Submitted: 05 September, 2025 Accepted: 05 January, 2026 Published: 30 April, 2026 DOI:10.22514/jomh.2026.035

ORIGINAL RESEARCH

JOMH Journal of Men's Health

Effect of increased male body mass index (BMI) on semen quality and IVF-ET clinical outcomes

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Abstract

Background: This study aimed to determine how elevated male body mass index (BMI) influences semen characteristics, embryo quality during *in vitro* fertilization-embryo transfer (IVF-ET), and subsequent reproductive outcomes in infertile couples. **Methods**: We retrospectively collected and analyzed clinical records from all couples who underwent IVF-ET at our center between January 2022 and September 2025. The study included couples infertile for one year with normal karyotypes and excluded those using donor sperm, with major lifestyle/medical issues, or specific male reproductive disorders. Based on BMI classification standards for Chinese adults, participants were divided into a group of normally weight, overweight, and obese men. A total of 243 IVF-ET cycles (81 randomly selected per BMI group) were analyzed for semen parameters, embryo quality, and clinical outcomes; group differences were assessed using the Kruskal-Wallis (H statistic) for continuous data *vs.* χ^2 tests for categorical outcomes. **Results**: It was only observed that significant differences were identified in male sperm progressive motility (H = 7.203, $p = 0.027$), total sperm motility (H = 8.173, $p = 0.017$) among the three BMI groups ($p < 0.05$). It was observed that significant differences were identified in miscarriage rate ($\chi^2 = 11.383$, $p = 0.003$), live birth rate ($\chi^2 = 6.045$, $p = 0.049$) among the three BMI groups ($p < 0.05$). **Conclusions**: These findings indicate that increased male BMI has a detrimental effect on sperm progressive motility and may adversely influence the clinical outcomes of assisted reproductive treatment.

Keywords

High male BMI; Semen parameters; *In vitro* fertilization-embryo transfer

1. Introduction

In recent decades, infertility has remained a major public health concern worldwide [1]. It is estimated that approximately 10%–15% of couples of reproductive age have trouble achieving conception, with male factors accounting for nearly 40% of these cases [2]. In parallel, the global rise in obesity has raised increasing concerns about its impact on human fertility [3], as reports indicate that obesity affects up to 20% of couples of childbearing age and that approximately 15% of infertility cases can be attributed, either directly or indirectly, to excessive body weight [4–6].

It is well known that overweight and obesity increase the risk of metabolic and cardiovascular complications such as hypertension, dyslipidemia and type 2 diabetes, and accumulating evidence indicates that they also adversely affect paternal reproductive health by altering endocrine hormones and other physiological mechanisms [7]. The potential pathways involved may include dysregulation of the hypothalamic-pituitary-gonadal (HPG) axis and impaired testicular thermoregulation, which together can lead to reduced serum concentrations of sex hormone-binding globulin, inhibin B, and androgens, accompanied by elevated estradiol levels [8]. In addition, obesity-related metabolic disorders and obstructive sleep apnea syndrome may indirectly aggravate oxidative stress, promote sperm DNA fragmentation, and result in reproductive dysfunction [9]. Body mass index (BMI) is widely used as a simple and practical indicator for assessing the degree of obesity, and, according to the most recent BMI classification standards for Chinese adults, normal weight, overweight, and obesity are defined as $18.5 \leq \text{BMI} < 24.0$ kg/m^2, $24.0 \leq \text{BMI} < 28.0$ kg/m^2, and BMI ≥ 28.0 kg/m^2, respectively [10]. In recent years, there has been growing interest in the relationship between male BMI and fertility; however, research findings regarding the association between male BMI and conventional sperm parameters remain highly variable, and only a limited number of studies have examined the impact of male BMI on assisted reproductive outcomes, with some even reporting contradictory conclusions [11]. This present study aims to provide more specific evidence on the influence of paternal BMI on fertility and Assisted Reproductive Technology

(ART, a medical technique designed to assist couples who are unable to conceive naturally in achieving pregnancy) outcomes, to inform clinical decision-making, particularly in relation to preconception counseling and individualized weight management strategies for men undergoing ART. Given these mechanisms, we hypothesised that male BMI would impair sperm motility and IVF success.

