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ORIGINAL ARTICLE

Male Infertility

Body mass index effects sperm quality: a retrospective study in Northern China

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Excess weight and obesity have become a serious problem in adult men of reproductive age throughout the world. The purpose of this retrospective study was to assess the relationships between body mass index and sperm quality in subfertile couples in a Chinese Han population. Sperm analyses were performed and demographic data collected from 2384 male partners in subfertile couples who visited a reproductive medical center for treatment and preconception counseling. The subjects were classified into four groups according to their body mass index: underweight, normal, overweight, and obese. Of these subjects, 918 (38.3%) had a body mass index of >25.0 kg m^{-2}. No significant differences were found between the four groups with respect to age, occupation, level of education, smoking status, alcohol use, duration of sexual abstinence, or the collection time of year for sperm. The results clearly indicated lower sperm quality (total sperm count, sperm concentration, motile sperm, relative amounts of type A motility, and progressive motility sperm [A + B]) in overweight and obese participants than in those with normal body mass index. Normal sperm morphology and sperm volume showed no clear difference between the four groups. This study indicates that body mass index has a negative effect on sperm quality in men of subfertile couples in a Northern Chinese population. Further study should be performed to investigate the relationship between body mass index and sperm quality in a larger population.
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Keywords: body mass index; male infertility; obesity; sperm quality

INTRODUCTION

Infertility is a serious health problem that affects about 10% of all families worldwide and possibly more in developing countries.[1] Although assisted reproduction technology has addressed this issue to some extent, it does not solve the underlying problem for infertile couples. Identifying risk factors for subfertility is a serious task for clinicians and researchers.

Obesity is a global health problem that has reached epidemic levels not only in Western countries but also in developing countries.[2] In 2008, one in every three adults in the world was overweight and one in every nine was obese, and the increase in the prevalence of obesity has accelerated in the last decade.[3] It has also been reported that there are considerable numbers of overweight and obese infertile couples in their reproductive years,[4] and infertility affects approximately 15% of couples of which 40% is attributed to the male factor.[5]

Obesity has been associated with adult male infertility, as measured by a prolonged delay before conception.[6–8] It is generally accepted that obesity affects the GnRH-FSH/LH pulse, impairs the function of Sertoli and Leydig cells, and influences the release of sex hormones and of sperm maturation.[9] Serum testosterone, sex hormone-binding globulin (SHBG), and inhibin B are reported to decrease with increasing body mass index (BMI), whereas estradiol increased with increasing BMI.[10,11] Moreover, researchers in recent years[12–14] have found obesity in adult men to be linked to low sperm quality. Obese men are 3 times more likely to exhibit a reduction in semen quality than men of a normal weight,[15] and overweight and obesity has been associated with an increased prevalence of azoospermia or oligozoospermia.[16] One large single-clinic study of more than 10000 samples investigated the relationship between male BMI and semen parameters and demonstrated that there was a clear association between obesity and sperm production (volume, concentration, and total sperm count).[17] It has been accepted that obesity is linked to male fertility because of lifestyle changes, internal hormonal environmental alterations, and sperm genetic factors. Obesity negatively affects male reproductive potential not only by reducing sperm quality, but in particularly by altering the physical and molecular structure of germ cells in the testes, which ultimately affects the maturity and function of sperm cells.[18]

In contrast, there are other studies that show no significant correlation between BMI and semen parameters (concentration, semen volume, and total sperm motility).[19–21] A meta-analysis from MacDonald *et al.*[11] found no evidence of a relationship between BMI and sperm concentration or total sperm count, but there was a negative relationship between testosterone, SHBG, and free testosterone with an increased BMI. However, there are some study limits in these reports such as the small number of obese male subjects and the study populations consisting of multinationals. Therefore, this study was designed to explore the relationship between BMI and sperm parameters in a Northern Chinese Han population.

