



## Non-Thermal Effect of the Brand and Generic Mobile Phones Radiofrequency Radiation on the Antioxidant and Histology of the Epididymis after Prolonged Whole-Body Radiation on Sprague Dawley Rat

Oyedele Oyewumi Ajayi<sup>1,2</sup>, Johnson Orié Okoro<sup>3</sup>, Daniel Ugbomoiko<sup>1</sup>, Obhioze Augustine Akpoka<sup>4,5</sup>, Theophilus Ogie Eraméh<sup>1</sup>,  
Efosa Bolaji Odigie<sup>3</sup>

<sup>1</sup>Department of Medical Laboratory Science, Igbinedion University, Okada, Edo State, Nigeria. <sup>2</sup>Department of Pathology, University of Uyo, Uyo, Akwa Ibom State, Nigeria. <sup>3</sup>Department of Medical Laboratory Science, University of Benin, Benin City, Edo state, Nigeria. <sup>4</sup>Department of Biological Science, Igbinedion University, Okada, Nigeria. <sup>5</sup>Igbinedion University Teaching Hospital, School of Nursing, Okada, Nigeria  
\*Email: oyedele.ajayi@iuokada.edu.ng. DOI: 10.31964/mltj.v8i2.471

**Abstract:** Mobile phone usage has significantly increased over time. There is also the proliferation of models by different manufacturers, some of which are known to produce popular brands, while other manufacturers of unknown brands have been identified. Health conditions such as glioma, schwannoma, and some degenerative tissue conditions have been reported by different researchers on short and prolonged exposure. The IARC report on radiofrequency radiation as a human "possible carcinogen, group 2B" has also prompted much research on the effect of mobile phone radiofrequency radiation on humans. The mobile phone placement in the pocket, which is close to the male reproductive organ, and the possible radiation emission levels from different models by manufacturers prompted this study. The goal was to investigate the histological alterations, spermatid epididymal contents, and changes in oxidative stress indicators that may occur following prolonged exposure to mobile phone radiofrequency radiation. Forty (40) male Sprague Dawley rats were used for this study. They were equally grouped into eight groups, two (2) control groups and six (6) groups that were exposed to six mobile phone models. The 8 hours of daily exposure for six months was done with phones in active mode. The finding showed a significant reduction of the mature sperms in the lumen of the epididymis. There was focal epithelial hyperplasia in two groups exposed to branded models. No degenerative epithelium was observed in the epididymis. There was also a significant ( $p < 0.05$ ) reduction in the motility and sperm count of the exposed groups. The superoxide dismutase (SOD) was significantly ( $p < 0.05$ ) depleted across the exposed groups. There are isolated changes that result from individual phone models, but changes cannot be dichotomized into changes that result from branded or generic phones. By adequately positioning phones or turning them off when not in use, people can reduce exposure to RF radiation.

**Keywords:** Antioxidant; epididymis; histological; mobile phone; radiation; radiofrequency.

### INTRODUCTION

Mobile phones are among devices that emit radiofrequency electromagnetic waves (RF-EMW) to execute their communication function. These waves carry signals

**Corresponding Author:** Oyedele Oyewumi Ajayi

Department of Medical Laboratory Science, Igbinedion University, Okada, Edo State, Nigeria.

Email : oyedele.ajayi@iuokada.edu.ng

from cell phones to base stations and antennas. The frequency of such waves is low, ranging between 800 - 22450 MHz. However, humans are still at risk since the body can act as an antenna, absorbing these waves and converting them into eddy currents (Oyewopo et al., 2017; Chaithanya et al., 2017; Agarwal, 2011). The electromagnetic fields (EMFs) that lack the energy to induce ionization directly are referred to as non-ionizing radiation. RF-EMFs of higher intensity produce tissue heating a basis for microwave oven operation (Belpomme et al., 2018). The US national toxicology programs released the findings on the prolonged exposure of laboratory rats to cell phone radiofrequency radiation-induced glioma. Some tumour, such as schwannoma and acoustic neuroma, has also been reported after intensive human exposure to mobile phone radiofrequency radiation (Wyde et al., 2016).

The resulting impact of the radiofrequency radiation interaction with the biological system has been defined as thermal and non-thermal effects. High-frequency electromagnetic wave has always been associated with heat generation due to the increased electrical conductivity of the tissues. This thermal mechanism could disrupt tissue cell functions and morphology (Deepinder et al., 2007). However, the effect of heat generated by the non-ionizing nature of radiofrequency radiation has caused institutions to consistently deprecate the health concern of radiofrequency radiation at intensities that do not cause tissue heating (WHO, 2014). The substantial evidence for human health risks from non-ionizing EMFs at intensities that do not generate detectable tissue heating, as outlined in the Bioinitiative report, could only be explained by non-thermal mechanisms (Bioinitiative Report, 2019).

