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OCCUPATIONAL HAZARDS TO MALE REPRODUCTION: A SYSTEMATIC REVIEW OF ENVIRONMENTAL IMPACTS

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ABSTRACT

Male infertility has emerged as a major global public health challenge, accounting for nearly 50% of all infertility cases worldwide. While etiology is often multifactorial, occupational exposure to physical, chemical, and environmental hazards represents a critical, yet frequently under-recognized, contributor to male reproductive dysfunction. This review-based analysis synthesizes published evidence to evaluate the precise impact of diverse workplace hazards, their underlying biological mechanisms, associated risk factors, and available preventive strategies. Occupational hazards—such as heavy metals, pesticides, industrial solvents, ionizing and non-ionizing radiation, excessive heat, and endocrine-disrupting chemicals (EDCs)—profoundly impair reproductive anatomy and physiology. Mechanistically, these toxic agents disrupt spermatogenesis, alter hormonal homeostasis, and destroy sperm DNA integrity. Chronic exposure results in reduced sperm count, poor motility, abnormal morphology, and elevated oxidative stress, which severely compromise overall fertility potential. Certain populations, including agricultural workers, welders, painters, long-distance drivers, laboratory personnel, and factory workers, are particularly vulnerable due to prolonged contact with hazardous environments. Furthermore, workplace-related lifestyle factors like chronic psychological stress, irregular sleep patterns, and substance use exacerbate these reproductive risks. Despite expanding epidemiological and clinical evidence linking occupational environments to fertility decline, awareness remains limited among both workers and healthcare professionals. Routine reproductive risk assessment is rarely incorporated into standard occupational health screenings. This study highlights the urgent need for targeted preventive approaches, stricter workplace regulations, enhanced clinical awareness, and robust education to mitigate occupational reproductive hazards and safeguard male fertility.

KEYWORDS: Male infertility, occupational exposure, endocrine disrupting recepting chemicals, oxidative stress.

1. INTRODUCTION

Numerous physical and chemical risk factors are produced due to occupational exposure which affects the health of employee and leads to several disease and physiological disorders including cancers and neurological and systemic abnormalities [1]. Humans are exposed to thousands of external (exogenous) chemicals through industrial activities, development processes, and the food chain. Over recent decades, concerns about reproductive disorders and threats to reproductive health have grown, as certain chemicals have been shown to impair reproductive function. The male reproductive system, in particular, is especially susceptible to harm from both chemical and physical stressors[2]. Over the past twenty years, male infertility has seen a notable increase, likely linked to environmental exposures. Reports from fertility clinics suggest that chronic illnesses and genetic conditions only partly account for the current rates of male infertility. Instead, both environmental and workplace environments expose men to complex mixtures of endocrine-disrupting chemicals, which are now believed to

play a key role in fertility issues. Infertility is typically defined as the inability to conceive after one year of unprotected sex; it affects about 10–15% of couples, with the proportion differing by country and region. Both men and women contribute equally to infertility, and in many cases, more than one factor is responsible [3]. Given how common male-factor infertility is, there is growing concern about the influence of diet, lifestyle, and environmental exposures on men's reproductive capacity. Numerous retrospective studies have reported a decline in semen quality across various countries over the past few decades, though the precise causes remain unclear. Public-health research has consequently focused on the long-term impacts of chronic, low-dose exposures such as those from food, drugs, and environmental pollutants because their cumulative effects appear to significantly affect both individual and population-level health. These exposures can impair spermatogenesis and male fertility by disrupting hormonal balance, inducing oxidative stress, or damaging germ cells. Indeed, male reproductive health is increasingly being recognized as a sensitive indicator of environmental pollution and chemical exposure [4].

At the National Institute for Occupational Safety and Health (NIOSH), there is a registry that includes roughly 104,000 chemical and physical agents found in workplaces, yet the toxicological profiles of most of these agents remain unknown or only partially examined. In humans, exposure to some of these substances can lead to cancer, reproductive and developmental issues, neurological and immune-system damage, and other health effects. Such effects on fertility and development are especially concerning for couples planning to conceive, because exposure during critical stages of fetal development might not only impact the child's health but could even carry consequences into adulthood or across generations. There are multiple reasons for the lack of data on occupational exposures: animal studies typically focus on only one chemical at a time, which makes it difficult to understand how different toxins interact with each other. Moreover, these animal studies often don't examine long-term or subtle effects, such as developmental delays. Human epidemiological studies, meanwhile, often struggle with limited or imprecise exposure data and health outcome measurements. On top of that, barriers like insufficient government funding and restricted access to exposure data—especially in developing countries—further limit research in this domain [5]. Fig 1 illustrates the major occupational exposures associated with male infertility and their adverse effects on the male reproductive system.

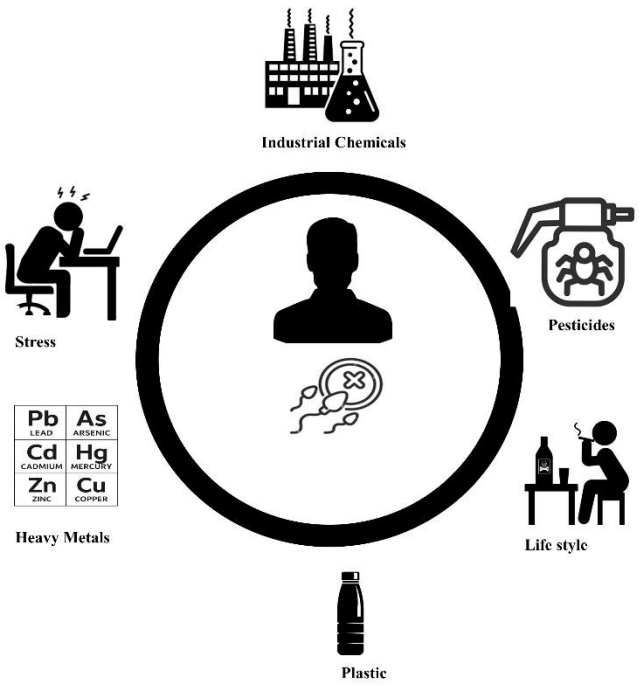

Fig.1: Occupational exposures and male Infertility

The human spermatogenic cycle lasts about 72–74 days, during which various stages of germ-cell development can be disrupted by toxic compounds (Endocrine disrupting chemicals and male fertility: from physiological to molecular effects Exposure to toxicants at any of these stages can reduce sperm numbers, increase the proportion of morphologically abnormal sperm, destabilize sperm chromatin, or damage sperm DNA. When toxins

accumulate in the epididymis, prostate, seminal vesicles, or seminal fluid, they can compromise the sperm's normal progressive motility [6].

