

Monitoring and Effect Analysis of Environmental Pollution on Oxidation Stress : A Review

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ABSTRACT

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Oxidative stress is well known to be involved in the pathogenesis of lifestyle-related diseases, including atherosclerosis, hypertension, diabetes mellitus, ischemic diseases, and malignancies. Oxidative stress has been defined as harmful because oxygen free radicals attack biological molecules such as lipids, proteins, and DNA. However, oxidative stress also has a useful role in physiologic adaptation and in the regulation of intracellular signal transduction. In this paper we are presenting review of literature related to analysis of stress oxidation, biomarkers and environment hazards and their treatments.

Keywords : Oxidative Stress, Free Radicals, Active Oxygen, Biomarkers

I. INTRODUCTION

Numerous epidemiological studies have shown an increased morbidity and mortality due to environmental air pollution. Environmental air does contain a complex mixture of toxics, including particulate matter (PM), irritant gases, and benzene. The chemical composition of particles does vary greatly and depends on numerous geographical, meteorological, and source-specific variables. Generally, environmental particles include inorganic components (sulfates, nitrates, ammonium, chloride, and trace metals), elemental and organic carbon, biological components (bacteria, spores, and pollens), and adsorbed volatile and semivolatile organic compounds. In addition, environmental particles, when mixed with atmospheric gases (ozone, sulfur

nitric oxides, and carbon monoxide) can generate environmental aerosols. Particles are usually defined as PM₁₀ and PM_{2.5} with diameter less than 10 and 2.5 μm , respectively. Any fraction may have different effects; that is, PM with aerodynamic diameter less than 10 to 2.5 μm does generate a bigger amount of hydroxyl radical due to the heavy metals adsorbed on the pores and surfaces of the particles, whereas particles of larger size (PM₁₀) deposit mainly in the upper airways and can be cleared by the mucociliary system. Recently, however, interest has also focused on the ultrafine particles (UFPs) with a diameter less than 100 nm; UFPs are considered important with respect to health effects because of their very high alveolar deposition fraction, large surface area, chemical composition, and ability to enter into the circulation and induce inflammation. Vehicle emissions, in particular related to diesel engines,

diesel exhaust particles (DEPs), are a major source of environmental UFPs, which in the presence of poor ventilation may penetrate indoor, where additional sources including environmental tobacco smoke, cooking, burning of candles, and chemical reactions are present. Long-term exposure to high levels of such particles can increase risk of cancer, respiratory diseases, and arteriosclerosis, whereas short-term exposure peaks can cause exacerbation of bronchitis, asthma, and other respiratory diseases as well as changes in heart-rate variability. The general consensus does indicate that the mechanism of air pollution-induced health effects involves an inflammation related cascade and oxidation stress both in lung, vascular, and heart tissue. Inflammation is initially a protective mechanism which removes the injurious stimuli and produces reactive oxygen species (ROS) able to induce cell killing. In the early phase of inflammation, oxidant stress does not directly cause cell damage and can induce the transcription of stress defense genes including antioxidant genes. This preconditioning effect of ROS enhances the resistance against future inflammatory oxidant stress and promotes the initiation of tissue repair processes. The additional release of cell contents amplifies the inflammatory process and consequently can induce tissue injury. Oxidation damage has been implicated in many degenerative and non-degenerative diseases, including cardiovascular and pulmonary diseases, diabetes, and Alzheimer disease. Oxidation stress derived from an unbalance between ROS formation and individual antioxidant activity potentially does lead to damage of lipids, proteins, and macromolecules such as DNA and RNA.

In this literature survey we are presenting some of the works done in past related to effects of oxidative stress on various cells, there positive and negative impacts. Detailed study of each literature paper has been put to justify the scope and present development in the field of antioxidants.

II. LITERATURE SURVEY

Roopesh Singh Gangwar et al (2020) the research paper provided an overview of the impact of particulate and gaseous pollutants on oxidative stress from human and animal. The research discussed current gaps in knowledge and evidence to date implicating the role of oxidative stress with an emphasis on inhalational exposures and stated that with the identification of gaps, and an exhortation for further studies to elucidate the impact of oxidative stress in air pollution mediated effects.

