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The Eating Disorders
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Chapter 21

The Etiology of Anorexia Nervosa

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The valid question in a discussion on the etiology of anorexia nervosa is: what caused the willful starvation that stems from the fear of fatness? It is thus important to clarify two fundamental issues at the outset: First, starvation is the cause of many of the features of anorexia nervosa, such as amenorrhea; constipation; insomnia; preoccupation with food, dieting, and shape; bulimia; food fads; increased consumption of coffee, tea, and chewing gum; and diminished sexual desire [1,2]. To suggest, for instance, that a fear of sexuality is the *cause* of amenorrhea or that a need to withhold is the *cause* of constipation is absurd. Second, the cardinal feature of anorexia nervosa is a fear of fatness, or a pursuit of thinness. The food avoidance and the vomiting and purging stem from this fear. The disorder is not due to a loss of appetite (anorexia) or a primary fear of eating or food. Thus, for instance, to postulate a link between anorexia nervosa and Crohn's Disease is absurd, because in the latter condition there is a true loss of appetite. There are currently seven main theories organized along different conceptual levels that attempt to explain the etiology of anorexia nervosa. Logically, they are not mutually exclusive, and they overlap to some degree. However, their explanatory power varies and so does the strength of the evidence for each theory. This review will discuss each theory.

THE SOCIAL-CULTURAL THEORY

This theory in essence states that social-cultural pressures contribute to the development of the disorder.

Evidence that supports this theory will be summarized according to (1) the current emphasis on slimness, (2) the contradictory role of women in modern society, and (3) the vulnerability of the white upper-social-class adolescent female.

The importance of physical attractiveness in Western society is undeniable. In the female this attractiveness has taken the form of slimness. Several surveys indicated that the majority of young women are unhappy about their weight and want to be slimmer [3-5]. In contrast, men prefer to be bigger and heavier [3-5]. Garner et al [6], reviewing data from Playboy centerfolds and Miss America Pageant contestants in the last 20 years, found a significant trend toward slimness. All measurements of Playboy centerfold women, except for height and waist, decreased significantly. Thus, for instance, the average Playmate in 1959 weighted 91% of average, while in 1978 they weighted only 83.5% of average. Since 1970, Miss America Pageant winners had a mean weight of only 82.5% of average. Meanwhile, the number of diet articles in six women's magazines have increased substantially over the last 20 years.

However, Garner et al [6] found that this emphasis on thinness and dieting occurs in a population that is becoming heavier. Weight statistics from the Society of Actuaries over the last 20 years indicated an increase in average weight for women in all height categories below the age of 30 years. The pressure on women, particularly those of the upper social class, to diet and appear slim thus seem relentless [7,8]. That such pressure may

precipitate the development of anorexia nervosa seems to be confirmed by the finding that the condition is much more common in women that must control rigorously their size and shape, such as ballerinas, modeling students, and athletes [9-11]. Competitiveness intensifies the pressure [11]. Under such circumstances it is perhaps not surprising that Branch and Eurman [12] found that the anorectic's friends and relatives actually admired her slimness, specialness, and control.

Boskind-Lodahl [13] regarded "the cultural heritage of sexual inequality" to be directly responsible for the development of eating disorders in women. However, since such inequity presumably exists also in developing countries, it is difficult to see why the disorder is so rare in these countries [14]. Selvini Palazzoli [15,16] emphasized the complex and contradictory roles women have to play in modern society. Self-definition and achieving a feminine identity may be particularly problematic for the modern woman [17,18]. High-achieving women may experience low self-esteem [17] and a heightened fear of success, which revolve around fear of loss of femininity and of interpersonal rejection [18,19]. Dunn and Ondercin [20] suggested that eating disordered patients may have difficulty integrating "masculine" ideals, such as independence and assertiveness, with traditional concepts of femininity. Such role diffusion presumably increases insecurity and intensifies the striving for perfection and control.