2. Subjects and methods

2.1 Participants and grouping

This study retrospectively reviewed the clinical records of all patients who underwent IVF-ET treatment at our hospital between January 2022 and September 2025. Inclusion criteria for participant selection were predefined in order to enhance the validity and reliability of the study findings. Couples who failed to conceive with unprotected intercourse for one year or longer. All male and female partners were required to have a normal karyotype as confirmed by chromosomal analysis.

Couples were excluded if they met any of the following conditions: (1) use of donor sperm; (2) Excessive smoking and alcohol consumption, diabetes, malignant diseases treated with radiation and/or chemotherapy; (3) male partners with anti-sperm antibodies, Y-chromosome microdeletions in the azoospermia factor region, testicular or epididymal inflammation, varicocele, a history of juvenile mumps or cryptorchidism. According to the most recent BMI classification standards for Chinese adults [10], male partners were divided into three BMI categories: normal weight ($18.5 \leq$ BMI $<$ 24.0 kg/m^2), overweight ($24.0 \leq$ BMI $<$ 28.0 kg/m^2), and obese (BMI $\geq$28.0 kg/m^2). After application of these inclusion and exclusion criteria, a total of 243 IVF-ET treatment cycles (81 cases randomly selected in each group) were included in the final analysis. All relevant clinical information data were extracted from the original case records of IVF patients through the principles of double data entry and independent review. All analyses were conducted by trained technicians who were unaware of patient groupings, thereby minimising observer bias.

2.2 Semen collection

Participants were instructed to abstain from ejaculation for 2–7 days before sample collection. Semen samples were obtained by masturbation into sterile containers and placed in a 37 °C incubator for liquefaction, during which the liquefaction time was recorded. After liquefaction, the samples were processed, 1 mL semen was aspirated with sterile pipettes for laboratory analysis.

2.3 Semen quality assessment

Following liquefaction, semen volume and pH were measured, and standard semen parameters, including sperm concentration, progressive motility, total motility, and overall sperm count, were analyzed using a Beijing Suijia Automated Semen Analyzer (SSA-II, Shenzhen Qianye Technology Co., Ltd, Shenzhen, Guangdong, China). All examinations were performed in accordance with the World Health Organization (WHO) Laboratory Manual for the Examination and Processing of Human Semen (2010 edition) [12].

2.4 Sperm morphology evaluation

Sperm morphology was assessed using Papanicolaou staining, with test kits supplied by Zhuhai Beisuo Biotechnology Co., Ltd. All procedures strictly followed the semen analysis criteria outlined in the WHO 2010 guidelines [12].

2.5 Sperm DNA fragmentation analysis

DNA fragmentation in spermatozoa was assessed using the sperm chromatin dispersion (SCD) assay, with detection kits purchased from Anhui Anke Biotechnology Co., Ltd. According to the assay criteria, spermatozoa without a halo, or with a halo measuring less than one-third of the sperm head diameter, were classified as DNA-fragmented.

2.6 IVF procedures, embryo development, and transfer

Oocyte fertilization was initiated approximately 39 hours after administration of human chorionic gonadotropin (HCG), and the expulsion of the second polar body was assessed 4 hours later. Pronuclear formation and fertilization status were evaluated the following morning, and embryos exhibiting two pronuclei (2PN) were considered normally fertilized. Embryonic development was monitored on days 2, 3, 5, and 6, with culture extended to day 5 or 6 when necessary. Embryo grading followed the Assisted Reproductive Technology Guidelines for cleavage-stage embryos and blastocysts.

During either fresh transfer cycles or frozen-thawed transfer cycles, one or two morphologically high-quality embryos were selected for transfer according to the clinical condition of each patient. Biochemical pregnancy was determined by serum β-HCG measurement 14 days after embryo transfer, whereas clinical pregnancy was confirmed by the presence of a gestational sac on ultrasound examination 28 days after transfer.

