

Medical Science

To Cite:

Goc E, Irzyk Z, Markuszka K, Rogala J, Lepak S, Polityńska K, Ruszel D, Samuła K, Sarzyńska M, Szabat M. Bulimia nervosa – Epidemiology, diagnosis, complications and contemporary treatment methods. *Medical Science* 2026;30: e172ms3865
doi:

Authors' Affiliation:

¹Faculty of Medicine, University of Rzeszów, Rzeszów, Poland

²Clinical Provincial Hospital No. 2, Rzeszów, Poland

*Corresponding author:

Emilia Goc,

Faculty of Medicine, University of Rzeszów, Rzeszów, Poland.

E-mail address: emilia.goc@wp.pl

Peer-Review History

Received: 16 February 2026

Reviewed & Revised: 07/March2026 to 09/August/2026

Accepted: 18 August 2026

Published: 29 August 2026

Peer-review Method

External peer-review was done through double-blind method.

Medical Science

pISSN 2321-7359; eISSN 2321-7367



© The Author(s) 2026. Open Access. This article is licensed under a [Creative Commons Attribution License 4.0 \(CC BY 4.0\)](http://creativecommons.org/licenses/by/4.0/), which permits use, sharing, adaptation, distribution and reproduction in any medium or format, as long as you give appropriate credit to the original author(s) and the source, provide a link to the Creative Commons license, and indicate if changes were made. To view a copy of this license, visit <http://creativecommons.org/licenses/by/4.0/>.

Bulimia nervosa – Epidemiology, diagnosis, complications and contemporary treatment methods

Emilia Goc^{1*}, Zuzanna Irzyk¹, Katarzyna Markuszka¹, Julia Rogala¹, Sylwia Lepak¹, Kinga Polityńska¹, Dominika Ruszel¹, Klaudia Samuła¹, Martyna Sarzyńska¹, Mateusz Szabat²

ABSTRACT

Bulimia nervosa is a complex eating disorder that mainly affects young women and teenagers. It develops due to a combination of social, psychological, and biological factors. People with bulimia often have episodes of binge eating followed by efforts to prevent weight gain. Frequent vomiting and purging can lead to serious metabolic issues and changes in blood electrolyte levels. Repeated exposure to stomach acid can cause tooth decay, mouth sores, and digestive problems. Many cases are not diagnosed because people are often reluctant to seek help, which makes it difficult to determine how common the disorder is. Diagnosis relies on standard criteria from international systems such as the DSM-5 and ICD-10. This review covers what is currently known about bulimia nervosa, including how common it is, how it is diagnosed, treatment options, and related physical complications. Studies indicate that cognitive-behavioral therapy is still the most effective treatment. Sometimes, medications like selective serotonin reuptake inhibitors such as fluoxetine are also used. Early diagnosis and a team-based approach to treatment help reduce complications and improve long-term outcomes for people with bulimia nervosa.

Keywords: bulimia nervosa; eating disorders; cognitive behavioral therapy; fluoxetine; somatic complications; epidemiology

1. INTRODUCTION

Eating disorders are mental illnesses that are important in clinical practice because they often last a long time and cause many physical problems. Studies show that bulimia nervosa is a common psychiatric disorder among young women. It involves repeated episodes of overeating and strict efforts to control weight. Although awareness of bulimia nervosa is increasing, it often goes undiagnosed, which delays treatment and raises the risk of long-term health issues. This paper reviews current information about bulimia nervosa, focusing on how common it is, how it is diagnosed, treatment options, and physical complications.

The name bulimia nervosa comes from the Greek word for intense hunger. People with this disorder have repeated episodes of binge eating and then use

different ways to compensate (American Psychiatric Association, 2004). The most common compensatory behaviors are self-induced vomiting, restricting food intake, using laxatives, diuretics, thyroid medications, appetite suppressants, exercising too much, and skipping insulin doses in people with diabetes (Fairburn, 1995; Jarema, 2021).

