Chapter 8
Eating Disorders

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Abstract

Anorexia and bulimia are diseases known since ancient times but, in recent years, their frequency has been continuously increasing in most industrialized countries. The etiology of these disorders can be traced back to the interaction between genetic predisposition, childhood experiences, and cultural pressures. As regards the course, a certain tendency to chronicity can be observed, and in extreme cases, they can cause death. According to the diagnostic classification of the DSM-5, Eating Disorders include Anorexia Nervosa, Bulimia Nervosa, Binge Eating Disorder (which, compared to DSM-IV, becomes a diagnostic category in its own right), and Other Specified Feeding and Eating disorders (OSFED). Both anorexia and bulimia cause potentially serious medical complications. To maximize the chances of good outcomes a multidisciplinary intervention is necessary with staff including professionally heterogeneous figures: a psychiatrist, a psychologist, and a nutritionist. Therapeutic success for these patients is limited. Eating disorders require, among psychiatric disorders, the greatest possible collaboration between different professional figures with different specializations.

Keywords: Eating disorders; Anorexia; Bulimia; Emotional health; Bingeing; Purging; Body image; Integrated treatment

8.1. Definition and epidemiology

Eating disorders are among the most common health problems in adolescents and young adults in Western countries. The incidence of Anorexia Nervosa (AN) is at least 8-9 new cases among women, while for men it is between 0.02 and 1.4 new cases per 100,000 people a year. Even more relevant is Bulimia Nervosa (BN), which every year records 12 new cases per 100,000 people among women and about 0.8 new cases per 100,000 people in one year among men (Italian Ministry of Health, 2020). DSM-5 defines feeding and eating disorders as follows: "Feeding and eating disorders are characterized by a persistent eating disorder or diet-related behaviors that result in impaired consumption or absorption of food and which significantly damage physical health or psychosocial functioning". However, the DSM-IV did not provide a precise definition of eating disorder, thus causing numerous issues in settling the diagnostic boundary of Eating Disorders Not Otherwise Specified (EDNOS). Thanks to the integrated approach (dimensional approach and categorical approach) of the fifth edition of the DSM, new diagnostic categories have been included, and some diagnostic criteria have been modified. This allows patients to receive a diagnosis that accurately define their symptomatology, to establish an adequate treatment plan. They, therefore, include, according to the new DSM-5: pica, rumination disorder, Avoidant/Restrictive Food Intake Disorder (ARFID), anorexia nervosa, bulimia nervosa, binge eating disorder (BED), Other Specified Feeding or Eating Disorders (OSFED).

8.2. Etiopathogenesis

Eating disorders are multifactorial disorders both in terms of the risk factors that determine them and in terms of treatment. This implies that taking in charge of the subjects who are affected must be carried out, from the beginning of the therapeutic path, on several fronts: psychiatric, medical, psychological, alimentary, familiar, and relational, in order to reduce the probability of the chronicization of the disorder and likewise decrease the likelihood of bad outcomes. Risk factors for EDs include social, family, psychological, developmental, and biological factors.

Among the *biological factors*, we find some obstetric complications such as maternal anemia, diabetes mellitus, pre-eclampsia, placental infarction, neonatal heart problems. It has been observed that the greater is the number of adverse neonatal events, the higher is the risk of developing an eating disorder. The importance of genetics in determining a vulnerability to DCA was then also emphasized

by several studies. Several papers report, for example, that the concordance rate for monozygotic twins is significantly higher (about 50-60%) than that for heterozygous ones, underlining the weight of genetic factors. Genetic factors could influence at the time of puberty the production of ovarian hormones, in particular estradiol, involved in the genetic transcription of neurotransmitters, such as serotonin, which regulate mood and appetite. Empirical data show that the *onset of puberty* must be considered as a relevant risk factor, and these data emphasize the association between anticipation of pubertal development and increased risk of developing an eating disorder. During early pubertal development, there is a state of greater anxiety related to physical change leading to a doubling of the fat mass in the body and the appearance of feminine forms. This change does not coincide with the maturation of the patient's identity and makes the subject vulnerable to the comments of parents and peers. The new body structure determines a conflict with the dominant aesthetic ideals and, in a condition of increased anxiety and impulsivity, can generate hyper-control behaviors that lead to a condition of fragility in which an eating disorder can easily develop. *Tolerance to distress* represents another element linked to a complex interaction between genetic and environmental factors. According to some authors, tolerance to distress is "the ability to resist and accept a negative emotion, to be able to develop a problem-solving process". A risk factor is represented precisely by a lack in the ability to choose cognitive and behavioral strategies for the management of experiences linked to both positive and negative emotions.

Other predisposing risk factors include the presence of familiarity for Eating Disorders (ED), pre-morbid temperamental predisposition to perfectionism (which indicates a greater risk for the restrictive subtype of AN), tendency to be excellent, fear of others' judgment related to fear to disappoint, overeating in the family (indicating a greater risk for the binge-purge subtype). Overeating in the family is a risk factor shared by both BN and BED.

The *socio-cultural and psychological aspects* are factors of primary importance in the development of the disorder as well. Among these, we can consider the female models present in our historical-cultural period, accentuated by the use of the female image in social networks, low nuclear self-esteem, marked interpersonal difficulties, and intolerance to emotions.

Among the *triggering causes* we can commonly observe: relational problems that have arisen within the family (separations, quarrels, high conflict), sentimental or school or dynamic delusions established within social networks, competition in some sports, comments between peers, and, very frequently, the starting an "occasional restrictive diet" or even choosing "healthier foods" in one's diet. Another relevant trigger is social exclusion, which can influence interpersonal relationships and interrupt the normal development of identity, potentially increasing the salience of competition, the pursuit of perfection, and a greater internalization of the ideals of beauty as a standard.

Among the *maintenance factors* of an organic type, the main place is occupied by the effects of fasting. Weight loss accentuates the idetic focus on food, causes a worsening in the distorted evaluation of body image, and alters the perception of internal hunger and satiety regulation signals. It negatively affects the mood and accentuates obsessive ruminations and social isolation. Finally, it can trigger hyperphagic crises that increase anxiety and the need for subsequent control.
According to the transdiagnostic cognitive-behavioral theory of eating disorders, developed by the Center for Research on Eating Disorders of the University of Oxford, today we tend to consider all eating disorders within a single diagnostic continuum, rather than separate disorders, since anorexia, bulimia, and binge eating disorder have predominantly common clinical characteristics and sometimes the patients who suffer from them tend to alternate various symptoms (from restrictive behaviors to bulimic crises and compensatory behaviors) migrating thus from one diagnostic category to another.

8.3. Clinical presentation

The psychopathological core of eating disorders consists of an overestimation of the body image and shape (an *excessive preoccupation with the body image*), responsible for a profound alteration in the way in which the subject experiences the relationship with his own body, with one's weight and with food. The overestimation of the weight and shape of the body affects the evaluation of one's self-esteem and the performance perceived by the subject in other domains of life. This symptom seems to have central importance in all three eating disorders (AN/BN/BED) and seems to affect the presence of the other symptoms and the global correlation between them, regardless of the specific diagnosis of the eating disorder. The overestimation of body weight and shape is speculated to be the direct causative factor of many, if not most, of the eating disorder symptoms. It thus becomes a key goal to consider in transdiagnostic treatment and a potentially useful severity specifier for the binge eating disorder.

The patient often overestimates the size of his body and adopts dysfunctional behaviors due to dissatisfaction and fear of gaining weight. Among these behaviors, according to the trans-diagnostic model of development and maintenance of eating disorders, we find “body checking” (BC) and “avoidance of body image” (BIA). Both are due to excessive worry and the need to control the shape and weight of the body, as well as the food to be consumed. Body checking and repeated behaviors aimed at evaluating one's shape, size or weight, perpetuate the pathology both by increasing affective dysregulation and reinforcing the belief that the continuous check of the various parts of the body facilitates the numerical control of weight. “Body checking” refers to the following aspects: the frequent measurement of the dimensions of the various parts of the body with a tape measure; weight

control several times a day; the continuous confrontation with the mirror and with the body of other people. Although some report feeling reassured after confirmation that their body shape has not changed, this does not stop their worry about the body shape and the frequent repetition of this behavior. On the contrary Behaviors aimed at avoiding body image (BIA) include avoiding looking in the mirror or exposing the body, refusal to be weighed, or a tendency to camouflage one's shape with baggy clothes. The other characteristic symptoms are related to the pervasive and obsessive thought of food linked to the "drive for thinness". Most of the patients collect recipes, count calories, have diaries in which they write the kcal consumed during each meal, spend hours eating, and take care of feeding family members, cooking for them. Some food rituals are also common, such as cutting food into small pieces, hiding the food, filtering the oil, and eating very slowly.

As symptoms persist, patients become more irritable, depressed, and socially isolated, and obsessive-compulsive symptoms worsen. In most cases, obsessive symptoms are subsequent and probably consequent to the reduction in caloric intake and conditioned by weight changes; however, in a minority of cases, psychiatric symptoms arise earlier. Affective symptoms and impaired interpersonal functioning are found in all changes in eating behavior. Depressive and anxious symptoms appear to manifest themselves homogeneously in the various disorders and play a central role in maintaining the symptomatic network. The transversal presence of these general psychiatric and interpersonal domains, in addition to the core symptoms described above, confirms the validity of the expanded transdiagnostic theory of ED. Furthermore, interpersonal distrust and a sense of personal ineffectiveness, together with impaired proprioceptive awareness and the urge to thinness, have been shown to be at the center of the network of symptoms in anorexia, bulimia, and BED, and they seem to have a role in determining the prognosis and the outcome at a 5-10 years follow-up. The family context must also be assessed in the evaluation of the maintenance factors of the disorders. Often, during the illness, the relationship with the parents becomes tense and hostile. Investigating the various models of family classes within which the disorder has developed and the impact that the disorder has had on the environment is thus crucial to set up a treatment aimed at changing family interactions and supporting the relatives with psycho-educational interventions about how to take charge even at home during meals and about the prevention of any critical comments that could negatively influence the course of the disorder. Most patients do not have insight into the disorder and/or experience it in an ego-syntonic way but do not recognize how weight loss/gain can be a threat to their survival. Therefore, most of the time, they manifest disinterest or open resistance to undertaking a therapeutic path.