The neuroendocrine regulation of spermatogenesis is also highly vulnerable to disruption. Toxic agents can interfere with androgen (testosterone) secretion from Leydig cells or inhibit Inhibin B production from Sertoli cells in the testes. They may also act on the central control systems—such as the hypothalamus (gonadotropin-releasing hormone) or the pituitary (gonadotropins)—disrupting hormonal signaling pathways .Toxins that target **stem cells** in the testes are particularly concerning, because they can lead to delayed, long-lasting, and potentially irreversible effects. Conversely, toxicants acting on later stages of development (e.g., more differentiated germ cells) tend to produce more transient damage [7]. NTP Technical Report on the Reproductive Dose Range-Finding Toxicity Study of Genistein). Experimental studies have indeed mapped specific sites of action for different chemicals, showing how certain endocrine-disrupting compounds selectively damage Leydig cells, Sertoli cells, or the germ cells themselves (Endocrine-disrupting chemicals and male reproductive health Aditi Sharma).

2 Occcupational exposure

Sometimes, individuals are unknowingly exposed to reproductive risk factors at work—such as toxic chemicals, radiation, or high heat—in much higher levels than from everyday environmental exposure. Yet, many only become aware of these hazards when they try to have a child. Research shows that over the past two decades, infertility in industrialized nations has risen from about 8% to 15%, and occupational exposures may be a contributing factor.

Workplaces offer a unique opportunity to study how reproductive toxicants impact fertility, because exposure measurement and risk assessment can be more controlled than in the general environment. Despite challenges like getting workers and employers to participate, well-designed studies at worksites can yield valid data. Moreover, findings from occupational settings can often be extrapolated to understand risks in the general population exposed to environmental pollutants [2].

2.1 Impact of heavy metals on male reproductive health

Heavy metal poisoning can arise from industrial wastewater, medicinal products, and polluted air or water. Smoking is also a significant source of metal toxins, such as cadmium (Cd^{2+}) and lead (Pb^{2+}), because tobacco plants tend to absorb these metals from soil and transport them from roots to their leaves. Improper recycling or dismantling of electronic waste (e-waste) can release toxic metals like cadmium, chromium, and copper into the environment.[8][9]

The severity of heavy metal toxicity depends on many factors, including; the type of metal and its oxidation state, the dose and duration of exposure, toxicokinetics and toxicodynamics,individual characteristics such as age, nutritional status, sex, and overall health [10] .

Heavy metals can harm the male reproductive system in several ways — for example, by disturbing the hypothalamic–pituitary hormonal axis or by directly impairing spermatogenesis, which can lead to reduced semen quality. Non-essential metals like lead (Pb) and cadmium (Cd) are widespread in the environment (water, air, soil), making exposure quite common [9][11][12]. When lead and cadmium accumulate in the body, they can be stored in tissues like bone, kidney, and gonads. Over long term, even at low levels, these metals may disrupt the oxidative balance in the testes, producing reactive oxygen species that damage sperm [13][12][14].

Several human and animal studies support this: for instance, one pilot human study found that seminal plasma levels of lead and cadmium were significantly associated with a higher percentage of immotile sperm [15]. Other research has also documented associations between occupational exposure to heavy metals and reduced sperm morphology, elevated DNA fragmentation, and signs of oxidative stress [16] [17][18]. Mechanistically, cadmium in particular can disrupt the blood–testis barrier, induce apoptosis (programmed cell death) in testicular cells, and impair sperm metabolism by interfering with key enzymes [13]. Finally, studies measuring metal levels in various biological fluids (blood, semen, urine) support these biological effects. For example, environmental exposure to multiple metals including Pb and Cd has been linked to lower sperm count, decreased motility, and poor

morphology [11].

Several studies have concentrated on how oxidative stress, apoptosis, and DNA fragmentation are affected. Over the years, elevated levels of metals such as lead (Pb), cadmium (Cd), copper (Cu), and zinc (Zn) have been associated with deterioration in semen quality[19]. As shown in Fig 2, oxidative stress plays a major role in male infertility by damaging sperm structure and function, reducing motility, and impairing DNA integrity.

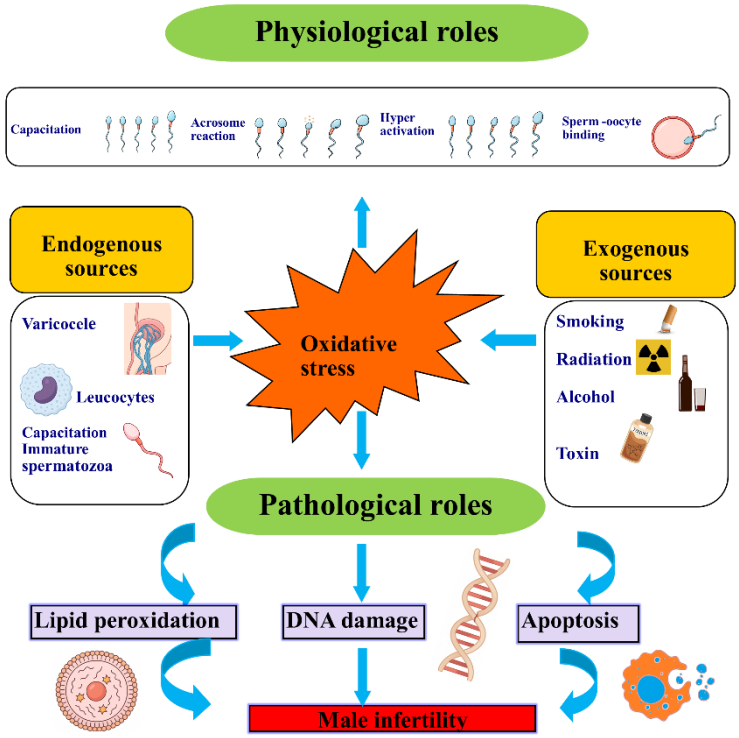

Fig. 2: Impact of oxidative stress on male infertility

As presented in Tab 1, exposure to heavy metals has been associated with significant reproductive toxicity across different model organisms, affecting spermatogenesis, hormonal balance, gamete quality, and overall fertility.

