J. C. Simpson (Eds), *Handbook of schizophrenia: Nosology, epidemiology and genetics* (Vol. 3, pp. 205-244). Elsevier: Amsterdam.

Wheaton, B. (1978). The sociogenesis of psychological disorder: Reexamining the causal

issues with longitudinal data. *American Sociological Review, 43,* 383-403.

Wheaton, B. (1980). The sociogenesis of psychological disorder: An attributional theory. *Journal of Health and Social Behavior, 21,* 100-124.

Whisman, W. A. (2001). The association between marital satisfaction and depression. In S. R. H. Beach (Ed.), *Marital and Family Processes in Depression (pp. 3-24).* Washington, DC: American Psychological Association.

Whisman, M. A., Sheldon, C. T., & Goering, P. (2000). Psychiatric disorders and dissatisfaction with social relationships: Does type of relationship matter? *Journal of Abnormal Psychology, 109*(4), 803-808.

Whitbeck, C. (1977). Causation in medicine: The disease entity model. *Philosophy of Science, 44,* 619-637.

White, S. L. (1986). Census tract of residence and he use of C. M. H. C. services. *Administration in Mental Health, 13,* 275-284.

Whitmer, D. A., Hawley, S. R., Orr, S. A., St. Romain, T., & Molgaard, C. A. (2006). Social networks and best practices in public health: The example of regional billing groups. *Public Health Nursing, 23*(6), 541-546.

Whittemore, R., Rankin, S. H., Callahan, C. D., Leder, M. C., & Carol, D. L. (2002). The peer advisor experience: Providing social support. *Qualitative Health Research, 10,* 260-276.

WHOQOL Group. (1994). The development of the World Health Organization Quality of Life Assessment instrument (WHOQOL). In J Orley & W. Kuyken (Eds.), *Quality of life assessment: International Perspectives (pp. 41-60).* Berlin: Springer-Verlag.

Wiersma, D., Giel, R., De Jong, A., & Slooff, C. J. (1983). Social class and schizophrenia in a Dutch cohort. *Psychological Medicine, 13,* 141-150.

Wiggins, J. S. (1988). *Personality and prediction: Principles of personality assessment.* Malabar, FL: Robert E. Kreiger.

Wiley, J., & Camacho, T. (1980). Life-style and future health: Evidence from the Alameda County study. *Preventive Medicine, 9,* 1-21.

Wilkinson, G. S. (1975). Isolation and psychological disorder. *Psychological Reports, 36,* 631-634.

Williams, R. M., Turner, A. P., Hatzakis, M., Chu, S., Rodriquez, A. A., Bowen, J. D., & Haselkorn, J. K. (2004). Social support among veterans with multiple sclerosis. *Rehabilitation Psychology, 49*(2), 106-113.

Williams, A., Ware, J., & Donald, C. (1981). A model of mental health, life events, and social supports applicable to the general population. *Journal of Health and Social Behavior, 22,* 324-336.

Willoughby, C., Brown, E., King, G., Specht, J., & Smith, L. (2003). Discovery of self – what helps and what hinders? In G. King, E. Brown, & L. Smith (Eds.), *Resilience: Learning from People with Disabilities and Turning Points in their Lives (pp. 89-128).* Westport, CT: Praeger.

Winefield, A. H., & Tiggemann, M. (1990). Employment status and psychological well-being: A longitudinal study. *Journal of Applied Psychology, 75*(4), 455-459.

Winnipeg Area Study. (1995).Winnipeg, MB: *Winnipeg Area Study,* University of Manitoba [producer and distributor].

Winnipeg Area Study. (1994).Winnipeg, MB: *Winnipeg Area Study*, University of Manitoba [producer and distributor].

Winnipeg Area Study. (1988). *The moral economy of family life [MDRF].* Winnipeg, MB: Winnipeg Area Study, University of Manitoba [producer and distributor].

Wirtz, P. H., von Kanel, R., Mohiyeddini, C., Emini, L., Ruedisueli, K., Groessbauer, S., & Ehlert, U. (2006). Low social support and poor emotional regulation are associated with increased stress hormone reactivity to mental stress in systemic hypertension. *Journal of Clinical Endocrinology & Metabolism, 91*(10), 3857-3865.

Wood, V., & Robertson, J. F. (1978). Friendship and kinship interaction: Differential effect on the morale of the elderly. *Journal of Marriage and the Family, 40,* 367-374.

Woogh, C. (1986). A cohort through the revolving door. 33rd. Annual meeting of the Canadian Psychiatric Association (1983, Canada). *Canadian Journal of Psychiatry, 31*(3), 214-221.

Wright, E. R., Gronfein, W., & Owens, T. J. (2000). Deinstitutionalization, social rejection and self esteem of former mental patients. *Journal of Health and Social Behavior,* 4(1), 68-90.

Wright, S. (1921). Correlation and causation. *Journal of Agricultural Research, 20,* 557-585.

Wright, S. (1934). The method of path coefficients. *Annals of Mathematical Statistics, 5,* 161-215.

Wu, T., & Serper, M. R. (1999). Social support and psychopathology in homeless patients presenting for emergency psychiatric treatment. Journal of Clinical Psychology, 55, 1127-1133.

Wykes, T., & Holloway, F. (2000). Community rehabilitation: Past failures and future prospects. *International Review of Psychiatry, 12,* 197-205.

Zadro, L., & Williams, K. D. (2003). In K. D. Williams, T. I. Case, and C. L. Govan, in *Responding to the Social World: Implicit and Explicit Processes in Social Judgments and Decisions.* J. Forgas, K Williams, and W. von Hippel, (Eds.). New York, NY: Cambridge University Press.

Zambon, A., Lemma, P., Borraccino, A., Dalmasso, P., & Cavallo, F. (2006). Socio-economic position and adolescents' health in Italy: The role of the quality of social relations. *European Journal of Public Health, 16*(6), 627-632.

Zautra, A., & Simons, L. S. (1978). As assessment of a community's mental health needs. *American Journal of Counseling Psychology, 6,* 351-362.

Zealley, A. K., & Aitken, R. C. B. (1969). Measurement of mood. *Proceedings of the Royal Society of Medicine, 62,* 993-996.

Zimmerman, F. J., & Bell, J. F. (2005). Income inequality and physical and mental health: Testing associations consistent with proposed causal pathways. *Journal of Epidemiology and Community Health, 60,* 513-521.

Zimmerman, M. A., Ramiriz-Valles, J., Zapert, K. M., & Maton, K. I. (2000). A longitudinal study of stress-buffering effects for urban African-American male adolescent problem behaviors and mental health. *Journal of Community*

Psychology, 28, 17-33.

Zohar, M., Hadas, Z., & Modan, B. (1987). Factors affecting the decision to admit mental patients in a community hospital. *Journal of Nervous and Mental Disease, 175*(5), 301-305.

Zubenko, G. S., Mulsant, B. H., Rifai, A. H., Sweet, R. A., Pasternak, R. E., Marino, L. J., Jr., & Tu, X. (1994). Impact of acute psychiatric inpatient treatment of major depression in late life and prediction of response. *American Journal of Psychiatry, 151,* 987-994.

Appendix A: Survey Measures

This group of questions is needed to determine how active you are socially and how many friends and relatives you can depend upon for support. (Unless otherwise indicated, please circle the number next to the answer you would like to give.)

CONTACT FREQUENCY

How many times in the last **TWO WEEKS** have you gone out socially with other people? For example, gone out shopping or to the movies?

```
MORE THAN THREE TIMES.....5
THREE TIMES.....4
TWICE.....3
ONCE.....2
I HAVE NOT GONE OUT SOCIALLY
DURING THE LAST WEEK.....1
```

Approximately, during the last **TWO WEEK**S, how often did you get together with your friends or relatives, either in your home or in their home?

```
MORE THAN THREE TIMES.....5
THREE TIMES.....4
TWICE.....3
ONCE.....2
DURING THE LAST WEEK THERE WAS
NONE OF THIS TYPE OF CONTACT.....1
```

In the last **TWO WEEKS**, approximately how many times have you had contact with your friends or relatives that did not include a personal visit? For example, you called them on the phone or they wrote you a letter (regardless of who initiated the contact).

```
MORE THAN THREE TIMES.....5
THREE TIMES.....4
TWICE.....3
ONCE.....2
DURING THE LAST WEEK THERE WAS
NONE OF THIS TYPE OF CONTACT.....1
```

During the last **TWO WEEKS**, how many times did you depend on your friends or relatives for help, advice, money, or friendship?

```
MORE THAN THREE TIMES.....5
THREE TIMES.....4
TWICE.....3
ONCE.....2
I DID NOT DEPEND ON MY FRIENDS OR
RELATIVES DURING THE LAST WEEK.....1
```

SATISFACTION WITH SUPPORT

Are you satisfied with the support you receive from others?

```
      VERY                                                          VERY
DISSATISFIED                                                     SATISFIED
      0      1      2      3      4      5      6      7      8      9     10
```

The following group of questions are about your employment (PLEASE INCLUDE SELF-EMPLOYMENT), education, and your household income. The actual source of your income is not important but we do need to know the amount of funds from ALL sources available to your household. If you are not in paid employment please enter "zero" to both parts of the next question.

In the last **SIX MONTHS**, how many weeks were you employed?

FULL TIME? __ PART TIME?__

What is your employment situation at the present time? Please circle the number next to all responses that apply to you. Are you:

a) EMPLOYED FULL TIME.....1
b) EMPLOYED PART TIME.....2
c) UNEMPLOYED, LOOKING FOR WORK.....3
d) UNEMPLOYED, NOT LOOKING FOR WORK.....4
e) RETIRED.....5
f) IN SCHOOL.....6
g) MAINTAINING A HOME.....7
h) VOLUNTEERING.....8
I) UNEMPLOYED DUE TO ILLNESS.....9
j) OTHER (PLEASE SPECIFY)_____99

If you are employed, how many hours have you worked in the last **TWO WEEKS**? If you are not employed for any reason, please circle zero hours. If during the last two-week period you were on holidays, please use the full two-week period prior to being on holiday.

ZERO HOURS.....8
1 TO 10 HOURS.....7
11-20 HOURS.....6
21-30 HOURS.....5
31-40 HOURS.....4
41-50 HOURS.....3
51-60 HOURS.....2
MORE THAN 60 HOURS.....1

Would you please indicate your **household's TOTAL** available income (before taxes) for the **1998 - TAX YEAR**? Please include **ALL** funds that come into your household regardless of the source. For example, if the rent on your principle residence was paid for you then add the value of the rent to your income. (Circle the closest alternative.)
to your income. (Circle the closest alternative.)

NO INCOME.....00	16,000-17,999.....08	34,000-35,999.....17
UNDER $3,000.....01	18,000-19,999.....09	36,000-37,999.....18
3,000-5,999.....02	20,000-21,999.....10	38,000-39,999.....19
6,000-7,999.....03	22,000-23,999.....11	40,000-44,999.....20
8,000-9,999.....04	24,000-25,999.....12	45,000-49,999.....21
10,000-11,999.....05	26,000-27,999.....13	50,000-54,999.....22
12,000-13,999.....06	28,000-29,999.....14	55,000-59,999.....23
14,000-15,999.....07	30,000-31,999.....15	60,000-(AND OVER)...24
	32,000-33,999.....16	DON'T KNOW.....98

FINANCIAL ADEQUACY PERCEPTION

The following questions deal with how satisfied you PRESENTLY are with your financial situation and how it influences your daily life. If you are very satisfied, you would circle the number 7. If you are very dissatisfied, you would circle the 1. If you are neither satisfied nor dissatisfied you would respond with a 4 which means you are as satisfied as you are dissatisfied. Now, please tell me how satisfied you are with the following items. (Please circle one of the responses.)

a) the amount of money you have for clothes and personal expenses.

