INVESTIGATION OF FAMILIAL TRENDS IN ANOREXIA NERVOSA AND BULIMIA

by

CATHERINE ELLIOTT-HARPER

A Thesis

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Abstract

It has been noted in the literature that eating disorders, as well as other psychiatric disorders, tend to run in families. Although different patterns of familial tendency are predicted by both genetic and psychological theories, to date no comparison of predicted and actual patterns has been undertaken. In the present study, data concerning eating disorders as well as other psychological/psychiatric problems in the families of eating disorder patients were compared with the predicted patterns under different genetic and psychological models, as well as with patterns seen in families of a matched normal group. This was an attempt to answer the questions of whether families of eating disorder patients are at higher risk for any eating disorders, and if so, whether the actual pattern of inheritance was best fit by psychological or genetic models. The results indicated that, overall, individuals in eating disorder families were at greater risk for developing an eating disorder. More specifically, there were a greater number of female relatives affected, a greater prevalence of other psychiatric pathology, and a greater prevalence of parental preoccupation with weight, food and appearance among the anorexic and bulimic groups' relatives than the control groups' relatives. These results were interpreted using three models of inheritance; single gene, multifactorial, and psychological. The data obtained were best fit by the psychological model.

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Introduction

Anorexia nervosa and bulimia are life-threatening disorders, the diagnoses of which have increased dramatically over the past 20 years (Dally & Gomez, 1979; Bruch, 1981). A recent prevalence study estimates the occurrence of anorexia nervosa to be as high as 1/250 school-aged girls (Crisp, Palmer & Kalucy, 1976). The apparent recent increase in these disorders has led to a heightened interest in the topic of eating disorders in general, as is indicated by several recent symposiums (e.g., Toronto, Ontario, October, 1981; Swansea, Wales, September, 1984), and the publication of a new journal, "The International Journal of Eating Disorders."

Although there have been recent controversies concerning the diagnostic definition and etiology of anorexia nervosa and bulimia, the most widely accepted definitions to date are those that serve as the basis for the American Psychiatric Association's diagnostic criteria (DSM-III, American Psychiatric Association, 1980), which consists of;

A) ANOREXIA NERVOSA;

1) intense fear of becoming obese, which does not diminish as weight loss progresses,
2) disturbance of body image, e.g., claiming to "feel fat" even when emaciated,
3) weight loss of at least 25% of original body weight or, if under 18 years of age, weight loss from original body weight plus projected weight gain expected from growth charts may be combined to make the 25%,
4) refusal to maintain body weight over a minimal normal weight

for age and height,

5) no known physical illness that would account for the weight loss.

B) BULIMIA;

1) recurrent episodes of binge eating,

2) at least three of a) consumption of high-caloric, easily ingested food during a binge; b) inconspicuous eating during a binge; termination of such eating episodes by abdominal pain, sleep, social interruption, or self-induced vomiting; c) repeated attempts to lose weight by severely restrictive diets, self-induced vomiting, or use of cathartics or diuretics; d) frequent weight fluctuation greater than ten pounds due to alternating binges and fasts,

3) awareness that the eating pattern is abnormal and fear of not being able to stop voluntarily,

4) depressed mood and self-depreciating thoughts following eating binges,

5) these episodes are not due to anorexia nervosa or any known eating disorder.

Since the earliest descriptions of anorexia nervosa, there has been some confusion as to whether it exists as a distinct disorder or whether there are diagnostic subtypes. Bruch (1973), King (1963), Frazier (1965), and Thoma (1967) have emphasized the description of anorexia nervosa as a discrete psychological syndrome. As well, all have emphasized a distinction between primary and secondary forms of the disorder (secondary forms reflecting weight loss secondary to some

other psychiatric illness).

Recently, Beaumont, George and Smart (1976) differentiated primary anorexics into two groups by their method of weight loss. "Dieters" or "restrictors" lost weight through caloric restriction alone. "Purger-vomiters" lost weight primarily through vomiting and laxative use. The first group represented those who are generally called anorexics, while the second group represented those who are generally called bulimics, in the literature. As many authors have noted that bulimia is often seen in conjunction with anorexia nervosa (although typically after a period of time of "restricting" only), the question has been raised as to whether bulimia is an end stage of chronic anorexia nervosa, whether bulimia may be considered to be a diagnostic entity of its own, or whether bulimia may be a distinct subgroup of primary anorexia nervosa.

Two recent articles have supported the existence of bulimia as a distinct subgroup of anorexia nervosa. Garfinkel, Moldofsky and Garner (1980) examined 141 eating disorder patients (one group of anorexics, the other group experiencing anorexia with bulimia), and found several characteristics that the two groups shared in common. Examination of the family histories showed an overrepresentation of anorexia nervosa in siblings and an overrepresentation of multiple births. Both groups pursued a thin body, regardless of weight, but the bulimic group dealt with this pursuit in a more inconsistent fashion (i.e., phases of starvation giving way to bouts of binging). The groups differed with respect to premorbid weight, impulse-related problems and maternal obesity associated with bulimic patients. They

suggested that, "These characteristics define a different group of women who are predisposed to develop the bulimic type of anorexia nervosa," (p. 1039), and that the clinical characteristics of their bulimic patients warrant the subcategorization of primary anorexia nervosa.

Casper, Eckert, Halmi, Goldberg and Davis (1980) conducted a study to determine the proportion of anorexia nervosa patients with bulimia, and to characterize this patient population to determine whether the symptoms of bulimia justified a distinction from restricting anorexics. They also concluded that bulimic patients had several personality and psychiatric characteristics which, while differentiating this group from "fasting-only" patients, supported their inclusion into a subgroup of anorexia nervosa patients.

An increasing number of diagnosed cases of anorexia nervosa and bulimia have been found in families with other psychiatric disorders. These disorders include alcoholism, studied by Halmi and Loney (1973) when they examined familial alcoholism in anorexia nervosa and found that, "... the frequency of alcoholism in the mothers ... is at least twice as high as expected, and ... in the fathers more than three times as high." (p. 53). Hall (1978) examined the family structures and relationships of 50 female anorexia nervosa patients and found that 19 of the parents had been diagnosed at some time with one of the following psychiatric illnesses; depression, alcoholism, schizophrenia, anorexia nervosa, or manic depression. Cantwell, Sturzenberger, Burroughs, Salkin and Green (1977) discovered a high percentage of affective disorder in their anorexic patients during a follow-up study. When

parents' and patients' reports were combined, the authors stated that 50% of their patients reported a clinical psychiatric diagnosis of affective disorder, and 56% to 67% of their patients reported the depressive symptom of dysphoric mood, at follow-up. They also noted that a family history of affective disorder was particularly common in the mothers of these patients. Fifty-eight percent (58%) of the mothers reported having a history of some type of affective disorder. Winokur, March and Mendels (1980) specifically examined the incidence of primary affective disorder in relatives of patients with anorexia nervosa, and in the families of anorexic patients. Their results indicated that a significantly greater number of the relatives in the anorexic group studied had histories of primary affective disorder, compared to the relatives of controls (22% compared to 10%). Also, in the anorexic group, of those relatives with primary affective disorder, 30% were female while only 13% were male. This difference was also statistically significant. When examining the incidence of primary affective disorder in the families of anorexics and controls, these authors found that 76% of anorexic families included at least one relative with primary affective disorder, compared to control families (48%). This difference was statistically significant as well. Crisp (1980), in his recent book, has documented the occurrence and influence of parental anorexia nervosa, depression, neuroses and affective disorders. He stated that, "... factors affecting the outcome ..." of anorexic patients such as, "sustained anxiety, neurotic avoidence patterns and vulnerability to depression in the parents, confer a worse outlook on the anorexic," (p. 156). Hudson, Pope, Jonas and Yurgelun-Todd (1983) reported on

420 first degree (1^{o}) relatives of 14 patients with anorexia, bulimia or both, who were evaluated for other psychiatric pathology using DSM-III criteria, by the family history method. They found that the morbid risk for affective disorders among relatives in families of eating disorder patients (28%) was significantly greater than that found in families of patients with schizophrenia (3%) or borderline personality disorders (3%). The eating disorder subgroup also differed significantly from the reference groups with schizophrenia and borderline personality disorder in the percentage of probands with a positive family history. Therefore, these authors concluded that, "... the prevalence of familial affective disorder was significantly greater in patients with anorexia nervosa and/or bulimia than in patients with schizophrenia or borderline personality disorder, but was similar to that found in patients with bipolar disorder."

These results are in agreement with Cantwell et al. (1977), Winokur et al. (1980) and also Gershon, Hamovit, Schreiber, Dibble, Kaye, Nurnberger, Anderson and Ebert (1982) who reported that family studies have found a higher than expected prevalence of affective disorders in the relatives of patients with anorexia nervosa.

Pyle, Mitchell and Eckert (1981) found 16 of 33 non-adopted bulimics reported depression in at least one 1^{o} relative. These results are in accordance with a study by Hudson, Laffer and Pope (1982) who reported 6 of 10 bulimics with the same type familial psychiatric history. Strober, Salkin, Burroughs and Morrell (1982) examined the prevalence of other psychiatric pathology in 1^{o} and second degree (2^{o}) relatives of bulimics and anorexics. They found a

significantly higher prevalence of affective disorders in the bulimic group than in the general population. Ten percent (10%) of combined 1^{o} and 2^{o} relatives had diagnosed cases of affective disorder. These authors state that this raw prevalence equates to a morbid risk of 15%, which is more than two times the average expected lifetime risk for affective disorders in the general population. As well, rates of familial alcoholism and drug abuse were higher in the bulimic group. These authors stated that a positive family history for alcoholism characterized 83% of bulimics and 49% of restrictors. They also found that drug use disorders, although less prevalent than either affective disorder or alcoholism, occurred significantly more often among bulimic relatives than anorexic relatives (7% compared to 3%), when the data was pooled across all relatives.

This tendency for several of these psychiatric disorders to "run in families" has been well documented in the literature, thus suggesting the possibility of genetic influences or common environmental factors involved in these psychiatric conditions. Examples of studies investigating the possibility of inherited factors of these disorders include genetic or family studies of: unipolar depression (Ashby & Crowe, 1978), bipolar manic-depressive psychosis (Angst et al., 1980; Baker, Dorzab, Winokur & Cadoret, 1972; Goetzl, Green, Whybrow & Jackson, 1974), schizophrenia (Essenmoller, 1977), affective disorder (Gershon, Mark, Cohen, Belizon, Baron & Knobe, 1975; Greenhill & Shopsin, 1980; Cantwell et al., 1977; Gershon et al., 1982; Halmi, 1983; Hudson et al., 1982, 1983; Pyle et al., 1981; Strober et al., 1982; Winokur et al., 1980), anxiety neurosis (Noyes, Clancy, Crowe, Hoenk & Slymen, 1978; Pauls, Noyes & Crowe, 1979), alcoholism (Goodwin, 1979a, 1979b; Penick,

Read, Crowley & Powell, 1978; Propping, 1978; Spalt, 1979), obsessive-compulsive neurosis (Welner, Reich, Robins, Fishman & Van Doren, 1976), and panic disorders (Pauls et al., 1980).

Eating disorders per se also have their own bodies of theoretical literature. The genetically based theories and the psychologically based theories have both focused on familial influences in the etiology of these disorders, although from different points of view. As the following studies indicate, the genetically based approaches have focused on the possible heritability of eating disorders. Dowson (1977) and Askevold and Heiberg (1979) reported on cases of anorexia nervosa in twins, both male and female pairs. The Dowson article discussed a male twin pair plus one sister with this diagnosis. The Askevold and Heiberg paper reported on two cases in discordant monozygotic twins. These authors also summarized the current data on all reported monozygotic twins with anorexia nervosa. They reported that about 1/3 of monozygotic pairs are in fact concordant for this disorder. Moskowitz, Belar and Dingus (1982) reported on one set of female twins who were concordant for anorexia nervosa. Their report concluded that, "... the anorexia was a final common pathway for a variety of psychodynamic patterns," and that the "... overlap of anorexia dynamics with those of adolescent twins would psychologically predispose twins toward concordance for anorexia nervosa," (p. 485). And, in a recent review of the previous twin data, Nowlin (1983) reported that sisters of affected females are at greater risk for developing an eating disorder. Possible explanations mentioned for this include;

- genetic etiology such as, "homozygosity for a very rare

recessive gene, or polygenic inheritance" (p. 103). However, if conclusions are to be drawn about the heritability of this illness from twin studies, two necessary requirements are; to have reliable diagnostic criteria for the disorder, as well as having the zygosity of each twin pair definitively established. Nowlin (1983) reports that many of the case studies listed in her article fail to meet one or both of these requirements.

The second possible etiology addresses the familial occurrence of anorexia nervosa and the terms;

- "induction" or "anorexia à deux" are used, where several authors have noticed parallels between anorexia nervosa and folie à deux (mimicking of sister's or mother's illness). This association between anorexia nervosa and another psychiatric problem is purely theoretical - no research has been done in this area to date.

In a more direct approach to genetic etiology, Wallinder and Mellbin (1977) had noted nine cases of anorexia nervosa described in the literature which were coincident with Turner's syndrome. In their study, they performed a chromosomal analysis on 30 women with anorexia nervosa, to determine if any abnormal chromosomal constitution existed. All of the women examined in this study had a normal chromosomal constitution.

The psychologically based theories have focused on the family interactions and on the mother-child relationship (Bruch, 1973, 1980; Rampling, 1980; Sperling, 1978; Story, 1976; Vigursky, 1977). Kalucy, Crisp and Harding (1977) have suggested that, "there are a number of

factors within the family system of patients with anorexia nervosa which predispose to greater than average problems in coping with the adolescent phase of development ... and which help to determine anorexia nervosa as the choice of adaptation style," (p. 382). With respect to the more analytic-psychological approach, the parents have been described as overprotective, overconcerned and overambitious, and they have (implicitly) expected obedience and superior performance from their children. This has been, in fact, achieved through over-compliance on the part of the child. With the approach of adolescence, the child has begun to make justified claims for independence. This has been unacceptable to these overcontrolling parents and the illness has then manifested itself as an expression of this underlying and unspoken struggle.

The parents may also "use" these children. The mothers need these "perfect" children as proof of their own perfection; the fathers need to measure their own achievements in terms of their children. Bruch (1973, 1980) has suggested that parents of these children may be older. These older parents may have had to wait (or chosen to wait) until their careers were well established before having children at all. Thus, they may not take the ability to have children as much for granted as younger parents might, resulting in overprotection and overconcern with the children. These parents' careers may also be quite time-consuming and/or pressured, and as a result the child may experience an environment where the child's needs are often secondary to career needs. These older parents will also likely find it more difficult to adjust to the different demands of parenthood. Their lifestyles will already

have been established, and they may feel resentful and angry at the disruption of having the child to care for.

Parental attitudes have also been related to the child's failure to develop a sense of self-identity (Bruch, 1981). Two forms of behaviour may be differentiated from birth on; 1) responses that are initiated within the indiviudal, and 2) responses to stimuli from the external environment. With anorexia nervosa patients, research has indicated deficits in confirmation of child-initiated cues. Therefore, growth and development may not be conceived of as the child's accomplishments but as those of the parents.

The confusion in hunger awareness may also be related to the mother's contradictory or inappropriate responses to the child's early feeding demands - the child therefore fails to learn to differentiate between being hungry and other sources of discomfort. The result is a lack of awareness of bodily sensations, and a sense of lack of control over those sensations that the child does experience. These intrapsychic disturbances leave the children feeling that they are deprived of living their own life - that they are only under the influence of external controls (Bruch, 1980).

With respect to the more behavioural-psychological approach, Slade (1982) has proposed a "functional analysis" model of anorexia nervosa and bulimia. This model has attempted to explain the development of eating disorders in terms of a set of hypothesized antecedent events/variables and its maintenance/exacerbation in terms of both positive and negative reinforcers. More specifically, he suggested that in anorexia nervosa, the initial dieting behaviour may be triggered

by apparently innocuous psychosocial stimuli (e.g.) social and cultural pressures to be thinner, weight loss associated with social success, and media coverage of eating disorders. Parental preoccupation with weight, food and appearance may also be a triggering stimulus. The successful dieting is then reinforced by (e.g.) feelings of being in "control" of self and others, increased social acceptance and implicit and possibly explicit parental approval. In this model, bulimia may follow on from anorexia nervosa, as an attempted strategy to cope with the biological pressures to increase the food intake, while still maintaining whatever reinforcement the individual receives from successful dieting.

These papers have suggested that both genetic and/or psychological factors may be involved in the etiology of anorexia nervosa and bulimia as well as other psychiatric disorders. However, the results and thus conclusions of the studies based on these theories are questionable due to methodological inadequacies and conceptual limitations. Several papers must be categorized as anecdotal reports based on clinical observations, and are therefore subject to the usual methodological criticisms of this type of study (viz. small samples, lack of controls, interviewer bias, etc.). Also, several of these papers have used arbitrary, idiosyncratic or unspecified diagnostic criteria when choosing subjects for their studies, and this may allow for inappropriate subjects to be included in their samples. The methods used in these previous studies have not exhausted all of the familial hypotheses possible as well.

Regarding the studies based on genetic theory, the twin intrapair

comparative method has been used for many years as a unique and valuable tool for studying genetic traits. This is due to the opportunity it provides for examining the interactions between genetic and environmental influences in the development of many disorders. The use of twin methodology has been based on two major assumptions;

1) that the zygosity of the twins has been correctly determined, and
2) that the environmental influence is the same for each twin, each type of twinship, and for twins and singletons.

Dibble, Cohen and Grawe (1978) examined these assumptions. Until 1955 methods of assignment of zygosity were inconsistent and unreliable. Siemen's (1927) "four levels of similarity" method, used after 1955, was criticized for subjective bias in rating of characteristics. Currently, tissue typing and blood factor analysis have proven to be the most reliable methods for determining zygosity. "However, ... there are often insurmountable constraints in obtaining blood specimens, or performing the more elegant tissue transplanting procedures." (p. 245).

Even the most recent instrument used (Dibble et al., 1978) for determining zygosity (a ten question form completed by parents) only validates and reliably discriminates monozygotic from dizygotic twinships, "... with an error rate ... approaching that of blood typing." (p. 246). Their results indicated that there were substantive differences in the environmental experiences of monozygotic and dizygotic twinships, and between twins and single births. On items in their questionnaire concerning confusion, they found that, "... mothers

rated 78% of MZ and 10% of DZ twins as experiencing confusion by mother and father, and 99% of MZ and 16% of DZ twins as experiencing confusion by strangers. The impact of such repeated confusion on individual twinships, or the effect of these differences between MZ and DZ twins is not known with certainty. However, such information must cast doubt upon the assumption of environmental equivalence," (p. 248). They also state that several authors have noted that twin populations appear to be more vulnerable to mental deficiency than singletons and that this vulnerability must be taken into account when generalizing from twin data to singleton data.

As well, these authors have addressed the effects of environmental influences before and shortly after birth, which may have later impact on development and which may be relevant to the definition of environmental equivalence for twins and singletons. Using their newly devised questionnaire, they had found that there were definitive differences noted between the environmental experiences of twins and singletons on the pregnancy, delivery and first month of life variables. Therefore, these authors concluded that the environmental differences experienced by twins and singletons must be accounted for in some way when investigating the mutual contributions of genetics and environment in psychological and developmental research.

Regarding the studies based on psychological theory, many of these are also anecdotal reports based on clinical observations, and are thus subject to the same criticisms mentioned above. Many of the theoretical notions inherent in current psychological theory have developed gradually from contact with patients seen in extensive

psychotherapy, and are therefore considered to be "soft" data. Also, reconstructing and defining the underlying psychological issues has proven to be additionally difficult, due to the extensive effect that starvation has on psychological and physiological functioning.

Taken together, these two bodies of theoretical literature leave us with a question: are the familial tendencies observed in eating disorders due to genetic factors, psychological factors, or both? For both genetic and psychological theories, familial patterns of inheritance may be predicted, based on the assumptions inherent in each theory.

Method

Predicted Genetic Models

Genetic theories include chromosomal, single gene and multifactorial modes of inheritance.

Chromosomal Inheritance

This includes both structural and/or numerical changes in the chromosomes, resulting in an alteration in the genetic makeup of the individual. Changes in the structure of the chromosome may be due to balanced or unbalanced translocations, deletions, insertions, duplications, rings or isochromosomes or mutations causing chromosome breakage. Structural alterations in the chromosomes may be detected through karyotyping and banding techniques. Numerical alterations of the chromosome may be due to meiotic or mitotic nondisjunction or translocations. Karyotyping and banding techniques are also used here for detection of numerical changes.

The question, of whether any chromosomal abberations are present

in those individuals developing eating disorders, would be very important in determining the etiology of anorexia nervosa and bulimia. The procedures necessary to answer this question are presently beyond the scope of this paper. However, if there were autosomal chromosomal abnormalities, we would expect the expression of the genes at the phenotypic level to include morphological abnormalities and mental retardation. Sex chromosome abnormalities would more likely be expressed as behavioural problems.

Single Gene Inheritance

Here, the distribution of phenotypes in the members of a family must follow a pattern that is predicted from the segregation and transmission of chromosomes through successive generations. The predicted outcomes are based on probabilities. There are four patterns of inheritance to be considered here; autosomal dominant, autosomal recessive, X-linked dominant and X-linked recessive. These Mendelian ratios are first examined to determine whether any pattern is evident, and then are tested using Segregation Analysis, by analyzing the family pedigrees to determine whether the pattern of inheritance observed in our sample fits the probabilities predicted from the theory. Factors altering these Mendelian ratios include; sex-related inheritance, sex-limited inheritance, pleiotropism, variable expressivity, lack of penetrance, phenocopies and lethality. The possible influence of each of these factors on this data is taken into account in the Discussion section.

Multifactorial Inheritance

This theory assumes that the additive effects of several genes at

different loci plus environmental influences determines whether an individual will reach a threshold level of liability during development or later in life after which the trait may appear. This theory is based upon Falconer's (1960, 1965) model, and assumes an underlying continuous normally distributed predisposition to a trait, with a liability derived from both genetic and environmental factors. If the individual's liability exceeds the threshold, they will express the trait. This model may be suggested by determining the population incidence of the eating disorders, and examining the incidence in second degree and third degree relatives for evidence of the predicted regression toward the population incidence. Caution must be taken with interpretation of the data using this model as well, due to the many assumptions inherent in this model (i.e., normal continuous distribution of liability in the population, liability derived from both genetic and environmental factors). The possible effects of these assumptions on this data will also be addressed in the Discussion section.

Predicted Psychological Theory

The expectations of the psychological theory are also tabled in Appendix A.

The psychological model may be tested by examining the answers to questions which are particularly relevant to the expectations of this model.

To date, this question, of whether familial tendencies observed in eating disorders are due to genetic factors, psychological factors, or both, has gone unanswered. It is the purpose of this thesis to examine this question, by outlining the possible modes of inheritance derived from both genetic and psychological theories, outlining the

expectations of each theory (see Appendix A) and then comparing observed data to these expectations.

Subjects

Group 1 was comprised of 15 female subjects, all of whom met the DSM-III criteria for anorexia nervosa. Group 2 was comprised of 20 female subjects, all of whom met the DSM-III criteria for bulimia. All of the subjects in each of these two groups had been patients in either Adult Psychiatry or Child Psychiatry at the Health Sciences Centre, Winnipeg, Manitoba. The control group (Group 3) was comprised of 20 female subjects. These subjects were recruited from the Psychology "pool", following the recommended procedure of the Department of Psychology and were matched with the other subjects for age, sex and average level of education. Approximately 75% of those contacted in each of the three groups agreed to participate. Since the compliance rate was fairly high, and approximately equal among the three groups, this argues against any response bias, and also supports the representativeness of each sample.

Procedure

The experimental subjects were asked, individually, to voluntarily participate in this research either by their therapist, or through a psychiatrist in Child Psychiatry. The researcher then contacted each patient by phone, explained the study and the procedure to them, and if they were still in agreement, arranged an individual interview with the patient and at least one parent in their home or in the researcher's office - whichever was most convenient for the patient. At the start of each interview, both subjects and parents were asked to sign consent forms (Appendices B and C). Then, a questionnaire (Appendix D) was completed and a three-generation pedigree was obtained from each

subject. The questionnaire used in this study was fairly extensive, with a total of 68 questions covering demographic variables of the subjects and the subjects' families, mothers' and fathers' psychiatric histories, their families' psychiatric histories, subjects' and families' occupations, parental involvement with feeding of the child, and any separations from the child. As well, questions were asked of the subjects' psychiatric histories, and perceptions of their home environment while growing up.

Group 3 subjects also volunteered to participate in this study. However, their participation was as partial fulfillment of their Introductory Psychology requirements, and therefore some potential biases must be addressed at this point.

1. Control subjects may be volunteering for this particular study because they know of a friend or relative with an eating disorder.

If this was the case, then the estimated incidence of eating disorders within this population would likely be higher than the estimated population incidence in the literature. Comparison of the two estimated population incidences in the Results section will determine whether there is any bias here or not.

2. The women volunteering as control subjects may have an eating disorder themselves, or may be concerned that they might have an eating disorder. Thus, they would be more likely to participate in a study of eating disorders, as they might gain more information about their own problem.

