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

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ABSTRACT

Introduction: Introduction: Male infertility is an important public health issue, and lifestyle factors are increasingly recognized as key determinants of reproductive health. Cigarette smoking may negatively affect semen quality through oxidative stress, DNA damage, and hormonal disturbances, although findings remain inconsistent. This systematic review aimed to assess the association between cigarette smoking and semen quality in men.

Methods: Data were selected using the PRISMA flow diagram. PubMed and EMBASE were searched for studies published between 2005 and 2025 using the keywords smoking, cigarette smoking, semen quality, sperm concentration, sperm motility, sperm morphology, and male infertility. After screening according to predefined criteria, 29 studies were included. The selected studies examined the association between cigarette smoking and semen quality parameters, including sperm concentration, motility, morphology, and semen volume, in relation to male infertility.

Results: Seventeen studies were included in the qualitative synthesis. Cigarette smoking was associated with poorer semen quality, particularly lower sperm concentration, total sperm count, motility, and abnormal morphology. Smoking was also linked to oxidative stress, sperm DNA fragmentation, altered chromatin remodeling, membrane dysfunction, hormonal changes, and epigenetic abnormalities. Several studies suggested a dose-dependent effect, while smoking cessation was associated with improvement in selected semen parameters, especially semen volume, sperm concentration, and total sperm count.

Conclusion: Cigarette smoking appears to be an important modifiable risk factor for impaired semen quality and reduced male fertility. Its harmful effects involve both conventional semen abnormalities and deeper molecular sperm damage. Smoking cessation may partially reverse these changes, supporting its role in male infertility prevention and management.

KEYWORDS

Cigarette Smoking, Semen Quality, Male Infertility, Sperm Motility, Sperm Concentration, Sperm DNA Fragmentation

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Introduction.

Infertility is a significant global health problem affecting approximately 15% of couples of reproductive age worldwide. According to the World Health Organization (WHO), male factors contribute to nearly 30–50% of infertility cases, highlighting the importance of understanding determinants that may impair male reproductive potential [1].

Semen analysis remains the primary diagnostic tool used in the evaluation of male fertility and includes parameters such as semen volume, sperm concentration, sperm motility and sperm morphology. Alterations in these parameters may reflect impaired spermatogenesis and reduced fertilizing capacity. Numerous environmental and lifestyle factors have been suggested to influence semen quality, including diet, alcohol consumption, environmental exposures and smoking habits. Several population-based studies investigating lifestyle determinants of male reproductive health have demonstrated that behavioral factors, particularly cigarette smoking, may significantly influence semen quality parameters in men of reproductive age [2–4].

A growing body of epidemiological and clinical evidence indicates an association between smoking and impaired semen quality. Population-based studies have shown that tobacco smoking may be associated with decreased semen quality parameters, including reduced sperm concentration and motility [4]. Similarly, research evaluating lifestyle factors in large cohorts of men has identified smoking as one of the key behavioral exposures associated with altered semen characteristics [2]. Investigations conducted among infertile men have also demonstrated that heavy cigarette smoking, particularly when combined with alcohol consumption, may significantly impair sperm parameters [3]. However, the magnitude of this association may vary depending on smoking intensity, duration of exposure and individual susceptibility [5].

The biological mechanisms linking cigarette smoking with reduced semen quality are complex and involve several physiological pathways. One of the most frequently proposed mechanisms is oxidative stress. Cigarette smoke contains numerous toxic substances and reactive oxygen species (ROS) that may disrupt the balance between oxidants and antioxidants in seminal plasma. Increased oxidative stress has been associated with decreased antioxidant enzyme activity, leukocytospermia and impaired sperm motility [6]. In addition to oxidative damage, cigarette smoking may also affect sperm DNA integrity and cellular repair mechanisms. Studies investigating molecular alterations in sperm cells have reported that smoking may be associated with increased sperm DNA fragmentation and altered regulation of checkpoint kinase 1 (Chk1), a protein involved in DNA damage response pathways [7]. Furthermore, cigarette smoking has been linked to structural alterations in sperm chromatin. Abnormalities in the histone-to-protamine transition process, which is essential for proper sperm chromatin condensation and genome protection, have been observed in smokers [8]. Additional studies have reported altered expression of protamine transcripts in spermatozoa of smokers, suggesting that smoking may interfere with sperm nuclear protein composition and chromatin stability [9]. Recent investigations have also explored potential epigenetic consequences of smoking exposure in sperm cells, including alterations in DNA methylation patterns that may affect sperm function and fertility outcomes [10]. Moreover, emerging evidence suggests that smoking-related oxidative stress may also affect telomere integrity and increase ROS levels in sperm cells, which may further compromise sperm function and fertilizing capacity [11].

