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Monitoring and Effect Analysis of Environmental Pollution on Oxidation Stress

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ABSTRACT

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. Therefore, a more useful definition of oxidative stress may be "a state where oxidative forces exceed the antioxidant systems due to loss of the balance between them." The biomarkers that can be used to assess oxidative stress in vivo have been attracting interest because the accurate measurement of such stress is necessary for investigation of its role in lifestyle diseases as well as to evaluate the efficacy of treatment. Many markers of oxidative stress have been proposed, including lipid hydroperoxides, 4-hydroxynonenal, isoprostan, 8-hydroxyguanine, and ubiquinol- 10. To prevent the development of lifestyle diseases, advice on how to lead a healthy life should be given to individuals based on the levels of oxidant and antioxidant activity assessed by pertinent biomarkers. Individual genetic information should also be taken into consideration.

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 PM2.5 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.

This research was focused on the mechanisms of oxidative stress induction and cellular damage by air pollution exposure on pulmonary and cardiovascular systems

Components of Air Pollution

Air pollution represents a diverse mixture of substances including PM, gases (e.g., ground- level ozone, carbon monoxide, sulfur oxides, and nitrogen oxides), organic compounds (e.g., polycyclic aromatic hydrocarbons and bacterial endotoxins), and toxic metals (e.g., vanadium, lead, nickel, copper, and manganese) that can be found in outdoor and indoor air. Among these, PM and ground-level ozone, which are formed primarily from nitrogen oxides and volatile organic compounds, appear to be the most widespread and harmful components. Of those, PM is especially relevant for nervous system damage and can be found as a mixture of solid particles and liquid droplets that are suspended in the air. Most individual components of atmospheric PM are not especially dangerous and some major constituents, such as sodium chloride, are harmless. PM is characterized by its size and aerodynamic property which is directly related to its biological effects. For instance, only particles less than 10 μm in diameter can be inhaled deep into the lungs, whereas larger particles usually get trapped in the upper airways. Generally, coarse particles with an aerodynamic diameter of 2.5 to 10 μm (PM10), fine particles of less than 2.5 μm(PM2.5), and ultrafine (UFPs), or nano-sized (NP) particles of less than 0.1 μmcan be classified.

Entry of Air Pollutants into the Central Nervous System

Sustained exposure to significant levels of airborne UFPs, PM, and LPS may result in the direct

translocation of these pollutants to the CNS where they can result in neuropathology through a variety of pathways and mechanisms (Figure 1). Alternatively, air pollutants might not enter the CNS directly, but could exert adverse effect on the CNS by triggering the release of soluble inflammatory mediators from primary entry organs or secondary deposition sites. The release of inflammatory agents could then lead to or alter the susceptibility for neuroinflammation and neurodegeneration in the CNS.

Once taken up by the body, fine PM or NPs could rapidly enter the circulatory system with the potential to directly affect the vascular system. For instance, NPs could be inhaled and cross the alveolar-capillary barrier in the lungs. The ability of NPs to cross this barrier is influenced by a number of factors that include the size of the particles, their charge, their chemical composition as well as their propensity to form aggregates. Even though the translocation of inhaled or instilled NPs across the alveolar-capillary barrier has been clearly demonstrated in animal studies for a range of NPs , it has been more difficult to directly demonstrate this mechanism in humans.

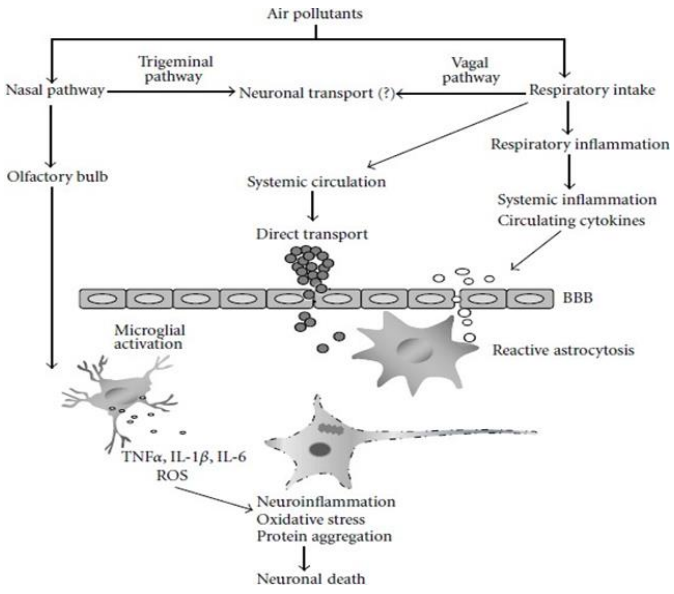

Fig 1 The impact of air pollution on the brain through multiple pathways

Objectives of the study

- Analyzing chemical and biological effect due to oxidative stress.
- Analyzing different the reactive oxygen species and their benefits.
- Descriptive analysis on air pollutants and their effects.