2. REVIEW METHODS

We reviewed research using databases such as PubMed. We searched for terms like "bulimia nervosa," "bulimia treatment," "physical complications of bulimia," "how common bulimia is," and "therapy for eating disorders." We included various types of studies published in English between January 1992 and January 2026 (Figure 1). We picked articles that were relevant to how often bulimia occurs, its causes, treatments, and how well they work.

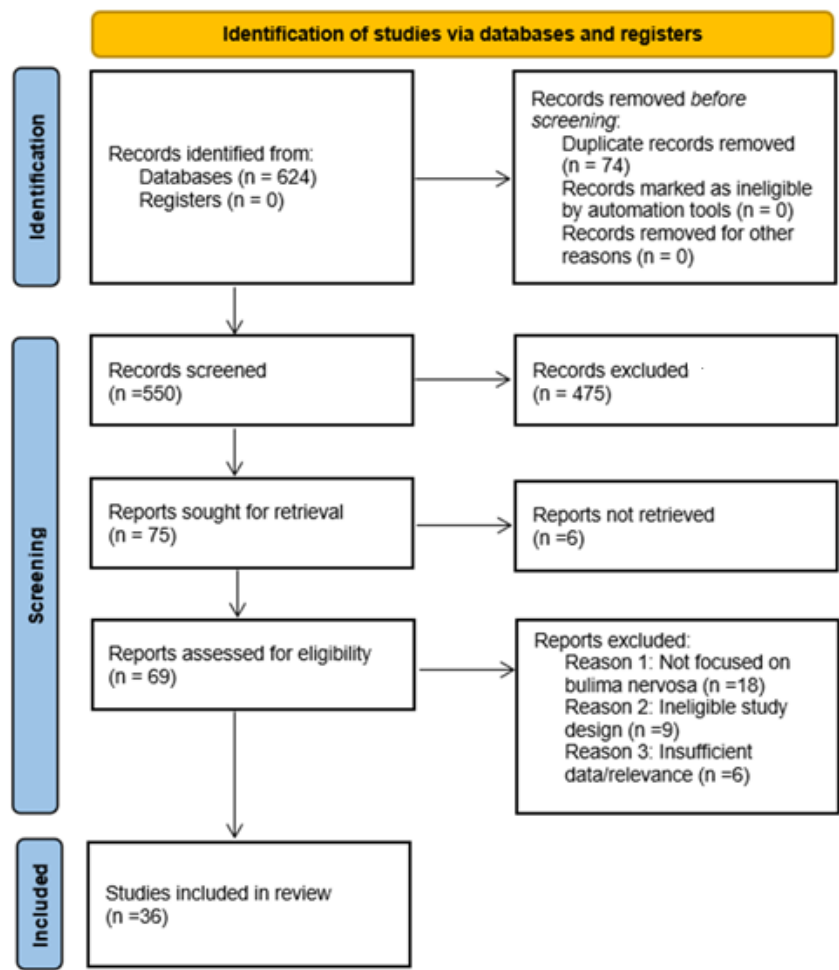


Figure 1. Flowchart for identification of studies

3. RESULTS AND DISCUSSION

Epidemiology

People with bulimia nervosa have a higher risk of death. The crude mortality rate for bulimia is about 2% per decade (Jarema, 2021). Studies in the United States show that bulimia affects about 1.3% of girls and 0.5% of boys (Smink et al., 2012). In Poland, about 3% of people have bulimia. The disorder is diagnosed much more often in women than in men, with about 90% of patients being female (Jarema, 2021; Hudson et al., 2007).

Eating disorder symptoms usually start during adolescence, and studies suggest that up to 13% of women have at least one eating disorder before age 20 (Stice et al., 2013). It is hard to know the exact rates because many people do not seek help or do not realize they have a problem (Smink et al., 2021).

