Review paper

The effect of weight reduction on the sexual function and reproductive health of obese men

Wpływ redukcji masy ciała w otyłości na funkcje seksualne i zdrowie reprodukcyjne mężczyzn

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Key words: obesity, obesity treatment, male sex hormones, sexual function, semen, bariatric surgery.

Słowa kluczowe: otyłość, leczenie otyłości, męskie hormony płciowe, funkcje seksualne, nasienie, chiurgia bariatryczna.

Abstract

Introduction: Obesity is a disease that significantly increases the risk of death in all ages. It is a risk factor for the development of many diseases that directly affect male reproductive health. Metabolic complications resulting from excess body weight affect testosterone levels and gonadal function in men. Morbid obesity causes hypogonadism in men and an increase in androgen secretion in women. The consequences of overweight and obesity in men are a decrease in testosterone and sex hormone-binding globulin (SHBG), an increase in oestradiol, sexual dysfunction, and reproductive health dysfunction. Surgical weight reduction improves the sexual function and reproductive health of men.
Aim of the research: To investigate the effect of weight loss in obesity on male sexual function and reproductive health.
Material and methods: Research material was collected by searching through databases with scientific articles on obesity, bariatric surgery, the effects of bariatric surgery on sex hormones, sexual function, and fertility in men. The Pubmed and Web of Science research databases were used. Scientific articles, systematic reviews, and meta-analyses were taken into account. The analysis of the included articles covered items from 2003 to 2021. One of the publications dates back to 1996.
Conclusions: Surgical weight reduction improves the profile of male sex hormones and sexual function. The problem of the associations between male sperm quality and surgical weight reduction requires further research.

Streszczenie

Wprowadzenie: Otyłość jest chorobą, która znacząco zwiększa ryzyko zgonów w każdym przedziale wiekowym. Jest czynnikiem ryzyka rozwoju wielu chorób, które bezpośrednio wpływają na zdrowie reprodukcyjne mężczyzn. Powikłania metaboliczne będące następstwem nadmiernej masy ciała wpływają na stężenie testosteronu oraz czynność gonad u mężczyzn. W otyłości patologicznej dochodzi do rozwoju hipogonadyzmu u mężczyzn oraz wzrostu wydzielania androgenów u kobiet. Następstwem nadwagi i otyłości u mężczyzn jest zmniejszenie stężenia testosteronu i globuliny wiążącej hormony płciowe (SHGB), podwyższenie stężenia estradiolu, zaburzenie funkcji seksualnych i zdrowia reprodukcyjnego. Chirurgiczna redukcja masy ciała wpływa na poprawę funkcjonalności seksualnej i zdrowia reprodukcyjnego mężczyzn.
Cel pracy: Zbadanie wpływu redukcji masy ciała w otyłości na funkcje seksualne i zdrowie reprodukcyjne mężczyzn.
Materiał i metody: Materiał badawczy zbierano, przeszukując bazy z artykułami naukowymi dotyczącymi tematyki otyłości, chirurgii bariatrycznej, wpływu chirurgii bariatrycznej na hormony płciowe, funkcje seksualne i płodność u mężczyzn. Wykorzystano bazę naukową PubMed i Web of Science. Uwzględniano artykuły badawcze, przeglądy systematyczne, meta-analizy. Analiza włączonych artykułów obejmowała pozycje z roku od 2003 do 2021. Jedna publikacja pochodzi z roku 1996.
Wnioski: Chirurgiczna redukcja masy ciała wpływa na poprawę profilu hormonów płciowych męskich i funkcje seksualne. Problem zależności pomiędzy jakością nasienia męskiego a chirurgiczną redukcją masy ciała wymaga dalszych badań.

Introduction

Obesity is a disease that is caused by various factors. It is a serious public health problem because it generates an increase in healthcare costs and social costs [1]. The European Association for the Study of Obesity (EASO) defines obesity as a growing challenge for public and clinical health, as well as for health sciences [2]. It most often affects the population of people living in developed countries; however, research results indicate that it is increasingly affect-

