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Implications of Environmental Toxicants on Male Reproductive Toxicity: A Mini ReviewSamuel Damilare Shoyinka*¹ and Mathias A. Emokpae²Faculty of Medical Laboratory Science, Achievers University, Owo, Nigeria¹, Department of Medical Laboratory Science, School of Basic Medical Sciences, University of Benin²Author for Correspondence*: shoyinkasamuel94@gmail.com/ shoyinkasamuel@achievers.edu.ng/
+234-813-486-3003/ ORCID:0009-0004-7408-0223<https://dx.doi.org/10.4314/sokjmls.v9i4.14>**Summary**

Male reproductive toxicity refers to harmful effects on the reproductive system that can impact sexual ability and fecundity in men, as well as adversely affect the development of their offspring. The male reproductive system is affected by environmental toxins. This review seeks to shed more light on the impact of environmental toxicants on male reproduction and its implications for Nigerians. Relevant articles used for this review were obtained from many internet search engines. Environmental toxicants are substances or agents produced or generated into the environment by human actions that are capable of causing damage. Among the pollutants found in the environment are toxic metals, chemicals used for pest, electromagnetic radiation, plastics and other hormone-disrupting substances, airborne pollutants, alcoholic beverages, and other drugs for recreation. The imitation of ions, disruption of cellular communication pathways, epigenetic control over the expression of genes, oxidative stress-triggered apoptosis, changed gene expression, and endocrine disruption have all been proven by literature as some of the processes by which they compromise male reproductive health. In exposed Nigerians, environmental toxins have been documented to reduce blood testosterone concentrations, number of sperm cells, overall motility, viability, and percent morphological characteristics. Male reproductive toxicity laboratory studies comprise spermogram tests, hormonal assessment, toxicological studies, and probable effects on children. Based on studies of the literature, the falling male reproductive potential

is linked to unrestricted exposure without sufficient prevention and management. In the infertile belt of sub-Saharan Africa, especially Nigeria, this can be worst. Therefore, bio-monitoring, lifestyle modification, and enforcement of preventive measures could help to lessen the effect of ecological toxins on male reproduction.

Keywords: Male, Hazardous Substances, Reproduction, Humans**Introduction****Male Reproductive Toxicity**

Toxicity in science is the degree to which a material could endanger human or animal health (Arzuaga *et al.*, 2019). These days, advancement in the industrial revolution, man-made chemical use, pollution of the environment, radioactivity or dangerous compound exposure while working influences reproductive wellness (Baidya, 2019; Ma *et al.*, 2019). Male and female genital systems experience disruption by these pollutants in the environment (Henriques *et al.*, 2019; Piazza & Urbanetz, 2019). It is vital to educate individuals about all the impacts of these environmental toxins upon organs of reproduction.

Toxicity of the reproductive organs is the harmful effects some compounds have on an individual's capacity for procreation and the growth of their children (Baidya, 2019). Male reproductive toxicity is the intoxication of the reproductive organs that could influence sexual activity and fecundity in men as well deleterious consequences on the development of their progeny (Baidya, 2019; Krzastek *et al.*, 2020).

Environmental factors including chemical exposure, strong metal element, and other toxins that induce damage of the reproductive system are designated as reprotoxic agents (Piazza & Urbanetz, 2019), these compounds interfere with regular reproduction processes. Changes in the genital system, negative effects on start of puberty, disruptions in the generation of gametes and transport, variability in reproductive cycles, complications with sexual behavior, fertility obstacles, or shifts in other processes that are contingent on the competence of the reproductive system are some of the possible consequences (Cremonese *et al.*, 2017; Ahmad *et al.*, 2024). Male infertility, which contributes over half of the incidences of barrenness in spouses, is the incapability of a man to produce a successful female pregnancy. Male factor infertility is the term used to describe infertility brought on by aberrant sperm concentration, motility, or shape (Mathur *et al.*, 2019).

