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# Relationship between sexual function, body mass index and levels of sex steroid hormones in young men

## Zależność pomiędzy funkcją seksualną a wskaźnikiem masy ciała i stężeniem hormonów steroidowych u młodych mężczyzn

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### Abstract

**Introduction:** In older men, sexual disorders may be the result of a decrease in testosterone and an increase in sex hormone binding globulin (SHBG) serum levels. Although obesity may enhance the decline of testosterone, it is also the cause of metabolic disorders, which are additional risk factors of erectile dysfunction. The purpose of this study was to investigate whether elevated body weight is associated with decreased serum testosterone concentrations and reduced sexual function in young men.

**Material and methods:** Data on general health, medication, depressive symptomatology and sexual life was obtained from 136 men aged 20–49 years. Blood levels of LH, total testosterone (TT), dehydroepiandrosterone sulfate (DHEA-S), oestradiol, SHBG, total cholesterol, LDL- and HDL-cholesterol, and triglycerides were determined. Body mass index (BMI), waist to hip ratio (WHR) and free testosterone index (FTI) were calculated.

**Results:** A significantly reduced occurrence of sexual fantasies, morning erections and erectile function scores was observed in the oldest group compared to the youngest men with normal BMI, although orgasmic function was unchanged. A significant decrease in TT serum levels was observed in obese 30-year-olds compared to men with normal BMI, while in obese 40-year-olds decreased LH and SHBG levels were also found. No differences in the levels of lipids and sexual achievements were found among men with different BMI. However, erectile function and morning erections significantly negatively correlated with age, BMI and WHR, and positively with FTI, but not with other studied hormones and lipids.

**Conclusions**: In young men, obesity can lead to a deterioration of erectile function as a result of lower testosterone levels as the only reason. **(Endokrynol Pol 2014; 65 (3): 203–209)**

**Key words:** *men; sexual function; testosterone; erectile dysfunction; obesity*

### Streszczenie

**Wstęp:** U starszych mężczyzn zaburzenia seksualne mogą być następstwem zmniejszania się stężenia testosteronu i wzrostu stężenia globuliny wiążącej steroidy płciowe (SHBG) we krwi. Otyłość może nasilać obniżanie stężenia testosteronu, ale jest także przyczyną zaburzeń metabolicznych, które stanowią dodatkowe ryzyko pojawienia się zaburzeń erekcji. Celem pracy było zbadanie, czy nadmierna masa ciała jest związana z obniżonym stężeniem testosteronu i pogorszeniem funkcji seksualnych już u młodych mężczyzn.

**Materiał i metody:** Dane na temat stanu zdrowia, stosowanych leków, objawów depresji i życia seksualnego uzyskano od 136 mężczyzn w wieku 20–49 lat. We krwi oznaczono stężenia LH, testosteronu całkowitego (TT), siarczanu dehydroepiandrosteronu (DHEA-S), estradiolu, SHBG, cholesterolu całkowitego, cholesterolu LDL i HDL oraz triglicerydów. Wyliczono wskaźniki: masy ciała (BMI), talia/biodra (WHR) i wolnego testosteronu (FTI).

**Wyniki:** Znamienne zmniejszenie częstości fantazji seksualnych i porannych erekcji oraz pogorszenie jakości erekcji, ale nie zdolności do osiągania orgazmu, obserwowano w najstarszej grupie badanych w porównaniu z grupą najmłodszych mężczyzn z prawidłowym BMI. Znamienne zmniejszenie stężenia TT stwierdzono u otyłych 30-latków w porównaniu z mężczyznami z prawidłowym BMI, a u 40-latków dodatkowo stwierdzono zmniejszenie stężenia LH i SHBG. Nie stwierdzono znamiennych różnic w stężeniu lipidów, a także w jakości reakcji seksualnych u mężczyzn z różnym BMI. Jednakże, jakość erekcji oraz częstość występowania porannych erekcji znamiennie negatywnie korelowały z wiekiem, BMI i WHR, a pozytywnie z FTI, ale nie z innymi badanymi hormonami i lipidami.