VERY DISSATISFIED						VERY SATISFIED	DO NOT KNOW
1	2	3	4	5	6	7	8

b) the amount of money that you have to spend on things for your home.

VERY DISSATISFIED						VERY SATISFIED	DO NOT KNOW
1	2	3	4	5	6	7	8

c) the amount of money you have to spend on the people you love.

VERY DISSATISFIED						VERY SATISFIED	DO NOT KNOW
1	2	3	4	5	6	7	8

d) the amount of money you have been able to save for future needs. (For example, retirement.)

VERY DISSATISFIED						VERY SATISFIED	DO NOT KNOW
1	2	3	4	5	6	7	8

e) the amount of money you have available for leisure activities.

VERY DISSATISFIED						VERY SATISFIED	DO NOT KNOW
1	2	3	4	5	6	7	8

f) the money you have available for your family's (household's) financial needs.

VERY DISSATISFIED						VERY SATISFIED	DO NOT KNOW
1	2	3	4	5	6	7	8

The following group of questions are necessary to evaluate how you have been feeling lately. Please tick the box that comes closest to the way you have been feeling during the past TWO WEEKS

EWB component of the and Health Survey see Hays, et al., 1993)

How much of the time during the last two weeks?	All of the time	Most of the time	A good bit of the time	Some of the time	A little of the time	None of the time
Have you been very nervous?	☐	☐	☐	☐	☐	☐
Have you felt calm and peaceful?	☐	☐	☐	☐	☐	☐
Have you felt downhearted and blue?	☐	☐	☐	☐	☐	☐
Have you been a happy person?	☐	☐	☐	☐	☐	☐
Have you felt so down in the dumps that nothing could cheer you up?	☐	☐	☐	☐	☐	☐

During the last **SIX MONTHS** if you have been ill with psychiatric symptoms and you were NOT admitted to the hospital, what did you do to care for yourself? For example, are there community resources you depend upon?_____

_____

_____

We would now like to ask you a few questions about your vital statistics that will aid in interpreting results.

Are you married or cohabitating with a partner at present? YES____1 NO____2

Are you? FEMALE_____1 MALE_____2

What is your age in years? _____

In total, how many years of schooling do you have? This includes the total of grade school, high-school, vocational, technical, and university training. NUMBER OF YEARS_____

What psychiatric diagnosis did you get either currently or in the past?_____

What type of health professional gave you this diagnosis?_____

During the last **TWO WEEKS** how severe were your psychiatric symptoms on your worst day?

NO SYMPTOMS — THE WORST THEY HAVE EVER BEEN

0 1 2 3 4 5 6 7 8 9 10

Have you ever been hospitalized for your psychiatric disorder/mental health difficulties? Please check whatever records you have available. **YES**_____ **NO** _____

In the last **SIX MONTHS**, to the best of your recollection, how many nights did you spend in the hospital for psychiatric reasons? Approximately how many nights in each of the following months? Please indicate the number of nights in the appropriately labelled boxes. **NUMBER OF NIGHTS** _____

Note: The boxes below represent the item on one of the surveys at a particular point in time and the verbiage would change for each point in time.

Nov 1995	Dec 1995	Jan 1996	Feb 1996	Mar1996	April 1996

Appendix B: Zero-order and Partial Correlations Through Time

Table B1.

Zero-order correlation coefficients between original Time-2 variables. N = 146.

	CF	SAT	SS	ED	EMP	INC	FAD	SYMP
CF	--	+.48 p < .001	+.83 p < .001	+.01 p = .930	+.06 p = .497	-.01 p = .913	+.08 p = .323	-.21 p = .009
SAT		--	+.89 p < .001	+.001 p = .990	+.08 p = .335	+.09 p = .238	+.22 p = .008	-.46 p < .001
SS			--	.004 p = .970	+.08 p = .333	+.06 p = .495	+.18 p = .029	-.405 p < .001
ED				--	+.23 p = .004	+.20 p = .014	+.01 p = .861	+.03 p = .701
EMP					--	+.22 p = .006	+.15 p = .070	-.10 p = .208
INC						--	+.37 p < .001	-.04 p = .633
FAD							--	-.22 p = .007
SYMP								--

Note. The abbreviations are as follows: p = two-tailed probabilities, CF = contact frequency, SAT = satisfaction with support, SS = social support compound score, ED = years of formal schooling, EMP = employment score, INC = Family income score, FAD = Financial Adequacy Score, and SYMP = Symptomatology measure. All probabilities are two tailed for the appropriate values related to directional predictions this value needs divided by two.

Table B2.

Partial correlation coefficients after controlling for the effects of age grouping, hospitalization history, postal code, marital status, DRG, and gender Time-2 variables. N = 146.

	CF	SAT	SS	ED	EMP	INC	FAD	SYMP
CF	--	+.48 p < .001	+.83 p <.001	-.03 p = .770	+.04 p = .680	+.08 p = .355	+.15 p = .069	-.24 p = .004
SAT		--	+.89 p < .001	-.023 p = .789	+.04 p = .610	+.11 p = .174	+.25 p = .003	-.47 p < .001
SS				-.027 p = .746	+.05 p = .589	+.11 p = .177	+.24 p = .004	-.42 p < .001
ED				--	+.15 p = .066	+.28 p = .001	+.09 p = .298	+.03 p = .696
EMP					--	+.21 p = .010	+.24 p = .005	-.073 p = .388
INC						--	+.35 p < .001	-.01 p = .908
FAD							--	-.19 p = .020
SYMP								--

Note. The abbreviations are as follows: p = two-tailed probabilities, CF = contact frequency, SAT = satisfaction with support, SS = social support compound score, ED = years of formal schooling, EMP = employment score, INC = Family income score, FAD = Financial Adequacy Score, and SYMP = Symptomatology measure. All probabilities are two tailed for the appropriate values related to directional predictions this value needs divided by two.

Table B3.

Zero-order correlation coefficients between original Time-3 variables. N = 123.

	CF	SAT	SS	ED	EMP	INC	FAD	SYMP
CF	--	+.41 p < .001	+.82 p < .001	+.07 p = .456	-.04 p = .632	-.03 p = .731	+.05 p = .554	-.27 p = .002
SAT		--	+.86 p < .001	-.03 p = .728	+.01 p = .945	+.05 p = .565	+.34 p < .001	-.54 p < .001
SS			--	-.05 p = .564	-.02 p = .786	+.02 p = .835	+.244 p = .006	-.49 p < .001
ED				--	+.17 p = .057	+.08 p = .370	-.13 p = .135	+.03 p = .754
EMP					--	+.27 p = .002	+.12 p = .195	-.07 p = .411
INC						--	+.35 p < 001.	+.06 p = .530
FAD							--	-.18 p = .041
SYMP								--

Note. The abbreviations are as follows: p = two-tailed probabilities, CF = contact frequency, SAT = satisfaction with support, SS = social support compound measure, ED = years of formal schooling, EMP = employment score, INC = Family income score, FAD = Financial Adequacy Score, DRG = diagnostically related groups, and SYMP = Symptomatology measure. All probabilities are two tailed for the appropriate values related to directional predictions this value needs divided by two.

Table B4.

Partial correlation coefficients after controlling for the effects of age grouping, hospitalization history, postal code, marital status, DRG, and gender. Time-3 variables. N = 123.

	CF	SAT	SS	ED	EMP	INC	FAD	SYMP
CF	--	+.50 p < .001	+.85 p < .001	-.11 p = .225	-.14 p = .153	+.02 p = .808	+.13 p = .207	-.35 p < .001
SAT		--	0.88 p < .001	+.03 p = .754	+.03 p = .792	+.11 p = .258	+.37 p < .001	-.51 p < .001
SS			--	-.04 p = .666	-.06 p = .536	+.08 p = 413	+.29 p 003	-.50 p < .001
ED				--	**+.07** **p = .521**	+.06 p = .543	+.02 p = .851	-.07 p = .754
EMP					--	+.17 p = .086	**+.34** **p = .001**	-.18 p = .080
INC						--	**+.41** **p < .001**	+.04 p = .723
FAD							--	-.15 p = .141
SYMP								--

Note. The abbreviations are as follows: p = two-tailed probabilities, CF = contact frequency, SAT = satisfaction with support, SS = social support compound measure, ED = years of formal schooling, EMP = employment score, INC = Family income score, FAD = Financial Adequacy Score, DRG = diagnostically related groups, and SYMP = Symptomatology measure. All probabilities are two tailed for the appropriate values related to directional predictions this value needs divided by two.

Table B5.

Zero-order correlation coefficients between original Time-4 variables. N = 96.

	CF	SAT	SS	ED	EMP	INC	FAD	SYMP
CF	--	+.58 p < .001	+.88 p < .001	+.15 p = .137	+.04 p = .717	-.01 p = .893	+.08 p = .428	-.20 p = .047
SAT		--	+.90 p < .001	+.09 p = .357	+.07 p = .488	+.04 p = .696	+.32 p < .001	-.24 p = .019
SS			--	+.13 p = .184	+.061 p = .546	+.02 p = .873	+.23 p = .020	-.25 p = .014
ED				--	**+.29** **p = .004**	+.16 p = .116	-.02 p = .809	+.12 p = .249
EMP					--	+.31 p = .001	**+.07** **p = .470**	+.07 p = .481
INC						--	**+.25** **p = .013**	-.05 p = .658
FAD							--	-.20 p = .052
SYMP								--

Note. The abbreviations are as follows: p = two-tailed probabilities, CF = contact frequency, SAT = satisfaction with support, SS = social support compound measure, ED = years of formal schooling, EMP = employment score, INC = Family income score, FAD = Financial Adequacy Score, DRG = diagnostically related groups, and SYMP = Symptomatology measure. All probabilities are two tailed for the appropriate values related to directional predictions this value needs divided by two.

Table B6.

Partial correlation coefficients after controlling for the effects of age grouping, hospitalization history, postal code, marital status, DRG, and gender. Time-4 variables. N = 96.

	CF	SAT	SS	ED	EMP	INC	FAD	SYMP
CF	--	+.58 p < .001	+.87 p <.001	+.04 p = .741	+.05 p = .700	-.01 p = .959	+.10 p = .393	-.18 p = .141
SAT		--	+.91 p < .001	+.05 p = .661	+.08 p = .534	+.13 p = .270	+.31 p = .008	-.24 p = .044
SS			--	+.05 p = .661	+.07 p = .562	+.08 p = .516	+.24 p = .039	-.24 p = .046
ED				--	+.13 p = .300	+.13 p = .281	+.14 p = .250	+.04 p = .739
EMP					--	+.25 p = .036	+.26 p = .027	-.12 p = .335
INC						--	+.40 p < .001	-.09 p = .467
FAD							--	-.15 p = .214
SYMP								--

Note. The abbreviations are as follows: p = two-tailed probabilities, CF = contact frequency, SAT = satisfaction with support, SS = social support compound score, ED = years of formal schooling, EMP = employment score, INC = Family income score, FAD = Financial Adequacy Score, and SYMP = Symptomatology measure. All probabilities are two tailed for the appropriate values related to directional predictions this value needs divided by two.

