



Ambient Air Pollution and Asthma-Related Outcomes in Children of Color of the USA: a Scoping Review of Literature Published Between 2013 and 2017

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Abstract

Purpose of Review Given racial disparities in ambient air pollution (AAP) exposure and asthma risk, this review offers an overview of the literature investigating the ambient air pollution-asthma relationship in children of color between 2013 and 2017.

Recent Findings AAP is likely a key contributor to the excess burden of asthma in children of color due to pervasive exposure before birth, at home, and in school. Recent findings suggest that psychosocial stressors may modify the relationship between AAP and asthma.

Summary The effect of AAP on asthma in children of color is likely modulated by multiple unique psychosocial stressors and gene-environment interactions. Although children of color are being included in asthma studies, more research is still needed on impacts of specific criteria pollutants throughout the life course. Additionally, future studies should consider historical factors when analyzing current exposure profiles.

Keywords Asthma · Air pollution · Health disparities · Race · Ethnicity

Introduction

Approximately 11 and 17% of all asthma cases and asthma-related deaths in children under age 20 across the globe are believed to result from environmental exposures [1]. One such environmental exposure, ambient air pollution (AAP), likely plays a contributory role in asthma pathogenesis among these individuals [1]. One of the growing AAP threats is

anthropogenic in nature, resulting from the burning of fossil fuels. At an international level, AAP is worst in low- and middle-income countries, with countries of Sub-Saharan Africa and South Asia bearing the worst burden [2]. While these locations unquestionably shoulder the highest exposure, AAP is a public health concern in high-income nations as well. A study published in 2012, conducted in California, found that between the years of 2001 to 2006, 30.6 and 36.1% of people experienced particulate matter with aerodynamic diameter < 2.5 μm (PM_{2.5}) and ozone levels, respectively, that exceeded the daily National Ambient Air Quality Standards set by the Environmental Protection Agency [3].

The link between AAP and respiratory health has been repeatedly demonstrated in the scientific literature, especially with regard to asthma [4–6]. The postulated mechanism for AAP-triggered asthma development or exacerbation (“attack”) in those with asthma is through oxidative stress to the respiratory tract. Oxidative stress leads to an immune response, thereby triggering an inflammatory cascade that can ultimately lead to acute and chronic obstruction of the airways [7]. Multiple genetic variations may also contribute to an individual’s susceptibility to develop asthma and increase exacerbation risk via

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AAP [5]. Furthermore, health disparities in asthma outcomes manifest among different racial/ethnic and socioeconomic groups in the USA; moreover, the communities that carry a disproportionate burden of asthma also carry a disproportionate burden of AAP exposure. Estimated at \$81.9 billion per year, the costs of asthma are felt most by low socioeconomic status (SES) communities of color, who experience two times more unplanned health care visits and missed school days compared with all other groups [8, 9•]. A recent study demonstrated that the prevalence of asthma in the USA is more than twice as high among African American children (15.7%) and nearly twice as high in children of Puerto Rican (12.9%) descent than in non-Hispanic white children (7.1%) [9•].

In any child, living with asthma can have wide-ranging impacts that affect not only the individual but also the family unit. For instance, uncontrolled asthma is associated with missed school days, poor academic performance, excess medical costs, and increased visits to the physician [10]. In addition to these challenges, children of color likely encounter other structural and social factors contributing to this uneven disease distribution. Studies from the past 5 years have suggested that children and youth of color are more likely to go to the emergency department (ED) for asthma-related care, are less likely to seek specialty care, and may have asthma-related priorities that are not addressed by their local policy makers [11•, 12, 13•]. Considering these factors in conjunction with environmental exposures allows for a clearer picture; children of color are more likely to live in neighborhoods with AAP levels that exceed the limits set by federal ambient air quality standards [14–16]. Even with an increase in the number of fuel-efficient vehicles on the roads and tightened emission regulations, a recent study suggested a continued disparity with mean nitrogen dioxide (NO₂) levels, a traffic-related air pollutant, still 37% higher in non-white populations [17•].

This review compiles articles published between 2013 and 2017 and investigates the association between AAP and asthma in children of color in the USA in addition to exploring potential effect modifiers and biologic mechanisms of these relationships.