Tab 1: Summary of heavy metals-induced reproductive toxicity on different model organisms

Heavy metal	Model organism	Adverse effects on male and female reproductive system	M/A	
Cd2+	Human	Teratozoospermia and asthenozoospermia. The sperm showed loss of head-actin. In testicular tissue, evidence of apoptosis was observed.	1.ROS and Oxidative stress. 2. Mimicking ions or essential molecules; substitution / interference with physiological ions / cofactors 3. Binding to thiol (-SH) groups and other reactive groups on proteins / molecules 4. Apoptosis 5.Involvement of Metallothioneins (MTs) / metal-binding proteins / redox buffers 6. Alteration of signal transduction / intracellular signaling	[10]
	Rodents (Wistar rat, mice)	Inhibition of StAR expression. Destabilization of sperm chromatin / increased DNA damage Alkalization of luminal fluid in seminiferous tubules / epididymal duct Necrosis of efferent ducts		

		Damage to Sertoli cells. Shrinkage / impairment of Leydig cells. Disruption of blood vessels, the testis barrier, and seminiferous tubules Disassembly of gap junctions, tight junctions, and desmosomes Decreased (reduced) rate of spermatogenesis	pathways 7. Alteration of signal transduction	
Pb2+	Human (occupational workers)	• Reduction in testicular weight. • Decline in semen volume, sperm count, and sperm motility. • Impaired sperm chromatin decondensation. • Increased sperm DNA damage. • Rise in the proportion of abnormal sperm. • Lower reproductive performance and decreased fertility rate.	• Generation of reactive oxygen species (ROS) and induction of oxidative stress. • Binding to thiol (–SH) groups on proteins / molecules. • Substitution or displacement of essential divalent ions. • Suppression of estrogen (eg, estradiol) activity.	[10]
	Rat	• Reduction in androgen levels and decrease in reproductive-organ weights. • Impaired germ-cell differentiation. • Decline in the number of Leydig cells. • Suppression of key steroidogenic enzymes (P450scc, P45017α, 17β-HSD).		
Hg°/Hg2+/ CH3Hg+	Rat and human male	• Reduced testicular growth and spermatogenesis, with increased testicular necrosis. • Increased number of immature sperm. • Lower sperm concentration in semen, reduced sperm motility, decreased ejaculate volume, and increased proportion of abnormal sperm. • Decline in testosterone levels.	• Excessive generation of reactive oxygen species (ROS), causing oxidative stress. • Binding/interaction with thiol (–SH) groups in cellular proteins and antioxidants, disrupting normal protein/antioxidant functio	[10]
	Monkey	• Reduced sperm count.		

		• Lower testosterone levels. • Damage to testicular structure and integrity.		
	Zebra fish	• Reduced transcription of genes including GnRH, FSHβ, LHβ, LHR, AR, CYP17, and CYP11B. • Development of hyperplasia in the seminiferous epithelium. • Delay in spermatogenesis and spermiogenesis. • Slower formation and maturation of sperm cells		
	Channa punctatus Murrel	• Altered sexual behavior via inhibition of Isotocin and Vasotocin systems. • Pyknosis (condensation/shrinkage) of interstitial cells. • Fibrosis of the interstitial tissue. • Suppression of spermatogenesis. • Reduced sperm motility and fertilization potential.		
As3+/As5+	Hamster	• Reduction in epididymal sperm count. • Overall low sperm count.	• ROS generation → oxidative stress • Binding to thiol (–SH) groups of proteins / enzymes Displacement/substitution of essential bivalent ions (cofactors) • Inhibition of key enzymes (metabolic / antioxidant / steroidogenic) • Suppression or impairment of androgen-receptor signalling/function	[10]
	Mice	• Reduction in sex-organ growth and overall testicular development. • Oligozoospermia (reduced sperm count). • Asthenozoospermia (reduced sperm motility). • Inhibition of steroidogenic enzymes (3β-HSD and 17β-HSD). • Chromatin abnormalities in sperm (chromatin margination). • Formation of abnormal large/“giant” spermatid cells. • Disruption of meiosis and post-meiotic stages of spermatogenesis, leading to impaired sperm maturation.		

	Rabbit	• formation of vacuoles in the sperm acrosome. • Asthenozoospermia (reduced sperm motility). • Teratozoospermia • Decreased fertility rate / reduced fertility potential.	
Cu2+	Human	• Poor semen quality. • Reduced sperm count. • Decreased sperm motility and lower proportion of normally shaped (norm-morph) sperm	Oxidative stress and apoptosis
	Wistar rat	• Reduction in sex-organ weight. • Decreased activity of the enzymes Δ5-3β-HSD and 17β-HSD.	
Zn2+	Mice	• Poor semen quality.	
	Capra hircus	• Increased apoptosis of male germ cells.	

