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Impacts of Selected Heavy Metals and Pesticides on Sperm Parameters Among Men Being Evaluated for Fertility in A Tertiary Hospital in South-West Nigeria

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Abstract

Background: Research indicates a worldwide decrease in male fertility with limitations in seminal fluid analysis (SFA), focus must shift to identifying underlying causes, including environmental toxins. Assessing seminal pesticide residues and heavy metal levels, as well as their influence on antioxidant status and sperm quality, provides a clear understanding of how environmental factors affect male fertility. Aim and Objective: To evaluate the influence of seminal concentrations of specific pesticides (Malathion, DDT, Permethrin, and Carbamate) and heavy metals (Pb, Cd, and Fe) on semen analysis parameters and antioxidant markers (vitamins C and E, glutathione peroxidase, and catalase) in men exhibiting abnormal compared to normal sperm profiles. Methods and Materials: Semen samples were obtained from 130 men each with abnormal sperm (study group) and healthy sperm (control group) following a minimum of three days of sexual abstinence. Samples were collected through either coitus interruptus or masturbation. After liquefaction and centrifugation, supernatants were digested and analyzed: pesticide residues by GC-MS; Pb, Cd, and Fe by Atomic Absorption Spectrometry; Vitamins C and E by spectrophotometry; and GPx and CAT by ELISA.
Result: Subjects had significantly elevated seminal concentrations of cadmium, lead, and iron compared to the control group ($p<0.001$). Pesticide levels were also higher: Malathion, DDT, Permethrin, and Carbamate ($p<0.001$). Conversely, antioxidants were lower in subjects: Vitamin C, Vitamin E, GPx and CAT ($p<0.001$). Seminal levels of Pb, Cd, and Fe showed significant negative correlations with sperm count and motility. Similar negative correlations were observed for pesticide residues, except Carbamate. Antioxidant levels were positively correlated with count and motility. None of the evaluated variables showed a significant correlation with sperm morphology. Conclusion: Quantifying seminal levels of pesticide residues and heavy metals may aid in evaluating male infertility–especially idiopathic cases–where current tools fall short, offering clinical value for both management and prevention of sperm quality decline.

Keywords

Heavy metals toxicity, Infertility, Male etiology, Pesticides toxicity, Semen Analysis

Article history

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1. Introduction

Over the past five decades, a growing body of evidence has documented a global decline in male fertility, marked by significant reductions in sperm count, motility, and morphology. This trend has emerged as a major public health concern; particularly as male factors are implicated in nearly half of all infertility cases worldwide. While multiple etiologies contribute to this decline, environmental exposures–especially those arising from industrialization–have garnered increasing attention. Heavy metals, pesticides, and other industrial pollutants are known to disrupt hormonal regulation, impair spermatogenesis, and trigger oxidative stress pathways, ultimately undermining sperm quality and function. These mechanisms highlight the urgent need to understand male infertility not merely as a clinical issue, but as one shaped by environmental, occupational, and socio-demographic determinants [1].

In countries undergoing rapid industrial and urban expansion, such as Nigeria, this intersection of environment and health becomes particularly salient. Economic growth in sectors like petrochemicals and telecommunications has undoubtedly generated employment and infrastructure gains [2], yet it has also introduced heightened exposure to toxicants with known reproductive effects. Urbanization compounds this risk: large-scale rural-to-urban migration often leads to overcrowded, poorly planned settlements where access to clean air, water, and sanitation is limited. These Peri-urban zones, typically characterized by informal housing and inadequate waste management systems, serve as concentrated points of environmental stress, with consequences that extend into population health–including male reproductive outcomes [3-5].

Moreover, the cumulative health burden of urban living is not confined to toxic exposures alone. Urban environments are increasingly linked to poor nutrition, chronic psychosocial stress, sedentary lifestyles, and limited access to quality healthcare–all of which intersect with reproductive health in complex ways. As cities continue to swell–projected to house 6.3 billion people, or 67% of the global population, in the near future–the scale of these challenges grows correspondingly [6,7]. Urbanization, though often celebrated as a sign of socio-economic advancement, is producing a new epidemiological landscape: one marked by environmental degradation, shifting disease patterns, and under-recognized fertility issues. Addressing male infertility within this context demands an integrated, multi-sectoral response–one that acknowledges the ecological determinants of health and prioritizes sustainability alongside development.

