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An Application of Spatially Autoregressive Models to the Study of US County Mortality Rates

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ABSTRACT

County mortality rates in the US tend to be associated with social and economic resources of counties and the unequal distribution of these resources across space. The processes that generate these social and economic inequalities are often tied to geographical location. In this paper, we present an application of spatially autoregressive models of US county mortality rates that control for the social and economic conditions that often influence mortality rates and the effects of spatial structure of counties in the US. We suggest that arguments are missing from the social science and demographic literatures to offer possible explanations for the spatial patterning of county mortality rates and ecological correlates of these rates at the county level. We find that, after controlling for spatial structure in the data, several key social variables become insignificant in the analysis. We suggest that spatial statistical models are valuable tools in the social and behavioural sciences but that the use of these methods needs to be well grounded in considerations about the spatial process inherent to the outcome studied, and the applications of these methods should not be used solely for post hoc statistical correction. Copyright © 2009 John Wiley & Sons, Ltd.

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INTRODUCTION

Problem Statement and Purpose

Variability in mortality rates across US counties and states has been noted in the literature (Clifford and Brannon, 1985; LeClere *et al.*, 1997; Geronimus *et al.*, 1999; McLaughlin *et al.*, 2001; McLaughlin and Stokes, 2002; Morton, 2004; Diez Roux *et al.*, 2007). Most often, studies examining county mortality rates either are largely descriptive or offer some evidence for correlations among certain ecological factors within counties and the county's subsequent mortality rate. More recent research has used basic exploratory spatial data analysis tools to map this variability and document the existence of spatial clusters in mortality rates. Spatial clusters for counties with both high and low mortality rates have been noted at a single point in time (Morton, 2004; McLaughlin *et al.*, 2007) and over several time periods (Cossman *et al.*, 2003).

Spatial clusters among ecological factors measured at the county level are also observed when conducting exploratory spatial data analysis. Spatial clusters can be found among counties with a high percentage of minority populations, high county poverty rates, and county levels of income inequality. However, current literature addressing the spatial distribution of aggregate mortality risks has not fully explained the mechanisms influencing this spatial pattern, based on the potential spatial distribution of other factors associated with higher or lower county mortality rates. This could partly be the result of

methodological and conceptual questions about the appropriate level for assessing aggregate mortality risks (Diez Roux, 2001), as well as how other ecological factors may lead to different aggregate mortality risks for certain members of the population, such as racial/ethnic minorities or persons living in poverty. It is also necessary to consider how health-promoting or health-harming resources are spatially located and how the uneven spatial distribution of these resources and population characteristics may impact mortality chances. However, empirical research exploring this uneven distribution of resources across space must consider the appropriate mechanisms that link these aggregate associations to the spatial process leading to the pattern being studied and observed.

The purpose of this paper was to address the use of appropriate spatial statistical modelling techniques to assess the role of space in influencing outcomes that have an inherent spatial dimension, in this case, US county mortality rates. Further, we argue that the missing component to the appropriate application of these methods to the study of aggregate health outcomes is poor or limited conceptual development on the existence of spatial heterogeneity in these outcomes. Therefore, we offer a more detailed argument based on spatial relationships in the econometric literature that differentiates between possible explanations for spatial heterogeneity in mortality rates. Specifically, this paper examines the relationships between race/ethnicity and resources, broadly defined, based on a spatial inequality framework. However, this paper moves beyond basic arguments made at the aggregate level that relate county resources and population composition to mortality rates to offer a conceptual and empirical model that specifies the nature of the spatial process at work. We first use a standard ordinary least squares (OLS) model to estimate the effects of county-level characteristics on mortality rates. We describe how this approach fails to incorporate possibly important explanatory factors into the model and explains little of the variation in county mortality rates. In particular, the OLS model fails to account for spatial structure and clustering in our data.

To extend the analysis, we use both weighted least squares and spatial linear regression models to examine the relationship between standardised mortality rates and county-level contextual variables using spatial error and spatial lag models, based on our understanding of the nature of the spatial process for this outcome. We are interested in how the influence of spatial proximity and connectivity affects the regression model estimates after the addition of spatially autoregressive parameters that model the effects of the spatial structure of the data. Our goal for this paper was to understand how the incorporation of spatial parameters in our statistical models, based on a more focused understanding of the spatial process at work, can better account for the effects of ecological factors on mortality rates in the US.

Background Information and Theoretical Development of Spatial Processes

Research in the public health literature has had an emerging focus on examining the impact of the environment, both physical and social, on a variety of health outcomes (Macintyre *et al*., 1993; Jones and Duncan, 1995; Kaplan, 1996; Robert, 1999). Numerous studies have examined the impact of structural, contextual, or ecological factors on individual health outcomes using multilevel modelling techniques (Jones and Duncan, 1995; O'Campo *et al*., 1997; Patel *et al*., 2003; Stuber *et al*.,. 2003; Chaix *et al*., 2005). These studies have found that variables measured at a higher level of aggregation may have independent or interactive effects on individual health outcomes (Soobader *et al*., 2006). Still, this statistical method has not allowed for the explicit spatial distribution of the aggregate level areas, or their populations and resources, to be examined as they relate to mortality risks for the US population as a whole. A recent study by McLaughlin *et al*. (2007) finds that the spatial patterning of county mortality rates is associated with economic characteristics, racial minority concentration, social conditions and safety, health-care services distribution, and environmental risks in counties. The inclusion of a spatial lag parameter in the spatial linear regression model indicates that several of the relationships between county mortality rates and the above-mentioned county-level factors become stronger in these models. This result suggests that more traditional linear regression models misspecify the possible relationships between county ecological factors and mortality rates if the inherent spatial dimension

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of these relationships is not considered. Further, the particular spatial model specified in their research assumes that a spillover type process is operating. However, this may not be the most appropriate conceptual model to use in studying aggregate mortality rates.

Often, the literature on spatial regression models fails in justifying why either a spatial lag or a spatial error model is most appropriate for examining specific outcomes. Additionally, in the social sciences, spatial regression models are often selected only to meet the analytical needs of data collected over space. A first step in determining the spatial analysis technique most appropriate for examining specific health outcomes must be considering conceptual matters with regard to the specific outcomes and then specifying the spatial process inherent to the research problem (Anselin, 2002; Morenoff, 2003). Anselin (1988) uses the term *spatial dependence* as a general term to refer to either a spatial lag model or a spatial error model. *Spatial heterogeneity* is another way to consider a spatial effect, and Baller *et al*. (2001: 566) define spatial heterogeneity as 'a situation in which coefficients or error patterns vary systematically across geographic areas'. Based on this definition, it would not be clear if the observed spatial pattern in county mortality rates is a result of errors associated with unmeasured variables at the county level or possible impacts of neighbouring mortality rates on a county's specific mortality rate. Therefore, spatial diagnostic tests can help to determine if a spatial error or spatial lag model would best approximate the spatial process underlying the observed pattern in county mortality rates with controls for spatial heterogeneity. Still, these diagnostic tests, which are based on the ordinary or Robust Lagrange multiplier statistic, do not offer a conceptual connection to the appropriate selection of either of these two spatial models.

