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EFFECTS OF CHILDHOOD EXPOSURE TO ENVIRONMENTAL POLLUTANTS: NURSING REVIEW

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Abstract:

A better understanding of the potential effects of environmental exposures on fetal and childhood growth is critical for public health because of the following factors: increased exposure levels to a variety of pollutants as a result of increased industrialization; the known ability of environmental pollutants to easily cross the placenta; plausible mechanisms linking environmental exposures to impaired fetal and childhood growth; and the influence of early life exposure. We search the electronic databases; PubMed, Embase for all papers that was published regarding our review's topic, up to middle of 2021. The relevance of early environmental exposures in the development of childhood asthma is becoming more widely recognized. Although outdoor air pollution is a known asthma trigger, it is unknown whether exposure influences incident illness. There is some evidence that environmental toxins may play a role in attention deficit hyperactivity disorder and autism (lead, PCBs, air pollution), respiratory and immunological health (dichlorodiphenyldichloroethylene - DDE - and PCBs), and obesity (DDE). Furthermore, there is now some evidence that some chemicals of recent concern, specifically perfluorooctanoate and foetal growth, and polybrominated diphenyl ethers and neurodevelopment, may be linked to poor child health outcomes.

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INTRODUCTION:

The development of wheeze and asthma is thought to be influenced by a complex combination of environmental exposures and genetic vulnerability (1). Asthma, the most common non-infectious condition in children (2), affects 235 million people worldwide, with a rising prevalence in low and middle-income nations. Particulate matter, chemical substances, and biological materials are all components of air pollution, which is a complex and omnipresent mixture of pollutants (3). There is growing recognition of the negative effects of air pollution on health after both prenatal and postnatal exposure (4). There is compelling evidence that postnatal air pollution can aggravate pre-existing wheeze and asthma (5). Postnatal exposure to the major components of air pollution (nitrogen dioxide (NO2), fine particulate matter (diameter 2.5 m, PM2.5; diameter 10 m, PM10), black carbon (BC), sulphur dioxide (SO2), and others) is linked to an increased incidence of wheeze and asthma (4,5).

Environmental tobacco smoke (ETS) exposure before and after birth is linked to an increase in asthma occurrence (6). Although air pollution exposures before the age of 2-3 years appear to be the most relevant for asthma development (7), the effect of prenatal (or in utero) exposure has not been studied to our knowledge. One exception is a study of polyclyclic aromatic hydrocarbon exposure that was conducted in tandem with ETS (6). Maternal smoke, folic acid, paracetamol, and biphenyl A use were all associated with an increased prevalence of childhood asthma in observational and systematic studies assessing prenatal exposure to drugs (8). Until far, there has been no conclusive evidence linking prenatal air pollution exposure to childhood wheeze and asthma (8).

DISCUSSION:

At enrolment, extensive socio-demographic information, obstetric history, anthropometric measures, and food intakes were gathered (9). A socioeconomic status (SES) index based on an income and assets questionnaire used in previous demographic studies in Mexico was created using main components analysis (10). Maternal education (in years) was self-reported at the time of enrollment. The Raven progressive matrices tests (11) were used to assess maternal IQ, which is a nonverbal test intended to measure abstract reasoning. It was designed expressly to be a "easy-to-administrate, easy-to-interpret" measure for assessing fluid intelligence, and it has been validated for usage in a variety of settings (11). This study's version had three sections of 12 problems each, with a maximum potential score of 36. A certified psychologist administered the test in a quiet room at the study clinic (9).

For all offspring, data on birth outcomes, child anthropometry, and newborn and young feeding behaviors, including breastfeeding, were gathered (9). Between 6 and 12 months, the Home Observation for Measurement of the Environment (HOME) questionnaire was utilized to assess household stimulation and learning environment (10)

A review of ten small studies found evidence that BPA exposure is associated to neurobehavioral disorders in children (12). However, there are several differences between studies in terms of the sex-specific directions of the correlations, the specific behavioral domains most affected, and the time window of exposure (pre-orpostnatal). These contradictions can also be detected in the findings of contemporary prospective research (13). In terms of cognitive development, two research (8) find a link between prenatal BPA exposure and cognitive performance, whereas one does not (14).

Respiratory and immune health:

Pollutants that disrupt the growing respiratory and immune systems may result in a diminished ability to fight illness, decreased lung function, and an increased chance of developing allergies (15). As the most investigated manifestations of pollution impacts on the developing respiratory and immune systems, this study focuses on respiratory infections, antibody responses, asthma, wheezing, lung function, and allergies and atopy.

