The Impact of Pollution on Children's Health: A Call for Global Accountability and Enforcement

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Abstract: Environmental pollution in large industrial centers has had a negative impact on the population's health, specifically among children.

Our objective is to provide a systematic review of the literature, focusing on the impact of environmental pollutants from urban and metropolitan areas on pediatric health.

Disregard for the Earth's atmosphere can negatively impact our fragile ecosystem and create a global toxicity. The impact of industrial growth and economic development has become paramount to modern society. Unfortunately, future generations will pay the consequences of the world's failure to implement regulations to secure a safe environment for our children's health and development.

Pollutants penetrate an organism through different routes and change physiological processes, which leads to a decrease in microbial resistance by weakening the child's immune system. The major contaminants are: polycyclic aromatic hydrocarbons, lead, manganese, sulfur dioxide, airborne fine particles, and nitrogen dioxide. Xenobiotics negatively affect the morphological, functional, biochemical parameters, genetics, and epigenetics of the body. It is well documented that the physical development and psychological well-being of children is adversely affected by pollution. The accumulation of heavy metals and other contaminants adversely affected a child's health has been found in the pediatric population.

An effort has been made to develop detoxicant remedies, in particular, some enterosorbents and natural adaptogens. Research is ongoing to improve medical rehabilitation of children, who already are affected. Public education and regulations regarding emerging non-pollutant industrial technologies is called for. A global system of accountability and enforcement regarding environmental protection needs to be implemented.

Keywords: Children, health, pollution, industry, environment, urban, economic stability.

INTRODUCTION

Is it possible for children to grow up healthy in a modern metropolitan area with a lot of industrial activity, if they are exposed to a variety of environmental pollutants? Industrial progress brought technological advancements to society, but simultaneously polluted our biosphere. Contamination permeates the air in densely populated urban and metropolitan areas: significantly impacting the health of children. Exposure is usually multifactorial, but certain contaminants may predominate, depending on the local industries. There are two aspects to pediatric exposure: living in an unfavorable, polluted environment; and/or by being around their parents, who work in industries where they are exposed to occupational hazards and inadvertently bring it home. A recent meta-analysis found an association between childhood leukemia and prenatal maternal occupational pesticide exposure [1, 2]. Creation of a system that continuously collects, stores, and analyzes information about environmentally-induced diseases, exposure, and risk factors, as well as a timely dissemination of this information to the decision-makers is an important approach being undertaken in some regions [3-5]. The Policy Interpretation Network on Children's Health and Environment (PINCHE) was set up within EU FP5 (QLK4-2002-02395) to protect the health of children and provide a prioritized list of risk factors and policy recommendations for action [6]. Ideally, such an approach should be adopted globally.

The role and mission of pediatricians and environmental scientists around the world is to recognize hazards and protect children's health. In this review we will address recently published data on urban contaminants that impact the pediatric population dwelling in industrialized areas around the world. Summarized approaches are presented in Figure **1**.

POLYCYCLIC AROMATIC HYDROCARBONS (PAHs) AND OTHER VOLATILE ORGANIC COMPOUNDS (VOCs)

VOCs are different groups of compounds such as n-alkanes, cycloalkanes, aromatic and chlorinated