The conclusion derived from the research stated that oxidative stress was a critical intermedator in the transduction of systemic toxicity associated with air pollution exposure. The role of endogenous antioxidant defenses particularly, with chronic exposure needed further exploration. The importance of personal protective measures in reducing air pollution exposure and their effects on key oxidative stress pathways and anti-oxidant defense mechanisms are important areas in future research.

D. Wilhelm Filho et al (2001) the research paper analyzed the livers of *Geophagus brasiliensis* collected from both a non-polluted site and a polluted site were analyzed for different antioxidant defenses, O₂ consumption, thiobarbituric acid-reactive substance (TBARS) levels, and histological damage. Compared to controls (116.6 ± 26.1 nmol g⁻¹), TBARS levels were enhanced at the polluted site (284.2 ± 25.6 nmol g⁻¹), as also was oxygen consumption (86.6 ± 11.3 and 128.5 ± 9.8 $\mu\text{mol O}_2 \text{ min}^{-1} \text{ g}^{-1}$, respectively). With respect to enzymatic antioxidants, increased catalase activities (8.7 ± 1.3 and 29.2 ± 2.4 mmol min⁻¹ g⁻¹, respectively), unchanged superoxide dismutase activities (767.2 ± 113.3 and 563.3 ± 70.2 U g⁻¹, respectively), and diminished glutathione S-transferase activities (29.0 ± 3.2 and 14.9 ± 3.2 $\mu\text{mol min}^{-1} \text{ g}^{-1}$, respectively) were detected. Reduced glutathione (1.91 ± 0.17 and 1.37 ± 0.25 mM,

respectively), oxidized glutathione (1.50 ± 0.20 and 0.73 ± 0.17 mM, respectively), and total glutathione (3.40 ± 0.26 and 2.07 ± 0.27 mM, respectively) concentrations were also below control values at the polluted site.

The analysis led to the conclusion that the seasonal influences need to be better understood in order to make further inferences regarding antioxidant defenses and biotransformation enzymes, especially in thermoconformers such as most fish and aquatic invertebrates living in non-polluted or polluted environments. Time of exposure and pollution levels seem to determine quantitatively the kind of response regarding biotransformation enzymes and antioxidants in fish, and this response depends therefore on the functional capacity of the organ and tissues involved. The use of biochemical indicators in environmental pollution studies at a lower organizational level such as in the present study is of high toxicological relevance, and oxidant-mediated responses are useful indices of environmental quality.

Achuba Fidelis Ifeakachuku et al (2014) the research paper investigated Oxidative stress biomarkers: levels of Lipid peroxidation as well as changes in catalase and superoxide dismutase activities in tissues of African catfish, *C. heterobranchus* inhabiting Warri River. Lipid peroxidation products in fish from the midstream and downstream parts of the river were significantly ($P < 0.05$) different from fish collected from upstream. Similarly, lipid peroxidation products in tissues of fish from midstream and downstream parts of the river were significantly ($P < 0.05$) different from fish in the reference hatchery. No significant difference was observed between fish in the upper part of the river and those from reference hatchery. Similar to lipid peroxidation, the activities of antioxidant enzymes, catalase and superoxide dismutases (SOD) were significantly ($P < 0.05$) different in fish from midstream and down- stream

parts of the river compared to fish collected from upstream and reference hatchery.

It was relevant to conclude that a polluted environment could result in increase lipid peroxidation, superoxide dismutase and catalase activities in tissues of *C. heterobranchus*. On the whole, the results presented suggest that environmental pollution could act as a mediator in the induction of oxidative stress in *C. heterobranchus*.

Soad M. Mohy El-Din (2020) the research paper investigated the toxicity and cellular stresses of nonylphenol and bisphenol to *Nannochloropsis salina*. The endocrine-disrupting compounds, nonylphenol and bisphenol attracted much attention because of their estrogenic activity and widespread environmental distribution. Both nonylphenol and bisphenol showed inhibited growth of *N. salina*, but with different degrees, in which the effective concentrations of nonylphenol 1.5 mg/l and bisphenol 10 mg/l for 5 days. Oxidative stress was induced in algal study when exposed to both endocrine-disrupting compounds as evidenced by increased malondialdehyde content than in untreated cells. Exposure also resulted in an over-expression of oxidative stress parameters was significantly affected, showing an increase in the antioxidant activities (catalase, superoxide dismutase and ascorbate peroxidase) and oxidative damage to lipids.