Each participant in this study also completed an Eating Attitude Test (EAT) (Garner & Garfinkel, 1979), (Appendix E). This scale has been shown to reliably discriminate those with eating disorders from

those without eating disorders. A score of 30 or greater indicates an eating attitude problem. The EAT scores for the Control group ranged from 4-23, with a mean of 12.79 and a standard deviation of 6.4, thus indicating that there were no Control subjects with an eating attitude problem. All subjects in both Experimental groups had EAT scores above 30.

The procedure for Group 3 was the same as that for Groups 1 and 2.

To determine the direction of specific differences between the groups, several comparisons were made for each variable under investigation. Therefore, Fisher's Exact Tests were performed on (e.g.) the data where the expected values of less than 5, and χ^2 tests were performed on the data while the expected values were greater than 5. This use of 2 x 2 tables resulted in the following comparisons being made for each variable;

a) Controls x Bulimics,

b) Controls x Anorexics,

c) Controls x both Bulimics and Anorexics, and

d) Bulimics x Anorexics.

The χ^2 value arising from each of these tables are no longer independent of each other, and this may result in an inflated significance level. Therefore, Brunden's (1977) correction has been applied to each set of comparisons. This results in a much more conservative level of significance, which has been applied to this data. Throughout the Results section, the uncorrected significance level reached will be indicated by an asterisk (*) and the significance level required by the corrected method will be indicated by a cross (†). Where all questions in the Questionnaire were answered, a discriminant analysis was performed

on the data. This analysis was done to determine which variables would be found to discriminate between the groups of Controls, and Anorexics and Bulimics combined. Also, to determine whether a pattern of responses could be found, which may be useful in predicting other (potential) individuals with eating disorders.

Results

Pedigrees

Tables 1, 2 and 3 show examples of pedigree diagrams from the Control group, Bulimic group and Anorexic group respectively. A pedigree diagram for each family in this study is included in Appendix F. As well, numerical representation of this family data is given in Appendix G. (For explanation of symbols, see p. 76.)

Table 1

Example of Control Pedigree

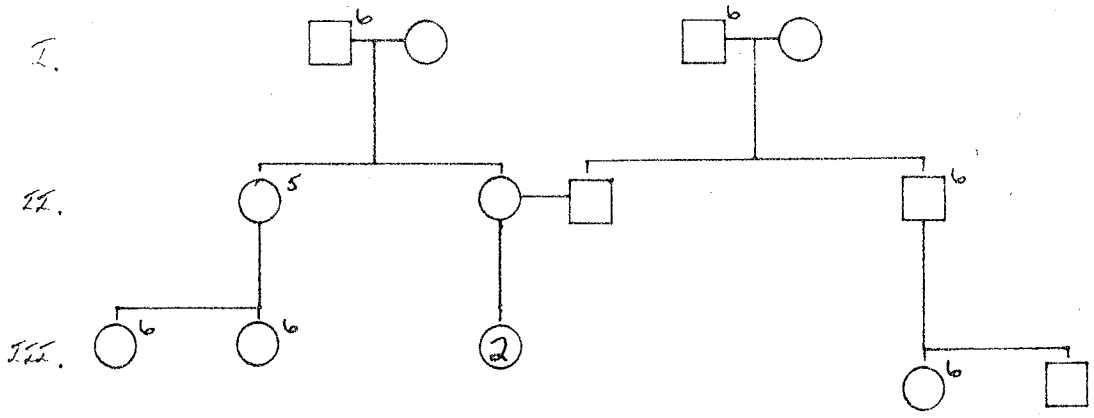


Table 2

Example of Bulimic Pedigree

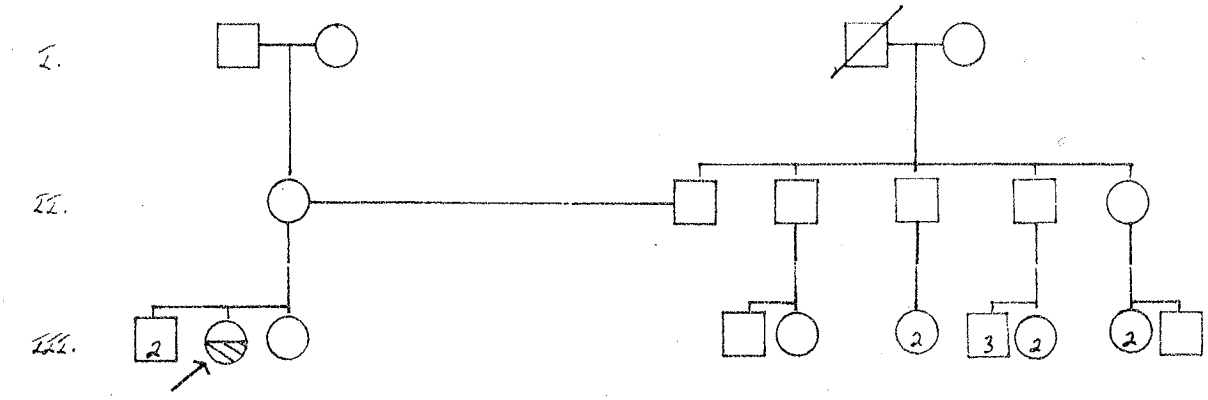
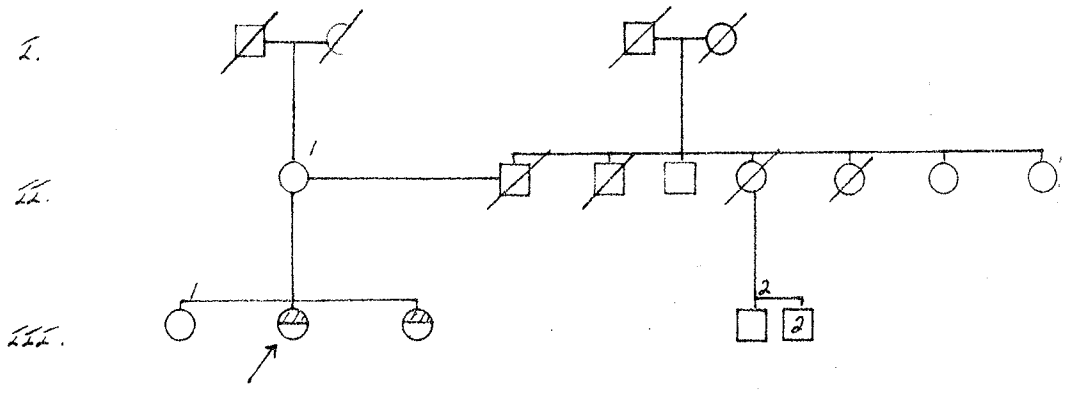


Table 3

Example of Anorexic Pedigree



Number and Percentage of Affected Males and Females

Table 4 indicates the number and percentage of affected males and affected females over the total number of males and total number of females in each group (excluding subjects). First, second and third degree relatives were combined in each group.

Table 4

Number & Percentage of Affected Individuals - 1^{o}, 2^{o} & 3^{o} Relatives Combined

Sex	Groups		
	Control	Bulimic	Anorexic
Males	0/297 (0.0%)	1/290 (0.3%)	0/252 (0.0%)
Females	1/260 (0.4%)	5/281 (1.8%)	4/276 (1.4%)

Fisher's Exact Tests revealed no significant differences between the three groups of males, the three groups of females, or the three groups (with males and females) combined.

Table 5 also indicates the number of affected males and affected females (over the total number of males and females in each group), but each group is broken down into 1^{o}, 2^{o} and 3^{o} relatives (excluding subjects).

Table 5

Number of Affected Individuals - 1^{o}, 2^{o} and 3^{o} Relatives

	1^{o}				2^{o}		3^{o}	
	Fathers	Mothers	Sisters	Brothers	Males	Females	Males	Females
Controls	0/20	0/20	0/19	0/22	0/118	0/102	0/137	1/119
Bulimics	0/20	0/20	2/21	0/25	0/98	0/99	1/147	3/141
Anorexics	0/15	0/15	1/20	0/15	0/87	3/86*†	0/135	0/155

* Sum p = .009; † p < .013

(For first degree relatives, mothers and sisters were combined, and fathers and brothers were combined.)

Fisher's Exact Tests revealed no significant differences between the groups in terms of numbers of affected 1^{o} males or 1^{o} females (separately or combined), or 3^{o} males or 3^{o} females (separately or combined). There were, however, significant differences noted between 2^{o} female Controls and Anorexics (Sum p = 0.009) and between 2^{o} female Anorexics and Bulimics (Sum p = 0.009). In both cases, the Anorexic

group had a significantly greater number of female 2^{o} relatives affected than expected.

Number of Affected Children per Family

Table 6 gives the total number of male and female children in each group, and the total number of affected male and female children in each group, amongst the 1^{o} relatives of the subject. Subjects were excluded from calculations in this table.

Table 6

Number and Percent of Affected Children per Group

	# Children/Group			# Affected (excluding Ss)			%		
	Females	Males	Total	Females	Males	Total	Females	Males	Total
Controls	19	22	41	0	0	0	0.0%	0.0%	0.0%
Bulimics	21	25	46	2	0	2	9.5%	0.0%	4.3%
Anorexics	20	15	35	1	0	1	5.0%	0.0%	2.9%

Fisher's Exact Tests revealed no significant differences between the groups with regard to the number of affected children per family.

Prevalence of Eating Disorders

Table 7 gives the prevalence of eating disorders observed in males and females, together, in 1^{o}, 2^{o} and 3^{o} relatives. This data excludes subjects. The prevalence was calculated as the number of affected individuals divided by the total number of individuals in each group.

Table 7

Prevalence of Eating Disorders

(Males plus Females per 1,000 Individuals)

	1^{o} Relatives	2^{o} Relatives	3^{o} Relatives
Controls	00.0	00.0	04.0
Bulimics	23.0	00.0	14.0
Anorexics	15.0	17.0	00.0

Table 8 shows the prevalence of eating disorders in males and females, in 1^{o}, 2^{o} and 3^{o} relatives. This data excludes subjects. The prevalence was calculated as the number of affected males or the number of affected females divided by the total number of males or females, among 1^{o}, 2^{o} and 3^{o} relatives.

Table 8

Prevalence of Eating Disorders

Between Males and Females per 1,000 Individuals

	1^{o}		2^{o}		3^{o}	
	Males	Females	Males	Females	Males	Females
Controls	00.0	00.0	00.0	00.0	00.0	08.0
Bulimics	00.0	49.0	00.0	00.0	07.0	21.0
Anorexics	00.0	29.0	00.0	35.0	00.0	00.0

Other Psychiatric Pathology

Table 9 indicates other psychiatric pathology, in general, amongst 1^{o} relatives in each group. Other psychiatric pathology was calculated as the number of affected individuals divided by the total number of each particular type of 1^{o} relative.

Table 9

Other Psychiatric Pathology - 1^{o} Relatives

(Weight problems excluded)

	Number and Percent with Other Psychiatric Pathology				
	Fathers	Brothers	Mothers	Sisters	Total
Controls	4/20 (20.0%)	1/20 (5.0%)	3/20 (15.0%)	2/19 (11.0%)	12.7%
Bulimics	7/20 (35.0%)	12/25 (48.0%) *†1,3	7/20 (35.0%)	12/21 (57.0%) †1*3,4	44.2%
Anorexics	6/15 (40.0%)	1/15 (7.0%)	6/15 (40.0%)	3/20 (15.0%)	24.6%
Anorexics & Bulimics Together	13/35 (37.0%)	13/40 (32.5%) *4	13/35 (37.0%)	15/41 (37.0%) *4	41.2%
		(Weight problems	only)		
Controls	1/20 (5.0%)	0/20 (0.0%)	3/20 (15.0%)	3/19 (15.8%)	8.9%
Bulimics	0/20 (0.0%)	1/20 (5.0%)	3/20 (15.0%)	3/19 (15.8%)	8.9%
Anorexics	1/15 (7.0%)	1/15 (7.0%)	5/15 (33.3%)	3/20 (15.0%)	15.4%
Anorexics & Bulimics Together	1/35 (2.9%)	2/35 (5.7%)	8/35 (22.9%)	6/39 (15.4%)	11.8%

* Comparison of groups by χ^2 or Fisher's Exact Tests $p < .05$.
† Significant at the corrected α level.
1 Controls x Bulimics.
2 Controls x Anorexics.
3 Anorexics x Bulimics.
4 Controls x Anorexics and Bulimics together.

χ^2 analyses revealed that there were significant differences between the groups in terms of 1^{o} relatives with other psychiatric pathology. Brothers in the Bulimic group had significantly more "other psychiatric pathology" than brothers in the Control group (Sum p = .0015) as well as brothers in the Anorexic group (Sum p = .0069). Sisters in the Bulimic group had significantly more "other psychiatric pathology" than sisters in the Control group ($\chi^2 = 9.53$, df = 1, $p < .005$).

Table 10 indicates the number of each specific other psychiatric pathology, with 1^{o}, 2^{o} and 3^{o} relatives combined, out of the total number of males and females in each group (indicated at the bottom of each column.

Table 10

Other Psychiatric Pathology (1^{o}, 2^{o} & 3^{o} Relatives Combined)

	Controls		Bulimics		Anorexics	
	Females	Males	Females	Males	Females	Males
Depression	12	7	28*[1,3]	9	13	6
Alcoholism	3	20	5	24	5	11
Drug Abuse	4	6	7	11	3	9
Other	13	24*[2,3]	15	21	7	8
Obese	16*[2]	5	14*[3]	5	5	3
Overweight	28†[1]*[4]	16†[1]*[3,4]	12	2	19	8
Underweight	4	8†[2]	5	2	9	0
All "weight" problems together	48*[1,2,4]	29*[2]†[1,4]	31	9	33	11
Total in each group	260	297	281	290	276	252

* Comparison of groups by χ^2 or Fisher's Exact Tests $p < .05$.
† Significant at the corrected α level.
[1] Controls x Bulimics.
[2] Controls x Anorexics.
[3] Anorexics x Bulimics.
[4] Controls x Anorexics and Bulimics together.

A χ^2 analysis revealed that there were significant differences between the groups in terms of other psychiatric pathology. There were significantly more males in the Control group, who were overweight, than in the Bulimic group ($\chi^2 = 10.89$, df = 1, $p < .001$). As well, there were significantly more females in the Control group, who were

overweight, than in the Bulimic group ($\chi^2 = 8.33$, df = 1, $p < .005$). There were also a significantly greater number of underweight males in the Control group than in the Anorexic group (Sum p = .000013). When all "weight-related problems together" were considered, there was a significantly greater number of Control males in this category than Bulimic males ($\chi^2 = 10.75$, df = 1, $p < .005$), and Bulimic and Anorexic males together ($\chi^2 = 12.87$, df = 1, $p < .001$).

Parental Preoccupation with Weight, Food and Appearance

A positive answer by the subject to question number 21 in the questionnaire was used as the indicator of parental preoccupation with weight, food and/or appearance. Examples of answers taken as "positive" included, "... my parents always forced me to finish everything, saying that I would need all of the food to grow up to be beautiful.", "... my parents always told us to have proper table manners, sit up straight - chest out, stomach in, don't speak at the table. We could never have dessert until we had finished everything else, because just having dessert would make us fat."

Table 11 indicates the total number and percents of positive and negative responses to Question 21 from each group.

Table 11

Parental Preoccupation with Weight, Food and Appearance

	Controls		Bulimics		Anorexics	
Yes	(1/20)	5.0%	(9/20)†	45.0%	(7/15)†	46.7%
No	(19/20)	95.0%	(11/20)	55.0%	(8/15)	53.3%

† $p < .008$

Fisher's Exact Tests revealed that there was significantly more parental preoccupation in the Bulimic (Sum p = .0042) and Anorexic (Sum p = .0057) groups than in the Control group. As well, the combined Bulimic and Anorexic group had significantly more (Sum p = .0013) parental preoccupation than the Control group.

Mean Parental Age

Table 12 indicates the mean parental age, in each group, when the subjects were born.

Table 12

Mean Parental Ages When Subjects were Born

	Controls		Bulimics		Anorexics	
	$\bar{X}$	S.D.	$\bar{X}$	S.D.	$\bar{X}$	S.D.
Mothers	27.3	(5.5)	28.2	(6.4)	26.3	(3.4)
Fathers	30.9	(7.0)	30.4	(5.8)	30.3	(5.1)

An analysis of variance indicated that there were no significant differences between the groups in terms of either mother's or father's ages when the subjects were born.

Questionnaire

Table 13 indicates each question in the questionnaire, where any significant differences between the groups were noted. (Please refer to Appendix H for a description of all questions in the questionnaire.)

Table 13

Significant Questionnaire Results

Question	Controls (N=20)	Anorexics (N=15)	Bulimics (N=20)	Combined Bulimics & Anorexics (N=35)
5. Subject's weight (lbs.)	126.7	99.1**† (p<.000)	117.1**†(AB) (p<.005)	109.1**† (p<.002)
16. Subject's occupation (Hollingshead-Redlick Index, 1958)	48.0	40.6** (p<.033)	41.7	41.2**† (p<.002)
18. Do you know of anyone else with this problem? Yes:	9	13* (p<.0116)	17*† (p<.008)	30*† (p<.0014)
19. Anyone else in the family have psych. problems? Yes:	0	5* (Sum p=.009)	9*† (p<.0006)	14* (Sum p=.005)
20. Anyone else in the family seen a psychologist/psychiatrist? Yes:	5	7	13* (p<.0066)	20* (p<.0161)
21. Food and/or eating was a "special issue" when growing up? Yes:	1	7*† (Sum p=.0057)	9*† (p<.0035)	16*† (p<.0017)
25. Mom's weight prior to pregnancy with the subject: (lbs.)	126.6	120.4	132.3**†(AB) (p<.007)	127.4
26. Number of pregnancies in total: ($\bar{X}$)	3.1	4.0** (p<.043)	3.9	3.9** (p<.047)
36. Was contraception practised (Mom)? Yes:	5	6	10*(CB) (p<.0233)	16* (p<.0467)
38. Any drugs taken during pregnancy? Yes:	9	3	9*(AB) (p<.0155)	12
39. Weight gain during pregnancy: (lbs.)	21.0	24.2	29.1** (p<.014)	27.0** (p<.031)

Table 13 cont'd ...

Question	Controls (N=20)	Anorexics (N=15)	Bulimics (N=20)	Combined Bulimics & Anorexics (N=35)
47. Type of illness that anyone in the family had been hospitalized for: Medical	7*(CB) ($p<.035$)	3	1	4
Psychological	1	3	4	7
Both	1	1	0	1
48. Any period of time when Mom was separated from daughter? Yes:	5	5	11* ($p<.0151$)	16
51. Anyone in Mom's family ever had a psych. illness? Yes:	7	4	11*(AB) ($p<.0314$)	15
52. Anyone in Mom's family ever seen a psychologist/psychiatrist? Yes:	5	10* ($p<.0186$)	9* ($p<.0475$)	19* ($p<.0116$)

* Comparison of groups by χ^2 or Fisher's Exact Tests.

** Comparison of groups by t-tests.

† Significant at the corrected α level.

Note: Unless otherwise noted (e.g., AB = Anorexic x Bulimic comparison), all significant comparisons for the Anorexic group were Controls x Anorexics, for the Bulimic group, Controls x Bulimics, and for the combined Anorexic and Bulimic group, Controls x Combined.

Table 14

Discriminant Analysis of Questionnaire Data

Discriminating Variables and Standardized Positive Coefficients

(in order of relative importance)

Question	Coefficient
21. Do you feel that food and/or eating was treated as a "special" subject when growing up?	.58930
11. Father's age.	.58117
18. Do you know of anyone else with this problem?	.40337
19. Does anyone else in the subjects' family have psychological/psychiatric problems?	.28519
9. and 10. Mother's socioeconomic status.	.22111
7. Mother's age.	.03147
22. Has the subject ever had any other psychological/ psychiatric/medical illnesses?	.02191

Control cases = 20 (100.0%)

Experimental cases = 35 (100.0%)

Percent of Grouped Cases Correctly Classified = 80.0%

Discussion

Are the familial tendencies observed in eating disorders due to genetic factors, psychological factors or both? This thesis has addressed this question by examining several factors specific to the possible heritability or familial tendency of eating disorders.

Familial Prevalence

An initial question, and one which is central to this thesis, is whether there is a greater than expected number of relatives of affected individuals who are also affected with an eating disorder. As may be seen in Table 4, the Control group has only one affected individual out of all of the 1^{o}, 2^{o} and 3^{o} female and male relatives in that group - one female 3^{o} relative (0.1%). The Bulimic group however has two 1^{o} female relatives affected, three 3^{o} female relatives affected and one 3^{o} male relative affected (1.1%). The Anorexic group also has more affected relatives than the Control group - one 1^{o} female relative and three 2^{o} female relatives (0.8%). Therefore, these data have supported this central question - there were more affected relatives in the two experimental groups. It is interesting to note as well that the prevalence of eating disorders in relatives of this Control group quite closely approximates that of previous incidence studies.

Although there is a definite trend towards more affected relatives amongst the Bulimic and Anorexic groups, (particularly amongst the females) these differences were not statistically significant. It is probable that a larger total sample would yield significant differences between the groups for females.

Table 5 indicates the prevalence of affected individuals among 1^{o}, 2^{o} and 3^{o} relatives. Again, no statistically significant differences were found between the groups of 1^{o} females, 1^{o} males, 3^{o} females or 3^{o} males. There were, however, significant differences noted between 2^{o} female Controls and Anorexics, and between 2^{o} female Anorexics and Bulimics. In both cases, the Anorexic group had a significantly greater number of female 2^{o} relatives than expected. These results lend support to the literature which suggests that there are more females affected with eating disorders than males.

Examination of Table 6 indicates that amongst 1^{o} relatives of the subject, there were a greater number of affected females in both the Bulimic and Anorexic groups than in the Control group. More specifically, the Bulimic group contained 4.3% affected siblings, while the Anorexic group contained 2.9% affected siblings. This is supportive of the hypothesis that there is some familial tendency observed in the development of anorexia nervosa and bulimia. These results also lend support to the above-mentioned question; there are more relatives of affected individuals who are also affected with an eating disorder.

Prevalence

Since these data are to be examined from a genetic point of view, the prevalence of eating disorders was calculated for 1^{o}, 2^{o} and 3^{o} relatives (males and females combined) (Table 7) as well as males and females separately (Table 8).

Table 7 indicates that there is clearly an increased prevalence of eating disorders among 1^{o}, 2^{o} and 3^{o} relatives of the experimental groups compared to controls. More specifically, the 1^{o} relatives of the Bulimic group had a higher prevalence of eating disorders than 1^{o} relatives of the Anorexic group, and both experimental groups had a higher prevalence than the Control group. Among 2^{o} relatives, the Anorexic group had a higher prevalence of eating disorders than either the Bulimic or Control groups. Among 3^{o} relatives, the Bulimic group had a higher prevalence than either the Control or Anorexic groups.

When 1^{o}, 2^{o} and 3^{o} relatives were divided into males and females, group by sex comparisons indicated an overall higher prevalence of eating disorders among the 1^{o}, 2^{o} and 3^{o} female relatives of both the Bulimic and Anorexic groups.

These results then also lend support to the central question of this thesis - that there is an increased prevalence of eating disorders among relatives of individuals with eating disorders. It is also clear that the greatest majority of those affected relatives are female, thus lending support to the literature which suggests that females are at increased risk.

Other Familial Psychiatric Pathology

Another question to be addressed in this thesis is whether there is a disproportionate number of other types of psychiatric problems amongst the family members of those individuals with an eating disorder. If so, this may suggest that eating disorder patients come from "psychopathological" families, and may therefore be predisposed to the development of some psychological problem.

Table 9 indicates the number of 1^{o} relatives with other psychiatric pathology in each group. It may be seen that, when weight problems are separated from other types of problems, the 1^{o} family members of the Bulimic group have the highest percentage of others with psychiatric/psychological problems (44.2%). Anorexics and Bulimics together have the second highest percentage (41.2%), Anorexics alone have the third highest percentage (24.6%), while Controls have only 12.7%. Almost all 1^{o} relatives in both the Bulimic and Anorexic groups have higher percentages of psychiatric pathology than Controls. The only exception is the Brothers of the Anorexics, whose prevalence of psychiatric pathology is equal to that of Brothers of the Control group. The statistical analyses indicated a significantly greater number of Brothers with other psychiatric pathology in the Bulimic group compared to the Control group, and Brothers in the Bulimic group compared to the Anorexic group. As well, there were a significantly greater number of Sisters with other psychiatric pathology in the Bulimic group compared to the Control group. When weight problems alone were considered, there were no statistically significant differences found amongst the groups. However, it may be seen from the Table that Fathers, Mothers and Brothers of the Anorexic group had the highest percentages of weight-related problems. Also, when the eating disorder groups were combined, Mothers and Brothers had higher percentages of weight-related problems than Controls.

Table 10 indicates the total number 1^{o}, 2^{o} and 3^{o} relatives with other psychiatric pathology in each group. It is interesting to note that the Control males and females had significantly greater numbers

of relatives with weight problems. Specifically, there were significantly more overweight male and female Controls than Bulimics, more underweight male Controls than Anorexics, and more male Controls with "all weight-related problems together" than either Bulimics alone or both eating disorder groups together.