II. Literature Review

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 intermediator 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 g1), 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 μmol O2 min-1 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 μmol min-1 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 superoxde 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.

Direct measurement of Reactive Oxygen Species

Reactive oxygen species (ROS) are the key molecules responsible for the deleterious effects of oxidative stress. Direct measurement of their cellular levels is therefore one approach to determine oxidative stress conditions.

One way to estimate the cellular levels of ROS is through the use of fluorogenic probes. Hydrogen peroxide (H2O2), hydroxyl radicals (OH-), and peroxyl radicals (ROO-) can be measured following staining with 5-(and -6)-carboxy-2′,7′-dichlorodihydrofluorescein diacetate (DCFDA). This membrane-permeable probe diffuses into the cells where it becomes hydrolyzed by intracellular esterase to DCFH. The latter remains trapped within the cells and reacts with H2O2, generating the fluorescent 2′,7′-dichlorofluorescein (DCF). Therefore, the amount of peroxide produced by the cells can be estimated by the fluorescence intensity of DCF (λexcitation = 488 nm and λemission = 530 nm) as analyzed by flow cytometry or by employing a fluorescence plate reader. On the other hand, superoxide molecules (O2-) can be detected following

staining with another fluorescent probe, dihydroethidium (DHE). The sodium borohydride-reduced form of ethidium bromide is also permeable to viable cells. Inside the cells, DHE is directly oxidized to ethidium bromide by the superoxide anion, which then fluoresces. The red fluorescence, measured using an excitation of 488 nm and an emission of 585 nm, is therefore considered to be proportional to the intracellular superoxide anion levels. Another way to quantify ROS molecules such as hydroperoxides (R-OOH), particularly in serum, is by assessing the derivatives of reactive oxygen metabolites (D-Roms) test. In this assay, a small amount of patient serum is dissolved in an acetate-buffered solution (pH 4.8). Transition metal ions (Fe2+, Fe3+), liberated from the proteins in the acidic medium, react with hydroperoxide groups converting them into alkoxy (R-O•) and peroxy (R-OO•) radicals by way of the Fenton reaction. These newly formed radicals become trapped chemically with a chromo-gen (N,N-diethyl-para-phenylendiamine) leading to the formation of the corresponding radical cation. The concentrations of these newly formed radicals, which are directly proportional to those of the peroxides present in serum, are then determined by spectrophotometric procedures at an absorption of 505 nm.

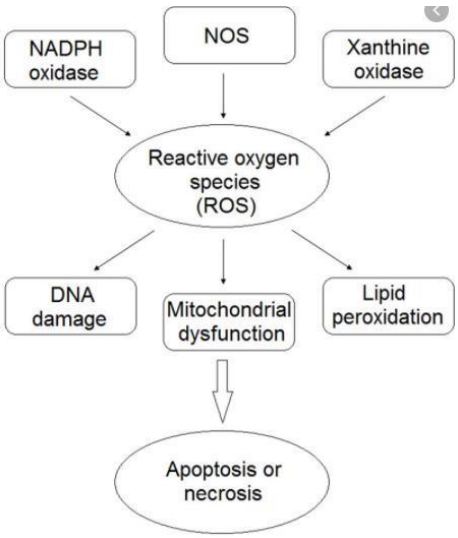

Fig 2. Reactive Oxygen Species (ROS)

The Interaction of Air Pollutants with Cells and Cellular Organelles.

Possible mechanisms by which air pollutants can interact with biological tissue depend on the size, the structure, and the composition of the componentsin thepolluted air,determining their spectrum of molecular activity and entry routes. PMs can be taken up by mammalian cells in different ways, including phagocytosis, pinocytosis, passive diffusion,receptor-mediatedendocytosis,directpenetration of the cell membrane, or transcytosis. Which route is taken largely depends on the physicochemical properties of the toxic components. PM that cannot enter cells directly could still interact with surface proteins and change cellular signalling and behavior.

