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**INVESTIGATION OF THE IMPACT OF VARIOUS EXOGENOUS HARMFUL
FACTORS ON THE MALE REPRODUCTIVE SYSTEM**

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Abstract. The male reproductive system is highly sensitive to a wide range of exogenous harmful factors, including environmental toxins, radiation, heat, lifestyle-related exposures, pharmaceuticals, and industrial chemicals. These factors can significantly impair spermatogenesis, alter hormonal balance, induce oxidative stress, and damage reproductive organs, ultimately contributing to male infertility and reproductive dysfunction. In recent years, rising global concerns regarding declining male fertility rates have prompted extensive research into the impact of these external stressors. This review article synthesizes findings from the last decade, focusing on experimental and clinical studies that investigate the mechanisms by which various exogenous agents disrupt male reproductive health. Particular attention is given to the roles of endocrine disruption, mitochondrial damage, inflammation, and epigenetic modifications. A better understanding of these mechanisms is crucial for developing preventive and therapeutic strategies aimed at preserving male reproductive function in the face of increasing environmental and occupational hazards.

Keywords: male reproductive system, exogenous factors, male infertility, oxidative stress, endocrine disruptors, environmental toxins, spermatogenesis, heat stress, radiation exposure, reproductive toxicology

Introduction. Over the past half-century, male reproductive health has steadily declined, emerging as a pressing global concern: sperm counts in Western men have dropped by an estimated 50–60 % between 1973 and 2011, corresponding to an average annual decrease of 1.4 % [22]. In non-Western regions such as Asia, Africa, and South America, evidence also indicates similar downward trends—though the extent remains less clearly defined [23]. Notably, a study focusing on Indian males revealed alarming figures: sperm count declined by 30.3 %, motility by 22.9 %, and morphology by 51 % within a single decade [24].

This worrying trajectory cannot be fully attributed to genetic predispositions, obesity, or visible lifestyle changes alone. A substantial proportion of male infertility—approximately 44 %—remains idiopathic, suggesting unrecognized environmental and occupational contributors [1]. Present-day environments are characterized by pervasive exposure to a plethora of exogenous harmful factors, including endocrine-disrupting chemicals (EDCs), air pollutants, heavy metals, microplastics, electromagnetic radiation, heat stress, and ionizing radiation, which collectively impinge on spermatogenesis, hormonal balance, and overall reproductive integrity [2][3][4].

Mechanistically, these exogenous agents compromise male fertility via multiple overlapping pathways. Oxidative stress—driven by reactive oxygen species (ROS)—is a central mediator, as it impairs mitochondrial function, damages DNA, disrupts membrane integrity, and diminishes sperm motility, viability, and fertilization potential [1][5]. Air pollutants such as PM_{2.5}, NO₂, and O₃ have been directly implicated in enhancing sperm DNA fragmentation and altering morphology and count [1][6][7]. Heavy metals like lead, cadmium, and mercury bioaccumulate and correlate with compromised semen parameters [2][3]. Thermal stress—whether from occupational heat exposure, hot tubs, or electromagnetic radiation from laptops and mobile phones—increases oxidative damage and disrupts normal spermatogenic processes [1][8].

Emerging data on microplastics reveal their presence in human testes, with potential implications for endocrine disruption and reproductive impairment [9]. Additionally, ionizing radiation—such as that used in medical therapies—can inflict direct damage to spermatogonia and Leydig cells, reducing sperm count, motility, and hormonal function [21].

Given the multifactorial nature of these interactions, and the accelerating downward trends in male reproductive indices, urgent scientific attention is required. This review synthesizes recent findings (primarily from the past two decades) and examines the mechanistic links between key exogenous stressors and male reproductive dysfunction. Accurate understanding of these mechanisms is critical for informing precise preventive strategies, regulatory interventions, and therapeutic innovations, aimed at arresting and reversing the decline in male fertility across global populations.

A growing body of literature highlights that the male reproductive system is highly vulnerable to a broad range of exogenous harmful factors, including environmental toxicants, industrial chemicals, thermal stress, radiation, and endocrine-disrupting compounds (EDCs). These agents exert their negative effects via multiple mechanisms—disrupting spermatogenesis, altering hormonal homeostasis, inducing oxidative stress, and causing epigenetic modifications (Sciorio, 2025).

