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Lifestyle causes of male infertility

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ABBRVIATIONS
AAS, anabolic–androgenic steroids; APA, advanced paternal age; ASIH, anabolic steroid-induced hypogonadism; ART, assisted reproductive technology; BMI, body mass index; Chk1, checkpoint kinase 1;

Abstract  Objective: To examine the potential effects of lifestyle factors on male reproductive health. Evidence of a global decline in human sperm quality over recent decades has been accumulating. Environmental, occupational, and modifiable lifestyle factors may contribute to this decline. This review focuses on key lifestyle factors that are associated with male infertility such as smoking cigarettes, alcohol intake, use of illicit drugs, obesity, psychological stress, advanced paternal age, dietary practices, and coffee consumption. Other factors such as testicular heat stress, intense cycling training, lack of sleep and exposure to electromagnetic radiation from mobile phone use are briefly discussed.

Materials and method: A comprehensive literature search was performed to identify and synthesise all relevant information, mainly from within the last decade, on the major lifestyle factors associated with male infertility and semen quality. Database searches were limited to reports published in English only. A manual search of bibliographies of the reports retrieved was conducted to identify additional relevant articles.

Results: In all, 1012 articles were identified from the database search and after reviewing the titles and abstract of the reports, 104 articles met the inclusion criteria. Of these, 30 reports were excluded as the full-text could not be retrieved and the abstract did not have relevant data. The remaining 74 reports were reviewed for data on association between a particular lifestyle factor and male infertility and were included in the present review.

Conclusion: The major lifestyle factors discussed in the present review are amongst the multiple potential risk factors that could impair male fertility. However,
Introduction

There has been increasing evidence on the global decline in human sperm quality over the past few decades [1–4]. In the most recent report, Levine’s group performed a systematic review and meta-regression analysis of the current trends in sperm counts. The comprehensive study involved 42,935 men with samples spanning over ~40 years. They reported a significant decline of 50–60% in sperm counts amongst men from North America, Europe, Australia and New Zealand [5]. This latest finding has sparked even greater concern over the reasons behind the apparent decline in the sperm count of Western men.

As male fertility can be influenced by a variety of factors, one possible explanation for the declining trend would be that there are environmental and/or occupational factors along with lifestyle practices that contribute to the deterioration of semen quality [6–9].

The present article reviews the available evidence examining the potential effects of lifestyle factors on male reproductive health. It focuses on the following lifestyle factors that are associated with male infertility: smoking cigarettes, alcohol intake, use of illicit drugs, obesity, psychological stress, advanced paternal age, dietary practices, and coffee consumption. Other factors such as testicular heat stress, intense cycling training, lack of sleep, and exposure to electromagnetic radiation from mobile phone use are also briefly touched upon.

Materials and methods

A systematic review of literature was conducted using PubMed over the last 10 years (from December 2008 to November 2017) for all published reports on the major lifestyle factors associated with male infertility (Fig. 1). The inclusion criterion was English language studies reporting on the lifestyle factors associated with male infertility such as ‘smoking’, ‘alcohol’, ‘marijuana’, ‘cocaïne’, ‘anabolic steroids’, ‘diet’, ‘obesity’, ‘BMI’, ‘psychological stress’, ‘advanced paternal age’, and ‘caffeine’. The keyword search terms for each of these factors were used in combination with the following search terms: ‘male infertility’, ‘male fertility’, ‘sperm quality’, ‘semen parameters’, ‘DNA fragmentation’, ‘paternal’, ‘maternal’, ‘ART’, and ‘IVF’. Pertinent studies that were published prior to the 10-year timeframe were included at the discretion of the author, as were some studies on testicular heat stress, intense cycling training, lack of sleep, and exposure to electromagnetic radiation from mobile phone use. Reference lists of the reports were searched for further relevant citations, which were subject to the inclusion criteria. All non-English language studies and those without a published abstract were not included.

