

Nature and Nurture in Anorexia Nervosa: A Study of 34 Pairs of Twins, One Pair of Triplets, and an Adoptive Family

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This paper reports observations following on a study of anorexia nervosa in 34 twins and one set of triplets, and within which approximately half the monozygotic (MZ) twins displayed concordance, compared with 7% of the dizygotic (DZ) twins. One additional case, involving childhood adoptions, is described, wherein neither genetic nor specifically assortative selection factors seem to have been at work. The findings suggest that genetic and environmental factors can interact to contribute to the development of this apparently culture-based and multifactorially derived condition. A number of possible genetic strands to the condition are proposed; probably not all of them are specifically necessary for its development. The same may apply to experiential influences. Elsewhere it has been argued that the one essential characteristic of anorexia nervosa is avoidance behavior and the factors permitting and facilitating it (Crisp, 1980). Experiential influences, in the form of psychological maturational challenges in adolescence impinging on constitutional propensities to the point of precipitating the disorder out, would seem often themselves to have been influenced by constitutional psychological characteristics and earlier childhood experiences. A further pooling and study of all known twin and adoptive cases might now advance our knowledge of this condition further.

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One hopes that twin studies can contribute to our understanding of anorexia nervosa. There is considerable evidence that the disorder is at least in part culture-bound. Evidence for any genetic component in the disorder has so far been inconclusive. At a clinical level, certain factors have been reported as being overrepresented in anorectic families. These include psychoneurosis (Kay & Leigh, 1954), obesity (Huenemann et al., 1966; Crisp, 1970a), depressive illness (Kalucy et al., 1977; Winokur et al., 1980), social anxiety and such responses as agoraphobia and family enmeshment behavior (Crisp & Toms, 1972; Kalucy et al., 1977), latent extraversion/impulsivity, alcoholism, a concern over weight and "middle class attitudes" (Crisp, 1980). In 1967, Crisp, studying 18 massively obese older females, found evidence of a clear previous episode of anorexia nervosa in two (10%) of them. Theander (1970) has reported a 6.6% sibling risk of anorexia nervosa in his series of 94 female anorectic patients. In a study of 102 anorectic patients, Crisp et al. (1980) found that 7% of the siblings provide clear evidence of having had or of still having anorexia nervosa, while 14% of the mothers and 9% of the fathers had a history consistent with the diagnosis of anorexia nervosa, albeit sometimes marginal; a further 10% of the mothers were obese. A recent study (Hudson et al., 1983) reported a 5.9% occurrence of eating disorders among the 70 first-degree relatives of 14 anorectic probands. In a study of 13 male anorectic patients, Crisp and Toms (1972) reported that anorexia nervosa was present in three siblings and suggested that the siblings were "taking turns" in stabilizing the family dynamics through their anorectic behavior.

The reports on twins with anorexia nervosa have been reviewed by four groups of authors (Askevold & Heiberg, 1979; Vandereycken & Pierloot, 1981; Garfinkel & Garner, 1982; Nowlin, 1983). Garfinkel and Garner (1982) list 27 pairs from the literature and add nine pairs of their own. Many of these reports can be criticized as zygoty was not clearly established and diagnostic criteria not stated. Depending on the strictness of the inclusion criteria, between 38 and 55% of the 27 monozygotic (MZ) pairs were concordant for anorexia nervosa. Nine dizygotic (DZ) pairs have been reported. The five in Garfinkel and Garner's series and four reported separately (Dickens, 1970; Nemeth, 1977; Beumont, Abraham, Argall, George, & Gluan, 1978) were all discordant. One pair reported by Morgan and Russell (1975), and also included in the present study, was concordant for anorexia nervosa. Crisp (1966), reporting on a MZ twin pair discordant for anorexia nervosa and reviewing the literature of the time, suggested that genetically influenced components in anorexia nervosa might include an underlying tendency towards obesity, high growth rate, and the accompanying sustained insulin response to a glucose load; also CNS "immaturity," as evidenced by more common than usual EEG spike and wave activity.

Table 1. Zygosity of the 34 twin pairs and one set of triplets and the mean age of each group (years), at the time of inclusion in the study.

	MZ		Female DZ		F/M DZ		Total
	Number	Age	Number	Age	Number	Age	
St. George's	5 ^a	29	8	24	2	25	15
Maudsley	6 ^b	27	4	24	1	22	11
Volunteers	7	25	2	25	—	—	9
Total	18	27	14	24	3	23	35

^aIncluding one set of male triplets.

^bIncluding one set of male twins.

The aims of the present study have been, first, to investigate concordance rates for anorexia nervosa and related disorders in a relatively unselected and larger series of anorectic twins and one set of triplets. The methodology and basic findings are reported elsewhere (Holland, Hall, Murray, Russell, & Crisp, in press). The study was collaborative between St. George's and the Maudsley Hospitals. A total of 34 pairs of twins and one set of triplets was identified in which the proband had anorexia nervosa. The twins and their parents were interviewed and relevant case notes examined. Diagnosis was based on standard criteria and the zygosity of the twin pairs was established by blood group analysis or by the use of the physical resemblance questionnaire.

In the 30 female twin pairs (nearly all by now past the age of being at serious risk of developing anorexia nervosa if it had not already arisen; see Table 1), 55% of the MZ and 7% of the DZ pairs were concordant for anorexia nervosa (Table 2). This difference was significant. Three of the non-anorectic co-twins had other psychiatric illness and two had a history of minor eating disorders. None of the male DZ co-twins had anorexia nervosa.

The present paper is concerned with further exploration of these findings in the light of clinical views concerning anorexia nervosa. The 7% concordance of the DZ twins is very similar to the occurrence of anorexia nervosa reported in non-twin siblings. It has been argued that DZ twins have a greater socially and existentially driven impulse compared with other siblings to differentiate themselves from each other. At the same time, they may be hampered in this by being treated by others as more similar than non-twin siblings. Then again, three of the DZ twin siblings were male. None had anorexia nervosa and the condition is very rare in the male. This finding is therefore difficult to interpret.

The 55% concordance rate in the MZ twins, all raised together, invites both genetic and environmental explanations. One might search for possible genetic predispositions which would become manifest under con-

Table 2. Concordance for anorexia nervosa among the 30 female twin pairs.

