# Climate, environment, neighborhood, & health: Pediatric asthma in Kansas City

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Climate, environment, neighborhood, & health:
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# Abstract

Public health is affected by social, environmental, and climatic factors, all of which vary through both space and time. Understanding the interactions between these factors and how they manifest in terms of public health vulnerabilities is key to reducing inequality and promoting environmental justice. Pediatric asthma is the most common chronic condition of childhood in developed nations and in the United States disproportionately affects minority and low-income children. We examine both the spatial and the temporal variation of acute pediatric asthma rates in the Kansas City Metro Area and propose guidelines for improving environmental health research. Our research shows that increasing the amount of greenspace in high-pollution neighborhoods may have a protective effect against acute pediatric asthma, particularly in neighborhoods of color. Additionally, we find that the effects of changing meteorological conditions are minor in comparison to the differences in rates between majority White and majority non-White neighborhoods. Based on these findings we recommend that environmental health research be informed by the communities of interest to ensure that the results of studies are interpretable, impactful, and actionable.

# Acknowledgements

For those I love, and who love me.

For the life I've lived, and the life I've yet to see.

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# Chapter 1

# Introduction

More than half of the world's population lives in urban areas with this number expected to increase to more than two-thirds by 2050 (United Nations, 2019). Within urban areas there are large disparities in public health outcomes and the burden of ill-health is borne disproportionately by those in socially disadvantaged groups (Rydin et al., 2012). Urban systems are coupled human-natural systems that emerge from the interactions between three covarying sectors: 1) the climate system comprising the measurable characteristics of the surface-energy balance with regional and local variability and the corresponding feedbacks; 2) the environmental system comprising qualities of the physical environment including land cover, fractional vegetation, zoning and land use, building characteristics, and transportation networks; and 3) the social system comprising the intersecting characteristics of individuals, neighborhoods, and communities like poverty, health-care coverage, and the socially-defined concepts of race and ethnicity. The interactions between these sectors influence the spatial variation of vulnerability, therefore advancing health equity and environmental justice in the face of global warming requires an understanding of the causal linkages in urban systems (Ferraro et al., 2019).

Disparities in public health outcomes are partially driven by the spatial and temporal variability of the interactions between climatic, environmental, and social systems in urban areas. These systems are highly interdependent and interact in complex ways that produce spatially heterogeneous landscapes of environmental exposure and vulnerability. Environmental health pathways are a conceptualization of risk, moving from source, to emissions, to environmental concentrations, to exposure, to health outcome (Mock et al., 2017). Many negative exposure-related health outcomes are identified, however, in order to effectively reduce risk the factors that

influence concentrations and exposure need to be better understood. These factors are complex and include environmental and social contexts and the ways these contexts are affected by climate variability. Climatic and environmental processes, as well as their interactions and feedbacks, manifest at many different spatial and temporal scales and unevenly across social gradients (Liu et al., 2007b; McPhearson et al., 2016; Pickett et al., 2001; Costanza et al., 1993; Wikle, 2003).

## 1.1 Research questions and objectives

The purpose of this dissertation research is to investigate the spatial and temporal variability of public health as a manifestation of the complex interactions between climatic, social, and environmental systems. As a case study, we employ long-term acute pediatric asthma observations to explore the structure of environmental health the Kansas City Metro Area. By using simple models and emphasizing Bayesian methods we aim to generate knowledge that is interpretable, impactful, and actionable for public health policy makers.

Specifically, to investigate the interactions between environmental and climatic variables and their effects on health and to diagnose racial disparities in asthma incidence we ask the following research questions:

- How is the spatial variability of acute pediatric asthma related to $PM_{2.5}$ concentrations and the amount of vegetated cover?

- Is there evidence of socioeconomic disparities in acute asthma incidence? If so, how do environmental and climatic effects vary along these axes of difference?

- How is the temporal variability of acute pediatric asthma related to meteorologic conditions?

- Are racial disparities evident in the manifestation of these temporal effects?

## 1.2 Organization of the dissertation

The spatial and temporal variation of acute pediatric asthma is explored in two studies. Chapter 2 explores the neighborhood-level spatial variability of asthma rates and investigates the effects of vegetation and air pollution exposure on asthma incidence. Additionally, Chapter 2 examines how these effects differ by socioeconomic characteristics of neighborhoods. Chapter 3 explores the temporal variability of asthma incidence and investigates the effects of meteorological conditions on daily asthma hospitalizations. Additionally, chapter 3 examines whether the temporal effects of environmental exposure on asthma exhibit racial disparities.

An additional chapter discusses the translation gap between research and policy. Chapter 3.5 provides opinionated recommendations for environmental health research to ensure that knowledge produced is interpretable, impactful, and actionable. Lastly, Chapter 4 provides a summary of the dissertation. Each chapter is formatted as a journal article complete with introduction, methods, results, discussion, and conclusions.

# Chapter 2

# Neighborhood effects on acute pediatric asthma

## Abstract

Urbanization produces spatially-variable landscapes where climatic, environmental, and social systems interact in complex ways that affect public health. Environmental exposure along with the associated health risks are unevenly distributed and communities of color are often disproportionately affected by poor health outcomes. Urban policy shapes urban form, and researchers should generate knowledge to support structural interventions for improving health. In this case study, we analyzed the spatial variability of acute pediatric asthma rates across the Kansas City Metro Area and investigated the impact of environmental factors. Using a Bayesian modelling framework, we found that increased levels of $PM_{2.5}$ were associated with increased asthma incidence and that increased vegetative cover helped to mitigate these effects in high pollution neighborhoods. The magnitude of the protective effect of vegetative cover was stronger in neighborhoods with a high proportion of non-white residents, indicating that investing in greenspace infrastructure would provide more benefit in neighborhoods of color. We also found significant racial disparities in asthma outcomes, even after accounting for neighborhood economic position. A first step towards improving health equity will be to focus on environmental justice.

## 2.1 Introduction

More than half of the global population lives in urban areas with this proportion expected to increase to two-thirds by 2050 (United Nations, 2019). As the population of an urban area

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increases so does the land area it occupies, a process known as urbanization (United Nations, 2019).

Urbanization is accompanied by a suite of surface modifications that affect the surface-energy balance, hydrological flows, and the abundance of vegetation (Pickett et al., 2001) thereby creating distinct and varied microclimates (Pickett et al., 2001). Urbanization produces a spatially-variable physical environmental with uneven landscapes of environmental externalities and benefits, and uneven access to urban infrastructure. These spatial variations in the urban environment in turn affect and are affected by community demographics, frequently with distinct inequalities across socioeconomic lines (Heynen et al., 2006; Wolch et al., 2014; Gee & Payne-Sturges, 2004). Systematic inequalities arise from discriminatory zoning practices and civic disinvestment, both of which have long historical legacies (Wolch et al., 2014; Williams et al., 2009; Hoffman et al., 2020; Gee & Payne-Sturges, 2004).

These three subsystems—climatic, environmental, and social—are highly interdependent and interact in complex ways that affect the functioning of urban systems (Liu et al., 2007a,b; McPhearson et al., 2016; Pickett et al., 2001; Costanza et al., 1993; Ferraro et al., 2019). Climate change coupled with increasing urban populations is projected to alter both the individual systems and their interactions (Ebi & McGregor, 2008). Understanding how these systems currently interact is imperative to understanding how to best manage urbanization in order to minimize functional disruption and increase equity (McPhearson et al., 2016). While urbanization is generally seen as a positive force for human development and poverty reduction, unmanaged urbanization has the potential for environmental degradation and inequitable sharing of beneficial outcomes (United Nations, 2019). Research into the ways urban systems affect individuals and society has the potential to improve health outcomes by supporting evidence-based policies.

Public health is defined as the health of entire populations and is best understood as a complex system comprised of multiple agents including citizens, governments, and non-governmental organizations (Trochim et al., 2006). As the world becomes increasingly urban, understanding the dynamics of public health in urban environments and how they are affected by climate, the environment, and society becomes increasingly important (Bai et al., 2018). Knowledge of the

structural and spatial variability of public health across these domains can support better public policies and resource allocation (McPhearson et al., 2016; Jia et al., 2019).

Sustainable cities require health equity (United Nations, 2019; McPhearson et al., 2016), however, large disparities in health outcomes exist across socioeconomic spectra, with minorities and the poor having the worst outcomes (Gee & Payne-Sturges, 2004; Samuels-Kalow & Camargo, 2019; Bailey et al., 2017). These social determinants of health vary spatially along with the physical and environmental determinants of health through residential segregation and structural racism (Gee & Payne-Sturges, 2004; Hoffman et al., 2020; Bailey et al., 2017). Environmental exposure along with the associated health risks are unevenly distributed and communities of color are frequently subject to civic disinvestment resulting in uneven landscapes of vulnerability (Schulz et al., 2016; Bailey et al., 2017). Even after accounting for economic factors, public health risks are often found to be higher in neighborhoods of color (Bullard, 1999). Neighborhoods that are characterized by poverty and a high proportion of minority residents often contain less greenspace and have increased exposure to pollutants, factors which contribute to disparities in environmental health (Jennings et al., 2021; Dai, 2011). Improving the inequitable distribution of healthful environments is a key issue of concern in the environmental justice movement (Bullard, 1999).

Pediatric asthma is associated with a host of social, climatic, and environmental factors. Asthma is a chronic inflammation of the airways characterized by recurrent episodes of wheezing, coughing, shortness of breath, and tightness of chest associated with airflow obstruction (Ford & Mannino, 2010; Won et al., 2016). It is not a disease but rather a diverse disorder that affects both children and adults although with somewhat different presentations (Trivedi & Denton, 2019). Asthma is the most common chronic condition of childhood in developed nations (Milligan et al., 2016). In the United States pediatric asthma is associated with 50 billion dollars in healthcare expenditures annually and causes significant disruptions to both children and parents due to absenteeism (Trivedi & Denton, 2019). Pediatric asthma doubled in the United States between 1980 and 1995 and although overall growth has slowed since then, racial disparities have increased (Akinbami et al., 2016). Minority and low-income children are disproportionately affected in terms

of prevalence rates, morbidity, and mortality (Trivedi & Denton, 2019; Won et al., 2016).

Exposure to higher levels of air pollution has been associated with increased asthma incidence although some studies report conflicting results. In clinical settings air pollution has been shown to affect asthma through oxidative stress which aggravates symptoms like cough and wheeze, decreases lung function, and induces airway inflammation (Delfino et al., 2014). Chang et al. (2019) found that short-term increases in $PM_{2.5}$ increased asthma emergency room admissions and Delamater et al. (2012) found that increasing levels of CO, $NO_2$, and $PM_{2.5}$ had a positive effect on asthma hospitalization rates. On the other hand, in a meta-analysis Buteau et al. (2019) found no evidence for the short-term effects of exposure to air pollution but did find indications of long-term effects. However, the authors note that the diversity of statistical design and exposure metrics precludes definitive conclusions. Additionally, in a review of studies on the impact of environmental factors on pediatric asthma, Pollock et al. (2017) found that the spatial variation of air pollutants has not been adequately investigated and Chen et al. (2007) found that their effects on health can be underestimated if spatial relationships are ignored.

Greenspace can have protective effects against asthma by reducing heat exposure and modifying pollution concentrations. Vegetation lowers temperatures by increasing the latent heat flux and reducing the urban heat island (UHI) effect (Oke, 1982; Wesley & Brunsell, 2019). Increased temperatures facilitate the formation of pollutants like ground-level ozone and have been shown to have a positive association with asthma incidence (Anderson et al., 2013; Kim et al., 2014; Lam et al., 2016; O'Lenick et al., 2017; Soneja et al., 2016). Vegetation can also impact concentrations of air pollutants by removing pollution through uptake, deposition, and dispersion (Nowak et al., 2006; Janhäll, 2015). Alcock et al. (2017) found that increased greenspace had a negative effect on asthma incidence rate. Ayres-Sampaio et al. (2014) found that lack of greenspace was positively associated with increased hospital admissions for asthma while Feng & Astell-Burt (2017) found that increased greenspace was protective against asthma incidence in areas characterized by high traffic volume. Additionally, greenspace is a modifiable factor of the urban environment and potential intervention point for improving public health. Understanding how

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greenspace interacts with air pollution to influence the spatial distribution of asthma can inform urban planners on the policy consequences and benefits of alterations to the urban land surface.

However, social factors remain a key determinant of pediatric asthma prevalence. Brewer et al. (2017) found that accounting for disproportionately higher air pollution levels in predominantly non-white neighborhoods did not attenuate the higher odds of asthma diagnosis among non-white children. Castillo et al. (2021) also found that there was significant racial/ethnic inequity in the distribution of both $PM_{2.5}$ exposure and associated health impacts. However, while identifying the social factors that predict asthma prevalence is helpful in identifying vulnerable neighborhoods, they are not themselves an intervention point. Many studies examine the effects of social factors on asthma in isolation, or the relationships between environmental and atmospheric characteristics and asthma prevalence, but few examine how the these environmental and atmospheric relationships vary across social factors. The social, environmental, and atmospheric systems in urban areas are intertwined through both the surface-energy balance and more than a century of racially-motivated housing policies, civic disinvestment, and growing inequality, and increasing environmental justice and promoting health requires understanding their interactions. In this study we characterize the neighborhood-level factors relating to acute pediatric asthma rates in the Kansas City Metro Area and how these factors vary by neighborhood social and environmental context.

## 2.2   Data and Methods

This study investigates the interactions between climatic, environmental, and social systems in the Kansas City Metro Area and how they affect acute pediatric asthma incidence. Although the distinctions between these systems are fuzzy, it is helpful to define the climate system as encompassing the measurable characteristics of the surface-energy balance including land surface temperature (LST), precipitation and weather, atmospheric gases, and particulate matter. The environmental system comprises qualities of the physical environment including land cover, fractional vegetation, zoning and land use, building characteristics, and transportation networks. The social system we define as the intersecting characteristics of individuals, neighborhoods,

and communities like poverty, health-care coverage, and the socially-defined concepts of race and ethnicity. These climatic, environmental, and social components should be examined simultaneously in relation to each other to improve our understanding of urban systems as a whole rather than the sum of their parts (Pickett et al., 2001).

### 2.2.1 Data

#### 2.2.1.1 Study area

The Kansas City Metropolitan Area is located at 39.0398°N latitude and 94.5949°W longitude and spans two states and six counties: Johnson and Wyandotte Counties in Kansas, and Platte, Clay, Cass, and Jackson Counties in Missouri. The Kansas City Metro Area exhibits characteristic patterns of urban sprawl (Fig. 2.1), which is generally defined as "geographic expansion over large areas, low-density land use, low land-use mix, low connectivity, and heavy reliance on automobiles relative to other modes of travel" (Stone et al., 2010) showing a 55% increase in built area between 1972 and 2001 (Ji, 2008).
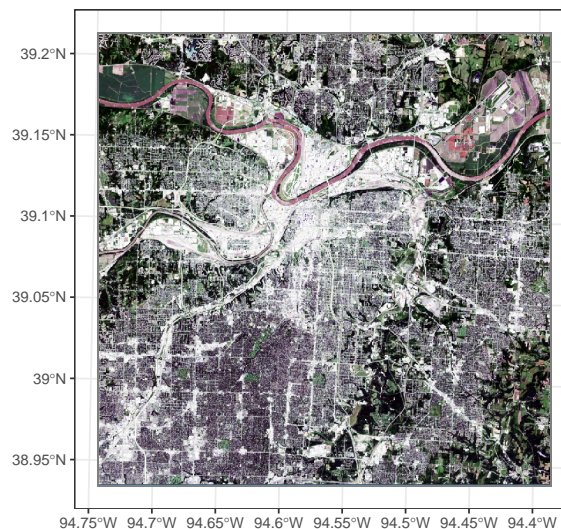


Figure 2.1: The Kansas City metro area exhibits classic patterns of urban sprawl. True-color composite image from Landsat (30 m) June 6, 2011.

Like many American cities, Kansas City has a history of race-based residential housing segregation and redlining (Gotham, 2014). In 2010 the black-white dissimilarity index—a

commonly used measure of community-level segregation—was 62.2, indicating a high degree of black-white residential segregation (Mid-America Regional Council, 2014). For reference, the U.S. Department of Housing and Urban Development (HUD) defines dissimilarity values of 55 and higher as indicating high segregation and values between 40 and 54 as indicating moderate segregation (2020). The overall non-white-white dissimilarity index in 2010 was 48 (Mid-America Regional Council, 2014). Additionally, the city is highly economically segregated and the poverty rate rose from 8.5% to 12.4% between 2000 and 2010 (Mid-America Regional Council, 2014). In 2010 the Urban Institute identified Kansas City as the $5^{th}$ most economically segregated metropolitan area out of 100 analyzed (The Urban Insitute, 2017). Evidence shows that living in racially and economically segregated communities can have detrimental impacts on individual health (The Urban Insitute, 2017).

#### 2.2.1.2 Pediatric asthma

KC Health CORE is a collaborative initiative between Children's Mercy Hospital (CMH) and the Center for Economic Information (CEI) at the University of Missouri, Kansas City created to investigate the geographic disparity of pediatric health outcomes. This analysis uses pediatric asthma data from 2002-2012 geocoded to street centerlines based on the patients' home address at the time of admission. The data comes from a retrospective collection of pediatric asthma encounters within the CMH network and includes residents of Clay and Jackson Counties in Missouri and Cass and Johnson Counties in Kansas. Only children ages 2-18 are considered as asthma is difficult to diagnose in very young children (Kane, 2020; Trivedi & Denton, 2019). The original medical records were formatted according to Table 2.1 (Kane, 2020).

The data were further classified into three severity levels according to the International Classification of Diseases, $9^{th}$ revision diagnoses codes (ICD-9) and the patient class: (1) controlled visit, (2) acute care visit, and (3) hospitalization. The patient class records both the location and the type of treatment received by the patient–e.g. controlled vs. acute care, inpatient vs. outpatient. Level-1 visits are controlled encounters that do not necessarily indicate presentation

Table 2.1: Structure of the original pediatric asthma data records submitted by CMH to UMKC-CEI.

| Category | Attributes |
|---|---|
| Diagnosis | Date of admission |
| | ICD-9 code |
| | Event account number |
| | Patient medical record number (MRN) |
| | Patient residential address |
| Demographics | Birthdate |
| | Sex |
| | Race |
| | Ethnicity |
| Visit characteristics | Payment type |
| | Patient class |

of symptoms, level-2 visits are acute care visits for immediate symptoms at same-day clinics and emergency departments, and level-3 visits are the most severe, requiring hospitalization and/or aggressive treatment. This analysis only considers acute care visits (levels 2 and 3). The CMH network contains the only facilities in the Kansas City metropolitan area that specialize in pediatric care and emergency patients are typically transferred there from other facilities. Thus, the network is likely to capture the majority of acute and severe cases of pediatric asthma. See Kane (2020) for more details on processing of the asthma data. The asthma observations were assigned to census tracts based on the location of the home where the child was living at the time of admission. Data were further aggregated to the total count of acute asthma cases per census tract allowing us to investigate broad spatial trends (Fig. 2.2). One census tract and 11 associated asthma observations were discarded due to missing data, resulting in a sample of 71,381 daily acute-care asthma cases over 11 years and 427 census tracts.