Interestingly, some studies suggest that smoking cessation may lead to improvements in semen parameters, including increases in sperm concentration and semen volume, indicating that the detrimental effects of smoking on male reproductive health may be at least partially reversible [12]. In addition, recent research has explored the combined effects of smoking and other substances, such as marijuana, further highlighting the complexity of lifestyle factors influencing male fertility [13].

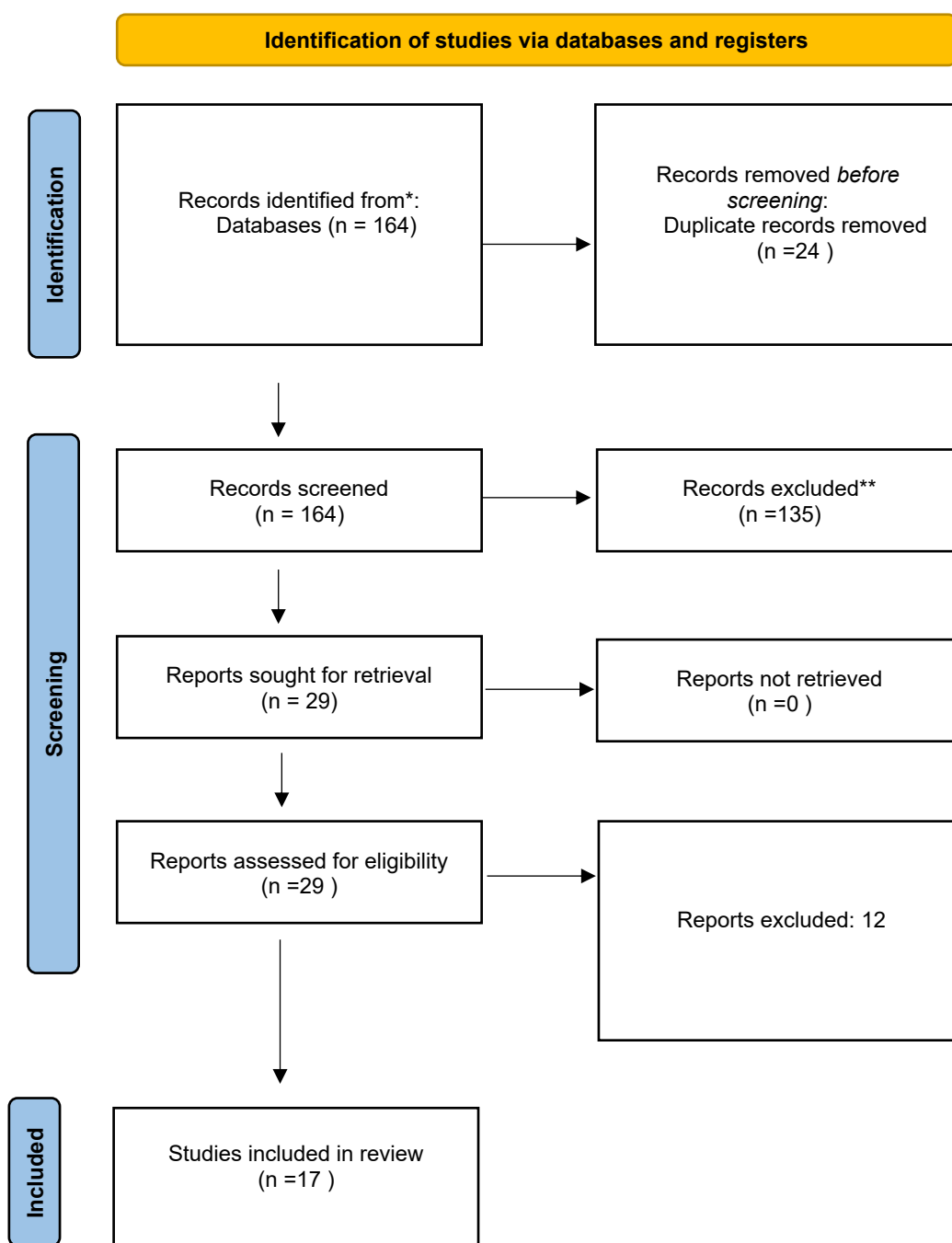
Although numerous studies have investigated the relationship between cigarette smoking and male reproductive health, the available evidence remains partially inconsistent. Some studies report significant deterioration of semen parameters among smokers, including decreased sperm concentration, reduced motility and increased sperm DNA damage, whereas others demonstrate weaker or non-significant associations depending on study population, smoking intensity and methodological differences. Furthermore, recent research has increasingly focused on molecular mechanisms such as oxidative stress, alterations in sperm chromatin structure, DNA fragmentation and epigenetic modifications. Despite the growing body of literature, the overall impact of cigarette smoking on semen quality and male fertility remains incompletely understood. Therefore, a comprehensive synthesis of available evidence is necessary to better characterize the relationship between cigarette smoking and semen quality parameters.