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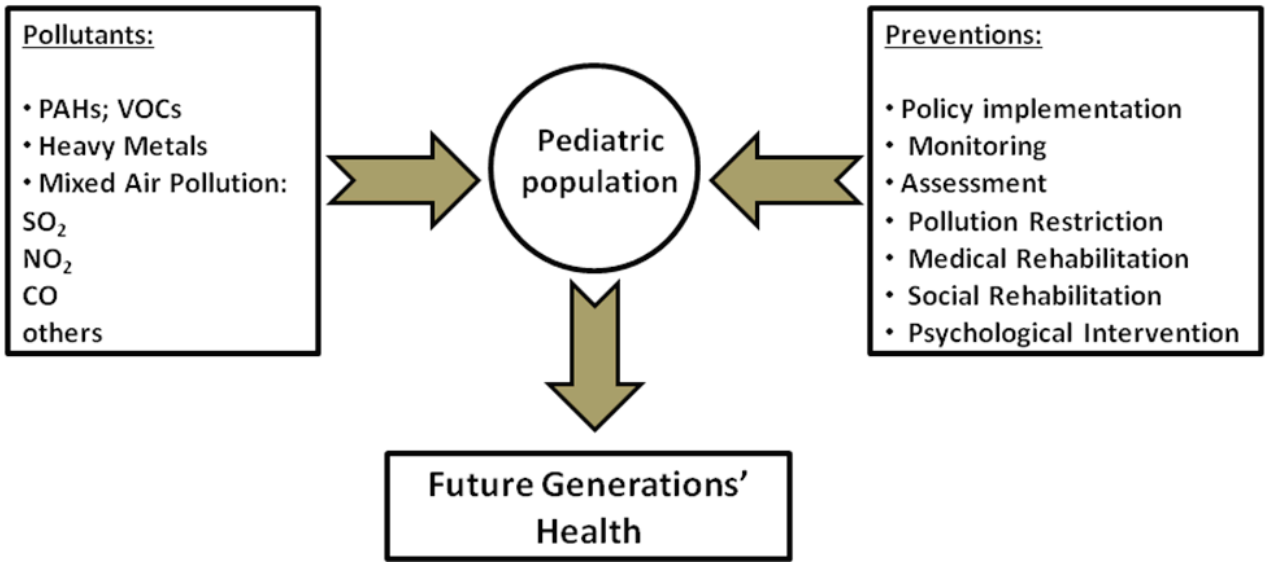

Figure 1: Impacts and Preventive Approaches to Improve Children's Health in Urbanized Areas. PAHs (polycyclic aromatic hydrocarbons); VOCs (volatile organic compounds); SO2 (sulfur dioxide); NO2 (nitrogen dioxide); CO (carbon monoxide).

hydrocarbons, and terpenes. Recent data from La Plata, Argentina showed the lifetime cancer risk-associated-value was related to benzene exposure indoors and outdoors with a doubling of risk for those living in industrial area *vs*. non-industrial urban, semirural, and residential areas [7]. Another study from the same region found that children living near a petrochemical plant had more asthma (24.8% *vs.* 10.1% to 11.5%), more asthma exacerbations (6.7 *vs.* 2.9-3.6 per year), more respiratory symptoms (current wheeze, dyspnea, nocturnal cough, and rhinitis), and lower lung function (>13% decrease in forced expiratory volume 1 minute) than those living in other regions. Length of residence in the area was a significant independent risk factor [8].

PAHs are persistent organic pollutants with carcinogenic, mutagenic, and toxic properties, which can undergo long-range transport and exist for a long time in the environment [9, 10]. In urban industrial environments, PAHs are emitted primarily by anthropogenic sources, such as vehicle emissions, coal and fossil fuel power generation, petroleum refining, burning of straw and firewood, industrial processing, chemical manufacturing, oil spills, and coal tar distillation [11]. PAHs are major pollutants of urban areas, that accumulate in soil and are poorly degraded [12-15]. Residential exposure to PAHs may pose an increased risk of cancer and mutations [16]. Data from Shenzhen, China showed that children aged 0 to 8 are the most sensitive sub-group to exposure to PAHs (59.2 ng per kg/d, which mainly occurs through inhalation) [17]. In the second French Total Diet Study, PAHs were analyzed in 725 foodstuffs habitually consumed by the French population [18]. Interestingly, the current highest levels of total PAHs were detected in mollusks and crustaceans, followed by different oil based products. Mean daily exposure to the sum of benzo[a]pyrene, benz[a]anthracene, chrysene and benzo[b]fluoranthene in this study was estimated to be 2.26 ng/kgbw/day in children [18].

Early life exposure to ambient PAHs can result in developmental delays [19]. The Columbia Center for Children's Environmental Health has reported a significant effect on prenatal exposure to ambient PAHs causing delayed mental development at three years of age. The group used the Bayley Scales in a cohort of low-income women and children in New York City [19]. Prenatal exposures to PAHs also adversely influenced fetal development including child weight, length, and head and chest circumferences [20]. In Eastern Europe, full-scale toxicological experiments were conducted to establish biochemical, clinical, and immunological parameters for the diagnosis of conditions in children exposed to hydrocarbons [21].