**Wnioski:** U młodych mężczyzn otyłość może prowadzić do pogorszenia erekcji na skutek obniżenia stężenia testosteronu jako jedynej przyczyny. **(Endokrynol Pol 2014; 65 (3): 203–209)**

**Słowa kluczowe:** *mężczyźni; funkcje seksualne; testosteron; zaburzenia wzwodu; otyłość*

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## Introduction

The incidence of obesity has increased dramatically over the last 30 years, especially in highly industrialised countries, where the lifestyle commonly includes a high calorie diet and low physical activity. A progressive decline of the age threshold of subjects with overweight and obesity has been observed. In the age group under 18 years, the prevalence of excessive body weight tripled in the last decade of the 20th century and now in Europe constitutes 16–22% [1]. In men, the frequency of obesity in the European population is approximately 15% [2]. The European Male Ageing Study (EMAS) revealed the prevalence of obesity among 2,736 men aged 40–70 years to be 24% [3]. In turn, our previous study revealed that as many as 50% of young men (20–39 years old) had excessive body weight: 36% were overweight and 14% were obese [4].

Links between obesity and cardiovascular artery disease, hypertension and diabetes mellitus are well documented in both sexes [5–7]. Obesity in men has also been shown to be associated with low plasma testosterone concentrations and erectile dysfunction (ED) [8, 9]. Decreased testosterone level and visceral adipose tissue accumulation in the ageing male probably represent the most important factors for comorbidities and mortality from cardiovascular disease [10, 11]. Hypoandrogenism in men is manifested, among other things, by a lack of interest in sex (no sexual fantasies), lack of spontaneous, morning erections, ED during sexual stimulation and difficulty in achieving orgasm [12, 13]. EMAS revealed that among men above 40, those with the lowest levels of total and free testosterone reported significantly lower sexual function compared to men with the highest testosterone levels [14]. Androgen deficiency in older men creates a clinical syndrome called late onset hypogonadism (LOH), which is associated with multiple end-organ deficits [15]. However, as this deficit of testicular function is not yet present in young men, the direct effect of obesity on androgen action may be clearly visible.

The purpose of this study was to investigate whether elevated body weight is associated with decreased serum testosterone concentrations and lower sexual function already present in younger men.

## Material and methods

### *Subjects and Study Design*

The study was performed after the approval of the Bioethical Committee of the Medical University of Lodz, Poland. A group of 300 men was randomly recruited from the Lodz city population register. The only criterion for selection was for the subject to be aged between 20–49 years. Subjects were invited by a letter to attend a screening visit at the Department of Andrology and Reproductive Endocrinology, Medical University of Lodz. The overall response rate for participation was 46%. Men who agreed to participate in the study (136 subjects, mean age 35.5 ± 10 years) were divided into age bands: 20–29 (44 subjects), 30-39 (36 subjects) and 40–49 (56 subjects).

### *Anthropometry*

Body weight and height, waist and hip circumference were measured using standard procedures. Calculated were body mass index (BMI = weight [kg]/height$^2$ [m]) and waist to hip ratio (WHR) [cm]. Each age band was divided according to BMI into a group with normal weight (18.5–24.9 kg/m$^2$), overweight (≥ 25.0–29.9 kg/m$^2$) and obesity (> 30.0 kg/m$^2$).

#### Hormone and lipid measurements

Single venous blood samples were taken in the morning hours (8.00–10.00) in a fasting state, 12 hours after the last meal. Serum was separated and stored at –80°C until assayed at the end of the study, not longer than six months.

Serum determinations of luteinising hormone (LH, normal range: 1.2–10.0 IU/L), total testosterone (TT, normal range: 7.5–28.2 nmol/L), dehydroepiandrosterone sulfate (DHEA-S normal range: 30.0–333.0 $\mu$g/dL), oestradiol (normal range: 19.7–242.0 pmol/L), and sex hormone binding globulin (SHBG, normal range: 13.0–71.0 nmol/L) were performed. All hormones were measured using chemiluminescence immunoassay (Immulite 1000, DPC, USA). Detection limits were for: LH — 0.1 IU/L, TT — 0.5 nmol/L, DHEA-S — 3 $\mu$g/dL, oestradiol — 20 pmol/L, and SHBG — 0.2 nmol/L. Free testosterone index (FTI) was calculated as follows: total testosterone [nmol/L]/SHBG [nmol/L] × 100.

