

Nonspecific eating disorders – a subjective review

Aneta Michalska, Natalia Szejko, Andrzej Jakubczyk, Marcin Wojnar

Chair and Department of Psychiatry, Medical University of Warsaw

Summary

Aim. The aim of this paper was to characterise nonspecific eating disorders (other than anorexia nervosa and bulimia nervosa).

Method. The Medline database was searched for articles on nonspecific eating disorders. The following disorders were described: binge eating disorder (BED), pica, rumination disorder, avoidant/restrictive food intake disorder, night eating syndrome (NES), sleep-related eating disorder (SRED), bigorexia, orthorexia, focusing on diagnosis, symptoms, assessment, comorbidities, clinical implications and treatment.

Results. All of the included disorders may have dangerous consequences, both somatic and psychological. They are often comorbid with other psychiatric disorders. Approximately a few percent of general population can be diagnosed with each disorder, from 0.5–4.7% (SRED) to about 7% (orthorexia). With the growing literature on the subject and changes in DSM-5, clinicians recognise and treat those disorders more often.

Conclusions. More studies have to be conducted in order to differentiate disorders and treat or prevent them appropriately.

Key words: eating disorders, DSM-5

Introduction

Nowadays different eating disorders are described more and more frequently. While anorexia nervosa (AN) or bulimia nervosa (BN) are diagnosed and treated frequently by clinicians, there are a lot of others, so called nonspecific eating disorders. Though they may seem uncommon, one would be surprised how many individuals have such problems. However, those lesser-known disorders can also be dangerous, mainly because they may lead to AN or BN, or may be associated with other psychiatric diseases such as depression or anxiety disorders. While it may be debatable if changes in

DSM-5 (recognising more and more disorders) are necessary, the need for thorough questions on patients' eating habits remains true.

Aim

The aim of the present paper was to characterise nonspecific disorders in the context of changes in DSM-5. We described: binge eating disorder (BED); pica; rumination disorder; avoidant/restrictive food intake disorder; night eating syndrome (NES); sleep-related eating disorder (SRED); bigorexia; orthorexia. We chose, in our opinion, the most interesting and clinically important disorders. We focused on disorders that were classified differently in DSM-5 than in its previous editions. Not all of them are classified as feeding and eating disorders, some are still not included in DSM-5.

Material and Method

We searched the Medline database for eligible studies published in English and Polish. The following search terms were used: nonspecific eating disorders, pica, rumination disorder, avoidant/restrictive food intake disorder, binge eating disorder, night eating disorder, sleep-related eating disorder, bigorexia, orthorexia. Search terms were combined with "DSM-5" and "treatment" in a title. The bibliographies of the retrieved papers were also reviewed. We included papers that met the following criteria: 1) reviews on chosen disorders; 2) retrospective and prospective studies, as well as case reports concerning prevalence, risk factors and treatment. Unpublished studies, abstracts of conference proceedings and dissertations were excluded.

Binge eating disorder

Binge eating disorder (BED) is defined as recurring episodes of eating significantly more food in a short period of time than most people would eat under similar circumstances, with episodes marked by feeling of lack of control [1]. Those episodes cannot be less frequent than once a week for 3 months. During such episodes people with BED eat too quickly, even without the feeling of hunger, up to the feeling of unpleasant fullness. Binges happen when a person is alone as individuals with BED tend to hide their behaviour. DSM-5 included BED as a formal diagnostic category.

The disorder is associated with feeling of guilt, shame, disgust, low self-esteem, stress, boredom, feeling that eating is out of control. Significant psychiatric comorbidities include major depression, generalised anxiety disorder, panic attacks, history of suicide attempts [2]. Aside from psychological comorbidities, there are potential somatic problems which are consequences of obesity (such as high blood pressure, coronary heart disease) as even up to 70% of BED patients is shown to be obese [2]. Taking the above into consideration, cooperation between specialists and multidisciplinary approach is highly recommended in case of BED.

BED may even be more common than anorexia nervosa and bulimia nervosa as its prevalence is about 2% in men and 3.5% in women according to Hudson [3].

However, it is more frequent in certain populations, for example 29% of those seeking treatment for obesity [4]. It is more prevalent among older adults, but the disorder starts in early twenties [3].