The results of the investigation stated endocrine-disrupting compounds and its oxidative stress efficiently increased the individual fatty acids to meet the biodiesel standards, so the promising biodiesel feedstock from *Nannochloropsis salina*. Also, the oxidative stress directly affected morphological and ultrastructure of cells.

Zaira Leni et al (2020) Air pollution remains a major factor for adverse health effects and premature death worldwide. Particulate matter with aerodynamic

diameter $<2.5 \mu\text{m}$ (PM_{2.5}), mainly originating from combustion processes, is considered most toxic. The respiratory and cardiovascular system are particularly affected. Despite all research efforts, the causative relations of air pollutants and exposure-associated health effects are not yet fully established. Recent studies using different methodologies have consistently shown peroxides and reactive oxygen species (ROS) to be crucial mediators of particle toxicity.

This review was an excerpt of results from experimental studies and methodological developments of the past 2 years that enhanced our understanding of oxidative molecules in particles, their transmission to the target organ, and the molecular pathways generating ROS in physiological and pathological processes. Further multidisciplinary research towards predicting toxicology from particle-related ROS transmitted to the target organ was required.

Frank J. Kelly and Julia C. Fussell (2015) Data strongly suggest that effects have no threshold within the studied range of ambient concentrations, can occur at levels close to PM_{2.5} background concentrations and that they follow a mostly linear concentration–response function. Having firmly established this significant public health problem, there has been an enormous effort to identify what it is in ambient PM that affects health and to understand the underlying biological basis of toxicity by identifying mechanistic pathways—information that in turn will inform policy makers how best to legislate for cleaner air. Another intervention in moving towards a healthier environment depends upon the achieving the right public attitude and behaviour by the use of optimal air pollution monitoring, forecasting and reporting that exploits increasingly sophisticated information systems. Improving air quality is a considerable but not an intractable challenge. Translating the correct scientific evidence into bold, realistic and effective

policies undisputedly has the potential to reduce air pollution so that it no longer poses a damaging and costly toll on public health.

Bernd Niemann et al (2017) the research paper reviewed In this paper, the specific role of several cardiovascular risk factors in promoting oxidative stress: diabetes, obesity, smoking, and excessive pollution. Specifically, the risk of developing heart failure is higher in patients with diabetes or obesity, even with optimal medical treatment, and the increased release of ROS from cardiac mitochondria and other sources likely contributes to the development of cardiac dysfunction in this setting. Here, we explore the role of different ROS sources arising in obesity and diabetes, and the effect of excessive ROS production on the development of cardiac lipotoxicity. In parallel, contaminants in the air that we breathe pose a significant threat to human health. This paper provides an overview of cigarette smoke and urban air pollution, considering how their composition and biological effects have detrimental effects on cardiovascular health.

Conclusion stated the biological mechanisms underlying the cardiovascular actions of smoking and air pollution must still be fully established, there is a clear role for oxidative stress as a key mediator that will exacerbate and potentially instigate the disease process. In particular, the biological pathways that link the initial lung response to air pollutants to that of the subsequent cardiovascular actions remain an important undetermined area for future research. Precise identification of the mechanisms at play will be extremely useful for identifying which constituents of air pollution are especially harmful and who is particularly susceptible to their effects. At present, though, reducing the prevalence and exposure to both these environmental risk factors remain the key means to preventing the significant burden that they inflict on health

Tania Rahman et al (2020) the research was designed to investigate the indoor environmental quality of textile industries and correlate its effect on the occupational health and well-being of the textile workers by measuring plasma oxidative stress status in textile workers and healthy control subjects. Environmental samples were collected from 15 textile industries located in Dhaka division, and 30 volunteer textile workers and 30 volunteer office workers (control) aged 18 to 57 years participated in the study. The concentration of plasma ascorbic acid (P-ASC), plasma malondialdehyde (P-MDA), and plasma conjugated diene (P-CD) was measured in both groups. The textile plants were found to have significantly elevated levels of indoor environmental pollutants compared with those in the control area, and the textile workers were significantly exposed to oxidative stresses compared with the control subjects. The use of noise pads and high-efficiency air filters is perhaps highly instrumental to put an end to this prevailing situation. Moreover, to overcome the oxidative stresses among workers, supplementation of antioxidant vitamins (ie, ascorbic acid and/or vitamin E) may be beneficial. In addition, to prevent serious health-related issues, proper precautions should be taken to protect the occupational health of the textile workers.