	MZ	DZ	Total
Concordant	9(55%)	1(7%)	10
Discordant	7	13	20
Total	16	14	30

^a $\chi^2 = 6.91, p < .01$.

ditions such as dieting or emotional stress. Such genetic vulnerabilities, not always necessary for the condition to develop, obviously include the gene determining *female* sex, and in addition may include specific nutritional/growth characteristics such as a *tendency to obesity, high growth rate* and *hyperactivity*. *High impulsivity/extraversion* (associated with high growth rate—see Crisp, 1970a—and sometimes a sense of loss of control arising in adolescence), on the one hand, and high *introversion*, on the other, have a genetic factor within them (Shields, 1962). Cognitive coping styles incorporated within an overall *unidimensional*, and therefore relatively inflexible and poorly adaptive *construct system*, which could come to accommodate ideas concerning personality in terms of body shape (Crisp & Fransella, 1972), may also have a genetically determined element to them. Less specifically, high levels of *anxiety, sadness, and/or low self-esteem* may be genetically shaped and certainly often seem inherent in and relevant to the condition, as may also be an *inability to cope with, tolerate or defend less self-destructively* against such experience and to react instead by *avoidance behavior*. Thus, MZ twins both carrying one or more vulnerability factors would have similar potential for developing anorexia nervosa under conditions of stress. This itself might be an experience any genetic vulnerability alone causes proportionately less frequently. Given the probandwise rate of the unselected Maudsley series and assuming a maximum prevalence of anorexia nervosa in young females of 1%, it is possible to attempt a calculation of the relative influence of genetic and nongenetic familial effects (Gottesman & Carey, 1983). This involves the use of a graphic solution, worked out by Smith (1974), in which the population risk is plotted along the horizontal axis and the risk observed in relatives on the vertical axis. The graph enables the correlation in liability between relatives to be estimated and thus the relative genetic and environmental influences calculated. This assumes that the particular disorder in question has a normally distributed liability and that this liability is the final common pathway for all genetic and environmental influences. The heritability calculated in this way, for anorexia nervosa, is .56 and the nongenetic familial effects, .38.

It may be, then, that there are potentially many strands of genetic contribution to anorexia nervosa. The matter may have much to do with the

way that these come together and become reinforced or muted by the nurturant developmental experience in early life and childhood, in the first instance. For example, at a physical level, potentially frightening high growth rate might be muted by childhood infection or undernutrition. Shields (1962) pointed out many years ago that, when major personality differences existed between twins, this was sometimes importantly related to the unilateral intervention of some disrupting physical factor. In another instance, the genetic potential for introversion might be minimized by a family structure characterized by a competent, adaptable and discriminating relationship to the outside world, while a young child's natural dependency and undifferentiated characteristics may be enhanced by overprotective family attitudes, generalized or specific to it [e.g., with one of a twin pair on the basis of real differences or through psychological splitting by the parent(s)], and excessive rewarding of compliant behavior. The potential for avoidance behavior in the child may be reinforced as natural fearfulness of the outside world by the parents' own enmeshed relationship and their associated fear of and avoidance of outside influences. This latter fear will be much enhanced in them as well as in their child when she/he reaches adolescence, begins to interact with the culture, discovers sexuality as the new interpersonal currency, and challenges their adjustments afresh (Crisp, 1980).

If the parents' reaction under these circumstances is, for instance, either for conflict to escalate between themselves or else for there to be a united and reinforced enmeshment, then perhaps anorexia nervosa as an avoidance response may precipitate out in the adolescent daughter as a necessary resolution to the new family problem. This may be especially so if, at this stage, she is perceiving her destiny or fate as importantly related to and threatened by her new-found puberty "fatness." She may have experienced her puberty as largely unsought and, in addition to her fearful nature, she may lack assertive or robust alternative capacities to adapt to the new social pressures and to her parents' reactions. Such a response may be delayed, even arising substantially later in life, and not until a particular crisis precipitates it out. Such personal adolescent experiences for her will presumably now be influenced genetically, not only by the genetic influences on childhood development, but also by genetic determinants of such pervasive states of mind and body as anxiety, poor self-esteem, doubt, and impulsivity, operating afresh within the context of adolescence, and now perhaps locking into a dominating and inflexible unidimensional construct system within which "fatness" becomes superordinate and related to "bad" things.

The findings in the present study cannot in themselves be conclusive in terms of the role of genetic, developmental and experiential factors in the determination of anorexia nervosa. It could be argued that the high concordance rate in the MZ twins is a function of their identical treatment by others during childhood, just as the discordance might be accounted

for by differences between them in respect of childhood experiences. Puberty might anyway, with its new, usually heterosexual, outgoing impulse, be a potential threat to the special bond between identical twins. Joint regression would reestablish the bond. Not only would puberty be banished, but, it has been argued elsewhere (Crisp & Bhat 1982), anorexia nervosa reduces individuals experiencing it to a diminished lowest common denominator, characterized by the overriding and individually undifferentiated impact of starvation on the human system. This undifferentiated state might have special relevance for MZ twins. Alternatively, if one such twin were to break away from the bond in adolescence, then the other, abandoned in the process, might find no better resolution than to backtrack into childhood via anorexia nervosa (Crisp, 1966). In searching for further clinical evidence for such interactions and especially for possible environmental determinants of the condition, which might offer the greatest hope for intervention at the social level, this paper now concentrates on three areas.

Table 3. MZ twin pairs.

	Age of Onset	
	1st born	2nd born
Concordant (<i>n</i> = 9)	20	14
	21	17
	13	13
	15	18
	20	17
	15	17
	15	17
	14	15
	15	15
Mean age of onset (<i>n</i> = 18)	16.1 ± 2.4 years	
Mean age of 1st presenting twin (<i>n</i> = 9)	15.0 ± 1.3 years	
Mean age of 2nd presenting twin (<i>n</i> = 9)	17.3 ± 2.7 years	
	Age of Onset	
Discordant (<i>n</i> = 7)	14	
	18	
	18 (male)	
	16	
	25	
	15	
	17	
Mean age of onset (<i>n</i> = 7)	17.6 ± 3.6 years	

First, let us consider the seven out of 17 MZ twin pairs discordant for anorexia nervosa. There was no correlation between being the anorectic twin and the birth order or birth weight of the twins, nor between being anorectic and educational success. In the seven pairs in whom there was a history of adverse perinatal factors, these had predominantly affected the anorectic twin in five pairs and both twins in two pairs. Similarly, childhood neurotic traits had been present in the anorectic twin alone in four out of eight pairs. In none of the pairs did the non-anorectic twin alone have such a history. In four out of five pairs in which a difference was reported, the anorectic twin had been heavier in childhood. The anorectic twin was second to reach the menarche in four out of six pairs and was the less dominant in six out of eight pairs and in the set of triplets. These differences were not statistically significant, and may also have been distorted by retrospective bias.