### 2.2.1.3 Environmental data

We characterized the physical environment of each census tract with the percent of vegetated cover estimated from the Moderate Resolution Imaging Spectroradiometer (MODIS) Terra Surface

Reflectance Daily Global 250 m product for the years 2002-2012. Data was acquired from and calculations performed using Google Earth Engine via the R package `rgee` (Aybar, 2021). Most research on the relationship between greenspace and asthma uses the Normalized Difference Vegetative Index (NDVI) as a proxy measure for "greenness", however, while correlated with the amount of vegetation in a pixel NDVI does not represent a physical quantity (Carlson & Ripley, 1997). A better measure is the fractional vegetation (Fr), a quantity derived by imposing physical constraints on NDVI (Gillies & Carlson, 1995). Like NDVI, Fr is easily derived from freely available satellite imagery but has the additional benefits of representing a physical quantity and being less dependent on atmospheric conditions (Carlson & Ripley, 1997).

To calculate Fr, first we calculated the NDVI which is defined as:

$$\text{NDVI} = \frac{\rho_{\text{NIR}} - \rho_{\text{red}}}{\rho_{\text{NIR}} + \rho_{\text{red}}} \tag{2.1}$$

where $\rho_{\text{NIR}}$ and $\rho_{\text{red}}$ are the surface reflectance values in the near-infrared and red bands, respectively. From NDVI we calculated Fr, which is the vegetated proportion of a pixel and is defined as:

$$\text{Fr} = \left( \frac{\text{NDVI} - \text{NDVI}_{\text{soil}}}{\text{NDVI}_{\text{veg}} - \text{NDVI}_{\text{soil}}} \right)^2 \tag{2.2}$$

where $\text{NDVI}_{\text{soil}}$ and $\text{NDVI}_{\text{veg}}$ are the NDVI values corresponding to bare soil and fully vegetated pixels, respectively (Gillies & Carlson, 1995). $\text{NDVI}_{\text{soil}}$ was assigned a value of 0.17 and $\text{NDVI}_{\text{veg}}$ was assigned a value of 0.85 based on the probability density functions of NDVI values from representative bare soil and fully vegetated pixels selected from the study area. Where there were multiple pixels intersecting a census tract, the daily median value was calculated. The NDVI value corresponding to the $99^{th}$ percentile of the time series of each census tract was chosen to represent the proportion of vegetated cover as a static land cover type (Fig. 2.2). Fr from the Landsat 7 8-Day NDVI Composite (30 m) product was also calculated to assess the sensitivity of model results to the resolution of Fr. The choice of MODIS (250 m) or Landsat (30 m) resulted in equivalent

model estimates for the effect of greenspace indicating that either spatial resolution can be used effectively. We chose the MODIS data because of the high temporal resolution, which may benefit subsequent research.

### 2.2.1.4 Climatic data

Daily land-surface temperature (LST) for each census tract was calculated from the MODIS Terra Land Surface Temperature and Emissivity Daily Global 1 km product for the years 2002-2012 using `rgee` and Google Earth Engine. Where there were multiple pixels intersecting a census tract, the daily median value was calculated. We then calculated the mean value for the time series for each census tract (excluding NA values). While LST values averaged over 11 years contain no information about the temporal association between acute asthmatic episodes and temperature, the LST averages reveal an uneven spatial distribution similar to that of asthma and the other risk factors (Fig. 2.2).

We used $PM_{2.5}$ concentration estimates developed by the Center for Air, Climate and Energy Solutions (CACES) using v1 empirical models as described in Kim et al. (2020). These models use land-use regression (LUR) to estimate annual air pollution concentrations at the census-block level by combining data from U.S. Environmental Protection Agency regulatory monitors, land-use characteristics, and satellite-derived estimates of air pollution. These estimates are then upscaled to the census-tract level with a population-weighted average. This dataset is freely available with coverage of the entire United States. We calculated the mean $PM_{2.5}$ concentration per census tract for the years 2002-2012. These calculated means for the census tracts containing the 13 EPA monitors in the study area show good agreement ($r = 0.71$) with the mean observed $PM_{2.5}$ concentrations and exhibit the expected spatial variation (Fig. 2.2). Additionally, because we used the $PM_{2.5}$ concentration estimates to measure intra-urban variation in environmental exposure rather than absolute exposure, we assigned $PM_{2.5}$ quintile ranks to the census-tracts rather than using the mean estimates directly. This has the additional benefit of reducing the correlation between Fr and $PM_{2.5}$. While the LUR does make use of land cover as a variable, covariates used

in the $PM_{2.5}$ modelling were combined using partial least squares (PLS) to reduce dimensionality and the estimated $PM_{2.5}$ concentrations contribute different information than Fr.

### 2.2.1.5 Socioeconomic data

Social data was acquired from the American Community Survey (ACS) 5-year estimates for 2010 (2006-2010) using Social Explorer and the 2010 Decennial Census using the `tidycensus` package for R (Walker & Herman, 2022). Variables include income to poverty ratios from the ACS (under 1.00: doing poorly, 1.00-1.99: struggling, 2.00 and over: doing okay) (Social Explorer, 2022b), the proportion of non-white residents from the 2010 Decennial Census (calculated as (Total Population - Total White Alone) / Total Population), and the population under age 18 per census tract from the 2010 Decennial Census (Social Explorer, 2022a) (Fig. 2.2). We aggregated the income to poverty ratio categories to under 2.00—doing poorly or struggling—and above 2.00—doing okay based on Social Explorer definitions. We opted to use the proportion of non-white residents to characterize neighborhood racial composition because studies repeatedly show that communities of color are disproportionately impacted by environmental exposure and that race-based residential segregation is linked to persistent and unequal health outcomes (Schulz et al., 2016; Bullard, 1999; Schlosberg, 2013).

### 2.2.1.6 IRB Approval

This study was approved by the University of Kansas and the Children's Mercy Hospital Institutional Review Boards (IRB #11120500). Access to the pediatric asthma data was obtained through an approved data use agreement with the Center for Economic Information at the University of Missouri-Kansas City.

### 2.2.1.7 Data processing

All data processing was done in the R software environment for statistical computing version 4.1.2 using the `tidyverse` suite of packages (R Core Team, 2020; Wickham et al., 2019).
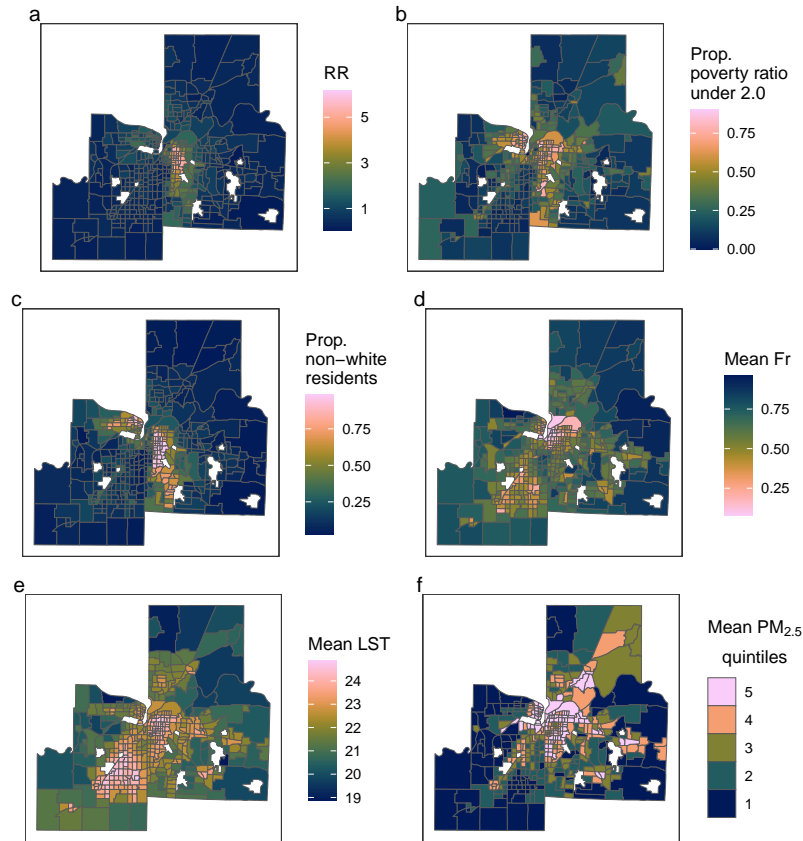
Figure 2.2: Spatial distributions of asthma rates and independent variables. Moving from top left to bottom right the plots show values per census tract: a) the relative risk of acute asthma incidence compared to the study area mean rate, b) the proportion of population living with an income to poverty ratio of below 2.00 indicating doing poorly or struggling, c) the proportion of the population who identifies as non-white, d) the mean fractional vegetation (Fr) indicating the amount of vegetative cover, e) the mean land-surface temperature (LST), f) and quintiles of the mean $PM_{2.5}$ concentration.

## 2.2.2 Analysis methods

### 2.2.2.1 Descriptive analysis

The relationships between variables were first explored through descriptive analysis. Mean values of the independent variables were calculated for quintiles of asthma rates (number of cases per population under 18) to examine how they vary between census tracts with high and low rates. Relationships were also visualized by categorizing each of the independent variables into deciles and then calculating the mean asthma rate for the tracts falling within each bin.

### 2.2.2.2 Statistical modelling

We estimated the effects of $PM_{2.5}$ on acute pediatric asthma using negative binomial regression with Bayesian methods. Additionally, we investigated how greenspace moderates the relationship between air pollution and asthma and how this moderating effect differs by neighborhood social context. We controlled for social characteristics of the census tracts and included the population under 18 as an offset to control for the differing pediatric populations per census tract. All analysis was accomplished using the `brms` package for R (Bürkner, 2017).

The asthma counts were generated by a Poisson process with the probability:

$$P(Y_i = y_i | x_i) = \frac{e^{-\lambda_i} \lambda_i^{y_i}}{y_i!} \qquad (2.3)$$

where $\lambda_i$ is the conditional mean count per census tract, otherwise known as the rate parameter. However, in a Poisson distribution the mean and the variance are assumed to be equal. In the case of the total number of acute care asthma visits per tract the variance far exceeds the mean $(E(Y) = \mu = 176.28, Var(Y) = \sigma^2 = 25478.31)$ indicating that the data is overdispersed, a quality of the distribution which if ignored can result in the underestimation of the uncertainty surrounding the parameters. The negative binomial distribution is a special case of the Poisson distribution which accounts for overdispersion by introducing a dispersion parameter $\phi$ which allows the mean $\mu$ and variance $\sigma^2$ to be estimated separately. As $\phi$ approaches infinity, $\sigma^2$ approaches $\mu$ and the negative binomial distribution resembles the Poisson distribution. Thus the data is represented by:

$$y_i \sim NB(u_i, exp(X_i, \beta), \phi) \qquad (2.4)$$

where $u_i$ is an exposure variable to account for uneven sampling—in this case the pediatric population per census tract, $X_i$ is the independent variables, and $\beta$ the regression coefficients (Gelman, 2021).

Because we are interested in describing the magnitude and direction of effects as well as explicitly accounting for uncertainty in effect estimates, we made use of a Bayesian modelling

framework. Bayesian methods explicitly quantify uncertainty, placing the focus on the presence and strength of effects rather than on null hypothesis significance testing (Kruschke & Liddell, 2018). Uncertainty is associated with the measurement of all variables, not least in regard to the asthma outcome data (Kane, 2022). Additionally, given that pediatric asthma is associated with a number of factors from genetics, to indoor allergens, to atmospheric conditions, the purpose of such an analysis is not to pinpoint exact effects but to understand the structure of relationships in a manner that allows for targeted intervention.

Bayesian methods make explicit use of probability distributions to quantify inferential uncertainty and incorporate expert knowledge through the specification of prior distributions for the estimated parameters (Lynch, 2007). These prior distributions represent the uncertainty of our knowledge about the data-generating process before seeing the data itself. The priors are then conditioned on the observed data, resulting in posterior estimates of the parameter distributions. These posterior distributions represent intervals with a high probability of containing the quantity of interest, which allows probabalistic statements to be made about the modelled effects, an advantage over the frequentist paradigm which interprets confidence intervals only in relation to a (hypothetical and infinite) repeated series of similar trials (Gelman et al., 2014).

The foundation of Bayesian inference is Bayes' theorem:

$$\underbrace{p(\theta|data)}_{\text{posterior}} \propto \underbrace{p(data|\theta)}_{\text{likelihood}} \times \underbrace{p(\theta)}_{\text{prior}} \tag{2.5}$$

which states that the probability of a parameter given the data is proportional to the probability of the data conditioned on the parameter multiplied by the marginal probability for the parameter. In other words, the posterior distribution is the the prior distribution weighted by the observed data.

The total count of acute care pediatric asthma visits between 2002 and 2012 per census tract was modelled with a log-link function to relate the mean count $\mu$ to a linear combination of $k$ variables:

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$$\mu_i = exp(log(exposure) + \alpha + \beta_1 * x_{1i} + \beta_2 * x_{2i} + ... + \beta_k * x_{ki}) \qquad (2.6)$$

where $i$ indexes the census tracts and the exposure is the total population under 18. Incorporating the exposure term accounts for the differing pediatric populations of census tracts and is equivalent to modelling the asthma rate. The independent variables considered were poverty (proportion of the census tract population with an income to poverty ratio of less than 2.0), race (the proportion of non-white residents), Fr, LST, and quintiles of $PM_{2.5}$. LST was found to have no effect in any model and so is not presented in the results.

Priors for the intercept and $\beta$ terms were selected to be weakly informative (Lemoine, 2019) and chosen based on prior predictive checks (simulating outcomes from the priors and comparing them to the observed data) (Gabry et al., 2019). Sensitivity of the posterior estimates to prior specification was also tested by changing the parameters of the priors and evaluating their affect on the posterior. Non-informative, weakly informative, and strongly informative priors all produce equivalent estimates of the parameters indicating that the data is sufficiently strong to dominate posterior estimation (Lemoine, 2019).

Bayesian estimation is accomplished by repeatedly sampling from the joint posterior distribution which in `brms` is accomplished using a Markov chain Monte Carlo (MCMC) algorithm. Effectiveness of the MCMC sampling was assessed visually by inspecting trace plots for chain convergence and numerically with Rhat ($\widehat{R}$) estimates. $\widehat{R}$ represents the amount of between chain variance relative to the within chain variance with a value of 1.0 indicating total convergence. Stable and accurate estimates of parameters depend partially on sample size which was evaluated numerically by the effective sample size (ESS) which estimates the amount of independent information available from the dependent chains as a ratio of the actual sample size by the amount of autocorrelation present in the chains (Kruschke, 2015).

We present the results of four models:

**Model 1**: asthma = poverty ratio + prop. non-white

Table 2.2: Mean values of independent variables in census tracts stratified by quintiles of asthma rate, increasing in prevalence from 1 to 5. Neighborhoods characterized by the highest rates of asthma have higher poverty ratios, higher proportions of non-white residents, lower fractional vegetation (Fr), and higher $PM_{2.5}$ concentrations compared to neighborhoods with lower asthma rates.

|  | Asthma rate (quintiles) | | | | |
| --- | --- | --- | --- | --- | --- |
|  | 1 | 2 | 3 | 4 | 5 |
| Mean proportion poverty-income ratio below 2.0 | 0.12 | 0.18 | 0.29 | 0.42 | 0.57 |
| Mean proportion non-white residents | 0.10 | 0.14 | 0.20 | 0.38 | 0.72 |
| Mean Fr | 0.63 | 0.58 | 0.55 | 0.57 | 0.46 |
| Mean $PM_{2.5}$ | 10.42 | 10.72 | 10.88 | 11.00 | 11.14 |

**Model 2**: asthma = poverty ratio + prop. non-white + $PM_{2.5}$

**Model 3**: asthma = poverty ratio + prop. non-white + $PM_{2.5}$ + fractional vegetation

**Model 4**: asthma = poverty ratio + prop. non-white + $PM_{2.5}$ x fractional vegetation

These models were designed to assess the degree to which asthma rates are effected by social, environmental, and climatic environments and to investigate racial disparities. Models were compared using the widely applicable information criterion (WAIC) and by visually assessing the model fit by simulating asthma counts from the joint posterior density.