A recent study quantified and assessed the human health risk associated with exposure to PAHs in street dust along the Trans-ECOWAS highway in West Africa [22]. This study showed that the cancer health risk associated with inhalation of benzo [a] anthracene, benzo [a] pyrene, benzo [k] fluoranthene and chrysene exists for the resident children in the Tamale metropolis. It was determined by the researchers that the children were at high risk from exposure to these chemicals [22].

Molecular mechanisms underlying PAHs toxicity showed that in the genome-wide expression profiles of rat livers exposed to benzo[a]anthracene, benzo[a]pyrene, phenanthrene and naphthalene that 158 outlier genes were identified [23]. These genes were considered to be early predictive and surrogate markers for exposure to these chemicals [23]. The interrelations genetic and epigenetic mechanisms suggest that shorter telomere length could be a central event in PAHs carcinogenesis [24].

CHILDHOOD LEAD EXPOSURE

Great efforts have been made to reduce environmental lead (Pb) pollution over the past several decades. Blood Pb levels (BLL) in children have been declining after implementing measures to control lead pollution in many countries [25]. Regardless, environmental Pb pollution continues to be a global problem. The most profound sources of contamination are around mining and smelting operations, since marked reductions in the use of lead in gasoline, paint, and many other sources has occurred [26-30]. Children in polluted areas continue to ingest lead through pica and can also assimilate relatively large amounts of inhaled Pb from their environment [31, 32]. Strong measures have been taken to prevent emissions from factories all around the world. But efforts to control environmental Pb pollution originating from the largest smelters have been only partially successful [33]. There is a direct correlation between Pb environmental pollution and its concentration in the bodies of children in the area. An increase of 0.44 microg/m^3 Pb in air led to a 155% increase in BLL, whereas blood Pb increased by about 63% if Pb in soil increased by 800 mg/kg [34].

Pb poisoning causes permanent neurologic and developmental disorders and remains an important environmental health problem for children all around the world. Interestingly, in urban areas in Nigeria a significantly negative association between BLL and malaria in the pediatric population was observed, and this association remained significant after controlling for confounding diseases and symptoms [35].

A large scale study from China that studied 246 children (3-8 years) showed that BLLs were negatively correlated with both height and weight, and positively correlated with bone reabsorption biomarkers [36].

In Ukraine, the level of blood Pb among 212 three-year-old children was measured and showed that the geometric mean Pb level was 3.15 microg/dL (range, 0.7-22.7). In an adjusted model, investigators observed a strong association between Pb levels in children and the likelihood that fathers worked as manual laborers in industries associated with lead exposures (adjusted odds ratio [OR] = 2.25. P = 0.025) [37]. There was also an increase in Pb levels in the children whose mothers smoked indoors (adjusted OR = 2.87, P = 0.047) [37]. Our previous data showed an association between children's lead levels (hair, nails) and residency near metallurgical factories, and roads with heavy traffic *vs.* the control group, who lived in recreational areas [38].

The neurotoxicity of Pb has been explained by multiple cellular "molecular targets" [39]. One possible target of the neurotoxic effect of Pb (at the synapse level) is N-methyl-D-aspartic acid receptors [39]. The cardiotoxicity cause of Pb exposure has been explained through cytochrome P4501A1-mediated mechanism [40]. The identification of molecular mechanisms underlying Pb toxicity on developing organism revealed that pre- and neonatal exposure of rats to Pb can decrease the number of hippocampus neurons, occurring concomitantly with ultrastructural alterations in this region [41].

Many countries are taking different approaches to deal with Pb environmental exposure. A good example of this is the Los Angeles (California, USA) model where a map of spatial distributions of soil Pb concentrations was created and shows great promise as a screening tool to evaluate continued Pb poisoning in children [42].

A Risk Communication Program was generated in San Luis Potosí (México), to educate the children about the environmental risks of pollution in their neighborhood, which diminished their exposure to Pb and arsenic (As) [43].