Cadmium (Cd), which has the atomic number 48 and a density of 8.7 g/cm³, is a naturally occurring heavy metal in the Earth's crust. Industrial activities such as mining, smelting, electroplating, as well as the manufacture of pigments, plastic stabilizers, Ni–Cd batteries, and lead-based paints are major contributors to the release of Cd^{2+} into the air, soil, and water. Human exposure to cadmium most commonly happens through smoking, via inhalation of tobacco smoke because tobacco plants readily absorb cadmium from soil. It can also come from dietary sources like contaminated foods like shellfish, rice, and certain vegetables are known to accumulate Cd [21][20][22]. Vegetables such as spinach, lettuce, potatoes, as well as peanuts, soybeans, and sunflower seeds, can contain cadmium at concentrations of about 0.05–0.12 mg Cd^{2+}/kg [21][23]**.** According to the ATSDR, cadmium is the eighth most hazardous chemical in terms of public health concern. According to WHO data, the average daily consumption of cadmium in a balanced diet ranges from 0.35 to 9.63 µg/kg body weight. The main sources of exposure include contaminated food, water, and tobacco smoke. (24)

Lead (Pb), with atomic number 82 and a density of 11.34 g/cm³, is a nonessential and highly toxic heavy metal that acts as an endocrine disruptor [24] [25]. Pb^{2+} is known to impair male reproductive health and has even been historically used as a spermicidal agent [25]. Its release into the environment occurs through various sources: industrial emissions, mining, galvanizing, mineral processing, automotive exhaust, and other human activities [25].

Even at low concentrations, lead can cause serious reproductive toxicity. For instance, blood lead levels (BLL) as low as ≥ 15 µg/dL have been linked to adverse effects on sperm count, motility, and morphology in men. The degree of accumulation in the body depends on factors such as exposure duration, frequency, lifestyle, and age. People living near lead-emitting industries, or those who smoke or abuse alcohol, typically show higher serum lead levels increasing their risk for lead-induced reproductive damage [26]. According to the ATSDR Substance Priority List, lead (Pb) is identified as a high-priority substance because of its widespread presence and toxicity [27].The WHO Guideline for Drinking-Water Quality sets a provisional limit for lead in drinking water at 0.01 mg/L (10 µg/L)[28]. This guideline is described as "provisional," primarily because of practical challenges in consistently achieving lower concentrations, not because lead is safe at that level [29]. Major routes of exposure

to lead include ingestion (e.g., from contaminated water or food), inhalation (e.g., lead dust or fumes), and, to a lesser extent, skin contact [30][29].

Mercury (Hg), with atomic number 80 and a density of 13.6 g/cm³**,** poses a major environmental threat. A significant portion of mercury pollution arises from the release of mercuric chloride ($HgCl_2$)**,** which contaminates water bodies even far from its industrial origins[31][32]. Mercury exists in three primary forms, each with distinct exposure pathways: Elemental mercury (Hg^0) — a liquid at room temperature that readily vaporizes; a common source of exposure is dental amalgam used in tooth fillings[33], inorganic mercury (Hg^{2+}) — such as mercuric chloride; exposure routes include antiseptic medicines, as well as contaminated air, water, and food[34][35], Organic mercury (e.g., methylmercury, CH_3Hg^+) — primarily found in fish and shellfish, as well as in some industrial uses (e.g., wood preservatives)[36]. Mercury is extremely toxic. It induces oxidative stress, increasing free radical production and causing structural damage to cells[35][34]. Among mercury compounds, $HgCl_2$ (inorganic mercury) is particularly potent in disrupting protein structure[34]. Major routes of human exposure include: Inhalation of elemental mercury vapor[32], ingestion of mercury via contaminated food, especially fish containing methylmercury[37], Dermal contact or mucous membrane exposure from inorganic mercury compounds. Mercury is particularly toxic for reproduction: it causes oxidative stress by boosting free radical production, which damages cellular structures. Among mercury compounds, $HgCl_2$ is especially potent in disrupting protein structure. Regarding toxicity, on the ATSDR substance priority list, mercury ranks 3rd in concern[37]. According to the WHO Guidelines for Drinking-Water Quality, the guideline value for inorganic mercury in water is 0.006 mg/L (6 µg/L)[28]. The tolerable daily intake (TDI) for inorganic mercury is 2 µg/kg body weight/day, as per WHO[28]. For methylmercury (CH_3Hg^+)**,** the provisional tolerable weekly intake (PTWI) set by JECFA (WHO/FAO) is 1.6 µg/kg body weight/week[38]**.** Main exposure routes include: inhalation (especially mercury vapor), skin absorption, eye contact, and ingestion of contaminated food.

Arsenic in its trivalent (As^{3+}) and pentavalent (As^{5+}) forms produces a wide spectrum of toxic effects, including hypertension, cancer, stroke, chronic lung disease, neurotoxicity, liver inflammation, genotoxicity, and reproductive dysfunction. Inorganic arsenic's health impact is a major concern due to its extensive industrial use, leaching into groundwater, and presence in drinking water. The main sources of exposure to As^{3+}/As^{5+} are mining, smelting, and manufacture of arsenic-containing products.According to the ATSDR, arsenic is a top priority chemical for public health risk. According to WHO guidelines, the provisional limit for arsenic in drinking water is 10 µg/L (i.e., 0.01 mg/L). [39][28][40] The primary routes of arsenic exposure are ingestion (via contaminated water and food) and inhalation of arsenic-laden dust or air[41].

Zinc (Zn), though essential, can be harmful when present in excess: chronic exposure can cause reproductive toxicity. Zn^{2+} plays a critical role in spermatogenesis, sperm motility, antioxidant protection, and the function of Zn-dependent enzymes, including those involved in protein phosphorylation. However, when Zn^{2+} levels become dysregulated, it can disrupt mitochondrial membrane potential, which in turn activates caspase-3 and triggers apoptosis in male germ cells. In addition, Zn^{2+} complexes have been shown to induce DNA fragmentation and cause degenerative changes in testicular tissue[42][43][44].