Although causes are unknown, there are a few factors that may increase the risk of BED, namely familial factors independent from obesity such as shared genes that are still unknown [3], and psychological problems (mainly due to being overweight). It is important to differentiate BED from BN and overeating. BED lacks the use of compensatory measures to prevent weight gain. The main symptom of BED is the loss of control over eating, not the focus on weight or appearance. It can be therefore discussed whether BED suits best in eating disorders or rather in impulse control disorders category.

The treatment includes self-help groups, psychotherapy, mainly CBT (cognitive-behavioural therapy), but also IPT (interpersonal therapy) and DBT (dialectical behaviour therapy), body mass reduction and pharmacotherapy: antidepressants (mainly SSRIs) or topiramate. Brownley reviewed 26 randomised controlled trials and concluded that SSRIs can reduce eating, psychiatric and weight symptoms; topiramate and sibutramine were promising and CBT can be effective in improving psychological features and reducing binge episodes, but not weight. According to this review, the literature regarding treatment of BED is inconsistent [5].

Pica, rumination disorder and avoidant/restrictive food intake disorder

First described by Hippocrates, pica for a long time has been diagnosed mostly in children, but since DSM-5 eliminated the category “feeding disorder of infancy and early childhood” psychiatrists started to assign pica to people of all ages. In ICD-10 it is classified in category “other eating disorders”. It is defined as the recurrent consumption of nonnutritive, nonfood items [1]. These behaviours must last at least one month. Moreover, it is not considered pica if consumption is developmentally or culturally appropriate (for example it is a common practice in Africa to eat white clay).

The disorder’s name comes from Latin name of magpie, the bird that is famous for stealing inedible items. There are various subtypes, depending on the substance consumed. Thus we recognise amylophagia (consumption of starch), coprophagia (faeces), geophagia (soil), hyalophagia (glass), litophagia (rocks), mucophagia (mucus), pagophagia (ice), trichophagia (hair; usually as a possible consequence of trichotillomania, which consists of hair pulling resulting in hair loss and other aesthetic, psychological and health disturbances), xylophagia (wood, paper) to name a few.

Pica can be caused by other disorders such as iron deficiency (mainly in children and pregnant women). In some cases, it is also associated with schizophrenia [6], autism spectrum disorders [7], and mental impairment or developmental delay [8] among others. Taking into consideration that eating is repetitive, egodystonic, intrusive, it has been suggested that pica lies in the obsessive-compulsive spectrum [9]. Another argument in favour of this theory is responsiveness to SSRIs [9]. On the other hand, some cases of pica can be diagnosed as impulse control disorders [9]. While exact aetiology of pica is still unknown, it has been suggested that hunger, nutrient

deficiencies, gastrointestinal distress or susceptibility to pathogens may play a role in its pathogenesis [10].

It is difficult to estimate the disorder's prevalence in general population. However, there are studies concerning populations in which it is more frequent: 1.3% of individuals being treated for eating disorders [11], 21.8% of persons with intellectual disabilities [12], 48% of autistic children [7] met the criteria for pica.

It often causes problems in daily functioning. What is more, it can cause some serious somatic consequences such as peptic ulcers, perforations, bowel obstruction, infections, anaemia, electrolyte imbalance and even poisoning. Cooperation between specialists (psychiatrists, general practitioners, surgeons, gastroenterologists) is necessary, as pica may be secondary to treatable internal diseases, as well as have somatic consequences that require urgent care.

If pica is secondary to other diseases, treatment of the underlying disease is necessary. In primary cases of pica, psychotherapy is recommended. Additionally, as other obsessive-compulsive spectrum disorders, pica can respond to SSRIs such as fluoxetine and sertraline [13].

Similarly, criteria for rumination disorder (RD) can be used with individuals of any age. RD is characterised by repeated regurgitation of food for at least 1 month. The behaviour is not attributable to any gastrointestinal condition. It does not occur exclusively during the course of AN, BN or any other eating disorder. It results in psychological distress and medical problems such as malnutrition and dental complications [11]. The overall prevalence is still unknown. It is more common in some populations, e.g. 5–10% of people with developmental disabilities [14].

Avoidant/restrictive food intake disorder (ARFID) replaced feeding and eating disorder of infancy or early childhood. The diagnosis can be used in adults who restrict their food intake and do not meet the criteria for other eating disorders.