Serum levels of total cholesterol (TC, normal range: 3.0–5.2 mmol/l), LDL-cholesterol (LDL-C, normal range: < 2.6 mmol/L), HDL-cholesterol (HDL-C, normal range: > 1.0 mmol/L), triglycerides (TG, normal range: < 1.7 mmol/L), and glucose (normal range: 3.3–5.5 mmol/L) were determined with the use of enzymatic methods (Cobas Integra 800, Roche Diagnostics, Poland).

#### Questionnaires

Subjects were asked to complete an interviewer-assisted questionnaire gathering information on general health, medications and lifestyle. General health was scored from 0, when very good, to 3, when very bad. To measure depressive symptomatology, the Beck Depression Inventory II (BDI-II) questionnaire was used [16].

To evaluate the quality of the sex life of the subject, a separate unassisted questionnaire was completed.

This questionnaire was validated and used as a scientific tool in the EMAS study in which we participated [17]. The questionnaire comprised a set of multiple choice questions. Questions concerning the previous four weeks were as follows: 1) Do you have a sexual partner? (Answer: yes, no); 2) Are you able to achieve and maintain an erection sufficient for sexual intercourse? (Answer: never, almost never, sometimes, usually, almost always/always; score 1–5); 3) How often do you reach orgasm during sexual stimulation? (Answer: never, almost never, sometimes, usually, almost always/always; score 1–5); 4) How often do you wake up with an erection? (Answer: never, 1 time/month, 2–3 times/month, 1 time/week, 2–3 times/week, 4–6 times/week, 1 time/day, > 1 time/day; score 0–7); and 5) How often do you think about sex (sexual fantasies)? (Answer: never, 1 time/month, 2–3 times/month, 1 time/week, 2–3 times/week, 4–6 times/week, 1 time/day, > 1 time /day; score 0–7).

### *Statistical analysis*

All statistical analyses were performed using Statistica for Windows PL software, version 10.0 (Statsoft Inc., Tulsa, OK, USA). Mean ± standard deviations (SD) were used to express group data. The ANOVA Kruskal-Wallis test was applied for the comparison of nonparametric variables between groups, after the distribution of values had been verified by the Shapiro-Wilk test. Correlations were examined using Spearman's rank correlation analysis. $P < 0.05$ was considered significant.

## Results

One or more morbidities were reported by 16 men. The most frequent were gastrointestinal ulcer (50%) and hypertension (37.5%). General health was significantly worse in men aged 40–49 (score 2.0 ± 1.3; $p < 0.001$) compared to younger ones (score for 20-year-olds: 1.1 ± 1.5; score for 30-year-olds: 1.8 ± 1.5). BDI-II revealed that one 20-year-old man presented symptoms of severe depression and four men aged 40 presented with moderate depression. All of them reported adverse life events during the previous weeks. Total scoring of BDI-II was not related to age, general health scoring, blood lipids and sex hormones serum levels. Details on general health and BDI-II are presented in our previous study on the same group of men presented by Kramek et al. [18]. Nobody used antidepressants, psychotropic drugs, narcotics, lipid-lowering medications or antiandrogens. None was denoted as an alcoholic or drug addict. A total of 62.5% of men were current or former smokers (20-year-olds — 52.3%, 30-year-olds — 63.9%, 40-year-olds — 65.2%). None used a special diet.

In the whole group, 38.8% of people led a sedentary life and did not show any other physical activity. Among professional working men, 74.4% had jobs that involved sitting.

Results of hormonal determinations and serum levels of lipids in men between 20 and 49 years of age with different BMI values are shown in Table I. Among men with a normal BMI, a significant increase in LH, SHBG and all lipid serum levels was observed in 40-year-olds compared to 20-year-olds. A significant decrease of FTI and DHEA-S, but not TT serum level, was visible in men 30 and above. It was also shown that oestradiol serum level is significantly lower in 40-year-old men.

In obese men, a significant decrease in TT serum levels was found in 30-year-olds compared to men with normal BMI, while decreased LH and SHBG levels were also found in 40-year-olds with higher BMI. A trend towards increased oestradiol and DHEA-S with growing weight was visible, but not statistically significant. No differences were found in the levels of lipids among men with different BMI values.

