Reproduction, Fertility and Development

Body mass index and human sperm quality: Neither one extreme nor the other

Journal:	*Reproduction, Fertility and Development*
Manuscript ID:	Draft
Manuscript Type:	Research paper
Date Submitted by the Author:	n/a
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Keyword:	spermatozoa, epididymus, fertility, semen

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Body mass index and human sperm quality: Neither one extreme nor the other

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Running head: Underweight, morbid obesity and sperm quality

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Abstract

Purpose: To study the still controversial association between BMI and seminal quality.

Methods: This study included 4860 patients (non-smokers or drinkers), classified according to their body mass index (BMI) as: underweighted (UW)=BMI<20, n=45; normal weighted (NW)=BMI 20-24.9, n=1330; overweighted (OW)=BMI 25-29.9, n=2493; obese (OB)=BMI 30-39.9, n=926 and morbidly obese (MOB)=BMI>40, n=57. Conventional semen parameters and seminal concentrations of fructose, citric acid and neutral alpha-glucosidase (NAG) were studied.

Results: Low weight and morbid obesity significantly decreased sperm concentration and total count (MOB=121.5±20.6 and UW=157.9±3.6 vs NW=157.9±3.6, OW=152.4±2.7 or OB=142.1±4.3), motility (MOB=42.6±2.6 and UW=41.8±2.5 vs NW=47.8±0.5, OW=48.0±0.4 or OB=46.3±0.6) and NAG (MOB=60.1±7.9 and UW=45.2±6.6 vs NW=71.5±1.9, OW=64.7±1.3 or OB=63.1±2.1); these parameters reflect epididymal maturation. Moreover, MOB patients showed a decrease in the percentage of morphologically normal spermatozoa (MOB=4.8±0.6 vs UW=6.0±0.8, NW=6.9±0.1, OW=6.8±0.1 or OB=6.4±0.2; $p<0.05$) and an increase (2.3 to 4.9 times) in the risk of suffering oligospermia and teratospermia ($p<0.05$).

Conclusions: Not only morbid obesity but also underweight have a negative impact on sperm quality, particularly in epididymal maturation. These results show the importance of an adequate/normal body weight as the natural best option for fertility, with both extremes of the BMI scale as negative prognostic factors.

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Introduction

Obesity is becoming a worldwide concern. According to data provided by the World Health Organization in 2010 and the European Association for the Study of Obesity in 2014, in North America and some European countries, the prevalence of overweight/obesity is greater than 60%. Nonetheless, this rise in the incidence of obesity is not restricted to industrialized societies, but has also become a health problem in developing countries (World Health Organization, 2010a, European Association for the Study of Obesity, 2014).

Somewhat in parallel with this obesity epidemic, numerous studies have warned researchers and physicians about a significant decline in male fertility and/or seminal quality (Carlsen, et al., 1992, Jouannet, et al., 2001, Swan, et al., 2000). Therefore, it was only a matter of time before these two phenomena were associated as a theoretical cause-effect fact. Finally, in 2004, Jensen and co-workers published probably the most renowned study attributing to overweight and obesity a detrimental effect on male reproductive function (Jensen, et al., 2004).

Between Jensen´s publication and today, many studies have intended to confirm or discard the hypothesis of obesity as etiological factor for male fertility decline (Cabler, et al., 2010, Du Plessis, et al., 2010, Hammoud, et al., 2008a, Hammoud, et al., 2008b, Mah and Wittert, 2010, Martini, et al., 2010, Teerds, et al., 2011). Although there is no total consensus, the hormonal profile of obese males is usually characterized by a decrease in total and free testosterone (T) levels, sex hormone binding globulin and gonadotropins concentration and by an increase of circulating estrogens (Cabler, et al., 2010, Du Plessis, et al., 2010, Hammoud, et al., 2008a, Hammoud, et al., 2008b, Mah and Wittert, 2010, Teerds, et al., 2011). Nevertheless, not all obese men have reproductive hormonal levels outside the normal range (Mah and Wittert, 2010).