Night eating syndrome and sleep-related eating disorder

Night eating syndrome (NES) is defined by evening hyperphagia with lack of hunger in the morning [15]. According to proposed criteria, such episodes happen at least twice a week over a 3-month period of time [15]. 25% or more daily food intake is consumed after the evening meal. Moreover, NES cannot be secondary to other somatic or psychiatric conditions nor the result of using drugs or psychoactive substances [15]. People with NES are aware of eating these foods and later remember doing so, contrary to patients with sleep-related eating disorder. Eating so much during late hours usually causes discomfort, problems in one's day-to-day functioning, depressive states and sleep disorders. Patients experience some kind of compulsiveness or loss of control, feel that they have to eat after supper in order to fall asleep [15]. Sleep of patients with NES was shown to be not effective, as they often arouse from non-REM sleep [16].

It is estimated that about 1.5% of general population can be diagnosed with NES [17]. However, it is more common among specific populations, namely people who are currently on diet – 4.3% [18], psychiatric outpatients – 12.3% [19], people with II and III class obesity – 10.1% [20]. The disorder usually starts in young adults. Patients with

NES have been shown to be 4.9 times more likely to have a first-degree relative also suffering from NES [19], which implicates the possible genetic contribution. It was shown equally frequent in men and women [21]. Although people do not necessarily gain weight, higher BMI was observed to be associated with increased risk of NES [18]. The disorder is comorbid with other eating disorders (25% in Lundgren's study) [22], depression [23], insomnia [24], harmful use of psychoactive substances [19]. It is important to differentiate NES from binge eating disorder, bulimia nervosa and sleep-related eating disorder.

Some studies suggest that NES is caused by circadian delay of food intake with normal circadian sleep onset [25]. These eating habits may also be the effects of poor behavioural control. As there is an increased activity of serotonergic transporter in mesencephalon, it has been proposed that the serotonergic system has a crucial role in the pathogenesis [26]. Nocturnal eating may also result in changes in hormone levels. According to one study, levels of ghrelin, a hormone suppressed by feeding, are significantly lower in NES subjects compared with healthy controls, while insulin levels are higher [27].

In treatment of NES, as in other eating disorders, psychological help and psychotherapy (e.g. CBT) is of utmost importance. However, patients may also benefit from pharmacotherapy. According to one trial, sertraline may be beneficial in the treatment as there was a significant improvement in symptoms of NES [28]. A case of successful treatment with topiramate has also been reported [29]. Melatonin [30] may also be effective. However, further studies on the pathogenesis and treatment options need to be conducted.

Sleep-related eating disorder (SRED), which is technically not an eating disorder but a form of NREM parasomnia, is worth mentioning. As described in International Classification of Sleep Disorders, it is a chronic disorder that consists of recurrent episodes of involuntary eating and drinking during the main sleep period which results in some health problems, usually due to being overweight. The episodes, in contrast to NES, are unintentional and occur after falling asleep and the patient is unaware of them and do not recall them the next day. The patients are overly focused on their weight. However, purging behaviours do not exist [31]. Moreover, important symptoms include: insomnia, sleep related injuries and dangerous behaviours during sleep, lack of morning appetite and sometimes eating inedible foods [31, 32]. SRED has been shown to be comorbid with mental disorders: affective disorders [33], anxiety [34], substance abuse [35], eating disorders. Sleep disorders that are comorbid with SRED include somnambulism [32] and RLS (restless legs syndrome) [36]. Additionally, SRED can cause obesity [34] and its health consequences such as diabetes mellitus type 2.

Its prevalence in general population is estimated to be 0.5–4.7% [33, 34], while it is more common among patients with other eating disorders – 16.7% of inpatients according to one study [33]. In a study by Winkelman et al., 26% of the patients had a first-degree relative with nocturnal eating behaviour [32]. The exact cause of SRED is still unknown, although dopaminergic dysfunction has been postulated [37]. Notably adverse effects of some drugs can present themselves as symptoms of SRED, these include most famously zolpidem [38].

A few drugs were investigated as a treatment option in addition to psychotherapy. According to one study, pramipexole can reduce some of the symptoms of SRED [39]. Topiramate was also found to be effective [40]. Moreover, it is essential to treat comorbid psychiatric disorders and modify the bedroom environment so that it is safe for the patient.