ing developing countries [1, 3]. The World Health Organization (WHO) defines obesity as a disease in which the excessive accumulation of fat has a negative effect on health, with a predisposition to the development of multiple diseases [1, 4]. It is a disease that significantly increases the risk of death in all age groups, both among women and men. Obesity/overweight is an epidemic of the 21st century that affects both adults and children [5]. This thesis is confirmed by statistical data provided by the WHO. According to data from the World Health Organization, approximately 1.9 billion people in the world are overweight and 650 million are obese. In 2016, 39% of the adult population was overweight and 13% of them were obese. In 2017, overweight and obesity caused the deaths of as many as 4 million people worldwide [1]. Over the years, the prevalence of overweight and obesity in both adults and children has steadily increased. From 1975 to 2016, this indicator quadrupled from 4% to 18% among children and adolescents aged 5 to 19 years [1]. The WHO reports that in 2019 as many as 38.2 million children under the age of 5 years were overweight or obese [1, 4, 6]. The National Health and Nutrition Examination Survey (NHANES) reports that about 66% of adults are overweight or obese, of whom 31% are obese and 5% are morbidly obese [7]. The continuing upward trend in the increase of overweight and obese people will ultimately result in 2.16 billion (38%) overweight people in the world and 1.12 billion (20%) obese adults worldwide by 2030 [3]. Obesity is a chronic disease that results from a disrupted energy balance. The energy balance is the ratio of energy in and energy out [1, 5]. The factors that may affect and disrupt the energy balance, and thus predispose a person to the development of overweight and obesity, are as follows: environmental, genetic, and hormonal factors, as well as the use of certain medications [3]. The environmental factor comprises the excessive supply of energy obtained from food in relation to the body's energy needs, the so-called positive energy balance. Mutations in the genes controlling appetite and metabolism are responsible for obesity [3]. The basic anthropometric index for the assessment of the nutritional status and classification of body weight used in clinical practice is the body mass index (BMI). It is a simple and easy to calculate index; however, it has many limitations and low sensitivity. The following are some of the limitations of the index: age, muscle mass, and ethnic origin, which influence the associations between BMI values and adipose tissue. Despite its imperfections, due to the ease of calculation and simplicity, it is the most frequently used index in clinical practice and in epidemiological studies [5]. According to BMI, obesity is diagnosed when it reaches or exceeds a value of 30 kg/m^2. Above 40 kg/m^2 it is diagnosed as morbid obesity [1]. Obesity is more common in women than in men [4, 8]. Other anthropometric measurements to assess nutritional status include skin fold thickness measurement, waist circumference, and waist-to-hip ratio (WHR). These are the indices that are increasingly used to assess the probability of health problems resulting from obesity or overweight [4, 9]. Overweight and obesity cause the development of a large group of comorbidities and psychosocial diseases, which in turn are associated with a reduction in the quality of life and have a negative impact on the healthcare system. They represent a high risk factor for many non-communicable diseases such as fatty liver, diseases of the cardiovascular system: arterial hypertension, thromboembolism, and circulatory failure, as well as obstructive sleep apnoea, hypercholesterolaemia, carcinoma, type 2 diabetes (T2DM), musculoskeletal and gallbladder diseases, and depression. The results of numerous studies show the negative impact of an excessive accumulation of adipose tissue on reproductive functions in both women and men [6, 10]. Obesity significantly influences disorders in the metabolism of sex hormones and disorders of the reproductive system [1]. Sex hormones significantly influence the composition of body mass and the metabolism of nutrients such as glucose or lipids [11]. Obesity affects ovulation disorders in women. It also shows a positive correlation with infertility that results from abnormalities in hormones and the quality of reproductive cells as well as in the receptivity of the endometrium [10]. Obesity influences the development of polycystic ovary syndrome (PCOS). The majority of women who qualified for bariatric surgery according to the validated Female Sexual Function Index (FSFI) suffer from Female Sexual Dysfunction (FSD) [10, 12]. Morbid obesity leads to the development of hypogonadism in men and an increase in androgen secretion in women [13]. About 40% of obese men develop Male Obesity Secondary Hypogonadism (MOSH). The consequence of being overweight and obese in men is a reduction in testosterone and sex hormone-binding globulin (SHBG) and an increase in oestradiol [10].

Research material was collected by searching through databases with scientific articles on obesity, bariatric surgery, the effects of bariatric surgery on sex hormones, sexual function, and fertility in men. The PubMed and Web of Science research databases were used. Scientific articles, systematic reviews, and meta-analyses were taken into account. The analysis of the included articles covered items from the period 2003 to 2021. One of the publications dates back to 1996.

The aim of the study is to investigate the effect of weight loss on the sexual function and reproductive health of obese men.

Obesity and available treatments

The life expectancy of people with morbid obesity is shorter by 5 to 20 years; therefore, the most effec-

tive treatments for the disease should be sought. In medicine, there are methods of conservative as well as surgical treatments of obesity. Conservative methods of obesity treatment include the modification of eating habits, increased physical activity, and the implementation of pharmacotherapy [14]. In clinical practice, the methods of conservative obesity treatment bring little effect, and there is a lack of long-term benefit – they only reduce body weight by 5% to 15% [7]. The goal of obesity treatment is the prevention of complications, as well as the prevention and treatment of comorbidities [2]. Lifestyle change is the most common treatment of overweight and obesity, but it is not always enough. Research results indicate that after an attempt to implement conservative treatment, the effect is short lived, and weight gain after the treatment is often much greater compared to the baseline [15]. An important element of disease treatment is the psychological aspect, which should be implemented by reducing the stigmatization of the patient and improving his/her self-esteem and well-being. Psychological factors play a key role in the treatment of obesity, especially in people with class 3 obesity, so-called morbid obesity. Pharmacotherapy of obesity and the treatment of comorbidities can be combined with other treatments. Pharmacotherapy should not be used as the sole method of treating the disease; it may be part of a multi-approach therapy, e.g. with a change of lifestyle. Patients undergoing pharmacotherapy are most often those with BMI > 30 kg/m^2 or with BMI > 27 kg/m^2 with comorbidities. Treatment with anti-obesity drugs is used and continued when the reduction of body weight in a patient reaches 5% after 3 months (non-diabetic patients) or > 3% (diabetic patients) [2, 14]. If the treatment fails to achieve its goals, it should be discontinued and alternative treatment should be implemented [16]. Another alternative treatment of obesity is bariatric surgery. Bariatric surgery has been in use since the 1950s and is of great benefit in the treatment of obesity and metabolic disorders resulting from the disease. Mortality after bariatric surgery is estimated at the level of 0.3% to 1.25%, depending on the type of surgery and the presence of comorbidities. In Poland, bariatric surgery has been performed since the 1970s and is considered the best method of treating morbid obesity. The loss of excess body weight is the result of deliberate changes to the digestive tract. The effect of these changes are a reduction of the amount of food consumed, disturbances in the process of digestion and absorption, as well as an influence on the hormones of the gut-brain axis [17]. The benefits of treating obesity with bariatric surgery not only include weight reduction, but also improved treatment of many metabolic disorders resulting from the disease, such as type 2 diabetes (T2DM) and hyperlipidaemia. Every year, around 500,000 bariatric surgeries are performed worldwide. Different types

