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Ambient air pollution and asthma-related outcomes in children of color of the United States: a scoping review of literature published between 2013 and 2017

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Structured 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 also suggest that psychosocial stressors may interact with the pathogenic pathways of AAP.

Summary: The effect of air pollution 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, current exposure profiles resulting from historical factors like redlining should also be considered in future analyses.

Keywords

asthma; air pollution; health disparities; race; ethnicity

Introduction

Approximately 11% and 17% of all asthma cases and asthma-related deaths across the globe in children under age 20 are believed to result from environmental exposures. One such

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environmental exposure, ambient air pollution (AAP), likely plays a key 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 greatest burden, AAP is a public health concern in high-income nations as well, especially in urban areas. A study published in 2012 found in California, the most populated state in the United States, that 30.6% and 36.1% of people experienced $PM_{2.5}$ (particulate matter with aerodynamic diameter <2.5 μm) and ozone levels, respectively, that exceeded the National daily Ambient Air Quality Standards set by the Environmental Protection Agency from 2001 to 2006.[3]

The link between AAP and respiratory health has been repeatedly demonstrated in the scientific literature, especially with regards 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 AAP.[5] Furthermore, health disparities in asthma outcomes manifest among different racial/ethnic and socioeconomic groups in the United States, and the communities that carry a disproportionate burden of asthma also carry a disproportionate burden of AAP exposure. Estimated at $81.9 billion, 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 United States 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 non-Hispanic white children (7.1%).[9]

In any child, living with asthma can have wide-ranging impacts that not only affect 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 five years have suggested that children and youth of color are more likely to go to the emergency department (ED) for asthma-related care, less likely to seek specialty care, and may have asthma-related priorities that are not addressed by their local policy makers.[11–13] 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 emissions regulations, a recent study suggested a continued disparity with mean nitrogen dioxide (NO_2) levels, a traffic-related air pollutant, still 37% higher in non-white populations.[17]

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

Ambient Air Pollution and Pulmonary Function

Exposure to AAP is been well established to be associated with decreased pulmonary lung function, 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] Other studies that have included a high proportion of children of color (>65% in each) with and without asthma have found associations between NO_2 and polycyclic aromatic hydrocarbons (PAHs) with decreased FEV_1.[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 United States (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 study, the impact of AAP on spirometric lung function was examined.[21] Exposure to NO_2, sulfur dioxide (SO_2), $PM_{2.5}$, PM_{10}, and ozone (O_3) were 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 1,449 Latino and 519 African American children with asthma by Neophytou *et al.* found that a 5 $\mu g/m^3$ increase in average lifetime $PM_{2.5}$ exposure was associated with a 7.7% (95% confidence interval (CI): −11.8%, −3.5%) decrease in forced expiratory volume in one second (FEV_1).[21] Although this finding was significant overall, the region-specific observed decrease in FEV1 in relation to lifetime PM2.5 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 two years preceding recruitment. After adjustment for confounders, a 5 parts-per-billion (ppb) increase in average NO_2 exposure in the first year and first three 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.[24–26] Berhane *et al.* longitudinally studied 4,602 children, aged 5 to 18 years in Southern California, among whom almost 20% had asthma and 59.1% identified as other than non-Hispanic white. The results of this study strongly suggested that decreases in annual averages of exposure to ozone, $PM_{2.5}$, PM_{10}, or NO_2 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 one, 3, 5, and 7 days were calculated for overall exposure to CO, NO_2, NO_x, O_3, 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 TRAP exposure, authors stratified by above and below the median of predicted TRAP using a California LINE Source Dispersion Model that uses wind patterns and nearby roadways. In seasonally-stratified analyses, exposure to ozone and $PM_{2.5}$ were associated with increased hospitalization and ED visits for an interquartile range increase in each pollutant in 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 $PM_{2.5}$, PM_{10}, NO_2, CO, and ozone with ED visits. The number of asthma-ED visits were 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 NO_2, $PM_{2.5}$, and O_3 exposures, no evidence for effect modification was seen with PM_{10} 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, Texas, Wendt *et al.* found that short-term

changes in NO_2, O_3, 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 O_3 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 in their bedrooms, schools, and on the playground. To measure indoor exposures, Dutmer *et al.* installed monitoring devices within the homes of inner-city participants and found that that NO_2 levels in the bedroom were at levels similar to those found curbside.[28] Past and recent studies demonstrate that indoor exposures at home and in school to AAP 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 also 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 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.[31–33]

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 five 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, Massachusetts, 54% of which included Hispanic mothers and 30% African American. Cases were determined by physician diagnosis by age six. 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 six. 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 United States and found that schools with >50% African American students were 18% more likely to attend a school 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 5,000 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. In the SAGE II and GALA II studies of African American, Mexican American, and other Latino children in the San Francisco Bay Area, 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.[42–47] 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-occur with high exposure to AAP. In Canada, where income equity is significantly better than in the United States, Pinault *et al.* were still able to demonstrate that exposure to NO_2 is inversely associated with socioeconomic status (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 have identified associations between parental stress and asthma as well as synergism between AAP and neighborhood violence on asthma risk.[52,53] In the last five 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 post-natal 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 (NO_3^-), 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 two and asthma by age six.[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 O_3 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 the likelihood that factors outside of heritability play a crucial role in disease pathogenesis. On the other hand, African American children still had higher rates of asthma than white children from the same neighborhood, which signifies 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 demonstrate 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 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] This may be the result of examining the association of global genetic ancestry estimates with AAP, wherein local ancestry estimates may play a more important role.

