

ALTERATIONS OF EATING BEHAVIOUR IMPACT OF BIOLOGIC, PSYCHOLOGIC AND ENVIRONMENTAL FACTORS AND PSYCHIATRIC DISEASE

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The relative importance of biologic, psychologic, environmental and sociocultural factors and of psychiatric disease in the development and course of eating disorders is, in contrast to many claims, not at all clear. There is even no general agreement as to which clinical entities have to be regarded as an eating disorder and as to whether the various eating disorders are clearly distinguishable from each other. By some investigators, they are rather viewed as lying on a continuum spread across a number of parameters which are partially independent of each other: undernutrition versus overnutrition, restriction versus indulgence, activity versus inactivity, abstinence behaviours such as dieting versus “purging behaviours” such as vomiting, and persistence of restraint eating versus intermittent reactive hyperphagia¹.

This review presents what is known about biologic, psychologic and environmental factors as well as psychiatric disease and the interactions of such factors, which may lead to and maintain a disordered eating behaviour.

Obesity

Is obesity, the excess accumulation of body fat, an eating disorder? Against such a notion speak the facts that the obese typically have not become fat because of a particular eating behaviour, and that their maintenance of a given body weight cannot be accomplished by a normalisation of eating patterns, but rather by a sustained restriction of energy intake, in particular of energy-dense high-fat food², and an augmentation of energy expenditure³. Obesity not only is one of the most stigmatised physical attributes in the eyes of the general public, but also is regarded as a disorder greatly determined by psychological factors. The latter notion is held despite the lack of indications for a specific

disturbance and the little evidence that obese individuals are psychologically more disturbed than others⁴⁻⁶ or eat more in response to emotional distress⁷. The psychopathology encountered in obese individuals seems not to be a cause but rather a consequence of obesity, specifically a consequence of the prejudice and discrimination to which the overweight are subjected in our society⁸. By contrast, data are accumulating⁹⁻¹⁴ to indicate that genetic factors play a major part in the development of obesity and this combined with the provocative environment in affluent societies. Genetic influences, however, are not so strong as to render dietary and behavioural treatments irrelevant¹⁵. The poor long-term results of dietary and behavioural treatments^{16,17} may be attributable to the fact that many programmes of that type do not include teaching of weight maintenance behaviours. Patients discover that they are unable to reach their goal weight which reinforces their view that they are incapable of lowering their weight to any meaningful extent¹⁸. Of central importance for dietary/behavioural treatment approaches, therefore, is to help patients to adopt more realistic weight goals and to shift their focus from changing their appearance to improving their physical health¹⁹.

Binge-eating disorder

Of obese individuals, 20% to 40% report significant problems with binge eating²⁰, that is, the eating of greater quantities than most people would eat during similar periods of time under similar circumstances^{21, 22}. In a multisite field trial, about 30% of obese patients taking part in weight control programmes met the then proposed diagnostic criteria for binge-eating disorder²³. These criteria in the meanwhile made their way into the Diagnostic and Statistical Manual of the American Psychiatric Association²⁴. They define individuals who eat an amount of food definitively larger than most people would eat in a similar period of time under similar circumstances, have a sense of lack of control over eating during the episodes, but do not engage, in order to avoid weight gain, in compensatory activities such as purging, fasting or excessive exercise. Among obese subjects accepted into treatment programmes, bingeing was found to become significantly more prevalent with increasing degree of adiposity²⁵. This was attributed to the facts that the excessive

diETING associated with being overweight leads to eating binges and that the weight cycling resulting from repeated dieting and refeeding not only gives way to more rapid weight gain, but also to a greater propensity toward binge eating^{26,27}.