Conceptually, the spatial lag and spatial error models imply very different spatial processes. A spatial lag model most closely represents a diffusive process in the outcome, implying that the value of a health outcome in one location is influenced by that in a neighbouring location for that particular health outcome. This means that the dependent variable being examined is lagged across all the neighbours for an area, while the spatial impact of unmeasured independent variables in the model is also considered. This type of spatial process for studying health outcomes is probably best suited to the study of infectious diseases and possibly social network processes as they spread among people in different spatial locations. This particular type of spatial model has been used to examine birth weight in Chicago neighbourhoods (Baller *et al*., 2001), hospitalisation rates for low-back problems in North Carolina counties (Joines *et al*., 2003), and US county morality rates (McLaughlin *et al*., 2007). However, we argue that the spatial process inherent to each of these health outcomes does not operate by a diffusive process. Joines *et al*. do the most to offer potential explanations of the diffusive mechanisms underlying their selection of a spatial lag model to examine hospitalisation rates of low-back problems. They first argue that patients in counties who are happy with their health-care service experience may talk to patients with similar low-back pain in neighbouring counties, and this stream of communication would lead to spatial clustering in their outcome. Second, they argue that standards for appropriate medical care among medical specialists may spread by referrals or interaction between doctors to lead to spatial patterns in hospitalisation rates for low-back problems. Still, this communication stream that is used to explain the existence of the spatial process in hospitalisation rates between neighbouring counties in treating low-back problems does not seem to best approximate the spatial process underlying this health outcome. It would seem that what is more likely to explain this particular spatial pattern at such a high level of aggregation and those in the other studies mentioned above are the result of unobserved independent variables being omitted from the model. A diffusive process, as captured by a spatial lag model, would probably best be operationalised at a very local level.

The spatial error model would use this logic to say that the spatial pattern observed is a result of unmeasured independent variables. For the particular research problem addressed here, the spatial error model would argue that the clustering of county mortality rates not accounted for by the independent variables included in the model is the result of correlated error terms among the independent variables and omitted independent variables from the model (Anselin, 1988; Baller *et al*., 2001). More specifically, this spatial model would indicate that the spatial

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process at work is not diffusive, meaning, that the mortality rate in one county does not increase the likelihood that a neighbouring county will have a similar mortality rate. Instead, the spatial process leading to spatial clusters in county mortality rates is a result of the spatial process inherent to the independent variables that are both measured and omitted from the empirical model specification (Morenoff, 2003). This type of argument makes it necessary to understand the spatial nature of ecological characteristics associated with county mortality rates and to better specify how these processes are likely to vary over space.

Recent research exploring obesity rates in ZIP codes areas of King County, Washington (Drewnowski *et al*., 2007), and pneumonia and influenza hospitalisations in Ontario, Canada (Crighton *et al*., 2007), used a spatial error model to test the spatial dependence in these two outcomes. Both papers note that the spatial error model offers the best solution to accounting for spatial dependence, but neither paper offers more detailed explanations for selecting this model. The paper by Crighton *et al*. does discuss the use of the Lagrange multiplier statistic as a diagnostic tool in selecting the spatial error model over the spatial lag model and potential issues with the scale at which the spatial process is examined. Still, more work is needed to specify the spatial pattern of ecological characteristics associated with health outcomes at an aggregate level to guide the development of spatially based empirical models.

When studying a process such as human mortality, we are interested in documenting if autocorrelation exists in both the causal factors that directly influence mortality rates and the underlying causes of unequal distributions of resources that are known to lead to difference in health and well-being. The study of human mortality rates within a regression framework assumes that we have measured independent variables that influence the observed rates. Much of the previous work on these ideas has focused on the inequality that exists between segments of the US population, based on race/ethnicity, income, educational levels, and household composition (Shin, 1975; Christenson and Johnson, 1995; McLeod *et al*., 2004; Pampel and Rogers, 2004; LaVeist, 2005a;Vinnakota and Lam, 2006). How these inequalities can contribute to the observed patterns of life expectancy and mortality has become a valuable line of enquiry in demographic studies of health (Waitzman and Smith, 1998; McLaughlin and Stokes, 2002; Vinnakota and Lam, 2006). When the factors that influence inequality are examined, recent work has suggested that place-based and spatially based studies of inequality can help us understand the process that leads to mortality differentials and, more generally, health inequalities from a broad income-inequality perspective (LaVeist, 2005b; Gibbons *et al*., 2007; Irwin, 2007; Lobao *et al*., 2007). Yet more work is needed to justify the appropriate use of spatial statistical tools in the study of place-based health outcomes and of how specific aggregate independent variables may partly lead to the spatial patterns observed for certain aggregate health outcomes.

This paper has three goals: (i) to document the spatial autocorrelation that exists in US county mortality rates, (ii) to estimate spatial autoregressive models for US county mortality rates based on an understanding of the spatial process in the outcome, and (iii) to estimate the effects of several social and economic inequality indicators on US county mortality rates after controlling for this spatial structure. Based on our understanding of the spatial process inherent to the study of county mortality rates, we hypothesise that a spatial error model will best approximate the spatial process in our outcome. Further, while we use diagnostic statistics as one means to make a decision between selecting a spatial lag or spatial error model, we argue that conceptual decisions should guide the selection of appropriate spatial statistical models prior to estimating these diagnostic statistics. This step is necessary because diagnostic statistics are often very similar in value, and it becomes difficult to select an appropriate spatial model based on this criterion alone. In the next section, we detail the data and methods that allow us to test the appropriateness of using a spatial error model to examine US county mortality rates.

DATA AND METHODS

Data for this analysis are taken from two sources: Compressed Mortality Files from Centers for Disease Control and Prevention (CDC) Wonder at the National Center for Health Statistics and the 2005 release of the Area Resource Files. Five-

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year age-sex-race standardised rates for the years 1998–2002 serve as the dependent variable in this analysis. Standardisation of mortality rates is used in order to facilitate the comparison of rates across groups. It is important in this analysis to standardise mortality rates based on age, sex, and race because mortality risks vary greatly based on these demographic characteristics. For this analysis, we use the age-sex-race distribution of the 2000 US population as our standard population. Specifically, the direct standardisation equation used to calculate the individual county-level mortality rates is

$$DS^{ASR} = \frac{\sum_{i=1}^{k} m^{ASR}(x)\,P^{2000}(x)}{\sum_{i=1}^{k} P^{2000}(x)}, \quad (1)$$

where $m^{ASR}(x)$ is each age – sex – race death rate in each county from 1998 to 2002 and $P^{2000}(x)$ is the age–sex–race distribution of the 2000 US population. In essence, direct standardisation utilises the same set of weights to the age-sex-race–specific mortality rates of each of the US counties, and the resulting standardised mortality rate is independent of variation in the age–sex–race distribution of each county's population.

