EUROPEAN JOURNAL OF BIOMEDICAL AND PHARMACEUTICAL SCIENCES
http://www.ejbps.com

ISSN 2349-8870
Volume: 7
Issue: 9
55-60
Year: 2020

TOTAL ANTIOXIDANT CAPACITY AND LIPID PEROXIDATION IN AUTOMOBILE MECHANICS AND PLASTIC INDUSTRY WORKERS IN NNEWI METROPOLIS, ANAMBRA STATE, NIGERIA

[1]*Patrick Onochie Manafa, [2]Jude Ndubuisi Onwuzulike, [3]Nkiruka R. Ukibe, [4]Ezihe Henry Chinagorom, [5]Enyi Ruhuoma Alice, [6]Izunna Somadina Okwelogu, [7] Ogbuowelu Ogechukwu Sabina, [8]Igwe Chigbo Anselem, [9]Eguogwu Franklin Chigozie and [10]Nnenna Amadi C.

[1]Department of Medical Laboratory Science, Nnamdi Azikiwe University, Nnewi Campus, Anambra, Nigeria.
[2]Deparment of Medical Laboratory Science, Aminu Kano Teaching Hospital, Kano, Kano State, Nigeria.
[3]Department of Medical Laboratory Science, Nnamdi Azikiwe University, Nnewi Campus, Anambra, Nigeria.
[4]Department of Medical Laboratory Science, Federal Neuropsychiatric Hospital, Enugu, Nigeria.
[5]Posgraduate Student, Rivers State University, Portharcourt, Rivers state, Nigeria.
[6]Department of Parasitology and Entomology, Nnamdi Azikiwe University, Awka, Anambra State, Nigeria.
[7]Department of Medical Laboratory Science, Nnamdi Azikiwe University, Nnewi Campus, Anambra, Nigeria.
[8]Department of Medical Laboratory Services, Crescent Diagnostics GRA, Onitsha, Anambra State, Nigeria.
[9]Department of Medical Laboratory Science, Nnamdi Azikiwe University, Nnewi Campus, Anambra, Nigeria.
[10]Department of Medical Laboratory Science, Nnamdi Azikiwe University, Nnewi Campus, Anambra, Nigeria.

***Corresponding Author: Patrick Onochie Manafa**
Department of Medical Laboratory Science, Nnamdi Azikiwe University, Nnewi Campus, Anambra, Nigeria.

Article Received on 30/07/2020 **Article Revised on 20/07/2020** **Article Accepted on 20/08/2020**

ABSTRACT
Automobile mechanics and plastic industry workers are exposed to a complex mixture of chemicals in their respective work places. Most of these chemicals have been adjudged to affect good health through the generation of free radicals. Inadequate body antioxidants in the presence of excess free radicals can result in oxidative stress which may lead to tissue damage. Oxidative stress has been implicated in the etiology of many diseases. **Materials and Methods:** This work is a cross-sectional study aimed at determining the total antioxidant capacity and lipid peroxidation in automobile mechanics and plastic industry workers. Lipid peroxidation was evaluated using malondialdehyde, a biomarker. The parameters were estimated spectrophotometrically. A total of 150 individuals which comprised 50 automobile mechanics, 50 plastic industry workers and 50 healthy individuals who were neither automobile mechanics nor plastic industry workers (controls) were recruited for this study by purposive sampling. A questionnaire was used to obtain important information from the participants and about 7mls of venous blood was drawn for analysis of the parameters from the cubital fossa. **Results:** The results showed significant lower mean serum TAC in automobile mechanics (884.50±146.27) and plastic industry workers (829.01±158.58) compared with the control group (994.43±164.18) ($p<0.05$). A significantly higher mean MDA levels were observed in automobile mechanics (2.88±0.80) and plastic industry workers (3.15±0.70) compared with the control (2.09±0.47) ($p<0.05$). No correlation was observed between TAC (r= -0.078, p=0.593) and MDA levels (r=-0.054, p=0.771) and age of automobile mechanics. In plastic industry workers, a weak positive correlation existed between TAC and age (r = 0.196, r=0.172), while no correlation was observed in MDA levels (r = 0.009, p=0.951). **Conclusion:** The industrial chemical exposures in automobile workshops and plastic industries have a widespread effect on the antioxidant system of automobile mechanics and plastic industry workers which is attributable to increased lipid peroxidation.

