A DEVELOPMENTAL STUDY OF STRESS AND PHYSICAL HEALTH IN CHILDHOOD

BY

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Abstract

Recent shifts in perspectives on health recognize the complex interplay among biological, psychological, and social factors. Psychosocial stress, including the socioeconomic environment, individual differences in self-evaluative cognitions and emotions, and the quality of social relationships have been found to be particularly potent stressors with strong associations to biological systems that mediate health and illness. Accumulating evidence suggests that the impact of stress on health begins in childhood when early environmental conditions program patterns of biological and behavioural processes that mediate health, and that health problems develop out of the cumulative dysregulating effects of exposure to multiple sources of stress over time. Using a bioecological framework, which conceptualizes child health in terms of the dynamic and interactive relationships among the child and his or her social context, the present study examined the combined effects of family socioeconomic stress, high biological reactivity to stress, exposure to negative parenting, and internalizing problems on mother's report of children's general health in a community sample of children participating in three phases of a longitudinal study. Results of hierarchical regression indicated initial child health and the interaction between children's behavioural inhibition and internalizing problems at Time 1 were significant predictors of child health at Time 2. Findings demonstrate the importance of controlling for initial health status in longitudinal research and to further assessment of the role of these proximal, individual child factors in health outcomes.

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A Developmental Study of Stress and Physical Health in Childhood

Over the past several decades it has become widely recognized that physical health cannot be understood simply in terms of biological processes (e.g., Engel, 1977). In contrast to traditional biomedical models, which viewed health simply in terms of biological processes (Engel, 1977), current conceptions acknowledge the social, psychological, and behavioral processes involved in physical health functioning (Miller, Chen, & Parker, 2011; Taylor, 2010). It is now well understood that physical health is best conceptualized in terms of the complex interactions among the biological, psychological, and social processes to which an individual is exposed.

In the past two decades, a growing body of work has found that psychological and social factors themselves constitute important sources of stress and have implications for health (Maggi, Irwin, Siddiqi, & Hertzman, 2010; Miller, Chen, & Cole, 2009; Taylor, 2010). The impact of stress on health is believed to begin in childhood when early environmental conditions may shape biological and behavioural processes that are important in the maintenance of health (Coe & Landenslager, 2007; Marin, Chen, & Miller, 2008). Although a large body of research provides evidence that early life experiences are related to health in adulthood (Lipowicz, Koziel, Hulanicka, & Kowalisko, 2007, Guralnik, Butterworth, Wadsworth, & Kuh, 2006; Taylor, Lehman, Kiefe, & Seeman, 2006; Lehman, Taylor, Kiefe, & Seeman, 2005; Lehman, Taylor, Kiefe, & Seeman, 2009), less is known about the nature of the relation and the processes through which early experiences of stress lead to effects on health. Developmental studies examining the impact of early experiences on health in childhood are few in number and

suffer from methodological shortcomings that limit the conclusions that can be drawn. Prospective investigations conducted during childhood examining health outcomes in childhood itself are needed to examine the early emergence of health effects and clarify the developmental processes through which early exposure to stress may influence health. Using a bioecological framework, which examines how biological, psychological, and social factors interact to affect outcomes (e.g., Anderson & Armstead, 1995; Bronfenbrenner & Morris, 1998), the present study examines whether exposure to multiple psychosocial risk factors in early childhood is associated with physical health effects in middle childhood.

The Link Between Stress and Health

Stress is a common human experience that occurs when environmental demands exceed an individual's perceived ability to cope with these demands (Lazarus & Folkman, 1984). A growing body of research has found exposure to psychological and social stressors to have important implications for health throughout the lifespan. A number of psychosocial factors, including socioeconomic status, social support, and individual differences in cognitive and emotional processing, have been studied in relation to health.

Stress and the Hypothalamic-Pituitary-Adrenal Axis.

The importance of studying stress has been recognized at least since the 1900s when Hans Selye (Selye, 1936, 1950) discovered that exposure to stress elicited a generalized set of physiological responses, and prolonged exposure to stress could influence the development of illness and disease. Selye was also the first to appreciate the role of the hypothalamic-pituitary-adrenal (HPA) axis in the stress response, which has become a focus of much research on how stress affects health. The HPA axis is the

principal pathway involved in the release of the stress hormone cortisol. In response to a stressor, the hypothalamus secretes corticotrophin releasing hormone (CRH), which triggers the pituitary gland to release adrenocorticotropin hormone (ACTH) into the bloodstream. In turn, ACTH stimulates the release of cortisol from the adrenal gland. Within seconds of exposure to a stressor, these circulating hormones activate a series of neurobiological, cardiovascular, metabolic, and immunological effects that increase attention to the perceived threat, increase cardiac output and respiration, and redirect blood flow to provide fuel to the aroused brain, heart, and muscles (Sapolsky, Romero, & Munck, 2000). Of the hormones released as part of the stress response, cortisol has been widely studied because of its widespread regulatory influence on a number of physiological systems in the body. Specifically, it is responsible for activating a series of stimulating and suppressive actions in the body, including increasing glucose in the bloodstream and brain and suppressing the immune, digestive, and reproductive systems in response to a stressor. At the same time, cortisol self-regulates, i.e., returns to its initial state, via a negative feedback circuit in which secreted cortisol suppresses the release of CRH and ACTH in turn reducing cortisol secretion (Levine, Zagoory-Sharon, Feldman, Lewis, & Weller, 2007).

Chronic stress and allostatic load

In the face of acute stressors, the HPA system allows the body to respond effectively to stress and it is essential to maintaining health. Over time, however, chronic stress can have a damaging effect on the body. In his theory of allostatic load, McEwen (1998) proposed that chronic stress affects health through the cumulative strain of repeated activation of physiological systems over time. The process of allostasis, which refers to the

body's ability to achieve stability through change, is essential to survival as it allows the body to adapt to stress. However, there is a cost of repeated adaptation over time. When an individual encounters multiple challenges, the repeated activity of physiological systems in response to chronic stress can damage associated tissues and organs and predispose an individual to disease. This process, called allostatic load, refers to the cost of repeated adaptation on the body (McEwen, 1998; McEwen & Gianaros, 2010; McEwen & Seeman, 1999; McEwen & Stellar, 1993). There has been considerable support for this theory, with evidence that high levels of allostatic load impair multiple domains of physical functioning. For instance, while cortisol plays an important role in metabolism by enhancing food intake and replenishing energy reserves, the overactivity of these processes due to repeated HPA activity can lead to allostatic load on the body in terms of insulin resistance, abdominal obesity, and atherosclerosis (McEwen & Seeman, 1999). Further, high and prolonged elevations in cortisol have been linked to decreases in bone mineral content (Freehill & Lenke, 1999), irreversible changes in the hippocampus (McEwen, 2006; Lupien et al., 1998), and suppression of the immune system (McEwen, 1998).

Although chronic stress was initially believed to be associated with continually high levels of cortisol, there is now evidence that chronic stress can lead to low or blunted levels of cortisol as well, which can also be associated with health problems. The finding of deficient cortisol associated with chronic stress was first described in a series of studies by Yehuda and colleagues (Yehuda, 1997; Yehuda, 2006) in which individuals who developed post traumatic stress disorder in response to a traumatic stressor also demonstrated a lack of cortisol production. Although initially thought to be a specific

correlate of post traumatic stress disorder, deficient cortisol production has also been observed in individuals living in conditions of chronic stress and linked to a range of physical illnesses, including autoimmune disorders, inflammation, chronic pain, asthma, and allergies (e.g., Heim, Elhert, Hellhammer, & Hellhammer, 2000). Such observations have led researchers to propose that the direction of the relation between chronic stress and cortisol activation depends on the time since stress onset (Fries, Hesse, & Hellhammer, 2005; Heim et al., 2000; Miller, Chen, & Zhou, 2007). That is, stress may initially result in elevated cortisol, but after extended exposure the HPA axis may develop a counter-regulatory response causing cortisol to rebound below normal. In a meta-analysis providing support for this idea (Miller et al., 2007), a negative association was found between time since onset of the stressor and HPA activity, with transitory stress tending to be associated with increased levels of cortisol and more chronic stress tending to be associated with blunted levels of cortisol. Thus, while the HPA axis is an adaptive self-regulating system that allows the body to effectively deal with stress in the short-term, repeated activation appears to dysregulate the system. When stress is acute or short-lived, the system tends to be well-regulated, but when it is chronic, the HPA axis can become dysregulated either upward or downward. A well-regulated system appears to be needed for good health, and chronic stress may be harmful to health by leading to either hyper- or hypocortisolism.

Chronic stress and the development of stress regulatory systems

Although an early assumption was that all stressors elicit the same physiological reaction (Selye, 1950), much has been learned about the particular features of the stressor and characteristics of the person facing it that affect stress reactivity. It is also now

understood that people differ in how they cope with chronic stress and that some individuals regulate their physiological reactions to stress better than others. In a meta-analysis examining chronic stress and patterns of HPA activity (Miller et al., 2007), stress responding was found to vary according to the nature of the threat posed, with stressors threatening the physical self exerting a different effect on cortisol output than stressors threatening one's psychological or social integrity. Results also indicated that HPA activity is shaped by an individual's response to the situation and that cortisol output depends on the core emotions elicited and the level of subjective distress. These findings suggest that individual psychological factors are important determinants of an individual's response to stress. Moreover, developmental models suggest that these individual differences in reactions to stress are present from very early in life and are influenced by early life experiences.

Biological programming of stress regulatory systems

According to critical period models, stress regulatory systems are programmed early in life in ways that affect health throughout the life span (Ben-Shlomo & Kuh, 2002; Hertzman, 1999; Hertzman & Boyce, 2010; Lupien, McEwen, Gunnar, & Heim, 2009). In this view, early life experiences during sensitive developmental periods permanently alter biological processes involved in stress reactivity and regulation. Thus, early adversity could be said to be biologically "embedded" or programmed into physiological processes. This process of biological embedding is viewed as one explanation for how exposures early in life may translate into health consequences decades later (Shonkoff, Boyce, & McEwen, 2009).

A considerable body of research has found support for the notion that early experiences shape neuroendocrine regulation. Consistent with critical period or biological programming perspectives, research with both animal and human infants has found that stress regulatory systems are susceptible to programming during prenatal and postnatal periods (Matthews, 2002; Meaney, 2001; Weinstock, 2001). In animals, exposure to prenatal stress modifies the development of brain regions involved in regulating the HPA axis such that they become more sensitized and reactive to stress (Lupien et al., 2009), i.e., programmed with a lower set point for reacting to stress. Similarly in humans, prenatal exposure to maternal anxiety is associated with greater cortisol reactivity to the first day of school at age five (Gutteling, de Weerth, & Buitelaar, 2005) and high overall cortisol activation in the course of a day at age 10, even after controlling for factors other than prenatal exposure that might account for these associations (postnatal anxiety and depression; O'Connor et al., 2005).

There is evidence that the programming of stress regulatory systems continues in infancy and childhood, with environmental experiences continuing to influence the development of these systems during these sensitive developmental periods (Coe & Laudenslager, 2007; Meaney, 2001). Postnatal developmental changes in the HPA axis have been particularly well-documented in rodents. The infant rat has been found to undergo a period of relative stress hypo-responsivity, whereby it is difficult to elicit HPA axis reactivity in the first two weeks of life (Gunnar, 1998). This period is thought to involve functional changes at various levels of the axis that downregulate its activity in order to protect the brain from the impact of elevated adrenocortical hormones. Similarly in humans, there is evidence that the HPA system becomes less responsive throughout

infancy. While newborns demonstrate significant cortisol reactivity to stressors (Gunnar, 1992), the reactivity of the HPA system diminishes in the first few months of life (Gunnar 1992; Lewis & Ramsay, 1995). In a longitudinal analysis of infants' response to stress, Lewis and Ramsay (1995) examined cortisol responses to standard physical examinations and inoculations at 2, 4, and 6 months of age. Results provided evidence for a developmental shift in adrenocortical functioning from 2 to 6 months, with cortisol responsivity inversely associated with age (Lewis & Ramsay, 1995). In an attempt to more closely delineate the emergence and patterns of this shift in HPA axis functioning, Gunnar and colleagues (Gunnar, Brodersen, Kruger, & Rigatuso, 1996) followed infants longitudinally from 2 to 16 months. They found significant pre-post inoculation increases in cortisol levels at 2, 4, and 6 months, but not at 15 months. Results also revealed a significant decrease in post-test cortisol levels from 2 to 4 months and again from six to 15 months, suggesting a decline in the responsivity of the HPA system to stressors over the first year of life. Behavioural indicators of distress (fussing and crying) were not found to decrease with age, suggesting that the decreasing response in HPA axis activity is not related to decreases in overall levels of distress, but rather to changes in HPA axis regulation (Gunnar et al., 1996).

Of particular interest is the finding that these early developmental changes in HPA axis functioning are highly sensitive to the social context, with early environmental experiences shaping the basal rhythms and reactivity of the system throughout infancy and childhood (Dettling, Parker, Lane, Sebanc, & Gunnar, 2000; Gunnar & Donzolla, 2002; Meaney, 2001; Tarullo & Gunnar, 2006; Watamura, Donzella, Kertes, & Gunnar, 2004). The strongest evidence for early environmental regulation of the stress response comes

from animal models, where daily handling and separation of rat pups from their mothers in the first few weeks of life has been found to alter stress reactivity in adulthood (Meaney, 2001). Similarly, research with human infants has found the quality of the early caregiving environment to moderate cortisol responses to stressful situations, even during the relative hyporesponsive period (Gunnar & Donzella, 2002). Early adversity in childhood has also been linked to dysregulation of stress regulatory systems (Carlson & Earls, 1997; DeBellis et al., 1993; Gunnar & Donzella, 2002; Kaufman et al., 1997). For instance, Romanian children raised in orphanages characterized by social deprivation, lack of physical stimulation, and little opportunity for attachment to caregivers have shown decreased levels of cortisol production and blunted daytime cortisol rhythms compared to their counterparts adopted at an early age (Carlson & Earls, 1997; McEwen, 1998; Taylor, 2010). Other less extreme characteristics of the caregiving environment also have been linked to HPA reactivity and regulation. Dettling and colleagues (Dettling et al., 2000) examined whether patterns of daily cortisol production in preschool children were related to the quality of care in home-based childcare settings. Results revealed that children in high quality childcare settings, defined by quality of focused attention and stimulation, showed the typical decreasing pattern of cortisol production from morning to afternoon, whereas those in low quality settings showed a reversal in the typical pattern. These findings suggest that the quality of caregiving can influence the cortisol response in infants and children.

