

A REVIEW ON OXIDATIVE STRESS AND ANTIOXIDANTS IN SPERM HEALTH AND MALE INFERTILITY

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Abstract

Background:

Male infertility is a critical health issue in the world and oxidative stress has been identified as a major causal factor influencing the functionality of sperm. The ratio between antioxidant defenses and reactive oxygen species (ROS) has been reported to be involved in the impaired quality of semen, especially in idiopathic infertility.

Objective(s):

To evaluate the association between oxidative stress markers and male infertility outcomes, and to review their diagnostic and therapeutic implications with respect to antioxidant balance.

Methodology:

Electronic databases were used to conduct a systematic literature review based on PubMed, Google Scholar, ScienceDirect, and SpringerLink. The inclusion and exclusion criteria were pre-established by challenging the quality of peer-reviewed articles published since 2020 and 2026. The number of reviewed studies, clinical studies, and experimental studies analyzed was 50 pertinent articles. The

qualitative synthesis of data was done in order to evaluate sources of ROS, oxidative damage mechanisms, sperm functionality effects, and antioxidant system role.

Results:

The results showed that oxidative stress was common in a considerable percentage of infertile men and it was closely related to low sperm count motility, morphology and integrity of sperm DNA. Endogenous sources (leukocytes, immature spermatozoa, mitochondrial dysfunction) as well as exogenous factors (smoking, environmental toxins, radiation) led to the rise in the level of ROS. Oxidative stress caused damage to the sperms using lipid peroxidation, DNA fragmentation, protein oxidation and mitochondrial dysfunction. Protective role was played by antioxidant defense system including enzyme (superoxide dismutase, catalase, glutathione) and non-enzyme (vitamins C and E, zinc, selenium). Whereas antioxidant supplementation was associated with a positive change in semen parameters, clinical outcomes were not uniform.

Conclusion:

Oxidative stress is the focus of male infertility as it is a pathogen that disrupts both the structure and the functioning of sperms in various ways. Although the antioxidant therapy is promising, more studies are needed to come up with standardized diagnostic tools and evidence-based treatment plans.

INTRODUCTION

The process of organ development and maturation, neuroendocrine regulation, hormone synthesis, meiosis and mitosis are all biological processes that are involved in the ability to reproduce ⁽¹⁾. Fertility refers to a clinical pregnancy ⁽²⁾. Infertility on the other hand is the capacity to conceive following 12 months of usual, regular and unguarded intercourse. Approximately 85 percent of infertile couples has a specific cause of infertility with the best frequent causes being male element infertility, tubal pathology, and ovulatory dysfunction. The rest 15 percent cases are scattered as unexplainable infertility. The obesity, smoking, and other environmental and lifestyle conditions might impair the fertility ability ⁽³⁾.

Male infertility is a complicated disease that can arise because of the malformation at different levels of the male reproductive system. There are broad categories of male infertility that are chosen based on the localization of dysfunction namely pre-testicular, testicular and post-testicular categories ⁽⁴⁾. The importance of these categories in recognizing underlying mechanisms and offering proper clinical control is important.

Causes of Male Infertility

Pre-testicular

- Acquired endocrinopathies
- Genetic endocrinopathies
- Disorders of GnRH secretion
- Disorders of LH, FSH & androgen function

Testicular

- Varicocele
- Genetics
- Cryptorchidism
- Exposure to gonadotoxins

Post-testicular

- Obstruction
- Immunologic infertility
- Disorders of ejaculation
- Erectile dysfunction

Figure 1.1: Classification of causes of male infertility into pre-testicular, testicular, and post-testicular factors ⁽⁵⁾

Pre-testicular causes of male infertility are primarily related to the issues of the malfunction of hypothalamic-pituitary-gonadal (HPG) axis to which the production of reproductive hormones is regulated. This axis is very crucial in the process of spermatogenesis since it controls the secrets of follicle-stimulating hormone (FSH), the luteinizing hormone (LH), and testosterone ⁽⁴⁾.

The testicular infertility is also very dependent on the genetic disorders. Klinefelter syndrome is a disease, which is associated with chromosomal disturbances (47,XXY karyotype as a rule) resulting in impaired testicular differentiation and demyelogeny. Other genetic factors like deletion and mutation of spermatogenesis of the Y-chromosome are other causes of infertility. Infections of the

testes such as orchitis may also inhibit normal sperm production. Orchitis is generally believed to be brought about by viral infections like mumps and can lead to inflammation, testicular atrophy and permanent destruction of the seminiferous tubules ⁽⁵⁾.