GxE studies postulate that response and potential development of disease as a result of one’s environmental exposures is 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 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 suggesting that environmental factors may play a key role during this time period. However, significant differences in asthma risk, medication use, and hospitalization between 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, the findings, at the very least, suggest 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-hour time points over 6 months in 163 Dominican and African American children from New York City. Buccal cells and FeNO were also collected and used as a proxy 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 United Kingdom offered four primary mechanisms in the pathway between AAP and asthma 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 children of color in the United States has led us to the following conclusions:

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

2) Variation in criteria air pollutant selection

Although multiple studies stratify by race and at large sample sizes, findings are mixed and inconsistent with regards to asthma development, risk of exacerbation, and lung function (Table 1) in study populations that were primarily children of color. The most widely studied and most consistent criteria pollutant is NO_2. In the seven studies we identified, none were inconclusive; six 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 $PM_{2.5,}$ 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 trans-placental AAP exposure and maternal stress.[44,48,49,54,55] Of these studies, three focused solely on AAP exposure, and included exposure to prenatal $PM_{2.5}$ and nitrates. Each of these studies also noted a higher risk of poor respiratory outcomes in boys than 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.[70,71] In addition, other prenatal exposure metrics should be considered such as exhaled FeNO, which could provide more direct indication of maternal respiratory inflammation. 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.[72,67,73] Future studies should attempt to quantify an association between prenatal paternal 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.[74–76]

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, starting in the 1930s, forced people of color into less-desirable neighborhoods by refusing to offer loans to investors seeking financial assistance in neighborhoods of color or by refusing to offer loans to people of color.[77] Furthermore, this process was enabled by the federal government through the Home Owner's Loan Corporation, created in 1935 by the New Deal and tasked with drawing maps of cities divided by neighborhood desirability, which largely depended on the presence of immigrants or people of color.[77] While certain ancestral genetic factors may offer minor contributions to increased asthma risk in children of color, research that focus solely on ancestral genetic factors risk appearing myopic; the divide in asthma burden that currently exists between children of color and white children likely exist due to disparities in access to resources, psychosocial stressors, and AAP exposures and are inextricably linked to historical practices and ongoing structural discrimination. (Mention income/wealth inequality across races in US) 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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REFERENCES

1. Institute for Health Metrics and Evaluation. GBD Compare | IHME Viz Hub [Internet]. 2018 [cited 2018 Mar 7]. Available from: https://vizhub.healthdata.org/gbd-compare/
2. WHO | Exposure to ambient air pollution. WHO [Internet]. World Health Organization; 2016 [cited 2017 Oct 31]; Available from: http://www.who.int/gho/phe/outdoor_air_pollution/exposure/en/
3. Hao Y, Flowers H, Monti MM, Qualters JR. U.S. census unit population exposures to ambient air pollutants. Int J Health Geogr [Internet]. BioMed Central; 2012 [cited 2017 Oct 30];11:3 Available from: http://www.ncbi.nlm.nih.gov/pubmed/22239864
4. D'Amato G, Vitale C, Lanza M, Molino A, D'Amato M. Climate change, air pollution, and allergic respiratory diseases. Curr Opin Allergy Clin Immunol [Internet]. 2016 [cited 2017 Oct 31];16:434–40. Available from: http://www.ncbi.nlm.nih.gov/pubmed/27518837
5. Guarnieri M, Balmes JR. Outdoor air pollution and asthma. Lancet [Internet]. 2014 [cited 2017 Oct 31];383:1581–92. Available from: http://linkinghub.elsevier.com/retrieve/pii/S0140673614606176
6. Ierodiakonou D, Zanobetti A, Coull B, Gold D. Ambient air pollution, lung function, and airway responsiveness in asthmatic children. J Allergy Clin Immunol [Internet]. Mosby; 2016 [cited 2017 Oct 31];137:390–9. Available from: http://www.sciencedirect.com.ucsf.idm.oclc.org/science/article/pii/S0091674915007691
7. Gowers AM, Cullinan P, Ayres JG, Anderson HR, Strachan DP, Holgate ST, et al. Does outdoor air pollution induce new cases of asthma? Biological plausibility and evidence; a review. Respirology [Internet] 2012 [cited 2017 Oct 31];17:887–98. Available from: http://doi.wiley.com/10.1111/j.1440-1843.2012.02195.x
8. Nurmagambetov T, Kuwahara R, Garbe P. The Economic Burden of Asthma in the United States, 2008–2013. Ann Am Thorac Soc [Internet]. American Thoracic Society; 2018 [cited 2018 Mar 20]; 15:348–56. Available from: http://www.atsjournals.org/doi/10.1513/AnnalsATS.201703-259OC