Caregiver responsiveness has also been shown to predict stress responding in infants and toddlers exposed to threatening situations (Nachmias, Gunnar, Mangelsdorf, Parritz, & Buss, 1996; Spangler & Grossman, 1993). In an experimental manipulation of

caregiver responsiveness to infant distress, warm and responsive care by a babysitter was found to completely prevent elevations in cortisol in nine-month old children separated from their mothers for 30 minutes (Gunnar, Larson, Hertsgaard, Harris, & Brodersen, 1992). In contrast, infants left with a babysitter who was unresponsive unless the infant cried showed significant increases in cortisol (Gunnar et al., 1992). Similarly, in an observational study of mothers and their three-to-nine-month old infants, maternal sensitivity was shown to influence adrenocortical functioning during free play, with higher levels of cortisol observed in infants with highly insensitive mothers compared to infants with more sensitive mothers (Spangler, Schiech, Ilg, Maier, & Ackermann, 1994). Others have found the security of attachment between parents and children (reflecting a history of sensitive and responsive care by the parent) moderates HPA activity, with insecurely attached children showing greater cortisol reactivity in response to a mild stressor than those having a secure relationship with their parent (Nachmias et al., 1996; Spangler & Grossman, 1993).

The effects of parenting early in life have also been shown to have a long-lasting impact on stress regulatory processes that persist into adulthood. For instance, young adults describing a more cold and unaffectionate early family environment had higher levels of negative emotional states (hostility, depression, and anxiety) and, in turn, showed higher basal cortisol levels (Taylor, Lerner, Sage, Lehman, & Seeman, 2004). In a recent retrospective study, adult reports of parental affection in childhood were associated with adult diurnal cortisol rhythms (Taylor, Karlamangla, Friedman, & Seeman, 2010). In one recent prospective study, parental structure in 6 to 11 year-old children predicted elevated

morning cortisol and cortisol reactivity to a social threat test seven years later at ages 13 to 21 (Ellenbogen & Hodgins, 2009).

Taken together, this body of research suggests that the HPA axis regulation is shaped by experiences prenatally, in infancy and childhood and that differences in HPA activity associated with these early experiences are still evident in adulthood. These alternations in the parameters of stress regulatory systems (e.g., the set point of HPA reactivity) result in individual differences in the sensitivity of these systems, making some children more reactive to stress leading to greater allostatic load and greater risk of effects on health.

Cumulative risk exposure and stress regulation

Models of cumulative risk (e.g., Chen, 2004; Taylor, 2010; Miller, Chen, & Parker, 2011) build upon the theories of allostatic load and biological programming by suggesting that health is influenced by the cumulative exposure to risk versus protective factors over time. The greater children's exposure to stress from multiple sources, the more the effects on stress regulation may accumulate over time contributing to dysregulation of stress regulatory systems and health consequences. Models of cumulative risk are consistent with Bronfenbrenner's (Bronfenbrenner, 1979; Bronfenbrenner & Morris, 1998) bioecological theory of human development, which suggests that development is shaped by the multiple contexts in which it occurs. Specifically, development occurs in multiple nested contexts from the biological to the cultural (e.g., genetic predispositions, family functioning, community values) and from the proximal to the distal (e.g., current beliefs and perceptions, childrearing history). According to bioecological models, risk to development and health can be conceptualized as occurring across these multilevel contexts, from

individual characteristics to stressful familial and socioeconomic disadvantages. As exposure to risk accumulates, the greater the allostatic load will be and the more likely it is that health problems will develop.

Recent life course models of health focus on the ways stressors at multiple levels accumulate to influence health over time (Miller, Chen, & Parker, 2011; Taylor, 2010). Miller and colleagues (Miller et al., 2011) have recently proposed a model of biological embedding of childhood adversity where they argue that early exposure to stress calibrates physiological systems and sets into motion biological and behavioural tendencies that have long-term health implications. They suggest that early stress contributes to greater interpersonal conflict through tendencies to be more vigilant to threat and mistrustful of others and to poor self-regulation skills, leading to a propensity for unhealthy behaviours that, together, amplify chronic inflammation in the body. Consistent with this view, Taylor (2010) suggests that health problems later in life originate from the cumulative dysregulating effects of exposure to low socioeconomic status in childhood, high physiological reactivity to stress attributable to predisposing genetic vulnerabilities, inadequate psychosocial resources, and psychological distress leading to allostatic load. Health problems are increasingly likely to develop when more of these dysregulating risk factors are present, and less likely to develop if risk factors are offset by the presence of protective factors. When harsh early environments, such as low socioeconomic status or negative parenting, interact with temperamental vulnerabilities, the ability to regulate stress may become impaired. Dysregulation at one level or in one domain (e.g, temperament) may increase the likelihood of dysregulation at subsequent levels (e.g., emotion self-regulation) and contribute to the development of cognitive and emotional

processes that increase psychological stress and, in turn, increase the risk of cumulative dysregulation over time. That is, the effects of dysregulation at one level increase the risk of dysregulation at another and together there may be compounding effects over time that propel the child towards poor health outcomes. It is these cumulative and interacting dysregulations that are believed to constitute risk for mental and physical health problems.

Effects of Psychosocial Stress on Health

Research examining associations between psychosocial stress and health has found psychosocial stressors to be linked to health outcomes (illness, diseases) and to biological markers of health (e.g., indicators of immune and endocrine functioning) or health risk (e.g., blood pressure as an indicator of cardiovascular risk). Biological markers are thought to be relevant to health research as they provide indirect evidence for stress-related health effects and reflect important mechanisms or pathways through which psychosocial stress may affect health over time. Psychosocial stressors have also been linked to health-damaging behaviours, including smoking, decreased exercise, or poor adherence to medical regimens, that represent important pathways through which stress influences health, particularly in adolescents and adults. It is important to note that many studies focus on biological markers rather than health outcomes, and thus provide only indirect evidence of stress-health associations as we do not know the extent to which biological markers may, in turn, contribute to the development of health problems over time. Given that health risk behaviours are most relevant to the study of health outcomes in adolescents and adults, the current review will focus on evidence linking psychosocial stressors to markers of health or health outcomes.

Studies of psychosocial stress effects on health in adults

An abundance of research from the field of psychoneuroendocrinology, which examines how psychosocial factors influence endocrine and immune function, has linked psychological stress to both biological markers of health and health outcomes in adults (Kemeny & Schedlowski, 2007; Kiecolt-Glaser, McGuire, Robles, & Glaser, 2002). A large body of research exists on the relations between social support, which reflects the level of available psychological and material resources in one's social network (e.g., Cohen, 2004; House, Landis, & Umberson 1988; Uchino, Cacioppo, & Kiecolt-Glaser, 1996) to health outcomes or health markers. Research has found social support to be associated with the stress hormone cortisol. In one study, individuals reporting greater social support over a 10-day period exhibited lower cortisol reactivity in response to a social stressor (Eisenberger, Taylor, Gable, Hilmert, & Lieberman, 2007). These findings suggest that individuals who experience their environments as more supportive may respond to stress with less biological reactivity. This stress buffering effect may, in turn, have implications for health given that higher cortisol can suppress immune responding, thereby increasing vulnerability to infectious illness (Cohen et al., 2002). There is evidence for the immunological effects of stress from studies examining response to vaccines (Glaser, Kiecolt-Glaser, Bonneau, Malarkey & Hughes, 1992; Kiecolt-Glaser, Glaser, Gravenstein, Malarkey, & Sheridan, 1996), which serve as proxy indicators of immune response to infectious agents (Glaser & Kiecolt-Glaser, 2005). For instance, Glaser and colleagues (1992) found that medical students reporting greater interpersonal support demonstrated a stronger immune response to a Hepatitis B vaccine than those

reporting less support. Thus, there is considerable evidence that social support is a buffer against stress effects on health.

Conversely, psychosocial stress appears to enhance the risk of health problems. Cohen and colleagues (Cohen, Tyrell, & Smith, 1991) exposed 394 healthy volunteers to common cold viruses and assessed exposure to psychosocial stress and respiratory infection. They found rates of respiratory infection and cold symptoms to be associated with greater psychological stress (comprised of the number of stressful life events in the past year, perceived ability to cope, and negative affect) in a dose-response fashion, even after controlling for baseline (pre-inoculation) immune function and a number of health-risk behaviours. In another study examining the types of stressors that increase susceptibility to the common cold, (Cohen, et al., 1998) healthy volunteers were exposed to a cold virus and tested daily for respiratory and nasal symptoms five days following exposure. Those who had been dealing with chronic interpersonal stress were more likely to develop upper respiratory infections than those who had not been dealing with such stressors. The finding of health effects even after controlling for pre-existing immune function provides evidence of a causal effect of psychosocial stress on immune function and illness.

Further evidence for the immunological effects of stress has been found in research examining the progression of HIV infection (Cole, Kemeny, Taylor, Visscher, & Fahey, 1996; Leserman et al., 1999; Leserman et al., 2000). In one study, psychosocial stress and HIV disease progression were studied bi-annually for 7 years in a sample of 82 homosexual men with HIV infection. Faster HIV disease progression to AIDS was associated with more stressful life events and less perceived interpersonal support, even

after controlling for baseline markers of HIV infection and a number of health risk behaviours (Leserman et al., 2000).

Taken as a whole, studies of the effects of psychosocial stress on infection, illness, and disease progression suggest that stress can increase susceptibility to infectious agents and influence the development and progression of infection and illness. Much of the research, however, has focused on establishing connections in adulthood, and few studies have examined the effects of psychosocial stress on general health during childhood. Examination of stress-health associations in childhood may be particularly important in view of models of allostatic load, biological embedding, and cumulative risk, which, together, suggest that stress response systems are shaped early in life and initiate processes that could affect health outcomes relatively early in life. The earlier in life stress is experienced and the more of it there is, the higher allostatic load may be, and the earlier health effects may appear.

Much of the developmental research to date has been based on life-course approaches that examine the long-term effects of early exposure to stress on markers of health and health outcomes in adulthood. Few of these studies examine health effects during childhood itself. Rather, most have been retrospective longitudinal studies examining the relation between adult health and early psychosocial stress assessed many years later by retrospective report. These studies provide some degree of support for the relation between early psychosocial stress and adult health. In a series of studies by Taylor and colleagues, retrospective reports of low family socioeconomic status and harsh family environments in childhood were linked to a number of biological processes known to have implications for health in adulthood, including markers of inflammation (Taylor et al.,

2006), metabolic functioning (Lehman et al., 2005), and markers of cardiovascular risk (Lehman et al., 2009). Similarly, in a recent study of early socioeconomic conditions on adult inflammation (Carroll, Cohen, & Marsland, 2011), retrospective reports of markers of parental wealth (home and vehicle ownership, number of bedrooms in the family home) were associated with higher levels of immune markers of inflammation in adults. While these studies examined markers of health, providing indirect evidence for health effects, other retrospective studies have directly assessed health outcomes, linking retrospective reports of childhood SES to cardiovascular disease at age 50 (Lipowicz et al., 2007) and retrospective reports of exposure to abuse and dysfunction in the first 18 years of life to adult disease conditions, including heart disease, cancer, chronic lung disease, and liver disease (Felitti, et al., 1998). In one study focusing on a younger age group (Marin et al., 2008), parents' retrospective report of socioeconomic status in the first three years of the child's life predicted the child's blood pressure in adolescence.

Although the findings of these studies support the idea that early experiences are linked to health outcomes or markers of health years later, the findings are limited by the retrospective design of the research. Retrospective designs are susceptible to biases that may influence the reliability and validity of findings in a number of ways that are relevant to the inference of causal connections (Hardt & Rutter, 2004). Specifically, reliance on adult retrospective reports may be limited by poor recall, recall biased by individual characteristics or mood state at the time of reporting, or biases associated with desire to portray the family in a socially desirable way. In addition, individual characteristics (e.g., personality traits, mood disorders, mental health concerns) reflecting sources of bias may

themselves be factors that influence health. In view of these potential biases, retrospective reports may not represent an optimal measure for assessing early-life environments.

A more rigorous approach is the use of a prospective longitudinal design, in which children are followed over time and data is collected at relevant developmental periods. There have been several such studies showing that psychosocial stressors assessed early in life predict markers of health in adulthood. Caspi and colleagues (Caspi, Harrington, Moffitt, Milne, & Poulton, 2006; Danese, Pariante, Caspi, Taylor, & Poulton, 2007) have found that early experiences of psychosocial stress predicted markers of health in adulthood in a birth cohort of 1037 children followed longitudinally. In one study (Caspi et al., 2006), children identified by their parents and teachers as socially isolated (peripheral or isolated in their peer groups) at ages 5, 7, 9, 11, and 15 years were at greater risk for cardiovascular disease at age 26 when compared to non-isolated children. Moreover, the effect was cumulative, with children who were socially isolated at multiple developmental periods being at greater risk of cardiovascular disease (assessed via biomarkers of cardiovascular risk, including weight, blood pressure, cholesterol) at age 26 than those who were socially isolated at only one developmental period. In another study (Danese et al., 2007), exposure to maltreatment from ages 3 to 11 (parental rejection, harsh discipline, disruptive caregiver changes, and physical and sexual abuse) predicted a biological marker of inflammation later in life. Specifically, a cumulative maltreatment exposure index was derived by summing the number of maltreatment experiences to which children were exposed. A significant graded association was found, with children who experienced more maltreatment demonstrating greater levels of inflammation at age 32. Although the associations in both these studies were maintained even after controlling for a host of

potentially confounding risk factors, the findings are limited in several ways. In addition to the reliance on indirect measures of health, child health status prior to exposure to psychological stress was not controlled. Initial child health status is an important factor to control in order to rule out a stability effect whereby continuity in health continues to be reflected in associations between stress and health, making it unclear whether the link between stress and health can be attributed to a *change* in health over time as a result of exposure to the stressor.

A further limitation of both the retrospective and prospective longitudinal research conducted to date is the long time spans over which relations were examined. Although these studies provide evidence that early life experiences are associated with markers of health and, in some cases, health outcomes later in life, collapsing across wide age ranges and focusing on health decades later makes it difficult to interpret the meaning of the associations found and leaves open the question of how and when early experiences influence health (Chen, Matthews, & Boyce, 2002; Matthews & Gallo, 2011; Miller et al., 2009).