There is a particular relationship between the particle size and the ways by which it can be taken up by cells. While the uptake of fine particles (0.1–2.5 μm diameter) by macro- phages is a specific receptor-mediated process (phagocytosis) the uptake of ultrafine particles (<0.1 μm diameter) can occur by other, nonspecific mechanisms. These mechanisms may include electrostatic, van der Waals, and steric interactions and are subsumed under the term adhesive interaction, although the exact mechanisms remain to be determined. As mentioned before, ultrafine PMs can cross red blood cell membranes rapidly and easily; a process that appears to be mediated by an unidentified non-phagocytic mechanism. Particles smaller than 100nm could be observed in intraluminal erythrocytes that were collected from frontal lobe and trigeminal ganglia capillaries from postmortembraintissue. UFP shave very large surface- to-volume ratio and are not enclosed by membranous organelles, which allow them to directly interact with intracellular proteins, organelles, or DNA. Such particles may reach specific organelles, such as mitochondria, lysosomes, and nuclei, where they could induce an oxidative burst within their membranes by interfering with NADPH-

oxidase activity. They may also induce the release of inflammatory mediators and cytokines by the cell. A recent study has shown that exposure to airborne UPMs is associated with mitochondrial damage, as reflected by an increase in the copy number of mitochondrial DNA (mtDNA). Damaged mitochondria may then contribute to increased oxidative- stress through altered ROS production and subsequently overloading the cell with ROSs, or by interfering with cellular antioxidant defense mechanisms.

Interaction of airborne PM with cellular proteins can also result in protein degradation and protein denaturation. Loss of enzyme activity and formation of autoantigens are possible consequences. Environmental NPs can also significantly increase the rate of protein fibrillation, whichprovides a possible link between air pollution and neurodegenerative disorders. If these findings can be confirmed under realistic in vivo conditions, it would have far-reaching consequences with respect to the mechanisms underlying neurodegenerative diseases. Other key molecular path- ways that are affected in neurodegenerative diseases lead to misfolding, aggregation, and accumulation of proteins in the brain. PMs that have the capability to enter nerve cells could contribute to these processes, so could oxidative stress that is induced by the air pollutants.

Cellular responses to oxidative stress can lead to changes in mitochondria and other organelles, notably the endoplasmic reticulum (ER), and eventually triggers the cell to enter a cell death pathway. Mitochondria, as regulators of cellular energy metabolism and apoptosis, are critical organelles in switching between different cellular responses leading to death or survival of the cell. Perturbed ER calcium homeostasis may also contribute to neuronal dysfunction and degeneration in neurodegenerative disorders. The ER is critical for early protein biosynthesis steps of secreted and membrane proteins, which occurs in the lumen of the ER,wherethe ER machinery assists in their folding.

Loss of ER homeostasis triggers stress responses, which are a hallmark of many inflammatory and neurodegenerative diseases. Recent studies have shown that exposure to airborne PM causes ER stress in lung tissue. Neurodegenerative disorders are often characterized by the aggregation and accumulation of misfolded proteins. Protein folding stress in the ER may lead to activation of the unfolded protein response (UPR). Organic DEP chemicals induce an UPR and proinflammatory effects in human bronchial epithelial cell line. However, the possible relationship between ER stress and exposure to air pollution has not been studied in the context of CNS cells. The interesting crosstalk between innate immune pathways and ER- signaling that regulates the intensity and duration of innate immune responses should also be considered in neuron flammation induced by air pollution.

III. CONCLUSION

Air pollutants have been, and continue to be, major contributing factors to chronic diseases and mortality, thereby dramatically impacting public health. Air pollution is a global problem and has become one of the major issues of public health as well as climate and environmental protection. The effects of air pollutants are thus at a high level of interest for scientific, governmental, and public communities. An in- creasing number of people are exposed to a complex mixture of inhalable NPs and toxic chemicals occupationally or as a result of manmade and natural disasters, such as war, fires, and volcanic eruptions. Air pollution is increasingly recognized as an important and modifiable determinant of cardiovascular and respiratory diseases in urban communities. Although adverse cardiopulmonary outcome shave been the focus of many studies, air pollution- related damage to the CNS has been widely neglected. However, there is mounting evidence that

air pollution also contributes to CNS damage or increased progression of neurodegenerative disorders.

The data discussed as part of this critical update high light that UFPs rapidly translocate from the lungs into the cells and into the blood circulation. There is good evidence that oxidative stress occurs in other organs, such as the heart and the brain. The breadth, strength, and consistency of the preclinical and clinical evidence provide a compelling argument that air pollution, especially traffic-derived pollution, causes CNS damage and that there is a clear link between air pollution and neurological diseases. Airborne particles cause neuropathology, which seem to be mediated by direct or indirect proinflammatory and oxidative responses. Both, the physical characteristics of the particle itself and toxic compounds adsorbed on the particle may be responsible for the damage. The time of exposure has a key role in damage. Minimum doses of pollution can be handled by the organism when this exposure is acute, but the same doses administered chronically lead to anoxidativestressstate that can produce neurodegeneration. Astroglia, cerebral endothelial cells, and microglia in particular respond to components of air pollution with chronic activation, inflammation, and oxidative stress. CNS effects can be chronic, can begin in early childhood, and may accumulate with age.