Prognosis

After 21 years:

- Anorexia:
    - 51% of patients in complete remission

SEVERITY	• **Mild**: BMI more than 17 • **Moderate**: BMI 16- 16.99 • **Severe**: BMI 15-15.99 • **Extreme**: BMI less than 15

    - 21% partial remission
    - 10% chronic
    - 16% death
- Bulimia: 70% remission in 11-12 years
- Binge Eating: 60% in remission after 6 years.

However, we frequently encounter the phenomenon of crossover that is the passage from one clinical picture to another, especially between subtypes of anorexia (from restrictive to binge).
Up to 30% of people with a history of eating disorder have a crossover phenomenon.

8.4. Anorexia Nervosa

8.4.1. Definition

In the most accredited international classification for eating disorders, the International Classification of Diseases (ICD 10), and in the Diagnostic and Statistical Manual of Mental Disorders (DSM-5), two main psychopathological characteristics are recognized for Anorexia Nervosa (AN): "fear fatness/fear of gaining weight" and "distortion of body image/disturbance in the way one's body weight is perceived". Fear of gaining weight is generally recognized as a central feature of anorexia, but its dependence on the patient's stage of development (e.g., less readily detectable in underage patients), culture, and stage of the disease question its clinical utility as a diagnostic criterion. For these reasons, DSM-5 extends the "fear" criterion to include "fearless reported" anorexia: even when the patient does not verbalize an "intense fear of gaining weight or becoming fat", the clinician can make a diagnosis of AN if “persistent behavior that interferes with weight gain is detected.” On the other hand, ICD-10 provides a more psychopathological definition of fears: it defines fear of fatness as a pervasive idea, intrusive and overrated, suggesting a strong link (corroborated by the literature) between AN and obsessive-compulsive spectrum disorders. As far as the distortion of the body image

is concerned, both ICD-10 and DSM-5 define it as a core aspect of the psychopathology of anorexia nervosa and require it as a necessary criterion for the diagnosis of AN.

Both classifications, however, do not investigate the usefulness of insight, or awareness of the disease, in AN. Scales such as The Yale-Brown-Cornell Eating Disorders Scale (YBC-EDS) and Brown Assessment of Beliefs Scale (BABS) may be useful to evaluate this construct.

In anorexia, a frequently present physical symptom is amenorrhea, traditionally considered a milestone in the diagnosis of AN and described by the ICD-10 within the category of "diffuse endocrine disorders involving the hypothalamus-pituitary-gonadal axis ". However, in the DSM-5, the requirement of amenorrhea was eliminated as a diagnostic criterion for various factors, including the increasingly common use of contraceptives among women, the presence of women with AN in

BOX 1: DSM-5 Diagnostic Criteria for Anorexia Nervosa

A. Restriction of energy intake relative to requirements, leading to a significant low body weight in the context of the age, sex, developmental trajectory, and physical health. Significantly low weight is defined as a weight that is less than minimally normal or, for children and adolescents, less than that minimally expected.

B. Intense fear of gaining weight or of becoming fat or persistent behavior that interferes with weight gain, even though at a significant low weight.

C. Disturbance in the way in which one's body weight or shape is experienced, undue influence of body weight or shape on self-evaluation, or persistent lack of recognition of the seriousness of the current low body weight.

SUBTYPES

Restricting type: During the last three months, the individual has not engaged in recurrent episodes of binge eating or purging behaviour (i.e. self-induced vomiting, or the misuse of laxatives, diuretics, or enemas). This subtype describes presentations in which weight loss is accomplished primarily through dieting, fasting and/or excessive exercise.

Binge-eating/purging type: During the last three months the individual has engaged in recurrent episodes of binge eating or purging behaviour (i.e. self-induced vomiting, or the misuse of laxatives, diuretics, or enemas).

SEVERITY

Mild: BMI more than 17

Moderate: BMI 16- 16.99

Severe: BMI 15-15.99

Extreme: BMI less than 15

menopause or prepubertal age, the small but relevant percentage of anorexic males, and, above all, the minority of women who continue to menstruate but meet the other criteria for AN.

8.4.2. Epidemiology

The disease affects about 0.3% of adolescents and young adults in Western countries (although the rates of a subclinical disorder would be higher), while it is rare in developing countries where there is less social pressure towards thinness. The disease is more common in women than in men with a woman-to-man ratio of 10: 1. The psychopathological onset takes place between the ages of 13-14 and 25, with peaks between 15 and 18 years. Currently, the disease rate is homogeneous in the different social classes and there is no evidence, as in the past research, of a higher prevalence in the middle-upper class.

8.4.3. Ethiopathogenesis

Anorexia nervosa has a multifactorial and multidimensional etiopathogenetic model typically characterized by the interaction of three classes of risk factors: *socio-cultural, individual and familiar.* Concerning socio-cultural factors, anorexia has been defined by the French Devereux as an "ethnic disorder", that is, as a phenomenon capable of expressing the anxieties and contradictions of society such as those typical of industrialized countries. However, although teenagers of Western civilizations are subjected to the psychosocial pressure of thinness, only a small part develop EDs.
We can, therefore, deduce that there are other factors, such as *individual* ones, that must coexist with environmental and social ones for a disorder such as anorexia nervosa to develop. It has been found that patients suffering from restrictive anorexia typically have an obsessive-compulsive personality, with social inhibition and emotional restraint, and a strong drive for perfectionism. On the contrary, patients with bulimic-purgative anorexia have an outgoing and impulsive personality. Among the predisposing factors to EDs, we also find reduced self-esteem. Regarding the *familiarity* in the etiopathogenesis of eating disorders, it is still not clear which genes are involved in the genesis of the disorder, but studies on twins highlight a higher incidence of the disease in homozygous twins compared to heterozygotes. As for the non-genetic family aspects, the family of the anorexic patient has traditionally been described, since the early 1980s, as an "anorexogenic family", characterized by the presence of an anxious, sometimes "oppressive" mother and an absent father. More recent scientific studies consider the cause-effect relationship between family characteristics and anorexia nervosa simplistic, emphasizing the point of view, universally valid for scientific studies, for which "correlation is not causation".

8.4.4. Clinical presentation

The onset of anorexia nervosa is often gradual and insidious, with a progressive reduction in food intake. In most cases, the disorder occurs because of a low-calorie diet that started to change the body weight and shape, excluding carbohydrates and fats from the daily diet. Sometimes even the selection of "healthy foods" can be related to the onset of anorexia nervosa. In other cases, the symptoms may appear after digestive difficulties, diseases (including depression), surgery, or traumas. Before the presentation of the clinical picture, stressful events, or life changes (e.g., losses, separations, job failures) are often observed. Patients decrease caloric intake by reducing food servings or by skipping snacks between meals and then, gradually, also the main meals, resulting in a significant weight loss. "In the first months, there is a phase of subjective well-being, due to the weight loss, the associated improvement of one's image, and the feeling of omnipotence produced by the ability to control hunger, while awareness of the problem is low and there is no request for help (the so-called "honeymoon phase" with the disorder). Later, concerns about body shape and weight become pronounced, and the fear of gaining weight does not diminish with weight loss".

To reduce weight, some people use self-induced vomiting or abuse of laxatives, diuretics, or, more rarely, anorectic drugs. This subgroup (bulimic/purgative subtype anorexia) has a worse prognosis, more frequent medical complications, and, from a psychopathological point of view, greater comorbidity, with a greater frequency of impulsive behaviors, self-harm, suicide attempts, alcohol, or other substances, abuse.

Family relationships can often deteriorate, becoming substantially blackmailing. Vicious circles are built between parents and patients: parents try to stimulate their daughter to eat more, spy on her, control her, limit her, so exasperating the behavior of the girl, who hides the food or acquires a particular skill in leaving the plate to give them the impression that the food has been consumed (chop, crumble, drain oil, etc.).

Despite the starved condition, the anorexic patient maintains physical hyperactivity, dedicating herself to long walks (up to 20-30 km per day) and exhausting workouts. She does not seem to feel fatigued and cold until the most advanced stages of the disease. In fact, in these patients, there is a difficulty in recognizing, perceiving, and responding to somatic needs: the patient does not feel hunger and cold or perceives them in an altered way. Another characteristic typically found in anorexia is the distortion of the body image, with a persistent tendency to see oneself fat, or with a physical appearance that does not satisfy the "ideal" one.

Anorexia nervosa has significant psychiatric comorbidity. The most frequently associated disorders are major depression, obsessive-compulsive disorder, and other anxiety disorders, alcohol or

substance addiction. Among the personality disorders, the most frequent are borderline, narcissistic, and avoidant disorders. Psychiatric comorbidity tends to decrease with weight recovery and normalization of nutrition.