9. Zahran HS, Bailey CM, Damon SA, Garbe PL, Breysse PN. Vital Signs: Asthma in Children — United States, 2001–2016. MMWR Morb Mortal Wkly Rep [Internet]. 2018 [cited 2018 Mar 7]; 67:149–55. Available from: http://www.cdc.gov/mmwr/volumes/67/wr/mm6705e1.htm?s_cid=mm6705e1_w
10. Nunes C, Pereira AM, Morais-Almeida M. Asthma costs and social impact. Asthma Res Pract [Internet]. BioMed Central; 2017 [cited 2018 Mar 7];3:1 Available from: http://www.ncbi.nlm.nih.gov/pubmed/28078100
11. Zhang Q, Lamichhane R, Diggs LA. Disparities in emergency department visits in American children with asthma: 2006–2010. J Asthma [Internet]. Taylor & Francis; 2017 [cited 2017 Nov 25];54:679–86. Available from: https://www.tandfonline.com/doi/full/10.1080/02770903.2016.1263315
12. Casale TB, Elward K, Pace W, Turner P, Walters R, Schatz M. Characteristics of Asthma Patients Seeking Specialist Care. J Allergy Clin Immunol [Internet]. 2013 [cited 2018 Mar 7];131:AB107 Available from: http://www.jacionline.org/article/S0091-6749(12)03053-9/pdf
13. Evans-Agnew RA. Asthma Disparity Photovoice: The Discourses of Black Adolescent and Public Health Policymakers. Health Promot Pract [Internet]. 2018 [cited 2018 Mar 20];19:213–21. Available from: http://www.ncbi.nlm.nih.gov/pubmed/29161900
14. Kravitz-Wirtz N, Crowder K, Hajat A, Sass V. THE LONG-TERM DYNAMICS OF RACIAL/ETHNIC INEQUALITY IN NEIGHBORHOOD AIR POLLUTION EXPOSURE, 1990–2009. Du Bois Rev Soc Sci Res Race [Internet]. 2016 [cited 2017 Oct 31];13:237–59. Available from: http://www.ncbi.nlm.nih.gov/pubmed/28989341
15. Abel TD, White J. Skewed riskscapes and gentrified inequities: environmental exposure disparities in Seattle, Washington. Am J Public Health [Internet]. American Public Health Association; 2011 [cited 2017 Oct 31];101 Suppl 1:S246–54. Available from: http://ajph.aphapublications.org/doi/10.2105/AJPH.2011.300174
16. Pais J, Crowder K, Downey L. Unequal Trajectories: Racial and Class Differences in Residential Exposure to Industrial Hazard. Soc Forces [Internet]. Oxford University Press; 2014 [cited 2017 Oct 31];92:1189–215. Available from: https://academic.oup.com/sf/article-lookup/doi/10.1093/sf/sot099
17. Clark LP, Millet DB, Marshall JD. Changes in Transportation-Related Air Pollution Exposures by Race-Ethnicity and Socioeconomic Status: Outdoor Nitrogen Dioxide in the United States in 2000 and 2010. Environ Health Perspect [Internet]. 2017 [cited 2017 Oct 31];125:97012 Available from: http://www.ncbi.nlm.nih.gov/pubmed/28930515
18. Gauderman WJ, Urman R, Avol E, Berhane K, McConnell R, Rappaport E, et al. Association of Improved Air Quality with Lung Development in Children. N Engl J Med [Internet]. Massachusetts Medical Society; 2015 [cited 2018 Feb 17];372:905–13. Available from: http://www.nejm.org/doi/10.1056/NEJMoa1414123
19. Gaffin JM, Hauptman M, Petty CR, Sheehan WJ, Lai PS, Wolfson JM, et al. Nitrogen dioxide exposure in school classrooms of inner-city children with asthma. J Allergy Clin Immunol [Internet]. 2017 [cited 2018 Mar 11]; Available from: http://linkinghub.elsevier.com/retrieve/pii/S0091674917315701
20. Padula AM, Balmes JR, Eisen EA, Mann J, Noth EM, Lurmann FW, et al. Ambient polycyclic aromatic hydrocarbons and pulmonary function in children. J Expo Sci Environ Epidemiol [Internet]. NIH Public Access; 2015 [cited 2018 Mar 18];25:295–302. Available from: http://www.ncbi.nlm.nih.gov/pubmed/24938508
21. Neophytou AM, White MJ, Oh SS, Thakur N, Galanter JM, Nishimura KK, et al. Air Pollution and Lung Function in Minority Youth with Asthma in the GALA II (Genes-Environments and Admixture in Latino Americans) and SAGE II (Study of African Americans, Asthma, Genes, and Environments) Studies. Am J Respir Crit Care Med [Internet]. American Thoracic Society; 2016 [cited 2017 Oct 22];193:1271–80. Available from: http://www.ncbi.nlm.nih.gov/pubmed/26734713
22. Nishimura KK, Galanter JM, Roth LA, Oh SS, Thakur N, Nguyen EA, et al. Early-life air pollution and asthma risk in minority children. The GALA II and SAGE II studies. Am J Respir Crit Care Med [Internet]. American Thoracic Society; 2013 [cited 2018 Jan 16];188:309–18. Available from: http://www.ncbi.nlm.nih.gov/pubmed/23750510