Bigorexia

Bigorexia, also called muscle dysmorphia, is an emerging problem, especially due to promotion of bodybuilding culture, excessive concentration on body, implementation of special dietary restrictions, dedication to sports, particularly body-building ones, and introduction of supplements or even drugs in order to achieve adequate body mass. The problem of this disorder is an important one, as eating disorders among men are underestimated not only in the entire society but also by doctors. According to DSM-5, muscle dysmorphia can be a variant of BDD (body dysmorphic disorder), in which an individual is preoccupied with the idea that his or her body is insufficiently muscular [1].

Bigorexia is promoted in culture but also in sport, as during the competition of body-builders, their appearance is assessed by the judges. Currently this problem affects approximately 10% of body-builders [41]. The term was used firstly by Pope, Katz and Hudson and was then designated as reverse anorexia, as it resulted in accumulation of mass and not its diminution. Notably, the prevalence of previous episodes of AN is higher in this group of patients and they share another important similarity with anorectic patients: reduction of fat and gain of muscle mass. Moreover, AN and bigorexia may be both perceived as disorders of self over-control, differently manifested in men and women. To the best of our knowledge, to date these similarities have not been studied thoroughly.

Pope et al. described diagnostic criteria helping to encounter the disease [42]:

- Strong preoccupation with appearance, especially body mass and dedication to extreme efforts to achieve it (dietary restrictions and practice in gym);
- Ignorance of social norms in order to follow restrictions to maintain the body mass;
- Elimination of situations that lead to body exposure (beaches, swimming pools) as the territory of social judgment;
- Continuation of dietary restriction, excessive exercises and implementation of drugs despite of adverse effects.

For clinicians, it is therefore important to remember about side effects of drugs implemented in bigorexia with special attention to anabolic steroids. The most important side effects include: raised cholesterol, prostate enlargement, acne, gynecomastia and testicular atrophy [43]. Moreover, Pope et al. concluded that sudden reduction of steroids can lead to depression. It is essential that the treatment includes psychotherapy [44].

Orthorexia

Orthorexia is described as extreme concern about healthy diet and is estimated to affect about 7% of general population and to be more frequent among men [45]. It is important, but underestimated problem. Moreover, fewer men than women seek help in this area, making orthorexia even more difficult to detect and treat.

The term was introduced by Bratman in 1997 and still remains of interest of specialists, particularly, regarding classification as it is not clear whether it belongs to eating disorders or obsessive–compulsive disorders. Orthorexia is diagnosed when the patient dedicates all his or her daily activity to plan the diet and healthy lifestyle and ignores social life and job. Bratman also underlined that orthorexia is related to determined pattern of emotional reaction and personality, particularly sensation of control achieved by restriction of diet. The high prevalence of orthorexia is a direct consequence of tendencies promoted in postmodern societies: focus on appearance and increasing concern about health. Although concentration on health seems to be a positive trend, extreme dedication of all activities to research on healthy diets could be manifestation of anankastic tendencies. Restrictive diet implemented in orthorexia leads, first of all, to nutritional deficits and, secondly, to social problems.

As in other nonspecific eating disorders, diagnostic criteria of orthorexia are neither clear nor well established. According to Bratman and Knight, the patient should answer affirmatively to at least 4 out of 10 questions [46]:

1. Do you dedicate more than 3 hours per day to think about healthy diet?
2. Do you plan your meals one day before?
3. What is more important to you: what you eat or the pleasure you have from eating?
4. Did your quality of life diminish with introduction of a healthy diet?
5. Are you more and more restrictive in your diet?
6. Are you capable of sacrificing pleasure derived from eating in order to maintain a healthy diet?
7. Does your self-esteem increase when you eat healthy? Do you dispraise people who do not follow healthy pattern of nutrition?
8. Do you feel guilty when you do not follow your diet?
9. Do you consider your diet as an isolating factor in social contacts?
10. Do you experience sensation of total control when you eat properly?

Recently, more restrictive and accurate diagnostic criteria of orthorexia were established [45]. The most important points are: obsessive-compulsive traits, health fanatic eating habits, permanent character of behaviour, negative effect on patient's quality of life enhanced by eating patterns.

