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Effect of petroleum based energy sources on human health

Uma Kant Sharma,[1*] Chandra Shekhar Singh[2] and Abhay K Pandey[3]

[1]*Department of Biochemistry, R. R. P. G. College Amethi, Dr. R. M. L. University, Faizabad, U.P., India*
[2]*Department of Biochemistry, R. R. P. G. College Amethi, Dr. R. M. L. University, Faizabad, U.P., India*
[3]*Department of Biochemistry, University of Allahabad, Allahabad, U.P., India*

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ABSTRACT

Exposure of environmental pollutant, especially which are produced from petroleum fuel brings various ailments in human. They are associated with formation reactive species or free radical when inhaled by human being. These reactive species are highly unstable and carry out oxidation of biomolecules such proteins, lipids and nucleic acids. Oxidation of lipid initiates a chain reaction through which all membranous lipids are oxidized leading damage in cells. Similarly oxidation of protein as well as nucleic acid obstructs their normal functions. Free radicals are also associated with dysfunction of liver, kidney and other organs of body part. The harmful actions of free radicals are prevented by antioxidant systems of our body. Antioxidants overcome the problems which are associated with free radical either by discontinuing chain reaction or quenching of free radicals. Therefore, in present review article we have discussed about the problems associated with exposure of environmental pollutants.

1. Introduction

In present days, we are surrounded by several environmental pollutants which are petroleum based energy sources. Usually these pollutants are obtained from combustion of crude oil which may cause severe toxic effects in living organism of earth as well as of aquatic habitats. Petrol based technologies become an essential constituents of human life because of their domestic and industrial uses[1]. Air pollution is associated with increased cardiovascular and pulmonary illness as well as mortality. The mechanisms of air pollution-induced health effects encompass oxidative stress and inflammation[2]. Exposure to crude petroleum and its fractionated products causes lesions in liver and kidney hence they do not perform their normal functions. That is manifested by imbalanced level of serum electrolytes[3,4]. Several studies have reported the hepatotoxic and hematotoxic nature of petrol based fuels viz., hydrocarbon[5,6,7].

2. Generation of reactive oxygen species by environmental pollutant

Liver is the first organ that initiates the metabolism of the xenobiotic including petro products which result in formation of free radicals including reactive species of oxygen and nitrogen also. They are highly active and unstable. They make interactions with biomolecules viz., nucleic acid, protein and lipids etc. and change their native conformation leading a pathophysiological condition i.e. oxidative stress. It can trigger redox-sensitive pathways that lead to different biological processes including cell death. This pathophysiological condition can be recovered by using antioxidants [8].

3. Biomarkers of hepatic and renal damage

Several serum biomarkers viz., serum glutamate oxaloacetate and serum glutamate pyruvate transaminases, carboxyl-esterase, lactate dehydrogenase as well as alkaline and acid phosphatase[9,10], serum electrolytes[4,5] can be used to determine hepatotoxicity and renal dysfunctions. The hematotoxic and hepatotoxic effects of hydrocarbons have been reported in several literature[5,6,7]. The condition of oxidative stress is associated with several biochemical reactions such as increase in level of lipid peroxidation and decreased level of reduced glutathione. In addition the increased level of lipid peroxidation is an indicator of hepatic, renal and heart damages[11]. In several literature it has been well mentioned that,[12,13,14] hematological and biochemical indices are reliable parameter for assessment of the health status of animals. Sese et al. also reported increase in AST and ALT level after administration of Bonny light crude oil to male Chinchilla rabbits[15].

Thrombocytosis (the overproduction of platelets) another parameter, is usually a transient event and may be related to inflammation, spleen disorder, oxidative stress, reverse effects of thrombopenia, iron efficiency and some types of anemia. It is revealed by disturbances in organism hemostasis and indicates about hazardous conditions to which the organisms are exposed. Evans reported that excessive increase in platelets could be related to stimulation of megakaryocytes in the bone marrow[16].

Nelson and Cox observed elevation in plasma level of bilirubin suggests hepatic cell damage, because these cells are responsible for removing bilirubin from serum[17]. Ovuru et al. reported an increase in total serum bilirubin concentration in semi adult rabbits exposed to crude oil contaminated diet and attributed this to a metabolic disturbance in the liver arising from efective conjugation and or excretion of bilirubin[5]. Momoh and Oshin demonstrated that administration of gasoline has severe effects on hematological parameters, causes impairment of the

∗ Corresponding author. Tel.: +91-8931826240; e-mail: sharmauk85@gmail.com

liver and kidney functions and result to oxidative damage in albino Wistar rats[1]. Bondy et al. reported that metabolism of major constituents of petroleum products [e.g. gasoline, kerosene and diesel] significantly increase the generation of free radicals or reactive species in several different tissues[18].