Physical complications and laboratory tests

- *Liver:* liver function is impaired, with high transaminases and very low proteins. If, on the other hand, we were faced with a patient in a phase of anorexia in which the muscle tissue is still minimally intact, we would find high values of proteins. Cortisol is the hormone that carries this proteolytic response and, in fact, subjects with anorexia in initial phases are always hypercortisolemic.
- *Blood:* mixed anemia is noted in the blood count, with low iron due to decreased intake (in a condition of anorexia, however, amenorrhea is present in the female sex, and therefore the amount of iron lost will be less). White blood cells are only increased in severe infections but are usually low due to prolonged fasting.
- *Kidney:* renal function is impaired, with low urea and creatinine, due to the reduced protein metabolism. Only in case of dehydration or intense physical activity, the values can be high.
- *Muscle:* in some cases, the CK increases much due to intense muscular exercise carried out on weakened muscles.
- *Gonads:* sex hormones are significantly reduced. Amenorrhea is frequently present in the female sex.
- *Hydro-electrolyte balance:* sodium is low in patients who drink a lot of water, while it is increased in patients who use many laxatives and are dehydrated. In the first case, the water intake will have to be restricted, because, if the patient continued to drink a lot, she would risk reaching a condition of cerebral edema due to hyponatremia.
- *Thyroid:* a state of hypothyroidism is almost always present since the body tends to save energy by slowing down metabolic functions (first, it eliminates the menstrual cycle, useless for survival in a starved condition). TSH and FT4 tend to be normal, while FT3 is low, being the hormone most closely related to the activation of peripheral metabolism. Therefore, since the activities that waste energy are reduced or eliminated, the active transport systems through cell membranes will also be automatically reduced. Therefore, in the case of administration of drugs, even of a psychotropic type, poor efficacy or paradoxical effects related to reduced membrane activity could occur.

Course and prognosis

The course of AN is variable: in about 50% of cases there is complete recovery, in about 30% of cases a partial but satisfactory remission, and in about 20% of cases there is a chronic course, without recovery of a sufficient quality of life. In the case of serious and chronic patients (20%), the risk of death from malnutrition and suicide is estimated at around 10%. Several prognostic factors have been identified:

Negative prognostic factors:

- Delay in treatment
- Onset in older age
- Bulimic-purgative subtype
- Poor family support

Positive prognostic factors:

- Short duration of illness
- Good premorbid socio-working adaptation.

Differential diagnosis

In the differential diagnosis, it is necessary to consider all organic causes of starvation, such as neoplastic diseases, endocrine disorders (diabetes mellitus, hyperthyroidism, Addison's disease), and gastrointestinal disorders (peptic ulcer, ulcerative colitis). Anorexia must also be distinguished from weight losses associated with other psychiatric conditions, e.g., Depressive Disorders, Somatoform Disorders, Social Phobia, Obsessive-Compulsive Disorder, Panic Disorder, Body Dysmorphism Disorder.

8.4.5. Treatment

The anorexic patient rarely asks spontaneously to be treated. This refusal to treatment is part of an extreme desire for control over herself, over her own body, and is dictated by the ego-syntony of psychopathological symptoms.

The treatment involves a multidisciplinary approach: dietary, psychological, and medical/pharmacological. From a nutritional point of view, with a BMI <10, enteral or parenteral nutrition must be set, given the too high risk for the patient. In cases with less severe BMI, an oral nutritional rehabilitation is set up in hospital, with the setting of customized meals: in this regime, the patient cannot choose the food to eat but can define 3 foods to be completely excluded from the diet. The nutritionist can define, in line with the medical and psychological staff, a weight recovery goal considered "acceptable" by the patient from a psychological point of view, and sufficient by the carers from a metabolic point of view. For this reason, in the first stages of treatment, meals must not aim

at "fattening" but must offer a caloric intake slightly above the basal metabolic rate. Carbohydrate loads must be avoided, which otherwise could cause a sudden increase in the minimum amount of insulin present in the body, increasing the risk of refeeding syndrome. In countries where there is famine, UNICEF offers bars called PlumpyNut, composed of 50% milk powder, 25% white sugar, and 25% peanut butter: this suggests how, in conditions of emaciation, the intake of fibers (e.g., from vegetables, whole grains, or legumes) should be minimized to prevent bloating and reduced absorption of other nutrients, and refined carbohydrates, proteins and fats should be preferred.

From a medical point of view, in the presence of underweight subjects, the general patient conditions must always be assessed, namely vital parameters, ECG, blood sugar, electrolytes, and kidney function. The medical condition of a patient with an eating disorder is very precarious and could worsen at any time, so it is essential to make the basic clinical, instrumental, and laboratory tests.

Another issue is disease insight: it is often difficult to establish a good working alliance with patients with poor insight. In these patients, the sense of imminent death and the perception of danger are absent. Therefore, it makes no sense to motivate the subjects with phrases such as "if you don't eat, you'll die", but rather we need to focus on issues such as school efficiency, aesthetics, and, more generally, quality of life, which are more taken into consideration by subjects with anorexia nervosa.

A further problem is the patient's manipulation of the environment ("if you forbid me to do this, I won't eat"), which must be managed by providing specific support to the caregivers and the patient's family members. With a seemingly "peaceful" position, like that of fasting, the patient puts those around her in a condition of total powerlessness. In these cases, the clinical staff must try to make patients understand how this behavior is not a real challenge to others but is instead a symptom of the disease itself that must therefore be managed and dealt with by the patient. The working alliance is very important and, to be able to achieve it optimally, it is necessary to create a neutral environment, without the judgments and reinforcements usually present in the family context. In addition, it must be remembered that anorexia is a multifactorial disease, so it is incorrect to look for a single "responsible" in the family or society (although they may be some of the factors to be taken into consideration).

Finally, it is necessary to find common goals with the patient, such as:

- The improvement of attention and concentration.
- The reduction of the obsessive ideation about food and body image.
- The return to acceptable levels of global functioning, given that the patient often feels in a condition of absolute solitude.

Concerning psychopharmacological treatments, the role of Serotonin Reuptake Inhibitors (SSRIs) in some phases of treatment and for the prevention of relapses has been highlighted. Antipsychotic drugs

are used to exploit their sedative and weight gain effects related to their intake. The use of these drugs is also supported by the hypothesis that there is an accentuated dopaminergic tone in the anorexic pathology. In general, there is currently no etiological psychopharmacological therapy for eating disorders. However, drugs can be used for the management of psychopathological symptoms concomitant with anorexia (anxiety, depression, obsessive-compulsive symptoms, and poor insight into illness).

The "Marsipan Study"

The English MARSIPAN (Management of Really Sick Patients with Anorexia Nervosa) Study defines the clinical guidelines for managing a patient with anorexia nervosa of considerable severity when this comes to the attention of the non-specialist doctor (e.g., in the case of access to the emergency room or medical departments not specialized in EDs).

The study stems from the observation that the anorexic patient runs, in extreme opposites, two types of risks:

1) In institutions experienced in treating frail and underweight subjects (e.g., medical departments that follow cancer patients or elderly patients), the risk of dying from malnutrition is much greater than for other sick people, because the clinical unit, accustomed to treating critically ill patients suffering from various diseases, tends to refeed the anorexic patient excessively slowly, so slowly that there is a risk that the patient will die from emaciation.

2) On the contrary, the anorexic patient who goes to a psychiatric unit, a unit accustomed to treating physically younger and healthier patients, runs the exact opposite risk, namely that of dying based on an excess of nutrition.

From these two opposed criticalities, from the UK Royal College of Physicians and the Royal College of Psychiatrists arose the need to establish guidelines for the diagnostic orientation and treatment of a severely underweight person who is at the same time attempting to resist medical treatment. Since these are life-threatening patients, treatment must be insisted upon, and it is impossible to give up in the face of the patient's refusal of treatment.

The MARSIPAN guidelines, therefore, evaluate six key points:

1) *Risk Assessment:* evaluates the risk, or how much the patient is at risk of life, through clinical, instrumental, and laboratory investigations.

2) *Avoid Refeeding Syndrome:* provides all the recommendations aimed at minimizing the risk of the re-nourishment syndrome, more easily found in psychiatric units, less accustomed to managing organ criticalities that can also affect young patients suffering from a chronic eating disorder.

3) *Avoid Underfeeding Syndrome:* provides all the recommendations aimed at minimizing the risk of undernutrition, which is often reached in the fear of overloading the patient from a nutritional point of view.
4) *Manage Behavioral* Problems (e.g., Sabotaging Nutrition): addresses behavioral problems. These are subjects who often "sabotage" the treatment, not following medical instructions and, for example, disconnecting the drip or throwing away the food supplement provided to them.
5) *Treat Under Compulsion* (in Italy, Trattamento Sanitario Obbligatorio or TSO): evaluates the mandatory treatment.
6) *Manage Family Concerns:* evaluate family aspects. In eating disorders, more than in any other medical or psychiatric pathology, it is essential to provide care and information also to family members. Family members often add important anamnestic information, left out by patients due to their lack of awareness of the disease.

Risk assessment

In the risk assessment, it is important to assess the BMI, weighing the patient in each case, even when the assessment of weight is considered a stressful event. When the BMI is below 13, the subject is in critical condition and may be metabolically unstable, even if the blood tests reveal nothing of significance. Other elements of risk are:

- Heart rate <40 BPM.
- Body temperature <35 °; it must be considered that almost all anorexic patients are hypothermic.
- Muscle strength: assessable with the Sit-Up-Squat-Stand test (SUSS Test), which evaluates the ability to flex in bed and to do a squat. When the score obtained is <2, or the patient is unable to do these exercises, it can be considered as a risk factor.
- A QTc on ECG> 450ms. We must therefore be careful when administering certain drugs.
- Blood pressure variation: it is necessary to consider that anorexic patients have rather low blood pressure. On the other hand, pressure variation (e.g., orthostatic hypotension) is worrying and constituting a risk factor rather than hypotension as such.
- Low sodium (<130mmol / L): it should suggest an occult infection or an excess of water assumption.
- Low potassium (<3.0 mmol / L): it should make us think about purging conducts, such as the abuse of laxatives or induction of vomiting.
- Hypoglycemia (glucose <54 mg / dL): when associated with low albumin and/or high CRP, it should lead to suspicion of an occult pulmonary infection.

- Urea and creatinine: they must be particularly monitored as these patients, being undernourished, often show low plasma values of urea and creatinine and, for this reason, finding them normal can already indicate a deficit of renal function.
- Leukopenia or high transaminases: these are two findings that often alarm general practitioners, who hypothesize hepatic or hematological problems and prescribe further tests, with the risk of delaying proper re-nutrition. In reality, these findings are directly related to fasting and/or physical hyperactivity.