23. U.S. Census Bureau. US Census Bureau 2010 Census Interactive Population Map [Internet]. 2010 [cited 2018 Mar 11]. Available from: https://www.census.gov/2010census/popmap/
24. Delfino RJ, Wu J, Tjoa T, Gullesserian SK, Nickerson B, Gillen DL. Asthma Morbidity and Ambient Air Pollution: effect modification by residential traffic-related air pollution. Epidemiology [Internet]. 2014 [cited 2018 Mar 11];25:48–57. Available from: http://www.ncbi.nlm.nih.gov/pubmed/24240657
25. Alhanti BA, Chang HH, Winquist A, Mulholland JA, Darrow LA, Sarnat SE. Ambient air pollution and emergency department visits for asthma: a multi-city assessment of effect modification by age. J Expo Sci Environ Epidemiol [Internet]. Nature Publishing Group; 2016 [cited 2018 Jan 21]; 26:180–8. Available from: http://www.nature.com/articles/jes201557
26. Berhane K, Chang C-C, McConnell R, Gauderman WJ, Avol E, Rapapport E, et al. Association of Changes in Air Quality With Bronchitic Symptoms in Children in California, 1993–2012. JAMA [Internet]. American Medical Association; 2016 [cited 2018 Mar 11];315:1491 Available from: http://jama.jamanetwork.com/article.aspx?doi=10.1001/jama.2016.3444
27. Wendt JK, Symanski E, Stock TH, Chan W, Du XL. Association of short-term increases in ambient air pollution and timing of initial asthma diagnosis among Medicaid-enrolled children in a metropolitan area. Environ Res [Internet]. NIH Public Access; 2014 [cited 2018 Jan 21];131:50–8. Available from: http://www.ncbi.nlm.nih.gov/pubmed/24657516
28. Dutmer CM, Schiltz AM, Faino A, Rabinovitch N, Cho S-H, Chartier RT, et al. Accurate Assessment of Personal Air Pollutant Exposures in Inner-City Asthmatic Children. J Allergy Clin Immunol [Internet]. Elsevier; 2015 [cited 2018 Mar 11];135:AB165 Available from: http://linkinghub.elsevier.com/retrieve/pii/S009167491403259X
29. McConnell R, Islam T, Shankardass K, Jerrett M, Lurmann F, Gilliland F, et al. Childhood incident asthma and traffic-related air pollution at home and school. Environ Health Perspect [Internet]. 2010 [cited 2018 Mar 21];118:1021–6. Available from: http://ehp.niehs.nih.gov/0901232
30. Gaffron P, Niemeier D. School Locations and Traffic Emissions — Environmental (In)Justice Findings Using a New Screening Method. Int J Environ Res Public Health [Internet]. 2015 [cited 2018 Mar 12];12:2009–25. Available from: http://www.ncbi.nlm.nih.gov/pubmed/25679341
31. McConnell R, Berhane K, Gilliland F, London SJ, Islam T, Gauderman WJ, et al. Asthma in exercising children exposed to ozone: a cohort study. Lancet (London, England) [Internet]. 2002 [cited 2018 Mar 21];359:386–91. Available from: http://linkinghub.elsevier.com/retrieve/pii/S0140673602075979
32. Lovinsky-Desir S, Jung KH, Rundle AG, Hoepner LA, Bautista JB, Perera FP, et al. Physical activity, black carbon exposure and airway inflammation in an urban adolescent cohort. Environ Res [Internet]. 2016 [cited 2018 Mar 11];151:756–62. Available from: http://www.ncbi.nlm.nih.gov/pubmed/27694044
33. Peters KO, Williams DAL, Abubaker S, Curtin-Brosnan J, McCormack MC, Peng R, et al. Predictors of polycyclic aromatic hydrocarbon exposure and internal dose in inner city Baltimore children. J Expo Sci Environ Epidemiol [Internet]. NIH Public Access; 2017 [cited 2018 Jan 21]; 27:290–8. Available from: http://www.ncbi.nlm.nih.gov/pubmed/27966668
34. Lakshmanan A, Chiu Y-HM, Coull BA, Just AC, Maxwell SL, Schwartz J, et al. Associations between prenatal traffic-related air pollution exposure and birth weight: Modification by sex and maternal pre-pregnancy body mass index. Environ Res [Internet]. NIH Public Access; 2015 [cited 2018 Mar 11];137:268–77. Available from: http://www.ncbi.nlm.nih.gov/pubmed/25601728
35. Jedrychowski WA, Perera FP, Maugeri U, Mroz E, Klimaszewska-Rembiasz M, Flak E, et al. Effect of prenatal exposure to fine particulate matter on ventilatory lung function of preschool children of non-smoking mothers. Paediatr Perinat Epidemiol [Internet]. Blackwell Publishing Ltd; 2010 [cited 2018 Feb 19];24:492–501. Available from: http://doi.wiley.com/10.1111/j.1365-3016.2010.01136.x
36. Jedrychowski WA, Perera FP, Spengler JD, Mroz E, Stigter L, Flak E, et al. Intrauterine exposure to fine particulate matter as a risk factor for increased susceptibility to acute broncho-pulmonary infections in early childhood. Int J Hyg Environ Health [Internet]. 2013 [cited 2018 Mar 11]; 216:395–401. Available from: http://www.ncbi.nlm.nih.gov/pubmed/23333083
37. Chiu Y-HM, Hsu H-HL, Wilson A, Coull BA, Pendo MP, Baccarelli A, et al. Prenatal particulate air pollution exposure and body composition in urban preschool children: Examining sensitive

