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Established and Emerging Environmental Contributors to Disparities in Asthma and Chronic Obstructive Pulmonary Disease

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Abstract

Purpose of review—Multiple respiratory diseases, including asthma and chronic obstructive pulmonary disease (COPD), display significant socioeconomic and racial/ethnic disparities. The objective of this review is to evaluate the evidence supporting a link between disproportionate environmental exposures and these health disparities.

Recent findings—Studies suggest that various co-occurring factors related to the home environment, neighborhood environment, non-modifiable individual factors, and individual behaviors and attributes can increase or modify the risk of adverse respiratory outcomes among socioeconomically-disadvantaged and racially/ethnically diverse populations. Pollutants in the home environment, including particulate matter, nitrogen dioxide, and pesticides, are elevated among lower socioeconomic status populations and have been implicated in the development or exacerbation of respiratory-related conditions. Neighborhood crime and green space are socioeconomically patterned and linked with asthma outcomes through psychosocial pathways. Non-modifiable individual factors such as genetic predisposition cannot explain environmental health disparities but can increase susceptibility to air pollution and other stressors. Individual behaviors and attributes, including obesity and physical activity, contribute to worse outcomes among those with asthma or COPD.

Summary—The root causes of these multifactorial exposures are complex, but many likely stem from economic forces and racial/ethnic and economic segregation that influence the home environment, neighborhood environment, and access to healthy foods and consumer products.

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Critical research needs include investigations that characterize exposure to and health implications of numerous stressors simultaneously, both to guard against potential confounding in epidemiological investigations and to consider the cumulative impact of multiple elevated environmental exposures and sociodemographic stressors on health disparities.

Keywords

asthma; chronic obstructive pulmonary disease; disparities; environment; housing; neighborhood

Introduction

Significant health disparities exist in multiple respiratory outcomes, with a recent policy statement by the American Thoracic Society and European Respiratory Society concluding that groups with lower socioeconomic status (SES) are up to 14 times more likely to have respiratory diseases than the highest SES groups (1). For example, low-income and racially/ethnically diverse populations in the United States (US) have higher rates of asthma and more frequent use of emergency departments (ED); Puerto Ricans, non-Hispanic blacks, and those below the poverty line have the highest asthma prevalence, and the asthma ED rate is 3.3 times higher for blacks vs. whites (2). Similarly, chronic obstructive pulmonary disease (COPD) is much more prevalent among low-SES populations (3), with far higher rates in the US among individuals with less than a high school education and those with household income < $20,000 (4).

These health disparities are complex and multifactorial, but may be partly explained by environmental exposures and characterized as environmental health disparities, defined as inequities in illnesses that are mediated by disproportionate exposures associated with the physical, chemical, biological, social, natural and built environments (5). The emphasis on inequities rather than inequalities implies that predefined subpopulations, including racially/ethnically diverse populations and those of lower SES, are of particular concern. The empirical evidence for environmental health disparities would therefore be found in two different domains – the evidence that there are disproportionate exposures to environmental stressors by SES or race/ethnicity, and the evidence that these exposures contribute to the underlying inequities in illness (i.e., that there is a causal relationship between the disproportionately distributed exposures and the health outcomes in question). Both lines of evidence are required to conclude that environmental health disparities are present, and there is a growing body of literature that provides empirical evidence for both the underlying exposure patterns and the linkage with specific respiratory outcomes.

In this article, we focus on four domains hypothesized to contribute to environmental health disparities for respiratory outcomes – the home environment, the neighborhood environment, non-modifiable individual factors, and individual behaviors and attributes (Figure 1). While there are numerous respiratory outcomes with strong evidence for health disparities, we focus on asthma and COPD to illustrate broad trends in the literature. For each domain, we discuss key articles in the recent literature as of February 2018 to evaluate the two lines of evidence required to reach conclusions about environmental health disparities – the evidence for disproportionate exposures for low-SES or racially/ethnically diverse populations, and

the evidence that the environmental stressors with evidence for exposure disparities are strongly associated with asthma or COPD. We conclude by determining the implications for environmental health disparities.

