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Is Sprawl Associated with a Widening Urban–Suburban Mortality Gap?

Yingling Fan and Yan Song

ABSTRACT *This paper examines whether sprawl, featured by low development density, segregated land uses, lack of significant centers, and poor street connectivity, contributes to a widening mortality gap between urban and suburban residents. We employ two mortality datasets, including a national cross-sectional dataset examining the impact of metropolitan-level sprawl on urban–suburban mortality gaps and a longitudinal dataset from Portland examining changes in urban–suburban mortality gaps over time. The national and Portland studies provide the only evidence to date that (1) across metropolitan areas, the size of urban–suburban mortality gaps varies by the extent of sprawl: in sprawling metropolitan areas, urban residents have significant excess mortality risks than suburban residents, while in compact metropolitan areas, urbanicity-related excess mortality becomes insignificant; (2) the Portland metropolitan area not only experienced net decreases in mortality rates but also a narrowing urban–suburban mortality gap since its adoption of smart growth regime in the past decade; and (3) the existence of excess mortality among urban residents in US sprawling metropolitan areas, as well as the net mortality decreases and narrowing urban–suburban mortality gap in the Portland metropolitan area, is not attributable to sociodemographic variations. These findings suggest that health threats imposed by sprawl affect urban residents disproportionately compared to suburban residents and that efforts curbing sprawl may mitigate urban–suburban health disparities.*

KEYWORDS *Mortality, Sprawl, Smart growth, Urban health penalty, Health disparities*

INTRODUCTION

Whether health disparities exist between urban and suburban residents has been debated in the field of public health for centuries. Early cities in the nineteenth or the early twentieth century were developed with rapid population growth in an environment without proper sanitation. High population density coupled with accumulation of city waste was likely to deteriorate air quality, contaminate water supply, provide new foci of infection, and create favorable conditions for the rapid transmission of disease from host to host—all of which led to elevated mortality risks among urban residents.[1–3] Modern city life, although offering health benefits through improved access to medical care, sanitation, education, jobs, social support, and higher income,[4] still threatens health via greater exposure to environmental pollutions, social stress, infections, violence, and accidents.[5]

Fan is with the Hubert H. Humphrey Institute of Public Affairs, University of Minnesota, Twin Cities, Minneapolis, USA; Song is with the Department of City and Regional Planning, University of North Carolina at Chapel Hill, Chapel Hill, USA

Correspondence: Yingling Fan, Hubert H. Humphrey Institute of Public Affairs, University of Minnesota, Twin Cities, 301 19th Avenue South, 295E HHH Center, Minneapolis, MN 55455, USA. (E-mail: yingling@umn.edu)

W. H. McNeil explicitly developed a conceptual model to explain variations in mortality rates between large metropolitan centers and their more thinly settled hinterlands.[6] Central areas, he argued, act as endemic reservoirs of diseases which spill over to their hinterlands in the form of recurrent epidemics. Urban populations thus experience generally higher and more stable levels of mortality. Hinterland mortality, by contrast, is less severe but is subject to violent short-term fluctuations.[6] Empirical studies conducted in the 1970s and 1980s concur with McNeil's model of the urban health penalty. Many suggest that there is a general increased risk of death for urban residents when compared to suburban residents.[7–10] However, studies in the 1970s and 1980s inadequately controlled for confounding variables such as race, ethnicity, and socioeconomic status. To some extent, "white flight"—the demographic shift in the first half of the twentieth century where middle-class families moved away from inner-city neighborhoods and where inner-city residents became equated with disadvantaged groups such as minority and low-income households—might explain the higher mortality rates found in urban areas in the 1970s and 1980s.[4]

More recent and rigorous studies on urban–suburban health disparities offer mixed and inconsistent findings. House et al. studied a national sample of 3,617 adults and found that significant urban mortality risk exists among white men, but not among white women.[5] Surprisingly, African Americans in suburban areas were found to have mortality risks as high as those in urban areas. Geronimus et al. selected several pairs of African-American communities and non-Hispanic white communities and investigated urban–rural disparities in mortality rates.[11] Their results contradict the findings of House et al., suggesting that African-American residents of urban communities suffer extremely high and growing rate of excess mortality. However, the contradiction may be due to the specific focus of Geronimus et al. on urban–rural disparities, which is different from the focus of House et al. on urban–suburban disparities. Smith et al. and Hayward et al. focused on a population of men 55 years or older and found that excess mortality existed among urban residents even after controlling for differences in social class and lifestyle factors between urban and suburban residents.[12,13]

Although the aforementioned studies performed adjustments for population composition and socioeconomic status, no study has yet examined how the extent of urban–suburban health disparities in a metropolitan area may be influenced by the region's built environment. The built environment, encompassing all of the buildings, spaces, and products that are created or significantly modified by people, not only forms a backcloth against which people live, work, and play,[14] but also to some degree determines residents' exposure to environmental risks and the associated physiological and psychosocial impacts.[15] Thus, the built environment has a profound impact on the health of its inhabitants, and different types of metropolitan environments (e.g., compact versus sprawling) each offer unique urban and suburban experiences, leading to a varied degree of urban–suburban health disparities.

Figure 1 illustrates a conceptual framework linking the built environment to health outcomes. The mediating factors and downstream pathways illustrated in Figure 1 are of particular interest as they outline possible connections between the built environment and health. Some of the meditating factors and downstream pathways are obvious: vehicle emissions, exposure to air pollution, and respiratory health; traffic congestion and noise, stress, and chronic diseases; and poorly maintained neighborhoods, crime, and homicides. Others are less direct but increasingly recognized as important, such as the relationships of land use patterns to human activity patterns and obesity-related diseases. In this research, we apply

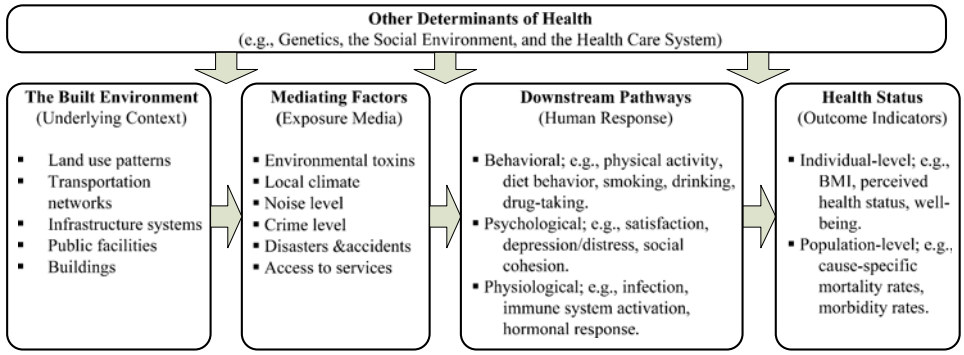


FIGURE 1. A conceptual model of how the built environment impacts health. The model presented in this figure is adapted from a conceptual model developed by Klitzman et al.[16] The model has been modified to highlight the mediating factors and downstream pathways by which the built environment influences health outcomes.

the conceptual framework in Figure 1 to study how sprawl may contribute to urban–suburban health disparities in cause-specific mortality.