Human exposure has been rapidly on the rise in recent years due to the proliferation of various models of mobile phones by manufacturers not approved by telecommunication agencies in some countries, particularly developing countries like Nigeria. An improper epididymal function has been linked to sperm DNA integrity and sperm parameters (Elbashir et al., 2020). The non-thermal mechanism is also said to induce free radicals and deplete antioxidant levels, which may cause tissue degeneration. Non-thermal nature of the ROS increased production resulting from radiofrequency radiation exposure has been previously reported. . Radiofrequency radiation intensity as low as 0.1 mW/cm<sup>2</sup> and absorbed SAR of 0.3 mW/Kg were shown to have induced significant oxidative stress in living cells (Burlaka et al., 2013; Oksay et al., 2014). Several studies performed on laboratory animals have shown unequivocally that non-thermal EMFs may result in oxidative damage (Esmekaya et al., 2011; Burlaka et al., 2013). Based on numerous animal experimental studies , short-term or long-term exposure to 900 MHz or 2.45 GHz RF-EMF has induced non-thermal effects in the brain (Sonmez et al., 2010; Megha et al., 2015; Shahin et al., 2017) through the induction of oxidative stress.

It has been reported that prolonged exposure to cell phones could cause adverse effects. However, the inclusion of the model and quality of mobile phone manufacturers has not been popular among researchers or considered a potential risk factor; hence this study aims to investigate the non-thermal effect of branded and generic mobile phone RF radiation on the testicular antioxidant concentrations and histomorphology of the epididymis' after prolonged whole body exposure of Sprague Dawley rats.

## MATERIALS AND METHODS

### Animals And Experimental Groups

A total of 40 Sprague Dawley rats were used. The rats were six weeks old with an average weight of 0.150 kg. Exposure duration was 8 hours/day for six months. The exposure time was between 10 am to 6 pm. The rats were kept in wooden cages on a 12-hour light/ dark cycle and were given free access to the rat's pelletized meal and good water. The rats were divided into eight groups, including two (2) control groups and six (6) exposed groups based on six different mobile phones used. The ethical committee approved the research on the use of animals, Department of Pharmacology, University of Uyo, Nigeria, in conformity with the National Institute of Health Guide for Care and Use of Laboratory Animals (Pub No. 85-23, revised 1985).

### Control Setup

Two (2) controls setup was done. In the negative control group (group 7), the rats were kept in the cage for six months. In the baseline control group (group 8), the rats were sacrificed after two weeks of acclimatization. The rats in the control groups were not exposed to mobile phones' radiofrequency radiation.

### Exposed Setup

Six (6) exposed setup was done in six separate wooden cages with five Sprague Dawley rats and one mobile phone per cage. The phone model used with the corresponding group number is shown in table 1 below. The phones were wrapped with aluminium wire mesh to prevent being damaged by the animal. The part upper of the aluminium wire mesh was attached to the top lid of the cage to stand the phone at the centre of the cage for whole-body exposure, with the farthest exposure distance being 27 cm. The phones were kept active to receive two text messages and social media notifications. An Android app (Moto Answer) was installed and enabled on each phone to automatically receive five (5) calls daily for at least 10 seconds. The exposure to emissions was done under the manufacturer's specified specific absorption rate (SAR) for six months at 8 hours per day. The phones' batteries were charged after daily exposures depending on the battery level. The cages were spaced at a distance of 2 meters from each other.

Table 1. Phone Model and Corresponding Group Number

Exposed groups	Class	SAR
1	Brd1	0.98
2	Gnr1	unknown
3	Gnr2	unknown
4	Brd2	1.67
5	Brd3	1.27
6	Brd4	1.67

### Necropsy and Sample Collection

No animal death was recorded at the end of the six (6) months experimental period. The epididymis was collected under chloroform inhalation anaesthesia. Part of the caudal epididymis was fixed in 10% buffered formalin for 48 hours, while part of the epididymis meant for the oxidative stress and antioxidant markers was preserved in phosphate buffered saline (PBS) and frozen, then transported in an ice pack to the analytical laboratory (Chemical Pathology Department, University of Uyo Teaching Hospital, Nigeria).

### **Preparation Of The Tissue Homogenates And Post Mitochondrial Supernatant**

The epididymis tissues (0.5 g) were homogenized with 0.1M phosphate buffer (pH 7.2). The resulting homogenates were centrifuged using Powerspin LX centrifuge (UNICO, Dayton, New Jersey) at 2500 rpm speed for 15 mins, were removed from the centrifuge, and the supernatants decanted and used for analysis.

### **Measurement of Lipid Peroxidation**

The amount of lipid peroxidation was measured by the quantity of malondialdehyde (MDA) in the testes. Tissue MDA was measured using the thiobarbituric acid reactive substance test, as described by Buege & Aust (1978). 1 mL of already prepared tissue homogenate was added to 2 ml of stock solution of 0.375% thiobarbituric acid, 15% w/v trichloroacetic acid and 0.24N of Hydrochloric acid. The reagent was heated in a boiling bath for 15 minutes and allowed to cool; the precipitate formed after cooling was removed by centrifugation at 3000 rpm for 10 mins. The obtained supernatant absorbance was determined using a UV recording spectrophotometer 2150 series (UNICO, Dayton, New Jersey) at 532 nm against a blank. MDA estimation was calculated using  $1.56 \cdot 10^5 \text{ mol}^{-1} \text{ cm}^{-1}$  as the molar absorbance coefficient for MDA TBA- complex.