These data suggest then, that the individuals in the two experimental groups do come from families with a higher prevalence of psychological problems than expected. As mentioned above, this may suggest that these are "psychopathological" families, and that the affected individual may therefore be predisposed to the development of their eating disorder. The basis of this predisposition, however, is only speculative at this time, and will be dealt with in a later section of this Discussion.

With regard to the weight-related problems only, it may be seen that overall, the relatives of the Control group had the greatest number of male and female relatives with "all weight-related problems" together.

These results may appear to be somewhat surprising, as one's initial expectation may be that the two experimental groups' relatives should have a greater number of problems with weight (thus resulting in more weight-related issues in the home, followed by the development of an eating disorder). However, there is an alternate explanation to be considered here. It may be that there is, in fact, a heavy emphasis on weight, food and appearance in the homes of the two experimental groups. This emphasis on weight and appearance may, however, result in an increased amount of time and attention focused

on maintaining an "acceptable" weight and appearance. This intense preoccupation with these issues would necessitate an additional preoccupation with food (i.e., preparation, calories, nutrients, etc.). Thus, the Anorexic and Bulimic groups' relatives would have less problems with obesity, overweight or underweight - because they would be much more preoccupied with these issues than the average individual. They would guard against these problems by their constantly focusing on them.

Parental Preoccupation

Support for this explanation comes from examining Table 11. The question of whether food and/or eating was a "special issue" when growing up was asked as the indicator of parental preoccupation with weight, food and appearance. It may be seen that the Bulimic group had significantly more positive responses to this question than the Control group. The Anorexic group had a greater number of positive responses than the Control group as well. This data lends support to the hypothesis that women in the eating disorder groups did come from familial environments where much more emphasis was placed upon weight and appearance than would be normally expected. Thus, they may have some "predisposition" to the development of their eating disorders in the sense that they have experienced a great deal of environmental pressure to be very aware of their weight, eating habits and appearance.

Some of this preoccupation may be reflected in the observation that the Mothers of the Bulimic women gained significantly more weight during pregnancy (with the subject) than did Mothers of the Anorexic or Control women. As well, Mothers of the Anorexic women gained more

weight during their pregnancies than Control Mothers. Yet, it must be noted that it was the female relatives of Controls who had a significantly greater number of problems with overweight. This may suggest that Mothers in the Bulimic and Anorexic groups were more conscious of their weight gains during pregnancy and strove to maintain their original weights after their children were born.

Parental Age

Another factor which may have possible influence on the child's development of an eating disorder is parental age when the child was born. It has been noted in the literature that the incidence of some physiological or psychological abnormalities has increased with increasing maternal or paternal age. As may be seen in Table 12, however, there were no significant differences between the groups in terms of mean parental age. Therefore, parental age does not seem to be an influential factor in the child's development of an eating disorder.

Questionnaire

With regard to the Questionnaire used in this study, Table 13 outlines the specific statistical analyses used with significant questionnaire results. Appendix H indicates the observed numbers and/or means for each question. As may be seen in Table 13, there were significant differences between the groups in terms of the subject's weight, with Anorexic subjects weighing significantly less than either Control subjects or Bulimic subjects. The weight for the combined Anorexic and Bulimic group was also significantly less than that of subjects in the Control group. There was no significant

difference between Control and Bulimic subjects' weights.

The subjects also differed significantly with their occupations. Using the Hollingshead-Redlick Index (1958), the lower the score, the higher the position. Thus, it appears that individuals in the Anorexic group had the highest positions, with individuals in the Bulimic group, the second highest. The individuals in the Control group had the highest scores, indicating a lower occupational position. The Combined Experimental group had significantly higher occupational positions than the Control group. This makes sense in light of the fact that all of the women in the Control group were students.

More women in the Bulimic group indicated that they "knew of someone else with an eating disorder" than did women in the Control group, and women in the Combined Experimental group "knew of someone else" more often than women in the Control group. This was likely due to the fact that the women in the two experimental groups had had previous clinical and/or hospital exposure to other patients with eating disorders. They may also be more aware of the symptoms to watch for, and may therefore notice another person's problem where a Control person might not.

Women in the Bulimic group also had a significantly greater number of positive responses than Control group women to the question of whether anyone else in the family had psychological/-iatric problems. Anorexic women had the second greatest number of positive responses. Women in the Control group had none. The same pattern of greatest to least number of positive responses was followed with the question of whether anyone else in the family had ever seen a

psychologist/-iatrist. Women in the Anorexic group had a greater number of positive responses to the question of, "Whether anyone in Mother's family had ever seen a psychologist/psychiatrist," (Question 52). Bulimic women had the second greatest number of positive responses. Women in the Control group had the least number of positive responses. There were more positive responses to the question of "anyone else in the family having seen a psychologist/-iatrist" by the Bulimic group than either the Anorexic group or the Control group. The Anorexic group had the second highest number of responses to this question. Significantly more Bulimics and Anorexics answered positively to the question of whether food and/or eating was a special issue when growing up. As well, there were more positive responses to the question of, "whether anyone in the Mother's family had ever had a psychiatric/psychological illness" by the Bulimic group than either the Control or Anorexic groups. These results are in keeping with the data presented earlier, indicating that there was an increased percentage of psychiatric pathology in the families of Bulimics and Anorexics.

Separation

A greater number of Mothers of the Bulimic group than the other two groups were also found to have had a period of separation from their daughters when their daughters were fairly young (approximately 8-9 years old). This is in keeping with the psychoanalytical literature which places the greatest emphasis in the development of eating disorders on the mother-child relationship. This theory suggests that either a significant lack of relationship or a too

highly dependent and controlling relationship between the mother and the child may lead to the development of this type of disorder in one of two ways;

(a) There is a lack of meaningful support, learning and bonding between the mother and child. Thus, the child does not develop a good, solid basic personality with a sense of self, self-worth and individual strengths, morals and ethics. If the mother leaves, even temporarily, this emphasizes the child's awareness of his/her lack of strength and individuality. Instead of relying on their own strengths and beliefs, the child will have to look to the outside to find direction in life. This, of course, is very confusing as there are so many different lifestyles available for the child to adopt. The confusion and lack of direction will likely be frightening to the child, and they may cope with this fear by regressing to a "younger" state in an attempt to force the mother into a more involved relationship.

(b) There is a highly dependent and controlling relationship between the mother and child. Here, the child is not given the opportunity to learn to respond to signals from within his/her self. The mother is such a dominating force in the child's life that the child does not have to learn to make decisions for him/herself - they have already been made by the mother. If the mother leaves, even temporarily, the child may feel suddenly lost and unable to cope with the fears and anxieties of having to make his/her own decisions.

Again, the child may cope with these fears and anxieties by regressing psychologically in an attempt to force the mother back into a dominating and controlling role in the relationship.

Pregnancy

Prior to their pregnancy with the subject, Mothers in the Bulimic group weighed significantly more than Mothers in the Anorexic group. Yet, the Bulimic Mothers gained the most weight with the pregnancy. Contraception was practised by more Mothers in the Bulimic and Anorexic groups than the Control group, yet there were more pregnancies in total reported by these two experimental groups. As well, more drugs were taken during pregnancy by the Bulimic group Mothers than the Anorexic group Mothers.

These results may suggest that there was at least some ambivalence on the part of the Bulimic and Anorexic Mothers to be pregnant. Since Bulimic and Anorexic Mothers were pregnant approximately one more time than Control Mothers, yet contraception was practised, this may have been an unplanned pregnancy. This may have lead to increased stress and heightened anxiety about the pregnancy, resulting in more difficulty with weight control for the Bulimic and Anorexic Mothers, and thus a greater weight gain during pregnancy.

This ambivalence and anxiety about the pregnancy may develop into an impoverished relationship with the child once it is born. This is also in keeping with the psychoanalytical model of the development of eating disorders.

Discriminant Analysis of the Questionnaire

Where all questions in the questionnaire were answered, a discriminant analysis was performed on the data. A total of seven variables were found to discriminate between two groups - Controls, and Anorexics and Bulimics combined. As may be seen from Table 14, the question that contributes most (as determined by the coefficients, which represent the relative contribution of their associated variables to the function) to the differentiation of the two groups is the same question used previously to indicate parental preoccupation with food, weight and/or appearance. Other questions which were found to differentiate the groups included whether the subject knew of anyone else with this disorder, whether anyone else in the subject's family had psychological/psychiatric problems, and whether the subject had ever had any other problems or illnesses. It is interesting to note that all four of these questions were "subject-answered" questions. Mother's age, Father's age and Mother's socio-economic status were also found to be discriminating variables. The discriminant analysis also indicated that, with these seven variables, the likelihood of identifying the correct group membership of another case is 80%. Thus, it appears that these seven questions (in the order presented in Table 14) may indicate a pattern of responses which may be useful in determining or predicting other individuals with (potential) eating disorders. This pattern also lends support to the hypothesis that women who have developed an eating disorder do come from different familial environments than women who do not develop an eating disorder. In this environment,

there seems to be more emphasis placed upon food, weight and/or appearance, a familiarity with eating disorders in general and familial psychological problems. As well, the Fathers and Mothers may both be older (approximately 30-32) with a higher socioeconomic status. It is also interesting to note that the univariate analyses did not reveal the parental age effect to be of importance, where the multivariate analysis did. This may suggest that the interactional nature of the multivariate analysis may be useful in determining important variables which may go unnoticed with other statistical techniques. Thus, this discriminant analysis has importance in that the pattern it reveals may have potential usefulness as a screening tool for "at risk" children and adolescents.

To this point, several differences have been noted between the Control, Bulimic and Anorexic groups in terms of prevalence, familial psychiatric pathology, parental preoccupation, subjects' weights, occupations, knowledge of others with a similar disorder, parent-child separation, and pregnancy. These results now need to be discussed in light of the possible themes and models described earlier.

Chromosomal Model

The question of whether any structural or chromosomal alterations were present in those individuals developing eating disorders cannot be directly addressed with the methods and procedures used in this thesis. The patterns of familial tendency observed here provide the wrong type of data with which to determine (e.g.) translocations or nondisjunction. Karyotyping and banding techniques must be used for detection of structural and/or numerical changes. However, past experience

with autosomal chromosomal abnormalities would lead us to expect morphological abnormalities and mental retardation. Sex chromosome abnormalities would more likely be expressed as behavioural problems.

Single Gene Model

Although the data does not precisely fit any of the four patterns of single gene inheritance, the results may be interpreted using either the X-linked dominant or autosomal dominant modes of inheritance.

X-Linked Dominant

When the total number of children (including probands) in each family is considered, it may be noted that 47.8% of children in Bulimic sibships and 45.7% of children in Anorexic sibships were affected with an eating disorder (Table 6). All of those affected were female. According to the theory, with an X-linked dominant condition, we would expect all daughters of affected fathers to be affected, all children of affected homozygous mothers to be affected, half of the children of heterozygous mothers to be affected and females to be affected twice as often as males. Thus, only the female criterion of this mode of inheritance has been met with this data - females were affected much more often than males.

It seems possible to interpret this data using the X-linked dominant mode of inheritance, but only when several factors which alter the expected Mendelian ratios are included in the interpretation. The familial tendency for the development of an eating disorder would be the result of an X-linked dominant gene with a lack of penetrance and/or variable expressivity and/or pleiotropism, which is either sex-related or lethal in males.

Autosomal Dominant

According to this theory, we would expect half of the children of an affected person to be affected, and males and females to be equally affected. Only the first criterion of this mode of inheritance has been met - approximately half of the children in the experimental groups were affected. However, only one of these affected children was a male.

Therefore; it seems possible to interpret this data using the autosomal dominant mode of inheritance, but only if the gene was sex-limited, (e.g., the presence of female hormones may be necessary for gene expression).

Multifactorial Model

The multifactorial mode of inheritance also needs to be examined in the interpretation of this data. This model assumes that it is the additive effects of several genes plus environmental influences which are necessary for an individual to reach a threshold point either during their development or later in life after which the trait will appear. Each individual has a predisposition for a given trait, or a liability. If their liability exceeds their threshold point, they will express the trait.

The first expectation of multifactorial theory is that there will be a non-linear decrease in the frequency of the disorder with a decrease in degree of relationship. Examination of Table 7 indicates that although there is an overall decrease in the incidence of eating disorders among the Bulimic group's relatives this decrease is not consistent. The incidence drops to 0/1,000 in 2^{o} relatives from

23/1,000 in 1^{o} relatives, and then increases to 14/1,000 in 3^{o} relatives. The same inconsistency applies to the Anorexic group. The incidence increases from 15/1,000 in 1^{o} relatives to 17/1,000 in 2^{o} relatives, then drops to 0/1,000 in 3^{o} relatives.

The second expectation of this theory is that, if the genetic liability is high, then the frequency of eating disorders in 1^{o} relatives will be approximately;

$\sqrt{I}$ I=population incidence

From this data, the observed incidence of eating disorders in 1^{o} relatives (per 1,000 individuals) of female controls is;

00.0 (0/39),

in 1^{o} relatives of female Bulimics is;

49.0 (2/41),

and in 1^{o} relatives of female Anorexics is;

29.0 (1/35).

However, these figures were calculated with female 1^{o} relatives of all ages combined.

The most recent and reliable estimate of the frequency of eating disorders in the population (1/250) was determined by examining groups of school aged girls only (Crisp et al., 1976). If we consider all female 1^{o}, 2^{o} and 3^{o} relatives together in the present study, we have incidences of;

Controls = 1/260

Bulimics = 5/281

Anorexics = 4/276.

In spite of differences between the two studies in terms of

population, sampling procedures and age of subjects, it is interesting to note that the population incidence estimate presented here agrees reasonably well with the figure of 1/250 school aged girls reported by Crisp et al. (1976). An average population incidence of 1/255 will be used here, to address the second expectation of multifactorial theory.

$$\sqrt{1/255} = 62.6/1,000$$

The observed incidence of eating disorders in 1^{o} female relatives of Bulimics was 49/1,000, and in 1^{o} female relatives of Anorexics was 29/1,000. Thus, it appears that the genetic liability for the development of an eating disorder is higher among Bulimics than Anorexics, although neither group is as high as the population. One possible explanation for the lower apparent liability among the Bulimic and Anorexic groups was the age range of females used to determine the incidence figures. Crisp et al. (1976) used females in the 14 to 18 year old range - typically the prime age range for the development of an eating disorder. Females in the present study had a much wider age range, 14 to 54. It is possible that the liabilities of 1^{o} affected female relatives in the Bulimic and Anorexic groups would have been higher, had the age range been more restrictive.

The next expectation of multifactorial theory suggested that the risk of having another affected child increases after having two affected children. However, in the observed data, the maximum number of children affected in any of the families was two. Therefore, this data is insufficient to test this expectation.

The multifactorial model also has the expectation of the risk of

affection in other children increasing with the severity of the disorder in the proband. Although the severity of the eating disorders was not formally assessed in this thesis, it appeared that there was very little difference in the severity of the disorder among members in any one of the families, or between families. Each of the affected individuals varied, of course, with respect to the length of time that they had been ill, but the symptoms of the eating disorders amongst these individuals was remarkably similar. Each appeared to experience the disorder to approximately the same degree of severity. Thus, as far as is testable, this expectation of the multifactorial model has not been met with this data either.

The final consideration of this model is that there may be sex differences in liability leading to different incidences of disease in each sex. With this data, females would have a higher mean liability than males for the development of an eating disorder, and therefore more females would fall beyond the threshold for the disorder. From an examination of this data, it is clear that there are more females than males affected with eating disorders, but in this model one must simply accept the presumption that the reason for this sex difference is "higher mean liability".

As with single gene inheritance, it seems possible to interpret this data using the multifactorial model of inheritance, but only when several a priori assumptions of the model are included in the interpretation. The first assumption is that it is the additive effect of influences which give rise to the development of a disorder. The second and third assumptions are that each individual possesses an

underlying continuous predisposition to a trait, and that this predisposition is normally distributed within the general population. The final assumption is that each individual has a liability for each trait, derived from both genetic and environmental factors.

With those assumptions in mind, the data may be interpreted as indicating an overall non-linear decrease in frequency of the disorder with a decrease in relationship, and definite sex differences in liability leading to a higher incidence of eating disorders for females. The third and fourth expectations of this model (increased risk of having another affected child after two affected children, and increased risk of the disorder developing in other children with a more severe disorder in one child) either were not directly testable with this data or were not met with this data. However, a study specifically assessing the severity of the disorder may help to address these two questions more directly.

Psychological Model

The psychological model of inheritance must also be examined in the interpretation of this data. The first expectation of this model is that only one child in the family (i.e., amongst 1^{o} relatives) should be affected. It may be seen from the data that amongst the Bulimic 1^{o} relatives there were only two families out of twenty, or 10%, where there was more than one child in the family with an eating disorder. Amongst Anorexics' 1^{o} relatives, there was only one family where more than one child was affected (7%). There were no families in the Control group's 1^{o} relatives with any children affected. Therefore, this data essentially supports this first expectation. The

fact that there were three families with more than one child affected may suggest that these were more pathological families to begin with - and hence a greater "opportunity" for more pathology to become evident in the children.

The second expectation of this model directly addresses the question of whether these families have a greater degree of psychiatric pathology or not. This second expectation states that families of affected children may have a higher frequency of psychiatric pathology than families of Controls. From the data (Table 9) it may be seen that both the Bulimic and Anorexic groups' 1^{o} relatives indicated much more psychiatric pathology than the Control group's 1^{o} relatives. When broken down by specific type of 1^{o} relative, there were a significantly greater number of Brothers in the Bulimic group and Sisters in the Bulimic group with some psychiatric pathology. Fathers in the Combined Experimental groups had the same percentage of psychiatric pathology as Fathers in the Control group. Mothers, in the Combined Experimental groups, did have a higher prevalence of psychiatric pathology than did Mothers in the Control group, but not significantly so. This expectation, then, is supported by the sibling data - there is overall more psychiatric pathology amongst families of those affected with an eating disorder than amongst Control families. The only exception is with the Fathers, where the percentage of psychiatric pathology in both the Control and Experimental groups is equal and is still fairly high - 20%.

The third expectation is that females are expected to be affected more frequently than males in this model. As may be seen from

Tables 4 and 5, and as has been discussed previously in this section, 1^{o}, 2^{o} and 3^{o} females were affected more often than males in both the Bulimic and Anorexic groups compared to the Control group. The fourth expectation is that parents of affected families were also expected to display subacute eating disorders, in the form of a preoccupation with weight, food and appearance. As may be seen in Table 11, there was significantly more parental preoccupation in the Bulimic and Anorexic groups than in the Control group. Thus, the third and fourth expectations of this model have clearly been met with this data.

The fifth expectation of the psychological model suggested that affected children may have older parents (i.e., parents over 30). This increased age of the parents was hypothesized to have been due to the parents having spent the first few years of their adult lives organizing their careers, finishing school, etc. This expectation was not met, as may be seen in Table 12. Parents in all three groups had their children at approximately the same age.

The final expectation of this model suggests that the observation of an eating disorder in a family should remain within that family unit - that is, fewer 2^{o} and 3^{o} relatives would be affected. Examination of the data reveals that 1.9% of Bulimic 1^{o} relatives were affected, 1.3% of Anorexic 1^{o} relatives were affected and 0.0% of Control relatives were affected. This compares with 0.0% of Bulimic 2^{o} relatives, 1.7% of Anorexic 2^{o} relatives and 0.0% of Control 2^{o} relatives, and 1.4% of Bulimic 3^{o} relatives, 0.0% of Anorexic 3^{o} relatives and 0.4% of Control 3^{o} relatives. Thus, overall

the Bulimic and Anorexic groups had a higher percentage of 1^{o} relatives affected than Control relatives affected. As well, when the two Experimental groups are combined, there are more 1^{o} relatives affected than 2^{o} or 3^{o} relatives (3.2% compared to 1.7% and 1.4%). Although these differences did not reach statistical significance, there was clearly a trend for the eating disorders in relatives to be observed primarily amongst the 1^{o} relatives of eating disorder families.

As with the single gene and multifactorial models, the psychological model may be used to interpret this data. But some of the expectations of the model were also not met directly, and therefore required explanation.

In summary, this thesis examined the question of whether the familial tendency observed in eating disorders was due to genetic factors, psychological factors, or both. The observed data was compared with the expectations of each of three models of inheritance. An interpretation of the data was made using each of the three models, taking into consideration the potential altering factors of each model as well. Although both the single gene and multifactorial models were useful in the interpretation of this data, several of the expectations of each of these models had to be qualified or amended before the data was explainable. The psychological model, on the other hand, had five out of six of its expectations met.

Heterogeneity in the Etiology of Eating Disorders

One other model should be considered briefly at this point. Although not directly addressed in this thesis, there is the possibility that the familial trend observed in the eating disorders

is not "genetically based" or "psychologically based" - but is heterogeneous, in at least some cases. Examination of the data revealed that in most cases there was a familial trend for either an eating disorder or other forms of psychological problems. However, there were a few sporadic cases where there were no apparent familial trends of this type. Therefore, the etiology of these sporadic cases may possibly be different from that of the familial cases (e.g., a genetic mutation, a mutation within a particular psychological environment). This potential difference must be examined in future studies of this kind.

Frank (1961) has stated that, "... acceptance of one model or theory over another strongly depends upon two criteria, 'agreement with observations' and 'simplicity'." Using these two criteria, he states that we remain completely within the domain of activities that are cultivated and approved by the community of scientists. Accepting these statements, the results of this thesis lend the strongest support to the psychological model of the development of eating disorders. This is not to say that genetics plays no part in the familial tendency for the development of anorexia nervosa and bulimia. It is clear from these results that there must be many more studies conducted in this area before more definitive conclusions can be made about the etiology of eating disorders.

References

American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders (3rd Edition), 1980.

Angst, J., Frey, R., Lomeyer, B. et al. Bipolar manic-depressive psychosis: Results of a genetic investigation. Human Genetics, 1980, 55, 237-254.

Ashby, H. B., & Crowe, R. R. Unipolar depression: A family study of a large kindred. Comprehensive Psychiatry, 1978, 19, 415-417.

Askevold, F., & Heiberg, A. Anorexia nervosa - two cases in discordant monozygotic twins. Psychotherapy and Psychosomatic Medicine, 1979, 32, 223-228.

Baker, M., Dorzab, J., Winokur, G., & Cadoret. Depressive disease: Evidence favouring polygenic inheritance based on an analysis of ancestral cases. Archives of General Psychiatry, 1972, 27, 320-327.

Beaumont, P. J. V., George, G. C. W., & Smart, D. E. "Dieters" and "vomiters and purgers" in anorexia nervosa. Psychological Medicine, 1976, 6, 617-622.

Bruch, H. Eating Disorders. New York: Basic Books, Inc., 1973.

Bruch, H. Brief communications: Preconditions for the development of anorexia nervosa. The American Journal of Psychoanalysis, 1980, 40(2), 169-172.

Bruch, H. Developmental considerations of anorexia nervosa and obesity. Canadian Journal of Psychiatry, 1981, 26.

Brunden. In B. S. Everitt (Ed.), The Analysis of Contingency Tables. New York: John Wiley & Sons, Inc., 1977, p. 45.

Cantwell, D. P., Sturzenberger, S., Burroughs, J., Salkin, B., & Green, J. Anorexia nervosa: An affective disorder? Archives of General Psychiatry, 1977, 34, 1087-1093.

Casper, R. C., Eckert, E. D., Halmi, K. A., Goldberg, S. C., & Davis, J. M. Bulimia: Its incidence and clinical importance in patients with anorexia nervosa. Archives of General Psychiatry, 1980, 37, 1030-1035.

Crisp, A. H. Anorexia Nervosa: Let me be. London: Academic Press Inc., Ltd., 1980.

Crisp, A. H., Palmer, R. L., & Kalucy, R. S. How common is anorexia nervosa? A prevalence study. British Journal of Psychiatry, 1976, 128, 549-554.

Dally, P., & Gomez, J. Anorexia Nervosa. London: William Heinemann Medical Books, Ltd., 1979.

Dibble, E., Cohen, D. J., & Grawe, J. M. Methodological issues in twin research: The assumption of environmental equivalence. In Twin Research: Psychology and Methodology. New York: Alan R. Liss, Inc., 1978, 245-251.

Dowson, J. H. Anorexia nervosa in a male identical twin. British Journal of Clinical Practice, 1977, 31(6), 80-81.

Essenmoller, E. Evidence for polygenic inheritance in schizophrenia. Acta Psychiatrica Scandinavia, 1977, 55, 202-207.

Falconer, D. S. Introduction to Quantitative Genetics. Edinburgh: Oliver and Boyd, 1960.

Falconer, D. S. The inheritance of liability to certain diseases, estimated from the incidence among relatives. Annals of Human Genetics, 1965, 29, 51-76.

Frank, P. G. The validation of scientific theories. New York, N.Y.: Collier Books, 1961.

Frazier, S. Anorexia nervosa. Diseases of the Nervous System, 1965, 26, 155-159.

