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The Impact of State Health Policy on Sub-national Mortality in the United States, 1990–2018

by

Denys Dukhovnov

A dissertation submitted in partial satisfaction of the

requirements for the degree of

Doctor of Philosophy

in

Demography

in the

Graduate Division

of the

University of California, Berkeley

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Professor Joshua R. Goldstein, Chair

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Abstract

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This dissertation embodies a series of four interrelated chapters, collectively aiming to uncover the role of state health policy (SHP) in population mortality outcomes across the US states and counties over the course of nearly 3 decades, from 1990 to 2018. Chapter 1 introduces necessary background literature on determinants of mortality, its causes and disparities across various sub-populations by socio-economic status, age, sex, place of residence, generational belonging, and health. Based on this evidence, in chapter 2 I propose a conceptual model with a particular focus on isolating assessed causal links between the key indicators, such as population age, health status, and state government ideology. I argue that these three concepts represent a minimal adjustment set to isolate the causal link between SHP and mortality. Additional variables broadly defining area-level socio-economic deprivation, income inequality, residential segregation are considered part of the conceptual model's expanded set that, in principle, ought to augment the precision of the target estimate in a quantitative analysis.

Chapter 3 follows with a detailed description of the method to generate health positivity index scores as well as an alternative, the rate of SHP innovation measure, from a selection of 110 groups of health-related policies, passed into law by states since 1970 through 2018. The main I score index of SHP health positivity takes advantage of the policies' cross-sectional and temporal cumulativity, its thematic relativity with respect to the global maximum in any year, and a double-weighting design to stem over-representation bias. I consider raw and transformed variations of the I score, its decade-lagged variant, and the indicator of the relative rate of new health policy passage. Preliminary testing using one- and two-way repeated measures ANOVA analyses demonstrates medium-to-large effect sizes for the associations between mortality and I score measures at the state level, with notable attenuation in the county group sample, whereas no such association exists for the alternative rate-of-health-policy-innovation indicator. Temporal dependence of the SHP-mortality association is established.

The aim of chapter 4 is to use spatial econometric modeling techniques to arrive at the best possible estimate of the state health policy effect on mortality, as measured by age-standardized death rate, and life expectancy estimates at birth and at age 65. Using mortality data from the United States Mortality Database (USMDB) chapter 4 thus builds

upon the previous chapters as follows. First, a set of quantitative indicators representing key concepts presented in chapters 1 and 2 are introduced from secondary sources or are estimated directly on the basis of publicly available secondary data from the US Census Bureau and National Center for Health Statistics. Next, I provide an overview to popular spatial econometric methodology approaches using spatial panel fixed effects designs and submit these variable sets to modeling 3 sex-specific mortality outcomes in various specification combinations, each of which includes one of the measures of state health policy derived in chapter 3. I use evidence from the model diagnostics to arrive at the most optimal model type for each combination of outcome-SHP predictor. My findings indicate that state health policy represented by the two I score measures is the most effective in predicting state-level outcomes. It is estimated that conditional on the socio-economic, political, health and demographic factors SHP could explain up to 30% of the longevity gap at birth and at age 65 for states on the extreme ends of the longevity spectrum. On the other hand, county group mortality is susceptible to the effects of the parent state health policy only to a limited degree, and is associated more with local socio-economic context. Approximately two-thirds of the SHP-mortality effect is direct and one-third is a by-product of spatial spillovers from the neighboring geography, whereas the reverse is true for the county groups.

The findings of this analysis produce several important contributions. First, the analysis highlights the utility of studying summary indicators of health policy at sub-national aggregate levels as these encompass valuable information that characterizes heterogeneous risk factors that impact the aggregate. Second, the effect of the policy action is inherently spatial, but depends greatly on the scale of aggregation. Longevity in smaller population sub-groups is more sensitive to its local and regional socio-economic and physical environments, with state policy effects percolating more slowly and in a reduced form. Lastly, I find that generalized synthetic measures of state health policy, as applied in the final part of the analysis, are best used in conjunction with summary measures of mortality at coarser geographic resolutions, such as states and regions. Future analyses involving county-level or finer resolution data would benefit from an application of thematic summary components of such measures.

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On the day I landed in Berkeley I neither had a clear understanding of demography, nor how transformative it would become to my view of all forms of human activity. I feel immense urge to give credit to the Berkeley Demography magicians, Robert Chung, Carl Mason and Carl Boe for instilling in me an out-of-the-box thinking, for enthusiastically and with a great deal of curiosity providing feedback on many of my project ideas and paper analyses with which I confronted them with increasing frequency. I thank Ken Wachter and Ron Lee for allowing me to develop my potential, for following me through the basic training at Berkeley Demography, and for offering valuable advice on navigating the field.

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— Denys Dukhovnov

Contents

Introduction **xiii**
Four decades of sub-national mortality in the United States . . . xiv
Health policy as a determinant of health and mortality . . . xv
Demographic data and estimation approaches . . . xvi
Structure of the thesis . . . xviii

1 Four Decades of Mortality in the United States, 1980–2018: Review of the Past Literature **1**
1.1 Geographic mortality trends . . . 2
1.2 Health and mortality in a spatial context . . . 6
1.3 Political, environmental, socio-economic and demographic components of area-level mortality . . . 8
1.3.1 Sources of mortality inequalities and resource deprivation . . . 11
1.4 Summary . . . 17

2 Uncovering Traces of State Politics in Death: A Conceptual Model **19**
2.1 A brief overview of causal inference using structural causal models . . . 22
2.2 Structural causal model: state health policy impact on sub-national mortality 26
2.3 Structural Causal Model assumptions and limitations . . . 32
2.4 Summary . . . 35

3 A Method for Generating State Health Policy Scores and Rankings **37**
3.1 Policy data . . . 38
3.2 Combining SPPD, SPID and CDC policy datasets . . . 38
3.3 State health policy score index . . . 41
3.4 Relative measure of the rate of health policy adoption . . . 43
3.5 Comparing various forms of SHP indicators and their associations with mortality 46
3.6 Summary . . . 49

4 Sub-national Analysis of the Impact of State Health Policy on Mortality **53**
4.1 USMDB data as input for sub-national mortality analysis . . . 55
4.2 Independent variables . . . 57
4.2.1 State health policy and state government ideology . . . 57
4.2.2 Age and level of population health . . . 58
4.2.3 SES measures: income, income inequality, education, occupation . . . 64
4.2.4 Demographic area composition and segregation . . . 64

4.2.5 Urban environment 66
4.3 Modeling strategy 67
4.3.1 Descriptive method 67
4.3.2 Model specification 68
4.3.3 Spatial models 69
4.3.4 Variable transformations and selection 73
4.4 Results 75
4.5 Discussion 107
4.6 Conclusion 116

Conclusion **119**

Bibliography **127**

Appendices **141**
A.1 Appendix to Chapter 3 142
A.2 Appendix to Chapter 4 151

List of Figures

1.1 Trends in national and state-level life expectancy (1980–2018), by select age groups. . . . 4
1.2 Trends in county-level age-adjusted mortality 1980–2014, by county metropolitan status, for major causes of death . . . 5
1.3 Trends in county-level age-adjusted mortality rate 1980–2014, by state and major cause-of-death contributor. For each state, 2 counties are shown, one that underwent most change and another that changed the least in the period of 1980–2014. Shading corresponds to the cause of death that contributed the greatest absolute increase or decrease in deaths between two consecutive years. . . . 7
1.4 Trends in county-level age-adjusted mortality 1980-2014, by sex, for major causes of death . . . 10

2.1 Illustrative examples of graphical representations of structural causal models (SCM) using directed acyclic graphs (DAG) . . . 24
2.2 Directed arrow graph depicting expected causal relationship between state health policy and sub-national mortality. . . . 28
2.3 Reduced directed arrow graph depicting expected causal diagram between state health policy and sub-national mortality, conditional on the minimal adjustment set (variables shaded in grey). . . . 29
2.4 Expanded and adjusted directed arrow graph depicting expected causal diagram between state health policy and sub-national mortality. . . . 31
2.5 Instrumental variable analysis using directed arrow graph, estimating expected causal effect of state health policy on sub-national mortality. . . . 33

3.1 State health policy positivity score 1970–2019. . . . 42
3.2 State health policy positivity rank for select years 1990–2018, sorted in order of decreasing rank in 2018. . . . 44
3.3 Geographic distribution of the state health policy positivity rank for select years 1990–2018. . . . 45
3.4 Frequency distribution of health policies passed in each decade leading up to the observation of mortality data panel. . . . 46

4.1 Detroit, MI. Disaggregation of county-level life expectancy at birth by zip-codes and census tracts unveils important spatial patterns given rise to by the local phenomena. . . . 54

4.2 Age-standardized mortality rate trends for the US counties, by year, sex, and state, 1990–2018. 56
4.3 Example using Alabama state forecasts to estimate poor/fair self-rated health prevalence, by sex. 62
4.4 Distribution of spatial neighbors under the queen(1) weighting configuration in the (a) state and (b) county group samples. 71
4.5 Univariate Local Indicators of Spatial Autocorrelation (LISA) cluster plot for state indicators of mortality, by year and sex. 82
4.6 Univariate Local Indicators of Spatial Autocorrelation (LISA) cluster plot for county group indicators of mortality, by year and sex. 83
4.7 State level bivariate Moran statistic for pairwise combinations of 3 mortality indicators and various independent variables, by year (both sexes combined). 86
4.8 County group level bivariate Moran statistic for pairwise combinations of 3 mortality indicators and various independent variables, by year (both sexes combined). 87
4.9 Comparison of state and county group fixed effects panel model fits for the three mortality indicators and three candidates for state health policy, by sex. 102
4.10 Spatial distribution of mean absolute percent error (MAPE) in fully adjusted spatial Durbin models (SDM) for the logarithm of the age-standardized mortality rate as the outcome and I score as the main state health policy predictor, by sex. 103
4.11 Spatial distribution of mean absolute percent error (MAPE) in fully adjusted spatial Durbin error models (SDEM) for the logarithm of the age-standardized mortality rate as the outcome and I score as the main state health policy predictor, by sex. 104
A.1.1 State-level correlation summary and plots for various measures of mortality, by year (1990–2018). 145
A.1.2 County group-level correlation summary and plots for various measures of mortality, by year (1990–2018). 146
A.1.3 State-level correlation summary and plots for select measures of mortality and indicators of state health policy, by year (1990–2018). 147
A.1.4 State-level correlation summary and plots for select measures of mortality and thematic components of the state health policy score (I_g), by year (1990–2018). 148
A.1.5 County group-level correlation summary and plots for select measures of mortality and indicators of state health policy, by year (1990–2018). . . . 149
A.1.6 State-level Q-Q plots for log-transformed age-standardized mortality rate, by year and sex. 150
A.1.7 County group level Q-Q plots for log-transformed age-standardized mortality rate, by year and sex. 150
A.2.8 Pearson correlation matrix and association box plots for select indicators in state data set, by year. 152
A.2.9 Pearson correlation matrix and association box plots for select indicators in county group data set, by year. 153

A.2.10 State bivariate Moran statistic for pairwise combinations of 3 mortality indicators and various independent variables, by year (females). 154
A.2.11 County group bivariate Moran statistic for pairwise combinations of 3 mortality indicators and various independent variables, by year (females). . . 155
A.2.12 State bivariate Moran statistic for pairwise combinations of 3 mortality indicators and various independent variables, by year (males). 156
A.2.13 County group bivariate Moran statistic for pairwise combinations of 3 mortality indicators and various independent variables, by year (males). . . . 157
A.2.14 State bivariate Moran plots for pairwise combinations of both-sex age-standardized mortality rate (ASDR) and various independent variables, by year. 158
A.2.15 State bivariate Moran plots for pairwise combinations of both-sex life expectancy at birth ($e(0)$) and various independent variables, by year. . . . 159
A.2.16 State bivariate Moran plots for pairwise combinations of both-sex life expectancy at age ($e(65)$) and various independent variables, by year. 160
A.2.17 County group bivariate Moran plots for pairwise combinations of both-sex age-standardized mortality rate (ASDR) and various independent variables, by year. 161
A.2.18 County group bivariate Moran plots for pairwise combinations of both-sex life expectancy at birth ($e(0)$) and various independent variables, by year. 162
A.2.19 County group bivariate Moran plots for pairwise combinations of both-sex life expectancy at age 65 ($e(65)$) and various independent variables, by year. 163
A.2.20 Spatial distribution of mean absolute percent error (MAPE) in fully adjusted spatial Durbin models (SDM) for the logarithm of the age-standardized mortality rate as the outcome and the 10-year lagged form of I score as the main state health policy predictor, by sex. 195
A.2.21 Spatial distribution of mean absolute percent error (MAPE) in fully adjusted spatial Durbin error models (SDEM) for the logarithm of the age-standardized mortality rate as the outcome and the 10-year lagged for of I score as the main state health policy predictor, by sex. 196
A.2.22 Spatial distribution of mean absolute percent error (MAPE) in fully adjusted spatial Durbin models (SDM) for the logarithm of the age-standardized mortality rate as the outcome and the relative fraction of new policies passed in prior decade as the main state health policy predictor, by sex. . 197
A.2.23 Spatial distribution of mean absolute percent error (MAPE) in fully adjusted spatial Durbin error models (SDEM) for the logarithm of the age-standardized mortality rate as the outcome and the relative fraction of new policies passed in prior decade as the main state health policy predictor, by sex. 198
A.2.24 Spatial distribution of mean absolute percent error (MAPE) in fully adjusted spatial Durbin models (SDM) for the life expectancy at birth as the outcome and I score as the main state health policy predictor, by sex. . . 199
A.2.25 Spatial distribution of mean absolute percent error (MAPE) in fully adjusted spatial Durbin error models (SDEM) for the life expectancy at birth as the outcome and I score as the main state health policy predictor, by sex. 200

A.2.26 Spatial distribution of mean absolute percent error (MAPE) in fully adjusted spatial Durbin models (SDM) for the life expectancy at birth as the outcome and the 10-year lagged form of I score as the main state health policy predictor, by sex. 201

A.2.27 Spatial distribution of mean absolute percent error (MAPE) in fully adjusted spatial Durbin error models (SDEM) for the life expectancy at birth as the outcome and the 10-year lagged form of I score as the main state health policy predictor, by sex. 202

A.2.28 Spatial distribution of mean absolute percent error (MAPE) in fully adjusted spatial Durbin models (SDM) for the life expectancy at birth as the outcome and the relative fraction of new policies passed in prior decade as the main state health policy predictor, by sex. 203

A.2.29 Spatial distribution of mean absolute percent error (MAPE) in fully adjusted spatial Durbin error models (SDEM) for the life expectancy at birth as the outcome and the relative fraction of new policies passed in prior decade as the main state health policy predictor, by sex. 204

A.2.30 Spatial distribution of mean absolute percent error (MAPE) in fully adjusted spatial Durbin models (SDM) for the life expectancy at age 65 as the outcome and I score as the main state health policy predictor, by sex. 205

A.2.31 Spatial distribution of mean absolute percent error (MAPE) in fully adjusted spatial Durbin error models (SDEM) for the life expectancy at age 65 as the outcome and I score as the main state health policy predictor, by sex. 206

A.2.32 Spatial distribution of mean absolute percent error (MAPE) in fully adjusted spatial Durbin models (SDM) for the life expectancy at age 65 as the outcome and the 10-year lagged form of I score as the main state health policy predictor, by sex. 207

A.2.33 Spatial distribution of mean absolute percent error (MAPE) in fully adjusted spatial Durbin error models (SDEM) for the life expectancy at age 65 as the outcome and the 10-year lagged form of I score as the main state health policy predictor, by sex. 208

A.2.34 Spatial distribution of mean absolute percent error (MAPE) in fully adjusted spatial Durbin models (SDM) for the life expectancy at age 65 as the outcome and the relative fraction of new policies passed in prior decade as the main state health policy predictor, by sex. 209

A.2.35 Spatial distribution of mean absolute percent error (MAPE) in fully adjusted spatial Durbin error models (SDEM) for the life expectancy at age 65 as the outcome and the relative fraction of new policies passed in prior decade as the main state health policy predictor, by sex. 210

List of Tables

3.1 Description of thematic categories and classes of state policies proximal to causes of mortality . . . 40

3.2 Post-hoc pairwise t test comparisons and repeated measures one-way ANOVA analysis of logarithm of both-sex mortality rate, for states and county groups. 49

3.3 Results of the state and county group two-way ANOVA for the logarithm of mortality rate, by sex, and state health policy measures: policy impact (I) score and the relative fraction of new policies passed in previous decade ($RFNPPPD$). . . . 50

4.1 Validation measures of the 2013–2018 both-sex forecast of mean prevalence of poor/fair health for states against the published CDC age-adjusted state-level estimates. . . . 63

4.2 Best fit time series model selection proportions by forecast direction and by sex. . . . 63

4.3 Population-weighted descriptive summary statistics for the provided and calculated variables in the state ($N = 50$) and county group ($N = 2472$) data sets. . . . 77

4.3 Population-weighted descriptive summary statistics for the provided and calculated variables in the state ($N = 50$) and county group ($N = 2472$) data sets. . . . 78

4.4 Univariate Moran I statistic estimates for presence of spatial autocorrelation for 3 mortality indicators. . . . 80

4.5 Spatial panel Lagrange Multiplier tests for type of global spatial dependence. 88

4.6 Results of likelihood ratio tests between minimally adjusted and fully specified model pairs for the logarithm of the age-standardized death rate and 3 state health policy indicators as regressors. . . . 89

4.7 Fully specified state-level non-spatial fixed effects models estimating both-sex mortality indicators ($log(ASDR)$, $e(0)$, and $e(65)$) featuring various specifications of state health policy. . . . 91

4.8 Fully specified county group-level non-spatial fixed effects models estimating both-sex mortality indicators ($log(ASDR)$, $e(0)$, and $e(65)$) featuring various specifications of state health policy. . . . 91

4.9 Fully specified state-level SDM models estimating both-sex mortality indicators ($log(ASDR)$, $e(0)$, and $e(65)$) featuring various specifications of state health policy. . . . 94

4.10 Fully specified state-level SDEM models estimating both-sex mortality indicators ($log(ASDR)$, $e(0)$, and $e(65)$) featuring various specifications of state health policy. . . . 95
4.11 Fully specified county group-level SDM models estimating both-sex mortality indicators ($log(ASDR)$, $e(0)$, and $e(65)$) featuring various specifications of state health policy. . . . 96
4.12 Fully specified county group-level SDEM models estimating both-sex mortality indicators ($log(ASDR)$, $e(0)$, and $e(65)$) featuring various specifications of state health policy. . . . 97
4.13 Post-regression residual Moran estimates for various models for the 3 mortality outcome indicators and I score as the state health policy indicator, by year and sex. . . . 101
4.14 Spatial Durbin model (SDM) state-level impact decomposition for the natural logarithm of age-standardized death rate, by sex and SHP indicator. . 108
4.15 Spatial Durbin model (SDM) county-group level impact decomposition for the natural logarithm of age-standardized death rate, by sex and SHP indicator. . . . 109
A.1.1 Description of policies used in the computation of the state health policy measures. . . . 142
A.2.2 Fully specified state-level fixed effects models estimating female mortality indicators ($log(ASDR)$, $e(0)$, and $e(65)$) featuring various specifications of state health policy. . . . 164
A.2.3 Fully specified state-level fixed effects models estimating male mortality indicators ($log(ASDR)$, $e(0)$, and $e(65)$) featuring various specifications of state health policy. . . . 165
A.2.4 Fully specified county group-level fixed effects models estimating female mortality indicators ($log(ASDR)$, $e(0)$, and $e(65)$) featuring various specifications of state health policy. . . . 166
A.2.5 Fully specified county group-level fixed effects models estimating male mortality indicators ($log(ASDR)$, $e(0)$, and $e(65)$) featuring various specifications of state health policy. . . . 167
A.2.6 Fully specified state-level fixed effects SAR models estimating both-sex mortality indicators ($log(ASDR)$, $e(0)$, and $e(65)$) featuring various specifications of state health policy. . . . 168
A.2.7 Fully specified state-level fixed effects SAR models estimating female mortality indicators ($log(ASDR)$, $e(0)$, and $e(65)$) featuring various specifications of state health policy. . . . 169
A.2.8 Fully specified state-level fixed effects SAR models estimating male mortality indicators ($log(ASDR)$, $e(0)$, and $e(65)$) featuring various specifications of state health policy. . . . 170
A.2.9 Fully specified state-level fixed effects SEM models estimating both-sex mortality indicators ($log(ASDR)$, $e(0)$, and $e(65)$) featuring various specifications of state health policy. . . . 171

A.2.10 Fully specified state-level fixed effects SEM models estimating female mortality indicators ($log(ASDR)$, $e(0)$, and $e(65)$) featuring various specifications of state health policy. 172
A.2.11 Fully specified state-level fixed effects SEM models estimating male mortality indicators ($log(ASDR)$, $e(0)$, and $e(65)$) featuring various specifications of state health policy. 173
A.2.12 Fully specified state-level fixed effects SLX models estimating both-sex mortality indicators ($log(ASDR)$, $e(0)$, and $e(65)$) featuring various specifications of state health policy. 174
A.2.13 Fully specified state-level fixed effects SLX models estimating female mortality indicators ($log(ASDR)$, $e(0)$, and $e(65)$) featuring various specifications of state health policy. 175
A.2.14 Fully specified state-level fixed effects SLX models estimating male mortality indicators ($log(ASDR)$, $e(0)$, and $e(65)$) featuring various specifications of state health policy. 176
A.2.15 Fully specified state-level fixed effects SDM models estimating female mortality indicators ($log(ASDR)$, $e(0)$, and $e(65)$) featuring various specifications of state health policy. 177
A.2.16 Fully specified state-level fixed effects SDM models estimating male mortality indicators ($log(ASDR)$, $e(0)$, and $e(65)$) featuring various specifications of state health policy. 178
A.2.17 Fully specified state-level fixed effects SDEM models estimating female mortality indicators ($log(ASDR)$, $e(0)$, and $e(65)$) featuring various specifications of state health policy. 179
A.2.18 Fully specified state-level fixed effects SDEM models estimating male mortality indicators ($log(ASDR)$, $e(0)$, and $e(65)$) featuring various specifications of state health policy. 180
A.2.19 Fully specified county group-level fixed effects SAR models estimating both-sex mortality indicators ($log(ASDR)$, $e(0)$, and $e(65)$) featuring various specifications of state health policy. 181
A.2.20 Fully specified county group-level fixed effects SAR models estimating female mortality indicators ($log(ASDR)$, $e(0)$, and $e(65)$) featuring various specifications of state health policy. 181
A.2.21 Fully specified county group-level fixed effects SAR models estimating male mortality indicators ($log(ASDR)$, $e(0)$, and $e(65)$) featuring various specifications of state health policy. 182
A.2.22 Fully specified county group-level fixed effects SEM models estimating both-sex mortality indicators ($log(ASDR)$, $e(0)$, and $e(65)$) featuring various specifications of state health policy. 183
A.2.23 Fully specified county group-level fixed effects SEM models estimating female mortality indicators ($log(ASDR)$, $e(0)$, and $e(65)$) featuring various specifications of state health policy. 184
A.2.24 Fully specified county group-level fixed effects SEM models estimating male mortality indicators ($log(ASDR)$, $e(0)$, and $e(65)$) featuring various specifications of state health policy. 185

A.2.25 Fully specified county group-level fixed effects SLX models estimating both-sex mortality indicators ($log(ASDR)$, $e(0)$, and $e(65)$) featuring various specifications of state health policy. 186
A.2.26 Fully specified county group-level fixed effects SLX models estimating female mortality indicators ($log(ASDR)$, $e(0)$, and $e(65)$) featuring various specifications of state health policy. 187
A.2.27 Fully specified county group-level fixed effects SLX models estimating male mortality indicators ($log(ASDR)$, $e(0)$, and $e(65)$) featuring various specifications of state health policy. 188
A.2.28 Fully specified county group-level fixed effects SDM models estimating female mortality indicators ($log(ASDR)$, $e(0)$, and $e(65)$) featuring various specifications of state health policy. 189
A.2.29 Fully specified county group-level fixed effects SDM models estimating male mortality indicators ($log(ASDR)$, $e(0)$, and $e(65)$) featuring various specifications of state health policy. 190
A.2.30 Fully specified county group-level fixed effects SDEM models estimating female mortality indicators ($log(ASDR)$, $e(0)$, and $e(65)$) featuring various specifications of state health policy. 191
A.2.31 Fully specified county group-level fixed effects SDEM models estimating male mortality indicators ($log(ASDR)$, $e(0)$, and $e(65)$) featuring various specifications of state health policy. 192
A.2.32 Post-regression residual Moran estimates for various models for the 3 mortality outcome indicators and 10-year lagged I score as the state health policy indicator, by year and sex. 193
A.2.33 Post-regression residual Moran estimates for various models for the 3 mortality outcome indicators and the relative fraction of policies passed in prior decade as the state health policy indicator, by year and sex. 194
A.2.34 Spatial Durbin model (SDM) state-level impact decomposition for life expectancy at birth, by sex and SHP indicator. 211
A.2.35 Spatial Durbin model (SDM) county-group level impact decomposition for for life expectancy at birth, by sex and SHP indicator. 212
A.2.36 Spatial Durbin model (SDM) state-level impact decomposition for the life expectancy at age 65, by sex and SHP indicator. 213
A.2.37 Spatial Durbin model (SDM) county-group level impact decomposition for the life expectancy at age 65, by sex and SHP indicator. 214

Introduction

The forces of demographic change are ubiquitous, but its existence is not closed off to outside influences. In contrast to the mathematical representation embodied by the balancing and renewal equations and the Stable Population Theory to produce estimates of fertility, mortality and migration, the root causes of these processes are abundant and diverse. A seemingly chaotic fabric of activity forming collective patterns of human behavior revolves and interacts with these three cornerstones of demography. Systems of beliefs, societal norms, physical and material needs all have a tremendous impact on how many children people have, how healthy and long are people's lives, and whether or not they move to become part of a different community or society at some point during their lives. Yet, relative to reproduction and migration, mortality is perhaps the one demographic outcome over which individuals have the least immediate agency. Since the ancient times the latter has been embraced as a feared but an inevitable outcome. Many have leaped onto a promise of mortality delay through the use of herbal medicine, nutrition, physical activity, or even rituals. In the modern era these measures have been largely replaced with science-based medicine, technology, understanding and practicing various forms of a healthy lifestyle. However, there is a limit to how much one person or a small group of people can achieve in attaining their ideal routine and in acquiring the necessary resources to that end. This is where societal mechanisms of institutional action come into play.

One purpose of a modern, democratically elected government is to provide various types of resources and its distribution for large scale measures targeting common population health concerns, beyond what single individuals can attain on their own. As such, political representation and its consequence in the form of health regulation are crucial for a given population's good health and, by extension, longevity. In the United States it is the responsibility of state governments to issue legislation aimed to promote better health outcomes and to reduce mortality. A systematic examination of state health policy effects on sub-national mortality across the entire country thus fits well with the agenda set forth by the recent research work on the patterns of sub-national and geographic disparities in mortality. Nonetheless, individual health policies, though targeted to remedy a particular health problem in a particular place, often create knock on effects that could spread spatially to different areas, trigger stimuli for additional intervention, and interact with other policy impacts to yield a different effect, distinct from the intended one for each individual policy. These considerations motivate a deeper examination of how state health regulation affects mortality outcomes on the state and sub-state levels. These also necessitate the change of focus away from individual pieces of health legislation toward health related policies operating in conjunction with one another over a considerable period of time and over a wider

area.

Four decades of sub-national mortality in the United States

The period of the past several decades in the United States bears the hallmarks of the final stage of the Demographic Transition. Fertility and mortality rates have both declined considerably since the beginning of the 20th century. The controversial McKeown thesis, once regarded as a mainstream working theory on the decline of mortality exclusively due to the rise in the standard of living, has become subject to scrutiny and criticism in the past decades (Colgrove, 2002). The postponement and compression of mortality toward old age was achieved in part through the evolution of diseases and their causes (Epidemiological Transition). The 2020 end-of-decade World Health Organization report outlines the top 10 causes of death for high-income countries and its evolution since 2000 (World Health Organization, 2020). Predictably, cardiovascular and Alzheimer's diseases occupy the top three spots, with the former continuing its gradual decline, whereas the latter is steeply on the rise since the turn of the 21st century. At the same time, the US has been lagging in this progress behind its high-income peer countries in terms of the high and increasing age variability in mortality since 1980, featuring higher peaks in the distribution of deaths during infancy and in mid-life (Rogers et al., 2020). The longstanding pattern of longevity disadvantage in the US, as compared to other developed nations, is contributed by the disproportionate number of deaths among the middle-aged adults, which has continued to persist through the early 2020s and well into the COVID-19 pandemic (Masters et al., 2022).

Locality based mortality differentials are also extensively documented in the existing literature (Kindig & Cheng, 2013). Murray et al. (2005) find that patterns of mortality in the US are represented by regional differentials, with at least 8 distinct cause set archetypes. Tobacco use, alcohol consumption and obesity are identified as some of the prominent distinguishing risk factors among the geographic subpopulations. More recently, in the 2010s, mortality from the so-called "diseases of despair" have been inflicting disproportionate damage to the communities across the country (Case & Deaton, 2015, 2017). Suicide, alcohol, and opioid drug abuse disproportionally targeted socio-economically disadvantaged population groups across the South and the Appalachia. Indeed, many studies find detrimental effects of a broad spectrum of deprivation aspects on mortality at fine geographic resolution, ranging from segregation to food deserts, to lack or poor infrastructure, education, to access to healthcare (Browning et al., 2011; Nandi & Kawachi, 2011; Seaman et al., 2019). The effects of income and its inequality on health and mortality outcomes are widely studied, often as part of cross-national comparisons (Lobmayer & Wilkinson, 2000; Preston, 1975). Notably, assessment of the impact of inequalities on health and mortality cannot exist independently from overarching institutional regulations that mitigate or promote such inequalities. County-level examination of racial inequalities in mortality since 2000 also demonstrates disproportionate disadvantage among the Black and American Indian and Alaska Native sub-populations (Dwyer-Lindgren et al., 2022).

It becomes evident through a rudimentary descriptive analysis that local cause-of-death anomalies and mortality differentials are often masked by national averages (Boing et al., 2020; A. S. Vaughan et al., 2017). Likewise, at a coarse resolution the underlying mechanisms

of many observed patterns of socio-demographic phenomena tend to become obscured, since it is not clear how aggregate results decompose into the respective local components. For instance, it is well understood that income and socio-economic status are key factors in a range of area-level outcomes in education, wealth accumulation, housing, health, well-being, safety, and so on. Placing the research on such mechanisms within a spatial context further affords a nuanced display of how various forms of disadvantage accumulate in the presence of environmental factors.

Health policy as a determinant of health and mortality

In the United States policy decisions related to healthcare and vital event registration are deferred to state governing bodies. In order to properly untangle the role that socio-economic factors and deprivation play in the aggregate outcomes, it is necessary to scale down the focal depth of analyses from the national level to that of states or smaller. Health and mortality on a small geographic scale, such as counties, are subject not only to socio-economic and environmental conditions of a given area, but are in large part influenced by knock-on effects from neighboring or otherwise spatially proximal counties, as well as by a parent state health policy. As a hypothetical exercise, consider passing of a tobacco tax increase legislation in a particular state with a goal of reducing prevalence of smoking related diseases. Depending on a particular circumstance such a regulation might actually not reduce the frequency of smoking among residents in that state. It could also reduce it to a lower-than-expected degree, if the residents begin to travel to the neighboring states to buy cheaper tobacco products, or if the residents' income is high enough to offset the tax increase. These dynamics would be more salient when viewing this scenario at the level of a county, especially if adjacent to the border with states without such tax regulation. In general, many analogous instances can be found throughout the nation with combinations of individual policy effects that are difficult to disentangle. Still, it is difficult to argue against the notion that the impact of local governance with regard to public policy has a tangible impact on health, well-being, and longevity of the population. Evidence shows that states with lower healthcare related spending report higher rates of morbidity and mortality (Beckfield & Bambra, 2016). The adoption of such landmark federal legislation as the Patient Protection and Affordable Care Act (ACA) (H.R.3590 - 111th Congress (2009-2010), 2010) induced differential impacts across the country due to the states' background differences and aims of the local political leadership with regard to the implementation of the ACA requirements. Regardless of the intent, state policy is one determinant that has been selectively incorporated into many studies on specific behavioral risk factors or causes of death (Haskell et al., 2009; Rodriguez et al., 2022; Slater & Carlton, 1985).

Quantitative policy research as a whole does not come without a multitude of challenges. Systematic cross-boundary analysis of state policies are notoriously difficult due to the differences in the policy wording, adoption times, target audience, method of enforcement and implementation, to name just a few obstacles. In addition, much like in every area of human activity, health policies often act in a bundle rather than independently, engendering a variety of impact attenuation and amplification responses (Montez & Grumbach, 2023). Fortunately, the ongoing work on the construction of grouped and harmonized repositories

of state policy metadata greatly aids in the effort of analyzing the effectiveness of regulation on a range of topics, including population health (Boehmke et al., 2018; Montez, 2022). It is my ambition to take this line of work further, to devise a method by which state health policies could be amalgamated into one or more area-level quantitative summary measures that could be analyzed on par with a variety of socio-economic, demographic, health, and other secondary data indicators commonly available to researchers. Putting state health policy in conversation with the aggregate contextual background is an indispensable part to uncovering the reasons behind the observed mortality/longevity patterns at most levels of sub-national aggregation.

Demographic data and estimation approaches

Traditional demographic methods for mortality estimation typically revolve around the patterns described by age and sex schedules within periods or cohorts (Riffe et al., 2020). Classical families of parametric and non-parametric mortality models, such as Gompertz, Brass, Kaplan-Meier, and more recent Lee-Carter approaches aim to describe the level and the shape of mortality curves. Studies at the intersection of demography and the fields of public health, political science, economics and applied sociology extend the models to include relevant frailty parameters, morbidity risks, and cause-specific hazards (e.g., Cox proportional hazards model). However, most studies employing formal demographic techniques seek to accurately estimate mortality, and not so much to explain its causes. For instance, small area death count estimates derived from negative binomial or Poisson models with additional predictors rely on such secondary covariates only insofar as to improve model fit when data are grouped or sparse (National Center for Health Statistics (U.S.), 2018). Nevertheless, a decent number of demographic studies pursue the examination of the origins of mortality in particular time intervals, age, sex, health and socio-economic status, as well as environmental conditions (Browning et al., 2011).

In the recent decades the arguments advocating for spatial analysis of socio-demographic phenomena have gained momentum with the gradual advent of computing power and suitable software tools for spatial analysis. Demography is an inherently spatial field, as demographic events are naturally the product of spatial dependence between individuals, groups, or entities, and often come about through the diffusion and interaction of location-tied ideas as well as the redistribution of the population (Matthews & Parker, 2013; Wachter, 2005; Weeks, 2004). As a consequence, many demographic studies are deemed spatially aware, even if no formal spatial analysis is conducted. Among other aspects of the unifying agenda of spatial demography, Raymer et al. (2019) note the need for a dynamic analysis of demographic phenomena that takes place in a particular space, with further ramifications for indirect estimation and forecasting of vital rates at the micro/local level. Macintyre et al. (2002) raise objections to the lack of theoretical framework in studying individual components of health and mortality outcomes, as opposed to the area effects, treatment of context as residual category, and the selection of scale. The problem arises when researchers are exclusively motivated by the data availability, rather than by parsimonious examination of theoretical/causal pathways of specific aspects of individual behavior, condition, and socio-physical environment. Instead, researchers are encouraged to consider, what collective aspects of an

area are being omitted from the data and what features indicate a healthy neighborhood. Likewise, spatial analysis has its proper place in determining the causes of health and mortality inequalities. These differentials are given rise to by the compositional and contextual effects of an area (Curtis & Rees Jones, 1998). The correspondence to the residents' needs, physical, social, cultural, and economic, with a due understanding of the variability in importance of these factors from one place or community to another is what ultimately determines their outcomes. Recent research work shows the growing gap in spatial inequalities in mortality among the middle-aged US adults in the 2010s, with clear indications of diverging trends between urban centers and the periphery (Vierboom et al., 2019).

Inherent challenges related to granular geographic data, such as small samples, uneven coverage, censoring for confidentiality, and others have all led to paucity of detailed sub-national estimates and publicly available geographically linked individual data. To solve this problem, recent methodological advances in small area estimation (SAE) enabled the generation of high-quality mortality estimates by age and sex at a sub-national level and over time (Alexander et al., 2017; Dwyer-Lindgren et al., 2022). While some effort has been directed toward the production of life tables for even smaller geographies, such as census tracts (Boing et al., 2020; National Center for Health Statistics (U.S.), 2018), the state and county estimates have been shown to be sufficiently robust to heterogeneous sources of bias (Winant et al., 2021). In parallel with the increasing availability of small area estimates (Dwyer-Lindgren et al., 2022; National Center for Health Statistics (U.S.), 2018) the recognition of the growing interest in exploring health dynamics at finer scales led to the introduction of tools and databases that provide high quality geographically linked data (Dush et al., 2023). Locality-specific municipal open data portals also allow researchers to extract detailed information about the contextual attributes of neighborhoods and cities that are highly pertinent to a given analysis.

From the estimation standpoint, today there is a number of methods available to researchers to position demographic phenomena within a spatial context. These include geographic discontinuity regression designs (Keele & Titiunik, 2015), geostatistical approaches (Goovaerts, 2009), Bayesian conditional autoregressive models (Alexander et al., 2017; Besag et al., 1991), and hierarchical linear models (Lacombe & McIntyre, 2017). One other method of socio-demographic analysis that has become increasingly popular in the recent past is in the field of spatial econometrics. The appeal of spatial econometric approach lies in the balance between the rigorous treatment of spatial and non-spatial components of the data generating process and in the intuitive inference from the results (Anselin, 2002; J. LeSage & Pace, 2009). This method is also very suitable for the study of area-level demographic phenomena (Campisi et al., 2020). Recent work on extending cross-sectional spatial econometric approaches into the longitudinal dimension is now firmly established in the literature (Bivand et al., 2021; Bouayad et al., 2018), thus presenting opportunities to study the causes of outcomes of interest.

In this dissertation I adopt an approach decoupled from individual-level data, independent of individual choices and background. My approach reflects the contribution of institutional factors to mortality outcomes of an aggregate. In so doing I aim to highlight the extent to which state health regulation shapes the mortality of the resident population, given local conditions. I place the analysis in the context of geographically linked effects

that many traditional analyses ascribe to unobserved heterogeneity. Here state health policy occupies a center spot within the relationship between the documented socio-economic, environmental factors and mortality. I seek to uncover the degree to which state health policy, namely through healthcare administration, healthcare and welfare provisions, and health behavior regulation in the US states impacts the mortality of states and county-level groupings. Explaining the collective action of these elements at multiple scales of aggregation and in the presence of contextual attributes would lead to a better understanding of how mortality outcomes are contingent on political, economic, and demographic dimensions of a place and its surroundings. In the course of this work I exploit an advantage associated with spatial approaches. The analyses presented in the chapters that follow do not depend on a singular source of data with all the required indicators, which would normally be expensive or laborious to obtain using conventional biomarkers or surveys. Instead, multiple unrelated data sets are linked to a geographic area with an administrative identifier, granting the scope and flexibility that is necessary to unravel the aforementioned relationship. Another important benefit of applying spatial treatment is that containment and spillover effects of social, economic, and demographic processes may be evinced through explicit accounting for the spatial structure of the state and county units. Like most phenomena, health behaviors and policy initiatives do not exist in a void: an implementation of one health policy in one area (e.g., tobacco tax, waste water treatment, etc.) may impact the health and mortality in a nearby area where no such policy was ever adopted. This consideration is particularly relevant to my present work, since health behaviors as well as certain forms of disease propagate through proximity between the actors.

Structure of the thesis

In the first chapter I present the relevant background reviewing to-date knowledge on a subnational mortality trends, and socio-economic and geographic disparities in mortality in the past 40 years. I introduce the background discussion on the interplay of political, social, economic and health factors to justify its use in the scope of the study of state health policy impact on mortality outcomes. In the second chapter, I propose a conceptual paradigm based on the structural causal modeling (SCM) framework considering the relationships between mortality, health policy, socio-economic, demographic, and political covariates reported in the literature. I provide an overview of the mechanics of causal inference using SCM and apply it to arrive at a pair of testable models: one with minimally-adjusted set of variables and another which I title a "fully specified" set. Both specifications are consistent with the SCM method, though I hypothesize the fully specified model might offer better precision when identifying the effect of state health policy on mortality due to loss of information when using fewer imprecise or indirect quantitative realizations of theoretical concepts (e.g., residential segregation).

In the third chapter I propose a method for calculating the scores aimed at quantifying state health policy as a singular annual state-level summary indicator. I introduce three sources of harmonized policy data that I synthesize into a unified set of select 110 thematic health-related policy groups, each assigned to one of 8 thematic categories and to one of 4 policy classes. To translate the qualitative character of the policies into a testable quantity,

I assign numerical values based on perceived impact of a regulation. From this set I create 2 measures of state health policy, of which the first is complemented with its time-lagged counterpart. The second type serves as a more naïve alternative to the first and measures the relative rate of health policy innovation. Following the exposition of the results, desirable properties and limitations of each proposed indicator are discussed in greater detail. Finally, using the state and county-group life tables from the United States Mortality Database (United States Mortality DataBase, 2023; Winant et al., 2021), I conduct preliminary one- and two-way repeated measures analysis of variance between these two indicators and the natural logarithm of sex-specific age-standardized death rates. The aim is to identify the differences in means in each indicator independently and to verify the temporal dependence of the two-way association between SHP and mortality at the state and county-group levels.