windows and sex-specific associations. Environ Res [Internet]. 2017 [cited 2018 Feb 19];158:798–805. Available from: http://www.ncbi.nlm.nih.gov/pubmed/28759881

38. Hsu H-HL, Chiu Y-HM, Coull BA, Kloog I, Schwartz J, Lee A, et al. Prenatal Particulate Air Pollution and Asthma Onset in Urban Children. Identifying Sensitive Windows and Sex Differences. Am J Respir Crit Care Med [Internet]. American Thoracic Society; 2015 [cited 2018 Feb 19];192:1052–9. Available from: http://www.ncbi.nlm.nih.gov/pubmed/26176842
39. Kingsley SL, Eliot MN, Carlson L, Finn J, MacIntosh DL, Suh HH, et al. Proximity of US schools to major roadways: a nationwide assessment. J Expo Sci Environ Epidemiol [Internet]. NIH Public Access; 2014 [cited 2018 Mar 19];24:253–9. Available from: http://www.ncbi.nlm.nih.gov/pubmed/24496217
40. Weaver GM, Gauderman WJ. Traffic-Related Pollutants: Exposure and Health Effects Among Hispanic Children. Am J Epidemiol [Internet]. Oxford University Press; 2018 [cited 2018 Mar 12];187:45–52. Available from: https://academic.oup.com/aje/article/187/1/45/3865653
41. Thakur N, Oh SS, Nguyen EA, Martin M, Roth LA, Galanter J, et al. Socioeconomic status and childhood asthma in urban minority youths. The GALA II and SAGE II studies. Am J Respir Crit Care Med [Internet]. American Thoracic Society; 2013 [cited 2018 Feb 19];188:1202–9. Available from: http://www.ncbi.nlm.nih.gov/pubmed/24050698
42. Exley D, Norman A, Hyland M. Adverse childhood experience and asthma onset: a systematic review. [cited 2018 Feb 19]; Available from: http://ow.ly/KiDUK
43. Thakur N, Barcelo NE, Borrell LN, Singh S, Eng C, Davis A, et al. Perceived Discrimination Associated With Asthma and Related Outcomes in Minority Youth: The GALA II and SAGE II Studies. Chest [Internet]. Elsevier; 2017 [cited 2018 Mar 18];151:804–12. Available from: http://www.ncbi.nlm.nih.gov/pubmed/27916618
44. Lee AG, Chiu Y-HM, Rosa MJ, Cohen S, Coull BA, Wright RO, et al. Association of prenatal and early childhood stress with reduced lung function in 7-year-olds. Ann Allergy, Asthma Immunol [Internet]. 2017 [cited 2018 Mar 12];119:153–9. Available from: http://www.ncbi.nlm.nih.gov/pubmed/28668548
45. Gentile DA, Sossong N, Morphew T, Presto A, Elliott J. Impact of Environmental Factors on Recurrent Asthma Exacerbations among Inner-CIty Schoolchildren from the Pittsburgh Region. J Allergy Clin Immunol [Internet]. Elsevier; 2017 [cited 2018 Mar 12];139:AB5 Available from: http://linkinghub.elsevier.com/retrieve/pii/S0091674916315883
46. Rosas-Salazar C, Han Y-Y, Brehm JM, Forno E, Acosta-Pérez E, Cloutier MM, et al. Gun Violence, African Ancestry, and Asthma. Chest [Internet]. 2016 [cited 2018 Mar 12];149:1436–44. Available from: http://www.ncbi.nlm.nih.gov/pubmed/26905363
47. Brehm JM, Ramratnam SK, Tse SM, Croteau-Chonka DC, Pino-Yanes M, Rosas-Salazar C, et al. Stress and Bronchodilator Response in Children with Asthma. Am J Respir Crit Care Med [Internet]. American Thoracic Society; 2015 [cited 2018 Mar 12];192:47–56. Available from: http://www.ncbi.nlm.nih.gov/pubmed/25918834
48. van de Loo KFE, van Gelder MMHJ, Roukema J, Roeleveld N, Merkus PJFM, Verhaak CM. Prenatal maternal psychological stress and childhood asthma and wheezing: a meta-analysis. Eur Respir J [Internet]. European Respiratory Society; 2016 [cited 2018 Jan 21];47:133–46. Available from: http://www.ncbi.nlm.nih.gov/pubmed/26541526
49. Brunst KJ, Rosa MJ, Jara C, Lipton LR, Lee A, Coull BA, et al. Impact of Maternal Lifetime Interpersonal Trauma on Children's Asthma. Psychosom Med [Internet]. 2017 [cited 2018 Feb 19];79:91–100. Available from: http://www.ncbi.nlm.nih.gov/pubmed/27359172
50. World Bank. GINI index (World Bank estimate) | Data [Internet]. [cited 2018 Mar 12]. Available from: https://data.worldbank.org/indicator/SI.POV.GINI?locations=CA-US
51. Pinault L, Crouse D, Jerrett M, Brauer M, Tjepkema M. Spatial associations between socioeconomic groups and NO2 air pollution exposure within three large Canadian cities. Environ Res [Internet]. 2016 [cited 2018 Mar 12];147:373–82. Available from: http://www.ncbi.nlm.nih.gov/pubmed/26950027
52. Shankardass K, McConnell R, Jerrett M, Milam J, Richardson J, Berhane K. Parental stress increases the effect of traffic-related air pollution on childhood asthma incidence. Proc Natl Acad Sci [Internet]. 2009 [cited 2018 Mar 21];106:12406–11. Available from: http://www.ncbi.nlm.nih.gov/pubmed/19620729

