RESEARCH PAPER

Primary Mechanisms of Environmental Pollutant-Induced Male Reproductive Disorders: AN updated literature review.

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ABSTRACT
Studies from recent times show that male reproductive health deterioration links to chemical contamination found in environmental and workplace settings. The widespread use of synthetic substances during the past few decades has led to a significant decrease in sperm quality and hormonal balance. The presence of these pollutants, numerous in their potential to disrupt endocrine functions, has sparked significant apprehension regarding their lasting effects on fertility and overall population health. A comprehensive search was conducted in the PubMed, Scopus, and MEDLINE databases using terms such as environmental pollutants, male infertility, endocrine disruptors, oxidative stress, and spermatogenesis. Research conducted from 1990 to 2025 has been revivewed and analyzed to outline the underlying mechanisms of disease and the results related to reproduction. Research shows that phthalates and bisphenols and pesticides and flame retardants disrupt the hypothalamic–pituitary–gonadal axis while blocking steroid production and creating oxidative stress that affects sperm shape and motility. The research shows that exposure to pollutants leads to male infertility according to the overall evidence despite varying results from different studies with different methods and participant groups. Environmental and occupational pollutants significantly impact the decline in male fertility. The protection of reproductive health requires three essential elements which include enhanced biomonitoring and strong regulatory systems and specific national research initiatives in Kyrgyzstan..
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INTRODUCTION

The inability to conceive a child during one year of unprotected intercourse defines infertility which affects 10–15% of couples worldwide at equal rates between men and women [1]. The worldwide decline of male reproductive health during the last few decades has created a major international health issue. This decline has been attributed by several experts to the increasing release of synthetic pollutants and industrial chemicals into the environment [2]. Carlsen et al. (1992) documented a twofold reduction in average sperm counts in men globally between 1940 and 1990. Concurrently, reports have indicated a rising incidence of testicular malignancies and congenital abnormalities such as cryptorchidism and hypospadias [3]. The 1992 research discovered that male reproductive issues became more common because of chemical substances containing estrogenic or endocrine-disrupting properties. Scientists later identified pesticides and herbicides and fertilizers and plasticizers and surfactants and industrial by-products as major environmental pollutants [4]. The environment now contains more than 80,000 new chemicals which stay in the air and soil and water and pass through food chains to reach human bodies. Scientists have found these compounds throughout sediments and water bodies and wastewater and human and wildlife food supplies which demonstrates their extensive and enduring environmental presence [5].

A strong correlation has been established between exposure to environmental pollutants and adverse reproductive outcomes such as reduced sperm quality, increased sperm morphological abnormalities, miscarriage, stillbirth, congenital anomalies, and testicular dysfunction [6]. The steady rise in male infertility rates over the past two decades cannot be fully explained by genetic factors or chronic diseases alone [7]. Emerging evidence points to the combined impact of environmental conditions and occupational exposures, where individuals are subjected to complex mixtures of endocrine disruptors (EDs) that interfere with hormonal balance and spermatogenic

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function [8]. Research has established links between these pollutants and male reproductive problems but scientists need to identify the specific ways these substances harm male fertility. The combination of lifestyle factors including alcohol use and smoking and obesity from high-calorie diets and drug consumption makes reproductive health more vulnerable [9]. The combination of chemical pollutants with occupational and environmental factors that include heat exposure and noise and vibration and electromagnetic radiation leads to spermatozoa DNA damage which worsens reproductive outcomes. Research continues to study how environmental factors interact with genetic factors to affect male fertility because scientists need to determine the exact impact of each factor [10].

The novelty of this review lies in its comprehensive analysis of the pathogenetic mechanisms by which environmental chemical pollutants impair male reproductive function. Research has shown that pollutant exposure leads to fertility decline but scientists continue to study the specific biological mechanisms that include endocrine disruption and oxidative stress and genetic damage. The research combines existing studies about environmental pollutant effects on spermatogenesis and hormonal regulation to create a comprehensive review of current knowledge. The research provides essential information for developing clinical treatments and prevention methods through its identification of environmental contaminants that can be targeted for intervention. The worldwide increase in infertility requires scientists to understand how synthetic chemicals affect male reproductive health because these substances exist everywhere in the environment.