The conclusion indicated that textile workers were exposed to significant level of indoor environmental pollutions as evidenced by significantly higher level of noise, formaldehyde, PM2.5, and PM10. Moreover, increased level of P-MDA and P-CD along with lower P-ASC in textile workers compared with the control subjects supports the opinion that environmental pollutants cause oxidative stress. To overcome oxidative stresses, the study suggests that vitamins (like ASC and vitamin E) can be supplied to the textile workers as a source of antioxidants. It was reported that supplementation with ASC protects against increased oxidative damage. Again, supplementation of ASC was found to be effective in

reducing oxidative stress among shift workers of Tehran Shahid Tondgoyan oil refinery.

Mohammed Othman Aljahdali and Abdullahi Bal Alhassan (2020) Industrial and domestic discharges of effluent is one of the major causes of heavy metal pollution in aquatic ecosystems. Samples of benthic sediment and freshwater mollusc *Bellamya unicolor* were collected from 5 sites in the River Kaduna to determine heavy metal concentration, their ecological risk, and antioxidant enzymes activities in *Bellamya unicolor*.

The ecological risk factor (ErF) revealed that Cd made the highest contribution to pollution, recording the highest ErF (2206.41). Moreover, the results of correlation base multivariate analysis showed that urban and industrial waste were the sources of Cu and Pb in the River Kaduna. The significant positive correlation between metal concentration and antioxidants catalase (CAT) and superoxide dismutase (SOD) was established, with maximum activities of antioxidants at site S5. Results from this study have revealed potential ecological risk as a result of heavy metals pollution in the River Kaduna. Hence the need for approaches and policies be put in place to prevent the discharge of untreated industrial and domestic waste into this aquatic ecosystem.

Ochuwa O. George et al (2017) the research paper investigated the effects of two commonly used antifouling paints (Berger TBT-free (A/F783 (H)), reddish brown color and Silka Marine lead based paint, pale orange color) on a non-target catfish species, *Clarias gariepinus*. The study involved an initial 96-hour acute toxicity assay followed by chronic toxicity evaluation (using 1/10th and 1/100th 96-hour median lethal concentration (LC50) values) for 28 days to determine the ability of the paints to induce micronucleus and red blood cell abnormalities, and histopathological as well as oxidative stress effects in the catfish. Examined anti-oxidative stress enzyme

activities include superoxide dismutase (SOD), catalase (CAT), reduced glutathione (GSH) and glutathione-s-transferase (GST).

Results from the biochemical assay indicated significantly higher ($P < 0.05$) levels of a lipid peroxidation product, malondialdehyde, in Silka-exposed catfish compared to the control. All enzymes showed significantly higher activities in Berger paint-exposed catfish compared to the control. There was evidence of micronucleated and binucleated cells in the red blood cells of fish exposed to both paints. Histopathological assessment indicated that the exposed fish gills showed evidence of abnormalities such as curved lamellae epithelial necrosis, epithelial lifting and hyperplasia. The liver samples of the catfish showed evidence of portal inflammation as well as mild to severe steatosis, while the gonads showed varying percentages of follicle degeneration. The research combined an array of biomarkers to determine the negative health impacts of two commonly used antifouling paints on non-target catfish inhabiting Lagos Lagoon. Further in situ studies are recommended to determine the current status of the lagoon fish.

Wenyuan Li et al (2020) the research paper measured myeloperoxidase and 8-epi-PGF2a were loge transformed using linear regression models and linear mixed-effects models with random intercepts for myeloperoxidase and indexed 8-epi-PGF2a, respectively. Models were adjusted for demographic variables, individual- and area-level measures of socioeconomic position, clinical and lifestyle factors, weather, and temporal trend. Results found positive associations of PM2.5 and black carbon with myeloperoxidase across multiple moving averages. Additionally, 2- to 7-day moving averages of PM2.5 and sulfate were consistently positively associated with 8-epi-PGF2a. Stronger positive associations of black carbon and sulfate with myeloperoxidase were

observed among participants with diabetes than in those without.