Home environment

Numerous exposures in the home environment have been shown to be disproportionately distributed by SES or race/ethnicity, either based on first principles or empirical evidence. Indoor residential exposures are driven by indoor sources (e.g., smoking, cooking), outdoor sources (e.g., traffic, industrial activity), physical structure (e.g., size of living space, heating systems), and activity patterns (e.g., smoking behavior) (6). Lower-income populations tend to have smaller and older homes located closer to major pollution sources and with greater prevalence of indoor sources. Empirical evidence showed disparities in exposure to multiple air pollutants (including nitrogen dioxide (NO_2), fine particulate matter ($PM_{2.5}$), and volatile organic compounds (VOCs)); secondhand smoke (SHS) exposure, which refers to exposure to a mixture of exhaled mainstream smoke and side stream smoke released from the burning of cigarettes or other smoking devices (cigar, pipe, etc.) and diluted with ambient air (7); lead; pest allergens; and semivolatile organic compounds in air and dust (6). Another review article (8) emphasized broad-based disparities in housing quality, with 7.5% of non-Hispanic blacks but only 2.8% of non-Hispanic whites living in moderately substandard housing. This has implications for numerous housing-based exposures with linkages to health outcomes, including heat/cold, noise, mold, SHS, lead, air pollution, and pest allergens (9). Studies have also shown that adverse housing conditions (e.g., housing disrepair factors including presence of mold, water damage, peeling paint) prevalent in low-income households in both urban and agricultural settings increase the likelihood of pest infestations and subsequent residential pesticide use (10–15). Pesticide misuse is also common in low-income households due to inadequate knowledge and training (13). Exposure disparities also exist for multiple non-chemical stressors in the home environment. For example, low-income populations may be more challenged by housing instability or financial limitations related to paying for living expenses, which can contribute to psychosocial stress and increased risk of depression, among other impacts (16, 17).

Given this array of stressors, there is significant risk for housing-based environmental health disparities in both asthma and COPD. We conducted a literature search in PubMed using the keywords (housing or nitrogen dioxide or air pollution or mold or environmental tobacco smoke or stress or allergen or temperature or noise or pesticides) and (asthma or COPD). Individual articles in the recent literature were selected that were most directly relevant to the outcomes and exposures in question. We emphasized systematic reviews and articles that considered multiple stressors simultaneously, given both concerns about confounding and the disparities implications of multi-stressor exposures, and we focused on articles since 2013 for stressors with numerous publications but included earlier dates for stressors with more limited publications.

The evidence linking indoor air pollution exposure to asthma is extensive. In particular, there is ample evidence that SHS exposure is linked to higher asthma morbidity, with a recent systematic review finding an odds ratio of 1.85 for asthma hospitalization and 1.66 for ED or

urgent care visits (18). Both prenatal and postnatal maternal and paternal smoking have been associated with increased risk of wheeze (19); and SHS exposure has been associated with not only morbidity, but also with incident asthma (20). In a systematic review, findings regarding the impact of domestic fuel combustion were mixed (20), although increases in both indoor particulate matter (PM) and NO_2 (primarily associated with stove use) have been associated with pediatric asthma-related outcomes in multiple recent studies, including nighttime inhaler use (21), asthma symptoms, and medication use (22). NO_2 was also associated with incident asthma in a population-based birth cohort study (23). In addition to indoor sources, home environments can also be a place of exposure to ambient pollutants infiltrating from the outdoors and lower-income homes are often closer to pollution sources (6). A general consensus is that traffic-related air pollutants are associated with asthma prevalence and exacerbations (20). Other housing-based exposures with evidence for an association with asthma include mold, VOCs and pesticides (20).

Pesticides may increase the risk of asthma through interaction with functional irritant receptors in the respiratory tract that promote neurogenic inflammation, increased bronchial hyper-responsiveness (24), cholinesterase inhibition which may promote bronchoconstriction (25), and through effects of autoinhibitory M2 muscarinic receptors on the parasympathetic nerves in the lung (26). Although epidemiologic studies linking residential pesticide use to respiratory effects are limited and some have relied on self-reported pesticide use rather than biological or environmental measurements of exposure (27–31), these studies have reported positive associations between early life exposures to residential pesticide use and adverse respiratory outcomes in children.