Results

In all, 1012 articles were identified from the database search and after reviewing the titles and abstract of the
reports, 104 articles met the inclusion criteria. Of these, 30 reports were excluded as the full-text could not be retrieved and the abstract did not have data on the association between the lifestyle factor of interest and male infertility. The remaining 74 reports were reviewed for data on the association between a particular lifestyle factor and male infertility. Available evidence pertaining to the potential adverse effects of these lifestyle factors on male fertility vary in strength. Certain factors, such as cigarette smoking and alcohol intake are likely to exert an additive effect, whilst other factors may pose a threat when exposed along with other environmental and occupational factors.

**Smoking**

Cigarette smoke contains >7000 chemicals, including highly carcinogenic tobacco-specific nitrosamines, [e.g. 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanone and N-nitrosonornicotine], polycyclic aromatic hydrocarbons (e.g. benzo[a]pyrene), and volatile organic compounds (e.g. benzene) [10]. Cigarette smokers have increased exposure to hazardous substances such as tar, nicotine (which is highly addictive), carbon monoxide, and heavy metals (e.g. cadmium and lead) [11].

Cigarette smoking is a known potential risk factor for decreased male fertility. Smoking is associated with leukocytospermia, a major endogenous source of reactive oxygen species (ROS). Moreover, tobacco smoke contains ROS at levels that can overwhelm the endogenous antioxidant defences. Increased seminal levels of ROS in smokers expose spermatozoa to oxidative stress, consequently impairing sperm function and ultimately compromising male fertility (reviewed in [12]). However, the mechanisms underlying the effects of smoking on sperm quality have not been fully clarified.

A large meta-analysis involving males from 26 countries/regions concluded that smoking causes a decline in sperm quality in both fertile and infertile men [13]. Sperm concentration in male smokers was reported to be typically 13–17% lower than that of non-smokers [14]. Moreover, cigarette smoking has been negatively associated with sperm count, motility, and morphology. The decline in semen quality was found to be more marked in heavy (>20 cigarettes/day) and moderate (10–20 cigarettes/day) smokers compared to mild smokers (1–10 cigarettes/day). The effect size was higher in infertile males than in the general population [15].

Besides its association with impaired male fertility, tobacco smoking is also responsible for increases in DNA damage, aneuploidies, and mutations in sperm [16]. One study suggested that the deterioration of semen quality amongst male smokers was correlated with increased DNA fragmentation rates and decreased expression of checkpoint kinase 1 (Chk1). Without the activation of Chk1 in response to DNA damage, there would be a decline in sperm repair leading to increased sperm apoptosis, which could lower semen quality [17].
Thus, the general effect of cigarette smoking on male fertility may result from the combined roles of elevated oxidative stress, DNA damage, and cell apoptosis, which could explain not only the reduction in semen quality, but also impaired spermatogenesis, sperm maturation, and sperm function reported to be present in smokers compared to non-smokers. Contributing factors leading to these effects in male smokers include the presence of nicotine and its metabolite, cotinine, benzo(a)pyrene, as well as cadmium levels [11]. For example, a meta-analysis of 13 317 men found that smoking was associated with higher mean testosterone levels, which could be attributed to the inhibition of testosterone breakdown by cotinine [18].

Pre-conception paternal smoking presents an increased risk of several morbidities in the offspring, which could perhaps be mediated via epigenetic modifications transmitted through spermatozoa. Male smokers showed a tendency towards increased alterations in methylation patterns genome-wide in their sperm DNA compared with never-smokers [19]. Maternal smoking during pregnancy and lactation could potentially cause harmful effects on male offspring fertility. Maternal cigarette smoke exposure during the gestation and weaning period was shown to cause diminished germ cell population, germ cell DNA damage, and defective sperm in the male offspring [20].