Studies of psychosocial stress effects on health in childhood

To address the question of how and when early health effects appear, research has begun to focus on the childhood years. Developmental research examining associations between early psychosocial stress and health outcomes or markers of health during childhood are emerging and suggest that the effects of stress can be seen in childhood. In one prospective study (Caserta et al., 2008), parental psychiatric symptoms were associated with frequency of illness, febrile illness, and changes in immune system parameters in 5- to 10-year-old children who visited an emergency department or other

pediatric services. Children with parents reporting clinically significant levels of psychiatric symptoms were at greater risk of illness in the following year. However, there was no control for the effect of child health status at the outset of the study. Consequently, it is possible that children at greater risk of illness had poorer health at the study outset. The recruitment of the sample from emergency departments or other pediatric services also limits the conclusions that can be drawn, given that children who visit these services may have poorer health or be exposed to other stressors that could account for the findings. It is possible that children visiting the emergency department may be a particularly at-risk group, limiting the generalizability of these findings to normative samples of children.

Other prospective studies have found evidence of associations between psychosocial stress and markers of health and general health functioning in more representative samples of children. In a large population-based study of over 8000 children, composite measures of maternal hostility and resentment assessed at several time points from ages two to four were found to predict mother's report of general health in children at ages seven to eight (Waylen, Stallard, & Stewart-Brown, 2008). In another study, lower SES and more negative parenting assessed in the first four years of the child's life predicted children's poorer general health at age six as reported by mothers (Belsky, Bell, Bradley, Stallard, & Steward-Brown, 2007). While these studies build on life-span developmental research by providing evidence that the effects of psychosocial stress may be evident by early childhood, both studies created composite scores on the psychosocial stressors measured by averaging across the multiple time points in childhood at which they were assessed. Collapsing across a number of years may limit understanding of how soon

after exposure to a stressor health effects may be seen. There is evidence that the timing of exposure to stress may be important. In a large population-based study, associations between parenting assessed at three time points throughout childhood (early, middle, and late) and cardiovascular risk assessed in late childhood were examined (Bell & Belsky, 2008). Results revealed parenting characterized by greater sensitivity and lower levels of conflict was associated with children's lower cardiovascular risk assessed in late childhood. Moreover, when compared to parenting assessed in middle or late childhood, measures of parenting collected prior to children's entry into kindergarten were more strongly associated with children's heart rate and blood pressure (Bell & Belsky, 2008), suggesting that psychosocial stressors experienced in early childhood may have particularly important implications for health compared to stressors experienced in middle or later childhood.

Finally, in a study examining immediate effects of stress on health over the course of a few days in early childhood (Watamura, Coe, Laudenslager, & Robertson, 2010), elevated cortisol levels in three-to six year-old children attending weekday childcare were associated with lowered antibody levels (reflecting immune function) on the subsequent weekend. Associations were also found between higher cortisol levels on weekend days and greater parent-reported infectious illness. Although initial presence of infection was controlled by measuring children's temperature at the time of cortisol sampling, there was no assessment of parent's report of child illness over time. It is possible that children with more frequent infections demonstrated greater cortisol levels as an effect rather than a cause of the illness given that illness itself is a type of stressor. While these studies increase the generalizability of findings by examining associations using more

representative samples, the failure to control for child health at the study outset limits the conclusions that can be drawn.

In summary, research to date examining the health implications of early psychosocial experiences has focused predominantly on health in adulthood and has been limited by retrospective study designs, assessment of markers of health and health outcomes over long periods of time, and failure to control for child health at study outset. In recent years, prospective developmental studies have begun to provide evidence of relations between early psychosocial stress in the child's environment and both markers of health and general health in childhood. Although these studies suggest that effects of early psychosocial stress can be seen by early childhood over periods of as little as two to three years after stress exposure, they have typically focused on markers of health rather than general health functioning, and have not controlled for child health at study outset. Research that controls for initial child health status is necessary to provide stronger evidence for a causal connection between psychosocial stress and health in childhood.

In addition, the studies to date have typically examined single rather than multiple sources of stress, although ecological models suggest that development occurs in multiple nested contexts involving systems of interrelated influences (Bronfenbrenner, 1979; Bronfenbrenner & Morris, 1998; Taylor, 2010). Singular risk factors will not predict developmental outcomes as well as multiple risk factors due to the cumulative interactive effects of multiple risks. Models of allostatic load, biological embedding, and cumulative risk suggest that stress response systems are shaped early in life and that the greater the exposure to stress early in life, the higher allostatic load may be, the more likely health effects are to appear and the earlier these effects are likely to appear. Prospective

longitudinal research examining the *combined* effects of multiple sources of stress in normative samples of children is needed to better understand the connection between psychosocial stress and health in childhood. Research informed by an ecological perspective points to some of the key sources of psychosocial stress that are likely to play an important role in this connection, particularly when they occur in combination.

Cumulative Risk Exposure and Health in Childhood

According to current life-course models of health (Miller et al., 2011; Taylor, 2010), risk to health in childhood is likely to increase as a result of the cumulative dysregulating effects of exposure to stress. Stressors associated with family socioeconomic stress, high biological reactivity to stress, exposure to negative parenting, and chronic psychological distress are key sources of stress that are likely to contribute to dysregulations by impacting developmental and biological processes involved in self- and stress-regulation. As children grow older, dysregulation in one domain increases the likelihood of dysregulation in another domain, resulting in excessive allostatic load and eventual effects on health. Research on the health effects of key sources of stress in childhood will be reviewed next.

Family socioeconomic stress

Socioeconomic status (SES), i.e., the social position and economic resources of the individual, is among the most widely studied sources of stress affecting child development and health (Bradley & Corwyn, 2002; Chen, Matthews, & Boyce, 2002). Epidemiological research with adults has consistently found the relationship between SES and health to be graded, with each incremental increase in SES accompanied by increasing health benefits (Adler et al., 1994; Bradley & Corwyn, 2002; Guralnik et al., 2006; Hertzman, 1999). In a

review of the literature on socioeconomic differences in child health, Chen and colleagues (Chen et al., 2002) concluded that, consistent with research on adults, studies of children and adolescents indicate that as SES decreases, mortality, morbidity, and rates of specific health problems increase in a monotonic fashion (Chen et al., 2002). For instance, low SES has been linked to growth retardation in utero, low birth weight, greater risk of congenital anomalies, injury-related deaths in infancy, specific health problems, and markers of health and general health functioning in children and adolescents (Chen, Fisher, Bacharier, & Strunk, 2003; Chen et al., 2006; DiPietro, Costigan, Hilton, & Pressman, 1999; Marin et al., 2008; Scholer, Hickson, & Ray, 1999; Vrijhead, Dolk, Stone, Alberman, & Scott, 2000). A number of studies have found childhood and adolescent socioeconomic status to be associated with markers of health, including body mass index (Chen & Paterson, 2006), heart rate and blood pressure (Marin et al., 2008; McGrath, Matthews, & Brady, 2006), basal cortisol levels (Chen & Patterson, 2006), and markers of immune function (Chen, Fisher, Bacharier, & Strunk, 2003; Chen et al., 2006).

While much of the research in childhood has focused on establishing concurrent associations, precluding conclusions about the causal connection between SES and health in childhood, there is some evidence from longitudinal data that SES may affect health. A majority of this research has been retrospective examining adult health and linking it to reported childhood SES. In a review of studies examining associations between childhood SES and adult mortality (Galobardes et al., 2008), lower childhood SES was associated with greater risk of premature death from cardiovascular disease, diabetes, certain cancers, and diseases of the digestive system, even after controlling for SES in adulthood. In another review of 49 studies, Pollitt, Rose, and Kaufman (2005) examined evidence of

associations between three life course models (early life SES, SES change, and cumulative SES) and adult cardiovascular outcomes. They found moderate support for the role of early life SES and strong support for effects of cumulative SES risk of adult cardiovascular disease, suggesting that risk increased with the number of years spent in low SES environments. While these reviews suggest an association between childhood SES and health later in life, the studies reviewed were limited by retrospective recall of childhood SES and the assessment of health outcomes decades after initial exposure to stressors. There is evidence of associations between childhood SES and adult morbidity from prospective longitudinal epidemiological data (Lawlor et al., 2006; Naess, Strand, & Davey Smith, 2007; Strand & Kunst, 2007), although the data has typically been collected for other purposes (i.e., census, mortality data; Cohen, Janicki-Deverts, Chen, & Matthews, 2011) and consequently initial child health was not controlled.

There has been some prospective longitudinal research examining health outcomes *in childhood* (Almqvist, Pershagen, & Wickman, 2005; Belsky et al., 2007; Kozyrskj, Kendall, Jacoby, Sly, & Zubrick, 2010). In one prospective longitudinal study (Belsky et al., 2007), lower levels of family income and maternal education assessed in the first four years of life predicted mother's report of poorer general health in their children at 6 years of age. Although initial health status was not controlled for in this study, limiting the conclusions that can be drawn, there has been some prospective research focusing on the childhood years that has controlled for health risk factors associated with specific illnesses. In a birth cohort study of 4089 infants followed longitudinally for four years (Almqvist et al., 2005), children with higher SES at two months old were at a decreased risk of asthma, allergic rhinitis, and sensitization to food allergins at age four even after controlling for a

number of known risk factors for allergic illnesses. Further prospective research has found evidence for cumulative effects of stress. In a birth cohort study of 2868 children (Kozyrskj et al., 2010) born to mothers enrolled in the study at gestational ages of 16 to 20 weeks, risk of asthma was assessed in relation to four family income trajectories from birth to age 14 (chronic low-income, increasing, decreasing, never low). After adjusting for asthma risk factors, results revealed an increased risk for asthma at age 6 and 14 in children living in chronic low-income families from birth, with stronger associations between chronic low-income and asthma at age 14 than at age 6 years. Moreover, when compared to children in chronic low-income families, children in households with increasing incomes from birth to age 14 had a significantly lower risk of asthma at age 14, suggesting that cumulative exposure to low SES is an important predictor of asthma. Although the findings of this study are consistent with cumulative risk exposure models suggesting that cumulative risk will have a greater impact than singular risks (Evans & Kim, 2010; Sameroff, Seifer, Zax, & Barocas, 1987), much of the research to date has focused on the impact of singular risk factors on health. No known prospective longitudinal research has examined the impact of the combined effects of exposure to multiple risks on children's general health functioning controlling for child health at study outset.

Bioecological models suggest it is the combined effects of stress exposure that will better account for allostatic load and health effects. With SES-health associations throughout the lifespan well established, attention has turned to models of cumulative risk exposure in explaining SES-health associations (Chen, 2004; Cohen et al., 2010; Evans & Kim, 2010; Matthews & Gallo, 2011). Models that focus on the impact of direct

mechanisms, such as limited environmental resources, inadequate living conditions, and malnutrition, or singular effects are inadequate in explaining the complexity of associations between SES and health. In two studies, Evans and colleagues found evidence of both cross-sectional and longitudinal effects of cumulative risk on allostatic load in children (Evans 2003; Evans, Kim, Ting, Tesher & Shannis, 2007). A cumulative risk index was comprised of nine domains of socioeconomic and family risk factors. Allostatic load was assessed by combining multiple physiological indicators of risk into a total allostatic load index. In the first study, 339 9-year old children with greater cumulative risk had higher allostatic load compared to children with lower levels of cumulative risk (Evans, 2003). At age 13, cumulative risk was associated with increased allostatic load, even after controlling for allostatic load at age 9. Further, the effect at age 13 was qualified by an interaction with maternal responsiveness, with cumulative risk elevating allostatic load in young adolescents only when maternal responsiveness was low (Evans et al., 2007). Others have also found evidence for combined effects, with differences in the quality of parenting influencing associations between SES and children's well-being and health (Belsky et al., 2007, Bradley & Corwyn, 2002; Chen et al., 2002). In one study, greater parental warmth/positive control and less parental negativity was found to reduce the predictive power of SES variables on child health by approximately half (Belsky et al., 2007). These findings suggest that children may be protected from the effects of cumulative stress when parenting is responsive. Alternatively, low SES combined with negative parenting may increase risk by adding to allostatic load. Recent work on the role of individual differences in cognitive and emotional processes in health (Chen, 2004; Gallo & Matthews, 2003; Taylor, 2010) suggests the impact of early exposures to stress

may not be the same for all children. Children with high biological reactivity to stress or chronic psychological distress may be more vulnerable to other stressors and to increased allostatic load. According to bioecological theories and evidence of combined effects of stress on health, family socioeconomic stress is most likely to predict poor health if it is combined with stress from other sources such as high biological reactivity to stress, negative parenting, or chronic psychological stress.

Biological reactivity to stress

Consistent with recent models of health suggesting that genetic predispositions interact with the environment to program stress-regulatory systems and influence health (Taylor, 2010), temperamental differences in biological reactivity to stress are likely to influence the impact of other psychosocial stressors on health. Certain temperamental characteristics may exacerbate the effects of exposure to stress and place individuals at heightened risk of mental and physical health problems (Boyce & Ellis, 2005; Ellis, Essex, & Boyce, 2005; Kagan, 1997). Temperament has been defined as constitutionally based individual differences in reactivity and self-regulation (Rothbart & Bates, 1998). Reactivity reflects the arousability of affect, motor activity, and attention, as measured by latency, intensity and recovery of a response, while self-regulation refers to the processes that modulate reactivity (Rothbart & Bates, 1998). Behavioural inhibition is a dimension of temperament that reflects individual differences in children's reactivity to novelty or unfamiliarity, with inhibited children showing a lower threshold for behavioural uncertainty and physiological reactivity to unfamiliar events (Kagan, Reznick, & Snidman, 1987; 1988). Behaviourally, temperamentally inhibited children demonstrate greater cautiousness and restraint in novel situations (Kagan et al., 1987). In a large body of

research (Kagan et al., 1987; 1988; Kagan, Reznick, & Gibbons, 1989; Nachmias, Gunnar, Mangelsdorf, Parritz, & Buss, 1996) children with greater temperamental inhibition have shown increased physiological reactivity to novelty unfamiliarity, or challenge. There is evidence of enhanced stress reactivity, including elevated cardiovascular responses to external stresses (Kagan, 1992), increased morning salivary cortisol levels (Kagan et al., 1987; Schmidt et al., 1997) and cortisol reactivity in response to stress (Nachmias et al., 1996).