Ambient Air Pollution and Pulmonary Function

Associations between AAP and decreased pulmonary function have been well established, yet few studies have investigated impacts solely in children of color. Improvements in air quality in Southern California were recently linked to improvements in lung function, although not specifically in children of color [18•]. Recent studies that have included a high proportion of children of color (> 65% in each) with and without asthma have found associations between both NO₂ and polycyclic aromatic hydrocarbons (PAHs) with decreased forced expiratory volume in 1 second (FEV₁) [19, 20]. However, the association between

PAHs and lung function was only statistically significant in children without asthma [20].

The Study of African Americans, Asthma, Genes, and Environments (SAGE II) and the Genes-environments and Admixture in Latino Americans (GALA II) were two case-control studies, the only of their kind to date, originally designed to examine the complex genetic and socio-environmental contributors to asthma prevalence and morbidity in children of color. GALA II and SAGE II consisted of self-identified Latino and African American children, respectively, with and without asthma aged 8 to 21 recruited from four different cities in the USA (New York City, Chicago, Houston, and the San Francisco Bay Area) and Puerto Rico. The SAGE II participants were solely recruited from the San Francisco Bay Area. In one GALA II and SAGE II analysis, the impact of AAP on spirometric lung function was examined [21•]. Exposure to NO₂, sulfur dioxide (SO₂), PM_{2.5}, particulate matter with aerodynamic diameter of <10 μm (PM₁₀), and ozone (O₃) was calculated per calendar year of each participant's life using distance-weighted averages from the nearest four air monitoring stations. A case-only analysis of 1449 Latino and 519 African American children with asthma by Neophytou et al. found that a 5 μg/m³ increase in average lifetime PM_{2.5} exposure was associated with a 7.7% (95% confidence interval (CI) – 11.8, –3.5%) decrease in FEV₁ [21•]. Although this finding was significant overall, the region-specific observed decreases in FEV₁ in relation to lifetime PM_{2.5} exposure was significant only in children from two of the five regions, San Francisco and Puerto Rico. Interestingly, these two areas also happened to be the regions with the lowest observed PM_{2.5} exposures.

Ambient Air Pollution and Asthma

Few studies have focused directly on the effects of AAP in communities of color; from the period of 2013 to 2017, we identified one study that examined the association of AAP and asthma diagnosis specifically in children of color [22•]. Similar to Neophytou et al., Nishimura et al. estimated the association between asthma and early-life exposure to AAP in the SAGE II and GALA II studies. Asthma cases were defined as physician-diagnosed asthma in addition to at least two of three respiratory symptoms (cough, wheeze, or shortness of breath) in the 2 years preceding recruitment. After adjustment for confounders, a 5 parts-per-billion (ppb) increase in average NO₂ exposure in the first year and first 3 years of life was associated with a 1.17 (95% confidence interval (CI) 1.04–1.31) and 1.26 (95% CI 1.07–1.48) increased odds, respectively, of having asthma. This study was the first of its kind to causally associate AAP exposure with asthma diagnosis in children of color.

Expanding the review to include studies that focused on urban populations, where on average about 50% of the population identifies as people of color, seven additional studies

were identified that examined the relationship between AAP and asthma [23]. Three studies in particular attempted to quantify an interaction between race and AAP on asthma morbidity, asthma-related hospitalizations, and respiratory symptoms in urban environments, although results were mixed [24, 25, 26••]. Berhane et al. longitudinally studied 4602 children, aged 5 to 18 years in Southern California, among whom almost 20% had asthma and 59.1% identified as a race other than non-Hispanic white. The results of this study strongly suggested that decreases in annual averages of exposure to ozone, PM_{2.5}, PM₁₀, or NO₂ were associated with decreased odds of bronchitic symptoms among children with and without asthma, yet tests for interaction between race and AAP exposure were insignificant [26••].