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MATERIALS AND METHODS

Study population

The participants were the male partners of subfertile couples who failed to conceive after 1 year of unprotected intercourse (mean infertile period was 1.20 ± 0.34 years). The participating men were recruited from those attending the Reproductive Medical Center of the First Affiliated Hospital of Zhengzhou University, China and who submitted semen samples for semen analysis or were attending for therapeutic procedures between March 2010 and December 2014. A total of 2384 healthy adult Chinese Han men of reproductive age were considered for the study. The fertility status of their female partners was not considered. Participants who had definitive pathological conditions capable of affecting sperm quality were excluded. These conditions included hypertension, diabetes mellitus, chronic kidney disease, atherosclerosis, orchidectomy for any reason, hypogonadism requiring medical treatment, and testicular cancer. In addition, participants with other factors interfering with male fertility such as heat exposure due to saunas, semen bacterial contamination, and azoospermia and oligozoospermia, which included a history of documented azoospermia or a semen analysis result of sperm concentrations of <4 spermatozoa per 400 × high power field (which correlates to a sperm concentration of ~1×10^6 ml^{-1}), as suggested by the World Health Organization Laboratory Manual for the Examination and Processing of Human Semen 2010,[22] were also excluded.

Trained staff described to the participants the hypothesis of the study and obtained their informed consents. All the subjects provided written informed consent and signed a standardized consent form. Research approval for this study was given by the Ethics Committee of the First Affiliated Hospital of Zhengzhou University.

Questionnaire

All the participants completed a questionnaire administered by an interviewer after the sperm was collected. The questionnaire covered personal information including background, ethnicity, period of abstinence, lifestyle factors, occupational exposure, genetic risk factors, and reproductive conditions. Subjects were also asked about their highest education level and current occupational status. Information was obtained on their alcohol and smoking intake in the previous week before completing the questionnaire. Smoking habits were calculated as the total number of cigars, pipes, and cigarettes smoked per day. Total weekly alcohol was estimated by combining the wine, beer, and liquor intake.

Physical examinations

A complete physical examination, including height (cm) and weight (kg), was performed by trained physicians using the same scale. Height was measured with the participant's ears and eyes in the same horizontal line and without shoes and socks. The BMI was estimated as the weight in kilograms divided by the squared height in meters (kg m^{-2}). Patients were divided into groups by BMI: underweight <18 kg m^{-2}, normal weight 18–25 kg m^{-2}, overweight 25–30 kg m^{-2}, and obese >30 kg m^{-2}. The possible status of varicoceles and hydroceles, consistency of the testis, the location of the testis in the scrotum and location of the epididymis were also recorded.

Sperm collection and analysis

Each participant was asked to provide a sperm specimen by masturbation into a wide-mouthed plastic container in a private room close to the sperm laboratory after a period of 3–7 days of sexual abstinence. Patients who provided incomplete samples were asked to repeat the process at another time or were excluded from the study. The period of sexual abstinence, spillage (if any), time of year of sperm collection, and history of recent fever were also recorded in the self-completed questionnaires after the sample was delivered. All the sperm samples were kept in a 37°C CO_2 incubator to allow them to liquefy and facilitate routine sperm analysis as described in the World Health Organization Manual of 2010.[22] The sperm smears were dried in the air, stained, and preserved at room temperature. All the smeared slides were used to determine sperm morphology and assessed by the same technician. The sperm variables used as outcome variables were as follows: sperm concentration (millions per ml), semen volume (ml), total sperm count (concentration × volume, in millions), relative number of sperm with rapid motility (Type A motility, %), relative number of sperm with linear progressive motility (Type B motility, %), percentage of sperm with sluggish motility (Type C motility, %), total motile sperm count (concentration × volume × relative number of type A + B, in millions), and relative number of normal sperm as assessed by morphology. All the sperm analyses were performed by trained laboratory technicians according to the guidelines of the World Health Organization Manual of 2010.[22] The laboratory was placed under strict quality control by trained staff. Laboratory quality controls were performed every week by analyzing the same sperm samples in blind fashion throughout the study. The observations and counting during the sperm analyses were automatic, and the origins of specimens were blinded to avoid bias.