2.7 IVF outcome calculations

The following indices were calculated:

Second meiotic division (MII) oocyte rate = (MII oocytes/total retrieved oocytes) $\times$ 100%;

Normal fertilization rate = (2PN oocytes/total retrieved oocytes) $\times$ 100%;

Normal cleavage rate = (2PN cleaved oocytes/2PN fertilized oocytes) $\times$ 100%;

Day 3 high-quality embryo rate = (number of high-quality D3 embryos/total 2PN cleaved embryos) $\times$ 100%;

Transplantable embryo rate = (transplantable embryos/total cleaved embryos) $\times$ 100%;

High-quality blastocyst rate = (high-quality blastocysts/total blastocysts cultured) $\times$ 100%;

Blastocyst formation rate = (blastocysts formed/total cleavage-stage embryos cultured) $\times$ 100%;

Biochemical pregnancy rate = (biochemical pregnancies/embryo transfer cycles) $\times$ 100%;

Clinical pregnancy rate = (clinical pregnancies/embryo transfer cycles) $\times$ 100%;

Implantation rate = (gestational sacs/embryos transferred) × 100%;

Miscarriage rate = (miscarriages/clinical pregnancies) × 100%;

Live birth rate = (live births/embryo transfer cycles) × 100%.

2.8 Statistical analysis

Statistical analysis was performed using IBM SPSS Statistics 22.0 version 25 (IBM Corp., Armonk, NY, USA). Continuous variables were evaluated for normality utilizing the Shapiro-Wilk test and assessed for homogeneity of variance through Levene's test. Continuous variates are presented as median (interquartile range), categorical variables are shown as the number (percentage), and the proportions are presented as rate (n/N). due to the non-normality, Kruskal-Wallis test was used to compare continuous variates. The chi-square test was used to compare categorical variables. When the overall test is significant, pairwise comparisons undergo Bonferroni correction. Correlation analysis was performed by the Spearman method. $p < 0.05$ was considered statistically significant, and $p < 0.01$ was considered extremely significant.

3. Results

3.1 Comparison of semen parameters among groups

In total, 243 couples were included in the study. There were 81 normal-weight male with a median BMI of 21.60 kg/m^2, 81 overweight male with a median BMI of 25.60 kg/m^2, and 81 obese male patients with a median BMI of 29.40 kg/m^2. The results of semen parameter analysis following Kruskal-Wallis test are presented in Table 1. It was observed that no significant differences were found in semen volume (H = 1.871, p = 0.392), sperm concentration (H = 1.806, p = 0.405), total sperm count (H = 2.429, p = 0.297), normal morphology (H = 0.321, p = 0.852), sperm DNA fragmentation (H = 5.835, p = 0.054) among the three BMI groups ($p >$ 0.05). While significant differences were identified in male sperm progressive motility (H = 7.203, p = 0.027), total sperm motility (H = 8.173, p = 0.017) among the three BMI groups ($p < 0.05$). When the overall Kruskal-Wallis test is significant, further pairwise comparisons employing Bonferroni correction revealed significant differences in the percentage of sperm with progressive motility (obese group 39.10% (30.90%, 48.00%) *vs.* normal group 45.50% (34.30%, 54.50%), p = 0.013) and total sperm motility (obese group 53.00% (45.20%, 62.90%) *vs.* normal group 61.20% (49.50%, 73.00%), p = 0.010) between the obese and normal groups (adjusted $p < 0.0167$).

3.2 Association of male BMI with semen parameters

Correlation analysis between male BMI and semen parameters showed no meaningful associations (Table 2), as all correlation coefficients were weak and did not reach statistical significance. Specifically, the correlations were as follows: semen volume (r = −0.052, p = 0.420), sperm concentration (r = 0.057, p = 0.381), progressive motility (r = 0.053, p = 0.415), total motility (r = 0.023, p = 0.716), total sperm count (r =

TABLE 1. Comparison of semen quality parameters in each group.