IMPACT OF OPEN MANGANESE (Mn) MINES

According to the World Health Organization data, the daily intake of Mn in the air by the general population in areas without Mn-emitting industries is below 2 μg/day [44]. In areas with major foundry facilities, intake may rise to 4-6 μg/day and in areas associated with ferro- or silico-manganese industries it may be as high as 10 μg/day, with peak values exceeding 200 μg/day [44]. A higher prevalence of nose and throat symptoms and reduced respiratory function were registered in school children exposed to Mn concentrations in air ranging from 4 to 7 μg/m^3 (five-day mean values) compared with an unexposed control group [44].

Ukraine has the world's main Mn mining operations, which are concentrated in the region of Dnipropetrovsk Oblast near the cities of Ordzhonikidze and Marhanets. The previous study showed that the major effects of Mn were observed on bone growth and development [45]. Among the 683 children studied from the Mn region, 53% had impaired growth with rickets-like skeletal deformities, changes in immune function (the children from the Mn mining region had increased mean salivary IgA), and somatic cell mutation (the mean level of mNUC in cells of the children in the Mn region was 5.8 times higher than the controls) [45].

The rehabilitation program was proposed and tested in the region, which included administration of humics, carotene oil, enterosorbents, and eubiotics. The results of the two-month rehabilitation study demonstrated that natural adaptogenes can promote a positive decrease in the frequency of occurrence of genetic pathologies in somatic cells and the concentration of Mn decreased significantly in the hair of children dwelling in the contaminated region. The implementation of social and medical rehabilitation measures may protect the next generation living in unfavorable environmental conditions from developing the symptoms of chronic Mn poisoning [45].

OTHER HEAVY METAL EXPOSURE

Heavy metals are among the major contaminants in industrial areas and may come from many different sources such as vehicle emissions and industrial discharges [46-48]. Metal smelting is one of the most important anthropogenic heavy metal emission sources [49-51]. During the smelting process, heavy metals in the ores are evaporated from the matrix, and eventually enter into the atmosphere if no pollution control technology is applied. Risk assessment models for residential exposure include: (a) direct ingestion of substrate particles; (b) inhalation of re-suspended particles through mouth and nose; (c) dermal absorption of trace elements from particles adhered to exposed skin; (d) or through inhalation of vapors [52]. A recent study conducted in China (Huludao City) concluded that in order to accurately estimate the health risk, there was a need for further research of street dust exposure parameters and transport factors that would help reduce the uncertainties associated with the risk calculations [52]. The exposure pathway that resulted in the highest levels of risk for children and adults exposed to street dust was ingestion, and Pb and cadmium (Cd) were regarded as the most likely culprits to increase health risks [52].

A significant relationship exists between respiratory diseases in children and the load of heavy metals accumulated in the body [53]. Heavy metals were the primary contaminants in large industrial centers of Ukraine for a long period of time [54]. We showed the positive correlations between the concentration of Cd in soils and malignant neoplasm, diabetes, and infant mortality, and between the concentration of Mn in soils and infant mortality [55].

Arsenic, cadmium, chromium, lead, and mercury rank among the priority metals that are of public health significance because they are systemic toxicants. They are known to induce multiple organ damage, even at lower levels of exposure [56]. Underlying molecular mechanisms are different and specific for the metals. Thus, Mn(2+) overload compromise specifically affected cellular integrity and Golgi organization, where the secretory pathway Ca(2+) /Mn(2+) -ATPase is localized [57]. Cadmium on the other hand, is a major cause of kidney disease. It could cause proximal tubule injury that involves specific changes in cell-cell adhesion, cellular signaling pathways, and autophagic leading to cells' necrosis or apoptosis [58]. Overall, population exposure to mixtures of environmental toxicants, engages three major signaling pathways: (i) activation of detoxification genes; (ii) induction of signal transduction effectors; and (iii) epigenetic modification of chromatin marks [59].

AIRBORNE MULTIFACTORIAL EXPOSURE

Mixed air pollution in industrial cities is a global problem. The strong or moderate correlations between particulate matter size and sulfur dioxide (SO_2), nitrogen dioxide (NO_2), and other environmental players make it difficult to distinguish the effects of individual air pollutants [60].

