December 30, 2020

PharmacologyOnLine

Archives • 2020 • vol.3 • 109-125

# Other Specific Feeding or Eating Disorders

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**Abstract**

Other Specific Feeding or Eating Disorders (OSFED) would be more appropriately named as "OTHERS SPECIFIC NUTRITION OR NUTRITIONAL DISORDERS " to indicate how some of the disorders that are grouped together represent pathologies in themselves with their own characteristics, albeit still little demonstrated. Many of the studies relating to these diagnostic categories date back to the years prior to the promulgation of the American Psychiatric Association (APA) criteria in 2013 and contained in the fifth edition of the Statistical Diagnostic Manual of Mental Diseases.
One of the main objectives of the revision of the criteria for the diagnosis of Eating Disorders (ED) previously in use by the APA, has been to reduce the use of the category Residual Eating Disorders Not Otherwise Specified (ED-NAS; Eating Disorders Not Otherwise Specified, EDNOS) to improve diagnoses and therefore specific treatments. In the DSM-5, DA-NAS have been replaced by two residual categories: OSFED and Unspecified Feeding or Eating Disorders (UFED, nutrition and nutrition disorder without specification) diagnose when choosing not to specify why the criteria for a Feeding and Eating Disorder) are not satisfied or there is not enough information to define a particular one pathology.
In this article we will discuss the possible causes and what are the treatments to solve the problem. Also, We will briefly describe the symptomatic peculiarities (from the point of view psychiatric, food and internistic) of the OSFED.

**Keywords:** *ARFID-Avoidant, Bigorexia, Drunkorexia, Other Specific Feeding, NES, Orthorexia, Unspecified Feeding*

## Introduction

The definition of OSFED applies when the specific symptoms for a FED predominate and they significantly compromise the different areas of the individual's life, but do not satisfy fully the criteria for a specific disorder and the clinician chooses to communicate why not the diagnostic criteria for any of the different FEDs are satisfied by following the wording Nutrition or nutrition disorder with other specification the specific reason (e.g. bulimia low frequency nervous).

DSM-5 (1) provides some specification examples such as:

1. A*typical anorexia nervosa* (ANa), which applies when all the diagnostic criteria for are met Anorexia Nervosa, but despite an important weight decrease, the subject still has a weight in the range or above the normal range.
2. *Bulimia nervosa* (low frequency and/or limited duration) (BNs), diagnosable when the frequency of binge eating and compensation behavior is less than that required and/or the duration is less than three months, however the other criteria for Bulimia Nervosa are met.
3. *Binge-eating disorder* (low frequency and/or limited duration) (BEDs), which refers to those cases in which the frequency of binges and/or their duration are less than what is required in the criteria for this ailment, while everyone else is satisfied.
4. Purging Disorder (PD) *disorder* , applicable to people who manifest elimination behaviors (self-induced vomiting, abuse of diuretics and/ or laxatives, enteroclysms) in the absence of binge eating.
5. *Nighttime feeding syndrome* (NES), which is diagnosed when the individual takes it significant quantities of food during the night or after the evening meal with awareness and memory of what happened (a specific part of this chapter will be dedicated to this pathology).

## Epidemiology

OSFED/UFED are the most common ED, they can suffer from males and females with a 1:4 ratio (2) and the typical age of onset is adolescence or early adulthood (3).

In a recent longitudinal study, carried out on almost 3000 Finnish twins (4) followed from 16 years up at 22-27 years old, only 292 tested positive for ED and of these only 71 have received a diagnosis of EDNOS. With the change of DSM-5 diagnostic criteria, the diagnoses residuals decreased by 62% and, while the lifetime prevalence of EDNOS would have been 3.9%, also including the BED and 3.3% without the BED, that of the OSFED / UFED dropped to 1.5%. This estimated data is in line with that found in other population samples in the Netherlands (0.8%) (5) and in Portugal (2.0%) (6). Mustelin et al. (2016) weighed the rate they estimated, not having been able to interview all the subjects of the sample used, obtaining a percentage of 4.2%, however very far from that obtained in US (11.5%) (7) and Australian (9.7%, of which 5% OSFED and 4.7% UFED) (8-9). In an Italian study (10) the DSM-5 criteria were applied to 206 patients, aged between 15 and 56, previously diagnosed according to DSM-IV, experiencing a 17% reduction in EDNOS and a 30.8% redefinition of EDNOS as OSFED. In a Californian study (11) it was analyzed retrospectively, with the diagnostic criteria of DSM-5 and DSM-IV, a clinical sample of males and the reduction of diagnoses of OSFED (45.5%) compared to those of EDNOS (62.6%). Applying to 1654 subjects of both sexes in school age (average of 13.4 years) the DSM-5 criteria through a structured interview, Hammerle e colleagues (2016) found that AN-atypical was present in 3.6%, the NL and the BED sub-threshold they were not diagnosed, while the Purging Disorder was present in 1.9%. The same authors (Hammerle et al. 2016), using a hierarchical approach, also measured the percentages partial syndromes (which meet only the main criteria of the disorder, but not all

those expected: 10.9% AN; 0.2% BN; 2.1% BED) and those defined below the threshold for reasons other than low duration or the low frequency (0.8% AN; 0.3% BN; 0.2% BED) and have discordant data.

