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Is Obesity Significantly Connected with Urogenital Disorders? – Systematic Review

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Abstract

Background: *Obesity was declared an epidemic of the 21st century by WHO. Currently, as many as 39% of adults in the world are overweight and 13% are obese. The number of obese people in the world has tripled since 1975. As is well known, excessive abnormal body weight has a significant impact on the functioning of the entire body. It is very common to emphasize education on cardiovascular implications related to obesity matter – however, overweight and obese patients may also be a group particularly vulnerable to problems related to the urogenital system.*

Objectives: *The aim of the study is to draw attention and discuss the problems related to the above-mentioned system, to which this group of patients is exposed more frequently than patients with normal body weight. They include dysfunctions that do not pose a threat to the patient's life, but significantly reduce the quality of life, such as urinary incontinence and impotence, and severe and even life-threatening diseases such as chronic kidney disease or cancer.*

Material and methods: *Based on numerous data available on this subject and collected from various research centers using Google Scholar and PubMed, the most recent and the most important information on this subject has been summarized. To better present and understand the problem, the mechanisms were also discussed, so that the work is a complete analysis, particularly useful in the current obesity epidemic.*

Results: *Obesity is associated with an increased risk of many diseases of the urogenital system. It causes a worse prognosis and makes treatment less effective. Treatment of obesity should be the first line of treatment of the discussed diseases of the genitourinary system.*

Conclusions: *Obesity is a serious and growing problem all over the world. It has an impact on the epidemiology of many diseases of the urogenital system, therefore paying special attention to its prevention and treatment seems to be one of the most important foundations for the prevention and treatment of dysfunctions of the genitourinary system.*

Key words: *obesity, fertility, uroge*nital, urology

Introduction

Obesity is defined as a complex chronic disease and it is a risk factor for many diseases including cardiovascular diseases, diabetes and cancer, and what is more it has a huge impact on overall health [1, 2]. The prevalence of obesity is increasing year by year and this phenomenon affects not only adults but also children and adolescents [1]. Overweight (including obesity) occurs in over 35% of adults and 11% are obese [3]. The prevalence of pediatric overweight (including obesity) in children aged 5 to 17 years is estimated at about 18%, obesity alone is approximately 2–3% [4, 6]. Obesity is defined as one of the biggest problems of public health [3]. BMI (body mass index) is commonly used as a screening tool to characterize and measure obesity [5]. **BMI can be calculated by dividing your weight (in kilograms) by your height in meters squared** [5]. Overweight is defined as a BMI of 25–30 kg/m^2 and obese is defined as a BMI ≥ 30 kg/m^2, **BMI between 30–34.9 is defined as 1st degree obesity, BMI 35–39.9 2nd degree obesity and over 40 3rd degree obesity** [5].

Mechanism and pathophysiological basis

Major function of ovaries is production, differentiation and release of oocytes. What is more, female gonads produce hormones that influence the development of secondary sex characteristics and the course of pregnancy [7]. One of the most common causes of infertility among women is Polycystic ovary syndrome (PCOS) [8]. PCOS is a reproductive and endocrine disorder and is frequently associated with insulin resistance, metabolic disorders, abdominal adiposity, cardiovascular risk factors and obesity [9]. It affects women of all ages, but most often it occurs in the reproductive age [10]. It is the most common cause of hyperandrogenization and the most common cause of ovulation disorders in women who are treated for infertility [9].