Refeeding Syndrome and Underfeeding Syndrome

Nutrition must be reintroduced gradually, and the gastric tube is useful in these cases because it makes it less easy for the patient to sabotage the "therapy". Calories are introduced very slowly (5-10 kcal/kg/day). The guidelines recommend checking even twice a day those blood chemistry indices that most easily suggest a refeeding syndrome. Refeeding syndrome arises when an organism accustomed to surviving in a condition of chronic food deficiency receives an excess of nourishment, especially sugars: this leads to cellular reorganization, following the arrival of insulin, which causes electrolytes such as magnesium and phosphorus to enter the cell. The electrolytes then enter the cell, causing hypomagnesemia, hypophosphatemia, and consequent risk of cardiac arrest. It is, therefore, necessary to check the plasma values of glucose, phosphorus, potassium, magnesium, calcium and increase nutrition, up to 40 kcal/kg/day, only if the electrolytes remain stable. If the wasting conditions are not extreme, a higher re-nourishment dosage, e.g., 20 kcal/kg/day, can be started, and calories can be increased more rapidly in the following days. As far as phosphorus is concerned, high-concentration supplements (e.g., potassium phosphate supplements) or foods, such as Parmigiano Reggiano or other aged cheeses, which contain high and readily bioavailable calcium and phosphorus, can be very useful. Since the intracellular migration of electrolytes is caused by the increase in insulin resulting from the intake of carbohydrates with a high glycemic load, it is necessary to implement a relatively low-sugar and high-fat re-nutrition to limit the problem. Finally, hyponatremia can be aggravated by hyperhydration, and very low sodium (<120 mMol/L) can put the patient at risk of cerebral edema and epileptic seizures. For this reason, in the first days of re-nutrition, it is often necessary to limit the patient's water supply.

Behavioral Problems, treatment under compulsion, family concerns

Regarding the tendency of patients to control the feeding, it is often necessary for a nurse to be specifically dedicated to a single patient, in order to minimize the risk of sabotage of re-nourishment. It is equally important to implement complete and transparent communication, both among the treating staff members and between staff and family members. As regards compulsory treatment,

differently, for example, from the UK, in Italy there are no specific rules for the treatment of the most severe anorexic patients against their will. The current Italian law on Compulsory Health Treatment (TSO) does not consider the specific needs of anorexic patients. It would be, therefore, desirable, for the future, to revise TSO legislation to allow a more effective and timely intervention in the case of patients suffering from severe anorexia and with little or no awareness of the disease.

8.5. Bulimia Nervosa

BOX 2: DSM-5 Diagnostic Criteria for Bulimia Nervosa

A. Recurrent episodes of binge eating. An episode of binge eating is characterized by both of the following:

- Eating, in a discrete period of time (e.g., within a two hour period), an amount of food that is definitely larger than what most people would eat during a similar period of time and under similar circumstances.

- Lack of control over eating during the episode (e.g., a feeling that you cannot stop eating, or control what or how much you are eating).

B. Recurrent inappropriate compensatory behavior to prevent weight gain, such as self-induced vomiting, misuse of laxatives, diuretics, or other medications, fasting, or excessive exercise.

C. The binge eating and inappropriate compensatory behaviors both occur, on average, at least once a week for three months.

D. Self-evaluation is unduly influenced by body shape and weight.

E. Binging or purging does not occur exclusively during episodes of behavior that would be common in those with anorexia nervosa.

SEVERITY

Mild: An average of 1–3 episodes of inappropriate compensatory behaviors per week.

Moderate: An average of 4–7 episodes of inappropriate compensatory behaviors per week.

Severe: An average of 8–13 episodes of inappropriate compensatory behaviors per week.

Extreme: An average of 14 or more episodes of inappropriate compensatory behaviors per week.

8.5.1. Definition

Compared to the classification of Bulimia Nervosa (BN) made in DSM IV, in DSM-5 there is:
- a reduction in the minimum required average frequency of binge eating and inappropriate compensatory behaviors (from 2 to 1 times per week for at least three months);
- the disappearance of subtypes (purging and non-purging).

BN can therefore be characterized by the presence of binge eating and purging behaviors.
In common language, the term binge is used to refer to an abundant and rich diet; in clinical practice, the term binge does not define only an excessive amount of food that the subject ingests but also refers to how the food is taken. The term binge means eating in a defined period (e.g., two hours) a significantly greater amount of food than most people in the same conditions would eat in that same time. The subject consumes the food quickly and voraciously, swallowing it without tasting it, and, sometimes, choosing foods that, under normal conditions, he/she does not even consider appetizing. During the episode, the subject has the feeling of losing control (e.g., feeling unable to stop eating or to control what or how much you eat). As for the eliminatory behaviors, these can occur through the use of laxatives, diuretics, enemas, self-induced vomiting, compensatory fasting, or motor hyperactivity.

8.5.2. Epidemiology

BN is an eating disorder that affects 1-3% of the general population, predominantly female subjects, with a male to female ratio of 1:10. Males represent only 10-15% of patients. Onset is around 18 and after 25 years of age with an age range of 12-35 years. The average duration of the disease at the time of referral is about 5 years. Some years pass before the patients come to the attention of a specialist because, unlike patients suffering from Anorexia Nervosa, whose physical impairment is more pronounced, patients suffering from bulimia nervosa are frequently normal-weighted.

8.5.3. Ethiopathogenesis

As for AN, the etiopathogenesis of BN is multifactorial, determined by the interaction between sociocultural, familial, and individual factors. However, in the pathogenesis of this disorder, some personal aspects acquire extreme relevance. Patients with BN are prone to experience frustrations as threats to their self-esteem, resulting in a feeling of discomfort towards their bodies. Compared to anorexic patients, bulimic patients are more impulsive, extroverted, and choleric. Narcissism, intended as the demand for attention from others, is considered among the risk factors for BN. As far

as family structure is concerned, we frequently find conflicts within the family, and bulimic patients often feel neglected and rejected, abandoned by parental figures.

8.5.4. Clinical presentation

The age of onset of BN is slightly higher than AN and the clinical presentation at onset is sometimes overlapping: in about 25% of cases, the diagnostic criteria for anorexia are met for the first period after onset, then binge eating episodes begin to interrupt dietary restriction, creating a cycle which tends to perpetuate over time. The onset of BN may occur after a strict restrictive diet for weight reduction or as a result of personal and emotional difficulties in managing situations of loss. In the early stages, the patient maintains absolute secrecy about her behavior, and, sometimes, years may pass before a family member notices the problem.

Binge episodes can be characterized by "objective binge eating" or "subjective binge eating", in which the feeling of loss of control is not associated with objectively high food intake. In both cases, however, the food ingested during the hyperphagic binge is high in sugars and fats, avoided during the dietary restriction, high in energy density, easy to ingest, and often lower in cost (Ministry of Health, 2017).

Binge eating is triggered by so-called "emotional hunger" linked to states of dysphoric mood, conflicts, stressful events, feelings of emptiness and loneliness, but also feelings of boredom and low tolerance to frustrations. It is also defined as "comfort eating " or "stress-induced eating". According to this theory, deficits in emotion regulation should translate directly into eating alterations. Overeating has been associated with unhealthy or extreme weight control conducts, dieting, non-suicidal self-harm, body dissatisfaction, low self-esteem, and depressive symptomatology.

The relationship between negative affect and binge eating in subjects with BN is well recognized, and current treatment strategies help normalize disordered eating conducts by promoting adaptive emotion regulation skills. Interestingly, studies have shown that dysfunctional eating behaviors in patients with BN not only include negative affect-induced overeating but also positive affect-induced underfeeding risk. This finding highlights the importance of maintaining a regular eating schedule as a primary goal, since individuals with BN may tend to underfeed when their mood improves during treatment. The idea that food may represent a tool used to regulate emotions (but those emotions can also regulate food intake) holds a prominent place among current theories on emotional eating and is the basis of several psychotherapeutic approaches to eating disorders.

Following the binge, most subjects, to compensate for feelings of guilt, shame, self-evaluation, inadequacy, and fear of gaining weight, use compensatory behaviors such as self-induced vomiting,

BOX 3: Clinical case

C. is a 27-year-old girl who at the first visit reports vague physical problems such as weakness and muscle cramps. She immediately admits that this is due to her "daily habits": 1-2 episodes of self-induced vomiting and heavy abuse of laxatives (an average of 40 tablets of senna derivatives every two days).

About five years before, after a "bad sentimental disappointment", C. discovers the immediate but temporary effect of a binge: she opens the fridge and quickly eats all the sweets she finds. After a momentary relief, she begins to experience bloating, weight in the stomach, and acid regurgitation; shortly after, she feels "empty inside" and guilty for what she did.

It is possible to observe typical aspects of bulimic disorder: the tendency to normalize the pathological behavior, the lack of awareness of the disease, the abuse of laxatives, the onset that goes back several years before the patient turns to the clinician, emotional and traumatic life events that trigger symptoms, the resort to impulsive actions to cope with the emotional tension arising from the trigger event.

In the following days, C. says to herself that she is well aware of the heavy discomfort resulting from the binge; nevertheless, after a phone call to a friend, with whom she remembers the disappointment experienced, she finds herself acting on impulse a new binge, this time more "chaotic": she first ingests sweets, then cold cuts, then sauces with bread, then more ice cream, then vegetables seasoned with lots of oil and vinegar... to finish two glasses of bitter "to digest".

In the space of two months, C. gained 15 kg (from 50 kg for 160 cm height and BMI: 19.5 to 65 kg AND BMI: 23.4).

At this point, she appears unable to control her binges and seeks alternative methods to "counterbalance" her food intake: she vomits immediately after a binge, takes laxatives every night and every few days fasts for a whole day. Moreover, she started drinking alcohol.