Results suggested positive associations of short-term exposure to PM2.5 and BC with plasma myeloperoxidase and of short-term exposure to PM2.5 and SO₄²⁻ with urinary 8-epi-PGF2a. The associations of BC and SO₄²⁻ with plasma myeloperoxidase appear stronger among participants with diabetes. Results further provided evidence suggesting potential intermediate biological mechanisms that may in part explain the observed associations between transiently higher air pollution levels and the increase of acute cardiovascular events.

Athanasios Valavanidis et al (2006) the importance of free radical reactions and ROS in the physiological processes of living organisms and in the mechanisms of toxicity by exposure to a variety of environmental pollutants stimulated an explosive increase of research and applications into the field of oxidative stress caused by ROS. The resulting oxidative damage to lipids, DNA, and proteins and the adverse effects on the antioxidant, enzymatic and nonenzymatic, defense mechanisms of aerobic organisms have been used in recent years as biomarkers for monitoring environmental pollution. The current knowledge that such processes of oxidative damage occur in aquatic organisms gave the impetus to extend environmental and ecotoxicological studies to aquatic organisms as sentinels of environmental contamination by toxic chemicals.

All these studies indicate that oxidative biomarkers in combination with other types of biomarkers (hepatic, genotoxic, hematoimmunotoxic) in aquatic organisms can be useful in large-scale environmental monitoring programs.

Mohamed YM Aly et al (2020) the research was controlled to assess the harmful effects of heavy metals on the biochemical and antioxidant defense

system in Nile tilapia collected from three sectors. The studied heavy metals in gills, liver, and muscles of the Nile tilapia (*Oreochromis niloticus*) collected from the eastern and western sectors of the lake was much higher than that of fish collected from middle sector. Biochemical profile and antioxidant defense system of Nile tilapia were significantly ($P < 0.05$) higher in fish collected from western and eastern sectors during summer and winter seasons compared to the middle sector.

The results obtained from the research also revealed that the meat quality of the collected fish from middle sector was more superior to those of fish collected from the eastern and western sectors of the lake. The investigation also indicated that the alterations in the biochemistry profile and antioxidant defense system of Nile tilapia can be used as biomarkers of metal pollution for monitoring aquatic life. As for the heavy metals, biochemical and oxidative stress parameters, they were higher in the western sector, followed by the eastern and then the middle, and in the summer they were higher than in the winter in the three sectors. The liver was the most polluted organs than the gills and muscles and Zinc is higher in fish organs than other heavy metals.

Ralph J. Delfino et al (2011) chemical components of air pollutant exposures that induce oxidative stress and subsequent inflammation may be partly responsible for associations of cardiovascular morbidity and mortality with airborne particulate matter and combustion-related pollutant gasses. However, epidemiologic evidence regarding this is limited. An exposure-assessment approach is to measure the oxidative potential of particle mixtures because it is likely that hundreds of correlated chemicals are involved in overall effects of air pollution on health. Oxidative potential likely depends on particle composition and size distribution, especially ultrafine particle concentration, and on transition metals and certain semivolatile and volatile

organic chemicals. For health effects, measuring systemic oxidative stress in the blood is one feasible approach, but there is no universal biomarker of oxidative stress and there are many potential target molecules (lipids, proteins, DNA, nitric oxide, etc.), which may be more or less suitable for specific study goals.

Concurrent with the measurement of oxidative stress, it is important to measure gene and/or protein expression of endogenous antioxidant enzymes because they can modify relations

between oxidative stress biomarkers and air pollutants. Conversely, the expression and activities of these enzymes are modified by oxidative stress. This interplay will likely determine the observed effects of air pollutants on systemic inflammatory and thrombotic mediators and related clinical outcomes. Studies are needed to assess the reliability and validity of oxidative stress biomarkers, evaluate differences in associations between oxidative stress biomarkers and various pollutant measurements (mass, chemical components, and oxidative potential), and evaluate impacts of antioxidant responses on these relations.