Sprawl is the prevailing land development pattern in the US, featured by low development density, segregated land uses, lack of significant centers, and poor street connectivity. Sprawl is found to be associated with higher levels of environmental pollution.[17,18] Thereby, according to Figure 1, residents living in sprawling metropolitan areas may experience higher levels of physical, chemical, and biological exposure and are likely to have elevated mortality risks from tumor, infection, or respiratory diseases. Sprawl is also found to be associated with a sedentary lifestyle, unhealthy eating habits, and risk behaviors such as smoking,[19–21] and based upon the downstream pathways illustrated in Figure 1, sprawl may lead to a higher mortality risk from cardiovascular/heart diseases. In addition, sprawl promotes extensive auto use and increases social polarization among communities, which may increase crime rates, worsen traffic conditions, and make residents more vulnerable to external causes of death.[22,23]

Having highlighted the underlying causes of death that are most relevant to health threats associated with sprawl, it is important to note that, within a metropolitan area, the health threats associated with sprawl are likely to affect urban residents disproportionately compared to suburban residents. Sprawl inevitably leads to decentralization and fragmentation of economic opportunities, dramatic reductions in population size, density, diversity, and resources in urban areas, and deprivation of economic, social, and political capitals in inner-cities, all of which create urban–suburban health disparities and increase excess mortality risks among urban residents. In contrast, compact development (i.e., the opposite of sprawl) promotes "smart growth"* and often has goals targeted to prevent

*"Smart growth" is antisprawl development that values long-range, holistic considerations of environmental protection, economic growth, and social equity over short-term fiscal considerations. The term of "smart growth" is often used interchangeably with "growth management." Examples of growth management/smart growth strategies include (a) urban containment boundaries that direct urban development into areas intended or needed for urban uses and protect rural land from urban spillovers, (b) capital improvements programming and adequate facilities standards that discourage developments farther away from existing civil infrastructure systems and encourage infill and redevelopments, (c) land preservation techniques (e.g., transfer of development rights and agriculture/forest buffers) that protect resource land from urban development pressures, etc.

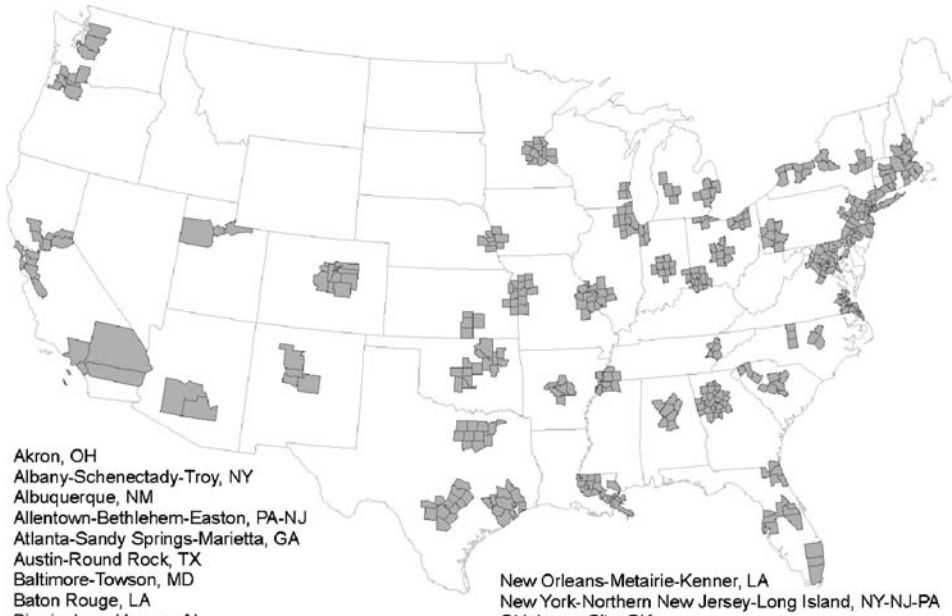
decentralization of economic opportunities, avoid inner-city decline, and advocate compact, transit-oriented, walkable, and bicycle-friendly land uses.[24] These policies embrace geographic equity and promote a more balanced allocation of resources within the metropolitan area between inner-cities and suburbs. Therefore, it is expected that sprawling regions may observe not only higher overall mortality rates but also a wider mortality gap between urban and suburban residents when compared to compact regions.

This paper presents a direct effort to test this a priori expectation. To ensure the robustness and thoroughness of the empirical test, two datasets are employed in this paper: one has cross-sectional population, mortality, and urban form information in the nation's 65 largest metropolitan areas from the year 2000; another has longitudinal population, mortality, and urban form information from 1989 to 2000 in the Portland metro's 71 zip code areas. Analysis of the national dataset attempts to quantify the size variation in urban–suburban mortality gaps across different metropolitan areas and how the magnitude of mortality gaps varies as a function of the extent of sprawl. Analysis of the Portland dataset comes with a longitudinal design that examines whether Portland's recent efforts on curbing sprawl are associated with decreases in urban–suburban health disparities. The two analyses complement each other and are intended to provide supporting empirical evidence on the hypothesis that sprawl is positively linked to the level of urban–suburban health disparities.

NATIONWIDE CROSS-SECTIONAL STUDY: DATA, METHOD, AND FINDINGS

This national study focuses on the 100 largest metropolitan areas during the year 2000. Boundaries of the metropolitan areas are specified using the Core Based Statistical Areas system defined by the U.S. Office of Management and Budget (OMB) in 2000. According to the US OMB, each metropolitan area consists of one or more counties, encompassing (1) the counties containing a core urban area of 50,000 or more population and (2) any adjacent counties that have a high degree of social and economic integration with the urban core. This operational definition offers opportunities of applying a parallel-group design (i.e., matched pairs of core urban versus suburban counties) to examine urban–suburban mortality gaps.

The final sample of this research is limited to 65 metropolitan areas because of data availability and matching suitability of county components in each metro. Single-county metros are either excluded from the sample (e.g., El Paso, TX and San Diego, CA) or combined into adjacent metros (e.g., the Oakland metropolitan area in CA is combined into the San Francisco metropolitan area). The final 65 metro areas in the sample, as shown in Figure 2, include a total of 458 counties. The 458 counties are categorized into core urban versus suburban counties based upon their urbanization level. Counties are coded as core urban counties if they are identified as large central counties in the 2006 Urban–Rural Classification Scheme by the National Center for Health Statistics (NCHS). If no counties in a metropolitan area are coded as large central by NCHS, counties with the largest city population in the metropolitan area are identified as the urban core county. For example, in the Portland metropolitan area, Multnomah County is identified as the core urban county while Washington, Clark, and Clackamas Counties are identified as suburban counties. Finally, 79 counties are identified as core urban counties and 379 are identified as suburban counties.