Garfinkel, P. E., Moldofsky, H., & Garner, D. M. The heterogeneity of anorexia nervosa. Archives of General Psychiatry, 1980, 37, 1036-1040.

Garner, D. M., & Garfinkel, P. E. The eating attitudes test: An index of the symptoms of anorexia nervosa. Psychological Medicine, 1979, 9, 1-8.

Gershon, E. S., Hamovit, J. R., Schreiber, J. L., Dibble, E. D., Kaye, W., Nurnberger, J. I., Jr., Anderson, A., & Ebert, M. Anorexia nervosa and major affective disorders associated in families. Paper presented at the Annual Meeting of the American Psychopathological Association, New York, March 1, 1982.

Gershon, E. S., Mark, A., Cohen, N., Belizon, N., Baron, M., & Knobe, K. E. Transmitted factors in the morbid risk of affective disorders: A controlled study. Journal of Psychiatric Research, 1975, 12, 283-299.

Goetzl, V., Green, R., Whybrow, P., & Jackson. X linkage revisited: A further family history study of manic-depressive illness. Archives of General Psychiatry, 1974, 31, 665-672.

Goodwin, D. W. Alcoholism and heredity: A review and hypothesis. Archives of General Psychiatry, 1979(a), 36, 57-61.

Goodwin, D. W. The cause of alcoholism and why it runs in families. British Journal of Addictions, 1979(b), 74, 161-164.

Greenhill, L. L., & Shopsin, B. Psychiatric morbidity in the offspring of patients with affective disorders: A preliminary report. Neuropsychobiology, 1980, 6, 159-169.

Hall, A. Family structure and relationships of 50 female anorexia nervosa patients. Australian and New Zealand Journal of Psychiatry, 1978, 12, 263.

Halmi, K. Anorexia nervosa and bulimia: Psychosomatic illness review (No. 6). Psychosomatics, 1983, 24(2), 111-129.

Halmi, K. A., & Loney, J. Familial alcoholism in anorexia nervosa. British Journal of Psychiatry, 1973, 123, 53-54.

Hollingshead, A. B., & Redlick, F. C. Social class and mental illness. New York: Wiley, 1958.

Hudson, J. I., Laffer, P. S., & Pope, H. G., Jr. Bulimia related to affective disorder by family history and response to the dexamethasone suppression test. American Journal of Psychiatry, 1982, 137, 695-697.

Hudson, J. I., Pope, H. G., Jr., Jonas, J. M., & Yurgelun-Todd, D. Family history study of anorexia nervosa and bulimia. British Journal of Psychiatry, 1983, 142, 133-138.

Kalucy, R. S., Crisp, A. H., & Harding, B. A study of 56 families with anorexia nervosa. British Journal of Medical Psychology, 1977, 50, 1057-1059.

King, A. Primary and secondary anorexia nervosa syndromes. British Journal of Psychiatry, 1963, 109, 470-479.

Moskovitz, R. A., Belar, C., & Dingus, C. M. Anorexia nervosa in identical twins. Hospital and Community Psychiatry, 1982, 33(6), 484-485.

Nowlin, N. S. Anorexia nervosa in twins: Case report and review. Journal of Clinical Psychiatry, 1983, 44(3), 101-105.

Noyes, R., Jr., Clancy, J., Crowe, R., Hoenk, P.R., & Slymen, D.J. The familial prevalence of anxiety neuroses. Archives of General Psychiatry, 1978, 35, 1057-1059.

Pauls, D. L., Bucher, K. D., Crowe, R. R. et al. A genetic study of panic disorder pedigrees. American Journal of Human Genetics, 1980, 32, 639-644.

Pauls, D. L., Noyes, R., & Crowe, R. R. Familial prevalence in second degree relatives of patients with anxiety neuroses. Journal of Affective Disorders, 1979, 1, 279-285.

Penick, E. C., Read, M. R., Crowley, P. A., & Powell, B. J. Differentiation of alcoholics by family history. Journal of Studies on Alcohol, 1978, 39(11), 1944-1948.

Propping, P. Alcohol and alcoholism. Human Genetics (Supplement), 1978, 1, 91-99.

Pyle, R., Mitchell, J. E., & Eckert, E. D. Bulimia: A report of 34 cases. Journal of Clinical Psychiatry, 1981, 42, 60-64.

Rampling, D. Single case study: Abnormal mothering in the genesis of anorexia nervosa. Journal of Nervous and Mental Disease, 1980, 168(8), 501-504.

Siemens, H. W. The diagnosis of identity in twins. Journal of Heredity, 1927, 18, 201-209.

Slade, P. Towards a functional analysis of anorexia nervosa and bulimia nervosa. British Journal of Clinical Psychology, 1982, 21, 167-179.

Spalt, L. Alcoholism: Evidence of an X-linked recessive genetic characteristic. Journal of the American Medical Association, 1979, 241(23), 2543-2544.

Sperling, M. Psychosomatic disorders in childhood. New York: Aronson, 1978, 129-173.

Story, I. Caricature and impersonating the other: Observations from the psychotherapy of anorexia nervosa. Psychiatry, 1976, 39, 176-188.

Strober, M., Salkin, B., Burroughs, J., & Morrell, W. Validity of the bulimia-restricter distinction in anorexia nervosa: Parental personality characteristics and family psychiatric morbidity. Journal of Nervous and Mental Disease, 1982, 170(6), 345-351.

Theander, S. Anorexia nervosa: A psychiatric investigation of 94 female patients. Acta Psychiatrica Scandinavia (Supplement), 1970, 214, 1-194.

Thoma, H. Anorexia nervosa. New York: International Universities Press, 1967.

Vigursky, R. A. Anorexia nervosa. New York: Raven Press, 1977.

Wallinder, J., & Mellbin, G. Karyotyping of women with anorexia nervosa. British Journal of Psychiatry, 1977, 130, 48-49.

Welner, A., Reich, T., Robins, E., Fishman, R., & Van Doren, T. Obsessive-compulsive neurosis: Record, follow-up and family studies. I. Inpatient record study. Comprehensive Psychiatry, 1976, 17(4).

Winokur, A., March, V., & Mendels, J. Primary affective disorder in relatives of patients with anorexia nervosa. American Journal of Psychiatry, 1980, 137(6), 695.

APPENDIX A

CHROMOSOMAL INHERITANCE

Expect;

- a karyotype to indicate any alteration in the genetic makeup of the individual (eg.)
  (a). structural changes - including balanced or unbalanced translocations, deletions, insertions, duplications, rings or isochromosomes, and spontaneous or induced mutations, or

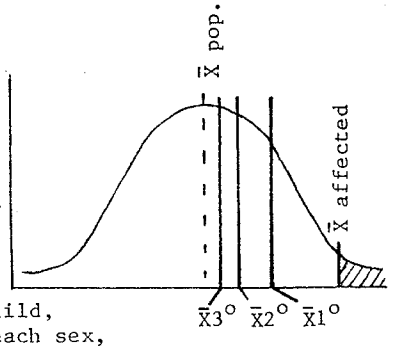
  (b). numerical changes - meiotic or mitotic nondisjunction or translocations.

MENDELIAN OR SINGLE GENE THEORY

Autosomal Dominant	Autosomal Recessive	X-Linked Dominant	X-Linked Recessive
Expect; -½ of children of an affected person to be affected,	-¼ of children of an affected family to be affected, -siblings affected, rarely parents or grandparents,	-all daughters of affected fathers to be affected, -all children of affected homozygous mothers to be affected, -½ children of heterozygous mothers to be affected,	
-males & females equally affected,	-males & females equally affected,	-females affected 2x more then males, -no male to male transmission,	-males affected much more often then females, -no male to male transmission.

GENETIC PLUS ENVIRONMENTAL PSYCHOLOGICAL THEORY-

MULTIFACTORIAL MODEL



Expect;

-a non-linear decrease in frequency of disorder with a decrease in relationship,
-if the genetic liability for eating disorders is high, then the frequency of the disorder in 1^{o} relatives will be approximately; $\sqrt{I}$ where I=general population incidence.
-risk of having another affected child increases after two affected children,
-risk of affection in other children increases with the severity of the disorder in one child,
-there may be sex differences in liability leading to different incidences of disease in each sex, (eg., females have a higher mean genetic liability, so more females would fall beyond the threshold for the disorder).

PSYCHOLOGICAL MODEL

Expect;

-the disorder to appear in one sibling in the family, that person being "used" within the family system, to fulfill the pathological needs of the other family members,
-families of affected children may have a higher frequency of psychiatric pathology than families of controls,
-females to be affected more frequently than males, due to societal preoccupation with women's weight control,
-sub-acute eating disorders in parents (i.e. parental preoccupation with weight, food and appearance),
-affected children to have older parents (i.e. over 30),
-(in direct contrast with Multifactorial hypothesis), the observation of eating disorders in an affected family to remain within the family unit (i.e. few 2^{o} or 3^{o} relatives affected).

APPENDIX B

INFORMATION TO FAMILIES

Your daughter/sister has agreed to help us in our study to try to determine how and why anorexia nervosa and bulimia, (psychiatric/psychological eating disorders), tend to run in families. She will be asked to fill out a questionnaire which asks about any medical, psychological or psychiatric problems that she or other members of the family have had. Two portions of the questionnaire are to be filled out by the parents as well, asking similar questions.

In order to determine the best answer to our question, it is important to answer as fully and truthfully as possible.

We would also like both the subjects and one of their parents to sign Consent forms for their participation in this study. Therefore, there are two Consent forms attached - Consent Form A, for the subjects to sign, and Consent Form B, for one parent to sign.

Thank you for your help and your support in this research study.

APPENDIX C

CONSENT FORM A - STUDENTS

I, _____, understand that I am going to voluntarily participate in a research project looking for familial trends in the development of anorexia nervosa and bulimia. I also understand that names will not be used at all in the study and that the information gathered here is for research purposes only. These consent forms will be kept separately from the pedigrees, (or family "trees"), so that the names of those participating will not be connected with any particular family.

I also understand that I may refuse to respond to any questions I choose, or withdraw from the study at any point in time, for any reason, without repurcussion to any ongoing or future psychological/ psychiatric treatment that I may receive.

_____
Signature

_____
Date

_____
Witness

_____
Address

_____
Phone number

CONSENT FORM B

I, _____, understand that I am voluntarily participating in a research project looking for familial trends in the development of anorexia nervosa and bulimia.

I understand that the information gathered here is anonymous and will be used for research purposes only.

I also understand that I may refuse to respond to any questions I choose, or withdraw from the study at any point in time, for any reason, without prejudice.

_____

Signature

_____

Date

_____

C.A. Elliott-Harper
Project Coordinator

APPENDIX D

ANOREXIA NERVOSA-GENETICS STUDY

QUESTIONNAIRE

1. Date of Interview _____ 2. Age _____ 3. Sex _____

SUBJECT:

Name _____ 4. Height _____

Maiden name (if any) _____ 5. Weight _____

Date of marriage (if married) _____

6. Ethnic origin _____

MOTHER:

Name _____ 7. Age _____

Maiden name _____

Date of marriage _____

8. Ethnic origin _____

9. Education and 10. occupation _____

FATHER:

Name _____ 11. Age _____

12. Ethnic origin _____

13. Education and 14. occupation _____

(for SUBJECT)

15. Age when this problem started _____

16. Occupation _____

17. Do any other members of your family have this problem? Who? For how long?

_____

_____

_____

18. Do you know of anyone else with this problem?

_____

_____

19. Does anyone else in your family have any psychological/psychiatric problems? Who? What?

_____

_____

_____

20. Has anyone else in your family ever seen a psychologist/psychiatrist? Who? Why? When?

_____

_____

_____

21. Do you feel that food and/or eating was treated as a "special" subject while you were growing up? (e.g., forced, made an issue of, neglected, emphasized, etc.?)

_____

_____

_____

22. Have you ever had any other psychological/psychiatric/ medical problems or illnesses? What? When?

_____

_____

(for MOTHER of subject)

23. Age at birth of affected child _____

24. Height _____ 25. Weight _____

26. Number of pregnancies in total

27. Livebirths _____ 28. Miscarraiges _____

29. Details of other pregnancies (difficulties, birth weights, outcome, dates, sex, etc.) in order of births (oldest to youngest)

_____

_____

30. Details of any other children with a medical or psychiatric problem

_____

_____

31. Occupation prior to birth of subject._____

32. Hazards? _____

33. Any major or chronic illnesses during pregnancy? (details - drugs, treatment, age, etc.)

_____

_____

34. Any acute illnesses during pregnancy? (details)

_____

35. Was this a planned pregnancy? _____

36. Was contraception practised? _____ 37. Type _____

38. Any drugs taken during pregnancy? Type and dose _____

_____

39. Weight gain during pregnancy? _____

40. Size and movement during this pregnancy compared to others? _____

_____

41. Any problems with delivery? _____

42. Was this child breast or bottle fed? _____ 43.Type of schedule (rigid or "on demand") _____

44. Feeding or other difficulties? _____

45. Type of baby/child. _____

46. Any major, chronic or acute illnesses after your pregnancy? What? When?

_____

_____

---

47. Anyone in the immediate family been hospitalized for a medical or psychiatric illness? Describe. _____

_____

_____

48. Any period of time when you were separated from your daughter? When and for how long? At what age of child? _____

49. Have you ever had a psychiatric/psychological illness? Describe. _____

_____

50. Were you hospitalized? Where and when? _____

_____

_____

51. Anyone in your distant family ever had a psychiatric/psychological illness? Describe. _____

_____

_____

52. Has anyone in your family ever seen a psychologist/psychiatrist? Who? When? Why? _____

_____

_____

(for FATHER of subject)

53. Age at birth of affected child _____

54. Details of any other children with medical or psychiatric problems

_____

_____

55. Occupation prior to birth of subject? _____

56. Hazards? _____

57. Was this a planned pregnancy? _____

58. Was contraception practised? _____ 59. Type _____

60. Did you participate in feeding? _____

61. Type of schedule _____

62. Difficulties? _____

63. Any major, chronic or acute illnesses after the birth of the child? Describe. When? _____

_____

64. Any period of time when you were separated from your daughter? When? How long? At what age of child? _____

_____

_____

65. Have you ever had a psychological/psychiatric illness? Describe

_____

_____

66. Were you hospitalized? When and where? _____

_____

67. Anyone in your distant family ever had a psychological/psychiatric illness? Describe. _____

_____

68. Has anyone in your family ever seen a psychologist/psychiatrist? Who? When? Why? _____

_____

_____

APPENDIX E

Eating Attitudes Test

1. Like eating with other people.
2. Prepare foods for others but do not eat what I cook.
3. Become anxious prior to eating.
4. Am terrified about being overweight.
5. Avoid eating when I am hungry.
6. Find myself preoccupied with food.
7. Have gone on eating binges where I feel that I may not be able to stop.
8. Cut my food into small pieces.
9. Aware of the calorie content of foods that I eat.
10. Particularly avoid foods with a high carbohydrate content (e.g., bread, potatoes, rice, etc.)
11. Feel bloated after meals.
12. Feel that others would prefer if I ate more.
13. Vomit after I have eaten.
14. Feel extremely guilty after eating.
15. Am preoccupied with a desire to be thinner.
16. Exercise strenuously to burn off calories.
17. Weigh myself several times a day.
18. Like my clothes to fit tightly.
19. Enjoy eating meat.
20. Wake up early in the morning.
21. Eat the same foods day after day.
22. Think about burning up calories when I exercise.
23. Have regular menstrual periods.
24. Other people think that I am too thin.

25. Am preoccupied with the thought of having fat on my body.
26. Take longer than others to eat my meals.
27. Enjoy eating at restaurants.
28. Take laxatives.
29. Avoid foods with sugar in them.
30. Eat diet foods.
31. Feel that food controls my life.
32. Display self-control around food.
33. Feel that others pressure me to eat.
34. Give too much time and thought to food.
35. Suffer from constipation.
36. Feel uncomfortable after eating sweets.
37. Engage in dieting behaviour.
38. Like my stomach to be empty.
39. Enjoy trying new rich foods.
40. Have the impulse to vomit after meals.

APPENDIX F

Pedigrees

Key:

 = Female

 = Male

 = Anorexic

 = Bulimic

 = Miscarriage

1 = Depression

2 = Alcoholism

3 = Drug Abuse

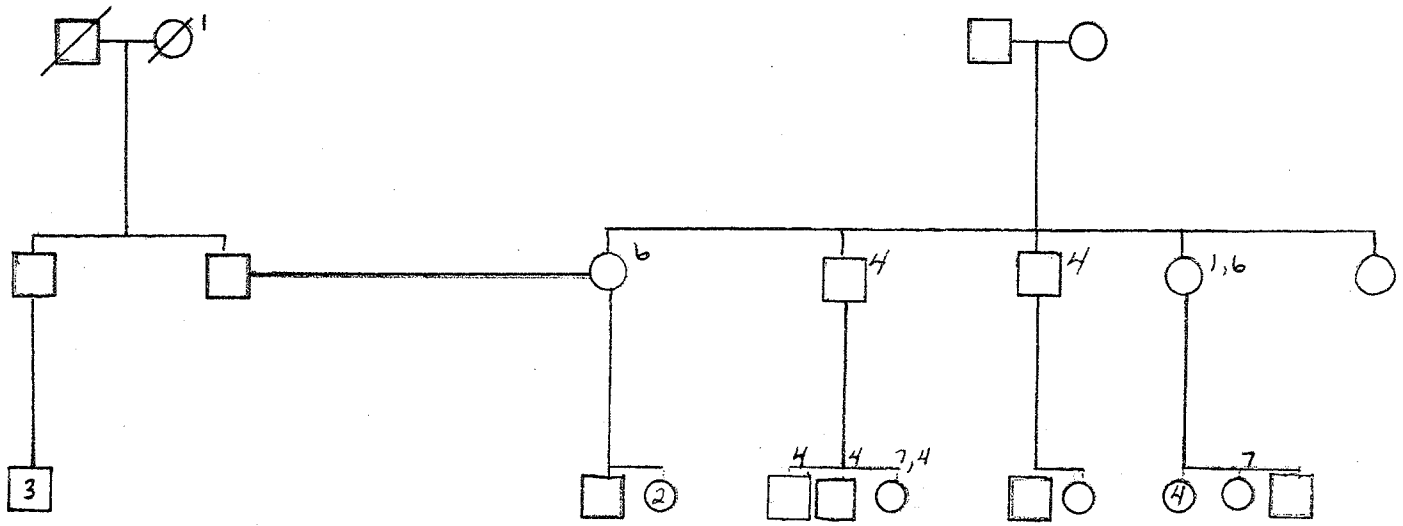
4 = Other

5 = Obese

6 = Overweight

7 = Underweight

Throughout the pedigrees, numbers within the symbol (e.g. ) indicate the number of males (or females) within that sibship. Numbers above the symbol (e.g.) indicate the types of psychiatric problem for that individual. For clarity, parents of subjects have each been placed at one end of their respective sibships. Thus, the order in which they appear does not necessarily reflect their birth order.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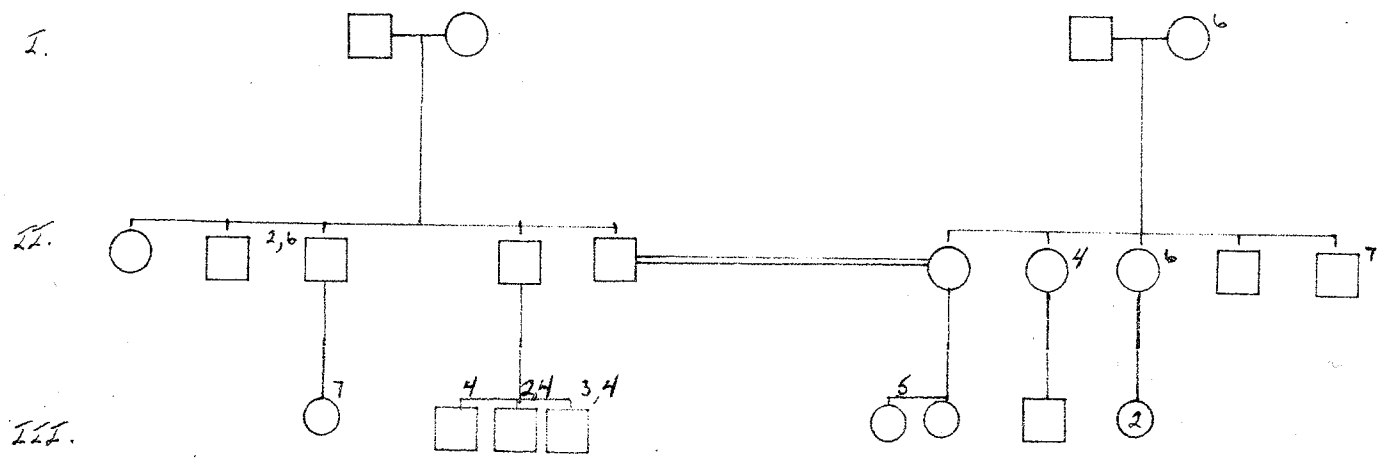
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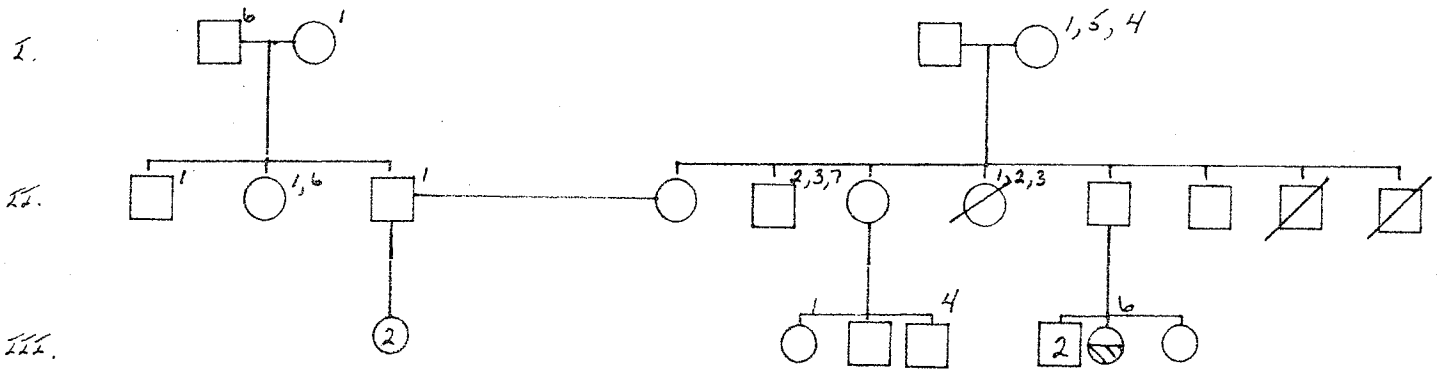

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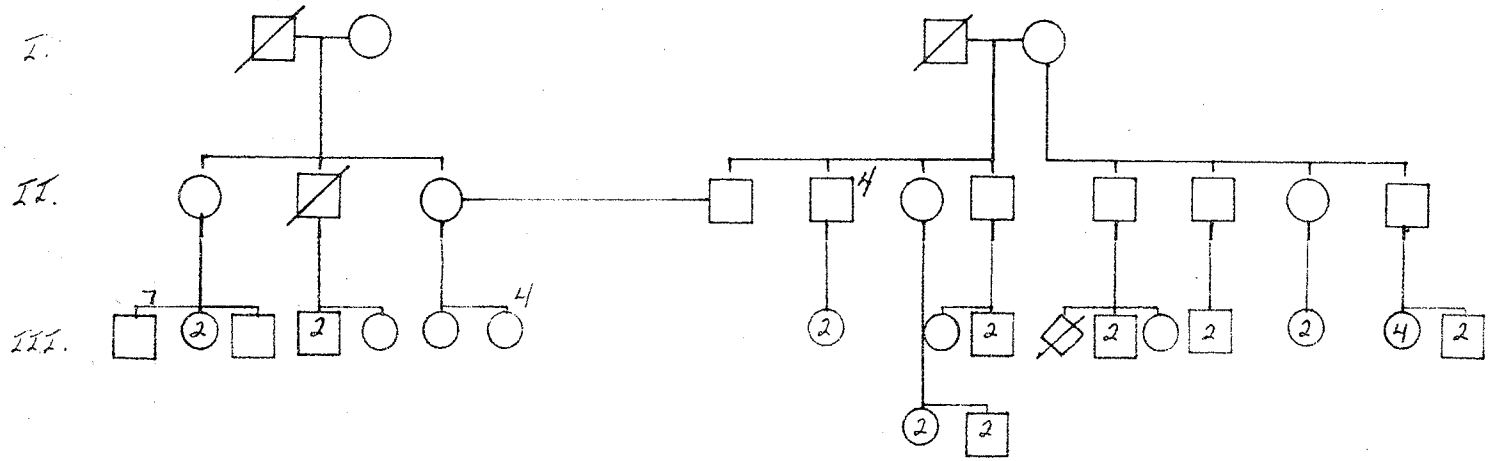