Boyce and Ellis (2005, 2008) have argued that individual differences in temperamental inhibition not only reflect increased arousal to novelty or challenge but rather an increased biological sensitivity to context that may be adaptive or maladaptive depending on the level of support or stress in the environmental context. The implications of high biological reactivity to stress for children's development and health appears to depend on the context in which it occurs, with biological reactivity serving as a risk factor in high-stress environments or a protective factor in low-stress, nurturing environments. Consistent with this view, temperamental differences in stress reactivity have been found to influence the impact of other psychosocial stressors on health. In prospective research with two separate samples of preschool aged children, Boyce and colleagues (Boyce et al., 1995) examined environmental stress and physiological stress reactivity as joint predictors of respiratory illness incidence in children. They found that children with low stress reactivity did not show higher incidence of illness in either low or high adversity settings. Children with high stress reactivity had higher rates of respiratory illness when exposed to more stressful environments but lower rates of illness in low-stress environments when compared to all other groups of children, suggesting that the greater sensitivity of more

reactive children can be either a protective or risk factor depending on the environmental context. In view of these findings, children with high behavioural inhibition, reflecting increased reactivity to stress, are likely to have greater allostatic load and health effects, but only if such inhibition occurs in a context of high stress. Thus, high behavioural inhibition is most likely to predict poor health if it is combined with other sources of stress such as high family socioeconomic stress, negative parenting, and/or chronic psychological stress.

Exposure to negative parenting

The family environment is widely recognized to be an important determinant of children's social, emotional, and physical health. Parenting style is believed to be an important factor (Grusec, 2011; Maccoby, 1992; Maccoby, 2007; Steinberg, 2001; Repetti, Taylor, & Seeman, 2002). As noted earlier, research suggests that the quality of early caregiving plays a role in shaping the reactivity and regulation of the HPA axis (Dettling et al., 2000; Gunnar & Donzolla, 2002; Watamura et al., 2004). For example, it has been shown that maternal responsiveness influences the stress response in children exposed to threat (Nachmias et al., 1996; Spangler & Grossman, 1993). The quality of caregiving is also believed to affect other processes that contribute to health. Taylor and colleagues (Repetti et al., 2002; Taylor et al., 2004) have argued that exposure to "risky" family environments (those characterized by conflict and aggression, a cold unaffectionate interaction style, or low nurturance) contributes to long-term health consequences by disrupting psychological processes and stress-responsive biological systems that have implications for health. Risky families not only act as interpersonal stressors themselves but also fail to provide opportunities for the development of effective coping and self-

regulatory skills, leaving children vulnerable to negative emotion and unequipped to manage stress.

Parenting styles have been widely studied in relation to child well-being. Authoritative parenting, characterized by warmth, reasoning, and autonomy granting, is associated with positive social, emotional, and behavioural outcomes for children (Steinberg, Elmen, & Mounts, 1989; Steinberg, 2001). Children from authoritative homes show greater competence, greater academic achievement, and fewer internalizing and behaviour problems than children raised in authoritarian, indulgent, or neglectful homes (Lamborn, Mounts, Steinberg, & Dornbusch, 1991; Steinberg, Blatt-Eisengart, & Cauffman, 2006). Conversely, children from authoritarian homes, characterized by high levels of hostility, directiveness, and punitive discipline, have been found to demonstrate lower levels of self-reliance and poorer self-concept (Lamborn et al., 1991) that persist over time (Steinberg, Lamborn, Darling, Mounts, & Dornbusch, 1994). Consistent with Taylor's (Taylor et al., 2004) notion of risky families, these findings suggest that the emotional climate created by an authoritative parenting style may serve as a buffer that provides resources for managing stress, whereas authoritarian parenting may undermine the development of these resources. As previously noted, there is some evidence from prospective longitudinal research that hostile or unsupportive parenting in early childhood is associated with markers of health and general health functioning by mid-childhood (Bell & Belsky, 2008; Belsky et al., 2007; Waylen et al., 2008) and that it may moderate the strength of the relation between other risk factors and health (e.g., Belsky et al., 2007; Evans et al., 2007). Given evidence of greater maternal than paternal involvement with preschool children (McBride & Mills, 1993; Yeung, Sandberg, David-Kean, & Hofferth,

2004), the majority of research with young children has focused on maternal parenting. Greater maternal resentment and hostility (Bell & Belsky, 2007; Waylen et al., 2008) and lower maternal warmth (Belsky et al., 2007) have been found prospectively to predict general health in children, although the effects were relatively small. While one study has found paternal hostility and supportive paternal presence to predict markers of cardiovascular functioning (Bell & Belsky, 2008), father's parenting has not been assessed in relation to children's general health functioning. Given the relatively small effect sizes and assessment of parenting primarily as a singular factor, further research is needed to determine whether children with low SES and/or greater biological reactivity to stress are likely to have greater allostatic load and increased risk of health problems if they also have mothers who engage in more negative parenting.

Internalizing Problems

Chronic psychological distress is another key source of stress that is likely to have cumulative dysregulating effects that have an impact on health. Individual differences in cognitive and emotional processing are thought to play an important role in the stress response and its effects on health (Dandeneau, Baldwin, Baccas, Sakellaropoulo, & Pruessner, 2007). Chronic experiences of psychological stress associated with internalizing problems such as anxiety and depression appear to be sources of health risk (Gross, 2002; Miller et al., 2007; Rohleder, Chen, Wolf, & Miller, 2008). Research suggests that psychological stress has potent effects on the HPA system (Dickerson, Gruenewald, & Kemeny, 2004; Gruenewald, Kemeny, Aziz, & Fahey, 2004; Dickerson, Gable, Irwin, Aziz, & Kemeny, 2009; Kemeny, 2003; Dickerson, S., Mycek, P., & Zaldivar, F., 2008). In a meta-analysis of 208 laboratory studies of psychological stressors in adults, Dickerson

and Kemeny (2004) found that tasks involving social-evaluative threat were associated with the largest cortisol changes and the longest times to recovery. In an experimental study, Gruenewald and colleagues (Gruenewald et al., 2004) compared cognitive, emotional, and physiological responses in 81 participants randomly assigned to perform a laboratory stress test (speech and mental arithmetic) either in the presence or absence of social evaluation. Results revealed that participants in the social evaluation condition exhibited greater self-conscious emotion (i.e., shame), reported lower social self-esteem, and produced greater levels of salivary cortisol compared to participants assigned to a non-social threat condition (Gruenewald et al., 2004). Furthermore, while greater cortisol increases were found in participants who experienced greater increases in shame and greater decreases in social self-esteem, cortisol increases did not differ as a function of the magnitude of other psychological states (anxiety, performance self-esteem), indicating that cortisol reactivity may have specific associations with the emotions and cognitions associated with threats to the self (Gruenwald et al., 2004). Similarly in childhood, behavioural displays of self-evaluative emotion have been linked to increased cortisol responses to stress (Lewis & Ramsay, 2002). Lewis and Ramsay (2002) observed behavioural expressions of self-conscious emotion in children and collected samples of salivary cortisol in response to an evaluative failure task (i.e., matching task structured so the child fails) and an exposure situation (i.e., the child is the focus of attention in a neutral or positive manner). Results revealed that negative self-evaluative and exposure embarrassment were differentially associated with cortisol reactivity. That is, while evaluative embarrassment was associated with greater cortisol responses, exposure embarrassment was related to lower cortisol responses, suggesting that cortisol reactivity

may have a specific association with negative self-evaluation as opposed to other self-conscious emotions (Lewis & Ramsay, 2002). These findings suggest that, as early as the preschool years, chronic feelings of personal distress may be a psychological stressor.

Research on internalizing disorders such as anxiety and depression also provides support for the idea that chronic psychological stress can have health implications. Anxiety and depression are more prevalent among individuals with chronic illnesses (e.g., diabetes, arthritis, and inflammatory bowel, cardiac, and thyroid diseases) than among those without such illnesses (Harter, Conway, & Merikangas, 2003; Kiecolt-Glaser et al., 2002; Graff, Walker, & Berstein, 2010; Sareen, Jacobi, Cox, Belik, Clara et al., 2006). They have also been linked to greater symptom severity (Katon et al., 2007) and increased functional and role impairment (Egede, 2007; Kessler, Ormel, Demler, & Stang, 2003; Sareen et al., 2006; Stein, Cox, Afifi, Belik, & Sareen, 2006) among individuals with chronic illness. In one of the largest population-based studies examining the impact of depression on functional impairment associated with chronic illness, the presence of co-morbid major depressive disorder was associated with nearly double the likelihood of health-care utilization and increased functional disability compared to the presence of a chronic illness without co-morbid major depressive disorder (Stein et al., 2006). Internalizing problems have become widely recognized as important risk factors for cardiovascular disease (Pennix et al., 2001; Surtees et al., 2008; Van der Kooy et al., 2007), with evidence from longitudinal cohort studies that internalizing problems contribute to increased risk of cardiac mortality even in individuals without clinical evidence of coronary disease at the time of study enrollment (Doering et al., 2010; Pennix et al., 2001; Surtees et al., 2008). While a number of behavioural mechanisms have been implicated in the association

between anxiety and depression and cardiac morbidity and mortality (Rozanski, Blumenthal, & Kaplan, 1999), there is evidence that internalizing problems contribute directly to heart disease risk through a number of physiological mechanisms, including HPA axis dysregulation, increased sympathetic nervous system activity, and inflammatory immune responses (Carney et al., 2005; Liukkonen et al., 2006; Pennix et al., 2005). Together these findings suggest that internalizing problems are comorbid with and may contribute to increased morbidity and mortality associated with many chronic health conditions.

Although less widely studied than in adults, research with children and adolescents has also found internalizing problems to be associated with chronic illness (Chavira, Garland, Daley, & Hough, 2008; Farrell, Turner, Donovan, & Walker, 2011; Katon et al., 2007; McQuaid, Kopel, & Nassau, 2001; Slattery & Essex, 2010), increased health care utilization and functional impairment (Chavira et al., 2008; Claar, Walker, & Smith, 1999; Mulvaney, Lambert, Garber, & Walker, 2006; Richardson, Russo, Lozano, McCauley, & Katon, 2008). Similar to research with adults, internalizing problems have been found to occur at higher rates in youth with chronic illness, including asthma and allergic disorders, functional gastrointestinal disorders, and diabetes. In one recent study, Chavira and colleagues (Chavira et al., 2008) examined the impact of comorbid medical illness and anxiety on mental health and functional health outcomes in 588 youth aged 6 to 18. Youth with comorbid anxiety disorders and medical illness were compared to anxious youth without medical illness and those with illness but no anxiety disorder. Results indicated that at least half of youth with anxiety disorders also had a comorbid medical illness. In addition, youth with comorbid anxiety disorders and medical illness were found to exhibit

greater emotional problems, somatic complaints, and functional impairment when compared to anxious youth without physical illness and those with physical illness alone. There is also evidence from longitudinal research that children with comorbid internalizing problems and illness exhibit greater functional impairment associated with their illness. In a study examining trajectories of symptoms and impairment associated with recurrent abdominal pain, 132 youth aged 6 to 18 years were followed longitudinally for five years (Mulvaney et al., 2006). Of the three trajectories identified (low risk, short-term risk, and long-term risk groups), the long-term risk group, which demonstrated the highest level of symptoms and impairment at five-year follow up, was characterized at baseline by the highest levels of anxiety and depression and the lowest levels of perceived self-worth. While the existing research provides evidence of comorbidity among internalizing and medical problems in youth and increased impairment associated with this comorbidity, the nature of these relationships is still not well understood. A majority of research has been cross-sectional in design and conducted in youth with chronic illness, limiting our understanding of the temporal relations among these conditions and the impact of internalizing problems in samples of healthy children without chronic health problems.

Internalizing problems may also exacerbate the effects of other stressors. Children with greater levels of self-evaluative cognitions and emotions associated with internalizing problems may perceive events as more threatening and respond with ineffective coping strategies, thus increasing their vulnerability to the effects of stress (Compas & Phares, 1991). For instance, cognitive information processing biases in individuals with internalizing problems have been found to contribute to increased perceptions of and sensitivity to threat. Individuals with anxiety disorders show biased processing of social

interactions, including selective attention to negative self-related information in interactions and greater recall of negative self-related information following interactions (Mellings & Alden, 2000). Anxious individuals process emotional stimuli more quickly (Ellenbogen & Schwartzman, 2009) and are more likely to perceive neutral or ambiguous social or emotional cues or events as threatening (Constans, Penn, Ihen, & Hope, 1999; Taghavi, Moradi, Neshat-Doost, Yule, & Dalgeish, 2000), making these individuals more sensitive to the effects of other stressors. There is also evidence that self-evaluative cognitions and emotions moderate the severity of physical complaints and health problems in children. In one study, self-perceived competence was found to influence symptoms of irritable bowel syndrome and disability in adolescents and adults with a history of recurrent abdominal pain (Claar et al., 1999). Specifically, for adolescents and young adults with lower perceived academic competence, irritable bowel symptoms were more strongly associated with functional disability than those with higher levels of perceived academic competence. Furthermore, the relation between symptoms and disability was stronger at lower levels of perceived social competence in girls and perceived athletic competence in boys. Walker and colleagues (Walker, Garber, & Green, 1993) examined whether perceived competence would influence associations between negative life events and somatic complaints in pediatric patients with chronic abdominal pain. The effect of negative family life events on somatic complaints was weaker among children with higher scores on a composite measure of social competence, comprised of child, teacher, and parent ratings of social skills and peer acceptance. Ratings of academic competence, in contrast, did not moderate the association between negative family life events and children's somatic complaints. These findings suggest that internalizing problems may

increase the effects of other stressors, contributing to increased allostatic load and consequences for health.

Summary and Hypothesis

In summary, bioecological models suggest that risk to health in childhood is likely to increase as a result of the cumulative dysregulating effects of exposure to multiple sources of stress and their synergistic effects. Family socioeconomic stress, high biological reactivity to stress, exposure to negative parenting, and chronic psychological distress have been identified as key sources of psychosocial stress in early life due to their dysregulating effects on developmental processes involved in self-regulation. To date, research on health effects in childhood has included few prospective studies examining health during childhood with initial health status controlled, and most studies have focused on the key psychosocial risk factors individually rather than in combination. However, the evidence reviewed provides some support for the idea that when more than one key psychosocial stressor is present early in childhood the risk to health will be elevated (e.g., Boyce et al., 1995; Evans 2003; Evans et al., 2007). Further prospective developmental research that examines the combined effects of multiple psychosocial stressors early in childhood is needed to more firmly establish that psychosocial stress has an impact on childhood health.