Obese binge eaters have been found to show more symptoms of affective disorders, i.e., depression, overall distress and anxiety, and also a greater life-time prevalence of psychiatric disorders than obese non-bingers^{6,28-30}. In states of depressed mood, eating, especially the intake of simple carbohydrates, can yield a temporary lifting of mood³¹⁻³³ and energy levels³⁴. Of grossly obese women with transient episodes of depression, 74% have been observed to crave carbohydrates in addition to their regular food intake³⁵. A specific hunger for carbohydrate-rich foods and an inability to control carbohydrate consumption was suggested as being one factor promoting weight gain or hindering weight loss. The craving, furthermore, was regarded as being characteristic of seasonal affective disorders³⁶. In some of the obese, by contrast, negative mood states may be overcome by the contrary of carbohydrate craving, i.e., the elimination of simple carbohydrates from the diet^{37,38}. There is no evidence to suggest, however, that obesity, as such, is systematically related to depression. Obese girls were found to be discontent with their weight and figure but not depressed³⁹.

Bulimia nervosa

Binge eating can be encountered not only in the obese but in normal-weight individuals as well. Of the latter, many have a morbid fear of becoming fat and try to get rid of the unwanted calories they voraciously ingest using compensatory manoeuvres, such as self-induced vomiting, the use of laxatives or diuretics, strict dieting between the binges, or vigorous exercise. If the binge-eating episodes occur at a minimum average of two per week for at least 3 months, the over-concern with body shape and weight is persistent, and the affected individual has the feeling of a lack of control over her/his eating behaviour during the binges, the criteria for bulimia nervosa²⁴ are met. This disorder has been reported to become more and more prevalent among young women, especially on college campuses^{40,41}, although clinically significant bulimia seems to be rare^{42, 43}.

The duration of a binge-eating episode in a patient with bulimia may vary from a few minutes to 8 hours^{44,45} and the ensuing energy intake from less than 100 kcal to several thousand kcal⁴⁶. In patients having 24-hour access to food, a daily intake of up to 42694 kcal was observed⁴⁷. Foods otherwise not eaten^{31,46} or perceived, because of their high-fat and energy-dense nature, as forbidden⁴⁸ or dangerous⁴⁹ have been reported as being more likely to be consumed. Others, however, found that the macronutrient composition of a binge did not differ from that of the diet eaten by the general population⁵⁰. Between their binges, patients with bulimia were observed to eat less at meal times than matched non-bulimics⁵¹. It is this self-control the patients are able to exert under normal conditions which leads them to consider a loss of control, and not having eaten too much, as the central feature of their disorder⁵².

Upon the bulimic ingestion of large quantities of food, not only distension and pain^{31,53,54}, but also infarction and rupture of the stomach are encountered⁵⁵⁻⁵⁷. Distension may be due not only to acute overeating but to a stasis of ingesta as well: in no less than 9 out of 24 consecutive patients with bulimia, the gastric emptying of a semisolid standard meal of a nutrient content of 1168 kJ was found to be grossly delayed⁵⁸. The emptying of liquids was found to be prolonged in 13 out of 23 and the emptying of solids in 7 out of 23 patients with bulimia⁵⁹. In another study⁶⁰, the emptying of a liquid meal, which was ingested until the subjects felt extremely full and which contained 1.68 kJ/l, was significantly slower in its initial phase in 9 bulimic women than in matched healthy women. The fact that the bulimics also had a significantly greater capacity of the stomach led the authors to suggest that patients with bulimia felt satiated only after larger and larger quantities of food. This deficit in the mechanisms which normally serve to turn off eating and the increased gastric capacity have been suggested to give rise to a delayed gastric emptying and a blunted postprandial cholecystokinin release, leading to an impaired satiety response and thus a perpetuation of the disorder⁶¹. By contrast, other workers found normal gastric emptying to prevail in bulimic individuals⁶²⁻⁶⁴. In one of the latter studies⁶⁴, however, the gastric emptying data of 11 patients were compared to those of a very heterogenous group of 16

premenopausal females, which included individuals with morbid obesity and inflammatory bowel disease and whose gastric half-emptying times were highly scattered. Shih et al.⁶⁵ found the emptying of cream of wheat to be delayed in 12 of 20 bulimic patients, but to be rapid in the remaining eight. The latter finding contrasts sharply with the results of other studies^{58,59,62-64}, in which rapid emptying had not been encountered in any patient.

