



ORIGINAL ARTICLE

Obesity and depression - a bidirectional relationship and clinical implications

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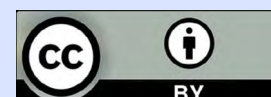
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ABSTRAKT

Background: Obesity has become a global civilization-related disease and constitutes an independent risk factor for numerous somatic disorders. Obesity can contribute to depression through low self-esteem, body image concerns, and stigmatization, while depression and chronic stress may promote obesity through emotional eating and binge eating. Both conditions share biological mechanisms, including HPA axis dysregulation, elevated cortisol levels, and chronic inflammation. Effective management requires an interdisciplinary approach that combines weight-loss interventions with psychological treatment. Weight reduction is associated with improved mood and quality of life.

Aim: The aim of this narrative review is to analyze and synthesize the current state of knowledge regarding the relationship between obesity and depression. The paper seeks to identify the biological and psychological mechanisms linking both conditions, as well as to assess the impact of obesity on the risk of depression and, conversely, the impact of depressive disorders on the development and course of obesity.

Material and methods: A literature review of scientific publications available in databases (including Google Scholar) was conducted, analyzing data on adolescents and adults with respect to pathophysiological and behavioral mechanisms. The review includes 24 articles published between 2006 and 2025.

Results: Individuals with obesity have a 55% higher risk of developing depression, whereas patients with depression are 58% more likely to develop obesity. The prevalence of depression in the population of individuals with obesity is increased, and a modifying effect of sex is observed. Key mechanisms include dysregulation of the HPA axis, chronic inflammation, and disturbances in neurotransmission.

Conclusions: Effective treatment requires an integrated approach combining weight reduction with psychological interventions. Treatment of obesity may lead to an improvement in mood.

KEYWORDS

obesity, depression, eating disorders

Introduction

Obesity has become a highly prevalent disease and currently represents one of the most common indirect causes of death ^[1]. It is defined as excessive accumulation of adipose tissue with a body mass index (BMI) exceeding 30 kg/m² ^[2,3]. Obesity is a chronic disease with no tendency for spontaneous remission and constitutes an independent risk factor for more than 200 disorders, including cardiovascular diseases and type 2 diabetes ^[1-6]. In parallel, depression represents a serious public health problem. It is the most common mental disorder ^[5]. Regardless of etiology, both obesity and depression are recognized as factors that increase mortality ^[4]. In recent years, increasing attention has been paid to the mutual, bidirectional relationships between these conditions, whereby obesity promotes the development of depression, and depression constitutes a predictive factor for the development of obesity ^[4,5]. A full understanding of the mechanisms underlying this mutual correlation is crucial for the development of effective, interdisciplinary therapeutic strategies ^[4,5].

1. Epidemiology and clinical significance

Contemporary scientific literature indicates the existence of a strong, bidirectional relationship between obesity and depression ^[7-10]. Meta-analyses of longitudinal studies show that individuals with obesity have a 55% higher risk of developing depression, whereas patients with depression are 58% more likely to develop obesity ^[7,9,10]. The prevalence of depression in the population of individuals with obesity is increased, while a modifying effect of sex is observed ^[4,5]. Studies suggest that women with obesity are more prone to depression and anxiety disorders than men with obesity ^[5,11,12]. In the authors' own studies conducted in a group of patients with BMI ≥ 40 kg/m², a significantly higher prevalence of severe depression was found compared to individuals with class I and II obesity ^[4]. The coexistence of both conditions increases the risk of mortality, as both depression and obesity are independent risk factors for cardiovascular diseases ^[4]. Studies using pseudo-panel analysis confirm that an increase in BMI significantly increases the number of days with depressive symptoms in the population ^[13].

2. Obesity as a risk factor for depression

The negative impact of obesity on mental health is explained primarily by psychological mechanisms ^[1,4]. Individuals with obesity often struggle with