Delfino et al. studied over 11,000 asthma-related hospital admissions or ED visits among 7492 individuals, 64% who identified as non-Hispanic white, aged 0–18 years in Orange County, California. Lag periods of 1, 3, 5, and 7 days were calculated for overall exposure to CO, NO₂, NO_x, O₃, and PM_{2.5} by using subject addresses and air quality data from the nearest US Environmental Protection Agency's Air Quality System monitoring station. In order to investigate heterogeneity of effect by traffic-related air pollution (TRAP) exposure, authors stratified above and below the median of predicted TRAP using a California LINE Source Dispersion Model that uses wind patterns and distance from nearby roadways. In seasonally stratified analyses, an *interquartile range-increase* in ozone and PM_{2.5} were associated with increased asthma-hospitalization and ED visits during the warm season (May–October), while the same was true for exposures to PM_{2.5}, NO_x, and CO during cooler seasons (November–April). The regression analysis stratified by above and below median predicted exposure to TRAP indicated greater risk of asthma-related hospital visit or admission among those living in areas with higher predicted exposures, even though exposure to TRAP was only a fraction of overall ambient pollution (< 10% in both cases). Hispanic and African American participants, as well as subjects without private insurance, were more likely to live in residences with higher TRAP. This suggests that even at low concentrations and regardless of season, living in an area with above-median TRAP may increase asthma hospitalization risk [24].

Lastly, Alhanti et al. conducted an age-stratified multi-city analysis on asthma-related ED visits with AAP in Dallas, TX; St. Louis, MI; and Atlanta, GA. Age-group specific rate ratios were calculated within each city and then combined across cities to estimate the variance-weighted rate ratios for the prior 3-day average exposure to PM_{2.5}, PM₁₀, NO₂, CO, and ozone with ED visits. The number of asthma ED visits was greatest among non-white participants across all age groups in every city except in those > 65 years old and stratification by white or non-white race suggested effect modification with respect to NO₂, PM_{2.5}, and O₃ exposures; no evidence for effect

modification was seen with PM₁₀ and CO [25•]. Similarly, Wendt et al. also found race-dependent risk effects of air pollution on respiratory health. Using data gathered from 18,289 medical records of asthma diagnoses of children under the age of 18 in Harris County, TX, Wendt et al. found that short-term changes in NO₂, O₃, and PM_{2.5} correlated with timing of first asthma diagnosis. These observed effects were strongest in children of color, as a 10-ppb increase in mean 6-day level of O₃ corresponded to increased odds of asthma diagnosis of 1.08 (95% CI 1.03–1.13) in African American children, 1.03 (95% CI 1.00–1.07) in Hispanic children, and 1.01 (95% CI 0.93–1.10) in non-Hispanic white children [27].

Nowhere to Hide: Exposures in the Womb, Playground, and Classroom

We identified six new studies from 2013 to 2017 that demonstrate the pervasiveness of AAP exposure. Some children, especially those living in urban environments, may be exposed while in their bedrooms, while at school, and while at play. To measure indoor exposures, Dutmer et al. installed monitoring devices within the homes of inner-city participants and found that NO₂ levels in the bedroom were at levels similar to those found in curbside [28]. Past and recent studies demonstrate that indoor exposures to AAP at home and in school may trigger asthma and asthma-related symptoms [19, 29]. Academic performance has also been shown to be inversely correlated with traffic-related PM_{2.5} emissions [30]. Recent studies have built upon prior work by McConnell et al. that demonstrated a negative effect of AAP on asthma risk in children exercising outdoors by finding that childhood activity levels are associated with markers of respiratory inflammation and pollution-related urinary metabolites. This body of work suggests that the positive benefits of active lifestyle may be diminished by AAP [31, 32•, 33].

Prenatal exposure to AAP is also implicated as a driver of asthma risk. Previous studies have identified associations between prenatal AAP exposure and subsequent decreased birth weight, increased risk of infection, and elevated BMI [34–37]. We found one study from the past 5 years that was focused on prenatal exposures and subsequent asthma diagnosis. A study by Hsu et al. indicates that prenatal exposure to AAP is associated with asthma by age 6 and suggests that fetal susceptibility to PM_{2.5}-associated asthma may vary throughout the pregnancy [38••]. The Asthma Coalition on Community, Environment and Social Stress cohort included 736 full-term births recruited in Boston, MA, 54% of which included Hispanic mothers and 30% African American. Cases were determined by physician diagnosis by age 6. Prenatal exposure was ascertained geospatially via satellite-derived aerosol optical depth calibrated daily through 78 air monitoring stations around New England. This technique allowed researchers to quantify daily AAP exposure over the course of

each participant's pregnancy with enhanced resolution relative to other studies in the field. Of the 736 observed births, 110 participants developed asthma by age 6. Using distributed lag models, the authors identified weeks 16 to 25 as the window of greatest susceptibility, a finding conserved after sensitivity analyses accounting for postnatal exposure to PM_{2.5} and birth weight-adjusted gestational age. Models were adjusted for a variety of confounders including postnatal secondhand smoke exposure, maternal age, race, and ethnicity. Additional analyses unveiled a potential sex-specific vulnerability to PM_{2.5} between weeks 12 and 26 in boys, but not in girls, as well as a statistically significant ($p = 0.01$) interaction between PM_{2.5} and sex.