Akron, OH
Albany-Schenectady-Troy, NY
Albuquerque, NM
Allentown-Bethlehem-Easton, PA-NJ
Atlanta-Sandy Springs-Marietta, GA
Austin-Round Rock, TX
Baltimore-Towson, MD
Baton Rouge, LA
Birmingham-Hoover, AL
Buffalo-Niagara Falls, NY
Chicago-Naperville-Joliet, IL-IN-WI
Cincinnati-Middletown, OH-KY-IN
Cleveland-Elyria-Mentor, OH
Colorado Springs, CO
Columbia, SC
Columbus, OH
Dallas-Fort Worth-Arlington, TX
Denver-Aurora, CO
Detroit-Warren-Livonia, MI
Grand Rapids-Wyoming, MI
Greensboro-High Point, NC
Greenville, SC
Hartford, CT
Houston-Baytown-Sugar Land, TX
Indianapolis, IN
Jacksonville, FL
Kansas City, MO-KS
Knoxville, TN
Little Rock-North Little Rock, AR
Los Angeles-Long Beach-Santa Ana, CA
Memphis, TN-MS-AR
Miami-Fort Lauderdale-Miami Beach, FL
Milwaukee-Waukesha-West Allis, WI
Minneapolis-St. Paul-Bloomington, MN-WI
New Haven-Bridgeport-Stamford-Waterbury-Danbury, CT
New Orleans-Metairie-Kenner, LA
New York-Northern New Jersey-Long Island, NY-NJ-PA
Oklahoma City, OK
Omaha-Council Bluffs, NE-IA
Orlando, FL
Philadelphia-Camden-Wilmington, PA-NJ-DE-MD
Phoenix-Mesa-Scottsdale, AZ
Pittsburgh, PA
Portland-Vancouver-Beaverton, OR-WA
Providence-Warwick-Pawtucket, RI
Raleigh-Cary, NC
Riverside-San Bernardino-Ontario, CA
Rochester, NY
Sacramento--Arden-Arcade--Roseville, CA
Salt Lake City, UT
San Antonio, TX
San Francisco-Oakland-Fremont, CA
San Jose-Sunnyvale-Santa Clara, CA
Seattle-Tacoma-Bellevue, WA
Springfield, MA
St. Louis, MO-IL
Syracuse, NY
Tampa-St. Petersburg-Clearwater, FL
Toledo, OH
Tulsa, OK
Virginia Beach-Norfolk-Newport News, VA-NC
Washington-Arlington-Alexandria, DC-VA-MD-WV
Wichita, KS
Worcester-Lawrence-Lowell-Brockton, MA-NH

FIGURE 2. Study area: 65 metro areas.

Data and Variables

County-level mortality data come from the Centers for Disease Control and Prevention (CDC). Using the online CDC WONDER platform, 2000–2005 mortality counts by underlying cause of death in the 458 study counties are acquired. As discussed in the "Introduction" section, we highlight the underlying causes of death that are most relevant to health threats associated with sprawl. They are (1) infections, (2) tumors, (3) cardiovascular diseases, (4) respiratory diseases, and (5) external causes such as injury, suicide, and homicide. Cause-specific mortality rates are identified using the International Classification of Diseases (ICD) published by the World Health Organization. To date, there have been ten revisions of the ICD. ICD-9 was used from 1979 to 1998 and ICD-10 has been used

TABLE 1 ICD codes for five specific underlying causes of death

Causes of death	ICD-9 (1979–1998)	ICD-10 (1999–present)
Certain infectious and parasitic diseases	001-139	A00-B99
Neoplasms/tumors	140-239	C00-D48
Heart diseases/diseases of the circulatory system	390-459	I00-I99
Diseases of the respiratory system	460-519	J00-J98
External causes of morbidity and mortality	E800-E899	V01-Y98

since 1999. Table 1 presents the ICD-9 and ICD-10 codes corresponding to each of the five death causes.

Sociodemographic information at the county level comes from the U.S. Census Bureau, including age, sex, race, ethnicity, marital status, income level, and poverty. Furthermore, a set of dummy variables are created to capture contextual differences such as weather and climate in the nine census divisions (i.e., New England, Middle Atlantic, East North Central, West North Central, South Atlantic, East South Central, West South Central, West Mountain, and West Pacific).

A sprawl indicator at the metropolitan level is incorporated into mortality models. Metropolitan-level sprawl indices have been developed by many sources, including USA Today, Sierra Club, and independent researchers (for example, Galster et al.).[25] This paper adopts the sprawl index developed by Ewing et al. for 83 US metropolitan areas because Ewing's index is the most recent and comprehensive effort of measuring sprawl,[26] incorporating various density, land use mix, centrality, and street connectivity dimensions. Ewing's sprawl index is a metropolitan-level factor extracted from six variables through principle component analysis: (1) gross population density (persons per square mile); (2) percentage of population living at low suburban densities; (3) percentage of population living at moderate to high urban densities; (4) net density in urban areas; (5) average block size; and (6) percentage of blocks with areas less than 1/100 square mile. This factor was transformed to a scale with a mean of 100 and a standard deviation of 25. Larger values of the sprawl index indicate more compact metro areas, whereas smaller values indicate more sprawling metro areas. The addition of Ewing's sprawl index to the national dataset allows us to examine whether Seattle (sprawl index=100.9) would have smaller urban–suburban mortality gaps if it were as compact as Portland (sprawl index=126.0) after controlling for sociodemographics.

Regression Model

At the first glance, Poisson regression is appropriate for this analysis because our dependent variables are mortality rates. However, descriptive analysis shows overdispersion in all-cause and cause-specific mortality rates, which contradicts the assumption of Poisson distribution (i.e., the assumption that the mean is equal to the variance). To address this issue, we estimate mortality models using generalized linear models (commonly referred to as GLZ)**—an approach that places fewer restrictions on model parameters. The GLZ approach allows the variance to be adjusted independently of the mean and thereby relaxes the requirement of equality

**The Generalized Linear Model (GLZ) is an extension of the General Linear Model (GLM) to be used when response variables follow distributions other than the normal distribution and when variances are not constant.

or constancy of variances in traditional Poisson regression. The specification of the GLZ model is shown below. An interaction term between the urban dummy variable and the sprawl index is added to the model to test the hypothesis that the urbanicity-related excess mortality is more evident in sprawling metros than in compact metros. In other words, inclusion of the interaction term allows us to infer whether the level of urban–suburban health disparities within a metropolitan area changes with the area's sprawl magnitude. Control variables of the model include age, gender, race, ethnicity, income, and geographical division. The standard errors in the model are adjusted to correct for correlation among counties in the same metropolitan area:

$$\ln\{E(Y)\} = \beta_0 + \beta_1 X_{Urban} + \beta_2 X_{Sprawl} + \beta_3 X_{Urban} X_{Sprawl} + \beta_C X_{Controls} + \varepsilon, \quad y \sim Poisson$$

where Y is the all-cause or cause-specific mortality rates within each study county in 2000–2005 (unit: deaths per person-year), X_{Urban} is the dummy variable as 1 represents core urban county and 0 represents suburban county, X_{Sprawl} is the sprawl index score, $X_{\text{Urban}}X_{\text{Sprawl}}$ is the interaction term of the urban dummy and the sprawl index, X_{Controls} is a set of control variables including age, gender, race, ethnicity, income, and geographical division, β_0, β_1, β_2, β_3, and β_{C} are regression coefficients, and ε is the error term.