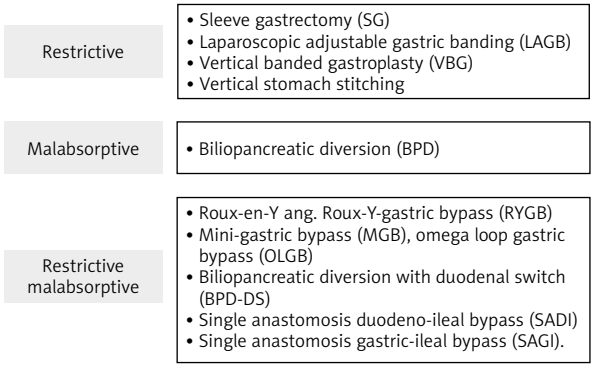

Figure 1. Types of bariatric surgery procedure [15, 18]

of bariatric surgery can be found: gastric restrictive surgery, which limits the volume of consumed food; malabsorptive surgery; and restrictive-malabsorptive surgery (Figure 1) [15, 17]. The most frequently performed procedures are sleeve gastrectomy (SG – 49%) and Roux-en-Y gastric bypass (RYGB – 43%) [18]. Currently, due to the impact of bariatric surgery on weight loss and metabolic disorders, the procedures are divided into those that achieve a bariatric effect and procedures aimed at treating metabolic disorders [15]. Weight loss varies depending on the type of bariatric surgery. In the case of the RYGB procedure, the reduction of excess body weight is 62%, after vertical band gastroplasty (VBG) it is 68%, and after laparoscopic adjustable gastric band (LAGB) it is 48% [18]. According to European guidelines, bariatric surgery procedures are performed in cases where conservative treatment has failed and the body mass index of the patient is above 40 kg/m^2, or the patient's BMI is between 35 kg/m^2 and 39.9 kg/m^2 accompanied by serious comorbidities related to obesity, such as diabetes. Patients with a BMI between 30 kg/m^2 and 34.9 kg/m^2 and with poorly controlled diabetes are also taken into account [2]. In patients over 60 years of age, the risk of complications should be weighed against the benefits of bariatric surgery.

The influence of obesity on sexual function and fertility in men

Obesity, which is considered to be a pandemic, is a disease that affects women twice as often as men. It is a risk factor for the development of, and a component of, many diseases, such as metabolic syndrome, cancer, diabetes, hypertension, heart disease, and infertility [17]. It affects over 1.9 billion adults worldwide. Epidemiological data show that in the United States, since the 1970s, the number of males in the reproductive period with obesity has tripled. This fact is alarming due to scientific reports which indicate that with the increase in obesity, the indicator of male infertility and low sperm quality increases proportionally [19].

Male infertility accounts for 45% to 50% of infertility in couples [19]. Excessive adipose tissue affects the functionality of the hypothalamic-pituitary-gonadal axis, leading to the development of hyperoestrogenic and hypogonadotropic hypogonadism [19, 20]. It probably influences spermatogenesis, also in the thermal effect mechanism, through diabetes and the epigenetic dysfunction of sperm. Hypogonadism is defined as a condition in which the gonadal function is diminished or aborted [21]. The association between hypogonadism and obesity is two-way. The results of studies of large populations indicate that obesity is a direct factor responsible for testosterone deficiency. In turn, low testosterone concentration accelerates and increases the process of adipogenesis and the development of visceral obesity. This leads to the conclusion that the balance of sex hormones plays a key role in energy homeostasis, energy expenditure, and then body mass composition. Visceral adipose tissue dysfunction is the cause of low testosterone levels, as a consequence of tissue insulin resistance, an ongoing inflammatory process and low sex hormone-binding globulin (SHBG) [22]. The study by Dallal *et al.* [23] showed that men with morbid obesity, who qualified for gastric bypass surgery, suffered from profound sexual dysfunction.