Endocrine-mediated effects of pollutants on the male reproductive system

A growing body of evidence demonstrates the reprotoxic effects of environmental pollutants through their disruption of the endocrine system. Endocrine disruptors (EDs) are present in a wide range of sources, including pesticides, food contaminants, cosmetics, and polluted water and air [11]. These substances interfere with the hypothalamic–pituitary–gonadal (HPG) axis, a tightly regulated hormonal network that controls male reproductive function [12]. Normally, the hypothalamus secretes gonadotropin-releasing hormone (GnRH), which stimulates the pituitary gland to release gonadotropins luteinizing hormone (LH) and follicle-stimulating hormone (FSH) [13]. These, in turn, regulate spermatogenesis by controlling androgen secretion from Leydig cells and supporting Sertoli cell activity. Because male reproductive processes are strictly hormone-dependent, any disruption in this axis can significantly impair sperm quality [14]. Endocrine disruptors that act as antiandrogens or mimic estrogens can disturb this delicate balance, leading to altered sperm production, motility, and morphology. EDs may exhibit estrogenic or antiandrogenic properties and act as either agonists or antagonists of endogenous hormones. Although these pollutants affect multiple organs, the testes remain one of the most vulnerable targets [15]. The increased expression of estrogen receptors (ERs) in testicular tissue, combined with the potent influence of estrogens on spermatogenesis and steroidogenesis, contributes to the heightened susceptibility of the testes to chemical insults. Several endocrine disruptors, including bisphenol A (BPA), exhibit potent estrogenic or antiandrogenic activity, disturbing reproductive function by altering the HPG axis. These substances interfere with GnRH release, gonadotropin secretion, and downstream signaling cascades within the reproductive system. The serum levels of LH and FSH and testosterone decrease when pollutants disrupt the normal operation of this axis. The testicular steroidogenesis process becomes blocked when pollutants cause Leydig cells to lose their LH receptor (LHR) expression [16].

The hormone testosterone faces suppression when perfluorooctane sulfonate (PFOS) binds to LHRs. The exposure to pollutants results in decreased receptor numbers and blocked adenylate cyclase and cyclic AMP (cAMP) activity and blocked steroidogenic enzyme function [17]. The fertility process depends on FSH and testosterone and estradiol and Leydig and Sertoli cell functions which become impaired when these components experience disruption. Research using rodents demonstrates that pollutants disrupt spermatogenesis by causing germ cell death and decreasing sperm production [18]. The Fas–FasL interaction and mitochondrial-dependent pathways activate apoptosis in primary germ cells. BPA activates its effects by changing ERα expression in Leydig cells and ERβ expression in Sertoli cells. BPA causes Sertoli cell death through two mechanisms which include actin filament aggregation and cytoskeletal framework disruption that leads to structural damage. The germinal epithelium and hematotesticular barrier (HTB) experience damage because pollutants attack the proteins which maintain their structural integrity [19]. BPA blocks junctional protein expression through the AP-dependent signaling pathway but PFOS and Aroclor 1254 increase HTB permeability through p38-MAPK signaling pathway activation. The damage of male reproductive function by pollutants occurs through oxidative stress as their main underlying mechanism. The increase of reactive oxygen species (ROS) prevents steroidogenic enzymes from functioning while triggering cell death pathways in testicular cells which results in impaired steroid hormone production and defective sperm development [20]. The epididymal antioxidant system disruption causes sperm maturation problems which result in decreased motility and abnormal morphology and DNA damage. The testicular development of germ cells depends on Sertoli cells because they provide essential metabolic and structural support. The Sertoli to germ cell ratio serves as an essential factor for maintaining proper energy homeostasis and sperm development. The synthetic insecticide cypermethrin causes seminiferous epithelium shape changes and Sertoli cell differentiation loss which damages their operational capacity [21].

The organochlorine pesticide endosulfan causes germ cell and Sertoli cell degeneration through oxidative stress and