Statistical analysis

The data from the questionnaires and physical examinations of the participants were compared between the four BMI categories to identify possible confounders. The potential confounders included age, occupation, level of education, smoking status (moderate, active, none), alcohol intake (moderate, active, none), time of year of sperm collection (April to September or October to March), and duration of sexual abstinence (day). Overall differences in potential confounders, semen volume, total sperm count, sperm concentration, relative numbers of normal morphology, and motile sperm ratio between the four BMI groups were analyzed by one-way analysis of variance. The risk of abnormal sperm quality between the different groups was compared with the use of *t*-test. The SAS 8.3 software package (SAS Institute, Cary, NC, USA) was used to perform data analyses.

RESULTS

A total of 2384 participants were included in the study. Subject characteristics are shown in **Table 1**. The average BMI of all subjects was 22.5 ± 2.7 kg m^{-2}, and 68 (2.9%) were classified as underweight, 1398 (58.6%) as normal weight, 620 (26.0%) as overweight, and 298 (12.5%) as obese. Of the total participants, 918 (38.4%) men had a BMI of >25.0 kg m^{-2}. Most of the study population did not smoke (81.3%) or drink alcohol (73.1%). No remarkable differences were found among the four BMI groups with respect to the duration of abstinence or semen pH. No differences were observed in the time of year of sperm collection (50.4% provided sperm from October to March and 49.6% from April to September).

There are other potentially independent risk factors that may confound or modify the association between BMI and sperm parameters, such as age, occupation, education level, smoking, and alcohol use as shown in **Tables 1** and **2**. Nevertheless, we found no associations between BMI and age, occupation, level of education, smoking status, or alcohol use. However, the results indicated there were significant relationships between BMI and the total sperm count, sperm concentration, total motile sperm, relative amount of type A motility and the relative amount of progressive motility (A + B). The sperm concentration of overweight and obese men was 34.1 ± 12.6 and 33.8 ± 10.8 million, respectively, which was lower than in the subjects with normal BMI. The total sperm count of overweight and obese men was 115.9 ± 33.2 and 113.9 ± 32.8 million, which was also remarkably lower than in men with normal BMI. Total motile sperm count for overweight 43.6 ± 13.3 and obese men 42.8 ± 14.8 was lower than in subjects with normal BMI. Furthermore, the relative amount of type A motility (%) for the overweight (10.3 ± 3.9) and obese individuals (11.9 ± 3.8) was also lower than in subjects with normal BMI. As displayed in **Table 2**, the overweight (37.9 ± 7.2) and obese (37.9 ± 8.5) groups had a slightly lower relative amount of progressive motility than the group with normal BMI. No obvious differences were found with normal sperm morphology and sperm volume between the four BMI groups.

Table 1: Characteristics of study population according to the different BMI groups (*n*=2384)

	BMI				*P*
	<18 (n=68)	*18–25 (n=1398)*	*25–30 (n=620)*	*>30 (n=298)*	
Age (year, mean±s.d.)	31.8±2.5	32.1±2.0	32.9±3.2	32.9±1.8	NS
Occupation, *n* (%)					
Farmer	17 (25.0)	346 (24.7)	157 (25.3)	80 (26.9)	NS
Worker	29 (42.6)	591 (42.4)	232 (37.4)	98 (32.8)	
Planners	13 (19.1)	288 (20.6)	133 (21.5)	75 (25.1)	
Not available	9 (13.2)	173 (12.4)	98 (15.8)	45 (15.1)	
Education level, *n* (%)					
Illiterate	1 (0.01)	16 (0.01)	9 (1.5)	4 (1.3)	NS
Elementary school	15 (22.0)	265 (18.9)	124 (20.0)	67 (22.4)	
Junior high school	26 (38.2)	567 (40.6)	265 (42.7)	132 (44.3)	
Senior high school	22 (32.3)	496 (35.5)	196 (31.6)	81 (27.2)	
College	4 (5.88)	54 (0.38)	26 (4.2)	14 (4.6)	
Smoking, *n* (%)					
None	56 (82.4)	1142 (81.7)	499 (80.5)	243 (81.5)	NS
Moderate	11 (16.1)	209 (14.9)	102 (16.5)	48 (16.1)	
Excessive	2 (1.5)	47 (3.4)	19 (3.1)	7 (2.3)	
Alcohol use, *n* (%)					
None	51 (75.0)	1042 (74.5)	423 (68.2)	229 (76.8)	NS
Moderate	17 (25.0)	326 (23.3)	192 (31.0)	66 (22.1)	
Excessive	0 (0)	30 (2.2)	5 (0.8)	3 (1.1)	
Season, *n* (%)					
April–September	31 (45.6)	674 (48.2)	325 (52.4)	153 (51.3)	NS
October–March	37 (54.4)	724 (51.8)	295 (47.8)	146 (48.7)	
Duration of abstinence (day)	4.01±0.29	4.0±0.26	4.0±0.28	3.99±0.22	NS