With paternal smoking being a significant risk factor for in vitro fertilisation (IVF) and intracytoplasmic sperm injection (ICSI) failure, paternal smoking could perhaps contribute to decreased assisted reproductive technology (ART) success rates as much as maternal smoking (a risk factor only for IVF failure) does or more [21]. Moreover, male smoking could even influence the clinical pregnancy rate per intrauterine insemination (IUI) cycle [22]. Amongst former smokers, every additional year following the male partner’s smoking cessation reduced the risk of ART failure by 4%, particularly between clinical pregnancy and live birth [23].

Despite there being no concrete potential relationship between smoking and male infertility as of yet, available evidence on cigarette smoking and male fertility support the recommendation of smoking cessation and minimising exposure to tobacco smoke amongst couples who are trying to conceive.

Alcohol

A meta-analysis involving 29 914 men reported a significant association between alcohol intake and lower semen volume, but not sperm parameters [13]. However, a recent meta-analysis involving 16 395 men reported that alcohol intake has a detrimental effect on semen volume and sperm morphology [24]. Direct exposure of spermatozoa to alcohol (at concentrations corresponding to that of serum after moderate and heavy drinking) was found to be harmful to sperm motility and morphology in a dose-dependent manner [25].

The actions of alcohol on the male reproductive system seem to occur at all levels of the hypothalamus–pituitary–gonadal (HPG) axis. Alcohol appears to interfere with the production of GnRH, FSH, LH, and testosterone, as well as impair the functions of Leydig and Sertoli cells. As a result, the production, morphological development and maturation of spermatozoa could be impaired [26]. Spermatogenesis appears to gradually decline with increasing levels of alcohol intake [27]. Partial or complete spermatogenic arrest and Sertoli cell-only syndrome were more commonly present amongst heavy drinkers compared to non-drinkers [28].

Chronic alcohol intake was found to have a detrimental effect on both semen quality and the levels of male reproductive hormones [29]. Conversely, a study comprising 8344 healthy male volunteers found that moderate alcohol intake was associated with higher testosterone levels but not with semen quality [30]. Chronic ethanol administration has been shown to decrease testicular steroidogenic and antioxidant enzyme activities resulting in increased oxidative stress [31], which could disrupt testosterone synthesis and compromise fertility.

A study on the male partners of couples facing primary infertility found that teratozoospermia was present in 63% and 72% of males who drank alcohol moderately (40–80 g/day) and heavily (> 80 g/day), respectively. None of the heavy alcohol drinkers were normozoospermic and most were oligozoospermic (64%), which is suggestive of progressive testicular damage in relation to increasing daily alcohol intake [32]. Similarly, another study found alcohol consumption rates to be significantly higher in men with severe oligozoospermia and with non-obstructive azoospermia compared to fertile controls [33].

Although the effects of alcohol on male reproductive function are dependent on the intake amount, a threshold amount of alcohol beyond which the risk of male infertility increases has not yet been determined. Moreover, it must be kept in mind that whilst alcohol intake and cigarette smoking alone did not affect sperm parameters, both habits together appear to exert an additive effect that could adversely alter sperm parameters [34].

Recreational drugs

Marijuana, cocaine, anabolic–androgenic steroids (AAS), opiates (narcotics), and methamphetamines are examples of illicit drugs that exert a negative impact on male fertility. The adverse effects of these drugs could impair the HPG axis, testicular architecture, and sperm function [35].

Cannabis or commonly referred to as marijuana, is the most abused illicit drug globally and has
predominantly male users. Regular marijuana smoking (more than once weekly within the last 3 months) was found to lower sperm concentration and total sperm count amongst young men, and this effect was further exacerbated when marijuana was used in combination with other recreational drugs [36]. Deregression of the endogenous cannabinoid system (ECS) was shown to significantly impair spermatogenesis resulting in lower total sperm count and motility [37]. Competition between the phytocannabinoids in marijuana and endocannabinoids for binding to the cannabinoid and vaniloid receptors leads to disruption of the homoeostasis of the ECS, which could consequently impair male fertility [38]. Sperm from infertile men have a marked modulation of endocannabinoid metabolism and reduction in endocannabinoid biosynthesis compared to sperm from fertile men [39].