stigmatization and discrimination in the social environment, which ultimately leads to psychological distress ^[4,6,11,14-17]. Stigmatization of depression also plays an important role ^[7,8,13]. The consequences of stigmatization may include lowered self-esteem, social isolation, and lack of acceptance of one's appearance ^[1,2,4,5,16]. Such stigmatization is based on deeply rooted and unfair stereotypes. These stereotypes attribute laziness, a lack of willpower, low intelligence, and a lack of self-discipline to individuals living with obesity ^[6]. This perception leads to a phenomenon of implicit discrimination, in which patients are judged by others (including, for example, employers) as less competent and less well-mannered ^[16]. Women with obesity frequently demonstrate strong dissatisfaction with their body image ^[16]. Such dissatisfaction often develops into feelings of helplessness and low self-efficacy. This hinders the process of changing eating habits and is further reinforced by previous unsuccessful attempts at weight reduction ^[2]. Women with obesity tend to devalue their own body image, which results in avoiding direct interpersonal contact and preferring remote forms of communication ^[16]. In the adolescent population, lack of body acceptance was significantly associated with the occurrence of depression, particularly in girls (even those with normal body weight) ^[18]. Such children often suffer from low self-esteem and discrimination from their peer group. Some of them attempt to cope with these negative experiences through excessive food consumption, which leads to a worsening of their condition ^[11]. Lack of body acceptance correlates with depressive symptoms and lower self-esteem ^[16]. Negative social attitudes cause affected individuals to begin believing that these harmful opinions about them are justified. In this way, they deepen their own suffering and isolation ^[6]. Severe obesity significantly worsens quality of life through numerous physical and psychological limitations. It may lead to the development of serious psychopathology, which can result in suicidal thoughts. The risk of such thoughts is significantly higher than in individuals with a high BMI ^[12]. Effective obesity treatment gives patients hope for a better future—one free from stigmatization—and leads to a rapid improvement in mood and self-image ^[15].

3. Depression as a risk factor for obesity

Depression may lead to the development of obesity due to changes in eating behaviors ^[4,5]. This phenomenon is particularly evident in the presence of so-called „reverse vegetative symptoms.“ These symptoms are characteristic of atypical depression,

in which patients experience an increased appetite (hyperphagia). This leads to rapid weight gain, often exceeding 5 kg within a period of three months [23]. Studies conducted among young women indicate that the severity of depressive symptoms is closely associated with BMI. Unhealthy dietary changes become a form of non-pharmacological attempt to cope with a lowered mood [5]. A key role in this context is played by emotional eating, which is defined as a tendency to overeat in response to negative emotions such as sadness, stress, or anxiety [2,5]. The biological explanation for this mechanism lies in the effect of carbohydrate consumption on increasing serotonin levels in the brain and stimulating the release of endogenous opioids. This allows for a temporary improvement in mood and a calming effect [4]. Patients subconsciously perceive food as a form of „reward,” which, under conditions of chronic stress, leads to neurobiological adaptation and the development of compulsive overeating [14]. In affected individuals, eating becomes a compensatory mechanism aimed at providing short-term pleasure and reducing depressive symptoms or relieving psychological tension [1,2,4,5,20]. This mechanism may in turn lead to compulsive eating and, consequently, to obesity, thereby creating a vicious cycle [1,2,4,14].

3.1. Eating disorders

Eating disorders often coexist with depression and obesity. Binge eating disorder (BED) affects approximately 5-30% of individuals with obesity [2,19,20] and consists of consuming excessive amounts of food in an uncontrolled manner. The occurrence of emotional eating was significantly associated with BMI level and depressive symptoms in a group of young women [5]. Patients often use high-energy „comfort foods” (rich in saturated fats and sugars) as a form of self-treatment for stress, which, however, exacerbates metabolic dysfunctions [8,21,22].

3.2. Specific types of depression

The occurrence of specific types of depression shows a stronger association with obesity. Atypical depression, characterized by increased appetite and weight gain [7,10,21], is particularly associated with overweight and obesity [4,23]. In the clinical picture of atypical depression, „reversed vegetative symptoms” are observed, including hyperphagia, weight gain, and hypersomnia [4]. In individuals with obesity suffering from depression, atypical symptoms occur more frequently and are more severe [4,23]. In patients with recurrent depression and bipolar disorder, a

high prevalence of metabolic syndrome (MetS), reaching 24-26%, has been observed, independent of the pharmacotherapy used [24]. The severity of increased appetite and weight gain correlates with waist circumference and BMI in the group of individuals with obesity. Waist circumference shows a positive correlation with the severity of symptoms measured by the Hamilton Depression Rating Scale (HAM-D) [24]. This suggests an association with abdominal obesity [23], which, according to increasing evidence, is a key predictor of anxiety and mood disorders [21,22].