### **Determination of the Activities of Antioxidant Enzymes (Superoxide Dismutase and Catalase)**

The superoxide dismutase activity in the epididymis tissue homogenates of the rats was assessed by its capacity to prevent the auto-oxidation of epinephrine measured by the rise in absorbance at 480 nm as stated by Sun and Zigma (1978). According to Sinha (1972), the Catalase activity was evaluated by measuring the reduction in absorbance at 620 nm owing to the decomposition of  $\text{H}_2\text{O}_2$  in a UV recording spectrophotometer 2150 series (UNICO, Dayton, New Jersey) at intervals of 60 seconds for 5 minutes.

### **Determination of Reduced Glutathione (GSH) Level**

Reduced glutathione (GSH) level in the epididymis homogenates of the rats was determined according to the method described by Sedlak and Lindsay (1968). To the homogenate, 10% TCA was added and centrifuged. 1.0ml of supernatant was treated with 0.5 mL of Ellman's reagent (19.8mg of 5,5-dithiobisnitro benzoic acid (DTNB) in 100 mL of 0.1% sodium nitrate) and 3.0 mL of phosphate buffer (0.2M, pH 8.0). The absorbance was read at 412 nm using a UV recording spectrophotometer 2150 series (UNICO, Dayton, New Jersey).

### **Histopathological Technique**

The epididymal tissues were fixed in 10% buffered formalin for 48 hours. The samples were thoroughly washed and grossed appropriately, placed in tissue cassettes and labelled according to the group. The sample was dehydrated in ascending graded alcohol, dealcoholized in 2 changes of xylene and infiltrated in two (2) changes of molten paraffin wax and embedded in paraffin. The tissue processing process was run in a Slee MTP Automatic Carousel Spin Tissue Processor for 18 hours. The paraffin tissue blocks were sectioned at 5  $\mu\text{m}$ , placed on a slide and stained with haematoxylin and eosin stains. The epididymal histoarchitectural alterations in the tissue were observed, and a photomicrograph was taken using a Leica microscope (DM750, fixed with Leica ICC50W camera and a LAS software program, Leica Microsystems, Wetzlar, Germany).

### **Sperm Quality Assessment Index**

#### **Sperm Motility**

The sperm motility assessment was performed according to Azu *et al.* (2014). The caudal epididymis is incised to expose fluid. 5  $\mu\text{L}$  of the epididymal fluid exposed

to 1000  $\mu\text{L}$  physiological saline is collected with a micropipette. Fluid was placed on a glass slide and covered with a 22 x 22 mm cover slip. The glass slides were mounted on a light microscope with a magnification of X400. Motility estimation is carried out at room temperature between 24 – 28  $^{\circ}\text{C}$ . The microscopic field is scanned systematically, and each spermatozoon encountered is assessed. Motility is recorded in percentage and classified as motile, non-motile, and actively motile (progressive). The procedure is repeated three (3) times, and an average is taken.

### **Sperm Cell Count and Concentration**

The sperm cell count estimation was done according to Osinubi *et al.* (2008). Harvested epididymis is minced in a petri dish using anatomical scissors. 50  $\mu\text{L}$  of epididymal spermatozoa was diluted in 950  $\mu\text{L}$  of diluent. A well-mixed solution was pipetted into both chambers of the hemocytometer. The hemocytometer is placed on the stage of the microscope. The specimen was viewed with x40 objective. The hemocytometer was viewed, and counting was done. Count values are recorded. Counting was repeated in each chamber, and the average count was documented. The average number of cells and cell concentration is now calculated by applying the formula.

### **Statistical Analysis**

Statistical analysis using specific statistical analyzing application. Data collected from the biochemical sample and sperm quality assessment were analyzed and expressed in mean and standard deviation. The mean comparison was done by One-way analysis of variance (ANOVA) for mean differences comparison between groups. The assessment of the epididymal indices data was typically distributed as assessed by the Shapiro-Wilk test ( $p > 0.05$ ). Games-Howell post-hoc analysis for unequal variance was used, as the equality of variance was not assumed. The outcomes were visualized by bar chart and table, respectively. Statistically, a significant difference was considered at a p-value less than 0.05 ( $p < 0.05$ ).

