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EFFECT OF PHENANTHRENE EXPOSURE ON THE TOTAL ANTIOXIDANT CAPACITY, CATALASE, SUPEROXIDE DISMUTASE AND XANTHINE OXIDASE, OF THE LIVER

Enemchukwu Onyinye Victory, C. Enemuo Ijeoma and H. Enemuo Emeka *

Department of Medicine, Nnamdi Azikiwe University, Nnewi Campus.

Keywords:

Phenanthrene; Polycyclic aromatic hydrocarbons; Oxidative stress; Wistar rats; Superoxide dismutase

Correspondence to Author:
Enemuo Emeka Hyacinth

Department of Medicine, Nnamdi Azikiwe University, Nnewi Campus.

E-mail: emekaenemuo875@yahoo.com

ABSTRACT: Background and Aim: Phenanthrene is an aromatic compound with three benzene rings forming the main structure. It represents the majority of polycyclic aromatic hydrocarbons found in the environment and is found in a variety of areas including, burnt smoky coal particles soil and sediment, diesel emissions, tobacco and food smoke. The aim of the study was to determine the effect of phenanthrene on oxidative stress markers in male Wistar rats. **Methodology:** Fifty healthy rats weighing between 100 and 180g were used for this study. The animals were randomly assigned into five groups of ten rats per group. Group A served as control and the animals were administered with 1ml of DMSO oil only orally for three weeks. Groups B animals were administered orally with 5mg/kg of phenanthrene dissolved in 9ml of DMSO oil and Group C animals were administered orally with 10mg/kg of phenanthrene dissolved in 13ml of DMSO oil for three weeks. Group D animals, had 180mg/kg of phenanthrene dissolved in 1ml of DMSO oil orally, once, while Group E animals were also, administered orally with 900mg/kg of phenanthrene dissolved in 1ml of DMSO oil, given once. **Results:** Results of the experiment showed significant increase in Superoxide dismutase, Xanthine oxidase levels and in Total antioxidant capacity. **Conclusion:** Phenanthrene increased oxidative stress, in male Wistar rats.

INTRODUCTION: Phenanthrene is an aromatic compound with three benzene rings forming the main structure. It was discovered in coal tar by E. Ostermayer in 1872. Phenanthrene derivatives are compounds obtained by transformation and derivation of new compounds from the already existing structure of phenanthrene. These derivatives consist of: mono-substituted phenanthrene, diametrical phenanthrene, poly substituted phenanthrene, phenanthrequinone and dihydrophenanthrene [36].

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Phenanthrene represents the majority of polycyclic aromatic hydrocarbons found in the environment. It is found in a variety of areas including: burnt smoky coal particles [37] soil and sediment, diesel emissions [63], tobacco and food smoke [43]. Phenanthrene has a low molecular weight and is not normally carcinogenic [41]. It is one of the most commonly found polycyclic aromatic hydrocarbons in the environment, largely due to the abundant amounts found in petroleum, creosote and coal tar in sediments [58].

Phenanthrene is a very dangerous chemical commonly found in air, soil and water [21]. It is a type of polycyclic aromatic hydrocarbon that poses a serious threat to the lives of people exposed to it [35]. Exposure to phenanthrene for a long period of time makes an animal highly susceptible to

hepatotoxicity [23]. The liver, the largest internal organ in the body, plays a crucial role in secretion, digestion, blood detoxification and nutrient storage [32]. It is extremely vulnerable to toxicants because constant exposure of hepatocytes to environmental toxins can significantly impair liver function and ultimately cause liver damage [65]. The effect of phenanthrene on the liver is not completely known, as most research done on the effects of phenanthrene in the liver has focused on aquatic animals [64], which do not share the same phenotype as humans and as such cannot be used to understand how phenanthrene will affect the human internal structure. For this reason, it is important to investigate the effect of phenanthrene on male wistar rats, which share similar internal structure to that of humans. The aim of the study was to determine the effect of phenanthrene exposure on oxidative stress markers of male Wistar rats.

