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Effects of air pollution on Human Cells (Review)

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ABSTRACT

Air pollution has become a world problem. And India stands on third position after China and United States. [1] 2013 study on non-smokers has found that Indians have 30% lower lung function compared to Europeans. [2] Air pollution is linked to urban centres, industrial activities and road traffic. Pollutants emitted have resulted deleterious health affects by numerous epidemiological and in vitro studies. Environmental air pollutants are a mixture of particles suspended into a liquid and gaseous phase which trigger the disruption of redox homeostasis, which results in inflammation and cell death via necrosis, apoptosis and activation or repression of apoptotic pollen as an adaptive response to xenobiotic might lead to alter chronic toxicity.

INTRODUCTION

As we know that life without Air cannot be possible. But when this life saving air - gets polluted by pollutants it become fatal either life taking. Impacts of air pollution results in increased health problems which can be cardiopulmonary, cancerous, reproductive failure [3, 4]

These pollutants are produced by Industrial activities, road traffic, and agricultural activities. [5]

These are almost 200 air pollutants which are quite harmful for us out of which only six are monitored by the Environmental Protection Agency (EPA). Additionally some other air pollutants also are hazardous. Like chlorofluorocarbons, polycyclic aromatic hydrocarbons, mercury, asbestos etc. [6]

Air pollution is maped on the basis of P.M. (Particulate Matter). Particulate Matter means suspended solid or liquid droplets in the air, which varies in size. P.M.'s are usually defined as PM_{10}, $PM_{2.5}$ $PM_{0.1}$ & that corresponding to airborne particles with an aerodynamic diameter, equal or less than 10, 2.5 & 0.1 microns respectively. PM_{10} & $PM_{2.5}$ are classified as the coarse fraction ,and Fine fraction of Particles (FP) and correspond to ultra-Fine Fraction of Particles (UFP) . Along these, there are Nano Particles (NP) having at least one dimension less than 100 nm , are in the same scale in size. They have capability to directly interact with biological molecules. And some particles emitted by Diesel which are stated as Diesel Exhaust Particles (DEP).

UFP and NP have high surface reactivity that is responsible for the production of free radicles . [7]

Basically pollutants are the fragments of large particles and these particles vary in size and environmental parameters. In urban areas it may contain molecules of different solids and gases, while in countryside it may contains spores, pollens along with other pollutant molecules. [8]

The urban aerosol mainly contains FP and UFP formed by fossil fuel combustion and tamed soot. In organic components like ammonium chloride, sulphates, nitrates etc. and organic compounds like alkanoic acid ,aliphatic acid, quinone,etc[9]

FP, UFP and DEP when inhaled with air, penetrate deeply into the respiratory tract, and are deposited in trachea bronchial and alveolar region. Exposure to $PM_{2.5}$, PM_{10}, CO_2 and black smoke also causes respiratory exacerbation in both adults and children. International agency for research and cancer classifies DEP as a possible carcinogen (group 2A). [10]

Carcinogenesis comes from prolonged exposure when mucocillary and alveolar clearance functions are exceeded, PM persist into lungs leading to the thickening of bronchial walls, there is hyperplasia of goblet cell and smooth muscle cells and sub epithelial fibrosis, as in the case of COPD (Chronic Obstructive Pulmonary disease). [11]

Most of the human cancers are carcinoma, derived from epithelial cells and in the case of lung cancers, the most common causes are squamous cell carcinoma, small cell carcinoma, large cell carcinoma and adenocarcinoma. [12] During multi-stage process of tumour genesis cancerous cells acquire six capabilities.

i. Self Sufficiency in growth signals
ii. Insensitivity to growth inhibitory signals
iii. Limiting replicative potential
iv. Evasion to apoptotic cell death
v. Sustained angiogenesis
vi. Tissue invasion & metastasis. [13]

The cell death often demonstrated in experiments performed with PM in normal human lung tissue or airway epithelial cells, was mitochondria - mediated apoptosis, characterized by a marked reduction of mitochondrial dehydrogenase activity and the cytoprotective effects of mitochondrial inhibitors. [14, 15]

Some have also studied that short term exposure with high doses of PM to respiratory cells lead to a consensus that health affects as well as cytotoxic impacts of particulate pollution mainly involve ROS production and oxidative stress. [16, 17]

Due to metallic environmental pollutants ROS is generated in mitochondria as well as Electron, transfer chain and from NADPH oxidase activity. [18, 19]

In addition to organic compounds, heavy metals like Cadmium, Lead, Aluminium, Iron etc. are often found in atmosphere pollutants. Some metals are both induce of ROS and essential cofactors for some antioxidant enzymes. As reviewed transition metals promote apoptosis through ROS generation, Mitochondria dysfunction, and activation of MAPK, p53 and cascades or down regulation of anti-apoptotic proteins of Bcl-2 family. [20]

Water soluble fractions of PM causes DNA damage as a consequence of ROS generation by Fenton reaction and result in cancer and myelination. In case of Cadmium metallothionein expresion determines the cell death, which is due to inhibition of antioxidant enzymes, mitochondrial dysfunction . In addition PM containing levels of non-Carcinogenic metals like Cobalt, Lead, Iron and Zinc provoke ROS production leading to apoptosis through the mitochondrial pathways. [21-22] Zinc is also able to increase p53 expression and function probably by stabilizing the proton which contains a tightly bound zinc atom necessary for its DNA binding activity. [23, 24]

All these outcome emphasize the possible dialogue between plasma membrane alterations and cell death. Among all the water soluble compounds of PM, responsible agents for the cytoprotective affects is Zinc; known to inhibit apoptosis and minimize the oxidative stress. [25] Zinc may protect cells both directly by stabilizing lipids and proteins of cellular and organelles membranes and indirectly via the maintenance of glutathione levels. [26]

It also reduces DNA fragmentation, processing of procaspase-3. And activation of cytoprotective signalling pathways.

CONCLUSION

Exposure to environmental pollutants may result in cellular disorder responsible for tissue damages. Minor stress will induce a cellular response characterized by metabolic morphological or signalling alterations in order to deal with it. The phenomenon involves many processes, such as hypertrophy or atrophy of cells. Also, persistent exposure may result in the replacement of one cell by another as in case of smokers these are replaced by squamous cells leading to squamous cell carcinoma. Air pollution can directly affect the respiratory epithelium resulting In loss of cilia until total desquamation change.

So it is better to keep check on air pollution by government agencies ,so that the hazardous affect of pollution can be lowered to come extent, the people by themselves should adopt non-polluting ways to keep atmosphere pollution free

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