4. Possible Mechanisms of Oxidative Stress Induced by Air Pollution Exposure

Air pollutants can exert its own specific toxicity in the several system of living being including human viz., respiratory and cardiovascular systems. Ozone, oxides of nitrogen, and suspended particulates all share a common property of being potent oxidants, either through direct effects on lipids and proteins or indirectly through the activation of intracellular oxidant pathways[19,20,21].

Air pollutants include particulate matter [especially ultrafine particles, PM< 0.1μm], oxides of nitrogen and oxygen, irritant gases, benzene and transition metal[22]. These are potent oxidants which are able to generate reactive oxygen species [ROS] viz., hydroxyl radical, superoxide anion radical, hydrogen peroxide etc. Small particulate matters (diameter less than 10 to 2.5μm) generate hydroxyl radicals which have very short half life time and are highly active[23], while larger particles are deposited mainly in the airways and can be cleared by the mucociliary system[24,25].

ROS can be generated from the surface of particles where polycyclic aromatic hydrocarbons and nitro derivative of polycyclic aromatic hydrocarbons are adsorbed, other than transition metals such as iron, copper, chromium, and vanadium that catalyzing Fenton's reaction to generate highly reactive hydroxyl radical which is more potent to induce oxidative DNA damage[26,27]. Particles bound benzo[a]pyrene has been shown to be bioavailable and can induce oxidative DNA damage in systemic target organs, including lung and kidney[28,29]. In addition it should also be noted that ozone and nitrogen dioxide are usually present together with particles in environmental air. They are also oxidants with potential effects in terms of oxidative DNA damage. Similarly, volatile compounds, such as benzene can also induce DNA oxidation[30,31].

Emission from diesel engine has diesel exhaust particle which are major source of ultrafine particles. Short-term exposure peaks can cause exacerbation of bronchitis, asthma, and other respiratory diseases as well as changes in heart-rate variability while long term exposure to high levels of such particles can increase risk of cancer, respiratory diseases, and arteriosclerosis[32-34].

Excess of reactive oxygen species results mitochondrial damage which leads activation of inflammatory cells [neutrophils, eosinophils, and monocytes] and increased production of macrophages capable of reactive oxygen species and reactive nitrogen species generation[35-37]. Initially, when oxidative stress is relatively low, various transcription factors, such as the nuclear factor erythroid-2 induce a series of antioxidant and detoxification enzymes e.g., catalase, superoxide dismutase, and glutathione S-transferase that counteract reactive species formation[38-39]. If the protective antioxidant response fails or is inadequate to deal with increasing reactive species production which leads to proinflammatory situation with various cytotoxic effects[40].

Gaseous pollutants contribute to a great extent in composition variations of the atmosphere and are mainly due to combustion of fossil fuels and to emission of motor vehicles[41]. In environmental conditions, nitric oxide is rapidly transformed into nitrogen dioxide by atmospheric oxidants such as ozone[42].

Various antioxidants act as powerful scavengers of free radicals in body fluids, likely to protect lung lining fluids against inhaled oxidizing air pollutants[43]. When such defense mechanisms are incredulous, ozone may injure the underlying cells by inducing lipid peroxidation and activating inflammatory gene expression[44].

Volatile organic compounds are a class of compounds which includes chemical species of organic nature such as benzene. Among gaseous pollutants, carbon monoxide has been pronounced as one of the main pollutants responsible for the development of cardiovascular diseases, while benzene can also induce haematological problems and cancer[45,46]. Benzene is a commonly used industrial chemical and a constituent of gasoline[31]. Inhalation is the most important route of absorption during occupation-related exposure. Benzene toxicity is attributed to its metabolism, which does lead to the formation of reactive metabolites such as hydroquinone and its oxidized form benzoquinone which are highly reactive molecules and, by means of redox cycling, produce reactive oxygen species[47]. Uzma et al. demonstrated that occupation-related exposure to benzene causes oxidative stress, immune suppression, and inducing the expression of tumour-suppressing gene p53 in gasoline filling workers. They hypothesized that the increase in the p53 expression may block the cell cycle at G1 phase and go on to repair DNA damage, which is the initial step in tumor suppression[31].