Copper (Cu, atomic number 29; density ~ 8.92 g/cm³**)** is the third most abundant essential trace metal in the human body, and it is vital for many biochemical processes. Cu^{2+} acts as a cofactor for enzymes involved in redox reactions, mitochondrial respiration (especially cytochrome c oxidase), iron metabolism, and antioxidant defenses (e.g., superoxide dismutase, (SOD)[45][46]. However, chronic copper overload is harmful, particularly for male reproductive health. Excess Cu^{2+} can accumulate in testicular tissue and seminal plasma, where it contributes to oxidative stress**,** activates apoptosis (programmed cell death), and damages sperm[47][48]. For example, in mice, long-term exposure to copper sulfate led to reduced sperm count and motility, increased expression of apoptotic proteins (e.g., caspase-3, Bax), and testicular degeneration — all without significantly affecting testosterone synthesis[48]**.** At the molecular level, copper homeostasis in the testis is tightly regulated by transporters and chaperone proteins; if this balance is disturbed, excess Cu can generate reactive oxygen species (via Fenton-like

reactions), impair DNA repair mechanisms, and cause structural damage in germ cells [45]. Chronic reproductive toxicity has also been documented by the Agency for Toxic Substances and Disease Registry (ATSDR)**:** high copper exposure in animals is linked to reduced testis weight, abnormal sperm morphology, and degeneration of testicular tissue[49]. Furthermore, epidemiological studies in humans show that elevated copper levels in seminal plasma are associated with increased oxidative stress and changes in immune signaling factors [50].In rats, although copper (Cu^{2+}) is essential at moderate levels, excessive amounts can be harmful. Animal studies suggest that very high dietary copper (~400–800 mg/kg) leads to reproductive toxicity. Excess Cu^{2+} can disrupt spermatogonial cell division and differentiation, impairing the early stages of spermatogenesis [51][52][47]. Overload of copper generates oxidative stress in the testis, reducing antioxidant defenses and increasing lipid peroxidation, which contributes to testicular tissue and spermatozoa damage. It also induces apoptosis in male germ cells (via caspase-3 activation) and causes DNA damage in testicular tissue [47].Therefore, while Cu^{2+} is critical for normal reproductive function, at high levels it exerts a dual role, protecting at low doses but causing toxicity and infertility when in excess.

2.2 Pesticide exposure and reproductive toxicity

About half of the of the world's workers is laboring in agriculture. Over the last half-century, agriculture has transformed dramatically, with widespread use of pesticides and fertilizers to boost crop protection, yields, food quality, and preservation. Pesticides are distinctive because they are inherently toxic to multiple biological targets, are intentionally released into the environment, and often have limited selectivity between species. Their toxic effects cause both immediate poisonings and long-term health problems, such as cancer and reproductive harm. Workers involved in the agricultural pesticide supply chain — distributors, mixers and loaders, applicators, bystanders, and field workers who return soon after spraying — face occupational exposure. Evaluating and controlling the health risks that pesticides pose to these workers is a complex but vital responsibility for occupational toxicologists. Experience from many countries shows that preventing pesticide-related health problems is both technically possible and economically beneficial for workers. Effective risk management is essential for the safe use of pesticides. Recent epidemiological studies have linked exposure to various pesticides with reductions in sperm concentration, motility, and morphology, and simultaneous exposure to multiple pesticides may further complicate risks[53]

Pesticides — both older organochlorines like DDT, hexachlorobenzene (HCB) and chlordane, and newer chemicals such as organophosphates, pyrethroids, and triazines — can negatively affect human fertility. At high exposures, animal and experimental studies show pesticides can disturb endocrine systems by altering gonadotropin, thyroid, and sex-steroid signaling [8,9], and can trigger oxidative stress [10,11], which can impair sexual development, reproductive capacity, and pregnancy maintenance. Nonetheless, evidence from humans is less clear when it comes to the low-level, chronic exposures common in the general population, and remains a matter of debate. [54].

Almost all pesticides can act as endocrine-disrupting chemicals (EDCs**)**, and in men, exposure to these EDCs islinked to a variety of reproductive harms. Studies show associations between pesticide/EDC exposure including cryptorchidism and hypospadias [55][56], reduced semen quality [57][58] and higher risk of testicular cancer[57][59].

Mechanistically, pesticides and other EDCs may mimic or block hormones (anti-androgenic or estrogen-like effects)[59], interfere with the hypothalamic–pituitary–gonadal (HPG) axis, disrupting testosterone production and other hormonal signals[60][61]., cause oxidative stress, mitochondrial dysfunction, DNA damage, and even epigenetic changes in reproductive cells[62]. Given these risks, especially when men are exposed to mixtures of different pesticides -managing and minimizing exposure is very important.

A number of epidemiological studies support the link between pesticide exposure and male reproductive dysfunction. In a well-known case–control study, men with poor semen quality were found to have significantly higher urinary levels of alachlor**,** atrazine**,** and diazinon metabolites. [63]. A cross-sectional study of Dutch fruit

growers and their spouses showed that during the spraying season, their time-to-pregnancy (TTP) was significantly longer, suggesting reduced fecundability [64][65]. In Danish men working in greenhouses**,** sperm density and motility were lower in those working more hours in sprayed greenhouses[66]. In Mexican floriculturists (flower growers), long-term exposure to organophosphate (OP) pesticides was associated with hormonal disturbances: a longitudinal study found increased FSH and prolactin and decreased testosterone and inhibin B levels[67]. Another cross–sectional study on the same population reported that urinary OP pesticide metabolites (dialkyl phosphates) were negatively correlated with inhibin B and FSH, suggesting a disruption of the hypothalamic-pituitary-gonadal (HPG) axis[68]. These findings strongly support the hypothesis that occupational exposure to certain pesticides — especially EDCs like OPs — can impair male reproductive health by altering hormone regulation and reducing semen quality. As shown in Tab 2, pesticide exposure adversely affects the male reproductive system by impairing spermatogenesis, altering endocrine function, reducing sperm motility, and increasing oxidative stress