Etiology

Bulimia nervosa develops from a mix of genetic tendencies and environmental factors (Culbert et al., 2015). Eating disorders are more common in families of individuals suffering from bulimia nervosa, implying a major role of genetic factors. Studies show that first-degree relatives of individuals with bulimia have an increased risk not only of eating disorders (3.4–19.8%) but also of affective disorders (9–37%), substance use disorders (8–40%), as well as personality disorders from cluster B according to DSM-IV (12%). Additionally, studies on monozygotic twins demonstrate higher concordance rates than in the general population, additionally supporting the importance of genetic predisposition. There are also indications showing a link between bulimia and a region on chromosome 10 (Wright et al., 2008).

The likelihood of developing eating disorders may increase in individuals raised in families where dieting and critical comments about body weight, body shape, or eating habits are common (Jarema, 2021). Some studies have also reported an association between higher levels of education among family members and more frequent hospitalization of girls due to eating disorders, which is explained, among other factors, by greater achievement pressure in the home environment (Maxwell et al., 2011; Alvarenga et al., 2013).

Social and cultural components also increase the risk of developing bulimia nervosa. The media play a big role by promoting thinness and sharing ways to achieve it. These messages can add social pressure and, according to studies, may raise the risk of bulimia nervosa to eight times (Keel, 2000; Martínez-González, 2003; Jarema, 2021).

Diagnosis

The diagnosis of bulimia in clinical practice is based mainly on the DSM-5 (Table 1), developed by the American Psychiatric Association, and the ICD-10 (Table 2), developed by the World Health Organization.

Table 1. Diagnostic Criteria for Bulimia Nervosa According to DSM-5

A.	Recurrent episodes of binge eating characterized by both of the following: - Consuming the amount of food within a discrete period of time, is definitely larger than what most people would eat in a similar period under similar circumstances, - a sense of lack of control over eating during the episode.
B.	Recurrent inappropriate compensatory behaviors to prevent weight gain, such as: - self-induced vomiting, - misuse of laxatives, diuretics, or other medications, - fasting, - excessive physical exercise.
C.	Engaging in binge eating and using unsuitable compensatory behaviors happens at least once a week for a duration of three months.
D.	Self-assessment is excessively swayed by body size and mass.
E.	The disturbance does not occur exclusively during episodes of anorexia nervosa.
All of the above criteria must be met for diagnosis.	

Table 2. Diagnostic Criteria for Bulimia Nervosa According to ICD-10

A.	Recurrent episodes of overeating (at least twice a week for three months), during which large amounts of food are consumed in a short period of time.
B.	Persistent preoccupation with eating and a strong desire or sense of compulsion to eat.
C.	Attempts to counteract the “fattening” effects of food by one or more of the following: - self-induced vomiting, - purging (inducing defecation), - alternating periods of starvation,

	- use of appetite suppressants, thyroid preparations, or diuretics.
D.	Self-perception as being overweight and an intense fear of gaining weight.
All of the above criteria must be met for diagnosis.	

Both classification systems highlight repeated binge eating episodes and compensatory behaviors. To meet the criteria, these behaviors must happen at least once a week for three months. The DSM-5 says that people with bulimia cannot restrict their food intake and are very focused on their weight. The ICD-10 describes a constant preoccupation with food and a strong urge to eat, and enumerates particular compensatory behaviors. Despite some differences, both systems describe bulimia in similar ways, and doctors use both for diagnosis.

Diagnostic Markers

There is no single test to diagnose bulimia nervosa. However, the effects of compensatory behaviors can cause changes in lab results that help with diagnosis, such as low levels of sodium, potassium, chloride, and calcium. Frequent diarrhea and long-lasting dehydration caused by overusing laxatives and diuretics may lead to a condition where the body becomes too acidic. On the other hand, losing stomach acid from self-induced vomiting can cause the body to become too alkaline (Jarema, 2021).

Differential Diagnosis

To rule out other conditions that cause chronic vomiting, clinicians must consider conditions such as:

- hiatal hernia,
- duodenal stenosis,
- esophageal achalasia,
- peptic ulcer disease of the stomach and duodenum,
- food allergies,

as well as infectious diseases, metabolic disorders, and poisoning (Hertl, 1993; Hetherington et al., 1994).