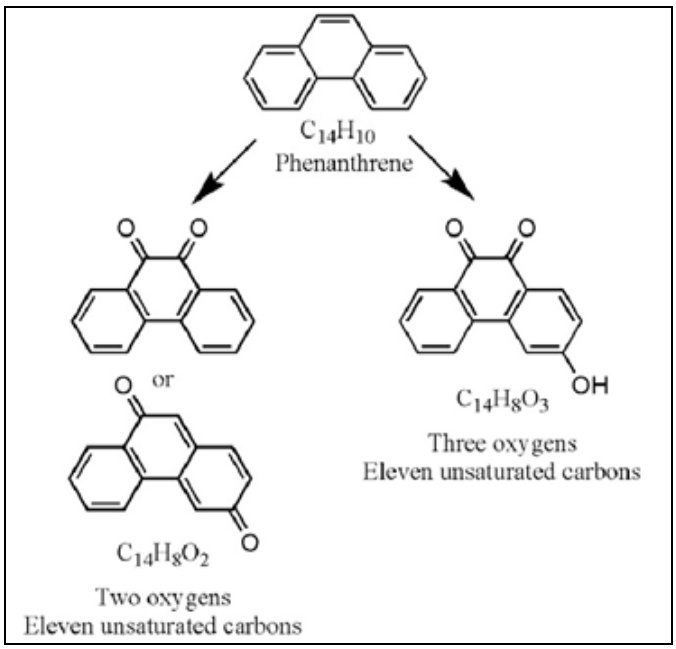

FIG. 1: STRUCTURE OF PHENANTHRENE (HINTZE*ET AL.*, 2010).

MATERIALS AND METHODS: Fifty healthy rats weighing between 100 and 180g were used for this study. The animals were obtained from the animal house of Igbinedion University, Okada, Edo State and they were distributed randomly into five well ventilated plastic cages with wood beddings with 10 rats each, where they were kept for two weeks before commencement of the experiment for acclimatization. They were fed with pelletized commercially prepared growers mash purchased in a local store in Okada, Edo State and had access to water. They were housed at standard laboratory conditions under 12h light/12h dark cycle. The cages were continuously kept clean every day to ensure good hygiene and prevent the animals from disease.

The animals were randomly assigned into five groups of ten rats per group.

Group A: Animals were administered with 1ml of DMSO oil only orally for three weeks. (DMSO only), and had free access to the pelletized commercially prepared growers mash and water *ad libitum*. This group served as the control group.

Group B: Animals were administered with 5mg/kg of phenanthrene that was weighed using Mettler balance and dissolved in 9ml of DMSO oil orally for three weeks. (PHE + DMSO)

Group C: Animals were administered with 10mg/kg of phenanthrene that was weighed using Mettler balance and dissolved in 13ml of DMSO oil orally for three weeks.

Group D: Animals were administered with 180mg/kg of phenanthrene that was weighed using Mettler balance and dissolved in 1ml of DMSO oil orally, once.

Group E: Animals were administered with 900mg/kg of phenanthrene that was weighed using Mettler balance and dissolved in 1ml of DMSO oil orally, once.

TABLE 1:

Groups	Treatments
A	DMSO oil only
B	5mg/kg Phenanthrene dissolved in DMSO oil
C	10mg/kg Phenanthrene dissolved in DMSO oil
D	180mg/kg Phenanthrene dissolved in DMSO oil
E	900mg/kg Phenanthrene dissolved in DMSO oil

Analytical Procedure: The Phenanthrene administration was done for 21 days. At the end of the experiment, the rats were weighed and blood samples collected through retro-orbital puncture for hematological analysis. Biochemical analysis was carried out using spectrophotometric method.