4. Pathophysiological mechanisms linking obesity and depression

The associations between depression and obesity share partially overlapping pathogenesis [4]. The common background of both conditions is based on several key metabolic processes:

4.1. Dysregulation of the HPA axis and stress

Chronic stress leads to excessive activation of the hypothalamic-pituitary-adrenal (HPA) axis, resulting in increased cortisol secretion [4,5]. This mechanism is primarily based on the increased release of hypothalamic corticotropin-releasing hormone (CRH) and vasopressin (AVP). These hormones act synergistically and intensify the secretion of adrenocorticotrophic hormone (ACTH) by the pituitary gland [4]. The corticotropin-releasing hormone produced exhibits psychotropic properties that may trigger anxiety responses, directly provoke appetite disturbances, and disrupt sleep architecture [23]. Hypercortisolemia promotes the accumulation of adipose tissue, particularly visceral fat [4,23], and insulin resistance [7-9,21,22]. Excess cortisol affects the level of satisfaction derived from eating by acting on neuroendocrine mediators (e.g., leptin and insulin). Under conditions of persistent stress, patients are more likely to consume high-calorie foods [4]. Chronic elevation of cortisol levels also increases the density of glucocorticoid receptors within visceral adipose tissue (VAT). This promotes the accumulation of visceral fat and perpetuates metabolic disturbances [23]. In turn, visceral adipose tissue secretes factors that affect the adrenal glands, which may lead to increased cortisol production [4]. This phenomenon is further amplified by a reduction in the concentration of cortisol-binding globulin (CBG) [4]. Studies have shown that women with visceral obesity exhibit significantly greater reactivity of the hypothalamic-pituitary-adrenal (HPA) axis to stress-related stimuli than individuals with gynoid obesity. This promotes further fat accu-

mulation in the abdominal region, face, and neck ^[23]. Excessive activation of the HPA axis is therefore one of the mechanisms linking obesity and depression ^[4].

4.2. Inflammation and adipokines

Adipose tissue, particularly visceral adipose tissue (VAT), is an active endocrine organ and a source of pro-inflammatory cytokines (such as IL-6 and TNF- α) ^[4,5] and C-reactive protein (CRP), which penetrates the brain and influences neuronal circuits responsible for emotions and motivation ^[7,10,21,22]. Increased inflammatory activation (elevated IL-6 and CRP levels) is observed in both obesity and depression, constituting another link between the pathogenesis of both diseases ^[4,5]. Leptin, a hormone secreted by adipocytes, provides information energy resources to the brain ^[1,5] and is associated with BMI and depressive symptoms ^[5]. Leptin resistance (the satiety hormone) and reduced adiponectin levels are closely related to the pathophysiology of both conditions ^[7,21,22].

4.3. Neurotransmission

In obesity and depression, disturbances are observed in the secretion and action of neurotransmitters such as serotonin, noradrenaline, and dopamine, which play a key role in the regulation of mood and appetite ^[4,5,19]. One of the underlying mechanisms of this phenomenon is a reduction in the number of serotonergic receptors in the brain and impaired sensitivity to the action of this neurotransmitter. This is observed in both individuals with

4.4. Gut microbiota

Diet-induced changes in the composition of the gut microbiota (dysbiosis) may modulate anxiety and depressive behaviors by affecting the permeability of the blood-brain barrier ^[21]. A fundamental role in this process is played by the bidirectional communication between the enteric nervous system and the central nervous system. The intestines contain approximately 100 million nerve cells that continuously transmit information influencing both the physical and psychological aspects of human functioning ^[27]. Nerve fibers located in the intestines can sense and respond to emotions and are also capable of regulating many vital functions under the influence of stress ^[27]. Contemporary research confirms that an unhealthy gut microbiota may be directly associated with a lower mood. Impairment of the intestinal bar-

rier constitutes an important mechanism that increases susceptibility to depressive states by intensifying the interaction between inflammatory processes and the brain ^[27]. Maintaining a healthy gut microbiota is an important preventive objective. A properly balanced diet and the responsible use of antibiotics are essential in this regard, as antibiotics can promote weight gain by destroying beneficial bacteria ^[6]. In patients with obesity and coexisting mood disorders, stabilization of the gut microbiota may become a key factor in breaking this vicious cycle. Inflammation will no longer be exacerbated by psychological symptoms, and psychological stress will cease to impair the functioning of the digestive system ^[27].

5. Clinical implications, interventions, and prognosis

The coexistence of obesity and depression is associated with poorer therapeutic outcomes ^[10]. High BMI may weaken the response to standard antidepressant treatment (e.g., fluoxetine) ^[10]. For this reason, a multidisciplinary approach is recommended, combining psychotherapy, dietary modification, and pharmacological interventions ^[7,9,21]. Effective therapy must be multidisciplinary, simultaneously addressing the treatment of obesity and depression ^[4,5].