## 2.3 Results

Census tracts with the highest asthma rates are characterized by high risk levels for all independent variables (Table 2.2). Moving from the census tracts with the lowest asthma rates (quintile 1) to the highest (quintile 5), the proportion of the population below a poverty-income ratio of 2.0 increases, the proportion of the population who identifies as non-white increases, Fr decreases, and the $PM_{2.5}$ concentration increases. This also is also evident in a visual assessment of bivariate risk. Figure 2.3 shows that asthma rates are highest in census tracts characterized by high poverty and high $PM_{2.5}$, high poverty and a high proportion of non-white residents, high poverty and low Fr, high proportion of non-white residents and low Fr, and high proportion of non-white residents and high
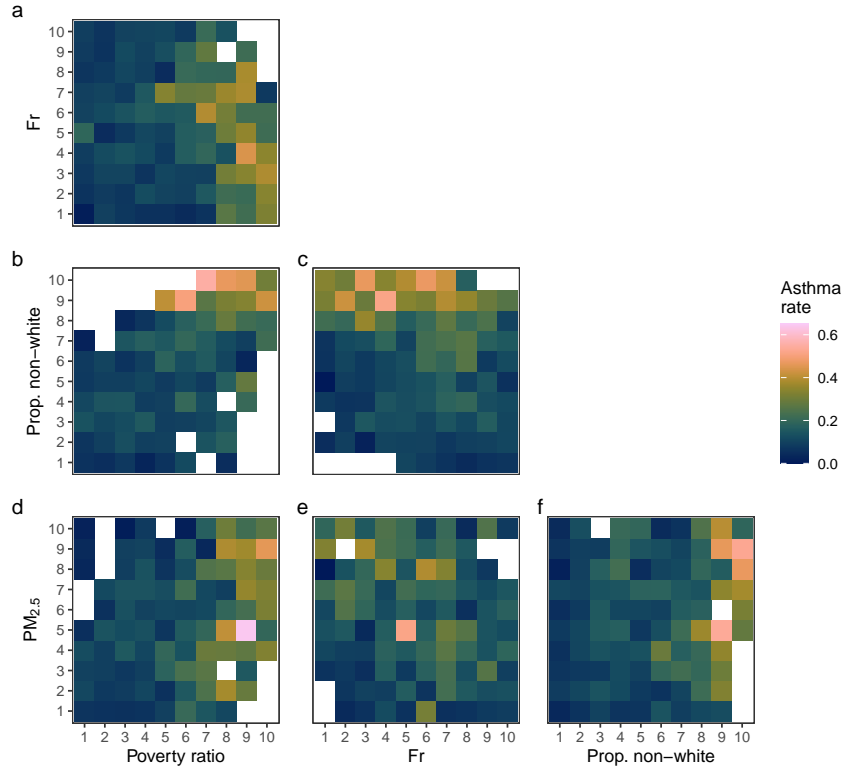
Figure 2.3: Descriptive plots showing bivariate means of asthma rates. Independent variables are divided into deciles and the mean asthma rate calculated for each bin. Asthma rates are highest in (a) neighborhoods characterized by high poverty and low fractional vegetation (Fr), (b) high poverty ratio and high proportion of non-white residents, (c) low Fr and high proportion of non-white residents, (d) high poverty ratio and high $PM_{2.5}$ concentration, (f) and high proportion of non-white residents and high $PM_{2.5}$ concentration.

$PM_{2.5}$ concentrations. This exploratory analysis indicates that census tracts with high asthma rates are characterized by high levels of poverty, high proportions of non-white residents, low Fr, and high $PM_{2.5}$ concentrations.

Table 2.3 presents results from all models as incidence rate ratios (IRRs) and Fig. 2.4 shows the posterior distributions of the model coefficients for Model 4 only. Because the negative binomial regression models the log of the asthma counts, the exponentiated model coefficients correspond to the percent change in asthma count associated with a one unit increase in the independent variable and are known as incidence rate ratios. An IRR of 1 indicates that a change in exposure produces no change in the asthma count, an IRR less than 1 indicates that an increase in exposure decreases

Table 2.3: Exponentiated estimates (IRRs) with 89% credibility intervals. Lower WAIC (widely applicable information criterion) indicates better model fit.

| | Model 1 | Model 2 | Model 3 | Model 4 |
|---|---|---|---|---|
| Intercept | 0.06 | 0.05 | 0.05 | 0.04 |
| | [0.06, 0.07] | [0.05, 0.06] | [0.05, 0.06] | [0.03, 0.06] |
| Prop. poverty ratio under 2.0 | 3.04 | 2.03 | 2.01 | 1.85 |
| | [2.40, 3.90] | [1.58, 2.68] | [1.54, 2.60] | [1.43, 2.44] |
| Prop. non-white | 6.77 | 7.53 | 7.57 | 7.57 |
| | [5.66, 8.20] | [6.20, 9.05] | [6.29, 9.08] | [6.29, 9.10] |
| Fr | | | 0.97 | 1.47 |
| | | | [0.79, 1.17] | [0.94, 2.38] |
| $PM_{2.5}$ Q2 | | 1.22 | 1.22 | 1.12 |
| | | [1.11, 1.36] | [1.10, 1.35] | [0.73, 1.76] |
| $PM_{2.5}$ Q3 | | 1.42 | 1.41 | 2.16 |
| | | [1.28, 1.58] | [1.28, 1.57] | [1.43, 3.20] |
| $PM_{2.5}$ Q4 | | 1.40 | 1.40 | 2.20 |
| | | [1.26, 1.56] | [1.26, 1.57] | [1.44, 3.41] |
| $PM_{2.5}$ Q5 | | 1.45 | 1.44 | 2.24 |
| | | [1.29, 1.63] | [1.28, 1.63] | [1.53, 3.28] |
| Fr x $PM_{2.5}$ Q2 | | | | 1.19 |
| | | | | [0.61, 2.30] |
| Fr x $PM_{2.5}$ Q3 | | | | 0.52 |
| | | | | [0.28, 0.93] |
| Fr x $PM_{2.5}$ Q4 | | | | 0.49 |
| | | | | [0.25, 0.99] |
| Fr x $PM_{2.5}$ Q5 | | | | 0.48 |
| | | | | [0.26, 0.87] |
| Bayes' $R^2$ | 0.807 | 0.818 | 0.818 | 0.817 |
| WAIC | 4567.4 | 4537.4 | 4539.6 | 4538.6 |
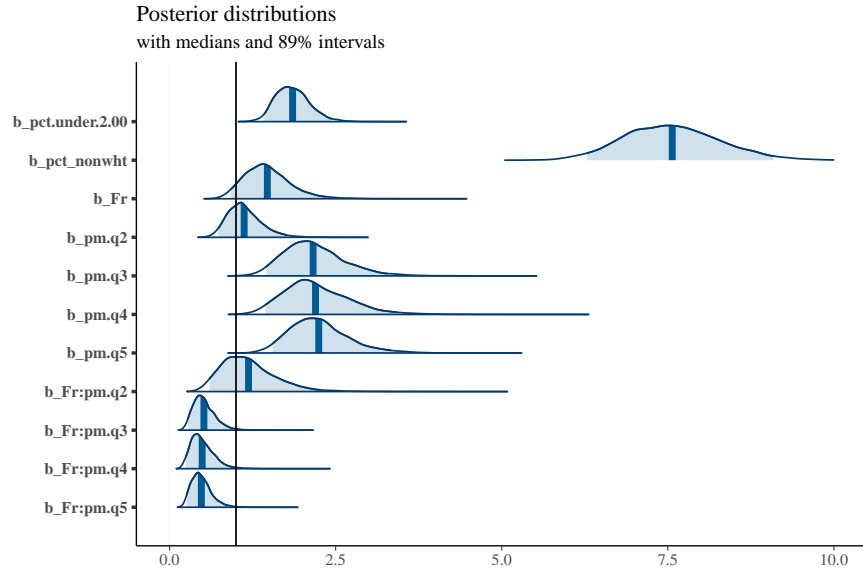
Figure 2.4: Posterior distributions of exponentiated model coefficients for Model 4. Acute asthma incidence per census tract is modelled on the proportion of residents with a poverty to income ratio below 2.00, the proportion of residents who identify as non-white, the fractional vegetation (Fr), quintiles of $PM_{2.5}$, and the interaction between Fr and the quintiles of $PM_{2.5}$.

asthma counts, and an IRR greater than 1 indicates that an increase in exposure increases asthma counts.

Neighborhood social characteristics have strong positive effects on asthma incidence (Model 1). A 1% increase in the proportion of the population with a poverty ratio under 2.0 is associated with a 3.04% increase in the rate of acute asthma while a 1% increase in the proportion of non-white residents is associated with an increase in asthma rates of 6.77%. With the addition of the environmental exposure variables, the effect of poverty ratio on asthma decreases by 1.01%, while the effect of proportion of non-white residents increases by 0.76%, indicating that the burden of environmental exposure is borne more heavily by neighborhoods of color even after controlling for neighborhood economic characteristics.

Models 2-4 additionally consider environmental exposure while controlling for neighborhood social characteristics. There is little certainty that the IRR for LST is different than 1 (model not shown) and so LST is not included in the interaction model. Models 2-4 indicate that higher levels of $PM_{2.5}$ increase asthma incidence, especially above the $40^{th}$ percentile of $PM_{2.5}$

(Q3-Q5). Adding Fr to the model without including an interaction between Fr and $PM_{2.5}$ does not significantly change the model estimates and the IRR for Fr indicates no effect on asthma. However, when including the interaction term, Fr demonstrates negative effects on asthma incidence in census tracts characterized by high levels of $PM_{2.5}$. Examining the conditional effects of both $PM_{2.5}$ and the interaction between $PM_{2.5}$ and Fr show that the magnitude of these effects are stronger in census tracts characterized by high poverty levels and high proportions of non-white residents (Fig. 2.5). As $PM_{2.5}$ concentration increases, Fr has a stronger negative effect on asthma and the magnitude of this relationship is stronger in census tracts characterized by poverty and a high proportion of non-white residents.
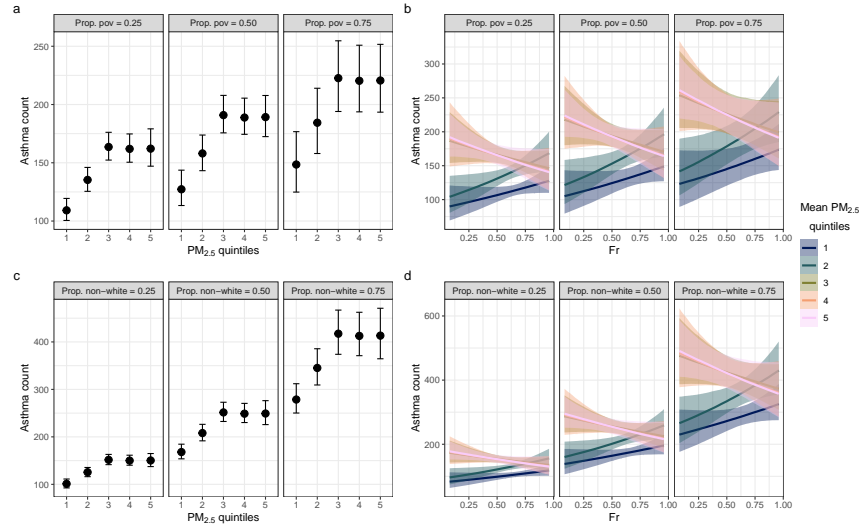


Figure 2.5: Effects of $PM_{2.5}$ quintiles on asthma incidence conditioned on the proportion of residents living in poverty (a) and proportion of non-white residents (c) and effects of the interaction between fractional vegetation (Fr) and $PM_{2.5}$ quintiles on asthma incidence conditioned on the proportion of residents living in poverty (b) and proportion of non-white residents (d). Conditional effects are shown for fixed values (0.25, 0.5, 0.75) of proportion of residents living in poverty (a, b) and proportion of non-white residents (c, d). The effect of $PM_{2.5}$ is stronger in neighborhoods characterized by high poverty rates (a) and a high proportion of non-white residents (c). There appears to be a threshold for $PM_{2.5}$ concentrations where above the $40^{th}$ percentile increased concentrations do not continue to increase asthma rates (a, c). This threshold is also apparent in the interaction effects (b, d). Fr has a negative impact on asthma rates in neighborhoods characterized by $PM_{2.5}$ concentrations above the $40^{th}$ percentile (Q3-Q5). This effect is stronger in neighborhoods characterized by high poverty rates (b) and a high proportion of non-white residents (d).

Model fit was assessed visually with posterior predictive plots (Fig. 2.6). $Y_{rep}$ indicates that the simulated values are replications of the outcome (using the same predictor values that informed the model) rather than predictions (Gabry et al., 2019). While there is some unaccounted for variation, predictions drawn from the joint posterior density of the model approximate the shape of the observed asthma incidence distribution and the medians of these predictions cluster around the median of the observed data. Mapping the means per census tract of the predictions from the MCMC draws also shows that the model adequately captures the spatial variation of the observed asthma counts (Fig. 2.7).
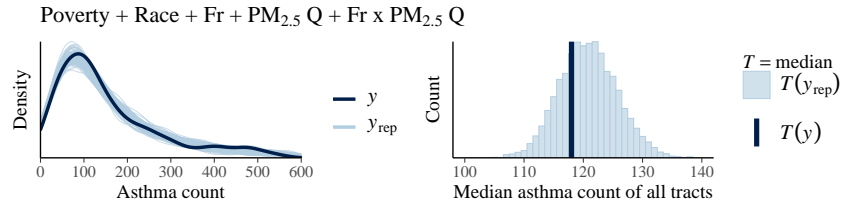


Figure 2.6: Posterior predictive checks. Plots on left show densities of simulated values overlayed on the density of observed values. The x-axis has been truncated to highlight the area of greatest density. Plots on the right show the ability of the model to represent the median (T(y)) of the observed data.
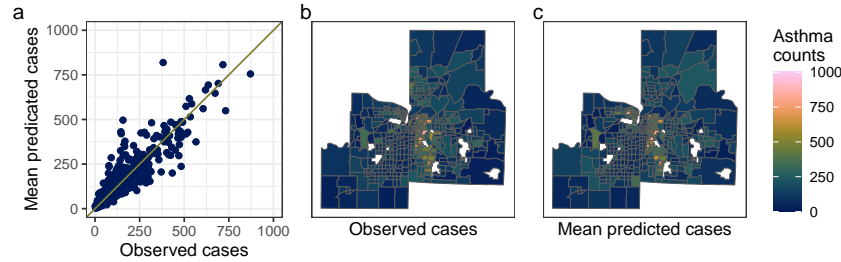


Figure 2.7: Mean acute asthma counts from the posterior replications for each census tract shown with the observed counts. From left to right the plots show: a) the predicted counts against the observed counts, b) the spatial distribution of the observed counts, and c) the spatial distribution of the predicted counts.

## 2.4 Discussion

The goal of this research was to describe the structure of the neighborhood-level variation in acute pediatric asthma rates in the Kansas City Metro Area. Using geolocated health data linked with census, satellite, and air pollution data, we explored the social, environmental, and atmospheric factors influencing asthma incidence. We found that while increased greenspace reduced asthma rates in areas characterized by high pollution levels, the strongest influence on asthma rates was the proportion of non-white residents in a census tract. We also found that average LST was not a significant factor influencing pediatric asthma.

Our research provides support for the positive association between rate of acute asthma incidence and long-term air pollution concentrations. Many studies point to such an association (Delfino et al., 2014; Chang et al., 2019) and while some research indicates contradictory results (Brewer et al., 2017), these may be due to the large variation in data and methods, including asthma outcome (acute vs. affirmative diagnosis), statistical design (count vs. binary outcome), spatial scale (census tract vs. county), and temporal scale (averages over study period, years, months, or weeks). We found that higher levels of $PM_{2.5}$ were associated with an increased incidence rate ratios with an apparent threshold effect above the $40^{th}$ percentile of $PM_{2.5}$ levels. Similar to Alcock et al. (2017) we found that increased Fr was associated with decreased asthma incidence when $PM_{2.5}$ levels were high but had little to no effect when they were low, possibly due to increased particulate deposition in high pollution areas (Janhäll, 2015; Nowak et al., 2006). Street-level geometry can affect deposition by altering turbulence and wind patterns and although we did not consider such fine resolution details, Fr is a key factor in air pollution mitigation (Kumar et al., 2019).

Our results also indicate that the protective effect of vegetation in high pollution areas is stronger in census tracts characterized by higher levels of poverty and higher proportions of non-white residents. Civic investment in public greenspace, especially in neighborhoods of color, can increase environmental justice by providing a range of positive health benefits (Wolch et al., 2014). These results provide evidence for targeted interventions to reduce air pollution by identifying

neighborhoods where greenspace investment will have the greatest positive impact on health.

We found that racial and socioeconomic disparities in acute pediatric asthma rates are significant even after accounting for neighborhood environmental and atmospheric factors. Research indicates that disadvantaged children have elevated exposure to deleterious physical conditions and elevated levels of psychosocial stress that accumulate over time (Williams et al., 2009). Environmental exposures associated with urbanization occur primarily at the community-level, and negative health risks are unequally distributed in cities (Mock et al., 2017; Rydin et al., 2012). Structural racism and residential segregation having been substantial factors in shaping the spatial distribution of environmental health and environmental health disparities in the United States (Bailey et al., 2017) and racial disparities in the prevalence of disease reflect disparities in environmental and social environments (Williams et al., 2009). Our research indicates that the proportion of non-white residents in a census tract was the strongest predictor of acute pediatric asthma in Kansas City, even after controlling for poverty and air pollution. This finding indicates that interventions targeted at reducing the health implications of structural racism are key to controlling the asthma epidemic. Future research should also consider the interaction effects between the proportion of non-white residents, air pollution, and poverty.

## 2.4.1 Limitations

This study relied on data that was averaged both temporally and spatially. Acute asthma incidences and the environmental variables were aggregated to the census-tract level for a so-called ecological analysis. These aggregations represent group exposures that are not readily measured at the individual level, however, this can result in misrepresentation of individual exposure and results from this study are not appropriate to extrapolate to individuals. Regardless, population-level effects—in this case neighborhoods represented by census tracts—are of primary concern to policy makers and an ecological study can provide easy to interpret information on the spatial distribution of environmental health risk. Additionally, we did not assess sensitivity of the results to different spatial scales of aggregation, i.e. zip codes or other municipal tabulation areas, and caution should

be used when interpreting the results at a different scale.

The temporal averaging of the data privileges the spatial variability of environmental health risk but neglects any yearly or seasonal trends in the relationships between acute pediatric asthma, greenspace, and air pollution. Changes to the built environment in established urban areas are generally quite slow and these averages well represent background exposure levels. Any neglected changes within census tracts are likely to be minor compared to the between tract variation in exposure and asthma outcomes.

### 2.4.2 Strengths

A major strength of this analysis is the fine spatial scale of the analysis. By looking at the rates of acute pediatric asthma per census tract we were able to investigate the relationship between asthma incidence and the social, environmental, and atmospheric structure of the neighborhood where a child was living when they were admitted to an emergency facility in the CMH network for acute asthma. The 427 census tracts in the analysis represent a range of neighborhood conditions. Understanding neighborhood-level variation not just in exposure but exposure-related health risks is crucial to creating informed and targeted policies and intervention strategies (Castillo et al., 2021). Additionally, the analysis was conducted using open source software and with freely available (with the exception of the confidential asthma) data. The methods are fairly straightforward and the results easy to interpret. These were conscious choices made to increase the portability of the methods and to provide analysis that is interpretable, impactful, and actionable.

### 2.5 Conclusions

Our findings show that there are significant racial disparities in neighborhood acute pediatric asthma rates in the Kansas City Metro Area. Neighborhoods that are characterized by a high proportion of non-white residents, and to a lesser extent a high proportion of residents living in poverty, exhibit higher rates of asthma incidence. We also found that increased levels of $PM_{2.5}$

were associated with increased asthma incidence but this effect was moderated by the fraction of vegetation (Fr). This interaction was stronger in neighborhoods with a higher proportion of non-white residents, indicating that increasing the amount of greenspace in neighborhoods of color would be protective against the deleterious effects of air pollution on pediatric asthma. Our research provides a clear mandate for planners and policy makers when considering the environmental health impacts of development and public policy: prioritize environmental justice to improve health equity.