Catalase (CAT): Catalase is a common enzyme found in nearly all living organisms exposed to sunlight, such as bacteria, plants and animals, which catalyzes the decomposition of hydrogen

peroxide to water and oxygen. It is highly essential in the protection of cells from oxidative damage by reactive oxygen species (ROS) [17].

Principles: The enzymatic reaction of catalase (CAT) in decomposing peroxide (H_2O_2) can be quickly stopped by ammonium molybdate. The residual H_2O_2 reacts with ammonium molybdate to generate a yellowish complex. CAT activity can be calculated by production of the yellowish complex at 405 nm against Blank.

Assay Procedure: Heparinized whole blood samples was diluted between 5 – 10 fold and tissue samples were homogenized in 10 vol. of 50 mmol/l phosphate buffer, pH 7.4 and centrifuged at 3000rpm for 20 minutes and the supernatant collected before starting the procedure. 200ul sample was added into a cuvette. 1ml of Substrate solution was added into the cuvette, mixed and allowed to stand for 1 minute. 1ml of molybdate solution was added into the cuvette and mixed for 2 minutes. The absorbance was read at a wavelength of 405nm and recorded. For the blank measurement, the samples were replaced with 200ul distilled water and the procedure was repeated.

Calculation:

Catalase Activity in U/ml (Serum or Plasma) = Abs Blank – Abs Sample × 162.5

Catalase Activity in U/mg Protein (Tissue) = Abs Blank – Abs Sample / Sample Protein Conc. in mg/ml × 162.5

Superoxide Dismutase: Superoxide dismutase (SOD), which catalyzes the dismutation of the superoxide anion (O2.-) into hydrogen peroxide and molecular oxygen, is one of the most important antioxidative enzymes [39].

0.5ml of whole blood was centrifuged for 10 minutes at 3500 rpm and then aspirated off the plasma. Red cells were washed 4 times with 5 ml of 0.9% saline and centrifuged for 10 minutes at 3500 rpm after each run. The washed centrifuged erythrocytes were then made up to 2.0 ml with cold distilled water, mixed and left to stand at 4°C for 15 minutes. Tissue samples were homogenized in 10 vol. of 50 mmol/l phosphate buffer, pH 7.4, then centrifuged for 20 min at 3000 rpm. The supernatant was collected and used in the assay.

Principles: The enzyme Superoxide dismutase has the ability to inhibit the autoxidation of pyrogallol. The autoxidation of pyrogallol in the presence of EDTA in the pH 8.2 is 50%. The principle of this method is based on the competition between the pyrogallol autoxidation by $O_2^{\bullet-}$ and the dismutation of this radical by SOD.

Assay Procedure: 50ul sample was added into a clean cuvette, 1ml of SOD assay buffer was added to the cuvette, 1ml of SOD chromogen solution was added to the cuvette and mixed. The absorbance was read immediately at 420nm, then read again after 1 minute. For Blank measurement, the sample was replaced with 50ul distilled water and the procedure was repeated.

Calculations: The change in absorbance of samples and blank was determined using the following equation:

Change in absorbance of Sample (ΔS) = Absorbance of sample (1 minute) – Absorbance of sample (Initial)

Change in absorbance of blank (ΔC) = Absorbance of blank (1 minute) – Absorbance of blank (Initial)

% inhibition of Pyrogallol autoxidation = ΔS / ΔC × 100%

SOD Activity In (U/ml) = % inhibition of Pyrogallol autoxidation / 50%

Total Antioxidant Capacity (TAC) Assay: Oxidative stress has been implicated in a number of diseases such as atherosclerosis, chronic inflammatory disease, chronic renal failure, and cancer. It is a condition where an imbalance exists between the production of reactive oxidizing species and the body's ability to neutralize these intermediates, resulting in cellular damage. The body has designed several physiological responses to oxidative stress including counterbalances such as enzymes and variously functionalized molecules that effectively neutralize these damaging species. These antioxidants can be either water or lipid soluble, and are localized transiently throughout various tissues, cells and cell types.