Independent variables for this analysis are taken from the Area Resource Files and serve as county-level social and economic inequality indicators. We include nine independent variables in our analyses: percentage of the county population that is rural; percentage of the county population that is black; percentage of the county population that is Hispanic; the percentage of the county population that lives below the federally designated poverty threshold; the percentage of households in the county with a female head; the county unemployment rate; the median household income in the county; the county's median house value; and the population density per square mile in the county. The percentage of the county population that is rural, defined as a population not classified as urban by the Census Bureau, is constructed by dividing the county's rural population by the total county population and multiplying this value by 100. Similarly the percentages of the county population that is black or Hispanic are constructed by taking the total number of black or Hispanic residents per county, dividing those numbers by the total county population in 2000, and multiplying the value by 100. The number of households with a female head is divided by the total number of households to obtain the percentage of households in the county with a female head. The unemployment rate is calculated by dividing the number of unemployed persons in the county by the civilian labour force in that county. Median home values are calculated by the Census Bureau using information contained in the 2000 Census Summary File 1. Population density per square mile was calculated by dividing the total population of the county by the area of the county in square miles. Finally, the Gini coefficient for household income in the county is included as a measure of relative income inequality within the county, with a value of 1 indicating perfect inequality and 0 indicating perfect equality in incomes. To overcome differences in variable scaling, we calculate *z*-scores for all variables in the analysis and use these in our regression models.

Measures of Spatial Autocorrelation

The first step in the analysis is an exploratory one, intended to discern if there is clustering in the data. We use the global Moran's *I* statistic to examine the variables in our data set for global autocorrelation. The interpretation of the Moran's *I* statistic proceeds much like the interpretation of a standard correlation. If the observed value of *I* is greater than its expected value, E[*I*], then an observation tends to be surrounded by neighbours with similar values, while if I < E[*I*], the observation tends to be surrounded by dissimilar values (Schabenberger and Gotway, 2005). Testing of the Moran statistic is carried out via randomisation methodology (Schabenberger and Gotway, 2005) and assumes a Gaussian distribution for the randomised observations of *I*. While the global estimate of the Moran statistic provides a single summary spatial correlation measure, the assumption of constant variance and homogeneity of the mean of the spatial process generating the data is often a weak one. We also consider the local version of the Moran statistic, referred to as a local indicator of spatial autocorrelation, or Local Indicator of Spatial Autocorrelation (LISA) (Anselin, 1995). Anselin's LISA statistic is calculated for each county in the data, taking into account the variation that exists in the

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surrounding observations (the local spatial neighbourhood defined by the spatial weights matrix, **W**). This local indicator is calculated as

$$I(y_i) = \frac{n}{(n-1)\sigma^2}(y_i - \mu_y)\sum_{j=1}^{n} w_{ij}(y_j - \mu_y), \quad (2)$$

where μ_y is the mean of the variable, σ^2 is the sample variance, and the local values at all locations i sum to the global Moran statistic. Once again, the interpretation of the LISA statistic is similar to the global Moran: if $I(y_i) > E[I(y_i)]$, then there is local positive autocorrelation at location i and a large (or small) value at that location tends to be surrounded by large (or small) neighbouring values. Likewise, if $I < E[I(y_i)]$, there is local negative autocorrelation and a large (or small) value at location i tends to be surrounded by small (or large) neighbouring values (Schabenberger and Gotway, 2005). As with the global Moran statistic, significance is judged by randomisation of the observed clusters. For example, if a county has a high mortality rate and neighbouring counties also have high mortality rates, there will be high local autocorrelation at that location. We present figures showing the distribution of counties classified as having significant high–high and low–low clusters. The cluster maps are useful for visualising the local clustering of mortality rates and geographical clusters of our other variables.

Spatially Autoregressive Models

When non-spatial data are analysed, the standard linear (OLS) model of analysis is the general choice for regression and prediction. Several of the basic assumptions of the OLS model are that the model residuals are normally distributed and that they have common unit variance. Spatial data, however, present a series of problems to the standard OLS regression model. These problems are typically seen as various representations of spatial structure within the data. By structure, we are referring to the ideas of autocorrelation and non-stationarity of the distributional parameters (mostly mean and variance) or heteroskedasticity (unequal variance) of the model residuals. Autocorrelation can be defined, in a general sense, as the co-occurrence of similar values at closely spaced spatial locations. This can be observed as neighbouring observations, both with high (or low) values (positive autocorrelation). We can also observe situations where areas with high values can be surrounded by areas with low values (negative autocorrelation). Because the standard OLS model assumes that the residuals are uncorrelated, as previously stated, the autocorrelation inherent to most spatial data introduces factors that violate the iid distributional assumptions and the assumption of common variance for the OLS residuals.

To account for the expected spatial association between our dependent variable (mortality rates) and our independent variables, we estimate a series of models that account for local structure in both the dependent variable (spatial lag model) and the autocorrelation in the model residuals (spatial error model). We run standard Lagrange multiplier diagnostic tests in order to show support for our hypothesis that a spatial error model best describes the spatial dependence present in our outcome. We begin by specifying the baseline OLS model, assuming a Gaussian distribution for our z-scored mortality rates specified as

$$\begin{aligned} &\mathbf{Y} = \mathbf{X}\beta + \mathbf{e}, \\ &\mathbf{e} \sim (\mathbf{0}, \Sigma) \end{aligned} \quad (3)$$

where **Y** is the vector of mortality rates, **X** is the matrix of independent variables, β is the vector of regression parameters to be estimated from the data, and **e** are the model residuals, which are assumed to be distributed as a Gaussian random variable, with mean 0 and constant variance-covariance matrix **Σ**. We specify the OLS model as

$$\begin{aligned} Mort_z = {} & \alpha + \beta_1 \%Rural_z + \beta_2 \%Black_z + \beta_3 \%Hispanic_z \\ & + \beta_4 \%FemHH_z + \beta_5 \%Unemploy_z + \beta_6 MedHomeValue_z \\ & + \beta_7 PopDensity_z + \beta_8 Gini_z + \varepsilon \end{aligned}$$

where $\%Rural_z$, $\%Black_z$, $\%Hispanic_z$, $\%Poverty_z$, $\%FemHH_z$, $\%Unemploy_z$, $MedHomeValue_z$, $PopDensity_z$, and $Gini_z$ are the z-scored variables described above, with α being the model intercept, and ε the model error term. Before the analysis, we examined the distribution of the mortality rates and found that it was not significantly skewed, so the normality assumptions of the OLS model are met. Also, we consider a Gaussian distribution for our data because they are represented as standardised rates instead of counts of events, which would necessitate a Poisson