Urbanization exerts a detrimental impact on the nutritional health of economically disadvantaged populations. Financial constraints and elevated food prices often result in inadequate diets, leading to illness and impaired nutrient absorption. Environmental pollution–especially from unsafe street food–further exacerbates undernutrition and contributes to foodborne illnesses such as botulism and salmonellosis [6]. Urban populations today face a complex mix of health challenges driven by rapid development and environmental degradation. One emerging concern is the increasing prevalence of over-nutrition and obesity among both middle- and lower-income groups, which is fueling a surge in chronic non-communicable diseases and presenting a significant public health concern [8]. At the same time, air pollution remains a major health threat, responsible for an estimated 6.5 million deaths each year–90% of which occur in developing countries [9]. Pollutants, especially carbon dioxide emissions, contribute to a broad spectrum of health issues including respiratory diseases, cardiovascular conditions, and various forms of cancer, affecting individuals across all socioeconomic strata.

In sub-Saharan Africa, these environmental and lifestyle shifts have been accompanied by a growing incidence of non-communicable diseases, including male infertility. Increasing evidence links this trend to widespread exposure to environmental toxins. Pesticides such as DDT and malathion, along with heavy metals like mercury, lead, and cadmium, are commonly found in household products and agricultural environments. These substances can contaminate food and water sources, contributing to long-term health complications, including reproductive health disorders. [3].

Chronic exposure can elevate blood toxin levels, leading to serious health issues, requiring specific investigations to link symptoms to these toxins [4]. Antioxidant systems in the body neutralize toxins, but excessive exposure overwhelms these defenses, causing cellular damage, including gonadotoxicity [5]. While previous studies on male fertility and environmental toxins have largely relied on self-reported data through questionnaires, there is a paucity of data that directly measures seminal levels of pesticide residues, heavy metals, and antioxidants. This study aims to fill this gap by objectively assessing these biochemical factors and comparing them with seminal fluid analysis parameters in both infertile subjects and fertile controls.

Around 7% of males globally experience infertility, with 40% linked to male factors and 20% still classified as idiopathic [6,7]. Environmental toxins, including heavy metals and pesticides, are implicated in infertility [8]. Annually, 4.6 million tons of pesticides are released, with developing countries facing 99% of pesticide-related deaths due to poor regulation [9].

Identifying a direct link between these toxins and male infertility could help address the global decline in male fertility. Previous studies have linked environmental toxins and heavy chemicals to infertility through questionnaires on occupational hazards, with few quantifying seminal concentrations of these chemicals [8,9]. Fewer still have assessed the combined effects of different toxic chemicals on antioxidant levels and sperm parameters. The data from this study

will aid regulators in setting limits on acceptable chemical quantities in both imported and locally manufactured products, and potentially banning items that pose health risks to the public.

This study explored the relationship between environmental toxins (pesticide residues, heavy metals) and seminal antioxidant status, as well as their impact on Semen Fluid Analysis (SFA) parameters in infertile males. The null hypothesis assumes no link between environmental toxins and quality of semen, whereas the alternative hypothesis proposes a significant relationship. The specific objectives included quantifying seminal concentrations of selected pesticides (Malathion, DDT, Carbamates, and Permethrin) and heavy metals (Cadmium, Iron and Lead), along with antioxidant markers such as glutathione peroxidase, vitamin E, catalase and vitamin C. Furthermore, the study evaluated sperm count, volume, and motility in both infertile men and normospermic controls.

2. Materials and Methods

This study was carried out at the Federal Medical Centre, Abeokuta, located in Ogun State, Nigeria–a referral institution serving neighboring states. The research was conducted within the Department of Chemical Pathology and Immunology, while biochemical analyses were performed at the Central Research Laboratory in Ilorin, Kwara State. The hospital's General Outpatient Department (GOPD) and specialist clinics manage infertility referrals, processing approximately 500 seminal fluid analysis (SFA) samples annually. The study population consisted of men in relationships with infertility issues who were referred for seminal fluid analysis (SFA), with participants categorized as subjects (those with abnormal SFA parameters) and controls (Those with standard seminal fluid analysis parameters). A cross-sectional analytical study design was employed, and participants were selected using simple random sampling. The principal aim of the study was to assess the influence of environmental toxicants on male reproductive function and fertility outcomes by quantifying seminal pesticide residues, heavy metal concentrations, and antioxidant levels.