Male infertility is also post-testicular and linked with a blockage of the sperm transport, maturation or ejaculation. The conditions can even stop such occasional access of the sperm to the female reproductive system despite normal spermatogenesis ⁽¹⁴⁾. A high percentage of obstruction to the male reproductive duct is part of the post-testicular causes. The obstructions could be in the epididymis, the vas deferens, or ejaculatory ducts either congenitally, due to infections, surgery or trauma. Indicatively, CFTR gene mutations in majority of the cases are associated with a congenital absence of the vas deferens which determines obstructive azoospermia ⁽⁶⁾.

It has been accepted that infertility is a major global health problem. Approximately 40 million infertile couples were treated in 2010. Among them nearly 34 million couples reside in the developing nations. Approximately there are 48.5 million infertile couples all around the world. The number is said to be high among female populations ⁽⁷⁾. Those most prevalent were in the South Asian region, sub-Saharan Africa, North Africa and the Middle East, Central and Eastern Europe and Central Asia. Although males were reported to be the cause of 2030 percent of infertility, it was not the case in the world. Actually, the optimal sperm parameters are observed in no more than 2 per cent of men and in as high as 50 per cent of infertility cases they have male factor infertility with considerable geographic variation ⁽⁸⁾.

roles ⁽²²⁾.

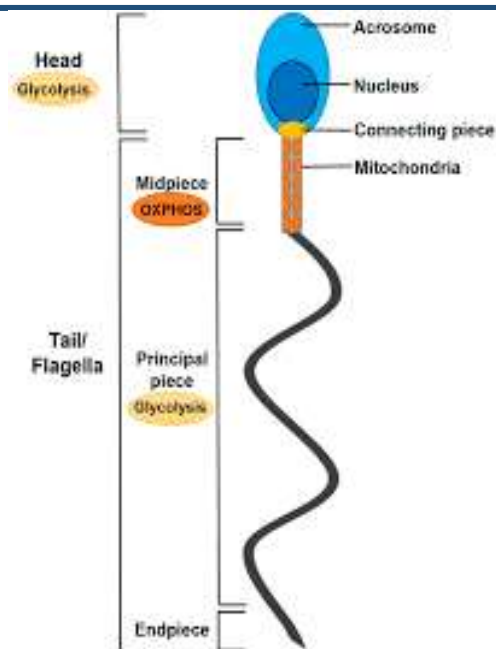


Figure 1.2: Structure of spermatozoon showing head, midpiece, and tail with energy metabolism sites⁽²³⁾

Sperm motility is a property of sperm to move actively and effectively. It plays a major role in determining the fertility rate because sperm has to pass through cervical mucus, uterus, and fallopian tubes to access the oocyte. The motility requires the production of ATP in the mitochondria and motion of the flagellum. The decrease in motility is generally linked to the existence of oxidative stress that ruins the functionality of the mitochondrion and the structural integrity of the flagellum⁽⁹⁾.

Overproduction of reactive oxygen species (ROS) leads to oxidative stress, which has a negative influence on the construction and activity of sperm by a variety of interdependent pathways. Spermatozoa are also highly susceptible to oxidative damage, as they contain large proportion of polyunsaturated fatty acids and lack an inherent defense against oxidants⁽¹⁰⁾. The significant changes through which oxidative stress may disrupt sperm functions are lipid peroxidation, DNA damages, oxidation of proteins, and mitochondrial dysfunction⁽¹¹⁾.