Race and Disproportionate Exposures

While existing evidence strongly suggests that AAP exposure, both short and long term, in and of itself is of serious concern, discussing exposure distribution among populations is also imperative. Kingsley et al. analyzed the distance of 114,644 schools from major cities in the USA and found that schools with > 50% African American students were 18% more likely to be located within two tenths of a mile from a major roadway than schools with ≤ 50% African American students [39••]. In addition to tracking with African American race, schools eligible for Title I funding, federal funding offered to schools where > 40% of student are from low-income families, and schools with > 50% of students eligible for free or reduced price meals also were associated with close vicinity to major roadways [39••]. Similar findings of disproportionate AAP exposures among children of color are corroborated by Weaver et al., who found that among a sample of 5000 children from California, Hispanic children were about one-and-a-half times as likely to live less than three tenths of a mile from a major freeway and less than one tenth of a mile from a major non-freeway roadway than non-Hispanic white children. [40••]

Psychosocial Stress and Air Pollution: an Avenue for Interaction

Similar to AAP, exposure to psychosocial stress is another potential driver of childhood asthma. Among African American, Mexican American, and other Latino children from the San Francisco Bay Area who participated in the SAGE II or GALA II studies, SES, as determined by maternal education, health insurance type, and household income, was predictive of greater odds of asthma in African American children, lower odds in Mexican American children, and no association in other Latino children [41]. In conjunction with low SES, exposures to other psychosocial stressors such as adverse childhood events, perceived discrimination, gun violence, and lack of quality insurance coverage are linked to

increased asthma risk and decreased pulmonary function in children [42, 43••, 44–47]. In addition, both pre- and postnatal stress experienced by the mother of the child has been linked to increased asthma and wheeze risk, suggesting a complex mechanism of stress-related respiratory pathogenesis [44, 48, 49]. This is meaningful, as exposure to psychosocial stressors often co-occurs with high exposure to AAP. In Canada, where income equity is significantly better than in the USA, Pinault et al. were still able to demonstrate that exposure to NO₂ is inversely associated with SES [50, 51].

Given these co-exposures of psychosocial stressors and AAP, researchers are increasingly focusing their efforts on elucidating potential interactions between the two on respiratory health. Prior work has identified associations between parental stress and asthma as well as synergism between AAP and neighborhood violence on asthma risk [52, 53]. In the last 5 years, we identified four articles that continue to build upon these findings, although none of the articles focused specifically on children of color.

We identified three studies that focused on the interactive effect of pre- and/or postnatal exposures to psychosocial stress and AAP on asthma outcomes. Two of these articles focused on prenatal exposure to AAP and stress on future respiratory health. Prenatal AAP exposure, specifically to PM_{2.5} and nitrates (NO₃⁻), coupled with maternal history of negative life events and community violence ascertained by validated surveys, respectively, is associated with increased risk of more than two wheezing episodes by age 2 and asthma by age 6 [54•, 55]. The final study, a meta-analysis, demonstrated an interaction between AAP and adverse childhood events (ACEs) on asthma risk; however, heterogeneity by race was not considered [42].

Kranjac et al. conducted an ecological analysis to identify neighborhood characteristics that correlated with pediatric asthma diagnoses [56•]. Neighborhoods in the Houston area were categorized into advantaged, middle-class, and disadvantaged communities based on socioeconomic factors, racial segregation, access to green space, and crime rates. This analysis revealed that children in the disadvantaged and middle-class neighborhoods shouldered a disproportionate burden of PM_{2.5} and O₃ exposures compared with those in the advantaged neighborhoods. Furthermore, middle-class and disadvantaged neighborhoods, unsurprisingly, demonstrated distinct racial and ethnic divides, and asthma rates tracked similarly; children in disadvantaged neighborhoods had nearly twice as many asthma diagnoses than those in the advantaged communities and about 33% more than those in the middle-class neighborhoods. Stratified analyses by race indicated that children in advantaged neighborhoods were less likely to have asthma than children of the same race in other neighborhoods, further establishing that factors outside of heritability play a crucial role in disease pathogenesis. However, racial disparities in asthma persist even when accounting for