Whether overweight/obesity affect sperm quality or not, is even less established; data reported by different authors are conflicting and do not exhibit a clear dose-response character

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(Hammoud, et al., 2008b). While some studies have documented a reduction in sperm count associated to obesity (Jensen, et al., 2004, Hammoud, et al., 2008a, Braga, et al., 2012, Sermondade, et al., 2012, Sermondade, et al., 2013, Stewart, et al., 2009), others have not (Martini, et al., 2010, Mac Donald, et al., 2010, Ramlau-Hansen, et al., 2010).

Regarding other parameters that reflect sperm quality (motility, viability, etc.), there are not many certainties so far. Moreover**,** despite the fundamental contribution of the epididymis, seminal vesicles and prostate to sperm fertilizing ability, and even being their secretory activity androgen-dependent, there are hardly any studies assessing the impact of overweight upon the function of male accessory glands. In a previous study, we found a negative association between BMI and seminal alpha-glucosidase levels (NAG) or sperm motility, supporting the hypothesis that obesity may affect epididymal maturation (Martini, et al., 2010).

In summary, there is still no consensus about the degree of damage provoked by obesity on male reproductive function; differences in selected populations may be responsible for these disagreements. There are several issues to consider when comparing studies: a) size and reproductive status of the analysed population (proven fertile or sub fertile males); b) degree of obesity (Martini, et al., 2013); c) inter-laboratory differences in the methods used to assess sperm quality; d) if possible confounding factors were taken into consideration, since smoking or drinking may affect sperm quality even more than body weight (Braga, et al., 2012) and e) the biological importance of the seminal damage provoked by obesity, since a significant but mild diminution in sperm parameters may have differential relevance on the fertility status of sub fertile patients or healthy volunteers.

Conversely, studies exploring the possible association between underweight and subfertility are scarce (Qin, et al., 2007); although this connection is well known in animal models (Wade, 1999, Schneider, 2004). Critical thresholds of fat reserves need to be reached in order to attain complete pubertal development and reproductive capability in adulthood.

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While this phenomenon is especially relevant in females (because of the energy invested in pregnancy and lactation), it has also been described in males (Wade, 1999, Schneider, 2004, Tena-Sempere, 2013). In a previous study performed in our laboratory using mice as an experimental model, we found that weight loss associated with chronic food restriction exerted detrimental effects on epididymal maturation; i.e. we detected a significant reduction in sperm count, sperm motility and NAG activity (Martini, et al., 2007).

Taking into consideration these findings, the objective of this study was to evaluate the possible association between BMI and seminal quality in a large sample of patients attending our andrology laboratory (Cordoba, Argentine). For this analysis, we took into consideration not only the effects of different degrees of obesity upon sperm quality (obese and morbidly obese patients), but also those of underweight. The strengths of our study are based on: a) the large number of patients (n=4860) whose semen samples were evaluated under the same methodologies and criteria; b) the exclusion of patients that smoked, drinked or had been exposed to heat or toxics; c) the statistical control of variables such as age and abstinence, and d) the evaluation of seminal markers from epididymis, seminal vesicles and prostate function.

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Materials & Methods

Semen samples were obtained from the male partner of couples that attended the Andrology and Reproduction Laboratory (LAR) in Córdoba, Argentina. This observational study was performed from November 2006 to August 2012. All patients agreed to participate and signed a written informed consent. Since our study includes non-invasive procedures and the semen samples were voluntarily provided by patients and kept rigorously anonymous, an IRB approval was unnecessary.

Patients' height and weight were measured on the same day that semen sample was obtained and processed. All patients filled out a form containing data on age, abstinence period, toxic exposure and genitourinary and/or other diseases that can affect the hypothalamic hypophyseal testicular axis. Exclusion criteria were: incomplete data, azoospermia, varicocele, cryptorchidism, parotitis, diabetes, hypothyroidism and/or other disease, smoking or drinking habits and exposure to heat or toxics. Only presumably healthy patients between 18 and 65 years old were considered.

From the 12018 patients who were asked to participate in the study, 40.4% were finally included; the final number of semen samples evaluated (one sample/patient) was 4860. Patients were classified into five groups according to their body mass index (BMI= weight (kg)/height^2 (m): underweighted (UW: BMI<20), normal weighted (NW: BMI=20-24.9), overweighted (OW: BMI=25-29.9), obese (OB: BMI=30-39.9) and morbidly obese (MOB: BMI>40). This classification is in accordance with previous publications (Jensen, et al., 2004, Thomsen, et al., 2014).