KEYWORDS: Total antioxidants, Malondialdehyde, Lipid Peroxidation, Plastic.

INTRODUCTION
Automobile mechanics are exposed to a wide range of chemicals, including heavy metals, contained in brake fluids, degreasers, detergents, lubricants, metal cleaners, benzene, solvents, and asbestos (from brake repair), as well as welding fumes and car exhausts.[1] The exposure to these chemical pollutants may lead to increase in the production of free radicals. The abnormal accumulation of reactive oxygen species (ROS) is the underlying pathology in a variety of human diseases such as neurodegenerative phenomena, inflammatory diseases, metabolic disorders, and cancer.[2] In low-income

countries, the number of temporary and unauthorized garages is high, and most workers are illiterate and work without any safety measures.[3] This further exacerbates the chemical exposures and potential consequences.

The overheating of plastic materials during processing, cleaning, purging and maintenance operations can expose workers to a complex mixture of combustion by-products. Some of these contaminants include: hydrogen chloride from Polyvinylchloride (PVC), styrene from polystyrene, nitrogen compounds from nylon and acrylonitrile, and cyanide from urethanes.[4] 1,3-Butadiene, a colorless gas regularly used in the production of plastics, thermoplastic resins, and styrene-butadiene rubber, poses an increased leukemia mortality risk to workers in this field.[5] Bisphenol A (BPA) can induce the production of free radicals and oxidative stress.[6] Biological monitoring of chemical exposure in the workplace has become increasingly important in the assessment of health risk as an integral part of the overall occupational health and safety strategy.[7]

Lipid peroxidation is a chain reaction initiated by hydrogen abstraction or addition of oxygen radical, which can lead to oxidative damage of polyunsaturated fatty acids.[8,9] Under physiological or low lipid peroxidation rates (subtoxic conditions), the cells stimulate their maintenance and survival through constitutive antioxidants defense systems or signaling pathways activation that upregulate antioxidants proteins resulting in an adaptive stress response.[10] By contrast, under medium or high lipid peroxidation rates (toxic conditions) the extent of oxidative damage overwhelms repair capacity, and the cells induce apoptosis or necrosis programmed cell death; both processes eventually lead to molecular cell damage which may facilitate development of various pathological states.[10] Malondialdehyde (MDA), one of several by-products of lipid peroxidation process, is a biomarker that provides an indication of lipid peroxidation level.[11,12]

The body possess a complex network of antioxidants defense system that collectively acts against free radicals. Total antioxidant capacity (TAC) is the sum of endogenous and food derived antioxidants and represents the total antioxidant activity of the system. The cooperation among different antioxidants provides greater protection against damage caused by reactive oxygen species or reactive nitrogen species, than any single compound alone. Thus, the overall antioxidant capacity may provide more relevant biological information compared to that obtained by the measurement of individual components, as it considers the cumulative effect of all antioxidants present in plasma and body fluids.[13] TAC is a sensitive and reliable marker to detect changes of in vivo oxidative stress, which may not be detectable through the measure of single "specific" anti-oxidants.[14]

MATERIALS AND METHODS

Study design

This is a cross-sectional study to evaluate total antioxidant capacity and malondialdehyde in automobile mechanics and plastic industry workers in Nnewi metropolis. A total of 150 people participated in the study. This comprised 50 automobile mechanics, 50 plastic industry workers and 50 subjects who were neither automobile mechanics nor plastic industry workers (controls). The subjects were recruited by purposive sampling and the ages ranged from 25 – 55 years.

The study was done in selected automobile workshops and plastic industries in Nnewi metropolis, Anambra State, Nigeria and the samples were analyzed in Nnamdi Azikiwe University Teaching Hospital, Nnewi, Anambra State, Nigeria. A questionnaire was used to generate necessary data from the participants. Ethical approval for this study was obtained from Ethics committee of Nnamdi Azikiwe University Teaching Hospital, Nnewi, Anambra State and informed consent was obtained from all participants enrolled in the study.