Overall, this analysis has a national scope that improves generalizability and uses a parallel-group design that prevents the metropolitan-level environmental context from confounding the impact of urban residence on health. In addition, integrating spatial factors such as the extent of sprawl into cross-sectional mortality models helps to understand the impact of sprawl on urban–suburban health disparities. It is expected that urban–suburban mortality gaps are more evident in sprawling metros than in compact ones.

Findings

Table 2 presents descriptive statistics on the variables used in the national study. Core urban counties and suburban counties differ with regard to crude mortality rates, population composition, and socioeconomic level. In core urban counties, the percentage of individuals aged 18–29 years tends to be higher. Population in suburban counties (median age=36 years) are generally older than those in core urban counties (median age=34 years). The population size and diversity of suburban counties are much lower than urban counties. Suburban counties on average have higher income levels than their urban counterparts.

In addition, Table 2 shows that all-cause mortality rates are higher in core urban counties, but when looking at cause-specific mortality rates, excess mortality among urban residents does not exist in any of the five cause-specific categories. While death rates associated with infectious and cardiovascular diseases are higher in core urban counties, death rates associated with tumor, respiratory, and external causes are lower in urban counties.

The discrepancy in mortality rates between urban and suburban counties presented in Table 2 does not adjust for demographic composition and socioeconomic characteristics. Table 3 presents the regression results from the estimated GLZ, which controls for sociodemographic confounding factors.

The results in Table 3 show that, after adjusting variations in age, race, ethnicity, income, and regional location, the urban dummy variable is significant and positive in all of the mortality models except the external cause model. Among the

TABLE 2 Demographic composition, socioeconomic characteristics, and mortality rates by urbanicity

	Core urban counties ($N=79$)				Suburban counties ($N=379$)			
Variable	Mean	SD	Min	Max	Mean	SD	Min	Max
County-level mortality rates (deaths per 1,000,000 persons per year)								
All-cause	8,267	1,774	4,741	13,357	8,111	2,097	2,510	15,339
Cause-specific								
Infectious	282	160	79	1,085	190	86	33	658
Tumors	1,904	396	1,072	3,018	1,944	471	674	4,018
Cardiovascular	2,997	737	1,530	4,637	2,960	936	719	6,696
Respiratory	753	181	405	1,211	789	239	144	1,722
External	569	164	306	1,177	575	186	211	1,215
County-level independent variables								
Population in 2000–2005 (1,000 persons)	6,940	7,828	806	58,700	1,199	1,649	29	11,000
Age under 5 (%)	7	1	4	9	7	1	3	11
Age 5–17 (%)	18	2	10	22	20	2	7	24
Age 18-21 (%)	6	1	3	11	5	2	3	37
Age 22–29 (%)	12	2	8	19	10	2	6	20
Age 30–39 (%)	16	2	14	23	16	2	8	22
Age 40–49 (%)	15	1	13	17	16	2	8	23
Age 50–64 (%)	14	1	11	17	16	2	10	23
Age 65 up (%)	12	3	7	23	12	3	4	31
Median age	34	2	29	43	36	3	23	50
Foreign born (%)	14	11	2	51	5	6	0	32
Hispanic (%)	14	13	1	57	6	8	0	59
Black (%)	20	15	1	67	9	12	0	70
Median family income ($)	50,933	8,961	30,682	81,717	55,281	12,825	25,443	97,225

TABLE 3 Regression results from the national cross-sectional study

Variables	Infectious diseases	Neoplasms/tumors	Cardiovascular diseases	Respiratory diseases	External causes	All causes
Interaction term	−0.0024***	−0.0011***	−0.0010**	−0.0016**	−0.0004	−0.0009*
Sprawl index	0.0026***	0.0009***	0.0005	−0.0007**	0.0002	0.0005***
Urban dummy	0.2462**	0.1104***	0.1181**	0.1539**	0.0473	0.0936**
% Age under 5	−14.9471**	−5.7679***	−5.2328**	−3.1119**	−7.2509**	−2.8675
% Age 5–17	−5.9328**	−1.9959**	−4.5074***		−3.6170***	−2.5069**
% Age 18–21	−8.7904***	−3.5969***	−5.2944***	−1.8986**	−5.8741***	−3.4247***
% Age 22–29	−6.3232*	−2.4140*	−7.4744***		−3.8317***	−2.3086**
% Age 40–49		2.8566***	4.3180**	5.6304***		5.9964*
% Age 50–64		6.1889***	9.8254***	10.7556***	3.7620***	8.3231**
% Age 65 up	5.9366***	8.2364***	11.3656***	10.5460***	3.3174***	10.6078***
Median age	−0.1257**	−0.1014***	−0.1815***	−0.1300***	−0.0895***	−0.1353**
% Foreign born		−0.0074***			−0.0112***	−0.0065**
% Hispanic			−0.1556**	−0.6139***		
% Black	2.0164***	0.2519***	0.3211***	−0.3386***	0.5163***	0.3269**
Family income ($10,000)	−0.0598***	−0.0289***	−0.0691***	−0.0743***	−0.1215***	−0.0547***
New England	0.4612***	0.0600***		0.1474***	−0.1671***	0.0479**
Middle Atlantic	0.4688***		0.0566***		−0.2482***	
Midwest East	0.2424***		0.0662***		−0.0906**	
Midwest West		−0.0269**				
West Mountain	0.1786**	−0.1633***	−0.1732***		0.2859***	−0.1002**
South Atlantic	0.3523***	−0.0662***	−0.0806***	−0.0861***		−0.0505***
West South Central	0.3963***	−0.0768***		−0.1400***	0.0767*	−0.0496***
East South Central	0.2331***	−0.0503**				
Constant	−1.9473	−3.6682***	−0.3133	−5.4057***	−2.7010	−2.0958***
Summary statistics						
N	458	458	458	458	458	458
LR statistic	2,657.87	4,738.90	2,855.05	1,544.17	2,219.10	6,430.52
P (alpha) = ~0	0.0000	0.0000	0.0000	0.0000	0.0000	0.0000

Among age groups, "% Age 30–39" is the reference age category. Among census divisions, West Pacific is the reference category. Variables were excluded from models if they are not significant at the 0.1 level

*$p<0.10$; **$p<0.05$; ***$p<0.01$

five cause categories, infectious diseases are associated with the highest excess mortality among urban residents (indicated by a coefficient of 0.2462), followed by respiratory diseases (0.1539), tumors (0.1181), cardiovascular diseases (0.1104), and external causes (0.0473). The coefficient of 0.2462 on the urban dummy variable in the mortality model of infectious diseases translates to an incident rate ratio of 1.279 (1.279=exp(0.2462)). This indicates that, while holding other variables constant and the sprawl index at 0 (note that a 0 value on the sprawl index indicates the highest level of sprawl), residents in core urban counties on average have an infection-related mortality risk 27.9% ($p<0.01$) higher than those in suburban counties. In the all-cause mortality model, the urban dummy variable has a coefficient of 0.0936, indicating that, while holding other variables constant and the sprawl index at 0, the overall mortality risk in core urban counties on average is about 10% ($p<0.05$) higher than that of suburban counties (10% = 100% × exp(0.0936) − 100%). Likewise, in the most sprawling metropolitan areas where the sprawl index is close to 0, urban residents on average have 11.7% higher risk of tumor-caused death, 12.5% higher risk of cardiovascular mortality, and 16.6% higher risk of respiratory mortality than suburban residents.