The dilemma confronting the modern female may explain why adolescence tends to be a particularly difficult phase for the young white girl who is more likely to be biologically fatter, to have a poor self-image, low self-esteem, and high emotional instability and self-consciousness. The Ten State nutrition survey [21,22], which in 1968 to 1970 studied more than 40,000 individuals from all age-groups with respect to their nutritional status, has provided a good deal of insight into why current cultural emphasis on slimness has the greatest impact on the middle- and upper-class white adolescent female. The survey findings may be summarized as follows:

1. There is a tendency for females of all ages to be fatter (as measured by the triceps fat fold) than males.
2. There is prepubertal weight gain in both sexes, but during adolescence proper, the female gains fat, while the male loses fat.
3. High income is associated with greater fatness for males at all ages and for females through early adolescence. In the female, however, there is an income-related reversal of relative fatness during adolescence, ie, during adolescence, females in the lower-income families started out being leaner but ended up being fatter than those in the higher-in-

come families. Garn and Clark [21,22] speculated that this reversal occurs as the result of conscious dieting on the part of the higher-income female.

4. White males tend to be fatter than black females at all ages. White females are also fatter than black females through early adolescence, but thereafter a reversal of relative fatness occurs similar to that for higher- and lower-income females. This differential fatness pattern between the two races, remains even after controlling for family income. Garn and Clark [21,22] again speculated that this reversal occurred as a result of conscious dieting on the part of the white female.

These findings suggest that fatness is related to being female, being white, and being from a high-income family. It is obvious that cultural emphasis on thinness has its most powerful impact on the white teenage girl from a higher-income family.

That such social-cultural pressure generates a greater likelihood for the development of anorexia nervosa in women is probably not in dispute. It is, however, obvious that not all women exposed to such pressure develop anorexia nervosa. Other factors must also occur to precipitate the final development of the illness.

FAMILY PATHOLOGY THEORY

The early investigators have all emphasized the family pathology in anorexia nervosa [23-25]. Charcot [23] advocated separation of the patient from family as part of treatment. Gull [24] found the relatives to be the worst attenders of the patient. Laseque [25] described the striking family enmeshment, and he urged clinicians not to overlook the family pathology. Other early investigators have likewise described the adverse influence of the family on the patient [26,27].

Attempts to identify a typical anorectic mother [28-30] or a typical anorectic father [30,32,33] have produced no consistent findings [34,35]. More recently, researchers have focused more on family interaction patterns that may be characteristic of the anorectic family. Bruch [36,37] emphasized the facade of happiness and stability that hid deep disillusionment and secret competition of the parents. She also found the parents to be enormously preoccupied with outward appearance and success. Palazzoli [15] studied 12 anorectic families and found rejection of communicated message, poor conflict resolution, covert alliance of family members, and blame shifting to be common. She also emphasized their rigidity in that they tried to preserve agricultural-patriarchal values and mores in an urban-industrial setting. Both Palazzoli [15] and Bruch [36,37] found the parents to be overprotective but also to involve the sick child in

their covert competition and conflict. They seemed to use the child to discharge some of their own unfulfilled longings.

Minuchin and his co-workers have written extensively on the psychosomatic family [38-40]. They advocated an open-systems model for psychosomatic illness, anorexia nervosa included. This system included parts such as extrafamilial stress, family organization and functioning, the vulnerable child, physiological and biochemical meditating mechanisms, and the symptomatic child. The system could be activated at any point, and the parts could affect each other. Nevertheless, these authors have emphasized almost exclusively the family pathology in this system and stated that, "When significant family interaction patterns are changed, significant changes in the symptoms of the psychosomatic illness also occur," [40]. They further hypothesized that (1) certain family characteristics were related to the development and maintenance of psychosomatic symptoms in children, and (2) the child's psychosomatic symptoms played a major role in maintaining family homeostasis. The family characteristics identified were enmeshment, overprotectiveness, rigidity, and lack of conflict resolution. Meanwhile, the child was used to maintain stability and avoid open conflict, and thus was often caught (triangulated) in the parents' covert conflict. The illness enabled the parents to submerge their conflicts in protecting or blaming the sick child, who was then defined as the sole family problem.

However, few studies confirm that such family interaction pathology occurs in anorexia nervosa. Crisp et al [41] used a standardized measure (the Middlesex Hospital Questionnaire) and found that the psychoneurotic status of the parents worsened significantly as the patient's weight increased with treatment. This was particularly so if the marital relationship was poor. Six-month outcome for the patient's illness was significantly related to the initial parental psychoneurotic morbidity. Case selection factors and family therapy effects were, however, uncontrolled. Foster and Kupfer [42] telemetrically recorded the nocturnal motility of a female patient with anorexia nervosa and found that nocturnal activity was correlated with visits by specific family members during the previous day. Visits by the father and the identical twin led to a decrease in nocturnal motility, while visits by the mother and older sister led to an increase in such "arousal." The extent to which such findings can be generalized is, however, questionable.