# Chapter 3

# Temporal variability of acute pediatric asthma

## Abstract

Pediatric asthma is the outcome of interactions between complex systems including social, climatic, and environmental factors. While studies report varying effects of meteorological conditions on daily asthma prevalence, localized studies are necessary for understanding the temporal variation of asthma as it relates to local conditions. Using a Bayesian modelling framework we examined the time course of acute pediatric asthma in the Kansas City Metro Area. We found that contrary to what is most frequently reported in the literature, asthma incidence is positively associated with maximum temperature in the winter and negatively associated with maximum temperature in the summer. We also found that the effects of meteorological conditions on daily asthma incidence were small compared to the difference in rates between predominantly white neighborhoods and neighborhoods of color. While interventions to improve public health should emphasize mitigating extremes of temperature, humidity, and air pollution, they are unlikely to significantly affect the rate of acute asthma incidence. Rather, specific interventions to improve asthma outcomes should focus on addressing the causes of environmental health disparities.

## 3.1   Introduction

Pediatric asthma is major global public health concern. Asthma is the most common chronic disease of childhood in developed nations and in the United States alone is responsible for 50 million dollars in healthcare costs annually and is a significant cause of school absences and loss

of workdays for parents (Milligan et al., 2016; Trivedi & Denton, 2019). While growth in the rate of pediatric asthma has slowed since the mid 1990's, racial and socioeconomic disparities have increased, with minority and low-income children disproportionately affected by both prevalence and severity of symptoms (Akinbami et al., 2016; Trivedi & Denton, 2019; Won et al., 2016). Understanding the factors contributing to the burden of disease can improve equity in asthma outcomes for children.

The Global Initiative for Asthma (GINA) defines asthma as a heterogeneous disease that is characterized by chronic inflammation of the airways characterized by a history of respiratory symptoms, including wheezing, coughing, and shortness of breath (Global Initiative for Asthma, 2022). Asthma has a complex etiology and there are numerous factors, both individual and environmental, that contribute to the development and expression of the disease. Asthma prevalence is the outcome of interactions between complex factors and while the most prominent environmental factors affecting pediatric asthma are related to indoor allergens and tobacco smoke (Milligan et al., 2016; Akinbami et al., 2016), there remains impetus to understand the neighborhood-level factors that contribute to the incidence of acute pediatric asthma. Social, climatic, and environmental variables have all been shown to affect asthma incidence (Ford & Mannino, 2010; Li et al., 2011; Mireku et al., 2009; Raun et al., 2014; Soneja et al., 2016; Trivedi & Denton, 2019; Won et al., 2016) and understanding the influence of the outdoor environment, especially weather and air pollution, on acute pediatric asthma is important for informing urban policy, especially when considering the trajectory of a changing climate.

Weather can affect asthma both directly via inflammation of the airways and drying of the mucosal membrane, and indirectly by influencing concentrations of allergens and air pollutants (D'Amato et al., 2015; Hu et al., 2020). Air pollution aggravates symptoms by increasing oxidative stress, decreasing lung function, and inducing airway inflammation (Delfino et al., 2014). However, contradictory results concerning the influence of weather and air pollution on pediatric asthma are frequently reported. Many studies find increased asthma hospitalization associated with high temperatures during the summer and throughout the year, and with low temperatures

during the winter months (Anderson et al., 2013; Kim et al., 2014; Lam et al., 2016; O'Lenick et al., 2017; Soneja et al., 2016; D'Amato et al., 2015). Other studies report no effects of heat or cold but rather dependence on the magnitude of change from one day to another (Lei et al., 2021). Other reported effects include increased humidity negatively affecting asthma in both the warm and cold seasons (Hu et al., 2020) and positive associations with $PM_{2.5}$ in both warm and cool seasons (Delfino et al., 2014; Chang et al., 2019). The literature concerning climatic and weather-related effects on pediatric asthma covers a wide array of geographical contexts, climate regimes, social structures, and uses a variety of statistical techniques. Combined with the physiological and etiological complexity of asthma presentation and incidence rates it is unsurprising that research shows mixed effects.

To ensure relevant and actionable results, it is incumbent upon municipalities to base strategies for improving asthma outcomes on localized research. To this end we partnered with Children's Mercy Hospital to examine the relationships between acute pediatric asthma in the Kansas City Metro Area and meteorological conditions. We investigated not only the time course of acute asthma, weather, and air pollution, but also how these relationships were modified by both season of year and neighborhood social context. In this study we model the temporal association between acute pediatric asthma incidence for census tracts in the Kansas City Metro Area and daily atmospheric conditions. Additionally, we stratify the analysis by both season and the proportion of non-white residents to examine if these weather-related effects vary by time of year and to investigate if these effects contribute to racial disparities in asthma prevalence.

## 3.2 Data and Methods

### 3.2.1 Data

#### 3.2.1.1 Study area

The Kansas City Metropolitan Area is located at 39.0398°N latitude and 94.5949°W longitude and spans two states and six counties: Johnson and Wyandotte Counties in Kansas, and Platte,

Clay, Cass, and Jackson Counties in Missouri. The region has a Köppen climate classification of humid sub-tropical (Cfa) and is characterized by hot humid summers and cold winters, year-round precipitation, and prevailing southerly winds. The Kansas City Metro Area is characterized by low-density land use and low connectivity (Fig. 3.1), requiring heavy reliance on automotive traffic, patterns commonly referred to as urban sprawl (Stone et al., 2010). Although annual mean levels of $PM_{2.5}$ decreased between 2001 and 2012 in Kansas City, the region increased from the $47^{th}$ percentile in 2001 to the $73^{rd}$ percentile in 2012 of annual mean $PM_{2.5}$ for the 200 Core-Based Statistical Areas with levels reported by the U.S. Environmental Protection Agency (EPA) (www.epa.gov/air-trends/air-quality-cities-and-counties).
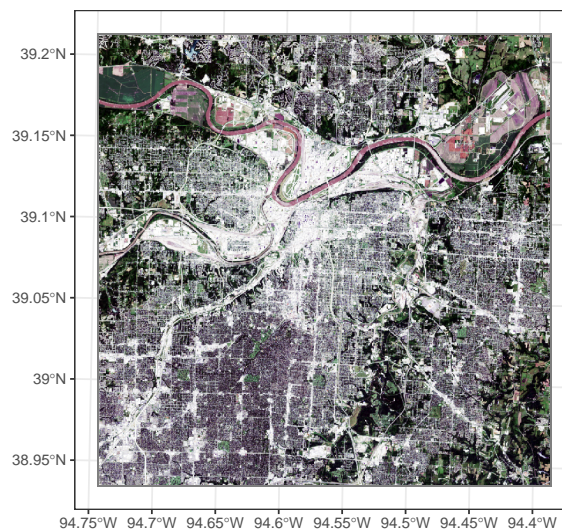


Figure 3.1: The Kansas City metro area exhibits classic patterns of urban sprawl. True-color composite image from Landsat (30 m) June 6, 2011.

#### 3.2.1.2 Pediatric asthma data

Long-term daily observations of acute pediatric asthma from the Children's Mercy Hospital (CMH) network in Kansas City offer a unique opportunity to investigate the effects of environmental exposure on health. The CMH network contains the only facilities in the Kansas City Metro Area that specialize in pediatric care and emergency patients are typically transferred there from other facilities. Thus, the dataset is likely to represent the majority of acute asthma incidents in the area.

The data comprise acute care visits for immediate symptoms at same-day clinics and emergency departments, and visits requiring hospitalization and/or aggressive treatment. Acute care visits are more likely to show sensitivity to fluctuations in atmospheric conditions than managed care visits. Only children ages 2-18 are considered as asthma is difficult to diagnose in very young children (Kane, 2020; Trivedi & Denton, 2019). The observations are from the years 2001-2012 and are geolocated to the home where the child resided at the time of admission; we aggregated these points to census tracts by location and calculated the daily total of cases per tract. One census tract and 11 associated asthma observations were discarded due to missing data for a total of 427 census tracts containing asthma observations in four counties: Clay and Jackson Counties in Missouri and Cass and Johnson Counties in Kansas. The total cases per day for the entire study area were calculated by summing the cases from each tract per day for a total sample size of 4,383 days over 12 years and 75,626 acute asthma incidents. See Section 2.2.1.2 and Kane (2020) for further details on the pediatric asthma data.

### 3.2.1.3 Environmental exposure data

To investigate the temporal dependence of acute pediatric asthma on environmental exposure, we selected variables with daily observations which have plausible physical pathways to affecting asthma. Variables were also selected to facilitate comparison with similar studies. We calculated daily summary values of environmental exposures for the entire study area: daily maximum temperature (Tmax, °C) was calculated as the maximum daily daytime (between 7 am and 8 pm) temperature; dewpoint depression (DDmed, °C) was calculated by subtracting the dew point temperature from the temperature and then finding the daily daytime median; $PM_{2.5}$ is the daily maximum concentration ($PM_{2.5}$, ppm); and wind speed (WDSP, knots) is the daily daytime maximum wind speed. We used dew point depression to quantify the level of moisture in the air because the measure is less dependent on temperature than relative humidity. The meteorological data come from the Automated Surface Observing System (ASOS) network run by the National Weather Service and the $PM_{2.5}$ data come from an EPA station in central Kansas City that is likely

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to be representative of general conditions in the area.

### 3.2.1.4   Social data

The pediatric population (under 18) per census tract was acquired from the 2000 and 2010 Decennial Censuses using the `tidycensus` package for R; we obtained yearly estimates through linear interpolation. Total pediatric population was calculated by summing the populations of the individual tracts per year. Pediatric population was considered static for the years 2011-2012 due to the 2020 Decennial Census results having not been released at the time of this analysis. The proportion of non-white residents (calculated as (Total Population - Total White Alone) / Total Population) per census tract was acquired from the 2000 and 2010 Decennial Census and then linearly interpolated to yearly estimates. To match the population data, we considered the proportions for the years 2011-2012 to be static.

In our previous research (see Section 2.3) we found that the proportion of non-white residents in a census tract was the dominant influence on the rate of acute asthma incidence in Kansas City. To investigate whether meteorological factors have a stronger impact on communities of color than on predominantly white communities we used quartiles of the proportion of non-white residents to stratify the data into four subpopulations of census tracts. We calculated daily counts of acute asthma for each strata, in addition to the total pediatric population per strata and ran separate models on each to compare effect sizes.

### 3.2.1.5   IRB Approval

This study was approved by the University of Kansas and the Children's Mercy Hospital Institutional Review Boards (IRB #11120500). Access to the pediatric asthma data was obtained through an approved data use agreement with the Center for Economic Information at the University of Missouri–Kansas City.

### 3.2.1.6 Data processing

All data processing was done in the R software environment for statistical computing, version 4.1.2 using the `tidyverse` suite of packages (R Core Team, 2020; Wickham et al., 2019).

## 3.2.2 Analysis methods

### 3.2.2.1 Descriptive analysis

The temporal variation of pediatric asthma and the exposure variables was first explored through descriptive analysis. Mean values of the independent variables were calculated for each season as well as the seasonal rates of asthma incidence for the stratified data. We also visualized the time series of asthma rates and all independent variables to look for common modes of temporal variability.

### 3.2.2.2 Statistical modelling

We estimated the temporal effects of atmospheric variables on acute pediatric asthma using negative binomial regression within a Bayesian framework. Additionally, we investigated how these relationships vary seasonally (spring, summer, fall, winter) and by quartiles of the proportion of non-white residents. We controlled for seasonality and time using a natural cubic spline, weekday effects with an indicator variable, and included the population under 18 as an offset to control for the differing pediatric populations per year. All analysis was accomplished using the `brms` package for R (Bürkner, 2017).

The daily asthma counts were generated by an overdispersed Poisson process where a log-link function and a negative binomial distribution are used to model the expected count on day $Y_i$:

$$E(Y_i) = \mu_i = exp(\beta, X_i + \delta, D_i + f(L_i) + log(u_i)) \sim NB(\mu_i, \phi) \tag{3.1}$$

where the expected count $E(Y_i)$ is equal to the mean $\mu_i$, $\beta$ is a vector of coefficients for the independent variables $X_i$, $\delta$ is a vector of coefficients for an indicator variable $D_i$ for the day

of week, $f(L_i)$ is a smooth function of time modelled with a natural cubic spline with 7 degrees of freedom per year to account for seasonal and long term trends, $u_i$ is an offset term for the yearly pediatric population, and $\phi$ is a dispersion parameter that relaxes the assumption of a Poisson distribution that the variance and the mean are equivalent. The use of a natural cubic spline to control for time effects and the choice of degrees of freedom for the time spline were based on the methods of similar studies (Anderson & Bell, 2009; Anderson et al., 2013; Chang et al., 2019). Additionally, the use of 7 degrees of freedom per year results in 3 knots per year, corresponding to 4 seasons (Perperoglou et al., 2019).

Because we are interested in describing the magnitude and direction of the effects of environmental exposure on asthma rather than provide exact estimates, we performed the analysis within a Bayesian framework. A distinct advantage of Bayesian methods is the estimation of coefficients as probability distributions rather than point estimates which allows probabalistic statements to be made about effects given the specified model and the data (Gelman et al., 2014). Bayesian methods also provide sufficient flexibility to specify the desired models (Kruschke, 2021).

For the unstratified dataset, three models were specified:

**Model 1**: asthma = weekday + ns(time)

**Model 2**: asthma = Tmax + DDmed + $PM_{2.5}$ + WDSP + weekday

**Model 3**: asthma = Tmax + DDmed + $PM_{2.5}$ + WDSP + weekday + ns(time)

Model 1 contains only the effects of time, both in terms of day of week variation and the seasonal and long-term trends in asthma cases and serves as a means of comparison to gauge the information contributed by adding subsequent environmental exposure variables to the model. Model 2 contains all exposure variables but does not control for the seasonal and long-term trends and also serves as a means of comparing goodness of fit. Model 3 is the full model including all environmental exposures and the time effects. To investigate whether the effects of environmental exposure vary seasonally, we ran Model 3 separately for each season (winter, spring, summer, fall). Additionally, we ran Model 3 for each strata of quartiles of the proportion of non-white residents

and for each strata by season. Research indicates that residential racial segregation contributes to differences in environmental exposure and racialized health inequalities (Bailey et al., 2017; Schulz et al., 2016; Bullard, 1999) and our previous research (see Section 2.3) found that the proportion of non-white residents in a neighborhood was the strongest predictor of acute asthma rates. The stratified models allowed us to investigate whether meteorological factors have a stronger impact on communities of color.

Model results are presented as incident rate ratios (IRR) which are the exponentiated model coefficients. The IRRs correspond to the percent change in the predicted asthma count associated with a one unit increase in an independent variable. An IRR of 1 indicates that a change in exposure produces no change in the asthma count, an IRR less than 1 indicates that an increase in exposure decreases asthma counts, and an IRR greater than 1 indicates that an increase in exposure increases asthma counts. Additionally, the widely applicable information criterion (WAIC) was calculated for each model to assess the fit of the models relative to each other for the same data. Lower WAIC values indicate a better model fit.

## 3.3    Results

### 3.3.1    Descriptive results

The daily count data show distinct annual and weekly trends. Figure 3.2 shows the annual variation of the daily asthma counts. Cases are lowest during the summer and highest during the fall when children go back to school. The seasonality of asthma also corresponds to allergen and respiratory infection seasons and is commonly reported in the literature (Won et al., 2016). There is also a distinct weekly trend to the data (Fig. 3.2). Cases are highest on Mondays and decline through the weekend, likely due to behavioral patterns of doctor's visits. Figure 3.3 shows smoothed estimates of the asthma counts and predictor variables by day of year. Tmax, DDmed, and $PM_{2.5}$ peak during the summer when cases are lowest, while WDSP is generally lowest during this time.

Table 3.1 shows the mean environmental exposure values for the study period. We defined

a
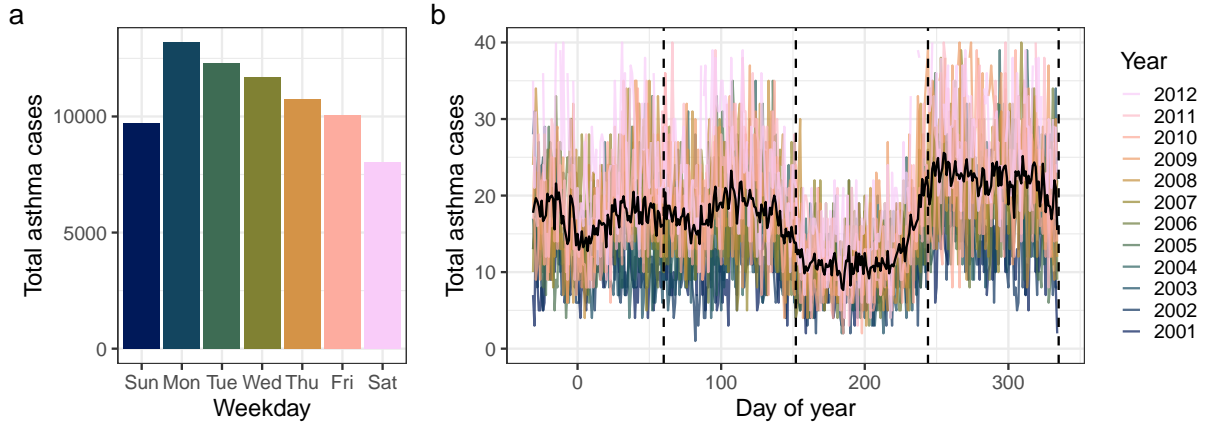


Figure 3.2: Temporal variability of acute pediatric asthma. Counts by day of week are shown on the left (a). Cases are highest on Mondays and decline through the weekend. Seasonality of asthma is shown on the right (b). Dotted lines show the meteorological seasons (winter, spring, summer, fall), the colored lines represent each year of the study, and the black line represents the mean count by day of year.