The final chapter serves as a forge to all the components hitherto introduced. The goal of this chapter is to identify the effect of state health policy on mortality outcomes at the two levels of spatial aggregation. It is ultimately achieved through a series of extensive step-wise modeling exercises, narrowing down the model selection to obtain the most optimal result. I facilitate this process by assembling the theoretical foundation developed in chapter 2, expatiating on the 3 mortality outcomes of interest (age-adjusted mortality rate, life expectancy at birth as well as at age 65) and state health policy indicators generated in chapter 3, and computing additional secondary variables to back up its prospective use in the models in accordance with the SCM developed in chapter 2. Suspecting spatial dependence in the data, I exploit spatial econometric models to select one model that would most closely describe the spatial dependence process governing the association between SHP and mortality.

I conclude the analysis with a discussion of the results, ramifications for future work, and implications of my approach and its findings.

Chapter 1

Four Decades of Mortality in the United States, 1980–2018: Review of the Past Literature

The notion of a healthy society is in part predicated on the role of government in containing and preventing public health crises and maintaining the general health welfare of the population. At least a couple of noteworthy examples from the recent decade underline the significance state actors have in managing major outbreaks. First is the ongoing mortality trend in the United States stemming from a collection of preventable causes of the so-called "diseases of despair" (Case & Deaton, 2015). Of those the opioid overdose stands out as the most prominent cause that reached its peak potency in the mid-2010s, claiming between 40,000 and 50,000 lives annually (Case & Deaton, 2015). The involvement of local health departments in curbing the proliferation of opioid-related deaths by providing necessary education to under-resourced communities could not be understated, in spite of the remaining challenges (Feuerstein-Simon et al., 2020). Likewise, federal and local health policy response to a public health emergency during the subsequent COVID-19 pandemic has been ubiquitous, though not unified in its aims (Adolph et al., 2021). However, by far not every local policy action is directed toward a universal health concern. Many places rely on the administration of healthcare resources and programs to alleviate the conditions resulting in pressing health issues in the immediate communities (Haskell et al., 2009; Roth et al., 2017). Evaluating the cumulative effects of multiple health policies in the context of existing health regulation differentials across state borders is inherently complex, yet important from the point of view of future interventions. Therefore, teasing apart the overall impact of state health policies in the presence of other drivers of the geographic mortality trends remains a sought-after result for many a public health and public policy researcher. The first step in establishing a causal link between the state health-oriented legislature and mortality is to define the structural relationships between the intervening factors. The aim of this chapter is to examine the existing body of literature on mortality disparities, its evolution over time and its causes. This evidence will be applied to construct a theoretical framework (chapter 2) to serve as a basis for numerical evaluation in the subsequent chapters. I begin by revisiting the relevant literature to identify the precursors to the condition of population health and

to document the pathways that ultimately lead to higher or lower morbidity and mortality in an aggregate, areal setting.

1.1 Geographic mortality trends

The national life expectancy registered a slow increase in the recent decades, rising roughly at a rate of a year per decade between 1980 and 2000, reaching 79.4 years at birth for women and 74.1 for men, thereafter slowing to down to a plateau of 81.4 and 76.2 years by the mid-2010s, respectively (United States Mortality DataBase, 2022). Despite the improvement, this overall increase was lagging significantly behind many developed countries since the turn of the century (Barbieri, 2022). The slow rate of progress in the US is at least partially attributable to the increase in sub-national variation in adult mortality, as compared to the other developed nations (Wilmoth et al., 2011). The new sub-national patterns form the backdrop of this progress, as shown in Figure 1.1. Notably, the broadening of the urban-rural gradient (Elo et al., 2019; Singh & Siahpush, 2014) and associated ethno-racial differentials likely point to the changing landscape of the effects of socio-economic (SES) disadvantage in the population (Dwyer-Lindgren et al., 2022; Vierboom et al., 2019). In fact, local and regional determinants of mortality demonstrated superior potential in explaining the variation in life expectancy in different population groups (Dobis et al., 2020). For instance, there was a sizable gap of nearly 21 years between the life expectancy of middle-aged Asian women and African American men on the national level in 2001, both when input data were grouped by state and by county (Murray et al., 2006, 2005). And while these ethno-racial groups represent the extreme ends of the life expectancy distribution, the causes of mortality are vastly different for the two groups, as a function of their occupational and socio-economic aspects. Ethnic and racial disparities in mortality to a large degree stem from social and material resource deprivation (Block et al., 2004; Browning et al., 2011). More recent trends indicate that life expectancy among high-mortality groups, such as males, most resource-disadvantaged segments of the population, and ethno-racial minorities have witnessed rapid increases, though with considerable variability among the states (Dwyer-Lindgren et al., 2022; Harper et al., 2014). The most positive developments have been recorded for African-American males in the decades since 1990, despite the relative disadvantage typically experienced by the lower SES members of minority groups (Currie & Schwandt, 2016). Singh (2003) corroborates the observation that in the last three decades of the 20th century racial and ethnic minority disadvantage began to matter less for explaining mortality inequalities, whereas the socio-economic gradient coinciding with the degree of rurality of an area began to matter more. At the same time, the 1980s have been the decade of rampant inter-county mortality inequalities along the sex axis, particularly for women in the most disadvantaged segment of the population (Ezzati et al., 2008). Female uptake of smoking, obesity, and high blood pressure, in addition to the both-sex HIV related deaths contributed to the stagnation or reversal of progress in female life expectancy in a number of regions (Ezzati et al., 2008). Kindig and Cheng (2013) documented the continued deteriorating trend in female mortality well into the period of the 2000s throughout the counties of the deep South, Appalachia, and the rural West. Indeed, female life expectancy declined significantly in the 154 counties within the aforementioned areas in the period of 1993-2002 thus widening the within-sex

gap, while shrinking the male-female gap top-to-bottom (Wang et al., 2013). Despite the overall improvement between 1990 and 2019, the age-adjusted mortality rates (AAMR) have deteriorated for both men and women aged 25–64 living outside the large metropolitan areas, and most remarkably in rural areas and among native Americans in the same age band (Cross et al., 2021). The widest mortality gap in 1990 has been registered between metropolitan Asians/Pacific Islanders (59% lower AAMR) and rural non-Hispanic Blacks, whereas in 2019 metropolitan Native Americans had 65% lower AAMR than rural non-Hispanic Blacks (Cross et al., 2021). Figure 1.2 illustrates that the overall mortality has declined since 1980 in both urban and rural areas. It is largely concurrent with respect to the directionality of the temporal trend between the two locales. The level of rural mortality has remained above that of metropolitan areas for most major causes of death and grew substantially for chronic respiratory diseases, mental and substance use disorders, and diabetes and related diseases of endocrine system. The exceptional lead of urban mortality from 1980 through 2014 was registered in the growth of neurological disorders, such as dementia, Alzheimer's disease, etc. This development has been preceded by the incremental increases in longevity and the late stages of the epidemiological transition. Cardiovascular diseases that dominated the contribution to mortality for decades have nearly halved over the 3 decades, in no small part due to the decline in smoking and due to the increases of living standards brought about by higher educational attainment (Graetz & Elo, 2022). This declining trend reached a point of stagnation in the 2010s (Cross et al., 2020). However, importantly, the variability of outcomes across the rural areas has been much greater overall and continued to rise at a more rapid pace than variability in mortality in the urban centers over the past 3 decades. This corroborates the findings of widening geographic inequalities on a sub-national level, as discussed in Vierboom et al. (2019).

These observations concur with the geographic patterns of demographic composition of the population. Neoplasms and cancers have emerged as one of the leading causes of death in the latter part of the 20th century, only second to the cardiovascular diseases (CVD), with the most prominent increase of 20%–46% from 1980 to 2014 in neoplasm-related deaths across multiple clusters of counties in the Appalachia region (Dwyer-Lindgren et al., 2016). Deaths from CVD remain the leading cause of death in the US, particularly in the southern Mississippi River valley and the southern part of the US, and in Oklahoma in particular (Roth et al., 2017). While CVD mortality is on a steady decline trend nationwide, county-level decomposition using flexible multivariate Bayesian space-time modeling demonstrated that aforementioned areas are indeed CVD mortality hotspots that dampen the reductions in the national trend (Quick et al., 2018; A. S. Vaughan et al., 2017). At the same time, diabetes, blood and endocrine diseases lead to progressively greater number of deaths over time, across the country, doubling in Appalachia, metropolitan areas of Portland, OR, and swaths of the central US. A notable exception to the trend is represented by a handful of counties in the area of Rocky Mountains and Denver, CO, where the death toll from diabetes and endocrine diseases declines by 20%–40% since 1980. Mortality due to neurological disorders has been rampant since the 1980s, doubling in the counties of the so-called Sun Belt, and increased to a lesser degree across the northern Midwest of the US. Many poverty-stricken areas of the South exhibited increased prevalence of chronic respiratory and infectious diseases. On the other hand, deaths due to violence and self-harm have remained relatively unchanged as a

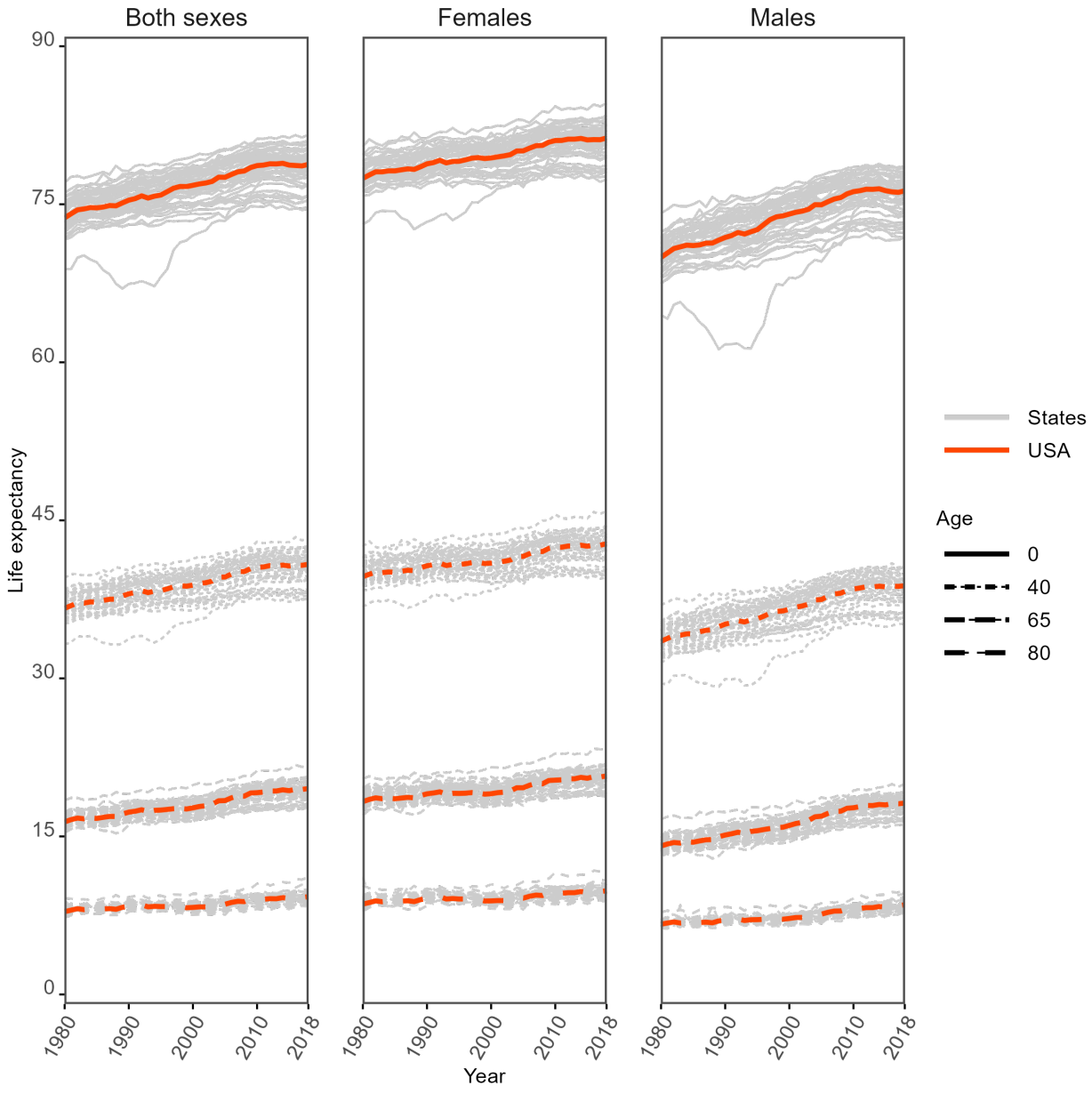


Figure 1.1. Trends in national and state-level life expectancy (1980–2018), by select age groups.

Source: Own elaboration on the data from the United States Mortality DataBase (United States Mortality DataBase, 2022, 2023)

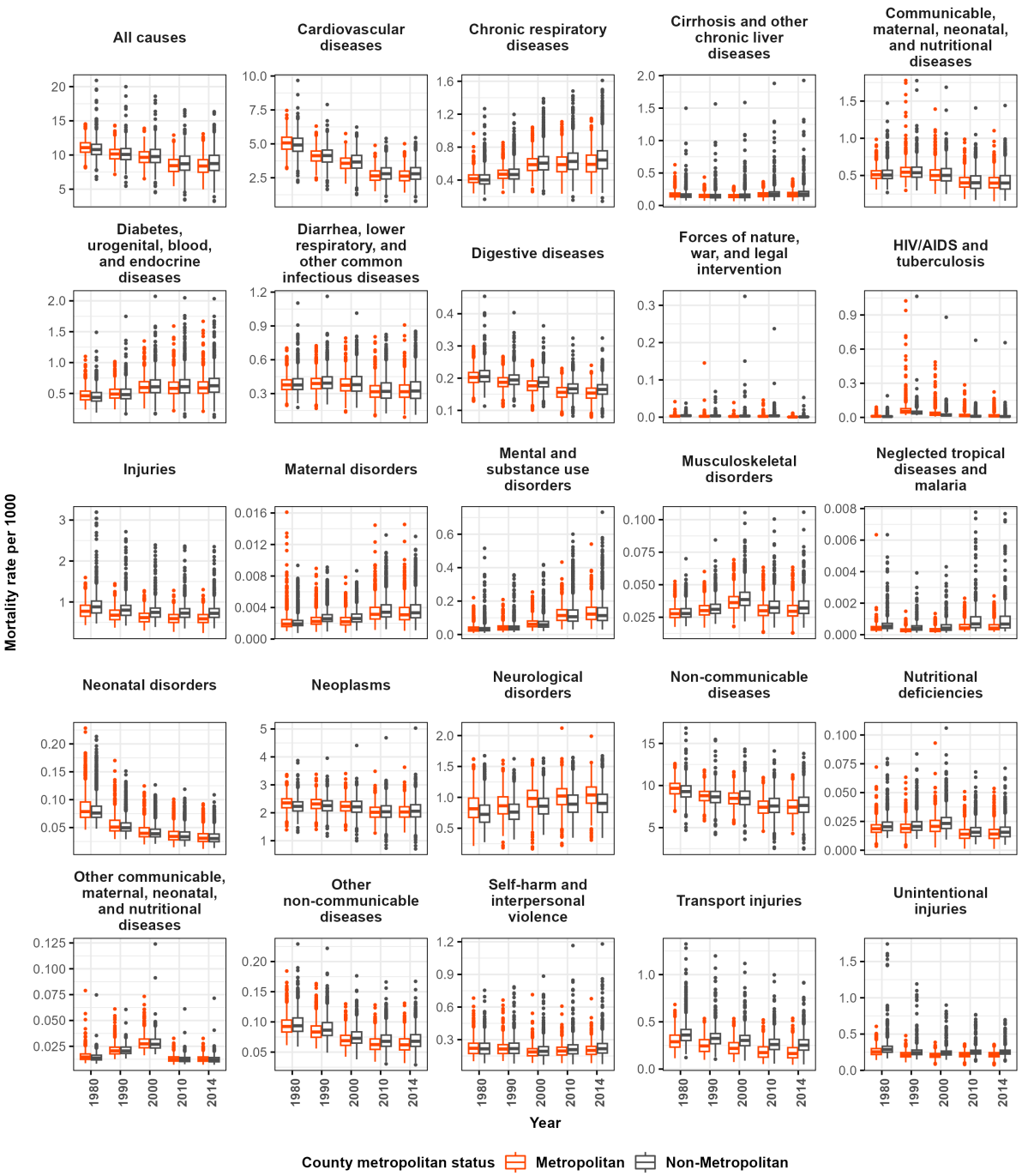


Figure 1.2. Trends in county-level age-adjusted mortality 1980–2014, by county metropolitan status, for major causes of death

Source: Own elaboration on IHME data. Institute for Health Metrics and Evaluation (IHME). United States Mortality Rates by County 1980–2014. Seattle, United States: Institute for Health Metrics and Evaluation (IHME), 2016. Data retrieved from http://ghdx.healthdata.org/record/ihme-data/united-states-mortality-rates-county-1980-2014 on May 25, 2020.

whole, yet saw large increases of more than 50% in the rural counties across the northern and western US, and equally large decreases in the major urban cores, such as Los Angeles, San Francisco, Houston, Miami, Chicago, Atlanta, and the cities along the Northeastern corridor. Like CVD, preventable causes such as transport injuries are on the steady decline and are being rapidly supplanted by the mortality from substance abuse and mental disorders in the counties of the former industrial Rust Belt. Figure 1.3 confirms that CVD decline was responsible for some of the highest gains in life expectancy on the county level throughout the country, with some exceptions. As expected, neurological disorders had led to the largest changes in mortality in the most recent years, though hardly large enough to offset the declines in the other causes. Some of the counties in Sun Belt states and rural Nevada, Texas, Montana, Tennessee and Kentucky with the most stagnant mortality have actually seen a slight drop in life expectancy, often for the same reason that the most successful counties have improved.

1.2 Health and mortality in a spatial context

Traditional analyses of demographic estimation of mortality are undegirded by a collection of modeling approaches. Most such models involve elaborate parameterization and extensive characterization of hazards and risks by age as sex (Chiang, 1984; Jarner & Kryger, 2011; Riffe et al., 2020), despite calls to consider demographic events in context with their surroundings (Matthews & Parker, 2013; Wachter, 2005; Weeks, 2004). Some such approaches exploit compositional group or areal attributes (European Commission. Eurostat, 2010), but overlook the valuable information offered by the nuances and interactions of the processes occurring in geographic space (e.g., due to proximity, adjacency, etc.). These models overlook the spatial dimension to explicitly account for unobserved heterogeneity, which is otherwise attributed to model error. The recent availability of data at finer geographic scales spurred the growth of popularity of small area estimation (SAE) techniques that tied the process of mortality estimation to geographic areas (National Center for Health Statistics (U.S.), 2018). However, so long as the mortality prediction is deemed accurate, the use of areal covariates in fitting the outcome often falls short of unraveling the complexity of independent structural interrelationships of which mortality and its spatial determinants are part.

Much health related research has focused on studying proximal individual determinants of health and mortality outcomes, such as smoking, stress, physical exercise, diet, and healthcare access, predominantly at coarse spatial resolutions (e.g., national and regional). However, historically physical and socio-demographic environmental conditions have received much less attention from the research community, particularly as contextual determinants at the local level. The new emerging studies are pushing this barrier aside (Graetz & Elo, 2022). The choice to study spatial units rather than individuals may stem from the fact that it is very difficult to study individual health outcomes without evoking concerns for individual confidentiality. In addition, the process of selection of individuals into spatial units is all but simple. Given that individuals can factor a multitude of considerations on whether to stay or to move from a defined area, such as environmental characteristics, these attributes may equally impact individual residential choices if the housing price is sufficiently justifiable for

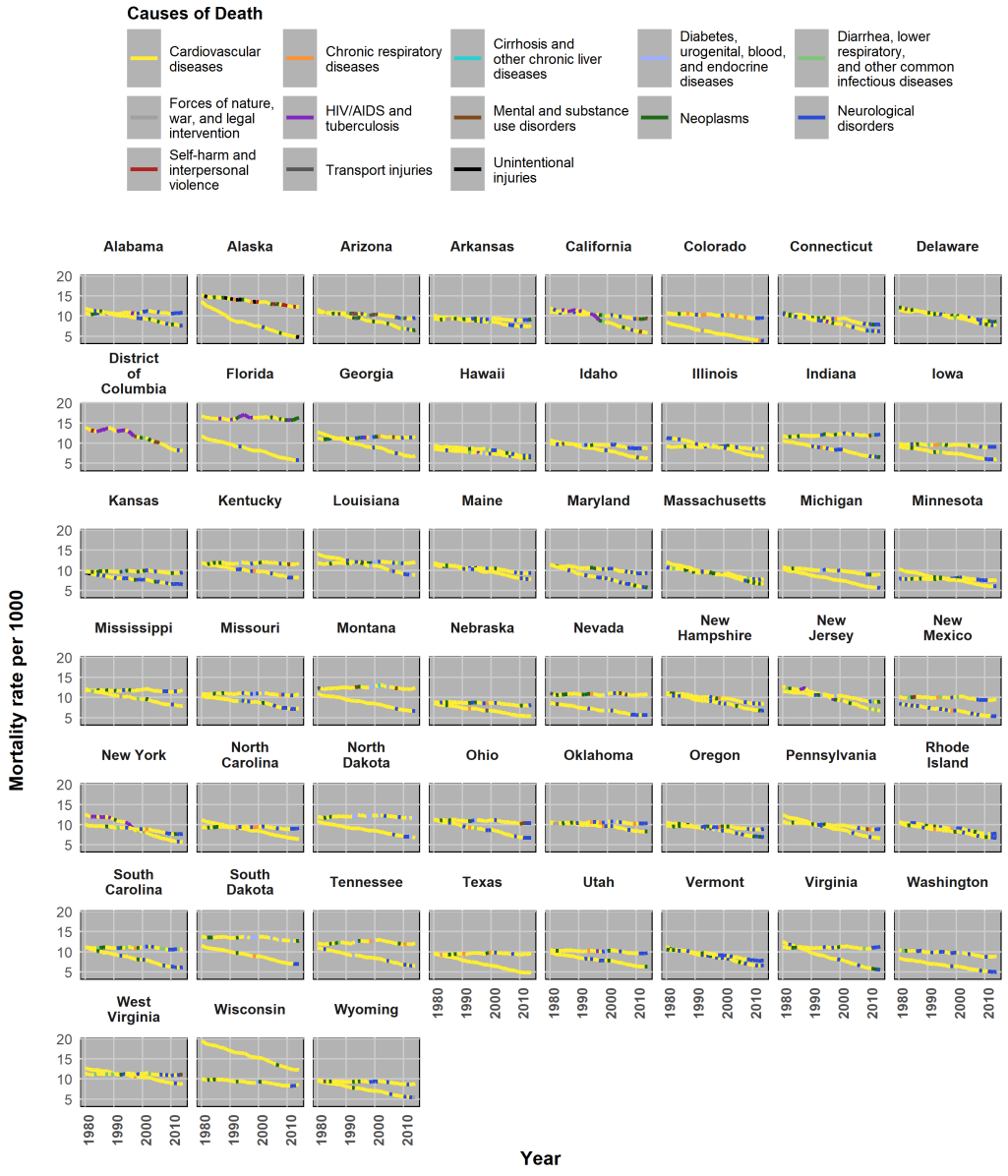


Figure 1.3. Trends in county-level age-adjusted mortality rate 1980–2014, by state and major cause-of-death contributor. For each state, 2 counties are shown, one that underwent most change and another that changed the least in the period of 1980–2014. Shading corresponds to the cause of death that contributed the greatest absolute increase or decrease in deaths between two consecutive years.

Source: Own elaboration on IHME data. Institute for Health Metrics and Evaluation (IHME). United States Mortality Rates by County 1980–2014. Seattle, United States: Institute for Health Metrics and Evaluation (IHME), 2016. Data retrieved from http://ghdx.healthdata.org/record/ihme-data/united-states-mortality-rates-county-1980-2014 on May 25, 2020.

a level of pollution that is different from the "usual" conditions people would expect to find in the general vicinity (Portney, 1981). Highly subjective decisions thus add another layer of complexity. The decisions are conditional on many unobserved characteristics that are typically not measured in conjunction with residential decisions by most large-scale survey studies. Absence of such detailed information introduces omissions of the actual exposure to various locations where people spend time. To mitigate such challenges, non-spatial or large-area research tends to focus on common individual-level predictors of health and mortality that highly correlate with unobserved exposure variables. As an example, Nandi and Kawachi (2011) present a large number of studies that demonstrate the impact of individual SES on mortality. However, given income inequality, individual physical and social mobility, the common aggregate measures of SES on the neighborhood level may be imprecise and entail even greater assumptions about the true life chances of an area's residents, given their education, occupation, income, etc. It is equally important to remember that inferring individual outcomes on the basis of an aggregate, no matter how small, renders the result vulnerable to ecological fallacy (Macintyre et al., 2002). It also makes inferences potentially unstable due to the modifiable areal unit problem (MAUP) (Openshaw, 1984). The nuanced use of finer geographic scales along with spatio-temporally linked data becomes a valuable resource for explaining the impact of compositional attributes of an area on health and mortality, independently and in conjunction with the other attributes, related through spatio-temporal links. Therefore, in an effort to establish a plausible and testable causal model for gauging the cumulative impact of state health policy on sub-national mortality I turn to the literature on *spatially aware* mechanisms through which socio-economic and demographic factors are associated with health and mortality.

1.3 Political, environmental, socio-economic and demographic components of area-level mortality

Like many phenomena health and mortality take place in space. After all, each is likely dependent on physical features and social and economic processes that co-occur in that space. For instance, Currie and Schwandt (2016) emphasize the significance of considering multiple aspects of deprivation (e.g., poverty or fraction of high school dropouts) over time in order to gauge the mortality inequalities on a group level. Neither one explanatory factor, nor one outcome fits all possible scenarios, where mortality rates as well as its inequality differentials at various ages are subject to period-cohort effects. The authors note the lingering mortality disparities on the geographic level go hand-in-hand with those by socio-economic status (SES). These inequalities are tied to behaviors such as smoking, lack of exercise, and unhealthy diet, in turn leading to progressively increasing lung cancer prevalence among older women with lower SES and to obesity rates among people of lower educational and occupational attainment (Currie & Schwandt, 2016).

Focusing on sex as one of the core demographic distinguishing factors, Figure 1.4 clearly demonstrates the impact of the sex component of declining overall mortality since 1980. Dominated by the declines in deaths from cardiovascular diseases (CVD) female mortality has seen sizable increases in deaths from respiratory diseases, diabetes, maternal, and neu-

rological disorders. In the same period men's survival outcomes have improved at a greater rate than among women, despite the fact that mortality from diabetes, mental and substance use, and neurological disorders grew in magnitude. The reasons for each increase for each sex and overall are distinct and merit individual examination. CVD decline in both sexes has been linked to the decrease in the prevalence of smoking in the US over time to less than one-fifth of the population at present time (Rostron, 2013). More women took up smoking in the latter part of the 20th century, but only recently the synchronous drop in smoking has produced about 22% and 27% decrease in male and female deaths from ischemic heart disease, respectively (Rostron, 2013). Another interesting observation can be made about maternal mortality and its increase. While this is a cause for policy concern to ensure that maternal care is readily available and that maternal mortality declines, the reasons for the relative hike may have to do with better diagnoses and reporting of maternal disorders in the recent couple of decades (Carroll, 2017).

Geography as a medium for mortality disparities

What becomes clear from Figure 1.4 is the increase in geographic inequalities by county in mortality outcomes for both sexes. Interestingly, the geographic variability for all-cause mortality remained fairly stable over the prior 3 decades, but the growth of cause-specific inequalities is apparent and, at times, glaring. Geographic variation in deaths from diabetes and endocrine diseases, as well as mental and substance abuse disorders has approximately doubled since 1980. In fact, county-level inequality has been rising for both sexes, with periods of plateau, but since the early 1990s it remained about 50% higher among men than among women, reaching as high as 19 years at birth in a given year (Wang et al., 2013). The areas of the South and the Appalachians remained consistently at a disadvantage relative to the rest of the country, particularly for males.

Examining the theoretical perspectives of living in urban or rural regions with a range of affluent or resource-deprived areas sheds light on the observed health disparities (Curtis & Rees Jones, 1998). Namely, one could ask, what advantages or disadvantages stem from living in a specific urban or a rural region? What aspects of social or economic/service infrastructure influence the degree of deprivation of a place? How is deprivation of an area related to morbidity and mortality of its population? On the conceptual level, research focusing on place and health should look beyond the context and composition of a geographical unit, and should also pay attention to the temporal variability in the two aspects while considering interactions that give rise to the observed contextual processes (Cummins et al., 2007).

The recent opioid overdose epidemic, notably documented by Case and Deaton (2015, 2017), was manifested by deteriorating mid-life mortality in the US, thus rekindling scientific interest surrounding local geographic variation in mortality outcomes. Specifically, more concerted effort has been devoted to the study of geographic disparities in mortality beyond just state and county borders and explaining the impact of contextual factors on mortality in general, as well as the so-called "deaths of despair" in particular. For instance, Vierboom et al. (2019) emphasize that geographic disparity in survival by age positions the individuals in their mid-adulthood at greater disadvantage in terms of the overall mortality improvement. The spatial variation in the characteristics of places and the underlying population is nevertheless substantial across the urban-rural spectrum, particularly for the leading causes

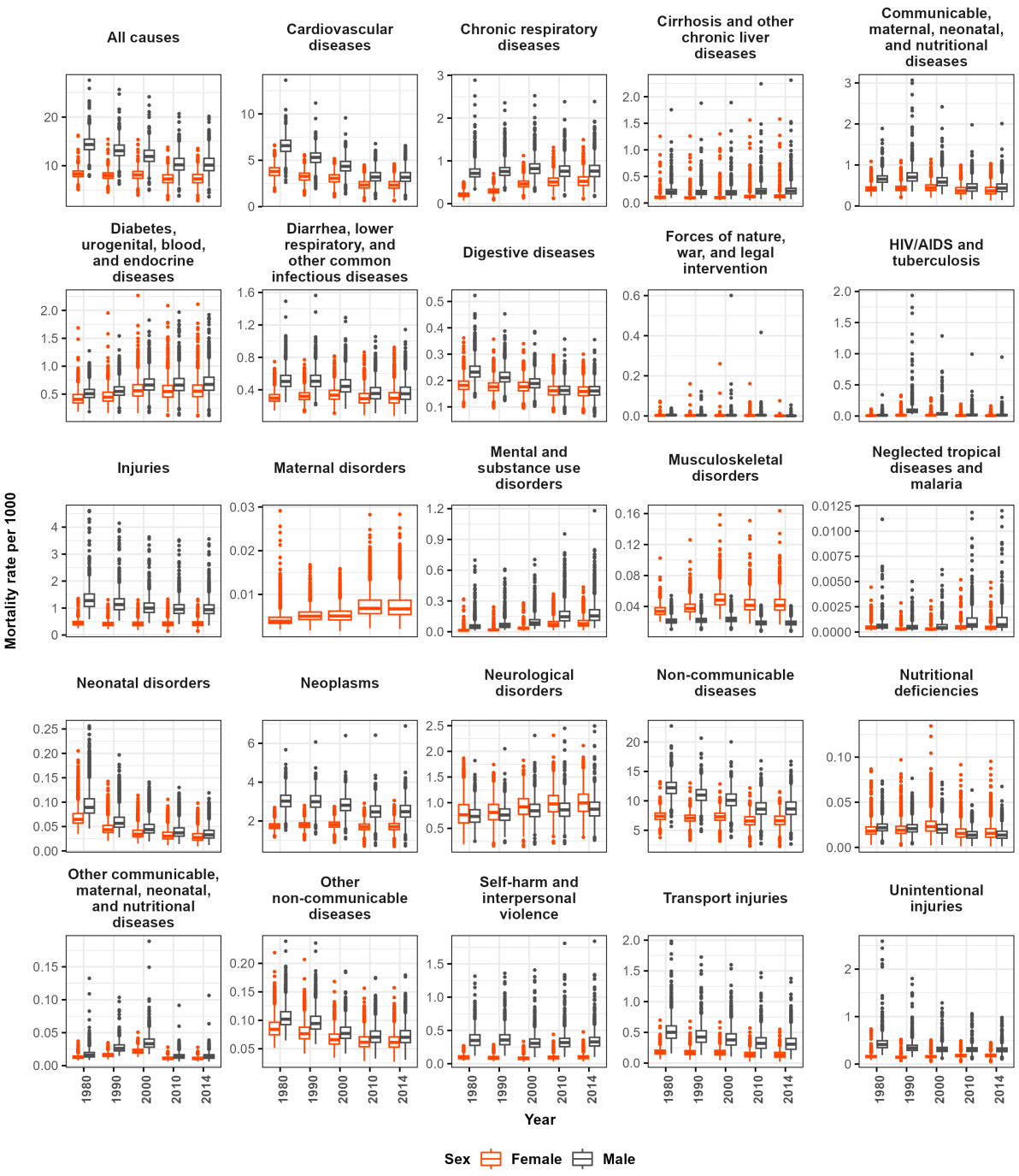


Figure 1.4. Trends in county-level age-adjusted mortality 1980-2014, by sex, for major causes of death

Source: Own elaboration on IHME data. Institute for Health Metrics and Evaluation (IHME). United States Mortality Rates by County 1980-2014. Seattle, United States: Institute for Health Metrics and Evaluation (IHME), 2016. Data retrieved from http://ghdx.healthdata.org/record/ihme-data/united-states-mortality-rates-county-1980-2014 on May 25, 2020.

of death, such as heart disease, lung cancer, and diabetes to name a few (Singh & Siahpush, 2014). Boing et al. (2020) demonstrate the extent of variation in mortality when considered at the level of census tract, county, and state, concluding that inequalities in health and mortality tend to be more local in nature than has been viewed by much of the prior research. In summary, much of the recent evidence from the literature on mortality disparities provides impetus to broaden the scope of analysis by including geographic identifiers at multiple levels of granularity.

1.3.1 Sources of mortality inequalities and resource deprivation

Naturally, a series of questions arises concerning the causes of mortality and its inequalities as local phenomena. The preponderant features found in the literature that are used to explain the differences in mortality across geographical areas are classified under the umbrella of determinants of resource deprivation and socio-economic disadvantage. At the same time, many research studies go further by examining the relationship between mortality and the components or proximal factors of deprivation and disadvantage. There has been a consistent effort to measure local mortality outcomes using indices of multiple axes of deprivation in the US and abroad (Centers for Disease Control and Prevention, 2013; Office for National Statistics, 2021). For instance, the evidence from the annual reports from the UK Office of National Statistics (IoMD, UK) determined the existence of a link between the relative deprivation rank and life expectancy on the postal code level. The annual multiple deprivation index has been informed by its weighted components:

- Income Deprivation (22.5%)
- Employment Deprivation (22.5%)
- Education, Skills and Training Deprivation (13.5%)
- Health Deprivation and Disability (13.5%)
- Crime (9.3%)
- Barriers to Housing and Services (9.3%)
- Living Environment Deprivation (9.3%)

An application of IoMD has been used to study variance in age at death and how deprivation accounts for the area-level heterogeneity in mortality outcomes in Scotland at the post-code level (Seaman et al., 2019). This study highlights the importance of the areal decomposition of mortality measures to identifying social determinants of mortality that would explain the differences in mortality inequalities. Yet, such otherwise valuable studies miss out on the interaction of both the covariates and the outcomes in a spatial context. Accounting for spatial associations would greatly broaden the understanding of how inequalities and its causes propagate across cities and towns. Similar initiatives to measure deprivation have been undertaken in the United States, albeit with all components being weighted equally (Centers for Disease Control and Prevention, 2013). It has been previously shown that various elements of "deprivation", such as food deserts are a by-product of various social ills in the local context (e.g., residential segregation along racial and socio-economic lines). For instance, food insecurity brought about by lack of access to high quality nutrition in the vicinity of the place of residence may precipitate in chronic conditions, such as obesity and diabetes often lead to long-term health complications and potentially decreased lifespan

(Block et al., 2004; Gucciardi et al., 2014).

Segregation, race, and gentrification

Earlier sources discuss the role of institutionalized racism as embodied by segregationist practices in driving up the mortality among the minority groups, by exposing them to lower quality of life and poor access to basic necessities, by virtue of poverty associated with their residential arrangements (Appel & Nickerson, 2016). Massey and Fischer (2000) elaborate on the mechanism by which the combination of the shape of income distribution, declines in average income, and residential segregation along socio-economic class lines act in tandem with respect to the poverty concentration in an area. Most importantly, the authors emphasize that given higher segregation levels in an area, the effects of income and its inequality would prove disproportionally harmful to a community, as compared to just a low-income neighborhood. The de-industrialization trend originating in the latter half of the 20th century has instigated the fall of life expectancy in many previously well-off blue collar worker communities and marked the starting point of the increase in health inequalities due to differential poverty and incarceration (Nosrati et al., 2018). Specifically for the poor but not for the wealthy, areas impacted by underlying dynamics of de-industrialization and incarceration are linked to the reduction in the average lifespan of as much as 2.63 years, and likely more so, given sufficient levels of spatial and socio-economic disaggregation (Nosrati et al., 2018). However, Huie (2001) suggests that collective aspects that define an aggregate (or an area) may undermine any individual advantages and disadvantages among the residents. Owing to the dynamics of the rampant residential segregation along the ethno-racial lines and its far reaching consequences for the inhabitants of many cities in the United States, multiple studies propose pathways originating at social aspects of a neighborhood that ultimately lead to differential well-being outcomes (Massey et al., 1987). Now long-defunct redlining practices still hamper residential choice and mobility along the ethno-racial lines due to the cumulative disadvantage that resulted from such practices (Appel & Nickerson, 2016). In conjunction with institutionalized racial discrimination this led to proliferation of inner-city ghettos with unenviable quality-of-life standards. The aftermath of these practices has continued to impact the residents' long-term health through the propagation of food deserts (Gucciardi et al., 2014), poor infrastructure and schooling (Collins & Williams, 1999), crime, and lack of critical health and safety services (Almond et al., 2006). Hence, health and mortality outcomes can often be linked to socio-economic decline of an area (Nandi & Kawachi, 2011).

As part of the general urban dynamic gentrification too may play an important role in determining health outcomes of a populated area. Considered in a less strict sense, relative to the social-class based definition originally coined by Ruth Glass (1964), a gentrified area may increase in either the value and condition of its housing and services, socio-economic improvement of the characteristics of its typical residents, or both. It may thus be hypothesized that gentrified urban areas have seen reductions in period mortality, particularly during the shift in demographic composition of the neighborhoods to younger, wealthier professionals. Simultaneously, the urban periphery might see stagnation or slower progress in life expectancy, where older and residents with lesser means tend to concentrate over time. This hypothesis is consistent with the recent findings on life expectancy changes by metropolitan status and

region (Elo et al., 2019). Much of the existing public health research to date has focused on environmental factors and urban sprawl dynamics that impact health and life expectancy by using microdata (Hamidi et al., 2018). Conversely, social science studies on gentrification and area inequality generally overlook quantifiable health hazards, instead focusing on sociological notions of inequality and stratification. Gentrification is a multidimensional concept, but not all aspects of areal upward socio-economic transition relate to gentrification, as many processes do not involve population displacement or radical changes to the built-up character, but rather an external influence (e.g., private interventions or government policies, infrastructure amelioration, etc.) (Owens, 2012). Recent cross-sectional investigation of county-level mortality outcomes in the US attributes the decline in life expectancy to the socio-environmental factors closely associated with urban sprawl (Hamidi et al., 2018). This study suggests that counties sprawling away from urban cores have higher prevalence of chronic diseases due to higher body mass index (BMI), increased travel and emergency response times, and higher number of traffic fatalities, unrelated to worse living conditions. Massey and Denton (1989) is one of the earlier examples of the use of such contextual local area characteristics to study the effects of segregation among non-Hispanic Black and Hispanic populations. With respect to mortality in metropolitan areas, a nearly omnipresent ethno-racial within-group gradient offers some evidence that segregation dynamics are responsible for the conditions leading to shorter lifespan among Black and Hispanic residents (Nuru-Jeter & LaVeist, 2011).