Second, we examined age of onset of the condition in twins. Tables 3 and 4 show these ages. It can be seen that the mean age of onset of all but

Table 4. DZ twin pairs.

Age of Onset		
	1st born	2nd born
Concordant (<i>n</i> = 1)	15	17
Age of Onset		
Discordant (<i>n</i> = 16)	16	
	16	
	18	
	11	
	16	
	20	
	10	
	16	
	40	
	22	
	15	
	14	
	20	
	15	
	17	
	21	
Mean age of onset (<i>n</i> = 16)	17.9 ± 6.7 years	
Mean age of onset (<i>n</i> = 15 excluding 40-years-old)	16.5 ± 3.4 years	
Reference population (Crisp, 1973) (non-twins)		
Mean age of onset (<i>n</i> = 86)	17.6 ± 5.1 years	

the first presenting MZ twin is fairly similar to the mean age of onset of the condition in general. However, the first presenting twin of the MZ pairs had developed anorexia nervosa at a significantly earlier age than her co-twin, than the MZ-discordant twins, and the general population of anorectics. We are currently searching for correlates of this early presentation. Perhaps it reflects the special vulnerability of identical twins when faced with puberty.

Third, we describe two pairs of twins and one set of triplets within the study initially assessed by the first author 10 or more years ago, and mention again (Crisp, 1970a) an adoptive family wherein there were three biologically unrelated anorectic members.

The first three cases (Figure 1) concern, respectively, a DZ discordant female twin, a MZ discordant male triplet, a MZ female twin pair concordant between themselves and with their mother for the condition. The names have been changed.

CASE NO. 1

These twins presented in 1974, aged 12 years, with Anne having had anorexia nervosa for about two years. She weighed 21 kgs and was 1.36 m tall. Pauline weighed 35 kgs and was 1.48 m tall. Until the age of eight, they had always been the same height, but Pauline, the firstborn and slightly heavier at birth, had been slightly plumper. Anne's increasing fussiness with food from the age of eight onwards had crystallized out as obvious anorexia nervosa two years later, by which time she demonstrably had a fear of her increasing weight, which she openly wished to curb—this being rooted in its associated pubertal fatness, which she was clearly intent on banishing. The twins were considered to have been identical, although this was to turn out not to be the case. Anne had always been more anxious and highly strung, and the more dependent of the two, especially upon Pauline. During early childhood, both had been shy; but in recent years, Pauline had become increasingly independent. She had acquired other friends while Anne, from the age of five onwards, had preferred to stay in the home, often helping her mother with the cooking.

The father was a hard-working and successful farmer, dominated by his wife. She nursed deep feelings of inadequacy behind a facade of competence in the home. Her fears had escalated during her pregnancy with the twins, on discovering that she was carrying them and not a single fetus. Her fears had again escalated as the children had gotten older and needed to be taken out and about to school and the nearby market town. She battled against her agoraphobia. Within the home, she was perceived as strong but controlling. Two years previously, the other and

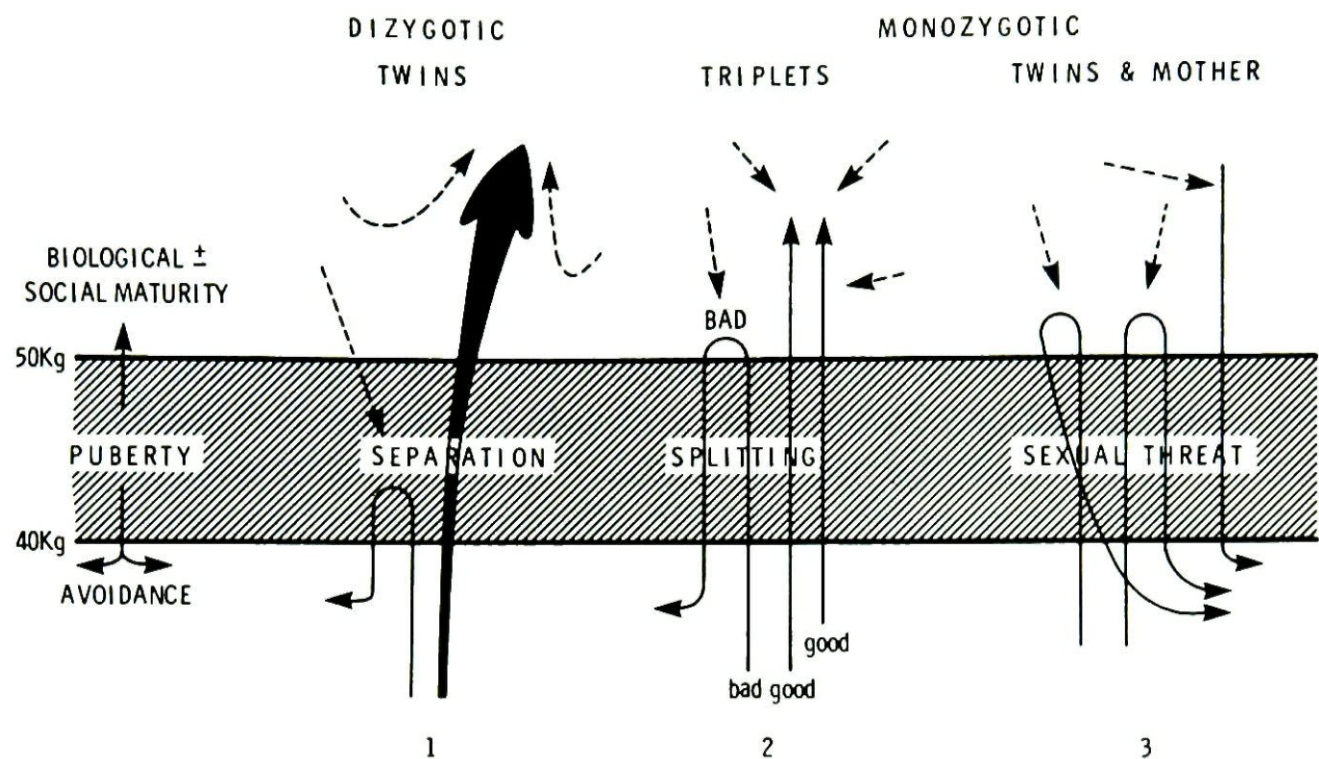


Figure 1. Three cases, two sets of twins and one set of triplets.

younger sibling, of whom Anne had always been jealous, was sent away to boarding school and, coincidental with this and the twins giving evidence of reaching puberty, the father had begun to take an active interest in political matters outside the home. His wife had felt that this had to be allowed, but her inner anxiety had again escalated. Within the last year, and within the context of Anne's anorexia nervosa, the husband had abandoned this venture and once again the parents rarely went out.