Appendix C: Cross-Sectional Tables of Effects Through Time

Table C1.

Cross-sectional time-1 direct, indirect, unexplained, spurious, and total effects for the symptomatology path model, including regression beta weights, N = 232.

Variable Pairs IV/DV	Direct Effect	Indirect Effect	Unexplained Effect	Spurious Effect	Total Effect
CF/SAT	β= .1949 Z= 24.40 p < .001	not applicable	-1.4 E-17	1.4 E-17	β= .1949 Z= 7.7150 p < .001
SAT/SCH	invalid path	invalid path	invalid path	invalid path	invalid path
CF/SCH	β= .0499 Z= 1.09 p= .138	not applicable	-1.7 E-18	1.7 E-18	β= .0499 Z= 1.09 p= .138
SAT/EMP	β= 3.3824 Z= 2.17 p= .015	not applicable	-1.22	-0.0193	β= 2.1464 Z= 1.48 p= .069
SCH/EMP	β= 3.692 Z= 4.28 p < .001	not applicable	-0.069	-0.0132	β= 3.6102 Z= 4.16 p < .001
CF/EMP	β= -1.3010 Z= -1.95 p= .026	β= .8434 Z= 2.40 p= .008	1.4 E-17	-8.6 E-7	β= -.4576 Z= -.74 p= .230
SAT/FIN	β= .3283 Z= 1.40 p= .081	β= .2114 Z= 2.05 p= .020	-0.022	0.006	β= .5115 Z= 2.22 p= .013
SCH/FIN	β= .3271 Z= 2.45 p= .007	β= .2307 Z= 3.54 p < .001	-0.006	-0.002	β= .5610 Z= 4.03 p < .001
EMP/FIN	β= .0625 Z= 6.29 p < .001	not applicable	0.007	5.8 E-4	β= .0702 Z= 7.33 p < .001
CF/FIN	β= .0330 Z= .33 p= .371	β= .0517 Z= .76 p= .224	1.4 E-17	-5.4 E-7	β= .0847 Z= .853 p= .197

Table C1. (continued)

SAT/FAD	β= 1.0956 **Z**= 2.92 **p**= .002	β= .3355 **Z**= 1.77 **p**= .038	0.187	-0.002	β= 1.6160 **Z**= 4.46 **p** < .001
SCH/FAD	β= -.4257 **Z**= -1.98 **p**= .024	β= +.3429 **Z**= +2.87 **p**= .002	0.0488	-0.007	β= -.0412 **Z**= .18 **p**= .430
EMP/FAD	β= -.0197 **Z**= -1.15 **p**= .125	β= .0466 **Z**= 4.68 **p** < .001	0.001	7.8 E-5	β= .0282 **Z**= 1.64 **p**= .051
FIN/FAD	β= .7451 **Z**= 6.99 **p** < .001	not applicable	0.0453	-0.00671	β= .6932 **Z**= 7.10 **p** < .001
CF/FAD	β= .1865 **Z**= 1.17 **p**= .121	β= .2644 **Z**= 2.52 **p**= .006	-1.4 E-17	5.4 E-7	β= .4509 **Z**= 2.8430 **p**= .002235
SAT/SYMP	β= -.2703 **Z**= 4.39 **p** < .001	β= -.0752 **Z**= -1.78 **p**= .038	-0.064	0.009	β= -.4009 **Z**= -7.22 **p** < .001
SCH/SYMP	β= -.0072 **Z**= -.21 **p**= .417	β= -.0253 **Z**= -1.75 **p**= .040	-0.013	0.002	β= -.0443 **Z**= -1.16 **p**= .122
EMP/SYMP	β= -.00763 **Z**= -2.98 **p**= . 002	β= -9.3E-4 **Z**= -1.01 **p**= .156	-0.001004	0.00005	β= -.0095 **Z**= -3.46 **p** < .001
FIN/SYMP	invalid path	invalid path	invalid path	invalid path	invalid path
FAD/SYMP	β= -.0345 **Z**= -3.53 **p** < .001	not applicable	-0.017	0.00013	β= -.0514 **Z**= -4.99 **p** < .001
CF/SYMP	β= -.0610 **Z**= -2.37 **p**= .009	β= -.0651 **Z**= 4.26 **p** < .001	1.40000e-17	0.002	β= -.1242 **Z**= -4.97 **p** < .001

Note: Abbreviations are as follows: β = path coefficients, **Z** = Z-scores , **p** = one-tailed probability values, IV/DV = independent/ dependent variable pairs for each path, CF = contact frequency, SAT = satisfaction with support, SCH = years of education, EMP = employment score, FIN = family income, FAD = financial adequacy score, SYMP = symptomatology score, and not applicable = it was physically impossible for an indirect effect to exist.

Table C2.

Cross-Sectional Time-2 Replication Model. Direct, indirect, unexplained, spurious, and total effects for the symptomatology valid path model, including regression beta weights.

Variable Pairs IV/DV	Direct Effect	Indirect Effect	Unexplained Effect	Spurious Effect	Total Effect
CF/SAT (+) 1	β =.285723 **Z** =6.625233 **p** <.0000001	not applicable	0	0	β =.285723 **Z** = 6.625 **p** < .0000001
SAT/ED (+) 2	invalid path	invalid path	invalid path	invalid path	invalid path
CF/ED (+) 3	β = .0067417 **Z** =.1088 **p** = .4566789	0	0	-0.001475	β = .004942 **Z** = .084 **p** = .466528
SAT/EMP (+) 4	β = .565955 **Z** = .755604 **p** =.2249433	0	0.0910904	-0.00817	β = .648875 **Z** = .967 **p** = .166772
ED/EMP (+) 5	β = 1.582801 **Z** = 2.88989 **p** = .0019269	not applicable	0.003337	-0.00233	β = 1.583809 **Z** = 2.901 **p** <.0018599
CF/EMP (+) 6	β = .10244 **Z** = .2301291 **p** =.4089956	β =.1717632 **Z** = .7319762 **p** = .2376	0	-0.002334	β =.271969 **Z** = .681 **p** = .2479357
SAT/FIN (+) 7	β = .305859 **Z** =1.322885 **p** =.09294	β =.0317337 **Z** = .7141 p = .2376	-0.088593	-0.002202	β = .246798 **Z** = 1.185 **p** = .1180087
ED/FIN (+) 8	β = .3378 **Z** = 1.94635 **p** = .02581	β = .08875 **Z** = 1.7424 **p** = .04072	-0.0002	-0.00086	β = .42546 **Z** = 2.488 **p** = .00642
EMP/FIN (+) 9	β = .05607 **Z** = 2.1841 **p** =.01449	not applicable	0.0125425	0.000713	β =.069326 **Z** = 2.763 **p** =.0028636
CF/FIN (+) 10	β = -.117991 **Z** = -.8603 **p** = .1948	β =.104939 **Z** = 1.391 p = .0821122	0	-0.00063	β = -.013681 **Z** = -.11 **p** = .45621

Table C2. (continued)

SAT/FAD (+) 11	β = .779644 **Z** = 2.0696 **p** = .019244	β = .216617 **Z** = 1.435 **p** = .07566	-0.060294	-0.00273	β =.935695 **Z** = 2.683 **p** = .003648
ED/FAD (+) 12	β = -.256316 **Z** = -.89296 **p** = .18594	β = .30903 **Z** = 2.3374 **p** = .009709	0.0026507	-0.003359	β =.052005 **Z** = .175 **p** = .43054
EMP/FAD (+) 13	β = .040719 **Z** = .9558115 **p** = .16958	β =.03215 **Z** = 1.938 **p** =.02633	0.0059977	0.000255	β =.079122 **Z** = 1.822 **p** =.03423
FIN/FAD (+) 14	β =**.57339** **Z** = 4.2004 **p** = .000013	not applicable	0.0335941	0.0012509	β =.608235 **Z** = 4.609 **p** = .0000020
CF/FAD (+) 15	β = -.01468 **Z** = .06538 **p** = .47393	β =.2248 **Z** = 1.6261 p = .05197	0	-0.0001	β = .210049 **Z** = .993 **p** = .16036
SAT/SYMP (-) 16	β =-.46729 **Z** =-5.143 p <.0000001	β =-.033836 **Z** =-1.3536 p = .087938	0.069574	0.0031882	β = -.49098 **Z** = -6.32 p <.0000001
ED/SYMP (-) 17	β = .033759 **Z** = .49284 **p** =.30948	β =-.005896 **Z** = -.256452 **p** = .3988	-0.001703	0.0019591	β = .028119 **Z** = .385 **p** = .35012
EMP/SYMP (-) 18	β =-.00936 **Z** = -.92841 **p** = .176599	β =-.001287 **Z** = -.5104 **p** = .3049	-0.003078	0.000154	β = -.013569 **Z** =-1.266 **p** = .10276
FIN/SYMP (-) 19	β = .025506 **Z** = .74725 **p** = .2275	β = -.021382 **Z** =-1.78193 **p** = .0427849	-0.021544	0.000848	β = -.016572 **Z** = -.48 **p** = .31562
FAD/SYMP (-) 20	β = -.03729 **Z** = -1.8843 **p** =.02976	not applicable	-0.016972	0.000206	β =-.054056 **Z** = -2.729 **p** =.00318
CF/SYMP (-) 21	β =.0095 **Z** = .1785 **p** = .4292	β =-.1441 **Z** = -4.1567 **p** = .000016	0	-0.00071	β =-.133877 **Z** = -2.63 **p** =.0042692

Note: Abbreviations are as follows: β = path coefficients, **Z** = Z-scores , **p** = one-tailed probability values, IV/DV = independent/ dependent variable pairs for each path, CF = contact frequency, SAT = satisfaction with support, ED = years of education, EMP = employment score, FIN = family income, FAD = financial adequacy score, SYMP = symptomatology score. Valence signs indicate the predicted direction of the relationship, and not applicable = it was physically impossible for an indirect effect to exist.

Table C3.

Cross-sectional Time-3 direct, indirect, unexplained, spurious, and total effects for the symptomatology valid path model, including regression beta weights, N = 123.