Hormonal disorders affecting the fertility of obese men

According to research results, one of the possible causes of sexual and fertility dysfunctions in people with excess body weight is hormonal imbalance, including an imbalance in reproductive hormones. An excessive amount of adipose tissue mainly located in the visceral and abdominal cavities leads to changes in the levels of reproductive hormones, changes in sexual behaviour, and the development of chronic inflammation [24]. In obese men, the sex hormone profile is significantly disturbed in comparison with the hormonal profile of men with normal body weight. The condition for the proper course of the spermatogenesis process is the correct testosterone concentration in the testes, which is > 70 nmol/l [19]. Therefore, even with a slight decrease in the concentration of the hormone in the serum, its level decreases in the testes as well, which disrupts the process. The physiological hypothalamus synthesizes gonadotropin-releasing hormone (GnRH) in a pulsating manner, and stimulates the secretion of luteinizing hormone (LH) and follicle-stimulating hormone (FSH). LH synthesized by the pituitary gland influences the steroidogenesis of testosterone by Leydig cells. Testosterone binds mainly to SHBG and is metabolized to estrogen by the aromatase enzyme. Follicle-stimulating hormone (FSH) is involved in enhancing the function of Sertoli cells [19, 25]. Excessive adipose tissue, especially visceral fat, is a strong risk factor for the development of male obesity secondary hypogonadism (MOSH). Secondary obesity-related hypogonadism affects approximately 45% of men in moderate-severe obesity and leads to endocrine dysfunction. It is hormonally characterized by a low level of free and total testosterone, below 12.1 nmol/l in plasma, a low level of sex hormone-binding globulin (SHBG), and an increased concentration of oestradiol. The clinical picture of MOSH also includes a reduced level of lean body mass, bone demineralization, depression, fatigue, erectile dysfunction, changes in sperm parameters, and sexual dysfunction, which is associated with male infertility [21]. Obesity-related hypogonadism is likely to include abnormal testes response to LH, decreased levels of SHBG, FSH, reduced pulse amplitude of cyclic LH secretion from the pituitary gland, higher levels of oestradiol (E2), and low levels of inhibin B [19, 24]. The high concentration of oestrogens is caused by the increased conversion of androgens to oestrogens as a result of greater bioavailability and increased activity of the cytochrome P450 aromatase enzyme, which is actively involved in the biosynthesis of oestrogens. High levels of the enzyme are found in white adipose tissue, which occurs in men with excess body weight. White adipose tissue is a large secretory organ responsible for the production of many hormones such as adiponectin, leptin, resistin, and adipokines, which are immunomodulating agents. Due to its excess, the concentration of adipose-derived hormones is high, and as a result of their action, changes in the body's homeostasis, insulin action, angiogenesis, vascular remodelling, lipid and glucose metabolism, as well as sexual functioning and fertility occur [21, 24]. Physiologically, adipocytes are responsible for the release of leptin, increasing the release of kisspeptin from KISS neurons [19]. The consequence of this action is an increased secretion of GnRH from the hypothalamus, LH and FSH from the pituitary gland, followed by an increase in testosterone synthesis in the testes [19, 25, 26]. Excessive adipose tissue leads to disturbances in the hypothalamic-pituitary-gonadal (HPG) axis [19]. Leptin is responsible for regulating food intake and for energy expenditure depending on the hypothalamus [24]. It affects the functioning of the hypothalamic-pituitary axis, which influences the reproductive system in both sexes, and is essential for its proper functioning. It affects the kisspeptin neurons, and stimulates the secretion of gonadotropin (GnRH) and luteinizing hormone (LH). Testosterone inhibits the secretion of leptin from adipocytes. As a result of adipocyte dysfunction caused by obesity, there is an excessive release of leptin from fat cells, causing central hormone resistance at the level of the hypothalamic-pituitary axis. The consequence of the process is the reduction of kisspeptin expression in the hypothalamus, and inhibition of the release of gonadotropin (GnRH) and luteinizing hormone (LH), which results in testosterone defi-

ciency [24]. The increase in the concentration of the hormone may contribute to a disturbance in the spermatogenesis process. Research results suggest that the hormone is actively involved in appropriate sexual maturation and reproduction and glucose metabolism [27]. Leptin receptors are located on the testicular tissue and in the sperm cell membrane, and perhaps the hormone directly influences sperm, regardless of the regulation of the hypothalamic-pituitary-gonadal axis, and only due to the hormonal function [24, 28]. Another factor contributing to the development of male infertility related to obesity is the low concentration of sex hormone-binding globulin (SHBG). SHBG is a glycoprotein synthesized by hepatocytes. It binds and transports sex hormones, including testosterone [24]. It is responsible for reducing fat deposits in macrophages and adipocytes and inhibits the development of inflammation [22]. High levels of lipids in the liver, insulin resistance, and high levels of the pro-inflammatory cytokines tumour necrosis factor tumor necrosis factor α (TNF-α) or interleukin-1 (IL-1) are probably responsible for the low concentration of SHBG in obese patients [22]. Testosterone is a hormone that has anti-inflammatory effects, reduces the concentration of C-reactive protein in the liver, and improves the sensitivity of tissues to insulin. Together with oestradiol (a product of testosterone aromatization), it takes an active part in the activation of androgen receptors and oestrogenic in visceral adipose tissue. This action reduces the release of adipokines and increases the secretion of adiponectin and visfatin [22]. Adipokines are substances secreted by white adipose tissue, some of them, such as TNF-α , IL-6, and plasminogen activator inhibitor 1, are believed to negatively affect sperm function and reproductive health in both men and women. They are inflammatory mediators that induce low-grade chronic inflammation, inhibiting GnRH secretion from the hypothalamus and LH secretion *in vitro*. The pathomechanism of inflammatory mediators is related to the release of reactive oxygen species (ROS) and reactive nitrogen species (RNS) [24, 29]. Increased levels of IL-1B, IL-6, IL-8, and TNF-α are associated with poor sperm quality. Adipokines contribute to the development of hypogonadism by interfering with kisspeptin signalling and impairing the release of gonadotrophin (GnRH) [22]. Interleukin-1 (IL-1), especially IL-1β, to a greater extent than IL1-α, is a potent blocker of GnRH secretion, thereby inhibiting LH release. C-reactive protein (CRP) may contribute to disturbances in the HPG axis and may increase insulin resistance by disrupting insulin signal transduction. Research reports indicate that many of these cytokines interfere with spermatogenesis and steroidogenesis [30]. The results of a study by Agarwal *et al.* [26] indicated that a high concentration of reactive oxygen species is an independent marker of male infertility. Insulin resistance is another important factor associated with hypogonadism in obese men. Insulin stimulates the activity of the hypothalamic-pituitary-gonadal (HPG) axis. May affect GnRH secretion by affecting the neurons of the hypothalamus. Therefore, it is extremely important to maintain adequate insulin metabolism to maintain the functional integrity of the HPG axis [26].