P value means the differences between the normal weight group and the obese group. Student's *t*-test. BMI: body mass index; NS: not significant; s.d.: standard deviation

DISCUSSION

The results of this study demonstrate that BMI is a factor affecting sperm parameters in adult men of infertile couples attending outpatient preconception clinics. Overweight or obese men were more likely to have lower total sperm counts, sperm concentration, total motile sperm, a relative amount of type A motility, and a relative amount of progressive motility (A + B) than men of normal weight. The study did not produce any evidence of an association between BMI and either sperm volume or normal sperm morphology. One previous study indicated that sperm motility was the parameter associated with excess weight and obesity.[14] The Jensen *et al*. group[10] found sperm concentration to be the one sperm parameter associated with BMI. Current findings indicate a negative association between a high BMI and sperm parameters. These are consistent with previous studies performed in Europe and the USA.[23–25] Most studies on sperm quality have focused on BMI as the main measure of obesity. However, there are other studies that did not indicate any relationship between BMI and any of the semen parameters.[19–21]

This study has a number of strengths and weaknesses. The strengths were that the study population was comprised of men from infertile couples trying to conceive who visited a reproductive medical center between March 2010 and December 2014. Sperm collection and analyses were measured at a single center and laboratory, reducing the potential of inter-laboratory variability. In addition, this study included underweight men, unlike the majority of studies published on this issue. The study populations had a large proportion of overweight (26.0%) and obese men (12.5%). The weaknesses of the study are that it was performed in men of infertile couples at a fertility center; therefore, the results may not reflect semen quality in the general population. This limits its external validity; therefore, its results should be interpreted with caution. The reasons for the association between BMI and sperm quality remain unknown, but there are several potential explanations. First, the increased fat of overweight and obese men produces more estrogen from testosterone, which suppresses the hypothalamic and pituitary hormonal secretion and can affect the testes directly.[23] Some studies have also reported the GnRH-LH/FSH pulses of overweight and obese men are different from those of normal weight men, which may impair the functions of the Sertoli and Leydig cells, and then

Table 2: Sperm analysis results of the men in the different BMI groups (*n*=2384)

	BMI				*P*
	<18 (n=68)	*18–25 (n=1398)*	*25–30 (n=620)*	*>30 (n=298)*	
Volume (ml)	3.4±1.1	3.5±1.2	3.5±1.1	3.5±1.1	0.56
pH value	7.3±0.5	7.4±0.6	7.5±0.2	7.4±0.3	0.81
Sperm concentration (10^6 ml^{-1})	40.2±12.9	38.1±12.0	34.1±12.6*	33.8±10.8*	0.01
Total sperm count (10^6)	126.8±33.5	124.1±34.0	115.9±33.2*	113.9±32.8*	0.001
Total motile sperm count (10^6 ml^{-1}) (%)	50.1±13.5	48.9±14.1	43.6±13.3*	42.8±14.8*	0.001
Type A motility	13.3±4.6	12.6±3.0	10.3±3.9*	11.9±3.8*	0.003
Type B motility	25.6±6.5	26.8±7.4	26.8±7.1	25.8±7.1	0.08
Type C motility	18.6±6.0	19.6±5.6	19.9±5.7	20.1±5.8	0.11
Progressive motility (A + B) (%)	39.8±11.2	39.5±8.3	37.9±7.2*	37.9±8.5*	0.02
Normal sperm morphology (%)	14.3±3.5	13.6±3.1	13.7±2.9	14.6±3.8	0.23