Environmental Toxicity

Any harmful agent or substance resulting from human activity or initiated into the natural environment by mankind is considered an environmental toxicant (Jiang *et al.*, 2024). There are several kinds of toxicants, which can come from either natural or manmade origin. Since it greatly influences the underlying mechanisms of numerous illnesses in humans, environmental contamination in any conceivable form has been of main health interest worldwide (Sokan-Adeaga *et al.*, 2023). Metals that are toxic such cadmium, lead, arsenic, chemical substances used in agriculture including herbicides, pesticides, substances of misuse such the stimulant caffeine, cannabis, nicotine, cocaine and heroin, electromagnetic radiation, pollution in the air, plastic materials and other disruptors of the endocrine system such phthalates, bisphenol A, alcohol, smoking, and recreational drugs (Krzastek *et al.*, 2020; Sokan-Adeaga *et al.*, 2023).

Mechanism of Actions of Environmental Toxicants Ideal Male Genital System Regulations

Different hormones coordinate basic reproductive process in males (Mínguez-Alarcón *et al.*, 2023). Fundamental to this

control is the hypothalamic-pituitary-gonadal (HPG) axis. Specialized GnRH-expressing neurones enable the rhythmic dispensation of gonadotropin-releasing hormone (GnRH) from the hypothalamus, hence starting this mechanism (Corradi *et al.*, 2016; Moreira *et al.*, 2021). Then the gonads generate estrogen and testosterone while the anterior pituitary gland generates luteinizing hormone (LH) and follicle-stimulating hormone (FSH). For proper physiological purposes, the male reproductive system mostly hinges on the actions of these several hormones generated by diverse bodily glands (Mathur *et al.*, 2019). Testosterone is highly crucial for the evolution and maintenance of male secondary sexual traits, increases sexual drive, and interacts in tandem with FSH to accelerate the generation of spermatic fluid (Concepción-Zavaleta *et al.*, 2022). Rising sperm counts cause the testes to synthesize the hormone, inhibin, that subsequently passes the bloodstream to block the release of GnRH. FSH and LH production, likewise as GnRH levels decline, therefore slowing down sperm assembly. One of the key feedback systems controlling and preserving sex hormones at quite constant levels is this one (Corradi *et al.*, 2016; Concepción-Zavaleta *et al.*, 2022). Some processes by which toxic metals compromise sexual health are programmed cell death, an ion imitating, inflammatory processes, change of cell communication pathways, epigenetic control of gene activity, a state of oxidative stress, obstructed genome regulation, wreckage to the testis-blood barrier and endocrine malfunction (Arzuaga *et al.*, 2019). Based on several forms of environmental toxicants, these systems are explored here.

Reproductive Toxicity of Some Environmental Toxins and Implications for Nigerians

1. Pesticides/Herbicides

Pesticide is any compound or mixture of chemicals utilized to avert or kill undesirable pests which includes carriers of disease in humans or animals, plants, microbes, or other organisms to improve agricultural output and aid manufacturing, handling, storing, or advertisements of the food and farming commodities (Ahmad *et al.*, 2024).

Among the most well-known pesticides linked to possible negative effects on fertility is dichlorodiphenyltrichloroethane, (DDT). DDT and its derivatives, especially 1,2-Dibromo-3-chloropropane (DBCP), have been shown to promote estrus in males via inhibition of androgen receptors (Fucic *et al.*, 2021). Reduced testosterone, lower male gonad mass, the quantity and proportion of active sperm cell in the epididymis, seminal vesicle weight, and rises in LH and FSH have been linked to DDT (Fucic *et al.*, 2021; Moreira *et al.*, 2021). Furthermore, more than hundred chemicals that kills pests have been categorized as inhibitors of the endocrine system with different suggested modes of action generating endocrine disturbance and possible consequences on fertility indices (Krzastek *et al.*, 2020; Yeiya and Emokpae *et al.*, 2022).

Male fertility has been investigated for impacts of pesticides as well as their mixtures. These substances can imitate biological hormones, interfere with their ideal operation and control the hormonal system (Cremonese *et al.*, 2017; Yeiya and Emokpae, 2022). Studies on organophosphates have connected them to aberrant semen indices including declines in count of sperm, motion, survival, and concentration as well as heightened damages to DNA and irregular sperm morphology. Furthermore, linked to morphological testicular alterations including diminished testicular volume is organophosphates (Ghafouri-Khosrowshahi *et al.*, 2019).