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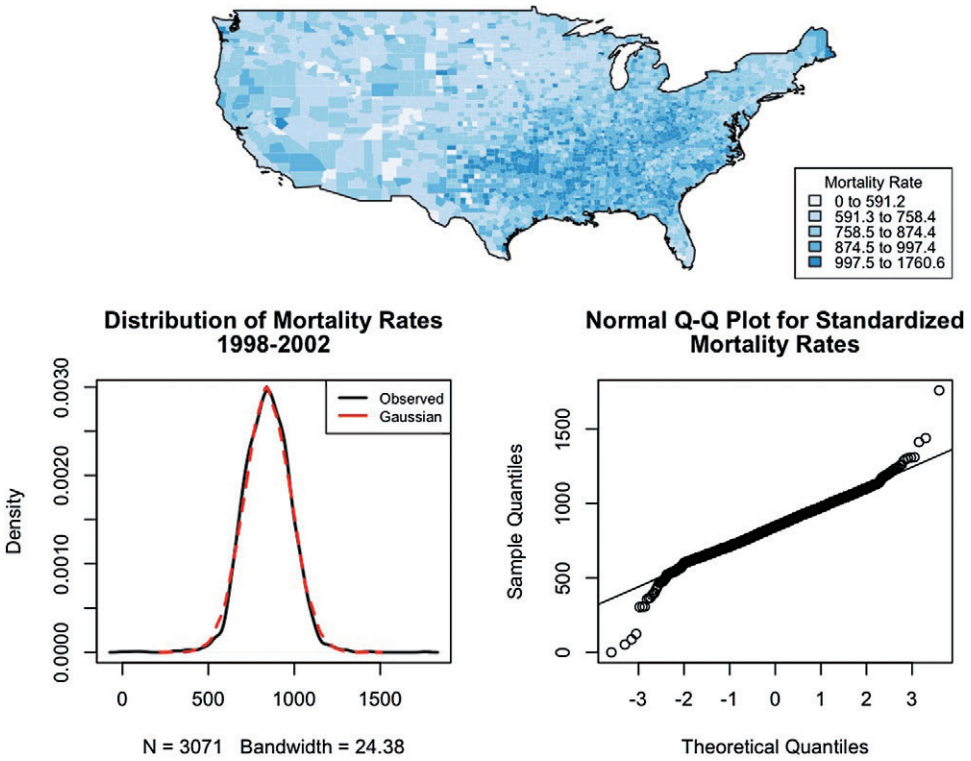

Figure 1. Geographical distribution and comparison to Gaussian distribution of US age, sex, and race standardised mortality rate, 1998–2002.

distribution to model such counts of events. We present the geographical distribution of our dependent variable and plots of its density and comparison to a Gaussian distribution in Figure 1. While an initial Shapiro–Wilk (Royston, 1995) test of normality shows a significant deviation from a Gaussian distribution (W = 0.987, P = <0.0001), we perform some further testing of our dependent variable. Because the sample size for our analysis is rather large (n = 3071), we have a high degree of statistical power to detect any deviation of our data from normality, but, on the other hand, the Shapiro–Wilk test may be overly sensitive in this case to our large sample size and reject the null hypothesis when, in fact, our data are fairly consistent with the Gaussian distribution. To explore this, we conduct a bootstrapping analysis of the Shapiro–Wilk test. We generate 9999 samples with replacement, each of size n = 1000 and compute the Shapiro–Wilk test for each sample to generate a distribution of the W statistic. When we compare our observed value of W with the bootstrapped distribution, we found that 43.9% of the bootstrapped values were greater than or equal to our observed value, giving a bootstrapped p-value of 0.453. Based on this, we conclude that, while our entire sample may deviate significantly from normality using traditional testing, further examination reveals that the deviation lies purely in the extreme tails of the distribution, and there is no marked skewness or kurtosis to the rates. Furthermore, because in a regression framework, our inference is really on the mean of our dependent variable, we believe we can make strong inference about (at least) the 95% confidence interval around the mean, if not the entire distribution. Following the basic fitting of the OLS, we estimate the value of Moran's I for the fitted model residuals. This test is equivalent to testing the assumption of the OLS model that the residuals from the model fit are iid (Cliff and Ord, 1981).

One major limitation to our dependent variable is the degree to which counties with small

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populations have unstable (meaning from year to year) rates because of their small population sizes. To accommodate the possible effect of this, we also fit a weighted least squares model to assess the effect of county population size to our dependent variable. We include a weighting variable that is the inverse of the average of the county's population size from 1998 to 2002. This effectively gives less weight to counties with large population sizes (Neter *et al*., 1985; McLaughlin and Stokes, 2002; Waller and Gotway, 2004; McLaughlin *et al*., 2007).

After examining the results of the OLS model, we estimate the spatial autoregressive models; first the autoregressive error then the autoregressive lag model. For the spatial autoregressive error model to be specified, the model residuals from the OLS model are regressed onto a set of spatial dependence parameters:

$$\begin{aligned} \mathbf{Y} &= \mathbf{X}\beta + \mathbf{e}, \\ \mathbf{e} &= \mathbf{B}(\mathbf{e} + \mathbf{v}) \end{aligned} \tag{4}$$

where **B** is a matrix of spatial dependence parameters and **v** is an uncorrelated and homoskedastic error term. **B** is defined as

$$\mathbf{B} = \rho\mathbf{W}, \tag{5}$$

where ρ is the spatial autoregressive parameter and **W** is the row-standardised spatial connectivity matrix defined as

$$\mathbf{W} = w_{ij} = \frac{w_{ij}}{\sum c_i}, \tag{6}$$

where c_i is the total number of neighbours a county has and $w_{ij} = 1$ if two counties share a border or an apex, and 0 otherwise (the Queen form of spatial contiguity). Counties are said to be Queen contiguous if they share a line segment or a single point of their border (Anselin, 2002). The process of row-standardising the spatial weights equally distributes the weight of neighbouring values of counties that have few versus many neighbours, effectively averaging the values of the neighbouring counties in this case (Tiefelsdorf and Griffith, 1999; Anselin, 2002).

The spatial error model is then specified:

$$\begin{aligned} \mathbf{Y} &= \mathbf{X}\beta + \mathbf{e} \\ \mathbf{e} &= \rho\mathbf{W}\mathbf{e} + \mathbf{v} \end{aligned} \tag{7}$$

with the full specification being

$$\mathbf{Y} = \mathbf{X}\beta + (\mathbf{I} - \rho\mathbf{W})^{-1}\mathbf{v}, \tag{8}$$

where **I** is an identity matrix and **v** is an independent error term. This model is often referred to as the one-parameter autoregressive error model because we only end up estimating the global autoregressive parameter ρ by maximum likelihood (Ord, 1975; Cliff and Ord, 1981; Anselin and Bera, 1998; Schabenberger and Gotway, 2005). This model, in effect, controls for the nuisance of correlated errors in the data that are attributable to an inherently spatial process or to spatial autocorrelation in the measurement errors of the measured and possibly unmeasured variables in the model (Anselin and Bera, 1998).