2.1 Sample Size Determination

The sample size for the study was determined using the Fisher formula [10]:

Given as:

n = The required minimum sample size.

z = The standard normal deviation is typically set at 1.96, which represents a 95% confidence interval.

p = The prevalence of male infertility in the target population, as estimated in the previous study, was 8.45% [11].

q = The proportion of the target population that lacks a particular characteristic, i.e.,

q = 1 – p = 1 – 0.0845 = 0.9155,

d = A tolerable margin of error, where an observed difference of 5% is considered statistically significant.

Hence, n=

An attrition rate of 10% was assumed (approximately 12 participants). Therefore, the minimum sample size required was 130.

One hundred and thirty (130) individuals whose semen parameters align with WHO's normal standards guidelines, were used as the control group.

2.2 Subject Selection

Research subjects were voluntary male participants directed from the Gynaecology, Urology, and General Out-Patient Department (GOPD) clinics to the Medical Microbiology laboratory for seminal fluid analysis (SFA). Males exhibiting at least one abnormal semen parameter were enrolled as subjects, whereas those with normal semen profiles were included as controls. Normal SFA parameters were based on the current WHO criteria [12]. Informed consent was obtained prior to enrolling participants in the study. Recruitment continued employing a random sampling method to ensure each individual in the target population had an equal chance of being selected until the desired sample size was reached. The study included male partners of infertile couples who gave consent. Participants with one or more abnormal semen parameters were categorized as cases, whereas those with normal semen profiles were considered controls. Excluded were males with cryptorchidism, atrophic testes, hypogonadism, on medications, azoospermia, aspermia, or those on antioxidant therapy. Participants were informed about the study, and ethical approval was obtained from the Federal Medical Centre, Abeokuta's IRB: NHREC/08/10-2015; Protocol Number: FMCA/470/HRE/10.

2.3 Collection and Processing of Samples

Semen specimens were obtained in sterile containers through either masturbation or coitus interruptus, following a minimum of 72 hours of sexual abstinence. The specimens were delivered to the laboratory within one hour of collection, kept at body temperature during transport, and then left to liquefy at room temperature for 45 minutes.

Seminal fluid analysis (SFA) was conducted under the supervision of a Consultant Medical Microbiologist. Following analysis, samples were centrifuged at 1000 × g for 20 minutes, and the resulting supernatants were stored at –30°C for subsequent determination of pesticide residues and heavy metal concentrations.

2.4 Biochemical Analysis

Lead, Cadmium, and Iron Analysis [13]: The levels of Pb, Cd, and Fe in the semen were measured using Flame Atomic Absorption Spectrophotometry (AAS) at their characteristic wavelengths. Quality control measures included blank checks, replicate samples, and the use of ultra-pure water to flush the instrument.

Pesticide Residues Analysis: Pesticide residues [14] (Malathion, DDT, Permethrin, and Carbamates) were quantified using Gas Chromatography/Mass Spectrometry (GC/MS), which isolates and characterizes compounds by their mass spectra.

Glutathione Peroxidase Assay [15]: Human GPX1 levels were measured using an ELISA kit, which utilizes a sandwich-ELISA principle to quantify GPX1 by measuring optical density at 450 nm. Assay Procedures:

Glutathione Peroxidase (GPX1): 100 µL of standard/sample was added to wells and incubated at 37°C for 90 minutes. Afterwards, 100 µL Biotinylated Detection Antibody was added, incubated for 60 minutes, and the plate was washed. HRP conjugate was then added, incubated for 30 minutes, and washed again. Substrate Reagent was added and incubated for 15 minutes subsequently, a Stop Solution was added, and the plate was then read at 450 nm.

Catalase (CAT) [16]: The sample and reagent were mixed and incubated at 37°C for 5 minutes.