Variable Pairs IV/DV	Direct Effect	Indirect Effect	Unexplained Effect	Spurious Effect	Total Effect
CF/SAT	β= .23217 Z= 4.9956 p < .0001	not applicable	0	0	β= .23217 Z= 4.996 p < .0001
SAT/ED	invalid path	invalid path	invalid path	invalid path	invalid path
CF/ED	β= -.04714 Z= -.7261 p= .23392	not applicable	0	-0.001384	β= -.4853 Z= -.747 p= .22753
SAT/EMP	β= .25318 Z= .29325 p= .38467	not applicable	-0.196181	-0.006962	β= .05004 Z= .064 p= .47449
ED/EMP	β= 1.1638 Z= 1.883 p = .0299	not applicable	0.0142557	-0.00036	β= 1.17768 Z= 1.92 p= .02718
CF/EMP	β= -.218835 Z= -.4446 p= .3283	β= .00393 Z= .01814 p= .49276	2.8 E-17	-0.001616	β= -.21653 Z= -.48 p= .31561
SAT/INC	β= .19083 Z= .7436 p= .2286	β= .02028 Z= .2918 p= .3852	-0.071337	-0.00093	β= .13885 Z= .577 p= .28196
ED/INC	β= .06164 Z= .3309 p= .3704	β= .09323 Z= 1.5934 p = .0555	0.0038483	-0.00053	β= .15819 Z= .841 p= .20017
EMP/INC	β= .08011 Z= 2.990 p = .00139	not applicable	0.0022006	0.00006	β= .08237 Z= 3.151 p= .0008
CF/INC	β= -.07469 Z= -.5103 p= .3049	β= .02418 Z= .3367 p= .3682	0	-0.00022	β= -.05072 Z= -.37 p= .35569

Table C3. (continued)

SAT/FAD	β= 1.5205 Z= 4.143 p= .000017	β= .11476 Z= .78587 p= .21597	-0.179378	0.0016586	β= 1.45757 Z= 4.064 p < .0001
ED/FAD	β= -.56371 Z= -2.120 p= .0170	β= .10711 Z= .95398 p= .17005	-0.00961	0.021728	β= -.44449 Z= -1.502 p= .0666
EMP/FAD	β= .023432 Z= .59165 p= .27704	β= .041298 Z= 2.329 p = .00836	-0.009348	0.000234	β= .05562 Z= 1.303 p= .09629
INC/FAD	β= .5150 Z= 3.991 p < .0001	not applicable	0.0328123	0.0028059	β= .55112 Z= 4.154 p < .0001
CF/FAD	β= -.21911 Z= -1.0479 p= .14734	β= .34853 Z= 2.479 p= .0066	0	-676	β= .12875 Z= .594 p= .27625
SAT/SYMP	β= -.57085 Z= -6.267 p < .0001	β= .00615 Z= .3943 p= .3467	-0.027602	0	β= -.59234 Z= -7.155 p < .0001
ED/SYMP	β= .01520 Z= .23035 p= .4891	β= -.008317 Z= -.5504 p= .29103	0.0150787	0.0021655	β= .02413 Z= .314 p= .36768
EMP/SYMP	β= -.01312 Z= -1.1335 p= .09101	β= .003595 Z= 1.2676 p= .1024	0.00036	0.00006	β= -.0091 Z= -.824 p= .20497
INC/SYMP	β= .04482 Z= 1.3996 p= .08087	not applicable	-0.020131	-0.00116	β= .02358 Z= .648 p= .25849
FAD/SYMP	invalid path	invalid path	invalid path	invalid path	invalid path
CF/SYMP	β= -.03692 Z= -.7144 p= .23842	β= -.1327 Z= 3.8199 p < .0001	0	0	β= -.1696 Z= -3.15 p= .00082

Note: Abbreviations are as follows: **β** = path coefficients, **Z** = Z-scores , **p** = one-tailed probability values, IV/DV = independent/ dependent variable pairs for each path, CF = contact frequency, SAT = satisfaction with support, ED = years of education, EMP = employment score, INC = family income, FAD = financial adequacy score, SYMP = symptomatology score, and not applicable = it was physically impossible for an indirect effect to exist.

Table C4.

Cross-sectional Time-4 direct, indirect, unexplained, spurious, and total effects for the symptomatology valid path model, including regression beta weights, N = 96.

Variable Pairs IV/DV	Direct Effect	Indirect Effect	Unexplained Effect	Spurious Effect	Total Effect
CF/SAT	β= .32383 Z= 7.0605 p < .0001	not applicable	0	0.004443	β= .32827 Z= 7.06 p < .0001
SAT/ED	invalid path	invalid path	invalid path	invalid path	invalid path
CF/ED	β= .1141 Z= 1.4999 p= .06681	not applicable	0	0	β= .114056 Z= 1.50 p= .06681
SAT/EMP	β= .58678 Z= .5812 p= .28025	not applicable	0.017068	0.0154095	β= .585124 Z= .688 p= .2457
ED/EMP	β= 1.787 Z= 2.935 p= .001669	not applicable	-0.005907	-0.602831	β= 1.17839 Z= 2.988 p = .0014
CF/EMP	β= -.220337 Z= -.3873 p= .34926	β= .39385 Z= 1.0901 p= .137845	-2.78 E-17	0	β= .17351 Z= .364 p= .35793
SAT/INC	β= .135348 Z= .3939 p= .3468	β= .05828 Z= .56969 p= .28444	-0.086686	0.0030431	β= .10992 Z= .377 p= .35309
ED/INC	β= .17563 Z= .8135 p= .20795	β= .17731 Z= 2.054 p= .01998	-0.011961	0.008732	β= .34187 Z= 1.625 p= .05208
EMP/INC	β= .099215 Z= 2.8761 p= .002013	not applicable	0.0077391	0.000717	β= .10737 Z= 3.000 p= .00135
CF/INC	β= -.102213 Z= -.52845 p= .2985	β= .081076 Z= .64501 p= .259461	0	0	β= -.02114 Z= -.13 p= .44828

Table C4. (continued)

SAT/FAD	β= 1.81933 Z= 3.44685 p= .0020836	β= .074190 Z= .529875 p= .298099	-0.420381	-0.001159	β= 1.4712 Z= 3.332 p= .000431
ED/FAD	β= -.269838 Z= -.83726 p= .201226	β= .1351577 Z= 1.37799 p= .0841022	0.0432615	0.0087318	β= -.082387 Z= -.212 p= .40439
EMP/FAD	invalid path	invalid path	invalid path	invalid path	invalid path
INC/FAD	β= .383799 Z= 2.55074 p= .005375	not applicable	0.0048675	0.0011575	β= .389824 Z= 2.503 p= .006157
CF/FAD	β= -.34353 Z= -1.1546 p= .124134	β= .550261 Z= 2.67684 p= .003716	-2.78 E-17	0	β= .206727 Z= .793 p= .21389
SAT/SYMP	β= -.142484 Z= -.992049 p= .16059	β= -.07006 Z= -1.2945 p= .09774	-0.076737	0.0073513	β= -.28163 Z= -2.595 p= .0047295
ED/SYMP	β= .118978 Z= 1.38926 p= .082377	β= .0107209 Z= .5383 p= .360054	-0.030747	-0.00098	β= .097971 Z= 1.175 p= .119998
EMP/SYMP	β= .007886 Z= .556695 p= . 28887	β= -003756 Z= -.88712 p= .18750	0.003481	0.00027	β= .007418 Z= .549 p= .2915
INC/SYMP	β= -.023652 Z= -.570514 p= .284164	β= -.01421 Z= -1.23706 p= .10803	0.014038	0.000968	β= -.02281 Z= -.578 p= .2816
FAD/SYMP	β= -.037024 Z= -1.414 p= .078599	not applicable	-0.014078	-0.002315	β= -.053417 Z= -2.21 p= .01355
CF/SYMP	β= -.104671 Z= -1.361998 p= .0863993	β= -.038356 Z= .7815 p = .21725	0	0	β= -.143026 Z= -2.285 p= .0111564

Note: Abbreviations are as follows: β = path coefficients, **Z** = Z-scores , **p** = one-tailed probability values, IV/DV = independent/ dependent variable pairs for each path, CF = contact frequency, SAT = satisfaction with support, ED = years of education, EMP = employment score, INC = family income, FAD = financial adequacy score, SYMP = symptomatology score, and not applicable = it was physically impossible for an indirect effect to exist.

Appendix D: Structural Equations for Cross-Sectional Models

Table D1.

Structural Equations for the Multiple Regressions for the Full Time-1 Cross-Sectional Model (Hypothesized relationships are indicated with valence signs for each path).

Effects		Endogenous Variables						EXV
Equation number	DV	SAT	ED	EMP	INC	FAD	SYMP	IV--CF
EQ#1	SAT	------	------	------	------	------	------	path #1(+)
EQ#2	ED	path #2(+)	------	------	------	------	------	path #3(+)
EQ#3	EMP	path #4(+)	path #5(+)	------	------	------	------	path #6(+)
EQ#4	INC	path #7(+)	path #8(+)	path #9(+)	------	------	------	path #10(+)
EQ#5	FAD	path #11(+)	path #12(+)	path #13(+)	path #14(+)	------	------	path #15(+)
EQ#6	SYMP	path #16(-)	path #17(-)	path #18(-)	invalid #19(-)	path #20(-)	------	path #21(-)

Note: Abbreviations: independent (IV)/dependent (DV) variable pairs for each path, and structural equation number (EQ); CF = contact frequency, SAT = satisfaction with support, ED = years of education, EMP = employment score, INC = family income, FAD = financial adequacy score, and SYMP = symptomatology score.

Table D2.

Structural Equations for the Multiple Regressions for the Full Time-2 Cross-sectional Model (Hypothesized relationships are indicated with valence signs for each path).

Effects		Endogenous Variables						EXV
Equation number	DV	SAT	ED	EMP	INC	FAD	SYMP	IV--CF
EQ#1	SAT	------	------	------	------	------	------	path #1(+)
EQ#2	ED	path #2(+)	------	------	------	------	------	path #3(+)
EQ#3	EMP	path #4(+)	path #5(+)	------	------	------	------	path #6(+)
EQ#4	INC	path #7(+)	path #8(+)	path #9(+)	------	------	------	path #10(+)
EQ#5	FAD	path #11(+)	path #12(+)	path #13(+)	path #14(+)	------	------	path #15(+)
EQ#6	SYMP	path #16(-)	path #17(-)	path #18(-)	invalid (-)19	path #20(-)	------	path #21(-)

Note: Abbreviations: EXV exogenous variable, independent (IV)/dependent (DV) variable pairs for each path, and structural equation number (EQ); CF = contact frequency, SAT = satisfaction with support, ED = years of education, EMP = employment score, INC = family income, FAD = financial adequacy score, and SYMP = symptomatology score.

Table D3.

Structural Equations for the Multiple Regressions for the Full Time-3 Cross-sectional Model (Hypothesized relationships are indicated with valence signs for each path).

Effects		Endogenous Variables						EXV
Equation number	DV	SAT	ED	EMP	INC	FAD	SYMP	IV--CF
EQ#1	SAT	------	------	------	------	------	------	path #1(+)
EQ#2	ED	path #2(+)	------	------	------	------	------	path #3(+)
EQ#3	EMP	path #4(+)	path #5(+)	------	------	------	------	path #6(+)
EQ#4	INC	path #7(+)	path #8(+)	path #9(+)	------	------	------	path #10(+)
EQ#5	FAD	path #11(+)	path #12(+)	path #13(+)	path #14(+)	------	------	path #15(+)
EQ#6	SYMP	path #16(-)	path #17(-)	path #18(-)	invalid (-)19	path #20(-)	------	path #21(-)

Note: Abbreviations: EXV exogenous variable, independent (IV)/dependent (DV) variable pairs for each path, and structural equation number (EQ); CF = contact frequency, SAT = satisfaction with support, ED = years of education, EMP = employment score, INC = family income, FAD = financial adequacy score, and SYMP=symptomatology score.

Table D4.

Structural Equations for the Multiple Regressions for the Full Time-4 Cross-Sectional Model (Hypothesized relationships are indicated with valence signs for each path).

Effects		Endogenous Variables						EXV
Equation number	DV	SAT	ED	EMP	INC	FAD	SYMP	IV--CF
EQ#1	SAT	------	------	------	------	------	------	path #1(+)
EQ#2	ED	path #2(+)	------	------	------	------	------	path #3(+)
EQ#3	EMP	path #4(+)	path #5(+)	------	------	------	------	path #6(+)
EQ#4	INC	path #7(+)	path #8(+)	path #9(+)	------	------	------	path #10(+)
EQ#5	FAD	path #11(+)	path #12(+)	path #13(+)	path #14(+)	------	------	path #15(+)
EQ#6	SYMP	path #16(-)	path #17(-)	path #18(-)	invalid (-)19	path #20(-)	------	path #21(-)

Note: Abbreviations: EXV exogenous variable, independent (IV)/dependent (DV) variable pairs for each path, and structural equation number (EQ); CF = contact frequency, SAT = satisfaction with support, ED = years of education, EMP = employment score, INC = family income, FAD = financial adequacy score, and SYMP=symptomatology score.