## **RESULTS AND DISCUSSION**

### **Estimation of Epididymal Sperm Characteristics Across the Groups**

The result of the epididymal sperm characteristic as shown in Table 2. It revealed average sperm motility in the two control groups, Negative control ( $91.67 \pm 2.89$ ) and baseline group 8 ( $76.00 \pm 8.94$ ). The sperm motility levels in the controls were significantly ( $p < 0.05$ ) higher than the exposed groups. The sperm motility estimation of negative control ( $91.67 \pm 2.89$ ) was significantly ( $p < 0.05$ ) higher than group 1 (Brd1-exposed), group 5 (Brd3-exposed) and group 6 (Brd4-exposed). The amount of non-motile sperm was considerably low in the control groups 7 and 8,  $8.33 \pm 2.89$  and  $24 \pm 8.94$ , respectively. The average non-motile value in group 5 (Samsung galaxy S8-exposed) was the highest, followed by group 6 (Brd4-exposed). The difference in the mean non-motile value between the exposed groups was not statistically significant. The post-hoc analyses showed that the average non-motile value of group 7 was significantly ( $p < 0.05$ ) lower than groups 1, 5 and 6. The mean active score showed that group 6 (Brd4-exposed) had the lowest active sperm score ( $19.00 \pm 39.75$ ) while the negative control group 7 had the highest ( $86.67 \pm 2.89$ ) active sperm score. The mean active score of the sperm across the groups was statistically significant. However, the pairwise comparison showed that the mean difference between the exposed groups was not statistically significant ( $p > 0.05$ ). The mean active score of the control groups 7 and 8 was significantly higher than exposed groups 1, 5 and 6. The epididymal sperm count of the unexposed control group 7 was the highest ( $120.5 \pm 20.38$ ). The post-hoc result showed that the average sperm

concentrations of the exposed groups were not significantly different. The mean sperm count was significantly higher in group 7 than in group 4 (Brd2-exposed) and 6 (Brd4-exposed).

Table 2. Estimation of Epididymal Sperm Characteristics Across the Groups

Groups	No	Motility Mean±SD	Non-motility Mean±SD	Active Mean±SD	Sperm Count Mean±SD
1	5	29.00±21.33 <sup>a</sup>	71.00±21.33 <sup>c</sup>	26.00±23.82 <sup>a</sup>	65.85±34.38 <sup>a,b</sup>
2	4	57.50±15.00 <sup>a,b</sup>	40.00±18.26 <sup>a,b,c</sup>	50.00±18.26 <sup>a,b,c</sup>	47.85±31.76 <sup>a,b</sup>
3	4	51.25±19.31 <sup>a,b</sup>	59.00±28.37 <sup>a,b,c</sup>	44.00±33.62 <sup>a,b,c</sup>	81.95±48.04 <sup>a,b</sup>
4	5	50.00±26.93 <sup>a,b</sup>	50.00±26.93 <sup>a,b,c</sup>	46.00±26.08 <sup>a,b,c</sup>	39.00±15.07 <sup>a</sup>
5	4	16.25±11.09 <sup>a</sup>	83.75±11.09 <sup>c</sup>	23.75±18.88 <sup>a</sup>	71.05±54.10 <sup>a,b</sup>
6	5	20.00±41.98 <sup>a</sup>	80.00±41.98 <sup>c</sup>	19.00±39.75 <sup>a</sup>	34.55±30.46 <sup>a</sup>
7	5	91.67±2.89 <sup>c</sup>	8.33±2.89 <sup>a</sup>	86.67±2.89 <sup>c</sup>	120.5±20.38 <sup>b</sup>
8	5	76.00±8.94 <sup>b,c</sup>	24.00±8.94 <sup>a,b</sup>	79.00±8.94 <sup>b,c</sup>	54.25±32.33 <sup>a,b</sup>
p-value		0.001	0.001	0.004	0.04

\*Key: a,b: values with the same alphabetic superscript were not statistically significantly different (p>0.05) at Post-hoc. **\*Exposed Groups:** 1(Brd 1- exposed), 2(Gnr 1- exposed), 3(Gnr 2- exposed), 4(Brd2- exposed), 5 (Brd3- exposed), 6 (Brd4-exposed). **Control groups:** 7 (Negative control) and 8 (Baseline).

### The Concentration of Antioxidants and Oxidative Stress Markers in Caudal Epididymis

The result of the antioxidants and free radical marker concentration in the epididymis is shown in Figure 1 (A-D). The table showed a statistically significant mean difference between epididymis MDA and the antioxidative enzyme markers (except GSH). The pairwise comparison expressed in superscript alphabet showed that the mean difference between smartphones RF radiation-exposed groups was not statistically significantly different for SOD level. However, the control group 7 (8.05 ± 0.03) was significantly (p < 0.05) higher than all the exposed groups except group 1. There was no statistically significant mean difference in the caudal epididymal SOD level of the branded-exposed and the generic-exposed groups. The mean epididymal catalase (CAT) level across the groups did not depict a specific pattern in differentiating the RF radiation exposed and control groups. Group 1, Brd1- exposed, has the lowest epididymal CAT concentration level (5.7±0.02), while group 5, the Brd3-exposed, has the highest CAT level (10.38±1.69). The mean epididymal catalase level of the negative control was not significantly different from the smartphones (branded and generic) RF radiation exposed group. The MDA level in the epididymis was lowest in the baseline control group 8, followed by negative control group 7. The MDA level in group 1 was significantly higher than in groups 2 and 5. The negative control group 7 was not significantly different from the entire RF radiation-exposed groups (1-6). The level of GSH across the groups was not statistically significantly different (p>0.05).

