MULTISCALE AND SPATIOTEMPORAL DYNAMICS OF SOCIOECONOMIC AND ENVIRONMENTAL EFFECTS ON MENTAL ILLNESS MORTALITY

by

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DISSERTATION ABSTRACT

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Title: Multiscale and Spatiotemporal Dynamics of Socioeconomic and Environmental Effects on Mental Illness Mortality

Mental illness is a pressing global and national public health concern, necessitating the identification of risk factors to develop effective prevention measures. In this dissertation, I attempt to fill two research gaps by revealing the spatial and/or temporal disparity in the impacts of unemployment and greenspace on mental illness mortality with spatiotemporal modeling and a causal analysis across three spatial scales.

In Chapter 2, the association between mental illness and substance use mortality and unemployment was examined using Bayesian spatiotemporal hierarchical models. The findings revealed heightened positive effects in rural Appalachian and Midwestern counties. Overall mild effects were observed during the Great Recession period. The patterns could be attributed to local contexts such as the availability of healthcare supply and relative deprivation. Chapter 3 challenges the assumption of a spatially constant effect of greenspace exposure on mental illness mortality, using census tract-level data from Oregon and Washington. Results indicated that the impact of greenspace exposure on mental illness mortality varies across census tracts, with protective effects more likely in areas between Seattle and Portland. Protective effects were more likely observed in areas

between Seattle and Portland. The contrast between urban and rural areas was explained through factors such as patient preference and differential availability and accessibility to greenspaces. Chapter 4 shed light on the spatial differences in the causal effects of greenspace exposure on mental illness mortality using data from the State of Washington. Dichotomized treatment settings and propensity score matching methods were leveraged to examine the spatial disparity in causal effects of greenspace exposure to mental illness mortality. The results elucidated that the causal effect differed significantly across regions within Washington state, emphasizing that spatial heterogeneity is a critical element when examining the causal effects of greenspace exposure on mental illness mortality.

By highlighting the spatial and/or temporal disparity in socioeconomic and physical environment factors' effects, this dissertation provides new perspectives to spatiotemporal mental health research and suggests a transition from disease mapping to effect mapping. This transition offers evidence to devise locally-focused measures that consider the spatial disparities of associative and causal effects.

This dissertation includes previously published and unpublished co-authored materials.

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To My Family

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CHAPTER I

INTRODUCTION

1.1 Background

Mental illness is a rising global public health problem. Studies reported mental illness mortality as a major pressure to the increase in overall mortality throughout the world (Campbell, 2010; GBD 2019 Mental Disorders Collaborators, 2022). In the United States, both the incidence and mortality of mental illness have increased in the last two decades (Chang et al., 2010; Fekadu et al., 2015; Jayatilleke et al., 2017; Walker et al., 2015). As a response, the mechanism and etiology of mental illness have been transformed in the literature, where scholars accounted for both compositional and contextual factors to explain mental health outcomes across various spatial scales[1].

Early literature suggested that the prevalence of mental illness notably differs by individual traits. For instance, women were found to be more susceptible to mental illness (Gove, 1972), and there was no evidence of racial differences (Warheit et al., 1975). These studies suggested an alternative perspective for the analysis of mental illness against perspectives emphasizing personal motives alone, although they were based on empirical evidence rather than a theory. Income, sex/gender, and race/ethnicity are often linked to mental illness in the literature (McGilloway et al., 2010; Ridley et al., 2020). Contextual factors have been extensively accounted for by early scholars in ecological studies. These factors are thought to influence personal factors on the onset of mental illness. The list of contextual variables has been expanded, which encompasses residential and

[1]Scale here refers to as a term that includes a nested hierarchy of geographic units.

built environments (Goldsmith et al., 1986), crime (M. White et al., 1987), and socioeconomic status (Gruebner et al., 2017; Leventhal & Brooks-Gunn, 2003).

The spatial and temporal dimensions of emerging compositional and contextual factors have been emphasized in the literature. Processual or incidental factors like events in a life course (e.g., post-stress trauma) highlight the significance of accounting for the temporality to understand mental illness. For instance, longitudinal study designs are usually employed to trace the changing relationship between outcomes and explanatory factors over time. Spatially, collective effects, which include the social network (the impact of the relationship with other people) and social grouping effect (the impact of group identification in individuals), are hypothesized to mediate other factors of mental illness. Environmental factors are actively explored in addition to neighborhood socioeconomic status in mental illness studies. As a surrogate of a specific or an integrated physical environmental feature, environmental factors have been gradually integrated into conceptual frameworks in mental health research (Bratman et al., 2019; Dzhambov, Markevych, Hartig, et al., 2018; Helbich, 2018b; Markevych et al., 2017), and numerous empirical studies supported the integration (Oh et al., 2020; Speldewinde et al., 2009; Van Haaften & Van de Vijver, 1999; F. Wang et al., 2018). Greenspace (e.g., parks and vegetation) and bluespace (e.g., rivers and lakes) exposures are pronounced as the major factors of interest in contemporary mental health research (Bratman et al., 2019; Helbich, 2018a; Labib et al., 2020; Su et al., 2019; M. P. White et al., 2021; World Health Organization, 2021).

The emergence of spatiality as a key element in the study of mental illness risk factors raised a crucial question: What role does geography play in

our understanding of mental illness? The answer to this question hinges on two fundamental insights offered by geography. First, geography underscores that the majority of factors influencing mental health–including the physical environment and almost all contextual factors–vary across space. These factors are therefore spatially organized. Examples of such factors include neighborhood-level income and ethnic composition, which are subject to spatial segregation (Bettencourt et al., 2019; Reardon & Bischoff, 2011). This variability across spatial scales (Catney, 2018) highlights the need of conducting mental health research with different spatial frameworks.

The second insight offered by geography lies in the spatial disparities in mental illness incidence and mortality rates. Such disparities, demarcated by jurisdictional or natural boundaries, bring geographic disparity to the forefront of mental health studies. Understanding these disparities not only helps explain the prevalence of mental illness across various regions but also guides the design of proactive, geographically tailored interventions. Such interventions are important given the growing burden of adverse mental health outcomes and the limited resources available for addressing them.

In recent years, mental health research has shifted from a focus on the spatial disparity in the outcomes (i.e., disease mapping) to an understanding of the spatial disparity in environment factors' impacts on mental health (i.e., effect mapping). This transition enables stakeholders in public mental health policies to identify risk factors specific to certain geographic areas. It has been widely accepted that spatial disparities in mental health outcomes exist at both national and subnational levels, often mirroring socioeconomic gradients (Griffith & Jones, 2020; Philo, 2005). Comparative studies have also demonstrated spatial differences

in mental health outcomes and explanatory factors across countries (Gissler et al., 2013) and subnational regions (Andrilla et al., 2018; Charlesworth et al., 2023; Cortina & Hardin, 2023; Gorski-Steiner et al., 2022; Hudson, 2012; Ma et al., 2009; Maas et al., 2019; Rodero-Cosano et al., 2016; Ryan et al., 2023; Sutarsa et al., 2021). Given this context, it becomes imperative to ask whether spatial disparities in the impacts of risk factors on mental illness remain consistent over time. This question is especially relevant for factors exhibiting high temporal volatility or variation. Exploring the spatial patterns in mental illness has been common in research, employing measures such as spatial autocorrelation indices and spatial clustering analyses (Grigoroglou et al., 2020; Ngui et al., 2013). However, the research is noticeably sparse on investigations of spatial disparities in the effect of risk factors across different spatial scales. Only a few studies hypothesized the spatially varying effects of selected factors including mental health service utilization (Law & Perlman, 2018), poverty, insufficient sleep, marital status (Yankey et al., 2021), and health behavior (Choi & Kim, 2017), where the first two studies were done at a small area level in a city scale, underscoring the need for further evidence on spatial disparity at larger spatial scales.

Regarding the spatiotemporal analysis of mental illness, two significant research gaps stand out. The first is the lack of attention given to the interaction between spatial and temporal dimensions in effects. While recent years have seen an increased availability of longitudinal data on mental health outcomes and explanatory factors, researchers have yet to fully leverage this data to understand the spatiotemporal interaction that affect mental health outcomes. There is a pressing need for modeling approaches that capture spatially varying effects of contributing factors (Janko et al., 2019; Labib et al., 2020; Shin et al., 2020). The

second gap lies in our understanding of the spatiality of causal effects in mental illness research. While there has been growing interest in establishing causal relationships between different factors and mental illness incidence and mortality (Ridley et al., 2020; Uher & Zwicker, 2017), the role of spatial variations in these effects remains largely unexplored. Thus, future research must aim to bridge these gaps by focusing on the spatiotemporal interplay of factors influencing mental health outcomes and investigating the spatially varying causal effects on mental illness. Such studies will contribute to expanding our understanding of mental illness, providing valuable insights to design effective interventions.

1.2 Aims and structure of the dissertation

The primary goal of this dissertation is to address two crucial gaps identified in current mental health research. I attempt to accomplish the goals by three approaches: scrutinizing the spatial and temporal disparities in the influence of unemployment and greenspace exposure on mental illness mortality, performing this analysis at different spatial scales, and extending the analysis to infer causal effects beyond mere associations. The dissertation can be conceptualized as an exploration of the spatiotemporal dynamics of socio-environmental factors and their associations and causal impacts on mental illness mortality through three case studies across different spatial scales: counties, census tracts, and individual residential locations. This approach offers an integrated and comprehensive understanding of the relationship between mental health outcomes and socio-environmental factors.

Unemployment rates and greenspace exposure, the primary factors of interest of this dissertation, are considered significant contributing factors of mental health outcomes (Bartley, 1994; Barton & Rogerson, 2017; Collins et al., 2020;

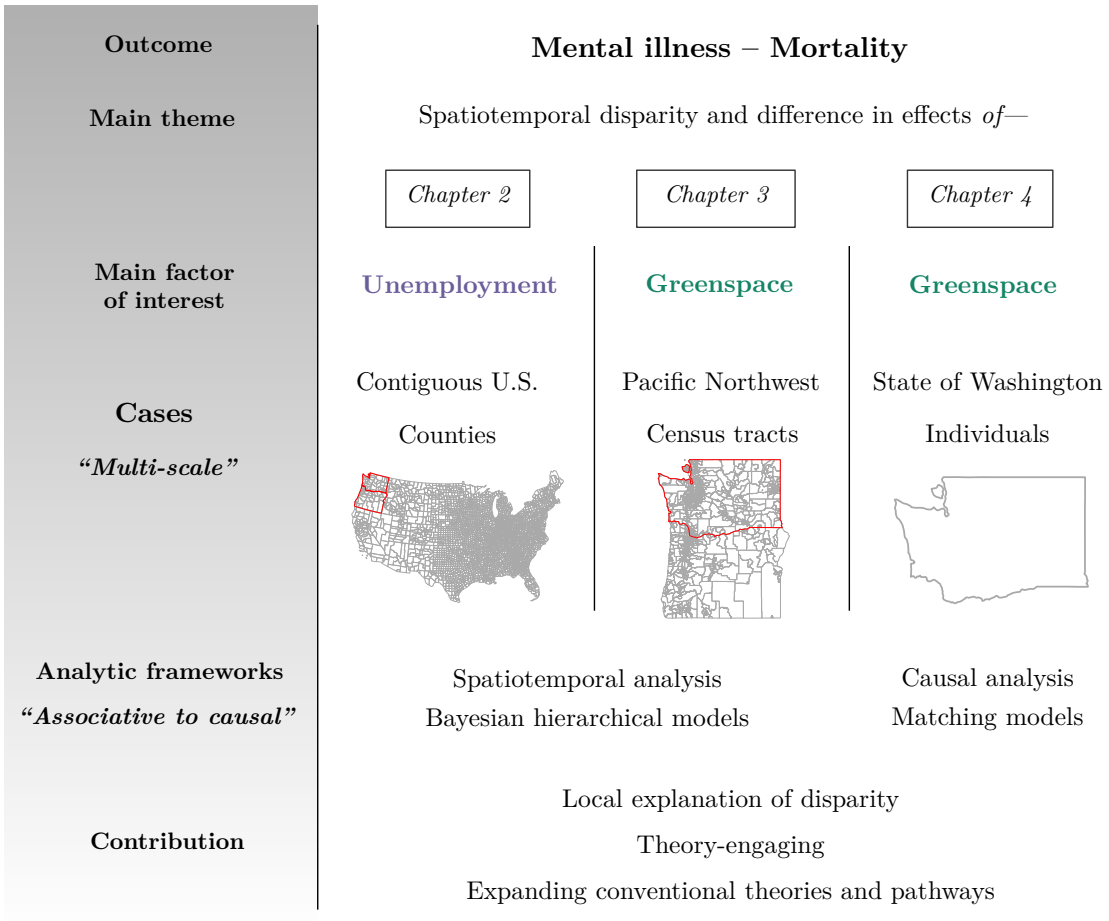
Marazziti et al., 2021; Uutela, 2010; Virgolino et al., 2022; Wan et al., 2022). The effects of these variables have been assumed to be constant across spatial entities within a single spatial framework. I challenge this assumption by proposing that these variables have spatially and temporally varying effects on mental illness. To reveal the spatial disparity in these associative and causal effects, the scope of the investigation is progressively narrowed down to focus on finer levels of spatial scale.

Chapter 2 presents a nationwide, county-level analysis of mental illness and substance abuse mortality rates, with a particular focus on the spatiotemporally varying effects of unemployment rates from 2001 to 2014. This fourteen-year span, encompassing two significant economic recessions including the Great Recession, provides a good case to trace the temporal shift in the spatial pattern of unemployment's impact on mental illness mortality. In Chapter 3, the analysis narrows down to a regional level, where I will examine the impact of greenspace exposure on mental illness mortality in the States of Oregon and Washington at the census tract level. This chapter employs spatiotemporal hierarchical linear models to analyze spatial variation in the effects of greenspace exposure, accounting for excess zeros in the aggregated mental illness mortality dataset. Chapter 4 takes the analysis a step further by attempting to estimate causal effects. It focuses on individual-level mental illness mortality data, using observational causal inference approaches (i.e., matching methods) to explore the spatial difference in the causal effect of greenspace exposure (defined as the average vegetation index value in a 15-minute walking area along the road network) on mental illness mortality. This chapter also uncovers challenges associated with estimating causal effects across multiple regions, particularly regarding the conditions for dichotomization to define the treated and controlled group from continuous greenspace exposure. The

diagnosis is conducted based on the theoretical conditions of equivalence suggested by Stitelman et al. (2010) (Stitelman et al., 2010).

In sum, this dissertation provides a comprehensive examination of the spatial disparities in the associative and causal effects of unemployment and greenspace exposure on mental illness mortality. Such examination enriches, corroborates, and challenges existing theories seeking a universal explanation for the effects of these factors on mental health (Figure 1). By investigating the local contexts that shape these effects, the dissertation contributes to a deeper understanding of the interplay between mental health outcome and socio-environmental factors.

Figure 1. Research flow and organization of this dissertation



CHAPTER II

THE SPATIALLY AND TEMPORALLY VARYING ASSOCIATION BETWEEN MENTAL ILLNESS AND SUBSTANCE USE MORTALITY AND UNEMPLOYMENT: A BAYESIAN ANALYSIS IN THE CONTIGUOUS UNITED STATES, 2001–2014

This chapter is based on the published work in volume 140 of the journal *Applied Geography* in March 2022. I chose the topic, collected data, conducted statistical analysis, and wrote the draft; Dr. Hui Luan, the dissertation chair and my advisor, supervised the statistical analysis and revised the draft. This chapter is a revision of the original work.

2.1 Introduction

The Great Recession led to a global deterioration of mental health status (Bacigalupe et al., 2016; Margerison-Zilko et al., 2016). During this time, mental illness mortality rates increased the United States, reaching a peak of 49.5 per 100,000 population in 2013 (CDC, 2021). The unemployment surge during the Great Recession reignited a broad scholarly inquiry to examine the impact of unemployment on mental health with its variable relationship during economic recessions (De Vogli et al., 2014; Frasquilho et al., 2015; Haaland & Telle, 2015; Norström & Grönqvist, 2015). Evidence suggested a clear negative association between mental health and unemployment, leading to increased substance use mortality and decreased life quality and satisfaction (Bartelink et al., 2020; Junna et al., 2020; Mousteri et al., 2018; Murphy & Athanasou, 1999; Paul & Moser, 2009). These effects seem more profound in early life stages, where unemployment

has been linked to heightened cigarette use (Lee et al., 2015), high-risk alcohol use (Henkel, 2011), and general substance use (Compton et al., 2014).

Few large-scale ecological studies have accounted for spatial variations in these associations, while an extensive number of individual-level studies indicated variability in the impact of unemployment on mortality across countries. The ecological studies attributed such variability to the difference in labor policy and workforce skills (McLeod et al., 2012; Norström & Grönqvist, 2015; van Lenthe, 2005). Despite suggesting spatial difference in the impacts, these studies assumed a spatially constant impact of unemployment on mental health outcomes, which may overlook the spatial autocorrelation of both mortality and unemployment rates (Halliday, 2014; Lorant et al., 2001; Molho, 1995; Patacchini & Zenou, 2007). Given these disparities, a spatially explicit approach is required to fully understand how local contexts may influence the unemployment and mental health relationship (Heutel & Ruhm, 2016; Sameem & Sylwester, 2017; Shoff et al., 2012; Trgovac et al., 2015)

Informed by these gaps in the literature, this chapter poses two key research questions. First, how does the relationship between mental illness, substance use (MISU) mortality, and unemployment vary across U.S. counties over time? Second, how has unemployment impacted MISU mortality spatiotemporally during the 2001 and 2008 economic recessions? By utilizing a multilevel regression model with spatiotemporal components, I seek to demonstrate how this relationship evolves across space and time. These research questions not only address the ongoing debate about the effect of economic recessions on MISU mortality rates, but also explore whether these impacts manifest differently at a county level given spatiotemporal variations in both mortality and unemployment rates. Furthermore,

this study revisits the claim that mortality rates decrease during recessions while shifting the focus from the mortality rates to the effect of unemployment rates on them. The study period, encompassing two economic recessions of 2001 and 2007, provides good cases to answer the research questions (Ariizumi & Schirle, 2012; Haaland & Telle, 2015; Miller et al., 2009; Neumayer, 2004; Ruhm, 2000; The National Bureau of Economic Research, 2020).

2.2 Methods

2.2.1 Data.

2.2.1.1 Mortality data. The primary outcome of interest is the county-level Mental Illness and Substance Use (MISU) mortality rates provided by the Institute for Health Metrics and Evaluation (IHME) (Institute for Health Metrics and Evaluation, 2016). These rates are cause-specific and age-standardized from 1980 to 2014 across twenty-one categories. The original work's authors categorized the raw mortality data by reclassifying codes in the International Classification of Diseases 9^{th} and 10^{th} (Dwyer-Lindgren et al., 2016). Given the consistency of county-level boundaries in the contiguous United States throughout the study period, the use of this dataset avoids potential misalignment problems (Gryparis et al., 2009). MISU were grouped into a single category due to confidentiality constraints associated with the small number of MISU deaths in this dataset.

2.2.1.2 Covariate data. The key explanatory variable is the unemployment rate, which is operationalized as the one-year lagged annual average unemployment rate. This one-year lag was chosen to assess the short-term impact of unemployment on MISU mortality rates. The unemployment data was sourced from the Local Area Unemployment dataset, which has been tracking monthly

unemployment rates for all U.S. counties since 1976 (U.S. Bureau of Labor Statistics, 2019).

Eight covariates were collected from the IPUMS National Historical Geographical Information System to control for potential confounding factors affecting MISU mortality (Manson et al., 2019; Ruggles, 2014). These were selected based on their relevance to MISU mortality as identified in previous ecological studies and other research that linked neighborhood socioeconomic status to MISU (Ballester et al., 2019; Catalano et al., 2011; Goldsmith et al., 1998; Jia et al., 2009; Kloos & Townley, 2011; Monnat, 2018; Moortel et al., 2018; Ridley et al., 2020; Silver et al., 2002; van den Berg et al., 2017). The covariates represent three indicators representing county-level economic status: median household income (adjusted to the year 2000 values using the consumer price index for consistency), renter-occupied housing ratios, and the ratio of households under the poverty line. Factors were included such as the elderly population, considered protective against MISU disorders (Silver et al., 2002), higher educational attainment, often associated with a protective effect against MISU (Goldsmith et al., 1998; Melchior et al., 2015; Seabury et al., 2019), ethnic composition represented by the ratio of the non-white population, and other demographic conditions like the ratio of the rural population and the ratio of single-person households (Solmi et al., 2017). Since each year's data contained eight variables from two decennial census during the study period, the 2001-2009 mortality rates were matched to the covariates of the 2000 Census and the 2010-2014 rates to the 2010 Census covariates.

It was found that the pairwise correlations of the covariates range between -0.727–-0.670, with only 5 (out of 36) having absolute values greater than 0.5. The variance inflation factors were well below 10, indicating no severe

multicollinearity (James et al., 2021). Thus, there was no need to compute a composite socioeconomic index using these covarites. County boundaries in 2014 were collected from the Census Bureau (U.S. Census Bureau, 2015). Some covariates, however, were incomplete due to small population sizes or confidentiality issues. Literature suggests that when the missing rate is less than 3 %, the imputation accuracy is acceptable even when employing a simple imputation method (Harrell, 2015). As such, I imputed missing values with the global median, given all the covariates had missing rates less than 1 %.

2.2.2 Statistical analysis. A set of spatiotemporal regression models were implemented to explore the spatial variation in the impact of unemployment rates on MISU mortality over time. The set was identified by adapting a hierarchical linear model with a spatiotemporal interaction component (Blangiardo & Cameletti, 2015, p. 240). Among four types of spatiotemporal interactions (often expressed with roman numerals I–IV), the interaction with the higher type order reflects the more complex relationship between the spatial and temporal components than the lower ones. I compared four types of spatiotemporal interaction terms in the random slope (Banerjee et al., 2015; Blangiardo & Cameletti, 2015; Haining & Li, 2020; Knorr-Held, 2000). The type-IV interaction models that the impact of the unemployment rate on MISU mortality rate in one county is similar to its spatial and temporal neighbors. Thus, the present year's MISU mortality rate is explained by the unemployment rates of neighboring counties and the previous year (Khana et al., 2018). The spatial, temporal, and spatiotemporal interaction terms were incrementally included in the random slope of unemployment rates, which is numbered from Model 1 to 5 (Table 1). Model 5 includes all components in the model, where the spatiotemporal interaction term

has four types, which were numbered with Models 5-I to Model 5-IV. The most comprehensive model, Model 5-IV, is specified in Formula 2.1.

Table 1. Model specifications of the baseline model (Model 1) and random effects with and without spatiotemporal interaction in random slopes (Models 2–5)

Model	Fixed and random main effects	Random slope components in β_{1it}		
		Spatial	Temporal	Spatiotemporal*
1				
2		$u_{1i\cdot}$		
3	$\beta_0 + \sum_{k=1}^{9} \beta_k x_{kit} + u_i + v_i + \gamma_t + \varepsilon_{it}$		$\gamma_{1\cdot t}$	
4		$u_{1i\cdot}$	$\gamma_{1\cdot t}$	
5		$u_{1i\cdot}$	$\gamma_{1\cdot t}$	I–IV (RW(1)**)

* Interaction type (assumption for temporal grouping). ** First-order random walk.

$$
\begin{aligned}
& y_{it} = \beta_0 + \beta_{1it} x_{1it} + \textstyle\sum_{k=1}^{9} \beta_k x_{kit} + u_i + v_i + \gamma_t + \varepsilon_{it} \\
& \beta_k \sim \mathcal{N}(0, \tau_{\beta_k}^{-1}) \\
& u_i \sim \mathrm{ICAR}(\mathbf{W}_S, \tau_u^{-1}),\ v_i \sim \mathcal{N}(0, \tau_v^{-1}),\ \varepsilon_{it} \sim \mathcal{N}(0, \tau_\varepsilon^{-1}) \\
& \gamma_t = \rho_0 \gamma_{t-1} + \varepsilon_{t0},\ \rho_0 \sim \mathcal{N}(0, 0.15),\ \varepsilon_{t0} \sim \mathcal{N}(0, \tau_{\varepsilon_0}^{-1}) \\
& \beta_{1it} = u_{1i\cdot} + \gamma_{1\cdot t} + \delta_{1it} \\
& u_{1i\cdot} \sim \mathrm{ICAR}(\mathbf{W}_S, \tau_{u_1}^{-1}) \\
& (\gamma_{1\cdot t} - \gamma_{1\cdot(t-1)}) \sim \mathcal{N}(0, \tau_{\gamma_1}^{-1}) \\
& \boldsymbol{\Delta} \sim \mathcal{N}(\mathbf{0}, \tau_\delta^{-1}[(\mathbf{D} - \mathbf{W}_S) \otimes \mathbf{W}_T])
\end{aligned}
\tag{2.1}
$$

y_{it} denotes the MISU mortality rate in county i and year t by including a spatiotemporal random slope of one-year lagged unemployment rate at each county i and year t, β_{1it}. The mortality rates were log-transformed for assuming the Gaussian likelihood. Thus, β_{1it} is exponentiated for interpretation. For example,

when a model yields $\beta_{1it} = 0.05$, it means that one standard deviation (2.78 %) increase of one-year lagged unemployment rate is associated with 5.1 % increase in MISU mortality rate ($\exp(0.05) - 1 = 1.051 - 1 = 0.051$). The county-level unemployment rate effect is modeled to have a random slope coefficient, that is, the sum of a spatiotemporally structured part δ_{1it}. The type-IV spatiotemporal interaction (δ_{1it}, $\mathbf{\Delta}$ in Formula 2.1 denotes the vector of δ_{1it}) is expressed in Formula 2.1. It models the spatiotemporal interaction by computing a Kronecker product of $(\mathbf{D} - \mathbf{W}_S)$ and the temporal weight matrix $\mathbf{W}_T$ to integrate the spatial intrinsic conditional autoregressive (ICAR) and temporal first-order random walk (RW1) processes (see Appendix A for an example) (Vicente et al., 2020; Williams et al., 2019). The term $\mathbf{D}$ denotes a diagonal matrix with the sum of each row's elements ($\text{diag}(\sum_j w_{Sij})$), where w_{Sij} is the (i, j) element of $\mathbf{W}_S$. The fixed effects of other covariates are denoted as β_k $(k = 2, \cdots, 9)$.