Cocaine is a highly addictive, strong stimulant drug. Male rats given high doses of cocaine chronically before mating had lower pregnancy rates and offspring birth weights [40]. Both acute and chronic exposure to cocaine disrupted spermatogenesis and damaged the testicular ultrastructure [40,41]. These changes could have been brought about by cocaine-induced apoptosis [42]. Long-term (≥5 years) cocaine users were associated with lower sperm concentration and motility, and a higher fraction of sperm with abnormal morphology [43]. Compared to other infertile men, infertile cocaine users are more likely to have other concurrent risk factors of male infertility such as smoking, (other) substance abuse, as well as a prior history of sexually transmitted infections [44].

Testosterone and its derivatives comprise a family of hormones called AAS. AAS are used primarily by males to enhance their athletic performance and/or personal appearance [45]. The use of AAS has expanded beyond that of professional athletes and the prevalence of anabolic steroid-induced hypogonadism (ASHI) amongst young men and teenagers is on the rise [46]. A retrospective study found that ASIH was the most common cause of profound hypogonadism (<50 ng/dL testosterone) amongst men who sought treatment for hypogonadism [47]. Increased levels of exogenous testosterone, resulting from AAS use, exert a negative feedback on the HPG axis causing reversible suppression of spermatogenesis, testicular atrophy, and infertility. This may result in transient azoospermia with a recovery period of possibly even up to 2 years. Additionally, ASIH induced by AAS abuse can also result in loss of libido and erectile dysfunction [48]. Consequences from AAS use and subsequently treatment of the resulting hypogonadotropic hypogonadism depend on the dose, duration, and type of AAS used [49]. Some studies have pointed towards a more permanent repercussion of steroid use on male fertility and as such, AAS use should be highly discouraged [50].

Obesity

Obese men who attempt ART have reduced rates of live birth per ART cycle [51]. Increased paternal BMI is associated with decreased blastocyst development, pregnancy, and live-birth rates, but not early embryo development [60].

One pilot study investigated if maternal obesity could influence the semen quality of her male offspring. Increasing maternal BMI had a negative relationship
with the offspring’s inhibin B levels. Although the study found a suggestive trend of impaired fertility status in the offspring of overweight mothers, it was not statistically significant [61].

The severity of the consequences of obesity on the hormonal profile, sperm parameters, and DNA damage, as well as pregnancy outcomes may be varied due to the presence of other co-morbidities. Weight loss and lowering of BMI have helped improve sperm quality in some, but not all men [62].

**Psychological stress**

Stress, in its many forms, may be detrimental to male reproductive potential. The classical stress response activates the sympathetic nervous system and involves the hypothalamus–pituitary–adrenal (HPA) axis [63]. Both the HPA axis and gonadotrophin-inhibitory hormone (GnIH) exert an inhibitory effect on the HPG axis and testicular Leydig cells. The resulting inhibition of the HPG axis reduces testosterone levels. This leads to changes in Sertoli cells and the blood–testis barrier, which ultimately causes spermatogenesis to be suppressed. Impairment of testosterone secretion forms the main basis underlying the detrimental effects of psychological stress on spermatogenesis [64]. Raised corticosterone levels in stressed rats were found to suppress both testosterone and inhibin levels [65].

One study evaluated the effects of psychological stress on reproductive hormones and sperm quality in the male partner of infertile couples. The level of psychological stress was assessed using the Hospital Anxiety and Depression Score (HADS) questionnaire and 27% of the study population was found to have significant psychological stress. Men who were significantly stressed (HADS ≥8) had lower levels of testosterone, and higher levels of FSH and LH than men with normal HADS (HADS <8). However, GnRH levels were unchanged. Testosterone was negatively correlated, whilst FSH and LH were positively correlated with their HADS. Men with HADS ≥8 had a lower sperm count, motility, and normal morphology compared to those with normal HADS. Serum testosterone levels were positively correlated, whilst LH and FSH were negatively correlated with sperm count and motility [66].

