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Industrial pollutions on the health effects of infants, children, and adolescents: A systematic review

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Abstract. Industrial pollution is considered especially harmful to juveniles' health. This paper analyzes the effect of common pollutants, including particulate matter, nitrogen oxides, persistent organic pollutants, and heavy metals, on three different types of diseases. It can be concluded that particulate matter, nitrogen oxide, and POPs are significantly associated with neurodevelopmental deficiencies in infants. Endocrine-disrupting effects and neuroinflammation are considered the leading causes, but the specific mechanisms remain questioned. Pollutants from heavy industries have a higher potential to cause children's respiratory diseases, and residency within 4 km of the industry is especially hazardous. Biomarkers VEGF and urinary 8-OHdG reflect a high glutathione-related oxidative burden for exposure to toxic metals attached to particulate matter. Deregulation in metabolism and DNA oxidative damage is shown to be responsible for increasing cancer risk, but the triggering pathway is unclear. The tendency to develop diseases in both genders is heterozygous, and a larger dataset is required to draw a conclusion.

Keywords: industrial pollution, $PM_{2.5}$, POPs, children, health.

1. Introduction

Industrial pollution is a nonnegligible part of worldwide pollution, causing severe damage to the environment and human health. Juveniles are especially susceptible to these detriments. Multiple types of research have shown that juveniles living near industrial areas have a higher risk of developing neurological development disorders, respiratory problems, cardiovascular diseases, and infant mortality [1-5]. There is also a relationship between industrial pollution and pediatric cancers [6]. Children who develop these diseases during their youth are often affected lifelong, resulting in lower quality of life and extra financial burdens.

Common sources of industrial pollution include fossil fuel combustion, refinery emissions, and improper disposal of industrial waste, resulting in air and water pollution. Pollutants caused by fossil fuel combustion and refinery emissions often include nitrogen oxides (NO_x), particulate matter with an aerodynamic diameter less than 2.5 μm ($PM_{2.5}$), particulate matter with an aerodynamic diameter less than 10μm (PM_{10}), ultrafine particles (UFPs), carbon monoxide (CO), sulfur dioxide (SO_2), ozone (O_3), and toxic metals such as lead (Pb) and beryllium (Be). Pollutants caused by improper disposal of industrial waste often include persistent organic pollutants (POPs) like Per- and Polyfluoroalkyl Substances (PFASs), Polycyclic Aromatic Hydrocarbons (PAHs), Polychlorinated Biphenyls (PCBs),

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benzene, and other aromatic hydrocarbons, as well as heavy metals like mercury (Hg) and nickel (Ni), often found in wastewater [7, 8].

This article provides a systematic review of various industrial pollutants and their health effects on infants, children, and adolescents, creating a better understanding of the hazards and mechanisms of industrial contaminants in environmental health.

2. Neurological development disorders

Researchers found that infant neurodevelopmental deficiencies are related to early life exposure to industrial air pollution, especially UFPs, NO_2, and $PM_{2.5}$, as well as POPs such as PFASs [2, 9, 10]. Several mechanisms for these contaminants have also been proposed to interpret the pathway that triggers them.

UFPs, due to their ultra-small size, are known to pass through lung epithelium and enter the bloodstream easily, causing multiple diseases. Recent studies have shown that both prenatal and postnatal exposure to UFPs is related to the increasing risk of neurological disorders in infants, such as low attention scores, hyperactivity, and autism spectrum disorder (ASD), while neuroinflammation and oxidative stress triggered by UFPs are considered the primary causes of these issues. Black carbon particles found in human placenta sample shows that UFPs can pass through the placental barrier and possibly the blood-brain barrier to the fetus's central nervous system, generating cell death and neuroinflammation [9]. At the same time, maternal oxidative stress prompted by UFPs will lead to even more damage to fetus development. Similarly, infants' postnatal exposure to UFPs can contribute to neuron damage through homogenous pathways, inducing neurodevelopmental disorders. However, the actual biological mechanism still remains ambiguous, and more research is needed to elucidate the specific pathways.