Seminal parameters evaluated

After abstinence of 2-7 days, semen samples were collected by masturbation in sterile containers. When necessary, samples were transported to the laboratory at ~37° C; in all cases, the samples were analysed within the first hour after collection.

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After liquefaction, semen analysis was performed according to the World Health Organization recommendations (World Health Organization, 2010b). Briefly, seminal volume was evaluated in a graduated conic tube and sperm concentration and motility in a Makler counting chamber. Sperm viability was determined with a supravital eosin Y technique and the hypoosmotic swelling test (HOS), incubating spermatozoa in a hypoosmotic solution. Sperm chromatin condensation was tested with the aniline blue technique. Sperm morphology was assessed with Papanicolaou staining. Seminal plasma concentrations of NAG, fructose and citric acid, functional markers of epididymis, seminal vesicles and prostate respectively, were assessed using colorimetric techniques.

Statistical analysis

Seminal parameters were expressed as Mean±SEM and analysed by multivariate analysis, using age and abstinence as co-variables. The probability and increased risk of suffering oligospermia, asthenospermia and/or teratospermia per group was evaluated with Chi-square analysis and Odds Ratio calculation respectively. The level of significance used was 5%. In all cases, n represents the number of samples evaluated (1/patient).

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Results

Table 1 shows some characteristics of the patients included in this study. More than 50% of them were overweight and only around 1% were underweight or morbidly obese. Patients classified as underweighted and normal weighted were significantly younger than the others. As expected, we found significant differences in the mean BMI between groups.

When comparing those parameters especially related to epididymal maturation (sperm concentration, total sperm count, motility and seminal NAG concentration), we observed that all of them showed a bell-shaped distribution with the lowest values in underweighted and morbidly obese patients (Figure 1). Moreover, morbid obesity significantly modified other seminal parameters: increased seminal volume and decreased sperm morphology and nuclear maturity. Underweighted patients showed a diminution on seminal volume and an augmentation in the percentage of spermatozoa with nuclear maturity (Table 2).

A significantly higher percentage of morbidly obese patients exhibited oligospermia (less than 15 millions of spermatozoa/ml) or teratospermia (less than 4% of morphologically normal spermatozoa) (Figure 2). The risk of suffering these pathologies in comparison with the other groups of patients can be seen in Table 3.

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Discussion

This study aimed to evaluate, in 4860 semen samples from men attending our andrology laboratory, the possible association between sperm quality and BMI. After a multivariate analysis we detected that the underweighted and morbidly obese groups showed a significant decrease in sperm concentration, total sperm count, motility and seminal NAG levels in comparison to normal, overweighted and/or obese patients. In concordance with a previous study (Martini, et al., 2010), these results point out the epididymis as a target organ for the reproductive alterations provoked by nutritional imbalances.

Additionally, morbid obesity reduced the percentage of spermatozoa with normal morphology and those with nuclear maturity. Underweight decreased semen volume. Finally, we observed that the risk of suffering oligospermia or teratospermia in MOB patients was, at least, two times higher than in other BMI groups.

In summary, although several authors have reported that obesity exerts negative effects on semen quality, only a few studies have explored the possible deleterious actions of low weight. In this study nevertheless, we confirmed what has been previously reported by other authors and by experimental results: underweight has a negative impact on semen quality, sometimes even higher than obesity and comparable with the detrimental effects exerted by morbid obesity.

Sperm concentration and/or total sperm count are probably the sperm parameters most affected by obesity. Although not unanimous (Mac Donald, et al., 2010, Qin, et al., 2007, Thomsen, et al., 2014, Pauli, et al., 2008, Shayeb, et al., 2011), several studies and some meta-analyses have reported these findings (Jensen, et al., 2004, Hammoud, et al., 2008a, Braga, et al., 2012, Sermondade, et al., 2012, Sermondade, et al., 2013, Stewart, et al., 2009). Probably, a key factor for this lack of consensus is the degree of obesity considered in each study. In a previous research performed in 794 men attending our andrology laboratory to evaluate their fertility status, we did not find any significant association between BMI and sperm

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concentration or total sperm count. Nevertheless, only nine of these patients had BMI>40; these morbidly obese patients showed a significant reduction in sperm concentration and count (Martini, et al., 2013). Although the small number of MOB patients did not allow us to make scientific valuable conclusions, those results supported the idea that the degree of damage on sperm quality (and probably of other male reproductive features) depends on the level of obesity. The current study is in accordance with this assumption.