In terms of multi-stressor studies or those considering the influence of housing more generally, a recent analysis from the Urban Environment and Childhood Asthma (URECA) study found that in addition to pollutant exposure, higher maternal stress and depression were associated with increased risk of developing asthma (32). Although the role of psychosocial stress in pediatric asthma has been challenging to define given the bidirectional nature of the relationship (i.e., severe asthma can itself be a stressor), psychosocial stress has been associated with asthma, through modifications to the activity of the hypothalamic-pituitary-adrenocortical (HPA) axis (33) as well as through behavioral pathways (i.e., caregivers who are unable to maintain the medication regimen for their children) (34, 35). Multiple studies have additionally shown interactions between air pollution exposures and psychosocial stress (36, 37). Regardless of potential mechanism or exposure, it has been shown that in general, poor housing quality has been associated with increased asthma diagnosis and morbidity (38), while “green” homes that reduce indoor environmental exposures have been associated with lower risks of asthma morbidity (39).

Although active smoking, which is more prevalent in low income communities (40), is the primary exposure leading to COPD in developed countries, the literature linking SHS exposure with COPD is less extensive. However, it is becoming more evident that residential and/or workplace SHS exposure also leads to impaired lung function and an increased risk of COPD in both never-smokers (41) and those with a heavy smoking history (42). A recent review also confirmed the role of both air pollution and temperature in contributing to COPD morbidity (43). In addition to outdoor air pollution, multiple additional stressors in

the indoor environment were also associated with COPD, including PM and NO_2 (44). Both hot and cold temperatures were associated with increased risks of COPD exacerbations, and the evidence for synergistic effects with air pollution appears suggestive, thus low-income populations who are more likely to have exposure to both air pollution and temperature extremes (6) would be at greater risk regardless of the nature of the interaction. Furthermore, children with asthma and lower respiratory infections are more likely to have chronic lung function decrements (45), implying that housing-related stressors that increase the risk of pediatric asthma and infections may also increase the risk of COPD later in life. There is also evidence of complex bidirectionality, wherein individuals with chronic lung disease are more likely to be housing insecure, attributable to the financial burden of medication for a chronic disease (46).

Neighborhood environments

Multiple aspects of the neighborhood environment continue to be unequally distributed across racial/ethnic and socioeconomic lines, as documented in the Institute of Medicine's updated report on Reducing Health Disparities (47). Residential segregation limits access to healthcare services, stores with healthy food choices, and outdoor spaces that encourage physical activity (47, 48). There is a higher density of tobacco outlets in low-income and racially/ethnically diverse neighborhoods (49), which has been associated with higher smoking rates in youth (50). Racially/ethnically diverse populations have long been shown to be exposed to higher rates of community violence (51), and have higher rates of youth group fighting and violence (52). Although total levels of air pollution have decreased in the last decade in the US, studies have shown an increase in ambient air pollution disparities across race/ethnicity, income and education for air pollutants such as $PM_{2.5}$ (53) and NO_2 (53, 54). Public parks located in low-income racially/ethnically diverse neighborhoods are closer to major roadways, with increased exposure to air pollutants (55), and nighttime and daytime noise levels are higher in areas with higher proportions of low-income and racially/ethnically diverse residents (56). Neighborhood green space has also been shown to be unequally patterned, and tied to community revitalization, affordable housing, neighborhood walkability, food security, job creation, and youth engagement (57). Green space disparities are driven by lack of amenities, but also by limitations in access because neighborhood conditions (i.e., high crime) discourage residents from using existing amenities (58, 59). To determine associations between neighborhood-level stressors that have been highlighted more recently and either asthma or COPD, we conducted a literature search in PubMed, using the keywords ((green space, crime, or violence) and (asthma, COPD, or respiratory disease)), limited to articles published since 2013. We also included articles that focused on the mediating impacts of neighborhood characteristics (e.g., green space and community violence) on the effects of air pollution exposures. Lastly, we touch upon the emerging evidence around residential segregation. Very limited COPD literature was available, thus results focus on asthma outcomes only.