The aim of this systematic review was to evaluate the current literature investigating the association between cigarette smoking and semen quality parameters in men, including sperm concentration, motility, morphology and selected molecular markers related to sperm function.

Materials and Methods:

The data for the analysis were selected using the PRISMA flow diagram. The search phrases used were: “smoking” OR “cigarette smoking” AND “sperm concentration” OR “sperm motility” OR “sperm morphology” and “male infertility”.

A literature review was conducted following the **Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines**. The databases PubMed and EMBASE were searched for the period 2002-2025 for original studies investigating the association between cigarette smoking and semen quality parameters in men. The search strategy included combinations of keywords related to smoking and semen quality parameters.



After screening the identified records and applying predefined selection criteria, studies evaluating the relationship between cigarette smoking and semen parameters such as sperm concentration, sperm motility, sperm morphology and semen volume were selected for further analysis.

Based on the above analysis, 29 original studies met the inclusion criteria and were included in the final analysis. Duplicate records were removed before screening and the remaining articles were assessed based on title, abstract and full-text evaluation.

These studies investigated the impact of cigarette smoking on semen quality parameters and selected molecular markers related to sperm function and male fertility.

Results

The follow articles formed the basis of the analysis:

1. Osadchuk L, Kleshchev M, Osadchuk A. Effects of cigarette smoking on semen quality, reproductive hormone levels, metabolic profile, zinc and sperm DNA fragmentation in men: results from a population-based study. *Front Endocrinol (Lausanne)*. 2023 Oct 18;14:1255304. doi: 10.3389/fendo.2023.1255304. PMID: 37920251; PMCID: PMC10619690. [14]
2. Boeri L, Capogrosso P, Ventimiglia E, Pederzoli F, Cazzaniga W, Chierigo F, Dehò F, Montanari E, Montorsi F, Salonia A. Heavy cigarette smoking and alcohol consumption are associated with impaired sperm parameters in primary infertile men. *Asian J Androl*. 2019 Sep-Oct;21(5):478-485. doi: 10.4103/aja.aja_110_18. PMID: 30829290; PMCID: PMC6732890. [3]
3. Asare-Anane H, Bannison SB, Ofori EK, Ateko RO, Bawah AT, Amanquah SD, Oppong SY, Gandau BB, Ziem JB. Tobacco smoking is associated with decreased semen quality. *Reprod Health*. 2016 Aug 5;13(1):90. doi: 10.1186/s12978-016-0207-z. PMID: 27496053; PMCID: PMC4974764. [4]
4. Davar R, Sekhavat L, Naserzadeh N. Semen parameters of non-infertile smoker and non-smoker men. *J Med Life*. 2012 Dec 15;5(4):465-8. Epub 2012 Dec 25. PMID: 23346252; PMCID: PMC3539834. [5]
5. Al-Matubsi HY, Kanaan RA, Hamdan F, Salim M, Oriquat GA, Al Hanbali OA. Smoking practices in Jordanian people and their impact on semen quality and hormonal levels among adult men. *Cent Eur J Public Health*. 2011 Mar;19(1):54-9. doi: 10.21101/cejph.a3629. PMID: 21526658. [15]