Here, then, is a pair of twins, believed by everyone to have been identical and treated as such, but who were not so biologically. On the brink of puberty and with the father for the first time declaring his interest in the outside world, one of them, the more dependent, had reacted with anorexia nervosa. At the time Anne's anorexia was considered by us as a regressive stance rooted in several factors: (a) her own incipient puberty; (b) Pauline's growth towards independence; (c) her own general insecurity/dependence; (d) more specifically, her fear that, as the other siblings dispersed, the family structure would rupture; (e) the resonance of this fear with the identical fears of the mother which, indeed, were probably importantly the origins of the patient's own fear; (f) the strong bond with the mother and especially with the mother's powerful underlying anxiety and defensive avoidance patterns; (g) the strong strand of nurturing/orality in this family with both its good (family-centered farming/cooking) and bad (alcoholism) aspects; (h) the rapidly emerging experience that anorexia nervosa resolved many of these potential conflicts.

Clearly, in this pair of twins, such factors relevant to anorexia nervosa could perhaps have many genetic and environmental determinants, and also psychodynamic determinants. For instance, the twin pair could be construed as having psychologically split itself through the mechanism

of the anorexia nervosa in Anne, this process being primed by puberty. It appeared that Anne has always been temperamentally more like her mother and Pauline more like her father. The discordance now expressed both aspects of this family's potential nature. Our attempts at intervention led to restoration of body weight to normal, during which time the family was involved in psychotherapy. The psychotherapy was intended to provide, as much as possible, a new interpersonal/social experience, both individually for the patient and collectively for the family, which might help to free up the growth within it necessary to release Anne from her anorexia nervosa. Ten years later, Pauline has matured normally (53 kg, 1.64 m, normal adolescent exploration). Anne, never fully recovered, is chronically ill, having relapsed after a three-year-long treatment intervention. She has shown some physical growth, mainly in terms of her height (35 kg, 1.55 m), but she remains, at the age of 22, with primary amenorrhoea. Recently we have attempted to help her adopt an anorectic lifestyle consistent with survival and with as good a social adjustment as possible; but otherwise, treatment is limited to occasional life-saving measures. The parents remain closely bonded, rarely venturing beyond the home. The younger sister is probably going to be all right.

CASE NO. 2

Michael, one of three male identical triplets, aged 23, presented with a two-year history of anorexia nervosa which, however, was insistently attributed by the family to the mother's death five years before.

The family lived in a house in which the father had been born and which he had lived in ever since, apart from a period during World War II, most of which he had spent as a prisoner of war in Japan, during which time he had been close to starvation. He was very strict in his manner, proud of his working-class background, and obese. One of his inconsistent characteristics was his fairly heavy drinking several nights a week at the local pub. The mother had devoted herself to him and to the triplets' care, rarely leaving the family home. Her previous marriage, ending in divorce and associated with many rumored sexual escapades at the time, was regarded as a taboo subject.

The family story was that, prior to the triplets' birth, the mother had been expecting twins. Michael, the last born, had been a surprise. The triplets became famous locally and claimed to have been irritated by the publicity and the insistence by many people that they could not be separately identified except by having their names printed boldly on their clothing. In fact, Michael was regarded by the family as different from the others, more adventurous, and, as adolescence progressed, more rebellious and dissolute. He came to embody the unspoken "bad" aspects of

the parents, especially, in their minds, those of his mother, to whom he was likened by most people. Within adolescence, he became obese, eventually reaching a body weight of 107 kg, and was given the nickname "Tug" as an apt anagram of "Gut". He came to be regarded as the black sheep of the family. The other triplets remained normal in weight. More compliant than Michael, they had always remained socially restricted and devoted their energies to leadership of the local Boys' Brigade. In his teens, Michael ("deservedly") contracted pulmonary tuberculosis and spent some time hospitalized. Within this context, his mother's imminent death was kept from him. His distress on learning of her death was overwhelming. He reformed, becoming very hard-working, and he began to diet in order to curb his obesity. During the next three years his weight fell to around normal and he did well in his job. Then, rushing across the road from his office, he suffered a traffic accident which left him unconscious for an hour or so. There were no neurological sequelae but classical anorexia nervosa now erupted. At our interview, he weighed 56.3 kg (1.79 m height).

At the time, his anorexia nervosa was viewed by us as a product of his unresolved experience of the loss of his mother. His reaction to the loss initially had been to feel guilt that his own impulsivity and sex-ridden behavior had contributed to her death; i.e., it had distressed her, not the least through kindling afresh her guilt over her own adolescent sexual conduct. He had sought to make reparation initially by hard work, clean living, and a curbing of his obesity, which he had recognized as explicit evidence of poor impulse control. His ever-increasing, frantic attempts to redeem himself by hard work decompensated following the accident, and anorexia nervosa then supervened as a further placatory stance. A propensity for impulsivity within the family, perhaps with a genetic basis, severely curbed in the mother in her own later life and expressed by the father solely through his drinking behavior—but basically contained through his enmeshment within his home and his image of strictness in the family—was a recognized danger. The father's overt obesity, sometimes also displayed premorbidly by Michael, had probably been shaped by many influences, including his wartime starvation, his current high alcohol intake, and his enmeshment within the "nourishing" home. This enmeshment, as a means of conflict avoidance, had long characterized him, his wife and the other two triplets. The family was seen as heavily defended against sexuality. In this context, Michael had been construed from his birth as different by the family, but not by the outside world. Puberty had enhanced this difference in their minds as he had begun to display those impulsive characteristics that so alarmed them. His anorexia nervosa, although a worry to the father, had now resolved many problems for them all.

Treatment of the usual kind enabled Michael to achieve a normal weight for him of 69.8 kg and he sustained this for some time following

discharge from the hospital. The residual tuberculous shadow in his lung field cleared. The remainder of the family remained heavily defended against our attempts at psychotherapeutic intervention with them. Ten years later, Michael remains marginally anorectic. All three triplets are leading very restricted social lives, with no heterosexual relationships. They remain dominated by their father.