Obesity affects the hypothalamic-pituitary-ovaries (HPO) axis. A normal energy balance, assuming average costs of thermoregulation, reaso-

nable energy consumption and sufficient food consumption, affects the pulsatile secretion of gonadotropin-releasing hormone (GnRH), which translates into the proper functioning of the reproductive axis [11]. GnRH stimulates the pituitary gland to secrete the following hormones: luteinizing hormone (LH) and follicle-stimulating hormone (FSH), which stimulate the growth of follicles in the ovaries and stimulate the production of estradiol and progesterone after ovulation [12, 13]. Estradiol works by feedback and mainly inhibits FSH. On the other hand, when estradiol reaches the concentration threshold and maintains it for at least 24–48 h, it activates increased LH secretion by reducing the amplitude and increasing the frequency of GnRH pulses [14]. Proper maturation is controlled by the hypothalamus and adrenal glands. The adrenal glands take part in adrenarche, which is associated with the increased secretion of androgens of the adrenal cortex in the period preceding puberty or in early puberty [15].

Adipose tissue secretes adipokines, these are bioactive cytokines that are involved in inflammatory processes and metabolic regulation. Due to the secretion of adipokines by adipose tissue, it is considered as an endocrine organ that has an important role in the regulation of physiological events such as immune response, reproduction, glucose and lipid metabolism. The normal level of adipokines is crucial for the functioning of the hypothalamic-pituitary-gonadal axis, as well as for the successful implantation of the embryo, regulation of ovulatory processes and physiological pregnancy. Adipokines specific for adipose tissue include leptin, adiponectin, resin, visfatin and omentin. It has been shown that adipose tissue dysfunction has been observed in patients with PCOS, leading to the overproduction of adipokines. Abnormal concentrations of these factors are strongly correlated with insulin resistance and type 2 diabetes (T2DM) [16].

By affecting the pituitary LH release, obesity leads to a disturbance in the luteal phase. Moreover, it not only affects many other components of the hypothalamic-pituitary-ovarian axis, but may have a direct, independent of the hypothalamus and pituitary effects on the ovaries [17]. Seve-

ral changes in the inflammatory and metabolic systems associated with ovarian dysfunction are outlined below.

Leptin

Leptin is one of the adipokines which, by signaling at the level of the hypothalamus, suppresses appetite and stimulates energy expenditure. Leptin was investigated as a potential slimming drug, but it turned out that the increased concentration of exogenous leptin did not cause weight loss [18]. This study suggested leptin resistance at the hypothalamus level. Another study showed a high concentration of leptin in obese women, which confirms the potential resistance to leptin [19].

Leptin also influences maturation, as proper energy balance is required for proper puberty [20]. Moreover, hyperleptinemia may initiate premature reproductive maturation, while the prolonged state of high leptin concentration may lead to abnormal ovulation [21]. On the other hand, low leptin concentration in the physiological period of puberty may delay or lack puberty [22].

Leptin affects the pulsatile secretion of GnRH; its non-physiological concentration is associated with delayed maturation and reduced fertility [23].

The optimal concentration of leptin is necessary for the proper functioning of the HPO axis, its low concentration may disrupt the physiological secretion of GnRh, on the other hand, high levels may disrupt ovarian folliculogenesis [21].

Ghrelin

Ghrelin is an enterokinase produced mainly by the stomach, but it is also detected in the pancreas, intestine, pituitary and hypothalamus [24]. Its concentration is increased on an empty stomach, and it stimulates the appetite to cover low nutrient intake [25].

In response to nutritional changes, ghrelin influences the homeostasis of reproductive function, which may reduce GnRH secretion and pulsation. In states of low nutrient intake, the concentration of ghrelin

increases, which inhibits the activity of HPO [26]. Moreover, ghrelin influences the secretion of other pituitary hormones, for example: adrenocorticotropic hormone and prolactin. Low levels of ghrelin in low energy states can stimulate the secretion of prolactin, potentially leading to disturbances in ovarian cyclicity. There was also a decrease in ghrelin concentration in children as people approached puberty [27].

In the case of obesity, ghrelin concentration decreases with increasing body weight in the pubertal period [28].

Neuropeptide Y and Agouti related protein (NPY/AgRP)

The arcuate nucleus, which secretes NPY and AGRP, connects peripherally circulating enterokines, adipokines, and the HPO axis. NPY is a neuropeptide that stimulates fat proliferation and angiogenesis, while AgRp stimulates the appetite. Both interact with ghrelin and stimulate the appetite [29].