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9. Cui X, Jing X, Wu X, Wang Z, Li Q. Potential effect of smoking on semen quality through DNA damage and the downregulation of Chk1 in sperm. *Mol Med Rep*. 2016 Jul;14(1):753-61. doi: 10.3892/mmr.2016.5318. Epub 2016 May 20. PMID: 27221653; PMCID: PMC4918538. [7]
10. Yu B, Qi Y, Liu D, Gao X, Chen H, Bai C, Huang Z. Cigarette smoking is associated with abnormal histone-to-protamine transition in human sperm. *Fertil Steril*. 2014 Jan;101(1):51-57.e1. doi: 10.1016/j.fertnstert.2013.09.001. Epub 2013 Oct 7. PMID: 24112532. [8]
11. Hamad M, Shelko N, Montenarh M, Hammadeh ME. The impact of cigarette smoking on protamines 1 and 2 transcripts in human spermatozoa. *Hum Fertil (Camb)*. 2019 Jun;22(2):104-110. doi: 10.1080/14647273.2017.1382733. Epub 2017 Oct 2. PMID: 28969455. [9]
12. Pasqualotto FF, Umezu FM, Salvador M, Borges E Jr, Sobreiro BP, Pasqualotto EB. Effect of cigarette smoking on antioxidant levels and presence of leukocytospermia in infertile men: a prospective study. *Fertil Steril*. 2008 Aug;90(2):278-83. doi: 10.1016/j.fertnstert.2008.02.123. Epub 2008 May 7. PMID: 18462724. [6]
13. Kulaksiz D, Toprak T, Tokat E, Yilmaz M, Ramazanoglu MA, Garayev A, Sulukaya M, Degirmen-tepe RB, Allahverdiyev E, Gul M, Verit A. Sperm concentration and semen volume increase after smoking cessation in infertile men. *Int J Impot Res*. 2022 Sep;34(6):614-619. doi: 10.1038/s41443-022-00605-0. Epub 2022 Aug 13. PMID: 35963898; PMCID: PMC9375087. [12]

14.Lin M, Feng Y, Zhang Y, He Q, Feng N. By ameliorating redox imbalance, patients of oligoasthenospermia increased sperm acrosin activity after smoking cessation in Chinese. *Rev Int Androl.* 2024 Dec;22(4):50-58. doi: 10.22514/j.androl.2024.030. Epub 2024 Dec 30. PMID: 39748770. [19]

15.Meri Z.B., Irshid I.B., Migdadi M., Irshid A.B., Mhanna S.A. (2013). Does cigarette smoking affect seminal fluid parameters? A Comparative study. *Oman Medical Journal*, 28(1), 12-15. <http://dx-1doi-1org-10000032x11d4.han3.wum.edu.pl/10.5001/omj.2013.03> [20]

16.Naeimi N., Mohseni Kouchesfehiani H., Heidari Z., Mahmoudzadeh-Sagheb H. (2024). Effect of smoking on methylation and semen parameters. *Environmental and Molecular Mutagenesis*, 65(1-2), 76-83. <http://dx-1doi-1org-10000032x11d4.han3.wum.edu.pl/10.1002/em.22583> [10]

17.Li W.W., Li N., Wu Q.Y., Xia X.Y., Cui Y.X., Huang Y.F., Yao Q. (2012)... *Zhonghua nan ke xue* = National journal of andrology, 18(12), 1093-1096. [21]

A total of 17 studies were included in the qualitative synthesis. Despite differences in study design, population characteristics, smoking intensity, and outcome measures, the overall direction of findings was largely consistent. Most of the included studies reported that cigarette smoking was associated with deterioration in at least one domain of male reproductive health. The adverse effects described in the selected articles involved conventional semen parameters, sperm functional capacity, oxidative balance, hormonal status, DNA integrity, chromatin remodeling, and epigenetic regulation. Importantly, several studies also suggested that the extent of semen impairment increased with smoking intensity, while the cessation studies indicated that some of these negative changes may be at least partially reversible after discontinuation of tobacco exposure.