Sample size

Sample size was calculated using the method of Taro Yamane (1967) as described by.[11]

$n = N/1 + (Ne^2)$ Where:

n = sample size, N = population limit, e = error limit = 0.05,

For automobile mechanics N = 48

$n = 48/1 + (48 * 0.05^2) = 48/1.1200 = 42.8571$

For plastic insustry workers N = 45

$n = 45/1 + (45*0.05^2) = 45/1.1125 = 40.4494$

The n was raised to 50 for each group

Inclusion and exclusion criteria

Individuals recruited for the study were automobile mechanics and plastic industry workers within the age range of 25 – 55years and who have worked for a period 5years and above. Healthy individuals of matching ages who were neither automobile mechanics nor plastic industry workers participated as controls. Excluded from the study were heavy smokers and alcohol consumers, individuals with known chronic diseases, plastic industry workers likely to be minimally exposed to industrial chemicals such as administrative staff, transporters and people living very close to automobile workshops or plastic industries.

Sample collection

Seven milliliters of whole blood was collected aseptically from each participant into a plain container, allowed to clot and centrifuged at 3000 revolutions per minute for 10 minutes. Serum was extracted and stored at 2-8°c before analysis.

Statistical analysis

Statistical package for Social Sciences (SPSS) version 20.0 was used for the analysis of the data. The data was

presented as mean ± standard deviation and analyzed using a two-way Analysis of Variance (ANOVA). Relationships between groups were analyzed using the independent Students' t-test and correlation studies were done using Pearson's correlation coefficient.

Sample Analysis

MDA levels were estimated using the method as described by Gutteridge and Wilkins (1982) and the level of MDA in the serum was expressed as nmol/ml using the molar extinction coefficient for MDA (1.56x105 M-1cm-1) [15]. Total antioxidant activity was estimated by FRAP method as described by Manafa *et al.*, 2017.[16]

RESULTS AND DISCUSSION

Results

Table 1.0 Total Antioxidant Capacity and Malondialdehyde levels in automobile mechanics, plastic industry workers and control group.

Parameters	Mechanics(n=50) (mean±SD)	Plastic workers(n=50) (mean±SD)	Control(n=50) (mean±SD)	F-value	P-value
TAC (µmol/L)	884.50±146.27	829.01±158.58	994.43±164.18	14.465	0.000
MDA(nmol/mL)	2.88±0.80	3.15±0.70	2.09±0.47	14.465	0.000

There was a significant difference in the mean serum total antioxidant capacities (TAC) and malondialdehyde (MDA) levels of automobile mechanics, plastic industry workers and the control group (P<0.05).

Key:
TAC = Total antioxidant capacity
MDA = Malondialdehyde
n = no of participants

Table 2.0 Post Hoc analysis showing Total antioxidant capacities and malondialdehyde levels among mechanics, plastic workers and control group (mean±SD).

Group	TAC (µmol/L)	MDA (nmol/mL)
Control group (A) n=50	994.43±164.18	2.09±0.47
Mechanics (B) n=50	884.50±146.27	2.88±0.80
Plastic workers (C) n=50	829.01±158.58	3.15±0.70
F-test	14.465	33.643
P-Value	0.000	0.000
A vs B	0.002	0.000
A vs c	0.000	0.000
B vs c	0.235	0.139

The mean serum TAC was observed to be significantly lower in automobile mechanics compared with the control group and in plastic industry workers compared with the control group (P<0.05). A significantly higher mean MDA was observed in automobile mechanics compared with the control group and in plastic industry workers compared with the control group (P<0.05). However, no significant difference was observed in the mean serum TAC and MDA levels of automobile mechanics compared with plastic industry workers (P>0.05).

Key:
TAC = Total antioxidant capacity
MDA = Malondialdehyde
n = no of participants