Case-crossover studies conducted in China to investigate the acute health effects of air pollution showed that short-term exposure to air pollution was associated with increased mortality [61]. Thus, a 10 $\mu g/m^3$ elevation of the 2-day moving average (lag 01) concentration in particulate matter with aerodynamic diameter of ≤ 10 μm (PM_{10}), SO_2, NO_2, and carbon monoxide (CO) has been shown to increase cardiovascular death [61]. High exposure to CO prior to birth (European study) could cause, on average, a 289g lower birth weight [62].

The strong impact of air pollutants on the respiratory system was demonstrated in a study from Seoul,

Korea. Analysis of the effects of air pollutants on children's respiratory and allergic symptoms demonstrated that CO affected all symptoms in all of the study areas. When the concentration of SO_2 or NO_2 was elevated, upper respiratory symptoms increased significantly. In contrast, when the concentration of ozone (O_3) rose, the symptoms decreased significantly [63].

We also studied multifactorial exposure of children in the Dnipropetrov region of Ukraine and implemented effective rehabilitation procedures which were offered in the region [64]. Methods involved in the study included clinical examination, functional ability of respiratory and cardiovascular systems, study of immunity indices, and determination of inflammatory enzyme activity [64].

We found that the response to environmental pollution among children was polymodal. Two groups were clearly identified: one with a strong reaction; and one with a weak response. Children living in polluted areas had hypertrophy of the tonsils, larger lymphatic nodes, and increased liver size, which pointed towards an increased strain on the protective barriers of the organism.

There was a direct connection between some anthropometric indicators, the functional state of cardiovascular/respiratory systems, and the level of excretion of amino acids in the urine. The negative influence of xenobiotics on the state of morphological, functional, biochemical parameters and genetics stressed protective barriers. Children living in unfavorable environmental areas showed a decrease of cellular and humoral immunity. Natural adaptones such as a humic substance normalized immune processes and improved functional activity of the cardiorespiratory system [64]. After the treatment, children had 1.5 times fewer respiratory diseases through the winter months than a year before treatment (independent of age). The number of frequently ill children decreased 1.3-2.0 times.

Another study from Ukraine, which included such unfavorably, industrially polluted cities such as Dneprodzerzhinsk, showed a strong correlation between environmental conditions and respiratory health of the children living in the area [65]. Data from Krivoĭ Rog (Dnipropetrovsk Oblast, Ukraine) also indicated that there was a reduction in the major immunological parameters in both healthy neonatal infants and 7 to 10 years-old children and a rise in pediatric morbidity in polluted urban areas [66]. The newborns and children under 14 years of age experienced the greatest danger of exposure to toxicants because their immunological systems were in an early stage of development [67].

Children with asthma might be the most sensitive to industrial air pollutants. Thus far, the data from the U.S. showed that children in urban areas have significantly decreased lung function following exposure to higher concentrations of air pollutants pollutants: SO_2, airborne fine particles, and NO_2 [68]. Higher NO_2 levels and higher levels of fine particles were associated with more school absences related to asthma, and higher NO_2 levels were associated with more asthma symptoms [68]. Because NO_2 is derived mainly from motor vehicle exhaust, this data provide evidence that car emissions may be causing adverse respiratory health effects among children who have asthma.

The French studied 4,907 children, who lived at the same address for 3 years and found that asthma (exercise-induced, past-year and lifetime) was significantly associated with benzene, SO_2, particles with a 50% cut-off aerodynamic diameter of 10 microns (PM_{10}), nitrogen oxides (NO_x), and CO [5]. In the same study, eczema (lifetime and past year) was also significantly associated with benzene, PM_{10}, NO_2, NO_x, and CO; and lifetime allergic rhinitis with PM_{10}, and sensitization to pollens with benzene and PM_{10} [5].