The spatial lag model uses a slightly different logic than the error model, where we model the autoregression of the mortality rates themselves, much like in a time-series approach (Anselin and Bera, 1998). The model is specified as

$$\mathbf{Y} = \rho\mathbf{W}\mathbf{Y} + \mathbf{X}\beta + \mathbf{e}, \tag{9}$$

where ρ and **W** are specified as in the spatial error model. In the lag model, we are specifying the spatial component on the dependent variables rather than the error structure. This leads to a spatial lagging of the mortality rates, where they are averaged over the surrounding neighbourhood defined in **W**.

Finally, following the procedure outlined in Waller and Gotway (2004), we estimate a weighted spatially autoregressive model that uses the inverse of the county's population size (see above) as the weighting variable. As with the weighted OLS model, this is primarily carried out to account for population size heterogeneity between the US counties.

We estimate the spatial autocorrelation statistics and the autoregressive models given in equations (8) and (9) using the spdep (Bivand *et al*., 2008) library in R 2.8.1 (R Development Core Team, 2009). For further discussion of the asymptotic properties and maximum likelihood estimators for both models, we encourage the readers to examine the following sources: Anselin (1988), Anselin and Bera (1998), and Ord (1975). We also present general and Robust Lagrange multiplier statistics (Anselin *et al*., 1996; Baller *et al*., 2001) to

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test for the presence of residual autocorrelation in the OLS model to test our proposition that the spatial error model is the more appropriate model in this circumstance.

RESULTS

We first present the results of the global spatial autocorrelation analysis for each variable in our analysis. Table 1 gives the observed and expected values of Moran's *I* for each of the 10 variables in the analysis. We see varying levels of global autocorrelation, with the percentages of the population that are black and Hispanic showing the highest degrees of spatial correlation, followed by the socio-economic indicators, while the percentage of the county population that is rural showing the lowest degree of spatial autocorrelation among the independent variables in our analysis.

The results of the analysis of local autocorrelation analysis are presented in Figure 2. Because the local Moran's *I* statistics refer to a specific geographical location (neighbourhood of counties), it is often useful to graphically depict the type of local autocorrelation detected as a cluster map showing counties that have high values of each variable surrounded by neighbours that likewise have high average values for the same variable (high–high clusters). In positive autocorrelation, you can also have a county with a low value surrounded by neighbours with a low average value (low–low clusters). Additionally, there can be spatial 'outliers', such as counties that have a high value for a variable but are surrounded by low average values for that variable, a so-called high–low outlier. Similarly, if a county's value is low, but the area surrounding it has a high average value, the county will be classified as a low–high outlier. Outliers are examples of locations showing negative spatial autocorrelation. We plot the cluster assignments for each of the US counties using all variables in this analysis in Figure 2, showing whether they are part of a high–high cluster, low–low cluster, an unclustered neighbourhood, or a high–low outlier, or low–high outlier. While we use the term *outlier* for our discussion, these observations may not be true statistical outliers, meaning influential observations, but instead show a pattern contra to the dominant pattern of positive spatial autocorrelation.

Table 1. Global Moran's *I* values for variables in the analysis.

Variable	Observed *I*	*E*[*I*]	*Z*[*I*]
Mortality rate	0.508	–0.0003	47.39
% Rural population	0.304	–0.0003	28.37
% Black population	0.797	–0.0003	74.27
% Hispanic population	0.825	–0.0003	77.05
% Female HH heads	0.589	–0.0003	54.96
Unemployment rate	0.467	–0.0003	43.6
Median house value	0.584	–0.0003	55.09
Population density	0.598	–0.0003	67.18
Gini coefficient (income)	0.394	–0.0003	36.81

HH = high–high cluster; *Z*[*I*] = the observed I value's standard deviate under the H0 of no association; *E*[*I*] = the expected value of Moran's *I*.

Mortality rates exhibit high–high clusters throughout the Southern Plains, Southeastern, and Appalachian regions of the US, and low–low clusters are observed in the western mountain regions of the country and in some areas of the southwest and Midwest. The high–high clusters indicate that counties in these clusters are observed to have high mortality rates and share boundaries with counties that have high mortality rates. The low–low cluster pattern indicates that counties that have low mortality rates tend to share borders with counties that also have low mortality rates. We suspect that high levels of economic inequality, high poverty, and poor economic development generally characterise the areas with high–high clusters.

Counties with a high percentage of the population that is rural have several areas of high–high clustering: Appalachia, the northern plains and some parts of the Deep South, while low–low clusters occur mostly in the highly populated, large metropolitan coastal areas of the east, and West coasts, south Florida, the Great Lakes region, and several areas of the southwest. The high-high clusters represent areas that are purely rural in character and generally show a high degree of isolation from urban centres, while the low-low clusters are generally places with high population densities and a more urban environment.

The percentage of the county population that is black exhibits high–high clusters in the Deep South, corresponding to the so-called 'black belt crescent' region, and low–low clusters are found in the northeast, northern plains, and western

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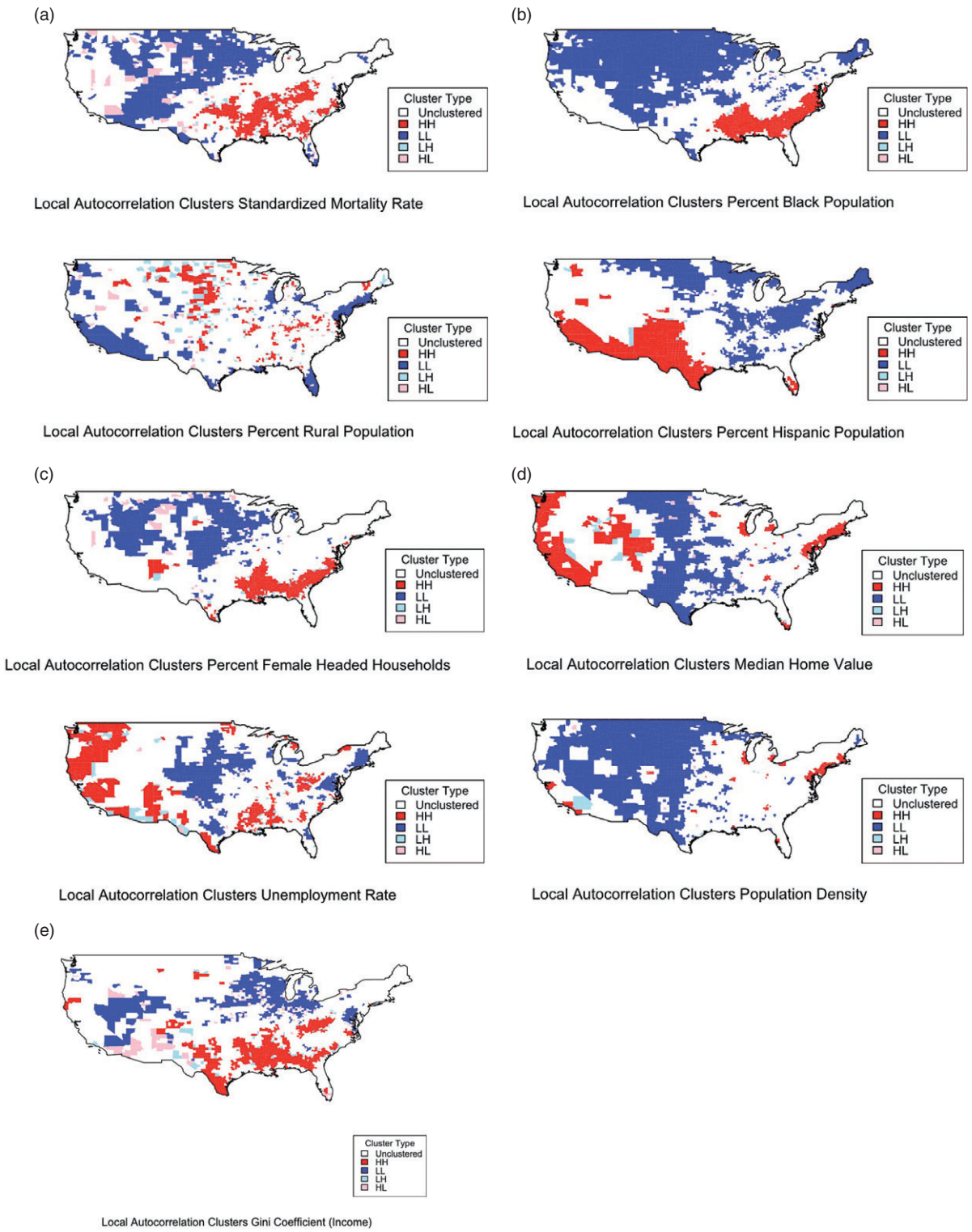