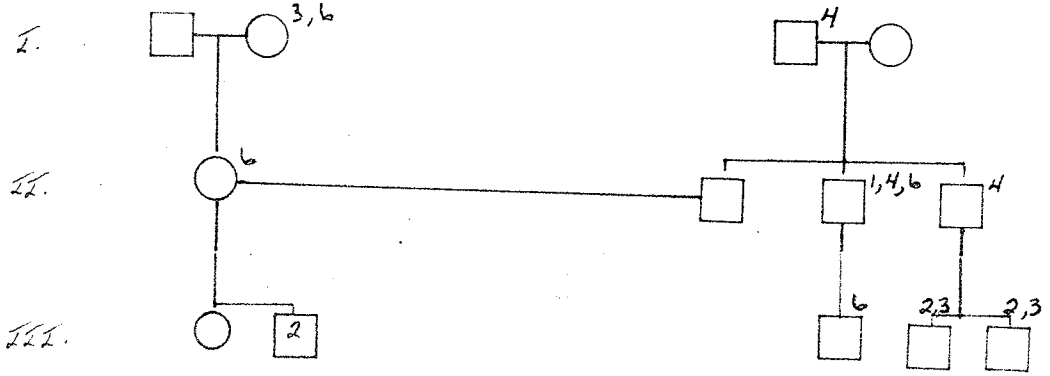
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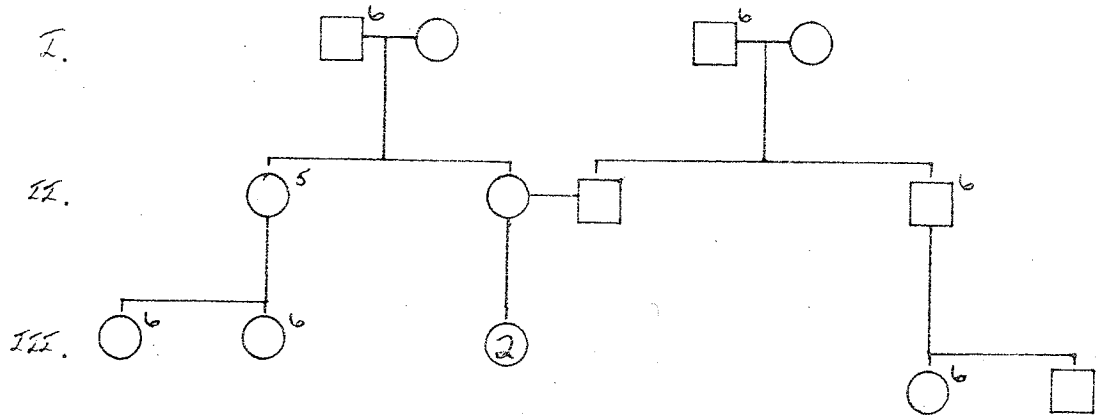

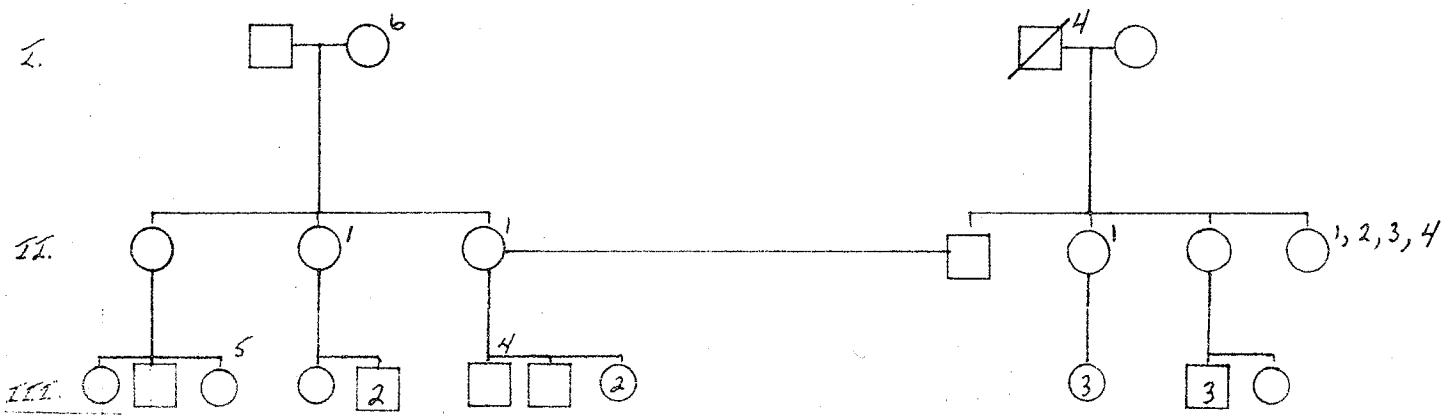
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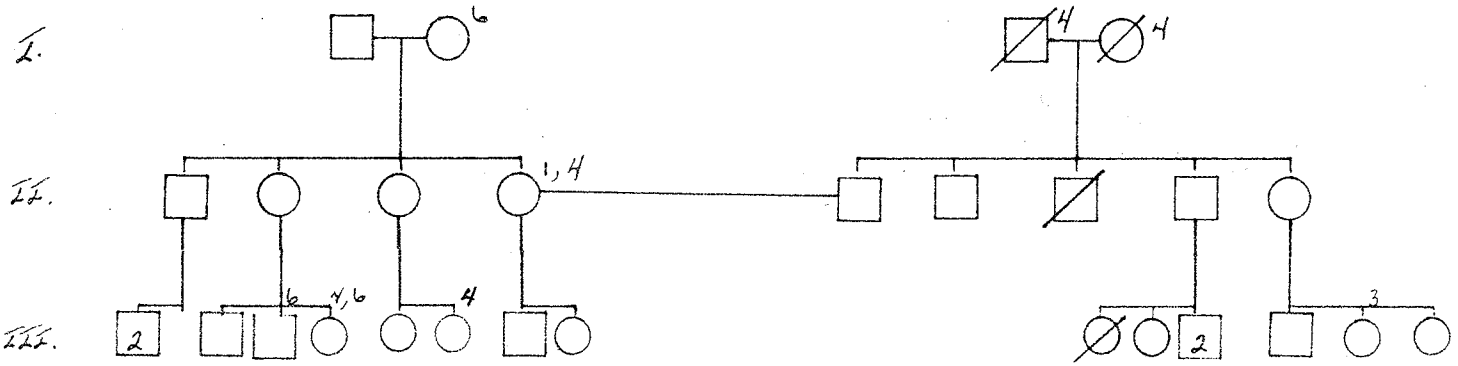
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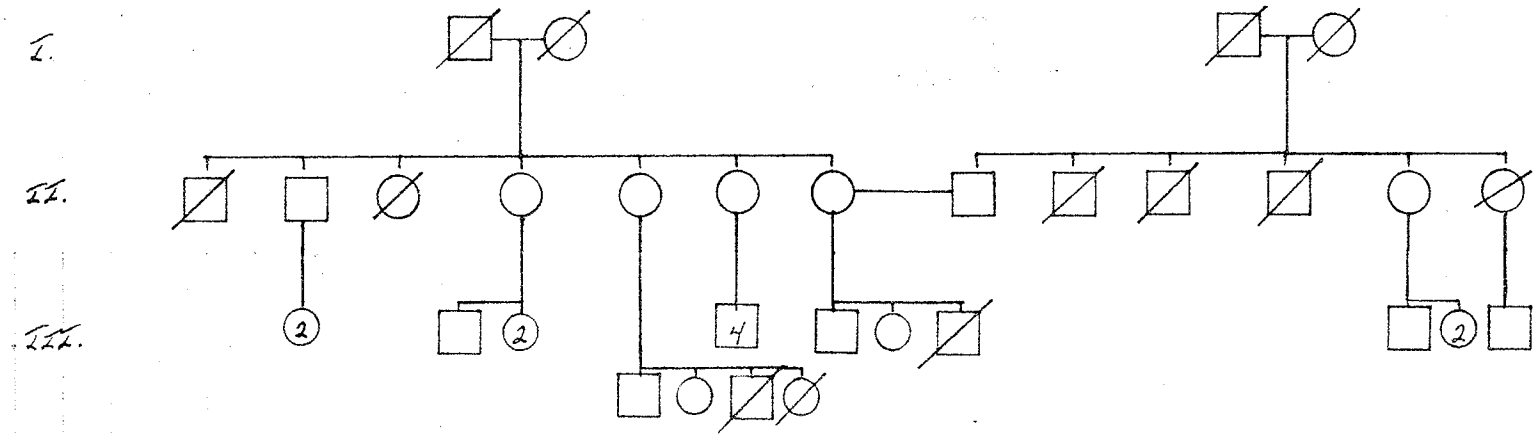
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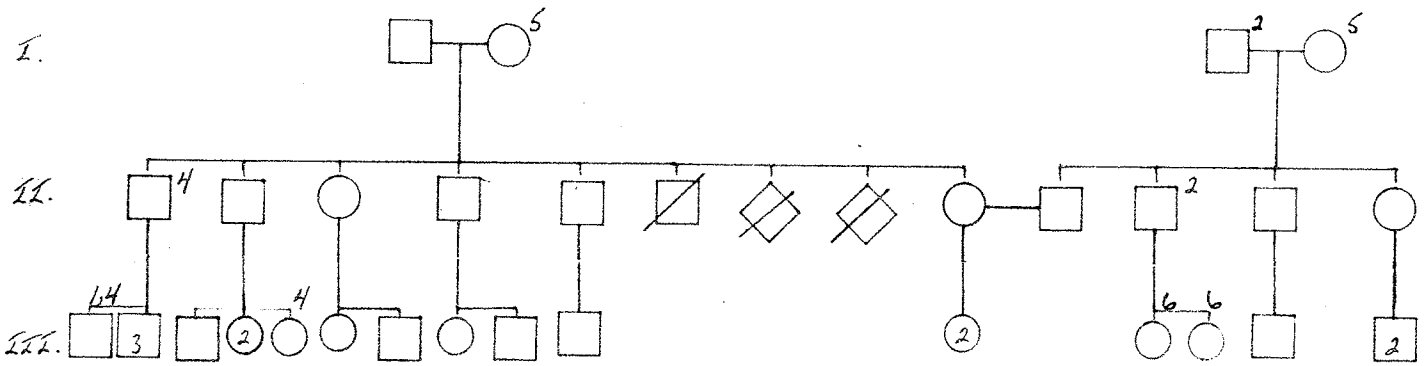
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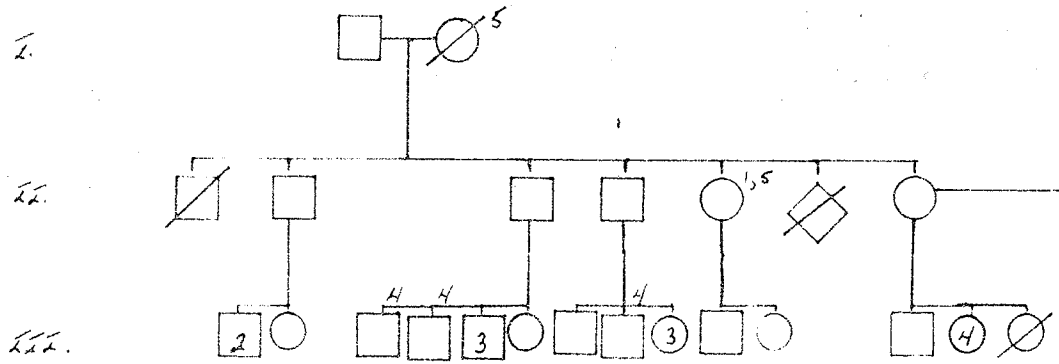

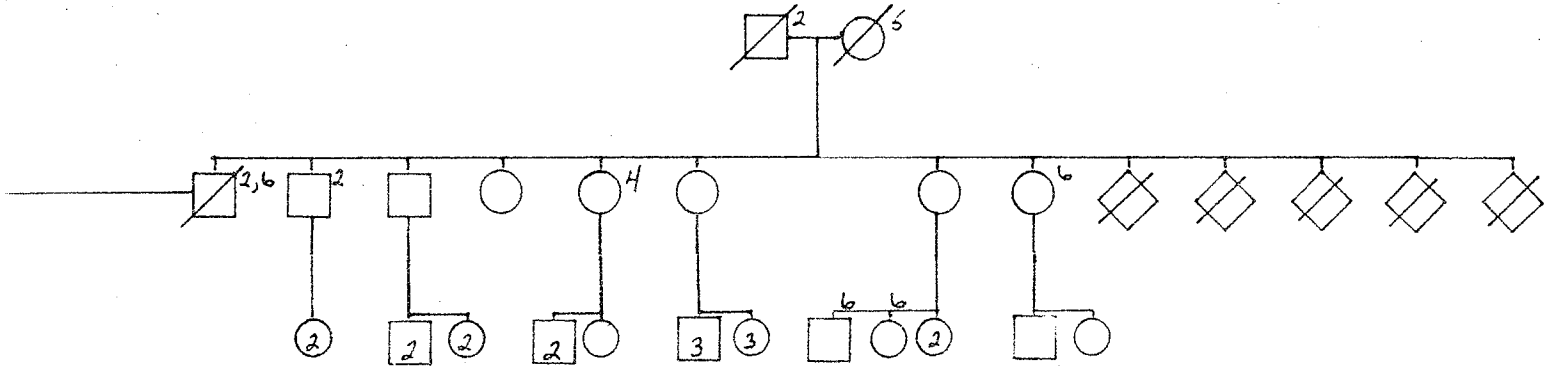
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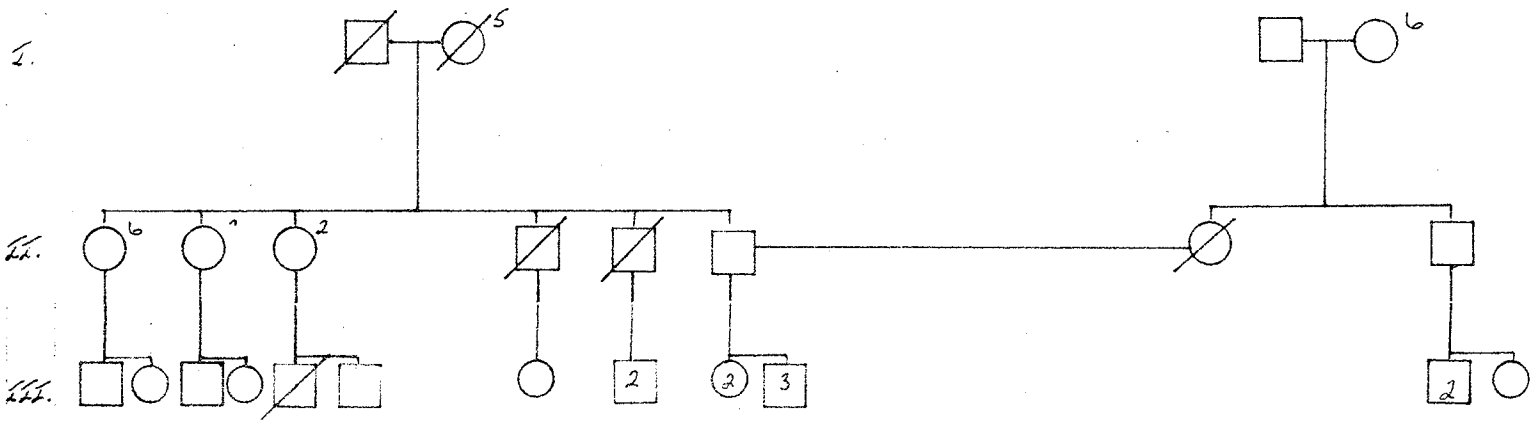

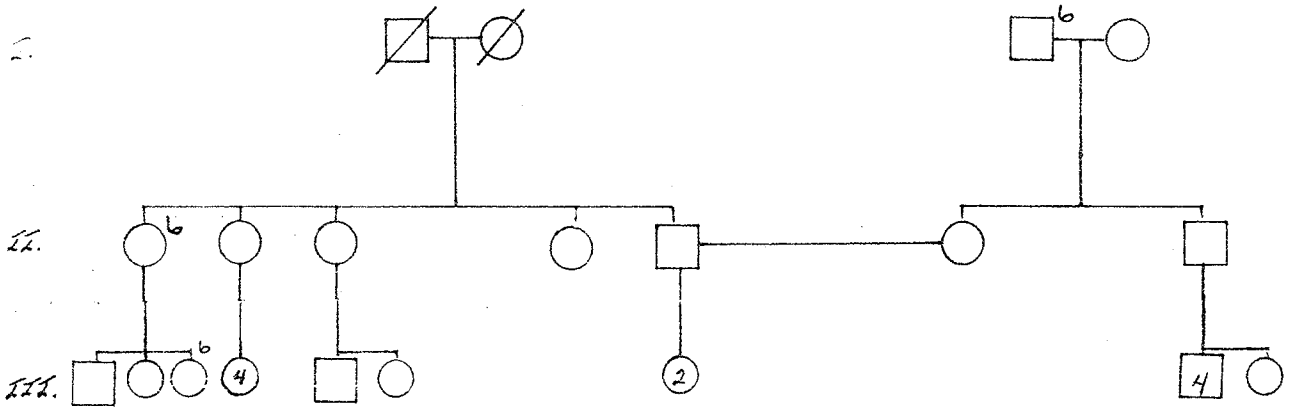
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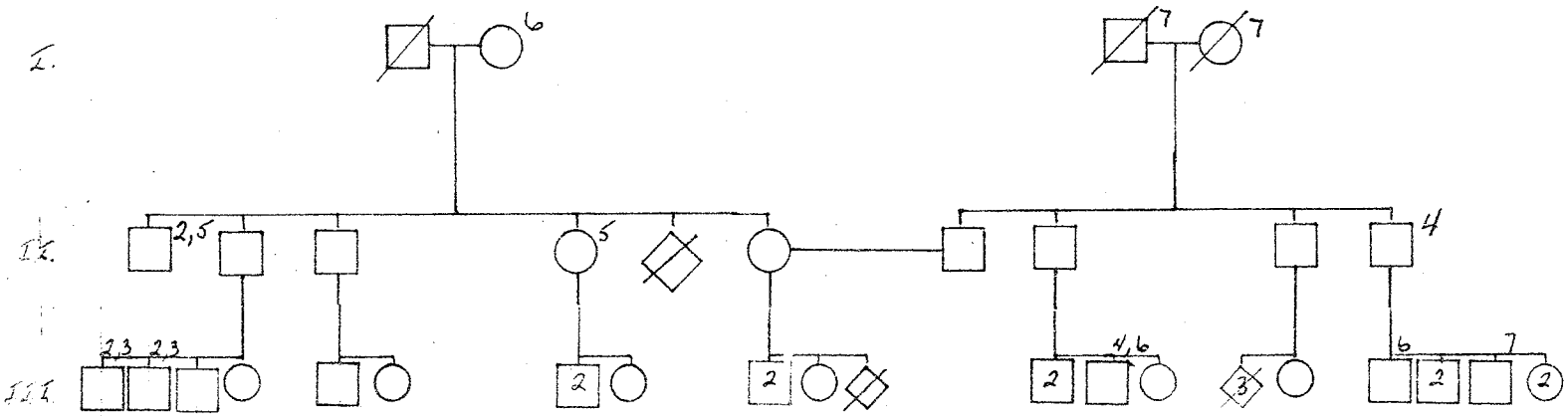
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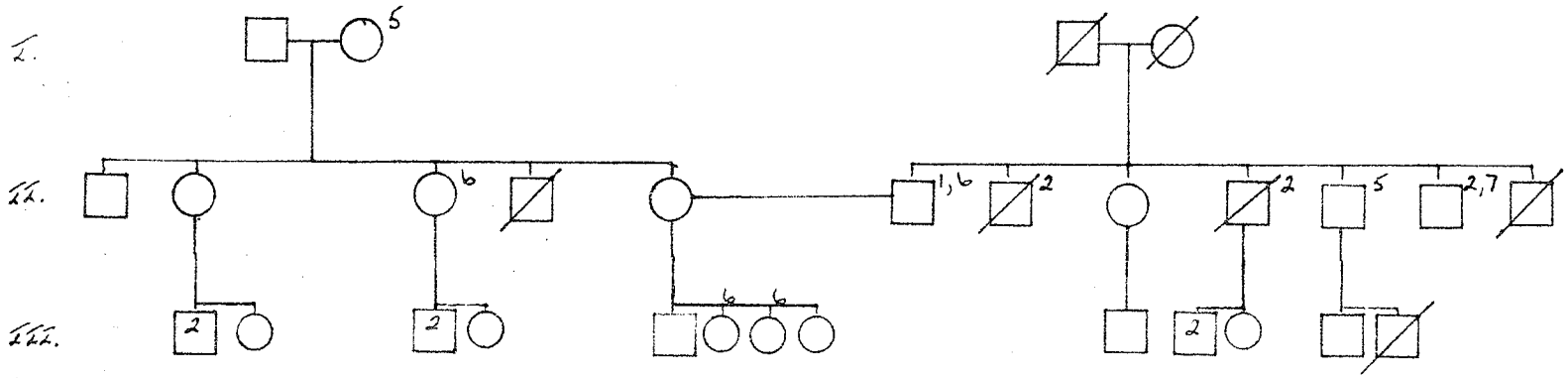
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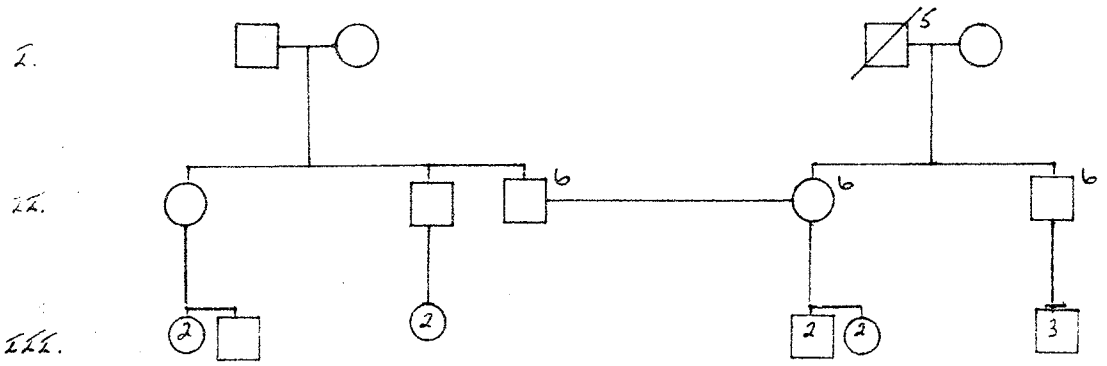
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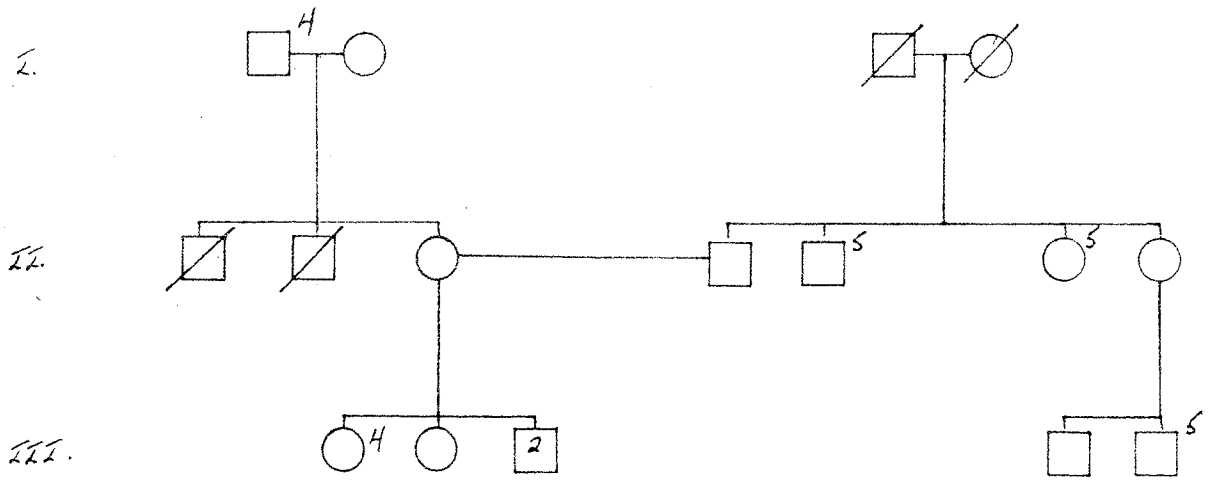
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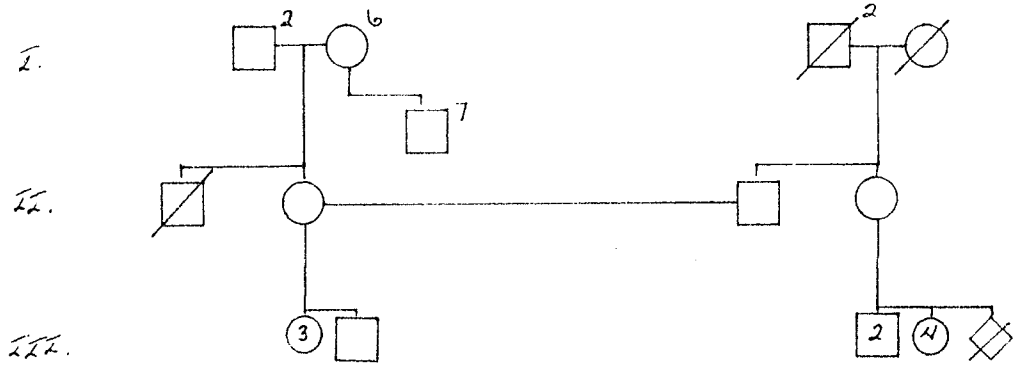