As one of the most common neurological disorders in infants and children, ASD has also shown a correlation to industrial air pollution. A meta-analysis of 13 different studies worldwide has assessed the risk ratio between $PM_{2.5}$, PM_{10}, NO_2, and O_3 and ASD and found that NO_2 and $PM_{2.5}$ play a major role in prenatal and postnatal exposure. Analysis shows that a 10 $\mu g/m^3$ increase in $PM_{2.5}$ and NO_2 prenatal exposure during the whole pregnancy has a risk ratio of 1.34 and 1.05, respectively; in comparison, postnatal exposure during the first year of birth has a risk ratio of 2.43 and 2.72, respectively [10]. The difference in exposure ratio risk suggests that infants are more susceptible to direct contact with pollutants during the neonatal stage, and nitrogen dioxide is especially threatening for new-born to develop ASD. Even though the risk ratio has shown a strong relationship between $PM_{2.5}$, NO_2, and ASD, the sample size is too small to draw a definite conclusion, and the methods used may be different in each study, causing deviation. Thus a larger and more systematic study is required to validate the result.

Many POPs often have the property of acting as endocrine disruptors, causing abnormal children's physical development. It is currently known that some industrial POPs, such as PFASs, can also lead to neurological disorders through animal trials and clinical studies. Studies show that due to bioaccumulation processes, ingesting water and food contaminated by industrial PFASs will lead to the long-lasting existence of PFASs in the human serum at low concentrations. Thus, maternal ingestion of PFASs will lead to continuous exposure to the fetus. Thyroid homeostasis disruption is thought to be the underlying mechanism for inducing neurodevelopmental disorders. The thyroid is responsible for myelination, synaptogenesis, and neurogenesis. Laboratory animal trials on mice have shown that PFASs can reduce thyroid hormone levels and inhibit related genes. Exposing PFASs to thyroid and FRTL-5 cells also reveals PFASs' cytotoxicity and genotoxicity [2]. Nevertheless, PFASs may perform differently on different species, and the conclusion drawn from mice may not be equally applicable to humans. The in vivo data in humans also showed a heterogeneous result, and the disrupting effects of PFASs still need to be studied.

3. Respiratory diseases

Industrial air pollution is well-known for causing respiratory abnormalities. Common pollutants, including $PM_{2.5}$, SO_2, and multiple hydrocarbons, have all been validated to induce children's decreasing

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pulmonary function and increasing oxidative stress. Different industries in different countries also show heterozygous results, including divergent effects on gender. Results from various studies have been compared and analyzed in the following sections.

Compared to other industries, the coal and oil shale industries are reported to be the most impactful. A study in Indonesia's coal industrial areas shows that the concentration of $PM_{2.5}$ is three times higher than in the non-industrialized region, impairing the lung function of more than 68% of children living in that area[3]. At the same time, research in Estonia's oil shale industry areas evaluates the fractional exhaled nitric oxide (FeNO), a marker of lung inflammation, of schoolchildren exposed to hydrocarbons like formaldehyde and benzene. The result found a 2 to 3 times higher FeNO level compared to the control area and a 2.5 times increase in the prevalence of asthma attacks [11]. In comparison, the investigation of Italy's chipboard industry suggests a 13% to 30% increase in emergency room (ER) admissions due to pneumology derived from mainly $PM_{2.5}$ and SO_2 pollution [12]. The difference in data shows that pollutants from heavy industry are relatively more hazardous for children than those from light industry due to their higher toxicity and concentration. However, since the socioeconomic status and environmental protection policies vary by region, more assessments are needed to reach a more accurate conclusion.

Industry's proximity to residences also plays a prominent role in triggering respiratory diseases. Multiple types of research show that living within a range of 3km from the industry plants will pose a significant health threat to residents. A study in North Lebanon suggests that the odd ratio of living 0-3 km from the industry in an exposed district is 4.63 for an annual experience of cough and phlegm, 3.40 for cough with a cold, and 4.12 for an annual chest cold while living outside the range will have a significantly lower risk of experiencing lung diseases [13]. Similarly, the study of the chipboard industry in Italy reveals a 5% decrease in the rate of ER pneumology admissions for those living every 1km away from the industry. Moreover, children living within 2km of the chipboard industry also show a 51% increase in the ER entry rate [12]. Thus, it can be concluded that living at least 4km away from industry is needed to prevent children, mainly under 4, from having frequent severe respiratory diseases.