Neighborhood crime – whether violent, property or drug abuse – has been associated with multiple asthma-related outcomes through hypothesized pathways including stress and depression (60–62). Children with asthma living in neighborhoods with higher rates of property, violent, and drug abuse crime have higher odds of lifetime asthma, lifetime

wheezing, and asthma hospitalization (63). Violent crime and physical disorder are associated with pediatric ED visits for asthma (64) and asthma utilization rates (65), and children who report being afraid to go out to their neighborhood have poorer asthma control (66). In addition, greater neighborhood problems have been associated with detrimental behaviors, including increased smoking and poor adherence to asthma controller medication use (67). Due to the heterogeneity in how exposure to community violence (ECV) is measured, no meta-analysis of ECV and asthma outcomes has been conducted (68).

A recent expert workshop hypothesized that biopsychosocial pathways linking green space to respiratory health fall into three domains: “reducing harm (e.g., reducing exposure to air pollution, noise and heat), restoring capacities (e.g., attention restoration and physiological stress recovery), and building capacities (e.g., encouraging physical activity and facilitating social cohesion)” (69). Exposure to green space is associated with reduced asthma hospitalizations (70), reduced wheezing and bronchitis (71), and reduced risk of asthma development in children living in areas of high pollution (72). Green space has also been shown to exert a protective effect on respiratory death (73) even after accounting for noise and air pollution, although a meta-analysis of related mortality studies did not confirm these results (74). Tree canopy density is associated with lower rates of asthma, even after controlling for green space (75). However, large variability in how greenness is measured across health studies to date may make definitive conclusions with a meta-analysis challenging (76, 77).

Multiple studies have started looking at interactions among neighborhood-level stressors on respiratory health, including between air pollution and neighborhood characteristics (often proxied by measures of SES). A meta-analysis found weak evidence of SES as an effect modifier of air pollution and asthma exacerbations (78); however more recent studies report children with asthma in low-income neighborhoods experience higher impacts when exposed to air pollution (79, 80). Large variability in how SES is measured may contribute to these conflicting results (79). Crowding and poor access to resources modified the association between NO_2 and pediatric ED visits for asthma (64). Even in non-asthmatic residents living in neighborhoods that are poorly maintained or experience stress from crime, those who have higher exposure to $PM_{2.5}$ have decreased lung function (66). Furthermore, exposure to green space may mitigate the impact of air pollution exposures on respiratory health (70, 72).

Residential segregation is hypothesized to contribute to asthma morbidity through multiple mechanisms at the neighborhood level such as neighborhood quality, medical care, and collective efficacy (81). A study looking directly at residential segregation found that neighborhood racial composition predicted asthma burden better than individual-level race/ethnicity, suggesting a strong effect of neighborhood conditions (82).

Non-modifiable individual factors

Environmental health disparities would be magnified if the same populations with elevated exposures to stressors were more susceptible due to individual factors. Several studies suggest that racial or ethnic subgroups have differing susceptibility to air pollution exposure

in regards to respiratory health (83). However, it has been difficult to discern whether these differences are due to socioeconomic factors, cultural differences (e.g., differences in dietary habits), and/or non-modifiable risk factors such as genetics. For example, there is evidence that genetic ancestry predicts asthma and chronic lung disease risk (84–87). Additionally, specific genetic polymorphisms have been shown to modify an individual's susceptibility to active smoking in terms of risk of developing asthma, COPD, and impairments in lung function (88–90). Limited studies investigating interactions between genetics and pollutant exposures also suggest that specific genetic polymorphisms may modify an individual's susceptibility to environmental exposures, including SHS, endotoxin and ambient air pollution. Specifically, several studies have investigated polymorphisms of the oxidative stress pathway, including glutathione-S-transferase M1 gene (GSTM1) and glutathione S-transfer P1 (GSTP1) gene polymorphisms. For example, children with asthma and the GSTM1 null genotype were more susceptible to ozone exposure (91). Further, findings from a recent genome-wide interaction analysis of air pollution exposure and childhood asthma suggest that several novel genes modify the impact of ambient NO_2 exposure on asthma development (92). Despite several studies highlighting the role of gene by environment interactions, differences in genetic risk are not sufficient to explain current environmental disparities observed in respiratory diseases, suggesting that other factors, including individual behaviors and attributes, may also play a role and warrant further examination. A recent study highlighted the complexity of such possibilities, in that children with asthma and genetic susceptibility thought to weaken antioxidant defense, combined with deficient dietary antioxidant intake, showed increased adverse effects of ozone on lung function (93). Therefore, exploration of individual behaviors and attributes and environmental disparities is also warranted.