Finally, an Australian retrospective study, noted the 23.5% reduction in OSFED / UFED diagnoses compared to those of EDNOS (12). A review on the Purging Disorder (13) identified one lifetime prevalence of this disorder in young women between 1.1 and 5.3%, confirmed figure recently from a population study on twins (3.77%) (14). The large gap between rates observed prevalence is due to survey methods and samples that are difficult to compare, as well as to lack of clarity in the distinction between OSFED and UFED.

In the Finnish study already cited (4) the incidence per 100000 inhabitants/year, calculated in the range of age at risk (between 10 and 24 years), it was 270 for EDNOS inclusive of BED (230 without BED) and 92 for OSFED/ UFED (38 OSFED and 64 UFED). Of the residual category only 37% were represented by patients who were diagnosed as OSFED and in particular: 13% Anorexia Atypical Nervous; 10% Bulimia nervosa (low frequency and / or of limited duration); 11% Noise from binge-eating (low frequency and / or limited duration); 3% Elimination conduct disorder. The 63% was instead in the UFED because it did not have the specifications required by DSM-5 to be recognized among OSFEDs (eg AN or BN without disturbing the body image).

According to Mustelin et al. (2016) the average age of onset of OSFED / UFED is 18 years (range from 18 to 25 years) (4), data confirmed by Stice et al. (2013) which found the peak incidence between 18 and 20 years (7). An interesting observation that the researchers made was that neither the average age of onset (18 vs 19 years, p=0.09), neither the average disease duration (3 vs 2 years, p = 0.57) nor the percentage of subjects healed at the time of the interview (57% vs 63%, p = 0.74) differentiated in significantly the OSFED from the UFED. Hay et al. have carried out a population study on a representative sample of Australian adolescents and adults finding that the OSFED / UFED they had an average age of around 40, an income and an average level of education, a distribution between the sexes less dependent on females than the other EDs. Furthermore, the diagnosis of BNs was higher than in the general population or in others OSFED and UFED (15).

The studies carried out so far are highlighting how the goal pursued in the DSM-5 to reduce the residual categories and making them more useful does not seem to have been fully achieved since OSFEDs represent only 1/3 of residual diagnoses and UFEDs 2/3, this too because it is not clear whether all the sub-threshold forms of FED can be included in the OSFED or only those specifically described in the DSM-5. Some authors (4) therefore criticize the opportunity of have divided the EDNOS into two categories, increasing complexity rather than reducing the confusion.

**Clinical manifestations and comorbidities**

The diagnosable patients in the OSFED category mimic the clinical characteristics of the disorders of which only have some criteria or present them at a lower level (by frequency or duration), consequently also the clinical manifestations and complications are superimposable.

People with an OSFED usually show very disturbed eating habits and an intense fear of gaining weight; some have a distorted body image and are extremely attentive to their weight and their body shapes. Clinical signs can include loss of weight, dehydration, compromised immune system for nutritional deficiencies and, in the females, amenorrhea. Kidney failure, osteoporosis, arrhythmias, infertility, esophagitis are all potential problems for long-term OSFED sufferers. The use of purgative behaviors, in particular, it can cause medical complications concerning the teeth,

the esophagus, the apparatus gastrointestinal, kidneys, skin, cardiovascular and musculoskeletal systems (16). The psychological characteristics include concern for food and appearance, dissatisfaction with weight and body shapes and increased levels of anxiety or agitation near meals. They are also warning signs of an OSFED restrictive behavior, take food at unusual times or wake up during the night to eat, compulsive and obsessive physical activity or ritualistic behaviors concerning the food and nutrition.

