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THE IMPACT OF CIGARETTE SMOKING ON SEMEN QUALITY AND MALE FERTILITY: A REVIEW OF CURRENT EVIDENCE

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ABSTRACT

Male infertility represents a major global health concern, with lifestyle-related factors increasingly recognised as contributors to lowering reproductive potential. Cigarette smoking is one of the most significant modifiable risk factors affecting male fertility due to the presence of toxic compounds capable of inducing oxidative stress, DNA damage, and impaired spermatogenesis. This literature review summarises current evidence published in articles regarding the impact of cigarette smoking and alternative nicotine products on semen quality and male fertility. The reviewed studies consistently demonstrated that smoking is associated with reduced semen volume, sperm concentration, total sperm count, and motility, alongside increased sperm DNA fragmentation and epigenetic alterations. Oxidative stress emerged as the central pathogenic mechanism, reinforced by mitochondrial dysfunction, abnormal DNA methylation, reduced antioxidant capacity, and formation of DNA adducts. Conventional semen analysis may underestimate smoking-related fertility impairment, as molecular and epigenetic abnormalities may persist despite apparently normal semen parameters. Smoking cessation was consistently associated with improvements in semen quality, particularly in sperm concentration and total sperm count, with longer abstinence producing greater recovery. Alternative nicotine products, including e-cigarettes, snus, and hookah, were also linked to adverse reproductive effects, although their degree of toxicity may differ from that of conventional cigarettes. Overall, current evidence supports smoking cessation as an important intervention for improving male reproductive health and highlights the need for further research into molecular markers of fertility impairment beyond routine semen analysis.

KEYWORDS

Cigarette Smoking, Male Infertility, Semen Quality, Public Health

CITATION

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Introduction

Infertility is struggling to conceive in 12 month of couple's regular unprotected sexual intercourse, factors can be male, female or other. According to World Health Organization (WHO), this issue might affect 1 in 6 couples in reproductive age, with about 50% of the cases being due to male's component. Male infertility is divided into primary and secondary one, depending on whenever the individual has previously achived conception. A decline in male fertility can be associated with lifestyle factors, due to the lessen quality of sperm (Ali et al., 2025; Cargnelutti et al., 2023; Fan et al., 2024; Jafari et al., 2021; Mai et al., 2023).

Semen analysis is one of the key diagnostic tools used to assess male fertility providing information on numbers, motility, vitality and morphology of spermatozoa. According to 2022 WHO manual, the minimum for parameters mentioned above are 1.4mL of volume, 39mln of total count, 16mln/mL of concentration, total motility of 42%, vitality of 54% and normal morphology of 4% (Baldi et al., 2022; Cargnelutti et al., 2023).

Smoking is a significant lifestyle factor impacting fertility. Cigarettes are a well-known addictive substance, with their smoke containing over 6,000 chemicals, including carcinogens, heavy metals (ex. cadmium, lead, mercury) and irritating or toxic compounds like hydrogen cyanide, which are causing oxidative stress, contributing to the decrease in male fertility by damaging sperm DNA and reducing sperm production (Fan et al., 2024; Mai et al., 2023; Osadchuk et al., 2023).

Methodology

This literature review was conducted to summarise current evidence regarding the effects of cigarette smoking on semen quality and male fertility. Relevant articles were searched using the following keywords and combinations: “cigarette smoking”, “smoking”, “male infertility”, and “semen quality” through PubMed. Original studies, review articles, and meta-analyses evaluating the relationship between smoking and male reproductive parameters, published in English between 2020 and 2025 were included. Articles not related to male fertility, duplicate publications, conference abstracts, and studies without available full text were excluded.