Furthermore, in an initial statistical evaluation of our data, we performed a linear regression analysis (with age, abstinence and BMI as independent variables). We found a significant and negative association between BMI and sperm concentration or total count (results not shown). Nevertheless, the truth is that these variables do not show a linear distribution; they display a bell-shaped one. Nonetheless, because of the markedly smaller amount of MOB or UW patients usually included in the studies, linear regression analyses yield results that are of statistical significance; however, this type of linear analyses do not show what actually happens with patients at BMIs extremes.

In a recent large cohort study (n=10665) performed in a single French laboratory, the authors found a significant negative association between BMI and sperm concentration or total count (Belloc, et al., 2014). Similarly, data from the LIFE study developed in the US showed that the percentage of men with abnormal concentration or sperm count increases along with BMI (Eisenberg, et al., 2014).

In our study, we also found that the risk of oligospermia in the MOB group was at least three times higher than in NW, OW or OB patients. In concordance, in a meta-analysis that included 21 studies and 13077 men, the risk for oligospermia or azoospermia for obese or morbidly obese men were 1.31 and 1.97 compared to men with normal weight (Sermondade, et al., 2013). Interestingly, these same authors found that the odds ratio for oligo/azoospermia of underweight men was 1.46, i.e. higher than that of obese men. This finding is in accordance with our study, in which the sperm concentration and the total count in the underweight

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population were significantly lower than that of the NW, OW and OB groups. Similar results were informed by Qin et al. in 2007. They found that after the adjustment for toxic habits, abstinence period and reproductive hormones, sperm concentration and total sperm count from underweighted healthy volunteers diminished significantly in comparison with normal, overweighted or obese men (Qin, et al., 2007). It is important to remark that the obese population of the above mentioned study had a mean BMI of 31.4±1.6, suggesting that there were not many men with morbid obesity within the recruited population.

With respect to sperm motility, in our study we found a significant reduction in the percentage of motile spermatozoa in UW and MOB patients. We also found such a profile for seminal NAG concentrations. These results are particularly interesting because: a) one of the epididymis functions is to storage and concentrate spermatozoa (Chauvin and Griswold, 2004), b) sperm motility is acquired in the epididymis (Yanagimachi, 1994) and c) NAG is a functional marker of this organ (Chauvin and Griswold, 2004, Cooper, et al., 1988, Li, et al., 2005, Mahmoud, et al., 1988). All these factors suggest that the epididymis may be a target tissue for obesity reproductive alterations. In addition, it is important to keep in mind that NAG secretion is androgen dependent and obesity, as previously mentioned, is often accompanied by hypogonadism (Cabler, et al., 2010, Du Plessis, et al., 2010, Hammoud, et al., 2008b, Mah and Wittert, 2010, Teerds, et al., 2011). In a previous paper published in 2010, we reported the same profile (Martini, et al., 2010); moreover, earlier studies developed in our laboratory using mice as animal models, have linked epididymis function and NAG secretion with nutritional alterations, particularly, undernutrition (Martini, et al., 2007).

Shayeb et al. (2014) found in a study of 2035 patients that the risk of showing motility percentages lower than 50% was 2.62, 0.96 and 0.75 for underweighted, obese and morbidly obese patients respectively, when compared to normal weighted individuals. On the contrary, other authors do not report motility reduction in underweighted patients or healthy volunteers (Qin, et al., 2007, Thomsen, et al., 2014). In our study, we found that those patients that were

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UW or MOB had higher risk of being asthenospermic. Nevertheless, probably because of the small number of patients in these groups, these differences did not reach statistically significance.