Under physiological conditions, NPY concentration increases during the ovulatory release to enhance the effect of GnRH, which stimulates the secretion of FSH and LH from the pituitary gland [28]. On the other hand, AgRP stimulates the hypothalamus to generate GnRH pulses, which regulates the release of gonadotropins [30]. NPY may have antiproliferative and pro-apoptotic effects on ovarian folliculogenesis [31].

Adiponectin

Adiponectin is an adipokine secreted by adipose tissue and its concentration increases with hunger [32]. It works by binding to the receptor in the arcuate nucleus, which leads to a reduction in energy expenditure and higher food consumption [33]. Moreover, the production of adiponectin makes cells more sensitive to insulin, and its concentration is inversely correlated with obesity [34]. Low levels of adiponectin are associated with insulin resistance, type 2 diabetes and metabolic syndrome [35].

TNF-α, IL-6 and IL-8 are pro-inflammatory factors that contribute to the development of insulin resistance [36]. Adiponectin inhibits the pro-

duction of TNF-α in adipocytes by acting as an anti-inflammatory adipokine [34]. In obese women, pro-inflammatory factors are elevated, while the concentration of adiponectin is low [21].

Adiponectin receptors can also be found in the ovaries. During the preovulatory phase, adiponectin is involved in changes in the cells of the granular layer, interacting with insulin and IGF-1 [37].

Insulin

Insulin is produced in the beta cells of the pancreas, and glucose stimulates its release. Insulin concentration increases with obesity, and insulin sensitivity decreases [37]. Insulin synthesis is stimulated by adipose tissue. Hyperinsulinemia affects the liver, reducing the production of SHBG, leading to an increase in the concentration of free circulating steroid hormones – estrogens and androgens [38]. The effect of LH on the ovaries is enhanced by insulin, which increases the secretion and production of androgens [39]. Additionally, excessive LH secretion may inhibit follicle growth in the early stages, which promotes cell luteinization and, consequently, may reduce the quality of oocytes [43].

By acting on the pituitary gland, insulin stimulates GnRH, which in turn causes the release of LH [40]. Insulin increases FSH activity by stimulating ovarian steroidogenesis [41]. In genetic diseases, a relationship between the syndromic form of insulin resistance and gonadotropin-independent hyperandrogenism and ovarian enlargement is observed [42].

The state of insulin resistance in particular affects the muscles, liver and adipose tissue, while the ovaries remain insulin sensitive and are therefore exposed to the effects of hyperinsulinemia. Insulin stimulates the cells of the ovaries to produce androgens, and their excess may lead to premature follicular atresia, and thus anovulation [43].

Hyperinsulinemia and insulin resistance accompanying obesity in women may cause disturbances in ovulation, menstruation and fertility.

Obesity affects the reproductive apparatus by impeding the development of ovarian follicles, quantitative and qualitative disorders of oocyte maturation, and disturbs fertilization, meiosis and the appropriate

preimplantation of the embryo [44]. The high concentration of free fatty acids has a toxic effect on reproductive tissues, which contributes to chronic inflammation and cell damage. The excess fatty acids supplied to the body with food can be stored in the form of triglycerides in the adipocytes, which does not cause cell damage. On the other hand, in the case of adipocyte overload, fatty acids accumulate in other tissues, which causes a toxic effect called lipotoxicity [45].

Obesity in the male population

Obesity affects the reproductive functions of men in a multifactorial way and is associated with reduced fertility [46, 47]. In overweight and obese men, an increased probability of decreased fertility and abnormal sperm parameters was observed [48].