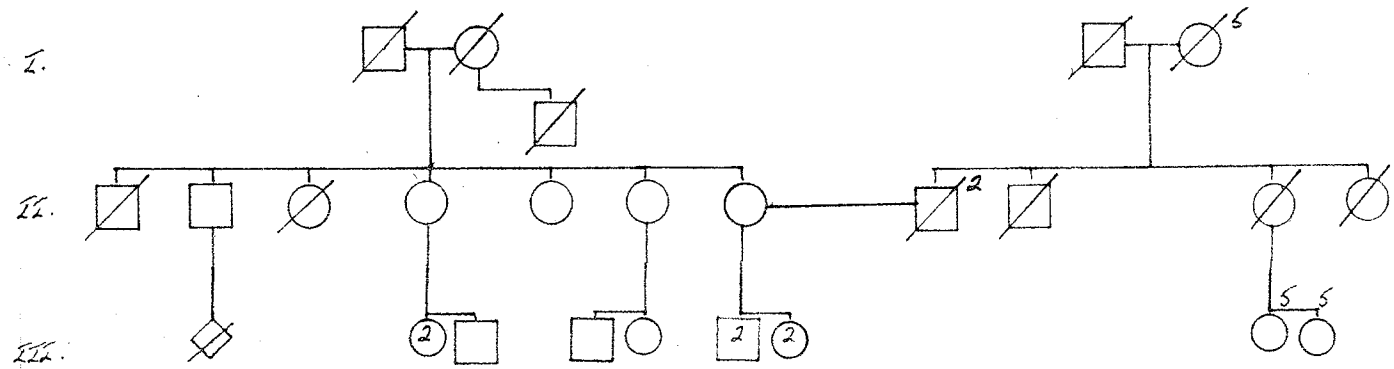
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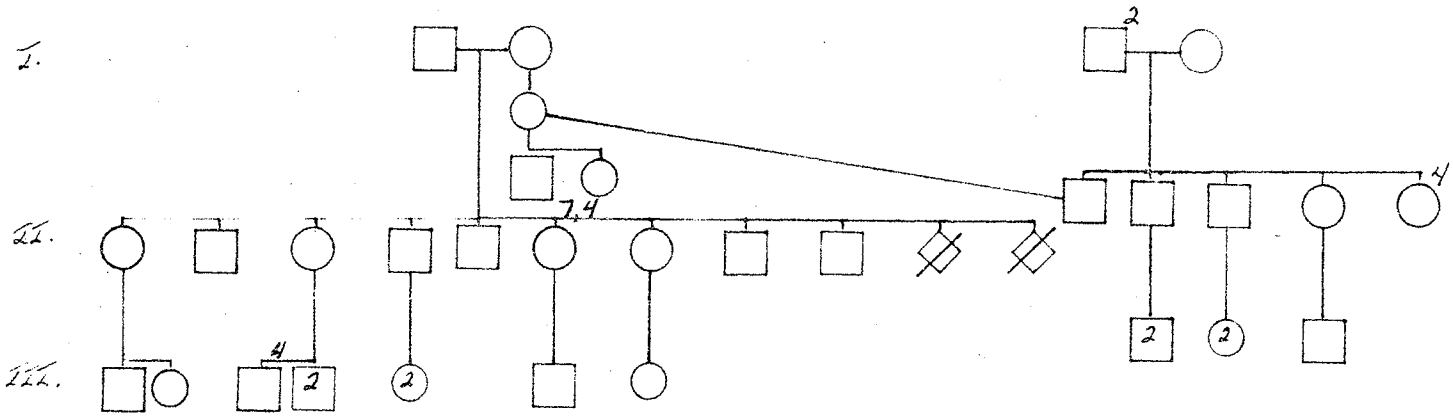

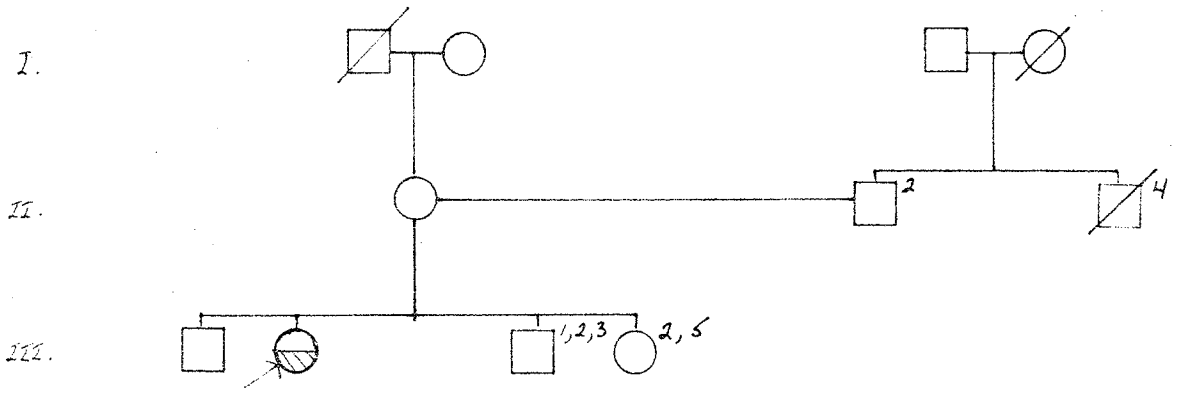
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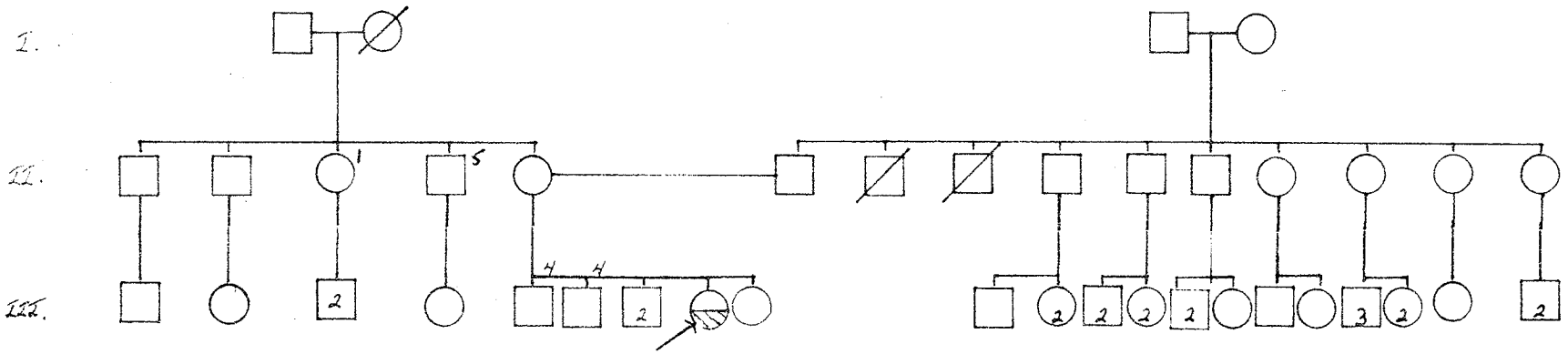
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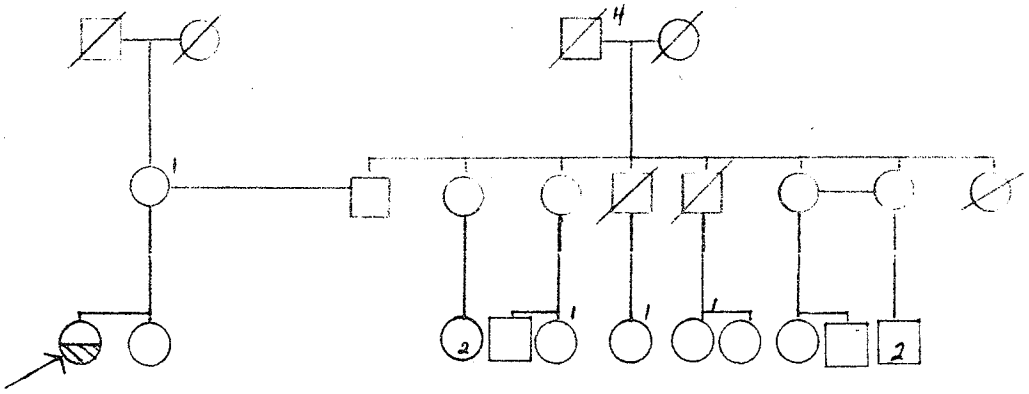

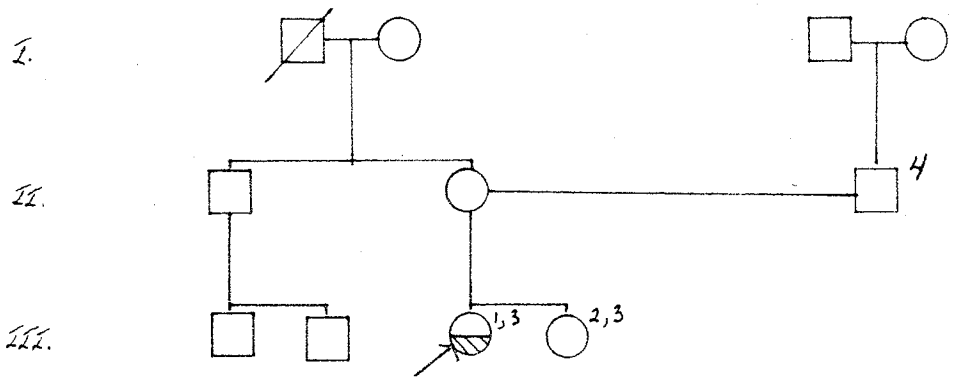
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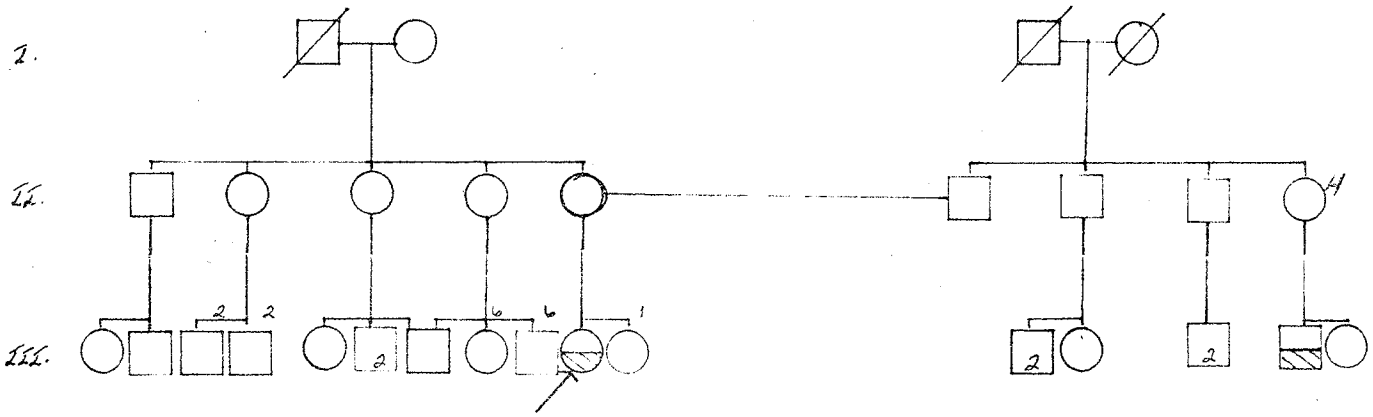
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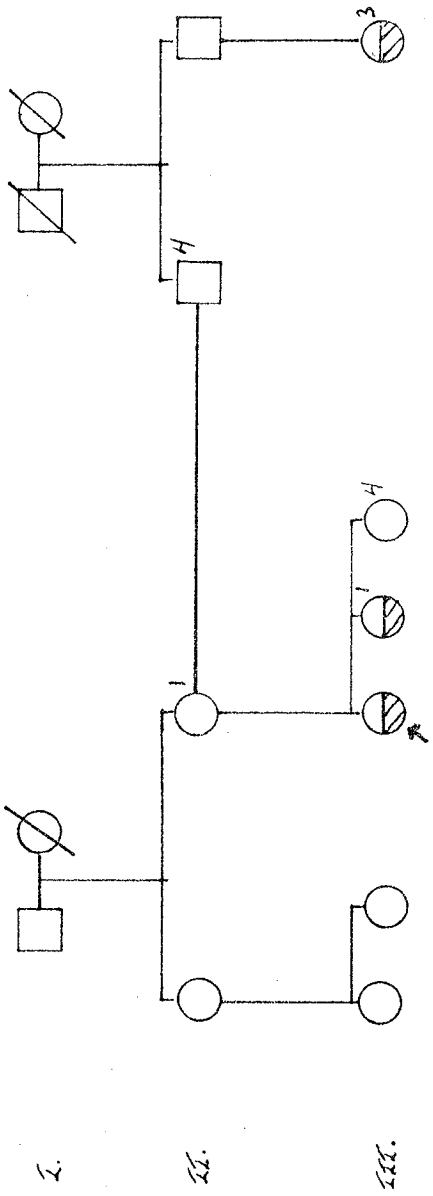
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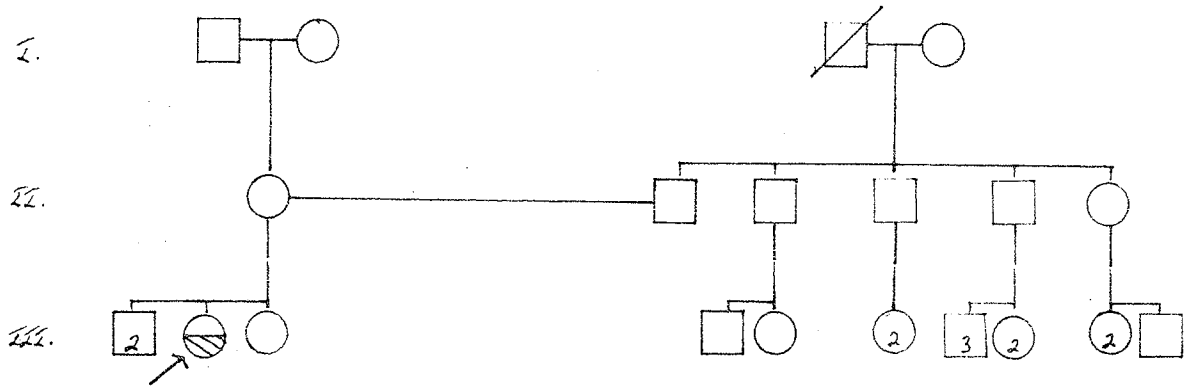