As happens with the association of BMI and sperm concentration, authors do not agree about a possible negative effect of obesity on sperm motility. While some studies have shown a negative association between obesity and motility (Hammoud, et al., 2008a, Belloc, et al., 2014, Samavat, et al., 2014), others have not been able to do so (Jensen, et al., 2004, Pauli, et al., 2008). The number of samples and the different degrees of obesity from the recruited population may be responsible for this lack of agreement. Moreover, even in the studies in which motility decreases along with obesity, the percentages of diminution are usually small. For instance, in their single center study, Belloc et al. (2014) found that the percentages of progressive motility diminished from 36.9 to 34.7 in normal weighted patients compared to extremely obese ones. In our own results, the percentage of motile sperm in the NW or OW group (47.8% and 48.0% respectively) diminished to 42.6% or 41.8% in the MOB and UW groups respectively. Nonetheless, we must keep in mind that in a sub fertile population, any improvement in semen quality (for example, those that may happen after weight loss) may benefit overall fertility.

Other sperm parameter that showed modifications in association with BMI was sperm morphology. Regarding this point, data are scarce and comparisons with other studies are difficult because of the different techniques used for assessing these parameters. Using the Kruger´s strict criteria, we found that MOB patients showed a significant reduction in the percentage of morphologically normal spermatozoa. Moreover, the risk of suffering teratospermia in this population was at least 2.3 times higher than in the other groups. Such a negative effect was not observed in UW patients.

Belloc et al., in their large cohort study (more than 10000 patients), found no correlation between BMI and sperm morphology; nevertheless, they used the modified

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David's classification method to evaluate morphology (Belloc, et al., 2014). On the contrary, Shayeb et al. (2011) informed a higher risk of presenting values under 15% in sperm morphology in obese patients compared to normal weighted ones. However, other authors have not been able to demonstrate such association (Jensen, et al., 2004, Pauli, et al., 2008). Anyway, since sperm morphology and density reflects spermatogenesis (Hirsh, 2003, Kühnert and Nieschlag, 2004, Spira and Multigner, 1998), our results suggest alterations in this androgen-dependent process as well.

Finally, in our study, the UW patients showed a significantly smaller semen volume while the MOB patients showed a higher one. The opposite happens with nuclear maturity, in which the UW group exhibited the higher values and the MOB one, the lowest. There are some publications that attribute to obesity a negative impact on DNA integrity. It is well known that inflammatory agents secreted by adipose tissue (Lampiao and du Plessis, 2008) and/or the insulin resistance and dyslipidaemia characteristic of obese patients, are associated with increased oxidative stress (Dandona, et al., 2005, Davi and Falco, 2005). This may alter sperm functional activity due to lipid peroxidation, protein adducts formation and DNA damage (Aitken, et al., 2012).

In conclusion, our results support the idea of a deleterious impact of obesity, particularly morbid obesity, upon seminal quality; not only in parameters that have usually been associated to obesity (like sperm concentration or total sperm count) but also in other variables of the spermogram that are not often evaluated (motility, morphology, NAG, etc). According to these results, we suggest that the negative impact of morbid obesity on the reproductive function is probably mediated, not only by alterations on the testicular function as has been proposed, but also on the epididymis.

Interestingly, we found that not only morbid obesity exerts negative effects, but also low weight. This feature has not been studied frequently, although some evidences may be obtained from obesity studies.

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These results draw attention to an adequate/normal body weight as the natural best option for fertility. It is important to consider that from an evolutionary point of view, reproductive functions (search and/or fighting for a couple, copula, pregnancy, lactation and parental care) require sufficient or excessive amounts of energy, in that sense, overweight (or even obesity) does not appear to be a negative issue. It must be taken to account that BMI classification and limits may adjust to death/cardiovascular risk but not necessarily to reproductive physiology. Perhaps in the future, with more evidences and adequate meta-analyses, andrologists may better establish the appropriate "reproductive" limits for BMI.

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Table 1: Characteristics, number and percentage of patients included in the study. Patients attended the Andrology and Reproduction Laboratory of Cordoba, Argentina, from November 2006 to August 2012. Exclusion criteria were: history of varicocele, cryptorchidism, parotitis, genitourinary infection, genitourinary surgery and toxic habits/exposure (including smoking, drinking, radiations, heat and pesticides). According to their body mass index (BMI) patients were included in one of the following categories: Underweighted: BMI<20; Normal weighted: BMI=20-24.9; Overweighted: BMI=25-29.9; Obese: BMI=30-39.9 and Morbidly obese: BMI>40.***:** $p<0.05$ vs other groups; #: $p<0.05$ vs obese and morbidly obese patients.