Individual behaviors and attributes

Multiple individual behaviors and attributes plausibly associated with respiratory health display significant racial/ethnic exposure disparities. In particular, higher obesity rates and increased physical inactivity have been reported among racially/ethnically diverse and low-income populations in the US (94–100). In addition, these same populations disproportionately reside within food deserts and food swamps, areas with limited access to fruits, vegetables, and healthful whole foods in favor of processed, ready-made foods (101, 102). There is also emerging evidence of disparities in exposures to chemicals in consumer products, including personal care products (e.g., perfumes, lotions), and links between exposure to these agents and respiratory health. For example, racial disparities in paraben exposure (an antimicrobial agent present in many personal care products) have been documented with African Americans reported to have higher urinary paraben biomarker concentrations compared to other racial/ethnic groups in the US general population (103). While exposure to phthalates in the US general population is widespread given their presence in many consumer products, including personal care products (hair and skin care products), cleaning agents, PVC flooring, building materials, toys, detergents, and plastic food packaging, phthalate metabolite urinary biomarker concentrations in Hispanic and black populations are generally higher than those reported in non-Hispanic whites. Among women, higher concentrations of urinary phthalate metabolite concentrations have been

reported among racially/ethnically diverse pregnant women (104) and women of reproductive age, including those from socioeconomically-disadvantaged backgrounds (105). We therefore conducted a search in PubMed using the keywords (asthma and (personal or individual) behavior), (COPD and (personal or individual) behavior), ((personal care products or phthalates) and asthma), (diet and (asthma or COPD)), (poor diet and asthma), and (obesity and (asthma or COPD)).

Most studies to date support a positive link between obesity and asthma (106, 107), including associations with greater asthma morbidity and severity, and increased asthma prevalence and incidence rates (108). It is also hypothesized that asthma among obese individuals may reflect a unique asthma phenotype, with more severe disease that does not respond well to conventional treatment (106). Similarly, obesity is linked to worse outcomes in patients with COPD (109). The exact mechanisms by which obesity could impact chronic lung disease remain elusive and may be attributed to multiple factors, arising from alterations of the airways and lung parenchyma, to systemic and airway inflammatory and metabolic dysregulation, which negatively affects lung function and/or response to treatment. Notably, obesity has also been identified as a potential determinant of susceptibility to air pollution exposure in both individuals with asthma and COPD, with a heightened adverse response to $PM_{2.5}$ and NO_2 exposure and an increased systemic inflammatory response among overweight individuals (110, 111).

There is also suggestive evidence that diet may play a role on respiratory and allergic diseases (112–117). In the US, increased consumption of fast food and highly processed foods has been coincident with the dramatic rise in asthma. Furthermore, consumption of a “Westernized diet”, characterized by processed foods, high fat intake, less produce consumption, and poor nutrient intake, has been associated with higher asthma prevalence rates in children, while a Mediterranean diet has been associated with reduced asthma-related symptoms (116, 118). Dietary intake has been shown to modify systemic inflammation and the Western diet is believed to promote a pro-inflammatory response resulting from factors such as low antioxidant intake and abundance of saturated fatty acids. It has also been suggested that a poor diet could lead to alterations in the gut microbiota (119). These factors may increase susceptibility to oxidative stress and innate immune activation (120, 121). Thus, it is also reasonable that nutrition may modify air pollution health risks. Limited human studies suggest that adherence to a Mediterranean diet or high fruit and vegetable intake attenuates airway inflammation and changes in lung function in response to ambient air pollution exposure in children with asthma (122).