Although OSFED diagnoses, or otherwise residual diagnoses, are often considered the consideration for minor illnesses and those who suffer from it feel less entitled to receive help from those who present a full diagnosis are actually just as serious diseases as the main FEDs for as regards food psychopathology, physical complications, long duration of symptoms and psychiatric comorbidity, present in one third of cases. In OSFED, in particular, the disturbance major depressive was found in 43% of subjects and Obsessive-Compulsive Disorder in 21%; also other psychiatric symptoms including those belonging to post-traumatic stress disorder, phobias and psychoses are frequent. Sawyer et al. compared the clinical characteristics (physical and psychological) of the affected patients atypical AN compared to those of patients with full diagnosis of AN failing to differentiate them for the severity level (17). In fact, the two groups (ANa vs AN) did not show significant differences regarding the frequency of bradycardia (24% vs 33%), orthostatic instability (43% vs 38%), psychiatric comorbidity (38% vs 45%) or suicidal ideation (43% vs 39%). A percentage higher than ANa patients, however, was previously overweight or obese (71% vs 12%), had lost more weight (17.6 kg vs 11.0 kg) in a longer period of time (13.3 vs 10.2 months) and manifested a greater discomfort in relation to nutrition and body image. Despite a healthier weight ANa appeared to be severely affected by dysfunctional metacognitions and a desire to thinness like the AN (18) and have shown a similar profile of serious complications of loss of weight (19). A study by Silén et al. found that the ANa were older ($p = 0.03$), with a weight higher (minimum body mass index 16.7 vs 15.1 kg / m 2 , $p = 0.003$) and less subject to comorbidity (38% vs 71%, $p = 0.04$) compared to AN (20). Older research (21) did not notice significant differences in general psychopathology and in personality traits between subjects with BN or BNs.

The young OSFED investigated by Stice and colleagues (7) reported more functional deficits, discomfort, suicidality, mental health treatments, unhealthy body mass indices healthy controls, with a smaller effect size in the AN and BN. As in the other FEDs, in the OSFED the quality of life is poor and the degree of disability worsens with the severity of the disease, particularly in the presence of binge eating/purgative behavior (22). Comorbidity with personality disorders was also investigated and appeared high, with proportions of 0.38 for all types of personality disorder, 0.38 for Cluster C and 0.25 for cluster B (23).

**Diagnosis**

Residual diagnoses are often considered of little importance and do not even come recognized and diagnosed. In the repeatedly cited sample of Mustlein and colleagues (4) only 11% of subjects with OSFED/UFED had received some form of attention.

The diagnosis of OSFED is basically clinical and can make use of structured interviews and questionnaires used for the diagnosis of full EDs taking into account the changes made to the criteria diagnostics from the DSM-5. The studies carried out so far are rather scarce and generally report having adapted the diagnostic tools already in use (eg Structured interview for anorexia and bulimia nervosa self-report, SIAB-s; Structural Clinical Interview for DSM-IV, SCID; Eating Disorders Examination) as well as having

also used tests to evaluate the specific psychopathology of AD (eg. Eating Disorders Inventory-2) or the general one (Symptom CeckList-90R). At the moment it is not clear whether all the sub-threshold forms of FED can be included in the OSFED or only those specifically described in DSM-5 and ample discretion is left to the clinician of diagnose as UFED an eating disorder that presents uncertainty in the diagnosis for any reason. In the absence of a clarification, discordant data will be possible in several areas epidemiological, clinical, research). In confirmation of this, it was observed that there was little agreement between researchers and clinicians with respect to a specific diagnosis of OSFED, although the introduction of the DSM-5 criteria had reduced the residual categories (according to the criteria of the DSM-IV: clinicians 28.7% and researchers 59.3%; according to DSM-5 criteria: 12.2% clinicians and researchers 31.3%) and had not generally changed the inter-rater reliability of clinicians' diagnoses with respect to those of researchers (24). As for the other EDs, the diagnosis must be multidimensional, considering not only the criteria diagnostic or psychological characteristics, but also medical comorbidities/complications and psychiatric.

**Prevention and screening**

Given the recent introduction of the diagnostic criteria for these pathologies, there are no specific studies on their prevention, but some summary documents have highlighted the opportunity of an integrated approach to the prevention of ED and obesity through the healthy modification of family lifestyle (25). The presence of food restriction, bodily dissatisfaction and depression in adolescence seems to be able to identify subjects with a phenotype that puts them at a high level risk of developing food symptoms, represented by a wide spectrum of beliefs and maladaptive behaviors concerning nutrition and weight. These behaviors and beliefs they are found in both full and partial or sub-threshold syndromes and identify their risk factors reasons will allow effective prevention of ED (26). In the case of OSFEDs, it would be helpful for general practitioners screened for attitudes and behaviors disturbed food, in particular purgative conduct and night feeding, associated with medical complications even in the absence of a diagnosis of ED (16). Recognize the link between the purgative behaviors and medical complications could help identify potential EDs, particularly those who often circumvent the diagnosis such as purging disorder (16).

