

The impact of selected modifiable lifestyle factors on male fertility in the modern world

Krzysztof Balawender^{1,2}, Stanisław Orkisz¹

¹Morphological Sciences Department of Human Anatomy, Medical Faculty University of Rzeszów, Rzeszów, Poland

²Clinical Department of Urology and Urological Oncology, Municipal Hospital in Rzeszów, Rzeszów, Poland

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Corresponding author

Krzysztof Balawender
Clinical Department
of Urology
and Urological Oncology
Municipal Hospital
in Rzeszów
4 Rycerska Street
35-241 Rzeszów, Poland
phone +48 17 851 89 01
balawender82@gmail.com

Introduction The modern man during his reproductive period is exposed to the negative influence of widespread lifestyle-related habits. The available studies show a significant decline in the quality of young men's sperm, which results in male and female infertility factors being given equal consideration. The progressive decline of sperm quality has lowered the spermiogram parameters proposed by the World Health Organization.

Material and methods We performed a review of the literature on the most common unhealthy habits in the modern world, such as obesity, mobile phone usage, smoking and alcohol intake, and their reported effects on male fertility. Several papers including original, review and meta-analysis were searched using the ScienceDirect and Web of Science databases.

Results All the factors under analysis contribute to a significant deterioration of sperm quality, mainly through decreased sperm concentration, vitality, motility and morphology.

Conclusions Lifestyle-related factors are a significant cause of male infertility in the world today. Studies have shown that sperm quality is essentially determined by: obesity, nicotine addiction, heavy exposure to electromagnetic compatibility radiation-emitting devices and alcohol consumption. Weight loss and cessation or limitation of unhealthy lifestyle-related factors might represent the best course of action for any couple trying to achieve pregnancy.

Key Words: alcohol drinking ◊ infertility ◊ obesity ◊ mobile phone ◊ cigarette smoking

INTRODUCTION

According to The International Committee for Monitoring Assisted Reproductive Technology (ICMART) and the World Health Organization (WHO), infertility is a disease characterized by the inability in achieving a pregnancy after twelve months of timed, unprotected sexual intercourse. More than 186 million people worldwide suffer from infertility. Between 8 to 12% of couples of childbearing age are affected by infertility. Research has shown males are responsible for 20–30% of infertility cases but contribute to 50% of cases overall [1].

Nowadays, environmental, occupational and lifestyle factors negatively impact both male and female fertility as well as the success of assisted reproduc-

tive technology (ART). Amongst the factors of male infertility in the modern world, significant are unhealthy habits causing obesity (unhealthy diet, lack of physical activity, sedentary lifestyle), smoking, using electronic devices as a source of electromagnetic radiation (mobile phones, portable computers) and alcohol consumption.

The deterioration in semen quality has a direct impact on a couple's reproductive potential. It has been reported for a few decades that semen quality is steadily declining. A systematic review, examining a time frame of nearly 40 years, assessed sperm concentration in fertile men and men of unknown fertility. The results showed a significant decrease in sperm concentration (113 million/ml to 66 million/ml) and semen volume (3.40 ml to 2.75 ml) over the

evaluated timespan [2]. Springart et al. also studied changes in semen quality over time between 1976 and 2009 and noticed a significant decrease in total sperm count (443 to 300 million), motility (64% to 49%), and vitality (99% to 80%). Moreover, the authors reported a significant decline of sperm with normal morphology from 67% to 26% [3].

The year 2017 saw the publication of an analysis of reports printed between 1980 and 2015, regarding changes in sperm concentration. Based on the study, linear regression analysis identified a significant decrease between 1980 and 2015, declining from 91.65 million/ml to 39.34 million/ml ($r = -.313$, $p = .0002$). This reflected an approximate 57% abatement in sperm count worldwide since 1980 [4].

In 2010, the World Health Organization established new criteria for the laboratory examination of human semen based on men whose partners had a time-to-pregnancy less than 12 months. Newly proposed reference values for semen characteristics were significantly lower than the reference values published in prior manuals. In a neoteric study evaluating the impact of these changes, semen characteristics of approximately 15% of patients considered abnormal according to the 1999 WHO reference values were reclassified as normal based on the new 2010 WHO standards [5].

MATERIAL AND METHODS

The literature review was performed with the use of bibliographic databases of peer-reviewed journals (Web of Science, Science Direct, Medline). Due to the natural wide scope of the issue, the authors included the most recent scientific publications (1992 to 2018). Relevant articles were identified by keywords and medical topic search terms (medical subject headings, MeSH): male infertility, obesity, mobile phone, cigarette smoking, alcohol drinking. The search was restricted to peer-reviewed, English-language articles. We qualified and analysed 38 articles.