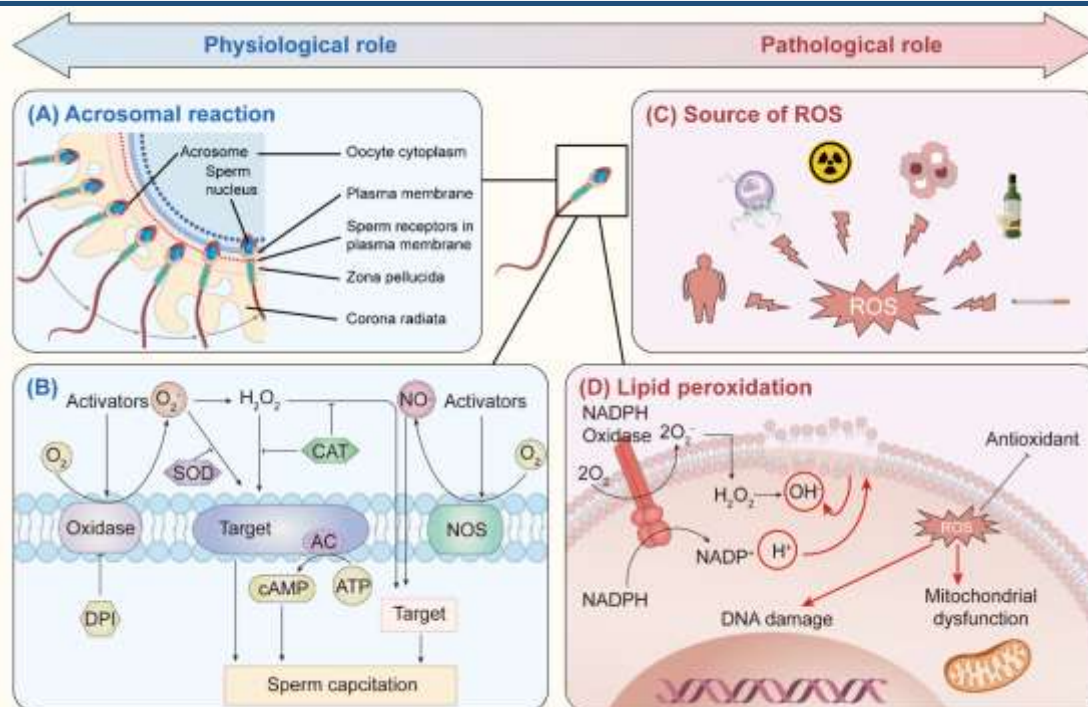


Figure 1.3: Physiological and pathological roles of ROS in sperm function and male infertility

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Lipid Peroxidation

One of the leading processes in which the cells of spermatozoa are damaged by oxidative stress is via lipid peroxidation. Polyunsaturated fatty acids (PUFAs) are abundant in the plasma membrane of sperm cells and prone to attack by reactive oxygen species especially hydroxyl radicals. The interaction between ROS and these lipids results in the formation of a chain reaction called lipid peroxidation⁽⁵⁸⁾. This results in the generation of lipid radicals and toxic by-products like malondialdehyde (MDA), which also continues to spread the damage of the membrane. Consequently, sperm membrane structure and fluidity are affected. Plasma membrane damage influences some vital sperm functions. It affects the permeability of membranes, ion exchange, and interferes with signal transduction events needed during capacitation and acrosome reaction

The main functions of the antioxidants are to counteract the surplus reactive oxygen species, and to preserve the fine balance between the oxidants and antioxidants in the male reproductive system⁽⁷⁶⁾. Antioxidants inhibit lipid peroxidation, fragmentation of DNA and oxidation of proteins in sperm cells by scavenging the effects of free radicals. Sperm integrity is important in ensuring proper sperm motility, membrane functioning, and fertilization⁽⁷⁷⁾. Antioxidants also facilitate the functioning of the mitochondria to supply sufficient energy to the sperm movement. Moreover, they contribute to the maintenance of structural and genetic stability of spermatozoa that is essential in fertilization and the development of healthy embryos.

Review of Literature

Krausz *et al.*, 2022 reported almost 7% of men suffer from disorders of fertility due to abnormalities at the pretesticular, testicular, or posttesticular level. Genetics seems to be responsible for about 20% of the cases. Genetic abnormalities like chromosomal defects and monogenic defects can make genetic screening a necessary part of the workup of infertile men presenting with severe oligozoospermia or azoospermia. Karyotypic abnormalities, like Klinefelter syndrome, chromosomal translocations or inversions, and Y-chromosome microdeletions, were the most important chromosomal contributors; all etiologic categories contained monogenic mutations. Congenital absence of the vas deferens was a significant posttesticular cause of male infertility and was often due to mutations in the CFTR gene. Advancements in next-generation sequencing technologies have helped identify more genetic determinants, especially in the case of pretesticular and testicular conditions, including congenital hypogonadotropic hypogonadism, which has been connected to more than 35 genes. Male infertility is observed as a manifestation of various other syndromic conditions such as Bardet-Biedl, Prader-Willi, primary ciliary dyskinesia, Noonan syndrome and myotonic dystrophy. An improved understanding of the genetic basis of male infertility is indispensable for accurately diagnosing male infertility and assessing the risk of genetic transmission by assisted reproductive techniques⁽¹²⁾.