5. Air pollution induced-oxidative damage in target organs

Diesel and gasoline vehicle emissions in the urban areas have dominant contributions to environmental particles, especially those located in the ultrafine range. Because of their small size and large surface area, ultrafine particles have demonstrated unique biochemical characteristics, such as enhanced ability to adsorb or absorb organic molecules and to penetrate into cellular targets in the human pulmonary and cardiovascular systems[48,49]. Ultrafine particles may be directly transported to the cardiac vasculature, where they can induce arrhythmias and decrease coronary blood flow[50]. Brook et al.[51] demonstrated that fine particulate air pollution and ozone cause acute arterial vasoconstriction in healthy humans. Urch et al., reported that fine particles exposure pollution raise blood pressure and impair vascular function[52]. In addition, ultrafine particle exposure depresses myocardial contractile response and coronary flow in both spontaneously hypertensive and wild type rats[53]. The same observation was found by Simkhovich et al. in young and old rat hearts[54].

6. Actions of antioxidant against pollutant induced oxidative stress

The antioxidant defense system of the body can be in the form of low molecular weight antioxidants such as vitamins E and C which block free radicals, or in the form of enzymes such as superoxide dismutase, catalase, and the glutathione system [glutathione, glutathione reductase, peroxidase, and transferase] that reduce the levels of reactive oxygen species[8,55,56]. Uric acid, end product of purine metabolism, is a powerful free radical scavenger in human being. Momoh and Oshin observed a significantly increased level of uric acid in animals, administered with petroleum products[1]. The elevated level of uric acid provides protection against increased in free radical activity. They also observed a significant increase in level of malondialdehyde [MDA], a product of lipid peroxidation and

reduction in activities of superoxide dismutase [SOD], catalase [CAT] and reduced glutathione [GSH].

Antioxidants help maintain lower levels of free radicals; thus they perform beneficial physiological roles[57,58]. Initially synthetic antioxidants were used to overcome the detrimental effects of reactive species or free radicals, but they are highly toxic. Hence currently we move towards natural antioxidant especially of plant origin[59].

According to the World Health Organization[60], 80% of the population of developing countries in Asia and Africa rely on traditional medicine for primary health care. Antioxidants contribute to the inhibition of many diseases including cardiovascular diseases and cancer[61]. Polyphenols are capable of scavenging free radicals by donating a hydrogen atom and are thus considered efficient antioxidants in a wide range of oxidation systems[62]. Antioxidant enzymes constitute the first line of defense against oxidative stress and damage caused by free radicals[63-65]. In addition to their antioxidant activity, flavonoids are also known to produce antitumor activity through inhibition of proliferation, metastasis and invasive effects, induction of apoptosis, suppression of protein tyrosine kinase activity, and anti-angiogenesis[66]. The identified flavonoids and phenolic acids have been reported to possess anticancer effects against different types of cancer cells[67].

7. Conclusion

In conclusion, several experimental and epidemiological studies have proved exposure to air pollution to be an important determinant of overall pulmonary and cardiovascular risk damage and possibly have an influence on traditional risk factors. Although each environmental pollutant has its own mechanism of toxicity, most pollutants, like ultrafine particles, particulate matter, ozone, nitrogen oxides, and transition metals, are potent oxidants or capable of ROS production. Consequently, the promotion of oxidative stress has been identified as one of the most important mechanisms responsible for toxic air pollutant effects. Oxidative stress can trigger redox sensitive pathways that lead to different biological processes like inflammation and cell death. Although we can prevent and cure of disease associated with oxidative stress, but we should care about our environment to keep it clean and healthy so that in future we will not face the problem of environmental pollution.