Group	Normal group (n = 81) median (1st, 3rd) quartiles	Overweight group (n = 81) median (1st, 3rd) quartiles	Obesity group (n = 81) median (1st, 3rd) quartiles	H	*p*
Male BMI (numbers)	21.60 (20.70, 22.95)	25.60 (24.65, 26.35)^a	29.40 (28.40, 31.00)^{ab}	215.187	<0.001**
Female age (yr)	32.00 (28.00, 36.00)	33.00 (29.50, 33.00)	33.00 (29.50, 36.50)	2.041	0.360
Male age (yr)	34.00 (31.00, 37.00)	34.00 (32.00, 37.50)	34.00 (31.00, 38.00)	0.255	0.880
Semen volume (mL)	3.00 (2.35, 3.55)	3.00 (2.15, 3.70)	2.50 (2.00, 3.45)	1.871	0.392
Sperm concentration ($\times 10^6$/mL)	109.20 (67.15, 203.15)	132.70 (74.65, 222.15)	130.80 (68.35, 197.15)	1.806	0.405
Sperm progressive motility (%)	45.50 (34.30, 54.50)	41.40 (29.80, 50.20)	39.10 (30.90, 48.00)^a	7.203	0.027*
Total sperm motility (%)	61.20 (49.50, 73.00)	56.40 (46.45, 70.75)	53.00 (45.20, 62.90)^a	8.173	0.017*
Total sperm count ($\times 10^6$)	337.90 (174.55, 582.35)	370.70 (233.60, 617.85)	315.00 (185.05, 577.05)	2.429	0.297
Normal morphology (%)	5.00 (3.00, 6.00)	5.00 (3.70, 7.00)	5.00 (3.00, 6.00)	0.321	0.852
Sperm DFI (%)	8.00 (6.00, 12.00)	9.00 (7.00, 13.00)	8.00 (6.00, 12.00)	5.835	0.054
Oocytes retrieved (numbers)	11.00 (8.00, 18.00)	12.00 (7.00, 17.00)	11.00 (7.00, 16.00)	2.212	0.331

*BMI: body mass index; DFI: DNA Fragmentation Index. Continuous variates are presented as median (interquartile range) due to the non-normality. *p < 0.05, **p < 0.01. ^{a}: indicates a statistically significant difference compared with the normal group. ^{ab}: indicates a statistically significant difference compared with the overweight group. When the overall Kruskal-Wallis test is significant, pairwise comparisons undergo Bonferroni correction.*

0.030, $p = 0.641$), normal morphology ($r = 0.070$, $p = 0.279$), and DNA fragmentation index ($r = -0.041$, $p = 0.524$).

TABLE 2. Correlation between male BMI and semen parameters.

Variables	r	p
Semen volume (mL)	−0.052	0.420
Sperm concentration ($\times 10^6$/mL)	0.057	0.381
Progressive sperm motility (%)	0.053	0.415
Total sperm motility (%)	0.023	0.716
Total sperm count ($\times 10^6$)	0.030	0.641
Normal morphology (%)	0.070	0.279
Sperm DNA fragmentation rate	−0.041	0.524

3.3 Comparison of IVF-ET outcomes across groups

The results of IVF-ET outcome analysis following the chi-square test are presented in Table 3. No significant variations were observed among the three BMI groups in the MII oocyte rate ($\chi^2 = 0.341$, $p = 0.843$), normal fertilization rate ($\chi^2 = 0.852$, $p = 0.653$), cleavage rate ($\chi^2 = 4.280$, $p = 0.118$), transplantable embryo rate ($\chi^2 = 0.786$, $p = 0.675$), high-quality embryo rate ($\chi^2 = 3.984$, $p = 0.136$), blastocyst formation ($\chi^2 = 1.535$, $p = 0.464$), high-quality blastocyst rate ($\chi^2 = 1.915$, $p = 0.384$), implantation ($\chi^2 = 1.358$, $p = 0.507$), HCG positivity ($\chi^2 = 0.424$, $p = 0.809$), clinical pregnancy ($\chi^2 = 1.912$, $p = 0.384$) ($p > 0.05$). While significant differences were identified in miscarriage rate ($\chi^2 = 11.383$, $p = 0.003$), live birth rate ($\chi^2 = 6.045$, $p = 0.049$) among the three BMI groups ($p < 0.05$). when the chi-square test is significant, further pairwise comparisons employing Bonferroni correction revealed significant differences in miscarriage rate (obese group 44.19% (19/43) *vs.* normal group 21.57% (11/51), $p = 0.002$) and live birth rate (obese group 33.33% (27/81) *vs.* normal group 51.85% (42/81), $p = 0.009$) between the obese group and the normal group (adjusted $p < 0.0167$).