5.1. Behavioral and psychological interventions

Cognitive-behavioral therapy (CBT) is the cornerstone of obesity treatment, particularly when combined with diet and physical activity ^[3,14]. CBT is effective in weight reduction, especially when it focuses on reducing uncontrolled and emotional eating ^[3,5]. Interventions should teach patients to recognize and cope with emotional eating (EE) ^[5,6].

5.2. Pharmacological and surgical treatment

Pharmacotherapy is indicated when lifestyle modifications are ineffective, especially in individuals with BMI ≥ 30 kg/m² or BMI ≥ 27 kg/m² with severe complications including hypertension, dyslipidemia, obstructive sleep apnea, cardiovascular disease, prediabetes/diabetes and others ^[3,6]. Modern medications (GLP-1 receptor agonists) result in significant weight reduction (>10-15%) ^[3,6] and protect against cardiovascular events ^[1,3,6]. Among currently available therapies, semaglutide (GLP-1 receptor agonist) and tirzepatide (dual GIP and GLP-1 receptor agonist) demonstrate the greatest efficacy and are recommended as first-line pharmacological treatment options

for patients with obesity ^[25,26]. Retatrutide is a new promising triple receptor agonist, targeting GLP-1, GIP and glucagon receptors. Clinical studies suggest that retatrutide can induce weight loss comparable to that observed following bariatric surgery, highlighting its potential as a highly effective pharmacological treatment for obesity ^[25]. In severe cases (morbid obesity), bariatric surgery remains one of the recognized therapeutic methods with proven long-term effectiveness in weight reduction. The most commonly performed bariatric procedures include sleeve gastrectomy (SG), Roux-en-Y gastric bypass (RYGB), one-anastomosis gastric bypass (OAGB), and adjustable gastric banding (AGB) ^[3,6]. Treatment of obesity (both surgical and pharmacological) may lead to improvement in mood and reduction of depressive symptoms ^[4,15,18].

6. Discussion

The relationship between depression and obesity is bidirectional in nature ^[5]. Contemporary medical knowledge indicates that obesity promotes the development of depressive mood, while the presence of depression simultaneously constitutes a significant risk factor for the development of obesity. This suggests the existence of a partially overlapping pathogenesis underlying both disorders ^[23]. Obesity increases the risk of depression by approximately 55% and depression increases the risk of obesity by 58% ^[7,9,10]. Data from meta-analyses emphasize that the coexistence of these two conditions significantly reduces patients' quality of life. It also increases the risk of mortality from cardiometabolic causes ^[5]. The key behavioral link is emotional eating (EE), which constitutes a form of „self-medication” of negative emotions and leads to uncontrolled weight gain ^[5,20]. Clinically significant is atypical depression, which shows a strong correlation with abdominal obesity, particularly in bipolar disorder ^[4,23]. In the psychosocial dimension, obesity induces depression through stigmatization and lack of body acceptance, which primarily affects women and adolescents ^[4,16,18]. The biological basis of both conditions includes dysregulation of the HPA axis (excess cortisol) and chronic inflammation induced by adipose tissue ^[4,5,7-9,13]. Effective therapy requires an integrated approach. The foundation of treatment must be an interdisciplinary team of specialists that incorporates pharmacotherapy, dietary habit modification, and psychological support. This approach enables patients to regain a sense of self-efficacy ^[6]. Weight reduction significantly improves mood, while psychotherapy focused on emotion regulation and modification of eating ha-

bits is essential for the durability of treatment effects ^[3,5,6]. Among patients undergoing surgical treatment for pathological obesity, a rapid reduction in both the frequency and severity of depressive symptoms, as measured by the Beck Depression Inventory, has been observed. This translates into a tangible improvement in self-image and future outlook ^[15].

7. Conclusions

The relationship between depression and obesity is strong, bidirectional, and requires simultaneous clinical attention and care. Obesity contributes to the development of depression mainly through psychosocial mechanisms. In contrast, depression leads to obesity primarily through behavioral changes, such as emotional eating ^[4,5]. Atypical depressive symptoms (hypersomnia, hyperphagia) are more strongly associated with obesity, including abdominal obesity, particularly in patients with bipolar disorder ^[23]. Complex pathophysiological mechanisms, including HPA axis dysfunction and chronic inflammation (cytokines and adipokines), constitute the biological basis of the coexistence of both conditions ^[4,5]. Effective weight reduction achieved through integrated therapy leads to significant improvement in mental health parameters and a reduction in depressive symptoms ^[4,6,15,18].