More interestingly, coefficients on the sprawl index show that, while infection-realted and tumor-related mortality is positively associated with ($p<0.01$) the sprawl index, respiratory diseases-related mortality is negatively associated with ($p<0.05$) the sprawl index. As lower sprawl index values represent more sprawling metros, the results indicate that when holding other variables constant and holding the urban dummy at 0 (note that the unit of analysis is the county and a 0 value on the urban dummy variable indicates the county to be a suburban county), suburban counties in compact metros have higher infection-related and tumor-related mortality but lower respiratory-related mortality than suburban counties in sprawling metros. The sprawl index in models for cardiovascular diseases and external causes is not significant. This is consistent with our earlier expectation that sprawl impacts health in a cause-specific way.

Negative coefficients on the interaction term provide evidence in support of our earlier hypothesis: urban–suburban health disparities are more evident in sprawling metros than metros implementing smart growth policies. Coefficients on control variables show consistency with previous studies on mortality. Poorer areas with more elderly and African Americans generally have higher mortality rates. In terms of regional location, the New England division has the highest mortality rates caused by infectious diseases, tumors, and respiratory diseases while the West Mountain division has the lowest. For external causes, the New England division has the lowest mortality rate while the West Mountain division has the highest. For all-cause mortality, New England has the highest and West Mountain has the lowest.

To gain a better view of how urban residence, sprawling land use patterns, and their interaction may affect mortality rates, we estimate all-cause and cause-specific mortality rates and the uncertainty surrounding them for urban versus suburban counties across the range of the sprawl index, while holding other variables at their median values. Statistical software packages including Clarify 2.0 and Stata 8.0 are used to estimate the expected values and their uncertainty.[27] For the case of a typical core urban county and the other case of a typical suburban county, expected value algorithm is repeated to approximate 90% confidence interval around mortality rates. We then plot the estimated expected values and the range of uncertainty in Figure 3, which illustrates the regression findings in Table 3 quite sharply. That is, the urban–suburban mortality gap, illustrated by the distance between blue dashed lines and black solid lines,

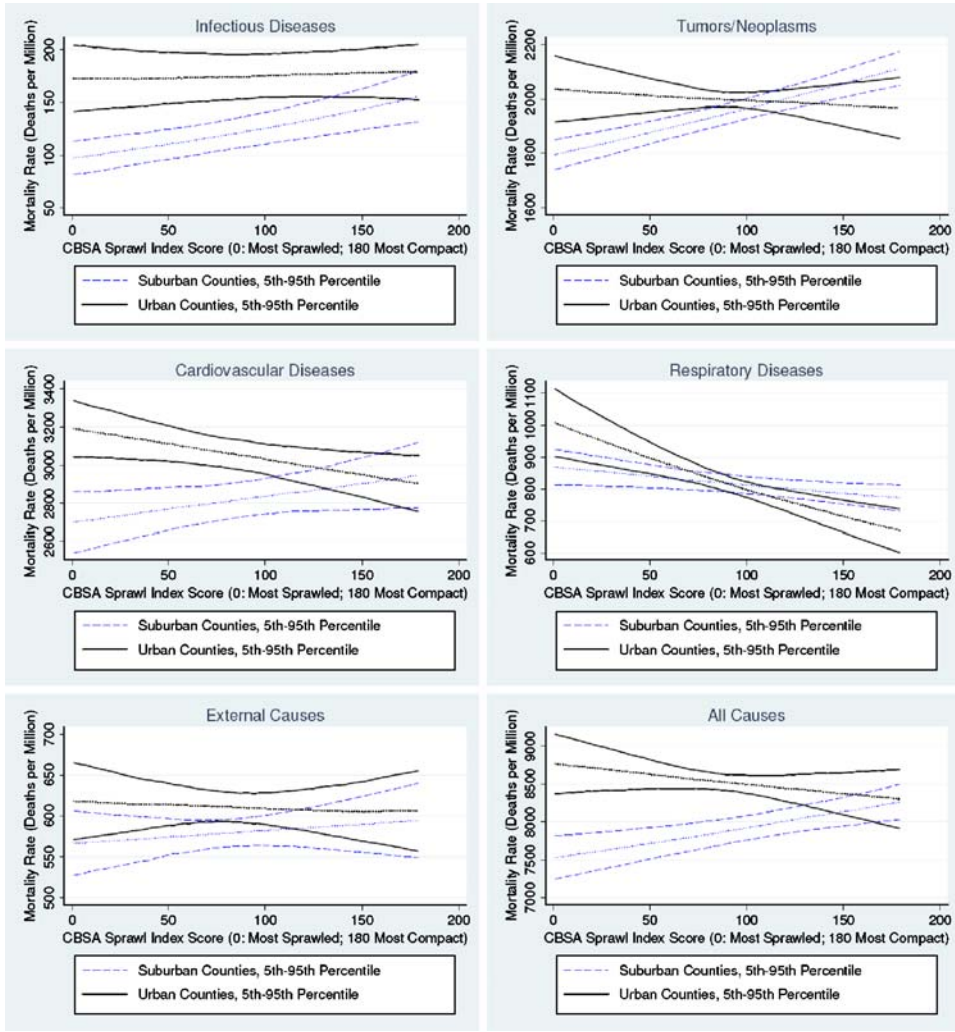


FIGURE 3. Mortality rates by the sprawl index and place of residence.

generally shrinks as the sprawl index increases (i.e., the built environment becomes less sprawling). Figure 3 shows that, in the most sprawling metros (sprawl index=0), mortality rates in core urban counties are significantly higher than those of suburban counties. In the least sprawling metros (sprawl index=180), no significant differences in mortality rates can be detected between core urban and suburban counties.

More specifically, in the U.S. most sprawling metros (e.g., Riverside, CA where the sprawl index score is 14.22), health disparities burdening urban residents (or the urban health penalty) translate to approximately 75 deaths per million persons caused by infectious diseases, approximately 200 deaths per million population caused by tumors/neoplasms, approximately 500 deaths per million caused by cardiovascular diseases, and approximately 100 deaths per million population caused by respiratory diseases. In terms of overall mortality, in the most sprawling metros, the model predicts an excess mortality of 1,300 deaths per million in core urban counties compared to suburban counties, after adjustment for socioeconomic characteristics and demographic composition. For less sprawling metros, Figure 3 shows converging confidence intervals between urban and suburban counties, indicating little urban–suburban mortality differentials in

metropolitan areas with compact development patterns. Among the five cause categories, only the model for external causes predicts neither urban–suburban mortality differences nor interaction between sprawl and urban–suburban health disparities. Evidence from all other models supports the association between sprawl and widening urban–suburban mortality gaps and that such mortality gaps mainly present an urban health penalty. This further indicates that health threats associated with sprawl tend to impact urban residents disproportionately compared to suburban residents.