Similarly, in an animal model study, acute restraint stress was shown to suppress sperm motility from 30 min of restraint onwards. Plasma levels of ACTH and corticosterone were elevated, whilst FSH, LH, and testosterone were decreased. It appears that the increased HPA axis activity had inhibited HPG activity [67].

A meta-analysis of 57 cross-sectional studies involving 29,914 participants reported that psychological stress could lower sperm concentration and progressive motility, and increase the fraction of sperm with abnormal morphology [13].

One study investigated the association between psychological stress in the form of occupational, life stress, family functioning, and semen quality. They found that occupational stress was negatively associated with semen quality, with a positive association between stress and percentage of sperm with DNA damage. Satisfaction with family functioning was negatively associated with the percentage of motile sperm cells. However, life stress did not correlate with semen quality [68]. Another study evaluated the associations between work-related stress, stressful life events, and perceived stress on semen quality. Of these, perceived stress and stressful life events were negatively associated with semen quality, particularly sperm motility and percentage normal morphology. However, work-related stress was not associated with semen parameters [69].

In another study amongst healthy volunteers, the effect of examination stress was investigated on seminal antioxidant content and sperm quality. During the stressful period just before examination, seminal glutathione and free sulphhydryl content, as well as sperm motility was reduced, whilst the percentage of sperm with abnormal morphology was higher compared to a non-stress period 3 months after examination [70]. Seminal antioxidant enzymes, superoxide dismutase (SOD) and catalase were also measured. During the stressful pre-examination period, stress scores and SOD activity was increased, whilst sperm concentration and motility were decreased compared to the post-examination non-stress period. However, catalase activity remained unchanged [71].

Psychological stress is associated with reduced paternity and abnormal semen parameters, and thus could be a causative factor in affecting male infertility.

**Advanced paternal age (APA)**

Advanced maternal age is defined as the age of 35 years, beyond which there is significantly increased risks of adverse reproductive outcome for women [72]. However, APA has not been as well-defined, with studies commonly defining it to be between 35 and 50 years of age or categorising it into age ranges of 5 years [73]. A meta-analysis of 90 studies involving 93,839 participants reported an age-associated decline in semen volume, sperm total, and progressive motility, normal sperm morphology along with an increase in DNA fragmentation. However, despite its decline over time, sperm concentration did not decline with increasing male age [74].

Analysis of semen parameters of healthy men over a wide age range (22–80 years) showed that semen volume and sperm motility declined gradually and continuously with age without a specific age threshold [75]. However, a retrospective study involving 5081 men (aged 16.5–72.3 years) suggested that the decline in sperm parameters and its corresponding age threshold were as follows:
total sperm count and total motile sperm - after 34 years of age; sperm concentration and fraction of sperm with normal morphology - after 40 years of age; sperm motility and progressive parameters of motile sperm - after 43 years of age; ejaculate volume - after 45 years of age; ratio of Y:X-bearing sperm in ejaculates - after 55 years of age. Thus, the authors proposed that independent of the woman’s age, the likelihood of pregnancy declines after intercourse with men aged > 34 years [76].

The exact mechanisms underlying the age-associated decline in male fertility have not been determined [77]. These age-dependent changes in semen quality could probably be attributed to normal physiological changes in the reproductive tract that occur with ageing, decreased capacity for cellular and tissue repair of damage induced by exposure to toxicants or diseases, and increased chances with age of having reproductive damage resulting from exogenous exposures such as smoking or infections [75]. The fact that both normal physiological and environmental factors could be held responsible for the effects of ageing on the male reproductive system adds to its complexity [78].

As men grow older, testicular function and metabolism deteriorates as the testis undergoes age-related morphological changes such as decrease in the number of germ cells, Leydig and Sertoli cells, as well as structural changes, including the narrowing of seminiferous tubules (reviewed in [78]). Concentrations of free and total testosterone steadily decline with increasing male age, leading to primary hypogonadism. Regulation of the HPG axis is also altered as men age. Accumulation of ROS in male germ cells throughout the course of ageing leads to oxidative stress and damage to sperm DNA. Apoptosis is also increased in the ageing testes (reviewed in [78]). These age-related changes inevitably lead to deterioration of sperm quality and quantity.