Urban-rural continuum defining socio-economic well-being

Urban-rural mortality divide is particularly stark in the context of preventable causes of death. Overall, controlling for local socio-economic factors, metropolitan status has been more predictive of changes in county-level mortality than only accounting for the state (Graetz & Elo, 2022). This implies that many of the socio-demographic causes of mortality differences vary across the urban-rural spectrum, rather than along the administrative boundaries. For example, institutional racial segregation of healthcare has been a major determining factor of disproportionately high infant mortality among African Americans in the areas of the rural South, the phenomenon that only began subsiding following the passage of the Civil Rights Act of 1964 banning segregation in health facilities (Almond et al., 2006). Indeed, CDC (Centers for Disease Control and Prevention, 2013) notes that non-Hispanic Blacks are 50% more likely to die of heart disease or stroke before the age 75 than non-Hispanic Whites. At the same time, non-Hispanic Whites and Asians as well as higher income and college educated populations generally experience lower prevalence of diabetes and obesity. African Americans exhibit nearly double the infant mortality of non-Hispanic Whites. Many of these disparities are deeply intertwined with social, behavioral, and health risk factors given rise to by the environment, conditions and composition of the populated areas (Elo et al., 2019). While the effects of residential segregation are more strongly associated with the urban and suburban landscape (L. Vaughan & Arbaci, 2011), the concept of resource deprivation, on the other hand, could be a hallmark of just about any densely or sparsely populated place. It is true that, on the whole, urban infrastructure ought to facilitate access to amenities improving the quality of life and access to healthcare. On the other hand, parts of the US rural landscape features a dynamic mimicking that of develop-

ing nations where healthcare is of lower quality or inaccessible. This is often coupled with elevated environmental health hazards, such as poor water quality and occupational hazards (Cutler et al., 2006). The onset of chronic health conditions has been linked to resource deprivation in isolated communities of color (Block et al., 2004). Buscemi et al. (2021) argue that inequalities experienced by African Americans and Hispanics in terms of their physical isolation and in association with the lack of healthy nutrition are reflecting on the ethno-racial inequalities in the population prevalence of diabetes. Spatial proximity and clustering of socio-economically disadvantaged communities was found to be significantly detrimental to infant mortality outcomes (Sparks et al., 2013). Some research evidence also suggests that poor overall level of health and psycho-social well-being are rooted in the low quality of one's living environment and its negative perception by the residents, all of which are commensurate with the one's SES (Wen et al., 2006). However, income and SES become increasingly influential determinants of health and mortality disadvantage in rural areas (Gong et al., 2019). Access to primary care is among the many features of urban environment that has significant potential in decreasing the health complications that lead to mortality among the most resource-deprived populations (Shi et al., 2003). Furthermore, the degree of area rurality increases the population exposure to traffic related health hazards as travel times increase (Hamidi et al., 2018). Much like in urban ghettos, timely physical access to quality healthcare in the rural areas is often limited, leading to higher mortality from injuries from accidents and treatable diseases (Hamidi et al., 2018).

Income, income inequality and poverty as pillars of deprivation

Income and income inequality are two widely cited facets of deprivation. The patterns of mortality decline with increasing income have been well documented in the literature (Cutler et al., 2006; Preston, 1975). More recently, and with an emphasis on the US sub-national perspective, state-level contextual income inequality has been the most deleterious for both non-Hispanic White and Black populations living in the poorest areas (Lochner et al., 2001). Likewise, but on the county level, Chetty et al. (2016) document a consistent finding that low-income individuals fare much better in terms of life expectancy if they live in cities with highly educated populations, incomes, and high levels of government expenditures (e.g., New York City and San Francisco). In fact, the mounting income inequality is a contributor, secondary to the income levels, to the growing deprivation in the US as a whole (Lobmayer & Wilkinson, 2000), and sub-nationally across the states (Lochner et al., 2001). However, there is a high degree of geographic variation in the association between life expectancy and income, partly driven by the composition of each individual area. Another analysis shows that 1980-2000 growth of life expectancy at a rate of about half a year per decade was also accompanied by the increase in regional variation of life expectancy, both on state and county levels, particularly due to the deterioration of progress among the least advantaged regions (Wilmoth et al., 2011). Ranked percentile rates of poverty at the county level were associated with an exponential increase in the number of years of life lost (YLL) within a group of the 10% poorest counties, whereas such an association is all but weak for the wealthiest counties (Egen et al., 2017). The gap in survivorship between the affluent metropolitan areas and the poor non-metropolitan areas has become increasingly evident in the period from 1969 to 2009 (Singh & Siahpush, 2014).

Educational attainment as a defining feature of SES

No discussion of the consequences of socio-economic status, be it individual or that of a population, can be complete without addressing educational attainment. The causal effect of individual education on health and mortality has been previously established, albeit with calls for exploration of detailed mechanisms through which the former operates on the latter (Lleras-Muney, 2005). An additional year of education at age 35 is expected to raise one's longevity by as much as 1.7 years (Lleras-Muney, 2005). Generally, more recent evidence amplifies the argument made in the prior literature that low educational attainment (e.g., less than high school diploma) is particularly characteristic of states with elevated mortality (Montez et al., 2019). The association between the proportion of low-educated adults and higher mortality across the states has strengthened considerably since 1980, though it moved in the opposite direction for those with at least a bachelor's degree (Graetz & Elo, 2022; Montez et al., 2019). A variety of intervening factors need to be mentioned in order to explain the evolving differentials by educational background. Sheehan et al. (2018) elaborate on the shifting functional forms of the causal association between educational attainment and mortality that vary in the regional setting among men and women. These forms dependent in large part on contextual factors, such as job opportunities, healthcare access, and policy landscape. Additionally, states of the US South with large African American populations might follow a distinct causal mechanism between education and mortality that has been rooted in credentialism (Hayward et al., 2015). Such a pattern contrasts that of garnering benefits of human capital resulting from higher levels of educational attainment, followed by a shift toward a more human-capital based returns from schooling in the first decade of the 21st century (Hayward et al., 2015). In other words, in the late 20th century, areas with low levels of educational attainment and high mortality benefited most from the population earning degrees, but more recent trend changes demonstrate a shift toward the human capital functional form, traditionally characteristic of advantaged non-Hispanic White populations. These factors follow in step with the negative selection of individuals into education. Lower proportion of adults without a high school degree suggests these people are already facing unfavorable conditions with regard to their income and living standards, adding to the backdrop of factors painting the context in an area (Montez et al., 2019). In this regard, there has been evidence to suggest that education and employment opportunities could be mediated by the physical and social environment. The quality of housing, such as the use of asbestos and lead in construction, overcrowding of the living quarters, mismanagement and neglect of rental housing by the landlords, and lack of local opportunities for recreation and stress abatement are all significant contributing factors to poor health and the lack of safe environment for education (Mueller & Tighe, 2007). The cumulative disadvantage thus leads to poor chances of employment in industries with lower occupational hazards and higher incomes (Mueller & Tighe, 2007). It is not difficult to imagine how aspects of social isolation induced by patterns of residential segregation would create barriers for professional development and diminish returns of human capital gained through schooling (Collins & Williams, 1999).

Access to healthcare

Education also plays an important role in raising awareness of various risk factors and ways of avoiding them. Individuals with an increased perception and a better understanding of risks involved in their daily activities are more likely to be proactive toward their health and more likely to have the financial means to do so. Indeed, prior to the adoption of the Affordable Care Act (ACA), uninsured adults and children were about 4 times as likely to not see a physician in at least a year as their counterparts with public or private coverage (Hoffman & Paradise, 2008). Access to healthcare through a health insurance subscription is extremely important to managing chronic conditions, the likes of diabetes and neurological disorders. Access to health insurance among marginalized populations is particularly instrumental in increasing their inpatient, outpatient, and emergency healthcare utilization (Farrell & Gottlieb, 2020).

Impact of government policy on health and mortality

Lastly, I review the research evidence illuminating the capacity of governments to improve the state of health in the population through legislation and health initiatives. To be rendered both effective and efficient policy interventions ought to be sensitive to the context and to the attributes of target populations (Cummins et al., 2007). A county-level examination of health and longevity disparities shows that in order to unpack the root causes of poor health and mortality inequalities, particularly along the ethno-racial lines, local data should be prioritized for analyses recommending meaningful policy interventions (Dwyer-Lindgren et al., 2022). Localized clustering of certain diseases and outbreaks in various populations informs policy responses and tends to explain the variation in all-cause mortality across the regions (Roth et al., 2017). Policies facilitating the development of compact cities provide better and more equitable access to transportation and services, improve walkability and use of bicycles as the primary means of transport, reduce traffic injuries, and non-communicable diseases (Giles-Corti et al., 2016). Regulatory responses have also been instrumental in alleviating the consequences of residential segregation on infant mortality and a range of other social and economic ills in the disadvantaged communities of the US South (Almond et al., 2006). Region-specific determinants of mortality are thus ideal points of intervention via geographically localized health initiatives (Dobis et al., 2020).

The first mechanism by which local governments may tackle specific health issues is through health spending and targeted programs. The reverse effect of inadequate social policies is documented by Beckfield and Bambra (2016) who find an association between mortality disadvantage and conservative healthcare spending by states. Moreover, studies attribute the observed ethno-racial disparities in mortality across state lines to local health spending differentials (Grembowski et al., 2010). Some of the most effective initiatives in mitigating mortality from opioid overdose were launched by local health departments by providing public awareness and education on topics surrounding the drug use, as well as stocking naloxone to reverse the fatal effects of overdoses (Feuerstein-Simon et al., 2020). More globally, the passage of the federal Patient Protection and Affordable Care Act (H.R.3590 - 111th Congress (2009-2010), 2010) has been arguably the most influential single piece of health legislation since the federal adoption of Medicare and Medicaid Act in 1965. However, when

it comes to the former, the responsibility for managing the tools for expansion of health insurance eligibility and benefits fell on states with contested political views on the reform and its components. As a result, the intended effect of ACA and its ramifications for population health remain variable along the state boundaries.

Another determinant of policy success or shortcoming is embedded in the support of local governments and the citizenry toward various health-related initiatives. An ideology of a population transcends the political fabric of its state legislature by virtue of electoral choice (Berry et al., 1998, 2010). Voting population's age and generational belonging, with all of its idiosyncratic experiences as a cohort, are deterministic of the population views on public policy decisions, including health reforms (Fisher, 2020). Self-rated health and the level of general health is a useful metric in gauging the demand for intervention for researchers and policymakers alike (Lee, 2014). In turn, a population's perception of its level of health can apply pressure toward health reform through ideological responses to government policy agenda (Oliver, 2006). As a consequence, population views on health issues often find reflection in laws that local and federal governments adopt and in official opinions voiced by state leaders (Quadagno, 1989). The recent coronavirus pandemic is only one of perhaps the most salient, if not extreme instances of politicization of a government response to an urgent health crisis (Adolph et al., 2021). Nevertheless, while some health policy decisions are ideologically charged (e.g., abortion laws) (Carroll, 2017), the other could be a product of local physical environmental conditions and challenges that arise due to incompatibility of population lifestyle and the intended outcome. For example, for many US residents driving is a necessity. Combined with sedentary lifestyle and reduced dependence on manual/blue collar jobs driving becomes a cause of reduced physical mobility among many populations living outside the metropolitan regions of the North-East. Public health policies promoting physical activity have been implemented in various places, albeit those struggle to offset the prevalence in chronic disease risks (Haskell et al., 2009). A great deal of public health policy work revolves around urban planning (Corburn, 2004). The vast majority of the US population lives in urban centers benefiting from improvements to city infrastructure, sanitation and living spaces, as these have a profound impact on population health, much like the century-old improvements that opened a new chapter in the epidemiologic transition in the US (Cutler et al., 2006).

In the next chapter I condense the reviewed evidence from the past research and convert it into a structural causal modeling framework, using a method developed by Pearl (2009). The output of this exercise will be a set of structural equations identifying the causal effect of state health regulation on mortality. These conceptual causal paradigms are quantitatively testable and will serve as inputs to inform the mathematical models in chapter 4.

1.4 Summary

The overall increase in longevity over the past four decades in the United States conceals important patterns and associations between the sub-national determinants of mortality. Though still prevalent, the causes of death have been gradually shifting away from cardiovascular diseases toward a growing fraction of obesity, cancers, and degenerative diseases associated with old-age survival. Overall, African American-White and female-male gaps

have been shrinking owing to higher rate of improvement in African American and male survival relative to non-Hispanic Whites and females, respectively. At the same time, regional differences have increased within most ethno-racial and sex categories, especially when examined across multiple SES attributes. In the more recent years localized outbreaks of suicide, opioid, alcohol and substance abuse within the middle-aged segment of the population have drawn more research attention to compositional and socio-economic aspects of mortality, linked to socio-economic decline. Given that these specific factors are placed within local geography, it is reasonable that area-level geographic disparities in mortality expanded in step with the disappearance of traditional blue collar occupations, with urban infrastructure decline, residential segregation, and living environment degradation. At the same time, political and health reform components of mortality have been examined to a much more limited degree. State governments are relied upon to ensure provision of access to healthcare, safety, and nutrition, as well as to regulate the causes of population health decline and mortality. However, the causal mechanism by which local health policy feeds into the system of associations between deprivation factors and mortality is not well articulated. The evidence gathered from this literature review serves as the stepping stone for the construction of a conceptual causal paradigm in which state health policy contributes to population-level mortality outcomes and its disparities.

Chapter 2

Uncovering Traces of State Politics in Death: A Conceptual Model

The level of population mortality is in large part a consequence of many of biological, social and economic processes that form a complex web of contextual interrelationships. As demonstrated by the outcome of the previous chapter, government health policy is a key institutional factor leveraging authority and resources to address pervasive issues in population health. Ultimately, the effects of legislative initiatives precipitate into potential health improvements, conditional on characteristics of the target population. In this chapter, I rely on the evidence from the reviewed prior studies to construct a testable framework with which one could estimate the impact of state health policy (SHP) on area-level mortality outcomes. I preface the main discussion by highlighting several important distinctions and considerations surrounding concepts to be later used in the construction of a functional paradigm. Next, I briefly review the foundational principles of structural causal modeling. I then restate some of the key findings from the literature on the relationships between various factors reviewed in the previous chapter, including mortality and health policy, thus grounding my theoretical modeling decisions in empirical evidence. The intended result of the following sections is one or more directed arrow graphs relating the concepts from the literature to the relationship between state health policy and mortality. Each graph is accompanied by a set of structural equations representing directional associations. Finally, I discuss the assumptions behind my representation of the relationships in a pair of structural models. I explain how my assumptions may or may not undermine the validity of the identified causal SHP-mortality association.

A model for an aggregate

Approaching the identification of the causal effect between health regulation and mortality requires several key distinctions to be made. First, the mechanism for individual pathway will likely differ from the population level pathways (Huie, 2001). Moreover, for the latter the scale of aggregation dictates which elements of a causal diagram would be sufficient for identification of the effect: some may become stronger and some may attenuate to the point where the existence of a path between the nodes is rendered obsolete. Each scenario would have ramifications for modeling of the target relationship. For example, access to

healthcare, as measured by the number of health professionals per capita, in distinct urban or rural neighborhoods within a county is a relevant variable confounding the relationship between injury or disease and mortality. However, when a population is grouped by larger administrative areas, such as states, local particularities dissolve in a state average. Albeit a theoretically pertinent determinant of mortality the access to healthcare may need to be operationalized differently at the level of a neighborhood, as compared to that of a county or a state. Second, the measurement of mortality in data where individuals are units of analysis could take form of time to death, odds, relative risks, and probabilities of dying of certain cause or before reaching a certain age. On the other hand, given that the aggregate units of analysis contain a mixture of population groups, a more appropriate measure of mortality is a mortality ratio or rate, typically standardized by age to account for the population age structure and related heterogeneous hazards. Silcocks et al. (2001) and Congdon (2009) also advocate for the use of life expectancy at birth ($e(0)$) as an alternative indicator of mortality particularly due to its interpretability and relevance to policy work. For the purposes of identifying the causal impact of state health policies on mortality I treat the two interchangeably. At the modeling stage in chapter 4 I will test the two types of indicators separately.

State health policy impact on period and cohort outcomes

When examining individual outcomes, a person may die at a particular age of a specific primary cause, conditional their exposure to a unique set of risks, environmental conditions, biological or genetic factors and so forth. A population on an aggregate level is evolving over time and is exposed to a different set of conditions than its predecessor, or period effects. In this manner individuals are experiencing a set of health and behavioral risks over time, specific to their birth cohort and their distinct life experiences. Populations are dynamic and contain an ever changing mix of individuals of various ages, sexes, walks of life and migration backgrounds. Thus, populations are more subject to period effects that impact the period-specific prevalence of a wide range of causes of death. When considered in the context of state health policy effects, individuals and birth cohorts are experiencing exposure to policy beginning at a certain age. This implies that the effect of a policy, such as a ban on smoking in public spaces, would have a variable impact on their survival chances. For somebody in their teens a recently passed health regulation may prevent their exposure to smoking early on, whereas a person in their 60s may not benefit from this policy as much, if they lived most of their lives exposed to tobacco smoke. Therefore, for a population, one way to look at a given policy or a set of policies might be through a temporal perspective, where the impact on the aggregate could be traced back to the time of adoption of those policies.

Prelude to linking state health policy with population-level processes

Despite the preponderance of evidence in the public health and epidemiology literature on the substantial role of specific policies impacting health outcomes in certain population groups, there is rarely a direct link between the two. Often policies act on proximal causes of health, but not directly *cause* a disease or its absence. For instance, the recent coronavirus pandemic

saw the introduction of a wide variety of rules and restrictions related to distancing and use of personal protective equipment (PPE) to curb the spread of the virus (Rauscher & Burns, 2021). The effectiveness of such policies in preventing the spread of the virus and the onset of disease hinged largely on the degree of public compliance and the availability of PPE. When it comes to mortality, as purposeful and effective a regulation might be, there often exists at least one factor on a causal pathway between a policy and an outcome, namely the presence of a disease or injury itself. The latter may or may not depend on other intervening aspects, such as behavioral risk factors, socio-economic conditions, or access to healthcare. Nonetheless, it is important to make a distinction between the risk factors on which health policies could have a tangible impact and those that exist in isolation from any policy action. For example, assuming full compliance, a smoking ban in indoor public establishments may not take any effect on the smoking behavior in a population that could substitute smoking the same amount of tobacco elsewhere. Then it follows that the risk factor for lung cancer and other health complications is maintained. It might simultaneously limit the second-hand smoke exposure among the non-smokers in public. Consequently, the risk for developing a life threatening disease as a result of the policy implementation follows via distinct pathways. For the purposes of the present analysis, I separate the risk factors into those that are not inherently shaped by the population-level policies and those that are. In other words, the former might be represented by idiosyncratic individual or small-group behaviors and health hazards that are beyond the practical scope of state-level policy to tackle (e.g., one may engage in an uncommon risky behavior that does not reach enough socio-political resonance in a population to draw regulatory attention).

Spatial and temporal dependence in a conceptual model

Prior to constructing a theoretical model, I emphasize several advantageous facets of a spatial and temporal approach in terms of its ability to contextualize predictive components of population mortality. Mortality outcomes are often a result of life course exposures to a variety of health hazards. Not many studies have formed all of these components into a unified system of analysis with the geographic breadth of a nation at the spatial granularity of a county or finer. It is also important to acknowledge the dynamic nature of such associations. As locality changes in composition and character over time, so do the determinants of health. New hazards may emerge simply in relation to the consequences of an aging population, thus changing the causes of certain health conditions and disease susceptibility. Some measures must therefore be time-lagged in order to fully capture the delayed effects of historical exposure. Health policy is one such key determinant, as it partly changes in response to the unmet needs and grievances of the population, albeit often taking time to attain the desired goals. The strength and directionality of health policy effects are dynamic and require examination through a longitudinal lens.

The nature of local policy is such that it concerns the health and mortality consequences of institutional actions beyond the immediate administrative borders. Tracing this cross-border impact and its interplay with place-specific compositional and contextual characteristics requires situating a proposed theoretical framework in geographic space. The key idea behind this approach is the interdependence of regional attributes due to adjacency and proximity, potentially resulting in spatial spillovers. Taking a hypothetical example of a tax

increase on tobacco products in one state, the residents of that state may choose to cross borders to purchase or to smoke tobacco products in the nearby areas. Alternatively, the residents from nearby states that previously traveled to the state in question for cheaper tobacco may now smoke less as they can no longer afford to purchase the same amount of goods. These two simplistic examples illustrate only a small fraction of potential spillover or containment effects that could take place within a spatial context.

Furthermore, the mechanism by which state health policy affects mortality may be sensitive to an area type or particular spatial support in the data. Policy impacts percolate differently through layers of administrative geography down to individual residents of an area. Aggregate outcomes in a sub-state geography may change depending on its local characteristics. To take matters to an extreme to illustrate the point, suppose one county has a highly skewed sex ratio in favor of men (e.g., coal mining area) within a state that recently banned abortion. Then mortality outcomes in the area would not be the direct result of the recent policy change. In this case a theoretical paradigm ought to be robust in describing the process at various scales of aggregation. Such models need to be subjected to spatial comparisons in order to assess the magnitude and sensitivity of health impacts on different geographic scales. To this end, I design the following framework with the spatial analysis across state and county boundaries in mind. It is crucial to note that causal pathways identified here would likely significantly differ for more granular spatial units.

In the following section, I review the foundational aspects of structural causal modeling. I then use this theoretical modeling framework to formalize the reviewed evidence from the literature on interrelationships between various socio-economic, political and demographic factors. These factors enter the model insofar as their impacts are deemed relevant to explaining the impact of state health policy on sub-national mortality outcomes.

2.1 A brief overview of causal inference using structural causal models

To infer the causal effect of state health policy on mortality I follow the technical framework based on the graph theory representation of structural causal modeling proposed by Pearl (2009). In the present section I provide a high-level overview and the pertinent details of the mechanics behind the framework. I enthusiastically refer the interested readers to Huntington-Klein (2022) and Pearl et al. (2016) for a solid but approachable introduction, featuring finer technical details and proofs. At the core of the approach is a graph consisting of nodes (random variables in a model) and edges (directed or undirected links) between them. The degree to which any two nodes are non-trivially associated with one another can be represented by an edge between them. What makes such graphical system useful is its ability to succinctly represent complex mathematical expressions in a systematic graphical form governed by a set of rules. A graph is often centered around a pair of elements of interest, a treatment or exposure and an outcome, with edges connecting the former to the latter either directly or through a series of chains and forks. Other nodes that impact or mediate the relationship between the treatment and the outcome may be included in the graph with edges linking one or more intermediary components. A chained collection of

nodes between a treatment and an outcome form paths. A presence or an absence of an edge between any two nodes is empirically testable, as is its (in)dependence. Inferring causality becomes possible when such a graph contains directed edges/arrows, informed by empirical research or theory, and no path that can be traced back to the original (parent or ancestor) node. These features thus render a graph directed and acyclic (see Figure 2.1(a)). For this reason it is henceforth referred to as *directed acyclic graph* (or DAG). The nodes are categorized into ancestors and descendants (equivalently, parents and children) in relation to each other. Like water in pipes, an effect is "flowing" through directed edges from ancestors to descendants and may or may not be split and diverted away to other connected nodes.

There are further important functional components to consider. Supposing there is a variable Z on the path in a relationship between otherwise independent nodes X and Y. As in Figure 2.1(b), if Z causes both X and Y, then the effect of X on Y is deemed non-existent or confounded by Z and cannot be measured unless Z is controlled/adjusted for. When this happens, it is said that X and Y are independent, conditional on Z. In case a direct causal link also exists between X and Y, within the graph theory framework a path from X to Y through Z ($X \leftarrow Z \rightarrow Y$) is called a back-door path, because the total effect of X on Y cannot be identified as some of it can be due to the variation in Z. Adjusting for Z in a regression model (here, adjustment is represented by a box) would effectively close off the back-door path and allow for the identification of the causal relationship between X and Y. It is said that X causes Y conditional on Z. Conversely, a front-door criterion is such when a variable Z is on the path from X to Y, but the causality is sequential from X to Z and from Z to Y, while Z intercepts the entirety of the direct effect on the path from X to Y. Examining panel (c) of Figure 2.1, controlling for a variable on the front-door path would close it and the effect of X on Y will become either biased (in case there is a back-door path containing the node W) or completely unidentifiable, if the entirety of the impact is contained within such a path. Another graph feature to consider in a DAG is the presence of colliders. If a path contains a collider Z, as shown in Figure 2.1(d), a type of node into which arrows on a particular path $X \rightarrow Y$ point, with none pointing outward, then conditioning on such a variable or any of its descendants (W in this case) would induce dependence between the pair of marginally independent nodes X and Y on a path where collider intervenes. This may introduce confounding bias, even when this node is present only on one of multiple paths that carry the effect from treatment to outcome. Figure 2.1(e) illustrates this, whereby the relationship between X and Y becomes biased, when the collider Z is conditioned on, and regardless of the status of A. Taking all of the aforementioned observations into consideration, when a path between a pair of nodes of interest is blocked by another intermediary node or a set of nodes which are either conditioned on or represent a collider that has not been conditioned on, these are termed as directionally separated (or *d*-separated). This concept is useful in assessing a likely (in)dependence between a pair of nodes in the model. Furthermore, to ensure the identification of the causal effect of exposure on outcome an intermediary node or a collection of nodes need to be adjusted to isolate causal paths and to remove sources of confounding and bias. These variables comprise a *minimal adjustment set*, which fulfills that purpose. For instance, a model shown in Figure 2.1(f) show an adjusted confounding set Z that is located on the back-door path from X to Y. Conditioning on Z effectively blocks the path $X \leftarrow Z \rightarrow Y$ and leaves only the variation

from X that affects Y through a front-door intermediate A. In this case, A should not be adjusted for, since it is solely endogenously dependent on X and has no other descendants, except Y.

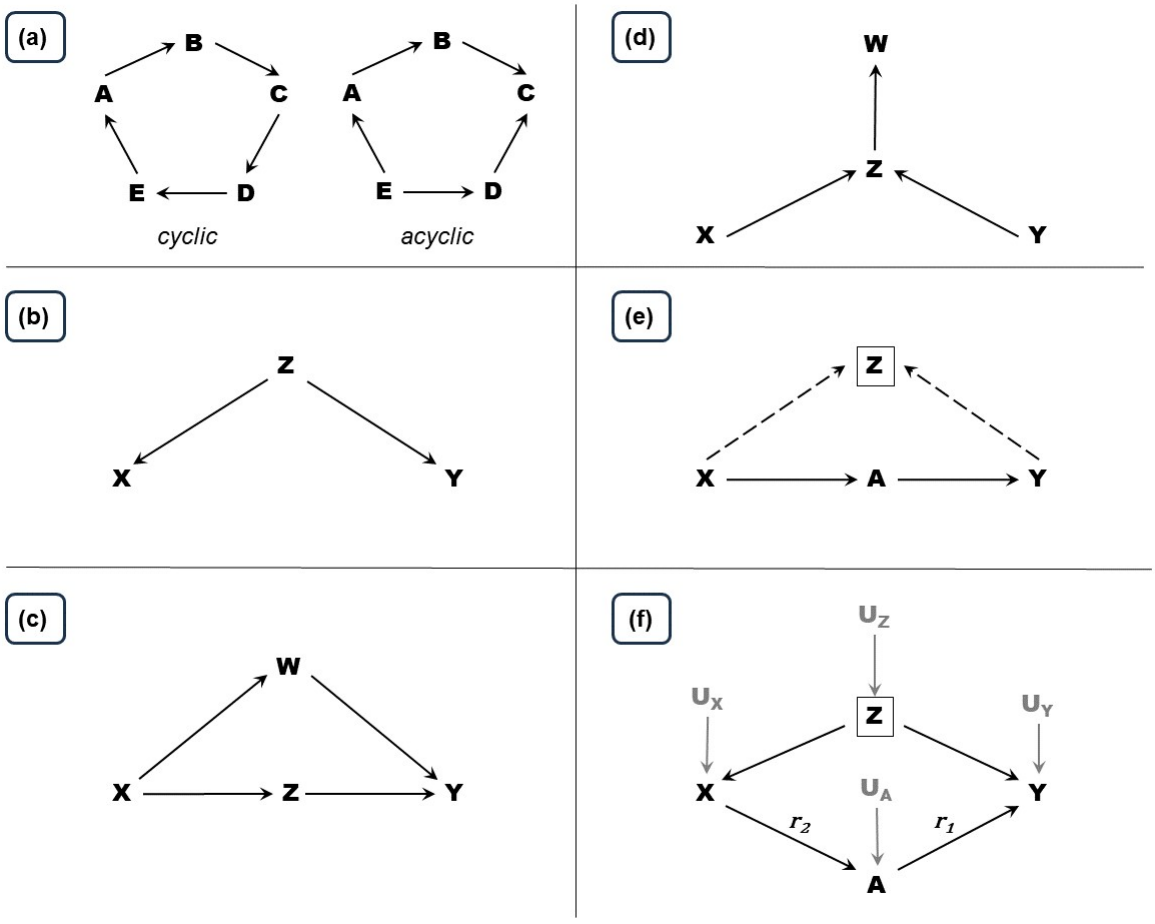


Figure 2.1. Illustrative examples of graphical representations of structural causal models (SCM) using directed acyclic graphs (DAG)

The last feature I would like to address is the presence and the impact of unmeasured components. In practice, few research studies could exhaustively measure or represent every component in a relationship between an exposure/treatment and an outcome. Therefore, all nodes on a graph that could be impacted by additional factors, exogenous to the system, are represented by U with an arrow pointed into the respective measured components (i.e., unmeasured parent nodes). This represents the effect of all the unmeasured heterogeneity through which a given descendant variable in a model is generated. An important consideration in building a DAG is to verify that components are fully or conditionally independent. For U-type components, researchers sometimes make assumptions of independence, akin to the independence of a nuisance parameter from measured variables in a regression model. These assumptions may be strong or weak, depending on a theoretical justification given in a study. In summary, a model represented by a DAG in Figure 2.1(f) demonstrates a properly adjusted model in which the causal effect of X on outcome Y is theoretically identifiable,

provided the model assumptions of independence and that the set of measured specified components is correct and sufficiently exhaustive.

A set of equations below mathematically represent the SCM shown in Figure 2.1(f):

$$V = \{X,\ A,\ Z,\ Y\}, \quad U = \{U_X,\ U_A,\ U_Z,\ U_Y\}, \quad F = \{f(1),\ f(2),\ f(3)\}$$
$$Z = U_Z$$
$$X = f_1(Z,\ U_X)$$
$$A = f_2(X,\ U_A)$$
$$Y = f_3(Z,\ A,\ U_Y)$$

Here the measured variables are gathered in a set V, unmeasured/exogenous ones—in a set U, and in a set of functions F representing the directed edges with structural coefficients r_1 and r_2 in the graph between the endogenous variables. In general, considering a structural relationship in a linear form such equations could be re-expressed in terms of equations with structural coefficients. The conditional probability $P(Y|X,S) = P(Y|S)$, where S is a conditioning set of variables, can be re-expressed as the conditional expectation of the outcome $E[Y|X,S] = E[Y|S]$. This enables the conversion of the structural system into a linear one, which facilitates the approach to numerical estimation of the causal effect using commonplace regression methods. A linear combination of conditional expectations is equivalent to a equation in which slope coefficients are partial regression coefficients.

$$E[Y|X_1 = x_1, X_2 = x_2, ..., X_k = x_k] = a_0 + a_1x_1 + a_2x_2 + ... + a_kx_k$$

However, statistical regression coefficients are not automatically equivalent to the structural coefficients that describe magnitude of pairwise causal associations along each edge on a DAG. To illustrate the causal effect through SCM with structural coefficients all the back door are to be closed first to isolate all the possible ways an exposure can affect an outcome through non-back-door paths. Consider a DAG in Figure 2.1(f) again. Conditioning on Z to remove all impact on X, the causal effect of X on Y can be written linearly, expressing the impact on Y in terms of X:

$$\begin{aligned} Y &= r_1A + U_Y \\ &= r_1(r_2X + U_A) + U_Y \\ &= r_1r_2X + r_1U_A + U_Y \end{aligned}$$

where r_1 and r_2 are structural coefficients related to edges $A \to Y$ and $X \to A$, and U are unmeasured variables pointing to nodes on the path from X to Y. If U terms are not dependent on X, then the total causal effect of one every unit change in X on Y is $\tau = r_1r_2$. Essentially the total causal effect is a product of the two structural coefficients associated with non-back-door edges $A \to Y$ and $X \to A$.

Finally, causal equations need to be translated into statistical ones where structural coefficients are replaced with regression coefficients. As Z has been adjusted for, it simply enters the regression model as a regressor with its own regression coefficient, whereas the total effect τ is represented by the regression coefficient on X. Note that in the regression model, A does not appear, since it was not adjusted for in the structural model, because its effect is fully dependent on X and the exogenous factors. Also, U are not part of the nuisance term ϵ and do not appear in the regression model, as they are absorbed by the respective β coefficients. In short, a statistical linear model representing the total causal effect of X on Y in Figure 2.1(f) can be expressed as:

$$Y = \beta_0 + \beta_1 X + \beta_2 Z + \epsilon$$

2.2 Structural causal model: state health policy impact on sub-national mortality

The body of literature on the associated links between various socio-economic, demographic, and political factors surrounding population health and mortality reviewed in chapter 1 enable the synthesis of a structural causal model describing these relationships, with an express focus on the state health policy as the exposure and mortality as the outcome of interest.

The following summary is an attempt to transform the previous research findings into a testable structural causal model. Beginning with mortality itself, there are multiple quantifiable forms it can assume based on the context of the analysis. Age-adjusted mortality rate (AAMR) is an indicator that captures the effects of the population structure and enables easy comparison across the sample and over time. Alternative metrics could include period life expectancy at birth and at various key ages (e.g., at retirement). Both AAMR and life expectancy could be computed separately for women and men to account for the sex differences in mortality. Regardless of an expression mortality takes, conceptually and inevitably it is linked to a direct cause. This association is illustrated in a DAG shown in Figure 2.2 where a directed edge is established between the injury/disease node and mortality.

Next, I shift focus onto the main exposure of interest, state health policy and its proximal determinants. First, generational views of the population impact the level of ideological and political preferences with ideas that percolate into the ranks of government (Fisher, 2020). A data realization of this process could be represented by an edge establishing a link between an indicator of population age (e.g., median age) and the state government ideology, and separately to health policy. In addition, population age is likely to directly impact the prevalence of various health disorders, disability, injuries and diseases (Chatterji et al., 2015). As a consequence of aging, population age is directly tied to the perceptions of the overall population health and susceptibility of the population to a number of behavioral risk factors. In turn, state government ideology impacts state health policy directly, as politicians holding specific world views would vote to pass a particular kind of legislation. State government ideology could likewise impact mortality indirectly by passing laws that would improve or

undermine socio-economic condition of the population cascading down multiple pathways until it could impact both mortality and heath policy. The latter pathway is denoted by arrows away from state government ideology.

Other central nodes on the DAG are the measures of income and its inequality. As discussed in the literature, income and its unequal distribution are important since it is a product of many constructs, such as one's educational attainment and social standing (Chetty et al., 2016). For many decades prior to the passage of the Patient Protection and Affordable Care Act, income would determine the level of healthcare coverage and the possession of health insurance policies as such (Hoffman & Paradise, 2008). At the same time, education would not only boost income directly, but also empower better educated individuals to be more cognizant and proactive in their health management, perhaps by seeking health resources and prioritizing health insurance acquisition. As noted in the background literature, educational attainment, expressed as the proportion of the population without a high school diploma, is particularly predictive of poor health and elevated mortality (Montez et al., 2019). Low educational attainment triggers mechanisms involving unstable income generation potential, higher occupational hazards (e.g., manual work) as well as exposure or lack of resilience in face of behavioral risk factors (Lleras-Muney, 2005). Returning to income, lower earnings are also associated with many social ills brought about by the dynamics of poverty and residential selection (Massey & Fischer, 2000; Nuru-Jeter & LaVeist, 2011), whereas the urban environment and professional occupations have the tendency to boost the income (Gould, 2007). Median household income and the Gini index of income inequality are two popular summary measures of population income and its distribution, respectively. These indicators represent the concepts of income and its unequal distribution on the DAG. As for the residential segregation, the technical representation is dependent on a variety of mathematical concepts that can be computed. Among the indicators that involve disassociation, dissimilarity, clustering, proportionality and evenness, Dissimilarity (D) and Information Theory (H) indexes are perhaps among the most recognized indicators in the research community (Reardon & Firebaugh, 2002). Recent extensions include features that additionally account for spatial proximity and connectivity (Roberto, 2018). Complementary to residential segregation, the proportion of ethno-racial minorities in an area (typically expressed through a proxy, the proportion of African Americans) has been associated with unfavorable socio-economic outcomes (Block et al., 2004). The context of residential segregation also impacts the infrastructural aspects, notably the quality of housing, physical access to healthcare, and risk factors that are associated with living in an environment with higher violent crime, drug abuse, poverty, food deserts, air and water pollution, etc. (Nuru-Jeter & LaVeist, 2011). All of these are represented by the corresponding directed edges on the DAG. In a similar fashion, denser urban environment impacts the employment, occupation, access to critical services (Corburn, 2004; Hamidi et al., 2018). Given the aforementioned background, Figure 2.2 summarizes the proposed theoretical framework incorporating state health policy and the proximal determinants. Granted, the graph is rather complex as-is. I choose not show the set of corresponding exogenous U_i effects for each node included in the graph. Nonetheless, these elements are non-trivial. I return to the discussion of these unobserved factors in the limitations section.

Crucially, the edge between state health policy and risk factors is absent. In the present

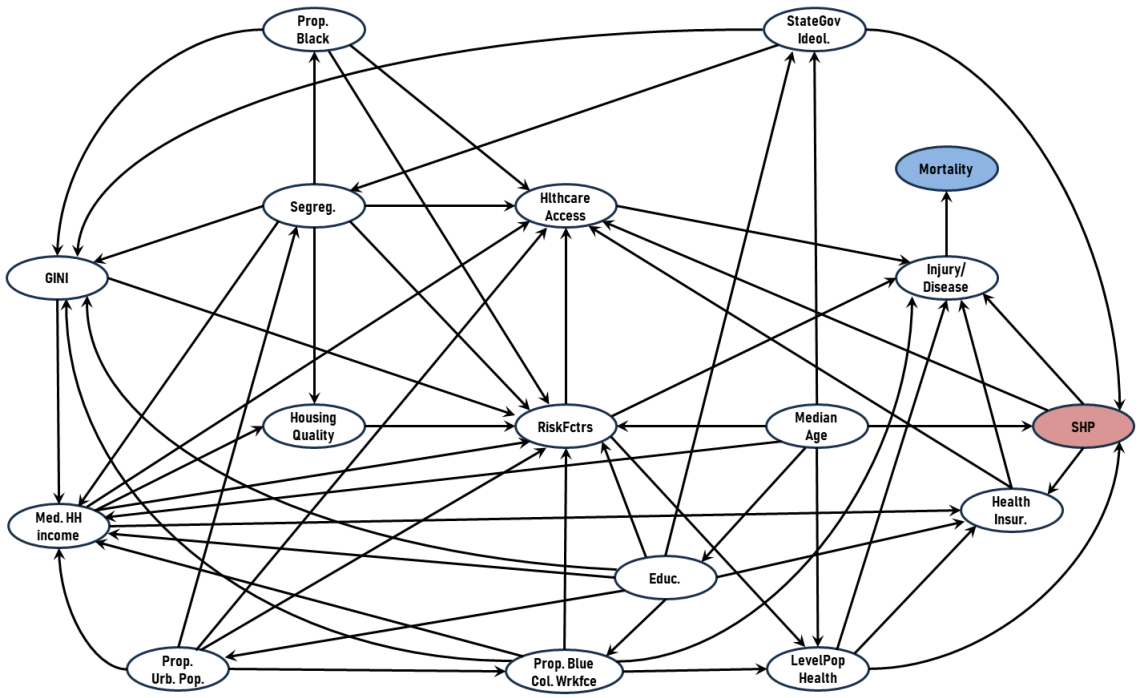


Figure 2.2. Directed arrow graph depicting expected causal relationship between state health policy and sub-national mortality.

analysis regulatory measures are designed to target population-level rather than individual outcomes. Therefore, I argue that state health policy is above all directed toward ensuring access, utilization, and efficiency of health care and enforcement, but not strictly tackling behavioral risk factors among individuals. However, this assumption may come close to being violated in certain instances, such as when ordinances banning smoking in public places are enacted, for example. At the same time, this may not reduce the risk factors associated with smoking, as individuals may avoid smoking in public establishments, but could still compensate with smoking elsewhere. The same could be true of those to whom the policy does not apply (e.g., abortion policies do not apply directly to males), but could have indirect impact via a separate pathway.

As before, the current model expressed in the graphical form can be equivalently expressed mathematically. Furthermore, a range of testable hypotheses of conditional independence of both the treatment (SHP) and the outcome ($Mortality$) from their common confounder variables is possible. In the interim, accepting the presented model as valid, the minimal adjustment set Z for the full model in Figure 2.2 is as follows.

$$Z = \{StateGovIdeol,\ MedianAge,\ LevelPopHealth\}$$

Adjusting for these variables closes off all the back-door paths leading from state health policy (SHP) to $Mortality$. Importantly, $Injury/Disease$ d-separates SHP and $Mortality$. If one

aims to isolating a direct effect, this setup could become an issue had there been an additional direct edge between SHP and $Mortality$ or its exogenous unobserved components U. Since the conceptual model does not envision a direct causal link between SHP and $Mortality$, conditioning on the mediating effects of injury or disease becomes unnecessary in view of the total effect estimation. By similar logic, to estimate the total effect, the $HealthInsur$ and $HlthcareAccess$ variables ought to remain unconditioned on in order to preserve the indirect paths that partially absorb the effect of SHP on the outcome. The model based on a minimally adjusted set of variables is presented in Figure 2.3.

Sometimes the measurement of certain variables is either beyond the capacity or scope of a study, or when the importance of such variables emerges only after data collection had occurred. Fortunately, not every element of the model must be represented in an observed/measured form, so long as it is on an open front-door path between pairs of nodes one such factor separates. For example, examining DAG in Figure 2.2, $RiskFctrs$ mediates relationships between many socio-economic summary measures in the full model and $Injury/Disease$ that feeds into mortality. While it would have been ideal, breaking apart all possible risk factors that may impact age-adjusted mortality rate could dramatically complicate the model. Measuring these risk factors accurately, consistently and with high degree of certainty at the level of a county for all counties in the US over a period of multiple decades would be practically unfeasible, despite the best efforts of the federal government and other agencies (Baker et al., 2013; Dwyer-Lindgren et al., 2017). Although not strictly necessary for the unbiased identification of the total effect of SHP on $Mortality$, adjusting for as many ancestor nodes of $Injury/Disease$ as practically possible will yield a more precise estimator of mortality, so long as $RiskFctrs$ and similar unobserved intermediary variables remain unadjusted for, thus keeping the paths open. This poses no problem for the causal effect identification, as I pursue the estimation of the total impact, not merely the direct or the indirect one.