Results

1. Mechanism

Tobacco smoke contains many toxic compounds, including polycyclic aromatic hydrocarbons, nicotine, cotinine, cadmium, lead, arsenic, and carbon monoxide (Farag et al., 2024). Nicotine and cotinine can penetrate the blood-testis barrier and accumulate in seminal plasma, affecting spermatogenesis and sperm function (Groppetti et al., 2024; Kleshchev et al., 2021; Osadchuk et al., 2023). Most commonly studies point to increased reactive oxygen species, reduced antioxidant capacity, and lower antioxidant enzyme activity after smoking (Amor et al., 2023; Henriques et al., 2023; Kaltsas et al., 2026; Osadchuk et al., 2023). The studies explicitly connect smoking-related oxidative stress to single-strand and double-strand DNA breaks, oxidative base damage such as 8-OHdG, chromatin damage, and increased sperm DNA fragmentation (Amor et al., 2023; Amor, Hammadeh, et al., 2022; Engel et al., 2021; Wu et al., 2025). Seminal zinc is described as part of the antioxidant defence, including support for superoxide dismutase, and smokers presenting lower seminal zinc alongside worse semen parameters and higher DNA fragmentation make a plausible feedback loop, where lower zinc levels cause weaker ROS buffering, contributing to sperm damage (Osadchuk et al., 2023). Beyond ROS, tobacco components can form DNA adducts, especially benzo[a]pyrene DNA adducts, which interfere with DNA replication and repair and contribute to mutations and DNA strand breaks (Amor et al., 2023; Osadchuk et al., 2023), making it a second route to fragmentation, separate from pure oxidative injury. Smoking is linked to altered DNA methylation and abnormal gene expression in sperm, including downregulation of PGAM5 and PTPRN2, which are helping protect cells from oxidative stress and supporting mitochondrial function. The study argues that smoking-related oxidative stress disrupts methylation patterns, producing aberrant gene expression and impaired sperm function (Amor et al., 2023; Naeimi et al., 2024). Nitric oxidate synthase is activated by smoking in regular smokers, further proving the oxidative stress mechanism, moreover mitochondrial uptake of fatty acids leading to inadequate energy supply, and disruption of phenylalanine and tryptophan metabolism suggesting explanation of compromised molecular machinery of the sperm even when conventional parameters appear normal (Yang et al., 2024) The stress-response study found increased HSP27 and phosphorylated HSP27, consistent with activation of the heat-shock response when sperm cells are under tobacco-induced stress (Henriques et al., 2023). The strongest unifying mechanism in these studies is oxidative stress, with DNA adduct formation, zinc loss, mitochondrial impairment, and altered gene regulation acting as reinforcing pathways rather than separate explanations.

2. DNA Fragmentation of Sperm

Sperm Chromatin Structure Assay (SCSA) can be used to evaluate male infertility, it is done by a flow cytometric test that detects sperm DNA sensitivity to denaturation, showing the degree of sperm DNA fragmentation in the DNA fragmentation index (DFI)(Ajayi et al., 2025). The clinical norm for DFI is <15%, 15-27% is considerable damage, and >27% is severe (Kleshchev et al., 2021; Nawale et al., 2025; Osadchuk et al., 2023). Smokers showed increased DNA fragmentation index by roughly 75% in heavy smokers compared to non-smokers, the DFI was higher in heavy smokers than moderate ones, leading to the conclusion of being dose-dependent (Osadchuk et al., 2023). Increased DFI was related to lower seminal zinc levels, supporting the mechanism of weakened antioxidant defence (Beigi Harchegani et al., 2020). In the Swedish snus study, authors measured DFI in both snus users and non-users, however did not find any significant difference between the two groups (Kimblad et al., 2022). In comparison of e-cigarette smokers and conventional cigarette smokers, the second group was found to have increased DNA fragmentation and teratozoospermia, although the DFI was not reported (Kim et al., 2025). That finding leads to the conclusion that the main factor of DNA strand breakage is cigarette smoke rather than nicotine itself (Du & Tuo, 2023).

3. Impact on Semen Quality

The most coherent conclusion of the studies is that smoking worsens the semen quality, especially lowering semen volume, sperm concentration or total count, and reducing motility in smokers (Al-Odat & Al-Fawaeir, 2025; Amoah et al., 2025; Fan et al., 2024; Kulaksiz et al., 2022; Osadchuk et al., 2023), but along with those findings are some differences. The Russian population study found smoking to cause lower semen volume, count, motility, higher DNA fragmentation, and lower seminal zinc, these findings showed broader harm of cigarettes than others the effects were not uniform across ethnic groups of this population study, showing the strongest impairment in Slavic men and weaker or absent in Yakut and Buryat men (Osadchuk et al., 2023). The Chinese infertility study showed a dose response, meaning heavy smokers had worse results, with primary infertility linked mainly to lower sperm concentration and secondary infertility linked mainly to lower progressive motility (Fan et al., 2024). The heavy smoking gene-expression study showed reduced standard semen parameters, along with downregulation of PGAM5 and PTPRN2, which supports a molecular damage mechanism rather than just a descriptive association (Amor et al., 2023). The Portuguese stress response study noted decreased semen volume and total sperm count, with only non-significant drops in motility and morphology (Henriques et al., 2023). The lifestyle study found smoking impaired native sperm motility, tho sperm telomere length was not clearly associated with lifestyle factors overall, meaning TL was not the main explanatory signal in that dataset (Sharqawi et al., 2022). The Greek study stands alone in reporting no association between smoking and any sperm parameter. Every other study that examined cigarette smoking found at least one significant adverse effect. The Greek finding may reflect methodological choices: the study used broad ordinal smoking categories and Spearman's rank correlation with no adjustment for confounders, and nearly 60% of participants were normospermic, which compresses the detectable range. The authors themselves note their correlation coefficients were very close to zero, rather than reporting formal hypothesis tests for smoking specifically. With the evidence from the other studies, the Greek results have less statistical power to prove no correlation between semen quality and smoking (Markantoni et al., 2022).

