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Assessment of the effect of gasoline fume on stress hormones, antioxidant status and lipid peroxidation in albino rat

F.O. Owagboriaye [a,*], G.A. Dedeke [b], A.A. Aladesida [b], J.A. Bamidele [b], W.E. Olooto [c]

[a] *Department of Plant Science and Applied Zoology, Faculty of Science, Olabisi Onabanjo University Ago-Iwoye, Ogun State, Nigeria*
[b] *Department of Pure and Applied Zoology, College of Bioscience, Federal University of Agriculture, Abeokuta, Ogun State, Nigeria*
[c] *Department of Chemical Pathology and Immunology, Faculty of Basic Medical Sciences, Olabisi Onabanjo University, Ago-Iwoye, Ogun State, Nigeria*

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Abstract Gasoline fume has been considered a major air pollutant affecting the heart, lungs, brain, liver and kidneys. Therefore, this study aims at investigating the effect of inhalation exposure to gasoline fume on some endogenic stress hormones and oxidative enzymes of albino rats. Forty adult male albino rats were randomly assigned to five experimental treatments (T) with eight rats per treatment (T1, T2, T3, T4 and T5). The control treatment, T1 was housed in a section of experimental animal house free from gasoline fumes while T2, T3, T4 and T5 were exposed to gasoline fumes in exposure chambers for one, three, five and nine hours daily respectively for twelve weeks. The levels of adrenocorticotropic hormone (ACTH), aldosterone and corticosterone were determined using enzyme-linked immunosorbent assay (ELISA) kits. Concentrations of oxidative stress marker (GSH, CAT, MDA and BuChE) were assayed using standard method. Levels of ACTH were recorded to significantly reduce in the gasoline fume exposed rats when compared to control. Aldosterone and corticosterone significantly increase with increase in the daily period of gasoline fume exposure relative to the control. Values of ACTH negatively correlate with those of corticosterone and aldosterone in the exposed rats. The values of GSH, CAT and BuChE were significantly higher in the control rats and significantly reduce with increasing daily exposure time to gasoline fume. MDA concentration was lower in control rats but significantly increased with increasing daily

* Corresponding author.
E-mail addresses: fobillion@yahoo.com (F.O. Owagboriaye), gabrieldedekson@gmail.com (G.A. Dedeke), aaladesida@gmail.com (A.A. Aladesida), Julius.bamidele@yahoo.com (J.A. Bamidele), olotoenny@yahoo.com (W.E. Olooto).
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exposure time to gasoline fume. Inhalation exposure to gasoline fume was observed to induce stress in the exposed animals.

1. Introduction

The volatile nature of petrol makes it readily available in the atmosphere any time it is dispensed, especially at petrol filling stations and depots. According to the Office of Environmental Health Hazard Assessment (2014), gasoline fuel contains toxic substances that can enter the environment and cause adverse health effects in people. Some of these substances, such as benzene, toluene and xylenes, are found in crude oil and occur naturally in fuels and their vapours. Petrol contains a mixture of volatile hydrocarbons and so inhalation is the most common form of exposure (Cecil et al., 1997). As reported by Takamiya et al. (2003), petrol vapour can reach supra-lethal concentrations especially in confined or poorly ventilated areas. Exposure to very high concentrations may result in rapid unconsciousness and death due to respiratory failure (Chilcott, 2007).

In Nigeria, there is an increase in the demand for petrol and other petroleum products which were used for various reasons at homes, in manufacturing and petrochemical industries. Some of these uses include fuel for vehicles, cooking and lighting fuel in homes and outside homes, as chemical feedstock for industries, therapeutic reasons (Huckabay et al., 1995) and as fuel for electricity generating machines at homes, offices and industries. This increasing daily use has increased the frequency at which individuals are exposed to its fume. Exposures to petroleum products both in and outside petroleum industries have been reported to have some effects on the users, with those who are occupationally exposed being more likely to be affected than their counterparts (Rothman et al., 1996; Carbello et al., 1994; Smith et al., 1993). Such effects include increased incidences of blood disorders and anaemia, higher cancer risk, renal function impairment and nephrotoxicity (Edokpolo et al., 2015; Riaz et al., 2014; Rothman et al., 1996; Festus et al., 2013).