In the population-based study by Osadchuk et al. (2023), the authors assessed the relationship between cigarette smoking and male reproductive parameters in a large cohort of men representing different ethnic backgrounds. Their analysis demonstrated that smoking, particularly when intensive, was associated with worse semen quality. Men who smoked heavily had lower semen volume, reduced sperm concentration, a lower total sperm count, and impaired motility compared with non-smokers. In addition to these conventional semen abnormalities, smoking was also linked with increased sperm DNA fragmentation, suggesting that the harmful effect of smoking extends beyond standard laboratory semen analysis and includes damage at the genomic level. The authors further reported that smokers had a less favorable metabolic profile and lower zinc levels, which may be relevant because zinc is important for normal spermatogenesis and antioxidant protection. An especially notable finding of this study was that the magnitude of smoking-related impairment was not identical across ethnic groups, implying that genetic or environmental modifiers may influence vulnerability to tobacco-related reproductive damage. [14]

The study by Boeri et al. (2019) focused on men evaluated for primary infertility and analyzed the relationship between heavy cigarette smoking, alcohol intake, and semen quality. The results showed that men with intense smoking exposure had poorer sperm parameters than non-smokers and lighter smokers. This impairment was particularly evident in sperm concentration, motility, and overall seminal quality. Moreover, the study suggested that the negative reproductive effects of smoking were even more pronounced when smoking coexisted with high alcohol consumption. In that sense, the article did not present cigarette smoking as an isolated factor only, but rather as one component of a harmful lifestyle profile associated with male infertility. An important clinical implication of this work is that men presenting with infertility and reporting heavy tobacco exposure may represent a subgroup at especially high risk of severe semen abnormalities. [3]

In the study conducted by Asare-Anane et al. (2016), tobacco smoking was clearly associated with a decline in multiple semen parameters. Compared with non-smokers, smokers had lower semen volume, lower sperm concentration, reduced total sperm count, and worse sperm motility and morphology. The authors also reported lower values of selected hormonal markers, including free testosterone and follicle-stimulating hormone, in the smoking group. One of the strongest aspects of this paper was that it linked smoking not only to average reductions in semen quality but also to a significantly higher probability of clinically relevant abnormalities such as oligospermia, asthenozoospermia, and teratozoospermia. The analysis therefore supports the conclusion that smoking is associated both with quantitative deterioration in semen characteristics and with an increased likelihood of pathological semen profiles. The authors additionally suggested a dose-related pattern, indicating that greater tobacco exposure may produce more severe impairment.[4]

Davar et al. (2012) evaluated semen quality in smoker and non-smoker men who were not necessarily selected from an infertile population. Their findings were somewhat less pronounced than in several other studies, as smokers showed lower sperm concentration, motility, and morphology, but the between-group differences did not reach statistical significance. Even so, the direction of the observed changes remained

consistent with the broader literature, with smokers tending to have worse semen quality than non-smokers. This study is relevant because it highlights that the strength of association may vary depending on the population examined, baseline fertility status, smoking load, and sample size. In other words, although not every study demonstrates statistically significant impairment in all parameters, the overall trend remains suggestive of a harmful influence of smoking on spermatogenesis and sperm quality. [5]

The Jordanian study by Al-Matubsi et al. (2011) provided additional evidence that smoking negatively affects semen quality. In their cohort, smokers had significantly lower sperm concentration and motility than non-smokers, whereas semen volume did not differ substantially between groups. The hormonal assessment revealed that smokers had altered levels of reproductive hormones, including higher testosterone and luteinizing hormone concentrations. The authors did not find a simple direct relationship between hormone concentrations and semen parameters, which led them to suggest that the reproductive harm of cigarette smoking is unlikely to be explained solely by endocrine mechanisms. Instead, their findings support the view that direct toxic effects of smoke constituents on the testes, epididymis, or sperm cells themselves may play a major role. The study therefore adds to the evidence that smoking-associated semen impairment can occur even when hormonal findings are not fully explanatory. [15]

The work by Collodel et al. (2010) approached the problem from a structural perspective by examining semen quality in idiopathic infertile smokers and non-smokers, including ultrastructural sperm assessment. Their observations suggested that smoking was associated with deterioration in sperm quality not only on routine semen testing but also at the level of sperm cell architecture. Ultrastructural analysis revealed abnormalities that may compromise fertilizing ability, supporting the idea that tobacco exposure disrupts the integrity of sperm organelles and subcellular organization. This study is important because it extends the concept of smoking-related reproductive toxicity beyond standard semen parameters and suggests that some smoking-induced defects may be invisible in routine analysis yet still biologically important for fertilization and embryo development. [16]