8. Summary

Obesity and depression are conditions whose coexistence entails serious health consequences as well as increased mortality risk ^[4]. Due to their complex, bidirectional etiology, treatment must be comprehensive and aimed at simultaneously addressing excessive body weight and accompanying mental disorders ^[4,5]. The implementation of non-pharmacological interventions is crucial, including lifestyle modification, physical activity, and dietary changes ^[3], additionally supported by behavioral interventions (e.g., CBT) ^[6]. In the absence of sufficient effects, pharmacotherapy (e.g., GLP-1 receptor agonists) is highly effective and may provide numerous benefits, including psychological ones ^[5,6]. In patients with morbid obesity, bariatric surgery is used ^[3,6] and demonstrates a positive impact on mental health ^[15]. Active screening for depression in patients with obesity and vice versa is necessary for early diagnosis and improved prognosis ^[5].

References

- Knyszyńska A., Bażydło M., Zabielska P., Karakiewicz B., Lubkowska A. Body composition and depression in people with metabolic syndrome aged over 55 years. *Family Medicine & Primary Care Review*. 2016;18(2):128-131. doi: <https://doi.org/10.5114/fmper.2016.43614>.
- Jaruć A., Wierusz-Wysocka B., Bogdański P. Psychological aspects of eating and excessive body weight. *Farmacja Współczesna*. 2011;4:119-126. https://www.akademiamedycyny.pl/wp-content/uploads/2016/05/201103_Farmacja_006.pdf
- Kołakowska K. Obesity pandemic — in search of the most effective treatment methods. *Biuletyn Główniej Biblioteki Lekarskiej*. 2024;383:143-156. doi: <https://doi.org/10.2478/bgbl-2024-0028>.
- Olszanecka-Glinianowicz M. Depression - cause or effect of obesity? *Endokrynologia, Otyłość i Zaburzenia Przemiany Materii*. 2008;4(2):78-85. <https://journals.viamedica.pl/eoizpm/article/viewFile/26040/20850>
- Suwała J., Skibińska M., Łojko D., Bogdański P. The assessment of selected factors associated with the co-occurrence of overweight/obesity and symptoms of depression in a group of young women. *Neuropsychiatria i Neuropsychologia*. 2024;19(1-2):1-8. doi: <https://doi.org/10.5114/nan.2024.141884>.
- Bąk-Sosnowska M., Białkowska M., Bogdański P., Chomiuk T., Dobrowolski P., Gałązka-Sobotka M., Holecki M. Clinical recommendations for the management of patients with obesity 2024. Position of the Polish Society for the Treatment of Obesity. *Medycyna Praktyczna*. 2024;1:1-116. <https://bazawiedzy.wsb.edu.pl/info/article/AWSBbb7adb5556154d60b424b92491392764/>
- Borgland S.L. Can treatment of obesity reduce depression or vice versa? *Journal of Psychiatry and Neuroscience*. 2021;46(4):E411-E414. doi: <https://doi.org/10.1503/jpn.210036>.
- Atlantis E., Goldney R.D., Wittert G.A. *Obesity and depression or anxiety*. *BMJ*. 2009;339:b3868. doi: <https://doi.org/10.1136/bmj.b3868>.
- Tzenios N., Tzanios M.E., Chahine M., Jamal P.O.B. The complex relationship between obesity and depression. *Specialty Journal of Medical and Academic Other Life Sciences*. 2023;1(3). doi: <https://doi.org/10.58676/sjmas.v1i3.22>.
- Villagrasa Blasco B., García-Jiménez J., Bodoano I., Gutiérrez-Rojas L. Obesity and depression: its prevalence and influence as a prognostic factor. *A systematic review*. *Psychiatry Investigation*. 2020;17(8):715-724. doi: <https://doi.org/10.30773/pi.2020.0099>.
- Joško-Ochojska J., Lizónczyk I. Occurrence of depression in overweight and obese adolescents. *Hygeia Public Health*. 2014;49(4):690-695. <http://www.h-ph.pl/pdf/hyg-2014/hyg-2014-4-690.pdf>
- Derkacz M., Chmiel-Perzyńska I., Marczewski K. Obesity as an interdisciplinary medical problem. Part II. Effect on human health. *Medycyna Ogólna*. 2010;16(2):175-183. <https://www.monza.pl/Otylosc-jako-interdyscyplinarny-problem-medyczny-Czesc-II-Wplyw-otylosci-na-zdrowie-czlowieka,73203,0,2.html>
- Ha H., Han C., Kim B. Can obesity cause depression? A pseudo-panel analysis. *Journal of Preventive Medicine and Public Health*. 2017;50(4):262-267. doi: <https://doi.org/10.3961/jpmph.17.067>.
- Pietrzykowska E., Wierusz-Wysocka B. Psychological aspects of overweight, obesity and slimming. *Polski Merkuriusz Lekarski*. 2008;24(143):472-476. <https://janiszewska.com/wp-content/uploads/2016/10/Psychologiczne-aspekty-nadwagi-i-otylosci.pdf>
- Kosatka D., Sobów T., Strzelczyk J. Frequency and severity of depression in obese individuals after surgical treatment of obesity using the RYGB method. *Wiadomości Lekarskie*. 2006;59(7-8):477-480.
- Brytek-Matera A., Charzyńska E. Cognitive and behavioral determinants of eating disorders in women with obesity. *Endokrynologia, Otyłości i Zaburzenia Przemiany Materii*. 2009;5(2):45-50. <https://journals.viamedica.pl/eoizpm/article/download/26016/20826>
- Bertrandt K. Self-assessment vs. quality of life and occurrence of depression symptoms among overweight and obese persons in comparison to persons of normal weight. *Problemy Higieny i Epidemiologii*. 2011;92(4):783-787. <http://www.phie.pl/pdf/phe-2011/phe-2011-4-783.pdf>
- Gębska-Kurczewska A., Wysocki M.J., Supranowicz P., Supranowicz T. Depression vs. overweight and perceived body appearance among adolescents living in suburban areas. *Roczniki Państwowego Zakładu Higieny*. 2011;62(4):427-431. <https://bibliotekanauki.pl/articles/876964.pdf>
- Kalinowski J., Kokociński S., Peszko M., Owecki M. The relationship of compulsive eating with obesity. *Postępy Biologii Komórki*. 2025;52(2):41-54. doi: <https://doi.org/10.59674/pbk25>.
- Bąk-Sosnowska M. Eating disorders accompanying obesity. *Forum Zaburzeń Metabolicznych*. 2010;1(2):92-99. https://journals.viamedica.pl/forum_leczenia_otylosci/article/view/28727