Toxic metals are always a concern for most industries, and they are primarily related to air pollution because these metals will attach to particulate matter, which will cause oxidative stress after being inhaled into the human body. Data collected have shown that when exposed to trace metals such as Ba, V, Al, and Fe, participants' FeNO levels have increased, suggesting a reaction of lung inflammation in the human body. At the same time, inflammation biomarkers such as vascular endothelial growth factor (VEGF) increased by 5.3% after exposure to nickel for an hour, and oxidative stress biomarkers such as urinary 8-hydroxy-deoxy-guanosine (8-OHdG) increased by 14% after an hour of exposure to copper [14]. Both VEGF and urinary 8-OHdG are significantly related to glutathione-related oxidative potential (GSH-related OP), a measure of GSH depletion in synthetic airway fluid. The GSH-related oxidative burden has been proven to have a strong connection with lung cancer mortality and pulmonary symptoms, which has further confirmed the hazardous impact of toxic metals and uncovered part of the triggering mechanisms [15].

Interestingly, different pollutants may also cause a different risk ratio among genders. A study of Indonesia's coal industry suggests that boys living in industrial areas exposed mainly to $PM_{2.5}$ have a 1.9 times greater risk of developing lung diseases than those living in unpolluted regions. In comparison, girls have a 1.3 times greater risk [3]. However, research on the natural gas industrial area in Bintulu indicates no significant difference for boys and girls in the relative risk between hospital admission and the cumulative lag effect of exposure to $PM_{2.5}$. Instead, they found the highest relative risk of cumulative lag effect of exposure to SO_2 for girls is 1.300, much higher than that of 1.195 for boys [16]. Nevertheless, the data size is too small to conclude the relationship between different pollutants and gender-specific vulnerability to having pulmonary diseases. The heterozygous results also require more examination of the mechanisms.

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4. Cancers

As one of the deadliest diseases, cancer has become highly prevalent since the 1800s. One of the linking factors in cancer is thought to be industrialization and modernization, although the specific reason is still a mystery. Recently, industrial pollution's effect on biomarkers has been studied to provide a better insight into the relationship and mechanisms.

Deregulation in metabolism and DNA oxidative damage are the leading risk factors for cancer, and they are induced by exposure to carcinogens emitted from fire plants. A study on adolescents living in Taiwan's petrochemical complex reveals a significant increase in IARC group 1 carcinogen As and Cd and PAHs biomarkers 1-hydroxypyrene (1-OHP) in the urine from the high-exposure group. They also show a substantial alteration of six acylcarnitine levels in serum and purine metabolism dysregulation, which is strongly associated with cancer development [17]. At the same time, a meta-analysis study of 13 passages relating childhood leukemia and air pollution indicates a strong correlation between benzene, NO_2, and blood cancer. The odd ratio of NO_2 and benzene exposure is 1.21 and 1.09 for acute lymphoblastic leukemia, respectively, and 1.06 and 2.28 for acute myeloid leukemia, respectively [18]. Benzene is shown to have a significant role in causing blood cancer, possibly because of its metabolites' oxidative damage to DNA.

Even though the positive relationship between cancer risk and industrial pollution has been proven, it still requires a larger dataset to validate the result to avoid publication bias. Moreover, a deeper analysis of the triggering pathway is also needed to assess the detriment better.

5. Conclusion

Industrial pollution has been linked to many diseases, including neurological development, respiratory diseases, and cancer. Common pollutants triggering diseases include particulate matter, nitrogen oxide, toxic metals, and POPs like PFASs, PAHs, and benzene. Most of the industrial contaminants displayed the property of inducing oxidative stress. UFP, $PM_{2.5}$, NO_2, and PFASs are thought to be responsible for infants' neurodevelopmental deficiencies due to oxidative burden, inflammation, and disrupted endocrine homeostasis, although the specific triggering pathway still remains ambiguous. Air pollution from heavy industries most significantly impacts juveniles' pulmonary function. Residency within 4km of the industry is especially hazardous for children due to the considerable risk of developing respiratory diseases. Toxic metals attached to particulate matter are often carcinogenic, causing residents' systematic inflammation and GSH-related oxidative burden, which are highly associated with lung cancer mortality. Different genders may have distinctive risks of developing pulmonary diseases if the major pollutant is different, but the dataset is too small—more assessments are required to draw a conclusion. Cancer risk and development are thought to be related to the deregulation of metabolism and DNA oxidative damage caused by industrial carcinogens and POPs. However, larger data sets and deeper analysis of the mechanisms are required to understand the relationship better.

This study only provides a systematic review of industrial pollution's impact on children, whereas industrial pollution affects people of all ages, not just juveniles. People in old age are also susceptible to industrial pollution, and its impact should also be studied. Moreover, diseases caused by industrial pollution in youths may not be limited to neurological development disorders, respiratory diseases, and cancers. Other non-communicable diseases, such as cardiovascular diseases, should also be studied.

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