When exposed to refinery emissions, children with asthma were significant affected. Data from Cape Town, South Africa, clearly showed an increased prevalence of asthma symptoms among children, who resided in areas of refinery emissions and provided a substantive basis for community concern [69]. Refineries are a classic multifactorial source of contamination because of the complexity of their emissions. The Cape Town scientists used a meteorologically-derived exposure metric calculation with the refinery as the putative point source [69].

Environmental pollution arising from electronic equipment waste disposal and recycling has received considerable attention in recent years. Although several studies have reported trace metals and polybrominated diphenyl ethers released from e-waste recycling operations, environmental contamination and human exposure to polychlorinated dibenzo-p-dioxins and dibenzofurans from e-waste recycling operations are less well understood [70]. There is a concern of health risk for humans, and particularly children, from dioxin exposures at e-waste recycling facilities.

Write & Brunst (2013) suggested that programming effects may result from pollutant-induced shifts in a number of molecular, cellular, and physiological states and their interacting systems resulting in sustained epigenetic changes [71]. Pollutant mixtures cause significantly greater biological effects and genetic alterations than the primary pollutants themselves, these factors need to be taken into consideration when studying adverse health effects caused by air pollution [72].

PSYCHOLOGICAL ASPECTS OF POLLUTIONS AND HOW IT IMPACTS CHILDREN'S HEALTH

Our discussion would not be complete without taking into consideration industrialization and its impact on the psychological aspects of children's health. Stress caused by city environment (noise, traffic, crowd *et ctr*.) could negatively affects maternal and child health. Other significant life stressors are poverty, lack of access to quality education, proper nutrition, and living in an unsafe environment [73]. It was also noted that indoor pollutant concentrations are affected by socioeconomic factors and status [74].

The study identified a complex relationship between actual levels of pollution, social deprivation, socio-behavioral factors and people's perceptions about pollution [75]. Interestingly, people's perceptions about the severity of pollution can impact their health [75]. Thus, many believed that childhood asthma was associated with central heating [75].

Many schools have educated their adolescent students about the dangers of environmental pollutants. For example, many teenagers were critical being exposed to secondary smoke inhalation by their parents. Non-smoking parents were most likely seen as good parents [76]. In another study Schuck *et al.* (2012) found that adolescent smoking rates increased when teens saw their parents' smoking [77]. For children living in polluted industrial cities, additional passive smoking exposure creates a more negative environment, full of cumulative pollutant mixtures. Behavior modification and educational programs should be provided at all levels of organization, for those responsible for children's well-being.

A group of children were studied to determine the effects of a large oil spill disaster (the Galician coast, Spain) on academic achievements. The effects of the disaster on academic achievement were relatively insignificant, but the researchers identified a marked variance in schoolroom behaviors [78]. Palinkas (2012) analyzed the results of the Deepwater Horizon oil spill and developed a conceptual model with three levels of impact: biopsychosocial that are direct consequences of the contamination of the physical environment; interpersonal that are direct consequences of the biopsychosocial impacts; and intrapersonal or psychological that are consequences of both the biopsychosocial and the interpersonal impacts [79].

CONCLUSION

Children are the most vulnerable population on this planet and it is our duty and responsibility to protect the future generations. The economic growth and competitive nature of the industrialized world has unfortunately led to significant contamination of the environment. Although rigorous efforts across the globe are being made to address health concerns directly related to environmental pollutions; we are losing the battle. Coordinated and collaborative research is needed to continue to assess the problem, implement protective measures, and provide rehabilitation. The best approach is to prevent further contamination of our fragile ecosystem. Promotion of the development of technologies to eliminate or minimize harmful exposures is essential. All countries need to be accountable for any emission of pollutants into the atmosphere; and a system of enforcement needs to be in place in order to support and maintain a healthy environment for children.

ABBREVIATIONS

As = arsenic

BLL = blood lead level

Ca = calcium

Cd = cadmium

Mn = manganese

NO_2 = nitrogen dioxide

O_3 = ozone

OR = odd ratio

PAHs = polycyclic aromatic hydrocarbons

Pb = lead

PINCHE = The Policy Interpretation Network on Children's Health and Environment

PM_{10} = particle pollution 10 micrometers

SO_2 = sulfur dioxide

VOCs = volatile organic compounds

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