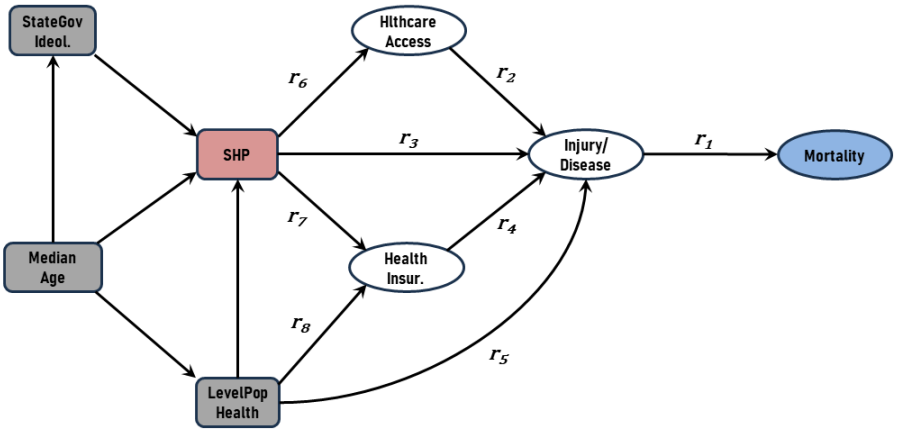


Figure 2.3. Reduced directed arrow graph depicting expected causal diagram between state health policy and sub-national mortality, conditional on the minimal adjustment set (variables shaded in grey).

Note: grey shading denotes an observed variable, unshaded nodes indicate unmeasured variables; a box around a variable represents adjustment or conditioning on that variable.

Similarly to the illustrative approach presented in section 2.1, the causal structural equations that comprise the minimal adjustment set can be combined to express the total causal effect of state health policy on mortality. The conditioning set Z from above serves the purpose of isolating SHP as the progenitor of the causal effect. Unpacking of the equation begins with the most proximal nodes to the outcome, for traceability.

$$
\begin{aligned}
Mortality &= r_1 Injury/Disease + U_{Mortality} \\
&= r_1[r_2 HlthcareAccess + r_3 SHP + r_4 HealthInsur + r_5 LvlPopHealth] \\
&\quad + r_1 U_{Injury/Disease} + U_{Mortality} \\
&= r_1[r_2 r_6 SHP + r_3 SHP + r_4(r_7 SHP + r_8 LvlPopHealth) + r_5 LvlPopHealth] \\
&\quad + r_4 U_{HealthInsur} + r_2 U_{HlthcareAccess} + r_1 U_{Injury/Disease} + U_{Mortality} \\
&= r_1[(r_2 r_6 + r_3 + r_4 r_7) SHP + (r_4 r_8 + r_5) LvlPopHealth] \\
&\quad + r_4 U_{HealthInsur} + r_2 U_{HlthcareAccess} + r_1 U_{Injury/Disease} + U_{Mortality}
\end{aligned}
$$

As a result, the total effect of SHP on $Mortality$ clearly traced on a DAG, in structural equation terms becomes $\tau = r_1(r_2 r_6 + r_3 + r_4 r_7)$. Of note, $Injury/Disease$, $HealthInsur$ and $HlthcareAceess$ could remain unmeasured (or simply unadjusted for), so long as the respective U terms are causally unrelated. The sought-after effect will remain identified for as long as the conceptual model is deemed accurate by construction, and will thus remain unbiased in this regard so long as no additional unadjusted edges enter the SHP node and no colliders exist on the front-door path from SHP to $Mortality$. Following the more trivial example in section 2.1, the βSHP regression coefficient represents a linear combination of individual pairwise effects on the path $SHP \rightarrow Mortality$. In statistical terms, the structural model implies the following linear regression setup:

$$Mortality = \beta_0 + \beta_1 SHP + \beta_2 StateGovIdeol + \beta_3 MedianAge + \beta_4 LvlPopHealth + \varepsilon$$

Figure 2.4 introduces an alternative, expanded structural model which respects the aforementioned minimal adjustment set Z. In other words, this model stipulates the inclusion and adjustment of the nodes, such as $Educ$, $MedHHIncome$, and so forth. At the same time, the unobserved intermediate nodes like $HousingQuality$ should remain outside of the adjustment set as long as its impact is dependent on the combination of the parent-of-outcome node effects that the former merely pass along an open path. The added benefit of identifying these expanded adjustment factors beyond the minimal adjustment set is that a model should yield a more precise statistical estimator of mortality, while preserving the identification of the causal impact in question. The expanded regression model based on Figure 2.4 becomes:

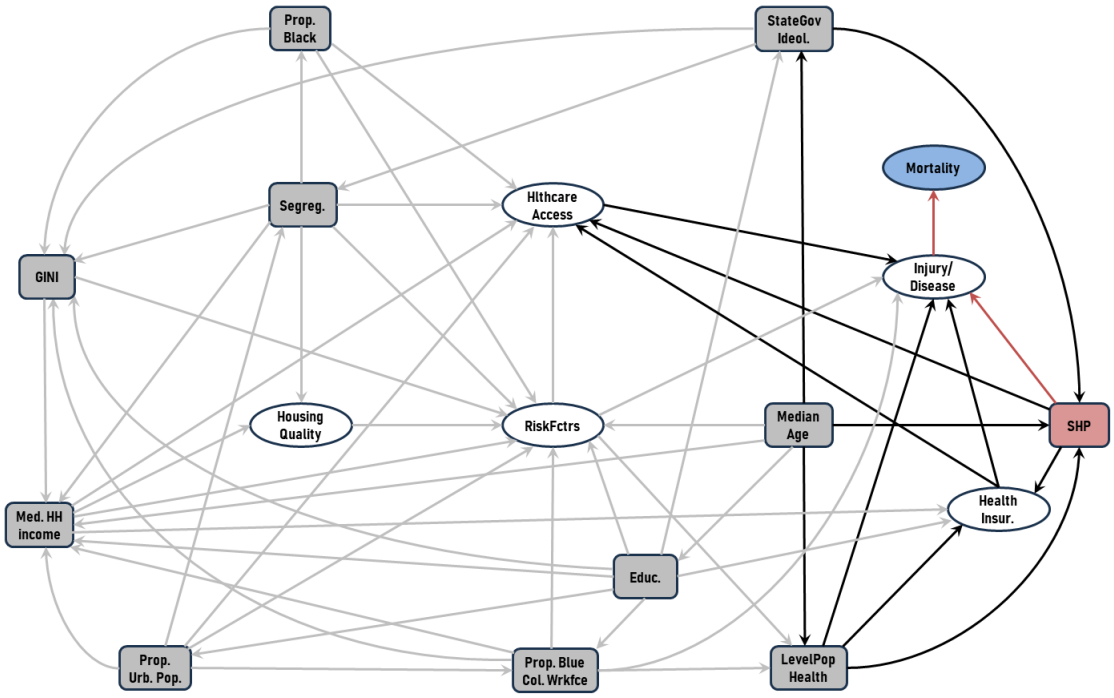


Figure 2.4. Expanded and adjusted directed arrow graph depicting expected causal diagram between state health policy and sub-national mortality.

Note: grey shading denotes observed variables, unshaded nodes indicate unmeasured variables; a box around a variable represents adjustment or conditioning on that variable. Grey arrows denote non-key relationships in identification of the SHP-mortality effect. Structural coefficients and unmeasured exogenous influences U are hidden for clarity of presentation.

$$
\begin{aligned}
Mortality = \beta_0 &+ \beta_1 SHP + \beta_2 StateGovIdeol + \beta_3 MedianAge + \beta_4 LvlPopHealth \\
&+ \beta_5 PrBlack + \beta_6 Segr + \beta_7 GINI + \beta_8 MedHHIncome + \beta_9 PrUrbPop \\
&+ \beta 10 PrBlueColWrkfce + \beta_{11} Educ + \varepsilon
\end{aligned}
$$

Ultimately, I select the minimal adjustment and the fully adjusted models for further investigation. The operational implementation and the explicit inclusion or isolation of additional parameters in a statistical model stand separate from the causal effect identification and merit its own careful discussion in the forthcoming chapters.

2.3 Structural Causal Model assumptions and limitations

At present stage it is imperative to divert the attention to a number of assumptions that are required to hold in order for the synthesized SCMs to be valid. Indeed, the validity of the relationships depicted as edges in a DAG with respect to a particular real-world data generating process can be tested empirically. For each pair of non-adjacent nodes in the graph an absence of an edge between them is equivalent to claiming their independence from each another, conditional on a set of intermediary nodes that *d*-separates the two. In practice, this can be tested numerically via an ordinary least squares fit in which one node in the pair is a dependent variable and the other is the main independent variable (ordering is not relevant due to pairwise symmetry), with the conditioning set included as controls. An effect that is statistically not different from zero offers a piece of evidence to the DAG discovery. For example, one such implication can be tested, where the value of state health policy is seemingly independent from the educational attainment of the residents, given the level of population health, medium age in the population, and the state government ideology score.

$$
(SHP \perp Educ \mid LvlPopHealth, MedAge, StateGovIdeol)
$$

The violations of this assumption may require alternative model specifications via the introduction of instrument variables. If there is a suspected bi-directional relational arc between unmeasured variables U of exposure and outcome, the estimated effect between two would become biased. In such a scenario there could exist a variable Z that is 1) causally related to the exposure and is not directly related to the outcome, 2) Z causes the outcome only through its causal effect on the exposure (condition also known as an *exclusion restriction*), and 3) Z is not involved in any confounding between itself and the outcome. A presence of such an instrument allows to enforce the causal effect identification in the presence of confounding and measurement error. In observational studies utilizing secondary data, or generally outside of a randomized control trial experiment setting where treatment/exposure assignment is predetermined, finding a valid instrument is a challenging task as most factors are not selectively unrelated. In the present case, an instrument such as

state legislative agenda or its proxy might theoretically fulfill this requirement. Additionally it must be conditional on the government ideology and level of population health, both of which are nodes in the minimal adjustment set presented above. Figure 2.5 demonstrates a potential way an instrument based on legislative agenda may aid in the effect identification of state health policy on mortality. In this specific case, legislative agenda could be influenced by the unmet need for health reform in the population (as determined by the population level of health) and the government ideology in the state legislative body. Importantly for this mechanism, the agenda does not induce confounding between the adopted state health policy and mortality. It is also theoretically uncorrelated with the unmeasured U terms of the exposure and the outcome. Further necessity and validity of some such instruments beyond the conceptual stage is to be empirically tested, while the effect is to be estimated through a two-stage least squares (2SLS) fitting (Huntington-Klein, 2022).

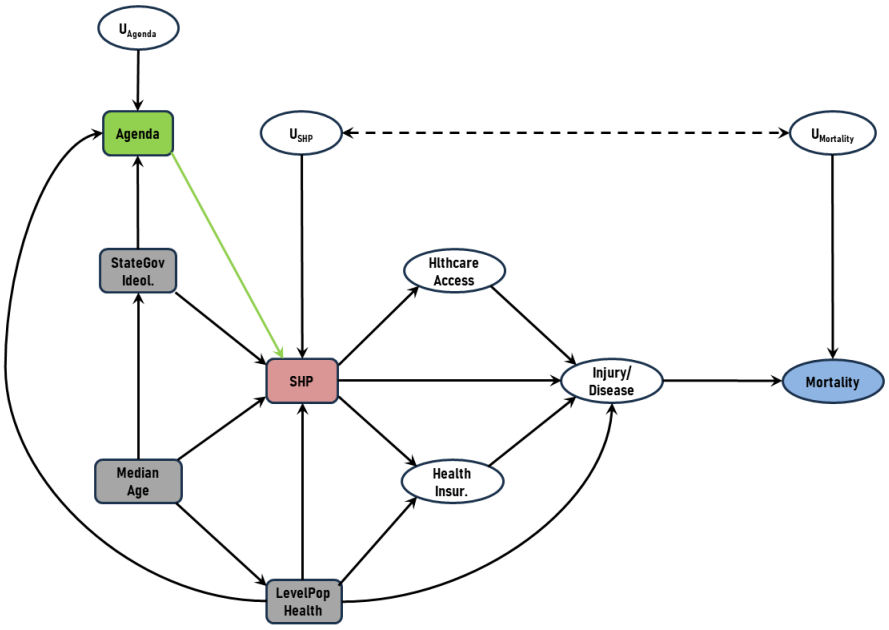


Figure 2.5. Instrumental variable analysis using directed arrow graph, estimating expected causal effect of state health policy on sub-national mortality.

Note: grey shading denotes observed variables, unshaded nodes indicate unmeasured variables; green shading represents an instrumental variable; a box around a variable represents adjustment or conditioning on that variable. Non-central structural coefficients and unmeasured exogenous influences U are hidden for clarity of presentation.

Another important limitation of both minimal and fully adjusted conceptual models, as presently described, is the applicability of the impact of certain effects. Among these health policy is one point of leverage, associated with residence in specific states. There is evidence to suggest that the state of residence is more important than the state of birth (Montez et al., 2019). However, the migration flows over a lifetime of a population are neither easy nor straightforward to measure, particularly with regard to evaluating cohort and period exposures to hazards and health policy effects at various times. Selective migration could alter composition of areal units, thus changing its population attributes used in the study.

For the purposes of this analysis I treat state populations as geographically static. This is a reasonable, albeit a nontrivial assumption. It is relevant if the window of potential exposure to both location-specific hazards and state health policy effects is extended over several decades during which small portions of the population would have changed states of residence.

The above idea extends to the broader notion of selection. It could be true that healthier populations end up in places that are also more health-promoting. I argue that the matter of selection is locally relevant, and greatly dependent on the scale of geographic aggregation. Regarding state populations as units of analysis, I argue that it is not likely that patterns of health selection *within* each unit would critically undermine the results of an area-level analysis. Where such patterns exist in each state, I expect a comparable mix of the baseline "healthy" people making up the state-level statistic. It is well known that better educated individuals are more likely to concentrate in urban centers, draw higher wages, and as a result lead healthier and safer lifestyles (Gould, 2007). However, this aspect of selection is weakly dependent on SHP that occurs at the state level, and not in specific areas where healthier populations congregate for other reasons than state health regulation. A greater danger is posed by interstate migration. In such cases, the selection of healthier individuals into healthier-promoting places ought to be insignificant for the SCM model to hold. It is important to be wary of interpreting the results of quantitative models, when considering isolated states and counties within those states. The issue of health selection is not easy to control for using aggregate data from secondary sources. In practice, given knowledge of the prevalence of health selection in various places, inverse probability weighting designs could be applied to address this issue. Alternatively, fixed effects regression designs could partially remedy this issue by controlling for it as part of idiosyncratic but unobserved process, assuming such and effect is temporally "fixed" and is linearly associated with the main outcome (i.e., mortality) across all units of analysis.

An important set of assumptions that underpin the pairwise associations described in DAGs in Figures 2.3 and 2.4 is related to timing of the events. Certain relationships imply temporal directionality. For example, the direction of the arrow from median age to poor health necessarily implies that a population needs to turn older on average in order to lead to higher prevalence of poor health. Likewise, a particular type of state health policy may be a result of a vote determined by a threshold representation in a state government with particular ideological views. The presence of a conservative majority in state government precedes a likely vote to ban abortion access for women, for instance. But not all population health matters are partisan. A traffic safety legislation may be supported by no particular government and at no particular time. In this sense, election of a majority of politicians with polar views on reproductive rights does not cause them to vote for or against traffic safety measures. The latter could be passed before or after certain politicians enter office. Thus, when multiple events (e.g., votes on the floor of a state assembly) are deemed to determine an outcome (e.g., a collection of health policies), it is not always the matter of a deterministic outcome, represented by an arrow. Instead, the arrows in a DAG in Figures 2.3 and 2.4 represent directional probabilities, informed by past research and empirical observations. The assumptions of magnitude and timing behind the DAG nodes are weaker for an analysis of an aggregate than those would be for individuals. In other words, a misattribution of a fraction

of a percent in a population summary indicator does not invalidate the overall relationship between any two variables comprising the causal mechanism. The causal diagram may still be sufficiently robust for an aggregate analysis where exceptions and outliers exist, so long as those can be identified and dealt with separately.

When it comes to the limitations of individual components or nodes in the structural diagram, aspects broadly associated with socio-economic deprivation may introduce conceptual instability into the model. This is due to the fact that the operationalization of various components of deprivation intends to measure its various facets that may or may not be equally applicable to different population segments. For instance, a racially segregated community in one town may not experience deleterious effects of societal isolation, perhaps due to community bonding and resource sharing, as compared to another segregated community in a different town. It may be expected that the effects of deprivation could be further attenuated with the decrease in size of the spatial unit. If any small area is considered resource-deprived in isolation relative to the surrounding areas, then individual residents of such an area may not be deprived as much due to their physical proximity to resources in the surrounding areas. Larger areas, such as states, counties and municipalities are often masking such effects by flattening the underlying heterogeneity. Coarse geographic units presume longer travel times for the socio-economic phenomena under study to be able to spill over, rather than be contained by default. Establishing the structural connections on a local level is therefore important. Parsing the spatial scale and relationships between the units exhibiting a certain phenomenon would reveal a multitude of patterns that nudge one toward a variety of conclusions and implications. While not one scale or size can fit the bill for the purpose, finding consistency across a range of geographic options should enable a proper identification of an effect of a systematic and deep-rooted dynamic, such as resource deprivation. Additional limitations may enter into play with the technical implementation of the model. I address those at each respective stage.

2.4 Summary

This chapter sets up a conceptual framework for assessment of the impact of state health policy on mortality via structural causal modeling approach. The associations and causal pathways between socio-economic, demographic, and political factors and mortality highlighted in the past literature are replaced with directed pathways on a graph. In turn, the directed acyclic graphs are re-expressed as a structural equations. Using the rules of *d*-separation, or declaring conditional independence between sets of graph nodes, as well as techniques of front-door and back-door adjustment I isolate the target relationship. The assumed causal effect of state health policy on mortality is minimally identified through adjustment for 3 auxiliary variables. The SHP-mortality effect identification hinges on population (median) age, political ideology in state government, and prevalence of poor health in a population. In addition to the minimal adjustment set I identify a range of supplementary socio-economic and demographic concepts from the literature to comprise an extended set (a fully adjusted specification). When adjusted for these additional factors the causal identification of SHP-mortality effect remains unaltered. I hypothesize that the use of the fully adjusted set would significantly enhance precision in quantitative models, as compared

to using only the minimal set. I discuss multiple assumptions enabling the two structural causal specifications. I conclude by outlining the limitations that might follow from the application of the conceptual model as presented in this chapter. For a future application, I propose a variation of the minimal adjustment model in the context of an instrumental variable analysis that could validate the proposed relationship.

Chapter 3

A Method for Generating State Health Policy Scores and Rankings

In this chapter I develop a method of representing health policy impact as a summary measure for areal units (i.e., states). Such a measure could be used to gauge the degree of involvement in population health improvement of regulatory institutions in an area. Such a summary measure would no longer be binary in nature in terms of its intended impact in the same manner as some individual pieces of health regulation could be. Instead, a summary indicator of state health policy (SHP) would encompass a certain qualitative component that could be expressed quantitatively and be used to assess the overall degree of positive impact on improving population health and reducing mortality. I henceforth refer to such an attribute of a potential SHP indicator as *health positivity*. Thus, I devote my attention to generating a pair of composite measures for state health policy. All indicators will be used in the following chapter as inputs to models evaluating its impact on mortality within the framework of conventional methods for causal analysis (Huntington-Klein, 2022).

The challenges of this exercise are many, not bounded by the mere fact that qualitative character of any regulation is difficult to assess in quantitative terms. Much of the public health research focuses on the impact of isolated policies in a particular geographic region. This typically limits the scope of study of targeted interventions, unconfounded with a multitude of idiosyncratic factors pertaining to other policies or to other places. The methodology of evaluating such a policy impact is specific to the case to which it is applied. Basu et al. (2017) discuss several such methods, involving difference-in-difference approaches, propensity score and synthetic control for areas without equal comparison, regression discontinuity, and instrumental variable approaches for unmeasured confounding in a single policy context. However, demographic work and social science research generally rely on aggregate group and summary measures to draw conclusions about population outcomes. This requires extension to the approaches discussed by Basu et al. (2017) to accommodate the possibility creating a synthetic measure for policies across multiple geographic sub-populations, for multiple policies that may or may not act on population health and mortality outcomes simultaneously, and/or at different geographic scales (Huie, 2001). Moreover, composite or summary indicators of mortality, such as all-cause age-standardized death rate and life expectancy at various ages, may require similarly encompassing explanatory measures to evaluate how the former

respond to the prevailing health policy context in an area.

3.1 Policy data

To accomplish the stated task I make use of the two sources of state-level policy data. The first is the State Policy & Politics Database (SPPD) (Montez, 2022). It is a compilation of state policy information dating from 1980 to 2021, concerning social safety net, taxation, labor and economy, health and behaviors most relevant to population health. Many policies have been harmonized by similarity of broader intent, as exact wording would likely differ across the states, and coded as binary values of 0 or 1 to indicate whether and when a state adopted a policy of a particular type and topic (e.g., ban on smoking in public places). The second source of data on state policies is State Policy Innovation and Diffusion (SPID) set composed of similar policies with binary coding by year of adoption (Boehmke et al., 2018). The difference between SPID and SPPD is that SPID includes a much wider range of policies that are not selected for inclusion due to its association with topics not relevant to the present analysis. Moreover, SPID data contains a series of 724 policy implementations dating as far back as 1691 and until 2017. While larger than SPPD, SPID data remains non-exhaustive. The two data sets have been compiled with distinct purposes in mind and thus complement one another in terms of the completeness of policy adoption information or the temporal coverage. Lastly, I procure several additional measures, specifically on tobacco legislation, nutrition, physical activity promotion, and obesity from Centers for Disease Control (CDC) (Centers for Disease Control and Prevention, 2023a, 2023b) to cover the gaps in SPPD and SPID data when it comes to highly relevant and detailed policies tackling the causes of major contributors of mortality.

3.2 Combining SPPD, SPID and CDC policy datasets

In this study I combine the sources by further harmonizing the policy coding structure by state and year, extending the historical range of years for SPPD policies that have been adopted pre-1980 and/or introducing the policies of interest from SPID and CDC that have been omitted in SPPD after 1980. I then sort through the policies to (1) eliminate those that overlap across multiple sources; 2) eliminate those regulations that may not be directly relevant to mortality outcomes of a substantial magnitude; and (3) introduce a cutoff to the range of years for policies in all the states that adopted those policies prior to 1970. In other words, for the latter point, if no new state adopted a policy after 1970, it is excluded from the analysis. The motivation for such a manipulation is to reduce the confounding effect of policies that legally overrode any previously adopted ones, while capturing the majority of the immediate and time-lagged impact of the more recent (post-1970 policies). The full list of policies selected for this analysis is presented in the Appendix to Chapter 3. Since the mortality data I use in this thesis is available for a more limited number of years than the policy data, I restrict the analysis of the state health policy impact on mortality to the 1990–2018 range of years. As far as the health regulation data is concerned, the period of 1970–1990 may thus be thought of as a "burn-in" period during which sufficient legislative

activity is accumulated from the 1970 baseline for a meaningful concurrent analysis. The primary purpose of this analysis is not to provide an indisputably exhaustive quantitative impact of the policies on mortality across the US state borders. Rather, the goal is to set a level field on which the cumulative impact of the rate of policy adoption acts as the relative distinguishing factor among the neighboring states with respect to their proactive approaches to population health improvement and mortality reduction (Dwyer-Lindgren et al., 2016; Egen et al., 2017). I categorize the policies by thematic components and by classes or types of legal action. These thematic categories and classes are outlined in Table 3.1.

Before introducing the details of the process of construction health policy score index, a formalization of several key assumptions is in order.

Assumption 1. The number of health policies of a particular thematic group passed by multiple states is cumulative and is non-zero over the long run.

Given this assumption, the relative distribution of the health policy scores reflects the cumulative nature of the policy effect. While some progressive states adopt laws early on, eventually the majority of the states may pass a thematically analogous set of policies. The differences at any given point in time are then due to the differential contemporaneous rate of adoption of such policies (including the states that ultimately never invoke analogous policies).

Assumption 2. The impact of a thematic policy group (i.e., not individual policies) is comparable to the analogous groups in different states and over time.

This assumption stems from the differences in the exact policy wording and implementation in different states. The policy databases use expert knowledge to group and harmonize the laws that had similar wording in state legislation, which alludes to a similar intended impact. I treat such policies as equivalent in the process of the construction of thematic groupings and the health positivity scores based on these groups. Deviations from this assumption would not necessarily invalidate the meaning of the component of these policies in the calculation of the score, but this may under- or overestimate the magnitude of the impact. Classification of these policies into thematic groups and further into policy classes enable criteria that could be used to decrease the leverage of any individual policies in potentially biasing the overall score. On the other hand, this requires that no single policy group addresses health/mortality concerns of disproportional prevalence, so as not to significantly diminish the true contribution of such a group within the composite score.

Assumption 3. The scope of application of individual policies (or single policy groups, as extracted from the secondary data sources) is identical across the thematic group and policy class spectra.

On the surface it appears as a rather strong assumption. Nevertheless, it is a reasonable and a convenient one to make in the absence of a more specific quantitative evidence of the true impact of a range of legislative initiatives that have variable targets and health consequences, conditional on the local demographic and economic context. Without full information, a quantity that could satisfy this assumption would need to be adjusted both

within the thematic group and then by weighting by the number of policies within a class. This would ensure minimization of the researcher bias, such that no policy type is over-represented in the final score. The health policy score is thus conceived of in a similar spirit and manner to the other summary measures that exist on an aggregate level.

Assumption 4. Adopted health policies remain in effect over time, unless explicitly revoked or amended.

It makes sense that major health policy initiatives are enacted for the durable impact on the population health. However, violations of this assumptions are possible when data obscures the fact of policy expiration or revocation. For example, if public mask wearing is implemented in response to an infectious disease, there may not be a phase-out policy or an explicit mention in the database to indicate that the policy has expired. Over time, the core aspect of such initiatives may be attenuated. On the whole, I expect the bias in the overall state health policy score indicator resulting from such lingering policies to be small in relative terms.

Table 3.1. Description of thematic categories and classes of state policies proximal to causes of mortality

Thematic categories	
1	Traffic and road safety, driving, cycling, etc.
2	Weapons, firearms, violent crime
3	Drug and opioid use, prescription regulation, monitoring and dispensation of controlled substances
4	Abortion, reproductive and maternal health
5	Alcohol and tobacco sale and consumption
6	Health insurance, regulatory acts against medical malpractice
7	Nutrition and safety net
8	Preventive health, screenings and tests, public health programs
Classes	
1	Legal regulation, barriers or limited restrictions
2	Fines, taxes, levies
3	Partial bans or a complete ban on an activity or product
4	Government and community health improvement initiatives

3.3 State health policy score index

With the aforementioned assumptions in mind, I use the policy data from SPID, SPDD, and CDC sources to compute a health positivity index. Inspecting the data, assumption 1 is satisfied. There is a sufficient, non-zero number of policies in all thematic categories and policy classes as defined in Table 3.1 by 1980. Assumptions 2 and 4 can be accepted within reason, as most secondary data are qualitatively grouped and harmonized, while no information on policy expiration or revocation is given. However, assumption 3 requires further adjustment to be made in order to minimize the qualitative influence of numerous fragmented policies for a given category topic on the overall score (e.g., many policies on abortion, but too few on safety net). I first propose to calculate an annual thematic policy ratio (R_g) for each thematic group g that takes into an account the relative health positivity of policies on a specific topic that are in effect in a given year and state.

$$R_{gst} = \frac{\sum_{i=1}^{N} p_{igst}}{\underset{s \in S}{\arg\max} \left[\sum_{i=1}^{N} p_{igt} \right]_s} \tag{3.1}$$

where p_i is a policy impact score in a value set $P = \{-1, 0, 1\}$ of N policies denoting an anticipated impact of a given policy i. R is then a relative ratio of health positivity of N policies in a given state s out of maximum possible in any state within a thematic policy category g and in the year of observation t. Further, I expect that different class of policies might have a quantitatively varying impact on mortality. For example, a legal barrier to tobacco sale or its use would likely produce different outcomes from that of a *tax* on tobacco products, or from that of a ban on *advertisement* of tobacco products. Therefore, I additionally group the calculated thematic policy ratios R_g in a given year by calculating its mean weighted by the number of effective policies in a given class, for each state. In the absence of additional information, I assign equal weight to each of the 4 policy classes. To obtain the final health policy score I_{st} for each state and year, the ratio R_{gst} for each thematic group is weighted the second time using the policy class c.

$$I_{st} = \frac{\sum_{c=1}^{C} \sum_{g=1}^{G} R_{gst} N_{cst}}{\sum_{c=1}^{C} N_{cst}} \tag{3.2}$$

A negative value of I represents a policy effect that is deemed to be detrimental to population health and mortality outcomes. A positive value indicates a likely beneficial policy environment. A policy impact value of 0 indicates that a qualitatively ambiguous potential impact. In this form the score I_{st} is additive and decomposable into its weighted thematic components I_{gst}, each of which may have a differential impact on mortality at various geographic resolutions.

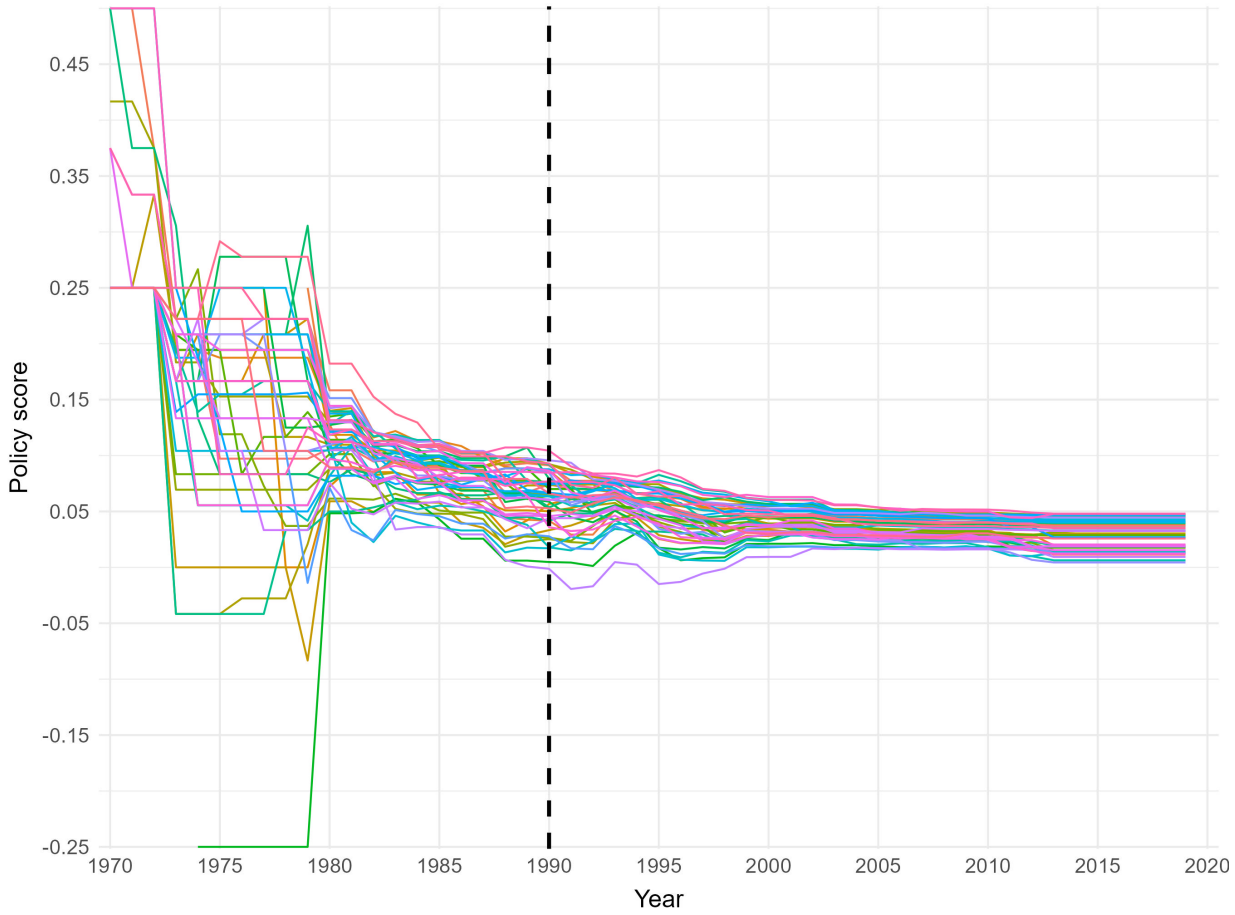


Figure 3.1. State health policy positivity score 1970–2019.

Note: each line represents a state-specific score. Scores shown are based on the assumption that the impact of policies is cumulative over time. Dashed line signifies the intended end of the "burn-in" period.

For instance, as shown in Figure 3.1, a value of $I_{s,1990}$ for any state s in 1990 is the weighted composite positivity score for policies that have been adopted since circa 1970. As a result, there is a seeming convergence of values toward the more recent years due to the fact that many similar policies have been enacted by most states, but at different times. In such a manner, the states are allowed to be subjected to time-lagged policy effects differentially. The visible reduction of variance in Figure 3.1 after 2000 and the eventual flattening of curves after 2013 is the artefact of the data in SPID and SPPD data sets. The flattening could be due to the fact that neither data source contains information about the more recent policies at the time of this analysis. While such omissions are clearly a limitation of the data, the relative distribution of the score at any given time point would nevertheless be useful in distinguishing the most and the least proactive states in terms of its health regulation agenda. Ranking these state-specific scores from the highest value at time t (1=most proactive) to the lowest value (50=least proactive) yields a rank plot shown in Figure 3.2, or an equivalent map showing the geographic distribution and the evolution of the state rankings over time (see Figure 3.3).

3.4 Relative measure of the rate of health policy adoption

To contrast the I score measure, I consider an alternative extension to the indicator of health policy environment, one that relaxes the assumption of cumulativity over time and disregards the assessed health positivity factor. It would be of interest to compare the rate of passage of policies by decade leading up to the point of observation of mortality data panel (i.e. 1990, 2000, 2010, and 2018). A potential strength of this approach is that it is not sensitive to the point of departure, which may be characteristic of the I score, as the year of policy impact accumulation is set to 1970. Figure 3.4 shows the distribution of the number of policies passed in each decade across all states. It is clear that the rate of passage of health regulation is lower in the 2000–2009 and 2010–2018 periods (14.8 and 3.9, respectively), whereas it is comparable for the 1980s and the 1990s decades (18.3 and 19.8, respectively). There is a concern that for the most recent decades the number of policies is lower due to an incomplete decade and omission of many regulations in the SPPD and SPID databases due to recency. However, assuming that omissions are equally probable across all states, the value of an alternative indicator is, once again, in its relative distribution. Thus the indicator of the relative fraction of new policies passed in the prior decade ($RFNPPPD$) could be expressed by Equation 3.3, or as a fraction of newly adopted policies in the decade leading up to a time point, relative to the maximum in any given geographic area during the same period.

$$RFNPPPD_{st} = \frac{\sum_{d=t-1}^{t-10} n_{sd}}{\underset{s \in S}{\arg\max} \left[\sum_{d=t-1}^{t-10} n_d \right]_s} \tag{3.3}$$

where n is a number of health policies in states s and data year t. Due to data sparseness, the

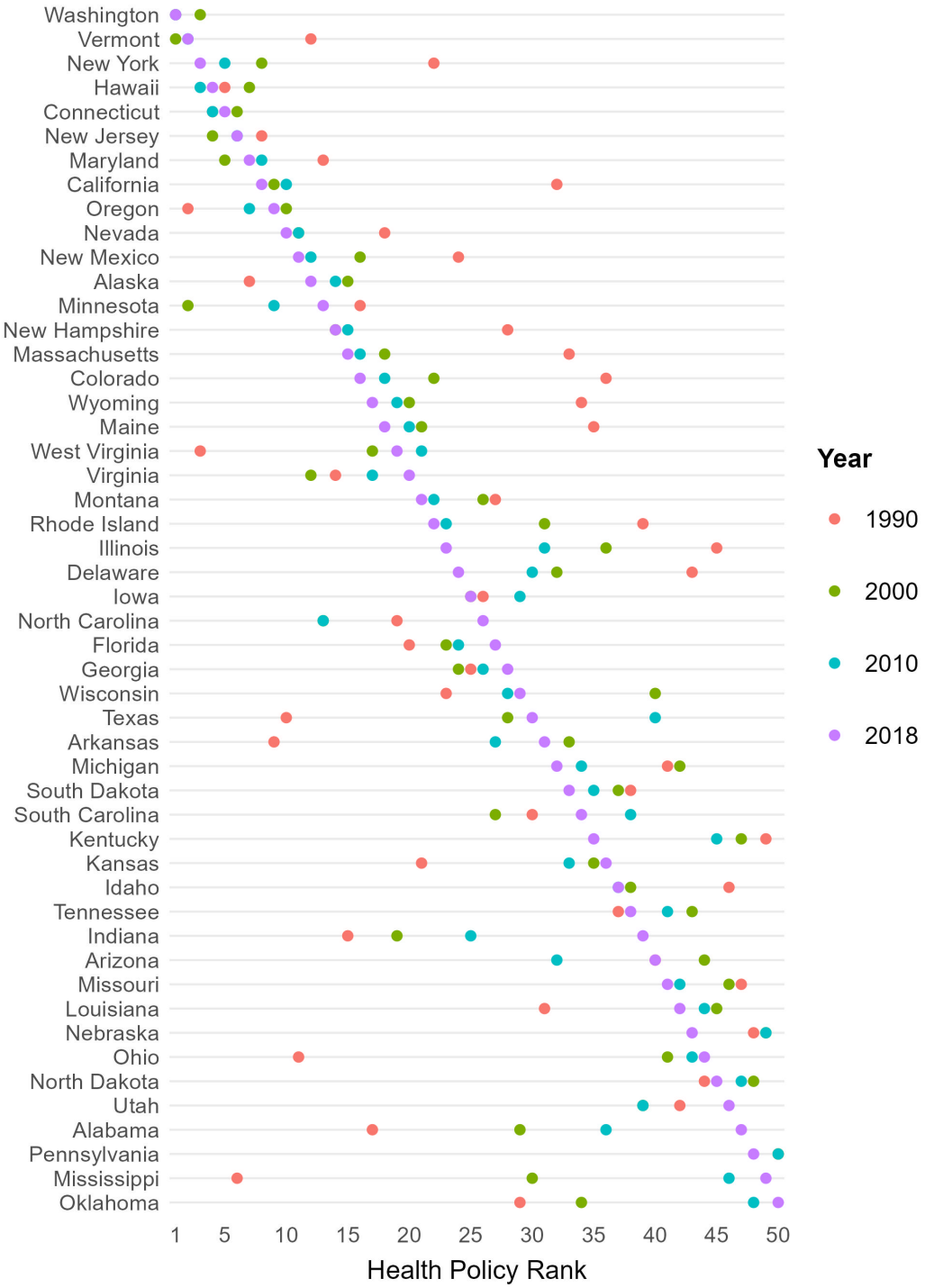


Figure 3.2. State health policy positivity rank for select years 1990–2018, sorted in order of decreasing rank in 2018.

Note: the rank of 1 represents the most health-proactive state and 50—the least health-proactive. Ranks shown are based on the assumption that the impact of policies is cumulative over time.

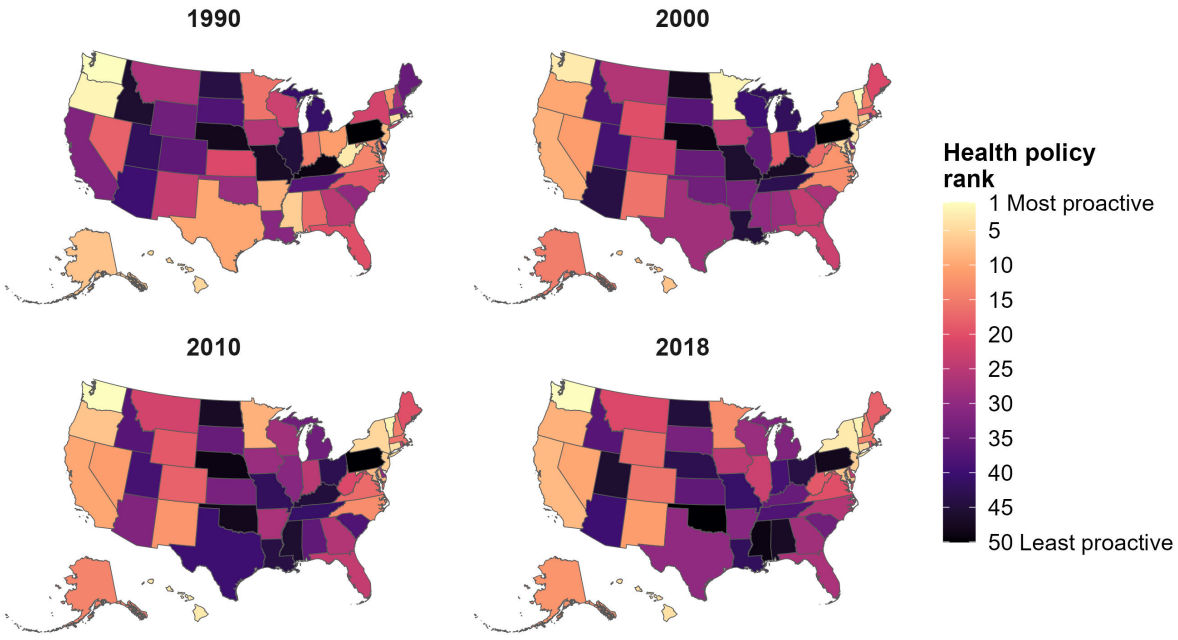


Figure 3.3. Geographic distribution of the state health policy positivity rank for select years 1990–2018.