Low FSH, LH and raised testosterone levels was observed among Iranian farmers in in a study (Ghafouri-Khosrowshahi *et al.*, 2019). Organophosphates have been suggested to directly influence spermatogenesis by lowering antioxidant status. They could potentially change testosterone metabolism in the body, impair the testicular synthesis of testosterone, or influence the central gonadotropin secretion (Ghafouri-Khosrowshahi *et al.*, 2019).

According to findings from an analysis conducted in Ondo state, Nigeria, around 40.5% of pesticide-exposed farmers had very low serum testosterone concentrations. Low testosterone subjects showed far less FSH, LH, and estradiol than individuals with appropriate concentrations. Among farmers who had contact with pesticide, the sperm quality was significantly reduced than in control participants (Yeiya and Emokpae, 2022).

Member of the phenylpyrazole family, fipronil has been demonstrated to bring about diseased tissues changes at the seminiferous tubules of rats. Along with a reduction in the activity of antioxidant enzymes, these changes reflect oxidative damage evidenced by elevated malondialdehyde and nitric oxide concentration (Saleh *et al.*, 2020).

Furthermore, a study has shown the ability of a pesticide, endosulfan to cause death in animal Sertoli cells by means of a built-in cellular death route mediated by mitochondria. This is resulting from a notable rise of MDA levels and oxygen radicals, which aggravate lipid oxidation and compromise cell wall stability (Rastogi *et al.*, 2014). Furthermore, a study found that endosulfan affects the testes, which results in changes within it alongside a reduction of testicular number of cells, numbers of sperm, and agility. Male rats susceptible to this chemical were especially badly damaged, compared to females. This variation might have something to do with endosulfan's impact upon androgen receptor, which is more prevalent in males (Sebastian & Raghavan, 2015).

2. **Plastics and Other Endocrine Disruptors**

By attaching to their receptors, endocrine disrupting chemicals (EDCs) either imitate or prevent the effects of naturally occurring hormones (Mínguez-Alarcón *et al.*, 2023). Beauty products, dental products, detergents, fabrics, floor coverings, bed linens, toys, perfumes, permanent makeup and tattoo ink all contain EDCs along with other possibly reproductive hazardous substances. Additionally, found in agricultural products as well as pollutants in the air and water are EDCs (Kubincová *et al.*, 2019; Mínguez-Alarcón *et al.*, 2023). We particularly address several typical EDCs found in plastic products.

3. **Phthalate**

Items made from plastic have phthalate incorporated to improve their resilience, clarity, and elasticity. Common in our contemporary surroundings, they show up in items from glues, wallpapers, to scents, skincare products, and home personal hygiene items. Phthalates may penetrate into an individual via contact with the skin, consumption, or inhalation (Cai *et al.*, 2015; Wang *et al.*, 2016).

It is yet unknown how precisely phthalates affect fertility. By impersonating natural hormones, attaching to or obstructing natural receptors for hormones, and altering receptor metabolic processes, these substances are considered to perturb the endocrine organs (Vandenberg *et al.*, 2012; Cai *et al.*, 2015). The levels of phthalates and their byproducts in body fluids were investigated in a systematic review comprising fourteen research findings together with their correlation with human semen quality. The findings revealed that elevated amounts of urinary phthalate metabolites were associated with a reduction in sperm concentration. Furthermore, linked with diminished motility and kinematic characteristics in the sperm was mono-2-ethylhexyl phthalate, and methoxyethyl phthalate, which additionally triggered more significant injury to sperm genetic material (Cai *et al.*, 2015).

4. Bisphenol A (BPA)

A chemical detected mostly in plastic items and epoxy resins is bisphenol A. It mimics naturally occurring estrogen (Wisniewski *et al.*, 2015). Its actions on androgen and estrogen receptors cause its toxicity, especially with relation to the replication process. Though it is an oxidant, it may cause redox imbalance by means of the generation of free radicals and the oxidation of lipids, therefore resulting in cellular alterations (Obialor *et al.*, 2023).