The influence of concomitant diseases resulting from obesity on male fertility

Obesity is a risk factor for the development of many diseases that directly affect male reproductive health. Metabolic complications resulting from excess body weight affect testosterone levels and gonadal function in men. Sleep apnoea is a medical condition that often coexists with obesity. Epidemiological data show that obesity affects approximately 4% of adult men, of whom 2/3 suffer from sleep apnoea. The clinical picture of sleep apnoea is associated with proximal airway obstruction, which leads to shallow breathing, chronic hypoxia, and hypercapnia. It causes the sleep cycle to be disturbed and fragmented. Consequently, there are changes associated with the synthesis of luteinizing hormone (LH), reduction of serum testosterone concentration, and functional weakening of the pituitary gland and gonads [19, 26]. Men have high deposits of adipose tissue in the scrotum, lower abdomen, and thighs, which act as a protective and thermal insulation layer for the testicles, increasing the temperature. The deposits are especially high in morbidly obese men. Increased temperature, low physical activity, and a sedentary lifestyle lead to impaired thermoregulation in the scrotum and testes, which results in disturbances in spermatogenesis and testosterone production [19]. The increased amount of adipose tissue generates the risk of high accumulation of environmental toxins. A large part of the toxic substances dissolve in fats, accumulating in the body and disrupting the hormonal balance, negatively affecting male sex hormones and spermatogenesis [19, 24]. Diabetes is another disease that is associated with obesity and affects the androgenic axis in men. It impairs fertility and lowers the sperm quality index [19]. Obese men with diabetes have central and peripheral tissue resistance to insulin, which results in decreased SHBG synthesis in hepatocytes. Low SHBG concentration means that a large part of the testosterone remains as free form, which is aromatized to oestradiol (E2) [19, 24]. Clinical studies have shown a positive association between diabetes and sperm disorders [19]. In their own study, Eisenberg *et al.* demonstrated the influence of diabetes on the reduction of fertility in men [31]. The results of the research by Svartberg *et al.* [32] indicate that low testosterone levels in men are associated with a high risk of hypertension and left ventricular hypertrophy. Haffner *et al.* [33] demonstrated that low levels of SHBG and testosterone may be a pre-diabetes state in men. Ding *et al.* [34],

in a systematic review of studies, showed that endogenous sex hormones can influence in different ways glycaemia and the risk of type 2 diabetes in men and women. High testosterone levels are associated with a higher risk of type 2 diabetes in women but with lower risk in men. The inverse association of SHBG with risk was stronger in women than in men. The eating habits of people with excess body weight often deviate from a healthy diet. High-fat and high-energy diets, constituting the basis of the nutrition of obese people, disrupt the protective barrier of the intestinal mucosa. As a result, they cause the risk of translocation of the bacterial flora from the inside of the intestine to the systemic circulation. The consequence is a chronic, low-intensity inflammatory process that favours the impairment of testicular function and impairs the reproductive capacity of both men and women [26].