APA-induced increase in sperm DNA fragmentation adversely affects the success rates of ART outcomes, as well as early embryo development [79]. A study of donor ovum cycles indicated a 26% lower odds of live birth with each 5-year increase in paternal age [80]. In couples undergoing IVF, implantation and pregnancy rates decreased with increasing paternal age when the maternal age was between 30–34 years [81]. However, paternal age did not seem to affect ART outcomes when ICSI maternal age was between 30–34 years [81]. However, paternal age did not seem to affect ART outcomes when ICSI rates amongst males [84]. For example, the Mediterranean diet, which is enriched with omega-3 fatty acids, antioxidants, and vitamins, and low in saturated and trans-fatty acids, were found to be inversely associated with low semen quality parameters [84]. Thus, greater compliance to the Mediterranean diet may aid in improving semen quality [85].

Another study defined a typical ‘Western’-style diet as one that was high in red and processed meat, refined grains, and high-energy drinks, whilst a more ‘Prudent’ diet comprised mainly of white meat, fish, vegetables, and whole grains. The healthier ‘Prudent’ diet was positively associated with sperm progressive motility, but not sperm concentration and morphology [86]. In fact, a healthy dietary intake was reported to improve at minimum one measure of semen quality [87]. To begin with, the practice of substituting processed red meats with fish may have a positive impact on sperm counts and morphology [88].

Vegetables and fruits, fish and poultry, cereals and low-fat dairy products were amongst the foods positively associated with sperm quality. However, diets consisting of processed meat, full-fat dairy products, alcohol, coffee, and sugar-sweetened beverages were associated with poor semen quality and lower fecundity rates [84].

**Caffeine**

Most studies have not found an association between moderate caffeine intake and male fertility. A large meta-analysis with 29 914 participants found no significant effects of coffee consumption on semen quality [13]. Another study involving 2554 young Danish men also found no association between moderate caffeine (≤800 mg/day) or cola (≤1 L/day) intake and reduction in semen quality [89].

However, Belloc et al. [90] found that nearly 76% of caffeine consumers (3.0 ± 1.8 cups of coffee/day) had a
slight increase in semen volume, whereas fertile vasectomy patients who drank ≥6 cups of coffee/day presented with higher sperm motility [91]. A recent systematic review involving 19,967 men found that in most of the studies, semen parameters were affected by cola-containing beverages and caffeine-containing soft drinks, but not by caffeine intake from mainly coffee, tea, and cocoa drinks [92].

Caffeine intake may impair male reproductive function possibly through sperm DNA damage. The Ricci et al. [92] meta-analysis suggests that caffeine intake could be associated with double-strand DNA breaks and sperm aneuploidy, but not DNA adducts. Independent of age, healthy non-smoking men whose daily coffee intake amounted to >308 mg (~2.9 cups) have shown increased double-strand sperm DNA damage [93]. By contrast, the Bellonc et al. [90] study found that caffeine intake was associated with a lower risk of elevated DNA fragmentation (odds ratio 0.92, 95% CI 0.92–0.99).

Some studies have even reported that coffee consumption in males was associated with prolonged time to pregnancy [92]. Amongst couples undergoing IVF, male caffeine consumption was not significantly associated with live-birth, fertilisation and implantation rates [94]. Male caffeine intake was associated with a lower probability of achieving a clinical pregnancy and live birth per ART cycle, particularly in men consuming ≥272 mg caffeine/day [95]. Male consumption of coffee had a negative relationship with ICSI fertilisation rate, but did not seem to affect implantation, pregnancy, and miscarriage rates [96]. Amongst couples with successful IVF/gamete intra-fallopian transfer (GIFT) outcomes, male caffeine consumption had no effect on fertilisation, pregnancy or live-birth rates. However, the odds of multiple gestations increased by 3.0 (95% CI 1.2–7.4) for men who consumed an additional 100 mg caffeine/day daily during the week of the initial clinic visit [97].