Several investigations in animal models have demonstrated that BPA exposure lowers measured spermatid fluid indices, alters sperm motility, and causes severe sperm genetic damage. Reduced activities of androgen receptors, according to the studies, helps explain the failure of spermatogenesis in experimental animals (Wisniewski *et al.*, 2015; Cariati *et al.*, 2019; Jeřeta *et al.*, 2019).

According to a recent study, in infertile Nigerian males, the amounts of BPA in seminal plasma were denary greater than in control participants. Reduced quality and number of spermatozoa were linked to elevated correspondingly rise of this chemical in seminal plasma. Moreover, the bisphenol A concentrations correlated positively with aberrant sperm cell shape and inversely with mobility, progressive motility, count of spermatid fluid (Obialor *et al.*, 2023).

5. Heavy Metals

Heavy metals (HMs) are diverse groups of elements and are somewhat common in the surroundings, their toxicity arises via natural processes or from actions taken by humans (Jan *et al.*, 2015). By altering the hormone regulator axis and hence compromising gonadal activity and sperm development, heavy metals add to reproductive toxicity. Of most worry are heavy metals like arsenic, cadmium, lead, and mercury (Rana, 2014; Krzastek *et al.*, 2020).

Numerous industrial activities emit cadmium into the atmosphere; tobacco fumes also contain this metal (Lei *et al.*, 2015). By changing occluding cell junctions and hence upsetting cell signaling pathways, this widely recognized reproductive toxin disturbs the HPG axis, reduces Leydig cell activity, and causes cytoplasmic injury and death of nursemaid cells of the primary spermatogonia. Thought to diminish sperm numbers, cadmium is said to cause sperm DNA damage (Famurewa and Ugwuja, 2017; Kumar & Sharma, 2019).

Comparatively to normospermic males, a Nigerian study uncovered that seminal and plasma levels of cadmium alongside plasma lead were much greater in azospermic and oligospermic men (Famurewa and Ugwuja, 2017).

Although various sources can also cause human mercury exposure, ingestion of seafood is the main way this is achieved (Ma *et al.*, 2019). Increased mercury levels associated with subfertility in male, poorer seminal fluid characteristics, and elevated sperm genome destruction according to a systematic review. The writers did admit, nevertheless, that research on the subject are varied and clear conclusions cannot be deduced (Henriques *et al.*, 2019). Mercury is presumed to be an endocrine toxicant, even if the precise processes by which it causes male infecundity are yet unknown (Baidya, 2019).

Affecting several organs and growing embryo, lead is a notable reprotoxic (Taha *et al.*, 2013; Kabamba and Tuakuila, 2020). Lead poisoning alters sperm activity and production. Lead-related excessive formation of oxygen radicals can adversely affect seminal fluid potency, ability to move, genome integrity, and

chemotaxis required for seminal and ovary cell binding, all these can help hinder conception (Gandhi *et al.*, 2017). Adult rats given lead showed lower reproductive hormone levels, testicular 17-beta hydroxysteroid dehydrogenase (HSD) activity, androgen receptor control, spermatic fluid characteristics, alongside changes in antioxidants levels when compared to control groups (Oyeyemi *et al.*, 2019). Predisposition to lead alters sex hormones concentration and function of enzymes that are free radical scavengers, according to a study among Nigerian men exposed to lead, potentially through lead triggered redox imbalance (Rabiu *et al.*, 2019).

Arsenic has been shown to affect the testis by causing free radical damage in the micro-testicular milieu that results in lowered blood testosterone levels in bucks, poor semen characteristics, and death of cells that nourish spermatids (Baidya, 2019).

6. Radiation

Either from nuclear disasters, radiation treatment, or radiology, ionizing radiation is widely believed to have adverse effects on sperm formulation by generating free radicals and gene alteration, hence killing sperm cell and sperm stem cells (De Felice *et al.*, 2019). Small doses that are as little as 30 Gy can trigger mutations in genes during sperm synthesis; inherited disorders may therefore linger, and mutations might be passed on to next generations (Wdowiak *et al.*, 2019).