Erectile dysfunction and obesity

Obesity affects not only changes in the profile of sex hormones, but also overall sexual health. Research suggests a positive association between obesity and erectile dysfunction in men. Erectile dysfunction is a serious clinical problem, and obesity is one of the main risk factors for its development. Erectile dysfunction is defined as the inability to achieve and/or maintain satisfaction during sexual intercourse. This disrupts the functionality of reproductive health and significantly reduces the quality of sexual life. Epidemiological studies are evidence of the scale of the problem that affects the male population. In 1995 the problem of erectile dysfunction affected approximately 150 million men worldwide, and by 2025 it is expected to affect as many as 322 million [11]. Bacon *et al.* [35] indicated that patients with a BMI higher than 28.7 kg/m^2 have a 30% higher risk of developing erectile dysfunction compared to men with a satisfactory BMI index. The effect of obesity on the ability to achieve and maintain an erection is a multifactorial process. It has been suggested that an important role is played by the ongoing inflammation presented by increased parameters of inflammation (TNF, IL-6) as well as decreased testosterone levels [19]. Achieving and maintaining an erection in men depends on the proper functioning of the central and peripheral nervous system, and endocrine and psychological factors. During sexual stimulation, non-adrenergic, non-cholinergic, and parasympathetic cholinergic fibres are activated, leading to the release of acetylcholine and nitric oxide. The consequence of this process is a reduction in the concentration of calcium inside the cell and an increase in the concentration of cyclic guanosine monophosphate (GMP). As a result, smooth muscles are relaxed, blood vessels widen, and there is increased blood flow to the corpus cavernosum. Excessive adipose tissue induces chronic inflammation, which leads to impaired function of the arteriolar endothelium, as well as a reduction in the activity of nitric oxide [29]. Insulin resistance and leptin resistance may contribute to the exacerbation of diabetes and the development of peripheral neuropathies, which is another factor that impairs erectile function [21]. Difficulties in obtaining and maintaining an erection are the cause of infertility in obese men with diabetes. In a study by Ho *et al.* [17], 72% of the studied cohort of men with morbid obesity had erectile dysfunction with frequent coexistence of diabetes. The results also suggest that people with excess body weight have an increased risk of azoospermia and oligozoospermia. Many studies on the association of BMI with sperm parameters, such as concentration and total sperm count, are not fully conclusive, which is a reason to continue research in this area [36]. Sarwer *et al.* [37], in their own study, showed that erectile dysfunction concerned 36% of men qualified for surgical weight reduction. Similar results were obtained by Moore *et al.* [38], who indicated that 45% of men with excess body weight had severe erectile dysfunction. Research clearly shows that excess body fat negatively affects sexual function, reproductive health, and quality of sexual life in men with morbid obesity. Therefore, it is extremely important to implement appropriate treatment methods, selected individually, which will in turn help to reduce body weight and improve the quality of sexual life in each area (Table 1) [39].

The influence of bariatric surgery on sexual function and fertility in men

The research results clearly indicate that obesity impairs both sexual function and fertility in men [17, 19–34, 36, 40–42]. In order to reduce the risk of developing infertility, to improve the sexual functions, as well as treat concomitant diseases dependent on an excessive amount of adipose tissue, the most effective methods of treatment should be sought. A promising treatment contributing to weight reduction, and improvement of reproductive health and quality of sexual life seems to be bariatric surgery. However, many of the available study results that present effects of surgical weight loss on the male hormone profile, sexual function, and sperm quality are inconclusive. Reis *et al.* [43], in their study, compared the extent to which sexual function, erectile function, and the hormonal environment change after significant weight loss in a morbidly obese man, using surgical and non-surgical methods. Weight reduction by surgery increased the quality of erectile function and increased the level of free testosterone (FT), total testosterone (TT), and follicle stimulating hormone (FSH) and decreased the concentration of prolactin (PRL). In comparison, lifestyle modification including exercise and diet only affected BMI without affecting the hormonal and sexual profile of morbidly obese men. The study demon-

Table 1. The influence of obesity on male fertility

Study site	Study group size (*n*)	Study group characteristics	Studied variables	Results	Literature
Great Britain	29	BMI > 40 kg/m^2	Sexual functions: sexual function, frequency of sexual thoughts, frequency of morning erections Symptoms of peripheral neuropathies Level of sex hormones: level of total testosterone, free testosterone, sex-hormone binding globulin, dihydrotestosterone, dehydroepiandrosterone, sulphate, androstenedione	– 55% of men had symptomatic erectile dysfunction – 45% of men had a decrease in the frequency of sexual thoughts – Low concentration of total testosterone, median = 9.0 nmol/l, erectile dysfunction is associated with obesity and small fibre neuropathy	Ho *et al.* 2019 [17]
North America	4440	Mean BMI = 27.6 kg/m^2	Hormone levels: concentration of testosterone, oestradiol (E2), FSH, LH, prolactin (PRL) Semen parameters: ejaculate volume, sperm concentration, mobility, morphological parameters	– Higher BMI negatively affects the reproductive hormones. Testosterone, E2, LH showed a significant association with BMI. Prolactin levels are not related to BMI values – As the body size increases, oestradiol levels increase and testosterone levels decrease, possibly affecting male fertility – Obesity affects the decrease in semen parameters: the incidence of azoospermia was 12.7%, and oligospermia was 31.7% in obese men	Bieniek J. *et al.* 2016 [39]
Spain	100	BMI ≥ 35 kg/m^2	Hormone levels: total testosterone (TT), SHGB, free testosterone concentration (FT) Semen parameters	– The incidence of MOSH in patients with moderate to severe obesity is high – Low testosterone in serum is associated with insulin resistance and low ejaculate volume with higher BMI and excess body weight	Calderón *et al.* 2016 [40]
Argentina 2006–2007	794 obese patients 155 overweight patients 388	BMI = median 33.2 kg/m^2	Semen quality parameters: volume, concentration, motility, morphology, viability, membrane integrity, functionality, and nuclear maturity Levels of functional markers of the epididymis and male accessory glands (seminal vesicles and prostate), testosterone levels	– No significant association between BMI and sperm concentration – Negative association between BMI and sperm motility (total and rapid motility) and between BMI and NAG levels ($p < 0.05$) – Reduced sperm motility in the high BMI group – No association between BMI and other semen parameters such as sperm viability, membrane integrity and/or functionality (HOS), testicular maturation or the concentration of citric acid in the semen	Martini *et al.* 2010 [41]

Table 1. Cont.