According to Casarini L et al. (2020), follicle-stimulating hormone (FSH) had been a major contributor to follicle spermatogenesis by acting on the Sertoli cells via its receptor (FSHR). They observed that hormonal agents acting on this pathway were used in the treatment of male infertility, specifically in the treatment of hypogonadotropic hypogonadism, where fertility had been successfully restored with agents such as gonadotropin-releasing hormone (GnRH) or human chorionic gonadotropin (hCG) or in combination with the follicle stimulating hormone (FSH). Nevertheless, the efficacy of FSH in the treatment of idiopathic male infertility had been less evident because of the scanty evidence. The review also talked about the new pharmacological strategies that were being developed including new compounds like allosteric ligands and chimeric gonadotropins which were promising to be used as therapy options in personalized ways later on in male infertility⁽¹³⁾.

According to Agarwal A et al. (2021), the pathophysiology of male infertility had implicated oxidative stress as its key element and that antioxidant treatment had presented a promising perspective in its treatment. A systematic review of 97 studies selected by them helped to evaluate the effectiveness of different antioxidants in different clinical situations and determine the effects these antioxidants have on the semen parameters and reproductive outcomes. The results showed that antioxidant supplementation had positively impacted the semen quality, which had included the sperm count, motility, and morphology and had specifically played a significant role in such conditions like varicocele, idiopathic, and unexplained male infertility. It also provided emphasis on the necessity of stronger randomized controlled trials, but suggested the evidence-based clinical guidelines to facilitate the proper usage of antioxidants to treat male infertility⁽¹⁴⁾.

Cilio S et al. (2022) stated that millions of people worldwide had already become infertile, and male factors contributed to a considerable percentage of the problem but stressed that oxidative stress had been closely linked with the low semen quality. They found in their review of 2011-2021 that an increase in reactive oxygen species had led to sperm DNA, protein, and lipid damage, resulting in impaired motility, morphology and vitality. The results indicated that the antioxidant

supplementation, inositol, alpha-lipoic acid, zinc, folate, coenzyme Q10, selenium and vitamin B12, had positively changed semen parameters, which entailed minimizing oxidative damage, increasing mitochondrial activity and maintaining hormone production⁽¹⁵⁾.

According to Ali M et al. (2021), oxidative stress, which is caused by an unequal balance of reactive oxygen species and antioxidants, had been one of the key factors causing male infertility because of its contribution to DNA fragmentation of sperm and aberrant semen characteristics. They emphasized that antioxidant therapy was suggested as a possible treatment that could lead to the minimization of oxidative damage and semen quality improvement, because of its availability and relative non-toxicity. Although earlier research had indicated positive results of using antioxidant supplementation in the treatment of male subfertility, recent studies had shown differing results, concern has been raised on whether it is effective in general⁽¹⁶⁾.

Amorini AM et al. (2021) stated that reactive oxygen species (ROS) had been crucial in the physiological functions of sperm maturation, although excessive production of reactive oxygen species, in most cases, was accompanied by the formation of reactive nitrogen species, which resulted in oxidative and nitrosative stress, damaging proteins, lipids, and DNA and compromising sperm functioning and viability. Their search of literature covering the last ten years revealed that despite the studies on antioxidant-based therapies as a treatment of male infertility having been done, the available evidence was scarce and unclear. Only few percent of studies had measured important outcomes like the rate of pregnancy or live births and very few had shown significant clinical advantages⁽¹⁷⁾.