Known as the Besag-York-Mollié (BYM) model (Besag et al., 1991), u_i and v_i separate spatially structured (i.e., ICAR) and exchangeable effects for every county. The spatially structured effect depends on the distribution of adjacent spatial units. For the spatial structure, counties sharing at least a vertex are considered as neighbors (i.e., first-order Queen's contiguity). γ_t is the temporal effect that incorporates temporally autocorrelated functions by specifying a RW1 structure. The random slope term β_{1it} consists of the spatially structured $u_{1i\cdot}$, the temporally structured $\gamma_{1\cdot t}$, and the spatiotemporal interaction term δ_{1it}.

Four types of spatiotemporal interactions in β_{1it} were compared to identify the term that fits the dataset best (Blangiardo & Cameletti, 2015; Haining & Li, 2020). The models were implemented with the Integrated Nested Laplace Approximation (INLA) approach. The INLA method assumes the latent Gaussian

distributions to estimate marginal posteriors, which is known to be effective in large data analysis (Rue et al., 2017). Models were compared with the Deviation Information Criterion (DIC) (Spiegelhalter et al., 2002) and the Watanabe-Akaike Information Criterion (WAIC) (Watanabe, 2009). The best-fitting model is the one with the lowest DIC and WAIC values. For hyperpriors, a weakly informative prior, $\log\Gamma(1, 0.00005)$, was used for the log-precision hyperparameters u_i, v_i and γ_t. A prior $\log\Gamma(100, 0.5)$ was applied to $u_{1i\cdot}$ and $\gamma_{1\cdot t}$ based on the assumption that the precision of the random slopes is larger than that of fixed and main random effects. All statistical analyses were performed with `R` 4.1.2 and `R` package `INLA` version 2021.11.01 (R Core Team, 2021; Rue et al., 2009; Verbosio et al., 2017).

2.3 Results

2.3.1 Descriptive analysis. The MISU mortality rates steadily increased during the study period, while unemployment rates exhibited higher variability than mortality rates, with a pronounced increase during the Great Recession (2007–2009). The county-to-county variation in mortality rates was greater than that of unemployment rates, as the county-specific mortality trends diverged from the median trend (Figures A.1–A.3). The temporal trends in MISU highly varied across counties, where some stagnated and others increased rapidly. Given the disparate scales of the covariates, the coefficient of variation (CV) was employed for comparative purposes. The ratios of the non-white and rural population showed the highest CVs, whereas variables such as median household income, the ratio of renter-occupied housing, and the ratio of the elderly population displayed relatively lower CVs (Table 2).

2.3.2 Spatiotemporal regression results. The best-performing model incorporated a type-IV interaction term, yielding the best goodness-of-fit metrics. Among all models with random effects, the full model (5-IV) had the lowest DIC and WAIC values (Table 3). Models incorporating only spatial random effects (Model 2) showed better fit than those including temporal random effects (Model 3). The global impact of the one-year lagged unemployment rate on MISU mortality gradually strengthened from Model 1 to Model 5-IV, with the impact increasing from 0.010 to 0.014. Notably, two covariates changed their coefficient directions: ratios of renter-occupied housing (from -0.004 in Model 1 to 0.008 in Model 5-IV) and single-person households (from -0.025 to 0.005). Meanwhile, two other variables exhibited diminishing impacts on MISU mortality: the coefficients for median household income shifted from -0.036 to -0.019, and the ratio of the elderly population moved from 0.018 to 0.009. The remaining variables

Table 2. Summary statistics of variables in 3,108 counties

Variable	Mean	Median	SD [1] (CV [2])
One-year lagged unemployment rate (%)	6.57	6.00	2.78 (0.42)
Median household income (Inflation-adjusted as of 2000)	35563.51	34085.42	9032.27 (0.25)
Ratio of non-white population (%)	15.76	9.42	16.17 (1.03)
Ratio of the population with or higher than bachelor's degree (%)	17.56	15.44	8.28 (0.47)
Ratio of the population below the poverty level (%)	14.39	13.41	6.31 (0.44)
Ratio of the rural population (%)	49.67	42.87	33.73 (0.68)
Ratio of renter-occupied housing (%)	26.52	25.22	7.60 (0.29)
Ratio of single-person households (%)	25.94	26.03	3.95 (0.15)
Ratio of the elderly population (65 years old or older, %)	15.22	14.85	4.15 (0.27)

[1] Standard deviation; [2] Coefficient of variation.

demonstrated consistent coefficients. The results reported in the next section are derived from the best-fitting model, Model 5-IV.

Table 3. Model fit statistics

Model	DIC	WAIC
1	-96382.50	-96295.92
2	-105528.97	-105591.72
3	-96539.23	-96450.34
4	-105680.51	-105735.67
5-I	-105633.44	-105678.01
5-II	-115598.62	-117514.14
5-III	-105657.35	-105700.21
5-IV	**-119361.94**	**-120751.66**

Note: the lowest value is highlighted.

Three key findings emerge regarding the random slope coefficients of unemployment rates in the spatiotemporal interaction term. First, the random slopes (β_{1it}) displayed the clear spatial variation (Figure 2), contrasting with the spatially constant coefficient of unemployment rates in Model 1 (the base model). The coefficient of one-year lagged unemployment rate in Model 1 was significantly positive (1.01 %, 95 % credible interval: [0.80 %, 1.21 %]) and invariant across all counties, while β_{1it} from Model 5-IV was between -0.666 and 0.479, which are equivalent to -47.63 % and 61.45 % change in MISU mortality rates by one standard deviation (2.78 %) increase of one-year lagged unemployment rate (Table 4). The positive random slope coefficients were found in the counties in Missouri, Illinois, Indiana, Ohio, and Pennsylvania in 2001–2004, among which counties near Cincinnati gradually shifted to the border of West Virginia and Kentucky after

Table 4. Regression coefficients and the 95 % credible intervals (CrI) of fixed and random effects of Models 1 and 5-IV

	Model 1	Model 5-IV
Intercept	2.268*	2.272
	(2.265, 2.272)**	(2.270, 2.275)
One-year lagged unemployment rate (%)	0.010	0.014
	(0.008, 0.012)	(0.011, 0.017)
Random slopes of one-year lagged unemployment rate		−0.666 (minimum)
		(-0.774, -0.557)
		−−0.479 (maximum)
		(0.374, 0.583)
Median household income (Inflation-adjusted as of 2000)	-0.036	-0.019
	(-0.041, -0.031)	(-0.024, -0.012)
Ratio of non-white population (%)	0.042	0.044
	(0.034, 0.050)	(0.035, 0.052)
Ratio of the population with or higher than bachelor's degree (%)	-0.007	-0.019
	(-0.012, -0.002)	(-0.025, -0.014)
Ratio of the population below the poverty level (%)	0.026	0.028
	(0.022, 0.029)	(0.023, 0.032)
Ratio of the rural population (%)	-0.015	-0.015
	(-0.020, -0.008)	(-0.021, -0.008)
Ratio of renter-occupied housing (%)	-0.004	0.008
	(-0.009, 0.001)	(0.001, 0.014)
Ratio of single-person households (%)	-0.025	0.005
	(-0.030, -0.020)	(-0.001, 0.010)
Ratio of the elderly population (65 years old or older, %)	0.018	0.009
	(0.013, 0.022)	(0.003, 0.015)

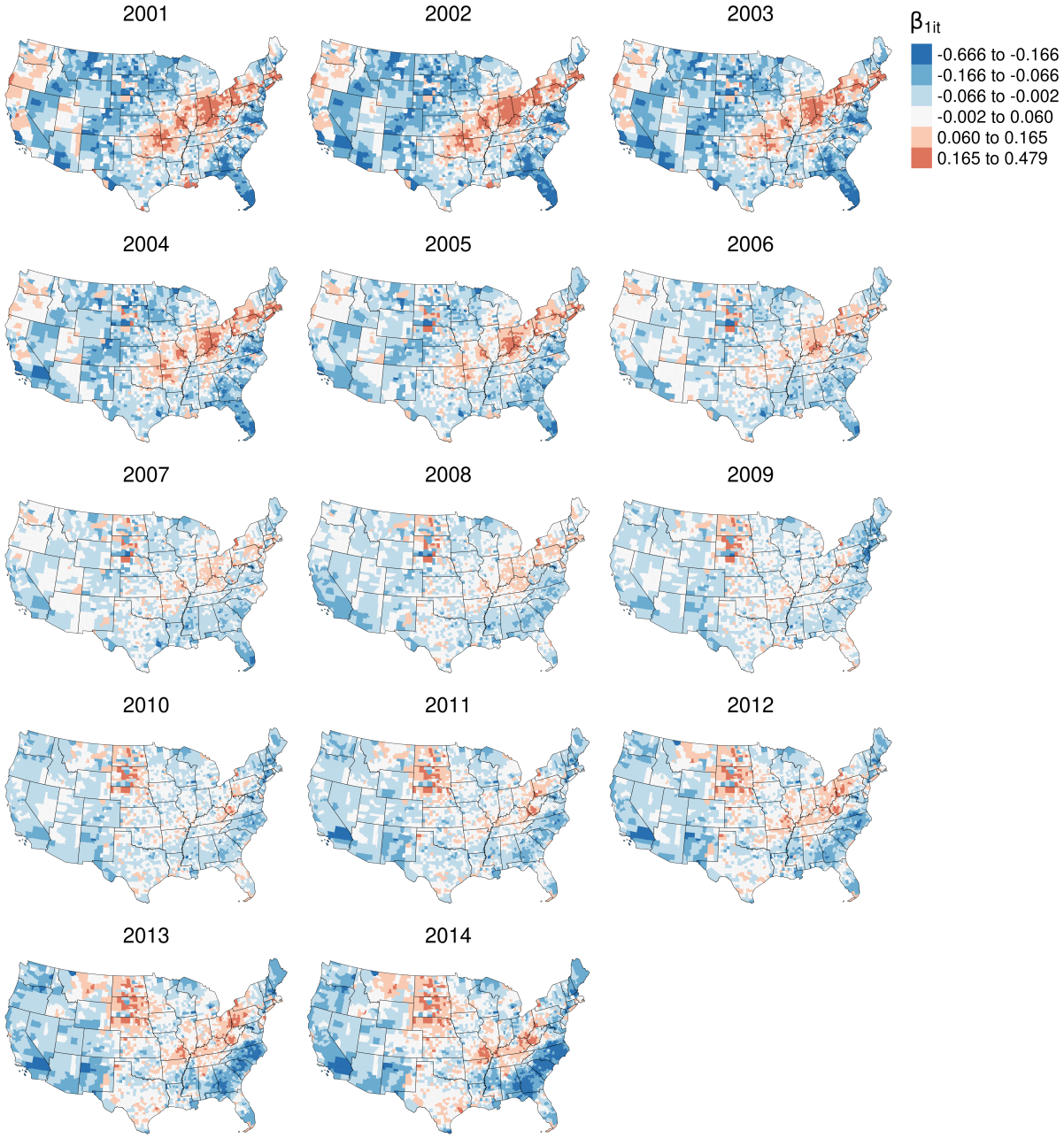
* Mean, ** 95 % CrI; Note: All explanatory variables were standardized.

the Great Recession. Second, regions with strong positive correlations between MISU mortality and unemployment expanded geographically throughout the study period. After Great Recession, an increase in counties with higher positive β_{1it} values was primarily observed in South and North Dakotas. Lastly, the association between MISU mortality and unemployment generally contracted during and in the wake of the Great Recession (2007–2010, between -0.1 and 0.1), compared to other study years (Figure 2). Summary statistics for the posterior distributions of hyperparameters are detailed in Table A.3.

2.4 Discussion

MISU mortality's association with unemployment varied both spatially and temporally. The results demonstrated that the association was dynamic rather than static (Towne et al., 2017). Considering that the study period spanned from 2001 to 2014, it becomes evident that this association underwent considerable changes, adapting and evolving with time and location. Through the period of 2002 to 2003, immediately after the 2001 recession, and again during 2009 to 2011, corresponding with the Great Recession, the unemployment rates rose consistently in most counties (Figure A.2). The manifestation of this trend was evident in the striking shifts seen in the coefficients of some covariates. In comparison to the base model, the best-fitting model yielded significant transformations in both the magnitude and direction of these coefficients. Such alterations in the coefficients underscore the crucial need to take into account spatiotemporal dependencies when analyzing multi-year, areal MISU mortality data. The shift observed in the spatiotemporal random slopes emphasized the significant impact of aligning the spatial and temporal resolution between the MISU mortality rates and one-year

Figure 2. Maps of the coefficient of β_{1it} from the mental illness and substance use mortality model 5-IV



lagged unemployment rates. The inversion of the coefficients of the two covariates is intriguing. The alteration in coefficients can potentially be ascribed to the impacts arising from resolution mismatches (Cross et al., 2019) and the mediating effects brought about by the spatiotemporal interaction.

The results of the study reinforce the importance of considering the role of spatiotemporal resolution in covariates when implementing spatiotemporal areal disease modeling. The evidence strongly suggests that such an approach is necessary for comprehending the complex interplay of factors contributing to the spatial and temporal changes in MISU mortality rates in relation to unemployment. Consequently, a more detailed and careful investigation into this matter is called for, promising to enhance our understanding of these dynamics.

Three primary patterns emerged from the analysis. First, a consistent positive correlation was found in rural Appalachia, potentially attributed to marginalized living conditions and economic hardship prevalent in these regions (Moody et al., 2017; Shamblin et al., 2012). Additionally, the economic stagnation in these areas, compounded by rising unemployment rates, might have contributed to increased MISU mortality levels. Limited access to psychiatric clinics or rehabilitation facilities could be another factor contributing to this correlation in these regions (Rural Health Information Hub, 2021). For instance, MISU care facilities are particularly sparse in the Midwest, central South, and western Appalachian regions. Most counties in these areas exhibit lower access to MISU care facilities compared to the national average (13.73 facilities per 100,000 population) (Figure A.6). This pattern can be further elucidated by socio-psychological deprivation impacting youth groups (Alcántara & Gone, 2007; Brockie et al., 2015).

Second, the influence of unemployment on mental health appears to have grown stronger in the Mountain West following the Great Recession. However, this observation warrants a measured interpretation due to the relatively low unemployment and mortality rates these counties maintained throughout the entire study period. The high sensitivity of mortality rates to fluctuations in unemployment rates indicates the existence of internal or endogenous factors, such as social interaction dynamics and local responses to economic recession. The relative deprivation hypothesis could offer a possible explanation (Wilkinson & Pickett, 2007). This theory suggests that deprivation arises not from an individual's absolute social status, but rather from their relative social circumstances. Therefore, individuals who become unemployed in areas with low unemployment rates may experience heightened adverse mental health outcomes due to a stronger sense of relative deprivation. This aligns with previous research that underscores the relationship between relative deprivation and mental health outcomes (Beshai et al., 2017; Kondo et al., 2008; Mishra & Carleton, 2015; Saito et al., 2014; Salti, 2010). Beyond this, it's important to recognize that policy responses to unemployment may vary across counties, which could influence the observed associations (Thiede & Monnat, 2016). The delayed response to the impact of the Great Recession could be one of the contributing factors to the increased association between MISU mortality and unemployment observed in the Mountain West. Should a definitive relationship between relative deprivation and the onset of mental illness be established, it would further substantiate this explanation. The examination of such internal dynamics and regional policy responses forms a critical part of understanding the intricate ways in which unemployment affects mental health outcomes.

Finally, in an intriguing pattern, virtually all counties exhibited a diminished influence of the unemployment rate on MISU mortality during the Great Recession compared to periods before and after. Additionally, there was a smaller disparity in these effects across counties during the recession years than in non-recession years. This suggests that the impact of economic recessions might actually reduce the detrimental effects of unemployment on MISU mortality, supporting the relative deprivation hypothesis. Interestingly, these findings align with the notion of spatiotemporally heterogeneous impacts of unemployment on MISU mortality, as well as the counter-intuitive idea that recessions may be associated with *benefits* for mortality rates (Ariizumi & Schirle, 2012; Miller et al., 2009; Neumayer, 2004; Ruhm, 2000). The key takeaway from this study is that this *benefit* of recession is not universal but is locally evident in the impact of unemployment on MISU mortality at the county level, and moreover, this county-level effect fluctuated over time. This understanding has crucial policy implications. It suggests that policy interventions need to be tailored to the specific needs of different counties. By identifying counties where the effect of unemployment on MISU mortality is particularly pronounced, resources can be allocated more effectively. Prioritizing these areas for intervention can enhance state-level unemployment benefits, which in turn could serve to alleviate the adverse health impacts of unemployment (Cylus et al., 2015). Thus, understanding the spatial and temporal dynamics of unemployment effects can guide more targeted and effective public health policies.

This chapter contributes to the existing literature in two significant ways. Firstly, it underscores the necessity for locally-tailored intervention policies to address areas with positive associations between MISU mortality and unemployment. If unemployment is considered an external factor, local

health policymakers can respond to the increased impact of unemployment on mortality by identifying areas experiencing the abrupt unemployment increase. Additionally, by considering individual-level factors like subjective health evaluation or behavioral adjustment post-unemployment, the findings can inform proactive policies aimed at weakening the link between increased MISU mortality and local unemployment. Secondly, this chapter offered a methodological contribution by comparing various spatiotemporal interaction terms in models for the association between MISU mortality and unemployment–an area rarely explored. I found that the type-IV interaction term best fits the data, suggesting that the association between mortality and unemployment in one area is correlated with associations in neighboring areas and times.

Several limitations of this study must be acknowledged. First, although a spatially varying relationship between MISU mortality and unemployment was observed, individual studies have shown that an explicit non-linear relationship (e.g., squared term of unemployment rates on mortality rates) might exist (Bonamore et al., 2015; Garcy & Vågerö, 2012). The model developed in this study can be adapted to incorporate a nonlinear term (e.g., Generalized Additive Model) to control the complex effect of unemployment rates. Second, future research need to consider the impacts of long-term unemployment, extending one-year lagged unemployment rate. It can reveal the impacts of long-term unemployment on MISU mortality by including multiple lags or cumulatively weighted unemployment rates in the model (Tapia Granados et al., 2014; Zheng, 2012). Third, I only considered mortality rates of both genders and all races/ethnicities in the model. This is largely due to the high missing rates or unavailability of gender- and race/ethnicity-specific mortality rates and covariates. The issue can be addressed by introducing

advanced imputation methods (for example, H. Quick, 2019) and by examining the impact of unemployment rates on gender-specific mortality rates (Dagher et al., 2015). Finally, residuals in several counties are particularly high, which indicates that there could be uncontrolled and mediating factors that could improve the model. Future research can account for additional spatial and spatiotemporal factors in the analysis, for example, the regime and the policy stance that affect the elasticity of unemployment (Norström & Grönqvist, 2015), the liberalization of trades (Pierce & Schott, 2016), and the economic diversity in the composition of industries and businesses (Farré et al., 2018; van den Berg et al., 2017; Watson & Deller, 2017).

2.5 Conclusion

This chapter provided insights into the spatiotemporal variation of the association between MISU mortality and unemployment at the county level in the contiguous United States between 2001 and 2014. I found that the impact of unemployment on MISU mortality weakened during the Great Recession period. High positive associations were observed in counties within Ohio, Kentucky, and West Virginia, while moderate positive associations emerged in counties within North and South Dakotas post-Great Recession. Negative associations were concurrently prevalent in southeastern counties. Such heterogeneity reflected the regional contexts such as the shortage of medical services, economic depression, and other local processes. These findings add to the literature by indicating that the impact of unemployment on mortality decreased during recession periods as well as support the prevailing view that economic recession might be negatively associated with MISU mortality. Future studies are warranted to examine the consistency of

these findings across different gender, race/ethnicity, age groups, and more recent data at various spatial scales.

CHAPTER III

LOCALIZED EFFECTS OF GREENSPACE EXPOSURE ON MENTAL ILLNESS MORTALITY IN THE PACIFIC NORTHWEST UNITED STATES

This chapter is under review for publication in *Applied Geography*. I conceived research questions, collected data, conducted statistical analysis, and wrote and edited the entire draft. Dr. Hui Luan advised to edit the draft.

3.1 Introduction

Mental illness is a leading contributor to the worldwide increase in overall mortality, thus became a global public health issue (GBD 2019 Mental Disorders Collaborators, 2022; Lawrence, 2015; Vigo et al., 2016; Walker et al., 2015). This trend is notably prominent in the United States, where incidence and mortality rates have surged over the past two decades (Chang et al., 2010; Fekadu et al., 2015; Jayatilleke et al., 2017; Zheng & Echave, 2021). Therefore, it is crucial to identify factors beneficial to promoting mental health. Recent research is increasingly focusing on the protective impacts of greenspace (e.g., parks and vegetation) and blue space (e.g., rivers and lakes) exposure on mental health outcomes (Bratman et al., 2019; Labib et al., 2020; Pearson et al., 2019; Su et al., 2019).

According to Wilson's biophilia hypothesis, human beings innately incline toward natural environments (Wilson, 1984). Expanding on this idea, Hartig et al. (2011) proposed that exposure to nature, including forests and greenspaces, is beneficial to human health and expanded this concept to mental illness by incorporating ecosystem services (Hartig et al., 2011). Empirical studies have

also documented the protective effect of residential proximity to greenspaces on mental health status, leveraging data from various study designs including cross-sectional, longitudinal, panel, randomized trials, twin studies, and ecological studies (Gascon et al., 2016; M. P. White et al., 2021). Theoretical underpinnings and empirical findings have informed the development of frameworks that consider the effects of greenspace and its potential confounding and mediating effects (Dzhambov, Markevych, Hartig, et al., 2018; Hartig et al., 2014; Markevych et al., 2017; R. Zhang et al., 2021). For instance, Markevych et al. (2017) presented a framework for examining the influence of greenspace to health and well-being outcomes, which are mediated by three functions of greenspace exposure including reducing harm, restoring capacities, and building capacities (Markevych et al., 2017). Another framework suggested by Dzhambov et al. (2018) included annoyance that is a response of perceived greenspace, noise, and air pollution, and they assumed that perceived greenspace takes effect in poor mental health status via restorative quality (Dzhambov, Markevych, Hartig, et al., 2018).

Most existing research on the relationship between mental health outcomes and greenspace relies on individual-level data. These studies often utilize mental health status or surrogate indicators such as antidepressant prescriptions (Helbich et al., 2018; McDougall et al., 2021; Reeves et al., 2011). While small-scale and individual-level studies provide valuable insights, their findings cannot readily be generalized to the broader population (Yoo et al., 2021). Conversely, data from population-scale sources such as death registers can inform universally applicable mental health measures at the population level. Death registers, which record deaths attributable to mental illness, offer opportunities for spatial analyses and the integration of neighborhood contexts (Flores et al., 2020). However, the lack

of control populations in these datasets hinders the examination of associations between mental illness mortality and greenspace exposures. Nevertheless, spatial-ecological data can still inform public health policies at administrative levels (e.g., county or state in the United States), providing valuable insights for public health professionals and urban planners to devise effective greenspace strategies for reducing mental illness mortality at the population level, for example, prioritizing the improvement of accessibility to greenspace for vulnerable population.