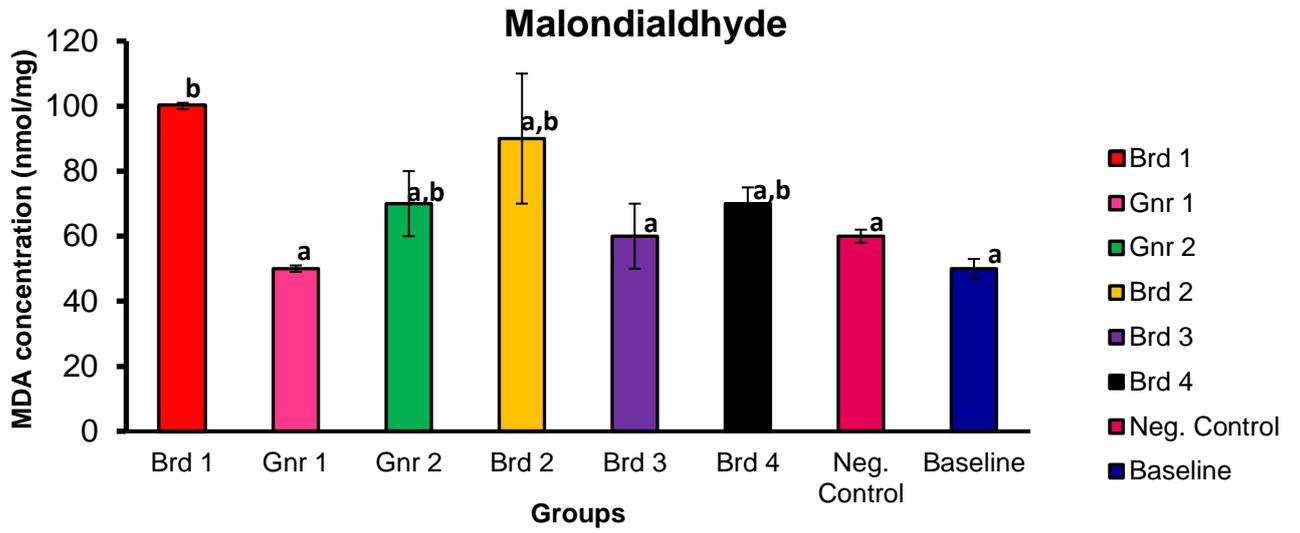


Figure 1.A. MDA

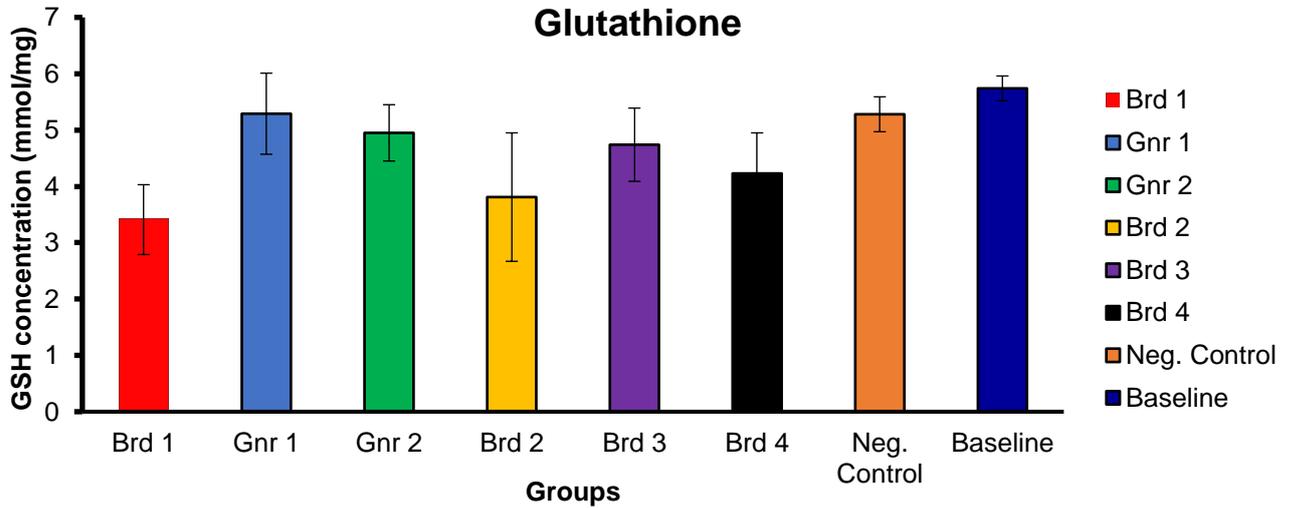


Figure 1.B. GSH

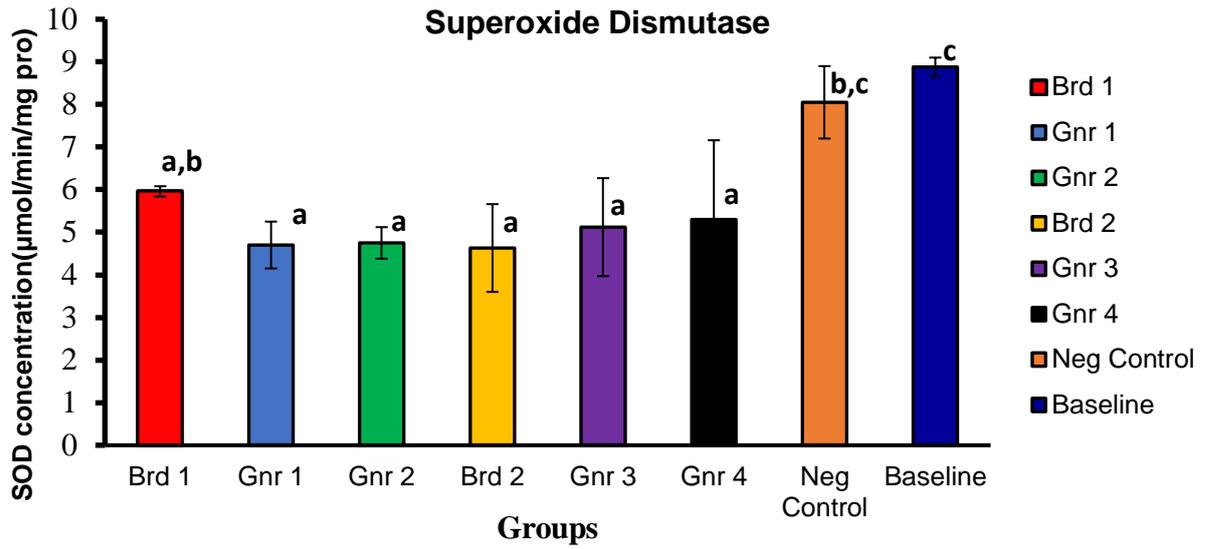


Figure 1.C. SOD

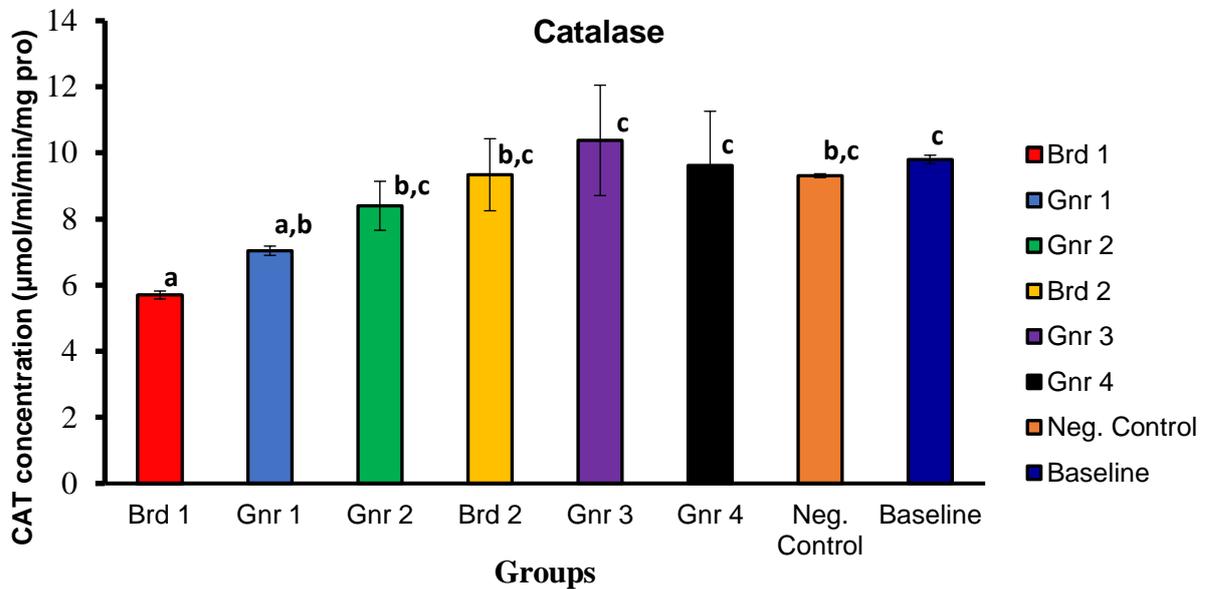


Figure 1.D. CAT

Figure 1.(A-D) Oxidative Stress Marker Level in Rats Exposed to Mobile Phone RF Radiation (Groups 1 - 6 and Control Groups (7&8)). (A) MDA, (B) GSH, (C) SOD and (D) CAT in the Epididymal tissue homogenate of the rats. All the groups were compared against each other using Game-Howell posthoc test. The posthoc is depicted with a lowercase alphabet on the bar. Two Groups with same alphabet(s) was not significantly different ( $p > 0.05$ ).

### Histopathological Changes in the Epididymis

The histopathological changes in the caudal epididymis section in the control groups 7 and 8 (Negative and baseline) and the smartphones (branded and generic) RF radiation-exposed groups are shown in Figure 2 below. The histopathological findings showed that branded model, the Brd1-exposed group showed normal epididymis with a significant reduction of spermatocytes in the lumen. Other branded phones, Brd2 (group 4) and Brd4 (group 6) RF-exposed groups, showed focal hyperplasia. The generic model phone, Gnr1 (group 2) and Gnr2 (group 3) exposed groups showed normal epididymis histology. The mature spermatocyte in the control lumen was normal and uniformly occupied the luminal space compared to the exposed groups, which showed reduced spermatocytes.