Lipid peroxidation, the oxidative catabolism of polyunsaturated fatty acids, is widely accepted as a general mechanism for cellular injury and death, and has been implicated in diverse pathological conditions (Garcia-Souza and Oliveira, 2014; Maruyama et al., 2014). Also, superoxide dismutase (SOD) and catalase (CAT) were referred to as endogenous antioxidant enzymes that act as free-radical scavengers and hence prevent and repair damage done by reactive oxygen species (Wu et al., 2013; Wyse et al., 2004). The roles played by the hormones secreted by the adrenal cortex (cortisol/corticosterone and aldosterone) in the mediation of physiological stress have also been documented (Franklin et al., 2012; De Kloet and Rinne, 2007; Jacobson, 2005; Neil, 2004).

Organs such as the heart, lungs, skin and kidneys have been reported to be affected by the toxic effects of gasoline fume exposure, resulting in various diseases and different forms of genotoxic, mutagenic, immunotoxic, carcinogenic and neurotoxic manifestations (d'Azevedo et al., 1996; Smith et al., 1996; Rabble and Wong, 1996; Rothman et al., 1996). Specific effect of gasoline exposure on some organs of the body has been studied by several authors. Ahmed et al. (2011) reported that gasoline vapour inhalation induced lung tissue injury and cellular damage, increasing the activities of antioxidant enzymes such as glutathione- S- transferase, glutathione peroxidase and glutathione reductase. Similarly, Elsayed et al. (2015) observed a damaging effect of gasoline exposure on the brain tissue, causing a significant reduction in the activities of antioxidant enzymes and an increase in lipid and protein oxidation levels in brain tissue. Exposure to gasoline has also been shown to demonstrate some toxicity to the liver, significantly increasing malondialdehyde concentration and hepatic enzymes (aspartate amino transferase (AST) and alanine amino transferase (ALT) activities) (Bokolo and Ligha, 2013; Uboh et al., 2005). Exposure to petroleum hydrocarbon was also linked with renal dysfunction via oxidative stress, increasing lipid peroxidation and reducing the antioxidant defence mechanism (Oyebisi et al., 2013). Uboh et al. (2013) therefore concluded that exposure to diesel and gasoline may be a risk factor for nephrotoxicity.

Odewabi et al. (2014) evaluated the effect of petroleum fume exposure on the plasma antioxidant defence system using human subjects (petrol attendants). Enhanced lipid peroxidation was observed in the petrol attendants when compared with control subjects. Similarly, a decrease in the antioxidant defence system (oxidative enzymes) was recorded in the blood of the petrol attendants. However, there is still paucity of information on the effect of different exposure times to gasoline fume on the antioxidant system of the blood. Also, previous studies on the stress effect of gasoline exposure have focused more on oxidative stress response in some specific organs of the body such as the lungs (Ahmed et al., 2011), liver (Bokolo and Ligha, 2013) and kidney (Azeez et al., 2013). In order to provide in-depth information on the status of stress-induction by exposure to gasoline fume, there is the need to study its effect on the hypothalamic–pituitaryadrenal (HPA) hormones which have also been reported to be involved in stress mediation (Neil, 2004; Jessica et al., 2006; Johnson and Grippo, 2006). Hence, the contribution of endogenic stress response hormones together with oxidative enzymes to the physiological effects of gasoline fume exposure needed to be evaluated, especially in the body circulating fluid. Therefore, this present study aims at evaluating the effect of varying daily periods of gasoline fume exposure on the levels of stress mediated hormones; adrenocorticotropic hormone (ACTH), aldosterone and corticosterone; and oxidative enzymes; reduced glutathione (GSH), CAT, malondialdehyde (MDA) and butyryl-cholinesterase (BuChE) in the blood of albino rats.

2. Materials and methods

2.1. Experimental animal

Forty adult male albino rats aged 9–10 weeks (200–250 g) were obtained from the breeding section of the animal house of the

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