A distinctive perspective was offered by Axelsson et al. (2013), who investigated the reproductive consequences of parental smoking in adolescent men. Their data suggested that parental tobacco exposure, particularly when only one parent smoked, was associated with reduced sperm concentration and lower total sperm count in the sons. The effect was especially apparent for paternal smoking among men not exposed to maternal smoking, and maternal smoking also showed a negative association with sperm concentration when paternal smoking was absent. Interestingly, the men's own current smoking status was not significantly associated with semen parameters in this cohort. This pattern implies that the effect of smoking on male reproductive health may in some cases be established earlier in life, possibly during fetal or preconception development, rather than resulting solely from adult smoking behavior. The article therefore broadens the interpretation of smoking-related male infertility to include developmental and possibly intergenerational mechanisms. [17]

In the cohort study by Yang et al. (2015), smoking was identified as one of the lifestyle-related factors associated with semen quality. Although the study evaluated a wider range of behaviors and exposures, cigarette smoking emerged as a relevant determinant of poorer semen characteristics. The authors presented smoking as part of a broader network of modifiable lifestyle variables affecting male reproductive potential. This is clinically meaningful because it places tobacco exposure in a realistic, multifactorial context and supports the notion that smoking contributes to semen impairment in the general male population, not only among patients already presenting for infertility care. Even when not analyzed as the sole exposure, smoking remained one of the reproducibly unfavorable lifestyle factors linked to reduced semen quality. [18]

The study by Cui et al. (2016) investigated the molecular basis of smoking-related semen impairment. The authors reported that smoking was associated with poorer semen quality and increased sperm DNA damage. In parallel, they observed downregulation of checkpoint kinase 1 (Chk1), a protein involved in DNA damage response pathways. These findings suggest that cigarette smoking may compromise not only the structural integrity of sperm DNA but also the cellular systems responsible for recognizing and responding to such damage. This is an important mechanistic contribution because it helps explain why smokers may present with reduced fertility even when some conventional semen parameters are only moderately altered. A sperm cell carrying DNA instability may still appear present in adequate numbers but possess diminished reproductive competence. [7]

Yu et al. (2014) examined one of the key stages of sperm nuclear maturation, namely the histone-to-protamine transition. Their study showed that cigarette smoking was associated with abnormalities in this process, indicating defective chromatin condensation during spermatogenesis. Proper replacement of histones

by protamines is essential for achieving a tightly packed sperm nucleus and for protecting paternal DNA from damage. By demonstrating disruption of this transition in smokers, the authors provided a biologically plausible explanation for the increased DNA vulnerability observed in other studies. This article is therefore highly relevant to the pathophysiology of smoking-related male infertility, as it indicates that tobacco exposure interferes with one of the core molecular events necessary for normal sperm maturation. [8]

A related finding was reported by Hamad et al. (2019), who focused on protamine 1 and protamine 2 transcripts in spermatozoa. Their results showed that smoking had a negative effect on both semen quality and the expression profile of protamine-related transcripts. Since balanced protamine expression is essential for normal chromatin packaging, altered transcript levels may reflect defective sperm nuclear maturation and reduced fertilizing competence. The significance of this study lies in reinforcing the observation that smoking affects the sperm nucleus at the transcriptional and packaging level, not merely through gross semen deterioration. In practical terms, this means that the reproductive consequences of smoking may include subtle but important defects in sperm genome organization, potentially contributing to infertility even when standard semen analysis alone does not capture the full extent of damage. [9]

The prospective work by Pasqualotto et al. (2008) concentrated on the antioxidant milieu of seminal plasma. The study showed that smoking was associated with lower antioxidant levels and reduced sperm motility. The authors also examined leukocytospermia, which is clinically relevant because inflammatory cells are a major source of reactive oxygen species in semen. Their findings support the idea that tobacco exposure creates a pro-oxidative seminal environment, favoring oxidative injury to sperm membranes and intracellular components. This article contributes to the overall review by linking smoking-induced semen impairment with a plausible biochemical pathway, namely oxidative stress. It also supports the concept that the harmful effect of smoking on fertility may be mediated not only by direct toxic exposure but also by altered seminal defense mechanisms. [6]