Note: the rank of 1 represents the most health-proactive state and 50—the least health-proactive. Ranks shown are based on the assumption that the impact of policies is cumulative over time.

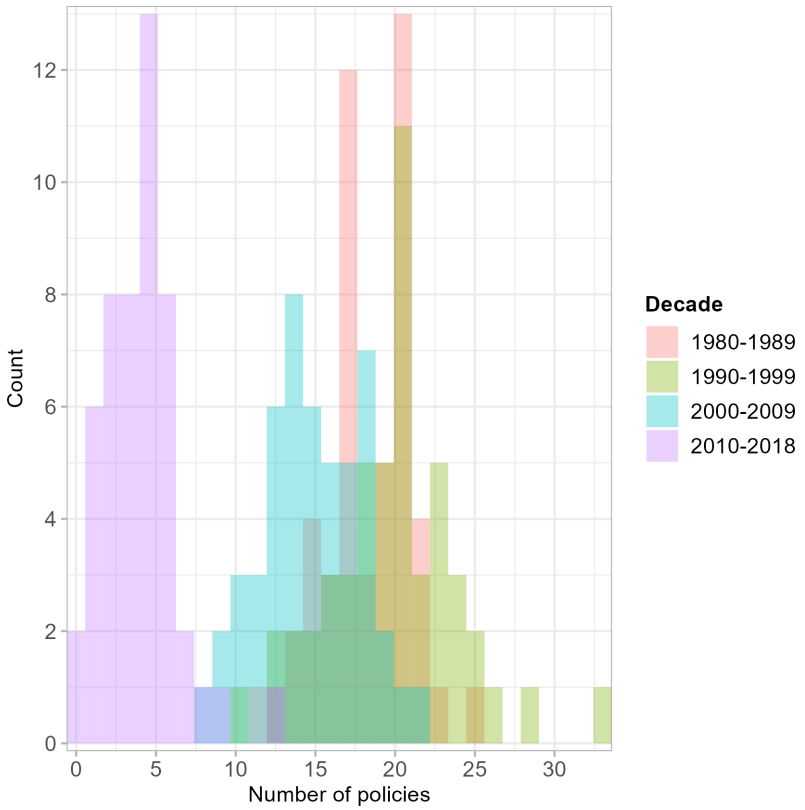


Figure 3.4. Frequency distribution of health policies passed in each decade leading up to the observation of mortality data panel.

thematic policy category form of $RFNPPPD$ is not possible, as it leads to empty cells. This would not be a problem had the period window been set to a longer range than one decade. However, at that point, such measure would lose its characteristic value of the rate at which new policies are being adopted. Under alternate circumstances where data contain a greater number of policies or fewer thematic categories the computation of the decomposition of $RFNPPPD$ by thematic category is feasible and straightforward.

3.5 Comparing various forms of SHP indicators and their associations with mortality

The two state-level indicators are weakly correlated ($r = 0.27$) in its raw forms and are practically uncorrelated when normalized ($r_Z = -0.1$). This is partly expected due to the fact that I score proposed in section 3.3 is a cumulative version of state health policy and is designed to measure the overall impact, weighted by its thematic group components. On the other hand, the the relative rate of passage of SHP intends to measure the speed at which health-related policies are passed, without regard for its qualitative impact. The two indicators are theoretically distinct but may contribute to a more holistic understanding of the impact of SHP on the state and county mortality. To this end, I present figures

showing the distribution and correlations between the various forms the two measures (raw and transformed) and run an analysis of variance (ANOVA) to gauge the potential effect size for each measure.

For the purposes of this descriptive analysis, I only briefly introduce the measures of mortality used in this thesis, which will be fully expanded upon in chapter 4. The mortality data has been obtained from the United States Mortality DataBase (USMDB). The series for states have been generated using the Human Mortality Database (HMD) version 6 methods protocol (Wilmoth et al., 2021) and the county mortality data have been derived from the Bayesian model analysis of county-level data ($N = 2472$) (Alexander et al., 2017; Winant et al., 2021). For the analysis I use 3 mortality indicators at state and county levels for each sex at 1990, 2000, 2010 and 2018 time points: life expectancy at birth and at age 65, ($e(0)$ and $e(65)$) as well as the age-standardized death rate (ASDR). I calculate the age-standardized mortality rates by sex using 2018 population as the reference in each case. Correlation summaries shown in Figures A.1.1 and A.1.2 in the Appendix demonstrate a high degree of correlation ($r > 0.9$) between all the mortality measures for states and county groups across the 4 time periods.

A similarly predictable consistency of association between the mortality measures and various forms of the two SHP indicators can be observed from the correlation summary plots in Figures A.1.3 and A.1.5. For states, variants of I score: raw form, ranked, and decade-lagged versions manifest a weak or negligible pooled correlation with mortality for all sexes. However, when considered over time, the association gradually strengthens to moderate from slightly negative in 1990 to about 0.6 in 2018. The raw I score induces marginally strongest growth in the association by 2018 with both life expectancy and ASDR indicators ($r_{e(0)} < 0.6$ and $r_{ASDR} > 0.6$, respectively) than the ranked version or the 10-year lagged version. A similar dynamic is present at the county level, albeit with an attenuated effect, such that 2018 absolute value of correlation strength between the I score and its variants does not exceed 0.43. At the same time, the $RFNPPPD$ measure does not feature a conclusive evolution of the association between itself and mortality outcomes over time. While the pooled association of $RFNPPPD$ is similar to that of the I score and its variants, where pooled correlations are weak or negligible, the longitudinal correlations of the alternative SHP measure oscillate around 0. These observations confirm three key findings about the SHP measures insofar as they relate to mortality outcomes at various time points. First, while correlation does not imply causation, there is some evidence to suggest that the I score might be more appropriate to study mortality outcomes, as the cumulative positive effects of the health policy are likely to improve population longevity in the longer term. This is contrasted by the temporally restricted and qualitatively insensitive measure such as $RFNPPPD$. The relative rate of passage of new policies may correlate with mortality slightly more or less in any given decade, but ultimately may be poorly associated with the outcomes that are products of extended exposure to certain health events. Second, county-level correlations are weaker than those of the states. This is important because sub-aggregate populations are subject to heterogeneous contextual factors that may intervene and confound the associations to which the parent groupings (i.e., states) may not be sensitive. And third, qualitative assessment of health policies matters for mortality. While the mere number or the relative rate of passage of policies in a given period may be useful in estimating state policy innovation

dynamics, those could become inefficient when it concerns the measurement of effects on outcomes within a thematic subset of policies.

Additionally, the decomposition of the I score presents a possibility of measuring strength of the association between the mortality outcomes and the thematic components of the I score measure (see Figure A.1.4). Policy groups 1, 2, 4 and 5, as described in Table 3.1, appear to be the most correlated with mortality in the most recent years. It is possible that the legislation in the areas of traffic and road safety, firearm regulation, reproductive health, and alcohol and tobacco control had the highest impact on mortality over time. Policy groups 3 and 7, relating controlled substance abuse and nutrition and safety net policies had the highest pooled effect on mortality, particularly out of proportion for males ($r^{male}_{ASDR,3} = 0.59$ vs. $r^{female}_{ASDR,3} = 0.35$; $r^{male}_{ASDR,7} = 0.63$ vs. $r^{female}_{ASDR,7} = 0.40$). Interestingly, there is no significant difference in the association between the reproductive health policies (e.g., abortion) and male and female mortality. This is likely due to a confounding of the effect of reproductive health with the effects of other policy categories.

To gauge the effects that both types of SHP could impose on mortality, it is first necessary to determine whether or not there is a significant temporal dependence in the relationship. Though mortality values are assumed to be independent across geography in a cross-sectional sense, those might not necessarily be independent across time. I first conduct one-way repeated measures ANOVA analysis to identify the dependence of temporal dependence of age-adjusted mortality values for states and county groups. The relevant F statistics, partial effect sizes η^2, as well as pairwise t tests with Bonferroni adjusted p-values are reported in Table 3.2. According to Cohen (2013), the effect sizes of 0.01 or below are negligible, between 0.01 and 0.06 are small, between 0.06 and 0.14 are medium, and those 0.14 and above are indicating a large effect size. Significant annual differences in means evaluated though Shapiro-Wilk test for normality show that state-level ASDR is approximately normal under the natural logarithm transformation, with marginal deviations from normality for 2010 and 2018 for males ($p < 0.04$). Substantial values of η^2 for both states and counties indicate that much of the variation in mortality values is linked to the year in which those are observed. As Shapiro-Wilk test is not suitable for evaluation in analyses with high number of degrees of freedom in large sample sizes, county-group level normality is assessed visually based on the histogram plots. The examination of outliers via Q-Q plots in Figures A.1.6 and A.1.7 confirms for both state and county measures of mortality that extreme outliers are eliminated through a log-transformation of the age-standardized mortality rate. Subsequently, Greenhouse-Geisser correction for violations of sphericity assumptions is applied to the ANOVA results.

Lastly, I consider a two-way repeated measures ANOVA analysis, by additionally interacting age-adjusted mortality with the two SHP measures. From Table 3.3 it is evident that I score measure captures a significant amount of variance from the temporal perspective, featuring medium-to-large effect sizes across sexes ($0.131 < \eta^2_p < 0.140$). The relative fraction of new health policies passed in previous decade has no significant interaction with the year for any mortality indicator by sex. Instead, all variation in two-way ANOVA with $RFNPPPD$ is accounted for by the temporal dimension alone, with large effect sizes. As a result, the I score could be more predictive of mortality outcomes in a longitudinal perspective than more rudimentary measures, such as $RFNPPPD$. Two-way ANOVA results for the county

Table 3.2. Post-hoc pairwise t test comparisons and repeated measures one-way ANOVA analysis of logarithm of both-sex mortality rate, for states and county groups.

	States				County groups			
	1990	2000	2010	2018	1990	2000	2010	2018
1990		13.1***	22.4***	18.7***		49.9***	87.2***	68.8***
2000			20.4***	19.9***			81.2***	58.7***
2010				3.46**				–0.661
2018								
F	$F(3, 196) = 150.127$***				$F(3, 9884) = 891.375$***			
η^2	0.434				0.213			
N	50				2472			

Note: pairwise t statistics reported in the table with standard errors corresponding the following p-values, ***$p < 0.001$, **$p < 0.01$, *$p < 0.05$

groups indicate an attenuation of the effect sizes from medium-large to small-medium at best, albeit with highly significant effects across both measures and sexes. This is consistent with the understanding that county group variation in mortality is more dependent on local explanatory factors than an overarching state health policy (Boing et al., 2020), though the latter has a part to play. Despite the attenuation of the effect size with the granularity of geographic scale, the I score effect sizes coupled with the temporal dimension in the data are nearly double those of the interaction of the data year with the $RFNPPPD$ measure.

The description of these two-way relationships lays an important groundwork for the conditional effect estimation in the scope of inferential modeling. In the next chapter I fit a series of quantitative models, compatible with the formal causal paradigm introduced in the previous chapter, with contextual factors for states and county groups.

3.6 Summary

Due to the differences in aims, scope of application, wording and implementation, comparative analyses of the relationship between health policy and its intended outcomes are challenging. Therefore, analytical syntheses of broad spectra of legislation must inevitably rely on assumptions and compensate for data or research design limitations. In this chapter, I develop a method to create a pair of quantitative summary indicators of state health policy for further inquiry into health policy impacts on area level mortality. Two types of indices are constructed on the basis of publicly available state health policy data: the I score index and the rate of policy innovation indicator ($RFNPPPD$). There are two main advantages of the I score over the simple rate of health policy innovation. Unlike the latter, the I score incorporates a qualitative component of "health positivity" (or the assessed qualitative benefit to remedying health issues and mortality). In addition, the I score index represents a cross-sectionally relative, and temporally cumulative effect of policy action. Finally, the

Table 3.3. Results of the state and county group two-way ANOVA for the logarithm of mortality rate, by sex, and state health policy measures: policy impact (I) score and the relative fraction of new policies passed in previous decade ($RFNPPPD$).

	Both-sex		Female		Male	
	F	η_p^2	F	η_p^2	F	η_p^2
	States					
Year	$F(3, 180) = 4.21^{**}$	0.066	$F(3, 180) = 1.95$	0.032	$F(3, 180) = 963^{***}$	0.138
I score quintile	$F(4, 180) = 0.25$	0.006	$F(4, 180) = 0.23$	0.005	$F(4, 180) = 0.30$	0.007
Year × I score quintile	$F(12, 180) = 2.45^{**}$	0.140	$F(12, 180) = 2.36^{**}$	0.136	$F(12, 180) = 2.27^{*}$	0.131
Year	$F(3, 180) = 16.64^{***}$	0.217	$F(3, 180) = 16.64^{***}$	0.140	$F(3, 180) = 28.28^{***}$	0.320
$RFNPPPD$ quintile	$F(4, 180) = 1.21$	0.026	$F(4, 180) = 1.03$	0.022	$F(4, 180) = 1.41$	0.030
Year × $RFNPPPD$ quintile	$F(12, 180) = 1.07$	0.067	$F(12, 180) = 0.98$	0.061	$F(12, 180) = 1.11$	0.069
	County groups					
Year	$F(3, 9868) = 70.19^{***}$	0.021	$F(3, 9868) = 29.45^{***}$	0.009	$F(3, 9868) = 198.24^{***}$	0.057
I score quintile	$F(4, 9868) = 6.80^{***}$	0.003	$F(4, 9868) = 5.39^{***}$	0.002	$F(4, 9868) = 6.46^{***}$	0.003
Year × I score quintile	$F(12, 9868) = 80.45^{***}$	0.089	$F(12, 9868) = 80.32^{***}$	0.089	$F(12, 9868) = 76.97^{***}$	0.086
Year	$F(3, 9868) = 285.20^{***}$	0.080	$F(3, 9868) = 142.18^{***}$	0.041	$F(3, 9868) = 520.79^{***}$	0.137
$RFNPPPD$ quintile	$F(4, 9868) = 36.28^{***}$	0.014	$F(4, 9868) = 30.34^{***}$	0.012	$F(4, 9868) = 42.95^{***}$	0.017
Year × $RFNPPPD$ quintile	$F(12, 9868) = 33.79^{***}$	0.039	$F(12, 9868) = 32.77^{***}$	0.038	$F(12, 9868) = 34.30^{***}$	0.040

Note: $^{***}p < 0.001$, $^{**}p < 0.01$, $^{*}p < 0.05$

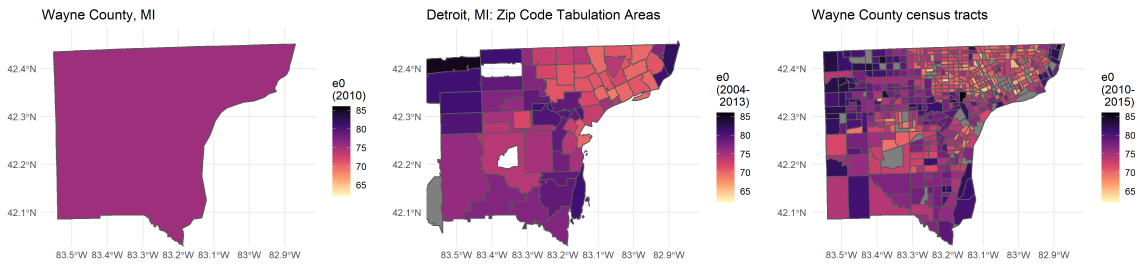
I score is decomposable into its thematic policy group components, should it be necessary. One plausible advantage of $RFNPPPD$ over the I score index lies in measuring the health policy introduction momentum. Lastly, I show that the association between mortality and both types of state health policy indicators is significant and time-dependent. This informs further application of appropriate analytical approaches, such as panel regressions, time fixed and random effects in the study of the causal impact of state health policy on mortality.

Chapter 4

Sub-national Analysis of the Impact of State Health Policy on Mortality

Sub-national estimation of mortality and its components with a focus on spatial and temporal domains has been gaining traction over the past few decades. The advent of programming tools, computing power to process the analyses, and geographically linked data at various levels of aggregation rendered such estimation feasible (Carracedo & Debón, 2021; Dwyer-Lindgren et al., 2022). It has been shown that variation in mortality is sensitive to scale of spatial aggregation (Boing et al., 2020). This finding carries forward an important implication. The degree to which mortality varies may not only be the function of a particular area, defined by its physical attributes, but also by communal and administrative determinants that align with state and county borders (Dobis et al., 2020; Nandi & Kawachi, 2011). Although the spatial dimension is clearly germane to an analysis of sub-national mortality and its socio-economic causes, the change of spatial support in the data lays the analysis open to the modifiable areal unit problem (MAUP) (Openshaw, 1984). This well known problem involves changes in the magnitude and direction of quantitative results, depending on the manner of spatial data aggregation due to limits on the geographic scope of the data imposed by a researcher or data included in a set (e.g., data on one state, but none from the neighboring states). Depending on a particular problem posed by MAUP this could lead to bias and making the conclusions of an analysis susceptible to ecological fallacy (Anselin, 2002; Huie, 2001). For instance, as may be inferred from Figure 4.1, the results of gradual aggregation from the census tract level to the county level clearly illustrate the sensitivity of modeling results to MAUP.

Aggregated mortality estimates not only fail to reflect the true heterogeneity of the underlying phenomenon, but also crucially omit the contextual structure of the patterns that give rise to the aggregate. It is therefore prudent to use the most granular data available to ensure reasonable interpretations can be made from model coefficients. However, making informed hypotheses about potential determinants of mortality becomes challenging without considering those alongside evidence from analyses on coarser geographic units. Placing the central aim of this thesis in conversation with the spatial treatment of data, in this chapter I make use of the state and county group estimates of mortality and relevant covariates at each geographic scale to determine the impact of state health policy on sub-national mortality



(a) **(b)** **(c)**

Figure 4.1. Detroit, MI. Disaggregation of county-level life expectancy at birth by zip-codes and census tracts unveils important spatial patterns given rise to by the local phenomena.

Sources: (a) Own calculations based on CDC WONDER data (Wayne County, MI, 2010 deaths by 5-year age groups); (b) Center on Society and Health (Virginia Commonwealth University), Detroit Life Expectancy Methodology and Data Table. Data retrieved from https://societyhealth.vcu.edu/media/society-health/pdf/LE-Map-Detroit-Methods.pdf on June 4, 2023; (c) NCHS USALEEP 2010-2015 census tract life tables for Michigan.

outcomes. I test the empirical fit of the data in relation to the conceptual SCM model developed in chapter 2 to attain the most optimal representation of the data generating process.

I seek to uncover the effects of state health policy on mortality in the presence of other contextual indicators. I ask whether and to what extent SHP is involved in spatio-temporal spillover impacts to neighboring states and counties. To this end, I extend the traditional linear and longitudinal panel modeling approaches by incorporating various spatio-temporal components, such that each source of variation could be accounted for explicitly. If effect spillovers exist, it is important to determine how the scale of aggregation impacts their proliferation (L. Vaughan & Arbaci, 2011). A strength of spatial modeling approaches is the ability to demonstrate how much unexplained variance exists in an observed pattern due to factors linked to a particular place or a cluster of places, and whether this effect is global or associated with local aberrations (J. LeSage & Pace, 2009). In the context of mortality modeling such an approach would greatly aid in identifying the underlying geographic causes of mortality. Furthermore, the proper quantitative representation of qualitative concepts and accounting for the determinants of mortality in accordance with the chosen theoretical paradigm boosts the explanatory power of a model. Spatial models have been previously evaluated in both Bayesian and traditional maximum likelihood estimation settings with hierarchical structure terms (Carracedo & Debón, 2021; Kamenetsky et al., 2019; Lacombe & McIntyre, 2016, 2017; Sparks et al., 2013). At the same time, there is a paucity of research incorporating health regulation holistically to study all-age mortality at multiple geographic scales and throughout the entire country. As such, this chapter is composed of a quantitative synthesis and an application of the well-developed technical framework for spatio-temporal panel approaches with the goal of determining how essential local health policy environment is to improving mortality conditions locally and overall.

4.1 USMDB data as input for sub-national mortality analysis

The present work relies on the data provided by the United States Mortality DataBase (USMDB) at the state and county group levels. State life tables are available as part of the USMDB output based on Human Mortality Database (HMD) Version 6 of the Methods Protocol (Wilmoth et al., 2021). For the county group output by sex, I use the results of the Bayesian model fit to the county-level death count data from the National Center for Health Statistics produced by the USMDB team (Alexander et al., 2017; Winant et al., 2021). The majority of the sample is composed of single counties. Of 3,221 counties under the 2010 US Census Bureau definition 1,084 counties with small populations were grouped into 401 county groups through adjacency and other population criteria to fit the requirements to reduce model parameter variability. The District of Columbia is excluded from the county group data due to the fact that by design no estimate was produced within the framework of the county-level USMDB mortality modeling. In other respects, the life table mortality indicators are of high quality, producing a close fit to the observed data and, when examined by state, bearing a high degree of fidelity to the aggregate. Age-standardized death rates associated with the generated county group life tables for 1990–2018 are presented in Figure 4.2. State-level estimates of life expectancy are associated with the life tables produced using the traditional HMD methods (United States Mortality DataBase, 2023; Wilmoth et al., 2021) are shown in Figure 1.1. Both figures provide comparable perspectives on the evolution of mortality conditions across the US sub-national geographies and over the three decades, yet reveal important differences on a more local level that are otherwise obstructed by aggregation to the state or national level. However, for the present analysis I use only 3 years of county-level data surrounding each decennial census year in the range (i.e., 1990, 2000, 2010) as well as 2018. The main hindrance to the analysis of the full series of mortality data is the unavailability of many important explanatory summary indicators that were measured consistently at decennial time points until the introduction of the American Community Survey in the mid-2000s.

I extract 3 measures of mortality from the USMDB data to represent the outcome. Each indicator will be used in the models that follow to draw inferences with respect to the impact of the state health policy and other key factors on mortality at the state and county levels. First, as commonly used in many demographic analyses and public health studies, I calculate age-standardized death rate (ASDR) using 2018 US population as a reference. The main advantage of ASDR over age-specific measures of mortality is its comparability across various geographic units and over time. In addition, it condenses mortality conditions brought about by variable age structures in different counties and states into a single quantity. The use of the ASDR indicator in analysis alongside other summary measures of phenomena that co-occur in a given space and time is convenient. I compute ASDR for each sex separately and for both sexed combined in order to gauge the degree to which sex-specific mortality responds to changes in state health policy, conditional on context.

Some studies advocate for the use of life expectancy as an alternative measure to ASDR, as the former is more interpretable and more readily understood by the audiences outside of the scientific community (Silcocks et al., 2001). For this reason I supplement the present

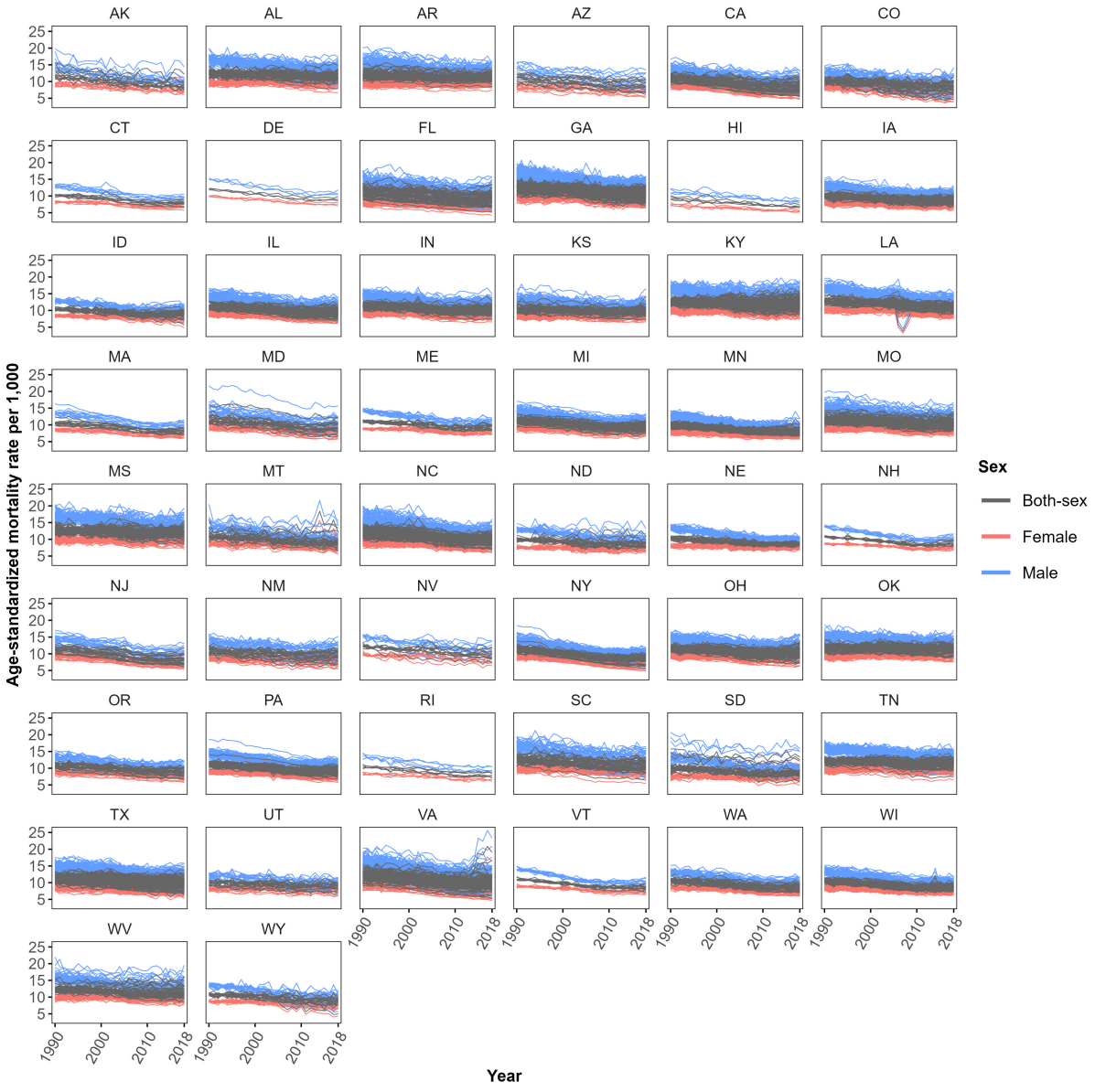


Figure 4.2. Age-standardized mortality rate trends for the US counties, by year, sex, and state, 1990–2018.

Source: Own calculations using USMDB county-group data (Winant et al., 2021). The District of Columbia, Puerto Rico and US territories have not been fit in the county-level model and hence are absent from the data. The national US population in 2018 was used as a reference for the computation of the rates.

analysis with measures of life expectancy at birth (e_0) and at age 65 (e_{65}). These may prove useful in discerning any age-sensitive responses of mortality to the contextual factors. Just as for ASDR, I extract these measures separately for females, males, and for both sexes combined.

In order to alleviate possible irregularities from temporal shocks I compute means of ASDR and life expectancy measures from 3 consecutive years, including an analysis time point itself and the 2 years surrounding it (e.g., 1989–1991 mean for the 1990 time point, etc.).

4.2 Independent variables

4.2.1 State health policy and state government ideology

The focal exposures in the present analysis are state-specific health policy indicators and state government ideology. State health policy indicators are such as presented in chapter 3 of this thesis. To summarize, these include the normalized I score index, its normalized 10-year lagged version, and the normalized relative fraction of new policies passed in a prior decade ($RFNPPPD$). For brevity, these will be henceforth referenced using a letter notation as I_Z, $I_{t-10,Z}$ and $\tilde{\pi}_{10,Z}$, respectively, and interchangeably with the corresponding full names. I test all these measures independently in the models that follow later in this chapter.

As far as government ideology is concerned, I do not consider the voter/citizen political ideology under the current conceptual framework. This is because the effect of citizen views on policies and health benefits that result from those are generally overridden by the function of state governing bodies. Elected officials are deemed to represent citizen views and to advocate for their needs. This may not be a strict case under a scenario where certain propositions pertaining to health find its way onto ballots in certain states, or temporary divergence between majority citizen ideology and that of the temporarily elected officials. However, I dismiss these instances as these occur with relatively rarity and tend to be very selective in nature. This issue notwithstanding, I use the government ideology measure as presented by Fording (2018), based on the methodology originally developed by Berry et al. (1998) and improved in subsequent studies (Berry et al., 2010). At its core is the weighted sum of components of political power shares (POW) between Republican (REP) and Democratic (DEM) parties in the lower (LOW) and upper (UPP) chambers of state legislative bodies, their ideology scores (ID) and the ideology score of assigned to a governor (GOV) in state s and in year t:

$$
\begin{aligned}
GOVTIDEO_{s,t} = {} & 0.25[(POW : DEM : LOW_{s,t})(ID : DEM : LOW_{s,t}) \\
& + (POW : REP : LOW_{s,t})(ID : REP : LOW_{s,t})] \\
& + 0.25[(POW : DEM : UPP_{s,t})(ID : DEM : UPP_{s,t}) \\
& + (POW : REP : UPP_{s,t})(ID : REP : UPP_{s,t})] \\
& + 0.50[ID : GOV_{s,t}]
\end{aligned}
$$

As becomes evident from the formula, one of the key assumptions accepted by Berry et al.

(1998) is that the ideological weight is distributed in equal parts between the two chambers of the state assembly and the governor. For the purposes of the present analysis it is a reasonable assumption, as the two branches of government ought to agree in order for a bill to be signed into law. Ultimately, the ideology data takes form of a score for every state between 1960 and 2017 ranging from 0 to 100, where 0 indicates extreme conservatism in state government and 100 represents a state where politicians hold extremely liberal views. Certainly, no state is ever fully conservative or liberal, however the values do change over time, as new state officials are elected and generational shifts take place. Since it takes time for many regulations, particularly controversial ones (e.g., gun reform or abortion), to be written, voted and passed into law state ideology score in any given year may not reflect the amount of support or resistance that any such policy may have been subject to in the process of its writing or debating. To smooth the effects of election cycles and to better describe the impact of ideological currents within state governments leading up to the passage of laws I compute a 10-year average of the score leading up to the year of observation. This cut off is purposefully arbitrary to represent the uncertainty and variability in the idiosyncratic manner in which aspects of a particular legislative context shape the agenda in state government at a given time.

4.2.2 Age and level of population health

I use median age in a population as the US Census Bureau's summary measure at both state and county levels (Social Explorer & U.S. Census Bureau, 2023a, 2023c, 2023e, 2023g). The more advanced population age is expected to negatively correlate with the actual and perceived health in the population, purely as a function of the onset of health conditions brought about by aging. I also employ it as a proxy for generational belonging at a given point of observation. Higher values of median age are expected to be weakly-to-moderately associated with ideological preferences, specifically insofar as state government ideology is concerned as well as the types of health policies passed by a state.

Next, I follow the tested precedent by Dwyer-Lindgren et al. (2017) by ascribing the prevalence of poor self-rated health in a population to the theoretical construct of population health (Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion, Division of Population Health, 2023). This option is conceptually consistent with the DAG (Figure 2.4) proposed in chapter 2. A crucial assumption behind this choice is that the experience of good or poor health at any one time as measured by its perception ratings is neither a *direct*, nor a likely immediate consequence of state health policy. Conversely, popular discontent and manifestations of unmet health needs are likely to impact the legislative health agenda directly through correspondence between the affected voters and the elected officials, for example.

Perceived level of health in the population can be measured on par with with attitudinal measures. To fit the conceptual model presented in chapter 2 it should measure the perceived or self-rated health, and not necessarily its factual manifestation, insofar as it is able to promote particular initiatives onto the voting floor in state government. Behavioral Risk Factor Surveillance System (BRFSS) is an annual national survey that measures self-reported health at sub-national level since 1993 onward. Self-reported health is an ordinal indicator of perceived health on the scale from 1 to 5, ranging from the "poor" to "excellent" overall

self-rated health, respectively. It is of particular interest to measure poor or fair health prevalence in populations, as this ought to be somewhat indicative of the true level of health in the population.

State-level estimates of self-rated health are available. Unfortunately, BRFSS data on county-level estimates are sparse with only 13%–15% of all counties represented in the data due to low sample sizes, complex survey design and confidentiality concerns. However, previous attempts were made to apply a multi-level Bayesian small-area model to fill the gaps in the geographic distribution at the state as well as county levels (Dwyer-Lindgren et al., 2017). The latter series do not include estimates for years before 1995, as BRFSS survey did not field the question on general health until 1993, while Centers for Disease Control and Prevention did not produce publicly available estimates of the prevalence of poor self-rated health until 1995. Dwyer-Lindgren et al. (2017) estimate series also stop in 2012. However, the estimates constructed by the authors are based on the 2010 US Census county boundaries, which are congruent with the counties featuring USMDB mortality estimates. In order to construct plausible estimates at the state and county levels I use the results of Dwyer-Lindgren et al. (2017) as a base for time series forecasting to extend the observed data backward to 1990 and forward to 2018. Time series forecasting offers an appealing alternative to the replication of much more complex methods, by exploiting the features of the data trend, deviations from the trend and potential seasonality in order to fit a model.

The method I employ for this task involves the individual fitting of 19,158 final models (one for each combination of 3,193 states/counties, 3 sex categories, and 2 forecasting/backcasting horizons), which ensures the most tailored fit to each series of the base estimates. To obtain the final model for each combination, I use an adaptive algorithm to fit 10 candidate models for each combination from the exponential smoothing and the autogregressive integrated moving average families of models. In order to achieve an optimal selection and subsequent forecasting for each combination of sex, state or county, and the forecast direction I identify the best fit using correlated Akaike Information Criterion (AICc) in addition to the traditional AIC and BIC criteria.

$$AIC_c = AIC + \frac{2k(k+1)}{T-k-1}$$

AIC_c is helpful as a selection criterion because it corrects for the small sample bias, which is bound to affect the models with only 18 years of observation. Hereby I provide a brief overview of the mechanics of the models used as candidates in this exercise.

Exponential smoothing time series (ETS) methods have gained popularity since the late 1950s (Hyndman & Athanasopoulos, 2021). These methods capitalize on assigning weights to the components of error, trend and seasonality of data, exponentially decaying as a function of elapsed time. The simplest exponential smoothing model ETS(A,N,N) (i.e., with an additive error, but no trend or seasonality component) can be characterized by the following forecast and smoothing equations.

$$y_t = l_{t-1} + \varepsilon_t \tag{4.1}$$
$$l_t = l_{t-1} + \alpha\varepsilon_t \tag{4.2}$$

where l_t is a level, or a smoothed value at time t, while the additive errors $\varepsilon_t \sim \mathcal{N}(0, \sigma^2)$ $i.i.d$ for each time point. Equations 4.1 and 4.2 describe a flat forecast function under $\alpha = 0$. Once the level has been estimated the only innovation in the forecast thereafter is governed through the smoothing parameter $\alpha \in (0, 1)$ which, if equal to 1, produces a forecast equivalent to a random walk model. One could additionally account for a trend component in the exponential smoothing. An additive trend b with its own smoothing parameter $\beta \in (0, 1)$ can be expressed as:

$$y_t = l_{t-1} + b_{t-1} + \varepsilon_t \tag{4.3}$$
$$l_t = l_{t-1} + b_{t-1} + \alpha\varepsilon_t \tag{4.4}$$
$$b_t = b_{t-1} + \alpha\beta\varepsilon_t \tag{4.5}$$

This approach is called Holt's linear method, or ETS(A,A,N), under which forecast errors implicate the prior values of the trend as well as the level, but are otherwise normal and independent with mean 0 and variance σ^2. However, in the long run forecast predictions may be unrealistic, if the forecast keeps increasing or decreasing. Hence a dampening coefficient ϕ is added to the trend component of Holt's linear model (ETS(A,Ad,N)) to ensure that a forecast converges to a particular value in the longer run, thus more realistically representing the lack of knowledge about the future direction of the forecast. Models from the exponential smoothing family used in the estimation of poor health prevalence are: simple exponential smoothing (SES) that includes only additive errors, Holt's linear method including additive errors and trend, as well as damped model with additive errors and additive damped trend. No seasonality component is considered here, as no pattern of cyclic/seasonal variation is apparent in the observed data.

An alternative approach is to fit models autoregressive integrated moving average models (also known as ARIMA), which generate forecasts on the basis of autocorrelation in the data trend, the residuals, and seasonality (with the latter omitted as per explanation above). The "integrated" differencing component is necessary to achieve stationarity in the trend so that autocorrelations could be captured without bias. Equation 4.6 is a generic specification of a non-seasonal ARIMA model.

$$y_t^{'} = c + \phi_1 y_{t-1}^{'} + \cdots + \phi_p y_{t-p}^{'} + \theta_1\varepsilon_{t-1} + \cdots + \theta_q\varepsilon_{t-q} + \varepsilon_t \tag{4.6}$$

where p is an order of the autoregressive part (AR), d is the trend differencing order, and q is the order of the moving average (MA), while ϕ and θ are the respective AR and MA lag coefficients, and c is a constant. From the family of ARIMA models I explicitly focus on ARIMA(0,1,0), ARIMA(0,1,1) and ARIMA(0,1,2) as the most likely candidates for the short 18-year series. To conclude this I rely on observations from the autocorrelation function (ACF) plots showing series non-stationarity before the first-differencing of the trend, and a likely moving average component. I also include an automated step-wise testing of models ranging from ARIMA(2,1,0) to ARIMA(0,1,2) and a *search* model that employs Hyndman-Khandakar algorithm for the deeper exploration of the parameter space to find the best ARIMA(p, d, q) fits with and without a constant beyond the explicitly specified set (Hyndman & Athanasopoulos, 2021). Exponential smoothing models do not require stationarity

within the trend, but incorporate additive error and trend components (including damped trend) and no seasonality as none is pertinent to the prevalence data. On the other hand, all explicitly specified ARIMA models incorporate first-order differencing of the trend in order to achieve its required stationarity, such that the residuals are indistinguishable from white noise. Unless indicated, a search-type model determines the components and its order automatically with Ljung-Box test statistic for white noise series, and from ACF and partial ACF values. For example, ARIMA(0,1,0) with a constant c at its core is equivalent to a random walk model with drift, with random noise as the only source of innovation. Figure 4.3 illustrates an example of fitting time series forecast models to the Alabama state-level estimates. The output for all the other states and counties is vast and is omitted for practical space considerations, but most show comparable results.

While validation options for the models are limited, especially at the county level, CDC publishes a both-sex age-adjusted prevalence of poor/fair self-rated health at a state level for the recent years (Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion, Division of Population Health, 2023). I use this data to evaluate the state-level forecasts with the official survey estimates for the period of 2013–2018. The results of the modeling validation are presented in the Table 4.1 with the best model fit to each of the state-year-sex combination. Despite the fact that errors may be compounded by some degree of bias embedded in the smoothed estimates from Dwyer-Lindgren et al. (2017), only about a quarter of the 2018 forecast point estimates are falling outside of the 95% confidence interval of the official CDC estimates. Moreover, as shown by the mean absolute percent error (MAPE) the deviations are consistently hovering around 5% away from the official estimate, and do not exceed 1 percentage point in magnitude throughout the period, on average, as shown by the mean absolute error (MAE). Though imperfect due to the model simplicity and short length of the base time series, given the consistency and relatively insubstantial inaccuracy (as per the root mean square error (RMSE)), the estimates of poor health prevalence appear plausible and are suitable for application in the present analysis as an explanatory indicator. Since similar models have been fitted to the county-level data for 1995–2012, I assume the model performance is comparable to that of the states for the backcast period of 1990–1994 and the forecast of 2013–2018. Moreover, only the estimates of population prevalence of poor/fair health at 4 time points compatible with the rest of the measures in the analysis of state health policy on mortality are kept: 1990 (backcast), 2000 and 2010 (used as found in Dwyer-Lindgren et al. (2017)), and 2018 (forecast).

As shown in Table 4.2, ARIMA family of models ensure superior fit to that of ETS models in virtually all cases. Specifically ARIMA(0,1,0), or a random walk with drift model, is indeed the preferred candidate in over 40% and up to 47% of observations in both 1990–1994 and 2013–2018 forecasting horizons. The automated stepwise ARIMA(p,d,q) is the second most common best fit model with nearly the same frequency as ARIMA(0,1,0), though the former is not a single model, but rather a set from the parameter space between ARIMA(2,1,0) and ARIMA(0,1,2) (with and without constant terms) that have not been explicitly tested. Interestingly, ARIMA(0,1,1) with MA(1) component is almost twice as common in fitting the time series for males than for females. These observations confirm the prior hypothesis that most state and county time series do not feature an autoregressive component.