apoptosis which results in severe sperm quality deterioration and reduced sperm production [22]. The endocrine system faces disruption because Leydig cells which produce testosterone under LH control become vulnerable to damage. The endocrine system and fertility become impaired when any factor damages Leydig cell function because testosterone plays a crucial role in spermatogenesis and genital development and secondary sexual characteristics [23]. The production of steroids becomes blocked at various points during the process. The exposure of BPA and triclosan leads to decreased LH receptor expression and adenylate cyclase activity which results in lower cAMP production and subsequently reduced testosterone synthesis. BPA exposure during preimplantation stages in mouse models leads to decreased StAR and P450scc gene expression which causes testicular development delays [24]. The exposure of Leydig cells to Aroclor results in reduced cell viability and blocked 3β-HSD and 17β-HSD enzyme activities which decrease testosterone production. The expression of StAR and androgen-binding protein (ABP) decreases while enzyme activity decreases when organochlorine compounds like lindane and methoxychlor are present which leads to increased ROS and hydrogen peroxide production and enhanced oxidative stress. Testicular tissue macrophage activation from arsenite exposure leads to testosterone metabolism disruption and complete inhibition of steroidogenesis [25]. The steroidogenic pathways of Leydig cells experience inhibition from phthalates which are widespread plasticizers through their direct interference with cell function. Research using adult testicular cell cultures demonstrates that DEHP and MEHP phthalates decrease androgen production which proves their negative impact on testicular steroidogenic pathways [26]. Table 1 summarizes the primary mechanisms through which environmental pollutants, including endocrine disruptors such as BPA, phthalates, and organochlorines, impair male reproductive function by targeting the HPG axis, Leydig and Sertoli cells, germ cells, and steroidogenic pathways

Table 1: Major Mechanisms of Environmental Pollutants Affecting Male Reproductive Health

Target / Mechanism	Pollutants / Examples	Effects	Reference
HPG axis & hormones	Bisphenol A (BPA), PFOS, triclosan	↓ GnRH, LH, FSH, testosterone → impaired spermatogenesis	[27]
Leydig cells	PFOS, BPA, phthalates (e.g., DEHP, MEHP)	↓ LH-receptor expression, blocked steroidogenesis → ↓ testosterone	[28]
Sertoli cells	BPA, cypermethrin, endosulfan	Cytoskeleton/junctional protein damage → impaired support for germ cells & sperm maturation	[29]
Germ cells & spermatogenesis	Pesticides, BPA	Apoptosis via Fas–FasL, mitochondrial pathways; ↓ sperm count, motility, morphology	[30]
Testicular tissue / HTB (hematotesticular barrier)	BPA, PFOS, Aroclor	Junctional protein disruption → ↑HTB permeability, testicular damage → impaired spermatogenesis	[31]
Oxidative stress	BPA, phthalates, organochlorines, heavy metals	↑ ROS → steroidogenic enzyme inhibition; sperm DNA damage, abnormal morphology & motility	[32]
Steroidogenic enzymes & pathways	BPA, Aroclor, DEHP/MEHP	Blocked StAR, P450scc, 3β-HSD, 17β-HSD → ↓ testosterone → impaired androgen signaling	[33]

Violations of the hematotesticular barrier under the action of environmental pollutants

The hematotesticular barrier (GTB) is formed by tight contacts (PS), gap contacts, and adhesive contacts (AK) between Sertoli cells. GTB divides the spermatogenic epithelium into 2 compartments basal and apical. Postmeiotic development of germ cells occurs in the apical compartment, while renewal of spermatogonia and differentiation to preleptothenic spermatocytes occur in the basal compartment.

GTB provides an optimal environment for meiosis and postmeiotic development of germ cells in isolation from the systemic circulation; otherwise, the formation of antibodies to spermatozoa would occur. The tightness of the barrier is explained by the presence of several types of connections. At the same time, GTB is one of the main targets of various environmental pollutants.

It was shown that exposure to a commercial mixture of PCBs Aroclor 1254 leads to disruption of GTB in rats and in Sertoli cell culture due to stimulation of endocytosis and degradation of connective proteins via the p38 mitogen-

activated protein kinase (MAPK) pathway. It was also found that $CdCl_2$ via the p38-MAPK signaling pathway can disrupt the function of GTB. Another study evaluated the negative effect of perfluorooctane sulfonate (PFOS) on GTB using in vivo and in vitro models. Exposure to PFOS resulted in increased GTB permeability, phosphorylation of p38/activating transcription factor 2 (ATF2), and expression of matrix metalloproteinase 9, with a parallel decrease in the expression of GTB proteins (occludin and connexin 43), which underlines the importance of the role of the p38/ATF2/MMP9 signaling pathway in PFOS-mediated GTB degradation.