*Significantly different from the normal weight group. *P* value means the differences between the normal weight group and the obese group. Student's *t*-test. BMI: body mass index influence the release of sex hormones and sperm maturation.[24,25] High BMI was also found to be associated with high levels of serum leptin, which inhibits testosterone synthesis and may cause poor sperm quality.[26] Second, several studies have demonstrated that overweight and obesity causes insulin resistance and dyslipidemia, of which the latter is associated with increased oxidative stress.[27,28] Oxidative stress is regarded as an independent marker of male factor infertility because it diminishes sperm production and maturation.[29] A study performed in mice showed that obesity could lead to increased oxidative stress and reduction in sperm motility.[30] Third, obesity may increase scrotal temperature because of the large amounts of suprapubic, thigh, and scrotal fat, which can decrease semen quality in adult men.[12]

CONCLUSION

A high BMI is associated with negative effects on sperm quality. Overweight and obese men are more likely to have abnormally low sperm concentrations, total sperm count, total motile sperm count, relative amount of type A motility, and relative amount of progressive motility (A + B) than men with normal weight. Further research is required to evaluate the relationship between male BMI and sperm quality in larger populations.

AUTHOR CONTRIBUTIONS

YPS conceived of the study, participated in its design, and helped to draft the manuscript. EYW participated in its design, performed the statistical analysis, and drafted the manuscript. YH and QYD handled the recruitment of patients, sample collection, and supervised the clinical aspects of work. GDY supervised all molecular laboratory work. All authors read and approved the final manuscript.

COMPETING INTERESTS

All authors declare no competing interests.