Similar symptoms may also occur in neurological conditions associated with increased intracranial pressure, including migraines. An important element of diagnosis is also differentiating bulimia from other eating disorders, particularly:

- anorexia nervosa, binge-eating/purging type,
- binge eating disorder (BED).

Unlike bulimia, binge eating disorder does not include compensatory behaviors like self-induced vomiting or using laxatives. BED also affects men and women more equally and is more commonly linked to being overweight or obese, while people with bulimia usually have a normal body weight (Ziółkowska and Mroczkowska, 2011; DeBate et al., 2005).

Treatment

Meta-analyses of randomized controlled trials show that cognitive behavioral therapy for bulimia (CBT-BN) is the most effective psychological treatment, especially compared to no treatment or other types of therapy (Hagan and Walsh, 2021; Yamada et al., 1996). The cognitive-behavioral approach is based on the idea that bulimia comes from unhealthy beliefs about body image, weight, and self-worth. Patients often judge themselves by their looks, which leads to distorted thinking patterns. This is known as the cognitive triad, which includes negative beliefs about:

- oneself,
- the future,
- relationships with others.

Binge-eating episodes are interpreted as acquired behaviors that reduce emotional tension. Within this system, while subsequent compensatory behaviors reinforce this mechanism (Pilecki and Józefik, 1999; Grzesiuk, 2006; Szymańska, 2002).

Studies comparing cognitive-behavioral therapy and psychoanalytic therapy show that after 5 months, 42% of CBT patients were in remission compared to 6% for psychoanalytic therapy. After 2 years, the rates were 44% for CBT and 15% for psychoanalytic therapy.

(Poulsen et al., 2014). CBT for bulimia should include at least 16 to 20 individual sessions over 4 to 5 months (Rabe-Jabłońska et al., 2008). If psychotherapy does not help enough after 6 weeks, treatment with the medicine fluoxetine at 60 mg per day is recommended (Crone et al., 2023; Palla and Litt, 1988). Fluoxetine works by increasing the amount of serotonin, a brain chemical, which helps improve mood and behavior (Fluoxetine Bulimia Nervosa Collaborative Study Group, 1992). The primary cause of the appetite reduction observed with fluoxetine is enhanced serotonergic transmission. Increased serotonin release can activate 5-HT_{2C} receptors on dopaminergic neurons in the ventral tegmental area (VTA) responsible for modulating the reward system (Yu et al., 2023). Research shows that this medication can reduce how often patients with bulimia binge eat and use compensatory behaviors. However, results are mixed. Some studies confirm its benefits, while others find no advantage over placebo and note that CBT is much more effective for achieving remission (Grilo et al., 2005). Research examines other antidepressants and antipsychotic medications in the treatment of eating disorders. Pharmaceutical companies funded some of these studies, which may introduce possible conflicts of interest and require prudent interpretation. Long-term studies (Romano et al., 2002; Lo Russo et al., 2008) suggest that ongoing fluoxetine treatment may delay relapse. However, its overall long-term effectiveness is limited and not clearly proven (Yu et al., 2023).

Complications

Treating the physical complications of bulimia should involve a team approach, including psychiatric, psychological, medical, and dental care. To reduce the risk of complications, it is important to follow specific treatment steps, such as rehydrating slowly and correcting electrolyte imbalances. Because of the risk of refeeding syndrome, calories and meal sizes should be increased gradually, starting with a low-calorie or easy-to-digest diet. To prevent compensatory behaviors early in treatment, medical staff should supervise patients during and after meals, as well as during bathroom visits. To reduce the risk of superior mesenteric artery syndrome, patients should lie on their left side after eating, limit physical activity, and rest as much as possible (Comerci, 1990). Bulimia nervosa causes many physical problems, often because of repeated vomiting and behaviors like overusing laxatives or limiting food. The mouth is often affected. Patients may have dry mouth, burning feelings, changes in taste, and thinning of the mouth lining. Tooth enamel erosion usually appears as shallow, smooth dents on the inside surfaces of the front teeth, which is a common sign. Some patients develop swollen salivary glands, especially the parotid glands. This swelling is usually not caused by infection but by ongoing irritation. Patients who make themselves vomit with their fingers may develop Russell's sign, which is inflammation and thickening of the skin on the back of the index finger. Repeated vomiting irritates the lining of the food pipe. It causes sores and bleeding. Digestive problems can also occur, such as slow stomach emptying and a lingering feeling of fullness after binge eating. Other complications can happen as well. If stomach contents are inhaled into the lungs, this can cause aspiration pneumonia (Paradowska and Sieja, 2010; Fisher et al., 2000).