Appendix E: Structural Equations for Cross-Sectional Reduced Models

Table E1.

Structural Equations for the Multiple Regressions for the Reduced Time-1 Cross-Sectional Model (Hypothesized relationships are indicated with valence signs for each path).

Effects		Endogenous Variables						EXV
Equation number	DV	SAT	ED	EMP	INC	FAD	SYMP	IV--CF
EQ#1	SAT	------	------	------	------	------	------	path #1(+)
EQ#2	ED	invalid (+)	------	------	------	------	------	path #2(+)
EQ#3	EMP	path #3(+)	path #4(+)	------	------	------	------	path #5(+)
EQ#4	INC	path #6(+)	path #7(+)	path #8(+)	------	------	------	path #9(+)
EQ#5	FAD	path #10(+)	path #11(+)	path #12(+)	path #13(+)	------	------	path #14(+)
EQ#6	SYMP	path #15(-)	path #16(-)	path #17(-)	invalid (-)	path #18(-)	------	path #19(-)

Note: Abbreviations: EXV = exogenous variable, independent (IV)/dependent (DV) variable pairs for each path, and structural equation number (EQ); CF = contact frequency, SAT = satisfaction with support, ED = years of education, EMP = employment score, INC = family income, FAD = financial adequacy score, and SYMP = symptomatology score.

Table E2.

Structural Equations for the Multiple Regressions for the Reduced Time-2 Cross-Sectional Model (Hypothesized relationships are indicated with valence signs for each path).

Effects		Endogenous Variables						EXV
Equation number	DV	SAT	ED	EMP	INC	FAD	SYMP	IV--CF
EQ#1	SAT	------	------	------	------	------	------	path #1(+)
EQ#2	ED	invalid (+)	------	------	------	------	------	path #2(+)
EQ#3	EMP	path #3(+)	path #4(+)	------	------	------	------	path #5(+)
EQ#4	INC	path #6(+)	path #7(+)	path #8(+)	------	------	------	path #9(+)
EQ#5	FAD	path #10(+)	path #11(+)	path #12(+)	path #13(+)	------	------	path #14(+)
EQ#6	SYMP	path #15(-)	path #16(-)	path #17(-)	path #18 (-)	path #19(-)	------	path #20(-)

Note: Abbreviations: EXV = exogenous variable, independent (IV)/dependent (DV) variable pairs for each path, and structural equation number (EQ); CF = contact frequency, SAT = satisfaction with support, ED = years of education, EMP = employment score, INC = family income, FAD = financial adequacy score, and SYMP = symptomatology score.

Table E3.

Structural Equations for the Multiple Regressions for the Reduced Time-3 Cross-sectional Model (Hypothesized relationships are indicated with valence signs for each path).

Effects		Endogenous Variables						EXV
Equation number	DV	SAT	ED	EMP	INC	FAD	SYMP	IV--CF
EQ#1	SAT	------	------	------	------	------	------	path #1(+)
EQ#2	ED	invalid (+)	------	------	------	------	------	path #2(+)
EQ#3	EMP	path #3(+)	path #4(+)	------	------	------	------	path #5(+)
EQ#4	INC	path #6(+)	path #7(+)	path #8(+)	------	------	------	path #9(+)
EQ#5	FAD	path #10(+)	path #11(+)	path #12(+)	path #13(+)	------	------	path #14(+)
EQ#6	SYMP	path #15(-)	path #16(-)	path #17(-)	path # 18(-)	invalid (-)	------	path #19(-)

Note: Abbreviations: EXV = exogenous variable, independent (IV)/dependent (DV) variable pairs for each path, and structural equation number (EQ); CF = contact frequency, SAT = satisfaction with support, ED = years of education, EMP = employment score, INC = family income, FAD = financial adequacy score, and SYMP = symptomatology score.

Table E4.

Structural Equations for the Multiple Regressions for the Reduced Time-4 Cross-sectional Model (Hypothesized relationships are indicated with valence signs for each path).

Effects		Endogenous Variables						EXV
Equation number	DV	SAT	ED	EMP	INC	FAD	SYMP	IV--CF
EQ#1	SAT	------	------	------	------	------	------	path #1(+)
EQ#2	ED	invalid (+)	------	------	------	------	------	path #2(+)
EQ#3	EMP	path #3(+)	path #4(+)	------	------	------	------	path #5(+)
EQ#4	INC	path #6(+)	path #7(+)	path #8(+)	------	------	------	path #9(+)
EQ#5	FAD	path #10(+)	path #11(+)	path #12(+)	invalid (+)	------	------	path #13(+)
EQ#6	SYMP	path #14(-)	path #15(-)	path #16(-)	path #17 (-)	path#18(-)	------	path #19(-)

Note: Abbreviations: EXV = exogenous variable, independent (IV)/dependent (DV) variable pairs for each path, and structural equation number (EQ); CF = contact frequency, SAT = satisfaction with support, ED = years of education, EMP = employment score, INC = family income, FAD = financial adequacy score, and SYMP = symptomatology score.

Appendix F: Variance Explained for Cross-Sectional Models

Table F1.

Multiple Regression Variance Explained for the Structural Equation System Based Upon Time-2 Reduced Path Model, N = 146.

Equation Number	R^2	MS_e	F-ratio value	p-value
EQ #1	0.2311	237.4	43.894	$p < .0001$
EQ #2	0	0.046	0.005	$p = .9955$
EQ #3	0.061	1386.9	3.131	$p = .0276$
EQ #4	0.085	139.7	3.322	$p = .0123$
EQ #5	0.1688	646.3	5.738	$p < .0001$
EQ #6	0.2378	45.7	7.332	$p < .0001$

Note: Abbreviations: EQ = equation number from the structural equation system, R^2 = proportion of the variance accounted for by the particular equation, and MS_e = mean square error.

Table F2.

Multiple Regression Variance Explained for the Structural Equation System Based Upon Time-3 Reduced Path Model, N = 126.

Equation Number	R^2	MS_e	F-ratio value	p-value
EQ #1	0.1664	132.7	24.956	$p < .0001$
EQ #2	0	5.8	0.559	$p = .4562$
EQ #3	0.03	637.4	1.287	$p = .2820$
EQ #4	0.079	114.5	2.617	$p = .0384$
EQ #5	0.2575	747.2	8.392	$p < .0001$
EQ #6	0.3107	59.8	10.906	$p < .0001$

Note: Abbreviations: EQ = equation number from the structural equation system, R^2 = proportion of the variance accounted for bu the particular equation, and MS_e = mean square error.

Table F3.

Multiple Regression Variance Explained for the Structural Equation System Based Upon Time-4 Reduced Path Model, N = 96.

Equation Number	R^2	MS_e	F-ratio value	p-value
EQ #1	0.3349	209.9	49.85	$p < .0001$
EQ #2	0.022	26.04	2.25	$p = .1638$
EQ #3	0.086	1291.7	3.04	$p = .0327$
EQ #4	0.1073	141.4	2.884	$p = .0265$
EQ #5	0.1746	587.9	5.078	$p = .0009$
EQ #6	0.1242	19.9	2.221	$p = .0476$

Note: Abbreviations: EQ = equation number from the structural equation system, R^2 = proportion of the variance accounted for bu the particular equation, and MS_e = mean square error.

Appendix G: Raw Correlations for Lagged Models

Table G1.

Raw correlations SF TIME-1 SYMP TIME-2 time-lagged model. N=146.

	SUPPORT TIME-1	FAD TIME-1	ED TIME-1	EMP TIME-1	INC TIME-1	SYMP TIME-2
SUPPORT TIME-1	- -	+ .28 p < .001	+ .05 p = .451	+ .05 p = .451	+ .11 p = .043	- .31 p < .001
FAD TIME-1		- -	- .02 p = .817	+ .11 p = .084	+ .43 p < .001	- .26 p < .001
ED TIME-1			- -	+ .24 p < .001	+ .24 p < .001	- .01 p = .889
EMP TIME-1				- -	+ .44 p < .001	- .09 p = .259
INC TIME-1					- -	- .07 p = .439
SYMP TIME-2						- -

Note. The abbreviations are as follows: p = one-tailed probabilities, SUPPORT= combined social support variable, ED =years of formal schooling, EMP = employment score, INC = Family income score, FAD =Financial Adequacy Score, SYMP = Symptomatology measure, and SF = social support and social functioning. All probabilities are two tailed for the appropriate values related to directional predictions this value needs divided by two.

Table G2.

Raw correlations SF TIME-1 SYMP TIME-3 time-lagged model. Social Causation Model N = 123.

	SUPPORT TIME-1	FAD TIME-1	ED TIME-1	EMP TIME-1	INC TIME-1	SYMP TIME-3
SUPPORT TIME-1	- -	+ .28 p < .001	+ .04 p = .504	+ .05 p = .451	+ .11 p = .086	- .38 p < .001
FAD TIME-1		- -	- .02 p = .817	+ .11 p = .084	+ .43 p < .001	- .26 p < .001
ED TIME-1			- -	+ .24 p < .001	+ .24 p < .001	+ .03 p = .714
EMP TIME-1				- -	+ .44 p < .001	+ .002 p = .982
INC TIME-1					- -	- .02 p = .805
SYMP TIME-3						- -

Note. The abbreviations are as follows: p = one-tailed probabilities, SUPPORT= combined social support variable, ED =years of formal schooling, EMP = employment score, INC = Family income score, FAD = Financial Adequacy Score, and SYMP = Symptomatology measure, and SF = social support and social functioning. All probabilities are two tailed for the appropriate values related to directional predictions this value needs divided by two.

Table G3.

Raw correlations SF TIME-1 SYMP TIME-4 time-lagged model. Social Causation Model. N = 96.

	SUPPORT TIME-1	FAD TIME-1	ED TIME-1	EMP TIME-1	INC TIME-1	SYMP TIME-4
SUPPORT TIME-1	- -	+ .28 p < .001	+ .04 p = .504	+ .05 p = .451	+ .11 p = .086	- .26 p = .011
FAD TIME-1		- -	- .02 p = .817	+ .11 p = .084	+ .43 p < .001	- .29 p = .005
ED TIME-1			- -	+ .24 p < .001	+ .24 p < .001	+ .15 p = .157
EMP TIME-1				- -	+ .44 p < .001	+ .05 p = .597
INC TIME-1					- -	- .06 p = .552
SYMP TIME-4						- -

Note. The abbreviations are as follows: p = one-tailed probabilities, SUPPORT= combined social support variable, ED =years of formal schooling, EMP = employment score, INC = Family income score, FAD = Financial Adequacy Score, SYMP = Symptomatology measure, and SF = social support and social functioning. All probabilities are two tailed for the appropriate values related to directional predictions this value needs divided by two.

Table G4.

Raw correlations SYMP TIME-1 SF TIME-2 time-lagged model. N = 146.