The factors contributing to an impaired gastric motor function in patients with bulimia have not been fully elucidated. Weakness and atrophy of gastric smooth muscle, caused by malnutrition and/or electrolyte depletion, primarily serum potassium^{66,67} but also total potassium depletion⁶⁸, resulting from repetitive self-induced vomiting and/or an abuse of laxatives or diuretics, might play a crucial role. In one study⁵⁸, there was a significant inverse relationship between gastric half-emptying times and serum-potassium levels, which were below the normal range in 4 out of 24 patients studied and near the lower borderline of normal in another 8. As delayed emptying can readily be combatted by prokinetic agents, its detection is of great importance for the successful management and the outcome of patients. However, a normal eating behaviour cannot be attained by the normalisation of gastric emptying alone. The feeling of abdominal distension resulting from the large quantities eaten during a binge is terminated by self-induced vomiting, which allows either the continuation or termination of the binge. The recurrent vomiting gives rise to a progressive decalcification of tooth enamel. This leads to a loss of enamel and dentin mainly at the lingual surface of the teeth, an increased caries propensity and dental fracture rate, as well as an increased sensitivity to hot, cold and acidic substances⁶⁹. Another feature frequently observed is an intermittent parotid swelling, occurring 2 to 6 days after the cessation of an overeating episode. The associated moderate elevation of serum amylase has been shown to be caused by an increased salivary-type amylase activity^{70,71}. The habitual self-induced emesis may further cause oesophagitis, erosions, ulcers and strictures, and also lead to oesophageal perforation, mediastinitis and mediastinal emphysema^{72,73}. Self-induced vomiting further may affect oropharyngeal swallowing. This is suggested by a videoradiographic study in 13 bulimic patients, all of whom were

found to have abnormal oropharyngeal swallowing patterns⁷⁴. In one study⁵⁸, four out of 32 consecutive patients diagnosed as having bulimia were revealed to have achalasia and one to have diffuse oesophageal spasms. All of the latter 5 patients reported experiencing two different types of vomiting: one self-induced, in which the vomited material tasted either acidic or non-acidic and like the preceding meal, and the other involuntary, in which the vomited material was always non-acidic and of the same taste as the preceding meal. Although the involuntary emesis occurred also in situations in which the patients did not intend to get rid of their ingesta, all of them thought that this vomiting, and their dysphagia and retrosternal pain, were consequences of the self-induced vomiting. This was the more plausible to them, as all had had no such symptoms prior to the onset of bulimia. Thus, the clinical evaluation of patients with bulimia should always include the taking of a thorough history with regard to swallowing and vomiting as well as to the nature and taste of the vomited materials. An important physiological consequence of binge eating is an increase in metabolic rate, which is partly due to an increased thyroid hormone activity⁷⁵.

For patients with bulimia nervosa, the bingeing and subsequent compensatory manoeuvres may provide relief from dysphoric moods⁷⁶ and feelings of anxiety. In one study, about three quarters of binge eaters stated that they were free of negative moods while eating³¹. However, eating may not only be precipitated but also maintained by negative mood states⁷⁷. Mood has been observed to worsen⁷⁸ and depression ratings to increase after a binge^{79,80}. Some authors suggested that bulimia was a variant expression of a primary affective disorder⁸¹. This concept seemed supported by reports that many patients, at least those presenting for treatment, were depressed⁸²⁻⁸⁷ or differed from control subjects in a significantly higher prevalence of personal and familial major mood disorders⁸⁸. The morbid risk for major depressive disorder was found to be significantly greater among first-degree relatives of patients with bulimia than among non-psychiatric control subjects⁸⁵ and similar to the risk in families of patients with bipolar disorder⁸⁹. However, many of the features generally regarded as being at the core of classic melancholia, that is, anhedonia, irritability, decreased concentration, lack of reactivity to the environment, diurnal variation in mood, marked motor

retardation, fatigability and diminished libido, were found to be conspicuously absent in most patients with bulimia⁹⁰. Moreover, such symptoms may be attributable to starvation⁹¹: the nutritional status of patients with bulimia tends to be, despite a regular body weight, quite abnormal. In fact, a series of authors reported depression to be secondary to bulimia and/or malnutrition⁹²⁻⁹⁴. Against the concept that bulimia is a variant expression of a primary affective disorder are also the results of other studies. In first- and second-degree relatives of bulimic women, no higher rates of affective disorders were detected than in relatives of women with no history of an eating disorder⁹⁵.