Kai's H Al-Gubory (2014) the use of animal models is still necessary to examine the adverse effects of cumulative exposure to various ubiquitous pollutants on prenatal development. The aims of such studies are to better understand the mechanisms by which environmental pollutants adversely affect conceptus development and to elucidate the impact of cumulative exposures to multiple pollutants on post-natal development and childhood health outcomes. The adverse effect of several chemicals on prenatal development at environmental concentrations is still a matter of controversy. Therefore, the toxicity of certain chemicals has yet to be proven by further properly designed studies. Wildlife and human investigations and observations in the natural and/or altered environmental conditions will also be needed

to understand if maternal preconception exposure to multiple environmental pollutants acts synergically to adversely affect maternal reproductive health, fertility and prenatal development.

In the context of current climate change and food security, as well as the constantly fluctuating environmental pollution, minimizing environmental impacts on prenatal and post-natal development and health outcomes due to cumulative exposure to various pollutants is a challenge and requires the development of international applied research programmes and preventive strategies. The potential of dietary antioxidants as an effective treatment of oxidative stress may provide sensible therapeutic means against female infertility and adverse prenatal developmental outcome mediated by maternal exposure to environmental pollutants. In addition, communication campaigns to educate and to provide basic medical information will be necessary because a reduction in the cost of health care is an increasingly important economic imperative.

III. CONCLUSION

Here in this review paper we reviewed several journal publications which illustrate the present condition of environment factors and how to resolve them In a unique technique.

IV. REFERENCES

- [1]. Roopesh Singh Gangwar, Graham H. Bevan, Rengasamy Palanivel, Lopa Das and Sanjay Rajagopalan, [Oxidative stress pathways of air pollution mediated toxicity: Recent insights], *Redox Biology* 34 (2020) 101545.
- [2]. D. Wilhelm Filho, M.A. Torres, T.B. Tribess, R.C. Pedrosa and C.H.L. Soares, [Influence of season and pollution on the antioxidant defenses of the cichlid fish acar (Geophagus brasiliensis)], *Brazilian Journal of Medical and Biological Research* (2001), ISSN 0100-879X.

- [3]. Achuba Fidelis Ifeakachuku, Ebokaiwe Peter and Peretiemo-Clarke Beatrice O, [Effect of environmental pollution on oxidative stress in African catfish (*Clarias heterobranchus*)], *International Journal of Environmental Monitoring and Analysis*, 2014; 2(6): 297-301.
- [4]. Soad M. Mohy El-Din (2020)
- [5]. Zaira Leni, Lisa Kunzi and Marianne Geiser, [Air pollution causing oxidative stress], *Current Opinion in Toxicology* 2020, 20-21.
- [6]. Frank J. Kelly and Julia C. Fussell, [Air pollution and public health: emerging hazards and improved understanding of risk], *Environ Geochem Health* (2015) 37:631–649
- [7]. DOI 10.1007/s10653-015-9720-1.
- [8]. Bernd Niemann, Susanne Rohrbach, Mark R. Miller, David E. Newby Valentin Fuster, and Jason C. Kovacic, MD, PHD, [Oxidative Stress and Cardiovascular Risk: Obesity, Diabetes, Smoking, and Pollution], *JOURNAL OF THE AMERICAN COLLEGE OF CARDIOLOGY* VOL. 70 , NO. 2 , 2017, ISSN 0735 - 1097 / \$ 36.00.
- [9]. Tania Rahman, Ar-Rafi Md. Faisal, Tahura Khanam and Hossain Uddin Shekhar, [Recurrent Indoor Environmental Pollution and Its Impact on Health and Oxidative Stress of the Textile Workers in Bangladesh], *Environmental Health Insights* Volume 14: 1–7, 2020.
- [10]. Mohammed Othman Aljahdali and Abdullahi Bal Alhassan, [Metallic Pollution and the Use of Antioxidant Enzymes as Biomarkers in *Bellamyia unicolor* (Olivier, 1804) (Gastropoda: Bellamyinae)], *Water* 2020, 12, 202; doi:10.3390/w12010202.

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