	SYMP TIME-1	FAD TIME-2	ED TIME-2	EMP TIME-2	INC TIME-2	SUPPORT TIME-2
SYMP TIME-1	- -	- .18 p = .032	- .03 p = .703	- .18 p = .027	- .09 p = .284	- .31 p < .001
FAD TIME-2		- -	+ .01 p = .861	+ .15 p = .070	+ .36 p < .001	+ .18 p = .029
ED TIME-2			- -	+ .23 p = .004	+ .20 p = .014	+ .01 p = .965
EMP TIME-2				- -	+ .22 p = .006	+ .08 p = .333
INC TIME-2					- -	+ .06 p = .495
SUPPORT TIME-2						- -

Note. The abbreviations are as follows: p = one-tailed probabilities, SUPPORT= combined social support variable, ED =years of formal schooling, EMP = employment score, INC = Family income score, FAD = Financial Adequacy Score, SYMP = Symptomatology measure and SF = social support and social functioning. All probabilities are two tailed for the appropriate values related to directional predictions this value needs divided by two.

Table G5.

Raw correlations SYMP TIME-1 SF TIME-3 time-lagged model. Social Selection Model. N = 123.

	SYMP TIME-1	FAD TIME-3	ED TIME-3	EMP TIME-3	INC TIME-3	SUPPORT TIME-3
SYMP TIME-1	- -	- .26 p = .004	- .01 p = .952	- .17 p = .203	- .05 p = .558	- .30 p < .001
FAD TIME-2		- -	- .13 p = .135	+ .12 p = .195	+ .35 p < .001	+ .24 p = .006
ED TIME-2			- -	+ .17 p = .057	+ .08 p = .370	- .05 p = .564
EMP TIME-2				- -	+ .27 p = .002	- .02 p < .786
INC TIME-2					- -	+ .02 p = .835
SUPPORT TIME-2						- -

Note. The abbreviations are as follows: p = one-tailed probabilities, SUPPORT= combined social support variable, ED =years of formal schooling, EMP = employment score, INC = Family income score, FAD = Financial Adequacy Score, SYMP = Symptomatology measure, and SF = social support and social functioning. All probabilities are two tailed for the appropriate values related to directional predictions this value needs divided by two.

Table G6.

Raw correlations SYMP TIME-1 SF TIME-4 time-lagged model. Social Selection Model, N = 96.

	SYMP TIME-1	FAD TIME-4	ED TIME-4	EMP TIME-4	INC TIME-4	SUPPORT TIME-4
SYMP TIME-1	- -	- .26 p = .010	- .02 p = .852	- .02 p = .817	- .09 p = .380	- .33 p < .001
FAD TIME-4		- -	- .02 p = .809	+ .07 p = .470	+ .246 p = .013	+ .23 p = .020
ED TIME-4			- -	+ .157 p = .116	+ .16 p = .116	+ .13 p = .184
EMP TIME-4				- -	+ .31 p < .001	+ .06 p = .546
INC TIME-4					- -	+ .02 p = .873
SUPPORT TIME-4						- -

Note. The abbreviations are as follows: p = one-tailed probabilities, SUPPORT= combined social support variable, ED =years of formal schooling, EMP = employment score, INC = Family income score, FAD = Financial Adequacy Score, SYMP = Symptomatology measure, and SF = social support and social functioning. All probabilities are two tailed for the appropriate values related to directional predictions this value needs divided by two.

Appendix H: Structural Equations for Lagged Models

Table H1.

Structural Equations for the Multiple Regressions for the SF1SY2 Time-2 Social Causation Reduced Model (Hypothesized relationships are indicated with valence signs for each path).

Effects	Endogenous Variables						EXV
Equation number	DV T-1	FAD T-1	ED T-1	EMP T-1	INC T-1	SYMP T-2	IV–SS T-1
EQ#1	FAD T-1	------	------	------	------	------	path #1(+)
EQ#2	ED T-1	path #2(+)	------	------	------	------	path #3(+)
EQ#3	EMP T-1	path #4(+)	path #5(+)	------	------	------	path #6(+)
EQ#4	INC T-1	path #7(+)	path #8(+)	path #9(+)	------	------	path #10(+)
EQ#5	SYMP T-2	path #11(-)	invalid (-)	path #12(-)	invalid (-)	------	path #13(-)

Note: Abbreviations: EXV = exogenous variable, independent (IV)/dependent (DV) variable pairs for each path, and structural equation number (EQ); SS = Social Support, FAD = financial adequacy score, ED = years of education, EMP = employment score, INC = household income, SYMP = symptomatology score, T-2 = measurement at Time -2, SF = social functioning variables , and SY = symptomatology variables.

Table H2.

Structural Equations for the Multiple Regressions for the SF1SY3 Social Causation Reduced Model (Hypothesized relationships are indicated with valence signs for each path).

Effects		Endogenous Variables					EXV
Equation number	DV	FAD T-1	ED T-1	EMP T-1	INC T-1	SYMP T-3	IV–SS T-1
EQ#1	FAD T-1	------	------	------	------	------	path #1(+)
EQ#2	ED T-1	path #2(+)	------	------	------	------	path #3(+)
EQ#3	EMP T-1	path #4(+)	path #5(+)	------	------	------	path #6(+)
EQ#4	INC T-1	path #7(+)	path #8(+)	path #9(+)	------	------	path #10(+)
EQ#5	SYMP T-3	path #11(-)	invalid (-)	invalid (-)	path #12(-)	------	path #13(-)

Note: Abbreviations: EXV = exogenous variable, independent (IV)/dependent (DV) variable pairs for each path, and structural equation number (EQ); SS = Social Support, FAD = financial adequacy score, ED = years of education, EMP = employment score, INC = household income, SYMP = symptomatology score, T-3 = measurement at Time-3, SF = social functioning variables , and SY = symptomatology variables.

Table H3.

Structural Equations for the Multiple Regressions for the SF1SY4 Social Causation Reduced Model (Hypothesized relationships are indicated with valence signs for each path).

Effects		Endogenous Variables					EXV
Equation number	DV	FAD T-1	ED T-1	EMP T-1	INC T-1	SYMP T-4	IV–SS T-1
EQ#1	FAD T-1	------	------	------	------	------	path #1(+)
EQ#2	ED T-1	path #2(+)	------	------	------	------	path #3(+)
EQ#3	EMP T-1	path #4(+)	path #5(+)	------	------	------	path #6(+)
EQ#4	INC T-1	path #7(+)	path #8(+)	path #9(+)	------	------	path #10(+)
EQ#5	SYMP T-4	path #11(-)	path #12(-)	invalid (-)	invalid (-)	------	path #13(-)

Note: Abbreviations: EXV = exogenous variable, independent (IV)/dependent (DV) variable pairs for each path, and structural equation number (EQ); SS = Social Support, FAD = financial adequacy score, ED = years of education, EMP = employment score, INC = household income, SYMP = symptomatology score, T-4 = measure at time-4 = measurement at times time 4, SF = social functioning variables , and SYMP = symptomatology variables.

Table H4.

Structural Equations for the Multiple Regressions for the SY1SF2 Social Selection Reduced Model (Hypothesized relationships are indicated with valence signs for each path).

Effects		Endogenous Variables					EXV
Equation number	DV T-2	FAD T-2	ED T-2	EMP T-2	INC T-2	SS T-2	IV–SYMP T-1
EQ#1	FAD T-2	------	------	------	------	------	path #1(+)
EQ#2	ED T-2	path #2(+)	------	------	------	------	path #3(+)
EQ#3	EMP T-2	path #4(+)	path #5(+)	------	------	------	path #6(+)
EQ#4	INC T-2	path #7(+)	path #8(+)	path #9(+)	------	------	path #10(+)
EQ#5	SS T-2	path #11(-)	invalid (-)	invalid (-)	invalid(-)	------	path #11(-)

Note: Abbreviations: EXV = exogenous variable, independent (IV)/dependent (DV) variable pairs for each path, and structural equation number (EQ); SS = Social Support, FAD = financial adequacy score, ED = years of education, EMP = employment score, INC = household income, SYMP = symptomatology score, T-2 = measurement at Times-2, SF = social functioning variables , and SY = symptomatology variables.

Table H5.

Structural Equations for the Multiple Regressions for the SY1SF3 Social Selection Reduced Model (Hypothesized relationships are indicated with valence signs for each path).

Effects		Endogenous Variables					EXV
Equation number	DV T-3	FAD T-3	ED T-3	EMP T-3	INC T-3	SS T-3	IV–SYMP T-1
EQ#1	FAD T-3	------	------	------	------	------	path #1(+)
EQ#2	ED T-3	path #2(+)	------	------	------	------	path #3(+)
EQ#3	EMP T-3	path #4(+)	path #5(+)	------	------	------	path #6(+)
EQ#4	INC T-3	path #7(+)	path #8(+)	path #9(+)	------	------	path #10(+)
EQ#5	SS T-3	path #11(-)	path #12(-)	cut (-)	path #13(-)	------	path #14(-)

Note: Abbreviations: EXV = exogenous variable, independent (IV)/dependent (DV) variable pairs for each path, and structural equation number (EQ); SS = Social Support, FAD = financial adequacy score, ED = years of education, EMP = employment score, INC = household income, SYMP = symptomatology score, T-3 = measurement at Times-3, SF = social functioning variables , and SY = symptomatology variables.

Table H6.

Structural Equations for the Multiple Regressions for the SY1SF4 Social Selection Reduced Model (Hypothesized relationships are indicated with valence signs for each path).

Effects		Endogenous Variables					EXV
Equation number	DV T-4	FAD T-4	ED T-4	EMP T-4	INC T-4	SS T-4	IV–SYMP T-1
EQ#1	FAD T-4	------	------	------	------	------	path #1(+)
EQ#2	ED T-4	path #2(+)	------	------	------	------	path #3(+)
EQ#3	EMP T-4	path #4(+)	path #5(+)	------	------	------	path #6(+)
EQ#4	INC T-4	path #7(+)	path #8(+)	path #9(+)	------	------	path #10(+)
EQ#5	SS T-4	path #11(-)	path #12(-)	invalid (-)	path #13(-)	------	path #14(-)

Note: Abbreviations: EXV = exogenous variable, independent (IV)/dependent (DV) variable pairs for each path, and structural equation number (EQ); SS = Social Support, FAD = financial adequacy score, ED = years of education, EMP = employment score, INC = household income, SYMP = symptomatology score, T-4 = measurement at Times-4, SF = social functioning variables , and SY = symptomatology variables.

Appendix I: Social Causation Reduced Lagged Models

Table I1.

Effects In Final Reduced Path Model(#) Social Causation SF1SY2. N = 146. Hypothesised Effect Valences in Brackets.