Her weight drops a little (from 65 to 60 kg) but she starts complaining about physical problems, such as acid reflux, muscle problems, increasing asthenia, caused by repeated vomiting.

Recourse to alcohol is not uncommon since it represents facilitation to emptying through emesis and on the other hand, it is a risk factor for further complications related to BN.

Among the compensatory behaviors, some patients choose complete abstention from food. However, this behavior only serves to reinforce the vicious circle because the subject, in a condition of fasting, becomes even more vulnerable to the urge to eat in an uncontrolled way (fasting -> uncontrolled eating -> guilt -> eliminatory behaviors -> fasting).

chewing it but before swallowing it, intake of drugs that control appetite . In a subgroup of people, self-injurious behaviors are present, sometimes aimed at pushing away the discomfort following the binge or punishing themselves for it. NSSIs (non-suicidal self-injurious behaviors) are often used as maladaptive coping strategies of emotional dysregulation. Such acts are seen more frequently in bulimic patients with greater body image distortion and body dissatisfaction. They are related to negative body-related feelings, reduced sensitivity to pain, and reduced ability to cope with distress.

In most patients the presence of compensatory behaviors has medical consequences, detailed in Table 2.

Physical complications and psychiatric comorbidities

- *Electrolyte imbalance:* One of the most dangerous complications of BN is the development of electrolyte imbalance, could result in cardiac arrhythmias. In particular, self-induced vomiting with the loss of gastric acid can lead to hypochloremic alkalosis and resultant hypokalemia. Laxative abuse may result in hypokalemia as well.
- *Subconjunctival hemorrhage and/or epistaxis:* They are caused by the rupture of small blood vessels induced by the increase in pressure that occurs during vomiting.
- *Dental complications:* Chronic exposure to stomach acid can cause dental damage (perimyolysis), in particular to the buccal and lingual surfaces. Other oral/dental consequences of self-induced vomiting include tooth discoloration, increased tooth sensitivity, oral mucositis, and cheilitis.
- *Larynx and vocal cords damage:* These organs may also be inflamed by acid exposure, leading to hoarse voice, chronic sore throat and cough, and difficulty swallowing.
- *Salivary glands:* Parotid and other salivary gland enlargement due to vomiting occurs in about 10-25% of patients.
- *Gastrointestinal tract disturbances:* Vomiting can lead to acid reflux, dyspepsia and dysphagia. Less frequently, it can cause small tears in the esophagus, and, rarely, esophageal rupture.

  Laxative abuse can instead lead to local effects such as chronic constipation or diarrhea, rectal prolapse, hemorrhoids, and hematochezia.

Medical consequences of Bulimia Nervosa
• ECG alterations (due to potassium depletion) • SALIVARY GLANDS HYPERTROPHY • HEROSION OF DENTAL ENAMEL AND DENTAL CARIES (dentists are sometimes the first to notice the presence of emesis) • FATIGUE AND WEAKNESS • DEHYDRATION, HYPOTENSION AND CAPOGIRIS • ABDOMINAL SWELLING and METEORISM, INTESTINAL MOTILITY ALTERATION (laxative abuse can lead eventually to PARALITHIC ILLEO) • ULCERS AND LACERATIONS OF THE ORAL AND PHARYNGEAL MUCOSA • GASTRIC CRAMPS, GASTRO-ESOPHAGEAL REFLUX, GASTRITIS • NUMBNESS AND TINGLING LIMBS • RUSSEL'S SIGN: calluses caused by the activity of gastric juices on the skin of the subject's hands

Table 2. Medical complications of Bulimia Nervosa

Finally, individuals with bulimia nervosa tend to be more prone to psychiatric complications. Comorbid psychiatric disorders can be detected at the time of diagnosis of BN or can develop later during the course of the disorder. The most common comorbidities are anxiety disorders (53%) and mood disorders (43%). Literature reports that approximately 90% of subjects with BN reported at least one episode of a mood disorder in their lifetime, usually a depressive one. The most frequent personality disorder associated with BN is Borderline personality disorder, particularly common in patients with history of childhood emotional trauma. At least 30% of patients with BN have consumed alcohol or stimulants in an attempt to control their appetite and weight. Besides, bulimia can increase the risk of suicide in patients.

In general, individuals with BN commonly have comorbid conditions that include acting-out behaviors, compared to the other subjects with eating disorders.

Course and prognosis

The course is usually chronic or remitting: often the disorder persists for several years, either continuously (chronicity occurs in 20% of subjects) or with alternating phases of remission and flare-ups. 5-10 years after the onset, 30-50% of patients have some eating disorder of clinical relevance, although, in many cases, it is an atypical form (forms of binge eating, eating disorder NOS, sometimes anorexia). Concerning severity and risk of hospitalization, we can say that BN is associated to a greater extent with self-harming behaviors and, as mentioned, with specific personality disorders, especially Borderline. Therefore, in general, these are subjects with high levels of impulsivity that can manifest through self-injurious and sometimes anti-conservative behaviors.

No unequivocal predictors of favorable prognosis have been identified; however, indicators of worse prognosis include childhood obesity, low self-esteem, and personality disorders.

Comorbidity with other disorders worsen the prognosis of these patients:

- Substance abuse that occurs in 55%

- Alcohol abuse 46%
- Agoraphobia 27-34%
- Social Phobia 15-55%
- Depression 31-90%
- Generalized Anxiety Disorder 23-70%

8.5.5. Treatment

The effect of pharmacological treatments in eating disorders is recognized as limited by the main international guidelines:

- APA (2006): drugs are not the first-choice treatment for BN; however, FDA recognizes the utility of fluoxetine's use for the treatment of bulimic disorders at dosages generally higher than those used for the treatment of depression (up to 60 mg). Similar utility, although with weaker evidence, is described for sertraline. However, the presence of purging behaviors sometimes does not guarantee the stabilization of plasma levels of pharmacotherapies. When concomitant anxiety or depressive symptoms are present, the use of pharmacotherapy helps to mitigate their effects.
- NICE (May 2017): medications are not the first choice in the treatment of EDs, but they may have a role as adjuvants to psychological therapies. It is recognized, however, that they can reduce the frequency of binge and purging in both BN and BED.

How then to manage the treatment of these patients?

1) First of all, it is necessary to implement a motivational intervention to the treatment, consisting of helping patients recognizing that they need specialist care and maintaining their motivation to get well over time. This goal is primary given the frequency of their reluctance to treatment.

2) Restoration of dietary regularity usually leads to a substantial improvement in the patient's general state.

3) The third aspect of the intervention consists of dealing with the poor evaluation that the patient has of his body shape and weight and helping her recognizing and managing dysfunctional eating habits.

4) Specialist treatment passes through the integration of different approaches (pharmacotherapy with antidepressants or antipsychotics, psychotherapy). Family therapy seems to be the most useful therapy for younger patients and is, therefore, the approach mainly used with adolescents.

An essential point in the psychiatric management of the patient with Bulimia Nervosa is a multidisciplinary approach, which aims to:

- offer and coordinate different types of care to the patient;
- involve different professional figures: psychologists, psychiatrists, dieticians, internists, dentists, and school staff in the management of the patient;

- promote team discussion and supervision.

This integrated intervention shows several advantages over a single intervention, such as the possibility of increasing the continuity, consistency, and effectiveness of the diagnostic process; the possibility of shortening the time of the subsequent therapeutic intervention; not to waste resources and energies of both therapists and patients; not to promote the maintenance of the state of disease.

Concerning the psychological interventions, the approach changes in the case of an adult or a minor patient.

In the case of subjects *more than 18 years old*, at first, self-help groups focused on bulimia can be proposed. This approach consists of providing material with cognitive-behavioral indications of self-help. This program can be supplemented with short support sessions (4 to 9 sessions of 20 minutes each for 16 weeks, starting at the beginning of the week).

If the treatment is ineffective after 4 weeks, or poorly accepted, or contraindicated, another possible approach is Cognitive Behavioral Therapy focused on bulimia (CBT-ED) (20 sessions in 20 weeks). In the first phase, CBT consists of motivational and educational interventions aimed at establishing a regular eating pattern, encouraging, counseling, and supporting the patient as he/she engages in this process. Then, CBT should focus on addressing the psychopathology of the eating disorder, such as extreme food restriction, concerns about body shape and weight, and the tendency to overeat in response to difficult thoughts and feelings.

Toward the end of treatment, meetings become less frequent and focus on reinforcing and maintaining positive change and reducing the risk of relapse. If therapists find it helpful, significant figures for the patient are involved for one-on-one support.

There are other possible psychotherapeutic approaches: interpersonal psychotherapy, dialectical behavior therapy, acceptance and commitment therapy, integrative cognitive-affective therapy, psychodynamic therapies, and family therapies.

Dialectical Behavior Therapy (DBT), is one of the most effective treatments in subjects with borderline personality disorder and has been extended, with appropriate modifications, to all disorders characterized by emotional dysregulation as a nuclear symptom, including eating disorders.

The term "dialectical" refers to the possibility of being in two different positions at the same time. For example, it is important for the patient to accept himself as he/she is but also to be motivated to change.

The treatment includes individual and group meetings, during which the patient learns skills to manage symptoms (preventing binge eating and refraining from eliminatory behaviors). It is a manualized treatment with versions available for both adolescent and adult clinical populations.