CASE NO. 3

This complex case may shed some special light on the nature/nurture issue. The mother presented to the clinic in 1973, and one of the twins (Jill) presented as a patient for the first time in 1979. The flavor of the problem is perhaps best introduced by extracts from the initial assessment letters sent at the time to the referring general practitioner.

Re: Mother aged 50 (17 December 1973); [Figure 2]:

"Dear Doctor . . . ,

"I saw Mrs. Brown on Friday last in my out-patient clinic when she came along accompanied by her husband and her twin daughters.

"We met with Mr. Brown and the two daughters first and they described the background of her illness. Their view was that it has been coming on for 3 or 4 years and had reached crisis point in August 1972 when she was down to 28 kg. Since then she has been up to about 51 kg following re-feeding treatment in hospital, but she was very distressed at that time by what she judged to be excessive and forced feeding and subsequently relapsed. She is currently 31.8 kg.

"Mr. Brown knew both her parents and indeed met her in India where she had been born and brought up. He described the father as a self-made, intelligent but very dependent man who worked as a teacher. He died in 1957 of what was said to have been a heart attack but there was some suggestion that he may have taken his own life. The patient's mother was described as a dominant, eccentric and very possessive woman, the patient being her favourite daughter. Mr. Brown's view was that the patient's father was devoted to his wife but that she treated him like a naughty child and never let him out of her sight. The patient has two brothers and two sisters, one brother and one sister being twins. This sister is said to be herself a very dominant and thin person and has also produced twins. The other siblings are said to be of normal weight.

"Mr. Brown met his wife-to-be when he was out in India and she was aged 19. At that time she weighed close to 63 kg although he has subsequently learned that around the age of 17 she had nearly died from ex-

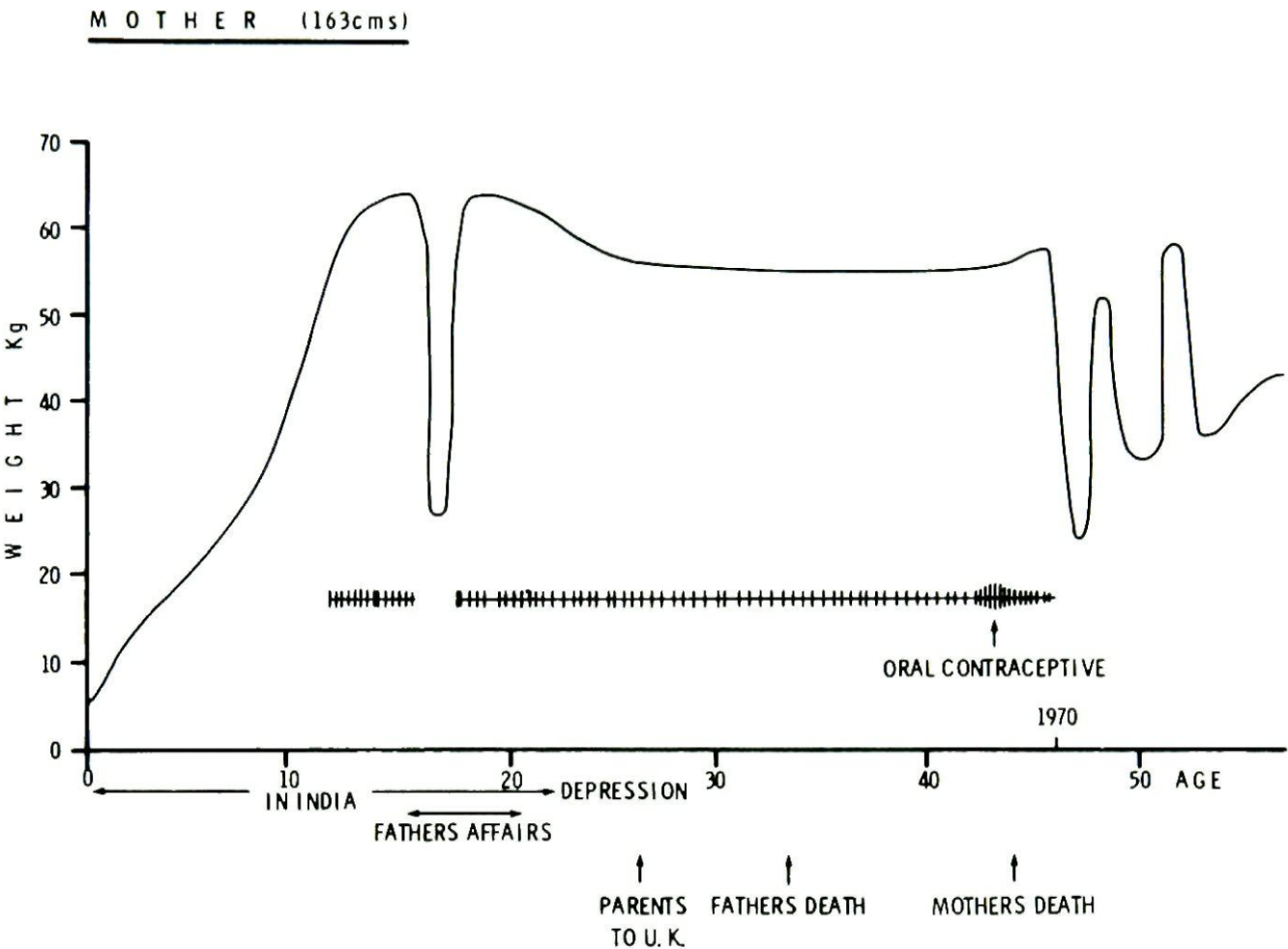


Figure 2. Weight fluctuation of mother in case No. 3.

cessive slimming. They married 8 or 9 months later and 9 months after this their first son was born. He is now aged 29 and on the plump side as is the next daughter who weighs about 70 kgs. Next come the twins who came along to the interview, and it is clear that they both have anorexia nervosa. They are both tall and weigh about 44 kgs now and have been amenorrhoeac for several years, but during their teens the more dominant one was about 70 kg and the other one about 60 kg. Lastly there is a surviving younger brother, tall and thin and who has lost 13 kg in weight recently. Both twins denied the possibility that they had anorexia nervosa and were very threatened by the proposition. They described the relationship between their mother and father as being a happy one, Mr. Brown being calm and placid and the patient excitable. Mr. Brown is himself slightly overweight and more akin in nature to his younger son, whilst the family sees the oldest son as being like the patient. They all emphasized how Mrs. Brown has, until the last few years, always been an attractive person, conscious of her figure, and usually taken as being a decade or more younger than she actually was. She has enjoyed acting, usually being given younger adolescent parts.