BMI category	Number of patients (%)	Age (years)	BMI
Underweighted	45 (1.0)	32.7±1.0*	18.8±0.1*
Normal weighted	1339 (27.5)	34.9±0.2#	23.6±0.1*
Overweighted	2493 (51.3)	36.1±0.1	27.2±0.1*
Obese	926 (19.0)	36.4±0.2	32.6±0.1*
Morbidly obese	57 (1.2)	36.6±0.9	44.5±0.6*

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Table 2: Multivariate analysis of the seminal parameters of patients included in the study. Patients attended the Andrology and Reproduction Laboratory of Cordoba, Argentine, from November 2006 to August 2012. According to their body mass index (BMI) patients were included in one of the following categories: Underweighted (**UW**): BMI<20; Normal weighted (**NW**): BMI=20-24.9; Overweighted (**OW**): BMI=25-29.9; Obese (**OB**): BMI=30-39.9 and Morbidly obese (**MOB**): BMI>40. HOS: Hypoosmotic swelling test. In parenthesis: number of patients included in each BMI category. Identical letters in each line indicate significant differences (p<0.05).

Seminal parameters	**Underweighted (~45)**	**Normal weighted (~1339)**	**Overweighted (~2493)**	**Obese (~926)**	**Morbidly obese (~57)**
Seminal volume (ml)	2.8±0.3 **a,b,c**	3.3±0.1 **a**	3.1±0.1 **b,d**	3.0±0.1 **e**	3.4±0.3 **c,d,e**
Viability (% of dead spermatozoa)	18.4±1.4	17.3±0.3	17.8±0.2	18.0±0.3	20.9±1.9
Strict criteria morphology (% of normal spermatozoa)	6.0±0.8 **a**	6.9±0.1 **b**	6.8±0.1 **c**	6.4±0.2 **d**	4.8±0.6 **a,b,c,d**
HOS (% of reactive spermatozoa)	78.8±1.8	78.1±0.3	78.4±0.2	77.5±0.4	74.5±2.4
Nuclear maturity (% of mature nuclei sperm)	70.1±2.4 **a,b**	67.6±0.5 **a,c**	69.1±0.3 **d**	68.6±0.5 **b,e**	64.0±2.4 **b,c,d,e**
Fructose (mg %)	277.2±20.3	298.7±4.0	300.7±2.9	300.1±4.8	323.6±17.9
Citric acid (mg %)	386.8±25.2	439.9±5.0	435.4±3.6	444.7±6.5	446.8±23.3

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Table 3: Increased risk of suffering oligospermia, asthenospermia and teratospermia in morbidly obese patients in comparison with other body mass index (BMI) groups.

Patients included in the study attended the Andrology and Reproduction Laboratory of Cordoba, Argentine, from November 2006 to August 2012. According to their body mass index (BMI) patients were included in one of the following categories: Underweighted (**UW**): BMI<20; Normal weighted (**NW**): BMI=20-24.9; Overweighted (**OW**): BMI=25-29.9; Obese (**OB**): BMI=30-39.9 and Morbidly obese (**MOB**): BMI>40. Criterion for oligospermia was: less than15 millions of spermatozoa/ml; for asthenospermia: less than 32% of motile spermatozoa and for teratospermia: less than 4% of morphologically normal spermatozoa. Unless mentioned (NS), odds ratio were statistically different ($p<0.05$).

BMI category	**Odds ratio (CI 95%) for oligospermia**	**Odds ratio (CI 95%) for asthenospermia**	**Odds ratio (CI 95%) for teratospermia**
Underweighted (n=45)	NS	NS	2.7 (1.1-6.8)
Normal weighted (n=1339)	3.1 (1.2-7.9)	NS	2.9 (1.7-5.3)
Overweighted (n=2493)	3.8 (1.5-9.4)	NS	2.9 (1.7-5.2)
Obese (n=926)	4.9 (1.8-13.2)	NS	2.3 (1.3-4.1)

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Figure 1: Analysis of variance of the seminal parameters of patients attending the Andrology and Reproduction Laboratory of Cordoba (Argentine), from November 2006 to August 2012. According to their body mass index (BMI), patients were included in one of the following categories: Underweighted (**UW**): BMI<20; Normal weighted (**NW**): BMI=20-24.9; Overweighted (**OW**): BMI=25-29.9; Obese (**OB**): BMI=30-39.9 and Morbidly obese (**MOB**): BMI>40. The number of seminal samples/group analysed for the parameters concentration, total sperm count and motility were: 45, 1339, 2493, 926 and 57 respectively. The number of seminal samples/group analysed for their neutral alpha-glucosidase concentrations were: 15, 580, 987, 362 and 22 respectively. Identical letters indicate significant differences ($p<0.05$).