A close relationship between bulimia and affective disorders seemed to be suggested by therapeutic successes obtained with the short-term medication of antidepressant drugs; with continued medication, however, only limited beneficial effects and a considerable relapse rate were observed^{96,97}. Treatment with imipramine yielded results inferior to those of structured intensive group therapy and psychotherapy plus imipramine had no better effect than psychotherapy alone⁹⁸. By contrast, two double-blind studies in 382⁹⁹ and 398¹⁰⁰ bulimic patients showed that 60 mg per day of the selective serotonin-reuptake inhibitor fluoxetine administered over 8 and 16 weeks, respectively, resulted in a significantly greater reduction of binge eating and vomiting than did placebo. An argument against the interpretation of bulimia as a variant of an affective disorder is provided by the fact that the sleep disturbances characteristic of patients with major depressive illness were found not to prevail in bulimic patients, who, by contrast, exhibited sleep patterns remarkably similar to those of healthy individuals¹⁰¹. A link between bulimia and affective disorders seemed to be indicated by the failure of about half of the patients studied to suppress their cortisol secretion in response to dexamethasone normally^{81,102}. In the meanwhile, dexamethasone non-suppression has been shown to occur in a variety of physical and psychiatric disorders and not to be specific for patients with depression¹⁰³. By contrast, the test proved to be highly sensitive to weight loss^{104,105}. This is demonstrated also by the results of a study in bulimic patients, in whom the sole clinical correlate of dexamethasone non-suppression was a history of weight loss and/or anorexia nervosa¹⁰⁶. To conclude the discussion about whether or not bulimia is associated

with affective disorders, the preponderance of evidence suggests that the two entities are distinct from each other. In one patient, a depressed mood or depression may result from an eating disorder, whereas in another depression may be an antecedent to abnormal eating behaviour. A series of studies suggested an association of bulimia nervosa with personality disorders. In one investigation¹⁰⁷, 19 out of 91 and in another¹⁰⁸ 15 out of 39 patients with bulimia fulfilled the diagnostic criteria for at least one personality disorder, most often a borderline disorder. A strong association between bulimia and a borderline disorder was also reported by two further studies^{109,110}. By contrast, Pope et al.¹¹¹ found the criteria for a borderline disorder to be met by only one out of 52 patients with bulimia. The latter finding, as well as the results of seven further studies¹¹², speak against a relationship between the two types of disorders.

A number of authors have suggested that experiences of childhood sexual abuse may contribute to the development of bulimia nervosa in adolescence or early adulthood. However, controlled studies, on the whole, did not support this hypothesis¹¹³. Sexual abuse was found to be a risk factor for the development of a psychiatric disorder in general, but not specifically for bulimia nervosa¹¹⁴.

A genetic predisposition for bulimia nervosa is suggested by a report that 5 out of 6 monozygotic and 4 out of 15 dizygotic female twin pairs were concordant for the eating disorder¹¹⁵.

Anorexia nervosa

The diagnostic criteria for anorexia nervosa²⁴ include a refusal to maintain body weight at or above a minimum weight for age and height, an intense fear of gaining weight or becoming fat even though underweight, a disturbance in the way in which body weight, size or shape are experienced, and the absence of at least three consecutive menstrual cycles when otherwise expected to occur. The term anorexia nervosa is a misnomer, as there is no loss of appetite. By contrast, affected individuals are engaged in a relentless pursuit of thinness and display a phobic avoidance of being fat¹¹⁶. The most important primary drive system, which yields pleasure or positive reinforcement with eating and negative reinforcement by hunger, is perverted: although hunger may

still cause discomfort, the consequence of sustained hunger, that is weight loss, causes pleasure. Food stimuli become something to be abhorred although they, at the same time, dominate the patients' mind¹¹⁷. An increased physical activity of patients with anorexia has consistently been commented on, but also has been found in a controlled study¹¹⁸. Clinical^{119,120} and cognitive studies^{121,122} showed that anorexic women chiefly rejected foods rich in fat. However, patients with anorexia nervosa also avoid other calorie-rich foods such as meats, milk products and sweets¹¹⁹, although their perceptions and preferences for a sweet taste do not differ from healthy control subjects¹²².