Prevention

Reducing the factors that lead to unhealthy eating habits and building resources that support good mental and physical health prevent the eating disorders (Szymańska, 2002). In practice, this means stopping behaviors like restrictive eating or binge eating, encouraging a positive body image, and teaching ways to handle anxiety and emotional tension.

Identifying risk factors and building protective factors are both important. These factors can be:

- individual, related to personality traits and mental condition,
- environmental, resulting from family relationships, social pressure, or the influence of popular culture (Kubacka-Jasiecka and Mudyń, 2003; Gonzales et al., 2007).

Researchers describe three levels of prevention:

- Primary prevention targets all children and teenagers who do not have eating disorders. The goal is to encourage healthy eating, build social skills, and help them handle difficult emotions.
- Secondary prevention focuses on people who are beginning to show signs of unhealthy eating or body image issues. The aim is to stop these problems from developing into full eating disorders.
- Tertiary prevention helps people who already have eating disorders by reducing negative effects, supporting their treatment, and avoiding relapse (Ziółkowska and Mroczkowska, 2011; Kozmér et al., 2006).

4. CONCLUSION

Bulimia nervosa is an eating disorder with complex causes. It usually begins during adolescence and is more common in women. The disorder can cause many physical problems, including changes in the mouth and metabolic issues. Cognitive-behavioral therapy is the most effective treatment for bulimia nervosa. Early diagnosis of bulimia nervosa and a team-based treatment approach can lower the risk of complications and improve patient outcomes.

Acknowledgments

We thank all co-authors and institutional staff for their support during the preparation of this review.

Authors' Contributions

Emilia Goc - contributed to the study conception and design, conducted the literature search and data analysis, wrote the manuscript, and approved the final version for publication.

Zuzanna Irzyk - conducted the literature search and data analysis.

Katarzyna Markuszka- conducted the literature search and data analysis.

Julia Rogala- conducted the literature search and data analysis.

Julia Rogala- conducted the literature search and data analysis.

Sylwia Lepak- conducted the literature search and data analysis.

Kinga Polityńska- conducted the literature search and data analysis.

Dominika Ruszel - conducted the literature search and data analysis.

Klaudia Samuła - conducted the literature search and data analysis.

Martyna Sarzyńska - conducted the literature search and data analysis.

Mateusz Szabat - conducted the literature search and data analysis.

Informed consent

Not applicable.

Ethical approval

Not applicable. This article does not contain any studies with human participants or animals performed by any of the authors.

Funding

This research did not receive any external funding like specific grant from funding agencies in the public, commercial, or nonprofit sectors.

Conflict of interest

The authors declare that they have no conflicts of interest, competing financial interests or personal relationships that could have influenced the work reported in this paper.

Data and materials availability

All data associated with this study will be available based on the reasonable request to corresponding author.

REFERENCES

- Alvarenga MDS, Lourenço BH, Philippi ST, Scagliusi FB. Disordered eating among Brazilian female college students. *Cadernos de Saúde Pública, Fundação Oswaldo Cruz (Fiocruz), Rio de Janeiro, Brazil* 2013;29(5):879–888. doi: 10.1590/S0102-311X2013000500006
- American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders: DSM-IV-TR (Text Revision)*. American Psychiatric Association Publishing, Washington, DC, USA 2004.
- Comerci GD. Medical complications of anorexia nervosa and bulimia nervosa. *Med Clin North Am* 1990;74(5):1293–1310. doi: 10.1016/S0025-7125(16)30517-X
- Crone C, Fochtmann LJ, Attia E, Boland R, Escobar J, Fornari V, Golden N, Guarda A, Jackson-Triche M, Manzo L, Mascolo