Few studies have empirically examined greenspace exposure may be either a mediating or a mediated factor in mental health outcomes (Engemann et al., 2019; Klompmaker et al., 2019; McDougall et al., 2021; Vries et al., 2016), even though the conceptual frameworks proposed by Markevych et al. (2017) and Dzhambov et al. (2018) suggest these relationships (Dzhambov, Markevych, Hartig, et al., 2018; Markevych et al., 2017). Recent research has started to incorporate greenspace and bluespace exposures into investigations of associations between mental health outcomes and the physical environment while accounting for socioeconomic factors (Labib et al., 2020; McDougall et al., 2021). However, comprehensive frameworks integrating these environmental factors have not yet been examined to generate reliable estimates of the effect of greenspace exposure on mental illness mortality, despite its having potentials to provide additional knowledge on the relationship between mental health outcomes and greenspace exposure.

Spatial disparity is another important consideration when examining the effect of greenspace on mental illness mortality. It is well-documented that access to greenspace is not evenly distributed across geographic units or socio-economic and ethnic/racial groups within a city (Boulton et al., 2018; Chen et al., 2022; Dai, 2011; Gruebner et al., 2017; H. Ha, 2019; I. Song & Luan, 2022; Y. Song et al.,

2021; Wolch et al., 2014). Therefore, another research question arises: do the effects of greenspace exposure on mental illness mortality vary spatially? This inquiry may provide insights into how spatial disparities in greenspace can explain mental illness mortality and expand our understanding of where greenspace exposure can effectively reduce (or even exacerbate) the risk of mental illness mortality. Also, revealing the spatial disparity in the greenspace effect is a means of discovery to new hypotheses of explaining such disparity.

Given these concerns and observations from the literature, I hypothesize a spatially varying effect of greenspace exposure on mental illness mortality. The research questions I seek to answer are: (1) does the effect of greenspace exposure on mental illness mortality differ across small areas (specifically, census tracts in this case)? (2) what is the general relationship between socioeconomic and environmental factors and mental illness? Using a Bayesian spatiotemporal model on data from the Pacific Northwest of the United States (comprising Oregon and Washington), I aim to address these research questions and draw related implications.

3.2 Methods

3.2.1 Study area. The study area is the northwestern region of the contiguous United States (42°–49° N, 125°–116° W), specifically the states of Oregon and Washington, which, as of 2020, had populations of 4,237,256 and 7,705,281 respectively. The terrain of these states is divided into the temperate western Cascades and the drier eastern Cascades by the Cascade Range (Figure B.1). Major cities such as Seattle, Washington, and Portland, Oregon, are situated in the western region, which is also home to the majority of the population. The

Columbia River serves as a boundary between the two states, shaping a basin that includes the central and eastern regions of these states and is surrounded by mountainous areas in southeastern Oregon and northwestern Washington. These two states are known for their equitable mental healthcare into legislation before the federal enactment of parity in mental health services (Brodey et al., 1995; Wallace & McConnell, 2013).

3.2.2 Main outcome. I obtained individual death registers from 2006 to 2018, courtesy of the Oregon Health Authority and the Washington Department of Health. These registers contain detailed information about the deceased, such as birth and death dates, sex, marital status, underlying and contributing causes of death (up to twenty), education attainment, and residential locations. Mental illness mortality was defined by examining the causes of death in selected F (Behavioral and Mental Disorders) sub-chapters of the International Classification of Disease 10^{th} revision. The included sub-chapter codes of causes of death were F20–29 (Schizophrenia, schizotypal, delusional, and non-mood psychotic disorders), F30–39 (Mood or affective disorders), F40–48 (Anxiety, dissociative, stress-related, somatoform and nonpsychotic mental disorders), F50–59 (Behavioral symptoms associated with physiological disturbances and physical factors), F60–69 (Disorders of adult personality and behavior), F70–F79 (Intellectual disabilities), F80–F89 (Pervasive and specific developmental disorders), F90–F98 (Behavioral and emotional disorders with onset usually occurring in childhood and adolescence), and F99 (Unspecified mental disorder), based on the literature (Engemann et al., 2019; Lundin et al., 2016; Yoo et al., 2021). Substance use disorders were excluded due to their unique association with greenspace exposure (Engemann et al., 2019; Weeland et al., 2019; Wiley et al., 2022). I incorporated decedents with determinable, non-

homicide related causes of death into the study. After integrating the attribute and location tables and cleaning the location data, I aggregated the number of decedents in each census tract. During the study period, Oregon and Washington reported 6,020 and 5,859 decedents respectively. The data was collected under the exemption approval of the Institutional Review Board at the University of Oregon (protocol number: 12212020.026).

3.2.3 Greenspace exposure and covariates.

3.2.3.1 Greenspace exposure assessment. I considered the total area of parks per 10,000 population in the study area to assess greenspace exposure. Parks that are accessible and free of charge were deemed available to the population, while larger areas like national parks and national/state forests were included separately as a binary variable in the model. This choice aligns with the goal of examining residential greenspace exposure at the census tract level, as vast parks and forests have limited spatial (e.g., entrance) and temporal (seasonal opening or prohibited access) availability. Notably, in Oregon and Washington, greenness measured with the normalized difference vegetation index, which is often used in the literature to assess greenspace exposure (Astell-Burt & Feng, 2019; Nutsford et al., 2013; Ribeiro et al., 2021; Wood et al., 2017; R. Zhang et al., 2021), presents a clear contrast between the west and east sides of the Cascade Range (Figure B.1). This difference could lead to misclassification and inflated estimates of greenspace exposure's effect in areas in the western Cascade Range, where most of the study area's population resides. Consequently, assessing greenspace exposure via parks can help avoid potential bias. This method is consistent with previous studies on the relationship between greenspace and mental health (Dzhambov, Markevych, Hartig, et al., 2018; Labib et al., 2020; Wood et al., 2017). The base population for

calculating the mortality rates is collected from the American Community Survey (ACS) five-year data tables for the years 2009–2018, adjusted with the non-prisoned population rate in the 2010 Census to account for the population who are not limited for daily activities.

Spatial datasets of parks were collected from both governmental and non-governmental sources, including the Protected Areas Database (PAD-US) 3.0 from the United States Geological Survey (U.S. Geological Survey Gap Analysis Project, 2022), state park databases, the ParkServe database from the Trust for Public Land (Trust for Public Land, 2022), and OpenStreetMap. PAD-US is a comprehensive database of protected areas in the US, while ParkServe encompasses local parks across 14,000 communities in the US and classifies park management agencies into private, public (federal, state, and city), and non-profit organizations. By utilizing PAD-US and ParkServe, I first combined unique park polygons data, then supplemented missing parks with auxiliary data like state park datasets and OpenStreetMap. Certain tracts have large expanse of national parks, forests, wilderness areas, preservation areas, and state forests, leaving little potential for accessible parks. To account for such mutually exclusive characteristics between park types, I controlled tracts located in and around large greenspace with a binary variable, indicating whether a census tract intersects with large greenspace. I performed spatial data operations using the `R` package `sf` (Pebesma, 2018).

3.2.3.2 Covariates. In addition to greenspace exposure, I accounted for ten census tract level covariates potentially associated with mental illness mortality. These covariates were divided into three categories: environmental, demographic, and socioeconomic, which were selected based on the local characteristics of mental illness mortality and the mechanisms. I considered the temporal availability of all

covariate data either to match the yearly mental illness mortality data or to avoid temporal misalignment issues (Hund et al., 2012).

First, environmental confounders, including air pollution and bluespace, were controlled (Labib et al., 2020; McDougall et al., 2021; Nutsford et al., 2016; Pearson et al., 2019; Triguero-Mas et al., 2015). I obtained high spatial resolution (0.01 by 0.01 decimal degrees) datasets of predicted surface particulate matter with an aerodynamic diameter less than 2.5 micrometers ($PM_{2.5}$) from the Atmospheric Composition Analysis Group at Washington University in Saint Louis (van Donkelaar et al., 2021) to calculate the average concentration. Bluespace data was collected from the United States Geological Survey National Hydrography Data (U.S. Geological Survey, 2019), from which only perennial freshwater features were selected to calculate the proportion of waterbodies to each census tract's total area.

Second, I controlled demographic covariates, including age structure and the residential characteristics of decedents. Given that raw counts are used, I considered the percentage of the senior (65 or older) population to account for the age structure. I also considered care facilities and retirement homes ("care facilities" onwards), which have a high prevalence of psychiatric disorders (Seitz et al., 2010). Care facility locations in the study area were collected from DataAxle using the ArcGIS Business Analyst and the North American Industry Classification System (NAICS) codes (Table B.1 for details). Ethnic or racial composition, accounted for by the percentage of non-white population, served as a proxy for the sociodemographic context in each census tract. Additionally, the marital trajectory and the presence of significant others, which could potentially associate with the severity of mental illness (Barrett, 2000; Vaingankar et al., 2020), were

accounted for with the percentage of negative marital status (separated, divorced, and widowed) from the ACS data.

Finally, socioeconomic factors were considered as contextual factors in the model. The five-year ACS estimates were assigned to the period's final year (e.g., 2014–2018 estimates were matched to the 2018 mental illness mortality and covariates). Socioeconomic deprivation was proxied by variables such as median household income, educational attainment higher than the bachelor's degree, unemployment rates, and the percentage of households below the poverty line. The deprivation index was derived from the principal component analysis of these four variables, with the first component used (Table B.2). Before calculating the deprivation index, median household income values were adjusted to 2010 dollar values using the consumer price index from the U.S. Bureau of Labor Statistics (U.S. Bureau of Labor Statistics, 2022). All variables were standardized to reduce skewness. The correlation matrix of eleven explanatory variables shows the Pearson correlation coefficient between any pair of variables ranges between -0.700 and 0.656, and the variance inflation factors are between 1.059 and 3.713, indicating that the data does not have severe multicollinearity (Tables B.3–B.4).

3.2.3.3 Spatial data integration. Given that the boundaries of census tracts in Oregon and Washington were adjusted in each census year, I utilized the crosswalk tables provided by the US Census Bureau to tackle the spatiotemporal misalignment issue in the dataset. These tables, which account for all possible matches of geographic areas between the origin and the destination census years, enabled the integration of the dataset spanning the 2000 and 2010 Censuses (U.S. Census Bureau, 2021b). Utilizing the area and population fractions for each 2000 Census tract, I computed the population-weighted mean of the covariates at the

2010 census tracts for the 5-year ACS estimates from 2005 to 2009. For the period 2006–2008, the data values from the 2005–2009 dataset were assigned, given the availability of 5-year ACS estimates only after 2009.

Twenty-eight census tracts–nine in Oregon and nineteen in Washington–were excluded upon examination of the ACS data. This exclusion was driven by various factors. Eighteen tracts were essentially water areas, while ten were excluded due to the fulfillment of the spatial model assumption and the need for a sufficient population size. An example of the scarce population size is a census tract in downtown Portland, Oregon, which only had a population of 0–6 throughout the study period. Another example is a census tract in the Fort McDermitt Paiute and Shoshone Tribes Reservation in southeastern Oregon, where the estimated population remained at zero for the entire study period. Additionally, four island tracts were excluded to satisfy an assumption of spatial modeling of each tract having at least one neighbor (Table B.5). Consequently, the final data comprised 845 and 1,439 census tracts in Oregon and Washington for thirteen years, totaling 10,985 and 18,707 census tracts in Oregon and Washington, respectively.

3.2.4 Model formation.

3.2.4.1 Spatiotemporal interaction model. The analysis carried out was a Bayesian hierarchical spatiotemporal regression, which was structured to accommodate spatiotemporal dependencies present in the mortality data through the inclusion of purely spatial, purely temporal, and spatiotemporal interaction components. The hierarchical models offered the advantage of accounting for the spatial and temporal autocorrelation in the spatiotemporal data. I could either uncover spatial disparities of greenspace effects or propose a new set of hypotheses to explain such disparities by using a spatially varying model approach

(Janko et al., 2019). It is important to note that the models were fitted separately for Oregon and Washington to avoid potential systematic differences in factors influencing both outcomes and covariates between the two states, such as variations in the diagnosis, treatment, and recording of mental illnesses and practices in patient support (Ganguli & Rodriguez, 1999; Gao et al., 2018).

I employed a zero-inflated Poisson likelihood, which divides zeros into structural zeros (p_{zero}) and sampling zeros ($(1 - p_{\text{zero}})$Poisson) within a Poisson distribution. This necessitated introducing the probability of excess zeros, resulting in Formula 3.1.

$$\begin{cases} p(y_{it} = 0) = p_{\text{zero}} + (1 - p_{\text{zero}})\text{Poisson}(\lambda_{it} = 0) \\ p(y_{it} > 0) = (1 - p_{\text{zero}})\text{Poisson}(\lambda_{it} > 0) \end{cases} \tag{3.1}$$

In this formula, y_{it} represents mental illness mortality in census tract i at year t. The terms $b_{i\cdot}$, $\gamma_{\cdot t}$, and δ_{it}, as given in the full model specification (Formula B.6), correspond to purely spatial, purely temporal, and spatiotemporal interaction components respectively. $\log(P_{it})$ is the population offset, and x_{itk} denotes the k^{th} covariate value in census tract i at year t. More specifically, x_{it1} denotes greenspace exposure in census tract i at year t. The total effect of greenspace exposure at each census tract β_{i1+} is made up of the fixed main effect β_1 and the spatially structured effect β_{i1}. The spatial random slope β_{i1}, which adjusts the fixed effect β_1, is the primary interest of the analysis.

$$\beta_{i1+} = \beta_1 + \beta_{i1} \tag{3.2}$$

In Formulae 3.1 and 3.2, the spatial components $b_{1.}$ in the main effect and β_{i1} in the random slope follow the modified Besag-York-Molli'e (BYM) formulation (Besag et al., 1991; Riebler et al., 2016). This approach was developed to distinguish between spatially structured and unstructured components and to scale the spatially structured component (M. Quick & Luan, 2021; Riebler et al., 2016). The main temporal component was structured as a first-order autoregressive model, accommodating the overall trend in the crude mental illness mortality rate with the autocorrelation parameter ρ.

The spatiotemporal interaction (STINT) term δ_{it} conveys the convolution of spatial and temporal processes. This term can be formulated in one of four distinct types (denoted as Roman numerals I–IV), depending on whether the spatial and temporal processes are independent or structured (Knorr-Held, 2000). These four (2×2) interactions arise from all possible combinations of these processes in each of the spatial and temporal parts (Blangiardo & Cameletti, 2015; Haining & Li, 2020). Models both without (Model 1) and with the interaction term in four types (Models 2–5) were fitted and compared. A comprehensive model specification, including details of the modified BYM for spatial random effect, the temporal effect, the spatial random slope, and the spatiotemporal interaction terms, is provided in the Appendix B.

3.2.5 Model fitting and sensitivity analysis. The models were implemented with the Integrated Nested Laplace approximation (INLA) method, a form of Bayesian computing that approximates posterior marginals with Gaussian marginal distributions (Rue et al., 2009). The INLA method, in contrast to sampling-based Monte Carlo approaches like Markov Chain or Hamiltonian, is known for its computational efficiency, particularly in fitting complex spatial and

spatiotemporal models (Blangiardo & Cameletti, 2015; Lindgren & Rue, 2013; Rue et al., 2017). The statistical analysis was performed using `R-INLA` package version 2023.03.17 in `R` version 4.2.2 (Niekerk et al., 2021; R Core Team, 2022). Five primary models and three sensitivity analysis models were compared using deviance information criterion (DIC) (Spiegelhalter et al., 2002) and Watanabe-Akaike information criterion (WAIC) (Watanabe, 2013), both of which account for model complexity and accuracy with the effective number of parameters and the log likelihood, respectively.

The default priors for hyperparameters in the modified BYM formulation within the R-INLA package are the penalized complexity (PC) priors. These priors account for model complexity by introducing the Kullback-Leibler divergence between model specifications with and without additional parameters (Simpson et al., 2017). The probability density function of a PC prior is defined as $P(\sigma_\theta > \theta_1) = \theta_2$, or more succinctly $\text{PC}(\theta_1, \theta_2)$, where σ_θ denotes the standard deviation of a hyperparameter θ. A larger θ_2 results in a flatter prior, similar to a non-informative uniform prior. In this study, I used a weakly informative prior PC(1, 0.01) for precision hyperparameters and PC(0.5, 0.5) for mixing hyperparameters. By comparing three priors with DIC and WAIC, I assessed the sensitivity of the prior configuration.

3.3 Results

3.3.1 Descriptive results. Census tract-level mental illness mortality rates in Oregon and Washington showed an increasing trend throughout the study period, with Oregon consistently reporting higher rates than Washington. The range of mortality counts by census tracts was 0 to 13 (mean=0.40, standard

deviation [sd]=0.76), and 20,864 tracts reported zero counts. The annual mortality rate per 10,000 population during the study period ranged from 0.89 to 1.75 in Oregon and from 0.59 to 0.82 in Washington. Oregon's rates were 1.44 (2009) to 2.13 (2018) times higher than those of Washington (Table 5).

Covariates showed different variations. Environmental factors, particularly greenspace and bluespace exposure, had higher coefficients of variation (CV; [standard deviation]/[mean]) (2.323–3.009) compared to other covariates, with the exception of the number of care facilities per 10,000 population (1.280–1.515). $PM_{2.5}$ had relatively lower CVs (0.272–0.342) than the other two environmental covariates. Among demographic covariates, the percentage of the non-white population and the number of care facilities per 10,000 population showed more dispersion than the percentage of the senior population and negative marital status. Socioeconomic covariates exhibited moderate relative dispersion (Table B.6).

Table 5. Annual rate of mental illness mortality per 10,000 population in Oregon and Washington in 2006–2018

Unit: per 10,000 population

State	2006	2007	2008	2009	2010	2011	2012
Oregon	0.87	0.92	1.02	0.95	0.99	1.03	1.16
Washington	0.59	0.58	0.60	0.66	0.61	0.63	0.63
State	2013	2014	2015	2016	2017	2018	Average
Oregon	1.12	1.23	1.41	1.48	1.57	1.75	1.17
Washington	0.60	0.64	0.67	0.71	0.84	0.82	0.66

3.3.2 Model results. Comparisons of Models 1–5 indicate that Model 5, featuring a type IV STINT component, provided the best fit for the data. The DIC

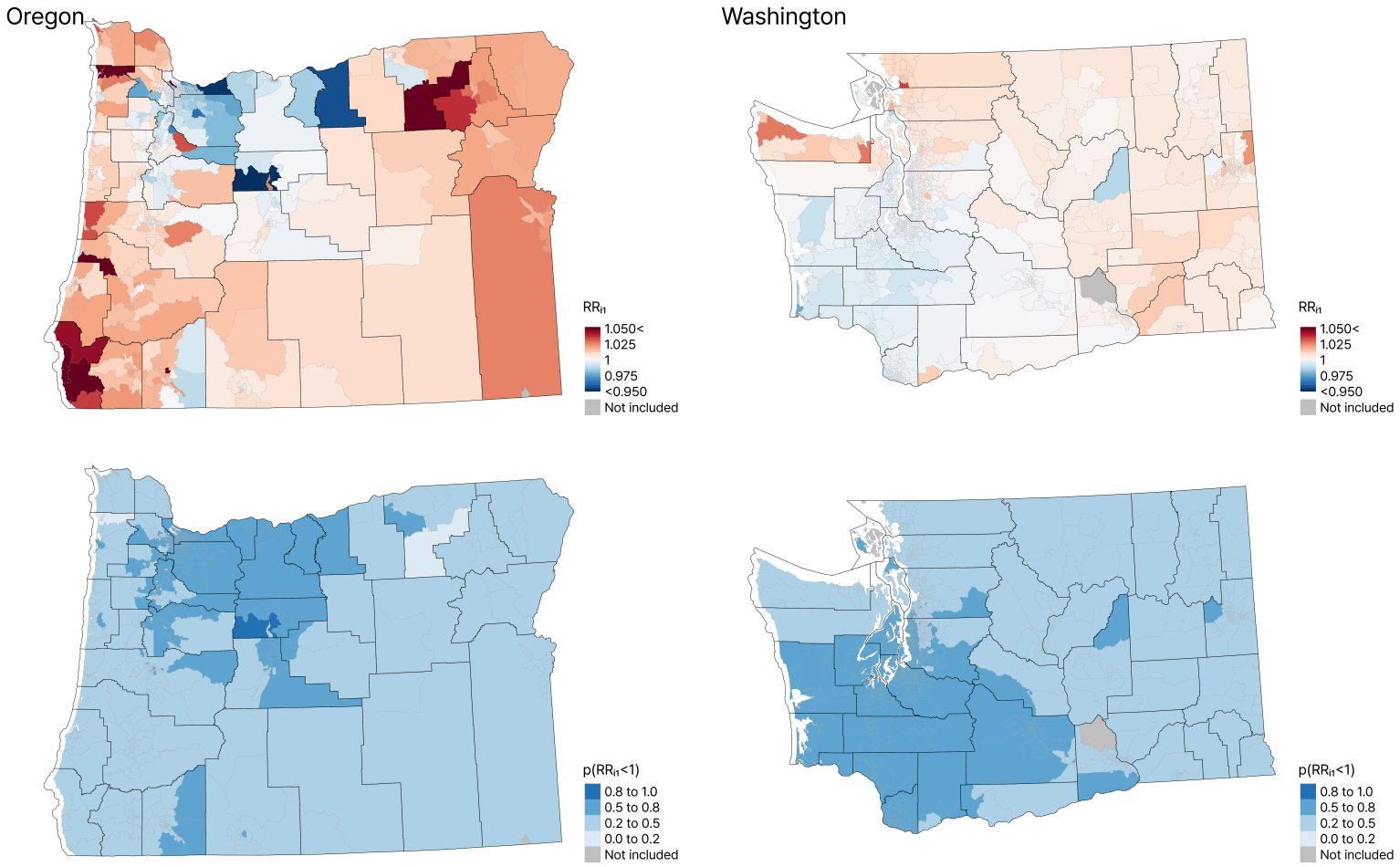
and WAIC values of Model 5 decreased in both states (Oregon: ΔDIC=-207.87, ΔWAIC=-208.98; Washington: ΔDIC=-180.85, ΔWAIC=-200.63) compared to those of Model 1 (Table 6). The area of parks per 10,000 population, the primary variable of interest, had a fixed effect of 0.908 (95 % CrI = [0.843, 0.978]) in Oregon and 0.949 ([0.903, 0.999]) in Washington, indicating that greenspace exposure had an overall protective effect on mental illness mortality in both states. However, the RRs of the spatial random slope ranged from 0.935 ([0.803, 1.090]) to 1.120 ([0.950, 1.321]) in Oregon and from 0.976 ([0.885, 1.076]) to 1.033 ([0.954, 1.119]) in Washington, adjusting the fixed effect RR of the greenspace exposure (Table 7 and Figure B.2). The posterior probability of the random slope RR being smaller than zero ($p(\mathrm{RR}_{i1} < 1)$) was between 0.5 and 0.8 near Portland in Oregon and southwestern Washington (Figure 3). The census tracts intersecting national and state forests were negatively associated with mental illness mortality, although the associations were marginal (0.996 [0.893, 1.111] in Oregon and 0.960 [0.831, 1.108] in Washington).

Table 6. Deviance and Watanabe-Akaike information criteria of five model settings with and without spatiotemporal interaction

	Oregon		Washington	
	DIC[1]	WAIC[2]	DIC	WAIC
Model 1: base model without spatiotemporal interaction	19528.87	19537.96	24737.75	24746.40
Model 2: spatiotemporal interaction type I	19543.06	19542.67	24796.34	24787.55
Model 3: spatiotemporal interaction type II	19507.28	19493.98	24759.35	24708.86
Model 4: spatiotemporal interaction type III	19426.61	19413.61	24797.70	24763.45
Model 5: spatiotemporal interaction type IV	19321.00	19328.98	24556.90	24545.77

[1] Deviance information criterion, [2] Watanabe-Akaike information criterion.

Figure 3. Tract-level random slope relative risk ($\text{RR}_{i1} = \exp(\beta_{i1})$) of one standard deviation increase of the park areas per 10,000 population to mental illness mortality and its posterior probability of being smaller than one in Oregon and Washington



Note: black lines are county borders.

The results from the analysis of environmental covariates suggest statistically significant effects of waterbodies on mental illness mortality. For example, in both Oregon and Washington, one standard deviation (SD) increase in the proportion of waterbodies (5.9 % in Oregon and 8.77 % in Washington) was associated with a marginal 2.1 % ([-6.0 %, 2.0 %]) and 3.4 % ([-7.6 %, 1.1 %]) lowering of risk in Oregon and Washington, respectively. However, one SD increase of average $PM_{2.5}$ (1.94 micrograms per cubic meter [$\mu g/m^3$] in Oregon and 1.60 $\mu g/m^3$ in Washington) was associated with a 6.1 % ([0.7 %, 11.8 %]) and 2.6 % ([-2.4 %, 7.8 %]) increase of mental illness mortality risk, indicating that $PM_{2.5}$ significantly raises the risk only in Oregon.