Figure 2: Differences in the frequency of oligospermia, asthenospermia and teratospermia of patients attending the Andrology and Reproduction Laboratory of Cordoba (Argentine), from November 2006 to August 2012. According to their body mass index (BMI), patients were included in one of the following categories: Underweighted (**UW**): BMI<20; Normal weighted (**NW**): BMI=20-24.9; Overweighted (**OW**): BMI=25-29.9; Obese (**OB**): BMI=30-39.9 and Morbidly obese (**MOB**): BMI>40. The number of patients/group evaluated for oligospermia or asthenospermia was: 45, 1339, 2493, 926 and 57 respectively. The number of patients/group evaluated for teratospermia was: 35, 1148, 2084, 776 and 48 respectively. Identical letters indicate significant differences ($p<0.05$).

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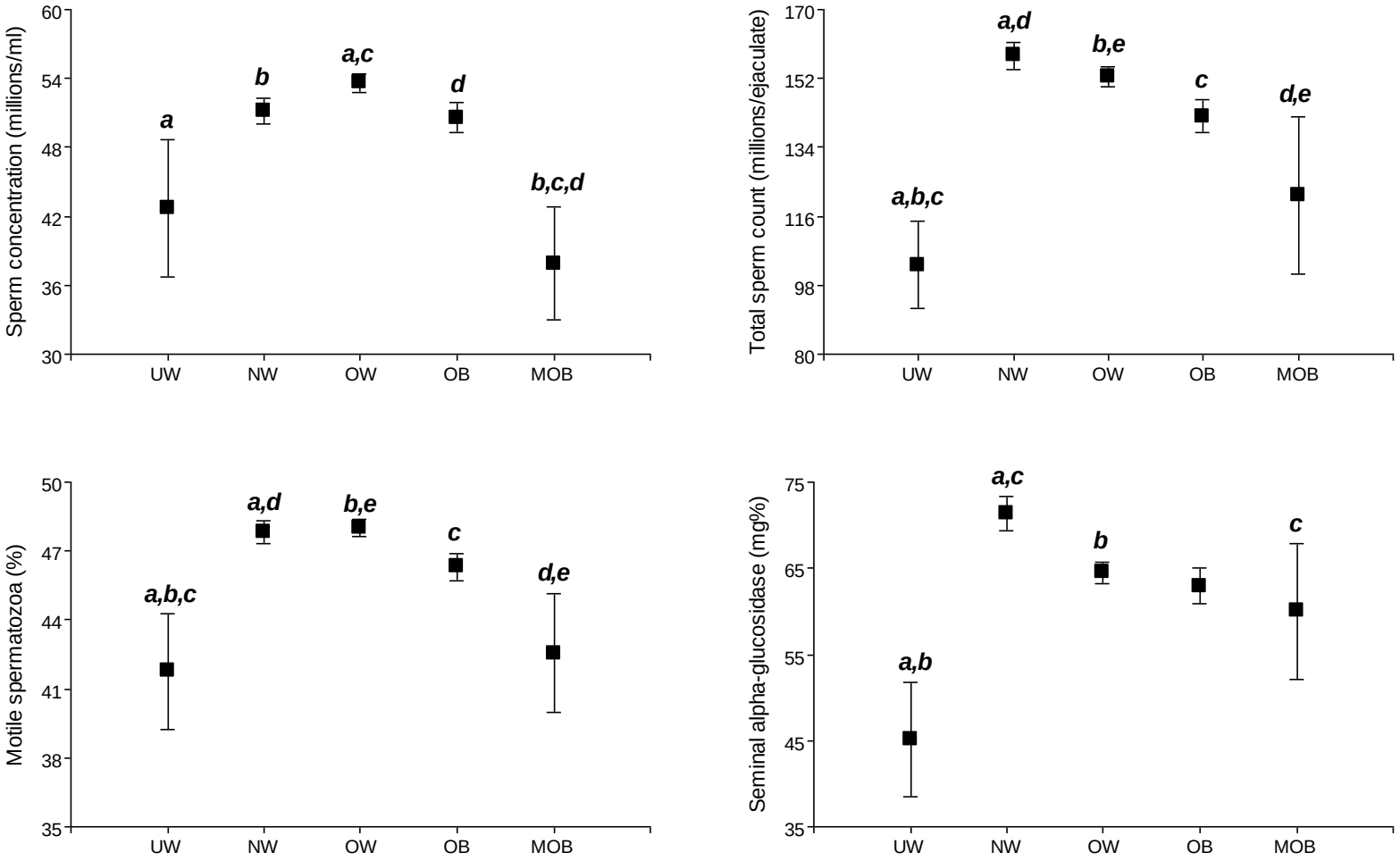
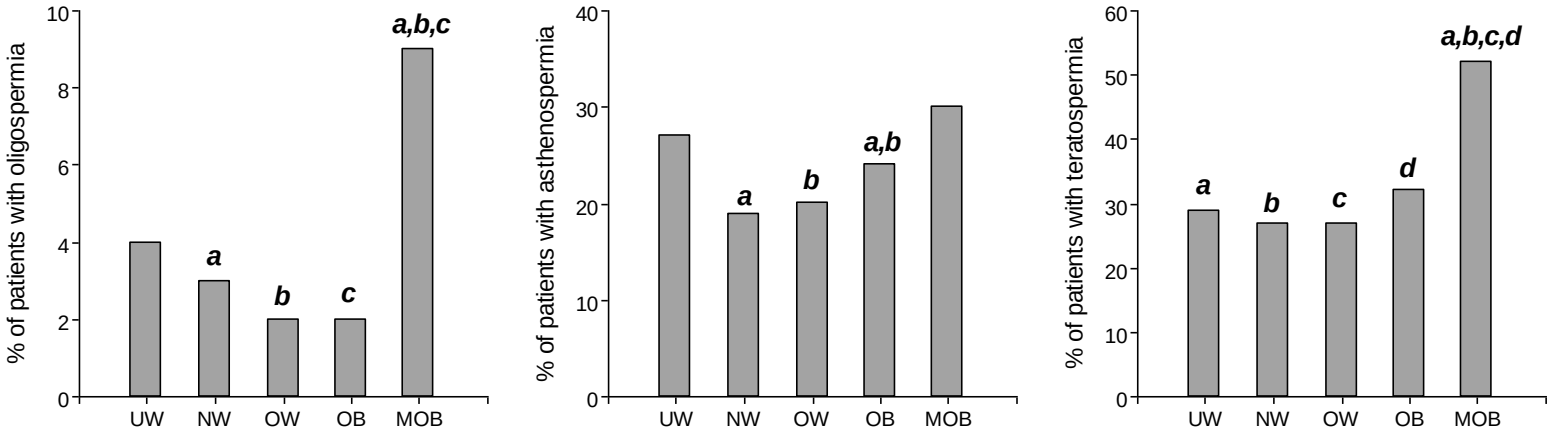

Sperm concentration (millions/ml)
60
54
48
42
36
30
a
b
a,c
d
b,c,d
UW
NW
OW
OB
MOB
Total sperm count (millions/ejaculate)
170
152
134
116
98
80
a,b,c
a,d
b,e
c
d,e
UW
NW
OW
OB
MOB
Motile spermatozoa (%)
50
47
44
41
38
35
a,b,c
a,d
b,e
c
d,e
UW
NW
OW
OB
MOB
Seminal alpha-glucosidase (mg%)
75
65
55
45
35
a,b
a,c
b
c
UW
NW
OW
OB
MOB


% of patients with oligospermia
0
2
4
6
8
10
a
b
c
a,b,c
UW
NW
OW
OB
MOB
% of patients with asthenospermia
0
10
20
30
40
a
b
a,b
UW
NW
OW
OB
MOB
% of patients with teratospermia
0
10
20
30
40
50
60
a
b
c
d
a,b,c,d
UW
NW
OW
OB
MOB