PORTLAND LONGITUDINAL STUDY: DATA, METHOD, AND FINDINGS

We choose Portland as the study area for our longitudinal study mainly because of Portland's recent success in reversing the trend of urban sprawl.[28] Portland, a living laboratory for efficient urban planning and living, is perhaps best known for its urban growth boundary (UGB) and its light rail system. The UGB was established in 1979 and has expanded little since then. Under the requirements of Oregon's land use statutes, all land outside the UGB—with exceptions—is designated for resource use and prohibited from urban development. All land both inside and outside the UGB must be planned by the appropriate city or county and implemented with corresponding zoning. Proponents argue that Portland's UGB has successfully served to contain urban sprawl.[29] In addition, Portland has adopted a set of plans to encourage a compact urban growth pattern. In 1995, the Portland Metro Council adopted the 2040 growth concept, which calls for putting newcomers into dense, mixed-use neighborhoods; developing vacant land inside the growth boundary into relatively dense residential areas; encouraging developers to take advantage of higher-density zoning, to redevelop existing neighborhoods, and to "infill" vacant lots; and increasing the share of multifamily housing. The adopted growth concept was fleshed out into a comprehensive "regional framework plan" by the end of 1997. The regional framework plan allocates Portland neighborhoods to such categories as "regional centers," "town centers," "corridors," and "inner" and "outer neighborhoods."[30] Furthermore, Portland's light rail transit system was established on the east side of the metropolitan area in 1986 and expanded on the west side in 1998. The light rail transit system runs along the corridors and connects the centers. Additionally, transit area overlay zones with minimum density requirements and several public/private partnerships are established to encourage high-density housing and employment growth around station areas. Proponents claim that the system has been an effective vehicle for creating a less auto-dependent urban development pattern.[31]

The fact that Portland's major growth management/smart growth efforts started in the late 1980s and became more evident in the 1990s offers an opportunity to perform hypothesis-driven research with a before-and-after design. Song and Knaap's study found out that several smart urban development elements including more connective neighborhoods and more accessible public transit happened since the early 1990s.[28] However, time lags must be considered when looking at the impact of the built environment on health outcomes. Evident changes in health outcomes are likely to begin in mid-1990s. To find the most appropriate intervention year, we tried 1993, 1994, and1995 when conducting before-and-after comparisons. The year of 1994 appears to have the most evident changes in mortality rates and thereby we use 1994 as the division line defining the before and after periods. If the smart growth movement in the Portland metro area has played a role in mitigating urban–suburban health disparities, smaller mortality gaps between urban and suburban residents are expected in the period after smart urban development than in the period before.

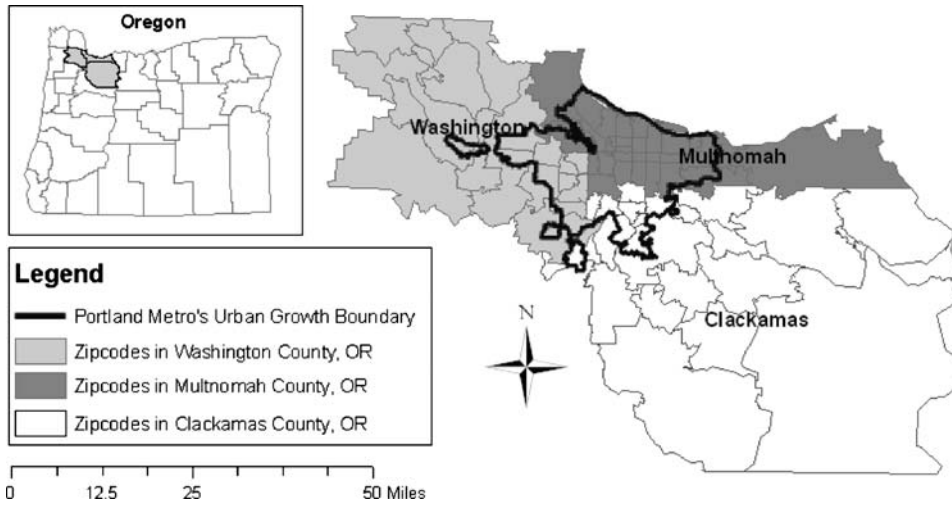


FIGURE 4. Study area: 71 zip codes in the Portland metropolitan area.

Due to issues in data availability, the study area comprises three out of the four counties in the Portland metropolitan area, including Washington, Multnomah, and Clackamas Counties in Oregon. Figure 4 shows the UGB and the 71 zip code areas in the three counties.

Data and Variables

The mortality data used in this study come from death certificates collected by the Oregon Center of Health Statistics in years from 1989 to 2000. The 1989–2000 death certificates provide individual information on causes of death, year of death, and residency identified by zip code. We aggregated the individual mortality data by year of death and zip code, resulting in a total of 142 data points (i.e., 71 mortality rates at the zip code level in the before period and another 71 in the after period). As the ICD codes for underlying causes of death changed in 1999, bridging codes in Table 1 are used to convert ICD-9 to ICD-10.

Population density at the zip code area level is used to quantify urbanicity—the degree to which a geographical unit is urban. All-cause and cause-specific mortality rates are regressed on the urbanicity indicator, a dummy code of time intervals, and an interaction term between them to quantify how the effect of urbanity on mortality rates changed over time, while controlling for sociodemographic composition of the zip code area. Census 1990 and 2000 data are used to generate values on population density and control variables, respectively, in the before (1989–1994) and after (1995–2000) periods.

Regression Model

GLZ are used again to model zip code-level mortality rates in the before and after periods. The model specification is shown below. The standard errors in the model are adjusted to correct for the correlation between before and after periods in the same zip code area:

$$\ln\{E(Y)\} = \beta_0 + \beta_1 X_{Urban} + \beta_2 X_{After} + \beta_3 X_{Urban} X_{After} + \beta_C X_{Controls} + \varepsilon, \quad y \sim Poisson$$

where Y is the all-cause or cause-specific mortality rates within the specific time period in the zip code area (unit: deaths per person-year), X_{Urban} is the urbanicity indicator represented by the population density at the zip code area level (unit: 100

TABLE 4 Before-and-after comparisons of demographic composition, socioeconomic characteristics, and mortality rates in the Portland metropolitan area

	1989–1994 ($N=71$)				1995–2000 ($N=71$)			
Variable	Mean	SD	Min	Max	Mean	SD	Min	Max
Zip code-level mortality rates (deaths per 1,000,000 persons per year)								
All-cause	8,814	4,420	265	26,846	7,725	2,806	1,414	18,113
Cause-specific								
Infectious	240	263	0	1,178	172	170	0	1,218
Tumors	2,116	1,149	0	8,501	1,785	559	202	2,967
Cardiovascular	3,387	1,606	139	10,067	2,800	999	404	5,627
Respiratory	834	398	0	1,854	716	284	0	1,370
External	334	280	6	2,122	334	355	72	2,892
Zip code-level independent variables								
Population density (person/square mile)	2,292	2,491	5	11,799	2,645	2,652	11	13,322
Age under 5 (%)	7	2	0	11	6	2	0	9
Age 5–17 (%)	18	5	0	29	18	5	1	25
Age 18–29 (%)	17	5	3	36	17	5	8	33
Age 30–49 (%)	34	4	25	43	33	4	28	53
Age 50–64 (%)	12	2	6	19	16	3	11	23
Age 65 up (%)	12	4	5	33	10	3	5	20
Foreign born (%)	10	6	0	24	10	6	0	24
Hispanic (%)	4	3	1	16	7	5	2	25
Black (%)	3	8	0	47	3	7	0	35
Male (%)	50	4	44	72	51	4	47	78
Married HHs (%)	56	16	7	77	51	16	3	75
Median family income ($)	57,700	14,383	24,844	101,190	57,700	14,383	24,844	101,190

Both before and after periods use median family income 1999 data downloaded from the Census Bureau because 1999 is the only year where income data are available

persons per square mile), X_{After} is the dummy code of time intervals (1 represents the after period from 1995 to 2000 and 0 represents the before period from 1989 to 1994), $X_{Urban}X_{After}$ is the interaction term of the urbanicity indicator and the time dummy, $X_{Controls}$ is the set of control variables including age, sex, race, ethnicity, marital status, and income, β_0, β_1, β_2, β_3, and β_C are regression coefficients, and ε is the error term.