4. Discussion

Obesity is a chronic medical condition characterized by an excessive accumulation of body fat, arising from a combination of factors that include unhealthy lifestyle habits such as high-fat diets and sedentary behavior and certain genetic predispositions [13]. At present, it is estimated that approximately 1.6 billion adults worldwide are classified as overweight and about 400 million are identified as obese, and because obesity is frequently associated with a wide range of health complications, it has become a major global health concern and a focus of considerable international attention [14].

Body mass index (BMI), calculated as weight (kg) divided by height squared (m^2), is the most commonly used index for assessing overweight and obesity. According to the most recent BMI classification standards for Chinese adults, BMI is categorized as normal weight ($18.5 \leq$ BMI < 24.0 kg/m^2), overweight ($24.0 \leq$ BMI < 28.0 kg/m^2), and obesity (BMI ≥ 28.0 kg/m^2) [10]. Although BMI is a widely accepted indicator of weight status, its impact on male fertility remains inconclusive. For example, MacDonald *et al.* [15] reported no differences in semen volume or sperm density between obese and normal-weight men, and similar findings were described by Thomsen [16], Anifandis [17], and Liu *et al.* [10], who observed that sperm concentration, total sperm count, semen volume, and motility were not significantly influenced by BMI. In contrast, Hammiche *et al.* [18] found that increasing BMI was associated with reductions in semen concentration and motile sperm counts, highlighting the ongoing inconsistency in the available evidence.

In this study, no significant differences were found among obese, overweight and normal-weight men for semen volume, sperm concentration, normal morphology, total sperm count, or DNA fragmentation, findings that are consistent with those reported by Eisenberg [19]. Furthermore, correlation analyses demonstrated that BMI showed no linear relationship with semen quality parameters, consistent with previous international studies [20, 21]. In contrast, both progressive motility and total sperm motility were significantly reduced in obese patients, in line with the observations of Bibi [22] and Wang [23]. These results suggest that obesity has harmful effect on fertility [24]. There are several possible scientific explanations for this phenomenon. Obese men show decreased serum concentrations of sex hormone-binding globulin, inhibin B and androgen, together with raised up levels of estradiol, correlating with the levels and severity of obesity [25, 26]. Apart from this endocrine context, this phenomenon could be further explained by oxidative stress due to obesity, which could supplementarily lead to elevated levels of sperm DNA damage and compromised sperm function [27].

Current literature on the impact of male BMI on IVF-ET outcomes remains inconsistent. Zhang [28] reported a negative association between BMI and fertilization rate, number of transferable embryos, and proportion of high-quality embryos, while Yang [29] found that male BMI ≥ 28 kg/m^2 was associated with significantly lower fertilization rates, poorer embryo quality, and reduced clinical pregnancy rates. In contrast, Merhi [30] did not observe such associations. In our cohort, neither the overweight group nor the obese group differed significantly from the normal-weight group in MII oocyte rate, normal fertilization rate, cleavage rate, transplantable embryo rate, proportion of high-quality embryos, blastocyst formation, or high-quality blastocyst development, which is consistent with the findings of Bibi *et al.* [22]. Large-scale analyses have also yielded conflicting results: a cohort including 6569 IVF-ET cycles reported no significant impact of male overweight or obesity on pregnancy, miscarriage, or live birth outcomes [10], and studies by Kim [31] and Liu [32] similarly did not identify differences across BMI categories. However, Zheng [33] and Yang [29] demonstrated a negative correlation between BMI and IVF-ET pregnancy outcomes among obese men. In our data, although the obese group showed a non-significant downward trend in clinical pregnancy, the obese group exhibited a marked decline in live birth rates, alongside a significantly higher miscarriage rate, consistent with the findings of Kort [34], Mushtaq [35], and Campbell [36].

This study has several limitations. First, the relatively

TABLE 3. Comparison of IVF-ET outcome parameters in each group.