Reagent 2 was added and incubated for 1 minute. Reagents 3 and 4 were added, mixed, and incubated for 10 minutes at room temperature. Optical Density (OD) was measured at 405 nm.

Vitamin C (Ascorbic Acid) [17]: 100 µL of the sample was mixed with reagent, incubated at room temperature, and centrifuged. A standard curve of vitamin C concentrations was prepared. The samples were introduced into the wells, followed by the addition of reagents, then incubated at 37°C for 30 minutes and measured at 536 nm.

Vitamin E assay [18]: involves preparing a standard curve by diluting a 100 µg/mL standard solution with ethanol to create serial concentrations (0–40 µg/mL). Samples are prepared in three tubes: standard tube (0.15 mL water + 0.3 mL standard solution), sample tube (0.15 mL seminal plasma + 0.3 mL ethanol), and blank tube (0.15 mL reagent + 0.3 mL ethanol), mixed with a vortex for 20 seconds. Subsequently, 0.5 mL of N-heptane was added to each tube, mixed using a vortex for 1 minute, and centrifuged at 3100g for 10 minutes; 0.2 mL of the upper layer is taken for the chromogenic reaction. In the chromogenic step, 200 µL N-heptane extract, 25 µL reagent 1, and 15 µL reagent 2 are added, mixed, and incubated for 5 minutes. After adding 15 µL reagent 3, the solution is mixed, 250 µL ethanol is added, and incubated for 2 minutes. The OD is measured at 533 nm to determine Vitamin E content.

Data analysis was performed using SPSS version 20. Normally distributed variables were expressed as mean ± standard deviation (SD), while non-normally distributed variables were presented as median and interquartile range (IQR). ANOVA was used for comparisons, and the relationships between variables were analyzed using Pearson's correlation coefficient. A p-value less than 0.05 was considered statistically significant.

3. Results

Table 1 displays the socio-demographic profiles of the study participants, the majority within the 41–50-year age range and predominantly urban-dwelling civil servants. Among the subjects, 43.8% reported alcohol consumption, compared to 42.3% of the control group. Similarly, cigarette smoking was reported by 31.5% of subjects and 45.4% of controls. Table 2.

Table 1. Demographic profile of participants.

Variable	Frequency (%)	
	Control	Subject
Age (years)		
21 – 30	29 (22.3)	34 (26.2)
31 – 40	40 (30.8)	42 (32.3)
41 – 50	52 (40.0)	49 (37.7)
>50	9 (6.9)	5 (3.8)
Occupation		
Artisan	34 (26.2)	33 (25.4)
Civil servant	54 (41.5)	38 (29.2)
Farmer	29 (22.3)	38 (29.2)
Student	9 (6.9)	20 (15.4)
Trading	4 (3.1)	1 (0.8)
Settlement		
Rural	53 (40.8)	51 (39.2)
Urban	77 (59.2)	79 (60.8)

Table 2. Social history of research participants.

Variable	Frequency (%)	
	Control	Subject
Alcohol consumption		
Yes	55 (42.3)	57 (43.8)
No	75 (57.7)	73 (56.2)
Cigarette smoking		
Yes	59 (45.4)	41 (31.5)
No	71 (54.6)	89 (68.5)

As seen in Table 3, the control group exhibited significantly higher spermatozoa counts and progressive motility compared to the study participants. Conversely, the subjects demonstrated an increased average proportion of non-motile sperm cells relative to those of controls. However, statistical analysis showed no considerable difference in the mean percentage of sperm with standard morphology between the two groups.

Table 3. Assessment of semen analysis parameters.

Variable	Mean ± SD		T	*p* value
	Control	Subject		
Sperm count (x10^6/ml)	65.38 ± 26.28	10.65 ± 7.87	22.748	<0.001
Active motility (%)	49.91 ± 12.98	21.97 ± 12.66	17.575	<0.001
Immotile (%)	21.77 ± 13.18	63.66 ± 15.24	-23.707	<0.001
Normal morphology %	72.69 ± 18.83	80.53 ± 82.95	-1.051	0.294

P≤0.05 is statistically significant

Mean seminal levels of Lead, Cadmium, and Iron were significantly elevated in subjects compared to controls. Table 4.

Table 4. Assessment of mean levels of heavy metals in semen.