- M, Pierce K, Riddle M, Seritan A, Uniacke B, Zucker N, Yager J, Craig TJ, Hong SH, Medicus J. The American Psychiatric Association Practice Guideline for the Treatment of Patients with Eating Disorders. *Am J Psychiatry*. 2023;180(2):167-171. doi: 10.1176/appi.ajp.23180001.
5. Culbert KM, Racine SE, Klump KL. Research review: What we have learned about the causes of eating disorders – a synthesis of sociocultural, psychological, and biological research. *J Child Psychol Psychiatr* 2015;56(11):1141–1164. doi:10.1111/jcpp.12441.
6. DeBate RD, Tedesco LA, Kerschbaum WE. Knowledge of oral and physical manifestations of anorexia and bulimia nervosa among dentists and dental hygienists. *J Dent Edu* 2005;69(3):346–354.
7. Fairburn CG. *Overcoming Binge Eating*. Guilford Press, New York, USA 1995.
8. Fisher M, Simpser E, Schneider M. Hypophosphatemia secondary to oral refeeding in anorexia nervosa. *Int J Eating Disorders*. 2000;28(2): 181–187. doi: 10.1002/1098-108X(200009)28:2<181::AID-EAT7>3.0.CO;2-K.
9. Fluoxetine in the treatment of bulimia nervosa. A multicenter, placebo-controlled, double-blind trial. Fluoxetine Bulimia Nervosa Collaborative Study Group. *Arch Gen Psychiatry*. 1992 Feb;49(2):139-47. doi: 10.1001/archpsyc.1992.01820020059008
10. Gonzales A, Kohn MR, Clarke SD. Eating disorders in adolescents. *Aust. Family Phys*. 2007; 36 (8): 614–619. doi: 10.3316/informit.357461588421464.
11. Grilo CM, Masheb RM, Wilson GT. Efficacy of cognitive behavioral therapy and fluoxetine for the treatment of binge eating disorder: a randomized double-blind placebo-controlled comparison. *Biol Psychiat* 2005; 57 (3): 301–309. doi:10.1016/j.biopsych.2004.11.002.
12. Grzesiuk L. *Psychotherapy: Practice – An Academic Textbook*. Eneteia – Psychology and Culture Publishing House. Warszawa, Polska, 2006; 381–453.
13. Hagan KE, Walsh BT. State of the Art: The Therapeutic Approaches to Bulimia Nervosa. *Clin Therapeutics* 2021;43(1): 40–49. doi:10.1016/j.clinthera.2020.10.012.
14. Hertl M. *Differential Diagnosis in Pediatrics*. Trans. P. Albrecht. Medical Publishing House, Warszawa, Polska 1993.
15. Hetherington MM, Altemus M, Nelson ML, Bernat AS, Gold PW. Eating behavior in bulimia nervosa: multiple meal analyses. *Am J Clin Nutrition* 1994;60(6):864–873.
16. Hudson JL, Hiripi E, Pope HG Jr, Kessler RC. The prevalence and correlates of eating disorders in the National Comorbidity Survey Replication. *Biol Psychiat* 2007;61(3):348–358. doi: 10.1016/j.biopsych.2006.03.040.
17. Jarema M. *Psychiatria. Textbook for medical students*. PZWL Medical Publishing, Warszawa, Polska 2021:436–443.
18. Keel PK. American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders*, 4th ed., Text Revision (DSM-IV-TR). American Psychiatric Association Publishing, Washington, DC, USA 2000.
19. Kozmér S, O'Rourke C, Lawrence NS, Smith JR, Beurden SB. Identification and Management of Binge-eating disorder and Bulimia Nervosa in Primary Care Settings: A Qualitative Systematic Review of Health Care Professionals' and Patients' Perceptions. *Int J Eat Disorders* 2026;59(1):16-34.
20. Kubacka-Jasiecka D, Mudyń K. *Crisis – Intervention and Psychological Support. New Perspectives and Possibilities*. Adam Marszałek Publishing House. Toruń, Polska 2003:17–27.
21. Lo Russo L, Campisi G, Di Fede O, Di Liberto C, Panzarella V, Lo Muzio L. Oral manifestations of eating disorders: a critical review. *Oral Diseases* 2008;14(6):479–484. doi: 10.1111/j.1601-0825.2007.01422.x
22. Martínez-González MA, Gual P, Lahortiga F, Alonso Y, Irala-Estévez J, Cervera S. Parental factors, mass media influences, and the onset of eating disorders in a prospective population-based cohort. *Pediatrics, Am Academy Pediatrics* 2003;111(2):315–320. doi:10.1542/peds.111.2.315.
23. Maxwell M, Thornton LM, Root TL, Pinheiro AP, Strober M, Brandt H i wsp. Life beyond the eating disorder: Education, relationships, and reproduction. *Int J Eating Disorders* 2011;44(3):225–232. doi:10.1002/eat.20804.
24. Palla B, Litt IF. Medical Complications of Eating Disorders in Adolescents. *Pediatrics*. 1988;81(5):613–623. doi:10.1542/peds.81.5.613.
25. Paradowska A, Sieja A. *Oral Imaging in Eating Disorders*. Department of Jaw Orthopaedics and Orthodontics, Wrocław Medical University. *Gastroenterologia Polska*, Poznań, 2010; 17 (2): 111–115.
26. Pilecki M, Józefik B. (red.). *Anorexia and Bulimia Nervosa*. Jagiellonian University Publishing House, Kraków, Polska, 1999.
27. Poulsen S, Lunn S, Daniel SI, Folke S, Mathiesen BB, Katznelson H, Fairburn CG. A randomized controlled trial of psychoanalytic psychotherapy or cognitive-behavioral therapy for bulimia nervosa. *Am J Psychiat* 2014;171(1):109–116. doi:10.1176/appi.ajp.2013.12121511.
28. Rabe-Jabłońska J, Pawełczyk T, Żechowska C, Jarema M. *Standards of Treatment for Eating Disorders*. *Psychiat Clin Psychol* 2008;8(1):20–40.
29. Romano SJ, Halmi KA, Sarkar NP, Koke SC, Lee JS. A placebo-controlled study of fluoxetine in continued treatment