To this end, the present study addressed the question of whether children with greater exposure to multiple sources of psychosocial stress would subsequently show poorer physical health, as rated by mothers. On the basis of bioecological theories suggesting that risk to health is likely to increase as a result of the cumulative effects of exposure to multiple psychosocial stressors, it was hypothesized that the greater children's

exposure to multiple psychosocial risk factors during early childhood, the poorer their health will be in middle childhood.

The hypothesis was tested through a prospective longitudinal study following a community sample of children from preschool age (3-4 years of age) to early elementary school age (5-7 years of age) and middle elementary school age (7-9 years of age). Psychosocial risk factors (SES, temperament, parenting, internalizing problems) were assessed at preschool age (Time 1) and maternal ratings of their child's global health and were assessed at preschool age (Time 1), early elementary school age (Time 2), and at middle elementary school age (Time 3). It was predicted that, with global health status at preschool age controlled, exposure to multiple risks at preschool age would predict poorer overall health by early or middle elementary school age. It was expected that the combined effects of multiple risk exposure would be greater than that of singular risk exposure.

Method

Participants

Participants were 252 children (111 girls, 141 boys) and their parents (245 mothers, 210 fathers) involved in a longitudinal study that began when the children were between 3.6 and 4.5 years of age (M = 4.1 years, SD = .27). Of the 252 children who participated at the first time point (Time 1), 221 (88%) participated at the second time point (Time 2) when they were between 5.3 and 7.3 years of age (M = 5.9 years, SD = .26), and 180 (71%) participated at the third time point (Time 3) when they were between 7.5 and 9.4 years of age (M = 8.1 years, SD = .28). The variation in age spans across the study time points was a result of differences in the pace of data collection. The interval ranged from 13 to 34 months (M = 21.2 months, SD = 2.43) between Time 1 and Time 2 data

collection and 18 to 36 months (M = 27 months, SD = 2.93) between Time 2 and Time 3 data collection, resulting in a slight increase in age span across time points. Children were predominantly first, second, or third born (34.5%, 34.1%, and 16.7% respectively), with a small proportion being an only child (4.8%).

Time 1 family demographic information is shown in Table 1. For ease of presentation, individual categories representing only a very small proportion of the sample were combined. As the table shows, parents were predominantly in their 30s, well-educated (48% of mothers and 42.9% of fathers had community college or some university), were married or cohabiting (87%), and were European in ethnic origin (75% of mothers and 75.4% of fathers). Annual total family income before taxes was above $60,000 for almost half the families, and was below $40,000 for one-quarter of the families.

The sample was recruited from a cohort of 3,500 children and their families drawn randomly from a population of 6,358 children living in Winnipeg, Manitoba, born between June 1, 1999 and May 31, 2000, and registered with Manitoba Health, the provincial health ministry. Recruitment was conducted with the assistance of Manitoba Health who mailed a letter, on the researcher's behalf, inviting families with healthy children to participate in a study examining the influence of children's emotions on health. Parents who were interested in participating were invited to contact the University by calling or by mailing an enclosed response card containing their name and phone number. Parents from 364 families inquired about the study. Of these 364 families, 257 volunteered to participate. Four of these families withdrew during the first phase and one was excluded from data analysis due to a lack of knowledge of English, leaving 252 families. Of the

participating families, 220 were two-parent families, with both parents participating in 204 cases.

From Time 1 to Time 3 of the study, a total of 76 families were lost to attrition: 31 from Time 1 to Time 2 (a loss of 12%, leaving 221 families in the sample) and 41 from Time 2 to Time 3 (a loss of 19% of the Time 2 sample, leaving 180 families in the sample). Families who continued to participate at Time 2 and Time 3 did not differ significantly from those who withdrew on child age, t (250) = -1.1, p = .28, mother's age, $\chi^2(3, N = 244) = 4.1$, p = .25, father's age, $\chi^2(4, N = 227) = 1.9$, p = .76, or mother's education, $\chi^2(4, N = 246) = 3.55$, p = .47. Families who continued to participate at Time 2 were more likely to have a son than a daughter in the study, $\chi^2(1, N = 252) = 4.26$, p = .04, had higher family incomes, $\chi^2(6, N = 240) = 18.71$, p = .01, were more likely to be two-parent families, $\chi^2(1, N = 249) = 6.54$, p = .01, and had more educated fathers, $\chi^2(4, N = 231) = 27.44$, $p < .001$, than families who dropped out between Time 1 and Time 2. Families who continued to participate at Time 3 had higher family incomes, $\chi^2(6, N = 240) = 13.64$, p = .034 and were more likely to be two-parent families, $\chi^2(1, N = 249) = 4.23$, p = .04, than families who dropped out between Time 1 and Time 3 but did not differ according to child sex, father education, or ethnicity. Given the finding that child sex, family income, and one- versus two-parent family structure were associated with attrition, it was decided to include these variables as auxiliary variables in the procedure used to handle missing values (Enders, 2010).

Table 1

Family Demographic Information

	Mothers		Fathers	
Variable	n	%	n	%
Age				
0-29	46	18.9	28	12.3
30-39	150	61.5	132	58.1
40 or above	48	19.6	67	29.5
Ethnicity				
European	183	75.0	169	74.4
Aboriginal	28	11.5	23	10.1
Other	33	13.5	35	15.5
Education				
12th grade or less	87	35.4	70	30.3
Community college/some university	118	48.0	99	42.9
University Graduate or higher	41	16.7	62	26.8
Family Income				
Under 20,000	22	9.1		
21– 40,000	42	17.5		
41-60,000	62	25.8		
Over 61,000	114	47.5		

Procedure

Data for the current study was drawn from the first three time points of a larger longitudinal study. In the first phase of data collection (Time one), mothers and fathers were mailed a package of questionnaires assessing family demographic information, child characteristics, and parenting practices. Parents were asked to complete the questionnaires independently of one another and to bring the completed questionnaires with them to the university laboratory where their child's emotional responding was assessed during a 1½ hour visit. The second and third phases of the study involved similar procedures. Specifically, in both the second (Time 2) and third (Time 3) phases, parents completed an additional series of questionnaires and children visited the laboratory to participate in an assessment of their psychosocial functioning. At each phase, families were provided an honorarium of $75 for their participation. The source of the data for the study was the demographic, authoritative parenting, and child temperament questionnaires completed by parents at time one and mother's report of child health at time one, two, and three.

Measures

Family socioeconomic stress at Time 1. Family socioeconomic stress was assessed from parents' reports of education, occupation, and family income (see Appendix A). Parents reported their highest level of education completed on a 5-point scale ranging from 1 (1^{st} to 8^{th} grade) to 5 (graduate or professional school). They reported their annual total family income before taxes using a 7-point scale ranging from 1 (under $10,000) to 7 (over $75,000). In the small proportion (12%) of two-parent families in which mother's and father's report of family income differed, an average of the two scale scores was computed. In all but three cases, parent's report of family income differed by only one

category. Occupational prestige was assessed using an updated version of the Standard International Occupational Prestige Scale (SIOPS; Ganzeboom & Treiman, 1996; Treiman, 1977) which provides a procedure for coding internationally comparable measures of occupational status based on the International Standard Classification of Occupation (ISCO88). The ISCO88 was developed in 1958 and revised in 1968 and 1988 by the International Labor Office of the United Nations to provide a standard hierarchical classification system for occupational categories. The occupations categorized in the SIOPS range from legislators, senior officials, and professionals to trade workers, machine operators, and laborers, with the highest scores given to the professional/senior occupations. For families in which both parents worked outside of the home, the score for the parent with the highest occupational prestige score was used. Each of the four components of family socioeconomic stress was recoded so that lower scores reflected lower stress (higher socioeconomic status) and higher scores reflected higher stress (lower socioeconomic status). The four components were all moderately and positively correlated with each other (lowest $r = .38$) and an overall measure of family socioeconomic stress was computed by transforming each of the four measures (mother's education, father's education, highest occupational prestige, and family income) into a standard (z) score and averaging the four z scores to create a single overall measure of family socioeconomic stress. Higher values reflected greater family socioeconomic stress.

Maternal negative parenting at Time 1. Mothers completed the Parenting Styles and Dimensions questionnaire by reading 62 statements and rating, on a 5-point scale, the frequency with which they exhibit the behaviour described (1 = *never*; 2 = *once in a while*; 3 = abo*ut half of the time*; 4 = *very often*; 5 = *always*). The PSDQ yields three global

parenting style factors (authoritative, authoritarian, permissive) and internally consistent subscales (Robinson, Mandleco, Olsen, & Hart, 2001). It has good factorial and predictive validity as indicated by relations with children's social and behavioural outcomes (Robinson et al., 2001; Russell, Hart, Robinson, & Olsen, 2003; Winsler, Madigan, & Aquilo, 2005). The authoritarian factor is comprised of three 4-item subscales assessing physical coercion (e.g., "slaps child when the child misbehaves"), verbal hostility (e.g., "scolds or criticizes to make child improve"), and non-reasoning/punitive (e.g., "uses threats as punishment with little or no justification"). A measure of maternal negative parenting was computed by averaging the 12 items comprising the authoritarian factor. Higher scores represented more negative parenting. Cronbach's alpha was .77.

Child behavioural inhibition at Time 1. Children's temperamental inhibition was assessed using the Children's Behavior Questionnaire (CBQ; Rothbart, Ahadi, Hershey, & Fisher, 2001), a parent-report measure designed to assess dimensions of temperament in children aged three to seven years. Parents were asked to respond to 194 items describing children's responses to situations on a 7-point Likert scale ranging from "extremely untrue of your child" to "extremely true of your child." The CBQ is comprised of 15 subscales assessing the following temperament dimensions: Activity Level; Anger/Frustration; Attentional Focusing; Discomfort; Fear; High Intensity Pleasure; Impulsivity; Inhibitory Control; Low Intensity Pleasure; Perceptual Sensitivity; Positive Anticipation; Sadness; Shyness; Smiling/Laughter; and Soothability. The subscales show internal consistency, with alpha coefficients ranging from .64 to .92 (Rothbart et al., 2001), and good stability over time (.54 to .77 over 12 months; Putnam & Rothbart, 2006). Convergent validity, as indicated by parental agreement, has been demonstrated by correlations between parent

ratings, with correlations ranging from .28 to .79 and a mean agreement across scales of .51 (Rothbart et al., 2001).

Factor analysis of the 15 temperament scales of the CBQ have consistently yielded three broad dimensions of temperament: Extraversion or Surgency (defined by positive loadings on the scales of Impulsivity, High Intensity Pleasure, and Activity Level and a negative loading on Shyness), Negative Affectivity (defined by positive loadings on the scales of Anger/Frustration, Sadness, Fear, and Discomfort, and a negative loading on Soothability), and Effortful Control (defined by positive loadings on Low Intensity Pleasure, Smiling/Laughter, Inhibitory Control, Perceptual Sensitivity, and Attentional Control; Ahadi, Rothbart, & Ye 1993; Rothbart et al., 2001). Behavioural inhibition has been assessed by aggregating the Fear, Shyness, and Discomfort subscales to obtain a measure of arousability/fearfulness (e.g., Kochanska, DeVet, Goldman, Murray, & Putnam, 1994). The three subscales were internally consistent as shown by high Cronbach's alphas for Fear (.74 for mothers, .76 for fathers), Shyness (.93 for mothers, .91 for fathers), and Discomfort (.74 for mothers, .72 for fathers). The three subscales also cohered (alphas of .89 for mothers and .87 for fathers) and thus were aggregated by averaging to create an overall measure of behavioural inhibition as assessed by each parent. Mother and father assessments were correlated, $r(252) = .64$, $p < .001$, and were averaged to create a single overall score reflecting behavioural inhibition as assessed by both parents (in two-parent households).

Child internalizing problems at Time 1. Children's internalizing problems were assessed using the Child Behavior Checklist, preschool version for ages 1.5 – 5 years (ASEBA CBCL; Achenbach & Rescorla, 2001). The ASEBA CBCL is a widely used

multiple-informant tool for assessing emotional and behavioural problems in children. Parents were asked to read a list of 99 items describing behavioural and emotional functioning and rate each item as 0 for *not true* of the child, 1 for *sometimes true,* or 2 for *very true or often true* based on the child's behaviour in the preceding two months. The ASEBA CBCL assesses seven domains of children's functioning: emotionally reactive, anxious/depressed, somatic complaints, withdrawn behaviour, sleep problems, attentional problems, and aggressive behaviour.

The ASEBA CBCL has good psychometric properties (Achenbach & Rescorla, 2001; Rescorla, 2005). One-week test-retest reliability for the scales has been found to range from .80 to .90, with a mean of .85 (Rescorla, 2005). Internal consistency has also been found to be high (Achenbach, 1992; Koot, Van Den Oord, Verhurst, & Boomsma, 1997). The validity of the scales has been supported by evidence showing that children referred for mental health services score higher on all scales compared to non-referred children (Achenbach & Rescorla, 2000; Koot et al., 1997; Rescorla, 1995) and by associations between scale scores and measures of social and emotional functioning, temperament, and clinically diagnosed emotional and behavioural problems (Koot et al., 1997).

Internalizing problems are assessed from 36 items evaluating emotional reactivity (9 items), anxiety/depression (8 items), withdrawn behaviour (8 items), and somatic complaints (11 items). Due to potential overlap between somatic items and physical health, in the present study the measure of internalizing problems was computed with the 11 somatic items excluded. Six items reflecting shyness/separation anxiety showed moderate correlations with items on the shyness scale of the CBQ and thus were also

excluded. The remaining 19 items from the emotional reactivity, anxiety/depression, and withdrawn scales were averaged to create separate scores for mother and father reports of internalizing problems. Cronbach's alphas were .81 for mothers and .84 for fathers. Given that the correlation between mother and father assessments was somewhat low, r (252) = .21, $p < .001$, it was not possible to aggregate the assessments into a single measure. Instead, separate analyses were performed using mother and father assessments.