It is also vital to remember about the discussion on classification of orthorexia. Janas–Kozik et al. underline the fact, that there are many similarities between orthorexia and anorexia nervosa. Among those, the most important ones are: rituals related to eating, concentration on food, strict dietary habits and very close relationship between eating and self-esteem. However, a distinctive characteristic is lower prevalence of patients with minimal BMI among individuals with orthorexia. On the other hand,

orthorectic patients demonstrate focus on food and often introduce rituals to control their daily routine and therefore orthorexia could be qualified as OCD. Nevertheless, contrary to OCD, orthorexia is related to egosyntonic behaviours, while OCD patients experience strong distress associated with the disease [47].

Although there are no studies concerning effectiveness of treatment options, CBT combined with SSRIs as well as olanzapine were reported to be effective [48, 49].

Conclusions

Nonspecific eating disorders presented in this paper have both similarities and differences. Pica, rumination disorder, avoidant/restrictive food intake disorder and BED are classified in DSM-5 as separate diagnostic categories. NES is classified as “other specified feeding and eating disorder”. On the other hand, SRED, orthorexia and bigorexia are not included in DSM-5. All of the included disorders may have some dangerous consequences, both somatic and psychological. They are often comorbid with other psychiatric disorders. Symptoms of nonspecific eating disorders may overlap with each other and it is therefore difficult to differentiate them. Also, similarities overlapping of symptoms may raise question if these disorders are independent nosological units or just variants of one or two basic disorders. Similarly, their prevalence is difficult to estimate. Approximately a few percent of general population can be diagnosed with each disorder, from 0.5–4.7% (SRED) to about 7% (orthorexia). Nevertheless, the diagnostic criteria of all nonspecific eating disorders remain the most difficult. Although symptoms vary, depending on an eating disorder, it is important to differentiate one from another, as well as from AN, BN and other psychiatric or somatic diseases.

Their presence in DSM-5 implies a degree of impairment in the quality of life. However, it is questioned by some, as it may bring unnecessary chaos to the classification with too many diagnoses. The disorders that are not yet classified were included because of their clinical or social importance and growing research. We believe that nonspecific eating disorders require further studies that would help in their detection and treatment.

References

1. American Psychiatric Association. *Diagnostic and statistical manual of mental disorders*. 5th ed. Arlington, VA: American Psychiatric Publishing; 2013.
2. Grucza RA, Przybeck TR, Cloninger CR. *Prevalence and correlates of binge eating disorder in a community sample*. *Compr. Psychiatry* 2007; 48(2): 124–131.
3. Hudson JI, Hiripi E, Pope HG Jr., Kessler RC. *The prevalence and correlates of eating disorders in National Comorbidity Survey Replication*. *Biol. Psychiatry* 2007; 61(3): 348–358
4. Spitzer RL, Yanofsky S, Wadden T, Wing R, Marcus MD, Stunkard A. et al. *Binge eating disorder: its further validation in a multisite study*. *Int. J. Eat. Disord.* 1993; 13(2): 137–153.
5. Brownley KA, Berkman ND, Sedway JD, Lohr KN, Bulik CM. *Binge eating disorder treatment: a systematic review of randomized controlled trials*. *Int. J. Eat. Disord.* 2007; 40(4): 337–348.