21. Fulton S., Decarie-Spain L., Fioramonti X., Guiard B., Nakajima S. The menace of obesity to depression and anxiety prevalence. *Trends in Endocrinology and Metabolism*. 2022;33(1):18-35. doi: <https://doi.org/10.1016/j.tem.2021.10.005>.
22. Hryhorczuk C., Sharma S., Fulton S.E. Metabolic disturbances connecting obesity and depression. *Frontiers in Neuroscience*. 2013;7:177. doi: <https://doi.org/10.3389/fnins.2013.00177>.
23. Buzuk G.A. Investigation of the relationship of overweight and obesity with the clinical picture of selected types of depression [doctoral dissertation]. Poznan: Uniwersytet Medyczny im. Karola Marcinkowskiego w Poznaniu; 2014. <https://www.wbc.poznan.pl/Content/347497/index.pdf>
24. Agarwal A., Agarwal M., Garg K., Dalal P.K., Trivedi J.K., Srivastava J.S. Metabolic syndrome and central obesity in depression: a cross-sectional study. *Indian Journal of Psychiatry*. 2016;58(3):281-286. doi: <https://doi.org/10.4103/0019-5545.192021>.
25. Katsi V., Koutsopoulos G., Fragoulis C., Dimitriadis K., Tsioufis K. Retatrutide-A Game Changer in Obesity Pharmacotherapy. *Biomolecules*. 2025;15(6):796. Published 2025 May 30. doi: <https://doi.org/10.3390/biom15060796>
26. Jastreboff AM, Aronne LJ, Ahmad NN, et al. Tirzepatide Once Weekly for the Treatment of Obesity. *N Engl J Med*. 2022;387(3):205-216. doi: <https://doi.org/10.1056/NEJMoa2206038>
27. Filanowicz, M., i Goldyn, A. (2023). Stany depresyjne w chorobie Lesniowskiego-Crohna. W: B. Jankowiak, B. Kowalewska i E. Krajewska-Kulak (red.), Promocja, edukacja zdrowotna oraz profilaktyka w naukach medycznych (t. 10, s. 141-149). Uniwersytet Medyczny w Białymstoku https://www.umb.edu.pl/wn-oz/o_wydziale/monografie_pracownikow_wnz

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