53. Clougherty JE, Levy JI, Kubzansky LD, Ryan PB, Suglia SF, Canner MJ, et al. Synergistic effects of traffic-related air pollution and exposure to violence on urban asthma etiology. Environ Health Perspect [Internet]. National Institute of Environmental Health Science; 2007 [cited 2018 Mar 21]; 115:1140–6. Available from: http://www.ncbi.nlm.nih.gov/pubmed/17687439

54. Bose S, Chiu Y-HM, Hsu H-HL, Di Q, Rosa MJ, Lee A, et al. Prenatal Nitrate Exposure and Childhood Asthma. Influence of Maternal Prenatal Stress and Fetal Sex. Am J Respir Crit Care Med [Internet]. American Thoracic Society; 2017 [cited 2018 Jan 21];196:1396–403. Available from: http://www.atsjournals.org/doi/10.1164/rccm.201702-0421OC

55. Chiu Y-HM, Coull BA, Sternthal MJ, Kloog I, Schwartz J, Cohen S, et al. Effects of prenatal community violence and ambient air pollution on childhood wheeze in an urban population. J Allergy Clin Immunol [Internet]. 2014 [cited 2018 Mar 12];133:713–722.e4 Available from: http://www.ncbi.nlm.nih.gov/pubmed/24200349

56. Kranjac AW, Kimbro RT, Denney JT, Osiecki KM, Moffett BS, Lopez KN. Comprehensive Neighborhood Portraits and Child Asthma Disparities. Matern Child Health J [Internet]. 2017 [cited 2018 Mar 12];21:1552–62. Available from: http://link.springer.com/10.1007/s10995-017-2286-z

57. Gilliland FD. Outdoor air pollution, genetic susceptibility, and asthma management: opportunities for intervention to reduce the burden of asthma. Pediatrics [Internet]. 2009 [cited 2018 Mar 22]; 123 Suppl 3:S168–73. Available from: http://pediatrics.aappublications.org/lookup/doi/10.1542/peds.2008-2233G

58. Jorde LB, Watkins WS, Bamshad MJ, Dixon ME, Ricker CE, Seielstad MT, et al. The distribution of human genetic diversity: a comparison of mitochondrial, autosomal, and Y-chromosome data. Am J Hum Genet [Internet]. 2000 [cited 2018 Mar 22];66:979–88. Available from: http://linkinghub.elsevier.com/retrieve/pii/S0002929707640245

59. Jorde LB, Wooding SP. Genetic variation, classification and “race”. Nat Genet [Internet]. 2004 [cited 2018 Mar 22];36:S28–33. Available from: http://www.nature.com/doifinder/10.1038/ng1435

60. Bamshad M, Wooding SP. Signatures of natural selection in the human genome. Nat Rev Genet [Internet]. 2003 [cited 2018 Mar 22];4:99–111. Available from: http://www.nature.com/doifinder/10.1038/nrg999

61. Pino-Yanes M, Thakur N, Gignoux CR, Galanter JM, Roth LA, Eng C, et al. Genetic ancestry influences asthma susceptibility and lung function among Latinos. J Allergy Clin Immunol [Internet]. 2015 [cited 2018 Jan 16];135:228–35. Available from: http://www.ncbi.nlm.nih.gov/pubmed/25301036

62. Bunyavanich S, Silberg JL, Lasky-Su J, Gillespie NA, Lange NE, Canino G, et al. A twin study of early-childhood asthma in Puerto Ricans. Dewan A, editor. PLoS One [Internet]. 2013 [cited 2018 Mar 12];8:e68473 Available from: http://dx.plos.org/10.1371/journal.pone.0068473