**Treatment**

The diagnosis of OSFED has no specific implications for choosing treatment or care from offer patients (27) and few studies offer indications. All EDs, including OSFED, are serious, potentially fatal and have long-term complications if left untreated, therefore, must be treated promptly. Healing is possible and the faster it is implemented therapeutic program all the more easily and quickly the complete resolution of the disorder. The treatment for OSFEDs should be similar to that of the corresponding DA and it should be offered by a professional or a team of professionals experienced in the care of these pathologies.

A special form of Cognitive-Behavioral Therapy (CBT) can be applied to OSFED called CBT-E (Enhanced) which has been studied for the treatment of all forms of DA. This approach it doesn't just focus on what is thought to be the central cognitive disorder of ED (the overestimation of food, weight and appearance), but also on the modification of the mechanisms that support food psychopathology such as perfectionism, low nuclear self-esteem, intolerance to emotions and interpersonal difficulties (28). CBT-E has also proven effective in follow-up (29) and is not specific to a given DA but is based on the concept that mechanisms municipalities are

involved in the persistence of all these pathologies (OSFED/UFED included). To try again of this, Ricca and collaborators (30) did not highlight differences in the response to a CBT treatment individual among AN patients with full or sub-threshold diagnosis, while the best predictor of outcome appeared to be concern for appearance.

**Assistance network**

The OSFED / UFED can use the same assistance network as the other DAs which provides levels of different care (outpatient clinic, day-hospital, ordinary hospitalization, rehabilitation, hospitalization, community therapy), by intensity and quality of care and duration of the care path, which can be activated depending on the severity of the pathology presented by the patient. The integrated or networked multidisciplinary approach should characterize nursing programs.

**Follow-up and social reintegration**

About 70% of subjects with OSFED heal over time and the best outcomes have been observed in individuals with high social support (31). The rapid response (less than 3 episodes of binge eating/vomiting in the first 4 weeks of treatment) the therapeutic program was predictive of remission at 6 and 12 months of follow-up in Purging disorder (32).

**Prognosis**

In OSFEDs the healing probability observed at 5 years is about 60% (95% CI 43-77%), with a average duration of symptoms of two years (4). The mean duration of disease, in months, in population samples was 11.6 for the ANa, 3.5 for the BNs, 3 for BEDs, 5 for Elimination Conduct Disorder (7) and percentages of remission to a year they seemed to vary from 71% for the ANa to 100% for the BNs and BEDs and to 94% for the PD. The recurrence rates of symptoms instead seem to fluctuate from 6% for PD to 33% for BEDs. These significantly more favorable data than those found in clinical specimens highlight the greater symptomatological and psychopathological severity of subjects requiring treatment. Stice e colleagues (7) reported a diagnostic progression, from ED below the threshold to those diagnosed full, higher for Bulimia nervosa and BED (32% and 28%) than AN (0%), suggesting a sort of exponential mechanism concerning binge eating. Silén et al. (20) have shown that ANa need shorter, less intensive and less intensive treatments cheaper than the AN. The full diagnosis of AN compared to that of ANa was found to be the only significant predictor of treatment outcome: healing in the ANa turned out to be 4.3 times (95% CI, 1.1-17.5) more likely than in typical NA.

Diagnosis of sub-threshold DA is frequent in patients with a history of AN or BN, it presents characteristics similar to the initial pathology, it is associated with a modest improvement in functioning psychosocial. It therefore appears that sub-threshold presentations may be part of the story natural of food pathologies (27-33). They certainly do not represent mild illnesses, but on the contrary, they condition psycho-social functionality a lot and have a serious prognosis that includes high mortality (27).

The reported results seem to indicate that the new diagnostic criteria for DSM-5 DAs are capturing clinically significant psychopathologies and usefully assign individuals with AD to categories homogeneous diagnostics.

**The Night Eating Syndrome Syndrome**

Although it was first described in 1955 by Stunkard (34), the Night Eating Syndrome (NES) was included only in 2013 in the DSM (35) among the OSFED, with the following definition: "Recurrent nocturnal feeding episodes that occur after waking up or with excessive consumption of food after the evening meal. There is awareness and I remember eating. Power nocturnal is not

justified by external influences such as changes in the individual cycle of sleep/wake or by specific social norms. Causes discomfort and/or impaired functioning. The disordered feeding patterns are not better explained by the diagnosis of binge eating disorder or from another mental disorder, including substance use, and not attributable to another medical or drug-related illness.

This delay was mainly due to the difficulty of uniquely defining the syndrome. In fifty years have followed twenty different sets of diagnostic criteria, sometimes even a lot different from each other. Finally in 2010 the main researchers interested in the syndrome have shared and published a table of diagnostic criteria, which since then are almost universally accepted and shared, which made it possible to have more reliable prevalence/incidence data, than compare the work of the different study groups and carry out studies on trans-national samples. Using these diagnostic criteria the prevalence of NES in student samples varies from 4.2% (1) to 5.7% (36), but excluding patients with BED the prevalence drops to 2.9% (35). Among patients affected by psychiatric disorders the prevalence of nocturnal eaters is around 22% (37), among those affected by depression the percentage rises to 25% (38). There do not appear to be differences between the sexes for regarding the prevalence of the syndrome (37).

