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Role of Exposome in Uncontrolled Asthma: An Update

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Abstract

Asthma is known to be one of the patient complaint and is caused by multi factors including genetics, diet, environmental exposures, respiratory infections, and other factors. Studies show that around 1 in 10 children are set up to be affected this condition worldwide, which is characterized by ongoing symptoms similar as patient cough, briefness of breath, gasping. It's a miscellaneous condition that varies from person to person grounded on factors like age of onset, treatment response, clinical signs, environmental factors, and overall progression. Exposomes synopsize the aggregation of internal and external sources of chemical substances, natural agents, and cerebral factors to which an existent is exposed over a lifetime. Internal exposomes involve factors like oxidative stress, inflammation, disordered metabolism and hormonal changes. Microbial exposure, unresistant smoking, air pollution and socioeconomic conditions are part of external exposomes. Asthma affects about 339 million individuals, out of which 33 are children under the age of 14, 27 are grown-ups who developed asthma in nonage, 40 are individuals diagnosed during adulthood. This review aims to interpret the environmental factors that leads to DNA damage in children suffering from uncontrolled asthma. It points out how the environmental exposures including air pollution, habitat , allergens and nutrition- related factors can increase oxidative stress and genomic stability, making asthma more habitual and inadequately controlled in children . Recent studies shown that the disabled regulation of DNA damage signaling pathways in the lungs can impact the development.

Keywords- DNA damage , Exposomes, uncontrolled asthma, Children, environmental factors

Introduction

Exposomes encapsulate the aggregation of internal and external sources of chemical substances, biological agents, and psychological factors to which an individual is exposed over a lifetime(1). Internal exposomes involve factors like oxidative stress, inflammation, disordered metabolism and hormonal changes. Microbial exposure, passive smoking, air pollution and socioeconomic conditions are part of external exposomes(2,3).

Asthma is known to be one of the persistent disease and is caused by numerous elements including genetics, diet, environmental exposures, respiratory infections, and other factors. Studies show that around 10% of children and young people are found to be affected this condition worldwide, which is known by ongoing symptoms such as persistent cough, shortness of breath, wheezing, and chest tightness (4). It is a heterogeneous condition that varies from

person to person based on factors like age of onset, treatment response, clinical signs, environmental factors, and overall progression (5).

PREVALENCE

Globally, asthma affects about 339 million individuals, out of which 33% are children under the age of 14, 27% are adults who developed asthma in childhood, 40% are individuals diagnosed during adulthood. Taking children into account, about 5% of them had severe asthmatic condition which is hard to manage. As a result, they also experience serious problems including increased risk of life- threatening episodes, medication side effects, poor academic performance and emotional stress (6)(7). The children are more prone to damage and less reparable because of their slower lung development and the exposure to pollutants also found to escalate due to faster breathing rates, higher activity levels, and longer time outdoors. In contrast to adults, they frequently breathe through their mouths, allowing the pollutants to reach the deeper airways. Earlier in the 1900s, Western countries often saw asthma as a mental issue, so doctors mainly focused on talk therapy as a treatment (8).

Recent studies shown that the impaired regulation of DNA damage signaling pathways in the lungs can influence the development, progression and therapeutic outcomes of chronic respiratory disorders, such as asthma, bronchopulmonary dysplasia (BPD) and chronic obstructive pulmonary disease (COPD) (10). DNA damage is a well-known condition occurring in cells, caused due to numerous external factors including UV radiation, chemical substances and so on. Oxidative stress, chronic disorders and metabolism are some of the examples of endogenous factors. On a daily basis, each human cell continuously repairs the DNA damage triggered by replication mistakes and metabolic processes (10).

This review aims to elucidate the existing insight on environmental factors that leads to DNA damage in children suffering from uncontrolled asthma. It points out how the environmental exposures including air pollution, passive tobacco smoke, allergens and nutrition-related factors can increase oxidative stress and genomic stability, making asthma more chronic and poorly controlled in children (11).

EXPOSOME AND ASTHMA

Gene- terrain relationships are highly linked in asthma. However, heritability studies also proved that the environmental factors and their limitations impact as well. This is significant since genetic mutations cannot be prevented, but chemical exposures, once identified, help to prevent asthma caused by environmental factors, by reducing its contact at crucial periods of disease development (12). An asthma episode develops in two stages- early and late phases. The early stage begins when IgE antibodies, which are bound and held within the airways, become activated. Certain environmental factors trigger the antibodies to react, by which IgE antibodies starts to attach to immune cells. The mast cells slowly produce cytokines and other substances like histamine, prostaglandins and leukotrienes, in response to an allergen. As a result, the released substances induce the airway muscles to tighten, narrowing the airways (12).