For demographic covariates, the proportion of the non-white population was marginally negatively associated in both states (RR=0.973 [0.924, 1.026] in Oregon and 0.975 [0.920, 1.034] in Washington) while the number of care facilities per 10,000 population in Oregon showed a marginal positive association (1.049 [0.974, 1.129]). SES was found to have a significantly protective effect on mental illness mortality, with its relative risk (RR) being 0.931 ([0.901, 0.962]) in Oregon and 0.926 ([0.891, 0.962]) in Washington (Table 7). Sensitivity analysis confirmed that model results with three alternative prior specifications were similar to those from Model 5 with the base prior PC(1, 0.01) (Table B.7).

Table 7. Estimated relative risks of explanatory factors from Model 5

	Oregon		*Washington*	
	Relative risk	95 % Credible Interval	Relative risk	95 % Credible Interval
Area of parks per 10,000 population				
Fixed effect coefficient	0.908	0.843, 0.978	0.949	0.903, 0.999
Random effect coefficients				
Minimum	0.935	0.803, 1.090	0.976	0.885, 1.076
Maximum	1.120	0.950, 1.321	1.033	0.954, 1.119
Combined effect coefficients				
Minimum	0.849	0.725, 0.994	0.927	0.832, 1.032
Maximum	1.017	0.856, 1.208	0.981	0.901, 1.068
National/State parks & forests				
No intersection	Reference		Reference	
Intersection	0.996	0.893, 1.111	0.960	0.831, 1.108
$PM_{2.5}$ ($\mu g/m^3$)	1.061	1.007, 1.118	1.026	0.976, 1.078
Waterbodies (%)	0.979	0.940, 1.020	0.966	0.924, 1.011
Number of care facilities per 10,000 population	1.049	0.974, 1.129	1.067	1.017, 1.120
65 years old or older (%)	1.233	1.178, 1.292	1.311	1.250, 1.375
Negative marital status (%)	1.125	1.081, 1.172	1.167	1.112, 1.225
Non-white population (%)	0.973	0.924, 1.026	0.975	0.920, 1.034
Socioeconomic deprivation	0.931	0.901, 0.962	0.926	0.891, 0.962

3.4 Discussion

The results revealed spatial variation in the effects of greenspace exposure on mental illness mortality across census tracts in Oregon and Washington. This finding expands the studies on the relationship between greenspace and mental health outcomes since most previous studies assumed the effect to be spatially stationary across the entire sample (Banay et al., 2019; Dadvand et al., 2015; Engemann et al., 2019; Nutsford et al., 2013; Nutsford et al., 2016; Tomita et al., 2017; Tost et al., 2019). The overall protective effect of greenspace exposure on mental illness mortality, which was found to be significant, adjusted to different directions when accounting for the random slopes. This variability in spatial random RRs of greenspace exposure implies that the impact of greenspace exposure on mental illness mortality is not spatially constant over the entire study region. Rather, local effects exist, and this supports the variability in local greenspace effects on mortality (Jiang et al., 2021; Richardson et al., 2010). Recognizing these local impacts of greenspace exposure has important implications for establishing geographically-tailored interventions, thus is equally important to assessing fine-grained greenspace exposure (Yoo & Roberts, 2022; R. Zhang et al., 2021).

The findings presented in this study provide evidence of different associations between mental health outcomes and covariates across the two states. This implies that conceptual models may operate differently by regions. For instance, $PM_{2.5}$ in Oregon and care facility rates in Washington exhibited significant positive associations. Other covariates showed consistent patterns of significance in both states. The ratio of individuals aged 65 years or older and negative marital status were significantly positively associated with mental health mortality, while socioeconomic deprivation was significantly negatively associated.

Bluespace exposure and the proportion of non-white population exhibited marginally negative associations. These findings offer limited evidence that racial and ethnic diversity may have a beneficial impact on mental health status (Henderson et al., 2005). However, these results do not reject the possibility that tracts with higher mental illness mortality may feature greater racial and ethnic diversity, considering that racial and ethnic diversity in Oregon and Washington marked below the national average based on the U.S. Census Bureau's diversity index (U.S. Census Bureau, 2021a).

The protective effect of greenspace is pronounced in contiguous census tracts lying between Seattle and Portland, aligning with previous research that reported beneficial effects of greenspace exposure in urban populations (Akpinar et al., 2016; Astell-Burt & Feng, 2019; Astell-Burt et al., 2022; Bijnens et al., 2020; M. P. White et al., 2013). The finding adds to the literature with evidence of the urban greenspace benefits in the combined urban-rural setting. Furthermore, areas with protective greenspace exposure effect on mental illness mortality are typically featured with flatter topography and milder climates than others in the study region. This suggests that the significance of the greenspace effect may be affected by the physical characteristics of an area. The presence of dense vegetation in these areas also implies a potential interaction between the quality, types, and physical features of greenspace and its effects on mental illness mortality. Future analyses should consider environmental barriers such as slopes and curvature to incorporate greenspace accessibility into the assessment of the health effects of environmental exposures on mental health outcomes (Liang et al., 2022).

Spatial random RRs exceeding 1.0, which offset the protective fixed effect, necessitate a careful interpretation. There are three possible explanations. First,

areas with a high spatial RR could be preferred by high-risk populations, implying that patients with critical mental illness might not have been hospitalized or moved into residential care facilities, leading to unprotective effect estimates. This hypothesis can be further examined through qualitative surveys on the residential preference of high-risk population and patients with mental illness, along with the analysis of spatial accessibility and economic affordability of mental healthcare systems, which often exhibit disparities along socioeconomic gradients (McConnell et al., 2020; Zhu et al., 2022). Second, parks in these areas might not be accessible to high-risk populations, limiting their effect on mental health outcomes. This lack of accessibility could be due to parks being located away from residential areas or parks having few entrances to the residential area. Accurate spatial information on park entrances and exits is needed to evaluate actual accessibility. Third, there might be no easily accessible greenspace for residents in certain areas. In such cases, tracts with higher mental illness mortality might also be more racially and ethnically diverse, suggesting a need for further investigation into the role of racial and ethnic diversity in mental illness. Third, there is no available greenspace that can be easily accessed by the residents in such area. The census tracts with elevated spatial risk exceeding 1.05 partly overlap or are surrounded by large National or State forests, which severely delimits usable land for both residence and parks. It may lead to biased effect estimates in tracts with some mortality counts surrounded by tracts with zero or very low mortality counts and less greenspace exposure.

There are four limitations that should be acknowledged in this study. First, the accuracy of information in death certificates relies on the expertise and experience of certifiers, such as physicians or nurse practitioners. The recording

of mental illness as a cause of death may differ between the two states. Although death certificates and preparation protocols of these are standardized across the United States, there may be systematic differences in diagnosing mental illness among patients in different geographic areas, such as counties or states. The data is not completely immune to such issues. Second, there may be unaccounted factors that could have influenced the severity or duration of mental illness and the actual utilization of greenspace. For example, incidental (e.g., trauma) and collective effects such as social networks (the impact of relationships with others), social grouping effects (the impact of group identification in individuals), and social cohesion can mediate both the progression of mental illness and the utilization of greenspace (Jennings & Bamkole, 2019). This limitation is primarily due to the nature of death register data, which do not provide actual residential circumstances beyond residence of decedents. Third, census tracts may not fully capture the actual characteristics of residential neighborhoods. Census tracts are delineated for administrative purposes (i.e., enumeration); thus, the actual activity space may not align with census tract boundaries. The relevant geographic context may differ across census tracts (Kwan, 2012; L. Zhang et al., 2021). Lastly, the uncertainty in covariates, such as margin of errors in ACS data, needs to be properly incorporated into models using measurement error models.

I suggest four directions for future research. The first direction is to examine spatially varying associations in other environmental and socioeconomic factors. This approach presents challenges in justifying mechanisms, which identify different spatial spheres of influence of such explanatory factors, and dealing with computational complexity. The second direction is to develop a suitable method for assessing greenspace exposure across regions with heterogeneous environments.

Similar to integrating ecological region information in a nationwide study (Coleman et al., 2022), future studies should develop a comprehensive measure of greenspace exposure that considers the fundamental environmental differences across regions. This aligns with the development of integrated environmental assessment measures (Marek et al., 2021). The third is to analyze the association between mental illness mortality and greenspace exposure separately or concurrently by gender and racial/ethnic subgroups. Such analyses will be feasible and warrant their results if mortality data in ethnic subgroups have sufficient sample sizes. Finally, the approach employed in this study can be expanded to other states in the United States, as well as subnational geographic areas in other countries, particularly in low- and middle-income countries (Nawrath et al., 2021). Such expansion is expected to provide additional evidence on the spatially varying relationship between mental illness mortality and greenspace exposure.

3.5 Conclusions

This chapter challenged the conventional assumption of spatially stationary effects of greenspace exposure on mental illness mortality. The relative risk of greenspace exposure on mental illness mortality varied from 0.935 to 1.120 across census tracts in the Pacific Northwest region. The protective effects of greenspace were higher in areas between Seattle and Portland. This suggests that interventions to improve greenspace exposure should be tailored to specific locations to reduce mental illness mortality in the region.

CHAPTER IV

LOCALIZED CAUSAL EFFECTS OF GREENSPACE EXPOSURE TO MENTAL ILLNESS MORTALITY: A PIONEERING STUDY IN THE STATE OF WASHINGTON, 2018

4.1 Introduction

In recent years, researchers have increasingly paid attention to the potential benefits of greenspace on human mental health (Gascon et al., 2016; Roberts et al., 2019; World Health Organization, 2021). Observational studies have concentrated on the association between greenspace and mental health outcomes, however, a growing body of literature explores causal relationship through experimental study designs (Collins et al., 2020). Numerous pathways and mechanisms have been developed to explain and conceptualize the impact of greenspace on mental health, many of which can be examined using causal frameworks (Dzhambov, Markevych, Hartig, et al., 2018; Markevych et al., 2017; Mueller et al., 2020; R. Zhang et al., 2021).

From a spatial standpoint, studies have found protective effects of greenspace across various study regions (M. P. White et al., 2021). Urban areas and a few national registries were the major spatial sample frame (Engemann et al., 2019, 2020; Luque-García et al., 2022). Although these studies have considerably broadened our understanding of the protective influence of greenspace exposure on mental health outcomes, there is limited exploration of the spatial distribution of greenspace exposure across various regions. The consideration of the spatial difference in greenspace exposure can help to comprehend the spatial disparity in greenspace exposure effects. The two research agendas of causal effect estimation

and spatial differences in the effect of greenspace exposure's effect on mental illness mortality lead to the following research questions of our study.

> First, is the causal effect of greenspace exposure on mental illness mortality different across subpopulation groups?
>
> Second, does the effect of greenspace exposure on mental illness mortality depend on the absolute quantity of greenspace across the entire study area or is it contingent on local area contexts?

To answer these questions, I employ matching methods on retrospective, state-wide death register data from the State of Washington in 2018. This approach allows me to examine the causal effect of greenspace exposure on mental illness mortality, under the socio-environmental pathways and mechanisms currently suggested in the literature (Aerts et al., 2022; Dzhambov, Markevych, Hartig, et al., 2018; Dzhambov, Markevych, Tilov, & Dimitrova, 2018; Helbich et al., 2018; Labib et al., 2020; Markevych et al., 2017). Furthermore, it facilitates the exploration of spatial differences in the causal effect of greenspace on mental illness mortality across regions. Through this study, I aim to build upon existing findings that highlight the association of greenspace with reduced cognitive decline in mid-age adults (de Keijzer et al., 2018) and lowered mortality in national cohorts (Wan et al., 2022) by investigating the causal effect of residential greenspace exposure on mental illness mortality.

The structure of this chapter is as follows. It begins with an introduction to the concepts of causal inference, their spatial expansion, and applications in assessing the association between mental illness mortality and greenspace exposure. Subsequently, the chapter delves into the methodology and data description,

followed by a comparison of causal effect estimates derived from both absolute and relative definitions of treatment conditions for continuous greenspace exposure in the entire study population as well as subregional populations. I will estimate the causal effect through propensity score matching to examine the variability of effect estimates across the subregions. Lastly, I reflect upon the implications and potential extensions of my study findings.

4.2 Spatial Causal Inference and Statement of Problems

The concept of causal inference from observational data is traditionally explored through two frameworks: the potential outcome and the probabilistic approach (Pearl, 2009). The potential outcome framework, in which researchers emulate randomized controlled trials, was developed to overcome observational study design limitations. One limitation is the inability to observe a true treatment effect, as both treated and controlled states cannot be observed in an entity simultaneously. This motivated the development of matching methods as a subset of causal inference methods. These methods aim to achieve balance between treated and control groups based on pre-treatment covariates, thereby enabling the estimation of counterfactual outcomes (Rubin, 1974; Splawa-Neyman et al., 1990). These methods assume three conditions: (1) random assignment of treatment, (2) the existence of only one treatment condition per unit (e.g., person), and (3) stability of treatment effect within the treated groups (referred to as the *stable unit treatment value assumption [SUTVA]*) (Imbens & Rubin, 2015). The average treatment effect (ATE) is the result of this counterfactual analysis, due to the aggregated comparison between treated and controlled groups. When there are

multiple subgroups in the population of interest, one can estimate *local* average treatment effect (LATE) within each subgroup.

Causal inference in geography has been addressed through dichotomous treatment and partial regression analysis (Davidson, 1976; Pringle, 1981). These methods were inspired by the Simon-Blalock method for analyzing spurious correlation in multivariate settings (Blalock, 1961; Simon, 1954). In the past two decades, causal inference methods such as difference-in-differences (DiD) and propensity score matching (PSM) oftentimes have been used in the literature in geography and cognate fields, most of which were published in economic geography and epidemiology (Li, 2022).

The geographical perspective in recent causal inference literature can be categorized into two major viewpoints (Akbari et al., 2023; Reich et al., 2021). The first treats *geography as a condition affecting study subjects*. Researchers focus on a set of small areas whose boundaries determine a treatment condition, for example, a local tax code on residential properties in one of two neighboring jurisdictions. This view has been integrated into natural experiment and regression discontinuity designs for causal inference in the literature (Keele & Titiunik, 2015, 2016; Keele et al., 2015). The second is *geography as a source of spatial dependence in measured or unmeasured variables for inferential models*. This branch inherited tradition in spatial statistics where scholars aimed to attain random errors by adding spatial components into models. Thus, handling spatial dependence with a set of spatial regression models (e.g., spatial error, spatial Durbin, and spatial autoregressive models) is often found in the literature. Examples include adding spatial autoregressive term into the DiD model (Dubé et al., 2014), combining spatial distance and propensity score matching (Papadogeorgou et al., 2019),

multiscale geographically weighted regression for instrumental variables (Bilgel, 2020), and removing spatial autocorrelation in covariates before performing regression analysis (Dupont et al., 2022).

Meanwhile, the concept of LATE derived from geographic subregions raises questions about spatial differences in LATE, especially in the context of greenspace-mental health research. Despite assertions that LATE generally differs from ATE due to the heterogeneity of subpopulations (Xie, 2013), this claim remains untested in cases where the subpopulation is geographically defined. Furthermore, as greenspace exposure is conditioned by physical and social environments and is highly spatially autocorrelated, there is potential for violation of SUTVA (Akbari et al., 2023). If these conditions are considered and the entire population is divided into subpopulations with similar physical environment characteristics, it becomes feasible to draw causal interpretations from these subpopulations. This also leads to the following question on the relativity of greenspace exposure in a subregion. For instance, if two subregions have distinctly different distributions of greenspace exposure, one might ask whether the absolute quantity of exposure metrics or intra-region relative greenspace exposure is more crucial in reducing the risk of adverse mental health outcomes.

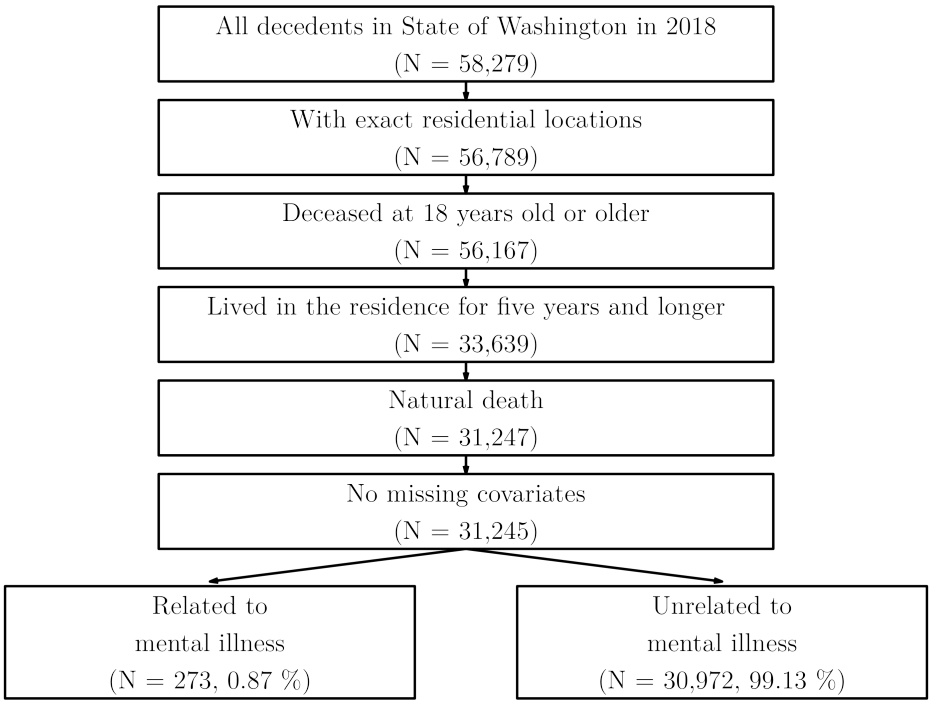
Taking these issues into account, I aim to examine the spatial differences in the effects of greenspace exposure on mental illness mortality, focusing on the boundaries of combined ecological regions and urban areas. To conduct a causal analysis, I introduce two additional assumptions: first, there are no unmeasured confounders; second, there are no interferences between individuals or mediations between any combination of covariates. These assumptions allow me to interpret the estimates causally after matching individuals in treated and control groups.

4.3 Methods

4.3.1 Data. Individual death register data for the year 2018 were gathered from the Washington State Department of Health. This dataset includes essential personal details such as sex, race, age, marital status, educational attainment, length of residence at the time of death, manner of death, and up to twenty recorded causes of death. The residential locations of the decedents were provided in a separate file and were integrated into the main dataset using unique death certificate identifiers.

I selected decedents who were 18 years or older at the time of death and had died naturally (e.g., disease or aging). The underlying and multiple causes of death, coded in the tenth revision of International Classification of Disease system, were screened to include subchapters of F20–29 (Schizophrenia, schizotypal, delusional, and non-mood psychotic disorders), F30–39 (Mood or affective disorders), F40–48 (Anxiety, dissociative, stress-related, somatoform and nonpsychotic mental disorders), F50–59 (Behavioral symptoms associated with physiological disturbances and physical factors), F60–69 (Disorders of adult personality and behavior), F70–F79 (Intellectual disabilities), F80–F89 (Pervasive and specific developmental disorders), F90–F98 (Behavioral and emotional disorders with onset usually occurring in childhood and adolescence), and F99 (Unspecified mental disorder). Substance use disorders were excluded as their association with greenspace exposure is inconsistent across studies (Weeland et al., 2019; Wiley et al., 2022). I included decedents who had resided in their reported residence for five years or more for exposure assessment. As a result, $31,245$ decedents met my study population criteria, with 273 of these related to mental illness (Figure 4).

Figure 4. Data selection flowchart



The dataset was obtained under the exemption approval of the Institutional Review Board at the University of Oregon (protocol number: 12212020.026).

4.3.2 Exposure assessment. The Normalized Difference Vegetation Index (NDVI) from the Landsat-8 OLI sensor (with a spatial resolution of 30 meters) was used to assess greenspace exposure, which is widely recognized in previous research. NDVI quantifies greenness at each pixel, with its values impacted by waterbodies or impervious surfaces such as buildings or barren land (Cardinali et al., 2023). Consequently, Therefore, we considered modified normalized difference water index (MNDWI) for water features (Xu, 2006) and normalized difference built-up index (NDBI) (Zha et al., 2003) for buildings to balance their distributions in subsequent matching analysis. NDVI data is based on eight-day average data from level 1 images (orthorectified), while the other two indices were directly derived from near

and shortwave infrared bands in eight-day raw level 2 images (orthorectified and atmospherically corrected) without cloud cover. Images collected every eight days were used to calculate the five-year (2013–2017) average. All spectral indices from satellite data were obtained from the United States Geological Survey via Google Earth Engine.

Residential exposure was assessed at individual residential locations. The exposure was defined as the area within a 15-minute walking distance along the road network, assuming a walking pace of 5 kilometers per hour. This approach, yielding a mean of 1.77 km^2 and a standard deviation of 0.70 km^2, aligns with the standard definition of a residential neighborhood of an 800-meter radius circular buffer from residences ($0.8^2 \times \pi = 2.01$ km^2) (Astell-Burt et al., 2022; Hartley et al., 2021; Sturm & Cohen, 2014). Using the Open Source Routing Machine (OSRM) and the OpenStreetMap road network data, the walking distance area was derived from each residence (Giraud, 2022; Luxen & Vetter, 2011).

4.3.3 Covariates. Individual and neighborhood covariates were included in the propensity score estimation model. Three groups of covariates were identified from the mechanisms and pathways in the literature: individual characteristics, environmental exposures, and neighborhood contexts. Individual characteristics that were recorded in the death certificates include sex, age, marital status, educational attainment, and smoking status. Environmental exposures were assessed within the 15-minute walking area and consisted of NDBI, MNDWI, traffic noise, and air pollution exposure. Traffic noise estimation data for 2018 came from the United States Department of Transportation Bureau of Transportation Statistics, which includes road, rail, and aviation noise across the continental United States (United States Department of Transportation Bureau of

Transportation Statistics, 2020). Particulate matter with aerodynamic diameter less than 2.5 micrometers ($PM_{2.5}$) air pollution prediction data was sourced from Washington University at Saint Louis, covering the continental United States at 1-kilometer spatial resolution from 2000 (van Donkelaar et al., 2021). Five-year (2013–2017) $PM_{2.5}$ average was calculated from these datasets, and the individual exposure was calculated as the average of pixel values in each walking distance area. The neighborhood-level contextual variables were extracted at the decedents' residential census tract from the American Community Survey five-year (2013-2017) data tables. These variables–rate of below poverty line, rate of non-white population, median household income, and unemployment rate–reflect neighborhood socioeconomic characteristics.