An important practical perspective was provided by Kulaksiz et al. (2022), who evaluated infertile men before and after smoking cessation. The authors reported significant improvements in semen volume, sperm concentration, and total sperm count after cessation. Although motility and morphology also tended to improve, these changes were less marked statistically. This study is highly relevant clinically because it indicates that smoking-related reproductive impairment is not necessarily permanent and that quitting smoking may produce measurable benefits within a relatively short timeframe. The findings also strengthen the causal interpretation of the association between smoking and poor semen quality: if semen parameters improve after cessation, it becomes less likely that the observed abnormalities were due only to fixed confounders unrelated to smoking itself. [12]

The study by Lin et al. (2024) further explored the consequences of smoking cessation, particularly in men with oligoasthenospermia. The authors showed that cessation was associated with improved sperm acrosin activity and a more favorable redox state, reflected by lower oxidative burden and improved antioxidant balance. These results suggest that quitting smoking may restore at least some specific sperm functions that are directly relevant for fertilization, even if the overall magnitude of improvement in standard semen parameters is not always dramatic. This article is especially valuable because it points to recovery at a functional and biochemical level, not solely in conventional semen counts. It also reinforces the central role of oxidative imbalance as a mediator of smoking-induced reproductive dysfunction. [19]

In the comparative study by Meri et al. (2013), smoking was associated with deterioration in seminal fluid quality, especially in relation to sperm motility, abnormal morphology, and leukocyte content. The authors also noted that more intense smoking exposure was linked to lower sperm concentration and more pronounced semen abnormalities. The increase in leukocytes is particularly important because it suggests a potential inflammatory or pro-oxidative component of smoking-related semen damage. This study therefore complements the oxidative stress literature and supports the interpretation that smoking impairs fertility through multiple overlapping mechanisms, including poorer sperm motility, altered morphology, and a less favorable seminal microenvironment. [20]

The epigenetic study by Naeimi et al. (2024) extended the evidence base by investigating methylation changes in infertile smokers. Their findings indicated that smoking was associated with worse semen parameters and with altered methylation status of the CHD5 gene. Smokers had lower sperm concentration than infertile non-smokers, and aberrant methylation patterns were more frequent in the smoking group. These results suggest that cigarette smoking may adversely affect male fertility not only through direct oxidative or toxic pathways but also through epigenetic modifications that could alter gene expression during spermatogenesis. This is particularly important in the context of modern reproductive medicine, because

epigenetic abnormalities may have implications beyond fertilization itself, potentially affecting early embryonic development and offspring health. [10]

The final article in the set, corresponding to Li et al. (2012), addressed sperm plasma membrane integrity. The study indicated that cigarette smoking adversely affected membrane stability and function. This is a meaningful addition to the overall picture because sperm plasma membrane integrity is critical for motility, capacitation, acrosome reaction, and successful fusion with the oocyte. Damage at this level may therefore reduce reproductive potential even if basic semen counts remain within a range that appears only mildly abnormal. By focusing on membrane function, this paper adds another mechanistic layer to the evidence that smoking impairs male fertility through multiple distinct but interconnected biological pathways. [21]

Across the included studies, cigarette smoking was most consistently associated with poorer conventional semen parameters, particularly reduced sperm concentration, motility, total sperm count, and abnormal morphology. Several articles also showed that smoking-related reproductive impairment extends beyond routine semen analysis and involves oxidative stress, sperm DNA damage, altered chromatin remodeling, membrane dysfunction, and epigenetic changes.

Discussion

The findings of this systematic review indicate that cigarette smoking has a negative effect on semen quality and male reproductive potential. Across the included studies, smoking was most consistently associated with deterioration in conventional semen parameters, particularly sperm concentration, total sperm count, motility, and morphology. Although the strength of these associations varied between studies, the overall direction of the findings was largely uniform, supporting the view that smoking is an important modifiable risk factor for impaired male fertility.

One of the main observations from the reviewed literature is that the harmful effect of smoking extends beyond routine semen analysis. In addition to poorer conventional semen parameters, several studies demonstrated associations between smoking and increased oxidative stress, reduced antioxidant capacity, sperm DNA fragmentation, abnormal chromatin remodeling, altered protamine expression, impaired membrane integrity, and epigenetic abnormalities. This suggests that cigarette smoking affects multiple stages of sperm production, maturation, and function. Therefore, the impact of smoking on fertility may be underestimated when evaluation is based solely on standard semen parameters.