**Diagnosis**

The diagnosis of NES is essentially clinical, but there are some useful test tools. The Night Eating Questonnaire (39) of which the Italian version is also available, edited by Vinai et al. (in validation course) and the Night Eating Syndrome History and Inventory (NESHI) (40), an interview semi-structured that allows to evaluate the severity of the syndrome. The main symptom of NES is the circadian delay in the introduction of food, which moves towards evening/night hours, while morning feeding decreases or disappears. Within patients affected by this syndrome are distinguished evening eaters (who eat after dinner, but before going to bed) and those at night (which feed during night awakenings). To diagnose NES, it requires that it be introduced after the evening meal, at least twice a week, before going to read, at least 25% of the entire daily calorie intake, while a quantity of food necessary to be defined "nocturnal eaters" and any quantity of food introduced during the nocturnal awakenings are considered potentially pathological. This is because the physiological situation neurohormonal at night (the prevalence of the levels of the anorectic hormone Leptin in the compared to that oressizzante Grelina) should make the income of food unlikely. Anorexia morning, another pivotal symptom of NES, is actually common among obese patients and in itself is not enough to diagnose.

**Differential diagnosis**

The NES differs from the Binge Eating Disorder for the time in which the food is consumed and for the different method of introduction of the same: compulsively and quickly in the BED; in the form of snacking in the NES. Another pathology that can be confused with NES is Sleep Related, Eating Disorder (SRED) a parasomnia described for the first time by Schenk and Mahowald (41) characterized by similar episodes sleepwalking, during which the patient can also eat, but unconsciously, without often to remember. For the altered state of consciousness in which these episodes occur, it often happens that the patient ingests inedible substances or cuts or burns while trying to get food, characteristics that allow quite easily to distinguish the two syndromes.

Unfortunately in some cases the two diseases can coexist and an accurate differential diagnosis is necessary to better set up the treatment.

**Comorbidities**

NES has been associated with many psychiatric disorders, mainly with depression, BED (39), insomnia and obesity. NES is more common in patients with other behavioral disorders food (ED), especially in those with BED and bulimia nervosa (BN), compared to the population general. Additionally, individuals with NES are more likely to have other eating disorders. There estimated prevalence of NES among ED patients ranges from 5% to 44% (42-45). NES has been reported in 15% -44% of patients with BED (42); subjects with BED and NES have more serious psychopathologies and BMI higher than those with only one syndrome (43,44). Few studies have investigated the relationship between NES and AN or BN: the prevalence of NES was estimated in 9% -47% of patients with BN (46). The NES it can cause and / or maintain sleep disturbances, but sleep disturbances can also precede the onset of NES (47). In obese patients NES is associated with greater severity of symptoms psychiatric, social functioning reduced lower levels of employment and higher prevalence of depressive symptoms. Furthermore, the diurnal behavior of obese NES patients is more chaotic than that of those not affected by the syndrome (48).

**Treatment**

There are few studies on NES therapy, both pharmacological and psychotherapeutic and using bright-light-therapy. A psycho-educational /behavioral approach associated with relaxation techniques it has been shown to be effective in a group of 44 night-eating patients (16). Clinical trials have mainly focused on antidepressant drugs, particularly SSRIs, in the treatment of NES. Improvements in symptoms have been reported with paroxetine and fluvoxamine. The effects of sertraline and escitalopram have been tested in two double-blind randomized placebo-controlled studies controlled (49,50) and three other open-label uncontrolled studies (49-52). Sertraline has been shown to improve NES symptoms, mood disorders and quality of life in patients by reducing intake night calorie and BMI, even in uncontrolled studies (52-56), as well as Escitalopram.

**ARFID-AVOIDANT**

ARFID is a new eating disorder included in DSM-5 (57). It can manifest itself at all ages, but it is more frequent in childhood and adolescence with a male / female ratio of 2: 1; the patients are younger than those with anorexia or bulimia nervosa and often exhibit psychiatric comorbidity with high levels of anxiety and depression, perfectionist tendencies; may fall within the autistic spectrum or have an attention deficit disorder. At the moment there are few studies on the prevalence of ARFID. An English study (2005-2006) documented an incidence of ARFID of 0.57 new cases per 100,000 (58). A Canadian study (2003-2005) suggests an incidence of ARFID in a population of 5 to 12 years of 0.69 new cases per 100,000 (59). In a Swiss study in a sample of 1444 subjects 8 to 13 years the prevalence is 3.2% (60). Three recent studies on early-onset eating disorders (12 years or less) have reported that in this age group there are numerous cases in which avoidance deliberate food with severe weight loss is not accompanied by fear of gaining weight and sprains body image: 1 in 5 to 1 in 3. The diagnosis of anorexia nervosa, although medical complications from malnutrition and psychiatric comorbidity are similar; instead, according to DSM-5, the diagnosis of ARFID (61) is applied. The epidemiology of ARFID in the general population is still uncertain, given the recent definition. The diagnostic criteria of ARFID according to DSM-5 are as follows:

1) Disturbance in food intake as a lack of interest in food; avoidance based on the sensorial characteristics of food products such

as color, consistency, taste; concern about harm related to food intake. This causes an inability to satisfy adequate nutritional and/or energy needs associated with:

a. Weight loss or failure to achieve expected weight gain or slowdown growth in children.

b. Nutritional deficiency.

c. Addiction to enteral nutrition or oral nutritional supplements.

d. Marked interference in the psychosocial sphere.

2) The disturbance is not explained by the lack of available food and is not connected with a consolidated one cultural practice.

3) The disorder does not occur exclusively in the course of anorexia or bulimia nervosa. Patients do not they fear weight gain, are not dissatisfied with body weight, with their physical fitness, of size, of one's own body image.

4) The disorder is not attributable to a concomitant medical condition or is not explained by another mental disorder. Children eat few foods and refuse to taste new ones. In front of a food unknown react with disgust and sometimes with retching. They get distracted at the table, they consume meals slowly, take some foods only if "masked" inside the food and favorite drinks.

A "picky eater" child must be distinguished from one with ARFID. The "Picky eater" or *picky-fussy eater* is defined as a child of normal weight but that he consumes an inadequate variety of foods by refusing both familiar and unfamiliar foods (62). There common feature includes limitation in food intake, inability to experiment with new ones (*food neophobia*). This neophobia peaks between two and six years of life with a gradual reduction over time enough to contract drastically in adulthood. The prevalence of *picky eating* varies from 14 to 50% of preschool children and from 7 to 27% of older children. There is also an increase of the 15-16 year olds who say no to food for fear of indigestion or suffocation or on the basis of the sensory characteristics of food.

Sometimes ARFID lasts over time, even in adulthood. Social life is especially undermined with waivers of dinners, parties, aperitifs. The first point of contact for many patients is the general practitioner or the general practitioner community pediatrician; other children are referred to dieticians or dieticians, pediatricians of developmental age, gastroenterologists, psychologists, psychiatrists. In consideration of the heterogeneity of the clinical picture, they are possible difficulties for healthcare professionals in knowing how to present them ARFID to reach the most appropriate diagnosis and therapy. At the moment they do not exist for ARFID *evidence-based* therapeutic recommendations ; however clinical experience suggests that the approach may vary depending on the factors that induce the eating disorder. For example patients who experience food restriction and weight loss for fear of suffocation respond better at cognitive strategies. On the other hand for children who have a poor history growth for food selectivity, combined psychological-behavioral. The duration of nutritional and psychological interventions varies from 1 to 3 years. Currently there are no prospective studies reporting results of targeted therapeutic interventions for patients with ARFID (62).

**Drunkorexia**

Drunkorexia is a made-up term reflecting an alarmingly real trend among young women. The non-medical slang term refers to women who choose to eat less so they can party hearty without gaining weight (63). The web article published in 2008, of which the first lines are reported, initials the birth of the "Drunkorexia", a term apt to describe that set of behaviors (calorie restriction, exercise, dysfunctional eating habits) put deliberately implemented by the person to compensate for the calories

taken with alcohol (64). Also known as Inappropriate Compensatory Behavior to Avoid Weight Assumption due to alcohol consumption (Inappropriate Compensatory Behavior to avoid Weight Gain from consuming Alcohol, ICB-WGA), this phenomenon first took hold in the campus environment American university students, then arriving in Europe, starting from Great Britain and interesting gradually all other countries, including Spain, France and Italy. As an emerging phenomenon, Drunkorexia does not yet represent a recognized pathology from the scientific community. However, the spread and seriousness of this behavior model make it diagnostic framework required. To this end, the link has recently been confirmed between ICB-WGA and other patterns of disturbed behavior, such as Behavioral Disorders Food (BDF) and Substance Use Disorder (DUS) (65).

Like other eating disorders, such as Anorexia Nervosa (AN), Drunkorexia originates from:

a) intense fear of gaining weight or gaining weight, or persistent behavior that it interferes with weight gain, despite a significantly low weight; and

b) anomaly in the way it is perceived the weight and shape of your body; inappropriate influence of weight and body shape on one's self-esteem, or persistent loss of the ability to assess the severity of the current loss weight (66).