Variables IV/DV	Direct Effect	Indirect Effect	Unexplained Effect	Spurious Effect	Total Effect
SUPPORT/FAD 1(+)	β = .369 Z = 3.4822402 p = .0002486	not applicable	0	0	β= .369 Z= 3.48 p= .0002507
FAD/ED 2(+)	β= .00605 Z= .243099 p= .4039644	not applicable	0.000326	0	β= .00638 Z= .27 p=.3935801
SUPPORT/ED 3(+)	β= .00155 Z= .0470322 p= .4812438	β= .0022328 Z= .2425088 p= .404193	0	0	β= .00378 Z= .12 p= .4522416
FAD/EMP 4(+)	β= .53251 Z= 1.5951062 p= .0553442	β= .0191187 Z= .2421993 p= .4043129	-0.09611	0	β= .45553 Z= 1.39 p= .0822644
ED/EMP 5(+)	β= 3.16012 Z= 2.8177045 p= .0024184	not applicable	0.0293468	0	β= 3.189 Z= 2.84 p= .0022557
SUPPORT/EMP 6(+)	β= -.46158 Z= -1.044313 p= .1481703	β= .2084768 Z= 1.232803 p= .1088247	0	0	β= -.25309 Z= -.58 p= .2809573
FAD/INC 7(+)	β= .26033 Z= 6.042544 p= 7.585E-10	β= .0347873 Z= 1.4399595 p= .0749394	-0.011127	0	β= .28399 Z= 6.11 p= 4.92E-10
ED/INC 8(+)	β= .30936 Z= 2.0986136 p= .0179255	β= .1885644 Z= 2.5132772 p= .0059808	0.0213624	0	β= .5193 Z= 2.93 p=.0016948
EMP/INC 9(+)	β= .05967 Z= 5.5589198 p= 1.3572E-8	not applicable	0.0120924	0	β= .07258 Z= 6.22 p= 2.486E-10
SUPPORT/INC 10(+)	β= -.0261 Z= -.459889 p= .3227979	β= .0821423 Z= 1.7752539 p= .037928	0	0.0008	β= .05604 Z= .81 p= .2089701

Table I1.	(continued)				
FAD/SYMP 11(-)	β= -.05869 Z= -2.772295 p= .0027831	β= -.004827 Z= -1.169928 p= .1210151	-0.017557	0.013844	β= -.06723 Z= -3.24 p= .005976
ED/SYMP 12(-)	INVALID PATH	**********	********	*********	**********
EMP/SYMP 13(-)	β= -.00875 Z= -1.704552 p= .044139	0	-0.00084	0.00346	β= -.00613 Z= -1.14 p= .1271432
INC/SYMP 14(-)	INVALID PATH	**********	**********	*********	**********
SUPPORT/SYMP 15(-)	β= -.08742 Z= -3.13546 p= .0008579	β= -.019445 Z= -1.736988 p= .0411947	0	0.00272	β= -.10414 Z= -3.84 p= .00000615

Note: Abbreviations are as follows: β = path coefficients, **Z** = Z-scores , **p** = one-tailed probability values, IV/DV = independent/ dependent variable pairs for each path, SUPPORT = combined social support measure, ED = years of education, EMP = employment score, INC = family income, FAD = financial adequacy score, SYMP = symptomatology score, and not applicable = it was physically impossible for an indirect effect to exist.

Table I2.

Effects In Final Reduced Path Model(#) Social Causation SF1SY3. N = 123. Hypothesised Effect Valences in Brackets.

Variables IV/DV	Direct Effect	Indirect Effect	Unexplained Effect	Spurious Effect	Total Effect
SUPPORT/FAD 1(+)	β= .34022 Z= 3.1097949 p=.0009361	not applicable	0	0	β= .34022 Z= 3.11 p= .0009354
FAD/ED 2(+)	β= -.02012 Z= -.665889 p= .2527411	not applicable	-0.003831	0	β= -.02395 Z= -.83 p= .2032694
SUPPORT/ED 3(+)	β= -.01761 Z= -.582818 p= .2800079	β=–.006845 Z=-.651129 p=.2574817	0	0	β= -.02495 Z= -.67 p= .2514289
FAD/EMP 4(+)	β= .68113 Z= 1.7256142 p= .0422084	β= -.056454 Z= -.645371 p=.2593434	-0.145312	-4116000	β= .47936 Z= 1.24 p= .1074877
ED/EMP 5(+)	β= 2.8058 Z= 2.6200147 p=.0043963	not applicable	-0.064673	0	β= 2.74085 Z= 2.3 p= .0107241
SUPPORT/EMP 6(+)	β= –.61859 Z= -1.254318 p= .1048632	β= .16311664 Z= .9536698 p= .1701254	0	0	β= -.45547 Z= -.97 p= .1660232
FAD/INC 7(+)	β= .27987 Z= 5.4874703 p= 2.0387E-8	β= .0245632 Z= .9530697 p=.1702774	-0.020781	0	β= .28365 Z= 5.3 p= 5.79E-8
ED/INC 8(+)	β= .33185 Z= 2.1351744 p= .0163734	β= .1403206 Z= 2.2338036 p= .012748	-0.059942	0	β= .41193 Z= 2.26 p=.0119106
EMP/INC 9(+)	β= .05001 Z= 4.2745916 p=9.5744E-6	not applicable	0.0125018	0.0007	β= .06324 Z= 5.02 p= 2.58E-7
SUPPORT/INC 10(+)	β= -.05628 Z= -.888314 p= .187186	β= .0643237 Z= 1.3703763 p= .0852847	0	0	β= .00805 Z= .11 p= .4562047
FAD/SYMP 11(-)	β= -.05447 Z= -2.137374 p= .0162838	β= .0093411 Z= .8031463 p= .2109451	-0.021356	0.0002	β= -.06626 Z= -2.91 p= .0018071

Table I2.	(continued)				
ED/SYMP 12(-)	INVALID PATH	**********	**********	********	**********
EMP/SYMP 13(-)	β=-.00205 Z= -.363173 p= .3582377	β= .0017448 Z= .8373068 p= .2012101	0.000534	0	β= .0001209 Z= .02 p= .4920217
INC/SYMP 14(-)	β= .03489 Z= .8538476 p= .1965947	not applicable	-0.031697	-0.01208	β= -.00889 Z= -.25 p=.4012937
SUPPORT/SYMP 15(-)	β=-.09621 Z= -3.345813 p= .0004102	β= -.017317 Z= -1.677905 p= .0466829	0	0.0002	β= -.1133 Z= -4.11 p= .0000198

Note: Abbreviations are as follows: β = path coefficients, **Z** = Z-scores , **p** = one-tailed probability values, IV/DV = independent/ dependent variable pairs for each path, SUPPORT = combined social support measure, ED = years of education, EMP = employment score, INC = family income, FAD = financial adequacy score, SYMP = symptomatology score and not applicable = it was physically impossible for an indirect effect to exist.

Table I3.

Effects in Final Reduced Path Model(#) Social Causation SF1SY4. N = 96. Hypothesised Effect Valences in Brackets.

Variables IV/DV	Direct Effect	Indirect Effect	Unexplained Effect	Spurious Effect	Total Effect
SUPPORT/FAD 1(+)	β= .36763 Z= 2.8279015 p= .0023427	not applicable	0	0	β= .36763 Z= 2.83 p= .0023274
FAD/ED 2(+)	β= - .03742 Z= -1.169017 p= .1211983	not applicable	0.0033222	0	β= -.0341 Z= -1.11 p=.1334995
SUPPORT/ED 3(+)	β= .01573 Z= .3724665 p= .3547728	β= -.013757 Z= -1.080347 p=.1399939	0	0	β= .00197 Z= .05 p= .4800612
FAD/EMP 4(+)	β=.52433 Z= 1.3455619 p= .0892219	β=-.092069 Z= -1.005832 p=.1572483	-0.250223	0	β= .18205 Z= .47 p= .3191775
ED/EMP 5(+)	β= 2.46043 Z= 1.9737424 p= .0242055	not applicable	-0.210731	-0.003	β=2.24669 Z= 1.78 p=.037538
SUPPORT/EMP 6(+)	β= −1.22347 Z= -2.39523 p= .008305	β=.1976146 Z= 1.0760098 p=.1409614	0	-0.23275	β= −1.2586 Z= -2.06 p= .0196993
FAD/INC 7(+)	β= .25466 Z= 4.5878365 p= 2.2393E-6	β=.0070251 Z= .267688 p= .3944694	-0.010355	0	β= .25133 Z= 4.44 p= 4.4979E-6
ED/INC 8(+)	β= .36709 Z= 2.0449201 p= .0204314	β= .1181745 Z= 1.6915921 p=.0453619	-0.105076	-0.001	β= .3787 Z= 1.86 p=.0314428
EMP/INC 9(+)	β= .04803 Z= 3.2831374 p= .0005133	not applicable	0.008175	0	β= .05647 Z= 3.65 p= .0001311
SUPPORT/INC 10(+)	β= .0021 Z= .0282819 p= .4887186	β=.0450732 Z= .8473279 p= .1984062	0	-3193000	β= .04717 Z= .58 p=.2809573
FAD/SYMP 11(-)	β= -.06046 Z= -1.996685 p= .0229297	β= -.003959 Z= -.830005 p= .2032679	-0.012605	0.0025	β= -.07433 Z= -2.87 p= .0020524

Table I3.	(continued)				
ED/SYMP 12(-)	β= .10093 Z= 1.117385 p=.1319148	not applicable	0.02179	0.0041	β= .12684 Z= 1.43 p=.0763585
EMP/SYMP 13(-)	INVALID PATH	**********	**********	********	**********
INC/SYMP 14(-)	INVALID PATH	**********	**********	********	**********
SUPPORT/SYMP 15(-)	β= -.06127 Z= -1.676746 p= .0467961	β= -.022028 Z= -1.624963 p= .0520852	0	0	β=-.08284 Z= -2.4 p= .0081975

Note: Abbreviations are as follows: **β** = path coefficients, **Z** = Z-scores , **p** = one-tailed probability values, IV/DV = independent/ dependent variable pairs for each path, SUPPORT = combined social support measure, ED = years of education, EMP = employment score, INC = family income, FAD = financial adequacy score, SYMP = symptomatology score, and not applicable = it was physically impossible for an indirect effect to exist.

Appendix J: Social Selection Reduced Lagged Models

Table J1.

Effects In Final Reduced Path Model(#) Social Selection SY1SF2. N = 146. Hypothesised Effect Valences in Brackets.

Variables IV/DV	Direct Effect	Indirect Effect	Unexplained Effect	Spurious Effect	Total Effect
SYMP/FAD 1(-)	β= -.80413 Z= -2.182802 p= .0145252	not applicable	0	0	β= -.80413 Z=-2.18 p= .00146287
FAD/ED 2(+)	INVALID PATH	**********	**********	**********	**********
SYMP/ED 3(-)	β= -.03644 Z= -.309892 p= .3783215	0	0	0	β= -.03644 Z= -.31 p= .3782805
FAD/EMP 4(+)	β=.2137 Z=1.3763866 p= .0843515	0	0.0548914	-0.031781	β= .23681 Z= 1.69 p= .045514
ED/EMP 5(+)	β=.98843 Z= 2.0320604 p= .0210738	not applicable	0.0276899	-0.01047	β= 1.00565 Z= 2.04 p= .0206752
SYMP/EMP 6(-)	β= -1.34217 Z= -1.923362 p= .0272173	β= -.207861 Z=-1.100164 p= .1356302	-2.2200e-16	0	β= -1.55004 Z= -2.23 p= .0128737
FAD/INC 7(+)	β=.19766 Z= 4.2507669 p= .0000107	β= .0100332 Z= 1.1106664 p= .133356	0.002785	-0.00145	β= .20903 Z= 4.52 p= 3.092E-6
ED/INC 8(+)	β= .25298 Z= 1.7232325 p= .0424232	β=.046468 Z=1.3801845 p= .0837649	0.0041229	-0.010181	β= .29334 Z= 1.89 p= .029379
EMP/INC 9(+)	β= .04695 Z= 1.8804908 p= .0300204	not applicable	0.020971	0.00058	β= .0685 Z= 2.69 p= .0035726
SYMP/INC 10(-)	β= .004 Z= .0190204 p= .4924124	β= -.240937 Z= -2.332669 p= .0098328	-2.7800e-17	0	β= .23693 Z= -1.07 p=.1423097
FAD/SUPPORT 11(+)	β= .10774 Z= 1.7999246 p= .0359363	0	0.0378762	-0.00135	β= .14437 Z= 2.36 p= .0091375

Table J1.	(continued)				
ED/SUPPORT 12(+)	INVALID PATH	**********	**********	**********	**********
EMP/SUPPORT 13(+)	INVALID PATH	**********	**********	**********	**********
INC/SUPPORT 14(+)	INVALID PATH	**********	**********	**********	**********
SYMP/SUPPORT 15(-)	β= -.95349 Z= -3.545123 p= .0001962	β = -.086637 Z= -1.388691 p= .0824634	0	0.016787	β= -1.02334 Z= -3.84 p= .0000615

Note: Abbreviations are as follows: β = path coefficients, **Z** = Z-scores , **p** = one-tailed probability values, IV/DV = independent/ dependent variable pairs for each path, SUPPORT = combined social support measure, ED = years of education, EMP = employment score, INC = family income, FAD = financial adequacy score, SYMP = symptomatology score, and not applicable = it was physically impossible for an indirect effect to exist.