Related to the issue of family environment is the role of genetic factors in the pathogenesis of anorexia nervosa. Several large-scale studies have found an increased incidence of anorexia nervosa in the family members of the patients [34,43-45]. Twin studies have found a concordance of about 50% for monozygotics

and 10% for dizygotics [46-48]. Adoptive studies, however, are needed to tease out environmental versus genetic factors. In this connection, Crisp and Toms [49] described a remarkable case of a male chronic anorectic whose adoptive son as well as the girl who stayed with the family as a war evacuee both developed anorexia nervosa.

Crisp and his co-workers have suggested that family weight pathology may be specifically related to the pathogenesis of the illness [35,50]. In a well-controlled study of the parents of anorectic patients, Halmi et al [8] failed to confirm this.

Several detailed studies of affective disorders in the families of eating disordered patients have been published recently. Cantwell et al [51] reported an increase in the familial prevalence of depression in anorectics. Strober et al [52] compared the relatives of 35 bulimic with 35 restrictive anorectics, and they found that affective disorder, alcoholism, and substance abuse disorders were more prevalent among the first- and second-degree relatives of bulimics. In a study of 420 first-degree relatives of 89 eating disordered patients, Hudson et al [53] found an increased risk of affective disorder among the first-degree relatives of both anorectic and bulimic patients to a level similar to that of the families of bipolar disordered patients. Winokur et al [54] also found that there was a doubling of affective illness in the parents of anorectics in comparison with a controlled population. Thus, the familial prevalence of affective illness, and perhaps of alcoholism as well, appears to be increased in the eating disordered patients.

In summary, it remains to be substantiated that specific and abnormal family interaction patterns occurred in anorexia nervosa and that they are causally related to the development of the condition. Most of the studies quoted ignored the effect of this exasperating illness on family interaction, and the investigators failed to distinguish between family pathology occurring as stress reactions as opposed to pre-existing patterns [55]. Familial affective disorder and perhaps familial alcoholism and other impulse-controlled disorders may predispose an individual to develop anorexia nervosa [48,52,56]. The mechanism for such predisposition is, however, unclear.

INDIVIDUAL PSYCHODYNAMIC THEORY

Early psychoanalytic interpretation of anorexia nervosa considered it to be related to a rejection of female genital sexuality and oral impregnation fantasies by means of refusal to eat. This was accompanied by a regression to pregenital defense mechanism in the face of conflict revolving around primitive, sadistic, and cannibalistic oral fantasies [57,58]. Object relation theorists

considered it to be related to the introjection and repression of a bad object consequent upon the early ambivalent relationship with an aggressively protective, unresponsive, castrating, domineering, and controlling mother [15,59,60]. On the more superficial level, it is generally recognized that starvation serves as an expression of hostility, control, and aggression toward the family [57,59]. In 1931 Brown [61] observed that anorexia nervosa was a pathological manifestation of the detachment of the growing individual from parental authority. Bruch [36,37,62,63] has repeatedly stated that anorexia nervosa was a struggle for a self-respecting identity. That such a struggle took the form of willful starvation suggested serious psychological developmental defects. Central to such defects was the failure of the parents to regard the patients as individuals in their own right; they failed to transmit a sense of competence and self-value to their children. The youngsters were instead treated as something to complement the parents' needs. Their sense of worth and value were thus derived from being needed by each parent. In short, they felt that they were the property of their parents. The illness thus represented an effort to escape from such a role and to establish control. Because of their disturbed perception of bodily sensation related to their lack of autonomy, and a paralyzing sense of ineffectiveness, such patients misinterpreted their biological functioning and social role, and came to interpret thinness and starvation as specialness and self-control in an exaggerated and concrete way. Palazzoli [15] and Boskind-Lodahl [13] echoed such views.

The individual psychodynamic theory is extremely plausible, but it has never been empirically tested; its emphasis on early parent-child interaction does not lend itself easily to such testing. A recent single case study [64] provided some evidence of abnormal mothering in a case of male anorexia nervosa.