Table 2: Effect of pesticides on male reproductive system

Pesticide	Effect	References
Chlorpyrifos, carbamates	• Reduced sperm motility, immature sperm, oligozoospermia, genome damage	[68]
Organophosphorus (animal study)	• Reduced sperm density and motility, reduced testicular and epididymal sperm density Reduced level of testosterone (malathion), lessened testis weigh, amplified abnormal sperm morphology, declined plasma FSH and LH, Seminiferous tubule degeneration,declined number of implantations and live fetuses	[69]
Pyrethroids	• Chronic exposure may disrupt endocrine function by interfering with human androgen receptors and with Sex Hormone-Binding Globulin (SHBG), altering androgen-mediated hormonal activity	[69]
Organochlorines (Methoxychlor, 2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCDD), Hexachlorocyclohexane, Lindane, DDT and DDE)	• Decrease in the weights of the testes, epididymis, seminal vesicles, and ventral prostate; increased generation of hydrogen peroxide and enhanced lipid peroxidation in mitochondrial- and microsome-rich fractions of the testis; number of abnormal sperm increased; testis weight was reduced; In the epididymis there were increased inflammatory-cell foci; daily sperm production declined; mounting and intromission latencies increased; In adulthood, serum testosterone concentrations decreased	[69]
	• Altered activity of enzymes including sorbitol dehydrogenase, glucose-6-phosphate dehydrogenase, γ-glutamyl transpeptidase and β-glucuronidase.; treated rats showed accumulation of	

	Hexachlorocyclohexane (HCH) and its isomers in the testes and in sperm; serum testosterone levels declined; epididymal sperm count and sperm motility dropped; number of abnormal sperm increased.	
	• Increases in Fas, FasL and caspase-3 levels; cytoplasmic level of NF-κB p65 decreased; and NF-κB showed altered localization: there was maximal nuclear translocation in germ cells	
	• Reduction in serum testosterone; decreased testicular weight; lower epididymal sperm count and motile-sperm percentage; reduced seminal-vesicle weight. • Serum levels of Luteinizing hormone (LH) and Follicle-Stimulating Hormone (FSH) increased; response of Sertoli cells to FSH was inhibited; the survival rate of Sertoli cells was decreased	
	• Decline was observed in the fertility index, along with reduced weights of the testes and male accessory sexual glands. • Serum testosterone levels dropped, and both sperm count and sperm motility were diminished. • Proportion of abnormal sperm increased. Histological examination revealed lesions in the seminiferous tubules and signs of incomplete or arrested spermatogenesis	[69]
Carbamates (Methymol,Propoxur, Carbaryl)	• Decrease in sperm density per gram of cauda epididymis, • Distortion of the seminiferous tubule architecture, with disrupted spermatogenesis. Many tubules showed accumulation of cellular debris/mass in their lumens, interstitial edema (swelling of the spaces between tubules), and varying degrees of sperm loss	
Dichlorophenoxyacetic acid (2,4-D)	• Asthenospermia , necrospermia, and teratospermia • Increased percentage of chromosomal aberrations in bone-marrow cells and spermatocyte cells	[69]
dibromochloropropane (DBCP)	• Decreased sperm count, elevated serum FSH but not LH; Azoospermia; decreased libido or impotence; selective atrophy of the germinal epithelium (intact Sertoli cells); reduced sperm counts and high LH/FSH; oligospermia; germinal	[69]

	epithelium damage; male subfertility; spontaneous abortion; hormonal imbalances; altered sex ratio in offspring.	
Benomyl	• Necrosis of dividing germ cells; abnormalities in the formation of spermatid nuclei; occlusion of the efferent ductules; sloughing (shedding) of germ cells; an estrogen-like (estrogenic) effect; blockage of the efferent ducts; and obstruction preventing sperm passage to the epididymis	[69]
Atrazine	• Reduction in body weight, as well as a decrease in the relative weights of the pituitary and the ventral prostate. • Testicular sperm number, sperm motility, and α-glucosidase activity in the epididymis were also decreased	[69]
Mancozeb	• Testis, epididymis, seminal vesicle and ventral prostate weights declined. Sperm counts (testicular and epididymal), fertility and serum testosterone also decreased. Acid-phosphatase (ACP) activity rose, while gonadal ACP and SDH activity fell. ALP and LDH activities increased, and seminiferous and epididymal tubules showed degeneration	[69]

Pesticides acting as EDCs can block the androgen receptor**,** reducing normal androgen signaling. [70][71][72]. They also increase oxidative stress**,** damaging testicular cells and triggering apoptosis[73][74]. Disruption of the HPG axis leads to altered LH/FSH production and decreased testosterone synthesis[75]. They can also interfere with steroidogenic enzymes, changing how testosterone is metabolized[72].

2.3 Plastic compounds and other endocrine disruptor

Endocrine-disrupting chemicals (EDCs) can mimic or block natural hormones by binding to receptors like estrogen, androgen, or thyroid receptors[76][77][78]. They're found in many everyday products ; for example, plastics, cosmetics, toys, and packaging as well as in foods, water, and air[79]. EDCs disrupt hormone signaling via multiple mechanisms: they act as agonists or antagonists at nuclear receptors, modify DNA methylation, interfere with hormone synthesis, transport or metabolism, and even impair cell–cell communication[80], [81]. Their impact on male reproductive pathways especially on hormone balance and fertility is a major concern.

Phthalates used in plastics to improve flexibility are widespread in products like flooring, cosmetics, and food packaging, and can enter the body via ingestion, inhalation, or skin contact. Urine and semen studies have linked metabolites such as MBP, MBzP, and MEHP to reduced sperm concentration, motility, and increased DNA damage[82], [83]. Mechanistically, phthalates may disrupt the endocrine system by blocking hormone receptors and reducing testosterone production in Leydig cells, impairing steroidogenic proteins, and triggering germ cell apoptosis. [84], [85][73] In vitro studies also show that MEHP increases germ cell death without significantly impacting testosterone in fetal human testes, suggesting direct toxicity to germ cells[86].