Principles of Procedure: The reduction potential of the sample or standard effectively converts Cu+2 to Cu+1, thus changing the ion's absorption characteristics. This reduced form of copper will selectively form a stable 2:1 complex with the chromogenic reagent with an absorption maximum

at ca. 450 nm. A known concentration of Trolox is used to create a calibration curve, with the data being expressed as mMTrolox equivalents or in µM copper reducing equivalents.

Assay Procedure: 50ul of samples and each standard were placed into the cuvette supplied. Blank contained 50ul of Dilution buffer in place of Standard or Sample. 1ml Assay Buffer was added to the cuvette containing samples, standards and blank. Background absorbance was read at 450nm. 100 µL of Chromogen was added and incubated for 5 minutes at room temperature. The cuvette was read a second time at 450 nm.

Standard Preparation: The trolox powder was diluted with 1 ml of TAC Dilution Buffer to make a 2mM Trolox solution. Serial dilutions were carried out to prepare the following concentration; 2mM, 1mM, 0.5mM, 0,25mM, 0.125mM, 0mM.

Calculations:

1. The change in absorbance of Sample and standard was calculated by subtracting the first absorbance readings from the second.
2. A calibration curve was plotted by plotting the Absorbance of the Standards (Y-axis) against the given concentration of the Standards (X-Axis).
3. The Total antioxidant Capacity of the samples was extrapolated from the calibration curve
4. The Total antioxidant capacity of the samples is expressed in mMTrolox equivalents

Xanthine Oxidase (XO) Assay: The rate of formation of urate from hypoxanthine was determined by measuring increased absorbance at 290 nm. A unit of activity is that forming one micromole of urate per minute at 25°C [62].

Procedure: Into cuvettes the following was pipetted:

TABLE 2:

	Test	Control
Buffer	1.9 ml	1.9 ml
Reagent grade water	----	1.0 ml
Enzyme	0.1 ml	0.1 ml
Substrate (at zero time)	1.0 ml	----

Increase in absorbance was recorded and ΔA_{290} was determined from the linear portion of the curve. The rate was proportional to enzyme concentration within limits of 0.01 to 0.02 units per test.

Calculations:

$$\text{Units/ mgP} = \Delta A/\text{ min} \times 1000/\ 1.22 \times 10^4 \times \text{mg/ ml rection mixture}$$

$$\text{Units/ ml} = \Delta A/\text{ min} \times 1000 \times 3 \text{ ml} \times \text{dilution} / 1.22 \times 10^4 \times 0.1 \text{ ml}$$

The molar absorbancy of uric acid = 1.22 X 10^4 cm^{-1} [62].

PICTURE ABOVE SHOWED THE FEED GIVEN TO THE RATS, THE RAT CAGE THE TOXICANT ADMINISTERED TO THE RATS (PHENANTHRENE)

RESULTS: Fig. 1 showed the effect of Phenanthrene on Superoxide Dismutase (SOD) in the liver. There was a significant increase in the levels of SOD in the 180mg and 900mg groups when compared with control. There was significant increase in levels of SOD in the 180 and 900mg groups when compared with 5mg group. There was significant increase in levels of SOD in the 180 and 900mg groups when compared with 10mg group. Meanwhile, there was no statistically significance difference between 900mg group when compared to 180 group. There was no significance difference between 10mg group when compared to 5mg group and control. There was also no significant difference between 5mg group when compared with control.