MATERIAL AND METHODOLOGY

This systematic review analyzed the role of oxidative stress and antioxidants in sperm health and male infertility. Conducted over four months in the Department of Medical Laboratory Technology, Superior University Lahore, the study used a non-probability purposive sampling technique to select 40–60 peer-reviewed articles published between 2020 and 2026 from databases such as PubMed,

Google Scholar, ScienceDirect, SpringerLink, Wiley Online Library, Nature, and Elsevier. Inclusion criteria focused on English-language studies addressing oxidative stress, reactive oxygen species (ROS), antioxidant defenses, and sperm function, while articles before 2010, non-peer-reviewed sources, and unrelated or duplicate studies were excluded. As a secondary data analysis, ethical considerations included proper citation and adherence to university guidelines. Data were collected using structured searches with Boolean operators (e.g., “Oxidative stress AND male infertility”), and articles were screened by title, abstract, and full text. Qualitative synthesis categorized findings into themes: ROS sources, oxidative damage mechanisms, effects on sperm function, and antioxidant defenses, comparing consistencies, contradictions, and research gaps without statistical software.

Main Body

Study Selection and Characteristics

A total of **50 peer-reviewed studies** published between **2020 and 2026** were included in this review following systematic screening. The selected literature comprised **systematic reviews, meta-analyses, clinical studies, and experimental investigations**, providing comprehensive evidence on the relationship between oxidative stress, antioxidant systems, and male infertility.

Across the included studies, there was a consistent and strong association between elevated oxidative stress and impaired sperm function, particularly in cases of idiopathic male infertility.

Table 5.1: Summary of Studies evaluating the Results

S. No	Author (Year)	Study Type	Key Focus	Major Findings
1	Krausz et al. (2022)	Review	Genetic factors	Genetic abnormalities contribute ~20% of male infertility
2	Agarwal et al. (2021)	Review	Male infertility causes	OS is a major contributor to sperm dysfunction
3	Takeshima et al. (2021)	Review	ROS prevalence	30–80% infertile men show elevated ROS
4	Dutta et al. (2022)	Review	Antioxidant therapy	Risk of reductive stress with overuse
5	Ayad et al. (2022)	Review	OS mechanisms	Lipid peroxidation & DNA damage are key
6	Mottola et al. (2024)	Review	Biomarkers	8-OHdG & isoprostanes indicate oxidative damage
7	Kumar & Singh (2022)	Review	Environmental factors	Pollution & toxins impair sperm quality
8	Imran et al. (2025)	Review	Multifactorial causes	Genetic + environmental interaction
9	Selvaraju et al. (2021)	Review	Environmental toxins	Pollutants induce oxidative stress
10	Zafar et al. (2023)	Meta-analysis	Nutritional therapy	Antioxidants improve semen parameters

Prevalence of Oxidative Stress in Male Infertility

The analyzed studies revealed that 30–80% of infertile men have elevated reactive oxygen species (ROS) levels, consistently linked to reduced sperm concentration, decreased motility (asthenozoospermia), abnormal morphology (teratozoospermia), and increased sperm DNA fragmentation, supporting oxidative stress as a key mechanism in male infertility. Endogenous ROS sources include activated leukocytes in semen, immature or abnormal spermatozoa, and mitochondrial electron transport chain leakage. Exogenous contributors—such as cigarette smoking, environmental pollution, and exposure to pesticides, heavy metals, and radiation—were repeatedly associated with deteriorated semen quality and heightened oxidative burden.⁽¹⁸⁾

Table 5.2: Summary of Sources, Mechanism and Impact on Sperm of Reactive Oxygen Species

Category	Source	Mechanism of ROS Production	Impact on Sperm
Endogenous	Leukocytes	Immune activation → ROS burst	DNA damage, reduced motility
Endogenous	Immature sperm	Residual cytoplasm enzymes	Increased oxidative stress
Endogenous	Mitochondria	Electron leakage	ATP reduction, motility loss
Exogenous	Smoking	Free radicals + toxins	DNA fragmentation
Exogenous	Pollution	Heavy metals, particulates	Reduced sperm quality
Exogenous	Radiation	Cellular damage	Increased ROS levels
Exogenous	Pesticides	Chemical toxicity	Hormonal & oxidative disruption

Mechanisms of Oxidative Stress-Induced Sperm Damage

Four principal mechanisms of ROS-mediated sperm damage were consistently reported:

Lipid Peroxidation

ROS-induced lipid peroxidation of sperm plasma membranes led to:

- Loss of membrane fluidity
- Disruption of ion exchange
- Impaired sperm motility and viability

DNA Damage

Oxidative stress caused:

- DNA strand breaks
- Base modifications (e.g., 8-OHdG formation)
- Increased DNA fragmentation

These alterations were strongly associated with **reduced fertilization rates and poor embryonic outcomes.**