References

1. J. Momoh and T. T. Oshin, *American J of Biochem*., **2015**, 5[1], 6.
2. M. Lodovici and E. Bigagli, *J of Toxicol*., **2011**, 2011, Article ID 487074.
3. E. Orisakwe, A. A. Njan, O. J. Afonne, D. D. Akumka, V. N. Orish and O. O Udemezue, *Int J Environ Res. Public Health*, **2004**, 1, 106.
4. F. E. Uboh, M. I. Akpanabiatu, J. I. Ndem, Y. Alozie and P. E. Ebong, *J Toxicol. Environ. Health Sci.*, **2009**, 1, 68.
5. S. S. Ovuru, N. A. Berepubo and M. B. Nodu, *Afr. J. Biotechnol*., **2004**, 3, 343.
6. M. I. George and O. A. Adegoke, *J Sci Res.*, **2011**, 3, 641.
7. A. H. Adebayo, G. Z. Zeng, Y. M. Zhang, C.J. Ji, A. A Akindahunsi, and N. H. Tan, *Afr. J Biotechnol.*, **2010**, 9, 2938.
8. G. Manda, M.T. Nechifor, and T. M. Neagu, *Current Chemical Biology*, **2009**, 3[1], 22.
9. M. G Baron, K. A Charron, W. T. Scott, and S. E. Duvall, *Environmental Toxicology and Chemistry*, **1999**, 18, 2506.
10. F. Basaglia, *Comparative Physiology and Biochemistry*, **2000**, 126, 503.
11. K. V. Sudakov, *Pathophysiol. Exp. Ther*., **1992**, 4, 86.
12. K. C Patrick-Iwuanyanwu, M. O Wegwu, and E. O. Ayalogu, *Asian J. Biochem.*, **2007**, 2, 409.
13. C. C. Ohaeri, and M. C. Eluwa, *Vet. Parasitol*., **2011**, 177, 199.
14. D. P. Sexena, S. K. Shukla, and R. Kumar, *Res. J. Med. Plant*, **2011**, 5, 312.
15. B. T. Sese, O. S. George, and N. O. Wariboko, *Food Sci. Qual. Manage*., **2013**, 14, 27.
16. G. O. Evans, Boca Raton: CRC Press, Taylor Francis., **2009**, 206.
17. D. L. Nelson, and M. M. Cox, Lehniger Principles of Biochemistry [4th ed.]. W.H Freeman and company Publication, New York, **2005**, 716.
18. S. C. Bondy, H. R. Lam, G. Ostergard, S. X. Guo, and O. Ladefoged, *Arch Toxicity*., **1995**, 69, 410.
19. H. A. Jeng, *American Journal of Physiology Lung Cell Molecular Physiology*, **2007**, 293, 170.
20. A. Valavanidis, K. Fiotakis, and T. Vlachogianni, *Journal of Environmental Science and Health C*, **2008**, 26[4], 339.
21. P. Moller and S. Loft, *Environmental Health Perspectives*, **2010**, 118[8], 1126.
22. R. M. Harrison and J. Yin, *Science of the Total Environment*, **2000**, 249[1-3], 85.
23. U. K. Sharma, A. K. Sharma, and A. K. Pandey, *Comp. and Altern. Med.*, **2015**, DOI; 10.1186/s12906-016-1147-4.
24. J. Ferin, G. Oberdorster, and D. P. Penney, *American Journal of Respiratory Cell and Molecular Biology*, **1992**, 6[5], 535.
25. Environmental Protection Agency, Air Quality Criteria for Particulate Matter, vol. III of EPA/600/P-95/001CF, National Center for Environmental Assessment, Research Triangle Park, NC, USA, **1996**.
26. J. H. Park, A. B. Troxel, R. G. Harvey, and T. M. Penning, *Chemical Research in Toxicology*, **2006**, 19[5], 719.
27. H. Andersson, E. Piras, J. Demma, and B. Hellman, *Toxicology*, **2009**, 262[1], 57.
28. K. B. Kim, and B. M. Lee, *Cancer Letters*, **1997**, 113[1-2], 205.
29. P. Gerde, B. A. Muggenburg, M. Lundborg, Y. Tesfaigzi, and A. R. Dahl, *Research Report*, **2001**, 101, 5.
30. M. Sorensen, H. Skov, H. Autrup, O. Hertel, and S. Loft, *Science of the Total Environment*, **2003**, 309[1–3], 69.
31. N. Uzma, S. S. Kumar, and M. A. H. Hazari, *American Journal of Industrial Medicine*, **2010**, 53 [12], 1264.
32. A. Peters, D. W. Dockery, J. E. Muller, and M. A. Mittleman, *Circulation*, **2001**, 103[23], 2810.
33. A. Brunekreef and S. T. Holgate, *The Lancet*, **2002**, 360[9341], 1233.
34. Y. J. Li, H. Takizawa, and T. Kawada, *Inflammation and Allergy*, **2010**, 9[4], 300.
35. A Baulig, M. Garlatti, V. Bonvallot, *American Journal of Physiology*, **2003**, 285[3], L671.
36. N. Li, C. Sioutas, A. Cho, *Environmental Health Perspectives*, **2003**, 111[4], 455.
37. N. Amara, R. Bachoual, M. Desmard, *American Journal of Physiology*, **2007**, 293[1], L170.
38. K. W. Kang, S. J. Lee, and S. G. Kim, *Antioxidants and Redox Signaling*, **2005**, 7[11-12], 1664.
39. J. D. Hayes and M. McMahon, *Trends in Biochemical Sciences*, **2009**, 34[4], 176.
40. A H. Sprague and R. A. Khalil, *Biochemical Pharmacology*, **2009**, 78[6], 539.
41. K. Katsouyanni, *British Medical Bulletin*, **2003**, 68, 143.
42. WHO, World Health Organization Regional Publications, **2001**, 91, 1.
43. W. Yang, and S. T. Omaye, *Mutation Research*, **2009**, 674[1-2], 45.