Figure 2. Local autocorrelation cluster map for (a) US county mortality rates and percentage of the county population that is rural; (b) percentages of US county populations that are black and Hispanic; (c) percentages of US county households that are female headed and county unemployment rates; (d) median housing value and county population density; and (e) Gini coefficient for income for US counties.

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mountain regions. The percentage of the county population that is Hispanic exhibits high–high clusters along the US–Mexico border, south Florida, and some parts of the southwestern US, and low–low clusters are found in the northeast, southeast, and northern plains. The spatial patterns observed for both blacks and Hispanics are consistent with settlement patterns in the US, based on racial residential segregation, although recent work has determined that foreign-born Hispanic migration streams are shifting to other areas of the rural South specifically (Lichter and Johnson, 2006).

Female-headed households exhibit high–high clusters in the southeast, south Texas, and some parts of the southwest, areas that also exhibit high clustering of minorities, while low–low clusters are generally observed in the central and northern plains. County unemployment rates show many areas of high–high clustering in the northwest, southwest, Deep South, and northern Appalachia. Low–low unemployment clusters exist in the central plains, some areas of the Midwest, central Texas, and areas in the Northeast around large cities.

Housing values show high–high clusters on the east and west coasts, central western mountains, southern Florida, and some areas of the Midwest around large cities. Low–low housing value clusters are found throughout the northern, central, and southern plains. Population density shows high–high clustering around large cities throughout the country but particularly on the east and western coasts, the Midwest, and some areas of the south. Low–low clusters are found throughout the western mountains, northern plains, small areas of Appalachia, and the southwest. Finally, the Gini coefficient of income inequality shows high–high clustering throughout the southeast and Appalachia, the Rio Grande Valley, and some areas of the far west. Low–low clusters exist around the Mid-Atlantic, the Great Lakes, and Northern Plains region and areas of the Rocky Mountains. Most of the high–high clusters of income inequality correspond with high–high clusters of the percentage of the county population that is black or Hispanic. This may indicate that economic and social resources are shared less equally in areas with high concentrations of minority populations, which should predict higher mortality rates for these areas as well.

Based on these results, we proceed with the estimation of the OLS and weighted OLS, spatial lag, spatial error, and weighted spatial regression models using the empirical specification described earlier. Table 2 presents the results of all OLS and weighted OLS regression models. Because all variables in the model are standardised to mean zero and unit variance, the estimated model coefficients are standardised coefficients and can be interpreted readily. We should note that we calculated variance inflation factors for all of our independent variables in our OLS model to examine possible mulitcollinearity in our predictors and we found no evidence of interdependence within our predictors.

The OLS model indicates that mortality rates increase as the percentage of female-headed households in the county and county income inequality increases. While, as the median value of homes in a county and the percentage of the population that is Hispanic or black increase, the county mortality rate tends to decrease. When we control for the variability in the population size using the weighted least squares model, we see a mortality advantage for counties with a high percentage of the population that is rural, that have a higher percentage of the population that is black, and for counties that have higher-than-average median home values. We observe a mortality disadvantage for counties with a high percentage of female-headed households and high county unemployment rates. We also notice the effect of income inequality dropout of the model when we add the weighting variable; this is an effect of the weighting process.

To assess the autocorrelation in the model residuals, we estimate Moran's I for the residuals of the OLS and weighted least squares models following the procedure outlined in Cliff and Ord (1981), which controls for the fact that the residuals are from a linear model fit, not regular spatial data. The resulting value of Moran's I for the OLS model is 0.366 ($z = 34.34$, $P = <0.0001$), indicating a highly significant degree of global autocorrelation in the OLS model residuals. The value of Moran's I for the weighted least squares model is lower at 0.192 ($z = 18.09$, $P = <0.0001$) but still shows autocorrelation in the model residuals.

Based on these results, we proceed with estimating the spatial regression models. The results of these models are presented in Table 3. We compare the OLS with the spatial error and lag

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Table 2. Results of OLS and weighted OLS models for US county mortality rates, 1998–2002.

	Estimate	Standard error	Robust standard error	95% Confidence interval[1]	z-Test, Pr(>|z|)[1]
OLS model					
(Intercept)	0.000	0.015	0.014	(–0.015, 0.015)	0, 1
% Rural population *z*	–0.019	0.019	0.028	(–0.037, 0)	–0.98, 0.327
% Black population *z*	**–0.237**	0.028	0.058	(–0.265, –0.21)	–8.55, <0.0001
% Hispanic population *z*	**–0.198**	0.016	0.019	(–0.214, –0.182)	–12.341, <0.0001
% Female HH heads *z*	**0.611**	0.032	0.077	(0.579, 0.643)	18.911, <0.0001
Unemployment rate *z*	0.025	0.017	0.022	(0.008, 0.043)	1.458, 0.145
Median house value *z*	**–0.230**	0.018	0.022	(–0.248, –0.212)	–12.812, <0.0001
Population density *z*	0.008	0.017	0.030	(–0.009, 0.024)	0.454, 0.65
Gini coefficient (Income) *z*	**0.144**	0.017	0.044	(0.127, 0.161)	8.411, <0.0001
Weighted OLS model					
Intercept	–0.073	0.036	0.036	(–0.109, –0.037)	–2.013, 0.044
% Rural population *z*	**–0.180**	0.029	0.064	(–0.209, –0.152)	–6.317, <0.0001
% Black population *z*	**–0.182**	0.035	0.098	(–0.217, –0.147)	–5.227, <0.0001
% Hispanic population *z*	0.015	0.018	0.085	(–0.003, 0.032)	0.835, 0.404
% Female HH heads *z*	**0.747**	0.034	0.121	(0.713, 0.781)	21.958, <0.0001
Unemployment rate *z*	**0.074**	0.021	0.081	(0.052, 0.095)	3.453, 0.001
Median house value *z*	**–0.314**	0.027	0.078	(–0.341, –0.288)	–11.826, <0.0001
Population density *z*	0.334	0.206	0.151	(0.128, 0.539)	1.623, 0.105
Gini coefficient (income) *z*	–0.042	0.010	0.069	(–0.052, –0.032)	–4.195, <0.0001