In the case of *minor patients*, the starting approach is represented by Family therapy, a family and patient support course that is developed in 18-20 sessions over 6 months.
It aims to establish a good therapeutic relationship with the patient, the family members, and caregivers and support and encourage the family to help the patient, maintaining a non-judgemental approach.
It also aims to: give information about body weight regulation, dieting, negative effects of self-induced vomiting, laxative abuse, or other compensatory behaviors; foster a collaborative approach between the patient and his/her parents to establish regular eating habits and minimize compensatory conducts. It includes regular one-on-one meetings with the patient during treatment, interventions to encourage self-control of bulimic behaviors, and discussion sessions with family members.
In the later stages of treatment, when the eating behavior has stabilized, the focus of care is directed toward supporting the person to establish a level of independence appropriate to the developmental stage of the subject. In the final phase of treatment, the focus shifts to dealing with any problems the patient and his/her family may have and relapse prevention.

8.6. Binge Eating Disorder

8.6.1. Definition

Binge-eating disorder (BED) was first described in 1959 by psychiatrist Albert Stunkard to describe the characteristics of a subgroup of subjects with obesity and recurrent episodes of excessive and uncontrolled eating: a behavior he called binge eating. However, its existence as a distinct diagnostic entity was ignored until the second half of the 1980s, when research on the prevalence of BN in the population highlighted a conspicuous subgroup of patients who did not adopt compensatory behaviors after binge eating episodes. At the same time, it was observed that about one-fourth of individuals seeking obesity treatment reported recurrent episodes of binge eating but did not meet the diagnostic criteria for bulimia nervosa. In 1994, the American Psychiatric Association included BED among eating disorders not otherwise specified and, in Appendix B of the Diagnostic and Statistical Manual of Mental Diseases (DSM-IV), provided a list of diagnostic criteria for further study. Subsequent studies confirmed that BED has distinctive features compared to bulimia nervosa and obesity, and supported the validity and clinical utility of the diagnosis of BED. However, it was not until 2013 that the disorder was recognized by DSM-5 as a distinct diagnostic category within nutrition and eating disorders.

Binge eating disorder (BED) is a psychopathological condition that affects 2.5% of adults and 1.6% of adolescents. This disorder is characterized by recurrent episodes of uncontrolled eating.

BOX 4: DSM-5 Diagnostic Criteria for Binge Eating Disorder

A. Recurrent episodes of binge eating. An episode of binge eating is characterized by both of the following:

- Eating, in a discrete period of time (e.g., within any 2-hour period), an amount of food that is definitely larger than most people would eat in a similar period of time under similar circumstances
- The sense of lack of control over eating during the episode (e.g., a feeling that one cannot stop eating or control what or how much one is eating)

B. Binge-eating episodes are associated with three (or more) of the following:

- Eating much more rapidly than normal
- Eating until feeling uncomfortably full
- Eating large amounts of food when not feeling physically hungry
- Eating alone because of being embarrassed by how much one is eating
- Feeling disgusted with oneself, depressed, or very guilty after overeating

C. Marked distress regarding binge eating is present.

D. The binge eating occurs, on average, at least 1 day a week for 3 months

E. The binge eating is not associated with the regular use of inappropriate compensatory behavior (e.g., purging, fasting, excessive exercise) and does not occur exclusively during the course of anorexia nervosa or bulimia nervosa.

SEVERITY

Mild: 1 to 3 episodes per week

Moderate: 4 to 7 episodes per week

Severe: 8 to 13 episodes per week

Extreme: 14 or more episodes per week

8.6.2. Epidemiology

BED has a prevalence of 0.7%-4.6% in the general population; 20-30% of obese patients, in general, have BED, while up to 70% of obese patients who come to hospitalization have BED. Symmetrically, 75% of BED patients are overweight or frankly obese. The male: female ratio is 1:1.5. The onset occurs between 30-40 years, regardless of diets or other specific reasons.

8.6.3. Etiopathogenesis

Binge-eating disorder is more common in female sex. Although people can develop a binge-eating disorder at any age, the onset often occurs in the late teens or early 20s. Factors that can increase the risk of developing a binge-eating disorder include:

- *Family history.* An individual is much more at risk of developing an eating disorder if parents or brothers and sisters have an eating disorder. Family history can indicate a genetic predisposition but it can also represent an environmental risk factor (parent modelling).
- *Dieting.* Many people with a binge eating disorder have a history of unsuccessful diets. Diet or limitation of calories intake during the day can cause a need for a binge to eat.
- *Psychological issues.* Many people suffering from a binge eating disorder feel negative about themselves and undervalue their capacities and achievements. Triggers for a binge eating episode can include stress, poor self-image, and the availability of favourite foods.

Factors that have been linked to the maintenance of the disorder are emotional, social, and cognitive dysfunctions. Binge-eating episodes are more likely to occur within in individuals with reduced emotional awareness, difficulties in emotion regulation, and frequent interpersonal problems. Moreover, binge eating is associated with neurocognitive dysfunction, including difficulties in inhibitory control and reward processing.

8.6.4. Clinical presentation

Binge eating disorder frequently co-occurs with obesity and metabolic syndrome. 40% of patients with binge eating disorder have a BMI > 30, and, of this percentage, 15% have a BMI > 40. More than 40% of patients with BED have metabolic syndrome.

Because of this comorbidity, individuals with BED are at increased risk for obesity-related medical consequences, such as type 2 diabetes mellitus, hypertension, and dyslipidemia, as well as premature mortality. Moreover, individuals with BED show a higher prevalence of other health conditions, including asthma, gastrointestinal symptoms, sleep disorders, musculoskeletal problems, neurologic problems, and gynecologic conditions.

However, BED is distinct from obesity for several reasons. Individuals with BED, compared to those with obesity without BED:

- Consume more calories in food intake studies and are generally more sedentary;
- More frequently report overestimation of weight and body shape, concern for their appearance and body weight, dissatisfaction with their physical appearance;
- Show greater cognitive deficits on neuropsychological tests;
- Have greater functional impairment and psychological distress, worse quality of life, and a higher prevalence of mental health disorders.

Indeed, BED is frequently associated with psychiatric comorbidities. 50% of BED patients have at least one depressive episode in their lifetime and, in general, increased recurrence of dysthymia, Panic and other anxiety disorders, Borderline Personality Disorder, substance and alcohol abuse.

Course and prognosis

The average age of onset of BED is about 21 years. However, BED has a wide distribution of age of onset, from 14 to 30 years. In typical cases, BED begins with episodes of binge eating, often associated with stressful events. Episodes of binge eating determine, in most cases, an increase in weight, which leads some individuals to undergo diets to try to lose weight, but generally without obtaining lasting results. This process is the opposite of what happens in bulimia nervosa, where usually the diet precedes the appearance of episodes of binge eating. Sometimes, however, BED can also begin after a period of strict dieting. The disorder generally has a chronic course. In most cases, individuals alternate prolonged periods characterized by recurrent episodes of binge eating with others characterized by a good control over eating. Although little information is yet available on the course of BED, it appears that migration of the disorder to bulimia nervosa and anorexia nervosa or other eating disorders is rare. In contrast, a longitudinal study of adolescent females observed that BED increases the risk of developing overweight, obesity, and depression by approximately twofold. Finally, it has been estimated that the average persistence of BED is approximately 16 years. Overall, forty to fifty percent of patients experience an improvement, but with an 80% chance of relapse. Integrated treatment, consisting of pharmacological treatment with SSRIs, psychological and nutritional intervention, is associated with a better prognosis.

8.6.5. Treatment

As in other eating disorders, to ensure that the patient has a good chance of achieving an effective and lasting recovery, it is essential to provide a multidisciplinary approach. This should be based on the coordinated involvement of internists (to manage the organic disorders and define a dietary plan appropriate to the weight loss required) and psychiatrists (to correct the mental and behavioral patterns typical of the disorder).

The treatment starts from "assumptions" different from those for the treatment of bulimia nervosa.

The patient must work on:

- Motivation to reduce weight and not binge;
- Willingness to carry out diets without eliminatory behaviors, although a dietary restriction is a rare event in these patients;
- Concerns about overweight.

The *psychotherapeutic approach* that seems to give the best long-term results is cognitive-behavioral therapy (CBT), aimed at redefining the relationship with food and providing the patient with the tools to react favorably to negative stimuli that can be commonly encountered in everyday life and represent the main trigger for binge eating.

NICE guidelines (2017) have recommended CBT as the treatment of choice for this disorder. In particular, CBT-based Guided Self Help (GSH) should be considered as the intervention of the first choice. It consists of working through a book about binge eating, and having short sessions with a therapist (between 4 and 9 sessions, each lasting 20 minutes) to monitor developments. If this treatment is not accepted, is contraindicated, or ineffective, group CBT-ED (behavioral therapy for eating disorders) may be considered. If group CBT-ED is not available or the person refuses it, individual CBT-ED should be considered. The same indications are provided for binge-eating disorder in children and young adults as for adults.

Some research supports other specialized psychological treatments for BED, most notably interpersonal psychotherapy (IPT). In general, psychological treatments result in remission of binge eating episodes in approximately 50-55% of individuals affected and improvement in associated eating disorder and depressive symptoms. These beneficial effects are maintained at two and four years. Unfortunately, the main disadvantage of these approaches is that they generally do not produce significant weight loss.

Depending on the severity of the disorder, in the first phase, it may be necessary to provide a hospitalization of a few weeks or a period of Day hospital, followed by periodic psychotherapeutic sessions for several months.

A model of treatment aimed at producing a permanent weight loss is represented by the following, which provides two types of intervention that can also be complementary:

1) Hospitalization: a hospital stay of 4 weeks aimed at behavioral intervention with total reliance on the multidisciplinary team of nutrition management;

2) High-intensity outpatient pathway (2 to 3 times per week): this is also a multidisciplinary intervention, including the implementation of a physical activity program.

Medications should never be indicated as the sole treatment for BED; however, in some patients, association with drug therapy is imperative. Indeed, regardless of the presence of a concomitant depressive disorder, CBT can be associated with pharmacological treatment with antidepressants, which has been shown to enhance the effectiveness of the psychotherapeutic intervention. Numerous medications have been tested in the treatment of BED, including antidepressants (SSRIs, serotonin, and norepinephrine reuptake inhibitors, and bupropion), anticonvulsants (topiramate), weight-loss agents (sibutramine), and agents for the treatment of substance use disorders (naltrexone). Although some antidepressants may reduce the frequency of binge episodes, they are sometimes associated with weight gain. Topiramate, which has been shown to reduce both frequencies of binge episodes and weight, is no more recommended because it may worsen cognitive dysfunction.