Outdoor air pollution and substances:

There is substantial evidence that present levels of air pollution can aggravate pre-existing asthma (16). Recent systematic reviews and meta-analysis studies add to the evidence that air pollution plays a role in the development of childhood asthma, allergies, and associated symptoms. The recent ESCAPE study (17), which combined data from multiple European birth cohorts, the longitudinal Southern California Children's Health Study (18), and systematic reviews and meta-analyses (17,18), found links between traffic-related air pollution and pneumonia (19), lung function decline, asthma prevalence and development, and, less clearly, eczema, allergic rhinitis, and sensitization.

A systematic review of 41 studies classified evidence as "limited" (several good qualities, independent studies report an association, but with some inconsistencies) for the association between prenatal exposure to DDE, PCBs, and dioxins and risk of

respiratory infections, as well as postnatal exposure to PCBs and decreased immune response after childhood vaccination (20). For additional organochlorine chemicals and results, the evidence was deemed insufficient. A later meta-analysis of 4608 participants from ten European birth cohort studies reveals that prenatal DDE exposure is linked to respiratory health problems in young children, but not PCB153 exposure (21). A long-term follow-up investigation discovered high maternal PCB118 and HCB concentrations were related with an elevated risk of asthma medication use in kids aged 20 years.

There is accumulating evidence that PFASs have an effect on the developing immune system, with research indicating that PFAS exposure may reduce antibody response to childhood immunizations and raise the chance of having low levels of the antibodies required for long-term protection. Other outcomes studies have yielded conflicting results, including claims of increased, decreased, and no risk of asthma, wheezing symptoms, and atopy in relation to PFAS exposure (20).

There was no difference in levels of PBDEs and other flame retardants in mattress dust between asthmatic children and healthy controls, according to the sole study available so far (22). We classified the situation as "no evidence." 5.6. Currently used pesticides Only two research have looked at the respiratory and immunological consequences of commonly used pesticides.

Prenatal organophosphate exposure was associated with respiratory symptoms at age 5 and 7 years inone recent study, and pyrethroid exposure with cough, wheeze, and IgE in early childhood in another (23). Several cross-sectional and case-control studies have found a link between phthalate exposure and the development of respiratory and allergy illness. Longitudinal studies have also revealed relationships in recent years, however these have not always been consistent: One prospective birth cohort study found a link between one high molecular weight phthalate metabolite (MBzP) and early onset eczema (before the age of 24 months), but not late onset eczema (23). Three studies examining prenatal phthalate exposure and the incidence of wheeze, asthma, and respiratory infections in children aged 5-11 years each found a positive connection, but the phthalate congeners involved differed (19,21,22).

The most striking feature of catch-up growth is a disproportionately higher rate of fat increase in comparison to lean tissue gain. Those who are growth restricted throughout fetal life but then develop swiftly and attain a larger body weight are the most affected and exhibit increased adiposity in childhood and later adult life (24). Aside from alterations in adipose tissue mass, LBW patients prefer to store excess adipose tissue centrally. There is mounting evidence that the developing fetus and infant are programmed for obesity risk in the utero and neonatal environments. Obesity in childhood raises the risk of type 2 diabetes, cardiovascular disease, and metabolic syndrome, as well as having a negative impact on pulmonary, musculoskeletal, and psychosocial functioning (25). Because there are few usually effective interventions to reduce excess adiposity after it is established, finding and developing interventions for modifiable risk factors for reduced prenatal growth and rapid catch up is a public health priority. It is suggested that prenatal PFC exposure may have an aftereffect on weight homeostasis. Environmental stresses, such as PCFs exposure, may result in a 'thrifty phenotype,' in which excess calories are stored in an inefficient manner (26).

CONCLUSION:

In recent years, the use of exposure biomarkers in the study of environmental pollutants and child health has increased fast, frequently indicating a significant improvement over previous exposure assessments, such as questionnaire-based ones. However, there are certain uncertainties in the usage of biomarkers. For example, it is difficult to describe exposure to non-persistent chemicals (phthalates, BPA, currently used pesticides) since they have short biological half-lives and are removed from the body in a few hours or days. A biomarker measurement at a single point in time thus only provides an estimate of relatively recent exposure, making study results difficult to interpret.

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