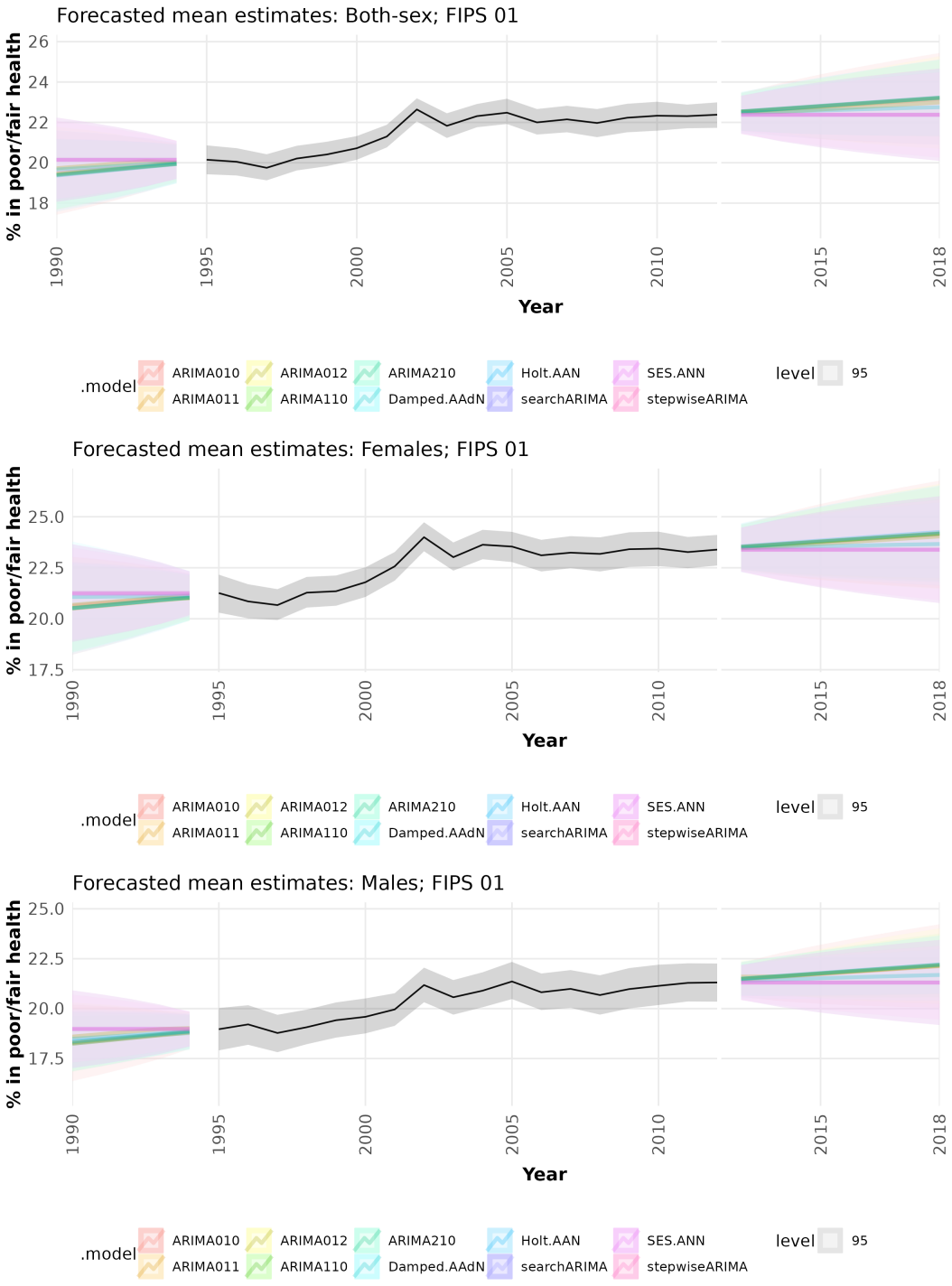


Figure 4.3. Example using Alabama state forecasts to estimate poor/fair self-rated health prevalence, by sex.

Note: black solid line indicates data from Dwyer-Lindgren et al. (2017).

Table 4.1. Validation measures of the 2013–2018 both-sex forecast of mean prevalence of poor/fair health for states against the published CDC age-adjusted state-level estimates.

Year	RMSE	MAE	MAPE (%)	$\pi_{states} \notin$ CDC 95% CI	Mean % CDC 95% CI contained within forecast 95% CI
2013	0.921	0.728	4.7	0.24	89.5
2014	1.190	0.894	5.6	0.24	59.4
2015	1.248	1.027	6.4	0.35	49.0
2016	1.245	1.034	6.4	0.33	36.4
2017	1.295	0.947	5.7	0.33	39.2
2018	1.203	0.954	5.6	0.27	46.7

Table 4.2. Best fit time series model selection proportions by forecast direction and by sex.

	1990–1994			2013–2018		
Model	Both-sex	Female	Male	Both-sex	Female	Male
ARIMA010	0.426	0.472	0.407	0.427	0.472	0.407
stepwiseARIMA	0.426	0.424	0.402	0.425	0.424	0.401
ARIMA011	0.102	0.075	0.129	0.102	0.075	0.129
ARIMA110	0.027	0.018	0.025	0.028	0.018	0.026
ARIMA012	0.012	0.005	0.019	0.011	0.005	0.019
searchARIMA	0.006	0.005	0.014	0.007	0.004	0.014
ARIMA210	0.000	0.001	0.004	0.000	0.001	0.004
Damped.AAdN	0.001	0.000	0.000	0.000	0.000	0.000

Finally, the estimated 1990 and 2018 values for poor health prevalence by sex for each county and state are appended to the accepted-as-known 2000 and 2010 values from Dwyer-Lindgren et al. (2017) to form a panel for use in the present analysis of sub-national mortality. Given that certain smaller USMDB counties are grouped, the average estimates of poor health prevalence in the counties involved in grouping are weighted by the respective population sizes.

4.2.3 SES measures: income, income inequality, education, occupation

Median household income is a widely used summary measure of income in many social science studies. For a sufficiently small area this measure has an advantage of capturing the typical level of income of the local resident population while avoiding skewness bias due to uneven income distribution. However, at a coarse geographic scale, such as a state, median household income loses its associative properties since the heterogeneity in population attributes becomes large while the determinants of income may become unrelated among the various population groups contained within. To rectify this limitation and in correspondence with the conceptual model, I use the Gini coefficient of income inequality (US Census Bureau, 2012, 2020, 2021) to measure the unevenness in income distribution. Gini index value of 0 indicating equality of income levels among the residents, whereas 1 signifies the highest level of disparity. Due to the fact that Gini coefficient is only available at the county level in years 1990 and 2000, to obtain the corresponding state-level measure I weighted the estimates for counties that fall within each state by the number of households within those states. While an imperfect solution, it should be an adequate representation of income inequality at the state level in the absence of complete microdata. I adjust the values of median household income for inflation to reflect the annual amounts for state and county in 2018 dollars for a fair comparison (Social Explorer & U.S. Census Bureau, 2023b, 2023d, 2023f, 2023h).

In line with the literature on the risk factors of increased mortality insofar as educational attainment is concerned I use proportion of the population in a state or county group that has never graduated high school (Social Explorer & U.S. Census Bureau, 2023b, 2023d, 2023f, 2023h). Additionally, I test the proportion of the category of population with college degree or above to gauge potential protective effects of higher education from mortality.

To account for a potential impact of various types of occupational hazards in an area I include the data on the prevalence of manual, low or unskilled jobs (Social Explorer & U.S. Census Bureau, 2023a, 2023c, 2023e, 2023g). To create a category of *blue collar* occupations and to compute the proportions of state and county group populations that engage in them, I combine the occupational categories that involve manual work, manufacturing, traditional trades (e.g., plumbing, electric work), resource extraction (e.g., mining).

4.2.4 Demographic area composition and segregation

As briefly touched upon in chapter 1 uneven distribution of characteristics in the general population, often characterized by patterns of residential segregation, is responsible for socio-economic conditions leading to a shorter lifespan (Nuru-Jeter & LaVeist, 2011). Here I dis-

cuss the quantitative representation of the concept of ethno-racial residential segregation. There are many indices used to characterize group inequality, isolation, segregation, diversity, and dissimilarity. Conceptually, residential ethno-racial segregation shapes multiple facets of economic well-being of area residents and can thus be a complementary measure to income, its inequality, and socio-economic status (Massey & Fischer, 2000). For the purposes of identifying inequality of residential arrangement by ethno-racial background, an entropy-based measure, such as the Information Theory Index (also known as Theil's H) may describe how diverse an area is for an attribute of choice. An overall index value of 0 would indicate perfect evenness of a local group distribution relative the region, and 1 signifies maximal dissimilarity between the two (Reardon & Firebaugh, 2002). Theil's entropy based Information Theory Index (H) is a decomposable measure and is often used to compare discrete groups on the global scale (say, cities with constituent neighborhoods within). The groups may be represented by race or gender, which is useful in segregation studies (Massey & Denton, 1989). An alternative index, also highly correlated with the Dissimilarity Index, is the Divergence Index (D) that rectifies a number of limitations that other indices have with respect to measuring inequality (Roberto, 2016, 2018). The main distinction of the Divergence Index is that it measures how different the areas which the groups occupy are, whereas the Information Theory Index H measures its diversity. Like the Dissimilarity Index, the Divergence Index is bounded between 0 and 1, with extreme values denoting complete equality of distribution and maximum disparity, respectively. An integral part of the multi-group Information Theory Index is Theil's entropy E, which itself reaches the value of 0 when an area contains individuals of only one group and 1 when all the groups are evenly represented.

$$E = \sum_{m=1}^{M} \pi_m \log(\frac{1}{\pi_m}) \tag{4.7}$$

where π_m is the proportion of a group m in an area. The entropy E can be computed for an individual area, or as a jth component (E_j, with and additional j subscript assigned to π_m equation terms) of the regional entropy. Using both E_j and E, one could calculate the H index for a region as a weighted average of its entropy components.

$$H = 1 - \sum_{j=1}^{J} \frac{t_j}{T} \frac{E_j}{E} \tag{4.8}$$

where t_j is the total population in a unit j and T is the total population of a region encompassing multiple j units. The subtraction from 1 is necessary to invert the interpretation of the scale, where 0 would indicate full integration and 1—a level of maximum segregation.

Similarly, the Divergence Index can be computed using many of the same components and with the same interpretation in terms the magnitude (i.e., 0 would indicate maximum similarity of the local population composition to that of the parent region, whereas the value of 1 would indicate the maximum multi-group divergence or segregation of a local area, relative to the region):

$$D = \sum_{j=1}^{J} \frac{t_j}{T} \sum_{m=1}^{M} \pi_{jm} \log(\frac{\pi_{jm}}{\pi_m}) \tag{4.9}$$

In the present analysis I descriptively present the D index at the level of a state and that of a county group. The application of either the H or D indices at varying geographic scales would be complementary to another, as each level of aggregation and indicator attributes provide important nuanced perspectives on *how* each measure describes segregation in disparate contexts (Catney, 2018; Fowler, 2016). Although census blocks and block groups are the smallest geographic units for which basic population summary attributes are reported for decennial censuses, I choose census tracts as the primary sub-regional (i.e., sub-state and sub-county) geographic units j. Census tracts are sufficiently small and numerous to offer adequate sample sizes for the computation of indices thus providing stable estimates over time. I generate consistent racial categories as measured in 1990, 2000, 2010 censuses and the 2020 5-year American Community Survey (with 2018 as the mid-year): White only, African American or Black only, Asian only and Pacific Islander, and Other (Manson et al., 2022). My preliminary analysis shows a high degree of correlation ($r > 0.8$) between the two indices on both state and county levels for 1990, 2000, 2010 and 2018 and progressively higher toward the more recent years ($r > 0.9$). This similarity is consistent with empirical comparisons (Roberto, 2016), barring more extreme cases where proportions are very skewed toward one group, which is fairly rare on the scale of a county or larger. It is with this understanding of the fair equivalence between the two index candidates, I ultimately choose the Divergence index D in further modeling in order to avoid redundancy and unnecessary complexity in the final model selection phase.

4.2.5 Urban environment

There is not one way to measure how urban any one place is. Much in how measurement approaches are applied in practice depends on different concepts represented by the level of economic activity in an area, population density, population number, area classification for administrative and zoning purposes, the level and/or pace of urban infrastructure development, etc. (Balk et al., 2018). For the purposes of this analysis I use the proportion of the population that lives in areas defined as urban by the US Census Bureau. The criteria for the inclusion of an area in an urban classification has been revised at each decennial census, although the changes did not typically lead to substantial divergence at the spatial scale of a state, or even a county. For instance, in the 2020 US census, an area is defined as urban if it contains at least 2,000 housing units and 5,000 residents in addition to a range of other criteria for population density and distance between population clusters and certain infrastructural elements (US Census Bureau, Department of Commerce, 2022). However, regardless of the decade, the degree to which the fraction of the population in a county or a state is urban ought to be indicative of the general level of infrastructural development, access to health services, and population exposure to distinct health hazards from those of the more rural counties and states.

4.3 Modeling strategy

4.3.1 Descriptive method

To complement the preliminary results shown in chapter 3, I expand the description of the data structure by evaluating additional variables pertinent to this analysis. I begin by showing variable summary statistics, pairwise correlations and present descriptive plots to demonstrate the evolution of the associations with main mortality indicators over time. The central feature of the present analysis is the use of spatial methodology to expand on the conventional knowledge of mortality at various geographic levels and its relationship with the local health regulation. Specifically, identifying the presence of spatial dependence in the data allows to substantially increase the precision of regression models and thus make more educated inferences. Controlling for spatial dependence accounts for structural variability in the model coefficients and avoids bias by eliminating the effects of regressor-outcome pairs between observations that are unrelated. Assuming spatial structure and orientation play a significant part in the relationship between mortality and state health policy, which has been theoretically established in the previous chapters, it is now necessary to empirically verify such claims. One very common method to test for such dependence is to compute Moran's I statistic using the residuals from the ordinary least squares fit for a variable of interest. Per Anselin (1995), the global sample measure of Moran's I can be computed as follows:

$$I = \frac{n \sum_{i=1}^{n} \sum_{j=1}^{n} w_{ij} z_i z_j}{\sum_{i=1}^{n} \sum_{j=1}^{n} w_{ij} \sum_{i=1}^{n} z_i^2} \quad , \; \forall \; i \neq j \tag{4.10}$$

where n is the number of units, $z_i = x_i - \bar{x}$ and $z_j = x_j - \bar{x}$ are the deviations from the mean for each spatial unit i and its geographic neighbors j. Elements w_{ij} of a connectivity/adjacency weights matrix $\mathbf{W}$ (explained further in section 4.3.3 on spatial weights design) are describing a particular form of spatial relationships between any two pairs of observations in a geographic space. Moran's I can be computed in a univariate as well as in a bivariate setting. In the latter case $z_j = v_j - \bar{v}$, representing the deviations from the mean of a second variable. At its core Moran's I can be viewed as a spatial (auto)correlation coefficient and offers researchers a convenient descriptive method of assessing the presence and strength of dependence between observation values across geographic space. A further extension to Moran's I is the test of local dependence, also termed *local Moran* and is part of multiple local indicators of spatial autocorrelation (LISA). As shown in Equation 4.11, local Moran is computed in a particular neighborhood where $j \in J_i$, are the only neighbors of a given unit i, using familiar components to the global Moran's I (Anselin, 1995).

$$I_i = z_i \sum_{j \in J_i} w_{ij} z_j \quad , \; \forall \; i \neq J_j \tag{4.11}$$

Then, a sum of local Moran scores $\sum I_i$ is related to the global Moran by the proportionality factor $n / \sum i \sum_j w_{ij} z_i^2$. Using local Moran values, it is possible to draw a LISA cluster plot,

which could be thought of as a localized spatial version of a t-statistic. A hypothesis test determines whether or not local values of one unit are significantly different from zero and, if so, whether they are higher, lower or similar in magnitude from the average of the neighbors. Like Moran's I values LISA plots can depict clustering of values in a univariate and bivariate settings. I produce maps to visually assess the clustering of mortality indicators and to draw comparisons for levels of disaggregation between the states and county groups.

4.3.2 Model specification

Given the longitudinal component of the data I examine the model in the panel setup, initially net of any spatial effects. The sample consists of $N_c = 2472$ county group observations across $N_s = 50$ states and $T = 4$ time periods. Careful consideration and selection of analytic strategies is important for a proper and unbiased estimation of the underlying phenomena. For example, propensity score matching (PSM) is a popular method for an unbiased estimation of a causal average treatment effect. However, in the present case it is not suitable for a number of reasons. First, state health policy score is a composite measure which makes it highly unlikely to see a state that undergoes no treatment of any kind (i.e., no health regulations) in a given decade. Second, the SHP measures are continuous treatment indicators, which are difficult to dichotomize using a binary threshold to indicate the presence or absence of treatment. Doing so would impose strong assumptions about the similarity of contributions of individual policies as well as thematic policy groups between multiple states. If states A and B hypothetically increased their SHP positivity scores by 1 point each in one time period, it could have been a result of state A's comprehensive tobacco tax regulation efforts, whereas in the case of state B an increase might have been due to the increase in government health programs for the elderly. Although the two states could match quantitatively, the similarity conditional on a set of covariates is unlikely, as the two states improved due to qualitatively distinct stimuli. Moreover, using PSM in such a manner violates the Stable Unit Treatment Value Assumption (SUTVA) (Huntington-Klein, 2022) in that (1) a qualitatively similar health regulation across states and over time represent a similar degree of intent to treat and the scope of its applicability to the population in question, and (2) the dichotomization of treatment on virtually any pre-determined threshold would not be identified consistently through a set of covariates used for matching, since the causes of changes in SHP scores are as disparate cross-sectionally as those are longitudinally. The third reason against the application of PSM in the current analysis is the dimensionality problem. Due to the fact that there are only 50 states to consider, balancing even on a modest number of covariates would quickly exhaust the possible combinations for matching. Lastly, PSM only operates on a set of observed covariates. Due to the complex nature of the treatment variable, there is a variety of potentially unmeasured confounders that could conceal open backdoor paths in the structural causal model adopted in chapter 2, thus preventing the identification of the causal relationship between the state health policy and mortality under the stated assumptions (Pearl, 2009).

The longitudinal dimension presents an opportunity to include fixed and/or random effects for the states and county groups, thereby controlling for the within-variation induced by unobserved covariates in either unit, time, or both dimensions. The isolation of a dedicated model parameter in the framework of fixed and random effects to absorb variation

that cannot be practically measured within the geographic units opens a possibility to measure changes that occur for each unit over time, for two time points across the sample, or both, assuming unit- and/or time-invariant characteristics. Independent control variables could then be fairly typical correlates and are encompassing of the wider patterns of socio-economic dynamics for small groups or areas. Ultimately, it is this approach that I adopt to make inference about the effects of state health policy of sub-national mortality in the framework of the conceptual model presented in Figures 2.3 and 2.4 in chapter 2 and given a set of plausible data indicators described earlier.

With the above considerations in mind, I introduce a series of regression models with the ultimate aim to select the most appropriate specification, while make further adjustments on the basis of the post-regression diagnostic tests.

4.3.3 Spatial models

In line with the aforementioned theoretical underpinnings I explore several modeling approaches that account for various aspects of spatial and temporal relationships. If the claims postulated in the previous chapters are valid, then running a spatial analysis would uncover potential effects of geographically global and local effect propagation. Prior to all modeling I assess the presence of spatial dependence across the three mortality indicators alone and in combination with SHP and all the other covariates, using descriptive methods, such as Moran's I statistic and clustering of localized spatially autocorrelated values (Anselin, 1995). Upon detection, further tests on the *type* of spatial dependence are necessary to guide the selection of an appropriate model describing spatial data generating process. For the inferential stage, I consider the traditional panel approaches as well as panel extensions to cross-sectional spatial econometric models with various forms of spatial dependence (as introduced by Anselin (2002)). Like the traditional panel fixed or random effects regressions, spatial panel models have also been implemented in the maximum likelihood (ML) as well as the generalized method of moments (GMM) estimation paradigms (Le Gallo & Pirotte, 2017). By definition spatially aware models and operations require a component that governs the relationships between geographic units. This effect may be achieved with a set of pairwise spatial weights between connected pairs of units akin to nodes on a graph.

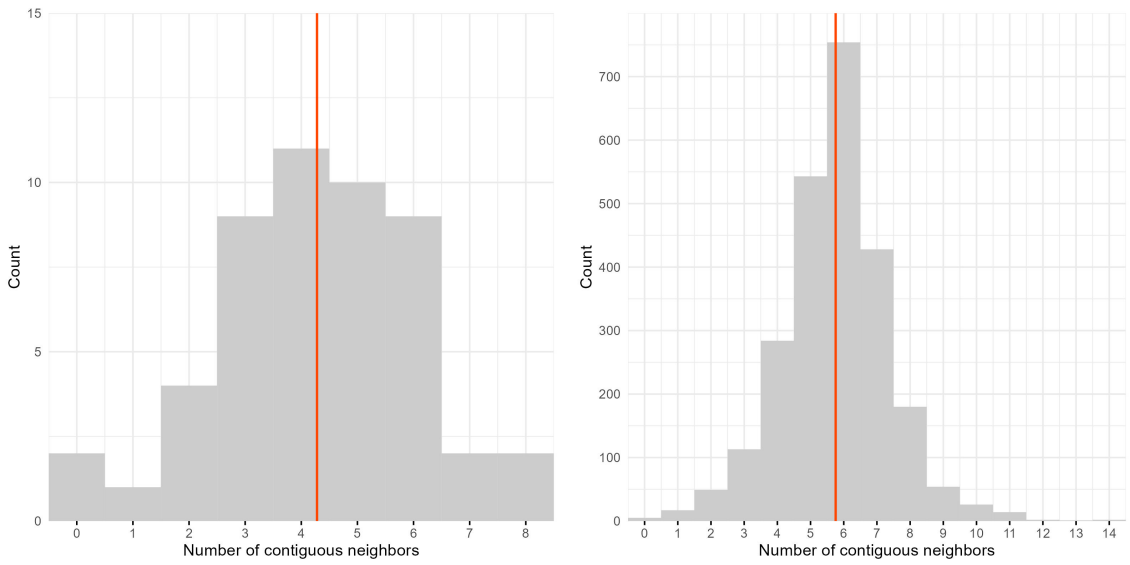
Spatial weighting design

The exact specification of a spatial connectivity structure depends on particularities of the data and the analysis at hand. However, a structure that respects adjacency or proximity of the units to one another is suitable in the majority of cases. The data that are naturally proximal share more similarities than its more distant neighbors. Specifically, one could create a matrix of binary spatial wights on the basis of connectivity or adjacency, such that an effect could pass from unit i to unit j if and only if they are adjacent. Mathematically, the binary nature of the matrix allows to evaluate the effect that is passed at "full strength" and/or in equal measure to all the adjacent units. This might be an oversimplification in the present analysis, given that counties and states vary in size and shape, at times separated by bodies of water, making the spatial orientation and adjacency more complex. Furthermore, this raises the question of the effect of contiguity that determines how strongly an impact could propa-

gate from a given unit given the neighboring structure of geographic units. In this analysis, I test spatial weights based on a *queen* orientation of the first degree of contiguity (i.e., the counties or states are considered neighbors if they share a common boundary or point, such that only immediate neighbors are impacted) and a k-nearest neighbors weighting structure. In most cases this would be equivalent to simple adjacency, except where adjacent states and counties are separated by significant natural obstacles, such as rivers, lakes or an ocean. I deem the $queen(1)$ configuration optimal as I expect the effects of mortality and its proximal determinants to attenuate in strength over distance, particularly as counties and states are fairly sizable. At the same time, one could consider distance as a weighting attribute, despite that Euclidean or other geographic distances may lack consistency in identifying the same set of neighbors. However, distance based connectivity structures, such as k-nearest neighbors (knn) impart desirable properties onto the structure of the data. Namely, since counties and states vary greatly in size and orientation relative to its respective neighbors, knn spatial weights consistently identify the k closest units. This improves stability of spatial model estimates as each areal unit will have a predictable number of connections through which an effect may or may not diffuse. Additionally, stable spatial structure mitigates the deleterious impact of the modifiable unit problem (MAUP) where the units, particularly those close to the edge of a geographic analysis extent, exhibit increased variability in the computation of zonal statistics, depending on the level of disaggregation/grouping. The disadvantage of knn relative to contiguity based weights is that it may potentially obscure meaningful links (a major road connection, for example) responsible for the diffusion of effects of an outcome and explanatory indicators. However, I expect this not to be an issue for geographic scale of counties or larger, as even the most geographically remote areas such as Hawaii could be well connected by air or sea to the mainland. In an attempt to strike a balance between the two approaches, I proceed as follows. I initially compute the $queen(1)$ adjacency matrix separately for states and the county groups. I then examine the distribution of the number of neighbors under the adjacency criterion in each sample and assign its rounded mean to k in order to compute the weights based on proximity for the average number of links. This ensures that the neighborhood of any given unit is not affected by an unusual orientation or disconnectedness of a county group or a state from the rest. The most extreme example of this difference may be the islands of Hawaii or the counties of Alaska and the mainland US, none of which would be considered neighbors under the $queen(1)$ weighting, but would be connected to some of the mainland counterparts under the k-nearest neighbors configuration. An example of k selection criterion is demonstrated in Figure 4.4 showing the distribution of the state and county group neighbors under the first-order adjacency. As a result, I derive a connectivity structure with k=4 for the states and k=6 for the county groups. Finally, I row-standardize the pairwise weight matrices for the two spatial resolutions, which ensures *a priori* equal neighborhood impact distribution across the local units (i.e., such that the weights for each unit i add up to 1). This is also an important feature for the technical implementation of the weights in models in the forthcoming sections.

Specifications of spatial models

Building upon the non-spatial specification of panel fixed and random effect models, spatial regression forms include a variety of geographic proximity and adjacency effect dynamics that



(a) **(b)**

Figure 4.4. Distribution of spatial neighbors under the queen(1) weighting configuration in the (a) state and (b) county group samples.

could be identified through testing and comparison of models with additional terms. The rudimentary forms of spatial econometric panel models include Simultaneous Autoregressive Regression (SAR), Spatial Error Model (SEM), Spatial Lax-X (SLX) processes. SAR accounts for a spatially endogenous response via a global spillover effect of the dependent variable, as described by Equation 4.12. The estimation of this process is recursive and requires a sizable number of simulations to obtain convergence in the outcome variable.

$$y_{it} = \lambda \sum_{i \neq j} w_{ij} y_{jt} + x_{it}\beta + \alpha_i + \phi_t + \varepsilon_{it} \tag{4.12}$$

where λ is a global autocorrelation parameter adjusting the strength of the endogenous spatially lagged response $\sum_{i \neq j} w_{ij} y_{jt}$. Here w_{ij} is the vector from a row-standardized spatial weights matrix $\mathbf{W}$ for neighboring county groups or states $i \neq j$, while α_i and ϕ_t are county group/state-specific and time fixed effects, respectively, and $\varepsilon_{it} \sim \mathcal{N}(0, \sigma^2)$ an *i.i.d.* nuisance term. By contrast, SEM offers an alternative perspective for spatial modeling, such that spatial data generating process is exogenous and unaccounted for by the regressors in the design matrix $\mathbf{X}$. SEM model (Equation 4.13) thus features a decomposable error term with portion attributable to spatial autocorrelation in model residuals and a non-spatial variation component $\varepsilon \sim \mathcal{N}(0, \sigma^2)$.

$$y_{it} = x_{it}\beta + \alpha_i + \phi_t + u_{it} \tag{4.13}$$

$$u_{it} = \rho \sum_{i \neq j} w_{ij} y_{jt} + \varepsilon_{it} \tag{4.14}$$

where ρ is a spatial error autocorrelation parameter adjusting the strength of the spatial dependence in model residuals. SEM is thus useful to account for spatial dependence in data

containing variation from multiple unobserved, exogenous factors. SLX is a separate model that describes local spatial dependence only, as defined by the spatial weights matrix $\mathbf{W}$, and differs from SAR and SEM models that focus on a global spatial impact propagation. This is a computationally simpler model that is advantageous in instances when spatial spillover effects are contained within a local neighborhood and do not propagate further in a recursive manner (Hall et al., 2022). Exogenous local spillover as featured in the SLX models, often titled spatial Durbin effect, motivates its further use as an additional component in more complex models (Vega & Elhorst, 2015).

$$y_{it} = x_{it}\beta + \sum_{i \neq j} w_{ij} x_{jt}\theta + \alpha_i + \varepsilon_{it} \tag{4.15}$$

SLX is a unique model in that the impacts of the regressors on the outcome are decomposable into direct and indirect ones. Direct impacts are generated via a geographic unit's own regressor values, whereas the indirect impacts stem from the weighted average of the neighboring units' values, or $\sum_{j=1}^{J} w_{ij} x_j$. The statistical significance of the total impact is tested independently of the direct and indirect components.

Further combinations of the three models give rise to more complex specifications. Two popular models are Spatial Durbin (SDM) and Spatial Durbin Error Models (SDEM), the parameters of which describe a global endogenous spillover in the case of SDM, and local spatial spillover process via spatially lagged explanatory variables, as well as through spatially autocorrelated residuals in the case of SDEM. SDM fixed effects panel regression with endogenous spatial autoregressive terms uses previously defined equation terms and can be written as follows:

$$y_{it} = \lambda \sum_{i \neq j} w_{ij} y_{jt} + x_{it}\beta + \sum_{i \neq j} w_{ij} x_{jt}\theta + \alpha_i + \phi_t + \varepsilon_{it} \tag{4.16}$$

where θ is a vector of coefficients regulating the local spillover effect of spatially lagged covariates $\sum_{i \neq j} w_{ij} x_{jt}$, .

Alternatively, Spatial Durbin Error Model (SDEM) specification assumes no endogenous feedback in the dependent variable ($\lambda = 0$). Rather, this model accounts for unobserved heterogeneity in the spatial process by decomposing the error term into the effect carried through spatial autocorrelation in the model residuals and the nuisance parameter, thus yielding:

$$y_{it} = x_{it}\beta + \sum_{i \neq j} w_{ij} x_{jt}\theta + \alpha_i + \phi_t + u_{it} \tag{4.17}$$

$$u_{it} = \rho \sum_{i \neq j} w_{ij} y_{jt} + \varepsilon_{it} \tag{4.18}$$

where $\mathbf{X}$ is the matrix of independent covariates for county group or state i at time t, α_i is a county-group fixed effect. Comprising the residual term u_{it} is a spatially lagged error term, the spatial impact of which is scaled by the ρ parameter, and a nuisance term ε_{it}, such that $\varepsilon_{it} \sim \mathcal{N}(0, \sigma^2)$ *i.i.d.*

In the present analysis I avoid less parsimonious and more complex models such as spatial autoregressive model with autoregressive heteroskedastic disturbances (SARAR), among other candidates, that combine elements of both the endogenous outcome variable and the spatial dependence on unaccounted heterogeneity (Cliff & Ord, 1981). While some such models may potentially offer better fits, these nevertheless present great hindrance to interpretability of the coefficients. This is an important drawback in research aiming to disentangle the demographic, political, and socio-economic causes of the outcome of interest. In the course of the analysis I use statistical tools to isolate the best model candidates. I examine the results of fit and the residuals after each successive model and subject those to a number of statistical tests. Among these are spatial Hausman test (Millo & Piras, 2012; Pace & LeSage, 2008) to identify the presence of fixed or random effects in a panel setup, F tests for the presence of individual and time effects as well as randomization-based test for cross-sectional dependence (Millo, 2017) and Baltagi-Song-Jung-Koh LM test for serial autocorrelation in spatial panels (Baltagi et al., 2007). To interpret the models with global and local spatial spillover components, I make inferences based on the coefficient impact decomposition as per Piras (2014). In such cases the total effects incorporate the combination of direct (within) and indirect impacts (global and/or local regional spillovers). The equations terms containing the weights matrix $\mathbf{W}$ and the associated coefficients can be extracted via the decomposition of the matrix of partial derivatives $(\mathbf{I}-\lambda\mathbf{W})^{-1}\beta$ in the case of the SEM and SAR, or $(\mathbf{I}-\lambda\mathbf{W})^{-1}[\beta+\mathbf{W}\theta]$ in case of the SLX, SDM and SDEM models (Vega & Elhorst, 2015). In the former instance, the direct spatial effects of β will depend on the diagonal of the partial derivatives matrix, which has 0 on all off-diagonal elements, indicating the absence of indirect effects. In the latter case, the diagonal elements represent the direct effects, and the non-zero elements—indirect effects. The sum of the two constitutes the total effect. The significance of these impacts needs to be judged based on the outcome of a Monte Carlo simulation of the parameter estimates from the variance-covariance matrix, where each combination is drawn from the multivariate normal distribution (Golgher & Voss, 2016). Many details of the aforementioned panel models, the related diagnostics, and descriptive procedures implemented in this analysis are described Bouayad et al. (2018), Basile and Mínguez (2018), Bivand et al. (2021), and Millo and Piras (2012) and are run in version 4.2.2 of R software (R Core Team, 2022).

4.3.4 Variable transformations and selection

To help with the interpretability and to ensure that linear model assumptions of distributional normality, linearity, and absence of factor collinearity are satisfied, I transform many of the indicators mentioned in the preceding sections.

Perhaps the most pertinent of all are the proposed state health policy indicators. A unit increase in the raw overall SHP score or any of its thematic components generally represents some degree of increase in beneficial policy effect. However, since the positivity of SHP score I_s and its 10-year lagged version are longitudinally cumulative and cross-sectionally relative by design, the direct interpretation of one unit increase in SHP score would vary between time points. As such, using these measures in its raw forms would offer a misleading interpretation with respect to the ASDR or e_x outcomes. I standardize the raw overall SHP score $I_{s,Z}$ for every time point in the data to allow the interpretation to vary at standard

deviation increments of the health policy positivity distribution. Such a transformation offers an organic interpretation while respecting the cumulative nature of the indicator. The normal transformation of the time-lagged version of SHP score thus ensures that the changes in the SHP and mortality indicators at each time point can be evaluated on a level field.

Other explanatory indicators also require transformation prior to its use in the models to satisfy assumptions of linear modeling, to avoid collinearity and ensuring normality of continuous distributions. For instance, median household income across counties, and to a lesser extent among states, generally follows a somewhat right-skewed distribution as there is a small subset of counties with substantially higher household incomes throughout, albeit not to the extent observed in individual-level data. A natural logarithm transformation adjusts the distribution, approximating a normal one. Likewise, the government ideology score is skewed by virtue of shifting political landscape and the affinity of a state government toward the national political administration at any time. The prevalence of fair or poor health in the population, education, Gini index of income inequality and segregation measures also benefit from natural logarithmic transformations (though there is no apparent benefit for the transformation of the Divergence index in the state-level data).

Certain variable combination induce collinearity and thus lead to biased standard errors. According to the post-regression tests on variance inflation factors, 3 variables produce high degree of collinearity in all types of fully specified state-level models. Among them are the blue collar occupation prevalence ($VIF > 5$), the natural logarithm of Gini index ($VIF > 7$) and the proportion of college educated adults ($VIF > 8$). While centering of the blue collar occupation prevalence indicator brings the multi-collinearity in model coefficients to well within the acceptable threshold ($VIF < 4$), the latter two variables ought to be dropped from the fully specified state models. However, in the respective county group models only the proportion of the college-or-above educated adults needs to be dropped because it is highly collinear with the proportion of adults without a high school diploma, whether or not centering or transformations are applied. In my reading of the background literature on mortality determinants, I deem the proportion of the adult population without a high school diploma to be a more divisive factor in predicting mortality outcomes and, as such, I drop the proportion of college educated adults variable from the fully specified county group models as well. As far as the distinction between the state-level and county group models with respect to Gini income inequality coefficient is concerned, it is expected that the index would play a far more important role with increasing spatial granularity, as would other hyper-local phenomena such as residential segregation. Distinct local populations are more susceptible to various forms of socio-economic inequality than more aggregate groups. The statistical test results are in agreement with this postulation. Lastly, it is worth reiterating that neither transformations, nor dropping of the aforementioned variables contradicts the conceptual SCM models presented in chapter 2. These transformations only determine the forms of measurement but not the concepts that I judge to be of causal consequence for the outcome. Similarly, the exclusion of variables beyond the minimal adjustment set described in chapter 2 should not disrupt the causal links under the given theoretical paradigm, but rather may only impact the precision and fit of the models.

4.4 Results

In preparation for the discussion of the model performance in order to select the most optimal model, I first present the results of the of the descriptive analysis. Table 4.3 outlines the population-weighted summary measures for the indicators comprising the analysis data set, broken down by panel year and geography. Some notable differences can be registered between the states and county groups, specifically for the age-standardized mortality rates. ASDR for all sexes is lower among the county groups due to the low numerator counts, relative to the states. In contrast, the means for the life expectancy at birth and at age 65 are comparable across the state and the county group samples. Both-sex e(0) and e(65) increased between 1990 and 2018 from 75.39 to 78.83 years and from 17.27 to 19.58 years in states, respectively, and from 75.53 to 77.51 years and from 17.25 to 19.03 years accordingly for the county groups. As can be observed from the results, the growth in the weighted average life expectancy in states exceeds that of county groups by approximately half a year over the 3 decades but with nearly double the spread in values across the county group sample. This suggests the presence of and the increasing gap in geographic inequalities in life expectancy, particularly due to the deterioration in longevity progress in low-populated and rural counties. In terms of the differences by sex, the state-level gap between the weighted average female and male life expectancy at birth shrank from 6.9 years in 1990 to only 5.0 years in 2018. The same gap across county groups shrank from 7.04 years to 5.08 years. A similar dynamic by sex can be observed for the average life expectancy among older adults, albeit smaller in magnitude, down from 3.90 to 2.57 years for states and down from 3.87 to 2.63 years for the county groups. As with the ASDR and other measures discussed further on, the spread in county groups is somewhat greater due to higher heterogeneity in disaggregated outcomes.

Moving on to the three measures of state health policy, defined at the state level, the means and standard deviations for all the unstandardized SHP indicators at both geographic resolutions are generally concordant, notwithstanding the variability in the number of county groups contained within different states. A notable observation about the means of the I score and its time-lagged counterpart is that both decreased over time and roughly converged to a particular value close to 0.03 in the later years. This is an expected result given the design of the composite scores, as discussed in chapter 3, hence standardized versions of all SHP measures will be used within the scope of modeling to eliminate the effect of the absolute value convergence. Similarly, the RFNPPPPD indicator is seen to decline over time, but not monotonically. Rather, as explained previously, this occurred likely due to the data latency bias, as there has been an uptick in the fraction of new policies passed in the decade 2000–2009, relative to that of 1990–1999. State government ideology has seen a gradual shift from marginally liberal (i.e., scores above 50) to predominantly conservative between 1990 and 2018, though the highest shift has occurred in the 2010s.

The median age in the population for both men and women has increased from lower 30s to upper 30s during the 3 decades under study. This too is an expected outcome, owing to the population aging and increases in longevity over time. By association with aging, the projected prevalence of poor health has increased somewhat, likely due to the increase in the relative number of period person-years lived in the population with chronic health

conditions at older ages. The average increase in median age from 1990 to 2018 amounted to approximately 6 years and has been consistent across both levels of geographic disaggregation for both women and men. Concurrent with population aging the prevalence of poor health has increased by about 3% points, with 1%–1.5% points higher among women than among men. This is consistent with the notion that women's lifespan has increased due to lower mortality at older ages, which gave rise to the onset of chronic health conditions among older adults. The peak average increase of 1.2% points has occurred in the 2000s decade. In certain counties the maximum prevalence was double that of the highest prevalence among the states, reaching close to 45%–50% in the 1990, but decreased steadily in the following decades. These observations corroborate the trend where the prevalence of poor overall health has increased, albeit with fewer outliers that make high prevalence values exceptional, particularly across the county group sample.

Next, the area economic indicators revealed a widening gap in household income and the gradual increase in income inequality over the course of the 3 decades. This gap was wider at the county group level. The difference between the minimum and the maximum median household incomes in the 2018 inflation-adjusted dollars grew from around $95,000 in 1990 to about $121,000 at the end of the observation period. At the same time the gap has remained fairly stable at the state level, oscillating about $40,000. The increase in the scale-specific Gini coefficient by 3.5%–4% points in the same period has marked a trend toward a less equal distribution of income at both geographic resolutions. Additionally, the US state and county populations have shifted away from the traditional blue collar employment, with nearly 30% of the 1990 blue collar labor force shifting toward professional and highly skilled occupations by 2018. Nevertheless, the spread in blue collar occupation prevalence is considerable and highly location-specific at the county group level with up to 50% of the local workforce employed in unskilled or semi-skilled manual operations.

Examining the evolution of socio-economic changes across time and geography, the most monumental changes have occurred in the realm of educational attainment. The fraction of the state and county population without a high school diploma dropped by more than 50% since 1990, amounting to a mere 11.5% in 2018 in both the state and county group samples. While there has been a non-trivial spread around the average, county groups with high fractions of adults without a high school diploma represent outliers that are also exceptional in other ways (e.g., largely consisting of retirement communities). Conversely, the average proportion of the adult population with a college degree or higher has increased from 20.3% to 32.9% in the span of 28 years. Interestingly, while the distributional spread has been shrinking for the population proportion without a high school diploma, the variability in the proportion of state and county group populations with college-or-higher degrees has been on the rise. This gives credence to the idea that educational gap has been widening across the geography.

Lastly, I make a few remarks about the factors typically associated with deprivation in urbanized areas. Specifically, when it comes to the proportion of urban and African American populations, both indicators have remained broadly stationary. The slight increase in urbanization observed between 1990 and 2010 could have been a mixed result between the decennial census revision of classification for urban geography as well as a natural growth and/or migration of the population to the urban areas. Some evidence to the latter was

manifested though the slight fluidity in the Divergence index, where gentrification on one hand, and ethno-racial marginalization of particular places on the other led to shifts in the patterns of residential segregation. On the whole, the Divergence index was quite low in both state and county group samples (12%–22%), but individual instances and clustering of segregated ethno-racial enclaves might be potent predictors of socio-economic deprivation.

Table 4.3. Population-weighted descriptive summary statistics for the provided and calculated variables in the state ($N = 50$) and county group ($N = 2472$) data sets.