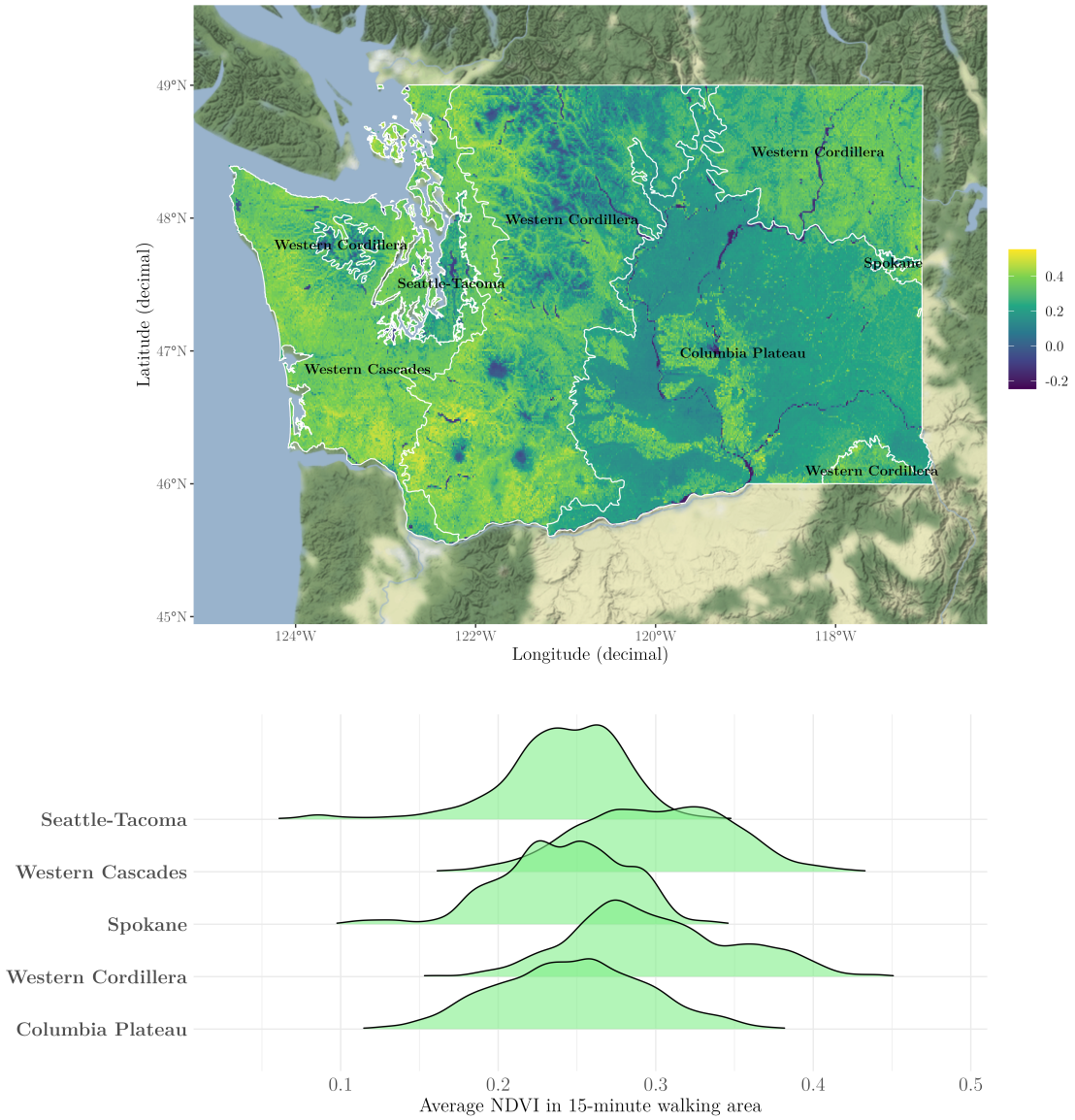
4.3.4 Propensity score matching. I used propensity score matching (PSM) to estimate causal effects. PSM seeks to minimize the difference in the probability of having been treated (*propensity score* or *distance*) between treated and controlled groups, and to balance the mean and variance of covariates. The process comprises three steps: estimating propensity scores using observed treatment information and covariates, matching units in the treated and controlled groups with minimal distance, and, after obtaining the matched dataset, estimating the causal effect by performing a simple regression of the outcome against the treatment variable. To balance covariates, the Covariate Balancing Propensity Score (CBPS) model was employed, which uses weights from inverse propensity score to achieve the minimum covariate mean difference between treated and controlled groups (Imai & Ratkovic, 2014). Decedents with the exact sex and race were matched given the different characteristics in mental health status and outcomes (Zheng & Echave, 2021). I evaluated and compared matching results using covariate balance, as measured by

the standardized mean difference for each covariate. These results are reported in Appendix C.

4.3.5 Subregion definition. The State of Washington, located in the northwestern part of the continental United States, is divided into two distinct ecological regions–the forested western region and the desert eastern region–by the Cascade mountain range. Given the higher-level impacts of ecology and climate on greenspace, ecoregions were used as a criterion to divide the entire region into subregions with similar ecological processes. For this division, I used the level 2 ecoregions from the Commission for Environmental Cooperation (Commission for Environmental Cooperation, 1997). Additionally, large urban areas where greenspace is sparse were separated. On top of the highly heterogenous nature of the spatial distribution of environmental exposure (Jarvis et al., 2020), the choice is supported by the preliminary finding of average NDVI values within 15-minute walking areas, which showed that urban areas such as Seattle and Spokane had lower NDVI values than other areas (Figure C.1). This also aligns with the literature on the protective effects of greenspace in urban areas (Astell-Burt & Feng, 2019; Collins et al., 2020; Dzhambov, Markevych, Tilov, & Dimitrova, 2018; Nutsford et al., 2013). Thus, I obtained Seattle-Tacoma and Spokane city boundaries from the urban growth areas in the Washington Geospatial Portal. Five subregions–Seattle-Tacoma, Western Cascades, Spokane, Western Cordillera, and Columbia Plateau–were established for the comparison of the effects of greenspace exposure. The Seattle-Tacoma area overlaps the Western Cascades, while Spokane extends across the Western Cordillera and Columbia Plateau (Figure 5). For a sensitivity analysis of subregion definition, the census core-based statistical areas were compared with the main results.

Figure 5. Five subregions in State of Washington based on level 2 ecoregions and urban growth areas with the distribution of five-year (2013-2017) average NDVI values (above) and the distribution of average NDVI values in 15-minute walking areas by subregions

(Note: the base map is from Stamen design)



4.3.6 Treatment setting with absolute and relative greenspace exposure.

Defining binary treatment poses a challenge as greenspace exposure is a continuous variable. Stitelman et al. (2010) suggested two necessary conditions for dichotomization of a continuous variable in a causal inference context: mechanism and effect equivalence. Mechanism equivalence implies that the probability of being treated remains constant regardless of exposure values, provided that these values surpass the predefined threshold (Stitelman et al., 2010). Stitelman et al. (2010) distinguished the *intended* and *observed* mechanisms in which they assumed intentionally different treatment probabilities across pre-dichotomization exposure groups (Stitelman et al., 2010). However, in our case of using observation data, we checked whether the treatment probability of each subgroup estimated by the covariates was identical to the treatment probability given various thresholds. The expectation is a negligible difference between these two estimated treatment probability values (Table 8). Effect equivalence requires that expected outcomes, which are estimated by covariates, remain constant across exposure subgroups above the threshold (Stitelman et al., 2010). To satisfy this condition, when plotting expected outcomes against exposure values, the data points should align horizontally. I investigated the fulfillment of these equivalence conditions in both the entire and subregional populations.

Equivalence conditions were examined as follows. I checked five threshold values, namely 50th, 60th, 70th, 80th, and 90th percentiles of greenspace exposure, for both the entire population and each subregional population. Using each threshold, I grouped the exposure values into 2 percent intervals and estimated the treatment probability for each interval group, with and without the binary variable indicating whether the value exceeds the threshold. The difference between

Table 8. Equivalence conditions for dichotomizing continuous exposure variables from Stitelman et al. (2010)

Conditions	Description	Mathematical expression
Mechanism equivalence	Probability of treatment assignment is the same regardless of dichotomization	$p^*(A'\|V) = p(A'\|A = 1, V)$ $\forall A' \in \{1, \cdots, j\}$
Effect equivalence	Expected potential outcomes are the same across the pre-dichotomization strata	$E\left[Y^{\Delta}(1)\|V\right] = \cdots = E\left[Y^{\Delta}(j)\|V\right]$

Notations
A': Multi-class treatment to be dichotomized
A: Dichotomized treatment
V: Covariates
Y: Potential outcome
$Y^{\Delta}(V)$: Potential outcome adjusted with covariates
p, p^*: Intended and observed probability of having been treated

the two treatment probability values were then compared. For example, when the 60th percentile was set as the threshold, I grouped the exposure values into 2 percentile intervals (e.g., [60, 62), [62, 64), $\cdots$, [98, 100] percentiles) and calculated the difference between the probabilities of each percentile group, with and without the indicator for values exceeding the 60th percentile. The mean difference was calculated at each 2 percentile interval for the five thresholds.

4.3.7 Examination of research questions. I assessed the fulfillment of equivalence conditions in each subregion to examine the research questions. Based on this assessment, I addressed the first research question by comparing causal effect estimates from propensity score matching in three subregions where both equivalence conditions were met. To answer the second question, I applied absolute (0.28–0.31) and relative (50th–90th percentiles) greenspace exposure to the entire population and subregional populations. The absolute values were chosen based on

the distribution of greenspace exposure to ensure non-empty treated groups in all subregions.

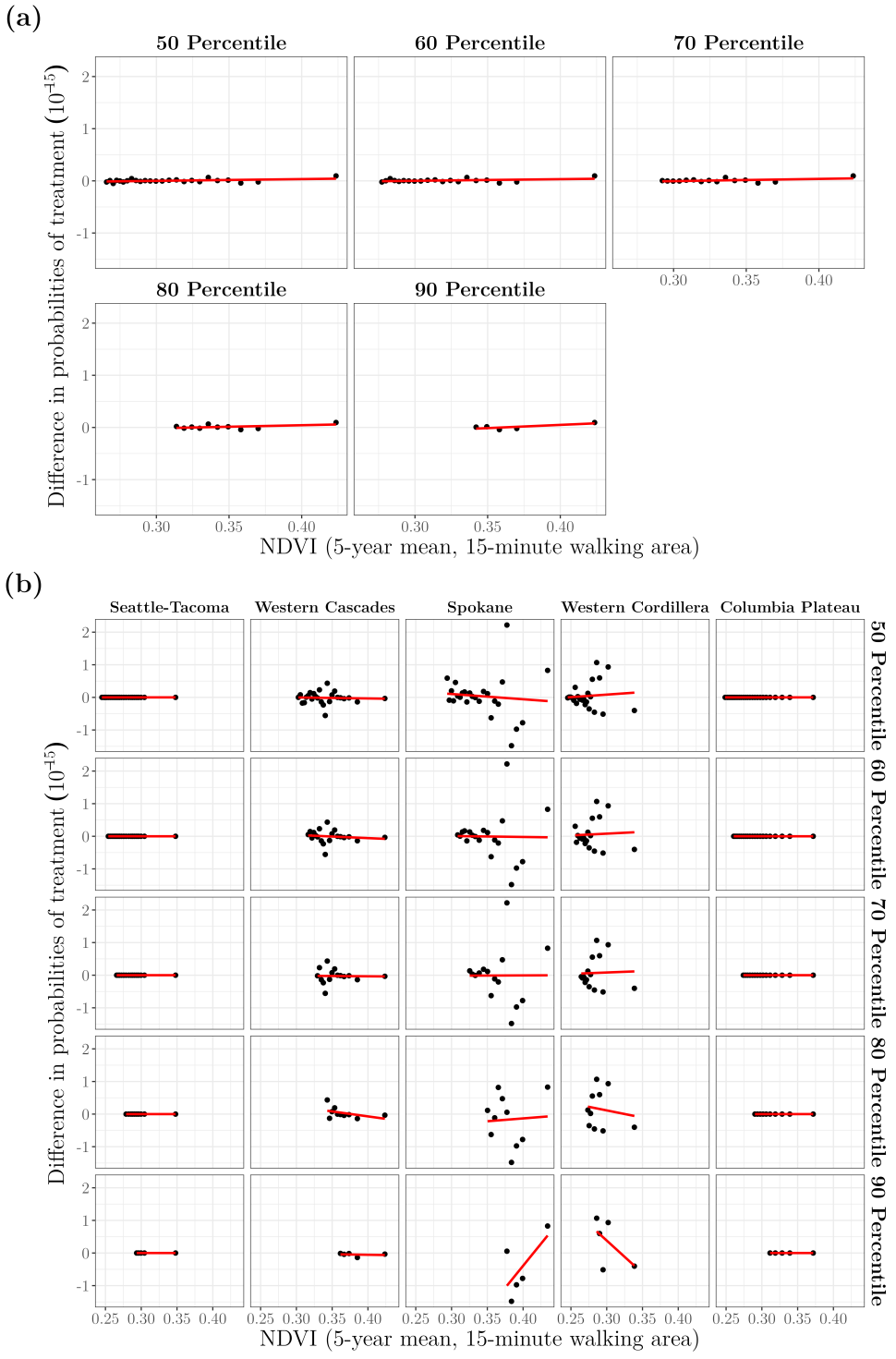
4.4 Results

4.4.1 Descriptive analysis. The majority of decedents in the State of Washington resided in the western part of the State, with 74.44 % living in the Seattle-Tacoma and Western Cascades areas (23,259 out of 31,245), which is proportional to the population distribution in the area (76.14 % or 5,867,317 out of 7,705,281 in the 2020 Census). The rate of mental illness mortality among the naturally deceased residents was 0.87 % (273 out of 31,245), resulting in a rate of 3.54 per 100,000 population (based on the 2020 Census population). The average age at death was 76.72 years, showing a mild right-skewness (mean = 76.72, median = 78.00). The racial composition was highly skewed, with 89.32 % of the decedents (27,909 out of 31,245) being non-Hispanic whites, while other racial groups accounted for 1.19 % (all others) to 3.85 % (Asian) (Table C.1). The decedents commonly had education beyond high school (86.65 %) and were married or widowed at the time of death (75.39 %). In terms of environmental exposures, NDVI exposure followed a normal distribution (mean = 0.27, standard deviation [sd] = 0.05). Similar normality was observed for MNDWI and NDBI. However, traffic noise exposure exhibited a highly right-skewed distribution (mean = 14.34, median = 8.90, sd = 15.58). Likewise, neighborhood socioeconomic covariates were predominantly right-skewed. To meet the linearity assumption in the matching analysis, these skewed variables were transformed using the square root (Table C.1).

4.4.2 Dichotomization conditions. The analysis demonstrated that the mechanism equivalence condition for dichotomization was satisfied across all thresholds. The mean difference between probability values in subgroups at 2 percentile intervals was consistently close to zero, with a small range in the entire study population. Similar results were observed in the subregional populations, except for Spokane and Western Cordillera, which exhibited higher variability in the differences compared to the other three subregions (Figure 6). Regarding effect equivalence, the probability of mental illness mortality remained stable across the entire population, ranging from 0.007 to 0.010 for every 2 percentile greenspace exposure interval. However, subregional results varied significantly. The probability of mental illness mortality remained stable in the Western Cascades and Columbia Plateau for every 2 percentile greenspace exposure interval. In Spokane, the probability showed random fluctuations along the greenspace exposure, while decreasing patterns were observed in the Seattle-Tacoma and Western Cordillera regions. Specifically, the probability mildly decreased at high exposure values in Seattle-Tacoma and sharply decreased up to NDVI = 0.3 in Western Cordillera. Consequently, I estimated the causal effects for the entire study population, Seattle-Tacoma, Western Cascades, and Columbia Plateau by ensuring the fulfillment of the equivalence conditions (Figure 7).

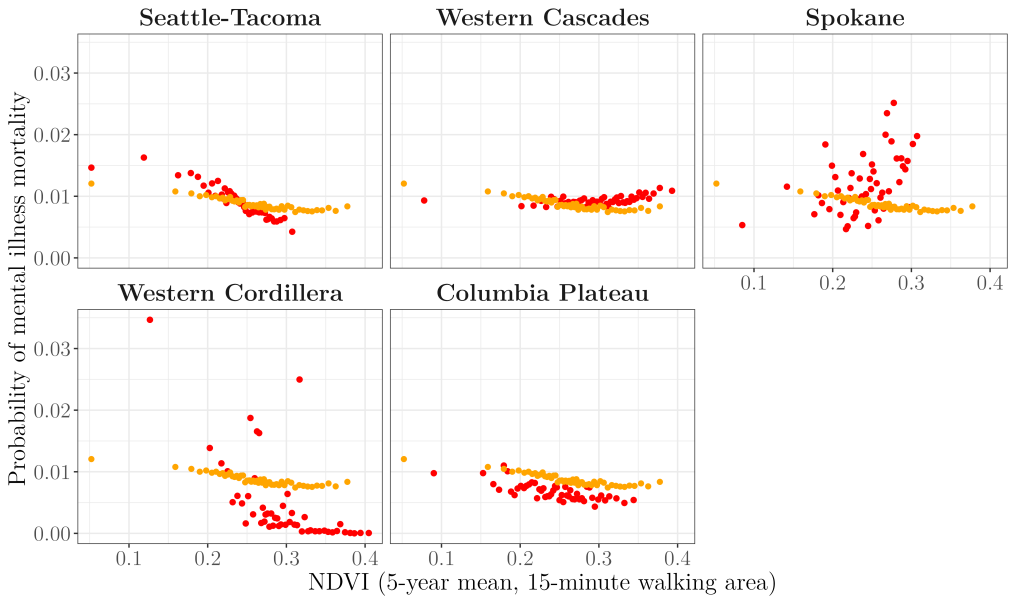
4.4.3 Matching analysis results. The results from the matching analysis performed on the entire study population revealed a mildly decreasing trend as the treatment was defined by higher percentiles. For instance, treatment defined by the 50th percentile or higher yielded an estimate of 0.30, while defining treatment by the 90th percentile or higher resulted in an estimate of -0.11. The subregional results helped to address the two primary research questions posed in this study.

Figure 6. Difference between treatment probability by dichotomization (a) in the entire study population and (b) among the five subregional populations at 2 percentile intervals across 50-90 percentile thresholds



Notably, these results showed that subregional causal effect estimates diverged from the global causal effect obtained from the entire study population (Figure 8). This observation provides evidence for spatial differences in causal effect estimates. More specifically, the effect estimates derived from relative treatment definitions using percentiles demonstrated a downward trend in the Seattle-Tacoma region (from 0.00 at the 50th percentile and higher to −0.41 at the 90th percentile and higher). Conversely, the Western Cascades and Columbia Plateau subregions showed an increasing trend in effect estimates, with a sudden drop observed at the 80th percentile (from −0.32 to 0.12 in Western Cascades and from 0.00 to 0.41 in Columbia Plateau) (Figure 8, panel (a)). On the other hand, defining the treatment in absolute terms by applying fixed NDVI values yielded highly volatile effect estimates. The range of estimates from the entire population fluctuated between −0.17 at a 0.29 threshold NDVI to 0.11 at a 0.28 threshold. Except for the Western

Figure 7. Average probability of mental illness mortality at 2 percentile intervals across 50-90 percentile greenspace exposure thresholds from local (red) and global (orange) data in the State of Washington



Cascades subregion, where estimates were consistently protective (ranging from -0.23 to -0.03) at thresholds above 0.29, subregional results showed variability. For instance, estimates for the Seattle-Tacoma region became zero at a threshold above 0.30 due to the absence of mental illness mortality cases in the treated group. Additionally, a sharp increase in effect estimates was observed in the Columbia Plateau subregion at thresholds above 0.29 (from -1.11 at a threshold of 0.29 to 0.00 at a threshold of 0.31) (Figure 8). In conclusion, the comparison between absolute and relative treatment definitions provided answers to the second research question.

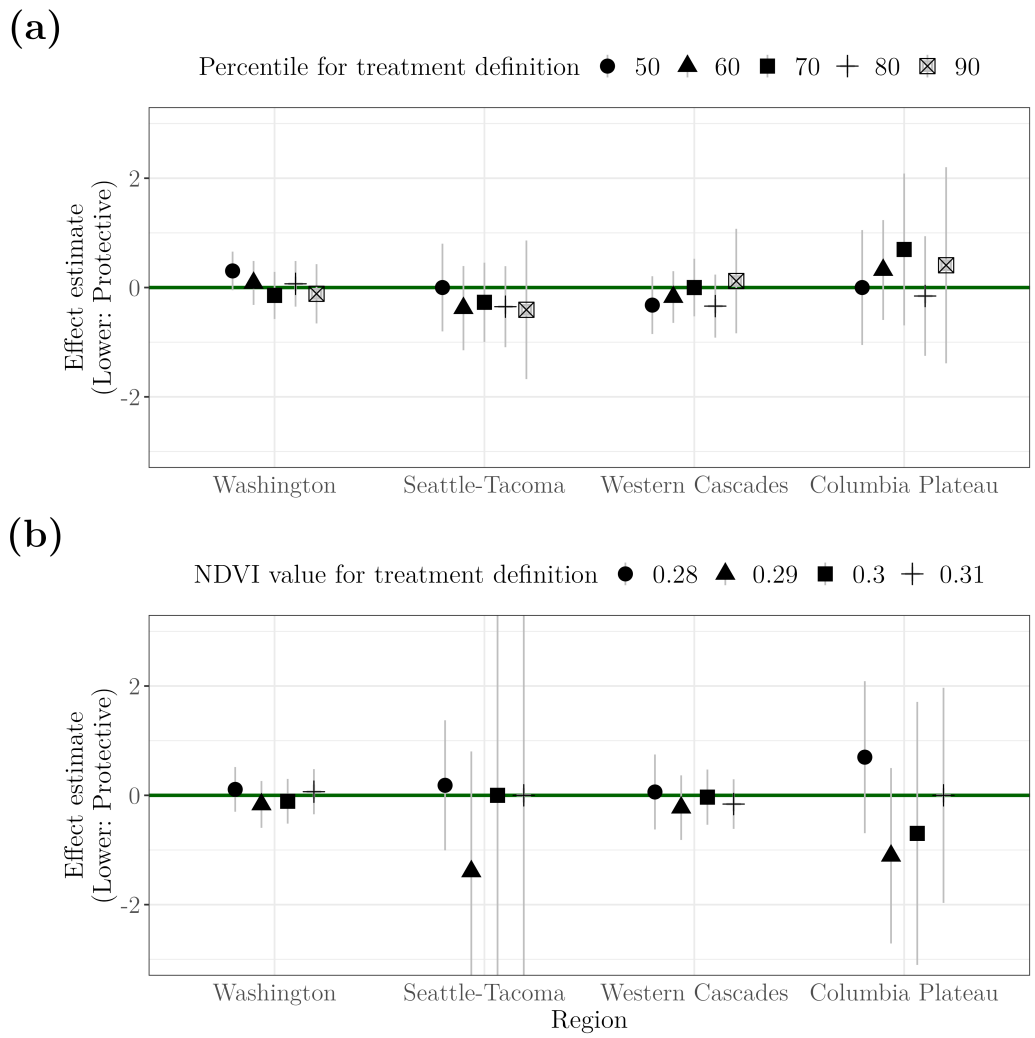
Individual-level covariates were well-balanced, whereas environmental exposures and neighborhood-level covariates exhibited varying magnitudes of difference. Most of the standardized mean differences between the treated and controlled groups ranged from -1 to 1, with NDBI being an exception (Figures C.1–C.2). Sensitivity analysis reveals that five metropolitan areas out of nineteen CBSAs met the conditions for reliable estimates. Among these, two metropolitan areas, Seattle-Tacoma-Bellevue and Spokane, showed consistent effect estimates. However, the remaining three areas displayed fluctuations in effect estimates and failed to produce reliable estimates at the 80th and 90th percentiles of the treatment definition (Figure C.2).

4.5 Discussion

This study examined spatial differences in causal effects of greenspace exposure on mental illness mortality. Additionally, the reliability of causal effect estimates using relative treatment definitions was assessed in subregions. By examining the equivalence conditions for the binary treatment definition, the

Figure 8. Causal effect estimates from the State of Washington and three subregions by percentiles (a) and values (b) of greenspace exposure thresholds

(Note: grey bars are 95 percent confidence interval from the simple regression of the NDVI treatment against mental illness mortality)



entire study population was divided into five subregions to estimate the causal effect of greenspace exposure on mental illness mortality. The results revealed that considering spatial subpopulations was crucial for identifying the causal effects of greenspace exposure on mental health outcomes. In other words, it is essential to compare greenspace exposure in a subregional context that is relevant to the greenspace itself, rather than using a globally fixed exposure value. This is particularly important in health effect studies of greenspace because its distribution is highly dependent on the physical environment. The use of relative treatment definitions in subregions allowed me to assess whether the effect of greenspace exposure on mental illness mortality varies depending on unique spatial contexts, supporting recent efforts to assess integrated environmental exposure assessment in a local context (J. Ha et al., 2022; Jarvis et al., 2020). The findings of this study suggest that locally defined greenspace treatment can effectively identify the causal effects, emphasizing the need to consider local environmental characteristics in exposure assessments for causal analysis.

The results of this chapter demonstrated that the causal effect of greenspace on mental illness mortality exhibits spatial variation. The causal effect estimates in subregions differ from those obtained for the entire study population. This finding contributes to the existing literature by identifying a spatially varying causal relationship between mental health outcomes and greenspace exposure, extending beyond previous findings on spatially varying associations (Labib et al., 2020). Furthermore, the results clearly demonstrate that the feasibility of the treatment definition is contingent upon the characteristics of the subregional population. It suggests that causal inference based on a binary treatment variable can be influenced by the spatial configuration of the subregional population. Further

research is warranted to examine more common mental health outcomes beyond mortality.

The balance diagnosis results of covariates provide additional insights into the intersection of greenspace exposure and socio-environmental composition. In Seattle-Tacoma and Western Cascades, the treated group exhibited much lower NDBI (approximately -2 standard deviations) compared to the control group. This can be partially attributed to the marked negative correlation between NDBI and NDVI. It implies that residential greenspace exposure is spatially segregated, similar to median household income and poverty rates. Environmental exposures, including $PM_{2.5}$ and traffic noise, are concentrated in urban centers or near roads, resulting in a slight imbalance in the matching results. This suggests that considering the spatial segregation of socio-environmental constructs is important in health effect studies of greenspace (Łaszkiewicz et al., 2021).