Variable	Mean ± SD		T	*p* value
	Control	Subject		
Cadmium (mg/L)	0.004 ± 0.002	0.012 ± 0.010	-7.525	<0.001
Lead (mg/L)	0.222 ± 0.087	0.432 ± 0.105	-17.419	<0.001
Iron (mg/L)	2.11 ± 0.75	3.85 ± 1.15	-14.488	<0.001

P≤ 0.05 is statistically significant

As illustrated in Table 5, the antioxidant levels—indicated by mean seminal concentrations of vitamin C, vitamin E, glutathione peroxidase, and catalase—were substantially greater in the control group than in the study subjects.

Table 5. Comparison of means of seminal antioxidants (log transformation).

Variable	Mean ± SD		T	*p* value
	Control	Subject		
Vitamin C (mg/100ml)	54.55 ± 2.78	21.74 ± 2.30	7.165	<0.001
Vitamin E (mg/100ml)	14.73 ± 1.62	5.62 ± 2.17	12.020	<0.001
Glutathione peroxidase (IU/L)	136.14 ± 2.79	74.28 ± 2.63	4.895	<0.001
Catalase (IU/L)	185.57 ± 1.24	102.28 ± 1.44	16.020	<0.001

As presented in Table 6, the mean seminal concentrations of the analyzed pesticide residues—Malathion, Permethrin, and DDT—were significantly higher in the study subjects compared to the control group.

Table 6. Comparison of means of seminal pesticide residue (log-transformed).

Variable	Mean ± SD (µg/ml)		T	*p* value
	Control	Subject		
Malathion	3.54 ± 1.81	13.57 ± 2.68	-12.844	<0.001
DDT	13.65 ± 1.75	24.03 ± 1.93	-7.467	<0.001
Permethrin	3.82 ± 1.78	28.99 ± 3.87	-14.675	<0.001
Carbamate	17.746 ± 3.90	35.75 ± 1.92	-5.291	0.001

As shown in Tables 7, 8, and 9, significant moderate negative correlations were observed between sperm count and seminal concentrations of both heavy metals.

Table 7. Correlation of heavy metals with sperm count.

Variable	R	*p* value
Cadmium (mg/L)	-0.334	<0.001
Lead (mg/L)	-0.573	<0.001
Iron (mg/L)	-0.565	<0.001

r: Pearson correlation coefficient

Table 8. Correlation of heavy metals with active motility.

Variable	R	*p* value
Cadmium (mg/L)	-0.357	<0.001
Lead (mg/L)	-0.519	<0.001
Iron (mg/L)	-0.519	<0.001

r: Pearson correlation coefficient

Table 9. Correlation of heavy metals with sperm morphology.

Variable	R	*p* value
Cadmium (mg/L)	-0.089	0.153
Lead (mg/L)	-0.045	0.474
Iron (mg/L)	-0.083	0.181

r: Pearson correlation coefficient

Pesticide residues were negatively correlated with sperm count and motility, except Carbamates. None of the pesticide residues correlated with sperm morphology, significantly, as shown in Tables 10, 11 and 12.

Table 10. Correlation of pesticide residues with sperm count.

Variable	R	*p* value
Malathion (µg/ml)	-0.448	<0.001
DDT (µg/ml)	-0.378	<0.001
Permethrin (µg/ml)	-0.344	<0.001
Carbamate (µg/ml)	-0.075	0.225

r: Pearson correlation coefficient

Table 11. Correlation of pesticide residues with active motility.

Variable	R	*p* value
Malathion (µg/ml)	-0.445	<0.001
DDT (µg/ml)	-0.324	<0.001
Permethrin (µg/ml)	-0.344	<0.001
Carbamate (µg/ml)	-0.085	0.174

r: Pearson correlation coefficient

Table12. Correlation of pesticide residues with sperm morphology.

Variable	R	*p* value
Malathion	-0.043	0.489
DDT (µg/ml)	0.043	0.495
Permethrin	-0.021	0.738
Carbamate	-0.006	0.928

r: Pearson correlation coefficient

Seminal anti-oxidant status levels (Vitamins C and E, Glutathione peroxidase and Catalase) were all positively correlated with sperm count, and sperm motility but not sperm morphology Tables 13, 14 and 15.