Oligozoospermia and low ejaculation volume were more frequent in men with increased waist circumference (WC) and BMI. **The study showed that in men WC > 101.6 cm there is a 7 times greater chance of oligozoospermia, and obese men (BMI > 30) had a 19 times greater chance of oligozoospermia** [49]. There was also a correlation between obesity in men and a higher percentage of abnormal sperm and a lower sperm concentration [50]. Moreover, studies of men undergoing fertility treatment with assisted reproductive technology (ART) showed a relationship between high BMI and a decrease in the pregnancy rate and the number of live births [51]. Despite extensive knowledge of obesity and infertility in women, the impact of obesity in men on the reproductive system is not fully understood.

It has been suggested that male infertility is influenced by several mechanisms that are not yet fully understood and elucidated. The negative impact of obesity on male fertility is mainly associated with a lower serum testosterone level and an increased concentration of estradiol, and therefore spermatogenesis is impaired. Moreover, obesity, through atherosclerotic influence on blood vessels, leads to erectile dysfunction. Increased inflammation and increased testicular temperature are also responsible for the disorders of spermatogenesis [52]. Normally in men,

the hypothalamus produces and releases gonadotropin releasing hormone (GnRH), which stimulates the anterior pituitary gland to produce and release luteinizing hormone (LH) and follicle stimulating hormone (FSH). LH and FSH then stimulate the nucleus to stimulate steroidogenesis and spermatogenesis. Obese men have normal or decreased serum LH levels, decreased total testosterone levels and decreased SHBG levels [53].

Obesity and the associated sedentary lifestyle cause an increase in testicular temperature, which contributes to the increase in estrogen production and disrupts the HPG axis [54]. Adipose tissue produces adipokines that contribute to inflammation and oxidative stress, which can contribute to damage to the structures of the testicles and epididymis. Testosterone production seems to be regulated by leptin, and in obese men a correlation has been shown between high leptin levels and hypogonadism [53]. It should be remembered that fertility is also influenced by other obesity-related factors, such as hyperlipidemia, pro-inflammatory states, metabolic syndrome and cardiovascular diseases [55].

Male obesity may disrupt the molecular and physical structure of sperm during spermatogenesis in the testicle, but also during sperm maturation in the epididymis. People with a high BMI may have a reduced semen quality, which is associated with a lower concentration and sperm motility, as well as a reduced acrosome response and a higher risk of DNA damage [53].

With the global obesity epidemic, there has been a sharp increase in male infertility. This phenomenon is mainly related to the imbalance of sex hormones, abnormal production of inflammatory factors and harmful effects on spermatogenesis [53].

Nephrolithiasis

Obesity and overweight are very significant risk factors for many diseases of the urinary system. Patients with an increased BMI show, among others, an increased tendency to develop kidney diseases,

problems with urinary incontinence and it can also cause growth in cancer incidence.

Urolithiasis is one of the most common urinary tract ailments. Stones in the urinary system may be a random find in an imaging examination (usually during ultrasonography) or may cause symptoms, most often in the form of renal colic. In the United States, urolithiasis affects about 8.8% of the population. It is more common in men than in women and was more often found among obese people (11.2%) compared to people with normal body weight (6.1%) [56]. The obesity epidemic observed in recent years and the change in eating habits mean that excessive body weight and the occurrence of metabolic syndrome can be considered a factor associated with the occurrence of urolithiasis. Urine tests in overweight people have shown that poor eating habits that lead to overweight and obesity may cause changes in urine biochemistry and because of that more kidney stones in these people [57].

There are likely three mechanisms by which obesity can be linked to an increased incidence of nephrolithiasis. The main mechanism is related to the fact that obesity is often associated with insulin resistance, which together lead to a low urine pH and thus uric acid precipitation and the formation of urate stones. The second mechanism is an increased amount of lithogenesis promoters and a decreased amount of its inhibitors in obese people. The last factor is improper diet often used by obese people. There was also a positive correlation between BMI and the amount of urinary oxalate, uric acid, sodium and phosphorus excreted in the urine [58].