Bold entries represent significant relationships at at least the $\alpha = 0.05$ level.
[1]The hypothesis tests are performed using the ordinary standard errors, not the robust estimates.
OLS = ordinary least squares; HH = high–high cluster; Pr = probability.

models using the standard likelihood ratio test $2*[-LL_{OLS} - (-LL_{Spatial})]$, with 1 degree of freedom because the only difference in the spatial and OLS models is the estimation of the ρ parameter in both spatial models. We compute the same likelihood ratio test for the weighted spatial regression model but we compare it only with the weighted OLS model. We also present the results of the general and Robust Lagrange multiplier statistic for the OLS model.

The spatial error model can be interpreted as controlling for the autocorrelation in the model error term. The ρ parameter is 0.635, with a standard error of 0.019, indicating significant autocorrelation in the model error terms of the OLS model. After controlling for the autocorrelation in the error term, we see overall decreases in the magnitude of the regression coefficient for the percentage of the county population that is Hispanic and observe a decrease in the importance female-headed households, median home values, and the county Gini coefficient in predicting county mortality rates. We return to these findings in our discussion. The spatial error model displays a highly significant improvement in total fit over the OLS model when the likelihood ratio test is examined (likelihood ratio is 840.47, with 1 degree of freedom, $P < 0.0001$).

The spatial lag model indicates slightly different results from the OLS or error models. Compared with the OLS model, we see that the percentage of the county population that is rural becomes significant and indicates a reduction in the mortality rate. We also observe an increase in magnitude for the effect of the percentage of the county population that is black. We see reductions in the magnitudes of all other predictor variables in the model. When we compare the spatial lag model with the OLS model using a likelihood ratio test, we see a significant improvement in fit of the spatial lag model over the OLS model (likelihood ratio is 870.35, with 1 degree of freedom, $P < 0.0001$). The spatial autoregressive parameter, ρ, indicates a significant autocorrelation in the lagged mortality rates themselves. This represents a model-based confirmation of the patterns seen in the map in Figure 1, where several clusters of high and low mortality rates exist across the US. The weighted spatial regression model shows some notable differences in coefficients compared

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Table 3. Results of spatial regression models for US county mortality rates, 1998–2002.

	Estimate	Standard error	Robust standard error	95% Confidence interval[1]	z-Test, Pr(>|z|)[1]
Spatial error model					
(Intercept)	0.002	0.033	0.033	(–0.032, 0.035)	0.048, 0.962
% Rural population z	–0.021	0.017	0.027	(–0.039, –0.004)	–1.255, 0.21
% Black population z	**–0.288**	0.033	0.065	(–0.322, –0.255)	–8.649, <0.0001
% Hispanic population z	**–0.073**	0.024	0.033	(–0.097, –0.048)	–2.981, 0.003
% Female HH heads z	**0.507**	0.031	0.078	(0.476, 0.538)	16.482, <0.0001
Unemployment rate z	0.034	0.018	0.027	(0.016, 0.052)	1.862, 0.063
Median house value z	**–0.232**	0.020	0.031	(–0.252, –0.212)	–11.404, <0.0001
Population density z	0.037	0.020	0.038	(0.017, 0.056)	1.884, 0.06
Gini coefficient (income) z	**0.097**	0.016	0.053	(0.081, 0.113)	6.207, <0.0001
ρ	**0.635**				
LR test vs. OLS	840.47, 1 df				
Spatial lag model					
(Intercept)	–0.003	0.012	0.012	(–0.016, 0.009)	–0.275, 0.784
% Rural population z	**–0.036**	0.016	0.025	(–0.052, –0.02)	–2.278, 0.023
% Black population z	**–0.259**	0.023	0.048	(–0.282, –0.236)	–11.108, <0.0001
% Hispanic population z	**–0.104**	0.014	0.018	(–0.118, –0.091)	–7.644, <0.0001
% Female HH heads z	**0.474**	0.028	0.066	(0.447, 0.502)	17.155, <0.0001
Unemployment rate z	0.009	0.015	0.019	(–0.005, 0.024)	0.636, 0.525
Median house value z	**–0.181**	0.015	0.017	(–0.197, –0.166)	–11.9, <0.0001
Population density z	0.022	0.014	0.017	(0.008, 0.036)	1.548, 0.122
Gini coefficient (income) z	**0.073**	0.014	0.047	(0.058, 0.087)	5.01, <0.0001
ρ	**0.555**				
LR test vs. OLS	870.35, 1 df				
Weighted spatial model[2]					
(Intercept)	**–0.133**	0.052	0.052	(–0.185, –0.081)	–2.555, 0.011
% Rural population z	**–0.109**	0.027	0.031	(–0.136, –0.082)	–4.024, <0.0001
% Black population z	**–0.205**	0.042	0.053	(–0.247, –0.163)	–4.885, <0.0001
% Hispanic population z	**0.084**	0.025	0.026	(0.058, 0.109)	3.304, 0.001
% Female HH heads z	**0.736**	0.035	0.076	(0.701, 0.771)	21.274, <0.0001
Unemployment rate z	0.036	0.023	0.025	(0.013, 0.059)	1.589, 0.112
Median house value z	**–0.239**	0.032	0.022	(–0.271, –0.207)	–7.436, <0.0001
Population density z	–0.088	0.240	0.076	(–0.327, 0.152)	–0.366, 0.715
Gini coefficient (income) z	**–0.049**	0.010	0.051	(–0.059, –0.039)	–4.95, <0.0001
ρ	**0.408**				
LR test vs. weighted OLS	269.71, 1 df				

Bold entries represent significant relationships at at least the $\alpha = 0.05$ level.
[1]The hypothesis tests are performed by using the ordinary standard errors, not the robust estimates.
[2]The robust standard errors reported for the weighted spatial regression model have been corrected for heteroskedasticity by using the method of White (1980), not the sandwich estimator as in the other models.
LR = likelihood ratio test; df = degrees of freedom; OLS = ordinary least squares; HH = high–high cluster; Pr = probability.

with the unweighted spatial regression models. We see a slight mortality disadvantage for counties with high percentages of the population that are Hispanic and we see a mortality advantage for counties with higher income inequality. This second finding seems at first to be erroneous, but, when we consider that the counties with the highest weight in this analysis were those with smaller population sizes, this could make sense, as these places often have marked inequality between residents. We see an autoregression coefficient of 0.408, lower than that of either the error or lag model. In total, we see that, by including a weighting factor for the population size of counties, we reduce some but not all of the autocorrelation in our data. We also see a significant

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improvement of the weighted spatial regression model over the weighted OLS model (likelihood ratio test (LR) = 269.71, $P = <0.0001$).