While the studies agree on cigarette smoking harming semen quality, ex. volume, concentration or count, and motility, the disagreements are mostly about which secondary markers change, the majority of the effect, and whether hormones, zinc, DNA fragmentation, or stress-response genes are the clearest downstream signals (De Brucker et al., 2020, 2025; Omolaoye et al., 2022).

4. Impact on Fertility

A crucial part of the impact on semen is its correlation with male fertility, as smoking being recognized as a major risk factor for infertility based on genotoxic components of tobacco (Amor et al., 2023). In a cigarette versus e-cigarette IVF study, among 296 couples undergoing IVF, conventional cigarette smokers had a live birth rate of 41.06% compared to 55.86% in the e-cigarette group ($p = 0.011$), and nearly three times the clinical miscarriage rate (36.27% versus 11.96%, $p < 0.001$). That stands in contrast to semen parameters, where semen concentration was higher in the conventional smokers group, but motility was better in e-cigarettes group, leading to the conclusion that conventional semen parameters were not as strong an indicator of live birth as male serum FSH and the number of 2PN embryos (Kim et al., 2025). Even tho it is observed that each decrease of 10 million sperm below a total count of 200 million correlates with about 3% lower chance of siring. Bringing that to the study of snus users, their chance of siring was decreased by approximately 10% (Kimblad et al., 2022). The correlation between worse semen parameters and lower expected fertility is more on a population level than an individual predictor for conception (De Brucker et al., 2025). The cessation of smoking is showing improvement of semen parameters, and then can be followed by better expected fertility outcomes, although they may not guarantee conception (Ragheb et al., 2025). Sawant et al. investigated sperm epigenetic age (SEA), a DNA methylation–based biomarker of biological sperm ageing, in two independent cohorts, including men from the general population and fertility clinics. The study found that SEA was not associated with conventional semen parameters, such as semen volume, sperm concentration, motility, total sperm count, or morphology, but advanced SEA was correlated to subtle sperm head abnormalities, which were increased head length and perimeter, greater prevalence of pyriform and tapered sperm, and reduced elongation factor (Sawant et al., 2024) suggesting that routine semen analysis might not find fertility-relevant changes seen in sperm epigenetic ageing, consistent with prior evidence linking advanced SEA to prolonged time-to-pregnancy. Smoking is known to accelerate epigenetic ageing, the study suggests a possible mechanism by which smoking may impair fertility even in men with apparently normal semen parameters. Importantly, the association between sperm epigenetic age and fertility-related changes remained significant even after adjusting for smoking status. In conclusion, the mechanism of worsened fertility is more due to the

damage of DNA, epigenetic changes, hormonal disruption or impaired capacitation than what can be measured by standard semen analysis (Amor et al., 2021, 2023; Amor, Jankowski, et al., 2022; Kim et al., 2025; Laqqan & Yassin, 2022; Osadchuk et al., 2023; Sawant et al., 2024).