Group	Normal group (n = 81)	Overweight group (n = 81)	Obesity group (n = 81)	χ^2	p
MII oocyte rate (%)	86.30 (869/1007)	87.09 (904/1038)	86.36 (817/946)	0.341	0.843
Normal fertilization rate (%)	59.88 (603/1007)	59.82 (621/1038)	61.6 (583/946)	0.852	0.653
Cleavage rate (%)	99.83 (602/603)	99.19 (616/621)	99.83 (582/583)	4.280	0.118
Transplantable embryo rate (%)	83.79 (610/728)	84.69 (603/712)	82.94 (564/680)	0.786	0.675
High-quality embryo rate (%)	70.60 (425/602)	66.56 (410/616)	65.46 (381/582)	3.984	0.136
Blastocyst formation rate (%)	64.50 (298/462)	67.32 (309/459)	68.25 (288/422)	1.535	0.464
High-quality blastocyst rate (%)	27.06 (125/462)	25.49 (117/459)	29.62 (125/422)	1.915	0.384
Implantation rate (%)	45.39 (64/141)	38.57 (54/140)	41.35 (55/133)	1.358	0.507
HCG positivity rate (%)	65.43 (53/81)	60.49 (49/81)	62.96 (51/81)	0.424	0.809
Clinical pregnancy rate (%)	62.96 (51/81)	54.32 (44/81)	53.09 (43/81)	1.912	0.384
Miscarriage rate (%)	21.57 (11/51)^a	13.64 (6/44)^a	44.19 (19/43)^b	11.383	0.003**
Live birth rate (%)	51.85 (42/81)^a	46.91 (38/81)^a	33.33 (27/81)^b	6.045	0.049*

*MII: second meiotic division; HCG: human chorionic gonadotropin; *$p < 0.05$, **$p < 0.01$. ^{a,b}: denotes different subgroups, when the chi-square test is significant, Bonferroni correction are employed for intergroup comparisons.*

limited sample size of this study might have skewed the results to introduce potential bias. Second, all data were derived from infertile couples of reproductive age treated at a single center, which may limit the generalizability of the findings to broader populations. Third, as a retrospective study based on existing medical records, the accuracy and completeness of some variables could not be fully ensured. Finally, our analysis did not adjust for potential confounding factors, such as lifestyle, dietary habits, smoking status, and comorbid conditions, which may have influenced both BMI and reproductive outcomes.

5. Conclusions

In summary, this study demonstrates that elevated male BMI adversely affects sperm motility and is associated with less favorable assisted reproductive outcomes. Accordingly, couples planning to undergo ART should be advised to maintain a healthy BMI in the male partner before treatment initiation, to enhance the likelihood of achieving favorable reproductive outcomes.

AVAILABILITY OF DATA AND MATERIALS

The data presented in this study are available on reasonable request from the corresponding author.

AUTHOR CONTRIBUTIONS

JP, QLQ, JW and YCS—conceptualised and designed the research study. JP—analysing and interpretation of the data as well as drawing the manuscript. QLQ—revised the paper and given final approval of the version to be published. JP, JW, YCS—was responsible for data collection.

ETHICS APPROVAL AND CONSENT TO PARTICIPATE

The study protocol was reviewed and approved by the Medical Ethics Committee of Pingxiang Maternal and Child Health Hospital in Jiangxi Province (No. 2025-007-08). The requirement for informed consent from patients was waived because the study was a retrospective analysis of existing data.

ACKNOWLEDGMENT

We express our sincere gratitude to all the dedicated staff members at the hospital who played an instrumental role in the collection of data for this study.

FUNDING

This research received no external funding.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

REFERENCES

[1] Zhang H, Zhu FY, He XJ, Tang SH, Long T, Peng L, *et al*. The influence and mechanistic action of sperm DNA fragmentation index on the outcomes of assisted reproduction technology. Open Life Sciences. 2023; 18: 20220597.

[2] Li Y, Liu X, Yu Z, Xu Y, Xue W, Du X, *et al*. Relationship between seminal plasma trace elements and sperm quality. Ecotoxicology and Environmental Safety. 2025; 297: 118240.

[3] Sahu A, Pajai S. The impact of obesity on reproductive health and pregnancy outcomes. Cureus. 2023; 15: e48882.