THE DEVELOPMENTAL PSYCHOBIOLOGICAL THEORY

Brown [61] stated that a fear of growing up and assuming adult responsibility was highly characteristic of anorexics. Crisp has repeatedly stated [50,65,66] that anorexia nervosa was rooted in the biological and consequently experiential aspects of normal adult weight. Starvation in the anorectic represented a phobic avoidance of adolescent/adult weight. Anorexia nervosa was thus a disorder of weight pivoting around specific maturational changes of puberty, both biological and psychological. The psychobiological regression reflected the individual's need to avoid adolescent and related family turmoil. The severe dieting was reinforced

by the relief that the control and the low weight brought, as biological and related psychological childhood was re-experienced and postpubertal experience was concurrently eliminated. Meanwhile, adolescence in the child threatened the rigid and experience-denying parents. The illness thus sometimes served to avert rekindling of buried and denied but unresolved parental conflicts and psychopathology. Needless to say, the illness brought its own problems, but they were deemed to be the price that the patient and her family had to pay to avoid deeper, more fundamental discord. Crisp has repeatedly emphasized that such maturational demands of adolescence and family pathology were not specific to the condition. Indirect and partial support for this view has come from several sources: 1. There is an immature pattern of gonadotrophin release in anorexics, which reverts to normal after weight gain [67,68], 2. Frisch [69,70] has found that puberty hinges on the individual attaining a critical amount of fatness, 3. Clinical experience suggests that anorexics will often agree to eat provided that weight gain does not occur, thereby indicating that weight rather than eating is involved in the issue of control, 4. Finally, Crisp et al [41] found that the parent's psychoneurotic status worsened after the patient's recovery, which seems to support the notion that the illness serves to reduce family tension. Nevertheless, this theory of "weight phobia" has never been empirically tested. One study found anorexia nervosa patients to be different from other phobic patients in terms of skin conductance changes [71].

PRIMARY HYPOTHALAMIC DYSFUNCTION THEORY

Russell [72-74] has repeatedly suggested that a primary hypothalamic dysfunction of unknown etiology and only partially dependent on weight loss and psychopathology occurred in anorexia nervosa.

Early onset of amenorrhea (sometimes before any appreciable weight loss), the incomplete recovery of hypothalamic function, and the persistence of amenorrhea despite weight gain have been cited as evidence for a primary hypothalamic disorder in anorexia nervosa [68,74]. Furthermore, several reports of hypothalamic tumor presenting as anorexia nervosa have appeared [47,75]. Finally, in animals the role of the ventromedial hypothalamic nucleus in the regulation of feeding and satiety appears to be well established [76].

However, the significance of all these findings remains controversial. An accurate dietary history is difficult to obtain in most eating disordered patients, and amenorrhea preceding the onset of eating disturbance probably occurs in only a small proportion of patients.

Furthermore, emotional disturbance, not specifically related to anorexia nervosa, may cause amenorrhea. Finally, patients who developed amenorrhea before appreciable weight loss do not seem to have a different outcome from those who develop the amenorrhea after the onset of weight loss. The incomplete recovery of hypothalamic function may be related to inadequate weight gain, abnormal eating habits, persistent preoccupation with food and weight, and the simple fact that recovery may take time. Simple weight loss without anorexia nervosa may lead to a functional hypothalamic disturbance [77]. The eating disorder that occurs in association with a hypothalamic tumor often has atypical features. Since the vast majority of anorectics have no demonstrable and anatomical hypothalamic pathology, in those rare but typical cases, it remains possible that this infrequent association is entirely due to chance.

The noradrenergic system in the medial hypothalamus in rats seems to regulate intake and body weight directly [76]. Thus, for instance, a decrease of noradrenergic innervation to the paraventricular hypothalamus leads to a decrease of daily intake (particularly carbohydrates) and body weight. Food deprivation, which stimulates intake and carbohydrate preference in the animal, increases the turnover rate of endogenous norepinephrine in the medial hypothalamus. However, extrapolating animal data to human psychopathology is hazardous, and there is no direct evidence for the existence of a medial hypothalamic noradrenergic system dysfunction in anorexia nervosa. Urinary MHPG, an important end product of norepinephrine, is reduced in anorexia nervosa, but its level normalizes with weight gain [78]. Reduced plasma levels of dopamine and norepinephrine in anorexia nervosa are also weight related [47]. In summary, while it remains possible that there is a hypothalamic dysfunction in anorexia nervosa unrelated to the starvation and weight loss, the evidence is by no means compelling. Furthermore, this theory cannot easily explain why the disorder affects selected groups in the population and is apparently increasing in incidence.