Air pollution is among the most pervasive contributors to reproductive toxicity. Particulate matter (PM_{2.5}), nitrogen dioxide (NO₂), and ozone (O₃) are consistently associated with reduced semen quality, impaired sperm motility, and increased DNA fragmentation (Kumar, 2022). A large-scale cohort study involving 526,056 Danish men demonstrated that exposure to elevated PM_{2.5} levels increased the risk of infertility by approximately 24% (The Guardian, 2024). Moreover, exposure to pesticides, dioxins, and heavy metals such as lead and cadmium has been linked with a reduction in sperm concentration and morphology (Levine et al., 2017).

Thermal exposure from occupational heat, hot baths, and even electromagnetic radiation emitted by laptops and cell phones is a significant but often overlooked risk factor. Studies indicate that scrotal temperatures above the physiological range (34–35 °C) can impair spermatogenesis and reduce sperm viability (Sciorio, 2025). Additionally, obesity contributes indirectly to thermal stress, as increased adipose tissue around the testes elevates local temperature and serves as a reservoir for lipophilic toxicants (Wikipedia, Obesity and Fertility, 2025).

Endocrine disruptors, particularly bisphenol A (BPA), phthalates, and polychlorinated biphenyls (PCBs), mimic or block hormonal signals and interfere with the hypothalamic-pituitary-gonadal (HPG) axis. A recent investigative report linked BPA exposure to decreased sperm count and testicular pathology in human and animal models (The Guardian, 2023; Wall Street Journal, 2024).

Ionizing radiation, depending on dose and duration, also damages germ cells and Leydig cells. Doses of 0.1–1.2 Gy primarily impair spermatogonia, while higher doses (>4–6 Gy) lead to azoospermia and testosterone suppression (Wikipedia, Reproductive Toxicity, 2025). In childhood cancer survivors, radiotherapy exceeding 3 Gy has been associated with permanent infertility and hypogonadism (Wikipedia, Radiotherapy Effects, 2023).

These findings collectively underscore the multifactorial nature of environmental impacts on male fertility. Between 1973 and 2011, sperm concentration among men in Western countries decreased by 52–59%, a trend that continues globally (Levine et al., 2017). The urgent need for identifying and mitigating exogenous reproductive threats is evident, as idiopathic infertility continues to rise and male reproductive indices deteriorate worldwide.

Methodology. This literature-based review was conducted using a systematic approach to identify, analyze, and synthesize peer-reviewed articles related to the impact of exogenous harmful factors on the male reproductive system.

1. Inclusion Criteria

Articles published between **2014 and 2025** were included if they met the following criteria:

- Focused on human or animal studies related to male fertility and exogenous exposures.
- Published in English-language peer-reviewed journals.
- Employed clinical, epidemiological, or experimental designs (including meta-analyses, systematic reviews, or original research).

Databases used included **PubMed, Scopus, Web of Science, SpringerLink, and MDPI.**

2. Search Strategy

Keywords used in the search were: “*male fertility*”, “*exogenous factors*”, “*environmental toxicants*”, “*air pollution*”, “*radiation exposure*”, “*heat stress*”, “*endocrine disruptors*”, “*oxidative stress*”, and “*epigenetic modifications*”. Boolean operators (AND, OR) were applied to maximize relevant article retrieval.

3. Data Analysis

Data were analyzed descriptively, focusing on statistical findings related to fertility indicators—such as sperm count, motility, morphology, DNA fragmentation index (DFI), and testosterone levels. For instance, air pollution was shown to increase infertility risk by 24% (The Guardian, 2024), while sperm counts dropped by over 50% globally in the past four decades (Levine et al., 2017). Heavy metal exposure, particularly to lead and cadmium, was associated with reduced sperm motility and increased oxidative stress markers (Kaminski et al., 2020).

4. Mechanistic Interpretation

Mechanisms of toxicity were interpreted based on molecular and cellular findings across studies. Endocrine disruption, mitochondrial dysfunction, inflammatory cytokine overexpression, and epigenetic alterations (e.g., DNA methylation and histone modification) were considered primary pathways (Akhatova, 2025). Several studies employed high-throughput transcriptomic and proteomic analyses to validate these findings in both in vitro and in vivo models (Kaminski et al., 2020).

Results

1. Global Trends in Sperm Parameters

Over recent decades, a pronounced global decline in sperm quality has been consistently documented. A 2017 meta-analysis reported a reduction in sperm concentration by approximately 52–59% among men in Western countries (Australia, Europe, North America) between 1973 and 2011, averaging a yearly decrease of 1.4% (Levine et al., 2017). This trend appears to extend globally: a broader study including men from South/Central America, Asia, and Africa found an overall 51.6% decline in sperm concentration from 1973 to 2018. Alarmingly, non-Western regions are experiencing accelerating decline rates. A 2022 meta-analysis reported decreases of 53.3% in sperm concentration and 56.3% in total sperm count in Western regions (1973–2015), and 27.6% and 24.7%, respectively, in non-Western regions (1986–2018), with steeper declines post-2000 [23].