63. Hüls A, Krämer U, Herder C, Fehsel K, Luckhaus C, Stolz S, et al. Genetic susceptibility for air pollution-induced airway inflammation in the SALIA study. Environ Res [Internet]. Academic Press; 2017 [cited 2018 Mar 19];152:43–50. Available from: https://www.sciencedirect.com/science/article/pii/S0013935116307174

64. Lee J-U, Kim JD, Park C-S. Gene-Environment Interactions in Asthma: Genetic and Epigenetic Effects. Yonsei Med J [Internet]. 2015 [cited 2018 Mar 19];56:877 Available from: https://synapse.koreamed.org/DOIx.php?id=10.3349/ymj.2015.56.4.877

65. Lau MYZ, Dharmage SC, Burgess JA, Win AK, Lowe AJ, Lodge C, et al. The interaction between farming/rural environment and TLR2, TLR4, TLR6 and CD14 genetic polymorphisms in relation to early- and late-onset asthma. Sci Rep [Internet]. Nature Publishing Group; 2017 [cited 2018 Mar 19];7:43681 Available from: http://www.nature.com/articles/srep43681

66. Gref A Interaction of genetic and environmental factors in childhood asthma and allergy. Institutet för miljömedicin / Institute of Environmental Medicine; 2017 [cited 2018 Mar 19]; Available from: https://openarchive.ki.se/xmlui/handle/10616/45571

67. Jung KH, Lovinsky-Desir S, Yan B, Torrone D, Lawrence J, Jezioro JR, et al. Effect of personal exposure to black carbon on changes in allergic asthma gene methylation measured 5 days later in urban children: importance of allergic sensitization. Clin Epigenetics [Internet]. BioMed Central; 2017 [cited 2018 Mar 12];9:61 Available from: http://clinicalepigeneticsjournal.biomedcentral.com/articles/10.1186/s13148-017-0361-3

68. Steptoe A, Hamer M, Chida Y. The effects of acute psychological stress on circulating inflammatory factors in humans: A review and meta-analysis. Brain Behav Immun [Internet]. Academic Press; 2007 [cited 2018 Mar 12];21:901–12. Available from: https://www.sciencedirect.com/science/article/pii/S0889159107000839

69. Chen E, Fisher EB, Bacharier LB, Strunk RC. Socioeconomic status, stress, and immune markers in adolescents with asthma. Psychosom Med [Internet]. [cited 2017 Oct 31];65:984–92. Available from: http://www.ncbi.nlm.nih.gov/pubmed/14645776

70. Gleason JA, Bielory L, Fagliano JA. Associations between ozone, PM2.5, and four pollen types on emergency department pediatric asthma events during the warm season in New Jersey: A case-crossover study. Environ Res [Internet]. 2014 [cited 2018 Mar 12];132:421–9. Available from: http://www.ncbi.nlm.nih.gov/pubmed/24858282

71. Nishimura KK, Iwanaga K, Oh SS, Pino-Yanes M, Eng C, Keswani A, et al. Early-life ozone exposure associated with asthma without sensitization in Latino children. J Allergy Clin Immunol [Internet]. Elsevier; 2016 [cited 2018 Jan 16];138:1703–1706.e1 Available from: http://www.ncbi.nlm.nih.gov/pubmed/27423496

72. Jung KH, Torrone D, Lovinsky-Desir S, Perzanowski M, Bautista J, Jezioro JR, et al. Short-term exposure to PM2.5and vanadium and changes in asthma gene DNA methylation and lung function decrements among urban children. Respir Res [Internet]. 2017 [cited 2018 Mar 12];18:63 Available from: http://respiratory-research.biomedcentral.com/articles/10.1186/s12931-017-0550-9

73. Somineni HK, Zhang X, Biagini Myers JM, Kovacic MB, Ulm A, Jurcak N, et al. Ten-eleven translocation 1 (TET1) methylation is associated with childhood asthma and traffic-related air pollution. J Allergy Clin Immunol [Internet]. 2016 [cited 2018 Mar 12];137:797–805.e5 Available from: http://linkinghub.elsevier.com/retrieve/pii/S0091674915015778

74. Dowen RH, Pelizzola M, Schmitz RJ, Lister R, Dowen JM, Nery JR, et al. Widespread dynamic DNA methylation in response to biotic stress. Proc Natl Acad Sci U S A [Internet]. National Academy of Sciences; 2012 [cited 2018 Mar 13];109:E2183–91. Available from: http://www.ncbi.nlm.nih.gov/pubmed/22733782

75. Meaney MJ, Szyf M, Meaney M. Environmental programming of stress responses through DNA methylation: life at the interface between a dynamic environment and a fixed genome. Dialogues Clin Neurosci [Internet]. 2005 [cited 2018 Mar 13];7:103–23. Available from: www.dialogues-cns.org

76. Essex MJ, Thomas Boyce W, Hertzman C, Lam LL, Armstrong JM, Neumann SMA, et al. Epigenetic Vestiges of Early Developmental Adversity: Childhood Stress Exposure and DNA Methylation in Adolescence. Child Dev [Internet]. Blackwell Publishing Ltd; 2013 [cited 2018 Mar 13];84:58–75. Available from: http://doi.wiley.com/10.1111/j.1467-8624.2011.01641.x