In the course of food restriction, at first glycogen stores are depleted. Then, muscle tissue is consumed and the necessary substrate for gluconeogenesis is provided by aminoacids. At last, primary fat is mobilised and free fatty acids serve as energy supply. As free fatty acids cannot pass the blood-brain barrier, the brain becomes dependent on the metabolites of the free fatty acids, the ketone bodies. Upon renutrition, these changes rapidly subside. The same applies to abnormalities in hypothalamic-pituitary-adrenal function¹²³, elevated growth hormone levels¹²⁴, impaired or absent responses in plasma growth hormone and cortisol levels to insulin-induced hypoglycaemia¹²⁵, reduced hypothalamic serotonergic responsiveness¹²⁶, impaired reproductive function¹²⁷ and raised levels of serum sex-hormone-binding globulin¹²⁸. Thus, the available data suggest that the afore-mentioned neurobiologic changes represent adaptive responses to starvation and weight loss, whereas there is no evidence for a role of a primary hypothalamic or a neuroendocrine disorder in the development of anorexia.

In contrast to subjects affected by other disorders associated with severe malnutrition, patients with anorexia nervosa do not have an increased infection propensity¹²⁹. This may be due to the maintenance of a normal number of T-lymphocytes of the CD4+ phenotype despite marked weight loss¹³⁰.

The emaciation associated with anorexia nervosa leads also to a reduced skin thickness and skin collagen content¹³¹. These processes may be triggered by the oestrogen deficiency accompanying emaciation. The same seems to be true for the genesis of osteoporosis and the reduced bone mass encountered in nearly all patients with anorexia^{132,133}. The

reduced bone density may persist despite nutritional repletion¹³⁴.

On primary evaluation of young females, vomiting and weight loss may be mistaken as indicating anorexia. The physician may be biased not only by the patients' emaciation but also by their young age and female sex, which may lead her/him to view certain aspects of history and behaviour as suggesting a pathological attitude towards eating. Another factor liable to contribute to misdiagnosis is that patients who are, or have been, in psychiatric or psychosomatically oriented treatment tend to learn "their" psychiatric or psychosomatic history. They thus may not only misinterpret their own sensations but also mislead their physicians. Symptoms such as epigastric distension and bloating are often overlooked or misinterpreted and one of the most renowned researchers in the field, Hilde Bruch¹³⁵, stated: "Anorexics will complain of feeling full after a few bites ... One gains the impression that this sense of fullness is a phantom phenomenon, projection of formerly experienced sensations". Sensations of that type, which were found to be significantly more intense than in healthy subjects¹³⁶⁻¹³⁹, are by no means phantom phenomena: a series of studies demonstrated that gastric emptying is markedly delayed in a high proportion of patients^{66,140-147}.

The mechanisms underlying disordered gastric motor function are still unclear, although malnutrition seems to play a crucial role. In depleted patients with anorexia, not only type 2-muscle fiber atrophy¹⁴⁸⁻¹⁵⁰, which is characteristic for cachexia, but also significant changes in muscle contraction-relaxation characteristics and fatigability properties have been observed¹⁵¹. These features all disappeared with refeeding and the restoration of muscle electrolytes^{151,152}. Muscular atrophy has also been observed to affect the heart, in particular the left ventricle¹⁵³⁻¹⁵⁶. Rhythm disturbances, mitral valve prolapse and diminished exercise capacity may ensue¹⁵⁷. Large U-waves in the electrocardiogram may be due to low serum-potassium levels¹⁵⁸. As to the effects of malnutrition on gastric smooth muscle, atrophies to the point that stomach and intestine appeared as "tissue paper thin" have been observed in Indians with mixed deficiency diseases¹⁵⁹. Muscular decompensation may underlie the acute gastric distension observed upon refeeding of patients with anorexia¹⁶⁰⁻¹⁶². Acute distension has also been found to develop upon the ingestion of large quantities of food in chronically starved prisoners of war¹⁶³,