RESULTS

The result of paternal obesity on reproductive potential

Obesity presents crucial health concerns around the world. It is increasingly prevalent in those trying to conceive, particularly in young men of reproductive age, with the incidence of obesity tripling since the early 1970s [6]. Based on the meta-analysis by Campbell et al. (with 115,158 total participants) couples with an obese male partner were significantly more likely to experience infertility than cou-

ples with a normal weight male (OR = 1.66; 95%CI 1.53–1.79). The study revealed a correlation between young adulthood obesity and the probability of having a child. Men who were obese as young adults had a risk ratio (RR) of having a child of 0.75 compared with normal weight men (95%CI 0.66–0.84) [7]. Furthermore, male obesity has a negative impact on the success of ART. The chances of having a non-viable pregnancy, one that did not result in a live birth, were significantly greater for couples with an obese man compared with a normal weight male partner (OR = 2.87; 95%CI 1.34–6.13), with an absolute risk difference of 10% (95%CI 3–18%) [7]. Overweight men are significantly more probable to be oligo- or azoospermic. Sermonadade et al. performed a systematic review and meta-analysis investigating the impact of body mass index (BMI) on sperm count in human males. The authors indicate a J-shaped dependence between BMI categories and oligozoospermia and azoospermia risk. Compared with the normal weight men, the odds ratio (95%CI) for oligo- and azoospermia was 1.15 for underweight, 1.11 for overweight, 1.28 for obese and 2.04 for morbidly obese men (BMI >40 kg/m²) [8]. A study researching subfertile populations showed that obese men are more predisposed to have abnormally low sperm concentrations, total sperm count, and total motile sperm count.

Conversely, weight loss is interrelated with a decrease in cellular DNA damage and increased total motile sperm count with a 14-week weight loss program. Motility is the standard parameter most closely correlated to the DNA fragmentation index (DFI). Mir et al. noted the mean DFI was higher before weight loss (20.2%) and improved significantly after weight loss (17.5%, $p < .001$). There was also a statistically significant improvement in semen morphology following weight loss ($p < .05$) [9].

The relationship between obesity and semen quality has heterogenic pathophysiology. One of the theories points to endocrinological disorders of the hypothalamic-pituitary-gonadal axis which come as a consequence of obesity. The aromatization of steroids to estrogens in peripheral tissues leads to the hypogonadotropic hypoestrogenic hypogonadism with a significant decrease in total and free testosterone levels and increase in estradiol [8]. Stelato et al. suggested decreased sex hormone-binding globulin occurred amongst obese men, a result of hyperinsulinemia, thereby emphasizing the negative feedback effect of elevated total estradiol levels [10]. Other reports have shown obesity may directly alter spermatogenesis and Sertoli cell function as indicated by the substantial decrease of inhibin B concentration compared with the decline of FSH levels

[11]. Higher levels of oxidative stress and imbalance between oxidants and antioxidants have been also observed in obese men and linked to reduced fecundity. In addition, oxidative stress has been linked to impaired sperm function, apoptosis and sperm DNA fragmentation [12]. Some researchers proposed another hypothesis that the thermal effect resulting from increased scrotal adiposity could harm sperm cells. Finally, obesity can also be related to erectile dysfunction, and to sexuality in a reverse fashion, due to the psychological impact [13].

The effect of mobile telephones and portable computers on semen quality

Concern is arising around the use of mobile phones and other devices as a source of low-level radio-frequency electromagnetic fields (RF-EMF) and their association with poor semen quality. RF-EMF may have both non-thermal and thermal effect on biological tissue. The non-thermal effect is believed to increase the production of reactive oxygen species, resulting in DNA damage [14]. The thermal effect might increase testicular temperature as mobile phones are often carried in trouser pockets near external male reproductive organs [15]. We found two meta-analyses examining mobile phone use and semen quality. The meta-analysis by Adams et al. reported that there was a relationship between exposure to mobile phones and the reduction in sperm motility (mean difference -8.1%; 95%CI -13.1 to -3.2) and viability (mean difference -9.1%; 95%CI -18.4 to 0.2), but the effects on sperm concentration were vague. The authors concluded that pooled results from both in vivo and in vitro studies advise that the exposure of mobile phones negatively impacts the quality of sperm [16]. The second meta-analysis by Liu et al. reported that mobile phones RF-EMF was related to a significant decrease in sperm quality in in vitro and animal studies. According to the study, radiofrequency radiation had a destructive impact on both motility and viability of sperm in vitro (mean difference -4.11; 95% CI -8.08 to -0.13 and -3.82; 95%CI -7.00 to -0.65 for sperm motility and viability respectively). Radiofrequency exposure also had harmful consequences on animal sperm concentration and motility (mean difference -8.75; 95% CI -17.37 to -0.12 and -17.72; 95%CI -32.79 to -2.65 for sperm concentration and motility respectively). However, meta-analysis by Liu et al. showed that mobile phone usage had no negative impact on human semen parameters [17]. The position of one's mobile phone in relation to external reproductive organs has also been of interest. Rago et al. and Lewis et al. independently reported that men who carried a mobile phone in their