77. Nelson RK, Winling L, Marciano R, Connolly N, Ayers EL. Mapping Inequality [Internet]. Am. Panor. [cited 2018 Mar 13]. Available from: https://dsl.richmond.edu/panorama/redlining/#loc=14/37.7791/−122.4259&opacity=0.96&city=san-francisco-ca&sort=20&area=D3&text=intro

78. Khreis H, Nieuwenhuijsen M. Traffic-Related Air Pollution and Childhood Asthma: Recent Advances and Remaining Gaps in the Exposure Assessment Methods. Int J Environ Res Public Health [Internet]. 2017 [cited 2018 Mar 11];14:312 Available from: http://www.ncbi.nlm.nih.gov/pubmed/28304360

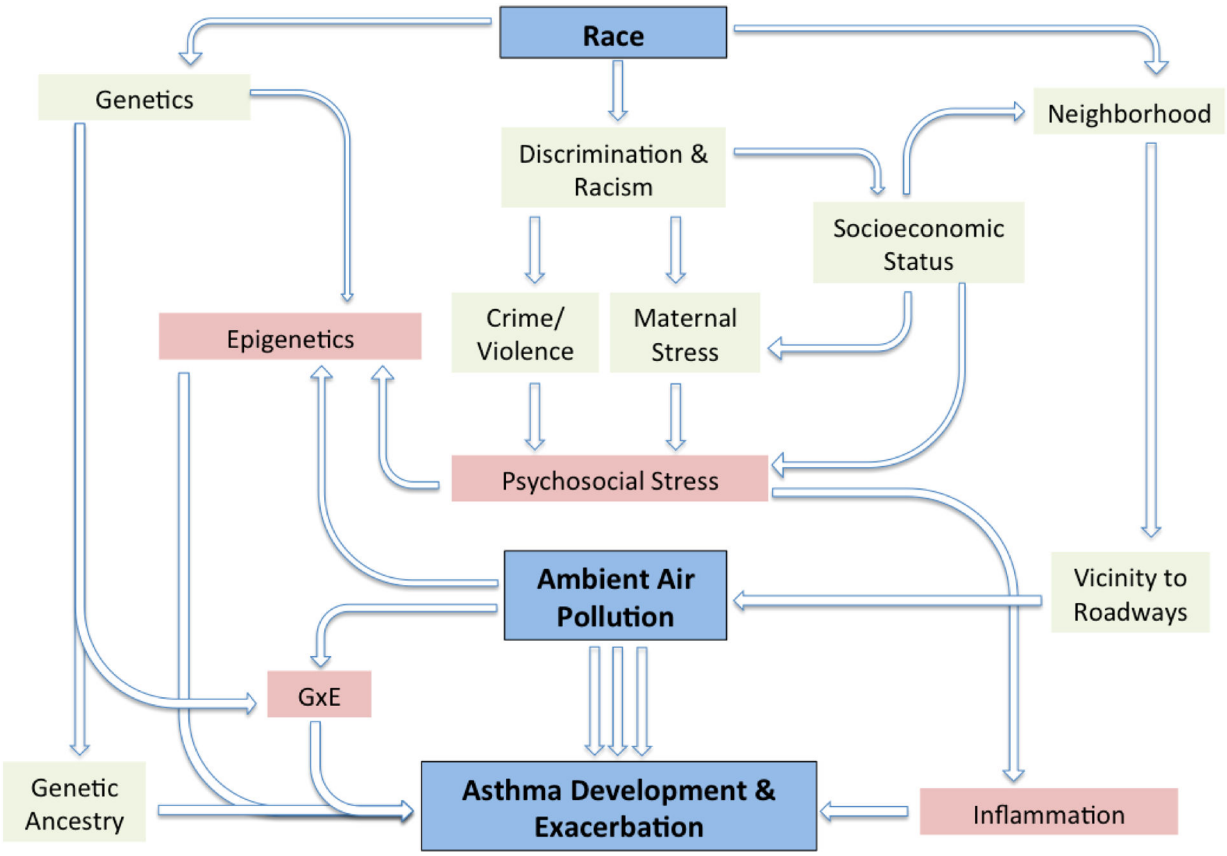

Figure 1.
depicts the relationships and pathways between race, AAP, and asthma as suggested by our recent literature search.

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	PM2.5	PM10	NO2	O3
Neophytou A [21]	26.4% African American 73.6% Latino	FEV1	Lifetime average	5 μg/m3 increase associated with 7.7% decrease in FEV1	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 three 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 [78]	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/m3 reduction	Decreased odds with 5.8 μg/m3 reduction	Decreased odds with 4.9 ppb reduction	Decreased odds with 0.66 μg/m3 reduction
Hsu HH [38]	29.6% African American 53.7% Hispanic 16.7% Other/white	Asthma diagnosis in child	Weekly PM2.5 concentration during pregnancy	Increased levels during weeks 16–25 associated with asthma	-	-	-