COGNITIVE BEHAVIORAL THEORY

The cognitive behavioral theory on the etiology of anorexia nervosa emphasizes cognitions and behaviors rather than early childhood experience, family interaction, or biological processes [47,79]. However, it does draw on individual psychodynamic and psychobiological concepts. Garner and Bemis [79] and Garfinkel and Garner [47] set out their views systematically on this theory, but they were careful to point out that cognitive behavioral theory explains only one aspect of what they consider to be a multiply determined disorder. As they

see it, anorexia nervosa is the final common pathway of a pathological sequence of events beginning with the introverted, sensitive, and isolated adolescent arriving at the idea that weight loss will somehow alleviate her distress and dysphoria. The dieting that follows is therefore a means to achieve slimness as well as an expression of ascetic control. It is reinforced by a gratifying sense of success and by approval and concern from others. The negative reinforcement of avoiding food and weight gain gradually becomes more prominent, and the increasing isolation that occurs as a result of starvation decreases the youngster's responsiveness to other issues and considerations. Progressive weight loss secures a "safety margin," [80] and the anorectic cognitions and behaviors become autonomous.

This theory is intuitively familiar and consistent with the patients' own reports. However, the more crucial question may be why more dieters do not move toward this final common pathway. While this theory may explain how the illness perpetuates itself, it does not explain what distinguishes between a normal dieter and a committed anorectic. Garner et al [81] recently provided some evidence to suggest that anorectics score higher than normal dieters on several subscales of the Eating Disorder Inventory, which measures ineffectiveness, interpersonal distrust, and lack of interoceptive awareness. Thus, certain vulnerable individuals may choose to starve themselves in an attempt to gain confidence and control.

THE AFFECTIVE DISORDER THEORY

Cantwell et al [51] have recently stated that anorexia nervosa may be an atypical affective disorder occurring in an adolescent female at a time in her life when body image issues were important. Two findings in their study supported this view: there was an increased incidence of affective disorder in the family, and on long-term follow-up the anorectics were more prone to develop affective disorder than to suffer a relapse of the eating disorder.

The former finding is supported by several studies [53,54], and the relationship of familial affective disorder to anorexia nervosa has been discussed previously.

Biological marker studies in the two disorders have yielded inconclusive findings regarding their relationship. Plasma cortisol level [82], dexamethasone nonsuppression [83], and low urinary MHPG have been found in primary affective illness, and these changes are also seen in some anorectics. However, the abnormalities in the latter are apparently reversible with weight gain [78,84]. Moreover, severe weight loss per se is known to produce dexamethasone nonsuppression [83,85]. The characteristic sleep EEG findings seen in primary affective

tive disorder [86] are different from those seen in anorexia nervosa [87].

Phenomenologic and outcome studies have likewise been inconclusive. Anorectics are not as overtly or persistently depressed as patients with affective disorder [88,89]. Depression may be associated with complex changes in weight and appetite [90,91], and it remains uncertain whether the dysphoria in anorexic nervosa is primary or secondary to the eating disorder [56]. No study exists to demonstrate that anorexia nervosa is more common in the family of affective disorder probands; most follow-up studies indicate that anorexia nervosa "breeds true" [92]. Finally anecdotal reports of the usefulness of anti-depressants in treating anorexia nervosa [93,94] have not been widely accepted [47,72,80].

While the complex relationship between the two disorders requires further study, there is no compelling evidence at present to believe that anorexia nervosa is a form of affective illness in an adolescent female.

CONCLUSION

While it is possible that anorexia nervosa has a single discrete cause, it is equally possible that complex chains of events interact to finally precipitate the illness. Kendell [95] clearly favored the latter view for psychiatric diseases in general. He even stated that, "The very idea of 'cause' has become meaningless, other than as a convenient designation for the point in the chain of event sequences at which intervention is most practicable," (p. 64). The overlap of the theories reviewed in this chapter certainly suggests that this argument is at least plausible in the case of anorexia nervosa. If this is so, the challenge will be to identify such events and how they interact. Needless to say, such events may include some or all of the proposed etiological factors already reviewed—or none of them. A possible strategy may be to prospectively study and follow a group of youngsters considered to be at risk for developing the illness—for example, professional dance and modeling students. The logistical and ethical problems involved in such a study may, however, be prohibitive.

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