neighborhood factors: African American children have higher rates of asthma than white children from the same neighborhood, signifying that other social and structural factors may be at play. As children in the disadvantaged communities likely experience greater psychosocial stress, shoulder a greater proportion of air pollution, and suffer a greater burden of asthma risk, it is logically and statistically plausible that these exposures interact, at least additively, to impact asthma incidence and exacerbation.

Genetic Susceptibility

Given the multifactorial and heterogeneous nature of asthma pathogenesis, understanding heritability factors is complexed, especially in consideration of environmental and social risk factors [57]. Several studies have examined the role of genetic ancestry, gene-by-environment (GxE) interactions, and epigenetic changes (specifically DNA methylation) in untangling susceptibility in high-risk populations. While genetic variation is greater between individuals than between racial/ethnic groups, specific genetic variations may cluster within certain ancestral groups [58–60]. Pino-Yanes et al. examined the differential susceptibility to developing asthma among Latino-American children with varying levels of global African, European, and Native American genetic ancestry and found that African ancestry is associated with greater odds of asthma and lower lung function [61]. However, in a related study of this population, no interaction between percent African ancestry and air pollution on lung function among children with asthma was found [21•]. The lack of an interaction between AAP and ancestry may result from the study's focus on global genetic ancestry estimates rather than on local ancestry estimates at specific loci, which may play a more important role.

GxE studies postulate that response and potential development of disease as a result of one's environmental exposures are dependent upon the individual's genotype, which dictates response and factors into susceptibility. Multiple studies have demonstrated that specific genetic polymorphisms attenuate response to environmental exposures, potentially augmenting individual risk for disease [62–66]. One GxE study by Bunyavanich et al. examined physician-diagnosed asthma, asthma medication use, and asthma hospitalization among twins born in a Puerto Rican birth cohort [62]. Twins were followed up at age 1 and age 3 and were categorized into monozygotic and dizygotic groups. There were no differences in asthma diagnosis risk and medication use during the first year of life between monozygotic and dizygotic twins. However, significant differences in asthma risk, medication use, and hospitalization were observed between the twin types at age 3, suggesting that even minor genetic differences between siblings may play a key role in asthma development. Although the investigators of this study acknowledge that physician diagnosis of asthma before age 6 is precarious, the findings, at the very

least, suggests that response to the same environmental conditions can be dependent on minor genetic differences.

In addition to GxE studies, researchers have also begun to investigate the impacts of air pollution in the context of epigenetics. Jung et al. measured black carbon exposure over two 24-h time points over 6 months in 163 Dominican and African American children from New York City. Buccal cells and exhaled nitric oxide, a marker of airway inflammation, were also collected, measured, and used as proxies for lower airway epithelial cell function and airway inflammation, respectively. Black carbon exposure 5 days prior was inversely associated with increased methylation of the promoter region for interleukin-4, a gene implicated in allergic asthma. Furthermore, the analysis revealed greater epigenetic sensitivity in children who were exposed to cockroach allergen, perhaps suggesting that those living in poorer housing conditions may be at increased susceptibility to black carbon exposure and that genetic expression of asthma genes may be altered in response to short-term exposures [67].

Hypothesized Biologic Mechanisms

As outlined above, the relationship between AAP and asthma is likely dependent on short- and long-term exposures and modified by a variety of co-factors such as genetic susceptibility and neighborhood, economic, and societal stressors. The Committee on the Medical Effects of Air Pollutants in the UK offered four primary mechanisms in the pathway between AAP and asthma exacerbations, including oxidative stress and damage, airway wall remodeling, inflammatory pathways and immunological effects, and enhancing respiratory sensitization to allergens [7]. While it is possible that genetic variations may contribute to the disparity in asthma prevalence between children of color and non-Hispanic white children, children of color are also more likely to experience greater exposures to AAP and prenatal and postnatal psychosocial stress. Psychosocial stress may interact with AAP to both cause and exacerbate asthma, as acute and chronic psychosocial stress increases circulating inflammatory cytokines in humans [68, 69].