Radiofrequency electromagnetic radiation (RF-EMR) is released by cell phones. Mobile phone radiation is believed to contribute to the breakdown of genetic materials and reactive oxygen species. RF-EMR irradiation is implicated in rats with sperm cell mortality and alterations inside the gonad (Adams *et al.*, 2014). According to a study, rats exposed to 10 GHz microwaves for two hours a day for over 45 days suffered DNA damage and a decline in testosterone levels (Kumar *et al.*, 2013).

Laptops also discharge RF-EMR, laptop tethered to Wi-Fi has been attributed to diminished sperm motility and more fragmented DNA. These effects are believed to stem from RF-EMR irradiation to the male gonad as well as heat influences (Kesari *et al.*, 2018; Avendano *et al.*, 2012).

7. Air Pollution

Respirable particles and gaseous contaminants make up air toxics. Chromosomal abnormalities, higher rates of genetic materials splitting, suboptimal seminal fluid formation, irregular sperm shape, and shorter spermatic fluid motion have all been linked to exposure to air contaminants. The presence of oxygen radicals, epigenomics alterations, and cell apoptosis most certainly have produced these modifications (Vecoli *et al.*, 2016; Jurewicz *et al.*, 2018).

8. Smoking

A widely accepted factor contributing to infecundity is smoking. Dose-dependent but reversible, smoking cigarettes has been tied to declines in sperm velocity, population, and volume. It also compromises the results of assisted reproduction technologies (ART) (Kovac *et al.*, 2015). A systematic review involving over five thousand male smokers established that lower sperm count, and motility is attributable to smoking (Sharma *et al.*, 2016).

Nicotine is regarded as a poisonous element of tobacco combustion that directly impairs male reproductive functions; its toxicological impact has been shown to be dose-proportional manner. Low amounts of nicotine or cotinine were not unequivocally detrimental to sperm migration according to an investigation. More concentrated levels of nicotine nonetheless significantly reduced spermatic fluid motility and healthy characteristics (Oyeyipo *et al.*, 2014). In another findings, it has been observed at higher concentrations to lower the proportion of active spermatozoa and induce sperm death, leading to disintegrated genome and changing the chromatin solidity (Condorelli *et al.*, 2013).

Detected in smoke, benzo(a)pyrene has also been demonstrated to reduce male fertility. Benzo(a)pyrene lowered the extent of acrosome halo development according to a research. The aforementioned result implies that this substance might be mostly responsible for genetic injury among smokers (Mukhopadhyay *et al.*, 2010).

In mouse male gonad, persistent cigarette fume has been shown to cause programmed cell death, which concurrently reduces the

germ cells, Leydig cells, and Sertoli cells count (La Maestra *et al.*, 2015). Furthermore, research concentrating on middle-aged men revealed that compared to non-smoking men, a greater proportion of active smokers suffered from erectile dysfunction (Scarneciu *et al.*, 2014).

9. Excessive Consumption of Alcohol

Used for ceremony and leisure since ancient times, alcohol is often misused. Habitual consumption of alcohol can raise prolactin, so weakening the control of the HPG pathway; this can lead to hypogonadism, minimized formulation of sperm, sexual dysfunction, and male breast enlargement (Rachdaoui and Sarkar, 2017; Finelli *et al.*, 2020). Alcohol abuse is linked to higher estrogen levels and lower testosterone levels (Ilacqua *et al.*, 2018; Mathur *et al.*, 2019). Alcohol intake is linked to impaired fertility; it is a recognized embryo toxicant (Oremosu and Akang, 2015; Mathur *et al.*, 2019). Reviewing information in eighteen cross-sectional studies, a meta-analysis established that regular alcohol intake lowers semen statutory characteristics (Ricci *et al.*, 2016).

10. Recreational Narcotics

Use of marijuana has been connected with improper regulation of the HPG axis yielding gonadal deficiency accompanied by low LH without modifying FSH (du Plessis *et al.*, 2015). By binding with endocannabinoid receptors, cannabis also directly influences the activity of seminal fluid and sperm characteristics (Amoako *et al.*, 2013). Furthermore, suppressing the sperm capacitance process during penetration and impregnation is the activation of cannabinoids 1 receptors (Krzastek *et al.*, 2020). Misuse of opiates and opioids could compromise seminal fluid production via blocking the production and action of male hormones. Lower sperm motility and viability were identified in a group of twenty-five males hooked to heroin when compared to an equal number of healthy men (Nazmara *et al.*, 2019).