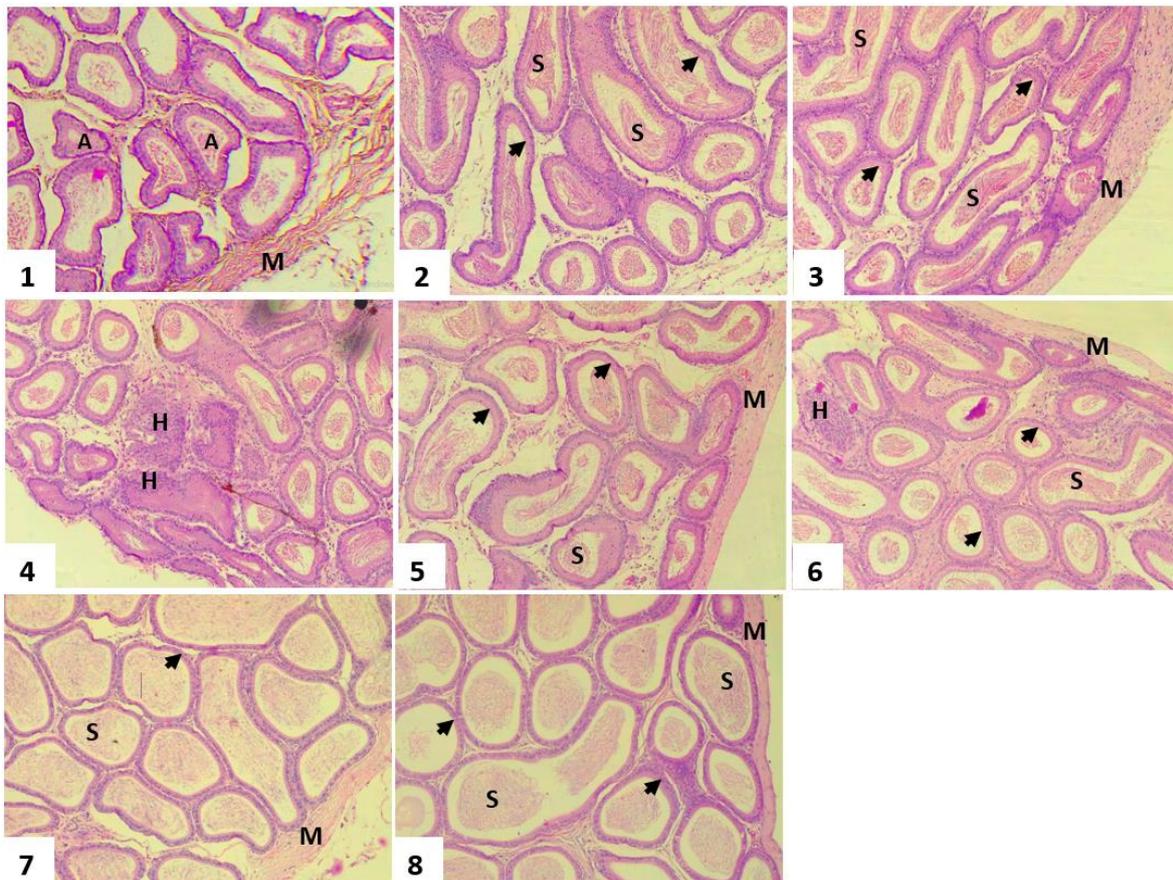


Figure 2. Photomicrograph of the Epididymal Tissue of Groups 1-8. Haematoxylin and Eosin(H&E) Stain, X100 Magnification.

The control groups 7 (Negative control) and group 8 (baseline) showed Normal epididymis with normal epithelial cuboidal to the columnar epithelium (**arrowhead**). The lumen showed evenly distributed mature spermatocytes (**S**) and the standard outer fibromuscular covering (**M**). The Brd1 depicts atrophy (**A**), while Brd2 and Brd4 showed focal hyperplasia (**H**). The epididymis lumen of the mobile phone RF radiation-exposed groups (1 - 6) showed few to moderate mature spermatocyte (**S**) distributions.

### Histopathological Evaluation of the Epididymis

As part of the male reproductive organ, the epididymis cellular changes in response to smartphone RF radiation exposure were examined; this study showed that control groups showed standard histological epididymal architecture and no

pathological lesion was observed. The branded smartphone (Brd2 and Brd4) RF radiation-exposed groups showed focal glandular epithelial hyperplasia, Figure 2 (slides 4 and 6). Other RF-exposed groups showed no epithelial and connective tissue degeneration. Figure 2 (slides 1, 2, 3 and 5). In this study, there were structural epithelial changes. However, no degenerative changes were observed in contrast to the study by Seymen *et al.* (2021), which exposed male Wistar rats to 2600 MHz RF radiation for 30 days and reported structural degenerations in the epididymis such as irregular tubule profile, impairment and vacuolization of epithelium, loss of stereocilia, separation in lateral and basal junctions of epithelium and immature sperm formation. No differential degenerative histopathology changes between animals exposed to branded and generic mobile phone radiation. It is worth knowing that there was a significant reduction in the epididymal luminal spermatocytes across the mobile phones RF radiation-exposed groups, figure 2 (slides 1 to 6).