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REFERENCES

1 Boivin J, Bunting L, Collins JA, Nygren KG. International estimates of infertility prevalence and treatment-seeking: potential need and demand for infertility medical care. *Hum Reprod* 2007; 22: 1506–12.
2 Stefan N, Haring HU, Hu FB, Schulze MB. Metabolically healthy obesity: epidemiology, mechanisms, and clinical implications. *Lancet Diabetes Endocrinol* 2013; 1: 152–62.
3 Stevens GA, Singh GM, Lu Y, Danaei G, Lin JK, *et al.* National, regional, and global trends in adult overweight and obesity prevalences. *Popul Health Metr* 2012; 10: 22.
4 Pasquali R, Patton L, Gambineri A. Obesity and infertility. *Curr Opin Endocrinol Diabetes Obes* 2007; 14: 482–7.
5 Guney AI, Javadova D, Kirac D, Ulucan K, Koc G, *et al*. Detection of Y chromosome microdeletions and mitochondrial DNA mutations in male infertility patients. *Genet Mol Res* 2012; 11: 1039–48.
6 Nguyen RH, Wilcox AJ, Skjaerven R, Baird DD. Men's body mass index and infertility. *Hum Reprod* 2007; 22: 2488–93.
7 Ramlau-Hansen CH, Thulstrup AM, Nohr EA, Bonde JP, Sorensen TI, *et al.* Subfecundity in overweight and obese couples. *Hum Reprod* 2007; 22: 1634–7.
8 Sallmen M, Sandler DP, Hoppin JA, Blair A, Baird DD. Reduced fertility among overweight and obese men. *Epidemiology* 2006; 17: 520–3.
9 Vermeulen A. Environment, human reproduction, menopause, and andropause. *Environ Health Perspect* 1993; 101: 91–100.
10 Jensen TK, Andersson AM, Jorgensen N, Andersen AG, Carlsen E, *et al.* Body mass index in relation to semen quality and reproductive hormones among 1,558 Danish men. *Fertil Steril* 2004; 82: 863–70.
11 MacDonald AA, Herbison GP, Showell M, Farquhar CM. The impact of body mass index on semen parameters and reproductive hormones in human males: a systematic review with meta-analysis. *Hum Reprod Update* 2010; 16: 293–311.
12 Aggerholm AS, Thulstrup AM, Toft G, Ramlau-Hansen CH, Bonde JP. Is overweight a risk factor for reduced semen quality and altered serum sex hormone profile? *Fertil Steril* 2008; 90: 619–26.
13 Koloszar S, Fejes I, Zavaczki Z, Daru J, Szollosi J, *et al.* Effect of body weight on sperm concentration in normozoospermic males. *Arch Androl* 2005; 51: 299–304.
14 Martini AC, Tissera A, Estofan D, Molina RI, Mangeaud A, *et al.* Overweight and seminal quality: a study of 794 patients. *Fertil Steril* 2010; 94: 1739–43.
15 Sharma R, Biedenharn KR, Fedor JM, Agarwal A. Lifestyle factors and reproductive health: taking control of your fertility. *Reprod Biol Endocrinol* 2013; 11: 66.
16 Sermondade N, Faure C, Fezeu L, Shayeb AG, Bonde JP, *et al.* BMI in relation to sperm count: an updated systematic review and collaborative meta-analysis. *Hum Reprod Update* 2013; 19: 221–31.
17 Belloc S, Cohen-Bacrie M, Amar E, Izard V, Benkhalifa M, *et al.* High body mass index has a deleterious effect on semen parameters except morphology: results from a large cohort study. *Fertil Steril* 2014; 102: 1268–73.
18 Shukla KK, Chambial S, Dwivedi S, Misra S, Sharma P. Recent scenario of obesity and male fertility. *Andrology* 2014; 2: 809–18.
19 Duits FH, van Wely M, van der Veen F, Gianotten J. Healthy overweight male partners of subfertile couples should not worry about their semen quality. *Fertil Steril* 2010; 94: 1356–9.
20 Eskandar M, Al-Asmari M, Babu Chaduvula S, Al-Shahrani M, Al-Sunaidi M, *et al.* Impact of male obesity on semen quality and serum sex hormones. *Adv Urol* 2012; 2012: 407601.
21 Relwani R, Berger D, Santoro N, Hickmon C, Nihsen M, *et al.* Semen parameters are unrelated to BMI but vary with SSRI use and prior urological surgery. *Reprod Sci* 2011; 18: 391–7.
22 Lu JC, Huang YF, Lu NQ. [WHO laboratory manual for the examination and processing of human semen: its applicability to andrology laboratories in China]. *ZhongHua Nan Ke Xue* 2010; 16: 867–71. [Article in Chinese].
23 Carreau S, Bourguiba S, Lambard S, Galeraud-Denis I, Genissel C, *et al.* Reproductive system: aromatase and estrogens. *Mol Cell Endocrinol* 2002; 193: 137–43.
24 Hammoud AO, Gibson M, Peterson CM, Meikle AW, Carrell DT. Impact of male obesity on infertility: a critical review of the current literature. *Fertil Steril* 2008; 90: 897–904.
25 Hammoud AO, Wilde N, Gibson M, Parks A, Carrell DT, *et al.* Male obesity and alteration in sperm parameters. *Fertil Steril* 2008; 90: 2222–5.
26 Hofny ER, Ali ME, Abdel-Hafez HZ, Kamal Eel D, Mohamed EE, *et al.* Semen parameters and hormonal profile in obese fertile and infertile males. *Fertil Steril* 2010; 94: 581–4.
27 Dandona P, Aljada A, Chaudhuri A, Mohanty P, Garg R. Metabolic syndrome: a comprehensive perspective based on interactions between obesity, diabetes, and inflammation. *Circulation* 2005; 111: 1448–54.
28 Davi G, Falco A. Oxidant stress, inflammation and atherogenesis. *Lupus* 2005; 14: 760–4.
29 Ebisch IM, Thomas CM, Wetzels AM, Willemsen WN, Sweep FC, *et al.* Review of the role of the plasminogen activator system and vascular endothelial growth factor in subfertility. *Fertil Steril* 2008; 90: 2340–50.
30 Bakos HW, Mitchell M, Setchell BP, Lane M. The effect of paternal diet-induced obesity on sperm function and fertilization in a mouse model. *Int J Androl* 2011; 34: 402–10.