Urinary incontinence (UI)

Another problem that obese patients may face more often than the general population is stress urinary incontinence [59]. Population studies have shown that UI is more common in women than in men, and that about 10% of all adult women suffer from it [60].

The cause of stress urinary incontinence is a loss of support for the pelvic floor muscles and connective tissue. The etiology of this disorder is multifactorial and includes, but is not limited to, connective tissue di-

sorders, chronic cough, obesity, pelvic floor injuries after vaginal delivery, pregnancy, menopause, constipation, weightlifting and smoking [61].

In the treatment of stress incontinence, one of the first recommendations is weight loss, giving up smoking and urorehabilitation. Studies have shown that weight loss intervention reduced the frequency of stress urinary incontinence episodes for 12 months and improved patient satisfaction with changes in urinary incontinence for 18 months [62]. Based on the results obtained, it may be suggested that maintaining body weight may have long-term benefits in the treatment of urinary incontinence.

Other renal dysfunctions

The diet of overweight and obese patients is often based on eating large amounts of carbohydrates and fats (especially saturated ones). As it turns out, the type of food you eat can have a direct impact on kidney damage. One study found that a long-term high-fat diet causes at least some kidney damage due to tissue lipid accumulation, increased oxidative stress, and mitochondrial dysfunction that promote excessive programmed cell death [63]. This may be associated with a greater incidence of various renal dysfunction, including chronic kidney disease [64]. The major physiological responses of the kidneys to obesity include increased glomerular filtration rate, renal plasma flow, filtration fraction and tubular sodium reabsorption. Considering obesity as a risk factor for kidney disease, obesity-related glomerulopathy (ORG) was distinguished, as a disease connected with excess body weight. The incidence of ORG is increasing in parallel with the worldwide obesity epidemic. The most common symptom is subnephrotic proteinuria in the urinalysis [65]. The important is that studies have shown that weight loss led to a reduction in proteinuria in patients with ORG [66].

Infections

Numerous studies have examined whether obesity is associated with an increased incidence of urinary tract infections. Many of them show that overweight people are at greater risk of developing **UTI (urinary tract**

infection) and pyelonephritis [67]. A similar dependence was observed in children [68].

The important fact is that adipose tissue is also actively involved in inflammation and immunity. Leptin is also involved in these mechanisms. A genetic defect with leptin deficiency in mice, which causes a severe obesity phenotype, is associated with impaired phagocytic functions and a decrease in T-lymphocyte function. In these mice, this increases the susceptibility to bacterial infections, e.g., Listeria monocytogenes, Klebsiella pneumoniae, etc. Leptin deficiency is associated with susceptibility for infections in both animals and humans [69].

Recurrent urinary tract infections may be associated with other causes, therefore in such patients, despite excessive body weight, in-depth diagnostics should be considered.

Renal cell cancer (RCC)

Overweight and obesity strongly correlate with the incidence of kidney cancer. Meta-analyzes suggest that overweight / obesity increases the risk of kidney cancer in both men and women [70]. Abdominal obesity can also be a risk factor in kidney cancer.

The mechanism of this dependency has not been fully explained. Also, as in the case of kidney stones, the presence of kidney tumors is associated with hyperinsulinemia. Insulin resistance, which is common in obesity and leads to an increase in IGF-1 levels. IGF-1 plays a role in carcinogenesis and leads to an increased risk of cancer [71].

An interesting phenomenon related to excess body weight and kidney cancer is the so-called obesity paradox. The obesity paradox is based on the observation that obesity is a risk factor for developing kidney cancer, but obese patients have an increased survival compared to normal BMI. However, the biological mechanisms underlying this observation are not yet fully understood. Recently, aspects of the tumor microenvironment that vary with BMI in tumor and peritumoral adipose tissue have been discovered, which may contribute to the apparent survival advantage in obese patients [72].