Table 3.1: Mean values of seasonal environmental exposure variables. Maximum temperature (Tmax) is lowest in the winter and highest in the summer, median dewpoint depression (DDmed) peaks in the spring and summer but is less variable than Tmax, windspeed (WDSP) peaks in the spring but is fairly constant year round, and $PM_{2.5}$ is highest in the summer and lowest in the fall.

|  | Winter | Spring | Summer | Fall |
|---|---|---|---|---|
| Tmax (°C) | 6.15 | 19.76 | 31.24 | 20.11 |
| DDmed (°C) | 4.81 | 6.01 | 5.85 | 4.96 |
| WDSP (knots) | 11.40 | 12.91 | 11.16 | 11.01 |
| $PM_{2.5}$ (ppm) | 13.65 | 13.39 | 15.64 | 12.38 |

the seasons according to the meteorological definition—winter comprises December, January, and February; spring comprises March, April, and May; summer comprises June, July, and August; and fall comprises September, October, and November. Tmax is highest in the summer and lowest in the winter, and while the other environmental exposure variables are fairly constant throughout the year, DDmed is lowest in the winter, WDSP is highest in the spring, and $PM_{2.5}$ is highest during the summer.

Our database of pediatric asthma includes 75,626 daily acute care visits to facilities in the Children's Mercy Hospital Network during the study period from 2001-01-01 to 2012-12-31. The
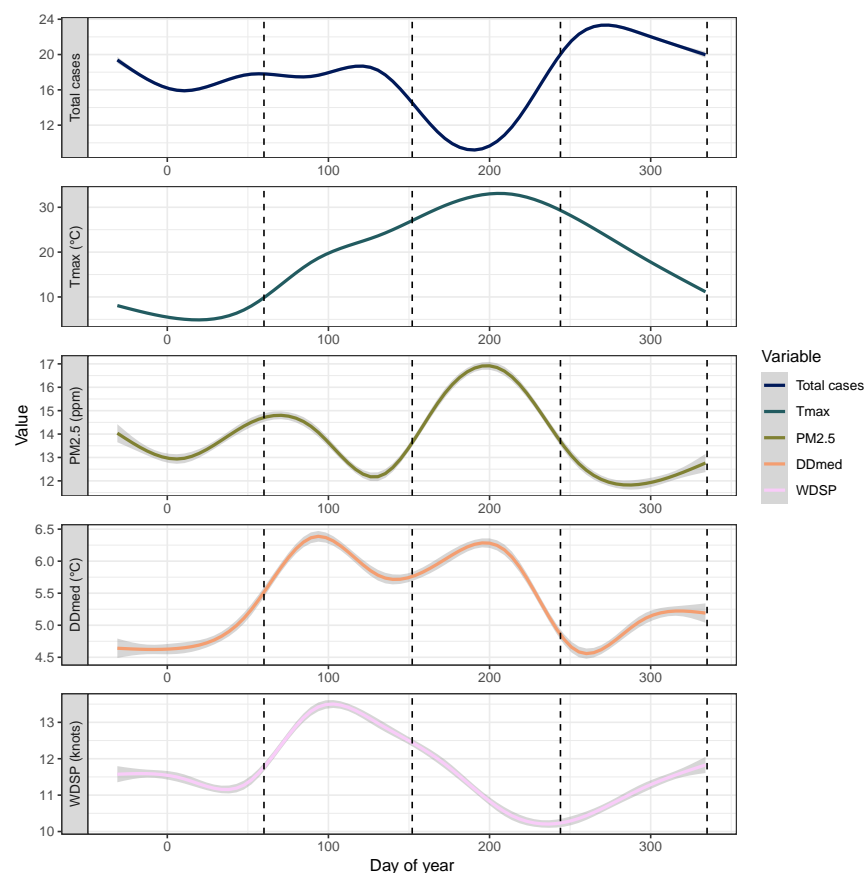
Figure 3.3: Asthma counts and predictors by day of year. Lines are smoothed estimates over the entire study period. Dotted lines show the meteorological seasons starting with winter and ending with fall. From top to bottom the plot shows time series of: total asthma counts, maximum temperature (Tmax), PM$_{2.5}$, median dewpoint depression (DDmed), and windspeed (WDSP).

pediatric population of the 427 census tracts comprising the study area grew from 385,727 in 2001 to 410,332 in 2010 corresponding to an increase in the overall incidence rate of acute pediatric asthma from 0.011 to 0.023. As reported in Chapter 2, the burden of pediatric asthma is not borne evenly by the population but is higher in census tracts with a higher proportion of non-white residents. Rates for all quartiles have increased slightly through time although neighborhoods in the upper quartile of proportion non-white residents have the highest rates overall (Fig. 3.4). Note that the rates for years 2011 and 2012 are not shown. Data from the 2020 Decenniel Census had not been released at the time of writing and so the pediatric population per census tract was assumed constant after 2010.

Table 3.2 shows the rates of acute pediatric asthma by season and quartile of proportion of non-
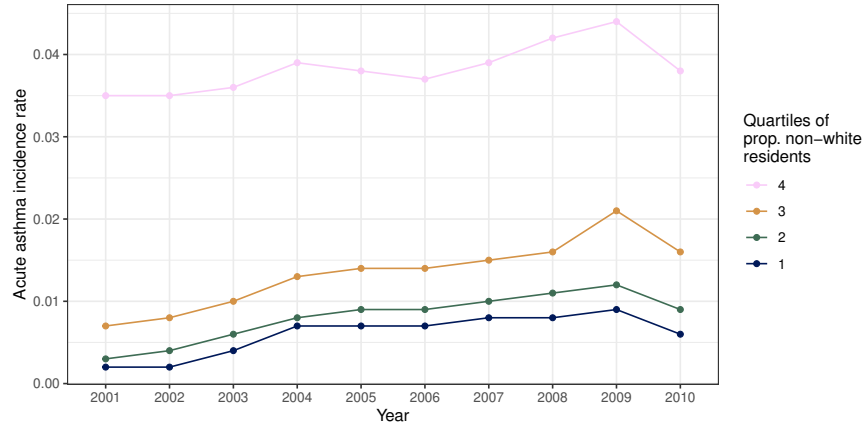
Figure 3.4: Yearly rates of acute pediatric asthma for each census tracts in each quartile of proportion of non-white residents. Rates increase from the lower quartile through the upper quartile and also through time. Note that the years 2011 and 2012 are not shown.

Table 3.2: Seasonal rates of acute pediatric asthma for quartiles of proportion non-white residents. Rates based on census tract pediatric populations in 2010.

| Prop. non-white | Winter | Spring | Summer | Fall |
|---|---|---|---|---|
| 0-25% | 0.02 | 0.02 | 0.01 | 0.02 |
| 25-50% | 0.03 | 0.03 | 0.02 | 0.03 |
| 50-75% | 0.04 | 0.05 | 0.03 | 0.06 |
| 75-100% | 0.12 | 0.13 | 0.09 | 0.16 |
| Total | 0.05 | 0.05 | 0.03 | 0.06 |

white residents. Rates increase in all seasons as the quartile range increases, with census tracts with 50-100% non-white residents showing significantly higher rates and also more variability in rates between the seasons. For these quartiles, rates are highest in the fall and lowest in the summer. These trends are mirrored in the overall rates for the study area.

### 3.3.2 Full models

Table 3.3 shows the associations between acute pediatric asthma and the environmental exposure variables for the full dataset and the entire study period. A comparison of Model 1 and Model 2 shows that accounting for the day of week and the seasonal and long-term temporal trends is necessary for explaining the temporal variability in pediatric asthma incidence. The Bayes

Table 3.3: Exponeniated estimates (IRRs) with 89% credibility intervals for all models and included variables. Model 1 includes only day of week and the time spline (coefficients not shown), Model 2 contains only the environmental exposure variables, and Model 3 contains both temporal and environmental exposure variables. Lower WAIC (widely applicable information criterion) indicates better model fit.

|  | Model 1 | Model 2 | Model 3 |
|---|---|---|---|
| Intercept | 0.000 | 0.000 | 0.000 |
|  | [0.000, 0.000] | [0.000, 0.000] | [0.000, 0.000] |
| Tmax |  | 0.993 | 1.002 |
|  |  | [0.992, 0.994] | [1.000, 1.003] |
| DDmed |  | 1.007 | 1.002 |
|  |  | [1.004, 1.011] | [0.999, 1.004] |
| WDSP |  | 0.997 | 0.998 |
|  |  | [0.994, 0.999] | [0.996, 0.999] |
| $PM_{2.5}$ |  | 0.998 | 1.000 |
|  |  | [0.996, 0.999] | [0.999, 1.002] |
| Num.Obs. | 3892 | 3892 | 3892 |
| Bayes' $R^2$ | 0.616 | 0.143 | 0.612 |
| WAIC | 22968.0 | 25753.0 | 23018.3 |

$R^2$ is much higher for the time-only model and the WAIC much lower when compared to the environmental exposure model that does not account for the temporal trends. While Model 3 includes both the temporal trends and the environmental exposure variables, adding the exposure variables does not improve the model fit and in fact decreases the Bayes $R^2$ and increases the WAIC compared to the time-only model. While an interpretation of the mean IRR for Tmax is that there is an approximately 0.80% increase in acute asthma cases for a 5°C (9°F) increase in maximum temperature, it is important to note that not only is this effect quite small, but also that Model 3 offers no improvement in explanatory power over the time-only model. Additionally, many of the posterior distributions of the IRRs contain 1, indicating that there is unlikely to be an effect. However, the lack of apparent effects is perhaps unsurprising considering that meteorological variables are likely to have different effects during different times of the year as the seasons are characterized by different average conditions.

Table 3.4: Exponeniated estimates (IRRs) with 89% credibility intervals for the seasonal models. Lower WAIC (widely applicable information criterion) indicates better model fit.

|  | Winter | Spring | Summer | Fall |
|---|---|---|---|---|
| Intercept | 0.000 | 0.000 | 0.000 | 0.000 |
|  | [0.000, 0.000] | [0.000, 0.000] | [0.000, 0.000] | [0.000, 0.000] |
| Tmax | 1.008 | 0.997 | 0.985 | 1.001 |
|  | [1.005, 1.011] | [0.994, 1.001] | [0.979, 0.991] | [0.998, 1.004] |
| DDmed | 1.010 | 1.001 | 1.018 | 1.001 |
|  | [1.003, 1.016] | [0.996, 1.005] | [1.011, 1.025] | [0.996, 1.006] |
| WDSP | 0.993 | 1.001 | 1.001 | 1.000 |
|  | [0.988, 0.997] | [0.998, 1.005] | [0.997, 1.005] | [0.997, 1.004] |
| $PM_{2.5}$ | 0.999 | 1.003 | 1.002 | 1.003 |
|  | [0.997, 1.002] | [1.001, 1.006] | [0.999, 1.004] | [1.000, 1.006] |
| Num.Obs. | 909 | 1027 | 1049 | 907 |
| Bayes' $R^2$ | 0.479 | 0.510 | 0.409 | 0.501 |
| WAIC | 5546.6 | 6209.6 | 5832.9 | 5639.8 |

### 3.3.3 Seasonal models

Table 3.4 shows the associations between acute pediatric asthma and the environmental exposure variables for each season (winter, spring, summer, fall). The strongest associations between Tmax and acute asthma incidence are in winter and summer. During the winter a 5°C increase in maximum temperature is associated with a 4.00% increase in asthma incidence and in the summer a 5°C increase corresponds to a 7% decrease in cases. DDmed also shows the strongest associations in winter and summer, although both are positive. An increase of 5°C in the median dewpoint depression (decreased humidity) corresponds to a 5.04% increase in cases during the winter and a 9.17% increase in the summer. WDSP only shows an association in the winter with a 3 knot (approximate step between lower levels of the Beaufort Scale) increase in windspeed corresponding to a 2.23% decrease in cases. $PM_{2.5}$ shows a positive association in the spring and fall, with an increase of 10 ppm corresponding to increases of 3.31% and 3.09% respectively.

Table 3.5: Exponentiated estimates (IRRs) with 89% credibility intervals for the stratified models. Census tracts were stratified by quartiles of the proportion of non-white residents. Lower WAIC (widely applicable information criterion) indicates better model fit.

| | Proportion non-white residents | | | |
| | 0-25% | 25-50% | 50-75% | 75-100% |
|---|---|---|---|---|
| Intercept | 0.000 | 0.000 | 0.000 | 0.000 |
| | [0.000, 0.000] | [0.000, 0.000] | [0.000, 0.000] | [0.000, 0.000] |
| Tmax | 1.000 | 0.999 | 1.000 | 1.003 |
| | [0.996, 1.004] | [0.995, 1.002] | [0.997, 1.003] | [1.001, 1.006] |
| DDmed | 1.008 | 1.000 | 1.002 | 1.001 |
| | [1.001, 1.015] | [0.994, 1.007] | [0.996, 1.007] | [0.997, 1.005] |
| WDSP | 1.002 | 0.999 | 0.995 | 0.997 |
| | [0.997, 1.006] | [0.995, 1.003] | [0.991, 0.999] | [0.995, 1.000] |
| $PM_{2.5}$ | 0.999 | 1.001 | 1.004 | 0.999 |
| | [0.995, 1.002] | [0.999, 1.004] | [1.001, 1.006] | [0.998, 1.001] |
| Num.Obs. | 3892 | 3892 | 3892 | 3892 |
| Bayes' $R^2$ | 0.339 | 0.388 | 0.428 | 0.405 |
| WAIC | 12955.3 | 14509.8 | 15541.1 | 19975.7 |

### 3.3.4  Stratified models

Table 3.5 shows the associations between acute pediatric asthma and the environmental exposure variables for strata of the census tracts defined by quartiles of the proportion of non-white residents. Our previous work (see Chapter 2) indicates that there are significant racial disparities in asthma outcomes in the study area and the purpose of stratifying these models is to investigate whether there are systematic differences in the strength of effects. While there is some variability in the estimated IRRs between the four stratified models, there is no consistent variation as the proportion of non-white residents increases. In fact, the only significant difference between the models is the WAIC which indicates that the full model including both temporal trends and the environmental exposure variables is a better fit for the bottom quartile compared to the top quartile. As for the results presented in Table 3.4, we should be unsurprised that there are no strong effects present. These models include data from the entire study period likely averaging out the seasonal variation in environmental exposure health effects.

Table 3.6: Exponeniated estimates (IRRs) with 89% credibility intervals for the winter models. Lower WAIC (widely applicable information criterion) indicates better model fit. Model 1 is for the bottom quartile of proportion of non-white residents and Model 4 is for the top quartile.

| | Proportion non-white residents—Winter | | | |
| | 0-25% | 25-50% | 50-75% | 75-100% |
| --- | --- | --- | --- | --- |
| Intercept | 0.000 | 0.000 | 0.000 | 0.000 |
| | [0.000, 0.000] | [0.000, 0.000] | [0.000, 0.000] | [0.000, 0.000] |
| Tmax | 1.003 | 1.004 | 1.010 | 1.009 |
| | [0.997, 1.009] | [0.999, 1.009] | [1.006, 1.015] | [1.006, 1.013] |
| DDmed | 1.032 | 1.013 | 1.003 | 1.005 |
| | [1.017, 1.048] | [1.001, 1.027] | [0.991, 1.015] | [0.996, 1.013] |
| WDSP | 0.993 | 0.998 | 0.998 | 0.989 |
| | [0.983, 1.002] | [0.989, 1.006] | [0.990, 1.005] | [0.983, 0.994] |
| $PM_{2.5}$ | 0.997 | 1.005 | 1.004 | 0.995 |
| | [0.991, 1.003] | [1.000, 1.010] | [0.999, 1.009] | [0.992, 0.998] |
| Num.Obs. | 909 | 909 | 909 | 909 |
| Bayes' $R^2$ | 0.319 | 0.360 | 0.380 | 0.233 |
| WAIC | 3134.2 | 3485.7 | 3738.1 | 4733.0 |

### 3.3.5 Stratified seasonal models

Tables 3.6-3.9 show the associations between acute pediatric asthma and the environmental exposure variables stratified by quartiles of the proportion non-white residents for each season. Comparing the coefficient estimates between quartiles within each season allows us to assess whether environmental exposure disproportionately affects communities of color. Results show that there are no consistent seasonal trends in coefficient variability associated with different levels of the proportion of non-white residents.