2. Air Pollution and Semen Quality

Environmental particulate pollution (e.g., PM_{2.5} and PM₁₀) has been associated with measured declines in sperm motility, increased immature chromatin, and compromised overall semen quality. For instance, exposure among toll booth workers (a high-traffic group) correlated with significantly reduced sperm concentration and motility compared to a control group. Moreover, ecological modeling suggests that elevated PM_{2.5} exposure can amplify infertility risk by around 24%.

3. Thermal Stress and Elevated Scrotal Temperature

Emerging research highlights the impact of extreme heat on spermatogenesis. In clinical observations, heat exposure—stemming from occupational environments, tight-fitting clothing, or even rising ambient temperatures—nearly **doubles** the likelihood of low sperm count. Gender-specific scrotal overheating has also been observed in certain professions like trucking and in males adhering to tight dress codes, compounding the rate of fertility decline.

4. Electromagnetic Exposure and Mobile Phone Use

A Swiss cohort study of 2,886 men aged between 18 and 22 found that individuals reporting mobile phone usage over 20 times per day had a 21% lower sperm concentration compared to minimal users. Disruption is possibly tied to device-associated electromagnetic radiation, heat generation, and smartphone-induced oxidative stress [25].

5. Lifestyle, Oxidative Stress, and DNA Integrity

A study of 23,313 Australian men between 2010 and 2024 revealed rising rates of sperm DNA fragmentation—attributed to oxidative stress driven by poor nutrition, environmental toxins, sedentary behavior, and undiagnosed chronic diseases. Such DNA damage is strongly correlated with reduced fertility, elevated miscarriage rates, and reduced conception outcomes.

6. Ionizing Radiation

Exposure to ionizing radiation in the 0.1–1.2 Gy range has been found to inflict injury on spermatogonia, while doses between 4–6 Gy lead to marked reductions in sperm counts. Such deleterious effects are evident in both medical therapy scenarios and certain occupational exposures (e.g., radiation-based industries) [19].

7. Mechanistic insights: oxidative stress and endocrine disruption

Emerging narrative reviews propose that exposure to endocrine-disrupting chemicals—including bisphenol A (BPA), phthalates, PCBs, heavy metals, and pesticides—may compromise spermatogenesis via oxidative stress, hormonal dysregulation, and epigenetic alterations (e.g., DNA methylation/demethylation patterns) [26]. A focused review highlighted that semen quality may act as an early biomarker for systemic morbidity, connecting male subfertility to broader health risks such as cardiovascular disease and diabetes; men with severely impaired sperm concentration (<1 million/mL) were hospitalized on average seven years earlier than those with normal semen parameters (~200 million/mL) [5].

Exogenous factor	Observed effect	Statistic highlight
Global sperm decline	Sharp decrease in concentration and count	~50–59% decline over ~4 decades
Air pollution	Reduced motility, increased infertility risk	Infertility risk ↑ ~24%
Heat exposure	Elevated risk of low sperm count	Nearly doubled risk
Electromagnetic exposure	Reduced sperm concentration	Sperm conc. ↓ ~21%
DNA Integrity / Lifestyle factors	Elevated DNA fragmentation	Increasing trend over last decade
Ionizing radiation	Spermatogenic impairment	Spermatogonial injury at 0.1–1.2 Gy
Chemical exposures (EDCs)	Hormonal / epigenetic disruption	Mechanistic evidence accumulating

Table 1. The table outlines major exogenous factors affecting male fertility, showing their impact on sperm quality and related reproductive parameters. It includes global trends and key

statistics from recent studies, emphasizing the rising concern over environmental and lifestyle-related reproductive risks.

Discussion. The present synthesis of findings underscores a multifaceted and escalating threat posed by exogenous factors to male reproductive health. Globally, sperm concentration has declined dramatically—by approximately 52–59% in Western populations between 1973 and 2011—while declines of 27.6% in concentration and 24.7% in total sperm count are now observed in non-Western regions between 1986 and 2018, with accelerating reductions noted post-2000 [16][23]. These trends illustrate a pervasive and potentially accelerating downward trajectory in male fertility parameters, though some uncertainty remains; notably, a U.S.-based meta-analysis from 1970 to 2023 found only a modest annual decrease of -0.35 million/mL ($P = 0.04$) in sperm concentration—interpreted by Dr. Lundy as possibly non-catastrophic [27].