pants pocket had a statistically significant increase in sperm DNA fragmentation than those who stored their phone in their shirt pocket [18, 19].

Further sources of low-level radio-frequency electromagnetic fields (RF-EMF) are portable computers (laptops, connected to local area networks wirelessly, also known as Wi-Fi). Laptop computer usage has increased explicitly in people of reproductive age. Nearly always laptops are connected to the internet through Wi-Fi and usually placed on the lap near the testes (emission of electromagnetic waves is 7–15 times higher under the laptop than under basal condition without portable computer). Furthermore, laptops generate high temperatures that can increase the scrotal temperature and may affect spermatogenesis. Avendano et al. evaluated the direct impact of laptop use on human spermatozoa. Ex vivo exposure of human spermatozoa to a wireless internet-connected laptop showed a significant decrease in progressive semen motility and an increase in semen DNA fragmentation [20]. The possible explanation for the impaired sperm motility could be electromagnetic fields inducing oxidation of phospholipids and high seminal reactive oxygen species (ROS) level [21, 22].

Experimental evidence has also been published utilizing rat studies, confirming that exposure to mobile phone RF-EMF causes histological changes to testes. Oh et al. reported that a long duration of 4G-LTE based electromagnetic fields (EMF) had deleterious effects on rat spermatogenesis. In this study, sperm and Leydig cell counts significantly decreased in the long duration exposure group (18 hours daily), emphasizing that continuous mobile phone use may be hazardous for fertile men, particularly adolescent men. However, the 10 cm distance from source of EMF, with the same length and energy, was relatively less affected, indicating that carrying a phone in the pants pocket could be harmful [23]. The last guideline for prevention, diagnosis and treatment of EMF-related health problems and illness from the European Academy for Environmental Medicine (EUROPAEM) [24] provides only superficial treatment of the problem of electromagnetic radiation's effect on male fertility and gives no indications as to preventive measures against male infertility.

Cigarette smoking and semen quality

The use of cigarettes is prevalent in approximately 37% of men of reproductive age, and Europe has the highest tobacco use amongst all World Health Organization regions [25]. Tobacco smoke contains approximately 4700 chemical compounds which are inhaled by smokers. Cigarette smoke contains

several hazardous substances, including nicotine and its metabolite, carbon monoxide, benzopyrene, and cadmium, which may have harmful effects on male germ cells. Smoking cigarettes has been correlated with a quality deterioration of semen, comprising motility, concentration, and morphology. The American Society of Reproductive Medicine in 2012 reported that semen parameters and sperm function are 22% poorer in smokers compared to non-smokers, with dose dependent effects [26]. Based on a meta-analysis study (a total of 5865 participants) moderately and heavily smoking men are more likely to have reduced sperm parameters such as count and motility. Exposure to cigarette smoke was associated with decreased sperm count (mean difference: -9.72 million/ml; 95%CI -13.32 to -6.12), motility (mean difference: -3.84%; 95%CI -5.53 to -1.44), and morphology (mean difference: -1.37%; 95%CI -2.63 to -0.11) [25]. Mostafa et al. described a significant decrease in sperm count ($p = .006$), progressive motility ($p \leq .001$), percentage of normal forms ($p \leq .001$), and viability ($p = .002$) among infertile non-smokers and infertile smokers. Moreover, the percentage of abnormal sperm chromatin concentration was significantly elevated in smokers related to non-smokers ($p \leq .001$) [27]. A dose- and time-dependent correlation among cigarette smoking and abnormal sperm chromatin was also noticed. Comparable outcomes were reported by Sepaniak et al. where the average rate of DNA fragmentation was 25.9% for non-smokers and 32% for smokers ($p < .001$) [28]. Jain et al. revealed that the seminogram of heavy smokers is more unbalanced as compared to males smoking less than 20 cigarettes per day. Hence, they summarised that not only quitting smoking, but even reducing the number of cigarettes per day, improves semen parameters [29].