“They dated the onset of her relapse 4 or 5 years ago to the time of her incipient menopause. At this time her periods became irregular; she was put on the Pill and began to gain weight. She became self-conscious once more and concerned about her advancing age and clearly began to curb

her appetite. Her weight slowly fell and it is only about 3 years ago that her emaciation commanded attention.

"Next we met with Mrs. Brown. She said that she wanted to get back to a normal weight, saying that she no longer minded being fat so long as she did not have a protruberant abdomen. In this latter context she commented on her treatment earlier this year which she felt had left her looking 7 months pregnant.

"She described the early family background in some detail, confirming the descriptions that I have already given of her mother and father. She saw her parents' marriage as an unhappy one from the time that she had reached her early teens. Up until then she had been the favourite child and her recollection of childhood is of her happiness during it. It was when she was in her teens that her father broke away from the marriage briefly and had a series of limited affairs and this kindled her mother's jealousy to an extent that the mother required the patient to scrutinize all of her father's mail in case he should be receiving any letters from other women.

"As the patient herself began to develop an adolescent interest in boys she became first of all self-conscious about her weight and then guilt ridden, and it was within this context that she developed acute anorexia nervosa at the age of 17 with all the attendant regression and childlike compliance. Subsequently she recovered spontaneously but her mother remained jealous of her plans to marry and throughout the rest of her life attempted to undermine the marriage, including trying to convince Mrs. Brown that her husband was having affairs.

"Mrs. Brown remained free from anorexia nervosa throughout her middle life although as I have said, she has always kept an eye on her weight by curbing her food intake. When she and her husband came to England after the war and she was thereby separated from her mother she became severely depressed for a year or two, but this remitted when the parents also came to England. When Mrs. Brown's father died the mother strove to come to live with her but she resisted this and their relationship became bitter. Her reasons for so doing were that she feared that her mother would come to dominate and destroy her husband. During the mother's terminal illness she was highly critical of the patient, blaming her for her plight. After her death the patient was depressed for some while and then the anorexia nervosa developed. Since then the patient has again felt closer to her mother, feeling, with her changed nature, that she is now more like her mother would have liked her to be. Over the last few years there has been no sexual intercourse in the marriage, although until 5 years ago it was an enjoyable and important aspect of the relationship.

"Mrs. Brown feels very much to blame for her twin daughters' illnesses, feeling that she has infected them.

“Mrs. Brown clearly has severe anorexia nervosa from a psychopathological standpoint. She maintains low body weight mainly by abstinence but also through the dehydrating effects of massive purgation. I think the pathology is particularly complicated, probably being contributed to by the imminent menopause and its meanings for her, and also by the relief the condition provides for her in the face of her mother’s death. It may be possible to help her and I have explained that she would need to come into hospital for about 4 months and have her weight restored to fully normal proportions in the first instance, in association with psychotherapy aimed at helping her to cope with the implications of such major weight change, which might well include a period of further and more sustained depression”

Re: Jill (and Sally) aged 31 (27 February 1979); [Figure 3]:

“Dear Doctor . . . ,
“I saw your patient Jill Brown when she came along today accompanied by her twin sister Sally and their father. Mrs. Brown had refused to come and I gather that she still has chronic anorexia nervosa. In fact I saw her back in 1973 and she subsequently came into Atkinson Morley’s Hospital for treatment, obviously without much success! I am enclosing