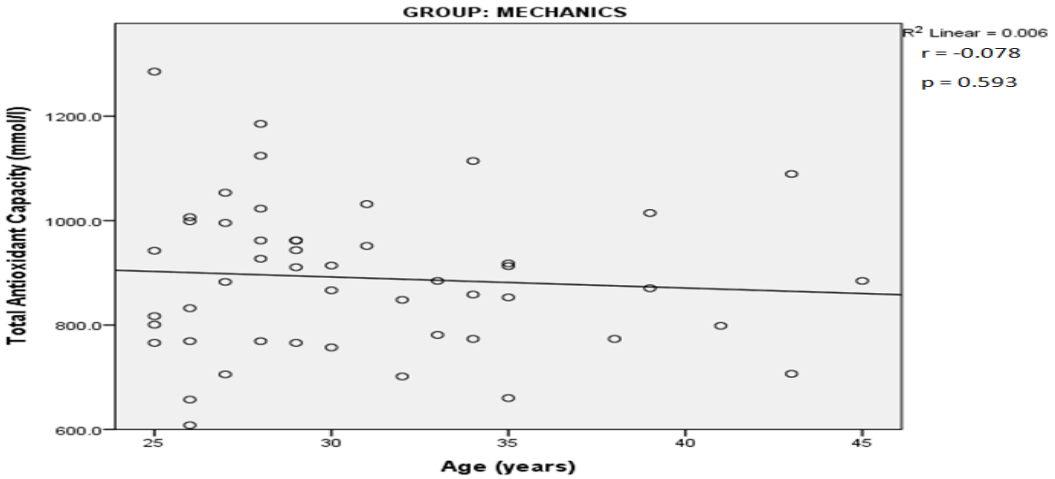

Fig 1.0 Correlation of TAC and age in automobile mechanics.

There was no correlation between the total antioxidant capacity and age of the automobile mechanics.

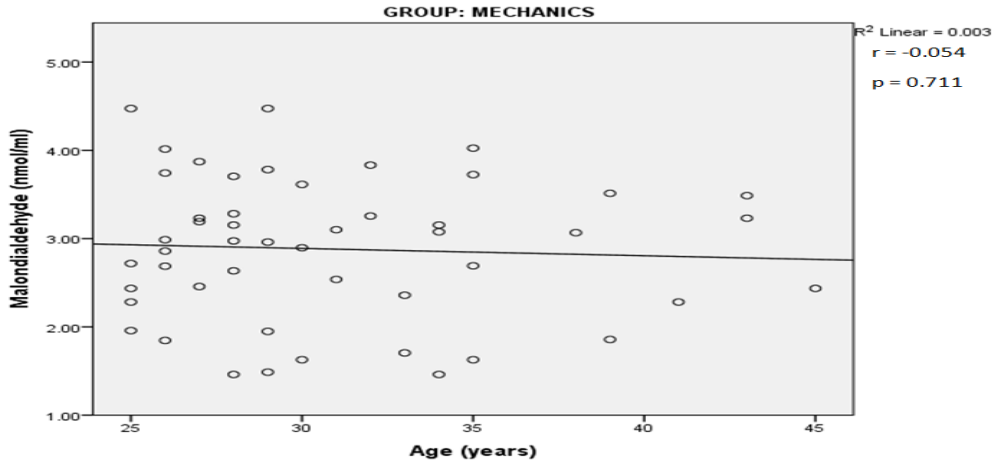

Fig 2.0: Correlation of MDA level and age in automobile mechanics.

There was no correlation between the levels of MDA and age of the automobile mechanics.

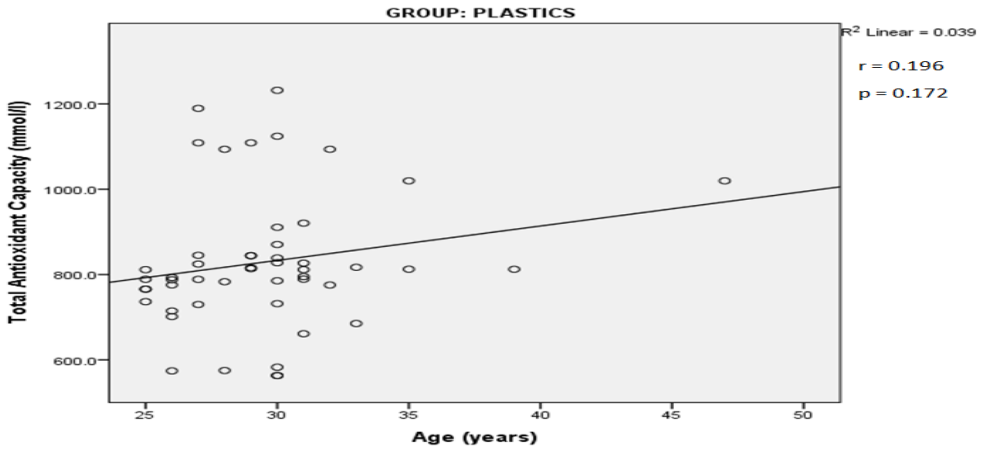

Fig 3.0: Correlation of TAC and age in Plastic industry workers.