		States				County groups			
Variable description	Year	Mean	SD	Min	Max	Mean	SD	Min	Max
ASDR per 1000 (both-sex)	1990	11.49	0.90	9.36	13.07	11.30	1.12	8.79	15.87
	2000	10.76	0.96	8.64	12.81	10.42	1.34	7.27	15.42
	2010	9.30	1.01	7.65	11.65	8.92	1.69	5.39	15.86
	2018	9.08	1.09	7.31	11.70	8.70	1.96	4.53	20.20
ASDR per 1000 (female)	1990	9.24	0.71	7.59	10.44	9.05	0.83	6.79	11.99
	2000	9.04	0.79	7.07	10.50	8.75	1.05	5.96	12.49
	2010	7.92	0.85	6.15	9.83	7.57	1.38	4.61	13.32
	2018	7.69	0.93	5.89	10.00	7.34	1.62	3.82	17.13
ASDR per 1000 (male)	1990	14.76	1.29	11.47	17.06	14.50	1.63	10.68	21.32
	2000	13.17	1.27	10.62	16.15	12.67	1.77	8.35	19.71
	2010	11.09	1.24	9.49	14.20	10.63	2.05	6.15	18.77
	2018	10.78	1.30	9.03	14.12	10.35	2.37	5.18	24.80
e(0) (both-sex)	1990	75.39	1.36	73.00	78.33	75.57	1.67	68.53	79.58
	2000	76.84	1.41	73.51	79.55	77.08	1.98	68.71	82.07
	2010	78.67	1.63	74.78	81.00	79.11	2.70	69.39	85.72
	2018	78.83	1.79	74.56	81.50	79.35	3.17	66.43	87.73
e(0) (female)	1990	78.81	1.15	76.86	81.39	79.06	1.38	73.82	82.92
	2000	79.44	1.24	76.74	82.34	79.72	1.70	73.29	84.37
	2010	81.04	1.49	77.65	84.00	81.52	2.43	72.62	87.64
	2018	81.33	1.66	77.30	84.48	81.89	2.90	69.81	89.88
e(0) (male)	1990	71.87	1.61	68.92	75.44	72.02	1.98	63.34	76.99
	2000	74.11	1.59	70.18	76.79	74.35	2.23	64.11	80.28
	2010	76.21	1.76	71.86	78.50	76.62	2.87	66.34	84.04
	2018	76.32	1.91	71.62	78.62	76.81	3.34	63.12	85.98
e(65) (both-sex)	1990	17.27	0.63	16.30	19.10	17.43	0.76	14.98	20.05
	2000	17.72	0.71	16.49	19.84	18.05	0.97	15.07	21.95
	2010	19.18	0.80	17.43	21.07	19.65	1.38	15.17	23.86
	2018	19.58	0.89	17.67	21.75	20.10	1.70	13.31	26.48
e(65) (female)	1990	19.01	0.66	18.10	20.79	19.16	0.75	16.93	21.97
	2000	19.08	0.73	17.83	21.48	19.39	0.94	16.51	23.53
	2010	20.36	0.81	18.61	22.73	20.89	1.36	16.47	24.99
	2018	20.78	0.91	18.90	23.35	21.36	1.66	14.53	27.58
e(65) (male)	1990	15.11	0.71	13.97	17.36	15.29	0.85	12.65	18.17
	2000	16.05	0.74	14.52	18.02	16.39	1.04	13.16	20.53
	2010	17.76	0.81	15.87	19.21	18.16	1.38	13.84	22.78
	2018	18.21	0.88	16.09	19.91	18.67	1.71	11.84	25.34
I score[†]	1990	0.06	0.02	0.00	0.10	0.06	0.03	0.00	0.10
	2000	0.04	0.01	0.01	0.06	0.04	0.01	0.01	0.06
	2010	0.03	0.01	0.02	0.05	0.03	0.01	0.02	0.05
	2018	0.03	0.01	0.00	0.05	0.03	0.01	0.00	0.05
I score (10-year lag)[†]	1990	0.11	0.03	0.05	0.18	0.10	0.03	0.05	0.18
	2000	0.06	0.02	0.00	0.10	0.06	0.03	0.00	0.10
	2010	0.04	0.01	0.01	0.06	0.04	0.01	0.01	0.06
	2018	0.03	0.01	0.02	0.05	0.03	0.01	0.02	0.05
RFNPPPD[†]	1990	76.59	11.67	44.00	100	76.58	11.01	44.00	100
	2000	64.70	13.53	30.30	100	64.70	13.31	30.30	100
	2010	69.09	14.56	36.36	100	69.09	12.78	36.36	100
	2018	36.24	20.50	0.00	100	36.25	21.02	0.00	100
Gov't ideology[†]	1990	54.00	11.14	24.17	69.84	54.00	9.46	24.17	69.84
	2000	47.26	14.24	23.48	71.41	47.26	13.07	23.48	71.41
	2010	48.67	15.17	17.56	73.62	48.67	15.05	17.56	73.62
	2018	41.70	16.89	17.78	70.38	41.70	16.52	17.78	70.38
Median age (both-sex)	1990	32.25	1.52	26.00	36.00	32.50	3.56	21.00	55.00
	2000	34.86	2.03	27.00	38.00	34.83	3.93	22.00	53.00
	2010	37.23	2.29	29.30	42.80	37.36	5.16	21.90	63.00
	2018	38.36	2.35	31.10	44.80	38.62	5.53	23.50	68.00
Median age (female)	1990	33.53	1.92	27.00	38.00	33.77	3.90	21.00	57.00
	2000	36.08	2.02	28.00	40.00	36.05	4.01	22.00	55.00
	2010	38.48	2.40	29.80	43.80	38.58	5.25	21.60	63.80
	2018	39.66	2.53	31.80	46.20	39.89	5.68	22.80	68.50
Median age (male)	1990	31.06	1.34	25.00	34.00	31.26	3.27	21.00	52.00
	2000	33.28	1.88	27.00	37.00	33.62	3.91	21.00	52.00
	2010	35.94	2.19	28.80	41.60	36.11	5.16	22.30	62.00
	2018	37.07	2.16	30.40	43.40	37.36	5.47	24.10	67.50
% in poor/fair health (both-sex)	1990	15.24	3.18	10.75	23.45	15.16	5.79	6.13	45.62
	2000	15.96	3.07	11.01	23.13	15.98	5.44	7.49	41.23
	2010	17.24	3.26	11.63	23.95	17.28	5.20	8.08	37.94
	2018	17.95	3.49	11.64	24.56	18.05	5.21	8.53	37.49
% in poor/fair health (female)	1990	16.12	3.45	10.69	23.64	16.08	5.89	6.38	49.67
	2000	16.70	3.24	11.16	23.64	16.73	5.52	7.81	44.79
	2010	17.89	3.52	11.73	24.81	17.94	5.40	8.38	39.74
	2018	18.49	3.68	11.66	26.00	18.60	5.46	8.77	39.24

Table 4.3. Population-weighted descriptive summary statistics for the provided and calculated variables in the state ($N = 50$) and county group ($N = 2472$) data sets.

		States				County groups			
Variable description	Year	Mean	SD	Min	Max	Mean	SD	Min	Max
% in poor/fair health (male)	1990	14.29	2.98	10.58	23.38	14.21	5.88	5.75	46.66
	2000	15.17	2.93	10.85	23.42	15.18	5.49	6.93	42.59
	2010	16.54	3.01	11.48	23.05	16.58	5.10	7.77	37.92
	2018	17.37	3.30	11.60	23.76	17.46	5.09	8.88	36.70
Median HH income (2018 $)	1990	59482	11173	39482	81803	60445	17729	19231	115617
	2000	64642	9798	44870	83277	65888	17479	24511	120911
	2010	59096	9546	42603	79985	60377	16287	23507	133750
	2018	64311	10355	45120	84459	66160	18624	21223	142712
Gini index (% form)	1990	42.89	2.12	38.42	47.27	42.83	3.65	30.55	58.67
	2000	44.51	2.21	39.83	47.67	44.45	3.86	33.46	60.40
	2010	46.47	2.19	41.32	50.05	45.18	3.63	34.41	59.94
	2018	47.45	2.16	42.30	51.27	46.04	3.68	34.71	61.09
% blue collar workers	1990	28.77	4.66	22.20	38.28	29.21	13.69	10.60	63.83
	2000	24.82	3.91	18.39	33.08	25.13	10.91	7.63	55.96
	2010	21.47	3.32	15.97	29.20	21.78	9.70	6.31	49.15
	2018	21.90	3.28	15.86	28.27	22.19	9.77	6.26	50.66
% population Black	1990	11.93	9.55	0.30	35.56	11.94	14.40	0.00	85.61
	2000	12.22	9.86	0.30	36.34	12.23	14.65	0.03	84.64
	2010	12.53	9.81	0.41	37.02	12.55	14.71	0.07	84.14
	2018	11.99	9.31	0.47	36.44	11.99	14.22	0.02	85.98
Divergence index (% form)	1990	19.01	9.50	0.48	34.72	14.20	11.65	0.02	52.25
	2000	19.43	9.68	0.87	36.20	13.37	10.73	0.02	50.87
	2010	18.24	8.50	1.38	33.97	12.18	9.55	0.01	49.28
	2018	21.80	10.26	1.95	36.87	13.94	10.39	0.05	46.87
% population urban	1990	75.15	16.27	32.19	92.65	75.15	42.99	0.00	100
	2000	78.97	16.62	38.18	94.44	78.97	43.13	0.00	100
	2010	80.69	16.24	38.66	94.95	80.70	43.51	0.00	100
	2018	79.96	16.64	35.12	94.24	79.96	48.58	0.00	100
% w/out HS diploma	1990	24.77	5.73	13.37	35.72	24.82	11.52	5.29	68.44
	2000	19.63	4.66	11.67	27.14	19.68	8.97	3.50	65.30
	2010	14.33	3.70	7.92	19.23	14.38	6.93	2.48	55.12
	2018	11.50	3.05	6.02	16.08	11.52	5.62	1.94	43.86
% w/ college ed. or above	1990	20.29	3.78	12.33	27.23	20.25	9.25	3.69	53.42
	2000	24.36	4.32	14.83	33.19	24.33	10.85	4.92	60.48
	2010	28.42	4.86	17.91	38.97	28.40	12.24	5.70	71.25
	2018	32.85	5.32	21.30	44.53	32.89	13.79	6.84	75.84

† Denotes measures defined only at the state level.

In the next phase I investigate the associations between the aforementioned indicators. Both state and county group bivariate Pearson's correlations, as shown in Figures A.2.8 and A.2.9, reflect similar patterns of association. However, the geographic scale matters for the manifestation and strength of the summary indicators. For instance, although the relationship between mortality and low educational attainment (i.e., less than high school) was positive and moderate in strength, attenuating somewhat over time, the association has been more pronounced in the state data set. Likewise, the correlation between mortality and the prevalence of the blue collar workforce in the state data set has been more pronounced than in a county group data, despite demonstrating an increasing level of positive association over time among the units in both spatial resolutions. Though both the prevalence of low educational attainment and the blue collar workforce depict various facets of lower socio-economic profile, the counter-opposing trends *vis-à-vis* the relationship with mortality outcomes hint at the complementary value of these measures for further analysis. The association between the prevalence of blue collar occupations and educational attainment has also diminished to an insubstantial value after 1990 at both state and county group levels, indicating a broadly weakening dependence between the two key socio-economic mobility processes and changes in how each could impact mortality outcomes. In fact, the prevalence of poor health in an area was consistently and strongly ($r \approx 0.9$) correlated with the proportion of the adult population without a high school diploma, all while it weakened over time with respect to the proportion of blue collar occupations. Such a pattern is largely in agreement with the observations made in the past literature that low educational attainment is a precursor to poor health and mortality outcomes at both group and individual levels.

Among other associations with moderate-to-high strength were the prevalence of poor health in the population and the median household income. In both cases, the relationship between each of these measures and mortality has strengthened considerably over time. Higher median household income gradually has become more associated with lower mortality ($r = -0.7$), whereas higher prevalence of poor health in the population has become more strongly associated with higher mortality by 2018 ($r = 0.8$). Interestingly, the association between mortality and income inequality has diminished to a weak one ($r \approx 0.22$) over the three decades. Likewise, the Gini index and median household income have become increasingly disassociated with one another, though more so at the state than at the county group level. It could be indicative of existing localized patterns of inequality that otherwise vanish at more global aggregate levels. These observations complement the narrative behind the interplay between the location-specific contextual factors that impact mortality outcomes at a given time and beyond. Another important association that ought to be mentioned is that between mortality and state government ideology. The 2000s decade has become an inflection point in this relationship which turned from weakly positive in 1990 ($r \leq 0.2$) to a moderately negative by 2018 ($r \leq -0.5$), hinting at an intensifying relationship between the inclination toward liberalism in state governing entities and the decline of mortality. Alternatively, this could point to a stall in the recent progress toward mortality reduction as a unique characteristic of conservative strongholds.

Some associations have remained static over time. The proportion of African American population in an area has been positively and consistently associated to a moderate degree with the prevalence of population in poor general health. Given that mortality has been declining for all ethno-racial groups since the beginning of the observation period and the association between the proportion of ethno-racial minorities and area's mortality grew weaker over time, this implies a growing intra-group inequality over time whereby mortality gap increased due to additional factors characterizing various racial and minority subpopulations. Residential segregation, as measured by the Divergence index has been consistent and moderately-to-strongly correlated with the proportion of Black population in an area. This is an expected finding, despite that much could hide under the facade of an aggregate, where the clustering or confluence of deprivation-related factors could be traced to an increased mortality.

The patterns of spatial dependence

In this section I demonstrate the results of the descriptive spatial analysis to complement the exposition presented in the previous section. For a better understanding of the phenomena underlying the evolving mortality landscape over time, it is also necessary to assess whether these phenomena are spatially linked. If spatial dependence is detected in the data, as anticipated from the theoretical discussion in chapters 1 and 2, then an application of spatial treatment will be in order. To this end Table 4.4 shows Moran I coefficient of global spatial autocorrelation present in the state and county group data for the three mortality indicators for each sex over time. Moran statistic is bounded between -1 and 1, where its higher values are indicative of spatial clustering of similar values or attributes (or its dispersion, in the case of the negative range), and values close to 0 denote an absence of any defined spatial pattern. From a cursory inspection of the table, it is obvious that all mortality indicators

for each sex in both data sets were spatially lagged to a non-trivial extent. The lowest values of I, 0.47–0.48 for state-level female life expectancy at age 65, represented a moderate degree of spatial clustering among states with similar values for female e(65). Some of the highest values of $I > 0.7$ were registered throughout all 3 indicators and 3 sexes in the county group data, which points to a moderate-to-high degree of spatial autocorrelation. It is expected that geographic granularity of spatial units at the county group level would manifest slightly more pronounced patterns, as there exists a number of common factors that bounded particular ranges of mortality values together in relatively small regions.

Table 4.4. Univariate Moran I statistic estimates for presence of spatial autocorrelation for 3 mortality indicators.

		States			County groups		
Sex	Year	ASDR	e(0)	e(65)	ASDR	e(0)	e(65)
Both-sex	1990	0.612***	0.600***	0.541***	0.670***	0.675***	0.672***
	2000	0.648***	0.659***	0.554***	0.702***	0.699***	0.699***
	2010	0.612***	0.622***	0.548***	0.705***	0.700***	0.690***
	2018	0.580***	0.573***	0.517***	0.633***	0.637***	0.634***
Female	1990	0.613***	0.614***	0.566***	0.675***	0.684***	0.681***
	2000	0.625***	0.642***	0.528***	0.703***	0.706***	0.699***
	2010	0.576***	0.596***	0.486***	0.710***	0.709***	0.691***
	2018	0.562***	0.567***	0.472***	0.638***	0.649***	0.628***
Male	1990	0.634***	0.609***	0.561***	0.693***	0.683***	0.714***
	2000	0.672***	0.672***	0.598***	0.711***	0.699***	0.717***
	2010	0.648***	0.641***	0.615***	0.705***	0.695***	0.695***
	2018	0.604***	0.582***	0.579***	0.631***	0.630***	0.641***

Note: p-values have been generated based on a two-tailed comparison to the null.
* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$.

The results of the global analysis leave much unexplored nuance with regard to the particular patterns of clustering and/or spatial disassociation. To determine the types of clusters mortality indicators form in geographic space I summarize the local Moran I values and its local z-score comparisons in LISA maps, for states and county groups, as shown in Figures 4.5 and 4.6. As expected, based on the review of the background literature, the clustering of high age-standardized death rate values (or lower life expectancy values) is a predominant feature of the southern states and county groups, areas broadly encompassing the eastern part of the so-called Sun Belt and the Appalachia economic region. The coastal regions as well as many areas of the North fused into low-mortality clusters (relative to the global mean). In comparison to its local mean, low male mortality was spatially associated more prominently in the northern areas of the country in the 1990–2000, while high male mortality delineated the grouping of southern states during the same period. However, by 2010 the geographic male mortality has become less uniform across the country, such that significant clusters of states and counties either diminished in size or were no longer

significant in relation to the regional average. This erosion of uniformity of male mortality is best evidenced by the panels in Figure 4.6(g). Spatial association patterns of female mortality largely mirrored that of the male counterparts, albeit with lesser intensity. Figures 4.5(d) and 4.6(d) present a largely similar picture. The spatial autocorrelation in life expectancy indicators depicted an inverse scenario of that described by the age-standardized death rate, but mimicking the patterns with a few exceptions. Despite this, notable differences were present for the life expectancy at age 65. In Figures 4.5(c, f, i) and 4.6(c, f, i) the vast extent of high older-adult life expectancy clusters in the North and West of the country have gradually eroded away toward the more recent years, while the lower life expectancy at age 65 remained clustered and even somewhat expanded in the South. This could be indicative of compression of old-age mortality over time, with longer left tail in a handful of southern states, in which the growth in e(65) did not keep up with the rest of the country. Where it concerns the remaining longevity past the age of 65 years, female and male e(65) indicators exhibited dramatically distinct patterns of spatial clustering in 1990. However, the two grew increasingly similar toward the end of the observation period, possibly suggesting that the contextual determinants of old-age mortality have become more alike for female and male populations (see the comparison of panels (f) and (i) in Figures 4.5 and 4.6).

Instances of persistent clustering merit investigation because these were likely characterized by a particular suite of time-enduring causes, such as socio-economic deprivation. Certain other states and county groups either altered their mortality clustering patterns over time relative to the immediate neighbors or remained significantly distinct from the proximal high- or low-mortality clusters. For example, states such as Texas, Nevada and Alaska, shown filled in lighter shades of blue or pink in Figure 4.5, represented such cases where neighboring mortality has been significantly higher between 1990 and 2010 than the respective local neighbor average (under the *knn4* weighting setup), but changed substantially with respect to its neighbors in the subsequent period. Similar instances of divergent regional patterns of mortality could be noted in the county group data (Figure 4.6). As an example, the predominantly mountainous, rural county groups to the east of Salt Lake City, Utah, over time have become clusters of stagnating progress in longevity relative to the neighboring urban cores of Salt Lake City and Denver, Colorado. The growth in life expectancy in these urban centers outpaced the surrounding rural areas by a vast margin. An analogous dynamic could be traced to the populated coastal counties of California and southern Arizona versus the rural parts of the Central Valley and Inland Empire (California). LISA maps are merely a descriptive technique that nevertheless complement the outcome of the global dependence analysis for each mortality indicator and demonstrate the non-uniform nature of its outcomes. The examination of both the global Moran and LISA plots inform the further application of spatial modeling in order to properly account for the effects that arise from geographically proximal determinants of mortality.

One could also explore the results of the bivariate spatial dependence between the mortality indicators and its potential predictors. Figures 4.7, 4.8, and its respective state and county group counterparts by sex in the Appendices illustrate the longitudinal association between the 3 mortality measures and the spatially lagged versions of all the secondary variables used in the present analysis. These sets of figures effectively shadow the Pearson's correlation matrices but with the focus on indirect (geographically proximal) influences of

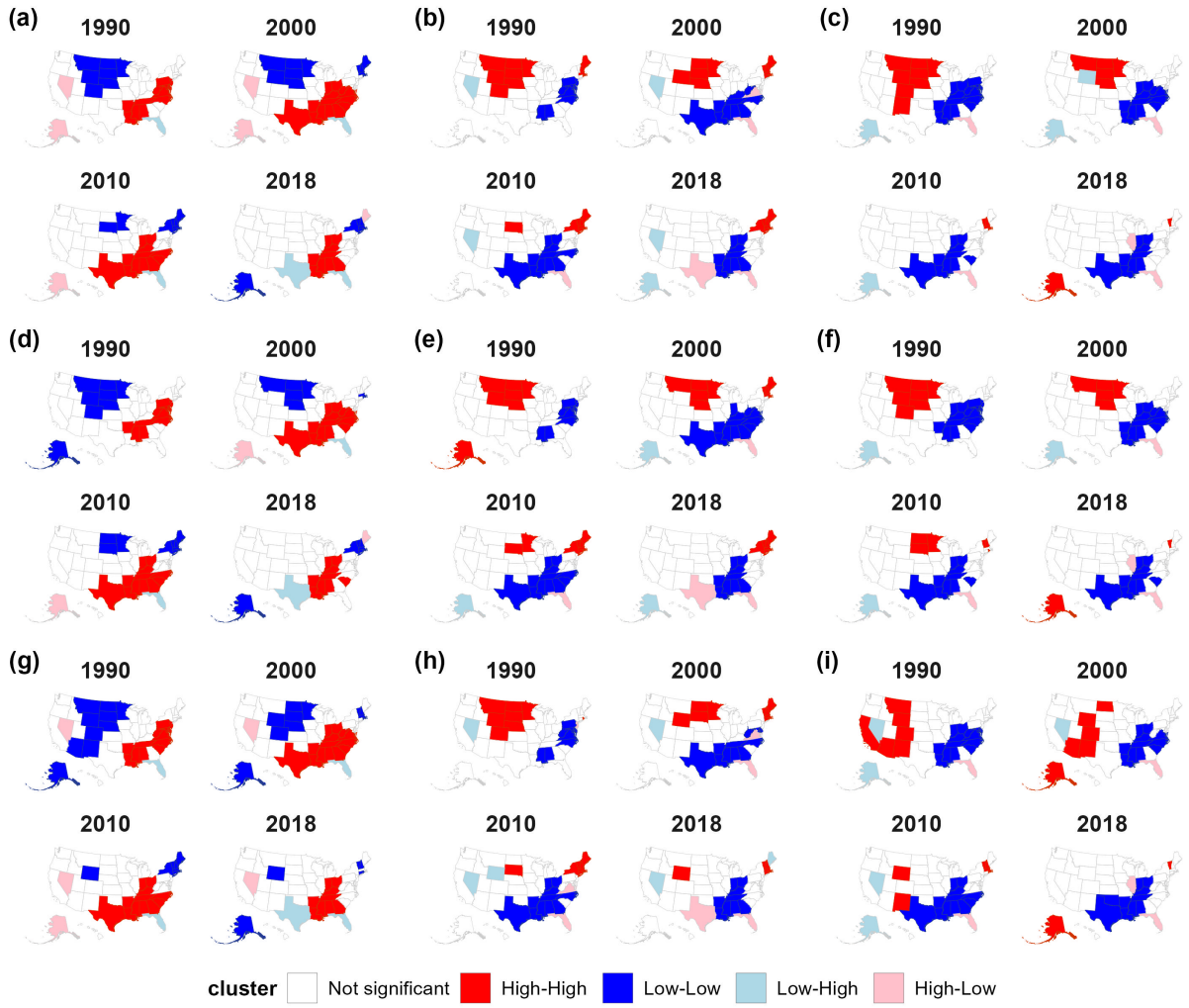


Figure 4.5. Univariate Local Indicators of Spatial Autocorrelation (LISA) cluster plot for state indicators of mortality, by year and sex.

First row: (a), (b), and (c) represent age-standardized mortality rate, life expectancy at birth, and life expectancy at age 65, respectively, for both sexes combined; Second row: (d), (e), and (f) represent the same indicators for females. Third row: (g), (h), and (i) represent the same indicators for males.

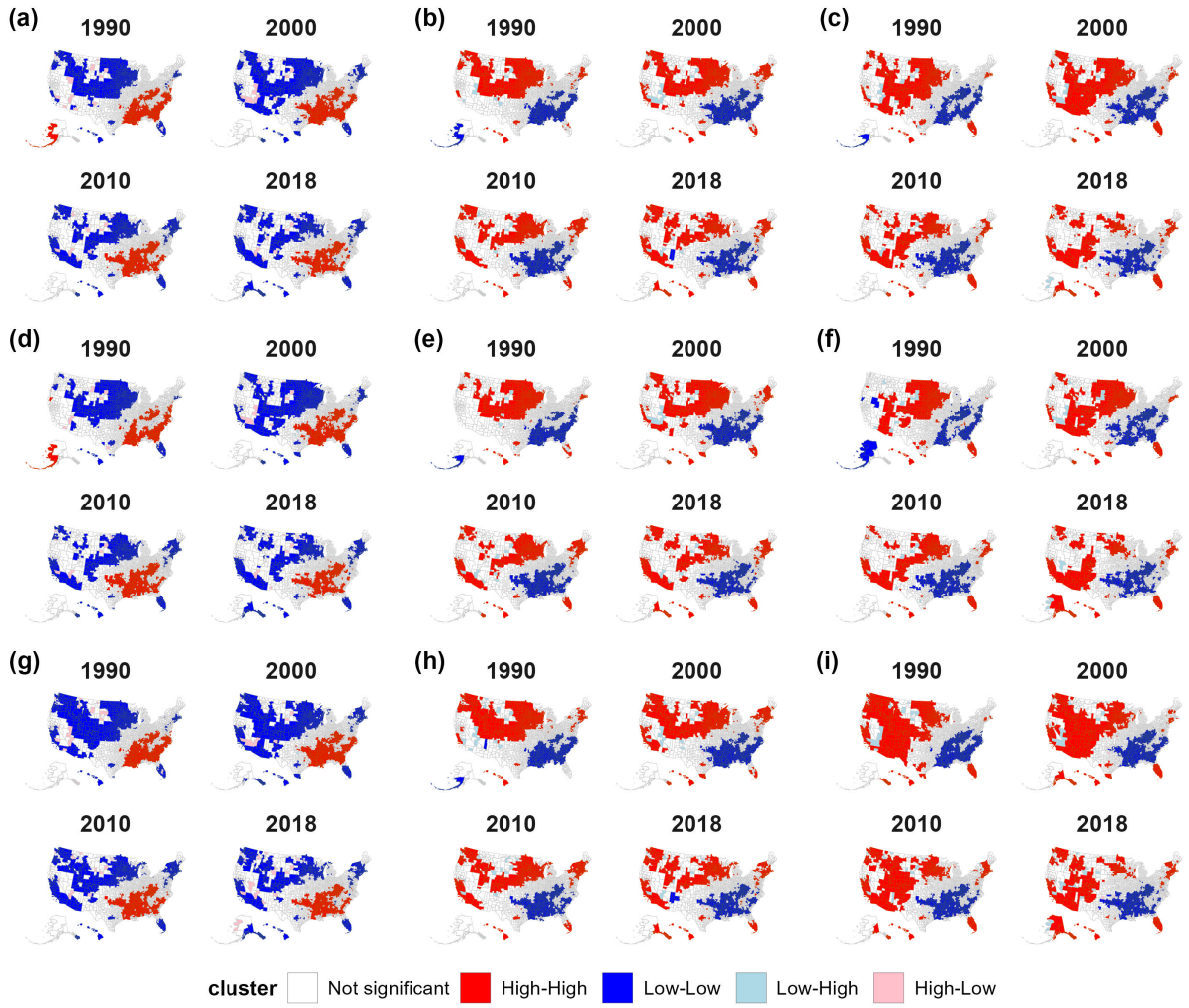


Figure 4.6. Univariate Local Indicators of Spatial Autocorrelation (LISA) cluster plot for county group indicators of mortality, by year and sex.

First row: (a), (b), and (c) represent age-standardized mortality rate, life expectancy at birth, and life expectancy at age 65, respectively, for both sexes combined; Second row: (d), (e), and (f) represent the same indicators for females. Third row: (g), (h), and (i) represent the same indicators for males.

the secondary variables on the 3 mortality outcomes. For instance, considering Figure 4.7(a), the overall relationship between the localized state SHP I score mean and ASDR changed from weakly positive to moderately negative over the 3 decades in the data. This opens a possibility that neighboring state health policies could have had a beneficial impact on mortality reductions in individual states. Of course, this is but a mere correlation, subject to further testing and inference in the presence of other factors. On the other hand, the RFNPPPD measure has been weakly associated with ASDR in every decade, as has been the median age and the proportion of urban population in a state. Interestingly, higher values of the neighboring state government ideology yielded a reversal in the association from a weakly positive to a weakly negative one over time. This observation highlights the reduction in the age-standardized death rate among states surrounded by less conservative governments. Simultaneously, the higher neighboring state and county group values for the proportion of blue collar occupations, proportion of the population that is African American, the proportion in poor/fair health, the Gini index, the Divergence index, and the percent of population without a high school diploma were all associated with increased levels of mortality to a weak and moderate degree. This confirms that selected measures are potentially useful for predicting indirect influences on both state and county group mortality for all 3 indicators for each sex.

The bivariate lagged associations with e(0) for both state and county groups largely resembled the reverse pattern of the ASDR for both sexes combined. Concerning the global associations with both-sex e(65), some variable pairings had marginally stronger magnitude than both-sex e(0), but not to a meaningfully significant extent. Likewise, male and female ASDR, e(0) and e(65) have all exhibited exceedingly similar strength of global spatial association with the aforementioned explanatory variables (see comparisons between Figures A.2.10–A.2.13). Additional "within" detail can be picked up from tracing the evolution of the associations in bivariate Moran scatter plots in Figures A.2.14–A.2.19. The dashed red lines represent spatially lagged means of the explanatory variables on the y-axes in all panels (one for each time period), the corresponding mortality indicator on the x-axes and the blue strokes representing the best fit lines, the slopes of which are the global bivariate Moran values shown earlier in the whisker plots (excluding the SHP indicators, as these have been examined exhaustively up until now). These sets of plots provide a deeper view of how susceptible the parts of the entire distribution were to the local spatial patterns of association with secondary variables. For instance, it could be observed in plot panels (e) that government ideology gradually shifted away from the negative spatial association with mortality to an increasingly positive over time, owing to the fact that more states have experienced a similar shift toward political conservatism concurrently with stagnating progress in regional mortality by 2018. This contrasts starkly with the tight distributions observed in 1990. The story behind educational attainment is equally peculiar. As most state and county group fractions of population without a high school degree (see panels (p)) have shrunk substantially from a band ranging between 17%–35% in 1990 to a range between 8%–15% in 2018. Although the slope of the best-fit line has become shallower between 1990 and 2018, the neighboring e(0) values extended from 5 to nearly 6 years apart among states and from 12 to over 16 years apart among county groups. This suggests that even marginally higher high school dropout rates were associated with an increasingly grim regional mortality prospects,

at the time when states and county groups with lowest high school dropout rates made incremental progress in population longevity. Yet, spatial associations with other factors, such as proportion of urban population and the segregation indices, did not change much overall, albeit the respective panels (k, l and m) demonstrate that the spread of values around the mean has broadened over the 3 decades. Much of the aforementioned evidence thus corroborates the narrative of broadening various forms of socio-economic inequalities, as noted in the background literature.

Characterizing the spatial data generating processes

All of the above empirical observations indicate the presence of substantial spatial dependence in the data. The purpose of this section is then to use statistical tools to identify the type of spatial dependence that would inform the choice of a spatial model. Table 4.5 summarizes the results of the spatial Lagrange multiplier tests for the types of spatial dependence based on the OLS fit for panel model residuals (Anselin & Bera, 1998) for minimally adjusted as well as the fully specified SCM models for natural logarithm of ASDR of every sex, each featuring one of 3 SHP indicators as main predictors. Analogous tables are not shown for e(0) and e(65) model outcomes as the respective results yield identical conclusions for each corresponding cell in the table. The first two numeric columns of the table contain the hypothesis test statistics indicating the preference for either the spatial lag or the spatial error process. Spatial lag is characterized by the endogenous dependence of the regressand (i.e., mortality indicators) and can be modeled via SAR and SDM models (non-exclusively), as described in the methods section of this chapter. The evidence of spatial autocorrelation in the error terms as indicated by the LM Error statistic may be accounted for by the SEM or SDEM models (again, non-exclusively). There is no analogous test for the spatial dependence among the exogenous regressors, but the rationale for using SLX, SDM, or SDEM models featuring spatial Durbin process relies primarily on a working theory and comparisons of fit with other models. Returning to Table 4.5, it is clear that both minimally adjusted and fully adjusted model results were inconclusive, as the null hypotheses assuming the absence of either respective spatial process were rejected with high confidence. For this reason Anselin et al. (1996) recommends the use of robust versions of the LM test. Examining the last two columns of Table 4.5 it becomes easier to assert preference for the spatial process selection. Amongst the minimally adjusted models the evidence for the presence of spatial lag was much more dominant, whereas only a couple of models could be fitted using spatial error components based on the preponderant failure to reject the null. At the same time, despite the strong indication that spatial lag is the correct paradigm for the fully specified SCM model, there was also more statistically significant support in favor of the spatial error process. This leaves a weak possibility that both types of spatial dependence could be valid for inclusion in and estimation of the aforementioned spatial models, rather than the spatial lag alone (with or without the spatial dependence in exogenous factors). From a purely speculative point of view, SDEM or SEM models perhaps seem more preferable than SDM, as the diffusion of mortality through an endogenous feedback is more difficult to explain. The error component in SDEM and SEM models then features a process accounting for the influence of the observed and unobserved heterogeneity in the spatial structure on the outcome.

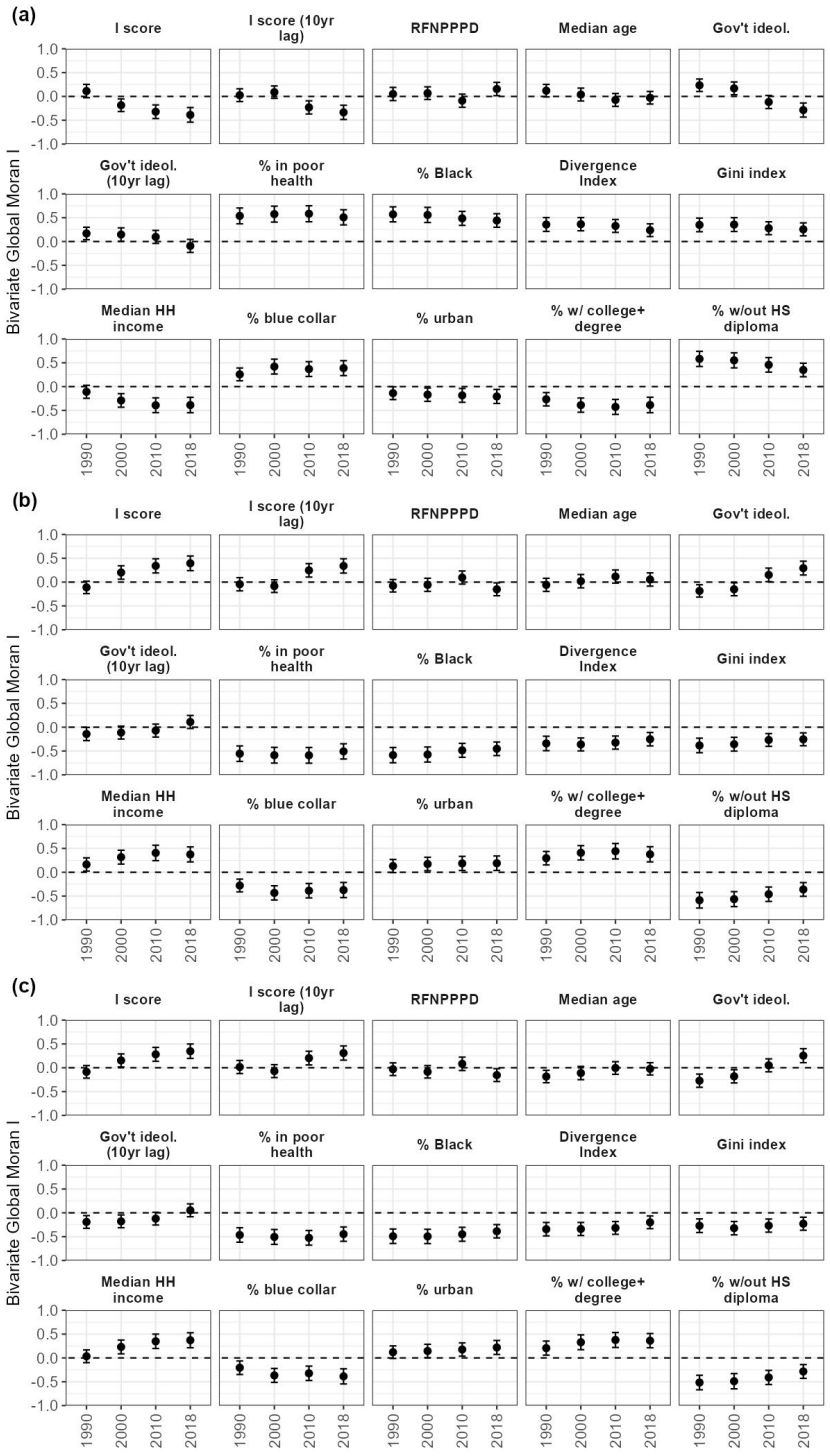


Figure 4.7. State level bivariate Moran statistic for pairwise combinations of 3 mortality indicators and various independent variables, by year (both sexes combined).

Note: (a) age-standardized mortality rate, (b) life expectancy at birth, (c) life expectancy at age 65

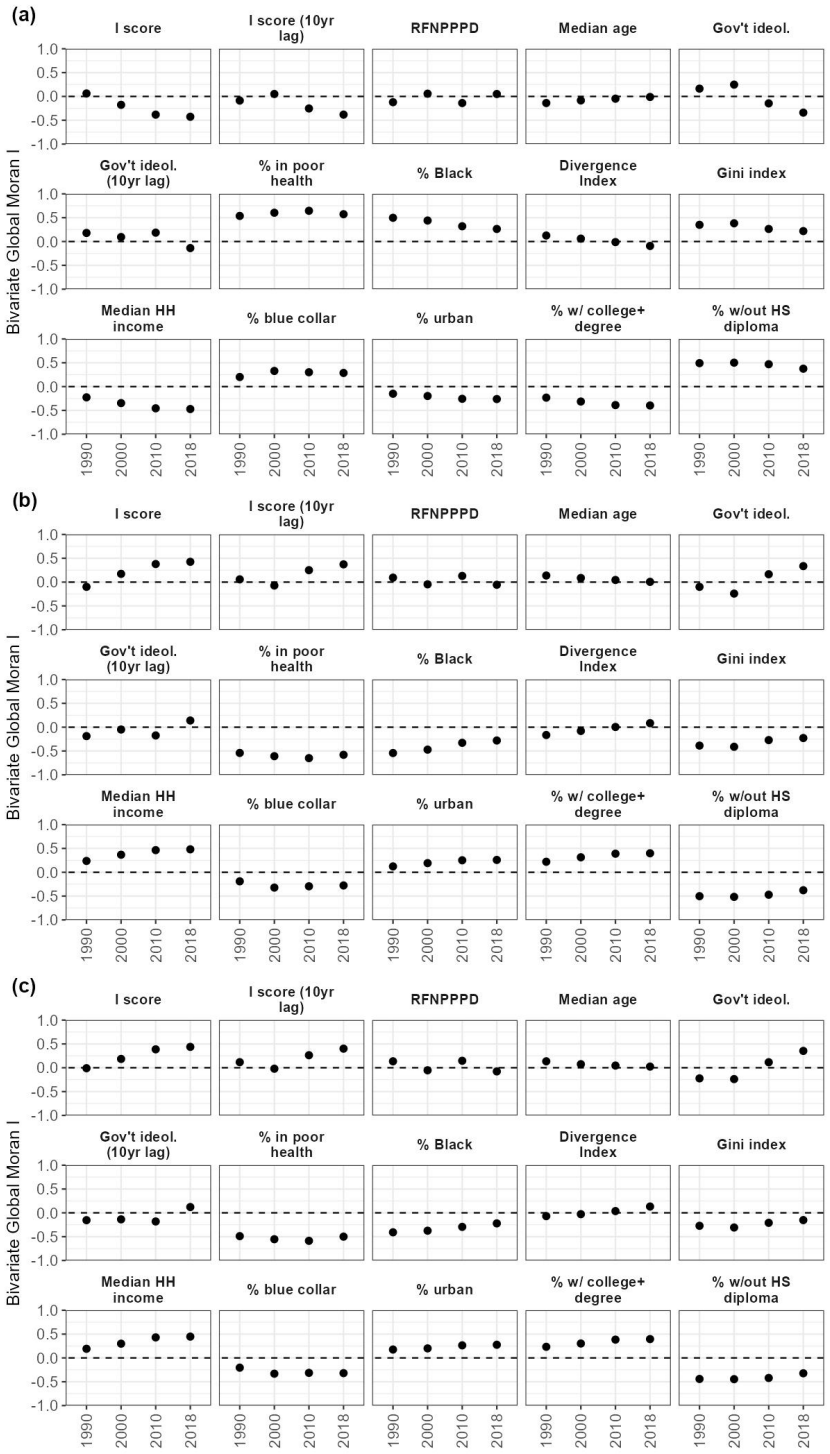


Figure 4.8. County group level bivariate Moran statistic for pairwise combinations of 3 mortality indicators and various independent variables, by year (both sexes combined).