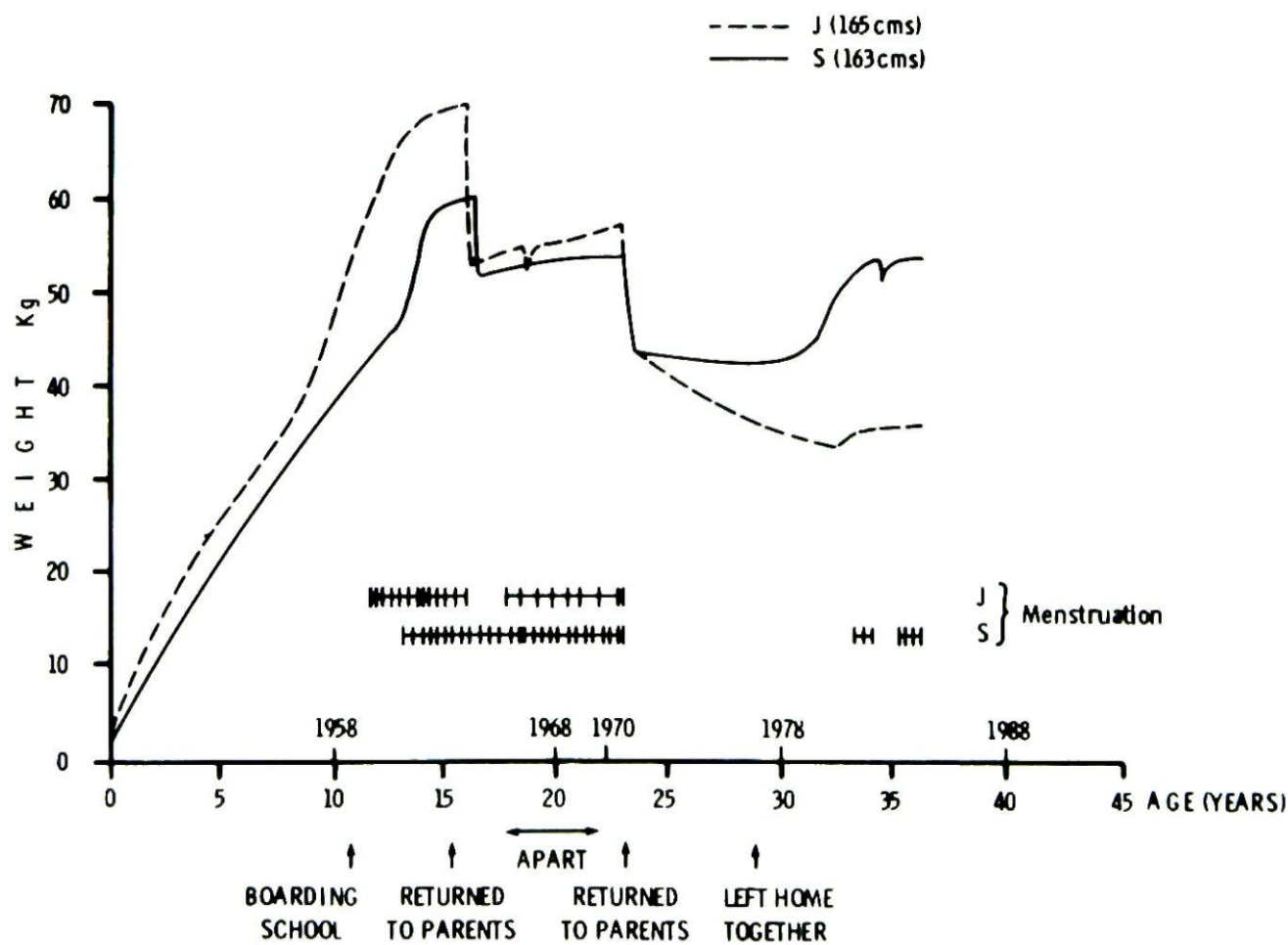


Figure 3. MZ twin daughters.

in confidence a copy of my initial letter concerning her. I think this is justified since it sheds light in my view on the problems that the twins have. At that time in 1973 and also during 1974 the twins visited their mother in hospital but always strenuously avoided discussion of their own condition.

"I met with Mr. Brown first and he described how his wife dropped to about 32 kg round about 1975 but has since levelled up to about 38 kg. She is now established on a stable self-selected low weight control diet and it sounds as though they have a fairly barren personal life. Mrs. Brown's case was of particular interest. She had an episode of anorexia nervosa in her teens, but then recovered in many senses although she had remained very weight conscious and controlled her body weight at a low level throughout most of her reproductive life. It was at the time of her menopause that she dipped down into anorexia nervosa again and my view of this was that it related to the death of her mother and was best construed in terms of a bereavement response rooted in the lifelong hostility and guilt that characterized their relationship especially in respect of matters sexual. Mr. Brown went on to describe their two sons who are said both to be well and married. They live close together up in Scotland and are farming. This family is of course riddled with anorexia nervosa as well as twins, and I think the sons' commitment to farming and to strenuous exercise, which is evidently the case, is another aspect of this former diathesis. Mr. Brown reminded me that Jill was the firstborn of the twins and throughout early life was the dominant and least anxious one. Sally's obvious childhood insecurity has led to her receiving greater attention than Jill. The twins shared childhood friends. In early adolescence Jill weighed around 70 kg whereas Sally weighed nearer 60 kg. Mr. Brown felt that it was the recent deterioration in Jill's state, with attention being directed at her abdominal symptoms, that had eventually precipitated her into a situation in which she, unlike Sally, was now acknowledging her anorexia nervosa and willing to come to this consultation. Mr. Brown was very doubtful whether Sally would want any help other than in respect of advice concerning the management of Jill's illness. He outlined how in recent years the sisters had lived together in a small village seven or so miles from Maidstone. Jill teaches; Sally works as a secretary in the school. She has obviously got very concerned recently because Jill is not eating so much and Sally suspects that she is giving her food to the half a dozen or so cats they have in the house.

"Mr. Brown went on to describe the nature of their relationship over the years and particularly since the onset of the anorexia nervosa, which as you know was in both of them concurrent with the mother's relapse into the same condition. He felt that they now had an absolute yearning to be totally identical in everything they do. The way he described it, it is evident that this need has been particularly condensed into their anorectic symptomatology. Thus they share out peas by the number, insist on

eating the same size apples and so on. During the last ten years since their anorexia nervosa developed they have had no boyfriends. They occasionally meet with their mother and appear to get on well with her from his vantage point. Mrs. Brown has obviously conveyed to them her own sense of deep distress on regaining normal body weight back in 1974. She has told the family that she felt bloated and disfigured and Mr. Brown is convinced that the daughters believe that this would merely also be their fate were they to submit to treatment.

"After this initial meeting with Mr. Brown the social worker working with me went off to talk with Sally whilst I was left with Jill. She weighed 37.5 kg with her clothes and shoes on. She was initially very wary in the interview but eventually settled down and was able to acknowledge that she had anorexia nervosa. She said "It has taken me a long time to accept it." She agreed that Sally also had anorexia nervosa but she felt that her own condition was worse, especially now since she finds herself restless and tense and also with the abdominal symptoms. She began by saying "Logically I would like to have an ideal figure, I get upset being called Twiggy." (It transpired that the weight she would like to be was about 41 kg.) "If I put on weight too quickly I get bored." I invited her to reconsider the word bored and she agreed that she meant depressed and panicky. Her main means of low body weight control has been through abstaining. Very occasionally when she finds that she has unavoidably overeaten, for instance at a dinner party, then she vomits. She denied taking purgatives but advanced the notion that the large amount of coffee that she drinks every day may account for her polyuria.

"In talking about her background she focussed on her early teens when she was plump, having experienced an early puberty. At that time she had a good appetite and would usually have second helpings. However, she had become very weight conscious and had started dieting by the age of 14 or so, and at the age of 15 she weighed about 54 kg and had amenorrhoea for two years. Subsequently her periods returned but then from 20 onwards, by which time she was down to 50.8 kg, she has had intractable and indeed progressively worsening anorexia nervosa. She gave a similar account of Sally's anorexia nervosa, which seems if anything to have followed on slightly after Jill's. She went on to insist that she had a fairly reasonable life with Sally, but sometimes wondered what it might all lead to. She felt that she could only be happy free of anorexia nervosa if Sally was also free of it. Under these conditions she could imagine them each being married although they would need to live close to one another. However, she qualified all of this by saying that she now finds that she is not at all attracted to men. This enabled us to discuss her late teens, which seems to have been the last time in her and Sally's lives when they were beginning to explore their adolescence. At this time Jill went up to Edinburgh to study at the university; she had several boyfriends and says that she enjoyed sexual contact. This would seem to

have been after her first episode of anorexia nervosa and before the present one, i.e. when she was aged 18, 19 and 20. The last of those relationships was with an older man aged 37. Around the same time, Sally was having similar experiences first of all in Oxford and then in Edinburgh with Jill. Sally had gone to Edinburgh to be with Jill and was studying for 'A' levels. This behaviour of the twins struck me as being highly reminiscent of their mother's behaviour in her own teens, which had precipitated out her own first bout of anorexia nervosa. At that time there were major sexual difficulties in her parents' marriage and her father was having an affair. Jill agreed that Mrs. Brown had not been able to discuss sexual matters with either of them or advise them how they might begin to handle themselves in peer relationships in general. Looking back now it would seem to me that there were several other precipitants to Mrs. Brown's relapse 10 years ago: thus, just preceding the death of her mother she had been put on to oestrogens and within this context had gained weight and was feeling unattractive and, I suspect, was nurturing ideas that people might regard her as pregnant despite her age. The 'permissiveness' associated with being on the Pill would also have had alarming connotations for her. I suspect that this problem was enhanced for her by the fact that her twin daughters were themselves now in their late teens and beginning to behave in the way that I have described. Mrs. Brown's solution to this was of course to develop her anorexia nervosa, and I would believe that she thereby inevitably provided this avoidance model for her daughters too.