Child global health at Time 1, Time 2, and Time 3. Mothers completed a short form of the Child Health Questionnaire (CHQ-PF; Landgraf, Abetz, & Ware, 1996), a parent-report measure of physical health and psychosocial well-being in children five years of age or older. Several versions of the CHQ varying in length from 28-98 items have been constructed. In the larger study from which the present data were drawn, the 50-item version of the CHQ was modified to include only those items assessing children's physical health functioning. For the purposes of the present study, children's health was assessed using the general health perception scale of the CHQ, which is comprised of six items assessing global health (1 item: In general, would you say your child's health is:) and general health (5 items: My child seems to be less healthy other children I know; My child has never been seriously ill; When there is something going around, my child usually catches it; I expect my child will have a very healthy life; I worry more about my child's health than other people worry about their children's health). Parents responded to the one global health item by rating their child on a continuum ranging from *excellent* to *poor*, and to the general health items by rating their child on a 5-point Likert scale ranging from *definitely true* to *definitely false*.

The CHQ has been normed on a representative sample of 914 children comprised of 16 normative subgroups (grouped according to child age, respondent, and demographics) and six clinical subgroups (including asthma, ADHD, cystic fibrosis, epilepsy, juvenile rheumatoid arthritis, and psychiatric problems; Landgraf et al., 1996). Studies of the factor structure of the CHQ have consistently found support for a two-factor model representing physical and psychosocial dimensions of health (Drotar, Schwartz, Palermo, & Burrant, 2006; Hepner & Sechrest, 2002; Landgraf et al., 1996). A minimum criterion for internal consistency of .40 was exceeded on average by 91% of the tests for the normative subgroups and 84% for the clinical samples (Landgraf et al., 1996). The internal consistency of the six-item general health perceptions scale was found to be .53 in one study (Rentz, Mattza, Swenson, & Revicki, 2005). There is also evidence for the validity of the general health perceptions scale. Concurrent validity is indicated by significant correlations between the CHQ scales and a parent-rated visual analogue scale rating of the child's health, with the strongest correlations being found for the global health item alone and the overall general health perceptions scale, $r = .50$, $p < 0.01$ and $r = .39$, $p < 0.01$, respectively (Raat, Botterweck, Landgraf, Hoogeveen, & Essink-Bot, 2005). Moderate correlations between the general health perceptions scale and doctor's assessment of symptom severity and global functioning in a sample of youth with juvenile chronic arthritis has also been found (Norrby, Nordhold, & Fasth, 2003). The CHQ has also been shown to discriminate between subgroups of children with and without parent-reported chronic conditions (asthma and frequent headaches) (Raat et al., 2005) and children's level of health care utilization (no visits to the doctor within one year compared

to three or more visits within one year; Raat, Bonsel, Essink-Bot, Landgraf, & Gemke, 2002).

An overall score at each time point was computed by recoding mothers' responses to the six items of the general health perception scale so that higher values represented better health, and averaging the six ratings. Cronbach's alphas were .63, .65, and .65 at Time 1, Time 2, and Time 3, respectively.

Results

Preliminary Analyses

The distributional properties of the variables were examined to identify and reduce the influence of any out-of-range or extreme values. Three variables with univariate outliers were identified, with each variable having no more than six extreme values. Following the recommendation of Tabachnick and Fidell (2001), extreme high or low values were decreased or increased as appropriate to the one unit larger or smaller than the next value that was not an outlier. The resulting variables were normally distributed, as indicated by low skewness and kurtosis.

An examination of missing data using SPSS Missing Values Analysis (MVA) revealed that, at Time 1, the proportion of missing data was less than 5% for family socioeconomic stress (0.8%), mother's negative parenting (2.8%), and behavioural inhibition (2.8%), internalizing problems as assessed by mothers (2.8%), and child global health (2.8%). The proportions of missing data were higher for internalizing problems as assessed by fathers (16.7%), reflecting fathers' lower rate of participation in the study. At Time 2 and Time 3, substantial proportions of data were missing for child global health (15.1% and 33.3%, respectively), due predominantly to attrition.

Families who were lost to attrition at Time 2 and Time 3 were compared to those who continued to participate on predictors at Time 1 and child health. When compared to families who continued to participate from Time 1 to Time 2, families who dropped out had significantly higher levels of family socioeconomic stress, $t(248) = 2.66$, $p = .01$. There were no significant differences between the two groups on Time 1 measures of child global health, $t(243) = -1.5$, $p = .13$, maternal negative parenting, $t(238) = .66$, $p = .51$, behavioural inhibition, $t(247) = .43$, $p = .67$, or internalizing problems as assessed by mother, $t(243) = -.15$, $p = .89$, and father, $t(208) = .22$, $p = .83$. Similarly for attrition between Time 2 and Time 3, when compared to families who continued to participate from Time 2 to Time 3, families who dropped out had significantly higher levels of family socioeconomic stress, $t(248) = 2.4$, $p = .02$. There were no significant differences between the two groups on Time 1 measures of child global health, $t(243) = -1.01$, $p = .29$, maternal negative parenting, $t(238) = .67$, $p = .50$, behavioural inhibition, $t(247) = -.10$, $p = .92$, or internalizing problems as assessed by mother, $t(243) = -1.36$, $p = .18$, or by father, $t(208) = -.99$, $p = .02$. There were no differences between families who continued to participate at Time 3 and those who dropped out on measures of Time 2 health, $t(212) = -.63$, $p = .53$.

Patterns of missing data were further assessed using SPSS MVA. Little's test of data missing completely at random (Little, 1988) simultaneously evaluates mean differences on every variable and provides a global assessment of missingness in the data set. Results of Little's test indicated that the overall pattern of missing data in the full data set did not reliably deviate from randomness, χ^2 (148) = 173.94. $p = .07$. Although Little's test provides information about the *overall* patterns of missingness in the data set, it does not assess specific patterns of missingness among particular variables. These can be

assessed using the MVA t-test procedure. T-tests are computed using an indicator variable that specifies whether a variable is present or missing for each case and provides information regarding how the pattern of missing data in one variable affects the values of another variable. The t-tests indicated that children with missing data for father assessment of Time 1 internalizing problems, compared to those without missing data, were in families under higher socioeconomic stress at Time 1, $t(51.7) = -3.3$, $p < .001$, and had better Time 2 child global health, $t(43.1) = -2.7$, $p = .01$. Children with missing values for Time 2 child global health, compared to those without missing data, were in families under higher socioeconomic stress at Time 1. Considered together with the results of Little's test, these findings suggest that, while the overall pattern of missing data in the data set was not systematic, child global health at Time 2 was related to two of the predictor variables, father-assessed internalizing problems and family socioeconomic stress. There were no other systematic relationships between data loss and study variables. Time 3 health was not systematically related to missingness on any study variables.

Given that attrition and low father participation were the predominant sources of missing values, multiple imputation was performed to reduce bias in parameter estimates and minimize the loss of power that can result from missing values. Multiple imputation is considered to yield more accurate parameter estimates and standard errors than traditional deletion or single imputation approaches for handling missing data (e.g., Enders, 2010; Graham, 2009; Jelicic, Phelps, & Lerner, 2009). Further, the inclusion of auxiliary variables (variables that may be a potential cause or correlate of missing data) in the multiple imputation procedure aids in reducing the biasing effects of attrition (Graham, 2009). Using regression-based procedures, multiple imputation creates multiple datasets,

each containing different plausible estimates of the missing values. Following recommendations about the number of imputations needed to maximize power and efficiency based on the proportion of data that are missing (Baraldi & Enders, 2010; Graham, Olchowski & Gilreath, 2007), 20 data sets were imputed. Although no specific rules regarding the number of imputations exist, simulation studies suggest that standard errors decrease as number of imputations increase and that power is maximized at 20 imputations, with little statistical benefit beyond 20 imputations (Enders, 2010; Graham, Olchowski & Gilreath, 2007). The imputation process includes all variables in the original data set that will be used in any subsequent statistical analysis as well as any auxiliary variables that may account for missing data. Thus, in the present study, the original dataset was comprised of all study variables as well as the demographic variables that were identified in attrition and missing values analyses as correlates of missingness.

Table 2 presents the descriptive statistics for the variables based on the pooled results. On average, children were perceived as having "very good" health (4 on a 5-point scale). They had intermediate scores on the assessment of behavioural inhibition ("neither true nor false" or 5 on a 7-point scale) and were assessed as low in internalizing problems ("not true of my child" or 0 on a 3-point scale). On average, maternal negative parenting was quite low, reportedly occurring less than "once in a while" (2 on a 5-point scale). Family socioeconomic stress ranged from quite low to quite high.

Table 2

Descriptive Statistics

Measure	*M*(*SD*)	Min	Max	Skewness	Kurtosis
Child global health					
Time 1 health	4.10 (.59)	2.00	5.00	-.81	.69
Time 2 health	4.16 (.61)	2.17	5.00	-.99	.86
Time 3 health	4.24 (.57)	2.33	5.00	-.91	.42
Time 1 Family socioeconomic stress (z)	.01 (.76)	-1.98	2.42	-.23	.06
Time 1 Child behavioural inhibition	3.73 (.68)	1.78	5.40	-.05	-.25
Time 1 Maternal negative parenting	1.78 (.39)	1.00	3.25	.98	1.5
Time 1 Child internalizing problems					
Mother assessment	.29 (.20)	.00	1.08	1.0	1.1
Father assessment	.28 (.23)	.00	1.33	1.5	3.1

Prediction of Child Global Health From Early Childhood Psychosocial Stressors

To test the hypothesis that children with greater exposure to multiple sources of psychosocial stress in early childhood would show poorer health in middle childhood, the effects of singular and combined exposure to psychosocial stressors in early childhood on children's health in early and middle elementary school were examined by means of two hierarchical regression analyses. In hierarchical regression, predictors are entered into the regression equation in specified steps so that higher-order effects can be evaluated after lower-order effects are accounted for and each effect can be assessed in terms of what it adds to the equation at its point of entry as well as uniquely after all other effects are taken into account.

To examine the singular and combined predictive effects of the psychosocial stressors on health, children's global health at each of two time points, early elementary school age (Time 2, age 5-7) and middle elementary school age (Time 3, age 7-9), was predicted from the main and interactive effects of family socioeconomic stress, maternal negative parenting, child behavioural inhibition, and child internalizing problems. Child global health at Time 1 was entered in the first step as a covariate so that the effects of the psychosocial stressors on later child health could be assessed over and above the child's initial health status. This was followed in the second step by the main effects of family socioeconomic stress, maternal negative parenting, behavioural inhibition, and internalizing problems (using mother assessment then father assessment in separate analyses), in the third step by the two-way interactions, in the fourth step by the three-way interactions, and in the fifth step by the four-way interaction. This order of entry permitted

assessment of whether the addition of interactions would improve the prediction of child health beyond that afforded by the singular effects of the psychosocial stressors.

The cases-to-variables ratio was 15:1. Given the conservative rule-of-thumb guideline for adequate power of 15 cases per effect, the power was considered to be sufficient for identifying moderate effects. Multiple regression assumes the variables are normally distributed, linearly related to the criterion variable, and similarly variable around the criterion variable (homoscedasticity). To determine whether the data met these assumptions, residual scatterplots were analyzed in the original data prior to data imputation. Results of these analysis did not reveal violation of assumptions of normality, linearity, and homoscedasticity.

Multiple imputation was performed using SPSS 19. Following the recommendation of Aiken and West (1991), the independent variables were centered in order to reduce problems of multicollinearity and provide more meaningful interpretation of the solutions when including interactions of independent variables in the prediction equation. In multiple imputation, centering is performed following imputation. Prior to imputation, the interaction terms were included in the dataset in the form of raw (uncentered) products of the main effects. Following imputation, the main effects and interaction terms were centered separately in each data file using procedures described by Enders (personal communication, January, 2011). The data analysis was then conducted on each of the imputed data sets and the resulting parameter estimates were pooled through averaging into a single set of results.

Before performing the regression analyses, correlations among the variables were examined. Pooled zero-order correlations are shown in Table 3. Correlations among the

measures of child health at the three time points were substantial, lowest $r(250) = .52$, $p < .001$, showing moderate stability across time. With regard to the relations between stressors and child health, poorer global health at Time 1 was associated with greater behavioural inhibition, $r\ (250) = -.13$, $p = .04$, and more internalizing problems as assessed by mothers, $r(250) = -.14$, $p = .03$, and fathers, $r(252) = -.17$, $p = .01$. At Time 2, poorer global health was associated with more internalizing problems as assessed by fathers, $r(252) = -.17$, $p = .02$ but not mothers. There were no significant associations between stressors and child health at Time 3. Contrary to expectation, family socioeconomic stress and mother's negative parenting were not significantly associated with child health at either Time 1, 2, or 3. There was some evidence that psychosocial stressors were correlated, with maternal negative parenting being weakly related to family socioeconomic stress, $r\ (252) = .14$, $p = .04$. Children higher in behavioural inhibition had more internalizing problems as assessed by mothers and fathers, $r(252) = .19$ and .23, respectively, ps $< .001$. The pattern of associations is somewhat consistent with the hypothesis that greater psychosocial stressors would be associated with child health. Specifically, while more distal factors (socioeconomic stress and negative parenting) were not associated with health in the current sample, more proximal child factors showed some association with child health at Time 1 and Time 2.

Table 3

Zero-order Correlations Among Variables (N=252).

Variables	1	2	3	4	5	6	7	8
1. T1 Child global health	...							
2. T2 Child global health	.59**	…						
3. T3 Child global health	.52**	.62**	…					
4. T1 Family socioeconomic stress	-.08	-.04	-.11	…				
5. T1 Child behavioural inhibition	-.13*	-.12	-.10	-.00	…			
6. T1 Maternal negative parenting	-.02	-.04	.-03	.14*	-.06	…		
7. T1 Child internalizing - Mother	-.14*	-.10	-.06	.12	.19**	.28**	…	
8. T1 Child internalizing - Father	-.17*	-.17*	-.05	.16	.23*	.06	.18*	…

* $p < .05$; ** $p < .01$.