Findings

Table 4 provides descriptive statistics of various variables in the before and after periods in Portland metro's 71 zip code areas. On average, when comparing the after period (1995–2000) with the before period (1989–1994), the Portland metro experienced a drop in all-cause mortality from 8,814 to 7,725 deaths per million persons per year. Mortality rates of the five cause-specific categories all decreased over time except externally caused mortality. However, without adjustment for demographic composition and socioeconomic characteristics and without relating mortality rates to urbanicity, decreases in crude mortality rates provide insufficient evidence that mortality risks among Portland residents have declined in the past decade.

Descriptive statistics in Table 4 also show time variations in Portland's demographic composition. During the past decade, the Portland metro became more populated and attracted more Hispanic/Latino immigrants. The area saw moderate changes in age distribution. The 50–64 age group increased from 12% in 1990 to 16% in 2000 while the 65+ age group decreased from 12% to 10%. Percentage of married households dropped from 56% in 1999 to 51% in 2000. The gender distribution did not change much in the Portland metropolitan area during the past decade. Large standard deviations and wide ranges shown in Table 4 suggest that substantial variation in mortality rates and sociodemographic attributes exists at the zip code area level.

Table 5 presents regression results from the Portland longitudinal study. Results suggest that, after adjustments for sociodemographic characteristics at the zip code area level, urbanicity-related excess mortality is only observed in the infection category. Only infection-caused mortality is positively related to the urbanicity factor measured by population density, shown by a positive regression coefficient of 0.0052. Coefficients on the urbanicity factor in other models are all negative and significant, indicating that all-cause mortality rates and most cause-specific rates such as tumor, cardiovascular and respiratory diseases, and external causes decrease as the urbanicity of a zip code area increases. In other words, in 1989–1994, Portland not only experienced an urban health penalty (indicated by results from the model for infectious diseases) but to some degree also experienced an urban health advantage (indicated by other models).

Coefficients on the time dummy variable are negative and significant in all the models, indicating that, while holding other variables constant, all-cause and cause-specific mortality rates decreased over time. This finding supports our early expectation that Portland's extensive efforts on curbing sprawl are associated with net decreases in the metro's mortality rates. Coefficients on the interaction term between time and urbanicity show different directions and magnitudes across the six models. However, the interaction term in each of the six models always has a different sign from the urbanicity factor. This pattern suggests narrowed urban–suburban mortality gaps (either urban penalty or advantage) in the after period. For example, in the infectious diseases model, the positive coefficient on the urbanicity

TABLE 5 Regression results from the Portland longitudinal study

Variables	Infectious diseases	Neoplasms/tumors	Cardiovascular diseases	Respiratory diseases	External causes	All causes
Interaction term	−0.0034*	0.0022**	0.0019**	0.0016	0.0021	0.0015*
Time dummy (1 if 1995–2000)	−0.2763**	−0.2953***	−0.4344***	−0.2318***	−0.2084***	−0.2249***
Urbanicity (100 persons per square mile)	0.0052**	−0.0027**	−0.0025*	−0.0034**	−0.0058***	−0.0029**
% Age under 5	−7.9326					
% Age 5–17	−8.6266***	2.5206**	2.5276**	3.3382**		1.6508*
% Age 50–64	−5.0240**	3.9351***	3.8088***	2.5325*		2.7417***
% Age 65 up		5.3751***	5.0376***	5.7367***	3.4160***	5.0588***
% Black	2.6545***		−0.6831**	−1.1124***		
% Hispanic	3.2915***		1.8667***			
% Male	−3.4285***		−2.5747**		4.4830***	
Median Family income ($10,000)	−0.0589**	−0.0554***	−0.0816***	−0.1044***	−0.1484***	−0.0778***
% Foreign born	−0.0210*		−0.0131**		−0.0203***	
% Married HH		−1.5021***	−2.0467***	−2.2834***	−1.5562***	−1.7320***
Constant	−3.9847***	−6.5735***	−4.2244***	−6.7634***	−8.7491***	−4.5714***
Summary statistics						
N	142	142	142	142	142	142
LR statistic	573.43	319.34	347.07	249.80	232.29	337.30
P (alpha) = ~0	0.000	0.000	0.000	0.000	0.000	0.000

Among age groups, "% Age 18–49" is the reference age category. Variables were excluded from models if they are not significant at the 0.1 level
*$p<0.10$; **$p<0.05$; ***$p<0.01$

factor suggests an urban health penalty in the before period and the negative coefficient on the interaction term suggests a decline of the urban health penalty over time. This further indicates that Portland's efforts on curbing sprawl are associated with mitigated urban–suburban health disparities.

Most coefficients on control variables show consistency with previous mortality studies. Poorer areas with more elderly and African Americans generally have higher mortality rates. Areas with higher ratio of married households are generally associated with lower mortality rates. Furthermore, larger male population is associated with higher externally caused mortality but lower infection-caused and cardiovascular-related mortality.

To better understand how the time and urbanicity factors affect health outcomes, all-cause and cause-specific mortality rates and the uncertainty surrounding them are estimated for the before versus after periods across the range of urbanicity, while holding other variables at their medians. For a typical zip code area in the before period (1989–1994) and a typical zip code area in the after period (1994–2000),