Despite often believed to be a subcategory of the AN, Drunkorexia, like the AN, is however characterized also by the presence of: a) recurrent episodes of binge eating and b) compensation behavior aimed at prevent weight gain, such as self-induced vomiting, abuse-misuse of laxatives, diuretics or other drugs; fasting or excessive exercise; c) inappropriately influenced self-assessment the shape and weight of the body (66).

This dysfunctional behavior model also presents a cluster of behavioral symptoms and typical DUS physicists (67), such as abstinence, tolerance and craving (strong desire to take the substance) (66). The reference is to the phenomenon of Binge Drinking (68), or the intake of multiple drinks alcohol in a more or less short time interval (69). The type of substance that comes is not important ingested nor any alcoholic addiction: the main purpose of these "alcoholic binges" is immediate intoxication and loss of control (70).

The existence of comorbidities between eating disorders and alcoholism has also been known for some time (72-74): alcohol is often used by bulimic subjects to more easily induce vomiting (purging) by compensating, thus, the food binges (75,76); and also people with AN, although obsessed with calorie counting, today they are often willing to take excessive doses of alcohol because they are able to calm anxiety related to food intake, provide a sense of satiety (77,78) and, once again, facilitate purging.

Attempting a summary, characteristic symptoms of Drunkorexia are:

1. alcohol abuse (with preference for low calorie drinks);
2. food restriction, with calorie counting, or fasting (as in the AN),
3. use of compensatory behaviors, such as self-induced vomiting (to empty the stomach of food before drinking alcohol or allowing more alcohol to be consumed during a social event), use of laxatives or excessive physical activity to counterbalance the calories eaten with alcohol (as in the AN),
4. constant concern about body image (67).

To push this behavior would be the pressures arising from the company, and above all from university environments, where being slim and having fun is an imperative in force. It happens like this that those who do not want to gain weight but cannot say no to cocktails and appetizers with friends decide to compensate for the calorie intake derived from alcohol by drastically reducing the food intake.

Since, then, the balance needle is a predominantly female obsession, they are

particularly the girls to be the victim of the disorder; also attracted by the so-called girlie drinks, colorful, sweet, packaged with cartoon images, but still with alcohol content. The fixation for the thinness, then combines with the example of famous people such as Paris Hilton, Lady Gaga or Lindsay Lohan who, by reporting similar patterns of behavior, do absolutely conduct inappropriate a fashion to emulate and a pass to be accepted by the group (79).

The age of onset of the disorder is estimated to vary between fifteen and thirty but, as with anorexia, is strongly lowering. In the same way data on the use of alcohol among young people always confirm earlier consumer experiences, and the family appears to have a key role in the etiopathogenesis of this problematic: the mourning of a loved one, the separation of the parents, the conflict between the walls domestic, up to the most serious cases of abuse and domestic violence.

Malnutrition associated with the effects of alcohol causes damage at many levels. The least serious are rushes mood, tachycardia and sweats. Similarly to what happens in the AN, if not in even to a greater extent, important electrolyte imbalances and pathologies may also develop kidney, neurological, cardiovascular, amenorrhea, ulcers of the esophagus and, as for those who abuse alcohol, the risk is to face alcoholic hepatitis and cirrhosis of the liver.

In girls, in particular, early use of alcohol induces emotional, learning, memory and causes morphological changes of some parts of the brain that do not appear in male drinkers, and the alcohol-hormonal structure interaction causes alteration of the menstrual cycle, decreases fertility and increases the risk of breast cancer.

In Drunkorexia, therefore, eating disorders and alcoholism coexist, and it is important that these are treated simultaneously through a multidisciplinary approach.

**Muscular dysmorphy or bigorexia**

Muscular Dysmorphy (DM), first described in 1997 by Pope, is a condition characterized by an intense and constant concern to be too thin, thin or not sufficiently muscular, by people with regular or already highly developed muscles, with consequent sense of inadequacy (80,81).

DM is included in DSM-5 as a variant of the body dimorphism disorder (82) characterized by unjustified concerns about body appearance, compulsive behavior, clinically uncomfortable significant with impairment of social functioning and absence of eating disorder.

However, this location remains controversial (80, 83). The various studies also use different tools psychometric that make univocal interpretation difficult.

The literature on the subject, flourishing but heterogeneous, states that DM mainly affects sex male, but it is also present in the female one.