In case the above-mentioned interventions are not sufficient, *bariatric surgery* can be considered if severe obesity needs to be addressed due to metabolic risks. The surgical approach should always, however, be integrated with psychological and nutritional support.

Types of Bariatric Surgery currently foreseen for BED:

- Gastric bypass (Roux-en-Y)
- Gastric banding
- Biliopancreatic diversion
- Sleeve gastrectomy

13.5% of patients who undergo bariatric surgery are BED. However, approximately 20% of patients who undergo these procedures do not benefit from them. Within 2 years, a high proportion of BED patients who have undergone surgery relapse, with at least 25% experiencing binge eating and 12% vomiting for reasons related to shape and weight.

Risks associated with the surgical procedure may include excessive bleeding, infection, adverse reactions to anesthesia, deep vein thrombosis, pulmonary or respiratory problems, leakage into the gastrointestinal system (i.e., fistulas), and death (rare). However, malabsorption syndromes are a frequent complication, resulting in nutritional deficiencies with consequent anemia (iron, vitamin B12, folate deficiencies), neurological disorders such as Wernicke's Encephalopathy (B12 deficiency), Beri-beri (B1 deficiency) alterations of vision (vitamin A and vitamin E deficiencies), skin alterations (vitamin A deficiency), osteoporosis, and secondary hyperparathyroidism. Finally,

the surgical intervention can be followed by the so-called "dumping syndrome" (also called "accelerated gastric emptying syndrome"). An early form and a late form of this disorder may occur. The early dumping syndrome is caused by an accelerated emptying of the stomach, that is a too-rapid passage of food from the stomach to the intestine through the communication created by the surgery, which, thanks to the gastric resection, reduces the volume of the stomach and alters to a greater or lesser extent the normal digestive processes. The passage in the intestine of food not yet fully processed by the gastric digestive phase determines the release of vasodilator substances and stimulators on nerve endings, which cause the classic symptoms of the syndrome (abdominal cramps, nausea, and diarrhea).

The late dumping syndrome is instead caused by hypoglycemia that follows the production of an excessive amount of insulin.

Finally, often as a complication of surgery, we can observe the development of a secondary eating disorder, for example, the appearance of BN.

8.7. Other eating disorders

Other specified feeding or eating disorder

The Other Specified Feeding or Eating Disorder (OSFED) corresponds to the diagnostic category of the DSM-5 that has replaced the Eating Disorder Not Otherwise Specified (EDNOS) present in the DSM-IV. This category includes 5 types of disorders: atypical anorexia nervosa, atypical bulimia nervosa of low frequency and/or limited duration, binge eating disorder of low frequency and/or limited duration, purging disorder, and night eating syndrome (NES). These disorders are quite common and account for 32% to 53% of all individuals with eating disorders.

In the DSM-5, these individuals present an eating disorder that has similarities with AN or BN and causes clinically significant impairment, but the diagnostic criteria listed for those disorders are not fully met.

For example, atypical disorders are those in which the patient's weight is just above the diagnostic threshold for AN, or binge eating and purging are less frequent than required for the diagnosis of BN. Although the diagnostic criteria are not met, these forms can also be very severe and incapacitating, and the treatment and prognosis can be virtually superimposable on those for AN and BN.

The following disorders are included among OSFED:

- *Atypical Anorexia Nervosa*: All criteria for AN are met, with the exception of significant weight loss, since the patient's weight is within or above the normal range.
- *Binge Eating Disorder (of low frequency and/or limited duration):* All of the criteria for BED are met, but at a lower frequency and/or for less than three months.
- *Bulimia Nervosa (of low frequency and/or limited duration):* All of the criteria for BN are met, but the binge eating and inappropriate compensatory behavior occurs at a lower frequency and/or for less than three months.
- *Purging Disorder:* It is characterized by recurrent purging behaviors that influence weight or shape (e.g., self-induced vomiting, abuse of laxatives, diuretics, or other medications) in the absence of binge eating.
- *Night Eating Syndrome (NES):* It is characterized by recurrent episodes of nocturnal food consumption (eating after waking from sleep or excessive food consumption after the evening meal). The patient has a conscious memory of eating. Nocturnal food consumption is not explained by external influences with changes in the sleep-wake cycle or local social norms and causes significant difficulties and/or problems with functioning. The prevalence of NES

in the general population is 1.5%, while in obese subjects is 6-16%. NES is characterized by the following symptomatology:

- Evening hyperphagia i.e., consumption of at least 25% of the daily intake after the evening meal and\or >2 nocturnal food intakes (defined as waking up at night to eat) per week.
- At least 3 of the following 5 characteristics:

  1) "Morning anorexia" (defined as no appetite in the morning).

  2) A strong urge to eat between dinner and the onset of sleep or during nighttime awakenings

  3) Insomnia at least 4-5 times a week

  4) The belief that eating is necessary to initiate or resume sleep

  5) Depressed mood that worsens during nighttime hours.
- Awareness and ability to remember the nightly or evening food intake. This criterion is necessary to differentiate NES from Sleep-Related Eating Disorder (SRED), a disorder in which the nightly food intake occurs without awareness and is not remembered by the subject.

Several studies show that the serotonergic system, involved in the regulation of appetite and circadian rhythms, has a key role in the pathophysiology of NES. Antidepressant treatments with sertraline and psychological therapies can be used for the optimal administration of patients with NES. The pharmacological treatment with sertraline shows a significant reduction in the number of awakenings every week, night intake of the week, and percentage of calorie consumption after the evening meal. Other treatment options such as melatonergic medications, light therapy, and topiramate represent promising treatment options.

PICA

It is an eating disorder characterized by the consumption of non-nutritious substances that are not typically thought of as food, such as hair, dirt, and paint chips.

It is characterized by the following diagnostic criteria:

- The consumption is continued for at least one month.
- The consumption of non-nutritious substances is inappropriate for the individual's developmental level. In children under the age of two, putting objects in their mouth is a normal part of development that allows the child to explore the senses. Mouthing can sometimes lead to swallowing. Children under two years of age should not be diagnosed with pica.

- The eating behavior is not part of a culturally or socially supported practice.
- If the eating behavior occurs in the context of another mental disorder (e.g., cognitive disability, autism spectrum disorder) or medical condition (e.g., pregnancy), it is severe enough to require additional clinical attention to avoid conditions of intoxication, poisoning, or bezoar formation.

The prevalence of PICA is difficult to establish due to differences in the definition. A high incidence of PICA (more than 50%) is reported in patients with intellectual disabilities (about 10%).

Mood disorders, anxiety disorders, and obesity are important predictors of PICA in adults. In children, Autism Spectrum (ASD) is the most significant PICA predictor.

Iron deficiency, anemia, and malnutrition are two of the most common causes of PICA, and some women can develop PICA during pregnancy because of the nutrient deficiency. In these cases, the disorder is a response of the body to a significant nutrient deficiency. The first-line treatment for PICA is thus to test for and correct vitamin or nutrient deficiencies. In many cases, PICA disappears once the deficiencies are corrected. If the disorder is not caused by malnutrition, several behavioral interventions are available.

Rumination disturbance

It involves the repeated regurgitation of food, which may be re-chewed, re-swallowed, or spit out.

It is characterized by the following diagnostic criteria:

- The repeated regurgitation of food over a period of at least one month
- The repeated regurgitation is not attributable to gastrointestinal or other associated medical conditions (e.g., gastrointestinal reflux).
- The disorder does not occur exclusively in the course of anorexia nervosa, bulimia nervosa, uncontrolled eating disorder, or avoidant/restrictive eating disorder.
- If symptoms occur in the context of another mental disorder (e.g., intellectual disability), they are severe enough to warrant additional clinical attention.

Some of the oldest observations of rumination disturbance suggested that the syndrome occurs primarily in children and adults with developmental delays but later studies have demonstrated that most patients with rumination are of normal-level intellect. Due to the lack of awareness of the many health care professionals, it is conceivable that the true occurrence of the rumination syndrome is underestimated.

Avoidant/restrictive food intake disorder (ARFID)

ARFID involves reductions in the amount and/or types of food consumed, but unlike anorexia, it does not involve any distress about body shape or size, or fears of fatness.

It is characterized by the following diagnostic criteria:

- A food and nutrition abnormality (e.g., lack of interest in food or eating; avoidance based on sensory characteristics of food) manifested by a persistent inability to take in adequate nutritional and/or energy intake associated with one or more of the following:
    - Significant weight loss or in children inability to achieve growth-related weight
    - Significant nutritional deficiency
    - Dependence on enteral nutrition or oral nutritional supplements
    - Marked interference with psychosocial functioning
- The disorder is not related to food deficiency or associated with cultural practices.
- The disorder does not occur exclusively in the course of anorexia or bulimia nervosa and there is no evidence of abnormality in the way one's weight and body shape are perceived.
- The abnormality is not best attributable to a medical condition or other mental disorder. If the eating disorder occurs in the course of another disorder, its significance exceeds that of the underlying disorder and requires specific clinical attention.

From the epidemiological point of view, the average age of onset is about 12 years, and in 30% of cases it affects male subjects. It is a clinical entity not to be underestimated since about 15% of patients who require treatment in centers for eating disorders meet the criteria of ARFID.
Children with autism spectrum conditions, ADHD, intellectual disabilities and anxiety disorders are much more likely to develop ARFID. Moreover, children who don’t outgrow normal picky eating appear to be at higher risk to develop ARFID.