Immune System

Th2 lymphocytes plays an important role because they produce GM- CSF and several interleukins (IL- 4, IL- 5, and IL- 13) which help the cells to communicate and sustain inflammation. IL- 13 contributes to hyperplasia, fibrosis, and redoing (13). Throughout the late phase, which extends for several hours, immune cells present in the body including neutrophils, basophils, eosinophils and the memory T-cells start migrating to the lungs, causing inflammation and bronchoconstriction. Mast cells are essential in

delivering the late-phase substances to the inflamed sites (14).

Understanding both pathways helps to guide the severe conditions of asthma, thereby reducing inflammation and bronchoconstriction. Over time, people with thicker airways tend to experience longer illness duration due to their narrower airways (15). Breathing becomes more difficult due to an occasional airflow restriction brought on by inflammation and bronchoconstriction.

Hyperresponsiveness

Airway hyper responsiveness can be caused by several processes. It may happen because the airway muscles get larger or because of the increased production of histamine by mast cells. The presence of high calcium levels inside airway muscle cells and stronger vagal tone make the muscles contract more (15). Tests like provocation test are performed to measure the degree of airway hyper responsiveness (16). This condition is often characterized with lower lung function and there is a high risk of asthma development from childhood ahead, which is recognized as clinically significant (17) and can be managed by early treatment. These changes leads to reduced lung elasticity, making difficulty in breathing.

Breathing becomes harder for people when more exudate, mucus, inflammation, and granular white blood cells fill the bronchioles. The smooth muscle layer and lamina reticularis shrink because of increased epithelial cells and myofibroblasts that produce collagen, which in turn narrows the airway walls. As a result, the basement membrane also thickens. This process, known as airway remodeling, is the cause of irreversible airflow restriction in humans (19).

Allergens

Extensive research identified dust mites as one of the major allergens linked to asthma and are a major source of indoor allergens. The two dominant species Der f 1 (*Dermatophagoides farinae*) and Der p 2 (*Dermatophagoides pteronyssinus*), are highly relevant. Der f 1 promotes inflammatory cell death in bronchial epithelial cells (19), and these allergens stimulate both the innate and adaptive immune responses 18. About 85% of children with allergic asthma exhibit sensitivity to these mites (20), which is associated with heightened inflammation, increased bronchial hyper reactivity, impaired lung function (21).

Mus m 1, the main mouse allergen, is found in mouse skin flakes, hair follicles, and urine. It binds to small particles that remain in the air for a long time. It is also present in household dust, with higher levels in kitchen compared to bedrooms (23). Previously, it was regarded an occupational hazard, but now it is strongly involved in asthma symptoms (24). Studies show that mouse allergen is highly prevalent in residential houses, with urban homes having higher amounts of the allergen than suburban and rural ones (25).

Pets

According to recent research, mice are more relevant urban allergens than cockroaches, even though dust mites are the most prevalent allergen sensitization in children with asthma (26, 27). According to the School Inner-City Asthma Study, mouse allergen was detected mostly in homes and schools, while it was much more common in schools. Independent of sensitization and exposure to home allergens, mouse allergen exposure was substantially linked to more days of asthma symptoms and a lower predicted FEV1 percentage, indicating the significance of the school environment in urban asthma (28).

The most commonly seen hairy pets in homes include dogs and cats, and allergies to these pets have been linked to severe asthmatic condition in children (29). About 12% of the general population and 25% to 65% of children with chronic asthma are sensitive to these allergens (30). The two main allergens are Fel

d 1 in cats and Can f 1 in dogs, are majorly found in saliva, hair follicles and skin. These allergens travel on tiny particles (less than 10–20 mm) that cling to clothes and remain in the air for longer periods (31).

Molds

Both the onset and severity of severe asthma can be attributed to mold exposure. Children exposed to mold show poorer lung function and more hyperresponsive airways compared to children who are not sensitive to mold (32,33,34). Studies show that newborns sensitive to mold experience recurrent wheezing. Recent Boston birth cohort study found a significant correlation between indoor dust-borne Alternaria at ages 2 to 3 months and the frequency of wheezing by the age of 1 year, even after controlling for outdoor airborne Alternaria concentrations (35).