A total of 18% of 20-year-olds, 8% of 30-year-olds and 2% of 40-year-old men reported a lack of sexual partner. Nobody reported severe dysfunction of erection or orgasm i.e. achievement "never" or "almost never". Table II shows the results of the sex life assessment and their comparisons between age groups and between men with different BMI scores. Less frequent sexual fantasies, reduced erectile function, and lower morning erection scores were observed in the oldest group compared to the youngest men, although orgasmic function was unchanged. No significant differences were found between men with different BMI values in each age group.

The correlations between age, FTI, and sexual fantasy, erectile function and morning erection results are presented in Table III. Erectile function and morning erections significantly negatively correlated with age, BMI and WHR, as did sexual fantasies with age and BMI. Sexual fantasies, erectile function and morning erections positively correlated with FTI. No correlations between sexual life parameters and other hormones were found, neither with lipids. Orgasmic function was not correlated with any studied parameters.

## Discussion

Assessment of male sexual function has been the subject of intense research in recent years. As the incidence of sexual disorders increases with advancing age, most studies have been devoted to older men. However, the risk factors for cardiovascular disease, as well as decreased testosterone serum concentration, may appear early in life, even in prepubertal children [19]. It has been shown that one of these factors is increased body mass. Our previous study found that BMI and

**Table I.** *Results of hormonal determinations and serum levels of lipids in men between 20 and 49 years of age with normal body mass index (BMI < 25 kg/m²), overweight (BMI 25–29.9 kg/m²) and obesity (BMI ≥ 30 kg/m²)*

**Tabela I.** *Wyniki oznaczeń hormonalnych i lipidów we krwi mężczyzn w wieku 20–49 lat z prawidłową masą ciała (BMI < 25 kg/m²), nadwagą (BMI 25–29,9 kg/m²) i otyłością (BMI ≥ 30 kg/m²)*

| Hormones and lipids | Age band (years) | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | 20–29 | | | 30–39 | | | 40–49 | | |
| | BMI < 25 kg/m² n = 28 | BMI 25–29.9 kg/m² n = 10 | BMI ≥ 30 kg/m² n = 6 | BMI < 25 kg/m² n = 13 | BMI 25–29.9 kg/m² n = 17 | BMI ≥ 30 kg/m² n = 6 | BMI < 25 kg/m² n = 26 | BMI 25–29.9 kg/m² n = 22 | BMI ≥ 30 kg/m² n = 8 |
| LH [IU/L] | 4.7 ± 1.6 | 4.6 ± 2.1 | 5.6 ± 1.3 | 4.5 ± 2.4 | 4.3 ± 1.5 | 3.6 ± 2.0 | 6.4 ± 2.5† | 5.0 ± 1.9* | 3.5 ± 0.9* |
| TT [nmol/L] | 21.1 ± 6.6 | 18.2 ± 4.4 | 16.8 ± 3.7 | 20.0 ± 4.9 | 18.7 ± 5.3 | 14.4 ± 6.0* | 19.0 ± 3.8 | 17.4 ± 6.2* | 14.5 ± 4.1* |
| FTI | 85.8 ± 30.2 | 84.9 ± 35.4 | 84.8 ± 22.0 | 74.1 ± 23.9† | 63.0 ± 40.4 | 63.9 ± 19.9 | 47.7 ± 14.9† | 45.7 ± 12.9 | 42.5 ± 12.2 |
| DHEA-S [μg/dL] | 271.6 ± 109.7 | 297.2 ± 106.2 | 289.0 ± 108.5 | 241.8 ± 95.3† | 258.2 ± 102.3 | 263.8 ± 91.9 | 227.1 ± 84.5† | 232.4 ± 76.4 | 233.0 ± 113.9 |
| Oestradiol [pmol/L] | 111.0 ± 47.4 | 125.1 ± 39.9 | 137.4 ± 37.3 | 95.4 ± 26.7 | 102.1 ± 44.4 | 107.8 ± 38.9 | 84.5 ± 28.8† | 87.4 ± 12.2 | 92.9 ± 27.8 |
| SHBG [nmol/L] | 24.5 ± 8.9 | 23.8 ± 8.9 | 20.8 ± 6.9 | 28.9 ± 11.7 | 27.5 ± 13.8 | 26.9 ± 10.1 | 52.6 ± 21.3† | 39.6 ± 16.9* | 33.1 ± 10.1* |
| TC [mmol/L] | 4.0 ± 1.0 | 4.3 ± 0.6 | 4.8 ± 0.7 | 4.5 ± 0.6 | 4.9 ± 1.2 | 5.3 ± 1.2 | 6.0 ± 0.9† | 6.1 ± 0.9 | 6.3 ± 0.7 |
| HDL-C [mmol/L] | 1.5 ± 0.3 | 1.5 ± 0.4 | 1.3 ± 0.4 | 1.6 ± 0.3 | 1.4 ± 0.4 | 1.4 ± 0.5 | 1.7 ± 0.3† | 1.6 ± 0.2 | 1.6 ± 0.2 |
| LDL-C [mmol/L] | 2.1 ± 0.9 | 2.3 ± 0.6 | 2.5 ± 0.6 | 2.3 ± 0.6 | 2.8 ± 0.9 | 3.2 ± 1.4 | 3.4 ± 1.3† | 3.4 ± 1.2 | 3.5 ± 1.1 |
| TG [mmol/L] | 0.9 ± 0.4 | 1.0 ± 0.3 | 2.2 ± 1.2 | 1.2 ± 0.8 | 1.6 ± 1.3 | 2.0 ± 1.6 | 1.6 ± 0.8† | 1.9 ± 1.2 | 2.3 ± 1.6 |