Laboratory Investigations of Male Reproductive Toxicity

To assess the impact of environmental factors on male reproductive health, specific laboratory tests may be conducted:

Biological Monitoring: Measuring the levels of specific chemicals or their metabolites in blood, urine, or other biological samples can provide evidence of exposure.

Genetic Toxicology Tests: Evaluating DNA damage in sperm cells can help identify the potential genetic impact of environmental exposures.

Oxidative Stress Markers: Measuring markers of oxidative stress in semen can help assess the extent of cellular damage caused by environmental factors.

Hormone Profiling: Assessing hormone levels, including testosterone, FSH, and LH, can help identify hormonal imbalances caused by environmental exposures.

Semen Analysis: Semen analysis (S/A) is a series of investigations assessing the performance of male gonads. The seminal fluid analysis should be done in laboratories according to WHO methodology (WHO, 2021), computer assisted seminal analysis can also be used. Computer-assisted histological image analysis including Sertoli and Leydig cells use (Sikka and Hellstrom, 2016). Biochemical tests comprise evaluation of zinc, fructose, citric acid in seminal fluid, reproductive hormonal assays measurements in body fluids as required (Concepción-Zavaleta *et al.*, 2022).

Oxidative Stress Markers: Measuring markers of oxidative stress in semen can help assess the extent of cellular damage caused by environmental factors.

Treatments and Prevention of Damage Caused by Environmental Toxins

Minimizing the detrimental consequences of ecological toxins mostly depends on avoiding or reducing exposure. Abolishing all feasible exposures would be quite challenging considering the great variety of available environmental contaminants (Krzastek *et al.*, 2020). Fascinatingly, unique silicone wristbands have been created to identify a range of toxic chemicals, therefore giving individuals hitherto unheard-of information about their individual predisposed account (Wang *et al.*, 2019).

In animal models, antioxidants have been found to have safeguarding and ameliorative effects on seminal fluid production, in human studies, they have been shown to lower reactive oxygen species

(ROS) and genetic disintegration (Asadi *et al.*, 2017; Krzastek *et al.*, 2020). Antioxidant qualities of melatonin, N-acetylcysteine, and green tea help protect cells from RF-EMF-induced damage (Roychoudhury *et al.*, 2017; Kesari *et al.*, 2018).

What should be done: The increasing prevalence of male infertility and reproductive disorders underscores the urgent need to address the detrimental effects of environmental toxicants. A growing body of evidence suggests that exposure to various environmental chemicals, including pesticides, heavy metals, and air pollutants, can significantly impair male reproductive health. These toxicants can disrupt hormonal balance, damage DNA, and induce oxidative stress, leading to reduced sperm quality, decreased sperm count, and impaired testicular function. The consequences of these effects extend beyond individual health, impacting fertility rates and potentially contributing to population decline.

To mitigate the adverse effects of environmental toxicants on male reproductive health, a multifaceted approach is necessary. Stricter regulations on the use and disposal of hazardous chemicals are essential to minimize human exposure. Educating the public about the risks associated with environmental toxins and promoting healthy lifestyle choices can empower individuals to reduce their exposure. Continued research is needed to identify emerging environmental threats, understand their mechanisms of action, and develop effective prevention and treatment strategies.

Implementing workplace safety measures and regular health monitoring can protect workers from occupational exposures to harmful chemicals.

Conclusion

By taking proactive steps to address environmental pollution and promote healthy lifestyles, male reproductive health can be safeguarded and ensure the well-being of future generations. Unconstrained mechanization and gentrification without appropriate regulations of environmental toxicants removal, and enforcement of preventive measures are the likely factors that have predisposed males to possibility of infertility, Nigerian men are not exempted. Monitoring and management of these environmental toxins could therefore be medically beneficial.

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