Bisphenol A (BPA), commonly found in plastics and resins, can weakly activate estrogen receptors. Animal studies show BPA exposure reduces sperm count and motility and increases DNA damage. [87], [88] In human sperm in vitro, BPA causes mitochondrial dysfunction, oxidative stress, apoptosis, and DNA damage[89]. Some epidemiological studies link higher urinary BPA to lower sperm concentration, motility, vitality, and total count, though findings are inconsistent. [90] Emerging evidence also suggests BPA exposure changes sperm protein and

miRNA profiles, potentially affecting fertility[91]. Tab 3 presents the effects of plastics on the male reproductive system, including their impact on hormone regulation, spermatogenesis, sperm quality, and fertility.

Table 3: Effect of plastic on male reproductive system

Plastics	Effect	Mechanism
Phthalates	Sperm concentration and motility declined, and DNA damage levels rose	They mimic endogenous hormones; bind to or block hormone receptors; alter hormone or receptor metabolism; reduce testosterone production; and trigger germ-cell apoptosis.
BPA	Sperm concentration and motility declined, and there was increased DNA damage	Acts as a weak estrogen-receptor agonist and reduces the expression levels of androgen receptors

Jobs with frequent contact with heavy metals, pesticides, solvents or other estrogenic chemicals can lower sperm production, increase abnormal sperm, and reduce androgen levels (occupational reviews) [91]. Spermatogenesis normally occurs ~3–4°C below core body temperature, and a sustained 1°C rise is associated with ≈14% fewer sperm[92], [93]. Occupations with prolonged heat or toxin exposure (e.g., bakers, welders, metallurgists, cooks, ceramic workers) have been linked to poorer semen quality in epidemiological studies [94], [95].

2.4 Work-related stress

According to McGrath and Altman, job stress arises when there's a significant mismatch between an individual's abilities and the demands of their work. It is linked to serious behavioral, physical, and psychological issues. This stress doesn't just affect health, it can also influence childbearing through three avenues: fertility (actual births), fertility intention (desire to have children), and infertility treatment[96]. Possible mechanisms include stress-induced reductions in sperm quality (e.g., lower motility, abnormal structure), hormonal disruptions (like reduced luteinizing hormone and testosterone), and impaired testicular function. [97] Social support at work may buffer some of these negative effects. Chronic job stress via elevated glucocorticoid levels can trigger apoptosis in germ cells, Leydig cells, and Sertoli cells[98], [99]. This not only reduces testosterone production by damaging Leydig cells [100], but also impairs spermatogenesis by disrupting Sertoli cell function and inducing germ-cell death[101]. Additionally, stress can raise oxidative stress (via reactive oxygen species), which further harms sperm quality[96]. Studies also suggest a two-way cycle: stress reduces sexual desire and performance, and worries about work or treatment can lower fertility intentions. Importantly, job stress may impair infertility treatment, partly by reducing the time available for treatment and partly by inducing physiological changes that interfere with it.

2.5 Psychosocial factors

Psychological stress is a major factor in idiopathic male infertility: it raises levels of cortisol and catecholamines (like epinephrine and norepinephrine), which boost intracellular reactive oxygen species (ROS) and trigger inflammation[102][103]. High glucocorticoid levels (from stress) act on Leydig cells via their receptors to suppress steroidogenic enzymes, reduce testosterone synthesis, and even induce Leydig cell apoptosis [104][100][98].

Furthermore, the enzyme 11β-hydroxysteroid dehydrogenase (11β-HSD) which regulates glucocorticoid metabolism inhibits key steroidogenic enzymes when stress is chronic, worsening testosterone decline[102], [105]. In acute stress, glucocorticoid excess can disrupt the hypothalamic–pituitary–gonadal (HPG) axis: over time, this may lower GnRH and LH secretion, compounding the suppression of testosterone production[102][103]. Finally, stress-mediated activation of the sympathetic (adrenergic) system may cause testicular vasoconstriction, further impairing Leydig cell function and thereby reducing spermatogenesis[102].

2.6 Lifestyle causes of male infertility

Male fertility is sensitive to many lifestyle and environmental factors. Research suggests that the decline in semen quality may be driven not only by aging, but also by modifiable behaviors and exposures. [106], [107]. Cigarette smokers often have lower sperm count, motility, and worse morphology. Smoking generates high reactive oxygen species (ROS), damaging sperm via oxidative stress, DNA fragmentation, and apoptosis[106]. Chronic heavy drinking disrupts the hypothalamic-pituitary-gonadal (HPG) axis, reducing testosterone and impairing Leydig and Sertoli cell function. It also raises oxidative stress, which can damage sperm[107], [108]. Use of marijuana, cocaine, steroids, and opioids can impair the HPG axis, reduce sperm production, alter testicular structure, and affect sperm function[106]. Excess fat leads to hormonal imbalances (like more estrogen via aromatization), increased oxidative stress, higher scrotal temperature, and inflammation all of which harm sperm quality [107], [109]. Diets rich in antioxidants (e.g., Mediterranean-style diet) are linked with better semen quality, whereas processed meat, sugary drinks, and high-fat diets are associated with poorer sperm parameters[106]. Some studies find no significant impact of caffeine on sperm count or motility[110]. Prolonged exposure of testes to elevated temperatures (sitting long hours, tight clothing, using a laptop on the lap) can induce oxidative stress, germ-cell damage, and impaired sperm production[110][106]. Advanced paternal age (e.g., beyond 35–40 years) is linked with lower sperm motility, increased DNA damage, and reduced viability[111]. Chronic psychological stress and exposure to electromagnetic radiation may also lower semen quality, potentially via oxidative stress and hormonal disruption[109][106]. Many of these factors increase oxidative stress, which damages sperm membranes, DNA, and reduces motility[107][106]. Also, hormonal disruption reduces sperm production and impairs spermatogenesis [107][108], and direct damage to germ cells such as from heat or toxins can lead to apoptosis or DNA fragmentation[112]. Because many of these risk factors are modifiable, lifestyle changes (like quitting smoking, reducing alcohol, improving diet, managing weight, minimizing heat exposure) could significantly improve male fertility. For men planning to conceive, assessing and addressing these factors can help optimize sperm quality and fertility outcomes.

