|| Print ISSN: 2589-7837 || Online ISSN: 2581-3935 ||

International Journal of Medical Science and Diagnosis Research (IJMSDR)

Available Online at www.ijmsdr.com

Volume 3, Issue 3; March: 2019; Page No. 111-114

crossref DOI: https://dx.doi.org/10.32553/IJMSDR/v3i3.20

HARMFUL EFFECTS OF AMBIENT AIR POLLUTION ON DEVELOPING CENTRAL NERVOUS SYSTEM OF GROWING CHILDREN: A NARRATIVE REVIEW

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Type of Publication: Review Article

Conflicts of Interest: Nil

Abstract:

Air pollution affects developing nervous system of growing children in various ways and it has adverse effects on cognition, mood and behaviours of children. Urban residents suffer from the harmful effects of air pollution more than rural children. Whenever the natural epithelial barriers are breached such as nasal, skin and blood brain barrier. How these toxicants in polluted air damage the developing nervous system is not fully elucidated. The most plausible hypothesis proposed is that neuroinflamamtion produced by these pollutants accelerate apoptosis and other cell death processes and thereby neuronal loss and cognitive impairment subsequently. Although the neuroinflammation caused by these pollutants often causes damage to the central nervous system of all age groups, but children are more prone to toxic effects of these pollutants. Carbon monoxide, nitrogen oxide and dioxide and lead are some of these pollutants. Various countries are trying several legislations and innovations to reduce air pollution, especially developed countries of Western world.

Keywords: air pollution; children; central nervous system

Introduction:

Air pollution refers to a mixture of chemicals, particulate matter (PM) and biological material in the air which can lead to harm and/or discomfort to living organisms including human beings. The source of air pollution can be natural (eg. Volcanic eruptions) or more commonly man-made (industrial, exhaust fumes).(1)

There are already countless hurdles to overcome in the outside world for a child. Air pollution has been added to these countless obstacles merely due to human errors in recent past. Mainly there are three types of air pollutant that cause environmental and health problems:

Particulate matter (PMs): The most dangerous tiny particles of air pollution can penetrate deep into our lungs, and can even get into the bloodstream. Particulates worsen heart and lung disease. Fine particle air pollution is responsible for 29,000 early deaths a year in the UK.

Nitrogen dioxide (NO2): A toxic gas that you might sometimes notice as an orange haze over a city. High levels of NO2 can cause a flare-up of asthma or symptoms such as coughing and difficulty breathing.

Ground level ozone (O3): Ground level or "bad" ozone is created by chemical reactions between oxides of nitrogen (NOx) and volatile organic compounds (VOC) in the presence of sunlight. It can irritate the eyes, nose and throat.

National Ambient Air Quality Standards(1997) define six “criteria air pollutants”: ozone, particulate matter, sulphur dioxide, nitrogen dioxide, carbon monoxide and lead.(2) Particulate matter and ground level ozone, formed predominantly from nitrogen oxide and volatile oxygen compounds are the most widespread and harmful of all the components of air pollution. Particulate matter can be classified on the basis of aerodynamic properties and size which has physiological relevance when studying effects in

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humans. Particles with size > 10 µm diameter are trapped in upper airways and do not reach the lungs. Coarse (2.5-10 µm), fine (<2.5 µm) and ultrafine particles (0.1 µm) particles reach the lungs.(1) Smaller particles (<2.5 µm) can readily cross the epithelial, endothelial and blood brain barrier. (2)

Neurotoxicity: Mechanisms and effects

The air pollutants gain entry into the body primarily through inhalation. After inhalation these particles can reach the olfactory bulb through the nasal mucosa. Alternatively, these can reach the alveolar surface; cause respiratory inflammation and breach of respiratory epithelium, with subsequent systemic release of inflammatory mediators. Blood brain barrier may be damaged secondary to the release of inflammatory mediators or by the direct access of the noxious agents to the blood brain barrier. The final common pathway for all these process is oxidative stress in the brain leading to neuroinflammation and neuronal cell

death (2).

Cellular & molecular mechanisms of neuronal injury

Interactions between air pollutants & cells/cellular organelles: The noxious agents (particulate matter) is taken up inside the cells by one of the following mechanisms depending on the size and characteristics of the PM- receptor mediated endocytosis, phagocytosis, pinocytosis, transcytosis (direct penetration of cell membrane) or passive diffusion. These then reach the various intracellular organelles like mitochondria, lysosomes & nucleus followed by various biochemical alterations. It may interact with NADPH oxidase resulting release of inflammatory mediators and cytokines. Mitochondrial damage may lead to increase in number of mitochondrial DNA with resulting alteration in the production of reactive oxygen species and cellular defence functions. Change in the oxidative stress leads to damage to other intracellular organelles, most notably the endoplasmic reticulum (ER) which can trigger cell death and apoptosis.

Neuronal and glial cell death can occur due to mitochondrial damage as detailed above or due direct neuronal and astroglial damage. It has been reported on high dose exposure to ozone; both neuronal apoptosis and necrosis can occur.Neuronal inflammation further leads to neuroglial activation with release of ROS, chemokines, nitrous oxide, reactive nitrogen species with progressive neuronal cell loss.

It is to be noted that brain is more susceptible to oxidative stress injury due to increased rate of metabolism, low activity of antioxidant enzymes (superoxide dismutase and catalase), low concentrations of endogenous radical scavengers and increased content of redox metals (copper & iron) which act as catalyst for reactive oxygen species (ROS) production.