Despite the novel approach used to examine spatial differences in the effect of greenspace exposure on mental illness mortality, several limitations of this study should be acknowledged. First, the data limitations imposed strong assumptions for causal inference, which may not hold in reality. I was only able to identify a subset of decedents who were reported to have died from mental illness and directly affected or contributed to their death. As the onset of mental illness may precede the 5-year greenspace exposure, the results are susceptible to attribution errors. Second, I assumed no interference and mediation between covariates, which should be evaluated using suitable methods that explicitly handle these factors (Hernán & Robins, 2006). Third, processes operating at a finer spatial scale should be considered. This issue relates to controlling unmeasured spatial confounding by incorporating spatial distance between subjects in the study population

(Papadogeorgou et al., 2019). Lastly, the exposure assessment remained static, and therefore, the effect estimates obtained using dynamic exposure assessment should be compared with the results of this study (Helbich, 2018a; Henson et al., 2020; Yoo & Roberts, 2022). Unfortunately, in this case study, static exposure assessment was the only feasible option given the nature of the single-year cross-sectional data.

I suggest three directions for future research. First, more analysis cases with quality data are needed to reexamine the questions investigated in this study. Second, the treatment definition should incorporate the complex relationship between environmental exposure measured by satellite images and visible greenspace assessed by street images (Giannico et al., 2022; Villeneuve et al., 2018; R. Wang et al., 2021). Third, unconsidered conditions such as interference and mediation should be taken into account using flexible causal inference methods.

4.6 Conclusion

In conclusion, this chapter examined the causal effect of greenspace exposure on mental illness mortality and investigated the spatial differences in this effect across regions. The findings indicated that the causal effect of greenspace exposure on mental health outcomes varies across subregional populations, highlighting the importance of assessing the impact of greenspace exposure in spatial contexts that uniquely characterize the study population. For future research, it would be beneficial to expand the causal approach to a spatiotemporal setting, analyze longitudinal cohort data, and investigate potential spatiotemporal differences in the causal effect of greenspace exposure on mental illness mortality.

CHAPTER V

CONCLUSION

Mental illness has become a major health issue in recent years, which demands a comprehensive understanding of the factors that affect mental illness, both negatively and positively. This dissertation investigated the impacts of two important risk/protective factors of mental illness mortality, unemployment and greenspace, through three case studies with the special focus on their spatial and temporal variations. The primary objective of this research was to emphasize the importance of recognizing the spatial and/or temporal disparity in the effects of socio-environmental factors, which takes a step further from the conventional approach of describing outcome patterns and assuming constant effects across space and time in mental health studies in health geography and cognate fields. I addressed two research gaps that were found in the literature by explicitly examining the spatially and temporally varying, and causal effects of unemployment and greenspace exposure on mental illness mortality in subregions.

Each case study demonstrated that the effects of unemployment or greenspace exhibited clear spatial disparity (Chapters 2 and 3), and the contrast between low and high effects varied over time in response to significant events that impact the factors of interest (Chapter 2). The feasibility of estimating causal effects is influenced by the spatial partition of the study population, and spatial disparity was found in regions where causal estimation is feasible (Chapter 4). These findings highlight the significance of considering spatially and temporally varying effects and providing researchers and public health practitioners with

locally-focused measures. They can use this finding as evidence to discover unique but unknown factors and develop effective regional mental health promotion policies. The effectiveness of policies could be guaranteed if the spatial patterns of the spatiotemporal effects intersect the boundaries for policy implementation, such as jurisdictions and special geographic divisions for public health purposes (e.g., public health districts in the State of Georgia).

The most significant contribution of this dissertation to the mental health research community is to emphasize the importance and value of directing research focus from the universality to the uniqueness of local contexts in the associative and causal effects of contributing factors on mental illness. The results collectively filled research gaps in the literature by demonstrating the spatial and spatiotemporal variation in effects and the conditioning role of regions in the causal effect estimation. The findings indicate a way to expand the theory on the influence of socio-environmental constructs on mental illness mortality and the application of spatiotemporal and causal modeling methods to geospatial analysis of mental illness. The other contribution to the field is the introduction of spatiotemporal interaction models to account for spatial and temporal dimensions and dependency in the data. Through Chapters 2 and 3, I extended the spatially varying coefficient models to spatiotemporally varying coefficient models, on top of explicitly including the interaction between spatial and temporal dependency in the outcomes, thereby demonstrated the usefulness of Bayesian spatiotemporal models for *effect mapping* beyond the traditional *disease mapping* in spatial mental health research contexts. Spatiotemporal models could prove its usefulness in more applications by the increasing availability of sizable spatiotemporal datasets in many countries. The

effect mapping approach is expected to reexamine the spatial disparity in socio-environmental effects on mental illness.

Future research, which takes into account an extensive set of contributing factors of mental illness such as individual-level medical history and genetic information, has the potential to corroborate the spatial disparities shown in this dissertation. Data with fine-grained spatiotemporal information, along with detailed individual attributes, will provide more opportunities to examine the spatially and temporally varying effects of socio-environmental factors on mental illness. Methodologically, it is feasible to expand the spatiotemporally varying coefficient models to multiple explanatory variables. However, this will require scalable computational infrastructure and efficient numerical methods, which are currently under development (Gaedke-Merzhäuser et al., 2022; Niekerk et al., 2021), to reveal simultaneous transitions in the effects of such variables. For spatial causal inference, I am working on developing a novel similarity matrix that combines a spatial weight matrix and Jensen-Shannon divergence for matching analysis to efficiently handle spatial confounding. A successful development will lead to the reliable estimation of the causal effect of explanatory factors on mental health outcomes. For exposure assessment, it is of utmost priority for researchers to develop a metric for the holistic assessment of exposure to physical and socioeconomic environments by accounting for their interaction. Such development needs to be in line with the critical re-engagement in the exposure's role along with considering physical activity, modalities of activities (e.g., leisure and commuting), and the effective exposures that actually affect mental health outcomes.

On the theoretical side, my focus is on spatial generalizability (or *external validity*) of associative or causal effects of contributing factors on mental illness.

In this context, spatial generalizability refers to the feasibility of extrapolating associative or causal findings from a local or higher-level spatial entity to other locals. This issue addresses two long-standing quests of academic geography: setting the relationship between nomothetic (seeking laws) and idiographic (describing characteristics) approaches and closely examining the meaning of geographic scales. There are virtually infinite possibilities that a local effect estimate may have no relationship with the global effect, or it may align perfectly with the global effect. All possibilities call for the reconciliation and positive feedback of nomothetic and idiographic approaches to understand the possible processes, if not the very reasons, of spatial disparities in the effects of socio-environmental constructs on mental illness and its mortality. Cross-scalar validity of an effect estimate will help us understand the tangible role of spatial scales in mental illness and to other mental health outcomes. Such investigations can encourage further efforts to gain deeper insights into tackling ecological and atomistic fallacies (Robertson & Feick, 2018). Based on this dissertation work, I will continue to reflect on the fundamental issues with empirical studies on mental illness and general mental health outcomes.

APPENDIX A

CHAPTER 2 APPENDIX

A.1 Modeling spatiotemporal interaction

Let there be spatial entities comprise nine square areas as below, with the temporal dependence of the first-order random walk and let the total length of the time series be three.

1	2	3
4	5	6
7	8	9

In this case, area 1 is assumed to be adjacent to areas 2, 4 and 5, area 2 is adjacent to areas 1, 3, 4, 5 and 6, and so on. Each structure can be expressed in matrices, say, $\mathbf{W}_S$ and $\mathbf{W}_T$:

$$\mathbf{W}_S = \begin{pmatrix} 0 & 1 & 0 & 1 & 1 & 0 & 0 & 0 & 0 \\ 1 & 0 & 1 & 1 & 1 & 1 & 0 & 0 & 0 \\ 0 & 1 & 0 & 0 & 1 & 1 & 0 & 0 & 0 \\ 1 & 1 & 0 & 0 & 1 & 0 & 1 & 1 & 0 \\ 1 & 1 & 1 & 1 & 0 & 1 & 1 & 1 & 1 \\ 0 & 1 & 1 & 0 & 1 & 0 & 0 & 1 & 1 \\ 0 & 0 & 0 & 1 & 1 & 0 & 0 & 1 & 0 \\ 0 & 0 & 0 & 1 & 1 & 1 & 1 & 0 & 1 \\ 0 & 0 & 0 & 0 & 1 & 1 & 0 & 1 & 0 \end{pmatrix} \tag{A.1}$$

$$\mathbf{D} = \text{diag}\left(\{3, 5, 3, 5, 8, 5, 3, 5, 3\}\right) \tag{A.2}$$

$$\mathbf{D} - \mathbf{W}_S = \begin{pmatrix} 3 & -1 & 0 & -1 & -1 & 0 & 0 & 0 & 0 \\ -1 & 5 & -1 & -1 & -1 & -1 & 0 & 0 & 0 \\ 0 & -1 & 3 & 0 & -1 & -1 & 0 & 0 & 0 \\ -1 & -1 & 0 & 5 & -1 & 0 & -1 & -1 & 0 \\ -1 & -1 & -1 & -1 & 8 & -1 & -1 & -1 & -1 \\ 0 & -1 & -1 & 0 & -1 & 5 & 0 & -1 & -1 \\ 0 & 0 & 0 & -1 & -1 & 0 & 3 & -1 & 0 \\ 0 & 0 & 0 & -1 & -1 & -1 & -1 & 5 & -1 \\ 0 & 0 & 0 & 0 & -1 & -1 & 0 & -1 & 3 \end{pmatrix} \tag{A.3}$$

$$\mathbf{W}_T = \begin{pmatrix} 1 & -1 & 0 \\ -1 & 2 & -1 \\ 0 & -1 & 1 \end{pmatrix} \tag{A.4}$$

Matrices $\mathbf{W}_S$ and $\mathbf{W}_T$ are sparse as seen above. By the definition of Kronecker product, their interaction term is

$$(\mathbf{D}-\mathbf{W}_S)\otimes\mathbf{W}_T = \begin{pmatrix}
3 & -3 & 0 & 1 & -1 & 0 & \cdots & 0 & 0 & 0 & 0 & 0 & 0 \\
-3 & 6 & -3 & -1 & 2 & -1 & \cdots & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & -3 & 2 & 0 & -1 & 1 & \cdots & 0 & 0 & 0 & 0 & 0 & 0 \\
-1 & 1 & 0 & 5 & -5 & 0 & \cdots & 0 & 0 & 0 & 0 & 0 & 0 \\
1 & -2 & 1 & -5 & 10 & -5 & \cdots & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 1 & -1 & 0 & -5 & 5 & \cdots & 0 & 0 & 0 & 0 & 0 & 0 \\
\vdots & \vdots & \vdots & \vdots & \vdots & \vdots & \ddots & \vdots & \vdots & \vdots & \vdots & \vdots & \vdots \\
0 & 0 & 0 & 0 & 0 & 0 & \cdots & 5 & -5 & 0 & -1 & 1 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & \cdots & -5 & 10 & -5 & -1 & 2 & -1 \\
0 & 0 & 0 & 0 & 0 & 0 & \cdots & 0 & -5 & 5 & 0 & -1 & 1 \\
0 & 0 & 0 & 0 & 0 & 0 & \cdots & 1 & -1 & 0 & 3 & -3 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & \cdots & -1 & 2 & -1 & -3 & 6 & -3 \\
0 & 0 & 0 & 0 & 0 & 0 & \cdots & 0 & -1 & 1 & 0 & -3 & 3
\end{pmatrix} \tag{A.5}$$

The result should have $(9 \times 3) \times (9 \times 3) = 27 \times 27$ elements. To note, when the spatial or temporal structure was imposed as identically independently distributed (i.i.d.), $\mathbf{W}_T$ equals to an identity matrix. In this study, the dimensions of the precision matrix will be $(3,108 \times 14) \times (3,108 \times 14) = 43,512 \times 43,512$.

Table A.1. Pearson correlation matrix of dependent and independent variables

	One-year lagged unemployment rate	Median household income (inflation-adjusted dollars as of 2000)	Ratio of population below poverty level	Ratio of rented housing	Ratio of population higher than bachelor's degree	Ratio of non-white population	Ratio of rural population	Ratio of single-person households	Ratio of elderly population
One-year lagged unemployment rate		-0.266	0.425	0.043	-0.221	0.282	-0.002	-0.001	-0.018
Median household income (inflation-adjusted dollars as of 2000)	-0.266		**-0.727**	-0.002	**0.670**	-0.159	-0.396	-0.331	-0.358
Ratio of population below poverty level	0.425	**-0.727**		0.237	-0.353	**0.518**	0.160	0.109	-0.025
Ratio of rented housing	0.043	-0.002	0.237		0.403	0.398	**-0.507**	0.265	-0.336
Ratio of population higher than bachelor's degree	-0.221	**0.670**	-0.353	0.403		-0.002	-0.435	0.106	-0.255
Ratio of non-white population	0.282	-0.159	**0.518**	0.398	-0.002		-0.185	-0.049	-0.339
Ratio of rural population	-0.002	-0.396	0.160	**-0.507**	-0.435	-0.185		0.085	0.411
Ratio of single-person households	-0.001	-0.331	0.109	0.265	0.106	-0.049	0.085		**0.576**
Ratio of elderly population	-0.018	-0.358	-0.025	-0.336	-0.255	-0.339	0.411	**0.576**	

Note: Absolute values higher than 0.5 were highlighted.

Table A.2. Variance inflation factor (VIF) of each variable

Variable	VIF
The one-year lagged unemployment rate (%)	1.293
Median household income (Inflation-adjusted as of 2000)	6.816
The ratio of non-white population (%)	1.814
The ratio of the population with or higher than bachelor's degree (%)	3.444
The ratio of the population below the poverty level (%)	4.365
The ratio of the rural population (%)	1.781
The ratio of renter-occupied housing (%)	2.596
The ratio of single-person households (%)	2.788
The ratio of the elderly population (65 years old or older, %)	2.729

Table A.3. Summary of model hyperparameters of from Model 5-IV: summary statistics of posterior marginals and 95 % credible intervals of precisions for each model component

Precision	Posterior mean	Standard deviation	2.5 %	Median	97.5 %
τ_{u_0}	5.01	0.14	5.22	5.49	5.78
τ_{v_0}	5005.34	50.52	4064.76	4989.64	6043.56
τ_{γ_0}	40.84	24.37	9.22	35.95	101.53
ρ_0	0.94	0.04	0.84	0.95	0.99
τ_{u_1}	2006.20	202.50	1638.24	1995.97	2433.87
τ_{γ_1}	33191.22	13185.79	14750.80	30751.57	65774.97
τ_δ	384.28	12.38	360.70	384.02	409.32

Figure A.1. Temporal trends of unemployment rates (grey lines) and mental illness and substance use mortality rates (red lines) in 3,108 contiguous US counties and the overall trends (dashed bold lines)

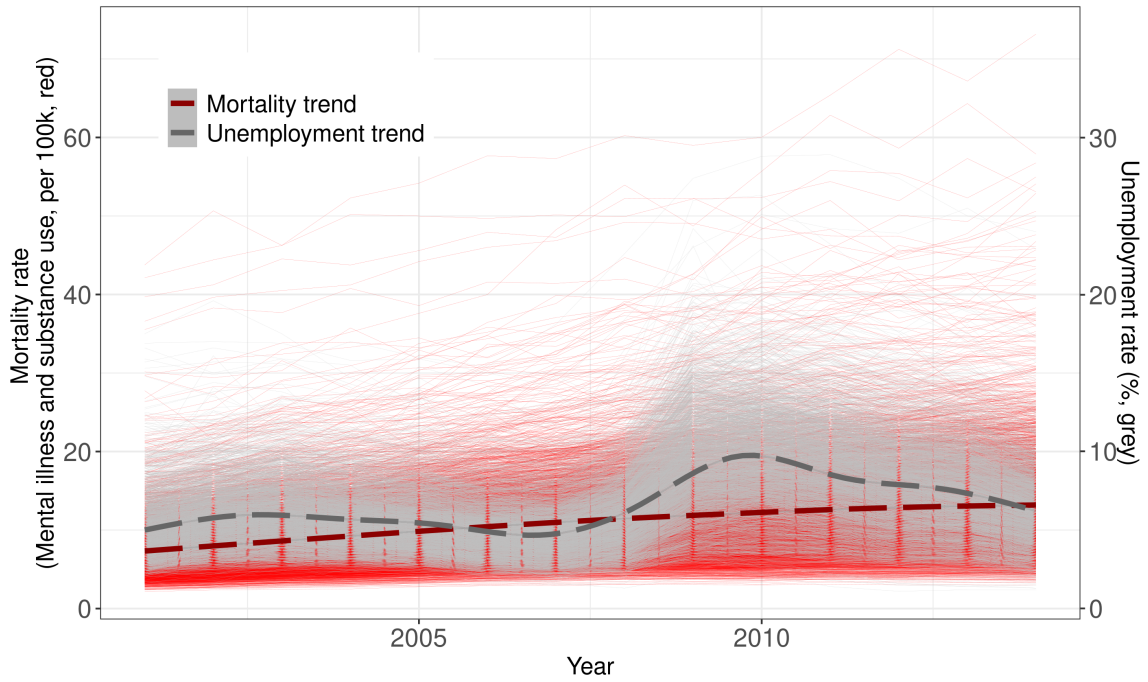


Figure A.2. Spatiotemporal distribution of one-year lagged unemployment rates in 3,108 contiguous US counties in 2001-2014

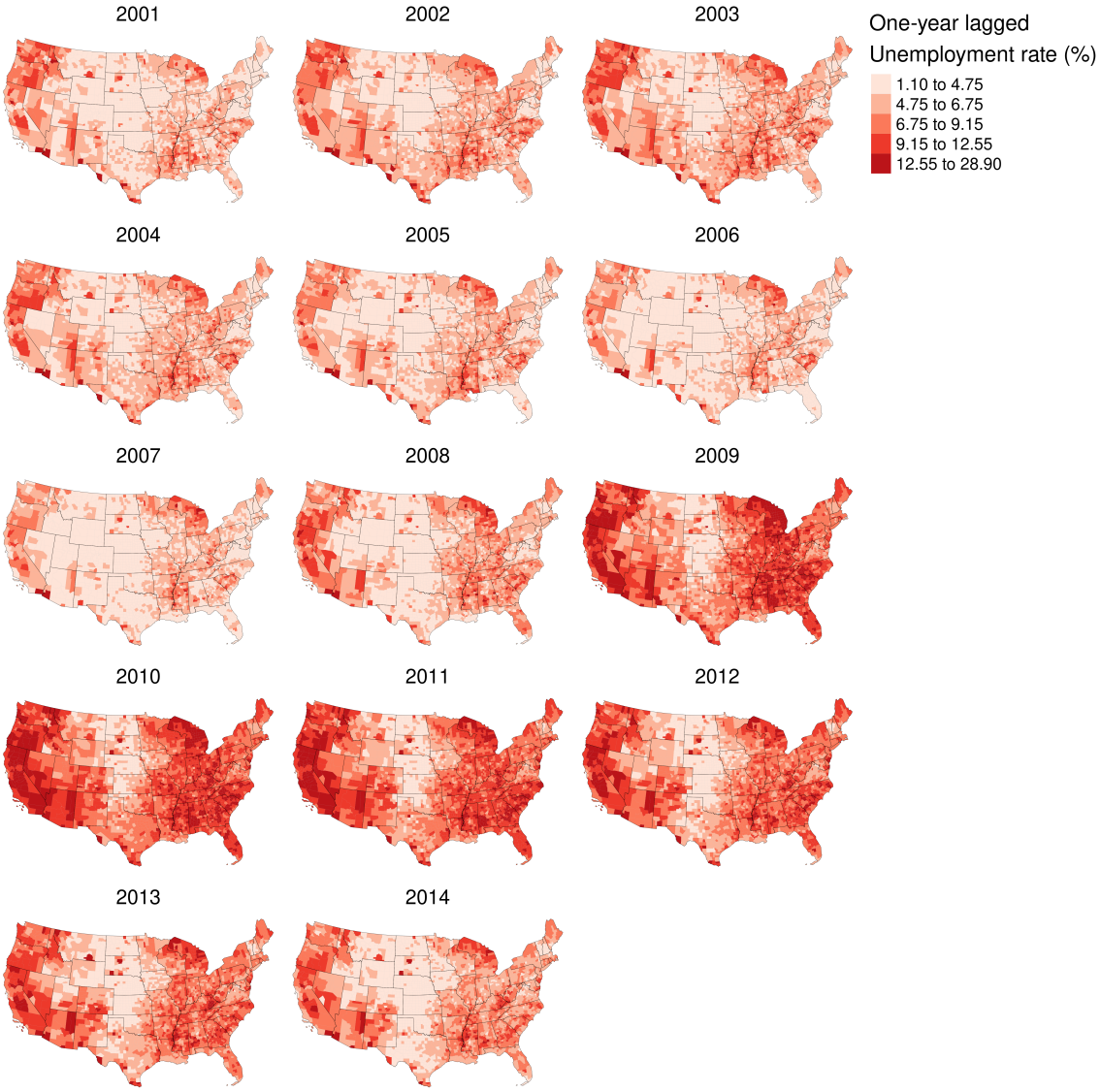


Figure A.3. Spatiotemporal distribution of mental illness and substance-use mortality rates (per 100,000 people) in 3,108 contiguous US counties in 2001-2014

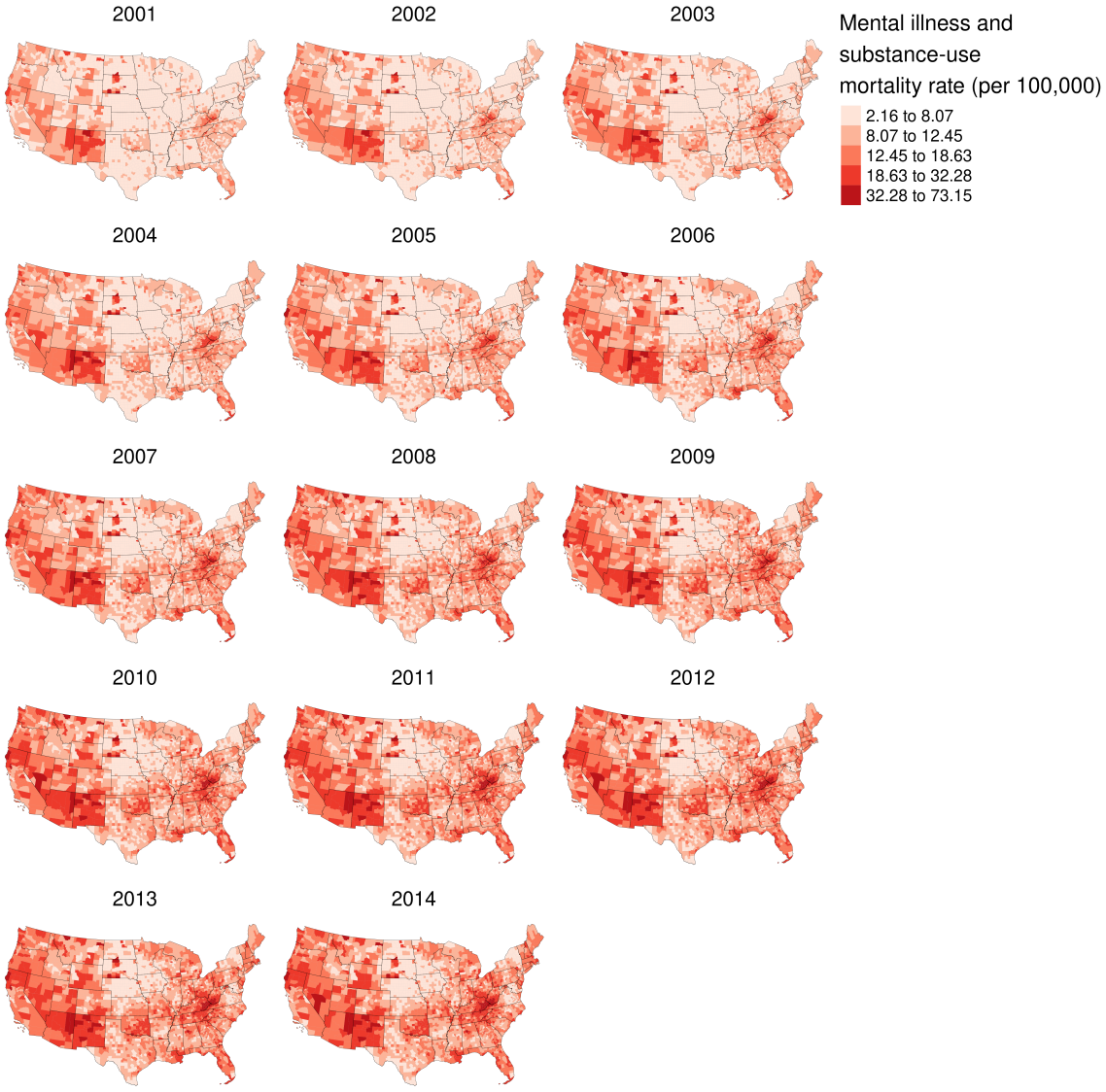


Figure A.4. Probability map of the random slope of unemployment rates in Model 5-IV

(Note: the map displays the probability of $\beta_{1it} > 0$)

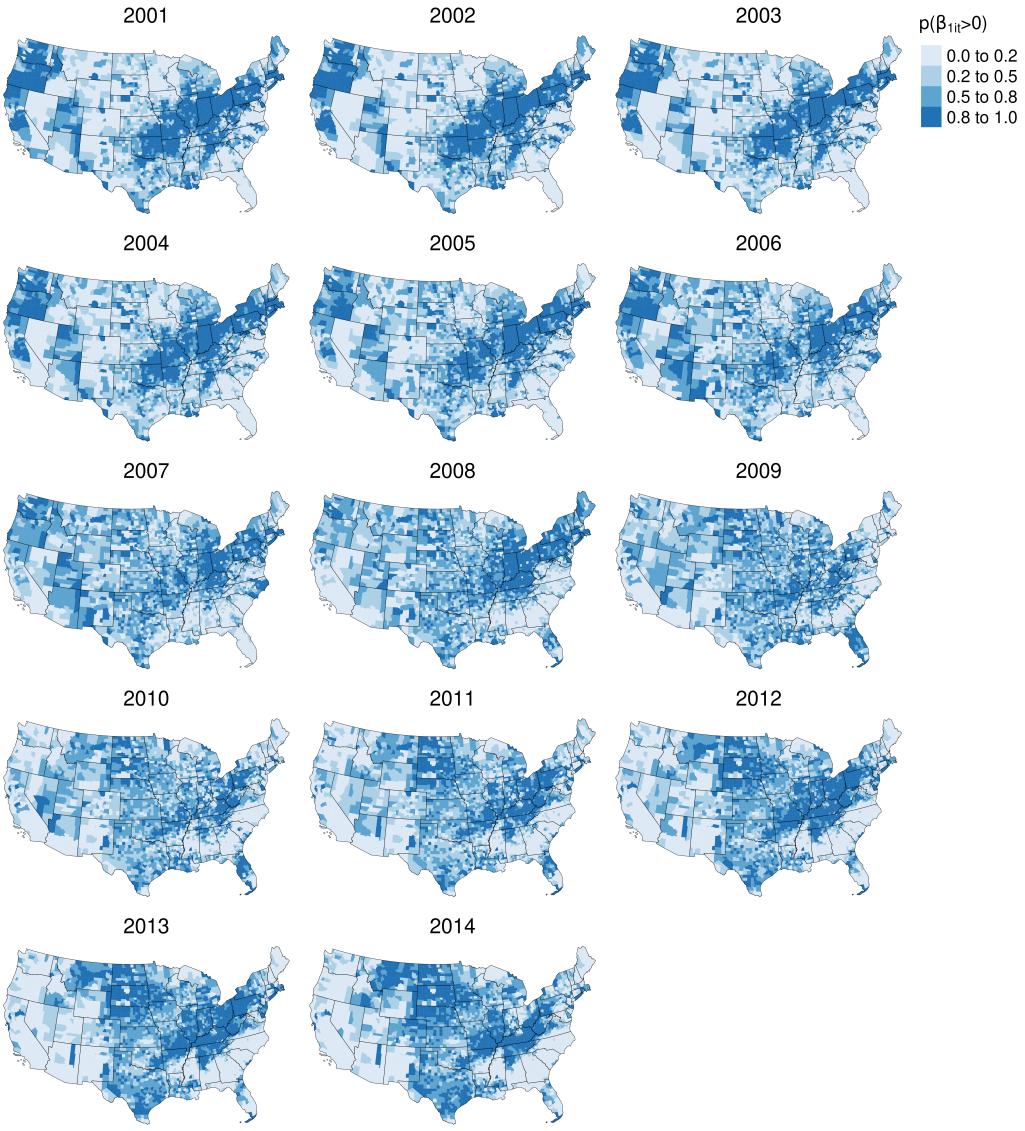


Figure A.5. Residual maps of Model 5-IV.