Table 13. Correlation of sperm count with antioxidant status.

Variable	R	*p* value
Vitamin C (mg/100ml)	0.408	<0.001
Vitamin E (mg/100ml)	0.496	<0.001
Glutathione peroxidase (IU/L)	0.262	<0.001
Catalase (IU/L)	0.586	<0.001

r: Pearson correlation coefficient

Table14. Correlation of seminal antioxidants with sperm active motility.

Variable	R	*p* value
Vitamin C (mg/100ml)	0.315	<0.001
Vitamin E (mg/100ml)	0.419	<0.001
Glutathione peroxidase (IU/L)	0.205	0.001
Catalase (IU/L)	0.531	<0.001

r: Pearson correlation coefficient

Table15. Correlation of seminal anti-oxidants status with sperm morphology.

Variable	R	*p* value
Vitamin C (mg/100ml)	0.066	0.289
Vitamin E (mg/100ml)	0.013	0.829
Glutathione peroxidase (IU/L)	0.028	0.657
Catalase (IU/L)	-0.061	0.323

r: Pearson correlation coefficient

4. Discussion

The socio-demographic profile of the research participants indicated that the majority were residents of Abeokuta, an urban area primarily inhabited by civil servants and artisans, with surrounding rural villages populated by farmers. All participants, regardless of their urban or rural status, sought secondary and tertiary healthcare services at the study site.

Sperm Count, Motility, and Morphology: The study revealed that the control group had significantly higher sperm count and proportion of actively motile sperm compared to the study subjects. However, no statistically significant differences in the normal morphology of sperm were observed between the two groups. These findings are consistent with the inclusion criteria and align with WHO guidelines, which emphasize that sperm motility and count are more significant indicators of fertility than sperm morphology, with a 5% normal morphology threshold considered adequate for fertility.

Heavy Metal Exposure and Sperm Parameters: A significant increase in seminal cadmium concentration was observed in the study subjects compared to the controls (0.012 ± 0.010 mg/L vs. 0.004 ± 0.002 mg/L, $p \leq 0.001$), which is consistent with findings from previous studies involving infertile males [19-22]. The concentration of cadmium showed significant inverse correlations with sperm count ($r = -0.334$, $p < 0.001$) and motility ($r = -0.357$, $p < 0.001$). Cadmium exposure has been associated with reduced testicular size, impaired testosterone synthesis, disrupted spermatogenesis, and testicular tissue damage through the generation of reactive oxygen species (ROS) and depletion of glutathione stores, leading to DNA and lipid peroxidation [23,24]. Additionally, cadmium has been linked to damage in testicular vessels, the blood-testis barrier (BTB), inflammation, and apoptosis [25,26].

Similarly, seminal lead concentration was significantly higher in the study subjects compared to controls (0.432 ± 0.105 mg/L vs. 0.222 ± 0.087 mg/L, $p < 0.001$), with negative correlations observed between lead concentration and sperm count ($r = -0.573$, $p < 0.001$) and motility ($r = -0.519$, $p < 0.001$). This finding is consistent with previous studies on lead exposure in both humans and mammals [27-29]. Lead exposure, like cadmium, increases ROS production, depletes antioxidant defenses, and disrupts sperm function through apoptosis [20,30]. Lead binds to –SH groups in proteins, leading to glutathione depletion and increased susceptibility to ROS-induced damage.

Seminal iron concentration was also significantly higher in the study subjects compared to the controls (3.85 ± 1.15 mg/L vs. 2.11 ± 0.75 mg/L, $p < 0.001$). Similar to cadmium and lead, iron concentration showed significant negative correlations with sperm count ($r = -0.565$, $p < 0.001$) and motility ($r = -0.519$, $p < 0.001$), consistent with previous findings [31,32]. Iron-induced sperm damage occurs through the generation of ROS, which leads to lipid peroxidation

and oxidative damage in sperm cells [32,33]. At low iron concentrations, iron-binding proteins such as lactoferrin on sperm membranes function to prevent ROS generation [34].