5. Smoking Cessation

Smoking cessation appears to improve semen parameters, in particular volume, sperm concentration, and total sperm count (Kulaksiz et al., 2022; Ragheb et al., 2025). In a prospective Turkish study, Kulaksiz et al. evaluated 90 infertile heavy smokers who smoked at least 20 cigarettes daily for a minimum of one year. After quitting, the improvements were noticeable, specifically an increase in semen volume (2.48 to 2.90 mL), sperm concentration (18.45 to 22.64 million/mL), and total sperm count (45.04 to 65.1 million). The prevalence of oligozoospermia dropped from 70.8% to 54.2%. However, sperm motility and morphology improved only modestly and did not reach statistical significance (Kulaksiz et al., 2022). Comparable findings were reported by Ragheb et al. in an Egyptian cohort of 60 men enrolled in a structured smoking cessation program, where abstinence was verified using urinary cotinine testing, and participants were followed for both three and six months. Their results were noted in all measured parameters: semen volume (3.33 to 4.34 mL), sperm concentration (14.77 to 19.27 million/mL), total sperm count (49.33 to 84.01 million), progressive motility (20.7% to 42.28%), total motility (41.48% to 67.67%), and abnormal morphology (69.32% to 40.82%) (Ragheb et al., 2025). Observed improvements were time-dependent, with even better results after six months of abstinence (Ragheb et al., 2025). The difference in morphology and motility changes can be due to either longer observation, biochemical verification, and/or the inclusion of light smokers alongside heavy smokers. In Vanegas et al. study, the assisted reproductive technology (ART) outcomes in men quitting smoking were connected to 4% reduction in ART failure risk for each year since cessation (Vanegas et al., 2017), supporting smoking cessation as an important clinical intervention for improving male fertility outcomes.

6. Electronic Cigarettes and Alternatives

Despite the increasing popularity of alternatives to conventional cigarette smoking, their effects on semen quality and male fertility are still a concern. Snus, a smokeless tobacco option, was reported to have inconsistent effects on male reproductive parameters. Earlier studies suggested the users of snuff were not noticed to have reduced sperm count like conventional cigarettes users (Richthoff et al., 2008), however, in Kimblad et al. study, it was reported that snus users had a 24% lower total sperm count compared with non-users, which suggests that even smoke-free nicotine products are not harmless for male fertility, particularly given evidence that nicotine and cotinine crossing the blood-testis barrier and accumulating in seminal plasma, and affecting accessory glands, reducing semen volume (Kimblad et al., 2022).

Hookah smoking has also been associated with adverse reproductive effects. In Alenzi et al. study, hookah smokers showed lower testosterone, worse sperm count and morphology, the number of hookah sessions per week correlated with worse sperm motility. Semen volume was significantly reduced in hookah smokers compared with controls, while cigarette smokers in the same study did not show a comparable reduction (Alenzi, 2021). No significant differences were observed in semen volume, progressive sperm motility, total motility, sperm count, sperm morphology, and sperm vitality between hookah and cigarette smokers (Alenzi, 2021). Furthermore, except for semen volume, these semen parameters were not statistically different in both tested groups when compared to the control group (Alenzi, 2021), which stands in contrast to Hamadneh et al. study, where hookah smokers showed only lower semen volume compared to the control group, with no other parameters having significant differences (Hamadneh et al., 2025). In Albeitawi et al. study, morphology was significantly worse in all smoking groups, and dual smokers (hookah plus cigarettes) had the worst morphology (Albeitawi et al., 2020, 2024). Although hookah might be perceived as less harmful, hookah tobacco has a higher lead concentration than cigarette tobacco, nicotine and cotinine levels in seminal plasma are significantly higher than in serum after hookah use, and the mechanism of the antioxidant response of in sperm caused by oxidative stress might be comparable to one activated by conventional cigarettes (Firouzabadi et al., 2024; Tofighi Niaki et al., 2023).

Electronic cigarettes are another increasingly common alternative to conventional cigarettes. E-cigarette use may negatively affect sperm count, although potentially to a lesser extent than conventional smoking. Holmboe et al. reported significantly lower total sperm counts among daily e-cigarette users compared with non-users (91 million vs 147 million) (Holmboe et al., 2020), while Kim et al. observed lower sperm counts in e-cigarette users relative to conventional cigarette smokers (71,78 million vs 81,55 million)(Kim et al., 2025), in oppose to that results, the sperm motility, live birth rate and clinical miscarriage rate was better

among e-cigarette users than among cigarette smokers, suggesting that e-cigarettes may be comparatively less harmful, but they still impact male reproductive parameters compared to non-users (Bandara et al., 2023).

To conclude, alternative nicotine and tobacco products are not without reproductive risk and may affect semen quality and male fertility.