Although we cannot perform the likelihood ratio test between the spatial error and lag models (df = 0), the Akaike Information Criteria (AIC) values of the two models indicate a slightly better model fit for the spatial lag model (AIC = 6552.16) than the error model (AIC = 6582.05). This suggests that the spatial process generating the data may operate more on the dependent variable than the error component of the data. While not comparable with the other spatial regression models, the weighted model has an AIC of 11,751.18, compared to the AIC from the weighted OLS model of 12,018.89, indicating an improvement in fit over the latter. The basic Lagrange multiplier statistics (Anselin *et al*., 1996) for the spatial lag model indicates that the spatial lag model is a slightly better model specification in this case than the error model [Lagrange multiplier (error) 1153.87, $P = <0.0001$; Lagrange multiplier (lag) 1198.78, $P = <0.0001$]. This is confirmed by the robust Lagrange multiplier statistics, which show a larger degree of differentiation between the models [Robust Lagrange multiplier (error) 70.85, $P = <0.0001$; Robust Lagrange multiplier (lag) 115.75, $P = <0.0001$]. This suggests that the spatial lag model may provide a better representation of the process in this setting. However, because of the very small difference between these values, we argue that conceptual considerations about the spatial process inherent to the outcome have to be considered when selecting the most appropriate spatial model for analysis. Based on the very small difference between these diagnostic statistics, we still think a spatial error model captures the spatial dependence in county mortality rates, largely based on the spatial clustering of the predictor variables noted in Figure 2. Lastly, we calculated the Breusch–Pagan test for the presence of heteroskedasticity in both the spatial error and lag models. Both models displayed significant heteroskedasticity in their residuals (Spatial error model Breusch–Pagan test = 307.98, $P = < 0.0001$; Spatial lag model Breusch–Pagan = 334.052, $P = < 0.0001$). To assess the impact of this on the hypothesis tests of the parameter values in Table 1, we calculated robust sandwich estimates of the regression parameter standard errors. For the OLS model, the significance of all relationships is confirmed when robust estimates are used. In the weighted model, the effects of population density and the unemployment rate become insignificant when robust standard errors are used. In the spatial error model, the Gini coefficient becomes insignificant when robust errors are used, and, in the spatial lag model, both the percentage of the county population that is rural and the Gini coefficient become insignificant when robust standard errors are used. When we use heteroskedasticity-corrected (White, 1980) standard errors for the weighted spatial regression model, we see the only parameter affected is the effect of the Gini coefficient, which becomes insignificant. When we consider these effects, we see the significant relationships become more consistent across model fits in our analysis.

DISCUSSION

Based on the analyses presented above, we conclude that there is significant spatial patterning in US county mortality rates, which we expected from previous work. There is also evidence of local spatial autocorrelation in all of our county-level social and economic independent variables. Significant local spatial clustering in each of these variables highlights the need for empirical research in the social sciences more broadly and in demography particularly that considers the relationships present within and between these spatial clusters. To date, most spatial analyses in demography examining health outcomes have remained fairly exploratory and descriptive. This could be a result of the lack of literature speaking to the spatial processes inherent to specific health outcomes or application of spatial statistical methods to inappropriate levels of geography leading to confusion about the existence or not of spatial patterns.

We argued that the econometric literature gives us a solid definition of the potential sources of spatial heterogeneity, which can be a result of errors associated with unmeasured variables in empirical model specifications or spatial processes that say neighbours have similar values of variables (Baller *et al*., 2001). However, a critical component of the definition of spatial heterogeneity speaks broadly to the variation in measures across space (Anselin, 1995). Therefore, many studies of spatial processes in health may mask the true underling spatial nature of a specific

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outcome by relying on more global measures of the spatial process.

Basing on our understanding of the spatial pattern in US county mortality rates and ecological correlates with these rates, we hypothesised that a spatial error model would best fit the data underlying the presence of spatial heterogeneity. While the standard diagnostic criteria used in most spatial data analyses indicated that a spatial lag model offered a better fit to our data, the very small absolute difference in the Robust Lagrange multiplier statistic between the lag and error models makes us question the reliance on this value in selecting the best spatial model for interpretation. We still believe that the spatial error model best speaks to the spatial pattern observed in US county mortality rates because it is unlikely that a county's total morality rate works in a diffusive manner to increase or decrease the mortality rate in a neighbouring county. Instead, we think that the spatial pattern noted in this analysis is largely a result of the existence of autocorrelation among omitted variables in our empirical model and the local variation in resources across the US that cannot be captured in this type of model specification. It would seem most appropriate to use a spatial lag model to examine health outcomes that have a true diffusive nature, such as the incidence of an infectious disease, or rely on a set of established network relationships among areas that would lead to spatial clusters in those outcomes. Likewise, a spatial lag model would probably best approximate a very local spatial process, while a spatial error model may best estimate spatial patterns in health outcomes that are found in much larger spatial units. When we consider the weighted spatially autoregressive model, we see that we further reduce the amount of autocorrelation in the data by allowing for the effect of heterogeneous population sizes, which itself has a significant amount of autocorrelation (Moran's $I = 0.360$, $P < 0.0001$).

We suggest that by using models that control for spatial variation and spatial structure in our empirical observations, particularly with topics that have an inherent spatial component, we can begin to separate the underlying spatial processes from the noise inherent in the data. However, we think research using spatial statistical techniques needs to focus on local spatial clusters in outcomes because the true mechanism underlying most spatial clusters in health outcomes varies greatly across space. Demographic analyses interested in spatial dynamics need to reach across diverse fields of study to better understand the application and interpretation of spatial statistical methods to local spatial patterns. The theoretical literature related to spatial processes in health or other outcomes is not likely to develop unless more local applications of spatial processes are considered more fully and developed at the appropriate geographical scale. We feel that it is the underlying processes inherent to spatial analysis that needs further development, especially when aggregate-level population processes are being considered. More focused, conceptually grounded applications of local spatial statistical methods may help in making decisions about the appropriate use of spatial regression models on aggregate data, or more specialised local spatial statistical models that consider much smaller areas may be warranted for certain outcomes. Beyond the study of county mortality rates, the examination of local spatial clusters will be valuable in future research considering the clustering of poverty, health-care access, and socio-economic inequality, given that these phenomena are unevenly distributed across areas of the US and in other settings and show aggregate associations with health differentials.

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