44. B. Kosmider, J. E. Loader, R. C. Murphy, and R. J. Mason, *Free Radical Biology and Medicine*, **2010**, 48[11], 1513.
45. M. L. Bell, R. D. Peng, F. Dominici, and J. M. Samet, *Circulation*, **2009**, 120[11], 949.
46. G. Barreto, D. Madureira, F. Capani, L. Aon-Bertolino, E. Saraceno, and L. D. Alvarez-Giraldez, *Environmental and Molecular Mutagenesis*, **2009**, 50[9], 771.
47. J. L. Bolton, M. A. Trush, T. M. Penning, G. Dryhurst, and T. J. Monks, *Chemical Research in Toxicology*, **2000**, 13[3], 135.
48. A. Peters, H. E. Wichmann, T. Tuch, J. Heinrich, and J. Heyder, *American Journal of Respiratory and Critical Care Medicine*, **1997**, 155[4], 1376.
49. A. A. Pope, J. B. Muhlestein, H. T. May, D. G. Renlund, J. L. Anderson, and B. D. Horne, *Circulation*, **2006**, 114[23], 2443.
50. R. J. Delfino, C. Sioutas, and S. Malik, *Environmental Health Perspectives*, **2005**, 113[8], 934.
51. R. D. Brook, J. R. Brook, B. Urch, R. Vincent, S. Rajagopalan, and F. Silverman, *Circulation*, **2002**, 105[13], 1534.
52. B. Urch, F. Silverman and P. Corey, *Environmental Health Perspectives*, **2005**, 113[8], 1052.
53. H. Hwang, R. A. Kloner, M. T. Kleinman, and B. Z. Simkhovich, *Journal of Cardiovascular Pharmacology and Therapeutics*, **2008**, 13[3], 189.
54. B. Z. Simkhovich, P. Marjoram, M. T. Kleinman, and R. A. Kloner, *Basic Research in Cardiology*, **2006**, 102[6], 467.
55. M. D. Evans, M. Dizdaroglu, and M. S. Cooke, *Mutation Research*, **2004**, 567[1], 1.
56. A. K. Sharma, S. Kumar, and A. K. Pandey, *Biochem. Anal. Biochem.*, **2014**, 2014[3], 153.
57. B. Halliwell, and J. Gutteridge, Free Radicals in Biology and Medicine, Clarendon Press, Oxford, UK, 4th edition, **2006**.
58. A. K. Sharma, U. K. Sharma, and A. K. Pandey, *Proc. Natl. Acad. Sci., India, Sect. B Biol. Sci.*, **2015**, DOI 10.1007/s40011-015-0578-x.
59. U. K. Sharma, and A. K. Pandey, *J. of Kalash sciences*, **2016**, 4[1], 7.
60. World Health Organization, Traditional Medicine, **2009**.
61. A. K. Pandey, A. K. Mishra, and A. Mishra, *Cell Mol. Biol.*, **2012**, 58[1], 142.
62. S. Kumar, and A.K. Pandey, *BMC Comp. Altern. Med.*, **2014**, 14, 112.
63. K. N. Agbafor, and N. Nwachukwu, *Biochemistry Research International*, **2011**, Article ID 459839, 4.
64. A. Mishra, S. Kumar, A. Bhargava, B. Sharma and A.K. Pandey, *Cell. Mol. Biol*., **2011**, 57[1], 16.
65. S. Kumar, U.K. Sharma, A.K. Sharma, and A. K. Pandey, *Cell. Mol. Biol*., **2012**, 58[1],171.
66. C. Kanadaswami, L.-T. Lee, P.-P. H. Lee, *In Vivo*, **2005**, 19[5], 895.
67. S. Kumar, and A.K. Pandey, *The Scientific World Journal*, **2013**, Article ID 162750.