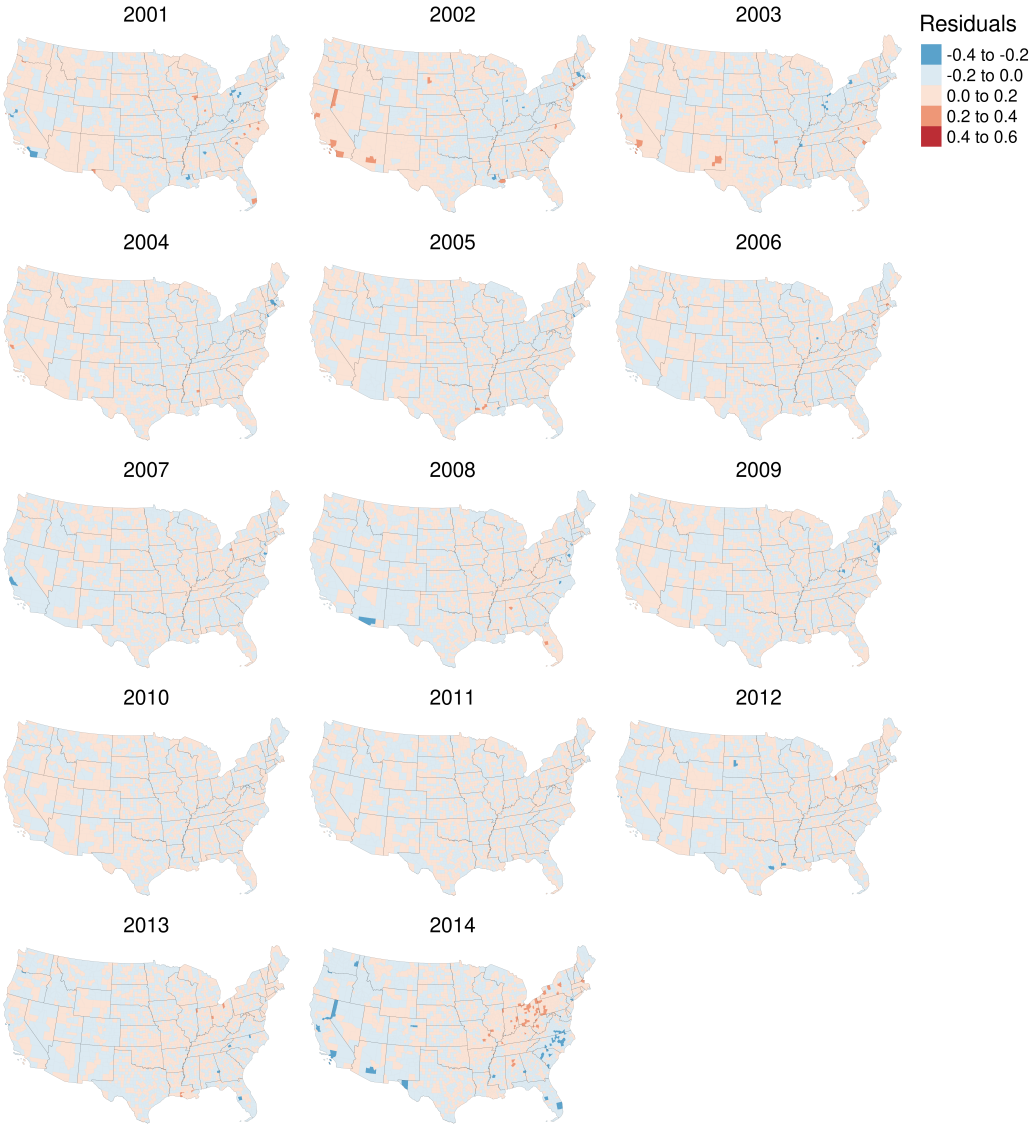
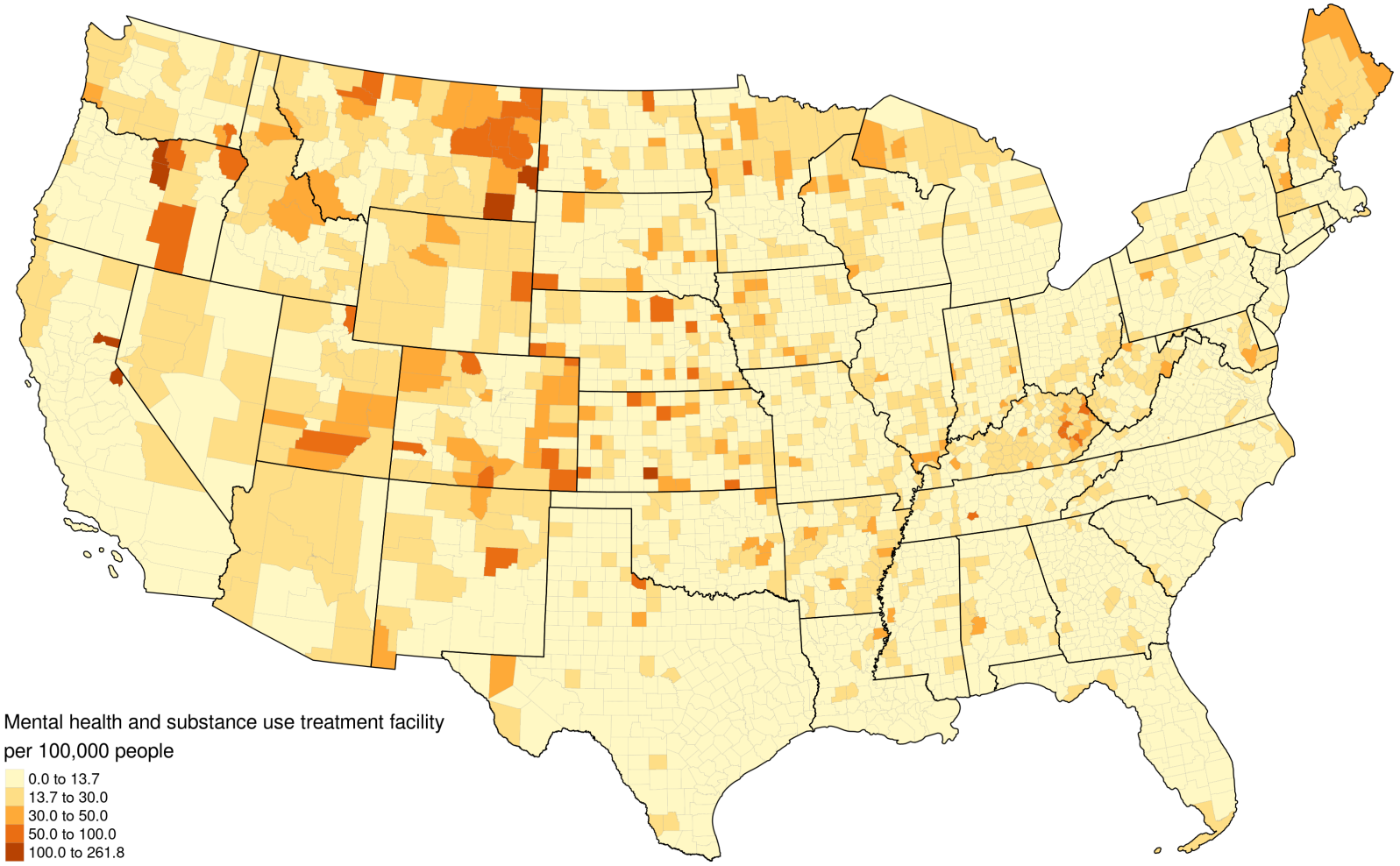


Figure A.6. Mental health and substance use treatment facility per 100,000 people in counties in the contiguous United States (2018)



Data source: Substance Abuse and Mental Health Services Administration

APPENDIX B

CHAPTER 3 APPENDIX

B.1 Detailed descriptions on spatiotemporal models

B.1.1 Spatiotemporal areal model formulation. Let $\mathbf{b}$ a vector of a spatial random main effect $b_{i\cdot}$ given a binary spatial weight matrix (also called graph) $\mathbf{W}$. $\mathbf{W}$ is usually defined by distance or sharing vertex between input spatial entities and has zeros in its diagonal, which means there is the self is not considered as a neighbor, and strictly being symmetric. The modified Besag-York-Mollié formulation (Besag et al., 1991; Riebler et al., 2016) is expressed as:

$$\mathbf{b}|\mathbf{W} = \frac{1}{\sqrt{\tau_b}}\left(\sqrt{1-\varphi}\mathbf{v} + \sqrt{\varphi}\mathbf{u}_*\right) \tag{B.1}$$

where $\mathbf{v}$ and $\mathbf{u}$ are vectors of spatially unstructured and structured components, respectively. The asterisk in the subscript of $\mathbf{u}_*$ means that the structured component is scaled. The mixing hyperparameter φ, which is bounded between 0 and 1, controls the contribution of each component to the variance of the random vector $\mathbf{b}$. The covariance matrix of $\mathbf{b}$ when the τ_b and φ are given turns out to be:

$$\mathrm{Var}(\mathbf{b}|\tau_b, \varphi, \mathbf{W}) = \tau_b^{-1}\left((1-\varphi)\mathbf{I} + \varphi\mathbf{Q}_*^-\right) \tag{B.2}$$

where $\mathbf{Q}_*^-$ is a scaled precision (inverse covariance) matrix and $\mathbf{I}$ is an identity matrix. The unscaled covariance matrix $\mathbf{Q}$ is modeled as a Besag model, which is also known as an intrinsic conditional autoregressive model given $\mathbf{W}$.

$$\mathbf{Q} = \mathbf{D} - \mathbf{W} \tag{B.3}$$

where $\mathbf{D} = \text{diag}(\text{vec}\left[\sum_{j=1}^{N} w_{ij}\right])$ in which diag(.) is a diagonal matrix with a vector of length N and w_{ij} is (i, j) element of $\mathbf{W}$. Formulae B.1–B.3 are also applied to the spatial modeling for the random slope component β_{i1}. Here a condition $\text{vec}\left[\sum_{j=1}^{N} w_{ij}\right]_i > 0 \quad \forall \ i$ should be fulfilled, which means all areas have at least one neighbor.

The main temporal component was structured in a first-order autoregressive model.

$$\gamma_{\cdot t} = \rho_0 \gamma_{\cdot(t-1)} + \varepsilon_{\gamma t} \tag{B.4}$$

The autoregressive parameter ρ_0 models the dependence of the value at the present time point (t) on the value of the previous time point $(t-1)$. $\varepsilon_{\gamma t}$ is a white noise process that models remaining variations in the temporal trend. In our analysis, Formula B.4 represents the overall trend in the crude mental illness mortality rate.

The spatiotemporal interaction (STINT) term δ_{it} represents the convolution between spatial and temporal processes in its precision. Four STINT types are identified by independent or structured spatial and temporal model precisions (formula B.5) (Knorr-Held, 2000).

$$\text{Precision}(\delta_{it}) = \mathbf{R}_\delta = \begin{cases} \mathbf{I} \otimes \mathbf{I} & \cdots \text{ STINT type I} \\ \mathbf{I} \otimes \mathbf{R}_\gamma & \cdots \text{ STINT type II} \\ \mathbf{R}_u \otimes \mathbf{I} & \cdots \text{ STINT type III} \\ \mathbf{R}_u \otimes \mathbf{R}_\gamma & \cdots \text{ STINT type IV} \end{cases} \tag{B.5}$$

$\otimes$ denotes Kronecker product, $\mathbf{R}_\gamma$ is a temporally structured precision matrix such as pth-order autoregressive or random walk, and $\mathbf{R}_u$ is a spatially structured precision matrix when $\mathbf{W}$ is given. Model 5 with STINT type IV is formulated as Formula B.6:

$$\begin{aligned}
&\begin{cases} p(y_{it} = 0) = p_{\text{zero}} + (1 - p_{\text{zero}}\text{Poisson}(\lambda_{it} = 0)) \\ p(y_{it} > 0) = (1 - p_{\text{zero}})\text{Poisson}(\lambda_{it} > 0) \end{cases} \\
&\psi_{it} = \log \lambda_{it} \\
&\psi_{it} = \log P_{it} + b_{i\cdot} + \beta_0 + \beta_{i1+} x_{it1} + \textstyle\sum_{k=2}^{p} \beta_k x_{itk} + \varepsilon_{it} \\
&\mathbf{b}|\mathbf{W} = \tfrac{1}{\sqrt{\tau_b}} \left(\sqrt{1-\varphi}\mathbf{v} + \sqrt{\varphi}\mathbf{u}_* \right), \quad \varphi_b \sim \text{PC}(0.5, 0.5), \ \tau_b \sim \text{PC}(1, 0.01) \\
&\gamma_{\cdot t} \sim \mathcal{N}\left(0, \left(\tau_\gamma (1-\rho_0)^2\right)^{-1}\right), \ \gamma_{\cdot t} = \rho_0 \gamma_{\cdot(t-1)} + \varepsilon_{\gamma t}, \ \rho_0 \sim \mathcal{N}\left(0, \tau_\gamma^{-1}\right), \varepsilon_{\gamma t} \sim \mathcal{N}\left(0, \tau_{\varepsilon_t}^{-1}\right) \\
&\beta_{i1+} = \beta_0 + \beta_{i1} \\
&\text{Precision}(\delta_{it}) = \mathbf{R}_\delta = \mathbf{R}_b \otimes \mathbf{R}_\gamma \\
&\{\beta_{i1}\} = \mathbf{b}_1 = \tfrac{1}{\sqrt{\tau_{b_1}}} \left(\sqrt{1-\varphi_{b_1}}\mathbf{v}_1 + \sqrt{\varphi_{b_1}}\mathbf{u}_{1*} \right), \ \varphi_{b_1} \sim \text{PC}(0.5, 0.5), \ \tau_{b_1} \sim \text{PC}(1, 0.01) \\
&\varepsilon_{it} \sim \mathcal{N}(0, \tau_\varepsilon^{-1})
\end{aligned} \tag{B.6}$$

The model is applied to the data of each state.

B.1.2 Description of hyperparameter estimates.

The estimated precision of main random effects was the lowest in the STINT component (mean=5.149–5.232), followed by that of the spatial component (9.28), and the temporal component (41.003–148.712). The estimated probability of structural zeros was 0.7–0.9 % (95 % credible interval [CrI] = [0.1 %, 2.2 %] in Oregon, [0.1 %, 3.4 %] in Washington) (Table B.7), which indicates that tracts had low probability of having no mental illness mortality. The precision of state-level random slope was around ten times higher (mean = 450.739–1338.804) than that of the temporal random effect (Table B.8).

B.2 Supplementary tables and figures

Table B.1. List of North American Industry Classification System (NAICS) codes for identifying care facilities

Code	Description
53111008	Retirement Apartments & Hotels
62311001	Adult Care Facilities
62311002	Convalescent Homes
62311008	Homes & Institutions
62311010	Homes-Personal Care Facility
62311011	Hospices
62311012	Independent Living Services for Disabled
62311013	Intermediate Care Facilities
62311014	Life Care Communities
62311015	Long Term Care Facility
62311016	Nursing Care Facilities
62311017	Nursing & Personal Care NEC
62311018	Nursing Home Services
62331101	Retirement Communities & Homes
62331103	Skilled Nursing Care Facilities
62331201	Adult Congregate Living Facilities
62331204	Senior Citizens Housing
62331205	Senior Citizens Service Organizations
62331206	Residential Care Homes
62399002	Other Residential Care Facilities
62399017	Sheltered Care Homes

Table B.2. Principal component analysis results for deriving socioeconomic deprivation index

	Correlation with the principal component	
	Oregon	*Washington*
Median household income (inflation-adjusted 2010 dollars)	0.892	0.893
Poverty rate(%)	-0.798	0.819
Unemployment rate (%)	-0.708	-0.734
Higher than bachelor's degree (%)	0.700	0.760

Table B.3. Correlation matrix of explanatory variables

Oregon

	MIM	NPARKS_SQKM10K	PM2.5	PCTWATER	CARE10K	PCTELDERLY	PCTNMARITAL	PCTNWHITE	PCTUNEMP	PCTPOVERTY	PCTEDUC
NPARKS_SQKM10K	-0.056										
PM2.5	-0.028	-0.190									
PCTWATER	-0.020	0.122	-0.045								
CARE10K	-0.070	0.609	-0.098	0.102							
PCTELDERLY	0.190	0.076	-0.416	0.069	0.054						
PCTNMARITAL	0.171	0.086	0.077	0.133	0.145	0.341					
PCTNWHITE	-0.044	-0.051	0.381	-0.060	-0.009	-0.454	0.067				
PCTUNEMP	0.064	-0.023	0.054	0.009	-0.005	-0.013	0.280	0.189			
PCTPOVERTY	0.069	-0.030	0.220	0.022	0.046	-0.169	0.259	0.376	0.454		
PCTEDUC	-0.150	-0.028	0.115	-0.021	-0.025	-0.172	-0.430	-0.145	-0.371	-0.272	
MEDINC10K	-0.156	0.049	-0.090	-0.061	-0.018	-0.152	-0.501	-0.183	-0.432	-0.700	0.578

Washington

	MIM	NPARKS_SQKM10K	PM2.5	PCTWATER	CARE10K	PCTELDERLY	PCTNMARITAL	PCTNWHITE	PCTUNEMP	PCTPOVERTY	PCTEDUC
NPARKS_SQKM10K	-0.038										
PM2.5	0.002	-0.195									
PCTWATER	-0.017	0.049	-0.011								
CARE10K	-0.030	0.107	-0.065	0.017							
PCTELDERLY	0.156	0.103	-0.210	0.047	0.059						
PCTNMARITAL	0.142	-0.045	0.189	-0.035	0.137	0.381					
PCTNWHITE	0.002	-0.139	0.202	0.001	-0.049	-0.321	0.091				
PCTUNEMP	0.018	-0.046	0.060	-0.038	0.038	-0.049	0.296	0.221			
PCTPOVERTY	0.058	-0.066	0.195	-0.052	0.038	-0.116	0.343	0.400	0.524		
PCTEDUC	-0.058	0.013	-0.047	0.243	-0.051	0.007	-0.426	-0.180	-0.418	-0.378	
MEDINC10K	-0.118	0.060	-0.164	0.216	-0.061	-0.118	-0.563	-0.234	-0.465	-0.700	0.656

Notes

- All correlation coefficients are statistically significant at (α=0.01).
- Variables except for mental illness mortality were standardized.

Abbreviations

MIM: mental illness mortality
PARKS_SQKM10K: the total park area (km^2) per 10,000 population
PM2.5: average $PM_{2.5}$ ($\mu g/m^3$)
PCTWATER: the proportion of waterbodies (%)
CARE10K: residential care facilities per 10,000 population
PCTELDERLY: 65 years old or older (%)
PCTNMARITAL: negative marital status (%)
PCTNWHITE: the non-white population (%)
PCTUNEMP: unemployment rate (%)
PCTPOVERTY: poverty rate (%)
PCTEDUC: higher than bachelor's degree (%)
MEDINC10K: median household income (inflation-adjusted 2010 dollars)

Table B.4. Variance inflation factors of explanatory variables

	Oregon	Washington
Parks (km^2 /10,000 population)	1.110	1.069
$PM_{2.5}$ ($\mu g/m^3$)	1.441	1.226
Area of waterbodies (%)	1.040	1.078
Residential care facilities (per 10,000 population)	1.099	1.059
65 years old or older (%)	1.950	1.824
Negative marital status (%)	1.745	2.012
Non-white population (%)	1.575	1.387
Unemployment rate (%)	1.343	1.445
Poverty rate (%)	2.935	2.837
Higher than bachelor's degree (%)	1.642	1.881
Median household income (inflation-adjusted 2010 dollars)	3.572	3.713

Table B.5. List of twenty-eight excluded census tracts and the reasons of exclusion

State	GEOID[1]	Full tract name	Reason	Key geographic feature
OR	41007990000	Clatsop County, Tract 9900	Water area	
OR	41011990101	Coos County, Tract 9901.01	Water area	
OR	41015990101	Curry County, Tract 9901.01	Water area	
OR	41019990000	Douglas County, Tract 9900	Water area	
OR	41039990000	Lane County, Tract 9900	Water area	
OR	41041990100	Lincoln County, Tract 9901	Water area	
OR	41045940000	Malheur County, Tract 9400	Missing or no population	Paiute and Shoshone Tribe Reserve
OR	41051980000	Multnomah County, Tract 9800	Missing or no population	Swan Island Industrial Park
OR	41057990100	Tillamook County, Tract 9901	Water area	
WA	53005012000	Benton County, Tract 120	Missing or no population	Hanford nuclear facility cleaning site
WA	53009990100	Clallam County, Tract 9901	Water area	
WA	53021980100	Franklin County, Tract 9801	Missing or no population	Tri-Cities Airport
WA	53027990000	Grays Harbor County, Tract 9900	Water area	
WA	53029992201	Island County, Tract 9922.01	Missing or no population	Smith Island
WA	53031990000	Jefferson County, Tract 9900	Water area	
WA	53033990100	King County, Tract 9901	Water area	
WA	53035990100	Kitsap County, Tract 9901	Water area	
WA	53049990100	Pacific County, Tract 9901	Water area	
WA	53055960100	San Juan County, Tract 9601	No neighbors	
WA	53055960500	San Juan County, Tract 9605	No neighbors	
WA	53055990100	San Juan County, Tract 9901	Water area	
WA	53057990100	Skagit County, Tract 9901	Water area	
WA	53061990002	Snohomish County, Tract 9900.02	Water area	
WA	53061990100	Snohomish County, Tract 9901	Water area	
WA	53067990100	Thurston County, Tract 9901	Water area	
WA	53071920400	Walla Walla County, Tract 9204	Missing or no population	Washington State Penitentiary
WA	53073010900	Whatcom County, Tract 109	No neighbors	
WA	53073011000	Whatcom County, Tract 110	No neighbors	

[*] Unique identifiers of census tracts are in Federal Information Processing Standards code as of 2010.