### **Epididymal Sperm Characteristics (Sperm Motility and Ount)**

The two testicular function includes spermatogenesis and steroidogenesis. However, various situations can interfere with spermatogenesis and impair sperm quality and quantity. Several variables, such as radiation, drugs, chemotherapy, pollutants, contaminated air, and lack of minerals and vitamins, might negatively influence spermatogenesis and sperm production (Krzastek *et al.*, 2020). Normal testicular parameters are partly evaluated by measuring spermatoc variables such as sperm count, motility, viability, and morphology (Zinaman *et al.*, 2000; Eliasson, 2003). The sperm motility report was classified as motile or non-motile, with motile sperm classified as active (progressiveness) or sluggish. In this study, the sperm motility and progressivity estimation of the mobile phone RF radiation-exposed groups showed poor distribution scores, evidenced by the high standard deviation values of the sperm motility estimation in this study. This may suggest the unequal differential responses by a different animal to radiofrequency radiation from mobile phones.

The sperm motility estimation in the controls groups showed uniform distribution scores, evident in the small standard deviation value, as seen in table 2 (groups 7 and 8). The sperm motility estimation grossly reduced in the high-end brands (Brd1 and Brd3) in Table 2 (groups 1 and 5) and an inexpensive brand (Brd4), Table 2 (group 6) RF radiation-exposed groups; this change was not significantly different from the sperm motility estimation of the generic phones exposed groups. This finding also showed that the average sperm motility of some of the RF radiation-exposed groups was significantly lower than the control groups. These results contradict the claim of (Dasdag *et al.*, 2003; Lee *et al.*, 2010; Nisbet *et al.*, 2012) of no significant effect of cell phone radiation on epididymal sperm characteristics. The average active sperm was significantly higher in control and exposed groups. Two of the branded exposed groups have the lowest average active sperm. There was no statistically significant difference in the mean active sperm between the classes of phones (generic and branded).

The results of a sperm count analysis depicted a non-uniform pattern in the scores. The non-uniform distributions of sperm count were evident in sizeable standard deviation in the radiation-exposed groups. The negative control group has the highest sperm concentration (sperm count) compared to the exposed one. The pairwise comparison of the mean values between the exposed groups shows that the average sperm concentration was not significantly different, as shown in Table 2. This result suggested that the impact of radiation from different cell phones on the sperm count was not significantly different. This finding from this study did not agree with the claim by (Tas *et al.*, 2013) that the RF radiation-exposed rat showed no statistically

significant difference from the control group. This research was in agreement with Avendano *et al.* (2012). They performed an in vitro investigation and assessed the wireless impact of laptop computers connected to local area networks on human spermatozoa. They observed a significant decline in progressive sperm motility. They suggested that keeping a laptop connected wirelessly to the internet on the lap near the testes may result in lower male fertility.

In this study, there was depletion in the epididymal superoxide dismutase (SOD) across the radiation-exposed groups, Figure 1c. The lipid peroxidase marker (MDA) level was found to be higher in most of the exposed groups but isolated significantly higher in one of the branded exposed groups (Brd1) (figure 1a) with the corresponding lowest GSH (Figure 1b) and catalase (Figure 1d) levels. This effect may be responsible for nearly empty epididymal lumen, ranging from few mature sperm cells to no luminal spermatocytes. The role of elevated ROS as the reason for the abnormal spermatozoa and the apoptotic figures was reported by Kesari and Behari (2012) in an investigation where Wistar rats were exposed to mobile phone radiofrequency radiations. Aitken and Curry (2011) reported in their review of sperm function redox regulation that elevated ROS levels may induce a reduction in sperm concentration, motility, and viability. They said that faulty spermiogenesis, which would result in the creation of abnormal gametes vulnerable to free radical assault and capable of atypical apoptosis, was caused by oxidative stress. This report was in concordance with the finding of this study, which showed reduced sperm concentration, motility, and activity in the mobile phone RF radiation-exposed groups. Limitation in this study is measuring tools to determine the specific absorption rate (SAR) of the generic phones and to confirm that the documented SAR of the branded phones was not accessible.

## CONCLUSION

In conclusion this study demonstrates that prolonged exposure to radiofrequency radiation emitted from both branded and generic mobile phones can severely impact male reproductive health, leading to gonadotoxicity. This exposure has been found to disrupt the delicate balance between oxidants and antioxidants in the epididymis, resulting in a decrease in superoxide dismutase (SOD) concentration and an increase in malondialdehyde (MDA) levels. As a result, this disruption can lead to a reduction in sperm count and motility, which are essential for male fertility. The observable changes were seen in the generic and branded mobile phone groups in equal measure the study. The epididymis epithelium did not show any degenerative effect across the groups. The study recommends that more studies be done on the non-thermal effects of mobile phone radiation, for a prolonged period with name-brand models and generic phones for more than six months. Measuring the actual value of the specific absorption rate and the emitting radiation parameters of the phones will help to establish the differences in the tissue response to the exposures.

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## CONFLICT OF INTEREST

The authors declare that they have no conflict of interest.

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