Protein Oxidation

ROS-mediated oxidation of proteins resulted in:

- Enzymatic dysfunction
- Structural protein damage
- Impaired sperm function and signaling pathways

Mitochondrial Dysfunction

Mitochondrial damage due to ROS led to:

- Decreased ATP production
- Reduced sperm motility
- Activation of apoptotic pathways

Table 5.3: Summary of Mechanism, Target, Process and Outcome of Oxidative Stress-Induced Sperm Damage

Mechanism	Target	Process	Outcome
Lipid Peroxidation	Sperm membrane	ROS attack on PUFAs	Loss of fluidity, reduced motility
DNA Damage	Nuclear DNA	Strand breaks, base modification	Infertility, poor embryo development
Protein Oxidation	Enzymes & structural proteins	Oxidative modification	Enzyme dysfunction
Mitochondrial Dysfunction	Mitochondria	ETC disruption	↓ ATP, ↓ motility
Apoptosis	Whole cell	Caspase activation	Sperm cell death

- **Fertilization potential:** Compromised by DNA and membrane damage

Table 5.4: Summary of Effect of ROS on Sperm and their consequences on the functionality of sperms

Function	Effect of ROS	Consequence
Motility	Reduced ATP production	Poor sperm movement
Capacitation	Disrupted signaling	Impaired fertilization readiness
Acrosome Reaction	Membrane damage	Failure to penetrate oocyte
DNA Integrity	Fragmentation	Reduced fertility outcomes
Viability	Increased apoptosis	Decreased sperm survival

Oxidative stress impairs sperm functional competence by reducing motility through ATP depletion and structural damage, disrupting capacitation, and compromising the acrosome reaction. These effects collectively lead to diminished fertilization potential due to DNA and membrane damage.⁽¹⁹⁾

Antioxidant Defense Systems

Table 5.5: Summary of Type Effect, Function Effect on Sperm of antioxidants

Type	Antioxidant	Function	Effect on Sperm
Endogenous	SOD	Converts superoxide → H ₂ O ₂	Protects membrane
Endogenous	Catalase	Converts H ₂ O ₂ → water	Prevents hydroxyl radicals
Endogenous	Glutathione	Detoxifies ROS	Maintains DNA integrity
Exogenous	Vitamin C	Scavenges free radicals	Protects DNA
Exogenous	Vitamin E	Prevents lipid peroxidation	Improves motility
Exogenous	Zinc	Stabilizes membranes	Enhances sperm count
Exogenous	Selenium	Cofactor for enzymes	Improves morphology

7.1 CONCLUSION

The review concluded that oxidative stress is a major contributor to male infertility, particularly idiopathic cases, driven by excessive ROS production overwhelming antioxidant defenses. This leads to lipid peroxidation, DNA fragmentation, protein oxidation, and mitochondrial dysfunction, resulting in impaired motility, membrane rupture, defective fertilization, and increased apoptosis. Key sources include endogenous factors (leukocytes, mitochondrial activity) and exogenous factors

(smoking, environmental toxins, radiation), highlighting modifiable risks for prevention. Although endogenous and dietary antioxidants help maintain redox balance and sperm integrity, clinical outcomes from antioxidant therapy remain inconsistent due to variations in study design, dosage, and treatment duration. Overall, while oxidative stress is a central pathophysiological mechanism, the lack of standardized diagnostic tests and evidence-based protocols limits effective clinical implementation.

7.2 LIMITATIONS & RECOMMENDATIONS

In the light of the results of this review, the following recommendations may be offered:

- There exists the necessity to establish the standardized diagnostic procedures to measure the oxidative stress in clinical practice.
- Large, adequately designed, clinical trials must be undertaken to formulate evidence-based recommendations on antioxidant therapy such as the most appropriate dosage, duration and combinations.
- To alleviate oxidative stress, healthcare professionals should work on achieving and/or altering risk factors related to lifestyle and environmental influences, including smoking, exposure to pollution, and unhealthy diet.
- Regular assessment of the oxidative stress indicators and sperm DNA integrity must be a potential consideration in the presence of unexplained male infertility.
- There should be public awareness campaign which will enlighten people on the effects of environmental toxins and lifestyle habits on male reproductive health.
- Further studies can be conducted to identify the molecular and genetic processes of oxidative stress and the ways it interacts with the environment.