## 3.4 Discussion

Understanding the temporal variability of acute pediatric asthma and its dependence on atmospheric conditions is key to both understanding the potential impacts of climate change on pediatric health and to devising strategies for mitigating these impacts and improving public health. Strategies such as these should be based on research relevant to the population and geography under

Table 3.7: Exponeniated estimates (IRRs) with 89% credibility intervals for the spring models. Lower WAIC (widely applicable information criterion) indicates better model fit. Model 1 is for the bottom quartile of proportion of non-white residents and Model 4 is for the top quartile.

| | Proportion non-white residents—Spring | | | |
| | 0-25% | 25-50% | 50-75% | 75-100% |
| --- | --- | --- | --- | --- |
| Intercept | 0.000 | 0.000 | 0.000 | 0.000 |
| | [0.000, 0.000] | [0.000, 0.000] | [0.000, 0.000] | [0.000, 0.000] |
| Tmax | 0.993 | 0.989 | 0.994 | 1.002 |
| | [0.986, 1.001] | [0.983, 0.995] | [0.989, 1.001] | [0.999, 1.006] |
| DDmed | 1.010 | 0.999 | 1.001 | 0.998 |
| | [0.999, 1.021] | [0.989, 1.008] | [0.992, 1.010] | [0.992, 1.003] |
| WDSP | 1.008 | 1.004 | 0.997 | 1.001 |
| | [1.000, 1.016] | [0.997, 1.010] | [0.990, 1.003] | [0.997, 1.005] |
| $PM_{2.5}$ | 1.002 | 1.002 | 1.007 | 1.002 |
| | [0.995, 1.009] | [0.996, 1.008] | [1.001, 1.013] | [0.999, 1.006] |
| Num.Obs. | 1027 | 1027 | 1027 | 1027 |
| Bayes' $R^2$ | 0.268 | 0.331 | 0.361 | 0.300 |
| WAIC | 3543.9 | 3908.5 | 4277.6 | 5335.5 |

Table 3.8: Exponeniated estimates (IRRs) with 89% credibility intervals for the summer models. Lower WAIC (widely applicable information criterion) indicates better model fit. Model 1 is for the bottom quartile of proportion of non-white residents and Model 4 is for the top quartile.

| | Proportion non-white residents—Summer | | | |
| | 0-25% | 25-50% | 50-75% | 75-100% |
| --- | --- | --- | --- | --- |
| Intercept | 0.000 | 0.000 | 0.000 | 0.000 |
| | [0.000, 0.000] | [0.000, 0.000] | [0.000, 0.000] | [0.000, 0.000] |
| Tmax | 0.993 | 0.986 | 0.984 | 0.983 |
| | [0.977, 1.008] | [0.972, 1.001] | [0.972, 0.997] | [0.976, 0.991] |
| DDmed | 1.013 | 1.014 | 1.020 | 1.019 |
| | [0.997, 1.032] | [0.997, 1.030] | [1.005, 1.033] | [1.010, 1.028] |
| WDSP | 1.015 | 1.000 | 0.995 | 1.001 |
| | [1.005, 1.025] | [0.991, 1.009] | [0.986, 1.003] | [0.996, 1.006] |
| $PM_{2.5}$ | 0.998 | 1.002 | 1.007 | 1.001 |
| | [0.991, 1.004] | [0.997, 1.008] | [1.002, 1.012] | [0.998, 1.004] |
| Num.Obs. | 1049 | 1049 | 1049 | 1049 |
| Bayes' $R^2$ | 0.233 | 0.263 | 0.266 | 0.215 |
| WAIC | 3106.8 | 3453.9 | 3705.1 | 5058.2 |

Table 3.9: Exponeniated estimates (IRRs) with 89% credibility intervals for the fall models. Lower WAIC (widely applicable information criterion) indicates better model fit. Model 1 is for the bottom quartile of proportion of non-white residents and Model 4 is for the top quartile.

| | Proportion non-white residents—Fall | | | |
| | 0-25% | 25-50% | 50-75% | 75-100% |
|---|---|---|---|---|
| Intercept | 0.000 | 0.000 | 0.000 | 0.000 |
| | [0.000, 0.000] | [0.000, 0.000] | [0.000, 0.000] | [0.000, 0.000] |
| Tmax | 1.000 | 1.001 | 1.000 | 1.001 |
| | [0.991, 1.008] | [0.994, 1.009] | [0.994, 1.007] | [0.996, 1.005] |
| DDmed | 0.999 | 0.995 | 1.004 | 1.002 |
| | [0.984, 1.012] | [0.983, 1.008] | [0.993, 1.014] | [0.995, 1.008] |
| WDSP | 1.000 | 1.004 | 0.998 | 1.000 |
| | [0.989, 1.009] | [0.995, 1.012] | [0.991, 1.006] | [0.995, 1.005] |
| $PM_{2.5}$ | 1.008 | 1.006 | 1.005 | 1.000 |
| | [1.001, 1.015] | [0.999, 1.012] | [1.000, 1.011] | [0.997, 1.004] |
| Num.Obs. | 907 | 907 | 907 | 907 |
| Bayes' $R^2$ | 0.288 | 0.292 | 0.407 | 0.264 |
| WAIC | 3269.4 | 3706.2 | 3839.7 | 4959.6 |

consideration. To this end, this research investigates the time course of acute pediatric asthma in the Kansas City Metro Area and seeks to elucidate any meaningful relationships with atmospheric conditions. Additionally, we investigate whether these relationships vary by proportion of non-white residents in an effort to understand the structure of public health vulnerability.

The daily acute pediatric asthma incidence shows a seasonal as well as day of week trend that is well documented in the literature (Won et al., 2016). Including variables for the day of week as well as a natural cubic spline to model the seasonality of asthma cases is necessary to control for these trends and produces a better fit to the data than only including the environmental exposure variables. Additionally, including both the temporal variables and the exposure variables does not improve model fit compared to the time-only model. Asthma is the result of environmental and immunological interactions and there are many factors associated with acute pediatric incidence including seasonal respiratory infections, seasonal allergies, indoor pollution, genetics, and tobacco smoke exposure and it is to be expected that these factors explain most of the variation in asthma incidence (Trivedi & Denton, 2019; Milligan et al., 2016; D'Amato et al., 2015).

We expected the environmental exposure variables to have effects that vary by season. These effects are averaged out in the full model which estimates coefficients for the entire time series; to understand the seasonal variability of environmental exposure effects we ran separate models for each season. Results are contrary to those most frequently reported in the literature, with Tmax showing a positive relationship in the winter and a negative relationship in the summer. Most studies report that an increase in Tmax during the summer results in an increase in acute asthma, however in Kansas City we see a decrease. We hypothesize that this is due to behavioral factors. Summers in Kansas City are hot and humid and there are few outdoor recreational activities that would provide a respite from these uncomfortable conditions (i.e. public swimming pools). Increased temperatures during the summer may encourage children to stay indoors where air conditioning can decrease any environmental exposure-induced inflammation that may contribute to acute asthma incidence. Likewise, most studies report that a decrease in Tmax during the winter results in an increase in acute asthma. Winters in Kansas City can be quite cold and an increase in winter temperature may encourage children to spend time outdoors, thus increasing exposure. In this case although temperatures have increased they are still likely to be quite cold, with breathing cold air being a well recognized trigger for constriction of the airways (D'Amato et al., 2015).

There is a relatively strong effect of DDmed in the summer, with an increase of $5°C$ in the dew point depression corresponding to a 9.17% increase in acute asthma incidence. A higher dew point depression corresponds to less humid conditions, indicating that dry heat during the summer increases asthma incidence. This again is contrary to what is generally reported in the literature, although both low and high humidity conditions can exacerbate asthma albeit via different biophysical mechanisms (Lam et al., 2016). $PM_{2.5}$ has a positive effect in spring and fall, times of generally pleasant weather when children are most likely to play outdoors.

Additionally, we ran separate models for each strata of proportion non-white residents to investigate whether there were disparities in the magnitude of the effects of environmental exposure. In previous research we found that neighborhood racial composition had the strongest effect on the relative risk of asthma incidence in census tracts as reported in Chapter 2. However,

we found no consistent pattern of variation in coefficient estimates between the strata, indicating that the temporally varying health effects of environmental exposure are consistent between neighborhoods. This does not mean environmental exposures disparities do not exist, but rather that the there is no systematic difference in the time course of health effects.

We further interrogated this result by running separate models for each combination of strata and season. While for the most part there is no consistent variation of coefficients, results indicate that the positive effect of Tmax in the winter is strongest in the neighborhoods with more than 50% non-white residents. This translates to a 4.80% increase in cases for a 5°C increase in temperature for neighborhoods with 75-100% non-white residents versus no effect for neighborhoods with less than 50% non-white residents. So while the models do not indicate that neighborhoods with a higher proportion of non-white residents are more vulnerable to variations in atmospheric conditions, the data do show that these neighborhoods have higher rates of acute pediatric asthma in all seasons.

### 3.4.1 Limitations

There are several limitations to this research. First, this study does not consider the individual and instead uses daily counts for the study area as the outcome of interest. And although in the stratified analysis the strata are comprised of counts per quartile of census tracts, neighborhood effects are otherwise not emphasized. Obviously this precludes examination of a host of asthma-related exposures and determinants experienced by both individuals and communities. Second, we assume that a daily environmental measure is sufficient to characterize exposure for the entire study population. With this assumption we have prioritized an understanding of the temporal characteristics of asthma over the spatial characteristics (see Chapter 2 for an investigation of the spatial variability of asthma). Both individuals and neighborhoods will differ in their exposure levels, however, the daily summary measures can be considered a central value around which exposure measured at the neighborhood level would cluster. While the neighborhood-level variation of exposure and effects are an important consideration, trends in the temporal dependence

of acute pediatric asthma on atmospheric conditions will still be apparent when considering average values. Additionally, we did not consider lag structures between the asthma counts and the temporal variables. While such structures may exist, they are difficult to interpret and the complexity of the estimated coefficients does not easily translate into effective policy.

### 3.4.2 Strengths

This study has three major strengths. First, the pediatric asthma data spans a period of twelve years and a geographical area that is comprised of 427 census tracts representing a range of social and environmental conditions. Second, we stratified the analysis to examine the effects of meteorological factors both seasonally and by subgeographies corresponding to quartiles of the proportion of non-white residents per census tract. Third, we used a Bayesian framework to understand the certainty of effects and created simple models that are relatively easy to interpret. While many studies use complicated lag models to describe the impact of temperature and air pollution on asthma, they often report estimates whose confidence intervals contain null effects and present models whose structure and interpretation are esoteric at best. Ultimately, the purpose of an analysis such as that presented here is to inform policy and strategic interventions to improve health. Communication and parsimony are both key components of such a goal.

### 3.5 Conclusions

Our findings suggest that while some small effects of meteorological conditions on acute pediatric asthma incidence exist in the Kansas City Metro Area, they are small compared to racial disparities. While interventions to improve public health should emphasize mitigating extremes of temperature, humidity, and air pollution, they are unlikely to significantly affect the rate of acute asthma incidence. Rather, specific interventions to improve asthma outcomes should focus on addressing the causes of environmental health disparities. Additionally, urban planners and other policy makers need information that is easy to interpret and actionable. Rather than focusing

on exact effect estimates and complicated lag structures, public health and environmental exposure research should focus on the magnitude and direction of effects in parsimonious models. Increasing the complexity of a model does not automatically reveal the complexity of the system but often obscures the structure of the system of interest. This study highlights that the most policy-relevant structure of acute pediatric asthma in Kansas City is the striking racial disparities in outcomes.

## Abstract

Well-managed urbanization can promote health, justice, and sustainability and environmental health research can provide the evidence necessary to support decision making by urban planners and communities. Too often, however, there is a translation gap between research and praxis. In an attempt to remedy this situation and to promote interpretable, impactful, and actionable environmental health research we present opinionated recommendations in five key areas: public participation, transdisciplinary collaboration, applied research, knowledge translation, and ethics & justice.

## 3.6 Introduction

Over half of the global population lives in urban areas and the United Nations predicts that this proportion will rise to more than two thirds by the year 2050 (United Nations, 2019). Urbanization is a complex process that alters both physical and social systems, transforming urban environments and demographic and social structures (United Nations, 2019). While generally considered a positive force bringing increased upward economic mobility, improved access to food and health care, and improved services (United Nations, 2019), urbanization can also result in increased income inequality and environmental degradation (Rohat et al., 2019). Additionally, urbanization emerges from myriad intersecting power structures and the benefits of urbanization are not evenly distributed either spatially or socially. Well-managed urbanization has the potential to minimize adverse impacts, improve health, and ensure the benefits of urban living are equitably shared (United Nations, 2019). Effective and equitable urban policy must be scientifically informed and understanding how urban environments affect health outcomes must be of the highest priority for researchers (Rydin et al., 2012; Prüss-Ustün, A et al., 2019).

Improving environments for health is an important aspect of the UN's Sustainable Development Goals (SDGs), including increasing good health and well-being for all (SDG 3), reducing

inequality (SDG 10), and creating sustainable cities and communities (SDG 11) (United Nations, 2021). Achieving these goals requires an understanding of how urban environments affect health outcomes (Rydin et al., 2012). Environmental health pathways comprise an understanding of risk moving from source, to emissions, to environmental concentrations, to exposure, to health outcome (Mock et al., 2017). Environmental risks from urbanization and industrialization occur primarily at the community-level, deriving from larger-scale organization including urban form, and include risk from air pollution and extreme heat events (Mock et al., 2017). These negative health risks are unequally distributed in cities and the burden of ill-health is borne disproportionately by those in socially disadvantaged groups (Rydin et al., 2012). Globally these risks are exacerbated by climate change with the the burden of ill-health borne primarily by populations with minimal contributions to greenhouse gas concentrations (Mock et al., 2017). Disparities in public health outcomes are partially driven by the interactions between climatic, environmental, and social systems in urban areas. These systems are highly interdependent and interact in complex ways that produce spatially heterogeneous landscapes of vulnerability (Ferraro et al., 2019; Liu et al., 2007b; Pickett et al., 2001). Reducing disparities in environmental health outcomes requires emphasis on both environmental hazards and social conditions, and a better understanding of how they interact (Schlosberg, 2013).

Established pathways through which racism affects health include adverse physical, social, and economic exposures (Bailey et al., 2017). Structural racism has had a substantial role in shaping the spatial distribution of health outcomes in the United States producing persistent inequalities (Bailey et al., 2017). Neighborhood physical environments, land use, health infrastructure, education, economic and political opportunities, and exposure to pollutants are all linked to race-based residential segregation and ultimately unequal health outcomes (Schulz et al., 2016; Bailey et al., 2017; Gee & Payne-Sturges, 2004). Structures of social stratification result in the unequal distribution of power and resources with environmental exposures and environmental injustice occurring along social axes, resulting in uneven distributions of health-damaging conditions (Smith & Laribi, 2021; World Health Organization, 2010).

Physical and social processes in urban areas interact across a multitude of spatial and temporal scales, with flows and feedbacks moving into and out of cities (Desouza & Flanery, 2013). Institutions, actors, resources, and processes interact in dynamic ways and it is through the intentional enhancement and suppressing of flows that we can create adaptive and equitable urban systems (Desouza & Flanery, 2013). Public health as it relates to environmental exposure in urban areas is shaped by economic, technological, demographic, and governmental structures (Desouza & Flanery, 2013) and understanding where leverage points to improve health exist within these structures is paramount. Vulnerability comprises the interactions between the spatial variability of exposure, sensitivity, and adaptive capacity and how these components respond to environmental stressors and urbanization scenarios (Wilhelmi & Hayden, 2010). Urban science for city policy should occur multilaterally across local, national, and international levels and involve evidence-based policy driven by environmental health research (Acuto, 2018). Theory and practice should be mutually informative requiring connections between communities and researchers (Schlosberg, 2013).

Social inequalities and health are not issues that can be separated from overall development policy (Costello et al., 2009). Policy makers must identify potential levels of intervention and leverage points for action on environmental health disparities (World Health Organization, 2010). Action can range from policies that tackle the upstream structural determinants to approaches that focus on community-based health outcomes (World Health Organization, 2010). Unfortunately, urban planning policy is frequently inconsistent with public health evidence (Lowe et al., 2022) and the purposes and values of urban planning are not necessarily aligned with those of public health (Pineo et al., 2020). The goal of public health is to equitably improve health while the goal of urban planning is to manage change in urban environments according to the objectives of city officials (Pineo et al., 2020). Bridging this potential disconnect is a key objective of environmental health research. The evidence-based public health (EBPH) paradigm emphasizes "making decisions based on high-quality, peer-reviewed evidence; systematic use of data and information systems; application of program planning frameworks based in behavioral science theory; community

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engagement; program evaluation; transparent dissemination of evaluation findings; and synthesis of sound decision making and scientific skill in decision making" (Hess et al., 2014).

Environmental exposure can impact health even at low levels and urban areas should be managed to promote healthy environments for all (Brook et al., 2018; Prüss-Ustün, A et al., 2019). Economic growth, development, and urban expansion cannot be relied upon to drive these improvements (Rydin et al., 2012) but rather governance for urban health requires a systems approach that considers the complex interactions between environmental and social systems that contribute to variation in health outcomes, involves transdisciplinary collaboration and community participation, and translates knowledge from research to practice (Pineo et al., 2020). Alleviating health disparities and promoting good health provides a foundation for social justice, economic prosperity, and environmental protection, all necessary components of sustainable cities (Dye, 2018; United Nations, 2021).

Policy making is a complex, contested, and nonlinear process (Pineo et al., 2020) and requires high-quality scientific information. However, the evidence necessary to support the development of healthy communities is often missing (United Nations, 2021). Research should be interpretable, impactful, and actionable but frequently is obtuse, esoteric, and inert (Choirat et al., 2019). To fill the lacuna between research and praxis, this paper provides opinionated recommendations for restructuring environmental health research.

## 3.7 Research priorities

Effective research for improving urban environmental health and decreasing environmental health disparities requires work in several areas. We have identified five components of environmental health research that are key to successful policy implementation to equitably improve health: public participation, transdisciplinary collaboration, applied research, knowledge translation, and ethics & justice.

### 3.7.1   Public participation

Scientific knowledge is usually conceived outside of the policy systems researchers seek to inform (Pineo et al., 2020). There should be clear identification and communication of shared goals between researchers and community members (Desouza & Flanery, 2013). The skills necessary for community involvement in the research process should be embedded in research curriculum (Fleming et al., 2018). Public involvement should be integrated with research at all levels, including formulating the research question, designing the study and data collection methods, deciding on statistical methods, and interpreting and communicating the results (Fleming et al., 2018; Choirat et al., 2019). Research should be co-designed with policy makers to ensure that studies are policy relevant (Lowe et al., 2022). Incorporating community voices into the generation of information builds scientific capacity and resilience in local infrastructure and attributes collective meaning to research findings and data (Costello et al., 2009; Lawrence et al., 2017). Collection of data is impacted by inequities and often areas where research may be the most impactful are data poor; community involvement in the data-generating process can promote equity and dismantle statistical bias.

Public participation also provides the opportunity to educate the public about science and to mitigate the perceived poor understanding of environmental systems (Kramer et al., 2017) while also educating researchers about the realities and priorities of the community. Environmental health disparities research should involve disadvantaged communities and those directly impacted by environmental change in research development (Rydin et al., 2012; Fleming et al., 2018). This co-creation of knowledge makes the research more directly impactful and applicable to involved communities (Fleming et al., 2018). Public participation ensures that the full diversity of interests are represented in the research process which supports the probability that complex problems can be solved effectively (Desouza & Flanery, 2013). Public participation also empowers communities; social power is vital to reducing health disparities and communities should have control over the decisions that affect their health (World Health Organization, 2010). Communities can also contribute experiential knowledge and help identify hazards (Rydin et al., 2012; Frank, 2021).

Additionally, involving at-risk communities when investigating environmental health disparities increases the chance of producing research that is respectfully framed and free of stigmatization (Frank, 2021). Researchers should also collaborate with policy makers and community members to evaluate the costs, consequences, and benefits of policies, both in terms of economics and health (Lowe et al., 2022). Realizing health equity requires empowering communities to exercise control over the factors that shape their health (World Health Organization, 2010).

### 3.7.2 Transdisciplinary collaboration

Researchers should strive to develop partnerships between academia, communities, environmental groups, and health service providers to support the effective translation of research into environmental health policy (Schulz et al., 2016). The development of urban science to support policy should be fully integrated between diverse academic disciplines in the natural sciences, social sciences, and the humanities and across methodological divides (Acuto, 2018; Lawrence et al., 2017). Urban environmental health challenges are complex and require a diversity of perspectives to formulate effective responses (Lawrence et al., 2017). Scientific knowledge is only one component of a larger domain where information, concepts, meaning, methods, and values converge into societal knowledge (Lawrence et al., 2017). Within this larger domain, researchers and academics have an important role in interfacing between scientific knowledge, public policy, and human behavior to catalyze the development of equitable and health-promoting environments (Lawrence et al., 2017; Costello et al., 2009).