3 Preventive & protective strategies

Hazard Assessment & Risk Control:

Employers should actively identify and evaluate reproductive hazards in the workplace such as chemical exposures, excessive heat, and radiation and conduct proper risk assessments (CCOHS). To manage these risks effectively, they should follow the established "hierarchy of control," which prioritizes eliminating or substituting hazardous agents, implementing engineering controls like adequate ventilation, isolating workers from the source of exposure, and enforcing safe work practices to minimize harm.

Engineering Controls and Work Practices:

Improving workplace ventilation or installing exhaust systems can significantly reduce airborne exposure to solvents, dust, and other reproductive hazards (CCOHS). Using physical barriers or enclosed systems around hazardous processes further limits direct worker contact with harmful agents. To minimize continuous exposure, employers can implement job rotation or scheduled breaks, which help reduce prolonged heat or stress-related risks. Heat exposure can also be controlled by providing cooling breaks, ensuring proper hydration, and maintaining temperature-regulated work areas.

Personal Protective Equipment (PPE):

Workers should be provided with appropriate personal protective equipment—such as gloves, respirators, protective clothing, and goggles—when handling substances known to harm reproductive health . In occupations involving high heat exposure, the use of heat-shielding gear or breathable, cooling fabrics is essential to reduce scrotal overheating and protect male reproductive function.

Training & Awareness:

Employers should train workers about reproductive hazards—what they are, how they can harm fertility, and practical steps to avoid or reduce exposure (CCOHS). They should also foster open reporting of reproductive-health concerns, symptoms, or unsafe conditions so issues can be investigated and controlled promptly.

Medical Monitoring:

Employers should establish biological monitoring when workplace reproductive hazards exist — for example, regular testing for heavy metals in blood or semen — and maintain records to detect patterns. They must also track, investigate, and respond to any clusters of reproductive health problems or reduced fertility among workers to identify causes and implement corrective measures[113].

Lifestyle & Nutritional Support:

Encourage a diet high in antioxidants — vitamin C, vitamin E, coenzyme Q10, zinc, and selenium — to help counteract oxidative stress from occupational toxins, alongside general health measures such as quitting smoking, cutting back on alcohol, and learning stress-management techniques; these steps support sperm health and treatment outcomes (PubMed). Promote maintaining a healthy weight, since obesity can worsen the harmful effects of chemical exposures on reproductive function[114][113].

Administrative & Policy Measures:

Employers can provide accommodations for workers planning to conceive, such as temporary transfers to lower-risk tasks, while also implementing reproductive-hazard policies that include clear risk communication, regular exposure monitoring, and worker reassignment when necessary[113].

Specific Measures for Common Risks

Heat exposure can be reduced by using cooling vests, providing shaded rest areas, and limiting continuous heat exposure. Proper PPE, safer substitutes, and hygiene facilities help minimize heavy-metal contamination. Radiation risks can be controlled by following safety protocols, using shielding, monitoring doses, and maintaining safe distances. For solvents and other chemicals, using low-toxicity alternatives, prohibiting eating or drinking in work areas, and ensuring safe storage and disposal are essential.[113]

By combining engineering controls, protective equipment, education, and medical surveillance, workplaces can significantly reduce the risk of occupational exposures impairing male fertility.

4 Policy and regulatory perspectives for occupational hazards on male infertility

Workplace exposures (chemicals, heat, radiation, solvents, metals, shift work) can reduce sperm quality and fertility; workplaces therefore offer the best point for prevention and surveillance [115] [116]. The WHO/ILO guidance on occupational health systems emphasizes the management of reproductive hazards , while OSHA and NIOSH provide detailed resources on reproductive risks, assessment methods, and workplace control measures and broader prevention strategies are reinforced through ILO standards and ISO 45001 occupational safety and health management system requirements[116][117][118].

Policies for reducing occupational contributors to male infertility should mandate reproductive-hazard risk assessment and implementation of the hierarchy of controls (Global Reporting Initiative), require WHMIS/SDS-style disclosure and reproductive-toxicity classification with phase-outs of high-risk EDCs (CCOHS), establish exposure limits and biological-monitoring guidelines for reproductive toxicants (CDC), ensure medical surveillance and confidential reproductive-health services including preconception counseling and job accommodations (PEHSU/DEOHS), obligate employers to maintain training, risk-communication, and exposure-record systems (PEHSU/DEOHS), strengthen inspection and compliance incentives (ILO), and expand surveillance and research through occupation-linked fertility registries and long-term studies. [119][117][115][120][118][121]

5 Conclusion

Occupational exposures including heat, heavy metals, pesticides, solvents, radiation and other endocrine-disrupting agents mcan disrupt hormones, impair spermatogenesis, and increase oxidative stress, leading to reduced sperm count, quality and function [122][123]. These risks are greatest with chronic, high-intensity, or

mixed exposures and are compounded by adverse lifestyle factors; multiple epidemiological reviews report consistent associations between workplace hazards and poorer semen parameters and fertility outcomes [124][125]. Accordingly, implementing workplace controls, exposure limits, medical surveillance, and targeted prevention policies (aligned with NIOSH/WHO/OSHA guidance) is essential to protect male reproductive health[115]. Raising awareness, funding targeted research, and enforcing occupational policies are essential to reduce work-related contributions to male infertility: education and risk communication help workers and employers recognize hazards, while focused epidemiology and longitudinal studies improve causal evidence and guide limits and controls [126][127]. Strong policy enforcement through national OSH laws, inspections, mandated biomonitoring, and alignment with international guidance (WHO/ILO/NIOSH/ACOEM) ensures prevention, surveillance, and timely interventions to protect reproductive health [120] .

Author Contributions

All authors contributed substantially to the preparation of this review article. **Kanan Gamit** conceived the idea, designed the review framework, and performed the primary literature search and manuscript drafting. **Umang Shah** contributed to preparation of figures. Milap Patel reviewed and edited the manuscript for intellectual content, ensured accuracy of references, and provided overall supervision. All authors read and approved the final manuscript and agree to be accountable for the content of the work.

Conflict of Interest

The authors declare no conflict of interest.

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