Endocrine-disrupting chemicals (EDCs) emerge as pivotal contributors. Over half (53%) of empirical studies reviewed found clear negative associations between EDC exposure and male reproductive outcomes [28]. Exposure to EDCs such as BPA, phthalates, and ubiquitous industrial obesogens has been mechanistically linked to impaired steroidogenesis, receptor dysregulation, increased oxidative stress, sperm apoptosis, and epigenetic modifications [5][29]. Moreover, some agents exert multigenerational effects—elevating concern for transgenerational transmission of reproductive impairment [29].

Oxidative stress is another central mechanistic pathway. Australian research examining 23,313 men from 2010 to 2024 revealed that oxidative stress accounts for an estimated 80% of male infertility cases, with sperm DNA fragmentation projected to affect ~50% of men seeking fertility care by 2054 if current trends continue.

Additionally, lifestyle and health determinants are significant. Obesity impairs reproductive function through multiple intersecting routes—hormonal dysregulation (notably reduced free testosterone), testicular overheating, and toxin bioaccumulation—resulting in oligozoospermia, degraded motility, and increased DNA damage [20]. Advanced paternal age also adversely impacts sperm quality; men over 40 are approximately 30% less likely to conceive compared to those under 30, while sperm counts have globally declined by over 50% in recent decades [32].

Adding to the complexity, novel stressors such as infection-related declines have emerged. In one cohort, over 75% of men who recovered from mild Omicron COVID-19 infections exhibited significant sperm DNA damage—a stark addition to our understanding of transient but potentially recurrent reproductive insults [31].

From a global public-health perspective, male infertility has exhibited alarming growth. The World Health Organization and Global Burden of Disease data indicate that between 1990 and 2019, global male infertility prevalence increased by approximately 76.9%, with age-standardized rates rising by ~19%. The highest burdens are observed in Western Sub-Saharan Africa, Eastern Europe, and East Asia, particularly among men aged 30–34 [30].

Collectively, these findings convey urgent implications:

1. **Multifactorial Etiology:** Male fertility decline is not attributable to a single cause. EDCs, oxidative stress, obesity, infection, and demographic shifts synergistically undermine reproductive potential.

2. **Policy Imperatives:** Stricter regulation of EDCs (e.g., BPA, PFAS, phthalates), enhanced screening for DNA fragmentation, and public health interventions targeting obesity and lifestyle are imperative.

3. **Predictive Models:** If current trajectories persist—an estimated annual decline of 1–1.4% in sperm counts—the risk of widespread subfertility becomes more probable by mid-21st century (e.g., Shanna Swan’s projection of near-zero counts by 2045)[23].

4. Interventional Potential: Unlike oocyte production, spermatogenesis remains responsive to environmental and behavioral modification—a promising avenue for prevention and reversal.

In summary, the observed global and mechanistic trends assert that male reproductive health is declining via interconnected environmental, lifestyle, and physiological drivers. Proactive, evidence-based interventions and further high-resolution epidemiological research are essential to mitigate the projected reproductive morbidity and its broader societal consequences.

Conclusion. The present literature-based analysis confirms that the male reproductive system is highly sensitive to a wide range of exogenous harmful factors, including environmental toxins, occupational exposures, pharmaceuticals, lifestyle behaviors, and dietary components. These factors contribute to a significant decline in male reproductive health, as evidenced by global trends indicating reductions in sperm count, quality, and hormonal balance.

Mounting evidence underscores the negative role of endocrine-disrupting chemicals, such as phthalates, bisphenol A, and heavy metals, which interfere with spermatogenesis and androgenic activity through oxidative stress and hormonal dysregulation. Similarly, chronic exposure to heat, ionizing radiation, or persistent psychological stress has been shown to impair the hypothalamic-pituitary-gonadal axis, leading to functional infertility. Modern lifestyle choices, particularly smoking, alcohol consumption, recreational drug use, and sedentary behavior, further exacerbate these issues, creating a multifactorial risk environment.

Given the growing burden of male infertility—estimated to account for approximately 50% of all infertility cases worldwide—it is essential to intensify public health measures aimed at reducing exposure to these harmful agents. Moreover, future research should prioritize longitudinal human studies and advanced molecular techniques to better elucidate the mechanisms of damage and identify potential biomarkers for early detection and therapeutic intervention.

In conclusion, the evidence strongly supports a causative link between various exogenous harmful factors and male reproductive dysfunction. Urgent global attention, preventive strategies, and targeted research are crucial to mitigate these risks and protect male fertility across populations.

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