As far as epidemiology is concerned, very few studies exist, the most recent estimates 427,000 cases equal to 0.7% of the population with a high prevalence, equal to 10%, among subjects who attend gyms. The strong focus on the muscularity of the body involves significant changes in the lifestyle of those who it is affected, such as carrying out a high number of hours of exercises in the gym, the implementation of unbalanced diet regimes, the excessive use of food supplements and often anabolic steroids All this, together with the perceived physical discomfort, causes social isolation, absenteeism from work, high risk of suicide (80).

In the development of DM, predisposing factors seem to exist (male gender, ages between 15 and 32 years, social pressures towards body worship, obsessive-compulsive tendencies and addictions, negative experiences related to the body, low self-esteem and insecurity in the sphere of intimacy, virility and sexual performance), triggers (traumatic events of

violence suffered as robberies, domestic violence, physical assault, bullying) and maintenance (social reinforcement, avoidance negative feelings related to body image and denial of obsessive thoughts, reduction anxiety through performing exercises) (80, 84).
DSM-5 lists depression, social phobia, obsessive compulsive disorder, comorbidities, medical complications secondary to substance abuse and inability to reduce intense activity physical, even in case of minor injuries. Currently the treatment is linked to the diagnostic classification, therefore the strategies are the same used for body dimorphism disorder and include cognitive behavioral therapy and use of selective serotonin reuptake inhibitor antidepressants. Anyway too this aspect remains controversial (80).

**Orthorexia Nervosa**

The term "orthorexia" derives from two words belonging to classical Greek: *orthos* , which means straight, correct, right and orexis which means appetite. In a non-literal way it could therefore be translated with "Proper nutrition". Orthorexia nervosa, on the other hand, is the expression that applies to people who don't they are limited to the moderate search for healthy foods, but which develop a form of obsession with food healthy or presumed such. In the mid-nineties, the first to coin this term to pinpoint the disorder, was Steven Bratman, an American dietitian who himself became a dietitian orthorexic. At that time he coined a test to try to identify subjects suffering from ON (test of Bratman) and published his book Health Food Junkies in which he traced the characters of the disorder comparing to pathological addictions (85).
Orthorexia nervosa is an emerging phenomenon but it is not classified in the DSM-5. Only recently, first from Moroze in 2015 and then from Dunn and Bratman, in a 2016 review (86), new diagnostic criteria were proposed that take into account the obsessive focus on healthy food and the resulting clinical compromises. So far, tests have been used to study the prevalence of the disorder in the various populations psychometric, in particular the ORTO-15 test validated for the Italian population by Donini (87) or some its variants and translations proposed in other countries. The prevalence of ON varies widely from 6% to 88.7%, with most studies accounting for between 30% and 70%. The maximum tips of prevalence is found precisely among operators in the clinical-nutritional area. Variability depends the populations studied, the psychometric tests administered and the cut-offs used. Anyhow the high presence of the disorder, highlighted by these studies, is questioned pending definitive diagnostic criteria and review of psychometric tests (86). The characteristics of the disorder can be summed up in fear of contracting diseases through the intake of food deemed impure/contaminated, selective choices towards "healthy-natural" nutrients, long time spent choosing, preparing and consuming food, ritual behaviors similar to compulsive ones, social functioning compromised by isolation and impaired judgment and altered physical well-being for extreme choices.
The ON has areas of difference and areas of overlap with Anorexia Nervosa and with obsessive compulsive disorder, while it does not seem attributable to the group of disorders Avoidant/ Restrictive Food Intake Disorder (ARFID), classified in DSM-5, which determine an avoidance of certain foods in response to traumatic events or difficulty feeding due to fear of development of adverse situations, not for concerns related to impure food (86). From a clinical-nutritional point of view, these patients, following specific restrictions, manifest themselves deficiencies similar to those that we can find in anorexia nervosa, such as electrolyte imbalances, avitaminosis, osteoporosis, muscle wasting and other physical problems that may also require

prolonged and expensive therapeutic interventions. The therapy of these patients is not simple because they are very reluctant to take drugs, which they consider them not pure, and are very resistant even to change their diet. However the appearance "Health" of the disease can prove to be a motivational push to accept treatment. They do not exist therapies codified for the ON but it seems that a multidisciplinary team work composed of doctors, psychotherapists and dietitians, combining cognitive behavioral therapy, nutritional rehabilitation and drug therapy with antidepressants selective serotonin reuptake inhibitors and antipsychotics such as olanzapine, when indicated, may lead to improvements (88).

Concerns about the ON for the foreseeable future there are concerns about the fact that still there is no clear definition and the fact that the orthorexic subject is not limited to rigidity personal food but you can be a promoter of extreme impositions even against minors or more fragile subjects that depend on him (89). For this reason it will be particularly important also identify who, among the operators in the sector, has orthorexic tendencies and recommends food plans too inflexible and unhealthy for fragile and influenceable patients.

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