A study of the effect of BPA and $CdCl_2$ on the culture of human Sertoli cells showed a violation of their adhesive properties due to changes in the F-actin network. Exposure of human Sertoli cells to monobutyl phthalate (MBP), which belongs to the group of substances that disrupt the function of the endocrine system, led to a decrease in the expression of the connective proteins occludin, ZO-1, beta-catenin, and androgen receptor (AR) [34]. It was found that MBP violates the structural and functional integrity of GTB through the AP-dependent pathway, and an increased susceptibility of connective proteins to the negative effects of various environmental pollutants was noted. Dankers et al. (2013) demonstrated that some of the endocrine-disrupting substances, including BPA, tetrabromobisphenol A (TBBPA), DEHP, MEGP, perfluorooctanoic acid (PFOA), and perfluorooctanoic sulfonic acid (PFOS), affect the mouse ATP-binding GTB cassette transporters, resulting in a decrease in testosterone secretion in Leydig (MA-10) cells [35].

Effects of environmental pollutants on the function of the testicular appendage

The testicular appendage, located in the distal part of the tail, creates the perfect conditions for the maturation and storage of sperm. The spermatozoa that exit the testicle are in a state of immobility and lack the capability to fertilize the egg. Spermatozoa gain motility and the capacity to fertilize as they traverse the testicular appendage [36]. The epithelium of the testicular appendage exhibits a distinctive array of dense compounds that play a crucial role in establishing the hematepididymal barrier, essential for preserving the optimal characteristics of the microenvironment within the lumen of the testicular appendage. The composition of proteins and enzymes within the intraluminal fluid exhibits variations as it traverses from the head to the tail of the testicular appendage [37]. The evolving properties of the microenvironment influence the morphological and biochemical traits of spermatozoa during their maturation process. Consequently, elements that interfere with the integrity and/or operation of the testicular appendage can hinder sperm development and subsequently impact male fertility [38]. Research examining the effects of pollutants on the testicular appendage has revealed a reduction in the mass of the appendage, degenerative alterations in the epithelium, and a decline in the quantity of spermatozoa. In adult rats, a combination of cadmium (a heavy metal) and diazinone (a pesticide) resulted in notable structural alterations in the testicular appendage tissue, including thickening of the epithelium, necrosis of epithelial cells, narrowing of blood vessels, interstitial edema, and infiltration by mononuclear cells [39] [40].

Investigations into the harmful effects of inorganic arsenic on young rats prior to puberty were conducted at concentrations of 0.01 and 10 mg/l. The findings from these studies, which correspond to concentrations detectable in the environment, indicated disruptions in the structural integrity of testicular and epididymal tissues, evidence of inflammatory infiltration, a reduction in spermatozoa within the lumen of the testicular appendage, and alterations in the expression of androgen receptors in the testicular appendage [41]. Research indicates that exposure to certain substances, specifically bisphenol A, leads to reproductive disorders in the offspring of female rats. Consequently, males that are offspring of these females exhibit a rise in spermatozoa count and an extended duration for their transit through the tail of the testicular appendage, which correlates with a heightened incidence of sperm abnormalities [42].

Effects of environmental pollutants on the prostate gland and seminal vesicles

The seminal vesicles and prostate gland serve as supplementary reproductive glands, contributing to the production of approximately 90% of seminal plasma, while the remaining 10% is generated by the testicular appendage [43]. Seminal plasma serves as a crucial medium that provides protection, nourishment, and transportation for spermatozoa from the instant of ejaculation until fertilization occurs. It contains essential substances that play a role in modulating the functions of spermatozoa [44]. The levels of pollutants found in seminal fluid show an inverse relationship with the quality of semen and the biochemical markers associated with other reproductive glands in cases of male infertility. PantN. et al. (2004) demonstrated elevated levels of chlorinated pesticides, specifically hexachlorocyclohexane (HCH) isomers and dichlorodiphenyltrichloroethane (DDT) metabolites, in the semen of infertile individuals when compared to their fertile counterparts [45].

A direct correlation was observed between the concentration of chlorinated pesticides and fructose levels in semen in cases of male infertility. Fructose plays a crucial role in maintaining normal metabolic functions and the motility of sperm. An elevation in its concentration within sperm suggests a reduction in sperm consumption, which consequently leads to a decline in their motility [46]. Pant N. et al. (2004) discovered a negative correlation between the levels of chlorinated pesticides in semen (α-HCH, β-HCH, and pp'-DDE, a metabolite of DDT) and indicators of prostate function (acid phosphatase and γ-glutamyltranspeptidase). A comparable association was observed regarding the levels of heavy metals (lead and

cadmium) in seminal plasma and indicators of seminal vesicle and prostate function in cases of male infertility [45].