neglected children^{164,165}, and healthy volunteers participating in starvation experiments⁹¹. With progressing gastric distension, venous occlusion, infarction and rupture may occur^{60, 166,167}. The delayed gastric emptying in patients with anorexia can readily be combatted by prokinetic agents^{142,143,168-170}. Their administration over prolonged periods of time may, by accelerating emptying, not only contribute to an amelioration of the nutritional status, but also help in the rehabilitation of patients¹³⁶. The ultimate goal of therapy, however, i.e., the resumption of a normal eating behaviour enabling the patients' social reintegration and restoration to an appearance acceptable to the social environment, can not be achieved by a normalisation of gastric emptying alone.

Malnutrition may not only affect the stomach but also more aboral parts of the intestine. Ten out of 20 patients with anorexia were found, upon barium-meal examination, to have proximal duodenal dilatation¹⁶¹. A marked dilatation of the duodenum and an absence of the, under normal conditions, regularly recurring activity fronts of the interdigestive migrating motor complex as well as a prolonged intestinal transit were observed in a young female, in whom these abnormalities vanished with renutrition¹⁷¹. In a further study, about one third of the patients were found to have moderate dilatation of small bowel loops¹⁷². The jejunum, which is exposed to high nutrient concentrations under physiological conditions, is much more at risk to undergo hypoplastic changes in states of starvation than is the ileum, which normally has to deal with nutrient-rich chyme. The colon, interestingly, is also very sensitive to withdrawal of food. Constipation associated with slow colonic transit¹⁷³ and abuse of laxatives¹⁷⁴ is frequent in anorexic patients.

A series of papers reported an increased prevalence of anorexia in the higher socio-economic classes¹⁷⁵⁻¹⁷⁹. In 100 consecutive patients presenting to an outpatient clinic in London, there was a definite skew of prevalence towards the upper classes; the skew, however, was less marked than in a survey carried out in the same institution 10 years earlier¹⁸⁰. A study in women taking part in bingo tournaments in Massachusetts suggested that anorexia may be at least as common amongst the more humble classes¹⁸¹. Thus, the assumption of an increased incidence in the higher classes may have arisen as a result of referral practices.

Much more under constant pressure than females of the upper social strata, to diet relentlessly, are professional dancers and athletes. A study in 183 professional dancers revealed that no less than 12 of them fulfilled the diagnostic criteria for anorexia nervosa¹⁸². However, a behaviour directed at inducing weight loss or maintaining a low body weight may promote unusual eating patterns, ritualised eating and obsessive food thoughts resembling those encountered in patients with anorexia⁹¹. A close relationship has been suggested to exist between anorexia nervosa and psychiatric disease. An association with affective disorders was hypothesised on the basis of a 5-year study showing that 18 out of 26 patients with anorexia had persistent dysphoric mood states and often a maternal history of depression¹⁸³. A controlled 10-year follow-up study in 62 women with anorexia revealed a high life-time prevalence of major depressive disorders and a significant comorbidity of anxiety disorders¹⁸⁴. Among these patients' first-degree relatives, the total number of psychiatric diagnoses as well as the prevalence of alcoholism were significantly higher than among age- and sex-matched individuals. Moreover, two of the patients' mothers had had bulimia and each two of other first-degree relatives, anorexia and bulimia, respectively. Of the control subjects' first-degree relatives, by contrast, none had an eating disorder¹⁸⁴. Others found that 46 out of 82 outpatients with anorexia fulfilled the diagnostic criteria for major depressive disorder⁸². One argument brought forward as supporting a close relationship between anorexia and affective disorders was based on the observation that a number of anorexic patients showed an inadequate suppression of cortisol secretion in response to dexamethasone¹⁸⁵. However, this test is highly sensitive for weight loss^{104,105} and normalises with relatively small weight gain.

Against an association of anorexia nervosa with affective disorders stand the results of a study⁸² in which the onset of an affective disorder post-dated, in the great majority of patients, the onset of the eating disorder by at least 1 year. This led the authors to conclude that the depression was secondary. Such an interpretation seems also to be supported by the fact that there is less than meager evidence that antidepressant drugs are of benefit to the majority of patients with anorexia^{97,186}. Notwithstanding, comorbid mood and anxiety disorders must be diagnosed and treated.