[4] Hoek J, Schoenmakers S, van Duijn L, Willemsen SP, van Marion ES, Laven JSE, *et al.* A higher preconceptional paternal body mass index influences fertilization rate and preimplantation embryo development. Andrology. 2022; 10: 486–494.

[5] Ennab F, Atiomo W. Obesity and female infertility. Best Practice & Research Clinical Obstetrics & Gynaecology. 2023; 89: 102336.

[6] Ameratunga D, Gebeh A, Amoako A. Obesity and male infertility. Best Practice & Research Clinical Obstetrics & Gynaecology. 2023; 90: 102393.

[7] Ma M, Zhang W, Zhang J, Liang Z, Kuang Y, Wang Y. Effect of paternal body mass index on neonatal outcomes of singletons after frozen-thawed embryo transfer cycles: analysis of 7,908 singleton newborns. Fertility and Sterility. 2020; 113: 1215–1223.

[8] Nikolic AZ, Dragojevic-Dikic S, Kocic J, Babic U, Joksimovic A, Radakovic-Cosic J, *et al*. Influence of male body mass index on semen analysis parameters and *in vitro* fertilization outcomes. Medicine. 2024; 103: e38949.

[9] Han JY, Yang C, Ren BN, Li PX, Shen R, Zheng W, *et al*. The impact of paternal body mass index on semen parameters and assisted reproductive outcomes in couples undergoing IVF/ICSI-ET treatment. Obesity Research & Clinical Practice. 2025; 19: 434–440.

[10] Liu X, Shi S, Sun J, He Y, Zhang Z, Xing J, *et al*. The influence of male and female overweight/obesity on IVF outcomes: a cohort study based on registration in Western China. Reproductive Health. 2023; 20: 3.

[11] Sharma R, Agarwal A, Harlev A, Esteves SC. A meta-analysis to evaluate the effects of body mass index on sperm parameters in infertile men. Fertility and Sterility. 2017; 108: e253–e254.

[12] World Health Organization. WHO laboratory manual for the examination and processing of human semen. 5th edn. World Health Organization: Geneva. 2010.

[13] Zheng L, Yang L, Guo Z, Yao N, Zhang S, Pu P. Obesity and its impact on female reproductive health: unraveling the connections. Frontiers in Endocrinology. 2023; 14: 1326546.

[14] Li Y, Dou K, Lv Q, Wu Y. Body-mass index and obesity in infertile couples in southwest China. Medicine. 2023; 102: e36494.

[15] 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. Human Reproduction Update. 2010; 16: 293–311.

[16] Thomsen L, Humaidan P, Bungum L, Bungum M. The impact of male overweight on semen quality and outcome of assisted reproduction. Asian Journal of Andrology. 2014; 16: 749–754.

[17] Anifandis G, Dafopoulos K, Messini CI, Polyzos N, Messinis IE. The BMI of men and not sperm parameters impact on embryo quality and the IVF outcome. Andrology. 2013; 1: 85–89.

[18] Hammiche F, Laven JS, Twigt JM, Boellaard WP, Steegers EA, Steegers-Theunissen RP. Body mass index and central adiposity are associated with sperm quality in men of subfertile couples. Human Reproduction. 2012; 27: 2365–2372.

[19] Eisenberg ML, Kim S, Chen Z, Sundaram R, Schisterman EF, Buck Louis GM. The relationship between male BMI and waist circumference on semen quality: data from the LIFE study. Human Reproduction. 2014; 29: 193–200.

[20] Pauli EM, Legro RS, Demers LM, Kunselman AR, Dodson WC, Lee PA. Diminished paternity and gonadal function with increasing obesity in men. Fertility and Sterility. 2008; 90: 346–351.

[21] Chavarro JE, Toth TL, Wright DL, Meeker JD, Hauser R. Body mass index in relation to semen quality, sperm DNA integrity, and serum reproductive hormone levels among men attending an infertility clinic. Fertility and Sterility. 2010; 93: 2222–2231.