Exposure to specific pollutants results in a reduction in the mass of seminal vesicles and the prostate gland. This happens either by directly causing degenerative changes and disrupting tissue structure, or indirectly by inhibiting testosterone production in the testis, which serves as the primary growth factor for the accessory sex glands [47]. In studies involving rodents, exposure to BPA and octyl phenol (OP) resulted in tubular atrophy and the emergence of intraepithelial neoplasia in the prostate, whereas cadmium led to vasoconstriction and degenerative cellular alterations in the seminal vesicles and prostate [48]. Exposure during prenatal development to a combination of phthalates, reflecting levels found in humans, led to a reduction in the size of the gonads, prostate, and seminal vesicles in F1 male mice. This was accompanied by a significant decline in the expression of steroidogenic genes and serum testosterone levels.

Effects of pollutants on sperm function

Male infertility assessment requires complete sperm quality evaluation through sperm count and motility and viability and spermatozoa morphology assessment. Scientists link environmental pollutant exposure to decreased sperm quality and increasing neonatal cryptorchidism cases which have been observed during recent times. The study by Lea G R. et al. (2016) showed that sperm quality in purebred dogs deteriorated during 26 years from 1988 to 2014 while cryptorchidism cases in male offspring rose from 1995 to 2014. The study found high levels of phthalates (DEHP) and organochlorine compounds (PCB153) in the testes of adult dogs and in commercial dog food [49]. Studies demonstrated that exposure of human and canine spermatozoa to PCB153 and DEHP in vitro at varying concentrations resulted in a significant increase in DNA fragmentation and a decrease in sperm motility. Studies have demonstrated an adverse correlation between the concentrations of specific pollutants in semen or urine namely PCBs, triclosan, bisphenol, and lead and multiple parameters of sperm quality in men, including concentration, total count, motility, viability, and morphology of spermatozoa [50]. WangY. et al. (2019) established a relationship between increased urinary phthalate concentrations and reduced semen quality, resulting from metabolic disturbances in seminal plasma predominantly associated with polyunsaturated fatty acids and acylcarnitine [51].

The study by MS Rahman et al. (2019) examining the impact of BPA on rodent spermatozoa at varying concentrations in vitro demonstrated a decrease in sperm motility and a disturbance in their movement kinematics, which was linked to a significant reduction in ATP concentration [52]. Additionally, it was noted that increased concentrations of BPA induce the activation of tyrosine phosphorylation of sperm proteins, resulting from modifications in the activity of protein-dependent kinase (PPC) associated with the acrosome reaction [53]. The changes in protein expression brought on by BPA led to an early acrosome reaction, which made it harder for fertilization to happen and messed up the development of the embryo.

Research conducted on mice in living environments demonstrated that BPA exposure causes sperm cell damage which results in poor sperm quality and function and abnormal sperm structure and leads to DNA damage and oxidative stress [54]. Human sperm cells exposed to DBP and MBP in laboratory settings experienced major reductions in their ability to move and penetrate cells. The sperm cells experience reduced functionality because phthalates disrupt their tyrosine phosphorylation processes which affects their ability to perform their regular tasks [55]. Studies demonstrate that in vivo experiments involving DBP result in diverse expression of various RNA elements, the induction of oxidative stress, and DNA damage in the spermatozoa of affected individuals. Chlorothalonil, a broad-spectrum fungicide, reduces the motility of wild boar spermatozoa and induces apoptosis, resulting in alterations to the mitochondrial membrane potential.

Epigenetic effects of environmental pollutants

To date, there is growing evidence that epigenetic changes may be an important mechanism mediating the impact of pollutants on male reproductive function [56]. The epigenetic mechanism involves DNA methylation, histone modification, and changes in miRNA gene expression. It has been reported that exposure to zearalenone (ZEA), a substance that disrupts endocrine regulation, during puberty in mice affects the process of meiosis and spermatogenesis signaling pathways, reducing sperm quality [57]. In addition, the concentrations of DNA methylation markers 5mC and 5hmC in mice exposed to ZEA were reduced, and the level of histone methylation marker H3K27 was increased [58]. A decrease in testicular ER expression was also observed in mice. Studies have shown the crucial role of the connection between the estrogen signaling pathway and genetic and epigenetic signaling pathways in the realization of the adverse effect of ZEA on spermatogenesis [59].