Prediction of child global health at Time 2. The results of the analysis predicting children's global health at Time 2 are presented in Table 4. The table shows the pooled unstandardized regression coefficients (B) for the relation of the predictors to child global health at Time 2, the standard errors of B (*SE* B), the standardized regression coefficients (β), and the squared semi-partial correlations (pr^2) indicating the unique contribution of each predictor to the total variance of the criterion variable at a given point of entry. Also shown are the increments in percent of variance accounted for by each step in the equation (ΔR^2), and the *F* test of the increment (ΔF). The addition of the three- and four-way interactions in steps four and five did not significantly improve the prediction of child health. Instead, a reduced model including only the covariate, main effects, and two-way interactions appeared to be the best fit and is the model shown in Table 4. In step 1, child health at Time 1 contributed significantly to the prediction of child health at Time 2, B = .65, β = .61, $p < .001$ (pr^2 = .38 indicating 38% of unique variance explained). Over and above the contribution of the child's health at Time 1, there was also a significant two-way interaction between behavioural inhibition and child internalizing problems, B = -.70, β = -.15, $p < .05$ (pr^2 = .02). To examine the interaction, simple slope tests were conducted following Aiken and West (1991) by performing regressions estimating the effect of children's behavioural inhibition on child health at Time 2 at 1 *SD* above (high) and 1 *SD* below (low) the mean on children's internalizing problems. The results are shown in Figure 1. As the figure shows, higher behavioural inhibition predicted poorer health at Time 2 for children high in internalizing problems, β = -.23, p = .03, but not for those low in internalizing problems, β = .05, p = .72. In summary, over and above the child's initial health status, the psychosocial stressors examined in the present study accounted for

approximately 2% of the variance in child health at Time 2, attributable predominantly to chronic psychological stress associated with high behavioural inhibition and internalizing problems. The child's initial health status itself accounted for fully 38% of the variance. A separate analysis performed with child internalizing problems assessed by father rather than by mother showed no significant effects, over and above Time 1 health, in the prediction of Time 2 health.

Prediction of child global health at Time 3. In the next regression, children's global health at Time 3 was predicted from global health at Time 1 and the main and interactive effects of family socioeconomic stress, maternal negative parenting, behavioural inhibition, and internalizing problems. Time 1 health was the only significant predictor of Time 3 health, $B = .55$, $\beta = .56$, $p < .001$ ($pr^2 = .25$ indicating 25% of unique variance explained). A separate analysis performed with child internalizing problems assessed by father rather than by mother showed no significant effects, over and above Time 1 health, in the prediction of Time 3 health.

Table 4

Summary of Hierarchical Regression Analysis Predicting T2 Child Health (N = 252).

Variables	ΔR^2	ΔF	*B*	SE *B*	*B*	pr^2
Step 1	.38	125.1***				
T1 Child health			.65	.06	.61***	.38
Step 2	.003	.230				
T1 Child health			.64	.06	.61***	.35
Family socioeconomic stress (F)			-.01	.05	-.01	.00
Behavioural inhibition (B)			-.03	.06	-.03	.00
Maternal negative parenting (A)			-.03	.10	-.02	.00
Child internalizing (I)			-.07	.18	-.02	.00
Step 3	.040	1.22				
T1 Child health			.63	.06	.60***	.34
Family socioeconomic stress (F)			-.03	.05	-.04	.00
Behavioural inhibition (B)			-.02	.06	-.03	.00

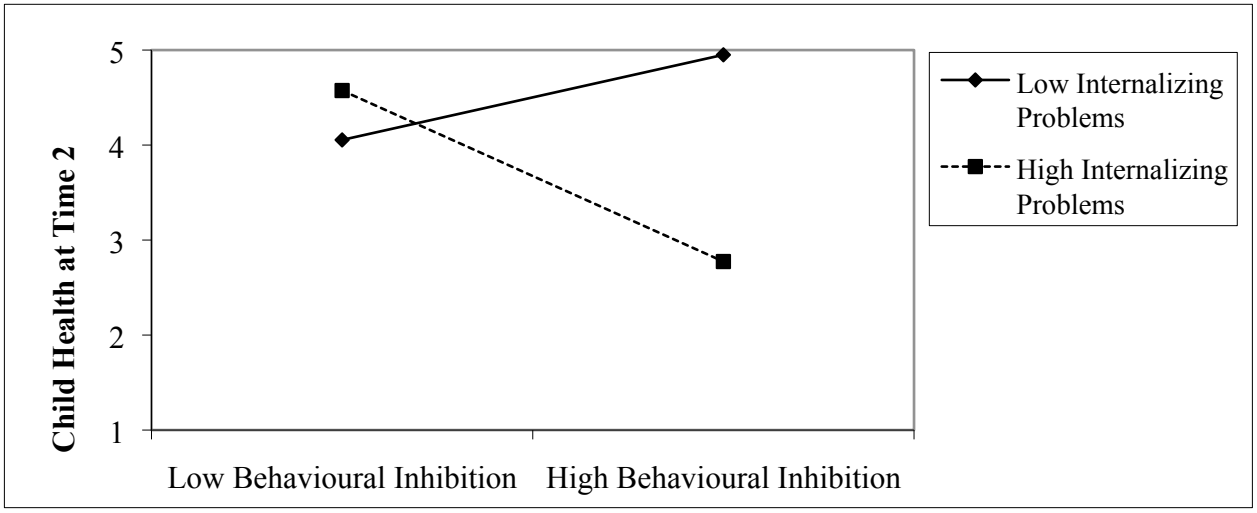
Maternal negative parenting (A)	-.00	.10	-.00	.00
Child internalizing (I)	-.03	.20	-.01	.00
F x B	-.03	.08	-.02	.00
F x A	-.06	.13	-.03	.00
B x A	.16	.16	.06	.00
F x I	.04	.27	.01	.00
B x I	-.69	.27	-.15*	.02
A x I	-.10	.52	.01	.00

Note. total R^2 = .40, F (4, 251) = 13.03, p = .000 for the full equation.

$p < .05$; ** $p < .01$; *** $p < .001$.

Figure 1

Interaction Between Behavioural Inhibition and Child Internalizing Problems



Discussion

Using a bioecological framework, which examines how biological, psychological, and social factors interact to affect outcomes, the present study examined whether exposure to multiple psychosocial risk factors in early childhood is associated with physical health effects in middle childhood. Family socioeconomic stress, behavioural inhibition, exposure to negative parenting, and internalizing problems are key sources of psychosocial stress that impact emotional and physiological processes associated with physical health. The current study extends the literature on developmental models of psychosocial stress and health by examining the effects of these multiple psychosocial stressors in childhood using a prospective longitudinal design and controlling for initial child health status. Based on bioecological models of health suggesting that risk to health in childhood is likely to increase as a result of the cumulative dysregulating effects of exposure to multiple sources of stress, the purpose of the present study was to examine the singular and combined effects of multiple individual and family level psychosocial stressors on mother's report of children's general health from early to middle childhood. Results of hierarchical regression indicated child health at Time 1 and the interaction between children's behavioural inhibition and internalizing problems as assessed by mothers were significant predictors of child health at Time 2. None of the stressors examined predicted a significant amount of variance in child health at Time 3 over and above the effects of Time 1 health.

The findings provide some support for the hypothesis that the combined effects of multiple risk exposure are greater than that of singular risk exposure. Bioecological models suggest that the combined effects of exposure to multiple psychosocial stressors on

health accumulate to impact health over time. Consistent with this view, while the main effects alone did not predict Time 2 health, the interaction between behavioural inhibition and child internalizing problems was a significant predictor of child health at Time 2. Higher behavioural inhibition predicted poorer health for children with greater levels of internalizing problems but not for those with lower levels of internalizing problems. These findings are consistent with bioecological theories suggesting that the impact of exposure to stress on health may not be the same for all children (Boyce & Ellis, 2005; Ellis et al., 2005; Taylor, 2010). Research suggests that high behavioural inhibition, reflecting greater reactivity to novelty or challenge, contributes to increased stress reactivity (Kagan, 1992; Kagan et al., 1987), particularly in high stress environments (Boyce et al., 1995). The findings of the current study extend past research and suggest that psychological vulnerabilities associated with internalizing problems may be another *context* in which high reactivity to stress is particularly detrimental. Cognitive and information processing biases that increase threat appraisals may create a context where even neutral situations are more likely to be perceived as threatening. When combined with increased physiological reactivity to stress, the cognitive and emotional processes associated with internalizing problems may increase vulnerability to allostatic load and health consequences. While the finding of a significant contribution of these individual difference characteristics supports theory indicating that these are important factors in child health and extends previous research by showing that they predict health over and above initial health status, it is important to consider the possibility that the significance of the sole interaction between behavioural inhibition and child internalizing problems in predicting health is due to a shared heritable dimension between these variables.

Further, it is noteworthy that, of the stressors investigated in this study, it was the proximal child factors that were predictive of child health. Although the contribution of variance explained by the significant interaction was small, it has been noted that even minor contributions to variance in health may represent meaningful effects given the many possible psychosocial mediators that play a role in health outcomes (Matthews, Gallo, & Taylor 2010). The majority of past research has examined the impact of more distal contextual stressors on child health and these findings add to existing research by finding effects of proximal child factors in a relatively low-risk sample. In studies where stress attributable to more distal child factors is greater (i.e., greater socioeconomic stress and more negative parenting), the presence of increased stress associated with more proximal child factors may be particularly significant for health.

Another characteristic of the child that significantly predicted health was the child's previous health status. The child's perceived health in early childhood was a singular and highly significant predictor of health in middle childhood, accounting for more than one-third of the variance in health in middle childhood. Although this finding may partly reflect the use of a single informant, it suggests that health, as reported by mothers, is moderately stable across childhood and is consistent with lifespan and critical period models that emphasize the lifelong health implications of early exposure to risk factors. Not only do early experiences play a role in programming stress regulatory systems in ways that increase vulnerability to health problems, (Ben-Shlomo & Kuh, 2002; Hertzman, 1999; Hertzman & Boyce, 2010; Lupien et al., 2009), but the experience of illness early in life has a lasting impact on health throughout the lifespan. This is believed to occur both directly through the illness itself and indirectly by restricting social

and developmental opportunities that increase other risk factors for health (Kuh & Wadsworth, 1993; Wadsworth & Kuh, 1997; Miller et al., 2011). Despite theory and evidence suggesting that early life experiences set into play a chain of physiological and social factors that influence health into adulthood, the majority of research has failed to fully account for early life health. The findings of the current research suggest controlling for child health at study outset is necessary to ensure the impact of psychosocial stressors on health in samples of healthy children are not overestimated and so that any changes in health can be attributed to the effect of psychosocial stressors examined.

The psychosocial stressors assessed in the current study predicted health two years later at Time 2 but not four years later at Time 3. There are several possible explanations for this finding that warrant further research attention. It is possible that changes in exposure to stressors over time influence health outcomes. In the current study, stressors were assessed at only one time point (Time 1) and it is possible that stressors present at one time may not persist over time, resulting in reduction in stress exposure. The theory of allostatic load (McEwen, 1998) suggests that it is *chronic* stress that affects health through the cumulative strain of repeated activation of physiological systems over time. According to Taylor's bioecological life-course model (Taylor, 2010), it is the cumulative effects of dysregulation in one domain that increases the likelihood of dysregulation in another domain and results in health effects over time. Although the current study provides some initial support for bioecological models by showing the combined effects of stress exposure on health in the short-term, further research examining the *accumulation* of stressors over time is needed. Future longitudinal research is needed that repeatedly

assesses multiple exposures to psychosocial stressors over time in order to understand the ways in which these exposures accumulate and interact to impact health.

It is also possible that the importance of particular risk factors changes over time, with psychosocial stressors influencing health in early childhood differing from those in middle childhood. In the current study, individual differences in children's temperament and internalizing problems were predictive of health in mid but not late childhood. It is possible that as children grow older, the emergence of cognitive resources allow for greater evaluation of threat or that other protective factors emerge with exposure to school and other contexts that alter or reduce the importance of individual difference variables. Indeed, there is evidence that health effects of psychosocial stress may not be the same for all physical health problems or stages of development (Chen et al., 2002; Chen, Martin, & Matthews, 2006; Marin et al., 2008). In a review of the literature on SES and child health, Chen and colleagues (Chen et al., 2002) concluded that, among children from lower SES families, the most common risk factors for mortality and chronic illness varied according to age. Young children from lower SES families were at greater risk for injury, asthma (prevalence and incidence), and elevated blood pressure. The risk of severe asthma was maintained for adolescents from lower SES families, but the risk for injury, asthma prevalence, and elevated blood pressure was no longer present in adolescence. These findings suggest that the particular psychosocial stressors or processes explaining the relationship between psychosocial stress and health may vary as children undergo normal developmental changes (Chen et al., 2002). For example, while peer relationships are a likely source of social stress (or support) worthy of research attention, the effects of peer relationships on health are likely to vary according to stage of development, having a

greater influence in adolescence. Further, there is evidence that negative emotions, such as hostility, may be important psychosocial mechanisms with implications for health, but the cognitive functions involved in these processes may not be developed until later in childhood or adolescence. In order to understand the developmental emergence and evolution of health problems associated with psychosocial stress, it will be important for longitudinal studies to examine a range of proximal and distal stressors across stages of development.

Two psychosocial stressors that were hypothesized to be important factors in child health were not found to be significant predictors of health either alone or in combination with other factors. Contrary to expectation, family socioeconomic status did not predict child health. The lack of a significant effect of family socioeconomic stress on health is inconsistent with a large body of previous research finding socioeconomic status to have a strong association with health throughout the lifespan (Adler et al., 1994; Bradley & Corwyn, 2002; Chen et al., 2002; Chen et al., 2006; Guralnik et al., 2006; Hertzman, 1999) and it may be related to the low levels of stress attributable to family socioeconomic status in the current sample. There is some evidence that the negative impact of family socioeconomic stress on health may not be evident until a certain level of disadvantage is exceeded (Nicholson, Lucas, Berthelsen, & Wake, 2010). In a recent study of the patterning of socioeconomic inequalities, five levels of socioeconomic disadvantage were examined in relation to children's health (Nicholson et al., 2010). Results revealed threshold effects of socioeconomic disadvantage on general health, with health effects only evident in the most disadvantaged group. Consequently, in view of the low levels of stress attributable to family socioeconomic status in the current sample, it is possible that

the threshold necessary for effects on health to be seen was not met. These findings also challenge the widely purported notion of gradient associations between socioeconomic disadvantage and health, which suggest that health decreases with each incremental increase in socioeconomic disadvantage and speak to need for further research into the developmental emergence and patterning of these associations. Much of the research on gradient effects has been limited by its failure to control for initial health status and the wide, composite age ranges across which the effects of SES have been examined (Chen et al., 2002; Chen et al., 2006). Further, much of the research examining gradient effects has used dichotomous measures of health. While dichotomous measures allow examination of whether a greater proportion of individuals in a poor health category are disadvantaged, it does not allow for determination of whether health *decreases* with increasing increments of socioeconomic disadvantage. Future research is needed to examine whether childhood health inequalities exist at all levels of socioeconomic disadvantage or emerge only after a certain threshold of disadvantage is exceeded. As patterns of socioeconomic status and health associations may differ according to the health outcome examined (Chen et al., 2002; Chen et al., 2006; Nicholson et al., 2010), gradient and threshold models should be examined in developmental studies using measures of both general health and specific health problems.