Table B.6. Descriptive statistics of the data

	Oregon				*Washington*			
	Mean	Standard deviation	Minimum	Maximum	Mean	Standard deviation	Minimum	Maximum
Mental illness mortality	0.56	0.89	0.00	10.00	0.31	0.65	0.00	13.00
Population	4681.02	1921.48	75.00	14619.00	4733.19	1756.68	48.00	14540.00
Mental illness mortality (per 10,000 population)	1.21	2.02	0.00	26.14	0.69	1.62	0.00	71.26
Area of parks (km^2 per 10,000 population)	4.17	12.55	0.00	356.07	2.65	6.16	0.00	82.97
$PM_{2.5}$ ($\mu g/m^3$)	5.66	1.94	0.63	13.50	5.88	1.60	0.55	12.84
Proportion of waterbodies (%)	2.48	5.90	0.00	56.78	3.64	8.77	0.00	75.36
Number of residential care facilities (per 10,000 population)	6.93	8.87	0.75	400.00	6.59	9.99	0.88	625.00
65 years old or older rate (%)	14.87	6.90	0.00	53.05	13.16	6.19	0.00	54.75
Negative marital status rate (%)	21.47	5.93	1.47	70.66	20.48	6.43	0.34	51.06
Non-white population rate (%)	20.58	13.10	0.65	97.13	26.39	17.91	0.00	96.44
Unemployment rate (%)	8.72	4.13	0.00	38.79	7.62	4.06	0.00	36.57
Poverty rate (%)	15.20	9.32	0.00	81.10	12.88	9.70	0.00	84.53
Higher than bachelor's degree (%)	30.00	17.36	0.00	85.64	30.87	17.77	0.00	87.44
Median household income (inflation-adjusted 2010 dollars)	51799.25	18832.92	7226.32	160170.05	60938.85	24030.67	4346.09	197166.29

Table B.7. Sensitivity analysis results

Hyperpriors $\tau_b, \tau_\gamma, \tau_\delta, \tau_{b_1}$	$\varphi_b, \varphi_\delta, \varphi_{b_1}$	DIC[1]	WAIC[2]
Oregon			
PC(1, 0.01) [3]	PC(0.5, 0.5)	19321.20	19329.04
PC(0.1, 0.01)	PC(0.5, 0.05)	19323.77	19333.37
logGamma(1, 0.0005)	logGamma(0.5, 0.0005)	19321.72	19331.36
Washington			
PC(1, 0.01)	PC(0.5, 0.5)	24555.82	24546.06
PC(0.1, 0.01)	PC(0.5, 0.05)	24552.89	24554.84
logGamma(1, 0.0005)	logGamma(0.5, 0.0005)	24553.51	24545.40

[1] Deviance information criterion; [2] Watanabe-Akaike information criterion; [3] Penalized complexity prior.

Table B.8. Summary of hyperparameter estimates

	Mean	Standard deviation	95% credible interval
Oregon			
p_{zero}	0.007	0.006	0.001, 0.022
τ_b	389.218	933.260	29.401, 2228.986
φ_b	0.313	0.193	0.033, 0.737
τ_γ	41.003	22.341	11.394, 96.719
ρ_0	0.836	0.105	0.570, 0.970
$\tau_\delta{}^*$	5.232	0.567	4.117, 6.345
$\varphi_\delta{}^*$	0.877	0.072	0.684, 0.964
$\rho_1{}^*$	0.895	0.020	0.853, 0.933
τ_b	450.739	1266.168	24.091, 2658.593
φ_{b_1}	0.314	0.263	0.009, 0.894
Washington			
p_{zero}	0.009	0.009	0.001, 0.034
τ_b	4.764	0.881	3.296, 6.754
φ_b	0.390	0.106	0.194, 0.605
τ_γ	148.712	89.331	41.570, 380.354
ρ_0	0.622	0.217	0.084, 0.915
$\tau_\delta{}^*$	5.149	0.878	3.631, 7.083
$\varphi_\delta{}^*$	0.439	0.097	0.257, 0.637
$\rho_1{}^*$	0.763	0.074	0.592, 0.881
τ_b	1338.804	2689.515	85.762, 7165.500
φ_{b_1}	0.295	0.173	0.052, 0.698

Figure B.1. Map of state, county, and census tract boundary and elevation in Oregon and Washington

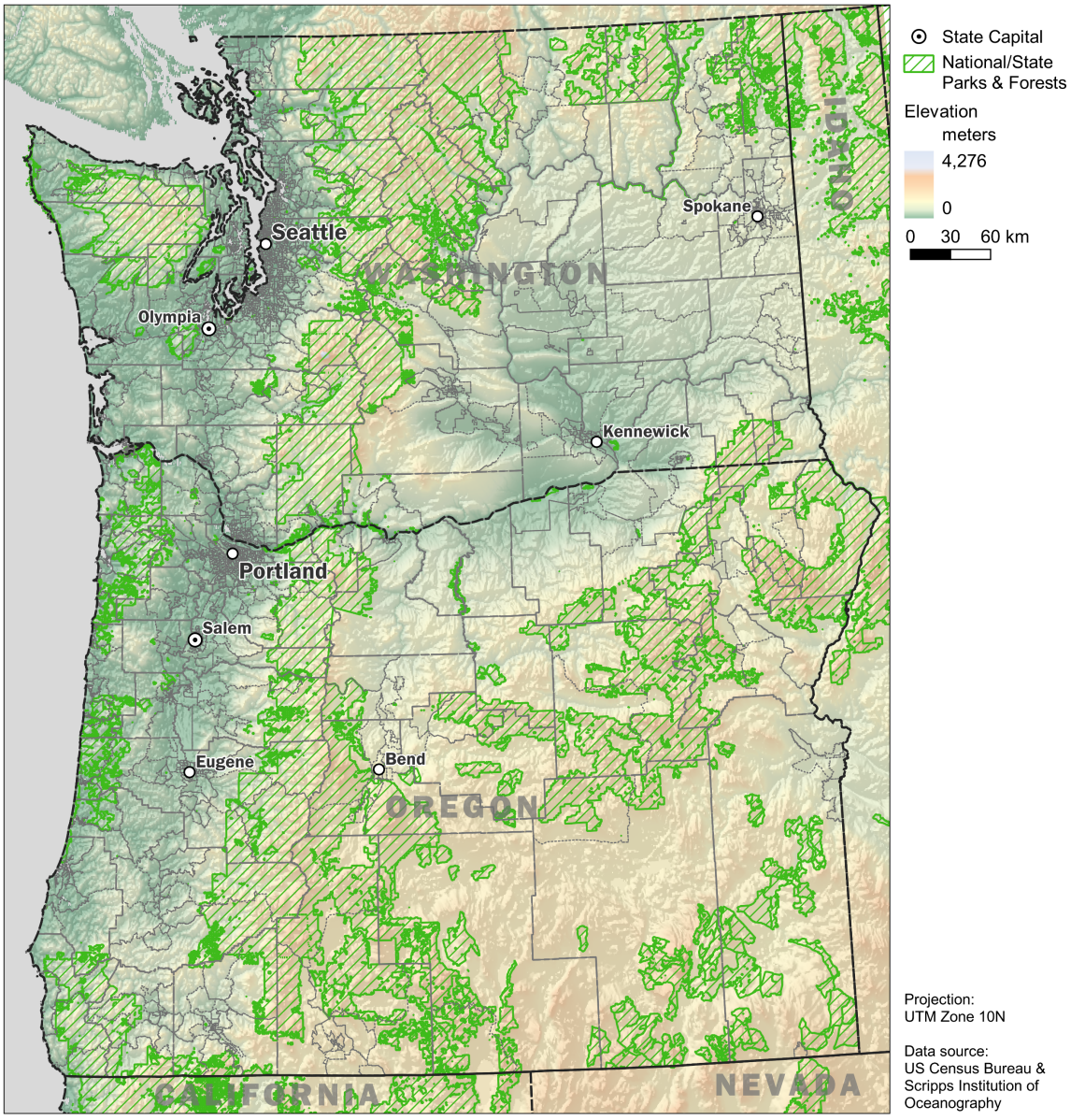
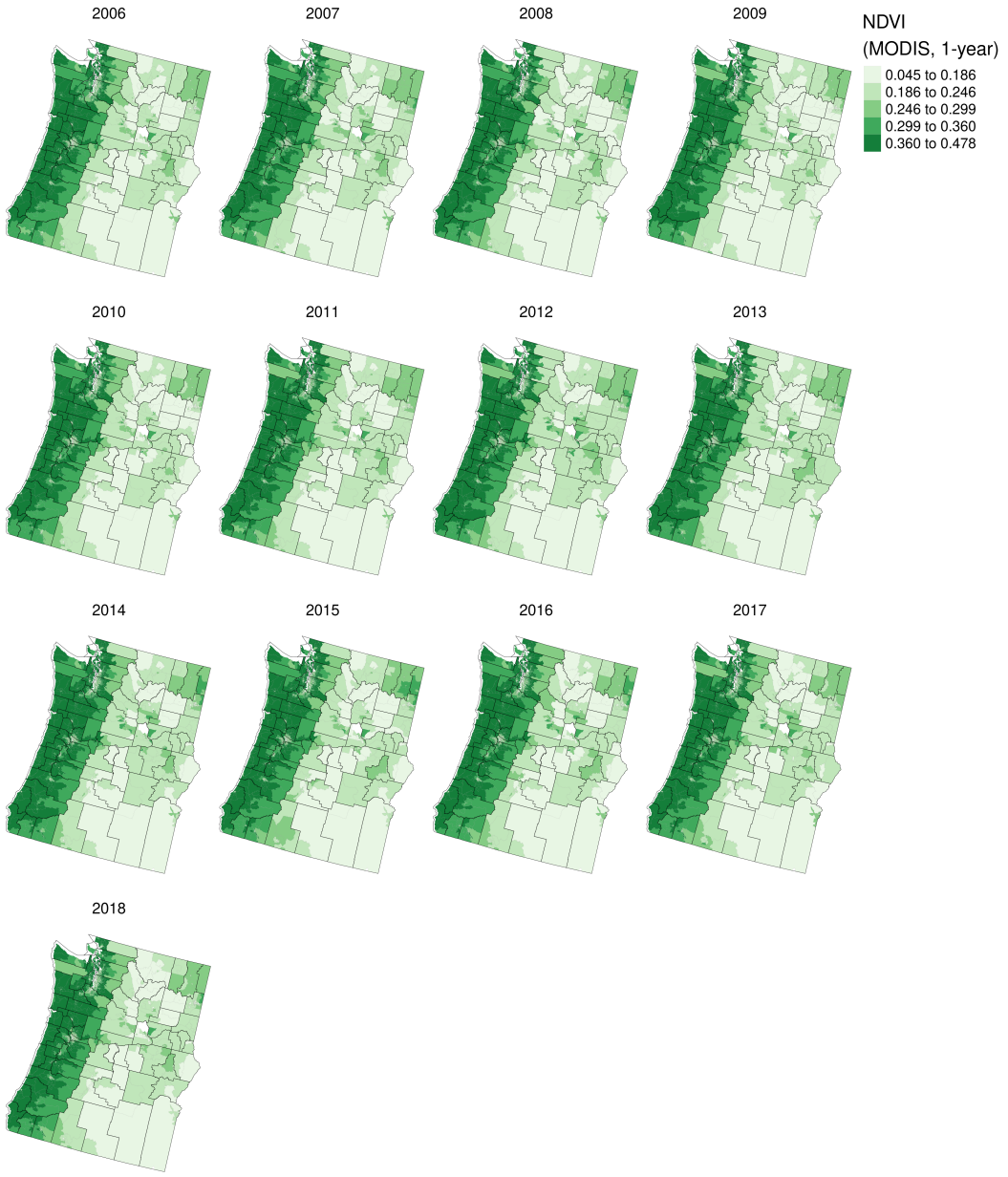


Figure B.2. One-year lagged annual mean normalized difference vegetation index (NDVI) at each census tract by year during the study period (2006–2018) from the moderate resolution imaging spectrometer (MODIS) Terra sensor



APPENDIX C

CHAPTER 4 APPENDIX

Table C.1. Descriptive statistics of the decedents who deceased in natural manners in the State of Washington, 2018

Characteristic	**N = 31,245**[1]
Mental illness mortality	273 (0.87%)
Subregion	
Columbia Plateau	4,586 (14.68%)
Seattle-Tacoma	11,569 (37.03%)
Spokane	1,706 (5.46%)
Western Cascades	11,690 (37.41%)
Western Cordillera	1,694 (5.42%)
NDVI (5-year average in 15-minutes walking area)	0.27, 0.26 (0.05)
Age	76.72, 78.00 (13.42)
Sex	
Female	15,036 (48.12%)
Male	16,209 (51.88%)
Race	
White (Non-Hispanic)	27,909 (89.32%)
Black / African American	812 (2.60%)
Native American	500 (1.60%)
Hispanic	449 (1.44%)
Asian	1,203 (3.85%)
Others	372 (1.19%)

(Continued on the next page)

Table C.1 (*Continued*)

Characteristic	**N = 31,245**[1]
Marital status	
Married / Partnered	14,020 (44.87%)
Separated / Divorced	5,266 (16.85%)
Widowed	9,536 (30.52%)
Single / Never Married	2,378 (7.61%)
Unknown / Not Reported	45 (0.14%)
Education	
No High School Diploma / GED	3,999 (12.80%)
High School Diploma to Associate Degree	20,153 (64.50%)
University Degree and Higher	6,920 (22.15%)
Unknown / Not Reported	173 (0.55%)
Smoking	
Yes	4,088.00 (13.08%)
Never	16,063.00 (51.41%)
Paused / Abstained	2,595.00 (8.31%)
Unknown / Not Reported	8,499.00 (27.20%)
MNDWI	-0.01, -0.01 (0.02)
NDBI	-0.13, -0.13 (0.02)
$PM_{2.5}$ ($\mu g/m^3$)	5.86, 5.65 (1.47)
Traffic noise (decibel)	14.34, 8.90 (15.58)
Median household income (US dollars)	67,357.96, 63,115.00 (25,034.76)
Non-white rate (%)	27.64, 22.88 (17.85)
Household income less than 150 % of poverty line (%)	12.58, 11.08 (7.85)
Unemployment rate (%)	6.24, 5.56 (3.13)
Bachelor's degree and higher (%)	31.12, 26.85 (16.95)

[1]n (%); Mean, Median (standard deviation)

Figure C.1. Standardized difference of covariates in the treated and controlled groups by five percentile thresholds for treatment definition in the State of Washington

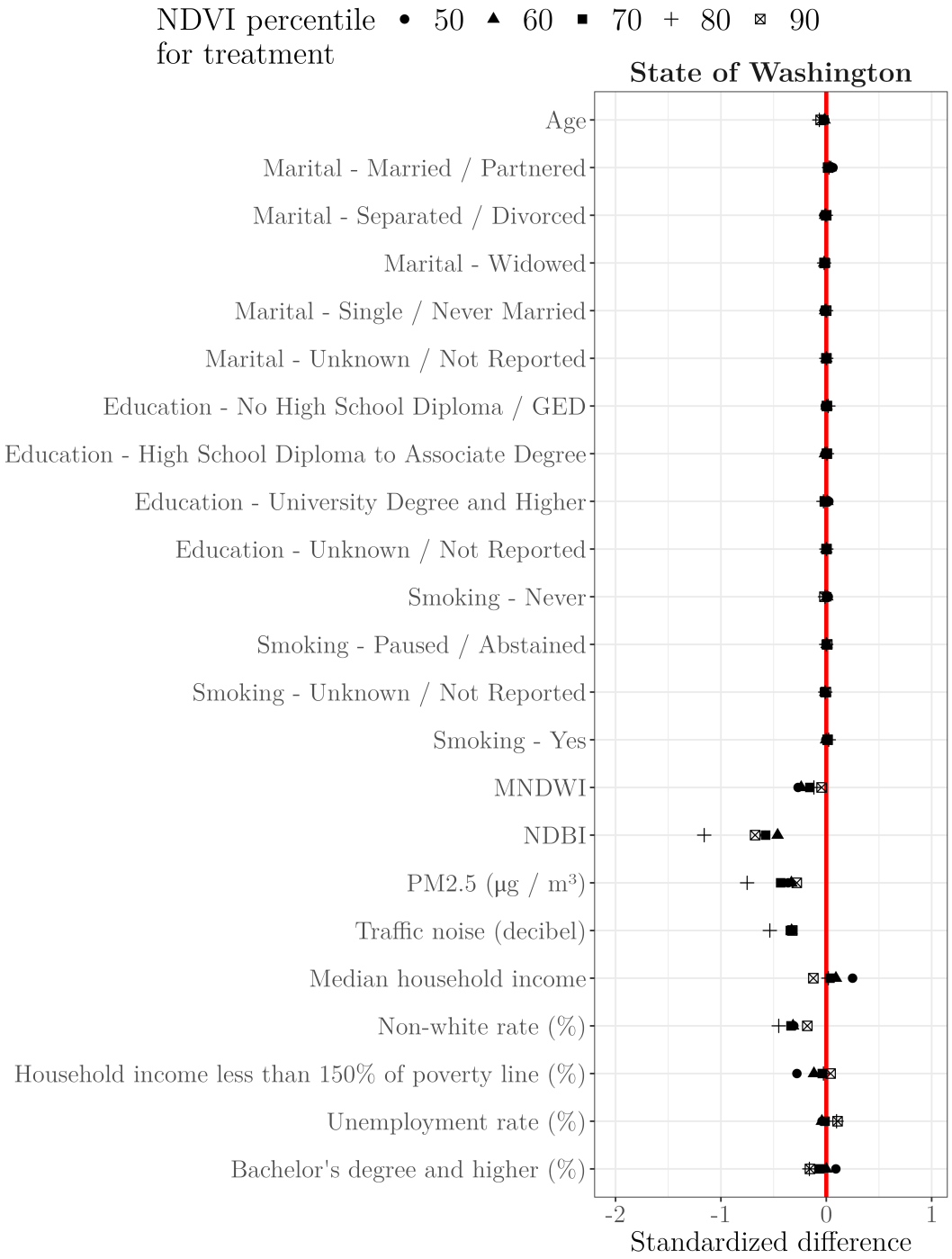


Figure C.2. Standardized difference of covariates in the treated and controlled groups by five percentile thresholds for treatment definition in subregions

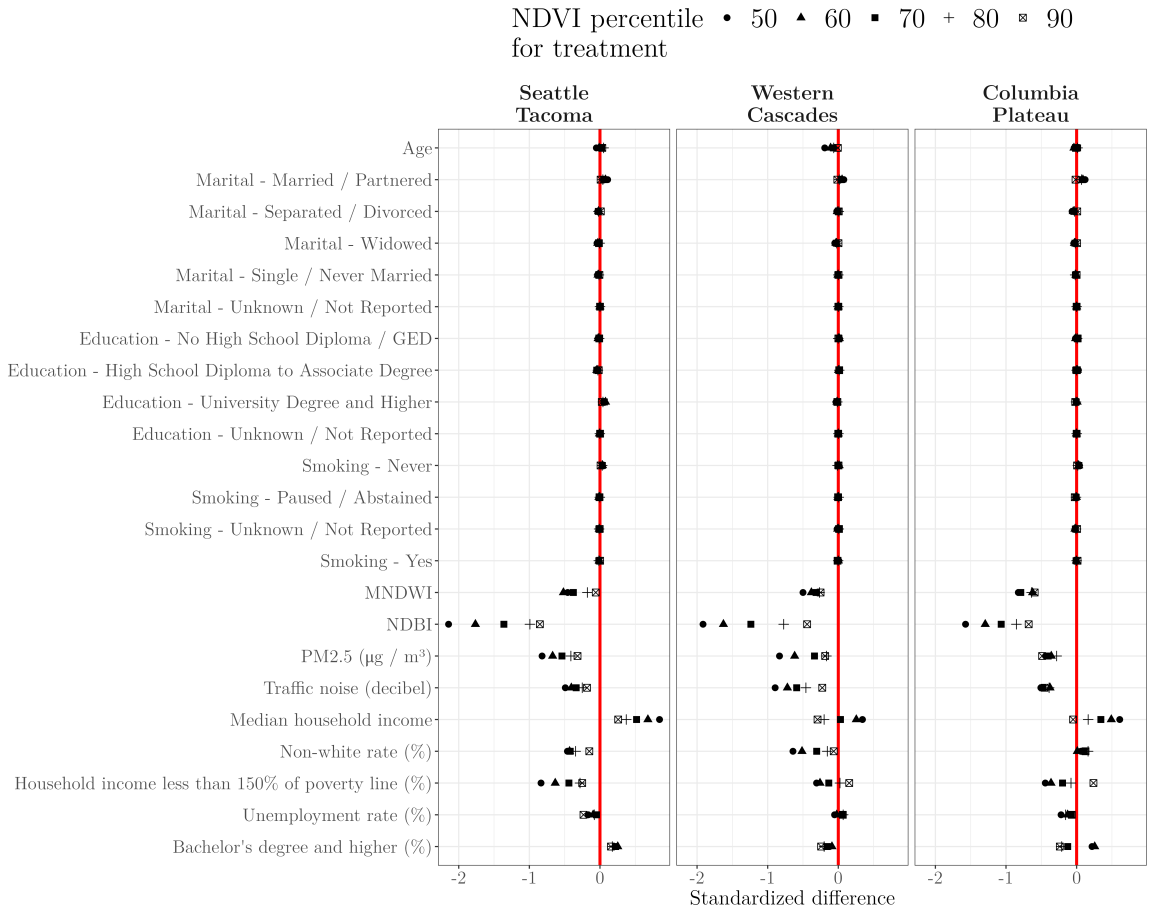
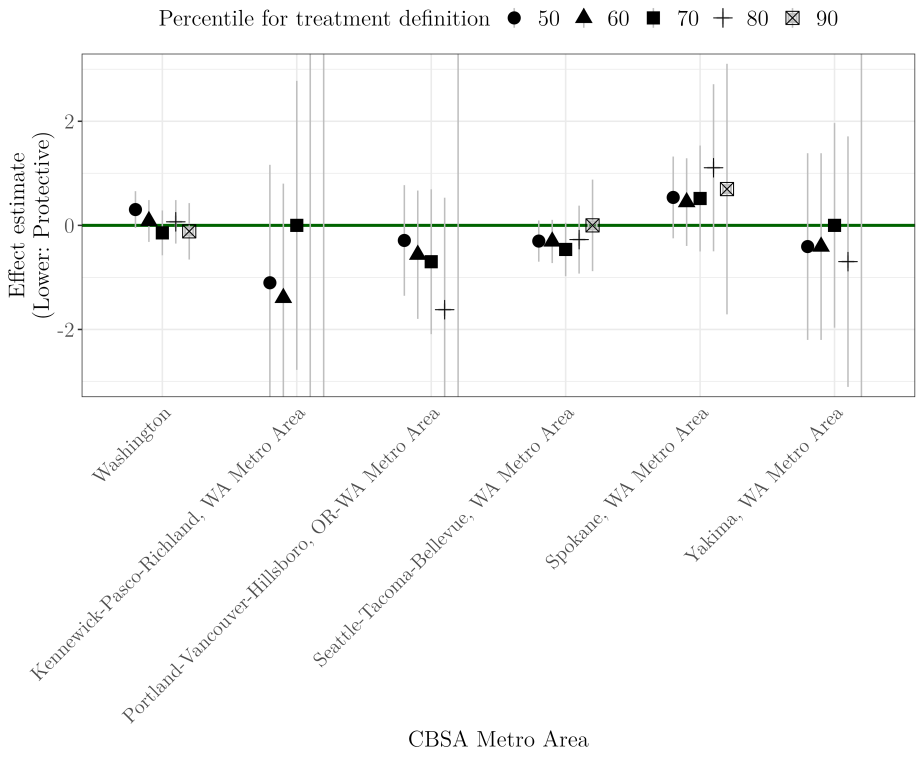


Figure C.3. Effect estimates from matching analysis by five percentile thresholds for treatment condition in five core-based statistical areas where dichotomization conditions were fulfilled



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