The potential developmental toxicity of caffeine in the reproductive function of the male progeny has also been examined. Men exposed in utero to increasing maternal coffee consumption displayed a tendency toward decreasing semen volume and testosterone levels. Whilst current caffeine consumption in adult males was not associated with semen quality, males with high caffeine intake had increased serum testosterone levels compared to those with low intake [98]. Maternal coffee consumption during gestation and lactation impaired gonadal development and seemed to exert permanent detrimental effects on the reproductive potential of male offspring rats [99].

In summary, based on the current available data, there is no firm potential relationship between caffeine intake and male infertility.

Other lifestyle risk factors

Genital heat stress resulting from scrotal hyperthermia is a substantial risk factor for male infertility. Prolonged hours of sitting or exposure to radiant heat, varicocele, and cryptorchidism can all lead to testicular heat stress [100]. Elevated scrotal temperatures lead to spermatogenic arrest, germ cell apoptosis, oxidative stress, and sperm DNA damage [59]. Cycling as a sport is associated with increased generation of testicular heat. Intense cycling training for 16 weeks in young healthy male road cyclists was found to induce an increase in seminal ROS and malondialdehyde levels, along with a decrease in enzymatic antioxidants and total antioxidant capacity. These changes were maintained even after 4 weeks of recovery [101]. Furthermore, seminal interleukin levels were raised in these non-professional cyclists and sperm parameters were suppressed despite the 4 weeks of recovery [102].

Sleep disturbances may possibly have adverse effects on male fertility, as semen volume was lower in patients with difficulty in initiating sleep, including those who smoked or were overweight [103]. Sleep loss was found to affect sperm function in an animal study [104]. Constant use of electronic devices also contributes to poor sleep hygiene. Exposure to radiofrequency electromagnetic waves radiation emitted by mobile phone use could potentially exert harmful effects on the testes [105]. One meta-analysis found that mobile phone exposure is associated with reduced sperm motility and viability [106], whilst another study found this adverse effect on sperm motility and viability occurs only in vitro [107].

Conclusion

There are a wide variety of risk factors that could potentially influence sperm quality. These include lifestyle factors such as cigarette smoking, alcohol intake, use of illicit drugs, obesity, psychological stress, APA, diet, and caffeine intake. The adverse effects of these factors could even become intensified from one generation to the next, and then passed on to the resulting offspring. However, their negative effects can be overcome to a large extent by behaviour modification and better lifestyle choices. In this manner, the harmful impact of these factors on the male reproductive potential could also be alleviated and thus result in a more favourable outcome.

The evidence supporting the adverse effect of each risk factor on male fertility, as discussed in the present review, are of varying strength. Almost all the studies focus on the specific effects of one or at most two risk factors that were under evaluation. However, in reality, exposure to these risk factors does not occur individually but rather simultaneously, with each one being at
a varying duration and severity of exposure. It could then be said that without insight into the broader picture of these complex exposures, we may already be underestimating the consequences of each exposure.

The additive effects from the risk factors of male infertility such as smoking and alcohol intake on sperm parameters have been recognised. Moreover, the other groups of risk factors of male fertility, such as environmental and occupational factors, may also pose a simultaneous underlying threat to male fertility. Exposure to the confounding factor(s) should also be taken into consideration when planning the study design.

Perhaps by maintaining an overall positive lifestyle, the burden of the multiple factors that could influence sperm quality and male fecundity, may begin to slowly improve. In that respect, awareness and recognition of the possible impact of risk factors present in daily life is crucial amongst couples seeking conception. As the influence of several of these factors governing male infertility may be reversible, therefore the couple may benefit from early counselling and clinical intervention.

Conflict of interest

None.

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Lifestyle causes of male infertility

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