- of bulimia nervosa after successful acute fluoxetine treatment. *Am J Psychiat* 2002;159(1):96–102.
30. Smink FRE, van Hoeken D, Hoek HW. Epidemiology of eating disorders: Incidence, prevalence and mortality rates. *Curr Psychiat Rep* 2012;14(4):406–414. doi:10.1007/s11920-012-0282-y.
31. Stice E, Marti CN, Rohde P. Prevalence, incidence, impairment, and course of the proposed DSM-5 eating disorder diagnoses in an 8-year prospective community study of young women. *J Abnor Psychol Am Psychol Assoc* 2013; 122(2):445–457. doi:10.1037/a0030679.
32. Szymańska B. Prevention in the local community. National Bureau for Drug Prevention. Warszawa 2002; 49-67
33. Wright P, Stern J, Phelan M. *Psychiatria. Sedno. Tom 1.* Elsevier Urban & Partner, Wrocław, Polska 2008.
34. Yamada Y, Fushimi H, Inoue T, Nishinaka K, Kameyama M. Anorexia nervosa with recurrent hypoglycemic coma and cerebral hemorrhage. *Int Med* 1996;35(7):560–563. doi: 10.2169/internalmedicine.35.560
35. Yu S, Zhang Y, Shen C, Shao F. Efficacy of pharmacotherapies for bulimia nervosa: a systematic review and meta-analysis. *BMC Pharmacol Toxicol*, 2023;24(1):72. doi:10.1186/s40360-023-00713-7.
36. Ziółkowska B, Mroczkowska D. *Bulimia from A to Z. A compendium of knowledge for parents, teachers, and educators.* Difin, Warszawa, Polska, 2011.