[22] Bibi R, Jahan S, Afsar T, Almajwal A, Hammadeh ME, Alruwaili NW, *et al*. The influence of paternal overweight on sperm chromatin integrity, fertilization rate and pregnancy outcome among males attending fertility clinic for IVF/ICSI treatment. BMC Pregnancy and Childbirth. 2022; 22: 620.

[23] Wang S, Wu B, Wang C, Ke Z, Xiang P, Hu X, *et al*. Influence of body mass index and waist-hip ratio on male semen parameters in infertile men in the real world: a retrospective study. Frontiers in Endocrinology. 2023; 14: 1148715.

[24] Guo D, Xu M, Zhou Q, Wu C, Ju R, Dai J. Is low body mass index a risk factor for semen quality? A PRISMA-compliant meta-analysis. Medicine. 2019; 98: e16677.

[25] Hammoud AO, Gibson M, Peterson CM, Meikle AW, Carrell DT. Impact of male obesity on infertility: a critical review of the current literature. Fertility and Sterility. 2008; 90: 897–904.

[26] Hofny ER, Ali ME, Abdel-Hafez HZ, Kamal Eel-D, Mohamed EE, Abd El-Azeem HG, *et al*. Semen parameters and hormonal profile in obese fertile and infertile males. Fertility and Sterility. 2010; 94: 581–584.

[27] Bakos HW, Mitchell M, Setchell BP, Lane M. The effect of paternal diet-induced obesity on sperm function and fertilization in a mouse model. International Journal of Andrology. 2011; 34: 402–410.

[28] Zhang X, Wu S, Qi X, Gao S, Qi J, Zhang S, *et al*. Effect of paternal body mass index on *in vitro* fertilization and neonatal outcomes among oligozoospermia and asthenospermia patients. World Journal of Men's Health. 2024; 42: 216–228.

[29] Yang Q, Zhao F, Hu L, Bai R, Zhang N, Yao G, *et al*. Effect of paternal overweight or obesity on IVF treatment outcomes and the possible mechanisms involved. Scientific Reports. 2016; 6: 29787.

[30] Merhi ZO, Keltz J, Zapantis A, Younger J, Berger D, Lieman HJ, *et al*. Male adiposity impairs clinical pregnancy rate by *in vitro* fertilization without affecting day 3 embryo quality. Obesity. 2013; 21: 1608–1612.

[31] Kim J, Patounakis G, Juneau C, Morin S, Neal S, Bergh P, *et al.* The appraisal of body content (ABC) trial: increased male or female adiposity does not significantly impact *in vitro* fertilization laboratory or clinical outcomes. Fertility and Sterility. 2021; 116: 444–452.

[32] Liu Z, Shi X, Wang L, Yang Y, Fu Q, Tao M. Associations between male reproductive characteristics and the outcome of assisted reproductive technology (ART). Bioscience Reports. 2017; 37: BSR20170095.

[33] Zheng CY, Wen Y, Xue ZW, Lei LJ, Yang HJ, Liu JJ. The correlation between sperm DNA damage and IVF-ET clinical pregnancy outcomes in different BMI populations with normal routine semen examination. National Journal of Andrology. 2023; 29: 490–497. (In Chinese)

[34] Kort HI, Massey JB, Elsner CW, Mitchell-Leef D, Shapiro DB, Witt MA, *et al*. Impact of body mass index values on sperm quantity and quality. Journal of Andrology. 2006; 27: 450–452.

[35] Mushtaq R, Pundir J, Achilli C, Naji O, Khalaf Y, El-Toukhy T. Effect of male body mass index on assisted reproduction treatment outcome: an updated systematic review and meta-analysis. Reproductive BioMedicine Online. 2018; 36: 459–471.

[36] Campbell JM, Lane M, Owens JA, Bakos HW. Paternal obesity negatively affects male fertility and assisted reproduction outcomes: a systematic review and meta-analysis. Reproductive BioMedicine Online. 2015; 31: 593–604.

How to cite this article: Jie Peng, Qinglin Qi, Jing Wang, Yingchun Su. Effect of increased male body mass index (BMI) on semen quality and IVF-ET clinical outcomes. Journal of Men's Health. 2026; 22(4): 60-65. doi: 10.22514/jomh.2026.035.