"Jill went on to describe her abdominal pain, and it did seem to me that one important element of this could be the hunger which she had experienced for many years, which has intensified recently, and which she is of course very reluctant to assuage. She still finds that if she can bring herself to drink some milk or eat some other food that this relieves it. I would be most interested to know whether or not there had ever been definite evidence of an ulcer.

"Following this, Jill began to negotiate ways in which she thought I might be able to help her. She said she would be willing to put on 6 kg if she could do it gradually, and she would like to know of a sensible diet to follow. I pointed out to her that she is actually an expert on diets and that there is a world of difference between good intention and ability to follow through. I personally have grave doubts that working with her in out-patients would be of any value, since psychotherapy in anorexia nervosa in my experience can only be of help once the person concerned is back in touch with a reasonably normal body weight, and prior to this it rarely helps in improving food intake for the better. In fact, it sometimes leads to a worsening of the condition. I went on to present to Jill my view about the situation. I confirmed for her that she has severe anorexia nervosa and that Sally and her mother also have this condition. I told her that the most likely thing was that she would continue to be handicapped

by it, given the natural course of events. I put it to her that intervention of a major kind would improve her chances of ultimately escaping from the condition. I also made it quite clear that she would be unlikely to achieve anything unless Sally was equally involved. She was eventually able to settle back and listen to my proposal that I would be willing to admit both of them to hospital on the basis of restoring their weights back to matched population mean levels, but on the basis of them trying to pick of the threads of life as 17 or 18-year olds rather than 30-year old women. I explained that, as they knew well from their observation of their mother's treatment, we would couple any such treatment with individual and joint psychotherapy involving them together and also probably involving their father. I pointed out that they would have major tasks of differentiation but that in my view there was a chance of them achieving this. Jill went on to tell me of a manuscript that she is currently preparing which is intended to be autobiographical and concerning her first term up at university. This struck me as an important admission on her part and perhaps rather hopeful in the sense that she is beginning again to wish to understand aspects of her more healthy self and maybe some of the problems with which she was trying to grapple at that age. She went on to describe her more sustained literary efforts which include the writing of books for children and about children, and she was able to accept that this, although it has its creative and rewarding aspects, is another aspect of her present psychopathology. Subsequently I met together with the three of them and expressed my views. As you can imagine I am not very optimistic but "

The subsequent evolution of anorexia nervosa in this family, in terms of body weight, is shown in Figures 2 and 3.

From a genetico/constitutional standpoint, the family is indeed riddled with anorexia nervosa (Figure 4). The mother's sister has it chronically as does the father's cousin's wife. The latter biologically unrelated case points to an assortative mating process at work. Both these women, though afflicted severely with the condition, were also capable of reproduction, as was our patient, the mother. Probably related strands of "pathology" in the family include the obesity within it, and the farmer son who is deeply involved in "physical fitness" at normal body weight, having previously been obese. Premorbid obesity is associated with a poor prognosis in anorexia nervosa (Crisp, Kalucy, Lacy, & Hardings, 1977) and this characterized Jill more than Sally. Sally's relatively mild premorbid obesity was characteristically associated with a phase of bulimia on being sent to boarding school (see Figure 3) together with Jill in their early teens.

On the experiential side, the mother's onset of anorexia nervosa seemed to arise in relation to her own mother's death and its activation

CASE NO. 4

This case is described because within this family, three biologically unrelated individuals had severe anorexia nervosa.

The first member of the family to present as a patient to the first author in 1964 was Mary, aged 14. She had a one-year history of severe anorexia nervosa. Her adoptive parents attended with her. Father, aged 60, had obvious and severe anorexia nervosa. Although unwilling to acknowledge it, he described having been obese in early adolescence. He had then slimmed and become emaciated, remaining like this ever since. He had made a fortune as a manufacturer of now nationally known biscuits. His wife, aged 56, slim and dressed in youthful manner, was superficially charming but essentially very anxious and "hysterical" in manner. The marriage was unconsummated. Mary had been adopted from birth and was much loved during her childhood. Her real mother had been a 19-year-old, lusty, unmarried university undergraduate of whom the family had photographs. At age 12, Mary had begun to act out as a young teenager. Her interest in and attractiveness to boys had triggered intense anxiety in the adopting parents and her mother had, for instance, screamed at her, "You're just like your mother." In the face of such conditional love and potential rejection, Mary's initially limited attempts at dieting had escalated within the context of the mother's mounting fantasy that she was pregnant. Anorexia nervosa, with its pubertal reversal and cessation of all adolescent activity, had resolved the issue. In-patient treatment, involving restoration of body weight to normal and psychotherapy, was associated with recovery over a three-year period. Now, 20 years later, Mary is married and has children.

In 1966 Carol, a 41-year-old anorectic, presented to the clinic. She had been severely ill since her teens and subject to some investigations from time to time, but with the diagnosis not previously made because of an effective smoke screen of complaints about abdominal pain and vomiting. It emerged that she had been evacuated as a refugee from London to the above family's household in 1940, aged five. She had then lived with them until well after the end of the war, at which point she returned to London, aged 12. Her anorexia nervosa had then erupted. Her billeting on the family had, under the prevailing conditions of mass evacuation from London at the time of the blitz, been an apparently random event.

This family contains three biologically unrelated members with anorexia nervosa, whose coming together would on the face of it seem to owe little to self-selection factors (assortative bondings) having to do with anorexia nervosa. The most obvious factors contributing to anorexia nervosa in the two females would appear to be the anorexogenic household within which they were brought up. This household was characterized by severe anorexia nervosa itself in the father, by enmeshment, and by the avoidance of all matters sexual.

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