Study site	Study group size (*n*)	Study group characteristics	Studied variables	Results	Literature
New Zealand 2008–2012	511	Median BMI = 27.1 kg/m^2	Sperm quality parameters: sperm concentration, total sperm count, sperm motility (total motility), sperm morphology, semen volume, total sperm motility Hormone levels: measurement of LH, FSH, oestradiol, total testosterone, free testosterone, and sex hormone binding globulin (SHBG) in a blood sample	– No significant association was found between BMI and semen parameters, except for normal sperm morphology – Overweight and obese men did not show a significantly increased relative risk of abnormal semen parameters – Among hormones, negative associations with BMI were found for total testosterone ($r = -0.35$, $p \leq 0.0001$), free testosterone ($r = -0.25$, $p \leq 0.0012$), and SHBG ($r = -0.44$, $p \leq 0.0001$). Multiple linear regression analysis also showed that BMI had a marginally effect on normal sperm morphology (effect estimate = 0.47, $p = 0.038$)	Macdonald *et al.* 2013 [42]

BMI – body mass index, FSH – follicle-stimulating hormone, LH – luteinizing hormone, PRL – prolactin, TT – total testosterone, SHGB – free testosterone and sex hormone binding globulin, FT – free testosterone concentration, MOSH - male-obesity secondary hypogonadism.

strates the advantage of surgical treatment over conservative treatment of obesity to improve the quality of sexual life and reproductive health. Regardless of the type of surgery, studies show a positive effect of bariatric surgery on the improvement of the sex hormone profile and treatment of MOSH in obese men. Samavat *et al.* [44] assessed the effect of bariatric surgery on waist circumference (WC), BMI, and TT levels in men with morbid obesity. The patients underwent gastric restrictive surgery with Roux-en-Y anastomosis, adjustable gastric banding laparoscopy, biliopancreatic diversion, and gastric sleeve. In patients with hypogonadism, an improvement in the profile of sex hormones was observed after surgery; a significant increase in TT, FT, and SHBG and a decrease in oestradiol (E2). A study by Pellitero *et al.* [45], similarly to the study by Samavat *et al.* [44], indicated the positive effect of permanent weight loss as a result of bariatric surgery on the treatment of hypogonadism. Research suggests that bariatric surgery plays a crucial role in treating MOSH [44, 46]. Lee *et al.* [47], in a systematic review, presented the results of the influence of bariatric surgery on male sex hormones, sexual function, and semen parameters. The review results, like the study by Reis *et al.* [43] and a study by Samavat *et al.* [44], showed an increase in the level of sex hormones: TT, LH, FSH, and SHBG and an increase in erectile function compared to the level before surgery. A decrease in the levels of free and total oestradiol (E2) and prolactin was also observed. The bariatric treatment did not affect the quality of sperm or the levels of DHEA, androstenedione, and inhibin B. A similar result from the study on the association between bariatric treatment and the quality of male sperm was obtained by Legro *et al.* [48]. The men enrolled in the study underwent gastric bypass surgery using the Roux-en-Y (RYGB) method. The study showed a significant improvement in total testosterone levels 3 months after the RYGB treatment. No changes in semen parameters or oestrogen levels in serum and urine were observed. A large area of research is the association between male sperm quality and surgical weight reduction. Many of the research findings are unclear and inconsistent. Most of the available studies regarding the influence of bariatric surgery on the profile of sex hormones indicate an increase in their level compared to the concentration before surgery. Weight reduction after bariatric surgery led to an increase in total and free testosterone levels, sex hormone-binding globulin, and a decrease in oestradiol levels [46]. Erectile dysfunction is a serious problem in men with obesity, especially those with morbid obesity. Fahmy *et al.* [46] assessed the effect of surgical weight reduction achieved with laparoscopic sleeve gastrectomy (LSG) on erection in obese men. There was a decrease in serum cholesterol and triglycerides, and a decrease in the level of IL-6 and C-reactive protein compared to the period before the surgery. The International In-

Table 2. The impact of weight loss as a result of surgery on the profile of sex hormones and erectile function in morbidly obese men