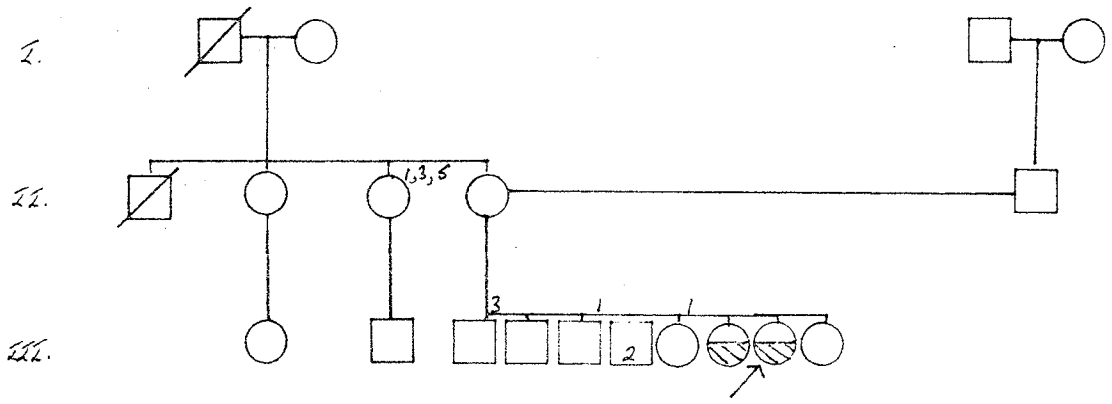
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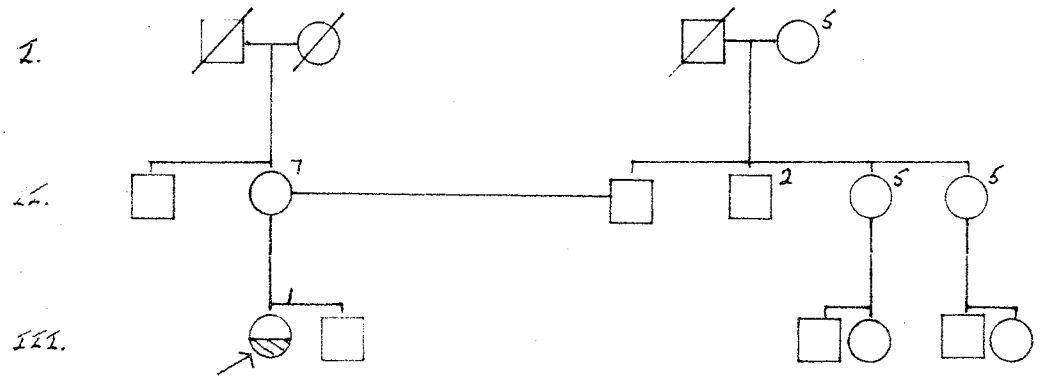
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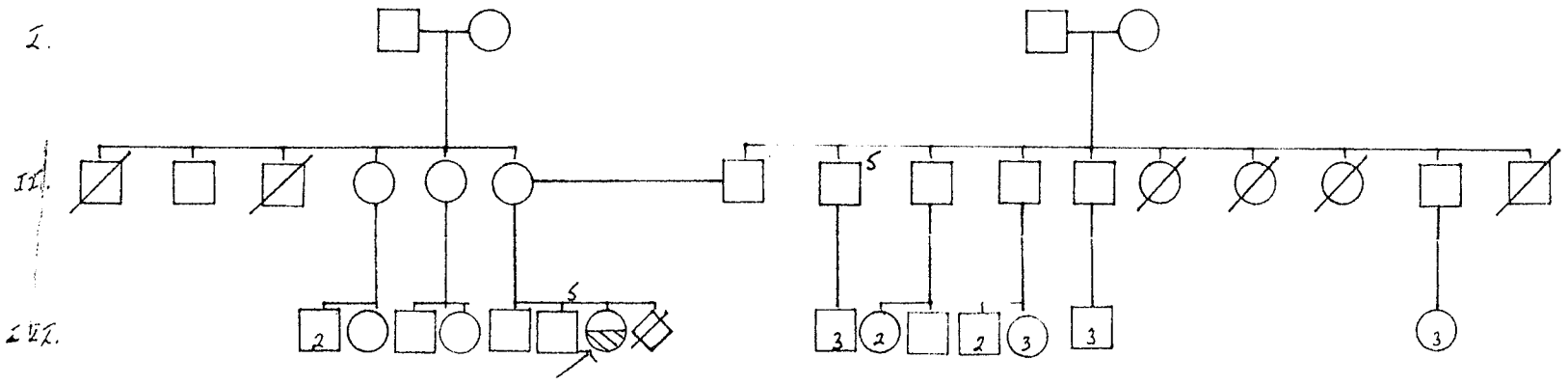
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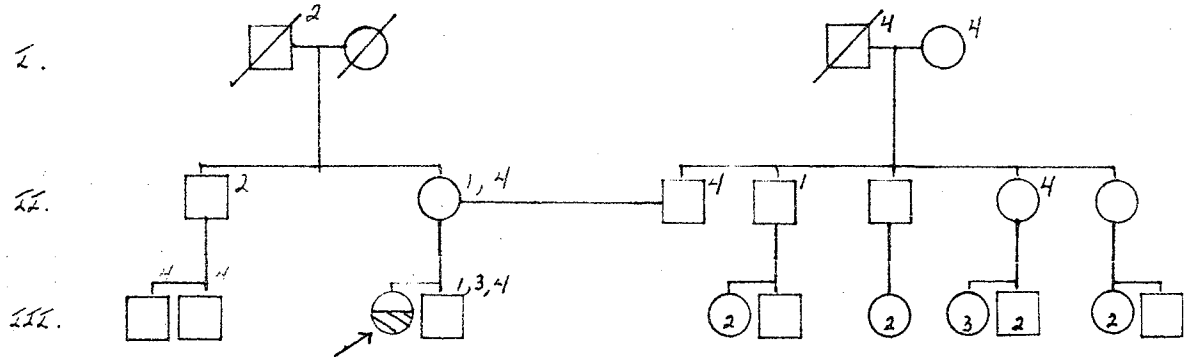
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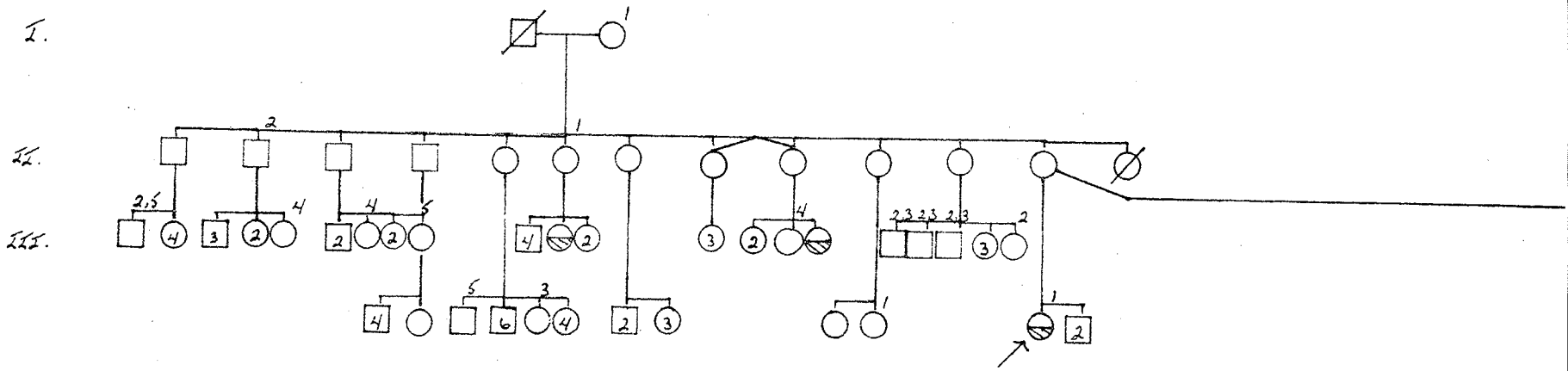
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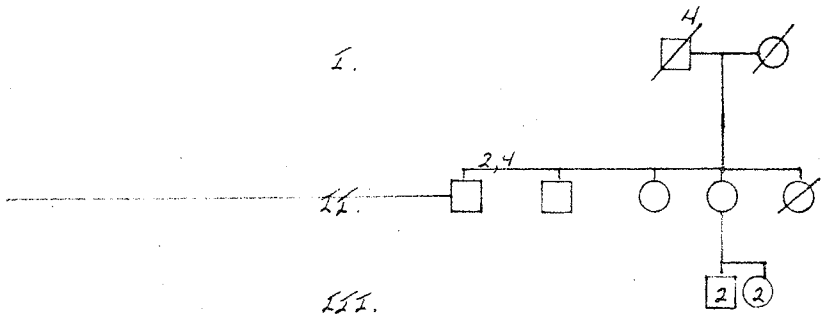
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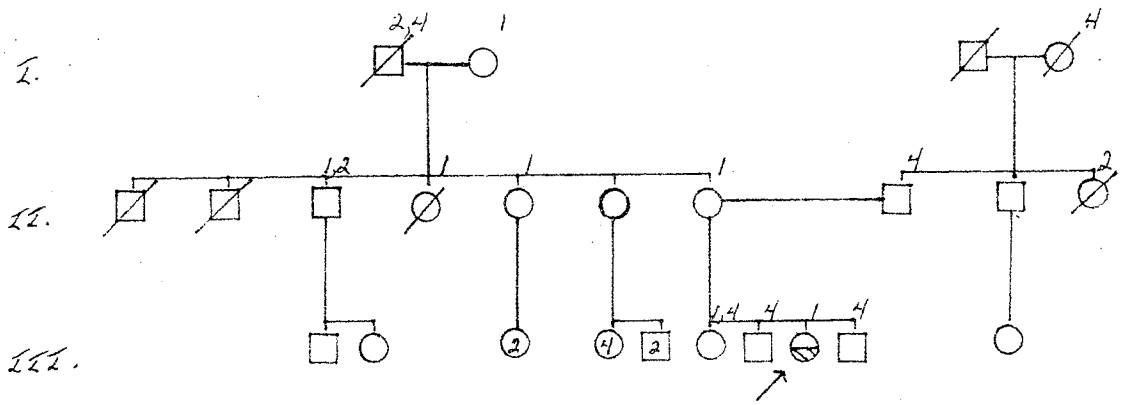
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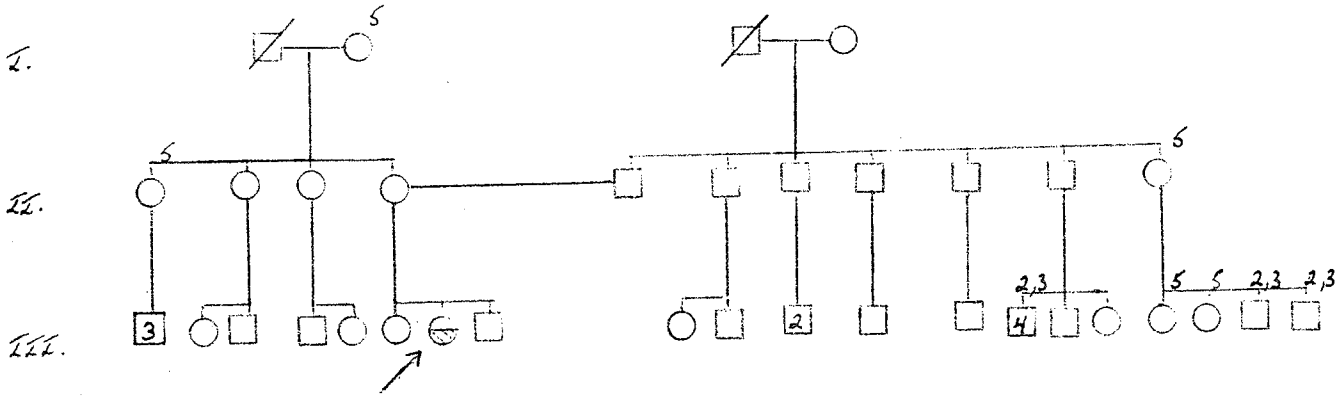
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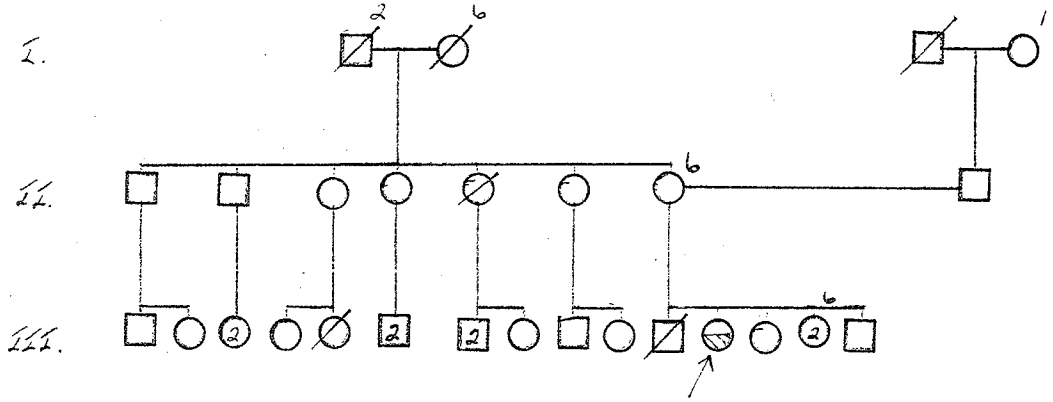
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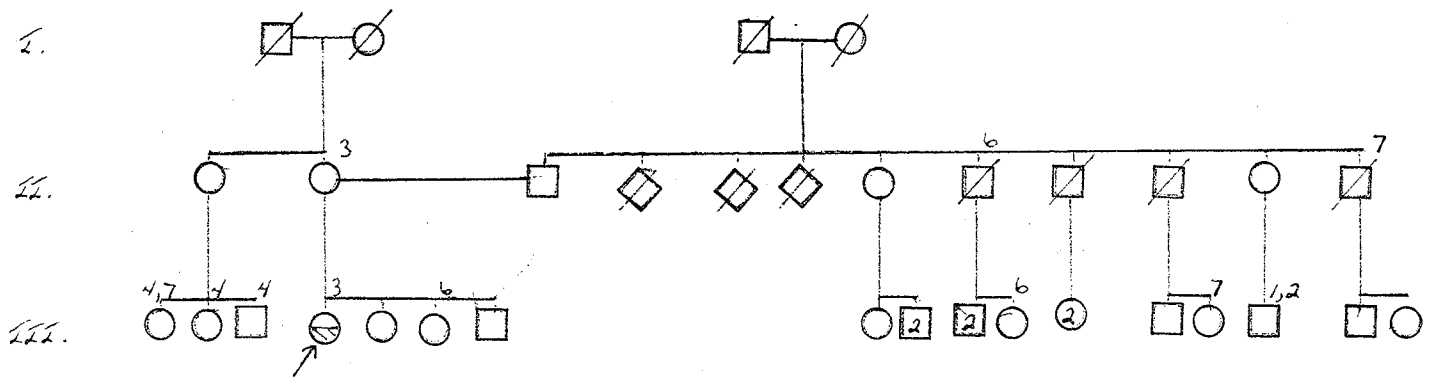
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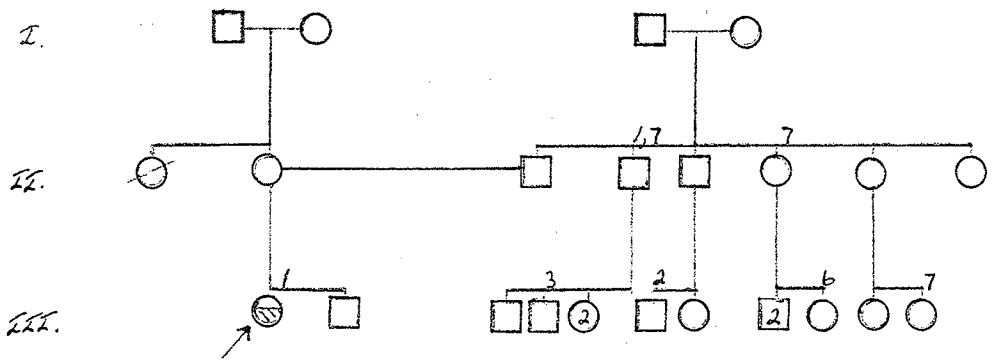
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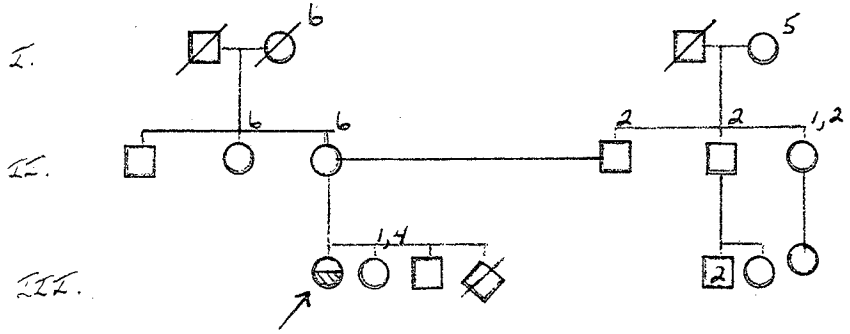
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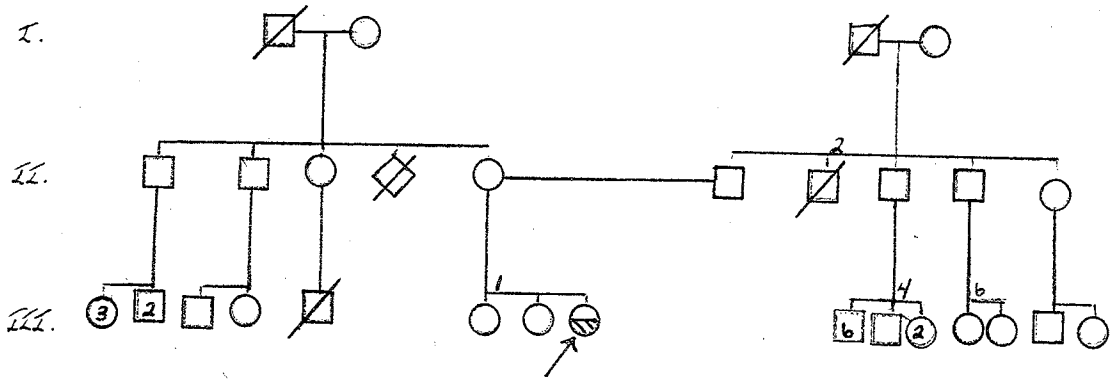
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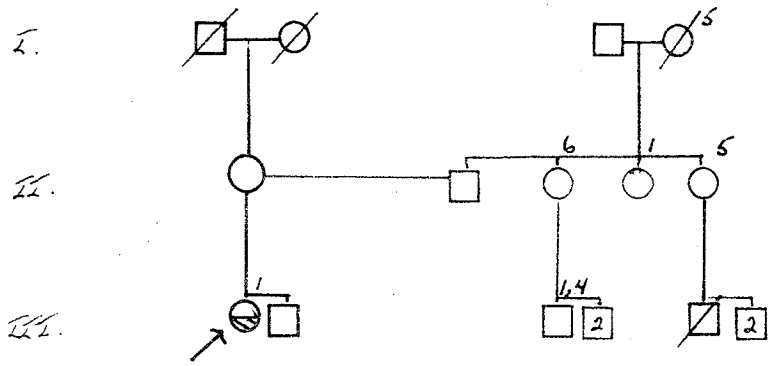
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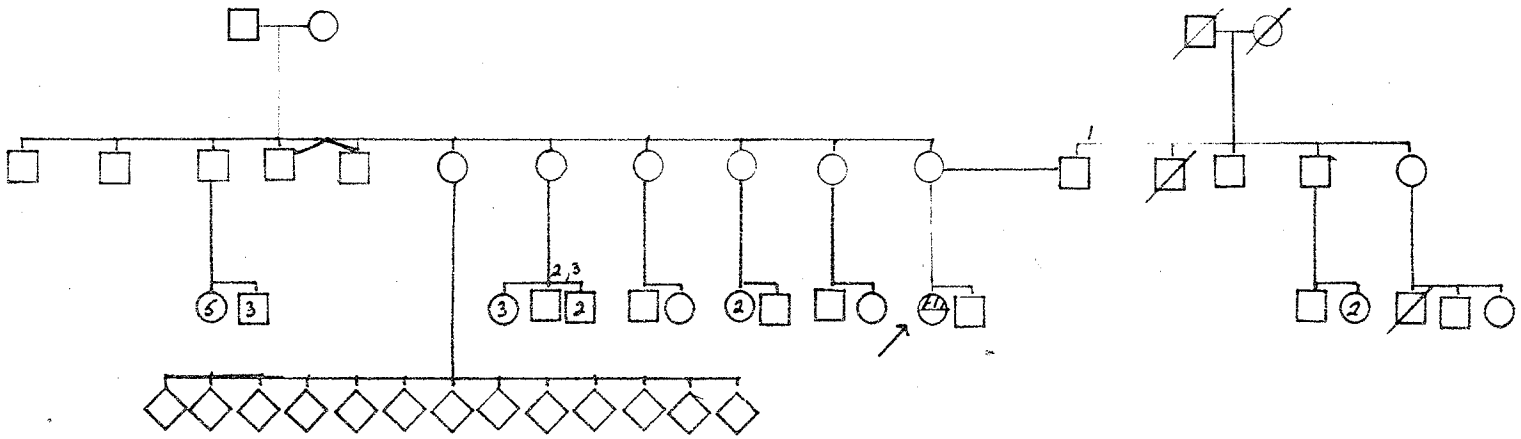
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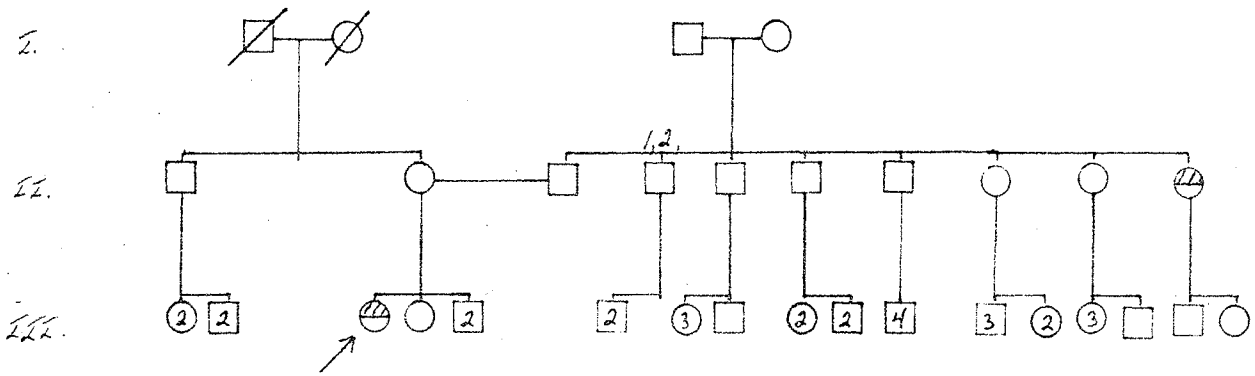
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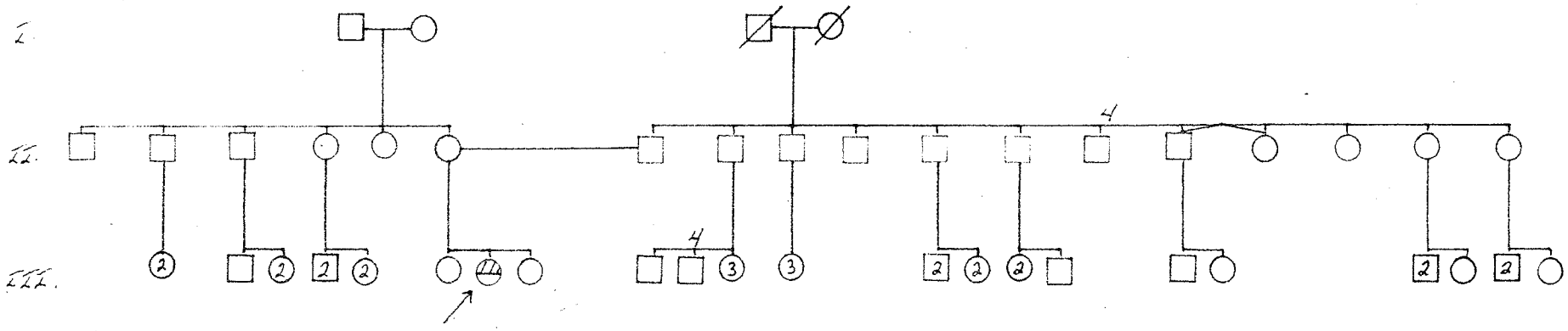
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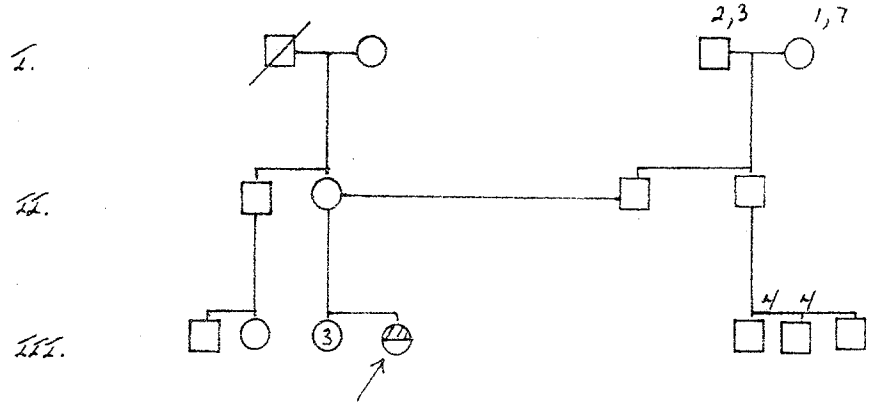
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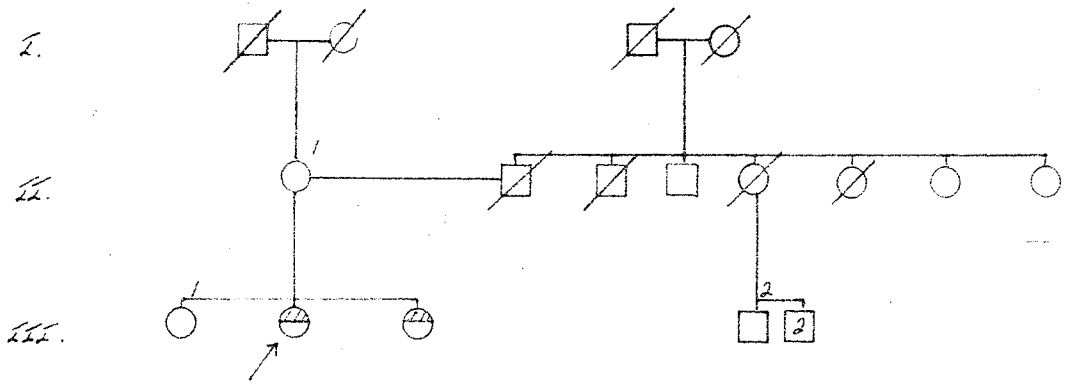
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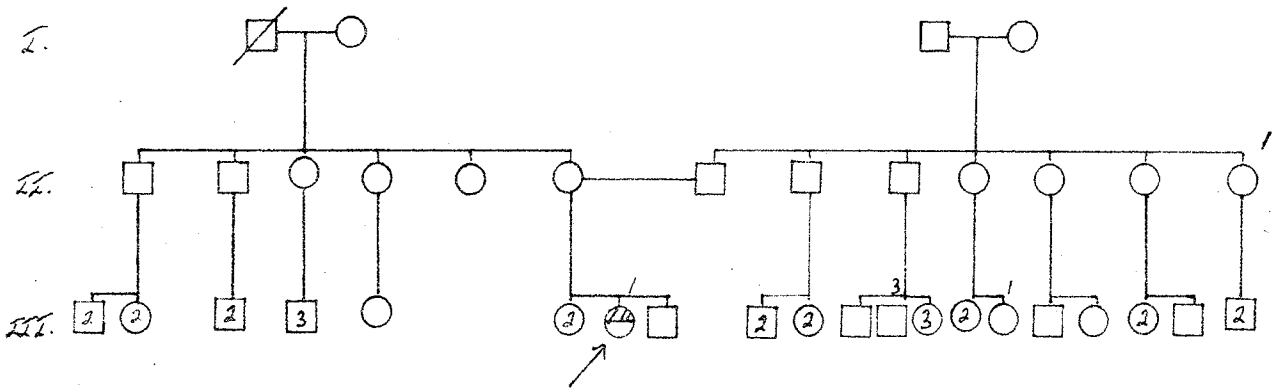

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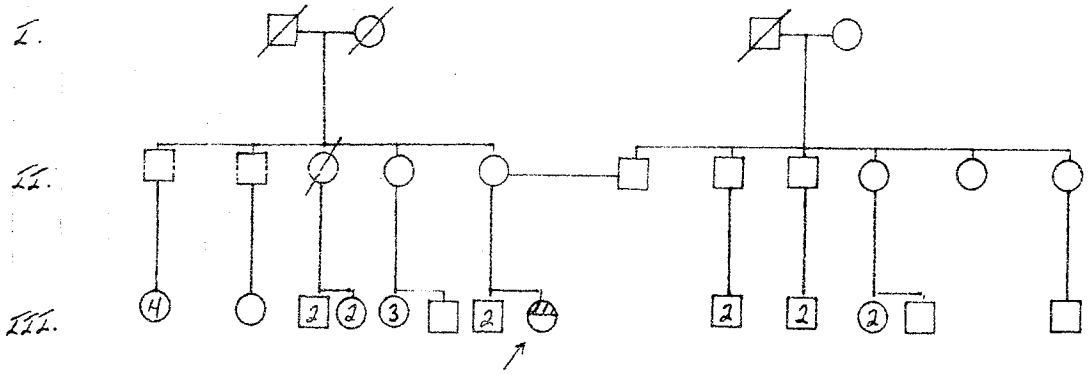

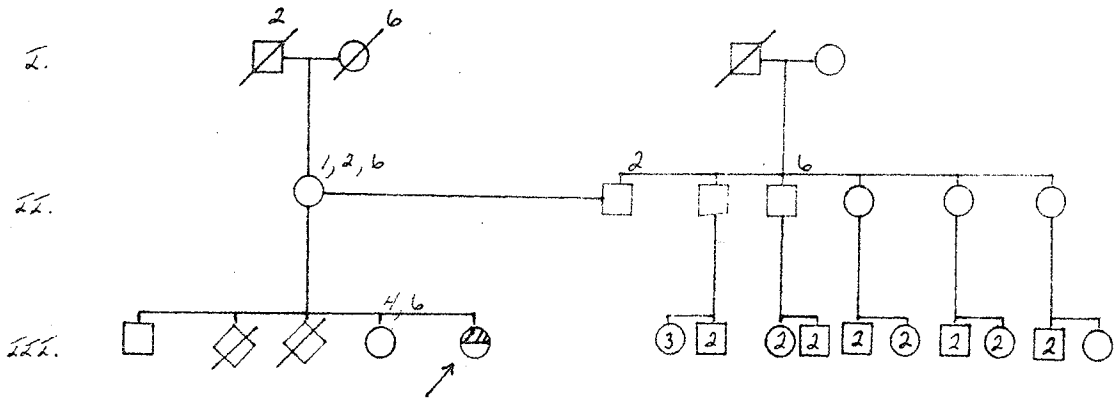
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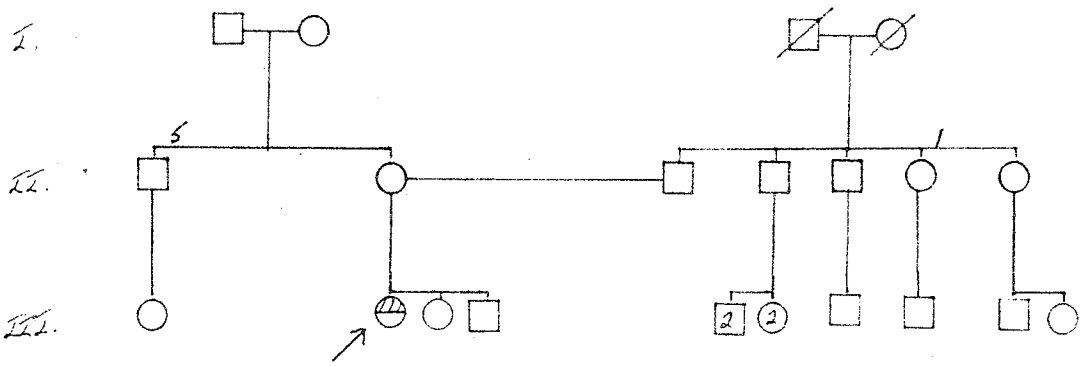
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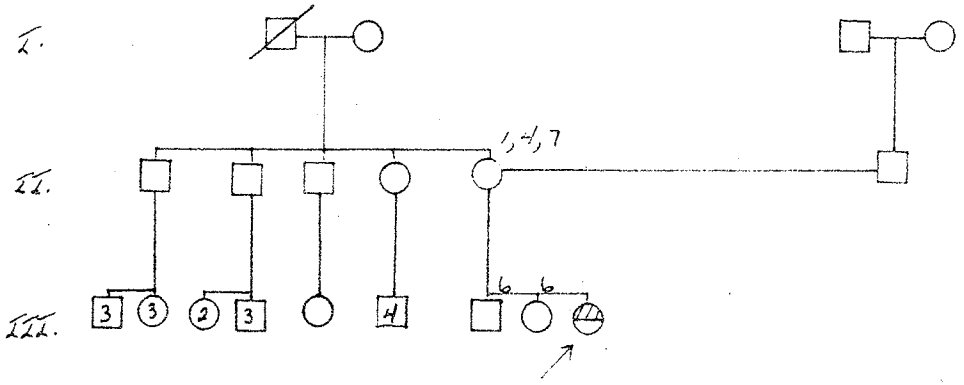