### 3.7.3 Applied research

Whatever the historical reasons for disdaining applied research within the academy (Acuto, 2018), such a paradigm is no longer ethically tenable. Environmental health and environmental health disparities will worsen with climate change without scientifically-informed policies. Research on environmental health needs to be suitable for policy purposes and should be designed to maximize the impact of results (Acuto, 2018). A focus on practical relevance should be incorporated from the

onset of a research project and public involvement from stakeholders and practitioners should guide development of the research question and all aspects of the study (Lowe et al., 2022; Choirat et al., 2019; Hess et al., 2014; Fleming et al., 2018). Robust methods that are reproducible, interpretable, and transparent should be prioritized and researchers must be accountable for methods, data, and results (Acuto, 2018; Choirat et al., 2019).

Effective scientific advice to cities must encompass a topical and geographical shift in environmental health research to focus on specific community needs and problems (Acuto, 2018). Localizing research projects promotes the use of methods that are sensitive to local conditions thus ensuring that results are directly actionable and supported by local health priorities and community opinion (Pineo et al., 2020; Rydin et al., 2012). Studies should be localized and spatially explicit to highlight interurban contrasts and also be linked to global scales to ensure robustness in a changing climate and consistency within national and international contexts (Rohat et al., 2019; Pineo et al., 2020). This includes locally extending and contextualizing global economic scenarios (Shared Socioeconomic Pathways—SSPs) and climate scenarios (Representative Concentration Pathways—RCPs) to project local drivers of vulnerability (Rohat et al., 2019).

Uncertainty exists about the scale and timing of the effects of environmental exposure on health, interactions within a changing climate system, and potential changes to social systems and demographic structures (Costello et al., 2009; Rohat et al., 2019). Regardless, policy responses and action must proceed in spite of this uncertainty and research should incorporate climatic and socioeconomic scenarios to assess future vulnerability (Rohat et al., 2019; Costello et al., 2009). Future environmental health risk will be affected by feedbacks between socioeconomic development, climate, and exposure and the influence of land-use change and urban morphology on health outcomes should be assessed to provide evidence for health-promoting development (Rohat et al., 2019; Schulz et al., 2016). A greater understanding of the range of potential interactions between the physical environment and health in urban areas can inform policy for reducing disease burdens, minimizing health inequalities, and lessening the negative impacts of changing environments (Brook et al., 2018).

### 3.7.4 Knowledge translation

Information has no worth without the capacity for its use and dissemination of relevant research findings is as crucial as the generation of knowledge (Costello et al., 2009). Planning professionals and other policy makers need clear guidance on the elements of urban systems that produce beneficial health impacts (Hooper et al., 2021), however, there is a considerable translation gap between environmental health research and planning policy and practice (Hooper et al., 2021). Knowledge translation encompasses "the process of synthesizing, disseminating, and applying knowledge to improve public health practice" (Hess et al., 2014). An emphasis on communication introduces an academic evidence base to policy makers for decision support (Hooper et al., 2021). Communicating research, sharing data, and creating tools to facilitate their use helps in four areas: visualization of proposed policy changes, understanding policy consequences, understanding health impacts, and engaging community members (Hooper et al., 2021). Providing policy makers with evidence about how their decisions may impact the health of their communities empowers them (World Health Organization, 2010).

Public health evidence is applied in a variety of settings (Hess et al., 2014) and thus knowledge translation plays a key role in ensuring evidence is accessible to those who require it. Much of the existing data and research relevant to environmental health and urban planning and policy resides in the academic literature and is inaccessible to the professionals whose work it should inform (Hooper et al., 2021). Making data and research available can help guide decision making and investments (Schulz et al., 2016) while simultaneously empowering communities through data ownership. Data about communities should belong to the communities.

Urban environmental health is a complex system and tools can simplify interpretation of the interactions between many interconnected parts and make data accessible to affected communities (Smith & Laribi, 2021; Pineo et al., 2020). Such tools include Urban Health Indicators (UHI)—metrics that quantify the impact of urban environments on health, and Planning Support Systems (PSS)—computer-based tools that integrate GIS and decision support by combining spatial data, models, and geovisualization to allow for simulation and testing of policy scenarios

(Hooper et al., 2021; Pineo et al., 2020). These tools can drive more equitable and inclusive policies for public health and reach a wider audience by putting data in a digestible form suited to the working processes and practices of planning professionals (Smith & Laribi, 2021; Kumar et al., 2019; Hooper et al., 2021). Tools have the additional benefit of allowing bespoke customization to test specific policy scenarios (Hooper et al., 2021). Creation of these tools should be transparent and make explicit the associated evidence and assumptions so that policy makers can make educated assessments about their suitability (Pineo et al., 2020). A co-design approach should be taken to tool development to ensure that the tool functionality meets the expectations and capabilities of end users (Hooper et al., 2021). Maps are particularly useful tools for decision makers to identify vulnerable populations and guide prioritizing communities for policy interventions (Schulz et al., 2016; Pineo et al., 2020). The abundance of innovative communication media provides opportunities for education and collaboration to support for decision making (Lawrence et al., 2017; Hooper et al., 2021). Clearly communicating relevant environmental health information can reframe problems and policy debates to motivate action (Pineo et al., 2020; Hooper et al., 2021).

## 3.7.5 Ethics & Justice

Power has a central role in the structure of environmental health disparities, thus the process of attempting to solve these problems is political and engages both the agency of disadvantaged communities and multiple levels of governance (World Health Organization, 2010). The policies that shape environmental health outcomes are guided by values, either implicit or explicit, and consideration of the value-laden nature of policy interventions is essential (Rydin et al., 2012). Scientists and researchers have ethical obligations to both the communities they study and the broader society whose resources support their research activity (Frank, 2021). Scientists are not objective observers but rather social actors who are implicated in "relations of social, political, economic, and technological power" and contextual values like human health, social justice, and sustainability are highly relevant throughout the research process (Frank, 2021). These

values justify choices and should be considered in methodological decisions (Frank, 2021). Methodological considerations raise real-life risks like reducing the likelihood of abatement of environmental exposures, and scientists have a moral obligation to consider the consequences of their research choices (Frank, 2021). Identification of environmental health hazards and quantitative measures of risk are all research choices that prioritize some causes of health over others, thus research decisions should explicitly include concerns of justice and the political and economic feasibility of policy interventions (Frank, 2021). Deeply embedded in how we choose to define a problem and what solutions the problem implies are ethical and political worldviews and different frames will yield different results (Chrisinger, 2022). Environmental health researchers should be transparent in their decision making process and realize that they cannot shield themselves from non-epistemic considerations (Frank, 2021). Indeed, given the nature and severity of environmental health consequences and the stark disparities that exist in outcomes, researchers should not seek to avoid or ignore the value-laden nature of scientific research but rather embrace the opportunity to produce evidence to inform policy for increasing environmental justice.

## 3.8 Conclusions

Improving environments for health must be a primary focus of both environmental health researchers and urban planners and policy makers. Well-managed urbanization to promote human health, social justice, and sustainability requires scientifically robust evidence but there is frequently a translation gap between research and praxis. We have laid out five components of environmental health research necessary to unite communities, practice, policy, and research in the common goal of improving well-being for all: public participation, transdisciplinary collaboration, applied research, knowledge translation, and ethics & justice. It is our sincere hope as researchers and concerned citizens that our recommendations will inform the change demanded by a global need for action to protect health and eliminate health disparities.

# Chapter 4

# Conclusions

## 4.1 Summary of findings

Disparities in public health outcomes are partially driven by the spatial and temporal variability of the interactions between climatic, environmental, and social systems in urban areas. These systems are highly interdependent and interact in complex ways that produce spatially heterogeneous landscapes of environmental exposure and vulnerability. Urban policy has the potential to shape health-promoting and equitable environments but must be informed by an understanding of how health disparities manifest from these interactions. This dissertation research investigates acute pediatric asthma in the Kansas City Metro Area as a case study for modelling these interactions.

Chapter 2 examines the neighborhood-level spatial variability of acute asthma incidence and climatic, environmental, and social factors. Results show that increased concentrations of $PM_{2.5}$ increased asthma rates but that this effect was mitigated in high-pollution neighborhoods by vegetated cover. Greenspace can reduce pollution levels by encouraging particulate deposition. We also found that the protective effect of vegetation was stronger in neighborhoods characterized by a high proportion of residents living in poverty and a high proportion of non-white residents. Additionally, we found that even after accounting for poverty, there were significant racial disparities in asthma outcomes.

Chapter 3 examines the temporal variability of daily asthma incidence and meteorologic conditions. Results indicate that contrary to findings typically reported in the literature, maximum temperature has a negative relationship with asthma in the summer and a positive relationship with asthma in the winter. Additionally, we found that a decrease in summer humidity was associated

with an increase in asthma incidence. We ran separate models for strata of the proportion of non-white residents but found no systematic variation in the magnitude of meteorological effects. However, the difference in rates of acute asthma incidence between strata was greater than any of the temporal effects of meteorological conditions.

Taken together Chapters 2 and 3 paint a clear picture of racial disparities in asthma outcomes. However, what is important to take away from this analysis is the clear identification of effective policy leverage points. Investing in greenspace infrastructure in neighborhoods of color is likely to decrease air pollution levels and decrease asthma incidence.

## 4.2 Summary of contributions

This dissertation builds upon a large body of work on environmental health and environmental health disparities. The purpose of this research is to provide interpretable, impactful, and actionable evidence for improving asthma outcomes in Kansas City. While there are many studies investigating the effects of social, environmental, and climatic conditions on pediatric asthma, they frequently present opaque methods and difficult to interpret results. To that end we utilized simple models in a Bayesian framework to promote portability of methods to other environmental health problems and attempted to present our results in easy to understand terms. Environmental health researchers should focus not just on the scientific merit of their work but also its use value. In Chapter 3.5 we present a set of recommendations to guide this endeavor.

## 4.3 Recommendations for future work

The research presented in this dissertation characterizes the spatial and temporal variability of acute asthma. However, asthma incidence varies concurrently in both time and space and future research should model the spatiotemporal variability of asthma rates. Additionally, the robustness of the models should be tested with additional years of data.

# References

Acuto, M. (2018). Global science for city policy. *Science*, 359(6372), 165–166.

Akinbami, L. J., Simon, A. E., & Rossen, L. M. (2016). Changing Trends in Asthma Prevalence Among Children. *Pediatrics*, 137(1), e20152354.

Alcock, I., White, M., Cherrie, M., Wheeler, B., Taylor, J., Mcinnes, R., Otte Im Kampe, E., Vardoulakis, S., Sarran, C., Soyiri, I., & Fleming, L. (2017). Land cover and air pollution are associated with asthma hospitalisations: A cross-sectional study. *Environment International*, 109, 29–41.

Anderson, B. G. & Bell, M. L. (2009). Weather-Related Mortality. *Epidemiology*, 20(2), 205–213.

Anderson, G. B., Dominici, F., Wang, Y., McCormack, M. C., Bell, M. L., & Peng, R. D. (2013). Heat-related Emergency Hospitalizations for Respiratory Diseases in the Medicare Population. *American Journal of Respiratory and Critical Care Medicine*, 187(10), 1098–1103.

Aybar, C. (2021). *Rgee: R Bindings for Calling the 'earth Engine' API*.

Ayres-Sampaio, D., Teodoro, A. C., Sillero, N., Santos, C., Fonseca, J., & Freitas, A. (2014). An investigation of the environmental determinants of asthma hospitalizations: An applied spatial approach. *Applied Geography*, 47, 10–19.

Bai, X., Dawson, R. J., Ürge-Vorsatz, D., Delgado, G. C., Salisu Barau, A., Dhakal, S., Dodman, D., Leonardsen, L., Masson-Delmotte, V., Roberts, D. C., & Schultz, S. (2018). Six research priorities for cities and climate change. *Nature*, 555(7694), 23–25.

Bailey, Z. D., Krieger, N., Agénor, M., Graves, J., Linos, N., & Bassett, M. T. (2017). Structural

racism and health inequities in the USA: Evidence and interventions. *The Lancet*, 389(10077), 1453–1463.

Brewer, M., Kimbro, R. T., Denney, J. T., Osiecki, K. M., Moffett, B., & Lopez, K. (2017). Does neighborhood social and environmental context impact race/ethnic disparities in childhood asthma? *Health & Place*, 44, 86–93.

Brook, J. R., Setton, E. M., Seed, E., Shooshtari, M., Doiron, D., & CANUE – The Canadian Urban Environmental Health Research Consortium (2018). The Canadian Urban Environmental Health Research Consortium – a protocol for building a national environmental exposure data platform for integrated analyses of urban form and health. *BMC Public Health*, 18(1), 114.

Bullard, R. D. (1999). Dismantling Environmental Racism in the USA. *Local Environment*, 4(1), 5–19.

Bürkner, P.-C. (2017). Brms: An R Package for Bayesian Multilevel Models Using Stan. *Journal of Statistical Software*, 80(1).

Buteau, S., Geng, X., Labelle, R., & Smargiassi, A. (2019). Review of the effect of air pollution exposure from industrial point sources on asthma-related effects in childhood:. *Environmental Epidemiology*, 3(6), e077.

Carlson, T. N. & Ripley, D. A. (1997). On the relation between NDVI, fractional vegetation cover, and leaf area index. *Remote Sensing of Environment*, 62(3), 241–252.

Castillo, M. D., Kinney, P. L., Southerland, V., Arno, C. A., Crawford, K., van Donkelaar, A., Hammer, M., Martin, R. V., & Anenberg, S. C. (2021). Estimating Intra-Urban Inequities in PM$_{2.5}$ -Attributable Health Impacts: A Case Study for Washington, DC. *GeoHealth*, 5(11).

Chang, H. H., Pan, A., Lary, D. J., Waller, L. A., Zhang, L., Brackin, B. T., Finley, R. W., & Faruque, F. S. (2019). Time-series analysis of satellite-derived fine particulate matter pollution

and asthma morbidity in Jackson, MS. *Environmental Monitoring and Assessment*, 191(S2), 280.

Chen, L., Mengersen, K., & Tong, S. (2007). Spatiotemporal relationship between particle air pollution and respiratory emergency hospital admissions in Brisbane, Australia. *Science of The Total Environment*, 373(1), 57–67.

Choirat, C., Braun, D., & Kioumourtzoglou, M.-A. (2019). Data Science in Environmental Health Research. *Current epidemiology reports*, 6(3), 291–299.

Chrisinger, B. W. (2022). Getting to Root Causes: Why Equity Must Be at the Center of Planning and Public Health Collaboration. *Journal of the American Planning Association*, (pp. 1–7).

Costanza, R., Wainger, L., Folke, C., & Maler, K.-G. (1993). Modeling Complex Ecological Economic Systems. *BioScience*, 43(8), 545–555.

Costello, A., Abbas, M., Allen, A., Ball, S., Bell, S., Bellamy, R., Friel, S., Groce, N., Johnson, A., Kett, M., Lee, M., Levy, C., Maslin, M., McCoy, D., McGuire, B., Montgomery, H., Napier, D., Pagel, C., Patel, J., de Oliveira, J. A. P., Redclift, N., Rees, H., Rogger, D., Scott, J., Stephenson, J., Twigg, J., Wolff, J., & Patterson, C. (2009). Managing the health effects of climate change. Lancet and University College London Institute for Global Health Commission. *The Lancet*, 373(9676), 1693–1733.

Dai, D. (2011). Racial/ethnic and socioeconomic disparities in urban green space accessibility: Where to intervene? *Landscape and Urban Planning*, 102(4), 234–244.

D'Amato, G., Holgate, S. T., Pawankar, R., Ledford, D. K., Cecchi, L., Al-Ahmad, M., Al-Enezi, F., Al-Muhsen, S., Ansotegui, I., Baena-Cagnani, C. E., Baker, D. J., Bayram, H., Bergmann, K. C., Boulet, L. P., Buters, J. T., D'Amato, M., Dorsano, S., Douwes, J., Finlay, S. E., Garrasi, D., Gómez, M., Haahtela, T., Halwani, R., Hassani, Y., Mahboub, B., Marks, G. B., Michelozzi, P., Montagni, M., Nunes, C., Oh, J. J. W., Popov, T. A., Portnoy, J., Ridolo, E., Rosário, N.,

Rottem, M., Sánchez-Borges, M., Sibanda, E., Sienra-Monge, J. J., Vitale, C., & Annesi-Maesano, I. (2015). Meteorological conditions, climate change, new emerging factors, and asthma and related allergic disorders. A statement of the World Allergy Organization. *World Allergy Organization Journal*, 8(1), 1–52.

Delamater, P. L., Finley, A. O., & Banerjee, S. (2012). An analysis of asthma hospitalizations, air pollution, and weather conditions in Los Angeles County, California. *Science of The Total Environment*, 425, 110–118.

Delfino, R. J., Wu, J., Tjoa, T., Gullesserian, S. K., Nickerson, B., & Gillen, D. L. (2014). Asthma Morbidity and Ambient Air Pollution: Effect Modification by Residential Traffic-Related Air Pollution. *Epidemiology*, 25(1), 48–57.

Desouza, K. C. & Flanery, T. H. (2013). Designing, planning, and managing resilient cities: A conceptual framework. *Cities*, 35, 89–99.

Dye, C. (2018). Expanded health systems for sustainable development. *Science*, 359(6382), 1337–1339.

Ebi, K. L. & McGregor, G. (2008). Climate Change, Tropospheric Ozone and Particulate Matter, and Health Impacts. *Environmental Health Perspectives*, 116(11), 1449–1455.

Feng, X. & Astell-Burt, T. (2017). Is Neighborhood Green Space Protective against Associations between Child Asthma, Neighborhood Traffic Volume and Perceived Lack of Area Safety? Multilevel Analysis of 4447 Australian Children. *International Journal of Environmental Research and Public Health*, 14(5), 543.

Ferraro, P. J., Sanchirico, J. N., & Smith, M. D. (2019). Causal inference in coupled human and natural systems. *Proceedings of the National Academy of Sciences*, 116(12), 5311–5318.

Fleming, L., Leonardi, G., White, M., Medlock, J., Alcock, I., Macintyre, H., Maguire, K., Nichols, G., Wheeler, B., Morris, G., Taylor, T., Hemming, D., Iacono, G., Gillingham, E.,

Hansford, K., Heaviside, C., Bone, A., & Duarte-Davidson, R. (2018). Beyond Climate Change and Health: Integrating Broader Environmental Change and Natural Environments for Public Health Protection and Promotion in the UK. *Atmosphere 2018, Vol. 9, Page 245*, 9(7), 245.