*p < 0.05 *vs.* BMI < 25 kg/m² in each age band; †p<0.05 *vs.* BMI < 25 kg/m² in 20-year-olds; Mann-Whitney U-test; n — number of subjects; LH — luteinising hormone; TT — total testosterone; FTI — free testosterone index; DHEA-S — dehydroepiandrosterone sulfate; SHBG — sex hormone binding globulin; TC — total cholesterol; HDL-C — HDL-cholesterol; LDL-C — LDL-cholesterol; TG — triglycerides

**Table. II.** *Results (mean ± standard deviation scores) of sexual life assessment in men between 20 and 49 years of age with normal body mass index (BMI < 25 kg/m²), overweight (BMI 25–29.9 kg/m²) and obesity (BMI ≥ 30 kg/m²)*

**Tabela II.** *Wyniki (średnia ± odchylenie standardowe punktacji) oceny parametrów życia seksualnego u mężczyzn w wieku 20–49 lat z prawidłową masą ciała (BMI < 25 kg/m²), nadwagą (BMI 25–29,9 kg/m²) i otyłością (BMI ≥ 30 kg/m²)*

| Parameters of sexual life | Age band (years) | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | 20–29 | | | 30–39 | | | 40–49 | | |
| | BMI < 25 kg/m² n = 28 | BMI 25–29.9 kg/m² n = 10 | BMI ≥ 30 kg/m² n = 6 | BMI < 25 kg/m² n = 13 | BMI 25–29.9 kg/m² n = 17 | BMI ≥ 30 kg/m² n = 6 | BMI < 25 kg/m² n = 26 | BMI 25–29.9 kg/m² n = 22 | BMI ≥ 30 kg/m² n = 8 |
| Sexual fantasies | 5.9 ± 1.4 | 5.3 ± 1.3 | 6.3 ± 1.6 | 6.3 ± 1.0 | 5.1 ± 1.3 | 6.4 ± 0.9 | 4.6 ± 1.2* | 4.5 ± 1.9* | 4.6 ± 1.5* |
| Erectile function | 3.9 ± 0.5 | 3.6 ± 0.9 | 3.8 ± 0.4 | 3.8 ± 0.7 | 3.7 ± 0.5 | 3.8 ± 0.8 | 3.3 ± 0.6* | 3.4 ± 0.6* | 3.4 ± 0.5* |
| Morning erections | 3.8 ± 1.3 | 3.3 ± 2.2 | 3.3 ± 2.9 | 3.2 ± 1.6 | 3.0 ± 2.3 | 3.0 ± 2.7 | 2.8 ± 1.9* | 2.7 ± 1.3* | 2.6 ± 1.8* |
| Orgasmic function | 4.6 ± 1.2 | 4.7 ± 0.4 | 4.6 ± 0.5 | 4.4 ± 1.5 | 4.2 ± 1.3 | 4.2 ± 1.8 | 4.3 ± 1.3 | 4.3 ± 1.3 | 4.7 ± 0.5 |