A weak positive correlation existed between total antioxidant capacity and age of the plastic industry workers.

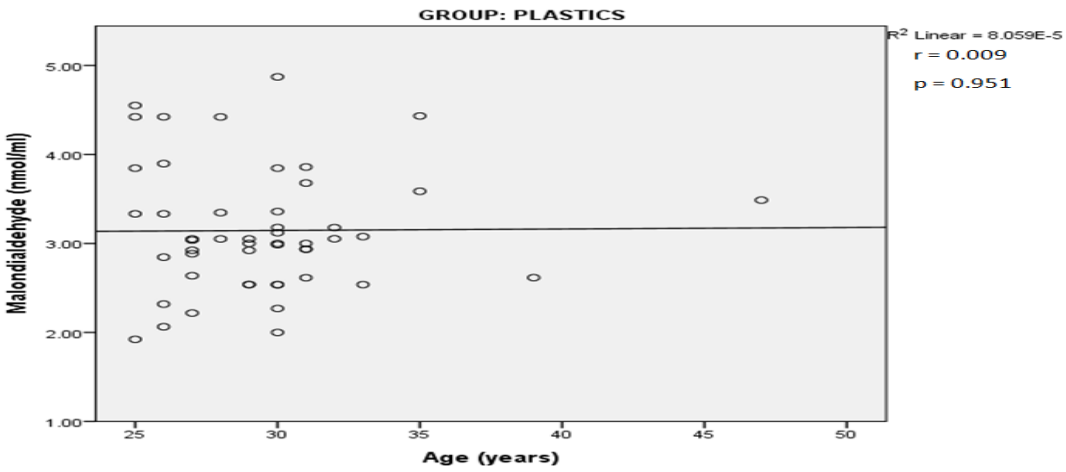

Fig 4.0: Correlation of MDA levels and age in plastic industry workers.

There was no correlation between the levels of MDA and age of plastic industry workers.

DISCUSSION

In this work, the mean TAC was observed to be significantly lower in both automobile mechanics and plastic industry workers compared with the control group. The increase in lipid peroxidation leads to changes in the antioxidant defense system which includes enzymes and nonenzymatic molecules. Lipid peroxidation (LPO) is one of the pathological effects of ROS that is associated with oxidation of membrane poly unsaturated fatty acid (PUFA) and causes damage to the lipids.[17,10] An agreement exists between this work and that of Dongre *et al.* (2011) who observed a decrease in TAC of automobile mechanics compared with the control group in their study to determine the activities of some antioxidant enzymes in automobile mechanics.[18] The decreased TAC observed in automobile mechanics in this work is in disagreement with the work of Arinola *et al.* (2006) which found no significant difference in total antioxidant capacity of automobile mechanics in Ibadan, Nigeria compared with the control group.[19] However, Arinola *et al.* (2006) suggested that the lack of significant difference in TAC may be due to compensation for low vitamin E or response to increased Pb and Cd observed in the same subjects.[19] Exposure to styrene results in its absorption into the body, where it is rapidly metabolized and eliminated. Styrene produces lung and liver damage that may be related to oxidative stress.[20] Most of the absorbed styrene is metabolized to styrene oxide.[21] Interestingly, inhalational exposure to styrene, ethylene glycol and other solvents has been found to be associated with increased free radicals imposing oxidative stress on cell.[22,23] The biological targets for highly reactive oxygen species are DNA, RNA, proteins and lipids.[21] Styrene-7,8-oxide, the main intermediate and highly reactive metabolite of styrene, may directly produce protein, RNA and DNA adducts.[24] The decreased TAC found in automobile mechanics and Plastic industry workers in this work supports the report of Ferrari, (2012) which stated that some occupational exposure can decrease TAC, rendering subjects less resistant to oxidative and nitrosative injuries and subsequent diseases.[25] In a study of a Taipei population group, Taiwan, exposure to arsenic was found to induce oxidative stress along with an inverse association between arsenic blood levels and TAC.[26]