Endotoxins

Endotoxins in homes can make wheezing worse (36) and can intensify asthmatics' airway reaction to allergens (37). A cross-sectional study found poverty, Mexican ethnicity, younger age, carpets, fuzzy pets, insects, and/or a household smoking as the markers of higher endotoxin levels (38). Endotoxins present in places like schools is also a factor that worsen asthmatic condition. Sheehan et al. found that inner-city schools had higher levels of settled-dust endotoxins than students' homes (39). In this group, After adjusting for exposures at home, children with nonatopic asthma showed greater symptoms of asthma when exposed to aerosol endotoxins in the classroom. (40).

Gut Microbiota

Research has shown that disruptions in the gut microbiota in early life is associated with increased risk of asthma later on (41). Arrieta et al. proposed that the first 100 days as a pivotal time during which altered gut microbiota induce newborns tendency to atopic wheezeThe gut–lung axis (42), which emphasizes the interaction between the gut and lung bacteria, is credited with causing the link. Due to the production of immune cells, bacterial ligands, and bacterial metabolites (including histamine and short-chain fatty acids), the gut microbiota can influence the lung immune response (42).

Lung Microbiota

Similar to the gut microbiome, The lung microbiota affects the lung's innate and adaptive immune responses through interactions with immune cells and the airway epithelium. The microbiome of the upper and lower airways, , which includes many microorganisms, contributes to asthma (41). Research show that certain groups of Patients with asthma often have bacteria. For example, microbial diversity was higher in patients with uncontrolled asthma, and this was positively correlated with bronchial hyperresponsiveness. (43).

Epigenetics

DNA methylation changes in genes directly associated with Th2 immunity and asthma are associated with allergic asthma in African American children living in inner cities (44). The IL4Rα-Q576R polymorphism is associated with both the severity and prevalence of asthma. . Recently, researchers found that the IL4Rα-Q576R genotype affects how endotoxin levels in urban classrooms is related to asthma symptoms days (45), showing a gene-environment interaction. Innate immunity genes (CD14, TLR4, and TLR2), which are crucial mediators of responses to bacteria in the extracellular space, are also a major component of gene-environment interaction studies of asthma-related phenotypes (46)

Chemicals

Particulate matter (PM), as well as chemical and biological compounds like sulfur dioxide (SO2), nitrogen dioxide (NO2), carbon monoxide (CO), and black carbon (CO) (47)

are all constituents of the omnipresent mixture of pollutants known as air pollution. Numerous studies indicate that exposure to air pollution can aggravate pre-existing asthma. Asthma has been linked to increased methylation changes in the Foxp3 gene promoter region (48), and exposure to elevated NO2, CO, and PM2.5 (aerodynamic diameter of 2.5 microns or less) levels. Taking into account the lag times between increases in air pollution and asthma exacerbations, a 2017 meta-analysis investigated the connection between outdoor air pollution and exacerbations (49).

Psychosocial

Later, people started to realize that the psychosocial environment also impacts asthma morbidity which include a person's social networks, family ties, locality, and socioeconomic status. According to Kopel et al., in inner-city school children with asthma, Childhood asthma morbidity is associated with the primary caregiver's perception of neighborhood safety, and asthma morbidity in children with asthma is associated with caregiver stress. (50,51).

CONCLUSION

In children, asthma is a major cause of morbidity, in which the development and its activity in children are greatly influenced by their surroundings. This review highlights the novel findings on how biological, physical, and psychological effects of the environment influence asthma. According to the research, depending on when an exposure occurs, certain environmental exposures may either be protective or a risk factor for asthma. Cohort studies should be the main focus of future study. and randomized trials to study important exposure periods during pregnancy, youth, and adulthood. Large sample size can be enrolled using a flexible study designs. Further more research is needed to find out the effective combined treatments and to validate the efficacy of single allergy treatments.

The laws and policies should be updated to control the pollution exposure and to address the social factors that cause unfavorable results. Health research should provide solid evidence required to support policy decisions. More creative and patient-centered therapies are required to understand the environmental impacts on asthma. Children with asthma are affected by many factors including biological, physical, and psychological influence. Depending on when exposure occurs, they may be preventive or a risk factor for developing asthma, or they may aggravate pre-existing asthma. Remediation is a crucial part of asthma care since allergen exposure is a major asthma trigger. However, there is a shortage of high-quality data on single component therapies.

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