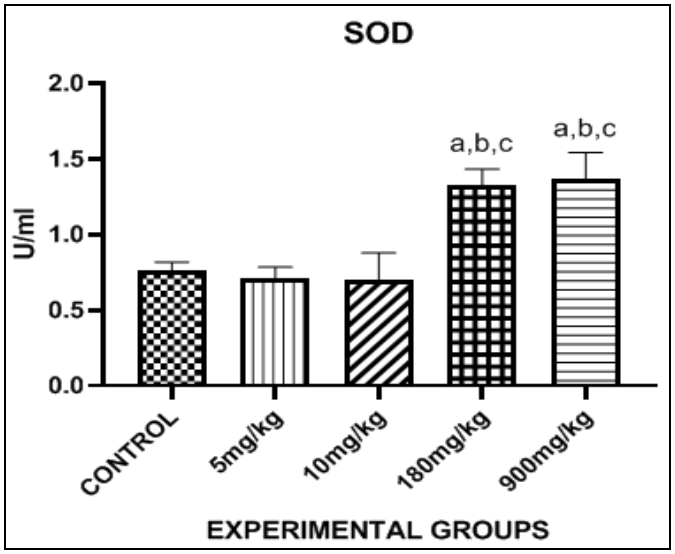

FIG. 1: EFFECT OF PHENANTHRENE (PHE) ON SUPEROXIDE DISMUTASE (SOD) IN THE LIVER. Bars are expressed in (1.327 ± 0.05302), (1.364 ± 0.07305). a, b, c represent significant differences when compared with control, 5mg and 10mg groups respectively ($p<0.05$).

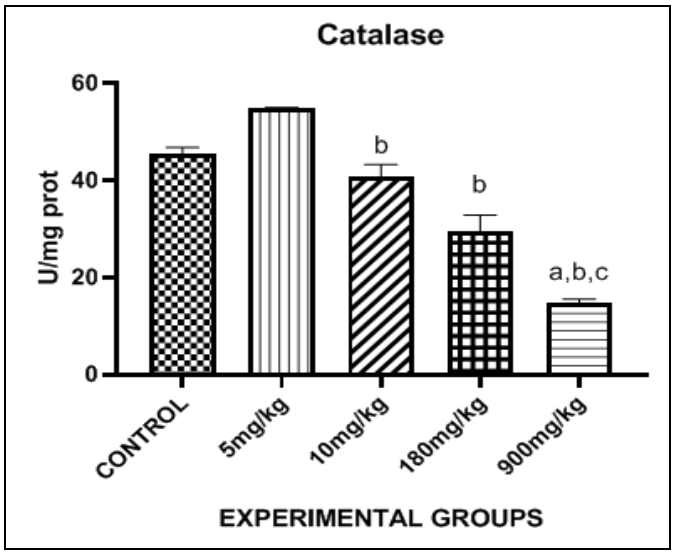

FIG. 2: EFFECT OF PHENANTHRENE (PHE) ON CATALASE (CAT) IN THE LIVER. Bars are expressed in (64.71 ± 10.22), (14.69 ± 0.4072). a, b, c, represent significant differences when compared with control group, 5mg group and 10mg group respectively ($p <0.05$).

Fig. 2, showed the effect of Phenanthrene on catalase in the liver. There was a significant increase in the level of catalase in the 5mg group when compared with control. There was significant decrease in the level of catalase in 900mg group when compared with control. There was significant decrease in 10mg, 180mg and 900mg group when compared with 5mg group. There was a significant decrease in 900mg group when compared with 10mg group. Meanwhile, there was no significance in 10mg and 180mg group when compared with control group.