6. Dumaguing NI, Singh I, Sethi M, Devanand DP. *Pica in the geriatric mentally ill: unrelenting and potentially fatal*. J. Geriatr. Psychiatry Neurol. 2003; 16(3): 189–191.
7. Clark B, Vandermeer B, Simonetti A, Buka I. *Is lead a concern in Canadian autistic children?* Paediatr. Child Health 2010; 15(1): 17–22.
8. Martindale JL, Bunker CJ, Noble VE. *Ingested foreign bodies in a patient with pica*. Gastroenterol. Hepatol. 2010; 6(9): 582–584.
9. Stein DJ, Bouwer C, van Heerolen B. *Pica and obsessive-compulsive spectrum disorders*. S. Afr. ed. J. 1996; 86(12 suppl.): 1586–1588, 1591–1592.
10. Young SL, Wilson MJ, Miller D, Hillier S. *Toward a comprehensive approach to the collection and analysis of pica substances, with emphasis on geophagic materials*. PLoS ONE 2008; 3(9): e3147.
11. Delaney CB, Eddy KT, Hartmann AS, Becker AE, Murray HB, Thomas JJ. *Pica and rumination behavior among individuals seeking treatment for eating disorders or obesity*. Int. J. Eat. Disord. 2015; 48(2): 238–248.
12. Ashworth M, Hirdes JP, Martin L. *The social and recreational characteristics of adults with intellectual disability and pica living in institutions*. Res. Dev. Disabil. 2009; 30(3): 512–520.
13. Gundogar D, Demir SB, Eren I. *Is pica the spectrum of obsessive-compulsive disorders?* Gen. Hosp. Psychiatry 2003; 25(4): 293–294.
14. Gravestock S. *Eating disorders in adults with intellectual disability*. J. Intellect. Disabil. Res. 2000; 44(Pt 6): 625–637.
15. Allison KC, Lundgren JD, O’Reardon JP, Geliebter A, Gluck ME, Vinai P. et al. *Proposed diagnostic criteria for night eating syndrome*. Int. J. Eat. Disord. 2010; 43(3): 241–247.
16. Miyaoka T, Yasukawa R, Tsubouchi K, Miura S, Shimizu Y, Sukegawa T. et al. *Successful treatment of nocturnal eating/drinking syndrome with selective serotonin reuptake inhibitors*. Int. Clin. Psychopharmacol. 2003; 18(3): 175–177.
17. Rand CS, Macgregor AM, Stunkard AJ. *The night eating syndrome in the general population and among postoperative obesity surgery patients*. Int. J. Eat. Disord. 1997; 22(1): 65–69.
18. Colles SL, Dixon JB, O’Brien PE. *Night eating syndrome and nocturnal snacking: association with obesity, binge eating and psychological distress*. Int. J. Obesity 2007; 31(11): 1722–1730.
19. Lundgren JD, Allison KC, Stunkard AJ. *Familial aggregation in the night eating syndrome*. Int. J. Eat. Disord. 2006; 39(6): 516–518.
20. Calugi S, Grave RD, Marchesini G. *Night eating syndrome in class II-III obesity: metabolic and psychopathological features*. Int. J. Obes. 2009; 33(8): 899–904.
21. Allison KC, Crow SJ, Reeves RR, West DS, Foreyt JP, DiLillo JG. et al. *Binge eating disorder and night eating syndrome in adults with type 2 diabetes*. Obesity 2007; 15(5): 1287–1293.
22. Lundgren JD, McCune A, Spresser C, Harkins P, Zolton L, Mandal K. *Night eating patterns of individuals with eating disorders: Implications for conceptualizing the night eating syndrome*. Psychiatry Res. 2011; 186(1): 103–108.
23. de Zwaan M, Roerig DB, Crosby RD, Karaz S, Mitchell JE. *Nighttime eating: a descriptive study*. Int. J. Eat. Disord. 2006; 39(3): 224–232.
24. Eiber R, Friedman S. *Correlation between eating disorders and sleep disturbances*. Encephale 2001; 27(5): 429–434.
25. Goel N, Stunkard AJ, Rogers NL, Van Dongen HP, Allison KC, O’Reardon JP. et al. *Circadian rhythm profiles in women with night eating syndrome*. J. Biol. Rhythms 2009; 24(1): 85–94.