Orthorexia

Orthorexia Nervosa (ON) is a disorder of very recent origin, which has developed simultaneously to the birth and spread of health philosophies of life (for example, vegetarianism, veganism, and organic food), and the increasing attention of our society towards healthy eating. This condition has always been difficult to define in clinical terms; in fact, it has not yet been clinically recognized as one of the disorders of nutrition and eating, as validated diagnostic criteria for ON have not yet been established. The term Orthorexia Nervosa was introduced by Steven Bratman, in 1997, to indicate a pathological preoccupation on healthy food consumption.
Nor DSM-IV and the DSM-5 recognize the ON as a proper disorder independent of others: it is placed together with "inverse anorexia" within the area of the avoidant/restrictive disorder of food intake. The obsession with "healthy eating" typical of orthorexia negatively affects the individual's relational, emotional, and physical spheres of life. According to the literature, the onset of ON is characterized by a desire to eat better in order to have a better physical shape. The pathological core of orthorexia

lies in a series of distorted beliefs about what is healthy and a sense of personal superiority resulting from dietary restrictions.

Many subjects who develop AN or BN exhibit orthorexic behaviors at early stages of their clinical history.

Orthorexia Nervosa is characterized by:

1) Devoting more than three hours a day to preparing and searching for food;

2) Feeling a sense of superiority over those with other eating habits;

3) Following a rigid regimen that is qualitatively controlled and putting in place compensatory restrictions in case of transgressions;

4) Associate one's self-esteem to the adherence to the diet;

5) Putting healthy nutrition at the center of one's life;

6) Overlooking values, relationships, previous interests and activities, and even physical health to achieve the goals described.

According to data released by the Italian Ministry of Health, orthorexics would be 300,000 in Italy (11.3% males vs 3.9% females).

For the assessment of orthorexic behaviors, it is possible to use the Bratman Test, a popular self-report questionnaire. If the answer is affirmative to at least 4-5 questions above, the subject is considered at risk. If the answer is affirmative to all questions, the attention to "healthy" food could configure a real obsession and increase the risk for the development of a proper eating disorder.

BRATTMAN TEST
1. Do you spend more than 3 hours a day thinking about your diet?
2. Do you plan your meals several days ahead?
3. Is the nutritional value of your meal more important than the pleasure of eating it?
4. Has the quality of your life decreased as the quality of your diet has increased?
5. Have you become stricter with yourself lately?
6. Does your self-esteem get a boost from eating healthily?
7. Have you given up foods you used to enjoy in order to eat the ‘right’ foods?
8. Does your diet make it difficult for you to eat out, distancing you from family and friends?
9. Do you feel guilty when you stray from your diet?
10. Do you feel at peace with yourself and in total control when you eat healthily?

Tabella 2. Brattman Test

Finally, abnormal eating behaviors can be present as a "symptom" of other psychiatric disorders:

- *Mood disorders:* the depressed subject often shows appetite and weight loss, while a subject with a manic episode can sometimes appear so busy that he does not feel the sense of hunger. On the contrary, in some cases, hyperphagia with weight gain can occur.
- *Schizophrenia:* altered eating behaviors can be found also in psychotic disorders, both related to the psychopathological symptoms and as a side effect of antipsychotic treatment. For example, a patient with a delusion of poisoning can refuse to eat, while a subject treated with antipsychotics can experience an increased appetite.
- *Anxiety disorders:* sometimes, a panic attack can occur while the subject is feeding, leading to the appearance of an intense fear of choking while eating.
- *Somatoform disorders:* in this case, the patient may fear that certain foods may lead to gastralgia or other gastrointestinal disorders.
- *Substance abuse:* the continuous intake of alcohol or drugs can be associated with a reduction in food intake, resulting in malnutrition.
- *Mental organic disorders:* the behavioral disorganization of the subject may lead to the inability to provide for their nutrition correctly.
- *Personality disorders:* Borderline personality disorder is often characterized by abnormalities of eating behavior (binge eating or food restriction), which usually have a short course in time and are the expression of the emotional dysregulation of the subject.

Suggested readings

1. Attia E, Becker AE, Bryant-Waugh R, Hoek HW, Kreipe RE, Marcus MD, Mitchell JE, Striegel RH, Walsh BT, Wilson GT, Wolfe BE, Wonderlich S. Feeding and eating disorders in DSM-5. Am J Psychiatry. 2013 Nov;170(11):1237-9. doi: 10.1176/appi.ajp.2013.13030326. PMID: 24185238.

2. Castillo M, Weiselberg E. Bulimia Nervosa/Purging Disorder. Curr Probl Pediatr Adolesc Health Care. 2017 Apr;47(4):85-94. doi: 10.1016/j.cppeds.2017.02.004. PMID: 28532966.

3. Fairburn CG, Cooper Z, Shafran R. Cognitive behaviour therapy for eating disorders: a "transdiagnostic" theory and treatment. Behav Res Ther. 2003 May;41(5):509-28. doi: 10.1016/s0005-7967(02)00088-8. PMID: 12711261.

4. Favaro A, Tenconi E, Santonastaso P. Perinatal factors and the risk of developing anorexia nervosa and bulimia nervosa. Arch Gen Psychiatry. 2006 Jan;63(1):82-8. doi: 10.1001/archpsyc.63.1.82. PMID: 16389201.

5. Gibson EL. The psychobiology of comfort eating: implications for neuropharmacological interventions. Behav Pharmacol. 2012 Sep;23(5-6):442-60. doi: 10.1097/FBP.0b013e328357bd4e. PMID: 22854304.

6. Godier LR, Park RJ. Compulsivity in anorexia nervosa: a transdiagnostic concept. Front Psychol. 2014 Jul 17;5:778. doi: 10.3389/fpsyg.2014.00778. PMID: 25101036; PMCID: PMC4101893.

7. Hilbert A, Pike KM, Goldschmidt AB, Wilfley DE, Fairburn CG, Dohm FA, Walsh BT, Striegel Weissman R. Risk factors across the eating disorders. Psychiatry Res. 2014 Dec 15;220(1-2):500-6. doi: 10.1016/j.psychres.2014.05.054. Epub 2014 Jun 6. PMID: 25103674; PMCID: PMC4785871.

8. Klump KL, Suisman JL, Burt SA, McGue M, Iacono WG. Genetic and environmental influences on disordered eating: An adoption study. J Abnorm Psychol. 2009 Nov;118(4):797-805. doi: 10.1037/a0017204. PMID: 19899849; PMCID: PMC2805262.

9. Lampard AM, Byrne SM, McLean N, Fursland A. Avoidance of affect in the eating disorders. Eat Behav. 2011 Jan;12(1):90-3. doi: 10.1016/j.eatbeh.2010.11.004. Epub 2010 Nov 10. PMID: 21184983.

10. Le Grange, D. Lock, J. Eating Bulimia in Adolescents: A Family Based Approach. New York: The Guilford Press; 2007.

11. Macht M. How emotions affect eating: a five-way model. Appetite. 2008 Jan;50(1):1-11. doi: 10.1016/j.appet.2007.07.002. Epub 2007 Jul 25. PMID: 17707947.

12. Meule A, Richard A, Schnepper R, Reichenberger J, Georgii C, Naab S, Voderholzer U, Blechert J. Emotion regulation and emotional eating in anorexia nervosa and bulimia nervosa. Eat Disord. 2019 Jul 25:1-17. doi: 10.1080/10640266.2019.1642036. Epub ahead of print. PMID: 31345125.

13. Pérez S, Marco JH, Cañabate M. Non-suicidal self-injury in patients with eating disorders: prevalence, forms, functions, and body image correlates. Compr Psychiatry. 2018 Jul;84:32-38. doi: 10.1016/j.comppsych.2018.04.003. Epub 2018 Apr 12. PMID: 29679850.

14. Racine SE, Horvath SA. Emotion dysregulation across the spectrum of pathological eating: Comparisons among women with binge eating, overeating, and loss of control eating. Eat Disord. 2018 Jan-Feb;26(1):13-25. doi: 10.1080/10640266.2018.1418381. PMID: 29384463.

15. Safer DL, Telch CF, Agras WS. Dialectical behavior therapy for bulimia nervosa. Am J Psychiatry. 2001 Apr;158(4):632-4. doi: 10.1176/appi.ajp.158.4.632. PMID: 11282700.

16. Stice E, Gau JM, Rohde P, Shaw H. Risk factors that predict future onset of each DSM-5 eating disorder: Predictive specificity in high-risk adolescent females. J Abnorm Psychol. 2017 Jan;126(1):38-51. doi: 10.1037/abn0000219. Epub 2016 Oct 6. PMID: 27709979; PMCID: PMC5215960.

17. Tozzi F, Thornton LM, Klump KL, Fichter MM, Halmi KA, Kaplan AS, Strober M, Woodside DB, Crow S, Mitchell J, Rotondo A, Mauri M, Cassano G, Keel P, Plotnicov KH, Pollice C, Lilenfeld LR, Berrettini WH, Bulik CM, Kaye WH. Symptom fluctuation in eating disorders: correlates of diagnostic crossover. Am J Psychiatry. 2005 Apr;162(4):732-40. doi: 10.1176/appi.ajp.162.4.732. PMID: 15800146.

18. Tremblay L, Lariviere M. The influence of puberty onset, body mass index, and pressure to be thin on disordered eating behaviors in children and adolescents. Eat Behav. 2009 Apr;10(2):75-83. doi: 10.1016/j.eatbeh.2008.12.001. Epub 2008 Dec 14. PMID: 19447348.

19. Udo T, Grilo CM. Prevalence and Correlates of DSM-5-Defined Eating Disorders in a Nationally Representative Sample of U.S. Adults. Biol Psychiatry. 2018 Sep 1;84(5):345-354.

doi: 10.1016/j.biopsych.2018.03.014. Epub 2018 Apr 17. PMID: 29859631; PMCID: PMC6097933.