The mean MDA level was found to be significantly higher in automobile mechanics and plastic industry workers compared with the control group ($P<0.05$). This may be consequent on the reduced TAC observed in automobile mechanics and plastic industry workers respectively. An increase in the production of free radicals unaccompanied by sufficient antioxidant capacity results in the overproduction of MDA.[27, 28] The increased MDA levels observed in this work agrees with the work of Dongre *et al.* (2011) who reported a serum lipid peroxides (MDA concentration) levels which were significantly increased in automobile workers compared with the controls.[18]

The positive correlation observed between TAC and age in plastic industry workers may be due to compensatory phenomenon acquired from years of industrial chemical exposures in the plastic industry. Limberaki *et al.* (2012) in a study found that total serum antioxidant status increases with age.[29] Nandana *et al.* (2017), in contrast reported a significant decrease in TAC with age.[30] The difference in the finding of Nandana *et al.* (2017) and this study may be attributed to other factors which can influence TAC such as the nutritional habits and inapparent health challenges of the participants enrolled for the respective studies.[30]

CONCLUSION

From the results obtained, it can be concluded that industrial chemical exposures in automobile workshops and plastic industries have a widespread effect on the antioxidant systems of automobile mechanics and plastic industry workers which is attributable to increased lipid peroxidation.

ACKNOWLEDGEMENT

Authors are grateful to everyone who participated in the work.

REFERENCES

1. El-Saadawy MS, Attwa EM, El-Tayeb IM, Zalat MM. Dermatoses and hematological disorders among car mechanics in Zagazig city and their effects on quality of life. *Zagazig University Medical Journal*, 2011; 17(2): 142–156.
2. Lee SF, Pervaiz S. Assessment of oxidative stress-induced DNA damage by immunoflourescent analysis of 8-oxodG. *Cell Biology*, 2011; 103: 99 - 113.
3. Basu R, Biswas A, Biswas K, Mukhopadhyay A, Talapatra SN, Ray SS, Ram SS, Sudarshan M, Dasgupta A, Chakrabarti S. An attempt to search the health status of garage workers: a neglected part in India. *International Journal of Advance Research*, 2015; 3(7): 1466–1471.
4. DeMatteo R. Chemical exposure and plastics production: issues for women's health | A Review of literature. *National Network on Environments and Women's Health*, 2011; 1.
5. Barajas TRL, Domínguez CMD, Borjas GC, Ramírez DML, Magaña TMT, González GJR. 1,2:3,4-Diepoxybutane Induces Multipolar Mitosis in Cultured Human. Lymphocytes. *Cytogenetic and Genome Research*, 2016; 148(2-3): 179 – 184.
6. Gassman NR. Induction of oxidative stress by bisphenol Aand its pleitropic effecys. *Environmental and Molecular Mutagenenesis*, 2017; 58(2): 60 – 71.
7. Ataro Z, Geremew A, Urgessa F. Occupational health risk of working in garages: comparative study on blood pressure and hematological parameters between garage workers and Haramaya University community Hara, Eastern Ethopia, 2018.
8. Schneider C. An update on products and mechanisms of lipid peroxidation. Molecular