Genetic susceptibility is also postulated to play role in the extent of air pollution associated adverse outcomes as different individuals respond differently to the same environmental toxins. Additionally, various pollutants can result in various epigenetic modifications of genes (eg- glutathione-S-transferase gene- for scavenging reactive oxygen species) resulting in altered cellular responses.

Polycyclic aromatic hydrocarbons (PAH), found in high concentrations in urban areas with high pollution index are potent carcinogens and have multiple endocrine and CNS effects. PAH is associated with early systemic dysregulation, brain and intrathecal inflammation, production of vasospastic substances and autoantibodies to various neuronal antigens. These also lead to abnormalities in the integrity of neurovascular unit, neural, olfactory, gastrointestinal and alveolar epithelium. It has been observed that it is also associated with accumulation of hyper phosphorylated tau protein, alpha synuclein and beta amyloid in anatomical distribution similar to that seen in early Alzheimer's disease and parkinsonism.(3)

CNS effects of air pollution

After exposure to air pollutants, a widespread inflammatory response is generated in the body, including neuroinflammation. Children are particularly predisposed to these effects: higher respiratory rate to body size ratio with consequent more air consumption per unit body weight as compared to adults – hence, higher exposure. The structures most commonly affected in children include prefrontal and frontal cortices and olfactory bulb, midbrain and hippocampus resulting in cognitive abnormalities. This inflammatory cascade has also been observed to result in areas of white matter hyperintensities or areas of demyelination resulting from reduced blood flow. This might impair with proper formation of synapses. In combination, these factors lead to cognitive deficits in otherwise healthy children.

Vehicular exhaust fumes, which are an important source of particulate matter pollution in developing and developed countries alike, have been found to be associated with higher executive function deficits in preschool (2-5 year) and school aged (6-14 year) children.

Adverse pregnancy outcomes

Increased air pollution exposure has been associated with premature delivery, low birth weight babies, intrauterine growth retardation, small for gestational age babies and abnormal length and head circumference at birth. (4)

Birth defects

Antenatal carbon monoxide exposure has been found to be associated with cardiac ventricular septal defects. Increase ozone concentrations have been associated with aortic artery and valvular dysfunction as well as pulmonary artery and valvular dysfunction. (4) Prenatal exposure to polycyclic aromatic hydrocarbons is harmful for the developing brain: high levels of DNA-PAH adducts have been found in cord blood of neural tube defect patients. (3)

Respiratory effects

Higher rates of childhood asthma, asthma medication use, emergency visits, hospitalizations have been temporally correlated with increase in ambient air pollution. (4) Higher ozone exposure to the developing lung has been associated with reduced lung function in children. These have also been associated with increase in respiratory symptoms in non-asthmatic children. Chronic bronchitis and chest illnesses have been well documented with exposure to particulate air pollution.(5)

Vitamin D deficiency rickets

Vitamin D deficiency rickets has been found to be more prevalent in areas with high air pollution index presumably due to decrease in the amount of solar light in the UVB range penetration to the ground level.(5)

Recent clinical studies on neurological effects of air pollution in children

Effect of prenatal exposure to polycyclic aromatic hydrocarbons was already studied on cohorts from USA, Poland, China, and Spain. All results indicate changes of child behaviour and neurodevelopment at the age of 3-9 years, decrease of IQ, increase of Attention Deficit Hyperactivity Disorder (ADHD), decrease of brain-derived neurotrophic factor (BDNF), reduction of left hemisphere white matter. Effect of traffic-related air pollution (TRAP) to neurobehavioral development in children, measured as PM2.5 (particulate matter <2.5 μm), PM10, elemental carbon (EC), black smoke (BC), NO2, NO_2, were studied in USA, Spain, Italy, and South Korea. Increased concentrations of TRAP were associated with the increase of ADHD, autism, affected cognitive development; PM2.5 decreased the expression of BDNF in placenta. Increased concentrations of PM2.5 affected adults cognition (episodic memory), increased major depressive disorders. Increased concentrations of NO_2 were associated with dementia, NO_2 with Parkinson's disease. Increased concentrations of PAHs, PM2.5 and NO_2 in polluted air significantly affect central nervous system in children and adults and represent a significant risk factor for human health.

Management and prevention

Air pollution, although can't be eliminated completely, but it can be reduced to a significant extent by simple, cost effective measures and precautions. Thus it constitutes one of the most common reversible factors at community level to reduce neurological morbidities in children. Sulfate particles, a larger proportion of the particulate burden in the air in urban areas, can be easily removed using scrubbers on powerplants (their largest source) at a cost that is <1% of the current price of electricity. NO_2 reduction, a major component of an ozone reduction strategy, can also be retrofitted onto powerplants. In India also catalytic converters on cars can be brought up to US standards. Traffic particles, NO_2, and so forth are dominated by diesel engines (6). Trap oxidizers and catalysts can reduce these emissions by up to 90%. Such devices have been on gasoline-powered vehicles for decades without ending industrial civilization as we know it. For many of these control strategies, it does not matter that we are not sure which component of the pollution mix is principally responsible (7). Oxidative catalysts reduce carbon soot, polycyclic aromatic hydrocarbons, CO, and so forth. Given the amount of money that we spend on the treatment of asthma and the difficulty that we have in reducing allergen exposures, such straightforward approaches need serious attention.

Source of funding: Nil

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