Pesticide Exposure and Sperm Parameters: The study also found higher levels of pesticide residues, including Malathion, DDT, Permethrin, and Carbamate, in the subjects compared to controls. These pesticide residues showed significant negative correlations with sperm count and motility, though no correlation was observed with sperm morphology, except for carbamate levels, which did not correlate with any sperm parameters. These findings align with previous research, which has linked pesticide exposure to a range of detrimental effects on male fertility, including seminiferous tubule degeneration, spermatogenesis inhibition, and alterations in reproductive hormone levels [34-37].

DDT, a well-known endocrine-disrupting chemical, interferes with androgenic and estrogenic receptors and has been shown to cause long-term male reproductive health issues [33,38]. While its use in malaria control programs is being reduced, its persistent presence in malaria-endemic regions, including Nigeria, remains a concern [39,40]. Chronic DDT exposure has been linked to sperm DNA fragmentation, likely due to disturbances in the programmed cell death pathways and malfunctions during spermiogenesis [41].

Malathion, another commonly used pesticide, shares a similar toxic profile with DDT. Despite most studies on Malathion being conducted in rodents, the current study's findings align with the limited human research available on its impact on male fertility [42,43]. The generation of ROS and disruptions to cell membranes due to Malathion's lipophilic properties contribute to its toxic effects on sperm.

Permethrin, while considered less toxic than other pesticides, showed significant negative correlations with sperm count and motility, consistent with previous studies [46,47]. However, some studies have reported no association between Permethrin exposure and sperm parameter alterations, which may be attributed to geographic differences, exposure levels, or varying methodologies, including the measurement of urinary metabolites instead of seminal fluid [48-50].

Seminal carbamate levels were elevated in the study subjects relative to controls, but no significant correlation with sperm count, motility, or morphology was observed. These discrepancies may be due to differences in geographic location, genetic variations, or study design [51,52].

Antioxidants and Sperm Quality: The seminal antioxidant system, comprising both enzymatic components (Glutathione Peroxidase and Catalase) and non-enzymatic components (Vitamins C and E), plays a crucial role in mitigating the harmful effects of oxidative stress induced by environmental toxins. In this study, the control group exhibited higher levels of Vitamins C and E, which were positively correlated with sperm concentration and motility, consistent with findings from previous studies [53-57]. Vitamins C and E scavenge ROS, preventing oxidative damage and maintaining a balance between beneficial oxidants and harmful stress. Supplementation with these vitamins has been shown to improve sperm quality and assist in the treatment of conditions like oligospermia and asthenozoospermia. Similarly, Glutathione Peroxidase and Catalase levels were significantly higher in the control group, positively correlating with sperm quality. This finding aligns with several studies that highlight the protective role of these antioxidants against oxidative damage in sperm cells [58,59]. However, a study with differing results observed a negative relationship between Glutathione Peroxidase levels and sperm motility and structural integrity, suggesting that the antioxidants in this study may not have been sufficient to counteract the overwhelming effects of oxidative stress [60].

5. Conclusion

This study assessed the impacts of two major products of industrialization and urbanisation and established sources of free radical generation i.e. Heavy metals and Pesticides, on sperm quality, in a non-occupational setting. We found that higher seminal concentrations of these chemicals correlate with deterioration of sperm quality in most cases, except for sperm morphology. Both seminal enzyme-based (Glutathione Peroxidase, Catalase) and non-enzyme-based antioxidants (Vitamins C and E) are confirmed to correlate with optimal sperm parameter qualities, reaffirming their role in the enhancement of male fertility.

Assessing seminal levels of environmental toxins in the laboratory may play a clinically significant role in evaluating infertile men, especially those with idiopathic infertility, and may be important for both managing and preventing the decline in sperm quality. Moreover, preventive oral antioxidant supplementation shows considerable potential in the management of male infertility. More studies will be required to further elucidate the roles of environmental toxins in aetiopathogenesis of male infertility and utility of anti-oxidants in its treatment.

6. Limitation of Study

Ideally biomarkers of oxidative damage like seminal malonlydialdehyde (MDA) and 8-hydroxydeoxy-guanosine should have been quantified along with the anti-oxidants, to elucidate the aetiopathogenesis of environmental toxins in male infertility. Also the effects of environmental toxins on sperm morphology needs future studies for further elucidation.

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