Table J2.

Effects In Final Reduced Path Model(#) Social Selection SY1SF3. N = 123. Hypothesised Effect Valences in Brackets.

Variables IV/DV	Direct Effect	Indirect Effect	Unexplained Effect	Spurious Effect	Total Effect
SYMP/FAD 1(-)	β= -1.05434 Z= -2.80115 p= .0025457	not applicable	0	0.0001	β= -1.05424 Z=-2.8 p= .0025551
FAD/ED 2(+)	β=-.04239 Z=-.473503 p= .3179273	not applicable	0.0030036	0	β= -.03969 Z= -1.45 p=.0735293
SYMP/ED 3(-)	β= -.052 Z= -.429921 p= .3336366	β= .0446935 Z= .4668797 p=.320293	0	0.0003	β= –.00698 Z= -.06 p= .4760778
FAD/EMP 4(+)	β=.24306 Z=1.246761 p= .1062426	β= -.052989 Z= -.460854 p=.324518	0.0483673	0	β= .23806 Z= 1.26 p= .1038347
ED/EMP 5(+)	β=1.25003 Z= 2.0072366 p= .0223622	not applicable	-0.098867	-0.0027	β= 1.14842 Z= 1.85 p= .0321568
SYMP/EMP 6(-)	β= -.77237 Z= -.935328 p= .1748097	β= -.265401 Z=-.928861 p= .1764806	0	0.0004	β= -1.03737 Z= -1.28 p= .1002726
FAD/INC 7(+)	β= .21145 Z= 3.773049 p= .0000806	β= .0053135 Z= .184317 p= .4268824	-0.04812	0	β= .21187 Z= 3.87 p= .0000544
ED/INC 8(+)	β= .1796 Z= .9930869 p= .1603338	β= .0850145 Z=1.5883057 p= .0561086	-0.096242	-0.0025	β= .16589 Z= .87 p= .19210502
EMP/INC 9(+)	β= .06801 Z= 2.5976421 p= .0046933	not applicable	0.0133374	0.0005	β= .08182 Z= 3.07 p= .0010703
SYMP/INC 10(-)	β= .14959 Z= .6319511 p= .2637094	β= -.294831 Z= -2.406057 p= .0080629	0	0	β= -.14516 Z=-.59 p=.2775953
FAD/SUPPORT 11(+)	β= .14876 Z= 2.1019905 p= .0177771	β= -.012064 Z= -.43713 p= .3310084	0.0484095	-0.0069	β= .17821 Z= 2.72 p= .0032641

Table J2.	(continued)				
ED/SUPPORT 12(+)	β= -.07575 Z=-.353751 p=.3617628	β= -.018647 Z=-.600161 p=.2741996	-0.052175	-0.03499	β= -.18156 Z= -.82 p= .2061081
EMP/SUPPORT 13(+)	CUT PATH	**********	**********	*********	**********
INC/SUPPORT 14(+)	β= -.07047 Z= -.659543 p= .2547736	not applicable	0.0768856	-0.01189	β=-.00547 Z= -.05 p= .480061
SYMP/SUPPORT 15(-)	β= -.83617 Z= -2.968891 p= .0014944	β= -.146055 Z= -1.616618 p= .0529804	0	0.30175	β= -.95205 Z= -3.46 p= .0002701

Note: Abbreviations are as follows: β = path coefficients, **Z** = Z-scores , **p** = one-tailed probability values, IV/DV = independent/ dependent variable pairs for each path, SUPPORT = combined social support measure, ED = years of education, EMP = employment score, INC = family income, FAD = financial adequacy score, SYMP = symptomatology score, and not applicable = it was physically impossible for an indirect effect to exist.

Table J3.

Effects In Final Reduced Path Model(#) Social Causation SY1SF4. N = 96. Hypothesised Effect Valences in Brackets.

Variables IV/DV	Direct Effect	Indirect Effect	Unexplained Effect	Spurious Effect	Total Effect
SYMP/FAD 1(-)	β= -1.28324 Z= -2.645879 p= .0040739	not applicable	0	0	β= -1.28324 Z=-2.65 p= .0040246
FAD/ED 2(+)	β= -.00888 Z=-.24077 p= .404864	not applicable	0.0021282	0	β= -.00675 Z= -.22 p=.4129356
SYMP/ED 3(-)	β= -.03979 Z= -.252917 p= .4001661	β= .0113952 Z= .239781 p=.4052481	3.46900e-18	0	β= .0284 Z= -.19 p= .4246546
FAD/EMP 4(+)	β= .11374 Z= -.598431 p= .2747762	β= -.015611 Z= -.239941 p=.4051881	0.004666	0	β= .1028 Z= .54 p= .2945985
ED/EMP 5(+)	β= 1.75797 Z= 2.8814345 p= .0019793	not applicable	-0.008261	0	β= 1.7497 Z= 2.89 p= .0019262
SYMP/EMP 6(-)	β= -.01729 Z= -.018362 p= .4926751	β= -.195873 Z=-.532711 p= .297116	2.77600e-17	0	β= -.21317 Z= -.23 p= .4090459
FAD/INC 7(+)	β= .15061 Z= 2.4081167 p= .0080175	β= .007745 Z= .332807 p= .369639	0.0041263	0	β= .16248 Z= 2.58 p= .00494
ED/INC 8(+)	β= .19824 Z= .9479319 p= .1715821	β= .170294 Z= 2.024255 p= .021471	-0.011244	0	β= .35729 Z= 1.69 p= .045514
EMP/INC 9(+)	β= .09687 Z= 2.8443841 p= .0022249	not applicable	0.0127905	0.00108	β= .11074 Z= 3.33 p= .00043242
SYMP/INC 10(-)	β= -.06081 Z= -.198894 p= .4211718	β=-.219547 Z= -1.43835 p= .075171	5.51000e-17	0	β= -.280335 Z= -.88 p=.1894297
FAD/SUPPORT 11(+)	β= .10447 Z= 1.3963861 p= .0812991	β=-.005457 Z= -1.4383 p= .07517	0.0539614	-0.00678	β= .1462 Z= 1.96 p= .0249979

Table J3.	(continued)				
ED/SUPPORT 12(+)	β= .37667 Z=1.5056953 p=.0660727	β= -.037832 Z= -.5195 p= .301677	0.005407	-0.014875	β= .32937 Z= 1.31 p=.0950979
EMP/SUPPORT 13(+)	β= -.02152 Z=-.52824 p=.2986663	not applicable	0.0213736	-0.00281	β= -.00296 Z= -.07 p=.4720968
INC/SUPPORT 14(+)	INVALID PATH	**********	**********	**********	**********
SYMP/SUPPORT 15(-)	β= -.9958 Z= -2.722758 p= .003237	β= -.140168 Z= -1.1576 p= .123512	0	0.0116783	β= -1.12429 Z= -3.17 p= .0007622

Note: Abbreviations are as follows: β = path coefficients, ***Z*** = Z-scores , ***p*** = one-tailed probability values, IV/DV = independent/ dependent variable pairs for each path, SUPPORT = combined social support measure, ED = years of education, EMP = employment score, INC = family income, FAD = financial adequacy score, SYMP = symptomatology score, and not applicable = is was physically impossible for an indirect effect to exist.

Appendix K: Lagged LOS Correlational Models

Table K1.

Cross-Lagged Pearson Correlation Coefficients (LOS Time-1, SS, and SF Time-2) for the Respondents with a LOS During the Last Three Years, N = 43.

	LOS Time-1	SYMP Time-2	FAD Time-2	ED Time-2	EMP Time-2	INC Time-2	SS Time-2
LOS Time-1	- -	r = -.309 p = .044/2	r = .162 p = .301/2	r = .077 p = .622/2	r = -.184 p = .238/2	r = .148 p = .343/2	r = .375 p = .013/2
SYMP Time-2		- -	r = - .356 p = .019/2	r = -.215 p = .167/2	r = - .266 p = .084/2	r = - .182 p = .242/2	r = -.533 p < .001/2
FAD Time-2			- -	r = -.044 p = .777/2	r = .112 p = .475/2	r = .366 p = .016/2	r = .321 p = .036/2
ED Time-2				- -	r = .339 p = .026/2	r = .096 p = .542/2	r = -.151 p = .334/2
EMP Time-2					- -	r = .026 p = .870/2	r = .165 p = .291/2
INC Time-2						- -	r = .080 p = .610/2
SS Time-2							- -

Note: Abbreviations are as follows: r = Pearson Correlation Coefficient, p = two-tailed probability value divided by 2 to obtain the one-tailed p-value, SS = compound social support, ED = years of education, EMP = employment score, INC = family income, FAD = financial adequacy score, SYMP = symptomatology score, and LOS Time-1 = number of nights in an inpatient psychiatric facility during the last three years as at Time-1.

Table K2.

Cross-Lagged Pearson Correlation Coefficients Controlling for Time-1 Symptomatology (LOS Time-1, SS, and SF Time-2) for the Respondents with a LOS During the Last Three Years, N = 43.

	LOS Time-1	SYMP Time-2	FAD Time-2	ED Time-2	EMP Time-2	INC Time-2	SS Time-2
LOS Time-1	- -	r = -.373 p = .015/2	r = .170 p = .281/2	r = .110 p = .488/2	r = -.165 p = .397/2	r = .159 p = .315/2	r = .392 p = .010/2
SYMP Time-2		- -	r = - .351 p = .023/2	r = -.134 p = .474/2	r = -.168 p = .287/2	r = - .156 p = .324/2	r = -.525 p < .001/2
FAD Time-2			- -	r = -.0735 p = .643/2	r = .090 p = .569/2	r = .360 p = .019/2	r = .313 p = .043/2
ED Time-2				- -	r = .270 p = .083/2	r = .0681 p = .668/2	r = .116 p = .464/2
EMP Time-2					- -	r = -.007 p = .966/2	r = .130 p = .413/2
INC Time-2						- -	r = .067 p = .674/2
SS Time-2							- -

Note: Abbreviations are as follows: r_s = Spearman Correlation Coefficient, p = two-tailed probability value divided by 2 to obtain the one-tailed p-value, SS = compound social support, ED = years of education, EMP = employment score, INC = family income, FAD = financial adequacy score, SYMP = symptomatology score, and LOS Time-1 = number of nights in an inpatient psychiatric facility during the last three years as at Time-1.