Ford, E. S. & Mannino, D. M. (2010). Considerations Regarding the Epidemiology and Public Health Burden of Asthma. In A. Harver & H. Kotses (Eds.), *Asthma, Health and Society: A Public Health Perspective* (pp. 3–17). Boston, MA: Springer US.

Frank, D. M. (2021). What is the environment in environmental health research? Perspectives from the ethics of science. *Studies in History and Philosophy of Science Part A*, 88, 172–180.

Gabry, J., Simpson, D., Vehtari, A., Betancourt, M., & Gelman, A. (2019). Visualization in Bayesian workflow. *Journal of the Royal Statistical Society: Series A (Statistics in Society)*, 182(2), 389–402.

Gee, G. C. & Payne-Sturges, D. C. (2004). Environmental Health Disparities: A Framework Integrating Psychosocial and Environmental Concepts. *Environmental Health Perspectives*, 112(17), 1645–1653.

Gelman, A., Carlin, J. B., Stern, H. S., Dunson, D. B., Vehtari, A., & Rubin, D. B. (2014). *Bayesian Data Analysis*. CRC press.

Gelman, author, A. (2021). *Regression and Other Stories*. New York, NY, U.S.A.: Cambridge University Press.

Gillies, R. R. & Carlson, T. N. (1995). Thermal remote sensing of surface soil water content with partial vegetation cover for incorporation into climate models. *Journal of Applied Meteorology*, 34(4), 745–756.

Global Initiative for Asthma (2022). *Global Strategy for Asthma Management and Prevention*. Technical report, Global Initiative for Asthma.

Gotham, K. F. (2014). *Race, Real Estate, and Uneven Development : The Kansas City Experience, 1900-2010*. Albany : State University of New York Press, second edition. edition.

Hess, J. J., Eidson, M., Tlumak, J. E., Raab, K. K., & Luber, G. C. (2014). An evidence-based public health approach to climate change adaptation. *Environmental Health Perspectives*, 122(11), 1177–1186.

Heynen, N., Perkins, H. A., & Roy, P. (2006). The Political Ecology of Uneven Urban Green Space. *Urban Affairs Review*, 42(1), 3–25.

Hoffman, J. S., Shandas, V., & Pendleton, N. (2020). The Effects of Historical Housing Policies on Resident Exposure to Intra-Urban Heat: A Study of 108 US Urban Areas. *Climate*, 8(1), 12.

Hooper, P., Boulange, C., Arciniegas, G., Foster, S., Bolleter, J., & Pettit, C. (2021). Exploring the potential for planning support systems to bridge the research-translation gap between public health and urban planning. *International Journal of Health Geographics*, 20(1), 36.

Hu, Y., Cheng, J., Jiang, F., Liu, S., Li, S., Tan, J., Yin, Y., & Tong, S. (2020). Season-stratified effects of meteorological factors on childhood asthma in Shanghai, China. *Environmental Research*, 191, 110115.

Janhäll, S. (2015). Review on urban vegetation and particle air pollution – Deposition and dispersion. *Atmospheric Environment*, 105, 130–137.

Jennings, V., Reid, C. E., & Fuller, C. H. (2021). Green infrastructure can limit but not solve air pollution injustice. *Nature Communications*, 12(1), 4681.

Ji, W. (2008). Landscape effects of urban sprawl: Spatial and temporal analyses using remote sensing images and landscape metrics. *The International Archives of the Photogrammetry, Remote Sensing and Spatial Information Sciences*, XXXVII(B7), 1691–1694.

Jia, P., Lakerveld, J., Wu, J., Stein, A., Root, E. D., Sabel, C. E., Vermeulen, R., Remais, J. V., Chen, X., Brownson, R. C., Amer, S., Xiao, Q., Wang, L., Verschuren, W. M. M., Wu, T.,

Wang, Y., & James, P. (2019). Top 10 Research Priorities in Spatial Lifecourse Epidemiology. *Environmental Health Perspectives*, 127(7), 074501.

Kane, N. (2022). Revealing the racial and spatial disparity in pediatric asthma: A Kansas City case study. *Social Science & Medicine*, 292, 114543.

Kane, N. J. (2020). *An Interdisciplinary Health Disparities Research and Intervention Strategy Applied to the Problem of Pediatric Asthma in Kansas City*. PhD thesis, University of Missouri - Kansas City, United States – Missouri.

Kim, J., Lim, Y., & Kim, H. (2014). Outdoor temperature changes and emergency department visits for asthma in Seoul, Korea: A time-series study. *Environmental Research*, 135, 15–20.

Kim, S.-Y., Bechle, M., Hankey, S., Sheppard, L., Szpiro, A. A., & Marshall, J. D. (2020). Concentrations of criteria pollutants in the contiguous U.S., 1979 – 2015: Role of prediction model parsimony in integrated empirical geographic regression. *PLOS ONE*, 15(2), e0228535.

Kramer, D. B., Hartter, J., Boag, A. E., Jain, M., Stevens, K., Nicholas, K. A., McConnell, W. J., & Liu, J. (2017). Top 40 questions in coupled human and natural systems (CHANS) research. *Ecology and Society*, 22(2), 44.

Kruschke, J. K. (2015). *Doing Bayesian Data Analysis: A Tutorial with R, JAGS, and Stan*. Academic Press, second edition edition.

Kruschke, J. K. (2021). Bayesian Analysis Reporting Guidelines. *Nature Human Behaviour*, 5(10), 1282–1291.

Kruschke, J. K. & Liddell, T. M. (2018). Bayesian data analysis for newcomers. *Psychonomic Bulletin & Review*, 25(1), 155–177.

Kumar, P., Druckman, A., Gallagher, J., Gatersleben, B., Allison, S., Eisenman, T. S., Hoang, U., Hama, S., Tiwari, A., Sharma, A., Abhijith, K. V., Adlakha, D., McNabola, A., Astell-Burt, T.,

Feng, X., Skeldon, A. C., de Lusignan, S., & Morawska, L. (2019). The nexus between air pollution, green infrastructure and human health. *Environment International*, 133, 105181.

Lam, H. C.-y., Li, A. M., Chan, E. Y.-y., & Goggins, W. B. (2016). The short-term association between asthma hospitalisations, ambient temperature, other meteorological factors and air pollutants in Hong Kong: A time-series study. *Thorax*, 71(12), 1097–1109.

Lawrence, R. J., Gatzweiler, F. W., & Gatzweiler, F. W. (2017). Wanted: A Transdisciplinary Knowledge Domain for Urban Health. *J Urban Health*, 94, 592–596.

Lei, X., Liu, L., Chen, R., Liu, C., Hong, J., Cao, L., Lu, Y., Dong, X., Chen, X., Qiu, X., Xia, M., Ding, B., Qian, L., Wang, L., Zhou, W., Gui, Y., Kan, H., Zhou, Y., & Zhang, X. (2021). Temperature changes between neighboring days and childhood asthma: A seasonal analysis in Shanghai, China. *International Journal of Biometeorology*, 65(6), 827–836.

Lemoine, N. P. (2019). Moving beyond noninformative priors: Why and how to choose weakly informative priors in Bayesian analyses. *Oikos*, 128(7), 912–928.

Li, S., Batterman, S., Wasilevich, E., Elasaad, H., Wahl, R., & Mukherjee, B. (2011). Asthma exacerbation and proximity of residence to major roads: A population-based matched case-control study among the pediatric Medicaid population in Detroit, Michigan. *Environmental Health*, 10(1), 34.

Liu, J., Dietz, T., Carpenter, S. R., Alberti, M., Folke, C., Moran, E., Pell, A. N., Deadman, P., Kratz, T., Lubchenco, J., Ostrom, E., Ouyang, Z., Provencher, W., Redman, C. L., Schneider, S. H., & Taylor, W. W. (2007a). Complexity of Coupled Human and Natural Systems. *Science*, 317(5844), 1513–1516.

Liu, J., Dietz, T., Carpenter, S. R., Folke, C., Alberti, M., Redman, C. L., Schneider, S. H., Ostrom, E., Pell, A. N., Lubchenco, J., Taylor, W. W., Ouyang, Z., Deadman, P., Kratz, T., & Provencher, W. (2007b). Coupled human and natural systems. *Ambio*, 36(8), 639–649.

Lowe, M., Adlakha, D., Sallis, J. F., Salvo, D., Cerin, E., Moudon, A. V., Higgs, C., Hinckson, E., Arundel, J., Boeing, G., Liu, S., Mansour, P., Gebel, K., Puig-Ribera, A., Mishra, P. B., Bozovic, T., Carson, J., Dygrýn, J., Florindo, A. A., Ho, T. P., Hook, H., Hunter, R. F., Lai, P.-C., Molina-García, J., Nitvimol, K., Oyeyemi, A. L., Ramos, C. D. G., Resendiz, E., Troelsen, J., Witlox, F., & Giles-Corti, B. (2022). City planning policies to support health and sustainability: An international comparison of policy indicators for 25 cities. *The Lancet Global Health*, 10(6), e882–e894.

Lynch, S. M. (2007). *Introduction to Applied Bayesian Statistics and Estimation for Social Scientists*. Springer Science & Business Media.

McPhearson, T., Parnell, S., Simon, D., Gaffney, O., Elmqvist, T., Bai, X., Roberts, D. C., & Revi, A. (2016). Scientists must have a say in the future of cities. *Nature*, 538(7624), 165–166.

Mid-America Regional Council (2014). *Fair Housing and Equity Assessment For the Greater Kansas City Region*. Technical report.

Milligan, K. L., Matsui, E., & Sharma, H. (2016). Asthma in Urban Children: Epidemiology, Environmental Risk Factors, and the Public Health Domain. *Current Allergy and Asthma Reports*, 16(4), 33.

Mireku, N., Wang, Y., Ager, J., Reddy, R. C., & Baptist, A. P. (2009). Changes in weather and the effects on pediatric asthma exacerbations. *Annals of Allergy, Asthma & Immunology*, 103(3), 220–224.

Mock, C. N., Smith, K. R., Kobusingye, O., Nugent, R., Abdalla, S., Ahuja, R. B., Apramian, S. S., Bachani, A. M., Bellis, M. A., Butchart, A., Cantley, L., Chase, C., Cullen, M., Dabestani, N., Ebi, K. L., Fang, X., Gururaj, G., Guttikunda, S., Hess, J. J., Hoe, C., Hutton, G., Hyder, A. A., Ivers, R., Jamison, D. T., Jawahar, P., Keay, L., Levin, C., Liao, J., Mackie, D., Malik, K., Meddings, D., Nguyen, N. P., Norton, R., Olson, Z., Partridge, I., Peden, M., Pillarisetti, A., Rahman, F., Rosenberg, M. L., Staples, J. A., Verguet, S., Ward, C. L., & Watkins, D. A. (2017).

Injury Prevention and Environmental Health: Key Messages from Disease Control Priorities, Third Edition. In *Disease Control Priorities, Third Edition (Volume 7): Injury Prevention and Environmental Health* (pp. 1–23).

Nowak, D. J., Crane, D. E., & Stevens, J. C. (2006). Air pollution removal by urban trees and shrubs in the United States. *Urban Forestry & Urban Greening*, 4(3-4), 115–123.

Oke, T. R. (1982). The energetic basis of the urban heat island. *Quarterly Journal of the Royal Meteorological Society*, 108(455), 1–24.

O'Lenick, C. R., Winquist, A., Chang, H. H., Kramer, M. R., Mulholland, J. A., Grundstein, A., & Sarnat, S. E. (2017). Evaluation of individual and area-level factors as modifiers of the association between warm-season temperature and pediatric asthma morbidity in Atlanta, GA. *Environmental research*, 156, 132–144.

Perperoglou, A., Sauerbrei, W., Abrahamowicz, M., & Schmid, M. (2019). A review of spline function procedures in R. *BMC Medical Research Methodology*, 19(1), 46.

Pickett, S. T. A., Cadenasso, M. L., Grove, J. M., Nilon, C., Pouyat, R., Zipperer, W. C., & Costanza, R. (2001). Urban Ecological Sysytems: Linking Terrestrial Ecological , Physical, and Socioeconomic Components of Metropolitan Areas. *Annual Review of Ecology and Systematics*, 32(2001), 127–157.

Pineo, H., Zimmermann, N., & Davies, M. (2020). Integrating health into the complex urban planning policy and decision-making context: A systems thinking analysis. *Palgrave Communications*, 6(1), 21.

Pollock, J., Shi, L., & Gimbel, R. W. (2017). Outdoor Environment and Pediatric Asthma: An Update on the Evidence from North America. *Canadian Respiratory Journal*, 2017, 1–16.

Prüss-Ustün, A, Wolf, J, Corvalán, C, Bos, R, & Neira, M (2019). *Preventing Disease through*

*Healthy Environments: A Global Assessment of the Burden of Diesase from Environmental Risks*. Technical report, World Health Organization, Geneva.

R Core Team (2020). *R: A Language and Environment for Statistical Computing*. R Foundation for Statistical Computing, Vienna, Austria.

Raun, L. H., Ensor, K. B., & Persse, D. (2014). Using community level strategies to reduce asthma attacks triggered by outdoor air pollution: A case crossover analysis. *Environmental Health*, 13(1), 58.

Rohat, G., Wilhelmi, O. V., Flacke, J., Monaghan, A. J., Gao, J., Dao, H., & van Maarseveen, M. (2019). Characterizing the role of socioeconomic pathways in shaping future urban heat-related challenges. *Science of The Total Environment*, 695, 133941.

Rydin, Y., Bleahu, A., Davies, M., Dávila, J. D., Friel, S., De Grandis, G., Groce, N., Hallal, P. C., Hamilton, I., Howden-Chapman, P., Lai, K.-M., Lim, C. J., Martins, J., Osrin, D., Ridley, I., Scott, I., Taylor, M., Wilkinson, P., & Wilson, J. (2012). Shaping cities for health: Complexity and the planning of urban environments in the 21st century. *Lancet*, 379(9831), 2079–2108.

Samuels-Kalow, M. E. & Camargo, C. A. (2019). The Use of Geographic Data to Improve Asthma Care Delivery and Population Health. *Clinics in Chest Medicine*, 40(1), 209–225.

Schlosberg, D. (2013). Theorising environmental justice: The expanding sphere of a discourse. *Environmental Politics*, 22(1), 37–55.

Schulz, A. J., Mentz, G. B., Sampson, N., Ward, M., Anderson, R., de Majo, R., Israel, B. A., Lewis, T. C., & Wilkins, D. (2016). RACE AND THE DISTRIBUTION OF SOCIAL AND PHYSICAL ENVIRONMENTAL RISK. *Du Bois Review: Social Science Research on Race*, 13(2), 285–304.

Smith, A. & Laribi, O. (2021). Environmental Justice in the American Public Health Context:

Trends in the Scientific Literature at the Intersection Between Health, Environment, and Social Status. *Journal of Racial and Ethnic Health Disparities*.

Social Explorer (2022a). *Age - Cumulative (Less)*. Technical report, Social Explorer, New York City, NY.

Social Explorer (2022b). *Ratio of Income in 1999 to Poverty Level*. Technical report, Social Explorer, New York City, NY.

Soneja, S., Jiang, C., Fisher, J., Upperman, C. R., Mitchell, C., & Sapkota, A. (2016). Exposure to extreme heat and precipitation events associated with increased risk of hospitalization for asthma in Maryland, U.S.A. *Environmental Health*, 15(1), 57.

Stone, B., Hess, J. J., & Frumkin, H. (2010). Urban form and extreme heat events: Are sprawling cities more vulnerable to climate change than compact cities? *Environmental Health Perspectives*, 118(10), 1425–1428.

The Urban Insitute (2017). The Cost of Segregation: National Trends and the Case of Chicago, 1990–2010. (pp.7̃8).

Trivedi, M. & Denton, E. (2019). Asthma in Children and Adults—What Are the Differences and What Can They Tell us About Asthma? *Frontiers in Pediatrics*, 7.

Trochim, W. M., Cabrera, D. A., Milstein, B., Gallagher, R. S., & Leischow, S. J. (2006). Practical challenges of systems thinking and modeling in public health. *American Journal of Public Health*, 96(3), 538–546.

United Nations (2019). *World Urbanization Prospects 2018: Highlights*. Technical report, Department of Economic and Social Affairs, Population Division.

United Nations (2021). *Sustainable Development Goals Report 2021*. S.l.: United Nations Department of Economic and Social Affairs.

U.S. Housing and Urban Development (2020). *Affirmatively Furthering Fair Housing Data and Mapping Tool (AFFH-T) Data Documentation*. Technical report, Office of Policy Development & Research.

Walker, K. & Herman, M. (2022). *Tidycensus: Load US Census Boundary and Attribute Data as 'tidyverse' and 'Sf'-Ready Data Frames*.

Wesley, E. J. & Brunsell, N. A. (2019). Greenspace Pattern and the Surface Urban Heat Island: A Biophysically-Based Approach to Investigating the Effects of Urban Landscape Configuration. *Remote Sensing*, 11(19), 2322.

Wickham, H., Averick, M., Bryan, J., Chang, W., McGowan, L. D., François, R., Grolemund, G., Hayes, A., Henry, L., Hester, J., Kuhn, M., Pedersen, T. L., Miller, E., Bache, S. M., Müller, K., Ooms, J., Robinson, D., Seidel, D. P., Spinu, V., Takahashi, K., Vaughan, D., Wilke, C., Woo, K., & Yutani, H. (2019). Welcome to the tidyverse. *Journal of Open Source Software*, 4(43), 1686.

Wikle, C. K. (2003). Hierarchical Models in Environmental Science. *International Statistical Review*, 71(2), 181–199.

Wilhelmi, O. V. & Hayden, M. H. (2010). Connecting people and place: A new framework for reducing urban vulnerability to extreme heat. *Environmental Research Letters*, 5(1), 14–21.

Williams, D. R., Sternthal, M., & Wright, R. J. (2009). Social Determinants: Taking the Social Context of Asthma Seriously. *Pediatrics*, 123(Suppl 3), S174–S184.

Wolch, J. R., Byrne, J., & Newell, J. P. (2014). Urban green space, public health, and environmental justice: The challenge of making cities 'just green enough'. *Landscape and Urban Planning*, 125, 234–244.

Won, Y. K., ho Hwang, T., Roh, E. J., & Chung, E. H. (2016). Seasonal patterns of asthma in

children and adolescents presenting at emergency departments in Korea. *Allergy, Asthma and Immunology Research*, 8(3), 223–229.

World Health Organization (2010). *A Conceptual Framework for Action on the Social Determinants of Health: Debates, Policy & Practice, Case Studies.*