26. Lundgren JD, Newberg AB, Allison KC, Wintering NA, Ploessl K, Stunkard AJ. *I-ADAM SPECT imaging of serotonin transporter binding in patients with night eating syndrome: a preliminary report*. *Psychiatry Res*. 2008; 162(3): 214–220.
27. Allison KC, Ahima RS, O'Reardon JP, Dinges DF, Sharma V, Cummings DE. et al. *Neuroendocrine profiles associated with energy intake, sleep, and stress in the night eating syndrome*. *J. Clin. Endocrinol. Metab*. 2005; 90(11): 6214–6217.
28. O'Reardon JP, Stunkard AJ, Allison KC. *Clinical trial of sertraline in the treatment of night eating syndrome*. *Int. J. Eat. Disord*. 2004; 35(1): 16–26.
29. Cooper-Kazaz R. *Treatment of night eating syndrome with topiramate: dawn of a new day*. *J. Clin. Psychopharmacol*. 2012; 32(1): 143–145.
30. Milano W, De Rosa M, Milano L, Capasso A. *Agomelatine efficacy in the night eating syndrome*. *Case Rep. Med*. 2013; 2013: 867650.
31. American Academy of Sleep Medicine. *International Classification of Sleep Disorders: diagnostic and coding manual*. 3rd ed. Westchester, IL: American Academy of Sleep Medicine; 2014.
32. Winkelman JW. *Clinical and polysomnographic features of sleep-related eating disorder*. *J. Clin. Psychiatry* 1998; 59(1): 14–19.
33. Winkelman JW, Herzog DB, Fava M. *The prevalence of sleep-related eating disorder in psychiatric and non-psychiatric populations*. *Psychol. Med*. 1999; 29(6): 1461–1466.
34. Schenck CH, Hurwitz TD, Bundie SR, Mahowald MW. *Sleep-related eating disorders: polysomnographic correlates of a heterogenous syndrome distinct from daytime eating disorders*. *Sleep* 1991; 14(5): 419–431.
35. Schenck CH, Hurwitz TD, O'Connor KA, Mahowald MW. *Additional categories of sleep-related eating disorders and the current status of treatment*. *Sleep* 1993; 16(5): 457–466.
36. Santin J, Mery V, Elso MJ, Retamal E, Torres C, Ivelic J. et al. *Sleep-related eating disorder: a descriptive study in Chilean patients*. *Sleep Med*. 2014; 15(2): 163–167.
37. Vetrugno R, Manconi M, Ferrini-Strambi L, Provini F, Plazzi G, Montagna P. *Nocturnal eating: sleep-related eating disorder or night eating syndrome? A videopolysomnographic study*. *Sleep* 2006; 29(7): 949–954.
38. Morgenthaler TI, Silber MH. *Amnesic sleep-related eating disorder associated with zolpidem*. *Sleep Med*. 2002; 3(4): 323–327.
39. Provini F, Albani F, Vetrugno R, Lombardi C, Plazzi G, Montagna P. *A pilot double-blind placebo-controlled trial of low-dose pramipexole in sleep-related eating disorder*. *Eur. J. Neurol*. 2005; 12(6): 432–436.
40. Winkelman JW. *Efficacy and tolerability of open-label topiramate in the treatment of sleep-related eating disorder: a retrospective case series*. *J. Clin. Psychiatry* 2006; 67(11): 1729–1734.
41. Pope HG Jr, Phillips KA, Olivardia R. *The Adonis complex: The secret crisis of male body obsession*. New York: Free Press; 2000.
42. Pope HG Jr, Gruber AJ, Choi P, Olivardia R, Phillips KA. *Muscle dysmorphia. An underrecognized form of body dysmorphic disorder*. *Psychosomatics* 1997; 38(6): 548–557.
43. Mosley PE. *Bigorexia: bodybuilding and muscle dysmorphia*. *Eur. Eat. Disord. Rev*. 2009; 17(3): 191–198.
44. Leone JE, Sedory EJ, Gray KA. *Recognition and treatment of muscle dysmorphia and related body image disorders*. *J. Athl. Train*. 2005; 40(4): 352–359.

45. Donini LM, Marsili D, Graziani MP, Imbriale M, Canella C. *Orthorexia nervosa: A preliminary study with a proposal for diagnosis and an attempt to measure the dimension of the phenomenon*. Eat. Weight Disord. 2004; 9(2): 151–157.
46. Bratman S, Knight D. *Health food junkies. Orthorexia nervosa: Overcoming the obsession with healthful eating*. New York: Broadway Books; 2000.
47. Janas-Kozik M, Zejda J, Stochel M, Brożek G, Janas A, Jelonek I. *Ortoreksja – nowe rozpoznanie?* Psychiatr. Pol. 2012; 46(3): 441–450.
48. Moroze RM, Dunn TM, Holland JC, Yager J, Weintraub P. *Microthinking about micronutrients: a case of transitions from obsessions about healthy eating to near-fatal “orthorexia nervosa” and proposed diagnostic criteria*. Psychosomatics. 2015; 56(4): 397–403.
49. Mathieu J. *What is orthorexia?* J. Am. Diet. Assoc. 2005; 105(10): 1510–1512.

Address: Natalia Szejko
Chair and Department of Psychiatry
Medical University of Warsaw
00-665 Warszawa, Nowowiejska Street 27