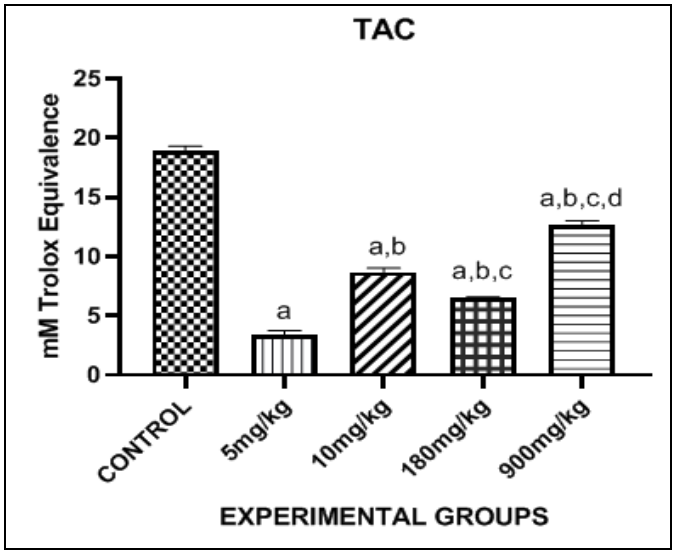

FIG. 3: EFFECT OF PHENANTHRENE (PHE) ON TOTAL ANTIOXIDANT CAPACITY ASSAY (TAC) IN THE LIVER. Bars are expressed in (3.450 ± 0.1559), (8.685 ± 0.1381), (6.537 ± 0.01827), (12.66 ± 0.1795). a, b, c, d represent significant differences when compared with 5mg/kg, 10mg/kg, 180mg/kg and 900mg/kg groups respectively ($p<0.05$).

Fig. 3 showed the effect of Phenanthrene on Total Antioxidant Capacity assay (TAC) in the liver. There was significant decrease in levels of TAC in 5mg, 10mg, 180mg and 900mg groups when compared to control. There was significant increase in levels of TAC in 10mg, 180mg and 900mg groups when compared with 5mg group. There was significant decrease in levels of TAC in 180mg group when compared with 10mg group. There was significant increase in levels of TAC in 900mg group when compared with 10mg group. There was significant increase in levels if TAC in 900mg group when compared with 180mg group.

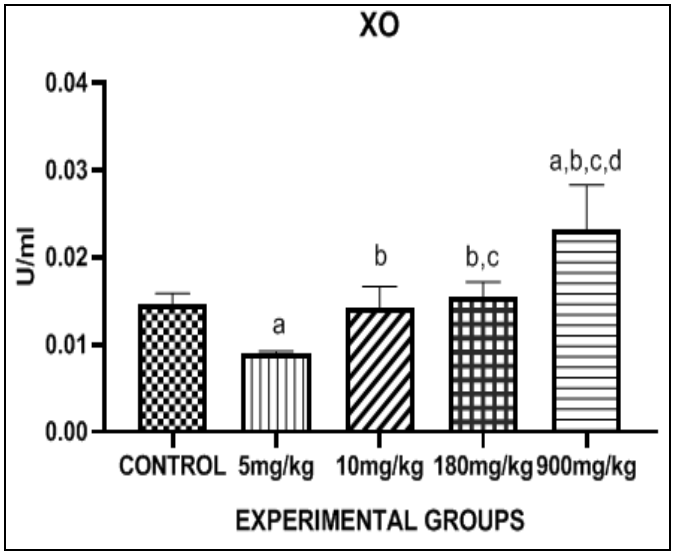

FIG. 4: EFFECT OF PHENANTHRENE (PHE) ON XANTHINE OXIDASE (XO) IN THE LIVER. Bars are expressed in (0.009 ± 9.364), (0.02321 ± 0.002071). a, b, c, d represent significant differences when compared to control groups, 5mg, 10mg and 180mg respectively ($p<0.05$).

Fig. 4, showed the effect of Phenanthrene on Xanthine Oxidase (XO) in the liver. There was a significant decrease in the level of XO in the 5mg group when compared with control. There was a significant increase in the level of XO in 900mg group when compared with control. There was a significant increase in level of XO in 10mg, 180mg and 900mg group when compared to 5mg group. There was a significant increase in level of XO in 180mg and 900mg groups when compared to 10mg group. There was a significant increase in level of XO in 900mg group when compared to 180mg

group. There was no significant difference between 10mg and 180mg group when compared with control group.