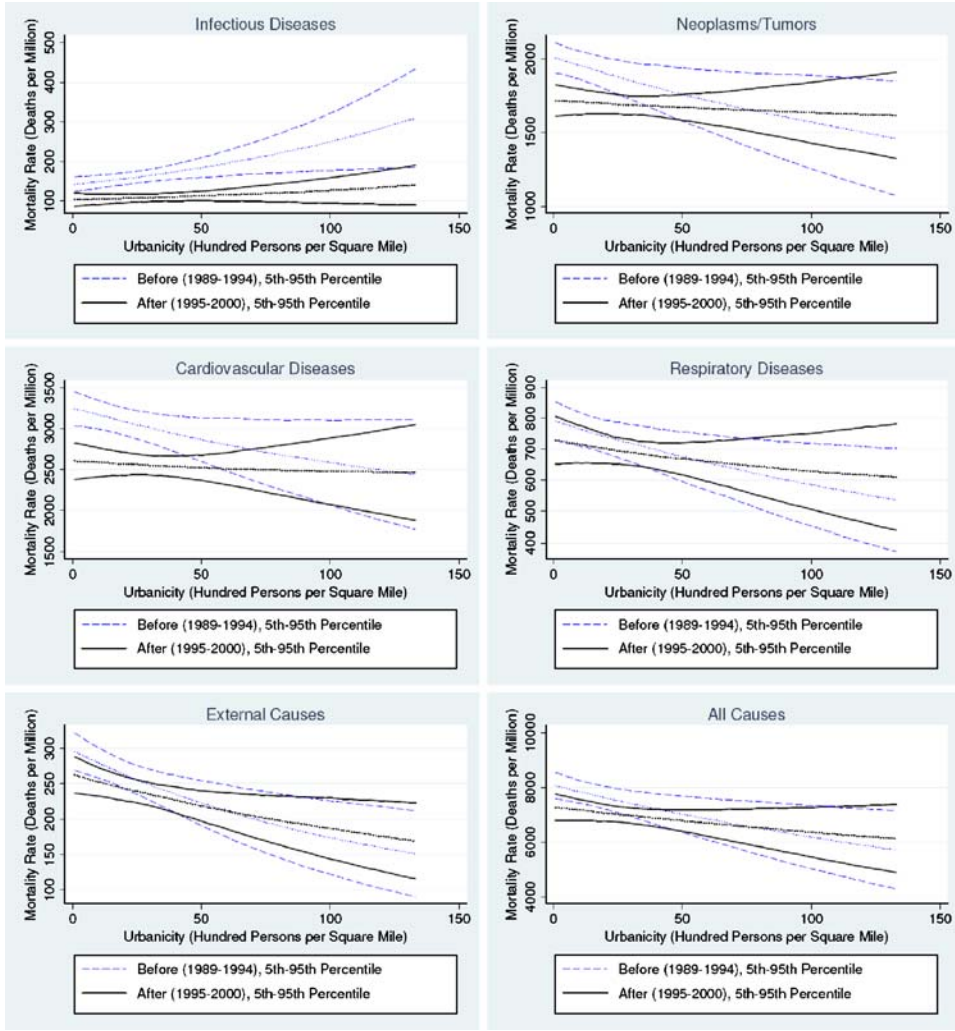


FIGURE 5. Mortality rates by time and urbanicity.

expected value algorithm is repeated to approximate how the 90% confidence interval of mortality rates changes by urbanicity. Statistical software packages including Clarify 2.0 and Stata 8.0 are used again to estimate the expected values and their uncertainty. The estimated results are visually presented in Figure 5.[27]

Figure 5 reinforces the regression findings presented in Table 5. Lines presenting the before period (blue dashed lines in Fig. 5) has steeper increasing or declining trends than the lines presenting the after period (black solid lines). This indicates that, in years 1989–1994, urbanicity-related mortality gaps existed in the Portland metro (i.e., area-based mortality rates change as urbanicity varies). However, urban–suburban mortality gaps existed in years 1989–1994—either urban penalty or advantage—all became less evident in the after period (1994–2000). Furthermore, Fig. 5 shows upward lines when relating infection-caused mortality to urbanicity but downward trends when relating other mortality rates to urbanicity. This suggests that, while the model for infectious diseases predicts excess mortality among urban residents in the Portland metropolitan area, all other models predict elevated mortality among suburban residents in the metro.

CONCLUSIONS AND DISCUSSION

In this paper, we examine whether sprawl is associated with a wider mortality gap between urban and suburban residents. Our national cross-sectional study and Portland longitudinal study provide the only evidence to date that (1) across metropolitan areas, the size of urban–suburban mortality gaps varies by the extent of sprawl: in sprawling metropolitan areas, urban residents have significant excess mortality risks than suburban residents, while in compact metropolitan areas, urbanicity-related excess mortality becomes insignificant; (2) the Portland metropolitan area not only experienced net decreases in mortality rates but also a narrowing urban–suburban mortality gap since its adoption of smart growth regime in the past decade; and (3) the existence of excess mortality among urban residents in US sprawling metropolitan areas, as well as the net mortality decreases and narrowing urban–suburban mortality gaps in the Portland metropolitan area, is not attributable to sociodemographic variations. The national and Portland studies, although very different in scope and approach, point to similar directions. Both suggest a positive association between sprawl and urban–suburban health disparities. This further indicates that growth management strategies and policies such as UGB and impact fees may reduce the mortality gap between urban and suburban residents through curbing and preventing sprawl.[32]

When looking at cause-specific mortality, infectious diseases and external causes show unique patterns from all other causes. Excess mortality among urban residents is most evident when examining infection mortality rates, shown by a much larger coefficient on the urban dummy variable in Table 3 and the positive relationship between infectious disease mortality and urbanicity in the Portland study. The high relevance of infection deaths to urbanicity suggests that crowding and density pose health risks to residents, perhaps potentiated by rapid transmission from one person or specie to another in populated urban environments. External causes also show unique mortality patterns. In the national study, the external causes model is the only model that shows no significant association among mortality rates, urbanicity, and sprawl. This finding contradicts the general perception that living in cities is equated with higher risks of accidental deaths due to exposure to more traffic accidents and violent crimes. However, the finding concurs with previous empirical

findings.[5,33] A possible explanation is that the model estimates mortality rates rather than the absolute number of deaths and people tend to mistakenly perceive higher risks in cities because of the high absolute number of accidental deaths.

Most importantly, while much research on sprawl has identified negative health consequences of sprawl,[20–22] our research has shown that it is also important to investigate the spatial distribution of health outcomes within the metropolitan area and especially the health disparity issues between central cities and suburbs. By looking at how sprawl and urbanicity may interactively affect mortality, we find that sprawl is associated with not only net mortality increases at the metropolitan level but also wider intrametropolitan mortality gaps between urban and suburban residents. Mortality risks imposed by sprawl affect urban residents disproportionately compared to suburban residents. In other words, this paper provides convincing evidence on the notion "sprawl is bad for health" without contradicting the fact "suburban residents are generally healthier."

Although the research provides a framework for examining the sprawl–health inequity association, the framework is largely preliminary and exploratory and raises important issues for future research. For example, when investigating health disparities burdening urban residents, both measuring health and defining urbanicity determine the results.[34] While level of urbanization can be conveniently used to categorize urban versus suburban areas, this scheme may mask important differences within urban or suburban areas.[35] An attempt should be made to develop alternative measurements of urbanicity. Furthermore, the national mortality analysis is conducted at the county level, which admittedly limits the interpretation of the analysis results. Within the same county, the built environment may vary considerably. Future research may be conducted at more disaggregate levels to develop a finer-grained understanding of how neighborhood-level built environment features may contribute to urban–suburban health disparities. In addition, information on housing location choice among urban/suburban residents may be collected to address self-selection bias—the issue that healthier people select themselves to "healthier" places. Finally, the transferability of findings from our Portland longitudinal study is somewhat limited. Researchers can apply the longitudinal approach to a much larger and more diverse sample of metropolitan areas. We hope that this study will stimulate research on the relationship between sprawl and health disparities.

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