Studies investigating the role of chemicals in personal care products on respiratory health are limited and, for some chemicals, findings remain inconclusive. However, two recent cross-sectional studies (123, 124) reported an increased risk of allergic sensitization with increasing exposure to antimicrobial agents (triclosan and parabens) present in personal care products. Another study reported sex differences from paraben exposure on allergic sensitization (125). These studies did not identify links between paraben exposure and asthma (123, 125). Still, phthalates, in particular diethyl phthalate (DEP) has been associated with airway inflammation as assessed via exhaled nitric oxide levels among inner-city children (126). DEP is used as a scent retainer in cosmetics, personal care products, and

fragrances as well as an excipient in medications and supplements (31, 127). Furthermore, prospective studies (128–134) have reported associations between prenatal phthalate exposure (mostly, butyl benzyl and di-(2-ethylhexyl) phthalate, high molecular weight phthalates commonly found in PVC flooring and food packaging) and an increased risk of developing asthma and allergic outcomes though two studies did not observe any associations with prenatal exposures (135, 136). Phthalates could play a role on respiratory and allergic diseases by altering immune or inflammatory responses (137, 138), including modulating Peroxisome Proliferation Activated Receptors (PPARs) (139) and suppressing cytokine production (140).

Conclusions

There is a substantial literature supporting the contribution of environmental exposures to health disparities in chronic lung diseases including asthma and COPD. Evidence indicates that low-SES and racially/ethnically diverse populations have elevated exposures to multiple stressors of concern, including, but not limited to, air pollution, SHS, pesticides, substandard housing, community violence, lack of green space, unhealthy diet, and chemicals in select consumer products. Genetic and other non-modifiable factors may compound the effects of these elevated environmental exposures. The root causes of these multifactorial exposures are complex, but many likely stem from economic forces and racial/ethnic and economic segregation that influence the home environment, neighborhood environment, and access to healthy foods and consumer products. Critical research needs include investigations that characterize exposure to and health implications of numerous stressors simultaneously, both to guard against potential confounding in epidemiological investigations and to consider the cumulative impact of multiple elevated environmental exposures and non-chemical stressors on health disparities.

Human and Animal Rights

This article does not contain any studies with human or animal subjects performed by any of the authors.

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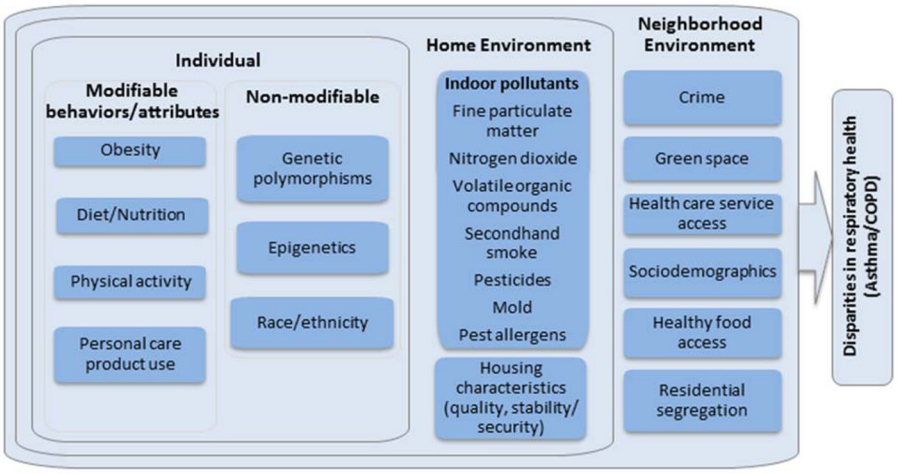

Figure 1.

Conceptual diagram indicating the contributions of four domains – the home environment, the neighborhood environment, non-modifiable individual factors, and individual behaviors and attributes – to environmental health disparities for respiratory outcomes.