REFERENCES

1. Sang Q, Ray PF, Wang L. Understanding the genetics of human infertility. *Science*. 2023;380(6641):158-63.
2. Vander Borght M, Wyns C. Fertility and infertility: Definition and epidemiology. *Clinical Biochemistry*. 2018;62:2-10.
3. Carson SA, Kallen AN. Diagnosis and management of infertility: A review. *JAMA*. 2021;326(1):65-76.
4. Eisenberg M, Esteves S, Lamb D, Hotaling J, Giwercman A, Hwang K, et al. Male infertility. *Nature Reviews Disease Primers*. 2023 Sep 14;9(1):49.
5. Ammar T, Sidhu PS, Wilkins CJ. Male infertility: the role of imaging in diagnosis and management. *The British Journal of Radiology*. 2012 Nov 1;85(Special Issue 1):S59-68. doi:10.1259/bjr/31818161
6. Li L, Lin W, Wang Z, Huang R, Xia H, Li Z, et al. Hormone Regulation in Testicular Development and Function. *International Journal of Molecular Sciences*. 2024 May 26;25(11):5805. doi:10.3390/ijms25115805
7. Lei T, Yang Y, Yang W. Luteinizing hormone regulates testosterone production, leydig cell proliferation, differentiation, and circadian rhythm during spermatogenesis. *International Journal of Molecular Sciences*. 2025 Apr 10;26(8):3548.
8. Sizar O, Leslie S, Schwartz J. Male hypogonadism. StatPearls Publishing; 2024. (StatPearls).
9. Sharma A, Minhas S, Dhillon W, Jayasena C. Male infertility due to testicular disorders. *Journal of Clinical Endocrinology and Metabolism*. 2021 Jan 23;106(2):e442-59. doi:10.1210/clinem/dgaa781
10. Ciarloni A, delli Muti N, Sacco S, Ambo N, Di Giacomo V, Perrone M, et al. Genetic, Epigenetic, and Non-Genetic Factors in Testicular Dysgenesis Syndrome: A Narrative Review. *Genes*. 2025 Dec 31;17(1):40.

11. Shi S, Chen W, Tian J, Liang Z, Wu J, Li J, et al. Risk factors associated with varicocele: a narrative review. *Translational Andrology and Urology*. 2025 Jun 30;14(6):1807–17. doi:10.21037/tau-2025-120
12. Xu Y, Hu P, Chen W, Chen J, Liu C, Zhang H. Testicular fibrosis pathology, diagnosis, pathogenesis, and treatment: A perspective on related diseases. *Andrology*. 2025 Sep;13(6):1322–32.
13. Akhigbe R, Dutta S, Hamed M, Ajayi A, Sengupta P, Ahmad G. Viral Infections and Male Infertility: A Comprehensive Review of the Role of Oxidative Stress. *Frontiers in Reproductive Health*. 2022 Feb 3;4:782915. doi:10.3389/frph.2022.782915
14. Barone B, Amicuzi U, Tamaro S, Olivetta M, Stizzo M, Musone M, et al. Male Infertility: A Comprehensive Review of Urological Causes and Contemporary Management. *Journal of Clinical Medicine*. 2026 Jan 5;15(1):397.
15. Achermann A, Esteves S. Diagnosis and management of infertility due to ejaculatory duct obstruction: summary evidence. *International Brazilian Journal of Urology*. 2021 Jul;47(4):868–81. doi:10.1590/S1677-5538.IBJU.2020.0536
16. Konstantinidis C, Zachariou A, Evgeni E, Çayan S, Boeri L, Agarwal A. Recent advances in the diagnosis and management of retrograde ejaculation: a narrative review. *Diagnostics*. 2025 Mar 14;15(6):726.
17. Obeagu EI, Njar VE, Obeagu GU. Infertility: Prevalence and consequences. *International Journal of Current Research in Chemistry and Pharmaceutical Sciences*. 2023;10(7):43–50.
18. Takalani NB, Monageng EM, Mohlala K, Monsees TK, Henkel R, Opuwari CS. Role of oxidative stress in male infertility. *Reproduction and Fertility*. 2023;4(3).
19. Bhattacharya I, Sharma SS, Majumdar SS. Etiology of male infertility: An update. *Reproductive Sciences*. 2024;31(4):942–65.