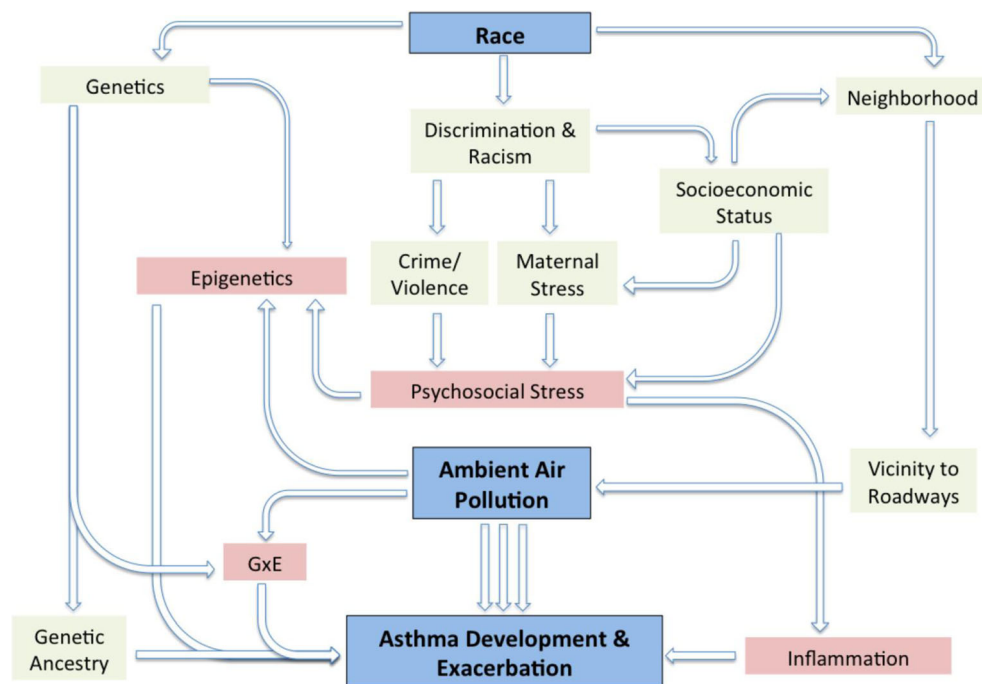
Conclusions

This review of recent literature on AAP and asthma in children of color in the United States has led us to the following conclusions:

- 1) Asthma disparities are worsened by a greater burden of AAP exposures in children of color and likely modified by psychosocial stressors.

Figure 1 depicts the relationships and pathways between race, AAP, and asthma as suggested by our recent literature search.

Fig. 1 Concept map of associations between AAP, asthma, and other risk factors as identified by this review. Blue boxes indicate key variables of interest of the review, green boxes indicate asthma risk factors or other exposure variables, and red boxes indicate biologic mechanisms through which asthma manifests



2) Variation in criteria air pollutant selection and findings

Although we have reviewed multiple AAP studies from the past five years that identified asthma development, risk for exacerbation, and lung function (Table 1) in children of color, different pollutants were studied and findings were mixed. The most widely studied and most consistent criteria pollutant in this population is NO_2 . In the six studies we identified, none were inconclusive; all suggested that with increased NO_2 exposure, risks for bronchitic symptoms, asthma, and asthma hospitalization increase. An equally common criteria pollutant in studies including children of color was $\text{PM}_{2.5}$, yet outcomes studied for this pollutant vary, including bronchitic symptoms, lung function, and asthma risk. Four studies included an analysis of O_3 and four studies included an analysis of PM_{10} . Two in each group identified direct relationships between the pollutant and asthma risk. Only two studies included CO and SO_2 , while no studies included lead. Future research should attempt to capture and quantify at least three of the four pollutants listed here in order to produce well-rounded literature that can be used to inform policy.