An association of anorexia nervosa with personality disorders has been suggested in view of the overlap between the phenomenologies of the two disorders, i.e., the rigid dietary routine and rumination about food as well as the repeated weighing and calorie counting. Premorbid obsessional personality traits may be over-represented in patients with anorexia, and the process of the illness may serve to exaggerate such traits¹⁸⁷. In 20 patients having recovered from anorexia nervosa, characteristics such as a need for order and precision were found to be significantly more accentuated than in healthy women¹⁸⁸. Of 31 patients, seven were found to fulfill the criteria for at least one personality disorder¹⁰⁷. A previous history of anorexia was found in 12 out of 105 patients with an obsessive-compulsive disorder¹⁸⁹; in these 12 patients, the two diseases had a similar time of onset, which may point to common vulnerability factors.

A genetic predisposition may play a role in the development of anorexia nervosa. Holland et al.¹⁹⁰ found that 14 out of 25 monozygotic and 1 out of 20 dizygotic female twin pairs were concordant for anorexia. Of the female first-degree relatives of these twins, nearly 5% also had a history of anorexia. This led the investigators to conclude that up to 80% of the variance in liability to develop anorexia may be accounted for by genetic factors. In a study in the family members of 97 patients, it was found that two out of the 98 sisters had anorexia and four had bulimia nervosa¹⁹¹. These prevalence rates were significantly higher than those occurring in families with no eating disorder and also significantly higher than the prevalence rates for anorexia in the families of 66 patients with primary affective disorder and 117 patients with various non-affective disorders. Against a genetic predisposition for anorexia nervosa stand the results of a study which revealed that none of the same-sex co-twins of 11 patients with anorexia was concordant for the eating disorder¹⁹². Another contra argument is provided by the finding that 58 mothers of girls with anorexia and 204 mothers of healthy girls of similar age and socio-economic status did not differ in their weight history and their attitudes towards weight-related matters¹⁹³. Furthermore, no differences, in terms of attitudes to weight control and dieting, were found between the parents of another group of patients with anorexia and matched healthy individuals¹⁹⁴.

In conclusion, there is no clear-cut evidence to suggest that distinct

biologic, environmental, psychologic, or psychiatric factors are of primary aetiological importance for binge-eating disorder bulimia nervosa and anorexia nervosa. The development and course of an eating disorder, however, should be seen as determined by an interplay of forces at the biologic, psychologic, familial and sociocultural levels of organisation¹⁷⁹. The thorough evaluation of these forces and their interaction may not only yield insight into the origin and course of the eating disorder in an individual patient, but also provide the basis for a successful therapy.

Summary

The relative importance of biologic, psychologic and environmental factors and psychiatric disease in the development and course of eating disorders is unclear. Obesity is widely regarded as being determined psychologically, although the psychopathology encountered appears rather as a consequence of the disorder. There is no evidence that obesity is related to affective or personality disorders, but genetic factors seem to be crucial. Obese patients with binge-eating disorder showed more symptoms of depression and anxiety, and a greater lifetime prevalence of psychiatric disorders than obese patients without binge-eating disorder. In bulimia nervosa, binge-eating and subsequent compensatory activities, such as self-induced vomiting and purging, may yield relief from dysphoric moods, but evidence for an interrelation with affective or personality disorders is meager. The bulimic behaviour and electrolyte depletion resulting from repetitive vomiting may impair oesophageal and gastric motility. Anorexia nervosa is associated with neurobiologic changes adaptive to starvation, but there is no evidence for a primary neuroendocrine disorder or an association with affective or personality disorders. By contrast, a genetic predisposition may play a role. Delayed gastric emptying develops secondary to malnutrition and subsides with refeeding. The development and course of eating disorders seems not to be determined primarily by psychological or psychiatric factors but by the interplay of such factors with forces at the biological and sociocultural levels, the evaluation of which may provide the basis for a successful therapy.

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