DISCUSSION: Oxidative stress is known to be an important mechanism of PAH-induced toxicity [60]. PAHs have been shown to be associated with a variety of toxic effects including oxidative stress, inflammation, and immunological disorders after they are metabolically activated to electrophilic intermediates. These reactive intermediates are able to covalently bind to DNA or participate in redox cycles. This process leads to an overproduction of reactive oxygen species, also known as ROS [47]. Antioxidant defenses include enzymes such as SOD and CAT. In a normal physiological situation, antioxidant defense systems are thought to prevent the production of ROS and other reactive oxygen species [33].

Antioxidant defenses can be induced by a mild oxidative stress as a compensatory response; however, a severe oxidative stress suppresses the activities of these enzymes and can lead to oxidative damage [64]. When OH is produced in excess or the cellular antioxidant defense is deficient, it can stimulate free radical chain reactions with proteins, lipids and nucleic acids, causing cellular damage and even disease [12]. Phenanthrene can accumulate in the liver and induce OH production, leading to oxidative stress that alters the activities of antioxidant enzymes such as catalase and superoxide dismutase [64].

Superoxide dismutase and catalase are antioxidant enzymes that play not only a fundamental but also an indispensable role in the antioxidant protective capacity of biological systems against the attack of free radicals. Superoxide dismutase (SOD) is the first detoxifying enzyme and the most powerful antioxidant in the cell. It acts as a first-line of defense system against reactive oxygen species (ROS) and catalyzes the dismutation of two superoxide anion molecules into hydrogen peroxide and molecular hydrogen, making the potentially harmful superoxide anion less dangerous [24]. Catalase is a widely distributed antioxidant enzyme that is found in almost all living tissues that consume oxygen. It utilizes iron (Fe) or manganese (Mn) as a cofactor and catalyzes the breakdown or reduction of hydrogen peroxide (H_2O_2) to water and molecular oxygen, thus completing the detoxification process initiated by SOD. It is abundant in cells, where it continuously searches for and breaks down hydrogen peroxide molecules, to produce oxygen and water [13].

The ability of CAT to effectively limit the concentration of hydrogen peroxide in cells makes it a more reliable tool in regulating the above physiological processes and, in addition, it is a first defense antioxidant enzyme [24]. Catalase deficiency in the cells has been linked to various diseases and abnormalities [55].

The decrease in catalase activity and total antioxidant capacity is a sure indicator of oxidative stress [49]. SOD deficiency is associated with a number of pathologies including neurodegeneration, myocardial damage and perinatal death in mice [34]. Unfortunately, SOD deficiency is quite common as it significantly promotes cellular health and protects the body's cells from excess oxygen radicals, free radicals and harmful agents that promote cell death [28].

Effect of Phenanthrene on Superoxide Dismutase: Superoxide dismutase (SOD) is the first detoxification enzyme and the most powerful antioxidant in the cell. It acts as a first line of defense against reactive oxygen species and helps neutralize the superoxide ion of the free radical [24]. In this study, the results show a significant increase in superoxide dismutase levels in the 180mg and 900mg groups in comparison to control.

This is possibly due to oxidative stress in the liver of these groups, as increased superoxide ion in the cells stimulates the first-order antioxidant enzyme superoxide dismutase to begin oxidizing superoxide anion to hydrogen peroxide and molecular oxygen [24]. However, this may cause the accumulation of hydrogen peroxide in the cells. This accumulation will lead to significantly increased amounts of hydrogen peroxide in the cells. This significant increase in the amount of Hydrogen Peroxide in the cells is also known as high levels of hydrogen peroxide in the cells, which is extremely toxic [24]. This finding is similar to that in the study carried out by (Ma *et al.,* 2020) [42], where administration of phenanthrene led to increased superoxide dismutase levels.

Effect of Phenanthrene on Catalase: Catalase is a common antioxidant enzyme that is found in almost all living tissues that utilize oxygen. Its function is to convert hydrogen peroxide to water and oxygen, thereby neutralizing it [24].