3) Emerging associations with prenatal exposures and effect modification by sex

Results from five studies published in the past several years indicate that fetal lung development is susceptible to transplacental AAP exposure and maternal stress [44, 48, 49, 54, 55]. Of these studies, three focused solely on AAP exposure and included exposure to prenatal $\text{PM}_{2.5}$ and nitrates. Each of these studies also noted a higher risk of poor respiratory

outcomes in boys than in girls, suggesting that boys may be especially susceptible to AAP in the in utero period. Given the connection of other AAPs such as O_3 to asthma risk, other exposures should be considered in future studies [71, 72]. In addition, other prenatal exposure metrics should be considered such as exhaled nitric oxide, which could provide more direct indication of maternal respiratory inflammation and, possibly, exposure. Future studies should also consider an interaction between prenatal AAP exposure and maternal stress.

4) Research on epigenetic changes in response to AAP is an unmet need

Although we identified several studies that measured how AAP may trigger epigenetic changes in children of color, we found no studies that adjusted for proxies of stress such as socioeconomic status, perceived discrimination, or maternal stress [67, 73, 74]. Future studies should attempt to quantify an association between prenatal parental stress and childhood stress with AAP-associated epigenetic changes in children, as plant, animal, and human models have demonstrated that stress can trigger both acute and heritable, albeit reversible, epigenetic changes [75–77].

5) A need to investigate how historical factors have contributed to disproportionate AAP exposure in people of color

The segregation of people of color during the last century alone likely contributes to, if not causes, the disproportionate burden of AAP exposures experienced by people of color today [15, 17, 30]. An example of this is redlining, which,

Table 1 Comparison measured effects of criteria pollutants on lung function and asthma risk in children of color

Author	Study population (age)	Outcome	Exposure type	PM _{2.5}	PM ₁₀	NO ₂	O ₃
Neophytou A [21••]	26.4% African American 73.6% Latino	FEV1	Lifetime average	5 µg/m ³ increase associated with 7.7% decrease in FEV ₁	No effect	No effect	No effect
Gentile DA [45]	53.6% African American	Asthma exacerbation		–	–	Increased odds	–
Nishimura KK [22••]	26.4% African American 73.6% Latino	Asthma diagnosis	First year and first 3 years of life	No effect	No effect	Increased odds	No effect
Wendt JK [27]	26.1% African American 2.9% Asian 61.3% Hispanic 0.5% Native American 7.9% White 1.4% Unknown	Asthma diagnosis	Short-term exposure	No effect	–	No effect	Increased odds
Khreis [70]	Meta-analysis of international birth cohorts	Asthma risk	Lifetime average	Increased odds	Increased odds	Increased odds	–
Berhane [26••]	3.7% African American Asian: 5.8% 45.2% Hispanic white 40.9% non-Hispanic white 4.3% Other	Bronchitic symptoms	Average yearly concentration	Decreased odds with 6.8 µg/m ³ reduction	Decreased odds with 5.8 µg/m ³ reduction	Decreased odds with 4.9-ppb reduction	Decreased odds with 0.66 µg/m ³ reduction
Hsu HH [38••]	29.6% African American 53.7% Hispanic 16.7% Other/white	Asthma diagnosis in child	Weekly PM _{2.5} concentration during pregnancy	Increased levels during weeks 16–25 associated with asthma	–	–	–

starting in the 1930s, forced people of color into less-desirable and oppressed neighborhoods by both refusing to offer loans to individuals seeking to invest in neighborhoods of color and by refusing loans to people of color [78]. Furthermore, this process was enabled by the federal government through the Home Owner's Loan Corporation, an office created in 1935 by the New Deal, tasked with drawing maps of cities divided by neighborhood desirability, which largely depended on the presence of immigrants or people of color [78]. While certain ancestral genetic factors may offer minor contributions to increased asthma risk in children of color, research that focuses solely on ancestral genetic factors risk appearing myopic; the divide in asthma burden that currently exists between children of color and non-Hispanic white children likely exists due to disparities in access to resources, psychosocial stressors, and AAP exposures. Moreover, the distribution of these factors is inextricably linked to historical and ongoing structural discrimination. Future studies should not only mention the likely role of these drivers of health disparities but also attempt to quantify the health impacts of structural discrimination when at all possible.

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Compliance with Ethical Standards

Conflict of Interest Dr. Thakur reports grants from the National Heart, Lung, Blood Institute and grants from Parker B. Francis Fellowship Program, during the conduct of the study. The other authors declare no conflicts of interest relevant to this manuscript.

Human and Animal Rights and Informed Consent This article does not contain any studies with human or animal subjects performed by any of the authors.

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- Of major importance

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