Diagnostics

According to the WHO, overweight in adults is diagnosed if the BMI value is within the range of 25–30 kg/m^2, while the diagnosis of obesity is based on a BMI ≥ 30 kg/m^2. Adults should be screened annually for overweight or obesity using BMI calculations. Please note that BMI is not an ideal indicator. In athletes, the elderly and the sick as well as the disabled (especially physically disabled) and people with sarcopenia, BMI is not an appropriate anthropometric indicator to assess the presence of excessive amounts of adipose tissue. The reason for this is the different ratio of muscle and fat mass in these people compared to a statistically healthy person. Based on the International Diabetes Federation (IDF) diagnostic criteria in adult Europeans with a waist circumference greater than (or equal to) 94 in men and 80 in women, visceral obesity can be diagnosed.

Patients with excessively abnormal body weight should be under the constant supervision of family doctors. Since an obese patient has an increased risk of diseases of the genitourinary system, the presence of this factor should be considered when reporting symptoms and informing the patient about the importance of weight loss at every stage of the diagnosis. One of the first and most important diagnostic tools that can guide us to the correct diagnosis is medical record.

A meta-analysis of 10 studies confirmed that women with a positive clinical history (presence of coughing, sneezing, walking or running as initiators of an incontinence episode) had a 74% chance of stress urinary incontinence, while women with a negative clinical history had a 34% chance [73]. There are also international standardized questionnaires that are routinely used in urinary incontinence clinical trials, including Incontinence, Impact Questionnaire (IIQ), King's Health Questionnaire (KHQ) and Urogenital Distress Inventory (UDI) [74]. In addition to the history and questionnaires, the initial evaluation includes physical examination, micturition diary, general urine examination, postvoidal residual urine assessment in ultrasound, and a pad test. Diagnostics is based mainly on the initial evaluation and in some patients on urodynamic tests

[75]. Doctors should not perform cystoscopy on healthy women who are considering surgery to evaluate stress urinary incontinence unless there are concerns about abnormalities in the urinary tract [76].

In the event of alarm symptoms such as haematuria, a tumor palpable through the abdominal wall, a positive family history, pain in the lumbar region, fever, and refractory arterial hypertension, the diagnosis should always be significantly deepened. A patient with unexplained hematuria should always be referred to a urologist for cystoscopy. There are no specific guidelines for the diagnosis of a patient with excess body weight. However, it should be remembered that excess body weight is a factor that increases the risk of both chronic kidney disease and urinary tract cancer. It is also important to inform at every stage of the diagnosis how important weight loss is. Weight reduction in obesity not only has a nephroprotective effect [77] but it is one of the main recommendations in the treatment of urinary incontinence.

Obesity treatment

One of the ways of losing weight is diet therapy, which assumes achieving a net energy deficit [78]. Obtaining a caloric deficit is possible in several ways, including by appropriate composition of macronutrients, limiting calories, changing meals [79].

In addition to a reduced calorie diet and exercise, drug treatment of obesity is recommended.

Orlistat

Orlistat acts on pancreatic lipases by irreversibly inhibiting their action, which reduces the absorption of free fatty acids and their excretion in the faeces. Orlistat reduces BMI, body weight, waist circumference, and cholesterol levels. Better glycemic control has been shown in people with diabetes. The most common side effect is gastrointestinal disturbance, which is often the reason why therapy is discontinued [80].

Liraglutide

Liraglutide is an agonist at the GLP-1 (glucagon-like peptide-1) receptor. GLP-1 is a released incretin hormone that is released from the gastrointestinal tract in response to the ingestion of glucose and fat. It works peripherally, by slowing down the passage of the digestive tract, and centrally, suppressing the appetite [81].

Naltrexone/bupropion

Naltrexone is an opioid antagonist used to treat opioid and alcohol dependence. Bupropion inhibits the uptake of norepinephrine and dopamine and is used as an adjunct to quitting therapy. When combined as a combined drug, they reduce appetite [82].