This is because Hydrogen Peroxide accumulated at high levels in the body can cause cell damage and oxidative stress [24]. Oxidative stress is a phenomenon that occurs when the number of reactive oxygen species in the cells is significantly greater than the number of antioxidants needed to neutralize them [59]. Catalase levels were measured using spectrophotometry. In this study, the results show a decrease in catalase in the 900mg group in comparison to the control group, which may have been as a result of oxidative stress [49]. This decrease in Catalase could have been as a result of the increase in Superoxide dismutase levels. Because the superoxide dismutase is "working overtime" to oxidize the ever increasing levels of superoxide ion in the cells, there becomes excess hydrogen peroxide for the catalase to "clean up". The more that catalase is being utilized to oxidize the hydrogen peroxide into oxygen and water, with an ever increasing amount of hydrogen peroxide levels, the less catalase there is to perform any antioxidant activity. Eventually, the number of hydrogen peroxide levels significantly increases more than the levels of catalase needed to oxidize it. This leads to oxidative stress [24].

This is similar to the work of (Ma *et al.,* 2020) [42] where administration of environmental toxicant Phenanthrene also led to decrease in catalase. It is also similar to the work of Yin *et al.,* 2007, whose catalase also reduced upon exposure to phenanthrene. The results also show a statistically significant increase in catalase in the 5mg group, which may have occurred as a result of an antioxidant defense being induced by slight oxidative stress in the liver of the 5mg/kg group. Little concentration of the toxicant can trigger slight increase in catalase levels [30].

Effect of Phenanthrene on Total Antioxidant Capacity: Total Antioxidant capacity is a measure of the ability of a biological system to neutralize oxidants and free radicals [59]. It is an important biochemical in medical and nutritional studies, as it provides insight into the overall antioxidant status and oxidative stress in the body [27]. In this study, there was a significant decrease in total antioxidant capacity levels in the 5mg, 10mg, 180mg and 900mg groups in comparison to the control group. This might have been as a result of oxidative stress in the livers of these groups [49], impairing the ability of their antioxidants to combat against the reactive oxygen species over produced as a result of the environmental toxicant phenanthrene in their livers [42]. Upon entry of environmental toxicant phenanthrene into the liver of the experimental groups, there might have been an increase in reactive oxygen species specifically to combat the effects of this strange toxicant [42].

However, upon unwillingness of this toxicant to leave the cells, the naturally occurring reactive oxygen species may have continued to multiply, in an effort to eradicate environmental toxicant phenanthrene. This leads to significant increase in amount of reactive oxygen species in the cells. This significant increase triggered the total antioxidant capacity to neutralize the suddenly excessive reactive oxygen species [59]. Unfortunately, the continued presence of phenanthrene in the cells kept triggering the reactive oxygen species to try to combat its effect, which continued to trigger antioxidants to neutralize these excessive reactive oxygen species until finally there is a significant decrease in the total antioxidant capacity [49].

Effect of Phenanthrene on Xanthine Oxidase: Xanthine oxidase is a form of xanthine oxidoreductase, a type of enzyme that generates reactive oxygen species. These enzymes catalyze the oxidation of hypoxanthine to xanthine and further catalyze xanthine to uric acid [7]. During severe liver damage, xanthine oxidase is released into the blood, so a blood assay for xanthine oxidase is a way to determine if severe liver damage occurred [52].

In this study there was a significant increase in xanthine oxidase levels in 900mg/kg group of the rats in comparison to control. This might have occurred as a result of severe liver damage [52], and might have resulted in accumulation of uric acid in the joints of the 900mg/kg group, causing various diseases [7]. There was a significant decrease in the 5mg/kg group in comparison to control. This might have occurred as a result of superoxide anion

suppression by the increase in superoxide dismutase levels in the study, and might have resulted in inhibition of uric acid formation by xanthine oxidase itself [5].

CONCLUSION: Phenanthrene increased oxidative stress markers in male Wistar rats.

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