Nutrition and Food Research, 2009; 53(3): 315-21.
9. Nam T.G. Lipid peroxidation and its toxicological implication. *Toxicology Res*earch, 2011; 27(1):1-6.
10. Antonio A., Mario F.M., and Sandro A., Lipid Peroxidation Products in Human Health and Disease. *Oxidative Medicine and Cellular Longevity*, 2014;
11. Singh Z, Karthigesu I, Singh P, Kaur R. Use of Malondialdehyde as a Biomarker for Assessing Oxidative Stress in Different Disease Pathologies: a Review (OPEN ACCESS). *Iranian Journal of Public Health*, 2014; 43(3): 7 -16.
12. Bhale D, Dhanashri S, Patil DS, Mahat R. Study of Malondialdehyde (MDA) As a Marker of Oxidative Stress in Obese Male Individuals. *International Journal of Recent Trends in Science And Technology*, 2014; 10(1): 51 – 52.
13. Rani A.J., Mythili S.V. Study on Total Antioxidant Status in Relation to Oxidative Stress in Type 2 Diabetes Mellitus. *Journal of Clinical and Diagnostic Research*, 2014; 8(3): 108 - 110.
14. Ghiselli A, Serafini M, Natella F, Scaccini C. Total antioxidant capacity as atool to assess redox status: critical view and experimental data. *Free Radical Biology* and *Medicine*, 2000; 29(11): 1106 - 1114.
15. Gutteridge J, Wilkins S. Copper dependant hydroxyl radical damage to ascorbic acid; formation of thiobarbituric acid reactive products. *Federation of European Biochemical Societies Letters*, 1982; 137(2): 327 - 330.
16. Manafa PO, Okafor CC, Okeke CO, Chukwuma GO, Ibeh NC, Ogenyi SI, Nwene EK, Aneke JC. Assessment of Superoxide dismutase activity and total antioxidant capacity in adult male cigarette smokers in Nnewi metropolis, Nigeria. *The Journal of Medical Research*, 2017; 3(1): 23 – 26.
17. Fraczek M, Szkutnik D, Sanocka D, Kurpisz M. Peroxidation components of sperm lipid membranes in male infertility. *Ginekologia Polska*, 2001; 72(2): 73-79.
18. Dongre NN, Suryakar AN, Patil AJ, Ambekar JG, Rathi DB. Biochemical effects of lead exposure on systolic & diastolic blood pressure, heme biosynthesis and hematological parameters in automobile workers of north karnataka (India). *Indian Journal Clinical Biochemistry*, 2011; 26(4): 400 - 406.
19. Arinola OG, Akiibinu MO. The levels of antioxidants and some trace metals in Nigerians that are occupationally exposed to chemicals. *Indian Journal of Occupational and Environmental Medicine*, 2006; 10(2): 65 - 68.
20. Meszka-Jordan A , Mahlapu A, Soomets U, Carlson G.P. Oxidative Stress Due to (R)-Styrene Oxide Exposure and the Role of Antioxidants in Non-Swiss Albino (NSA) Mice. *Journal of Toxicology and Environmental Health*, 2009; 72(10): 642 - 650.
21. Cabiscol E, Tamarit J, Ros J. Oxidative stress in bacteria and protein damage by reactive oxygen species. *International Microbiology*, 2000; 3(1): 3 – 8.
22. Sadi G, Eryilmaz N, Tütüncüoğlu E, Cingir S, Güray T. Changes in expression profiles of antioxidant enzymes in diabetic rat kidneys. *Diabetes/Metabolism Research and Reviews*, 2012; 28(3): 228 - 235.
23. Dlugosz A, Sawicka E., Marchewka Z. Styrene and ethylene glycol have a synergetic effect on lipid peroxidation that is better protected than repaired by CoQ10. *Toxicology In Vitro*, 2005; 19(5): 581 – 588.
24. Migliore L, Naccarati A, Zanello A, Scarpato R, Bramanti L, Mariani M. Assessment of sperm DNA integrity in workers exposed to styrene. *Human Reproduction*, 2002; 17(11): 2912 - 2918.
25. Ferrari CK. Effects of xenobiotics on total antioxidant capacity. *Interdisciplinary Toxicology*, 2010; 5(3): 117 – 122.
26. Wu MM, Chiou HY, Wang TW, Hsueh YM, Wang IH, Chen CJ, Lee TC. Association of blood arsenic levels with increased reactive oxidants and decreased antioxidant capacity in a human population of Northeastern Taiwan. *Environmental Health Perspective*, 2001; 109(10): 1011 – 1017.
27. Kurutas EB. The importance of antioxidants which play the role in cellular response against oxidative/nitrosative stress: current state. *Nature Journal*, 2016; 15(1): 71.
28. Cherian DA, Peter T, Narayanan A, Madhavan SS, Achammada S, Vynat GP. *Journal of Pharmacy and Bioallied Sciences*, 2019; (Suppl 2): 297 - 300.
29. Limberaki E, Eleftheriou P, Vagdatli E, Kostoglou V, Petrou CH. Serum antioxidant status among young, middle-aged and elderly people before and after antioxidant rich diet. *Hippokratia*, 2012; 16(2): 118 - 123.
30. Nandana M, Tanmay B, Suchetha K. (A Study on Effect of Ageing on the Levels of Total Antioxidant and Lipid Peroxidation. *International Journal of Contemporary Medical Research*, 2017; 4(12): 2454 - 7379.