Given the enormous complexity of the CNS and the complex nature of air pollution, the resulting CNS pathology can have many underlying causes and pathways and could be due to synergistic interaction of multiple pathways and mechanisms making it difficult to pinpoint a clear stimulus- response relationship. While epidemiological data link in-creased risk for stroke, MS, and PD to the exposure to specific air pollutants, further experimental and mechanistic studies aiming at the association between the components of air pollution and the development of CNS diseases are of pressing importance for mental health .The adverse effects of the complex mixtures of polluted air components are poorly understood. For instance, the contribution of direct effects of airborne UFP sto CNS injury remains to be worked out in detail, and data on the presence of UFPs in the human CNS are still lacking to date. The biological studies can be strengthened by the use of recent discovery tools and platforms, such as proteomics and genomics, to develop biomarkers for toxicity screening. The main problems that are encountered in testing air pollutants toxicity in human Sar dosimetry, the lack of appropriate standardized protocols, and good quantitative descriptions of real-world exposure conditions. Novel detection methods need to be developed for exposure assessment and dosimetry calculation.

Oxidative stress is implicated in the natural process of aging as well as in a variety of disease states. A detailed understandingregardingthelinkbetweenoxidativestressandpathogenesis can be exploited to assess disease status as well as to develop preventive and therapeutic strategies in humans. In this review, three approaches were suggested to assess oxidative stress in clinical samples:

(1) Direct measurement of ROS levels,

(2) Detection of the resulting oxidative damage to bio-molecules (DNA, lipids, and proteins), and

(3) Determination of antioxidant status (enzymatic antioxidant activities, non- enzymatic antioxidant levels, or total antioxidant capacity).

Direct ROS determination is a valuable and promising oxidative stress biomarker that can reflect on disease status. However, as we noted earlier, their measurement in biological systems is a complex task given the short half-life and high reactivity of these species. On the other hand, “foot- prints” of oxidative stress are extremely stable and provide a more reliable approach to evaluate oxidative stress in clinical samples. While some of these modifications only reflect the local degree of oxidative stress, aftershave direct effect on the function of target molecules. This functional significance or the causal role of oxidative

modifications further highlights the clinical applicability of these oxidative stress markers. However, sample processing shouldbe performed with caution to ensure their stability and to avoidthe possibility that biomolecules may become oxidatively damaged during their isolation. As for the correlation of the antioxidant status to the state of oxidative stress in clinical samples, the measurement of individual antioxidant levels/activities could yield conflicting results. For instance, some papers report low antioxidant status in cancer samples, explaining it as a loss of their protective capacity due to high oxidative stress, while others interpret the findings of high levels/activities of the antioxidants as an adaptive response mechanism to detoxify oxidative stress-related harmful metabolites. To overcome such biases, it is advisable to determine the total antioxidant status by evaluating all antioxidants simultaneously without excluding their interactions with each other. The choice of the oxidative stress biomarkers and the methods used to measure them in order toassess oxidative status in clinical samples should be decided based on the aim of the study and its design, as well as on the clinical relevance in the selected subjects. No single parameter has yet been recommended as a gold standard for defining redox status in clinical samples. Furthermore, the individual markers described above only partially reflect on oxidative status. Therefore, an integrative approach examining both pro- and antioxidant reactions has been recently suggested to obtain a comprehensive score with higher sensitivity to physiological and pathological alternations. Global redox status indexes such as OXY-SCORE or oxidative-INDEX, computed by subtracting the antioxidant capacity from ROS levels/ROS-induced damage, or oxidative stress index (OSI), which is the ratio of total oxidant status to total antioxidant status, reflect simultaneously on oxidative and antioxidant status in clinical samples and provide a better and more powerful index in the evaluation of overall oxidative stress in clinical samples and the establishment of a definitive relationship between oxidative stress and disease status.

As a final note, to our knowledge, no adequate comparison has yet been performed between different bio- markers and the methodologies used to measure them, making it difficult if not impossible to make a reliable comparison of findings from different groups. A critical evaluation and adaptation of proposed methodologies available in the literature should therefore be undertaken prior to carrying out a proposed study, so as to enable the investigators to choose the most suitable procedure for each chosen biomarker. In addition, such a comparison will enable careful meta-analysis of multiple scientific studies related to oxidative stress.

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