Oxidative stress appears to be one of the most plausible mechanisms underlying smoking-related reproductive damage. Sperm cells are especially vulnerable to oxidative injury because of the composition of their plasma membranes and their limited antioxidant defenses. The studies included in this review support the concept that cigarette smoke promotes a pro-oxidative seminal environment, which may impair sperm motility, damage membranes, and increase DNA fragmentation. This is consistent with the observation that some of the smoking-related abnormalities involved not only semen quality but also sperm function and genomic stability.

Another important finding is that several studies suggested a dose-dependent relationship, with heavier smoking being associated with more severe semen impairment. This pattern strengthens the biological credibility of the observed association and implies that smoking burden should be considered during clinical assessment. At the same time, cessation studies included in this review showed that some parameters, particularly semen volume, sperm concentration, and total sperm count, may improve after quitting smoking. Improvements in selected functional markers, such as acrosin activity and redox balance, were also reported. These findings suggest that at least part of the reproductive damage associated with smoking may be reversible, which is clinically relevant for men attempting conception.

The reviewed studies also indicate that the effect of smoking is not restricted to one biological pathway. While some papers emphasized conventional semen abnormalities, others highlighted molecular damage involving sperm DNA integrity, protamine regulation, chromatin condensation, or methylation changes. This multidimensional pattern suggests that tobacco smoke may affect fertility through overlapping toxic, oxidative, inflammatory, and epigenetic mechanisms. Hormonal disturbances were also observed in some studies, although these findings were less consistent than those related to semen quality and molecular sperm damage.

Despite the overall consistency of the evidence, several limitations should be considered. The included studies differed in design, population characteristics, smoking intensity, and laboratory methodology. Most were observational, which means residual confounding cannot be excluded. In addition, smoking often coexists with other lifestyle-related factors that may influence reproductive health, including alcohol use, diet, and environmental exposures. Another limitation is that not all studies assessed advanced sperm function markers, making direct comparison across publications difficult. Nevertheless, the repeated observation of poorer

reproductive parameters in smokers across different populations supports the reliability of the overall conclusion.

From a clinical perspective, the available evidence suggests that cigarette smoking should be routinely considered during male fertility evaluation. A detailed smoking history may be important in men with subfertility, abnormal semen analysis, or unexplained reproductive failure. Since some studies demonstrated improvement after smoking cessation, quitting smoking should be regarded not only as a general health recommendation but also as a potentially relevant element of fertility management.

In summary, the reviewed literature supports the conclusion that cigarette smoking adversely affects male reproductive health on both conventional and molecular levels. Its negative impact involves semen quality, sperm function, and genomic integrity, while smoking cessation may provide at least partial recovery. These findings underline the importance of including tobacco exposure among the major preventable factors in the assessment and counseling of men with fertility concerns.

Conclusions

This systematic review indicates that cigarette smoking is consistently associated with impaired male reproductive health. Across the included studies, smokers generally presented poorer semen quality than non-smokers, particularly with respect to sperm concentration, total sperm count, motility, and morphology. In addition to these conventional semen abnormalities, smoking was also linked to oxidative stress, sperm DNA fragmentation, disturbed chromatin remodeling, membrane dysfunction, inflammatory changes, hormonal alterations, and epigenetic abnormalities.

The reviewed evidence suggests that the effect of smoking on fertility is multifactorial and biologically plausible. Cigarette smoke appears to impair sperm not only through direct toxic exposure, but also through oxidative and molecular pathways that reduce sperm function and genomic integrity. Several studies further indicated that heavier smoking is associated with greater reproductive impairment, while smoking cessation may lead to partial recovery of semen quality and selected sperm functional markers.

Although heterogeneity between studies and the predominance of observational data should be taken into account, the overall body of evidence strongly supports cigarette smoking as a major modifiable risk factor for reduced male fertility. Therefore, smoking cessation should be considered an essential component of fertility counseling, prevention strategies, and the clinical management of men presenting with subfertility or abnormal semen parameters.

Further well-designed prospective studies are needed to better define the reversibility of smoking-related sperm damage, the timeline of recovery after cessation, and the long-term significance of molecular and epigenetic abnormalities associated with tobacco exposure.

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