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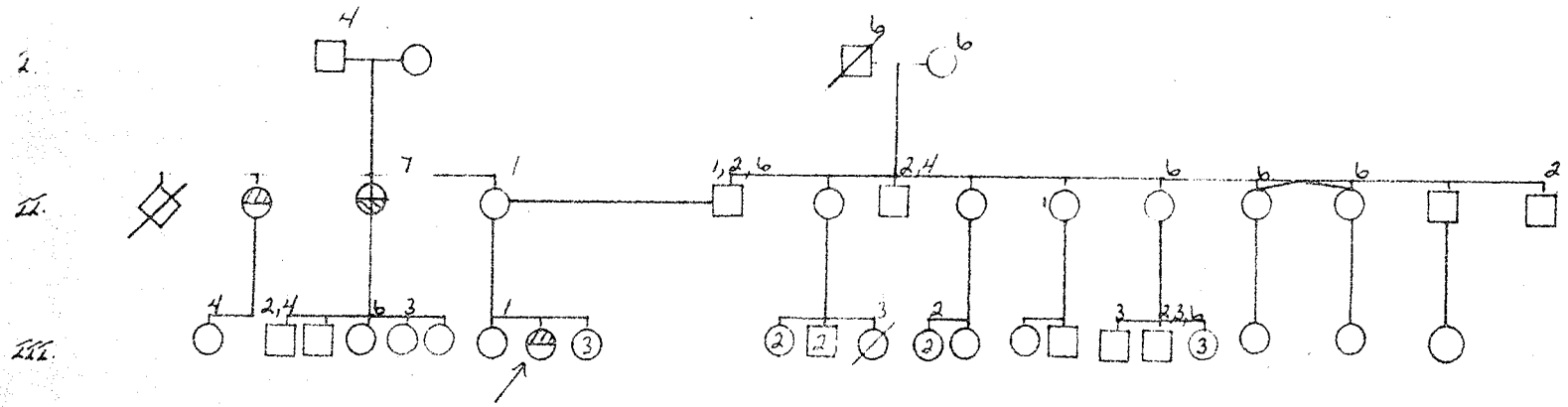

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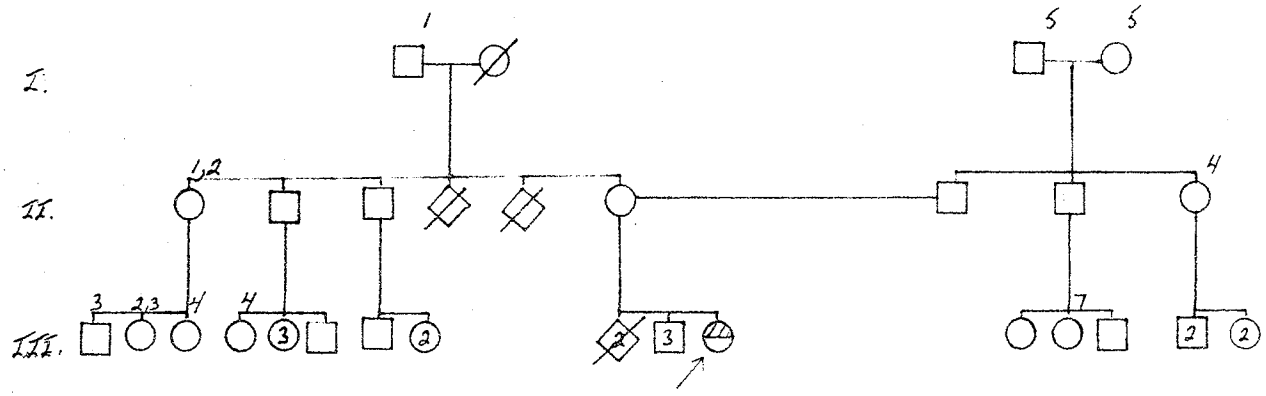

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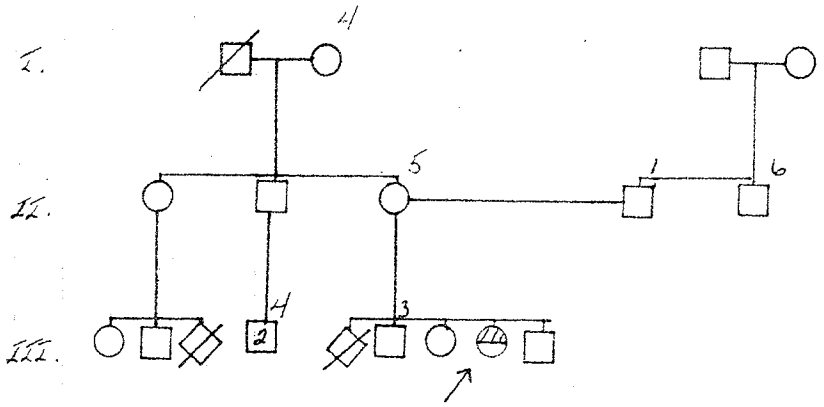

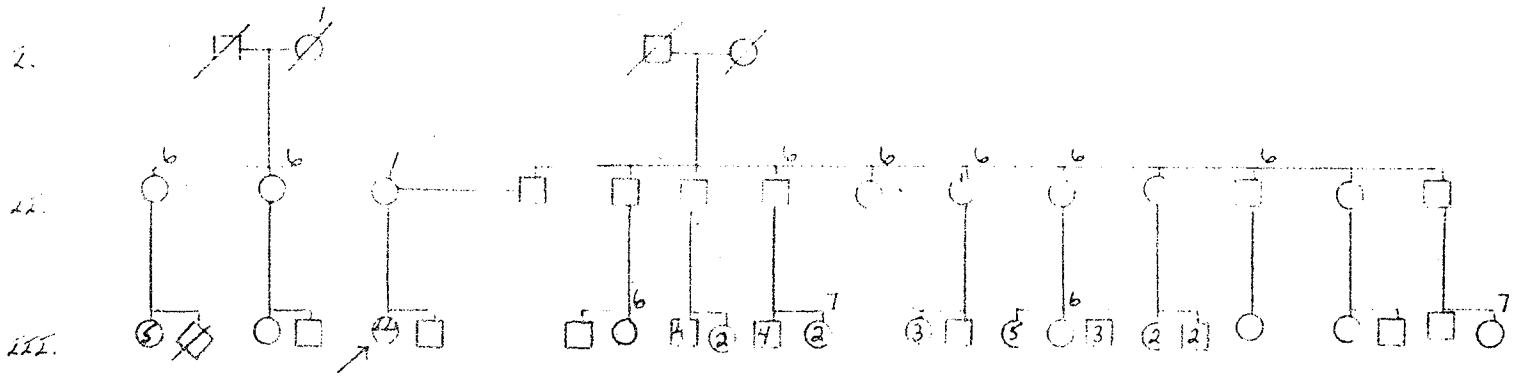
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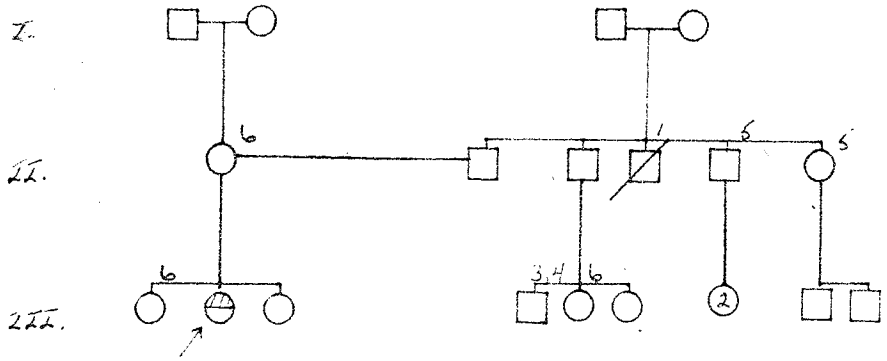

APPENDIX G

Pedigrees - By Group and Family - Controls

Family #	1° Relatives		2° Relatives		3° Relatives		1° Relatives			
							Females		Males	
	Female	Male	Female	Male	Female	Male	Mothers	Sisters	Fathers	Brothers
1.	2	2	5	6	7	8	1	1	1	1
2.	2	1	5	7	3	4	1	1	1	0
3.	2	1	5	7	3 *1	4	1	1	1	0
4.	2	1	5	8	15	15	1	1	1	0
5.	1	3	2	4	0	3	1	0	1	2
6.	2	1	3	3	3	1	1	1	1	0
7.	2	3	7	2	7	6	1	1	1	2
8.	1	2	5	6	7	7	1	0	1	1
9.	1	2	8	6	7	8	1	0	1	1
10.	2	1	4	8	7	11	1	1	1	0
11.	4	2	8	8	18	19	1	3	1	1
12.	2	4	5	6	4	7	1	1	1	3
13.	2	1	6	3	9	5	1	1	1	0
14.	1	3	3	8	7	13	1	0	1	2
15.	3	2	5	7	3	8	1	2	1	1
16.	2	3	3	4	4	4	1	1	1	2
17.	2	3	4	4	0	2	1	1	1	2
18.	3	2	3	5	4	2	1	2	1	1
19.	2	3	8	6	5	2	1	1	1	2
20.	1	2	8	10	6	8	1	0	1	1
Total	39	42	102	118	119	137	20	19	20	22
Plus Subjects	20									
Grand Total	59	42	102	118	119	137	0% affected	0% affected		

* = No. affected

Pedigrees - Bulimics

Family #	1° Relatives		2° Relatives		3° Relatives		1° Relatives			
							Females		Males	
	Female	Male	Female	Male	Female	Male	Mothers	Sisters	Fathers	Brothers
1.	2	3	2	2	0	1	1	1	1	2
2.	2	4	7	8	12	16	1	1	1	3
3.	2	1	6	4	7	4	1	1	1	0
4.	2	1	2	3	0	2	1	1	1	0
5.	2	1	6	5	5	12 *1	1	1	1	0
6.	3 *1	1	3	3	3 *1	0	1	2 *1	1	0
7.	2	3	3	5	7	5	1	1	1	2
8.	4 *1	6	4	2	1	1	1	3 *1	1	5
9.	1	2	4	4	2	2	1	0	1	1
10.	1	3	4	10	10	12	1	0	1	2
11.	1	2	4	5	10	5	1	0	1	1
12.	1	2	12	7	38 *2	28	1	0	1	1
13.	2	3	6	5	8	3	1	1	1	2
14.	2	2	6	7	6	17	1	1	1	1
15.	4	2	6	4	7	6	1	3	1	1
16.	3	2	5	6	8	8	1	2	1	1
17.	1	2	6	4	6	5	1	0	1	1
18.	2	2	4	4	2	2	1	1	1	1
19.	3	1	4	7	9	12	1	2	1	0
20.	1	2	5	3	0	6	1	0	1	1
Total	41	45	99	98	141	147	20	21	20	25
Plus Subjects	20									
Grand Total	61	45	99	98	141	147	0% affected	9.5% affected		

* = No. affected

Pedigrees - Anorexics

Family #	1° Relatives		2° Relatives		3° Relatives		1° Relatives			
							Females		Males	
	Female	Male	Female	Male	Female	Male	Mothers	Sisters	Fathers	Brothers
1.	1	2	8	8	15	12	1	0	1	1
2.	2	3	5 *1	7	13	16	1	1	1	2
3.	3	1	8	12	19	13	1	2	1	0
4.	4	1	2	4	1	4	1	3	1	0
5.	3 *1	1	6	4	0	3	1	2 *1	1	0
6.	3	2	9	6	14	15	1	2	1	1
7.	1	3	7	6	12	9	1	0	1	2
8.	2	2	5	4	12	10	1	1	1	1
9.	2	2	4	5	4	5	1	1	1	1
10.	2	2	3	5	6	10	1	1	1	1
11.	5	1	10 *2	5	17	7	1	4	1	0
12.	1	4	4	5	12	6	1	0	1	3
13.	2	3	3	4	1	3	1	1	1	2
14.	1	2	9	7	25	19	1	0	1	1
15.	3	1	3	5	4	3	1	2	1	0
Total	35	30	86	87	155	135	15	20	15	15
Plus Subjects	15									
Grand Total	50	30	86	87	155	135	0% affected	5% affected		

* = No. affected

APPENDIX H

Questionnaire Results

Question	Controls (N=20)	Anorexics (N=15)	Bulimics (N=20)	Combined Bulimics & Anorexics (N=35)
2. Age of subject ($\bar{X}$)(yrs.)	21.3 (SD=7.6)	21.1 (SD=6.3)	23.7 (SD=7.5)	22.6 (SD=7.0)
3. Sex: Males	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)
Females	20 (100%)	15 (100%)	20 (100%)	35 (100%)
4. Subject's height ($\bar{X}$)	65.0 (SD=2.4)	64.0 (SD=1.9)	64.9 (SD=3.2)	64.5 (SD=2.7)
5. Subject's weight ($\bar{X}$) (lbs.)	126.7 (SD=19.8)	99.1**† (SD=16.3) ($p<.000$)	117.1**† (SD=18.2) ($p<.005$)	109.1**† (SD=19.4) ($p<.002$)
6. Ethnic origin: Caucasian	20 (100%)	15 (100%)	20 (100%)	35 (100%)
7. Mother's present age ($\bar{X}$)(yrs.)	47.0 (SD=8.7)	47.3 (SD=7.7)	51.4 (SD=7.9)	49.7 (SD=8.0)
8. Mother's ethnic origin: Caucasian	20 (100%)	15 (100%)	20 (100%)	35 (100%)
9. Mother's SES: Class 1	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)
Class 2	4 (20.0%)	1 (6.7%)	3 (15.0%)	4 (11.4%)
Class 3	3 (15.0%)	4 (26.7%)	2 (10.0%)	6 (17.1%)
Class 4	7 (31.0%)	4 (26.7%)	10 (50.0%)	14 (40.0%)
Class 5	6 (30.0%)	6 (40.2%)	5 (25.0%)	11 (31.4%)
11. Father's present age ($\bar{X}$) (yrs.)	50.7 (SD=6.2)	50.9 (SD=8.6)	54.9 (SD=10.1)	53.2 (SD=9.5)

Question	Controls	Anorexics	Bulimics	Combined Bulimics & Anorexics
12. Father's ethnic origin: Caucasian	20 (100%)	15 (100%)	20 (100%)	35 (100%)
13. Father's SES: Class 1	6 (30.0%)	1 (6.7%)	2 (10.0%)	3 (8.6%)
Class 2	2 (10.0%)	4 (26.7%)	3 (15.0%)	7 (20.0%)
Class 3	3 (15.0%)	3 (20.1%)	4 (20.0%)	7 (20.0%)
Class 4	5 (25.0%)	6 (40.2%)	10 (50.0%)	16 (45.7%)
Class 5	4 (20.0%)	1 (6.7%)	1 (5.0%)	2 (5.7%)
15. Subject's age when problem started ($\bar{X}$) (yrs.)	-	17.6 (SD=4.7)	19.2 (SD=7.3)	18.5 (SD=6.3)
16. Subject's occupation: (Hollingshead-Redlick Index, 1958) ($\bar{X}$)	48.0 (SD=4.7)	40.6** (SD=11.6) (p<.033)	41.7 (SD=10.5)	41.2**† (SD=10.8) (p<.002)
17. Any other members of family have this problem? Yes:	0 (0.0%)	2 (13.4%)	3 (15.0%)	5 (14.3%)
18. Do you know of anyone else with this problem? Yes:	9 (45.0%)	13* (86.7%) (p<.0116)	17*† (85.0%) (p<.008)	30*† (85.7%) (p<.0014)
19. Anyone else in family have psych. problems? Yes:	0 (0.0%)	5* (33.3%) (Sum p=.009)	9*† (45.0%) (p<.0006)	14* (40.0%) (Sum p=.005)
20. Anyone else in family seen a psychologist/ psychiatrist? Yes:	5 (25.0%)	7 (46.7%)	13* (65.0%) (p<.0066)	20* (57.1%) (p<.0161)
21. Food and/or eating was a "special issue" when growing up? Yes:	1 (5.0%)	7*† (46.7%) (Sum p=.0057)	9*† (45.0%) (p<.0035)	16*† (45.7%) (p<.0017)

Question	Controls	Anorexics	Bulimics	Combined Bulimics & Anorexics
22. Have you ever had any other psych/med. problems? Yes:	5 (25.0%)	6 (40.2%)	9 (45.0%)	15 (42.9%)
23. Mother's age at birth of affected child ($\bar{X}$) (yrs.)	27.3 (SD=5.6)	26.3 (SD=3.4)	28.2 (SD=6.4)	27.4 (SD=5.3)
24. Mother's height ($\bar{X}$)(in.)	63.3 (SD=2.0)	64.4 (SD=2.5)	64.6 (SD=2.0)	64.5 (SD=2.2)
25. Mother's weight before pregnancy with subject: ($\bar{X}$)(lbs.)	126.6 (SD=16.1)	120.4 (SD=8.3)	132.3**† (SD=14.8) (p<.007)	127.4 (SD=13.7)
26. Number of pregnancies in total: ($\bar{X}$)	3.1 (SD=1.1)	4.0** (SD=1.4) (p<.043)	3.9 (SD=1.8)	3.9** (SD=1.6) (p<.047)
27. Livebirths:	58	49	65	114
28. Miscarriages:	4	11	7	18
29. Difficulties with pregnancies: Yes:	5 (25.0%)	4 (26.7%)	7 (35.0%)	11 (31.4%)
30. Any other children with psych/med. problems? Yes:	6 (30.0%)	4 (26.7%)	9 (45.0%)	13 (37.1%)
31. Mother's occupation prior to subject's birth: (Hollingshead-Redlick Index (1958) ($\bar{X}$)	32.9 (SD=14.7)	42.9 (SD=9.1)	37.2 (SD=14.5)	39.7 (SD=12.7)
32. Hazards: Yes:	1 (5.0%)	1 (6.7%)	1 (5.0%)	2 (5.7%)
33. Major of chronic illness during pregnancy with subject? Yes:	0 (0.0%)	0 (0.0%)	2 (10.0%)	2 (5.7%)
34. Acute illness: Yes:	0 (0.0%)	0 (0.0%)	1 (5.0%)	1 (2.9%)
35. Pregnancy planned: Yes:	8 (40.0%)	11 (73.7%)	7 (35.0%)	18 (51.4%)

Question		Controls	Anorexics	Bulimics	Combined Bulimics & Anorexics
36. Contraception practised Yes:		5 (25.0%)	6 (40.2%)	10* (50.0%) (p<.0233)	16* (45.7%) (p<.0467)
37. Type:	Condom	1 (5.0%)	1 (6.7%)	4 (20.0%)	5 (14.3%)
	BCP	0 (0.0%)	1 (6.7%)	0 (0.0%)	1 (2.9%)
	IUD	0 (0.0%)	0 (0.0%)	1 (5.0%)	1 (2.9%)
	Foam or jelly	1 (5.0%)	0 (0.0%)	1 (5.0%)	1 (2.9%)
	Rhythm	0 (0.0%)	0 (0.0%)	1 (5.0%)	1 (2.9%)
	Diaphragm	0 (0.0%)	3 (20.1%)	3 (15.0%)	6 (17.1%)
	Other	3 (15.0%)	1 (6.7%)	0 (0.0%)	0 (0.0%)
38. Any drugs taken during pregnancy with subject? Yes:		9 (45.0%)	3 (20.1%)	9* (45.0%) (p<.0155)	12 (34.3%)
39. Weight gain during pregancy: ($\bar{X}$) (lbs.)		21.0 (SD=7.8)	24.2 (SD=8.3)	29.1**† (SD=10.9) (p<.014)	27.0** (SD=10.0) (p<.031)
40. Size and movement of this pregnancy compared to others: Same:		15 (75.0%)	10 (67.0%)	10 (50.0%)	20 (57.1%)
41. Any problems with delivery? Yes:		0 (0.0%)	0 (0.0%)	3 (15.0%)	3 (8.6%)
42. Was this child breast or bottle fed?	Breast	11 (55.0%)	6 (40.2%)	8 (40.0%)	14 (40.0%)
	Bottle	5 (25.0%)	6 (40.2%)	11 (55.0%)	17 (48.6%)
	Both	4 (20.0%)	3 (20.1%)	1 (5.0%)	4 (11.4%)

Question	Controls	Anorexics	Bulimics	Combined Bulimics & Anorexics
43. Type of schedule: Rigid =	7 (35.0%)	2 (13.4%)	5 (25.0%)	7 (20.0%)
Demand =	13 (65.0%)	9 (60.3%)	13 (65.0%)	22 (62.9%)
44. Feeding or other difficulties: Yes:	1 (5.0%)	1 (6.7%)	2 (10.0%)	3 (8.6%)
45. Type of baby/child: Good =	17 (85.0%)	14 (93.8%)	14 (70.0%)	28 (80.0%)
Colicky =	2 (10.0%)	1 (6.7%)	5 (25.0%)	6 (17.1%)
46. Any major, chronic or acute illnesses after pregnancy: Yes:	3 (15.0%)	4 (26.8%)	3 (15.0%)	7 (20.0%)
47. Anyone in immediate family been hospitalized for a med/psych. illness: Yes:	9 (45.0%)	7 (16.9%)	5 (25.0%)	12 (34.3%)
*48. Any period of time when Mom was separated from daughter: Yes:	5 (25.0%)	5 (33.5%)	11* (55.0%) ($p<.0151$)	16 (45.7%)
Age of child at separation ($\bar{X}$) (yrs.)	8.2 (SD =6.1)	8.4 (SD=7.2)	8.6 (SD=7.5)	8.5 (SD=7.1)
Length of separation (weeks): ($\bar{X}$)	3.2 (SD=2.2)	4.0 (SD=1.9)	2.1 (SD=1.4)	2.7 (SD=1.8)
49. Has Mom ever had a psych. illness? Yes:	3 (15.0%)	3 (20.1%)	4 (20.0%)	7 (20.0%)
50. Were you ever hospitalized? Yes:	0 (0.0%)	2 (13.4%)	0 (0.0%)	2 (5.7%)
51. Anyone in Mon's distant family ever had a psych. illness? Yes:	7 (35.0%)	4 (26.8%)	11* (55.0%) ($p<.0314$)	15 (42.9%)

Question	Controls	Anorexics	Bulimics	Combined Bulimics & Anorexics
52. Anyone in Mom's family ever seen a psychologist/psychiatrist? Yes:	5 (25.0%)	10* (67.0%) (p<.0186)	9* (45.0%) (p<.0475)	19* (54.3%) (p<.0116)
53. Father's age at birth of affected child: ($\bar{X}$) (yrs.)	30.9 (SD=7.0)	30.1 (SD=5.1)	30.7 (SD=5.8)	30.4 (SD=5.5)
55. Father's occupation prior to birth of subject (Hollingshead-Redlick Index, 1958): ($\bar{X}$)	28.4 (SD=16.3)	23.9 (SD=12.3)	25.2 (SD=12.5)	24.5 (SD=12.2)
56. Hazards: Yes:	5 (25.0%)	3 (20.1%)	4 (20.0%)	7 (20.0%)
57. Was this a planned pregnancy? Yes:	8 (40.0%)	10 (67.0%)	5 (25.0%)	15 (42.9%)
58. Was contraception practised? Yes:	5 (25.0%)	6 (40.2%)	6 (30.0%)	12 (34.3%)
59. Type of contraception:				
Condom	1 (5.0%)	2 (13.4%)	2 (10.0%)	4 (11.4%)
BCP	0 (0.0%)	1 (6.7%)	0 (0.0%)	1 (2.9%)
Foam or jelly	1 (5.0%)	0 (0.0%)	1 (5.0%)	0 (0.0%)
Rhythm	0 (0.0%)	0 (0.0%)	1 (5.0%)	1 (2.9%)
Diaphragm	0 (0.0%)	2 (13.4%)	2 (10.0%)	4 (11.4%)
Other	3 (15.0%)	1 (6.7%)	0 (0.0%)	0 (0.0%)
60. Did Dad participate in feeding? Yes:	14 (70.0%)	10 (67.0%)	6 (30.0%)	16 (45.7%)
61. Type of schedule:				
Rigid =	3 (15.0%)	0 (0.0%)	1 (5.0%)	1 (2.9%)
Demand =	10 (50.0%)	6 (40.2%)	3 (15.0%)	9 (25.7%)

Question	Controls	Anorexics	Bulimics	Combined Bulimics & Anorexics
62. Difficulties: Yes:	0 (0.0%)	1 (6.7%)	0 (0.0%)	1 (2.9%)
63. Any major, chronic or acute illnesses after birth of child: Yes:	6 (30.0%)	4 (26.8%)	4 (20.0%)	8 (22.9%)
64. Any period of time when Dad was separated from daughter? Yes:	9 (45.0%)	6 (40.2%)	13 (65.0%)	19 (54.3%)
Age of child at separation: ($\bar{X}$)(yrs.)	5.7 (SD=5.5)	8.4 (SD=4.5)	7.9 (SD=5.9)	8.0 (SD=5.4)
Length of separation (weeks): ($\bar{X}$)	4.3 (SD=1.8)	3.7 (SD=2.3)	7.4 (SD=12.9)	6.2 (SD=10.8)
65. Has Dad ever had a psych. illness? Yes:	4 (20.0%)	2 (13.4%)	1 (5.0%)	3 (8.6%)
66. Were you hospitalized? Yes:	1 (5.0%)	1 (6.7%)	0 (0.0%)	1 (2.9%)
67. Anyone in Dad's distant family ever had a psych. illness? Yes:	4 (20.0%)	5 (33.5%)	5 (25.0%)	10 (28.6%)
68. Anyone in Dad's family ever seen a psychologist/psychiatrist? Yes:	3 (15.0%)	3 (20.1%)	5 (25.0%)	8 (27.9%)

* = Comparison of groups by χ^2 or Fisher's Exact Tests

** = Comparison of groups by t-tests.

† = Significant at the corrected α level.