*p < 0.05 *vs.* 20-year-old with BMI < 25 kg/m², ANOVA Kruskal-Wallis test; n — number of subjects

WHR levels increase in men between the 20th and 40th year of life [4], and this fact was confirmed in the present study. Moreover, it was seen that the number of adverse risk factors for cardiovascular disease rapidly increases after 40. It was visible that after the 40th year of life, men had significantly worse lipid parameters (increased

**Table III.** *Results of significant Spearman's correlation between parameters of sexual life and age, BMI, WHR, FTI in 136 men aged 20–49 years*

**Tabela III.** *Wyniki znamiennych statystycznie korelacji Spearmana pomiędzy parametrami życia seksualnego a wiekiem, BMI, WHR i FTI u 136 mężczyzn w wieku 20–49 lat*

| Parameters of sexual life | Age r | FTI r | BMI r | WHR r |
|---|---|---|---|---|
| Sexual fantasies | –0.43* | 0.28* | –0.21* | ns |
| Erectile function | –0.34* | 0.27* | –0.20* | –0.29* |
| Morning erections | –0.25* | 0.21* | –0.22* | –0.38* |

*p < 0.05; r — Spearman's correlation coefficient; FTI — free testosterone index; BMI — body mass index; WHR — waist to hip ratio; ns — not significant

serum levels of TC, LDL-C and TG), but no correlation was found between serum concentration of lipids and BMI or WHR in each age group. Although other studies note that dyslipidaemia accompanies obesity and has been linked to ED in older men [20–22], it may be the case that the participants of our study were younger and in better health.

The present study reveals that TT and FTI declines, and SHBG increases, with age from the 30th year of life while the mean hormone levels remain within the physiological range. Longitudinal studies in ageing males over 40 report that the decline in free testosterone is caused by a rise in SHBG [9, 23, 24]. However, the results of the present study indicate that mean TT level is lower in men with excessive body weight compared to men with a normal BMI, even by the age of 30. In 40-year-olds, LH and SHBG levels were also found to be significantly lower. The same observation in older men has been made by several prospective studies [4, 9, 25, 26]. Wu et al. [9] observe that the effect of increasing BMI and waist size on circulating testosterone was more substantial than that of age. The change in BMI from non-obese to obese may be equivalent to a 15-year fall in TT. One possible explanation of this phenomenon may be a failure of the hypothalamus and/or pituitary to compensate for hypoandrogenaemia. Weight gain appears to lead to blunted LH secretion, but with unchanged serum levels. Moreover, obesity is associated with lower SHBG as a result of insulin resistance, with the high levels of circulating insulin inhibiting hepatic SHBG production [27, 28]. Camacho et al. [3] suggested that fluctuations in SHBG concentrations can provide metabolic signal-linking variations to hypothalamo-pituitary-testicular axis function in adiposity, thereby regulating androgen bioactivity through insulin signalling. This is supported by the recent finding of Coviello et al. [29] that circulating SHBG is influenced by variations in genes involved in the regulation of lipid and carbohydrate metabolism. In addition, the delivery of bound hormones at target tissue sites is a further possibility of the active role played by SHBG in the regulation of sex steroid bioavailability [30]. In addition to peripheral and central insulin resistance, obesity is associated with proinflammatory cytokine production (TNF- and IL-6) from adipocytes and central nervous system endocannibinoid release, which can inhibit hypothalamo-pituitary-testicular axis function [31–33]. Increased plasma levels of leptin were found to be secreted from adipocytes in obese subjects. Leptin receptors are present in testicular Leydig cells and leptin may play a role in reduced androgen levels in obese men [34]. Lima et al. [35] found that massively obese men (BMI > 35 kg/m$^2$) had consistently low free testosterone levels. The authors concluded that functional decrease of LH pulse amplitude and serum LH levels, as well as the possible negative action of excess circulating leptin on steroidogenesis, may be related to the decreased androgen levels in obese men. In older men, multiple functional alterations in the action of reproductive hormones linked to distinct risk factors are shadowed by the progressive testicular impairment associated with increasing age [9]. In younger men, however, obesity may be the only risk factor for testosterone decline and poor sexual function.