When other obesity treatments have failed, bariatric surgery is the treatment of choice [83]. Bariatric surgery is recommended for patients who meet the following criteria:

- BMI ≥ 40 kg/m^2
- BMI ≥ 35 kg/m^2 with associated comorbidities that could be improved with weight loss
- BMI of 30–34.9 kg/m^2 who have recent-onset T2DM
- other weight loss options have been explored but have failed
- patient is receiving or will receive intensive management in a tier 3 service (a service-based weight loss program)
- patient is fit for anaesthesia and surgery propos
- patient shows commitment to long-term follow-up [84].

Proposed methods of bariatric treatment:

- **Adjustable Laparoscopic Gastric Banding** – is placed around the upper part of the stomach. The band narrows the stomach's lumen, limiting the amount of food that can be swallowed.
- **Roux-en-Y gastric bypass** – involves the simultaneous reduction of the stomach and intestinal absorption, thus enabling effective weight reduction. The procedure begins with the creation of a small stomach just below the cardia, i.e., connecting the stomach with the esophagus. The intestine is then divided into two parts

– the lower (Roux loop), which is then pulled up and attached to the new stomach, and the higher (enzyme loop), which is attached to the distal end of the Roux loop. From now on, gastric juices and digestive enzymes will flow through the enzyme loop to the Roux loop, where they will combine with the food and digestion will begin there.

- **Sleeve gastrectomy** – during the operation, 80% of the stomach is removed, while the 'sleeve' is left, in the narrow medial part. The reduced size of the stomach causes a reduction in the volume of food consumed.
- **Biliopancreatic diversion with a duodenal switch.**
- **Revision and reversal of bariatric surgery** [84].

Discussion

This article aims to emphasize the importance of obesity in the development of disorders of the genitourinary system, which not only increases morbidity and mortality, but also reduces the quality of life. Given that obesity rates are increasing worldwide, especially among women of childbearing age, this creates not only a clinical problem, but also a financial problem in health care [85]. That is why primary prevention and obesity prevention are so important, which will relieve health care. Moreover, the results of studies on increased fertility after weight loss are controversial, increasing the importance of primary prevention and promoting a healthy lifestyle [86]. Moreover, studies conducted on a group of men who underwent bariatric surgery showed a decrease in the number of already disturbed sperm. On the other hand, sperm parameters of men treated with diet and exercise improved [87].

Increased hemodynamics and the metabolic burden associated with obesity aggravate chronic kidney disease that has already developed [88]. What is important – non-surgical weight loss interventions reduce proteinuria and blood pressure and appear to prevent further deterioration of renal function [89]. Nevertheless, most patients believe that their

excess weight adversely affects their health and are aware of its consequences. They consider losing weight to be too difficult. Patients often mention the lack of motivation or money as a problem [90]. Therefore, it is most important that weight loss should be the mainstay of treatment of urogenital disorders in patients with excess body weight. Since health problems are a less motivating aspect for young adults than for middle-aged **adults** [91], the increased emphasis on primary prevention in young people seems to be the most effective idea, while reducing costs for the treatment of obesity complications.

Conclusions

Obesity and overweight are a disease of civilization, which in women of childbearing age may have a significant impact on their fertility, and thus may cause suffering due to the lower likelihood of pregnancy. The chronic inflammatory state of obesity can directly affect the normal function of the ovaries. Energy balance is closely related to the reproductive system and is controlled by many hormones: adipokines, cytokines and growth factors. The effect on reproductive function disorders occurs through neuroendocrine mechanisms that interfere with the functions of the ovaries and proper ovulation. Endocrine disorders, mainly decreased levels of gonadotropins and estradiol, also have a negative effect on reduced fertility. In turn, decreased fertility in obese men results from many genetic, physical and hormonal mechanisms that lead to abnormal sperm parameters and erectile dysfunction.

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