Contrary to expectation, negative parenting was not found to predict child health. It is has been argued that family environments characterized by conflict and aggression, a cold unaffectionate interaction style, or low nurturance contribute to long-term health consequences by disrupting psychological processes and physiological systems that have implications for health (Repetti et al., 2002; Taylor et al., 2004).While a large body of

research has found evidence linking early caregiving experiences to the reactivity and regulation of stress responding (Dettling et al., 2000; Gunnar & Donzolla, 2002; Gunnar et al., 1992; Meaney, 2001; Spangler et al., 1994; Tarullo & Gunnar, 2006; Watamura et al., 2004), the evidence linking parenting styles and practices to general health in children is less clear as research has been limited by retrospective study designs, failure to control for initial child health, collapsing across wide age ranges, and examination of parenting without considering the role of child characteristics. There is also little evidence on whether specific domains of parenting differ in the extent to which they impact health. Further, studies examining parenting in relation to children's general health have yielded only small effects (Belsky et al., 2007; Waylen et al., 2008), possibly as parenting has typically been assessed separately from other factors. In view of bioecological theories and evidence of the transactional nature of parenting in combination with child factors, it is possible that the impact of negative parenting will be greatest when combined with child characteristics that contribute to greater vulnerability to stress. Given the sample size and relatively low levels of negative parenting in the current study, the power to detect these cumulative or interactive effects may not have been sufficient. In future research, larger samples and greater heterogeneity in the quality of parenting may yield more information about the role of parenting in child health.

Limitations

Several limitations of the study should be noted. First, the reliance on mothers' assessments of both their child's characteristics and health raises concern about the validity of the results. It is possible that the significant associations that were found between child characteristics and child health were inflated by shared method variance due

to the use of a single source of information to measure these variables. The use of reports almost exclusively from mothers may have increased the degree of correspondence between child characteristics and child health. Somewhat mitigating this limitation, however, is that behavioural inhibition was assessed by both parents, indicating that the variance explained by this characteristic cannot be attributed entirely to shared method variance. Moreover, this finding is consistent with much previous research indicating that behavioural inhibition and internalizing problems are associated with enhanced physiological reactivity to stress, (Kagan, 1992; Kagan et al., 1987; Lewis & Ramsay, 2002; Nachmias et al., 1996; Schmidt et al.,1997) and health (Boyce et al., 1995; Chavira et al., 2008; Farrell et al., 2011; Katon et al., 2007; McQuaid et al., 2001; Slattery & Essex, 2010). Nevertheless, the present results may at least in part reflect shared method variance and are in need of replication using other types of measures such as observation and multiple informant reports of child characteristics, and more objective methods of children's physical health functioning.

The findings are also limited by the use of mothers' self reports of their parenting practices. Mothers may have been positively biased in their perceptions of their parenting behaviours and thus inclined to underestimate the frequency of their negative behaviours, limiting the extent to which negative parenting was related to child health. In future research, additional measures of parenting practices should be obtained through the use of other types of measures such as observation, spouse, or child report. Further, only parenting by mothers was assessed in the present study. Although research indicates that mothers have more impact on children's development than fathers in the preschool years (McBride & Mills, 1993; Yeung, Sandberg, David-Kean, & Hofferth, 2004), it is clear that

fathers do play an important role in children's development (Lamb, 2004) and there is some evidence that fathers' parenting may have implications for children's health (Bell & Belsky, 2007). From a cumulative risk perspective, it is possible that the presence of negative parenting by both mothers and fathers may provide an additional stressor, with implications for allostatic load and health problems over time. An important direction for future research will be to establish whether fathers' negative parenting contributes to increased risk of health problems in children and whether risk is increased further if children have both mothers and fathers who engage in more negative parenting.

The findings are also limited by reliance on mothers' report of children's health as the sole outcome measure. In addition to the problem of potential shared method variance associated with mothers' assessment of both child characteristics and health as noted above, the use of mother's report alone provides only an indirect assessment of child health. Reliance solely on this one outcome measure may have limited the likelihood that minor variations in health would be captured in this relatively healthy sample, particularly as variations in general health may be more difficult to capture than categorical measures of health. This may also have been reflected in the relatively modest reliability of this scale. While beyond the scope of the current study to use additional assessments of health, future research would be strengthened by using multiple measures of children's health functioning. Further, mothers' report of child health reflects mothers' subjective perception of illness severity and may be biased by their own mental or physical health functioning. Despite this potential bias, parent ratings of general health have been found to discriminate between subgroups of children with and without chronic health conditions (Raat et al., 2002; Raat et al., 2005) and children's level of health care utilization (Raat et

al., 2002). Further, there is some evidence that mothers' own psychological adjustment does not influence their report of their school-age children's health status (Dadds, Stein, & Silver, 1995). Nonetheless, it is possible that the sole significant interaction found in the current study reflects shared method variance and/or bias associated with reliance solely on mother's report and future research should replicate and extend the present findings using more objective health outcome measures, such as physician ratings, frequency and/or extent of health care utilization, physiological markers of health, and/or illness and disease morbidity.

Finally, the generalizability of the findings is limited by the relatively normative and high functioning nature of the sample. Families were, on average, relatively low in stress attributable to family socioeconomic status and negative parenting and children had few internalizing problems and very good health. The generally low levels of stress in the sample suggest there may have been selection bias, with higher functioning families more likely to volunteer to participate in the study. The generalizability was further limited by patterns of attrition in the sample. While attrition levels in the current sample were not greater than the anticipated rates of 10 to 20 percent per year in longitudinal research (St. Pierre, 1980), there was a greater loss of families with higher socioeconomic stress over time. It is possible that study demands contributed to increased attrition among lower SES families. Families were required to attend the University laboratory at each time point as part of the larger longitudinal study from which the data were drawn. This may have created an additional barrier to participation for lower SES families who may lack resources for travel or childcare or who are less familiar with the University setting. Families were also required to complete a lengthy series of questionnaires at each time

point. It is possible that the lower education levels of low SES families made the study questionnaires overwhelming or more onerous to complete. Given the large body of research linking high socioeconomic stress to poorer health (Adler et al., 1994; Bradley & Corwyn, 2002; Chen et al., 2002; Guralnik et al., 2006; Hertzman, 1999), the greater loss of families with higher levels of socioeconomic stress in the current sample likely further contributed to the disproportionately low levels of stress and good health in the sample and limits the generalizability of findings to samples of families with low levels of stress and children with good health. In future research, strategies should be employed to obtain and retain a more representative sample and minimize potential barriers to participation for families with fewer resources.

Summary and Potential Clinical Implications

Despite these limitations, the findings of the current study extend previous research by examining predictive relations among psychosocial stressors and child health using a longitudinal design and controlling for initial child health status. Much of the research to date has been correlational in nature and the longitudinal research conducted has frequently been based on retrospective reports of childhood health and failed to control for child health at study outset. The current study adds to the existing body of research by finding effects of psychosocial stressors over time after accounting for initial child health status and speaks to the importance of controlling for initial health status in longitudinal research in order to ensure that health effects can be attributed to the stressors assessed, rather than stability in health over time. Given that any individual psychosocial factor represents only one of many possible psychosocial mediators, the contribution of any specific factor is likely to be small, and even minor contributions to variance in health

explained may represent meaningful effects (Matthews, Gallo, & Taylor 2010). Consequently, it is noteworthy that associations between the psychosocial stressors and health in the current study were found in a low-risk sample of healthy children. Future research addressing associations between psychosocial stressors and health in larger samples and with greater heterogeneity in exposure to stress will help to clarify and expand the current findings.

While further research is needed to validate and extend the current findings, there are potential clinical implications of this research. The findings suggest that it is important to consider the role of psychosocial factors in strategies for health promotion and disease prevention. Although there is growing scientific consensus that psychosocial stressors experienced early in life affect health outcomes throughout the lifespan, current efforts in health promotion do not typically address the role of these factors. Rather, health promotion and disease prevention have typically focused on three strategies, including the provision of immunization, encouragement of health-promoting behaviours, and the reduction of health-threatening behaviours (e.g., smoking, alcohol consumption) in adults (Shonkoff et al., 2010). With increasing understanding of the role of social factors and psychological processes in health, it will be important for public policy to integrate such findings into intervention strategies for health promotion and prevention. The results of the current study suggest that strategies for moderating the effects of individual differences in stress reactivity and psychological processes may have important implications for intervention. Indeed, research examining the role of cognitive and attentional processes in stress responding and health provides interesting avenues for potential intervention strategies. In a series of studies examining the impact of individual cognitive and

emotional processes on the experience of stress, Dandeneau and colleagues found that modifying attentional processes related to social threat perception can impact the stress response (Dandeneau, Baldwin, Baccas, Sakellaropoulo, & Pruessner, 2007). Specifically, they found that, particularly among individuals with low self-esteem, selective attention towards negative social information was associated with increased cortisol reactivity and that a simple task involving repeatedly ignoring social threat and searching for cues of acceptance was effective in reducing the attentional bias (Dandeneau et al., 2007). In a recent study by Chen and colleagues (Chen et al., 2011), youth with an asthma diagnosis from low-socioeconomic status backgrounds had less asthma inflammation and impairment when they displayed higher levels of cognitive strategies for coping with stress. These findings highlight the impact of individual cognitive and emotional processes on stress responding and suggest that such intervention may have health implications for children identified as psychologically at risk. Further research on the role of these individual differences in child health is needed to provide evidence on which such interventions can be developed and implemented.

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Appendix A

Demographic Questionnaire

This questionnaire will take you about 5 minutes. Your answers to these questions will permit us to describe,
as a group, the families participating in this study.

1. What is the birthdate of **the child** who is participating in this study with you?

   ____/____/____ Check one: Girl?[ ]1 or Boy?[ ]2
   d m y

2. Is this child:

	Your biological child?	[ ]1	Your spouse's biological child?	[ ]1
	Your adopted child?	[ ]2	Your spouse's adopted child?	[ ]2
	Your stepchild?	[ ]3	Your spouse's stepchild?	[ ]3

3. Please state the age and sex of any other children you have:

Age (in years)	Sex
___	___
___	___
___	___

4.

What is the highest level in school or university you have completed (check one)?	What is the highest level your spouse has completed?
1st to 8th grade . . . . . .[ ]1	1st to 8th grade . . . .[ ]1
9th to 12th grade . . . . . .[ ]2	9th to 12th grade . . . . . .[ ]2
community college or some university [ ]3	community college or some univ.[ ]3
university graduate . . . . . [ ]4	university graduate . . . . . .[ ]4
graduate or professional school . . . . . .[ ]5	graduate or professional school .. [ ]5

5. Approximately how many hours do you work each week for pay (include home-based work, work outside of the home, hours self-employed)?

	Your spouse?:
Not working for pay . . . . [ ]1	Not working for pay . . . . .[ ]1
1 to 14 hours a week . . . .[ ]2	1 to 14 hours a week . . . .[ ]2
15 to 24 hours a week . . . [ ]3	15 to 24 hours a week . . . [ ]3
25 to 39 hours a week . . . [ ]4	25 to 39 hours a week . . . [ ]4
40 hours a week or more . [ ]5	40 hours a week or more .[ ]5

6. If employed, are you (check one): part-time? [ ]1 full-time? [ ]2
   If employed, is your spouse (check one): part-time? [ ]1 full-time? [ ]2

7. If employed, are you (check one): temporary/term [ ]1 permanent? [ ]2
   If employed, is your spouse (check one): temporary/term [ ]1 permanent? [ ]2

8. What is your present or most recent past occupation(s)? (Please be specific, e.g., "homemaker," "auto mechanic," "high school teacher"): _____

   What is your spouse's present or most recent past occupation(s)? (Please be specific, e.g., "homemaker," "auto mechanic," "high school teacher"): _____

9. Are you Canadian? If yes, check one: 1st generation: __ 2nd: __ 3rd or more: __

   How much do you feel you are a Canadian? (Please rate your feelings on a 10-point scale in which 1 = not at all and 10 = very much a Canadian): _____

   Is your spouse Canadian? If yes: 1st generation: __ 2nd: __ 3rd or more: __

10. To which ethnic or cultural group(s) did your ancestors belong? (check all that apply):

__ French	__ English	__ German	__ Scottish	__ Irish
__ Italian	__ Ukrainian	__ Metis	__ Jewish	__ Black
__ Chinese	__ Portuguese	__ South Asian	__ Polish	
__ North American Indian		__ Inuit/Eskimo	__ Dutch (Netherlands)	
__ Other (please specify: _____)				

To which ethnic or cultural group(s) did your spouse's ancestors belong? (check all that apply):

__ French	__ English	__ German	__ Scottish	__ Irish
__ Italian	__ Ukrainian	__ Metis	__ Jewish	__ Black
__ Chinese	__ Portuguese	__ South Asian	__ Polish	
__ North American Indian		__ Inuit/Eskimo	__ Dutch (Netherlands)	
__ Other (please specify: _____)				

11. What is your marital status? (check one):

Cohabiting	[ ]1	For how long? ____ yrs.
Married	[ ]2	For how long? ____ yrs.
Separated/divorced	[ ]3	For how long? ____ yrs.
Single	[ ]4	

12. How old are you? (check one):

Less than 20 years of age . . ....[	]1
20 to 29 years of age . . . . . [	]2
30 to 39 years of age . . . . . [	]3
40 to 49 years of age . . . . . [	]4

How old is your spouse? (check one):

Less than 20 years of age . . ….[	]1
20 to 29 years of age . . . . . [	]2
30 to 39 years of age . . . . . [	]3
40 to 49 years of age . . . . . [	]4

50 to 59 years of age . . . . . [ ]5	50 to 59 years of age . . . . . [ ]5
59 years of age or above . . . . . [ ]6	59 years of age or above . . . . . [ ]6

13. So that we can describe the group of families participating in this study, please indicate your **FAMILY** income for the past year (that is, total income before taxes of **ALL** members of the family residing in your household), by checking one of these income categories:

Under $10,000 . . . . . [ ]1
$10,001 to $20,000 . . . . . [ ]2
$20,001 to $30,000 . . . . . [ ]3
$30,001 to $40,000 . . . . . [ ]4
$40,001 to $60,000 . . . . . [ ]5
$60,001 to $75,000 . . . . . [ ]6
Over $75,000 . . . . . .[ ]7