Study site Study group size	Characteristics of the study group: age [years], weight [kg], BMI [kg/m^2], waist circumference (WC in cm)	Type of surgery	Total testo-sterone (TT)	Free testo sterone (FT)	Sex hormone binding globulin (SHBG)	Luteinizing hormone (LH)	Follicle stimulating hormone (FSH)	Oestradiol (E2)	Erectile function The International Index of Erectile Function (IIEF)	References
United States n = 32	Median: age: 48 years, weight: 149.3 kg, BMI: 45.1kg/m^2, WC: 145.4cm	Roux-en-Y gastric bypass	↑	↑	↑	↑	No information	No information	No statistical significance and no significant improvement in male sexual functioning 1 year after surgery: p = 0.910, 2 years after surgery: p = 0.099, 3 years after surgery: p = 0.357, 4 years after surgery: p = 0.395	Sarwer *et al.* [37]
Italy n = 44	Median: age: 43.45 years	SG 40/44 (90,91%), 4/44 RYGB (9,09%)	↑	No information	↑	No information	↑	Oestradiol is a significant negative predictive factor of sexual improvement. No statistical significance in the multivariate analysis	IEEF total score was significantly higher 12 months after surgery	Arolfo *et al.* [51]
Brazil n = 33	Age: 36.3 ±8.1 years, weight: BMI: 43.8 ±7.8 kg/m^2	Laparoscopic Roux-en-Y gastric bypass 25/33, laparoscopic sleeve gastrectomy 8/33	↑	↑	↑	↑	↑	↓	Total IIEF mean score increased by 5.2 points (p = 004). The erectile function subdomain increased by 2.4 points (p = 011), the sexual desire subdomain increased by 1.0 point (p = 0.006), and the intercourse satisfaction subdomain increased by 1.2 points (p = 0.012)	Machado *et al.* [52]
Italy n = 20	Median: age: 40.5 [27.2–46.7] years, BMI 43.6 [40.9–48.7] kg/m^2	Roux-en-Y gastric bypass	↑	↑	↑	↑	↑	↓	No information	Facchiano *et al.* [53]

dex of Erectile Function (IIEF) scores also improved, which indicates an improvement in erectile function. Similar research results were presented by Glina *et al.* [49] in a systematic review, indicating an improvement in erectile function in obese men after weight reduction by surgical methods. The results of Dallal *et al.* [23], similarly to the 2 previous studies, also indicate an improvement in sexual function, including sexual drive, erectile function, ejaculation, and sexual satisfaction in patients undergoing gastric bypass. El-Tholoth *et al.* [50] also assessed the effect of bariatric procedures on sexual function in men with obesity (average BMI: 46.8 kg/m^2). The results of the study clearly showed an improvement in erectile function. The IIEF score improved, and overall satisfaction was better (76.7%). However, conflicting results were obtained by Sarwer *et al.* [37], which indicated a lack of statistical significance in the improvement of sexual functioning, including the improvement of erectile function in men with obesity after bariatric surgery. Similarly, Legro *et al.* [48] did not find any significant improvement in erectile function and showed an improvement only 12 months after surgery (Table 2).

Summary

Obesity is a disease that significantly increases the risk of death in all ages. It is a risk factor for the development of many diseases. In medicine, there are methods of conservative and surgical treatment of obesity. The results of many studies indicate that after an attempt to implement conservative treatment, the effect is short lived, and the weight gain after the treatment is often much greater than the baseline values. Obesity affects changes in the profile of sex hormones, sexual function, and the state of reproductive health. Sexual dimorphism is clearly manifested in obesity-related gonadal dysfunction. Research results suggest a positive association between obesity and erectile dysfunction in men. Excessive adipose tissue, especially visceral fat, is a strong risk factor for the development of male obesity secondary hypogonadism (MOSH), which affects approximately 45% of men. It is characterized by low levels of free and total testosterone, low levels of sex hormone binding globulin (SHBG), and increased levels of oestradiol. The consequences of such a state are fertility disorders and lowering the quality of sexual life in men [17, 39–42]. Bariatric surgery is a widely used method of treating patients with morbid obesity and health complications caused by excessive amounts of adipose tissue. The results of many studies show that surgical weight reduction has a positive effect on the improvement of the hormonal profile, the cure of MOSH, the improvement of reproductive health, and the quality of male sexual life. A large area of research is the study of the association between male sperm quality and surgical weight reduction. Much research in this area is inconsistent. The reduction of body weight after bariatric surgery in the presented results of studies mostly led to an increase in total and free testosterone levels and sex hormone-binding globulin, and a decrease in oestradiol levels [37, 51–53]. Because the number of studies and the sample sizes are small, evaluation of the effects of bariatric surgery on various domains of male sexual function and the profile of sex hormones is still a large area for research.

Conclusions

Based on the presented research results, the following can be concluded:

1. Obesity affects changes in the profile of sex hormones, sexual function (erectile function), and reproductive health of men. It disturbs the functionality of the hypothalamic-pituitary-gonadal axis, leading to the development of hyperoestrogenic and hypogonadotropic hypogonadism [17, 19–36, 39–42].
2. Surgical weight reduction improves the profile of male sex hormones, leading to an increase in the level of total testosterone, free testosterone, and sex hormone binding globulin and lowers the concentration of oestradiol [37, 38, 43–53].
3. Surgical weight reduction improves erectile function. In most studies assessing the IIEF, it has improved compared to the condition before surgery. More research is needed on the direct effects of bariatric surgery on sexual function in multiple domains [37, 38, 43–53].
4. The association between the quality of male sperm and surgical weight reduction requires further research. Most studies in this area are inconsistent and unclear [37, 38, 43–53].

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Conflict of interest

The authors declare no conflict of interest.

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