Obese men have been shown to have elevated circulating levels of oestradiol predominantly derived from the aromatisation of circulating testosterone, mostly in adipose tissue where the enzyme aromatase is present in larger amounts [36]. The overall rate of aromatisation of testosterone to oestradiol increases with body mass. The non-significant trend for oestradiol serum level to increase with growing weight was also visible in our study. This phenomenon may result in a greater degree of testosterone deficiency by acting at the hypothalamus to decrease GnRH pulse frequency, at the pituitary to decrease LH secretion, and at the testicular level to inhibit testosterone biosynthesis [37, 38]. It has been found that oestradiol serum levels show a positive cross-sectional relationship with age and depressed mood related to declining sexual function in ageing men [39]. However, the level of serum oestrogen in men has been also reported to decline with age [40], and our study revealed the same observation in young subjects.

The results of the sexual parameter assessment, such as those concerning sexual fantasies, morning erections and erectile function, did not differ significantly between 20- and 30-year-old men. Only 40-year-olds showed significant deterioration of sexual function compared to 20-year-olds. General health worsened with age, but there were no serious diseases. Psychological causes were of minor importance. There were also no differences associated with excessive body mass. In turn, significant correlations were found between the above mentioned sexual parameters and BMI or WHR.

The results of our study are in agreement with other studies on cohorts of older men showing that several measures of obesity and central adiposity are significantly associated with ED prevalence [8, 41–43]. Orgasmic function was not correlated with any studied parameters. It is known that the incidence of OD is low (3–10% of men with sexual dysfunction) and increases with age [44–46]. Besides this, the pathophysiology of OD is not clear and needs more research.

Obesity belongs to the group of modifiable factors which can be reduced or completely eliminated. Physical activity and a low-calorie diet allow weight loss, self-image improvement and better quality of life, as well as better sexual function and satisfaction [47–50]. There is, however, limited data as to whether reduction in body weight by itself would improve sexual function. In the Massachusetts Male Aging Study (MMAS), ten year follow up data suggested that obese men aged 40 to 70, regardless of weight loss, had a greater risk of developing ED [51]. In turn, in a younger group of men (35–55 years) Esposito et al. [52] reported improvement of erectile function after at least 10% weight loss, healthy diet and increased exercise. Moreover, results are emerging from randomised and observational studies demonstrating that lifestyle changes and weight loss are associated with better sexual function, even in individuals with diabetes mellitus and in older men [46, 53, 54], which may be the result of increased testosterone levels. Camacho et al. [3] showed that weight gain was associated with suppression of TT, free testosterone and SHBG, while weight loss was associated with an increase in TT, free testosterone, SHBG and LH in men taking part in EMAS. Possible explanations for the difference between older and younger men with regard to the responsiveness of erectile function to weight loss include increased prevalence of cardiovascular disease, fewer urinary tract disorders, and other comorbidities at older ages. Corona et al. [55] revealed that more than 50% of EMAS subjects reported the presence of one or more common morbidities. Around 30% of men reported ED and 6% reported severe OD, both of which were closely associated with age and concomitant morbidities. Hence, sexual health declined while concomitant morbidities increased as a function of age.

## Conclusions

The general increase in obesity among men may result in more individuals with sexual dysfunction. ED in older men is the result of the decrease in testosterone and increase in SHBG serum levels. Obesity by itself may enhance the decline of testosterone but also represents a severe risk of cardiovascular disease and diabetes mellitus, which are also risk factors of ED. In younger men, excessive weight is not a reason for comorbidities, but may be the cause of lower testosterone action leading to worse sexual function in men over 40. Obesity is a reversible phenomenon. Hence, it is important to undertake preventive action in young people to reduce excessive weight and, in this way, reduce the risk of further sexual dysfunction.

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