When exposed to pollutants during the development of an organism, epigenetic changes can be transmitted between generations (transgenerational epigenetics). Kailiang Li. et al. (2020) showed that exposure to DBP in embryos disrupts testicular function in F1 and F3 generations through modulation of Sertoli cell function and spermatogenesis [60]. DBP affects the total hypomethylation of DNA in offspring, including suppressing hypomethylation of follistatin-like promoter 3 (Fstl3). Transgenerational epigenetic changes caused by exposure of the embryo to various pollutants were confirmed by the results of several studies [61]. In particular, it has been shown that chlordecone, DDT, vinclozoline, and DEHP cause

disturbances in testicular tissue architectonics and reduced sperm quality in F1–F3 offspring [62].

Future Directions and Policy Recommendations for Kyrgyzstan

Male infertility has become a growing reproductive health issue in Kyrgyzstan because the country faces distinct environmental and industrial and healthcare problems which affect male fertility rates. The country faces rising environmental pollution because of urban development and industrial activities and agricultural pesticide usage and inadequate waste disposal systems. The absence of research data about chemical exposure and reproductive health makes it difficult to create evidence-driven decisions. The solution to these problems needs national research collaboration with enhanced environmental oversight and healthcare programs that adapt to local requirements. The recommendations establish particular research and policy development paths to study environmental pollutant effects on male reproductive health in Kyrgyzstan.

Establish a national reproductive health and environmental exposure registry: Currently Kyrgyzstan does not have any unified system which tracks infertility rates and exposure to pollutants. A national registry which monitors reproductive health metrics and workplace contamination and environmental pollution would enable sustained tracking and policy development. The system needs to unite healthcare facilities with laboratories and environmental protection organizations for its operation [63] [64].

Conduct longitudinal population-based studies across regions: **Research needs to determine how pollutants affect different regions of the country and how infertility rates vary across industrial and agricultural zones including Chüy and Osh and Jalal-Abad. The Kyrgyz population needs extended cohort research to establish direct connections between chemical pollutants including pesticides and heavy metals and plasticizers and male reproductive health [65] [66].**

Strengthen environmental monitoring and regulatory enforcement: The current environmental control systems require additional support through regular monitoring of air and soil and water pollutants which should focus on areas receiving industrial waste and high farming activities. The implementation of international chemical safety standards together with reproductive toxicity criteria in environmental laws will help stop long-term exposure to small amounts of toxic substances [67] [68].

Integrate reproductive risk assessment into occupational health programs: **The workforce of Kyrgyzstan includes many men who face chemical exposure risks during their work in mining and metallurgy and agriculture and textile production. The process of occupational health surveillance needs to include reproductive health assessments together with hormone tests and sperm quality evaluations for all medical check-ups. The protection of workplace safety and exposure reduction requires both employer involvement and regulatory agency oversight [69] [70].**

Enhance public awareness and preventive healthcare services: Public health programs need to teach people about environmental pollutants that harm reproduction while teaching them to make fertility-friendly choices through smoking quit programs and proper eating habits and decreased usage of products containing proven endocrine disruptors. Healthcare providers need environmental risk factor training to deliver better preventive care through patient counseling [71] [72].

CONCLUSION

Male infertility has become a significant public health concern because it affects millions of men throughout the world with developed nations showing the highest incidence rates. The environmental factors that affect male reproductive health include phthalates and bisphenols and pesticides and flame retardants which show the strongest links to reproductive problems. The broad distribution of these chemicals throughout food products and cosmetics and air and water systems creates challenges for determining which substance causes the most harm. The current challenge in linking specific chemicals to infertility stems from widespread chemical exposure which hinders researchers from determining individual pollutant effects. The current exposure assessment methods need improvement because they fail to measure how different chemicals interact with each other when people are exposed to multiple substances. Available evidence indicates that the decline in sperm quality reported over recent decades is frequently accompanied by hormonal imbalance, increased DNA fragmentation, and epigenetic and mitochondrial disturbances. Elevated levels of endocrine disruptors are repeatedly observed in affected populations. Yet, the diversity of study designs, population characteristics, and epidemiological models contributes to inconsistent findings. Despite these variations, the overall body of data supports growing concern among clinicians, reproductive specialists, and policy makers about the long-term impact of environmental pollutants on male fertility and, by extension, on population demographics. Male infertility now receives recognition as a condition that develops from workplace and environmental contact with harmful substances. The solution to this problem requires multiple centers to conduct extensive research using standardized testing protocols which will generate reliable results for all cases. The research must continue because it enables scientists to create preventive measures and establish regulatory standards and integrate infertility monitoring into occupational and environmental health programs. The identification of environmental and occupational exposure as causes of infertility enables public health protection to become stronger while safeguarding reproductive health for upcoming generations.

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