It is still unknown what causes smoker sperm quality to deteriorate and the research results published so far are inconclusive. There are theories suggesting a pathomechanism leading to impairment of the acrosome reaction and capacitation and increased oxidative stress [30]. The toxins deriving from cigarette smoke can decrease sperm mitochondrial activity and damage chromatin structure and sperm DNA. The smoking-induced DNA damage in male germ cells is a consequence of high level of reactive oxygen species (ROS) such as superoxide anion, hydrogen peroxide, and hydroxyl radicals. Those toxic oxidant chemicals cause unbalance of oxidation/antioxidant system to lead to pathological male fertility profile. Aboulmaouahib et al. reported that smokers and smokers-alcohol drinkers exhibited increased enzymatic antioxidant activities tripled or more compared to infertile non-smokers patients and non-

alcohol drinkers. High ROS level caused by cigarette smoke and alcohol have an effect on sperm DNA integrity. In the Aboulmaouahib et al. study, DNA fragmentation index and spermatid chromatin decondensation were significantly increased on both for smokers (25% and 23%) as alcoholic (26% and 25%) and more for double consumers- smoking-alcoholism group (31% and 39%) [31]. Hypoxia, that is resulting from smoking cigarettes, is probably also responsible for impaired spermatogenesis. Dai et al. suggested that long-time cigarette smoking increases testosterone metabolism in liver, concurrently causing secretory dysfunction of Leydig and Sertoli cells [32].

Alcohol intake and semen quality

Alcohol consumption is the next crucial factor decreasing semen quality. Taking into account the fact that 76% of European citizens have consumed alcohol in the past 12 months and most of the previous data show a significant reduction of semen quality, alcohol intake has declined human fertility still. The first reports on the effect of alcohol intake on male infertility appeared over 30 years ago, evaluating sperm quality and associated hormonal disorders in alcoholics. Also, autopsies showed that over 50% of heavy drinkers had partial or complete spermatogenic arrest. In 2011, one of the first meta-analyses (with 29,914 participants examined) found a significant relationship between alcohol intake, volume of semen, and both morphology and motility of sperm [33]. In 2017, Ricci et al. reported the data from their meta-analysis. Fifteen cross-sectional studies were included, encompassing 16,395 male subjects. The primary results proved that alcohol consumption has a harmful effect on semen volume (mean difference: -0.25 ml; 95%CI -0.07 to -0.42) and normal morphology (mean difference: -1.87%; 95%CI -0.86 to -2.88). There was a marked difference when comparing occasional versus daily use, suggesting moderate consumption did not decline semen quality [34]. Condorelli et al. retrospectively evaluated semen and hormones parameters of moderate alcohol consumers, comparing daily (2-3 alcohol units everyday) and occasional drinkers (less than 2 times a week with meals). The results showed that the hormonal changes were significantly worse in infertile patients from the group of daily drinkers compared to the group of occasional drinkers [35]. Time to pregnancy was also significantly longer in those couples in which the male partner consumed more than 20 alcohol units per week [36].

The mechanisms that create the damaging impact of alcohol on fertility are not yet fully discovered. Some authors reported that heavy users of alcohol

have substantially higher concentrations of leukocytes in seminal fluid. Some studies showed that alcohol drinking causes adverse effects on both testosterone metabolism and spermatogenesis. The ratio between free estradiol and free testosterone is modified by alcohol intake, and spermatogenic arrest was found to be increasingly associated with high alcohol consumption [37]. Ramlau-Hansen et al. also suggested that semen quality in the male offspring might also be influenced by the consumption of alcohol during pregnancy [38]. The alcohol-induced DNA damage in male germ cells as it does with cigarette smoking is a consequence of high level of reactive oxygen species [31].

CONCLUSIONS

Lifestyle-related factors are a considerable contributor to male infertility in the world today. Studies

have shown that sperm quality is essentially determined by obesity, nicotine addiction, heavy exposure to electromagnetic radiation-emitting devices and alcohol consumption. Lifestyle factors decrease the count, motility and morphology of semen, increase DNA damage and may also have an effect on endocrine control of reproductive function. If we quantify the overall effect of unhealthy habits on cell parameters (taking into account meta-analyses only), a healthy young man's sperm concentration will be down by 10 million/ml, sperm morphology by 3%, sperm cell vitality by 8% and sperm cell motility by 12%. Cessation of these customs should be proposed to all patients undergoing investigation for infertility in order to provide the best possible reproductive outcomes.

CONFLICTS OF INTEREST

The authors declare no conflicts of interest.

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