Conclusions

Current evidence strongly supports cigarette smoking as a significant modifiable risk factor negatively affecting semen quality and male fertility. The reviewed studies consistently demonstrated reductions in semen volume, sperm concentration, total sperm count, and motility among smokers, together with increased sperm DNA fragmentation and molecular abnormalities. Oxidative stress appears to be the principal mechanism underlying smoking-induced reproductive damage, while additional pathways including DNA adduct formation, mitochondrial dysfunction, altered gene expression, epigenetic aging, and impaired antioxidant defense further contribute to sperm dysfunction and reduced fertility potential. Smoking cessation should be regarded as an essential component of fertility prevention and treatment strategies. Alternative nicotine delivery systems, including e-cigarettes, snus, and hookah, also demonstrated potentially harmful reproductive effects and should not be considered risk-free substitutes for conventional cigarettes. Further research is needed to clarify the long-term reproductive impact of alternative nicotine products and to establish clinically applicable biomarkers capable of detecting fertility impairment beyond standard semen analysis.

Discussion

Several important limitations should be considered when interpreting the current evidence regarding smoking and male fertility. Most available studies are cross-sectional in design, including studies from Portugal, Jordan, China, Ghana, and those evaluating hookah or dual tobacco use, which limits the ability to establish causality and allows only the identification of associations at a single point in time (Al-Odat & Al-Fawaeir, 2025; Amoah et al., 2025; Henriques et al., 2023; Yang et al., 2024). In addition, many studies were conducted on relatively small cohorts, such as the Portuguese oxidative stress study, the telomere-length study, and smoking cessation trials, reducing statistical power and limiting subgroup analyses or the detection of modest effect sizes (Henriques et al., 2023; Kim et al., 2025). Several investigations also relied on retrospective chart reviews, introducing potential inaccuracies in data collection and limiting control over unmeasured confounding factors (Kim et al., 2025).

Another major aspect is the assessment of smoking exposure itself. Most studies depended primarily on self-reported smoking status rather than objective biochemical verification, increasing the risk of underreporting and exposure misclassification (Kulaksiz et al., 2022). Only a limited number of studies, such as the smoking cessation trial by Ragheb et al., used urinary cotinine testing to confirm abstinence (Ragheb et al., 2025). Furthermore, accurate dose-response assessment remains challenging, particularly for alternative nicotine products such as hookah, where inhalation depth, session duration, tobacco composition, and smoking frequency vary substantially between users (Albeitawi et al., 2020, 2024). Many studies also lacked detailed information regarding smoking intensity, duration of exposure, or concurrent use of multiple nicotine products, limiting the precision of exposure quantification.

Interpretation of reproductive outcomes is additionally complicated by the limitations of conventional semen analysis. Although many studies demonstrated associations between smoking and impaired semen parameters, only a small number evaluated clinically meaningful fertility outcomes such as pregnancy rates, live birth, or miscarriage. Most studies therefore, extrapolated fertility potential from semen quality alone, despite evidence suggesting that standard semen parameters incompletely reflect male reproductive capacity (Ragheb et al., 2025). This concern is reinforced by findings from the sperm epigenetic age study, which demonstrated associations between epigenetic sperm ageing and prolonged time-to-pregnancy despite the absence of abnormalities in conventional semen analysis (Sawant et al., 2024). Similarly, relatively few studies directly assessed sperm DNA fragmentation, despite growing evidence that DNA integrity may represent a more sensitive marker of smoking-related reproductive damage than routine semen parameters.

Population characteristics also limit the generalizability of many findings. A substantial proportion of studies recruited participants from infertility clinics, making it difficult to extrapolate the results to fertile populations (Kulaksiz et al., 2022; Ramírez et al., 2022). Conversely, some studies included only healthy sperm donors, limiting applicability to infertile men (Ortiz-Vallecillo et al., 2024). Ethnic and geographic homogeneity represents another concern, as some cohorts consisted predominantly of White participants, while other studies suggested that the reproductive effects of smoking may differ between ethnic groups (Osadchuk

et al., 2023; Sawant et al., 2024). Passive smoking exposure was also rarely assessed, potentially leading to an underestimation of differences between smokers and non-smokers in populations with high smoking prevalence.

Finally, residual confounding remains a persistent issue throughout the literature. Lifestyle factors commonly associated with smoking, including alcohol consumption, diet, caffeine intake, occupational exposures, and recreational drug use, were inconsistently controlled across studies (Al-Odat & Al-Fawaeir, 2025; Kim et al., 2025). Some studies additionally failed to distinguish never-smokers from former smokers within control groups, which may have diluted observed associations between tobacco exposure and semen quality (Albeitawi et al., 2024). Collectively, these limitations highlight the need for larger prospective studies with standardised exposure assessment, molecular fertility biomarkers, and clinically relevant reproductive outcomes to better define the true impact of smoking and alternative nicotine products on male fertility.

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