Note: (a) age-standardized mortality rate, (b) life expectancy at birth, (c) life expectancy at age 65

Table 4.5. Spatial panel Lagrange Multiplier tests for type of global spatial dependence.

			States			
Adjustment	Sex	SHP indicator	LM lag	LM error	RLM lag	RLM error
Min. adjustment	Both-sex	I_Z	27 ($p < 0.0001$)	17 ($p < 0.0001$)	13 ($p < 0.0004$)	2 ($p < 0.1654$)
		$I_{t-10,Z}$	36 ($p < 0.0001$)	19 ($p < 0.0001$)	22 ($p < 0.0001$)	5 ($p < 0.0255$)
		$\tilde{\pi}_{10,Z}$	41 ($p < 0.0001$)	31 ($p < 0.0001$)	14 ($p < 0.0002$)	4 ($p < 0.0604$)
	Female	I_Z	36 ($p < 0.0001$)	22 ($p < 0.0001$)	17 ($p < 0.0001$)	3 ($p < 0.1088$)
		$I_{t-10,Z}$	44 ($p < 0.0001$)	23 ($p < 0.0001$)	27 ($p < 0.0001$)	6 ($p < 0.0115$)
		$\tilde{\pi}_{10,Z}$	49 ($p < 0.0001$)	37 ($p < 0.0001$)	16 ($p < 0.0001$)	3 ($p < 0.0764$)
	Male	I_Z	20 ($p < 0.0001$)	14 ($p < 0.0002$)	7 ($p < 0.0099$)	1 ($p < 0.4369$)
		$I_{t-10,Z}$	26 ($p < 0.0001$)	15 ($p < 0.0001$)	15 ($p < 0.0001$)	4 ($p < 0.0434$)
		$\tilde{\pi}_{10,Z}$	32 ($p < 0.0001$)	25 ($p < 0.0001$)	9 ($p < 0.0023$)	3 ($p < 0.1045$)
Full model	Both-sex	I_Z	24 ($p < 0.0001$)	14 ($p < 0.0002$)	10 ($p < 0.0015$)	0 ($p < 0.6767$)
		$I_{t-10,Z}$	32 ($p < 0.0001$)	20 ($p < 0.0001$)	12 ($p < 0.0006$)	0 ($p < 0.8038$)
		$\tilde{\pi}_{10,Z}$	32 ($p < 0.0001$)	22 ($p < 0.0001$)	10 ($p < 0.0016$)	0 ($p < 0.8075$)
	Female	I_Z	35 ($p < 0.0001$)	17 ($p < 0.0001$)	20 ($p < 0.0001$)	1 ($p < 0.2574$)
		$I_{t-10,Z}$	42 ($p < 0.0001$)	22 ($p < 0.0001$)	21 ($p < 0.0001$)	1 ($p < 0.3973$)
		$\tilde{\pi}_{10,Z}$	43 ($p < 0.0001$)	27 ($p < 0.0001$)	16 ($p < 0.0001$)	0 ($p < 0.6501$)
	Male	I_Z	15 ($p < 0.0001$)	10 ($p < 0.0015$)	5 ($p < 0.0246$)	0 ($p < 0.8288$)
		$I_{t-10,Z}$	19 ($p < 0.0001$)	13 ($p < 0.0003$)	6 ($p < 0.0162$)	0 ($p < 0.9125$)
		$\tilde{\pi}_{10,Z}$	21 ($p < 0.0001$)	15 ($p < 0.0001$)	7 ($p < 0.0095$)	0 ($p < 0.6199$)
			County groups			
Adjustment	Sex	SHP indicator	LM lag	LM error	RLM lag	RLM error
Min. adjustment	Both-sex	I_Z	5266 ($p < 0.0001$)	5057 ($p < 0.0001$)	215 ($p < 0.0001$)	7 ($p < 0.0107$)
		$I_{t-10,Z}$	5582 ($p < 0.0001$)	5389 ($p < 0.0001$)	203 ($p < 0.0001$)	10 ($p < 0.0018$)
		$\tilde{\pi}_{10,Z}$	5887 ($p < 0.0001$)	5695 ($p < 0.0001$)	198 ($p < 0.0001$)	6 ($p < 0.0133$)
	Female	I_Z	6406 ($p < 0.0001$)	6272 ($p < 0.0001$)	174 ($p < 0.0001$)	39 ($p < 0.0001$)
		$I_{t-10,Z}$	6844 ($p < 0.0001$)	6717 ($p < 0.0001$)	170 ($p < 0.0001$)	43 ($p < 0.0001$)
		$\tilde{\pi}_{10,Z}$	6998 ($p < 0.0001$)	6863 ($p < 0.0001$)	170 ($p < 0.0001$)	35 ($p < 0.0001$)
	Male	I_Z	4224 ($p < 0.0001$)	4044 ($p < 0.0001$)	181 ($p < 0.0001$)	1 ($p < 0.3655$)
		$I_{t-10,Z}$	4416 ($p < 0.0001$)	4258 ($p < 0.0001$)	157 ($p < 0.0001$)	0 ($p < 0.942$)
		$\tilde{\pi}_{10,Z}$	4789 ($p < 0.0001$)	4636 ($p < 0.0001$)	154 ($p < 0.0001$)	1 ($p < 0.3415$)
Full model	Both-sex	I_Z	4726 ($p < 0.0001$)	4262 ($p < 0.0001$)	466 ($p < 0.0001$)	2 ($p < 0.1318$)
		$I_{t-10,Z}$	4971 ($p < 0.0001$)	4526 ($p < 0.0001$)	453 ($p < 0.0001$)	8 ($p < 0.0056$)
		$\tilde{\pi}_{10,Z}$	5165 ($p < 0.0001$)	4635 ($p < 0.0001$)	530 ($p < 0.0001$)	1 ($p < 0.3246$)
	Female	I_Z	5621 ($p < 0.0001$)	5037 ($p < 0.0001$)	586 ($p < 0.0001$)	3 ($p < 0.0897$)
		$I_{t-10,Z}$	6025 ($p < 0.0001$)	5454 ($p < 0.0001$)	579 ($p < 0.0001$)	8 ($p < 0.005$)
		$\tilde{\pi}_{10,Z}$	6029 ($p < 0.0001$)	5368 ($p < 0.0001$)	662 ($p < 0.0001$)	1 ($p < 0.2511$)
	Male	I_Z	3797 ($p < 0.0001$)	3423 ($p < 0.0001$)	375 ($p < 0.0001$)	1 ($p < 0.3244$)
		$I_{t-10,Z}$	3917 ($p < 0.0001$)	3562 ($p < 0.0001$)	355 ($p < 0.0001$)	0 ($p < 0.8026$)
		$\tilde{\pi}_{10,Z}$	4183 ($p < 0.0001$)	3746 ($p < 0.0001$)	440 ($p < 0.0001$)	3 ($p < 0.0773$)

To further narrow down the list of model candidates I test the preference for the minimally adjusted SCM using the likelihood ratio (LR) test for nested models. Specifically, the minimally adjusted SCM specification is only a key subset of variables of the fully specified version and thus may not present value for inference beyond cursory inspection. LR statistics for all the proposed fixed effects panel models convincingly demonstrated the superior fit of the fully specified models over minimally adjusted models containing only 3 independent variables (see Table 4.6). Each cell in the table represents a statistically significant value of tests between the corresponding nested models for log-ASDR. This finding warrants omission of the outcomes of minimally adjusted models and places focus exclusively on the fully specified set, regardless of sex, geographic unit of analysis, or the main SHP predictor. Akin

to the panel LM tests, LR tables for e(0) and e(65) were not presented since they elicited the same conclusions as that with the models predicting log-ASDR based on its respective tests. However, for a better understanding of the difference between the two specifications, pooled ordinary least squares fits across all minimally adjusted models attained $R^2 \in (0.41 - 0.44)$ with only 3 degrees of freedom, thus capturing nearly two-thirds of the variance described by the fully specified models with at least 10 degrees of freedom. This is an encouraging result supporting the theoretical construct developed in earlier chapters. Finally, the non-spatial random effects (RE) model is presented for benchmarking, although, as I describe in the next section of the results presentation, random effects models are not appropriate within the scope of the current analysis.

Table 4.6. Results of likelihood ratio tests between minimally adjusted and fully specified model pairs for the logarithm of the age-standardized death rate and 3 state health policy indicators as regressors.

		States			County groups		
Sex	Model	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$
Both	Non-spatial FE	53.33(6)	50.35(6)	56.81(6)	704.48(7)	758.82(7)	793.05(7)
	Non-spatial RE	35.24(6)	50.32(6)	29.83(6)	1328.13(7)	1134.80(7)	1293.44(7)
	SEM FE	61.22(6)	59.69(6)	62.40(6)	427.47(7)	438.68(7)	434.84(7)
	SAR FE	52.11(6)	49.12(6)	52.33(6)	472.63(7)	493.47(7)	491.47(7)
	SLX FE	77.62(12)	70.74(12)	85.54(12)	1185.22(14)	1239.08(14)	1382.95(14)
	SDEM FE	83.62(12)	86.71(12)	90.16(12)	555.95(14)	558.17(14)	575.92(14)
	SDM FE	83.72(12)	82.13(12)	90.82(12)	607.27(14)	612.93(14)	641.87(14)
Female	Non-spatial FE	60.36(6)	57.27(6)	59.38(6)	732.42(7)	764.35(7)	817.23(7)
	Non-spatial RE	39.70(6)	47.28(6)	34.98(6)	1169.66(7)	1007.48(7)	1157.46(7)
	SEM FE	67.16(6)	68.06(6)	67.95(6)	344.35(7)	351.62(7)	350.88(7)
	SAR FE	62.33(6)	60.26(6)	58.95(6)	411.40(7)	426.66(7)	428.35(7)
	SLX FE	87.92(12)	81.55(12)	89.63(12)	1414.88(14)	1444.01(14)	1598.36(14)
	SDEM FE	91.27(12)	93.87(12)	92.95(12)	508.36(14)	500.30(14)	523.73(14)
	SDM FE	89.68(12)	88.57(12)	91.62(12)	584.29(14)	580.43(14)	612.56(14)
Male	Non-spatial FE	38.69(6)	38.88(6)	44.36(6)	660.73(7)	715.09(7)	755.22(7)
	Non-spatial RE	54.01(6)	70.38(6)	46.35(6)	1729.70(7)	1473.57(7)	1638.73(7)
	SEM FE	43.08(6)	43.64(6)	45.50(6)	420.69(7)	433.07(7)	428.61(7)
	SAR FE	35.28(6)	34.56(6)	37.65(6)	462.43(7)	482.67(7)	485.18(7)
	SLX FE	63.34(12)	63.79(12)	72.72(12)	1045.92(14)	1107.81(14)	1258.06(14)
	SDEM FE	67.55(12)	72.80(12)	76.38(12)	535.05(14)	544.08(14)	562.15(14)
	SDM FE	67.77(12)	69.99(12)	75.74(12)	576.12(14)	588.48(14)	618.95(14)

Note: All LR(df) statistics reported in this table are significant at the 95% confidence level.

Model comparisons

Having selected the final set of dependent and independent variables (i.e., the fully specified SCM set) and its respective transformations, I run all the proposed quantitative models and review the model outcomes in light of the associated pre- and post-regression tests. Though

I indicated that specific spatial models may be more preferable than other spatial or non-spatial ones, it is informative to examine and to interpret the similarities and differences that lead to advantages in one model over the other. Sometimes it is not possible to completely rule out all the options in favor of one model on the basis of one or two initial tests. I therefore present the results of all pre-selected fully specified model candidates predicting the 3 mortality outcomes at the state and county group levels, for each sex separately and both sexes combined, and for each state health policy indicator as a focal independent predictor.

Table 4.7. Fully specified state-level non-spatial fixed effects models estimating both-sex mortality indicators ($log(ASDR)$, $e(0)$, and $e(65)$) featuring various specifications of state health policy.

	$log(ASDR)$			$e(0)$			$e(65)$		
Variable	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$
SHP indicator	−0.023***	−0.020***	0.001	0.339***	0.323***	−0.001	0.169***	0.139***	−0.024
	(0.01)	(0.00)	(0.00)	(0.08)	(0.06)	(0.04)	(0.04)	(0.03)	(0.02)
Median age	−0.006	−0.006	−0.007	0.068	0.056	0.074	0.068**	0.071**	0.071*
	(0.00)	(0.00)	(0.00)	(0.06)	(0.05)	(0.06)	(0.03)	(0.03)	(0.03)
log(gov't ideology)	−0.017	0.009	−0.029**	0.166	−0.134	0.344*	0.160*	−0.051	0.250***
	(0.01)	(0.01)	(0.01)	(0.15)	(0.17)	(0.15)	(0.07)	(0.08)	(0.07)
log(% in poor health)	0.058	0.048	0.107	−0.242	0.072	−1.008	−0.756	−0.735	−1.078*
	(0.05)	(0.05)	(0.06)	(0.86)	(0.85)	(0.89)	(0.40)	(0.42)	(0.42)
log(% Black)	0.021	0.019	0.010	−0.334	−0.313	−0.177	−0.205*	−0.185*	−0.114
	(0.01)	(0.01)	(0.01)	(0.19)	(0.19)	(0.20)	(0.09)	(0.09)	(0.10)
D index	−0.004*	−0.004*	−0.003	0.065**	0.064**	0.054*	0.019	0.019	0.011
	(0.00)	(0.00)	(0.00)	(0.02)	(0.02)	(0.03)	(0.01)	(0.01)	(0.01)
log(med. HH income)	0.107	0.110	0.142*	−1.654	−1.762	−2.120	−0.978*	−0.970*	−1.258*
	(0.06)	(0.06)	(0.07)	(1.03)	(0.99)	(1.09)	(0.48)	(0.49)	(0.51)
% blue collar	−0.005*	−0.006**	−0.007**	0.049	0.065	0.081*	0.043*	0.054**	0.059***
	(0.00)	(0.00)	(0.00)	(0.04)	(0.03)	(0.04)	(0.02)	(0.02)	(0.02)
% urban population	−0.002	−0.002*	−0.003**	0.038*	0.038*	0.052**	0.017*	0.019*	0.026**
	(0.00)	(0.00)	(0.00)	(0.02)	(0.02)	(0.02)	(0.01)	(0.01)	(0.01)
log(% w/out HS diploma)	−0.098*	−0.082*	−0.114**	1.887**	1.512*	2.127**	0.616*	0.536	0.721*
	(0.04)	(0.04)	(0.04)	(0.64)	(0.64)	(0.67)	(0.30)	(0.31)	(0.31)
Joint Wald test	96***	93***	66***	84***	95***	60***	108***	94***	81***
df	10	10	10	10	10	10	10	10	10
Wooldridge test	85.43***	73.49***	114.68***	122.51***	110.8***	187.24***	42.15***	34.7***	50.7***
Hausman test	15***	16***	18***	18***	17***	20***	14***	15***	17***
LM Breusch-Pagan test	54***	25**	94***	68***	64***	66***	15	166***	4
Individual eff. F test	8.91***	8.97***	8.23***	7.87***	8.35***	7.22***	10.7***	10.47***	10.16***
Time eff. F test	51.87***	50.41***	50.72***	46.34***	44.81***	46.31***	53.43***	52.47***	51.98***
Two-way eff. F test	13.59***	13.26***	11.88***	11.49***	11.99***	10***	20.35***	18.85***	18.56***
R^2 (overall)	0.413	0.405	0.325	0.38	0.408	0.304	0.441	0.408	0.37
Adj. R^2 (within)	0.147	0.136	0.02	0.099	0.141	−0.011	0.188	0.14	0.085
AIC	−826	−823	−798	287	278	310	−17	−6	7
BIC	−618	−616	−591	495	485	518	190	202	214

$^{*}p < 0.05$, $^{**}p < 0.01$, $^{***}p < 0.001$.

Table 4.8. Fully specified county group-level non-spatial fixed effects models estimating both-sex mortality indicators ($log(ASDR)$, $e(0)$, and $e(65)$) featuring various specifications of state health policy.

	$log(ASDR)$			$e(0)$			$e(65)$		
Variable	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$
SHP indicator	−0.018***	−0.015***	0.003***	0.244***	0.214***	−0.028**	0.148***	0.119***	−0.034***
	(0.00)	(0.00)	(0.00)	(0.02)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)
Median age	−0.007***	−0.007***	−0.007***	0.073***	0.074***	0.078***	0.071***	0.073***	0.074***
	(0.00)	(0.00)	(0.00)	(0.01)	(0.01)	(0.01)	(0.00)	(0.00)	(0.00)
log(gov't ideology)	−0.017***	0.007**	−0.026***	0.162***	−0.126**	0.295***	0.181***	−0.049*	0.263***
	(0.00)	(0.00)	(0.00)	(0.04)	(0.04)	(0.04)	(0.02)	(0.02)	(0.02)
log(% in poor health)	−0.136***	−0.141***	−0.123***	2.148***	2.208***	1.964***	1.070***	1.124***	0.970***
	(0.01)	(0.01)	(0.01)	(0.16)	(0.16)	(0.16)	(0.08)	(0.08)	(0.08)
log(% Black)	0.005***	0.003*	0.003*	−0.100***	−0.077***	−0.078***	−0.031**	−0.017	−0.018
	(0.00)	(0.00)	(0.00)	(0.02)	(0.02)	(0.02)	(0.01)	(0.01)	(0.01)
log(D index)	−0.007***	−0.008***	−0.008***	0.120***	0.137***	0.132***	0.039***	0.049***	0.046***
	(0.00)	(0.00)	(0.00)	(0.02)	(0.02)	(0.02)	(0.01)	(0.01)	(0.01)
log(med. HH income)	−0.104***	−0.116***	−0.095***	1.496***	1.637***	1.386***	0.775***	0.894***	0.700***
	(0.01)	(0.01)	(0.01)	(0.15)	(0.15)	(0.15)	(0.08)	(0.08)	(0.08)
log(GINI index)	−0.184***	−0.191***	−0.206***	2.582***	2.616***	2.881***	1.488***	1.579***	1.668***
	(0.02)	(0.02)	(0.02)	(0.26)	(0.26)	(0.26)	(0.14)	(0.14)	(0.14)
% blue collar	−0.001***	−0.001***	−0.001***	0.017***	0.018***	0.019***	0.010***	0.012***	0.011***
	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
% urban population	−0.001***	−0.001***	−0.001***	0.019***	0.019***	0.020***	0.011***	0.011***	0.012***
	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
log(% w/out HS diploma)	−0.014*	−0.008	−0.010	0.189*	0.102	0.142	0.156***	0.108*	0.120**
	(0.01)	(0.01)	(0.01)	(0.08)	(0.08)	(0.09)	(0.05)	(0.05)	(0.05)
Joint Wald test	1476***	1368***	1237***	1166***	1153***	973***	1677***	1489***	1453***
df	11	11	11	11	11	11	11	11	11
Wooldridge test	1300.64***	1251.72***	1351.73***	1537.6***	1459.07***	1594.71***	959.32***	933.8***	994.72***
Hausman test	725***	711***	730***	772***	747***	773***	695***	695***	698***
LM Breusch-Pagan test	1972***	2067***	885***	2215***	2353***	1792***	1396***	1319***	1627***
Individual eff. F test	7.66***	7.23***	7.82***	7.46***	7.19***	7.54***	8.03***	7.55***	8.3***
Time eff. F test	745.42***	659.85***	742.06***	641.04***	571.34***	638.75***	894.1***	801.48***	896.14***
Two-way eff. F test	9.21***	8.79***	9.15***	8.73***	8.51***	8.62***	10.44***	9.81***	10.5***
R^2 (overall)	0.166	0.156	0.143	0.136	0.135	0.116	0.185	0.167	0.164
Adj. R^2 (within)	−0.114	−0.127	−0.144	−0.154	−0.156	−0.181	−0.089	−0.112	−0.117
AIC	−27972	−27851	−27702	26162	26177	26388	13912	14119	14159
BIC	−10076	−9954	−9805	44059	44074	44285	31809	32016	32056

$^{*}p < 0.05$, $^{**}p < 0.01$, $^{***}p < 0.001$.

Tables 4.7 and 4.8 show the output of panel fixed effects models for states and county groups, respectively, regressing each of the 3 SHP measures on 3 both-sex mortality/longevity indicators. Complementary models for females and males are presented in Tables A.2.2–A.2.5 in the Appendices. The coefficients of these models are not of much material significance from the point of view of inference, as it has been determined that spatial specifications enable superior fit. A series of tests and diagnostics that follow each model are instructive to validate the selection of model alternatives. First, the highly statistically significant result of the joint Wald test against the null hypothesis that all coefficients are zero indicated that the coefficients of the models are jointly non-trivial. In fact, all models with SHP indicators remained significant in the presence of the remaining contextual factors based on the state and county group data, whereas $RFNPPPD$ lacked significance across the 3 state-level models for each mortality indicator for females, males, and both sexes combined. This is an anticipated finding, given the preliminary analyses carried out in chapter 3. Based on the minimization of AIC and BIC criteria, in both state and county group data both-sex models featuring the I score as the main predictor yielded the best fit for the log-transformed ASDR and e(65). However, the lagged version of the I score offered a better fit among the models predicting e(0) at the state level, while it was on par with the concurrent I score measure among the county group models. Identical conclusions could be drawn from the AIC/BIC fit diagnostics for female and male models.

Aside from the cursory examination of the basic panel fixed effects model coefficients, the remaining post-regression tests revealed additional clues about the data generating process. Due to the longitudinal setup of the data, it is important that proper adjustments and procedures are followed to ensure the best possible fit and proper interpretability of the coefficients. The outcomes of the two-way non-spatial panel Hausman test for the fixed effects models in Tables 4.7, 4.8 and its counterparts by sex (see Appendices) indicated a strong preference for the fixed effects (H_a is that endogenous correlation between the predictors and the unique error term are present), as opposed to the random effects. In fact, none of the models contain independent time-invariant regressors (e.g., sex, race, religion, etc.) for which the coefficients would need to be estimated. The fixed effects approach is theoretically appropriate in the context where units of analysis are heterogeneous population groups in states and county groups, whose explicit exogenous attributes are not immutable over time. Fixed effects simultaneously strip away any unobserved time- and individual/group-invariant heterogeneity, thus absorbing the correlation between the regressors with the regressors and the error term into dedicated model parameters. This is a conclusion established for virtually all models and thus helped to further narrow down the list of potential candidates. Next, a series of tests for individual, time and two-way effects collectively pointed to the presence of both time and group effects in the data. A set of highly statistically significant results across all the models informed the decision to index the panel by years and units (state ID or county group ID). Finally, Wooldridge test for serial correlation (Wooldridge, 2010) and Lagrange multiplier adaptation of the Breusch-Pagan test for cross-sectional dependence (Baltagi et al., 2012) both demonstrated highly significant outcomes. Both of these tests highlighted a degree of correlation that, when unadjusted for through clustering and robust standard errors, could potentially render the estimated variances invalid. The collective evidence furnished by all the aforementioned tests completed the picture. It enables a

search of a much narrower spectrum of more advanced quantitative model specifications that would offer better accuracy and precision for the SHP-mortality effect identification. In summary, the fully specified (in terms of SCM) two-way fixed effects model with state-clustered, heteroskedasticity and serial correlation adjusted standard errors need to be applied.

Spatial panel 2-way FE models

In this part I assess the results of the spatial panel two-way fixed effects models. Earlier the LM tests on the type of spatial dependence indicated that spatial lag is a preferred type of association between the neighboring units as determined by the previously defined spatial weights matrices for each data set. However, the spatial error dependence could not be completely ruled out without examining additional evidence. Moreover, spatial lag X (SLX) process among the regressors could not be tested using the Lagrange multiplier tests, but it may be a crucial determinant of the fit, as partially evidenced by the bivariate Moran I associations discussed above. Moreover, the spatial lag X (also referred to as a spatial Durbin component) is also an integral part of the SDM and SDEM models. For the brevity of presentation in this section I present both-sex SDM and SDEM regressions for states (Tables 4.9 and 4.10) and county groups (Tables 4.11 and 4.12), the two most saturated model types being considered. All SAR, SLX, and SEM models as well as female and male versions of SDM and SDEM for states and county groups could be found in Tables A.2.6–A.2.31 in the Appendices.

One piece of evidence that distinguishes between the state and county models is the result of the randomization based test for cross-sectional dependence (RW test). This test is a modification of the Pesaran's cross-sectional test with adjustments introduced to yield consistent results in the presence of global spatial or serial autocorrelation (Millo, 2017). Furthermore, RW test is designed to test for local cross-sectional independence under the null hypothesis. The rejection of the null hypothesis based on the p-value provides evidence for a presence of local spatial dependence in panel data. Comparing the result of the RW test between Table 4.9 for states and Table 4.11 for county groups (the results of the test only differ between the corresponding combinations of mortality indicators and SHP variables in states and county groups. In other words, these are identical across all tables for states and separately for county groups featuring spatial fixed effects specifications. Examining the high p-values derived from the RW test for the state-level outcome-SHP predictor combinations, it is increasingly evident that the state data did not feature significant local cross-sectional dependence, whereas the county group data did. This is consistent with the expectation that local phenomena is likely to be associated among similar smaller units (in this case, spatially proximal counties). States as administrative entities are often too large to demonstrate significant cross-sectional similarities at a local level consistently across the board. Significant indication of local dependence warrants the addition of a Durbin effect generated by spatially lagged regressors, thus lending support for the use of SLX, SDM and SDEM models. Next, all tables containing spatial models include the result of the Baltagi-Song-Jung-Koh (BSJK C.2) test for panel serial correlation robust to the presence of spatial and random effects (Baltagi et al., 2007). Both state and county group data showed a non-trivial degree of serial autocorrelation, calling for the inclusion of robust errors in the models. The magnitude of the significant spatial autocorrelation parameters λ in SAR/SDM and ρ in SEM/SDEM

Table 4.9. Fully specified state-level SDM models estimating both-sex mortality indicators ($log(ASDR)$, $e(0)$, and $e(65)$) featuring various specifications of state health policy.

	$log(ASDR)$			$e(0)$			$e(65)$		
Variable	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$
SHP indicator	-0.014^{***}	-0.012^{***}	0.000	0.223^{***}	0.208^{***}	0.015	0.110^{***}	0.079^{***}	-0.016
	(0.00)	(0.00)	(0.00)	(0.06)	(0.05)	(0.03)	(0.03)	(0.02)	(0.01)
Median age	0.001	0.000	0.001	-0.043	-0.028	-0.050	0.011	0.019	0.008
	(0.00)	(0.00)	(0.00)	(0.04)	(0.04)	(0.04)	(0.02)	(0.02)	(0.02)
log(gov't ideology)	-0.010	0.016^{*}	-0.014	0.067	-0.224	0.130	0.098	-0.104	0.134^{*}
	(0.01)	(0.01)	(0.01)	(0.11)	(0.12)	(0.12)	(0.05)	(0.06)	(0.05)
log(% in poor health)	0.025	-0.005	0.049	0.051	0.615	-0.429	-0.404	-0.239	-0.535
	(0.04)	(0.04)	(0.04)	(0.63)	(0.64)	(0.64)	(0.28)	(0.30)	(0.29)
log(% Black)	0.037^{***}	0.040^{***}	0.036^{***}	-0.548^{***}	-0.590^{***}	-0.561	-0.293^{***}	-0.320^{***}	-0.265^{***}
	(0.01)	(0.01)	(0.01)	(0.16)	(0.16)	(0.17)	(0.07)	(0.07)	(0.08)
D index	-0.002	-0.002	-0.001	0.030	0.032	0.028	0.005	0.005	0.000
	(0.00)	(0.00)	(0.00)	(0.02)	(0.02)	(0.02)	(0.01)	(0.01)	(0.01)
log(med. HH income)	0.030	0.030	0.051	-0.387	-0.382	-0.684	-0.625	-0.661	-0.800^{*}
	(0.05)	(0.05)	(0.05)	(0.85)	(0.83)	(0.86)	(0.38)	(0.39)	(0.39)
% blue collar	-0.004^{*}	-0.004^{**}	-0.005^{**}	0.040	0.045	0.053^{*}	0.038^{**}	0.041^{***}	0.045^{***}
	(0.00)	(0.00)	(0.00)	(0.03)	(0.03)	(0.03)	(0.01)	(0.01)	(0.01)
% urban population	-0.001	-0.001	-0.001	0.010	0.012	0.016	0.005	0.005	0.008
	(0.00)	(0.00)	(0.00)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)
log(% w/out HS diploma)	-0.159^{***}	-0.152^{***}	-0.161^{***}	2.877^{***}	2.621^{***}	2.864^{***}	0.984^{***}	0.954^{***}	1.004^{***}
	(0.03)	(0.03)	(0.03)	(0.53)	(0.55)	(0.55)	(0.24)	(0.25)	(0.25)
Lagged indicators									
SHP indicator	-0.010	-0.013^{*}	0.000	0.028	0.165	0.003	0.088	0.090	-0.020
	(0.01)	(0.01)	(0.00)	(0.13)	(0.10)	(0.06)	(0.06)	(0.05)	(0.03)
Median age	-0.010^{*}	-0.005	-0.010^{*}	0.164^{*}	0.088	0.175^{*}	0.060	0.030	0.065
	(0.00)	(0.00)	(0.00)	(0.08)	(0.08)	(0.08)	(0.04)	(0.04)	(0.04)
log(gov't ideology)	-0.005	-0.023	-0.006	0.064	0.399	0.070	0.059	0.141	0.055
	(0.01)	(0.01)	(0.01)	(0.21)	(0.23)	(0.21)	(0.09)	(0.11)	(0.09)
log(% in poor health)	0.130	0.162^{*}	0.176^{*}	-2.347	-2.501^{*}	-2.901^{*}	-0.984	-1.254^{*}	-1.274^{*}
	(0.07)	(0.08)	(0.08)	(1.24)	(1.23)	(1.24)	(0.56)	(0.58)	(0.57)
log(% Black)	-0.038	-0.049^{*}	-0.071^{***}	0.735^{*}	0.705^{*}	1.146^{***}	0.163	0.313^{*}	0.422^{**}
	(0.02)	(0.02)	(0.02)	(0.34)	(0.32)	(0.32)	(0.15)	(0.15)	(0.14)
D index	0.000	0.004	0.002	-0.013	-0.060	-0.031	0.004	-0.017	-0.015
	(0.00)	(0.00)	(0.00)	(0.04)	(0.04)	(0.04)	(0.02)	(0.02)	(0.02)
log(med. HH income)	-0.050	-0.012	0.003	0.182	-0.129	-0.299	1.300	1.053	0.744
	(0.09)	(0.09)	(0.09)	(1.51)	(1.44)	(1.56)	(0.67)	(0.67)	(0.70)
% blue collar	0.007^{*}	0.004	0.006	-0.101	-0.068	-0.098	-0.060^{*}	-0.034	-0.044
	(0.00)	(0.00)	(0.00)	(0.05)	(0.05)	(0.06)	(0.02)	(0.02)	(0.03)
% urban population	-0.002	-0.001	-0.003^{*}	0.054^{*}	0.032	0.060^{*}	0.019	0.012	0.026^{*}
	(0.00)	(0.00)	(0.00)	(0.03)	(0.03)	(0.03)	(0.01)	(0.01)	(0.01)
log(% w/out HS diploma)	0.045	0.068	-0.003	-0.601	-1.083	0.166	-0.228	-0.279	0.045
	(0.06)	(0.06)	(0.06)	(0.96)	(1.05)	(0.99)	(0.43)	(0.49)	(0.44)
λ	0.365^{***}	0.414^{***}	0.445^{***}	0.358^{***}	0.358^{***}	0.391^{***}	0.401^{***}	0.464^{***}	0.480^{***}
	(0.08)	(0.08)	(0.08)	(0.08)	(0.08)	(0.08)	(0.08)	(0.07)	(0.07)
RW test (p-value)	0.4955	0.975	0.2018	0.9351	0.991	0.1059	0.1499	0.4635	0.957
BSJK (C.2) test	47^{***}	34^{***}	48^{***}	52^{***}	44^{***}	53^{***}	28^{***}	19^{***}	33^{***}
Pseudo R^2	0.979	0.98	0.978	0.974	0.976	0.973	0.986	0.985	0.985
AIC	-878	-884	-862	247	235	259	-73	-68	-57
BIC	-631	-636	-614	494	482	506	174	179	190

$^{*}p < 0.05$, $^{**}p < 0.01$, $^{***}p < 0.001$.

Table 4.10. Fully specified state-level SDEM models estimating both-sex mortality indicators ($log(ASDR)$, $e(0)$, and $e(65)$) featuring various specifications of state health policy.

	$log(ASDR)$			$e(0)$			$e(65)$		
Variable	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$
SHP indicator	-0.016^{***}	-0.015^{***}	0.000	0.234^{***}	0.243^{***}	0.012	0.121^{***}	0.104^{***}	-0.019
	(0.00)	(0.00)	(0.00)	(0.06)	(0.05)	(0.03)	(0.03)	(0.02)	(0.01)
Median age	0.000	-0.001	0.000	-0.024	-0.019	-0.029	0.022	0.027	0.022
	(0.00)	(0.00)	(0.00)	(0.04)	(0.04)	(0.04)	(0.02)	(0.02)	(0.02)
log(gov't ideology)	-0.011	0.016^{*}	-0.017^{*}	0.086	-0.210	0.162	0.109^{*}	-0.111	0.155^{**}
	(0.01)	(0.01)	(0.01)	(0.12)	(0.13)	(0.12)	(0.05)	(0.06)	(0.06)
log(% in poor health)	0.030	0.001	0.061	-0.055	0.500	-0.602	-0.447	-0.286	-0.617^{*}
	(0.04)	(0.04)	(0.04)	(0.64)	(0.64)	(0.66)	(0.29)	(0.30)	(0.30)
log(% Black)	0.034^{***}	0.036^{***}	0.029^{**}	-0.500^{**}	-0.543^{***}	-0.468^{**}	-0.281^{***}	-0.295^{***}	-0.228^{**}
	(0.01)	(0.01)	(0.01)	(0.16)	(0.15)	(0.17)	(0.07)	(0.07)	(0.08)
D index	-0.002	-0.001	-0.001	0.028	0.024	0.024	0.004	0.001	-0.003
	(0.00)	(0.00)	(0.00)	(0.02)	(0.02)	(0.02)	(0.01)	(0.01)	(0.01)
log(med. HH income)	0.033	0.038	0.063	-0.428	-0.491	-0.855	-0.604	-0.658	-0.850^{*}
	(0.05)	(0.05)	(0.05)	(0.83)	(0.80)	(0.85)	(0.38)	(0.37)	(0.39)
% blue collar	-0.004^{*}	-0.004^{**}	-0.005^{**}	0.039	0.043	0.056^{*}	0.037^{**}	0.041^{***}	0.047^{***}
	(0.00)	(0.00)	(0.00)	(0.03)	(0.03)	(0.03)	(0.01)	(0.01)	(0.01)
% urban population	-0.001	-0.001	-0.001	0.015	0.016	0.023	0.008	0.007	0.012
	(0.00)	(0.00)	(0.00)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)
log(% w/out HS diploma)	-0.156^{***}	-0.159^{***}	-0.160^{***}	2.844^{***}	2.707^{***}	2.855^{***}	0.978^{***}	1.038^{***}	1.008^{***}
	(0.03)	(0.03)	(0.03)	(0.52)	(0.53)	(0.54)	(0.23)	(0.25)	(0.25)
Lagged indicators									
SHP indicator	-0.021^{*}	-0.028^{***}	-0.001	0.204	0.380^{***}	0.014	0.178^{**}	0.213^{***}	-0.025
	(0.01)	(0.01)	(0.00)	(0.14)	(0.11)	(0.07)	(0.07)	(0.05)	(0.03)
Median age	-0.013^{*}	-0.006	-0.015^{*}	0.200^{*}	0.084	0.224^{*}	0.100^{*}	0.061	0.115^{*}
	(0.01)	(0.01)	(0.01)	(0.09)	(0.09)	(0.09)	(0.04)	(0.04)	(0.04)
log(gov't ideology)	-0.006	-0.016	-0.015	0.047	0.328	0.140	0.078	0.065	0.141
	(0.01)	(0.02)	(0.01)	(0.24)	(0.26)	(0.24)	(0.11)	(0.12)	(0.11)
log(% in poor health)	0.123	0.174^{*}	0.186^{*}	-2.048	-2.526	-2.902^{*}	-1.105	-1.557^{*}	-1.559^{*}
	(0.09)	(0.09)	(0.09)	(1.41)	(1.39)	(1.44)	(0.65)	(0.68)	(0.68)
log(% Black)	-0.026	-0.036	-0.063^{**}	0.534	0.515	1.051^{**}	0.054	0.187	0.337
	(0.02)	(0.02)	(0.02)	(0.38)	(0.36)	(0.37)	(0.17)	(0.17)	(0.17)
D index	0.000	0.003	0.002	-0.009	-0.062	-0.027	0.004	-0.021	-0.016
	(0.00)	(0.00)	(0.00)	(0.04)	(0.04)	(0.04)	(0.02)	(0.02)	(0.02)
log(med. HH income)	0.003	0.040	0.091	-0.714	-0.973	-1.582	0.904	0.639	0.063
	(0.10)	(0.10)	(0.11)	(1.71)	(1.64)	(1.76)	(0.78)	(0.80)	(0.83)
% blue collar	0.004	0.000	0.001	-0.067	-0.034	-0.043	-0.033	-0.001	0.000
	(0.00)	(0.00)	(0.00)	(0.06)	(0.06)	(0.06)	(0.03)	(0.03)	(0.03)
% urban population	-0.004^{*}	-0.002	-0.005^{**}	0.077^{*}	0.046	0.093^{**}	0.026	0.013	0.041^{**}
	(0.00)	(0.00)	(0.00)	(0.03)	(0.03)	(0.03)	(0.01)	(0.02)	(0.02)
log(% w/out HS diploma)	0.020	0.049	-0.043	-0.122	-0.764	0.903	-0.048	-0.128	0.345
	(0.06)	(0.07)	(0.07)	(1.04)	(1.09)	(1.06)	(0.47)	(0.52)	(0.50)
ρ	0.389^{***}	0.493^{***}	0.451^{***}	0.385^{***}	0.443^{***}	0.400^{***}	0.418^{***}	0.538^{***}	0.481^{***}
	(0.08)	(0.07)	(0.08)	(0.08)	(0.08)	(0.08)	(0.08)	(0.07)	(0.08)
RW test (p-value)	0.4955	0.975	0.2018	0.9351	0.991	0.1059	0.1499	0.4635	0.957
BSJK (C.2) test	47^{***}	34^{***}	48^{***}	52^{***}	44^{***}	53^{***}	28^{***}	19^{***}	33^{***}
Pseudo R^2	0.977	0.976	0.973	0.971	0.973	0.969	0.983	0.982	0.981
AIC	-384	-396	-366	740	723	753	423	420	440
BIC	-137	-148	-118	987	970	1000	670	667	688

$^{*}p < 0.05$, $^{**}p < 0.01$, $^{***}p < 0.001$.

models, respectively, governed the mean strength of the influence of the neighboring structure involved with the spatial processes in question. The discussions of statistical significance of these parameters are but trivial, since the conclusions were preempted by Moran, LM, and RW tests.

In terms of the model fit for all same-class spatial models for states and county groups, pseudo R^2, AIC and BIC criteria fell in agreement. Comparing the model fits by SHP indicator for each geographic unit-regression type-sex-mortality outcome combination the GoF indicators were very close indeed. In line with the expectation, models with RFNPPPD as the main SHP predictor offered arguably the worst fit of all the SHP indicators across all models in absolute terms, regardless of the mortality outcome. When it came to the two forms of the I score (the time-lagged and the original), the original version tended to outperform its lagged counterpart in corresponding specifications across various model types. However, the gap between the AIC/BIC estimates of the two SHP indicators across the models was quite narrow.

Table 4.11. Fully specified county group-level SDM models estimating both-sex mortality indicators ($log(ASDR)$, $e(0)$, and $e(65)$) featuring various specifications of state health policy.

	$log(ASDR)$			$e(0)$			$e(65)$		
Variable	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$
SHP indicator	−0.002	−0.005*	−0.001	0.057	0.066*	0.013	0.005	0.035*	0.012
	(0.00)	(0.00)	(0.00)	(0.04)	(0.03)	(0.02)	(0.02)	(0.02)	(0.01)
Median age	−0.004***	−0.004***	−0.004***	0.042***	0.042***	0.042***	0.050***	0.050***	0.050***
	(0.00)	(0.00)	(0.00)	(0.01)	(0.01)	(0.01)	(0.00)	(0.00)	(0.00)
log(gov't ideology)	−0.009	0.002	−0.009	0.093	−0.072	0.111	0.066	0.032	0.063
	(0.01)	(0.01)	(0.01)	(0.08)	(0.09)	(0.08)	(0.05)	(0.05)	(0.05)
log(% in poor health)	−0.052***	−0.052***	−0.050***	0.367*	0.374*	0.342*	0.626***	0.628***	0.612***
	(0.01)	(0.01)	(0.01)	(0.15)	(0.15)	(0.15)	(0.09)	(0.09)	(0.09)
log(% Black)	0.001	0.001	0.001	−0.022	−0.019	−0.019	−0.006	−0.005	−0.004
	(0.00)	(0.00)	(0.00)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)
log(D index)	−0.006***	−0.006***	−0.006***	0.097***	0.099***	0.098***	0.038***	0.040***	0.039***
	(0.00)	(0.00)	(0.00)	(0.02)	(0.02)	(0.02)	(0.01)	(0.01)	(0.01)
log(med. HH income)	−0.122***	−0.122***	−0.121***	1.879***	1.882***	1.875***	0.918***	0.923***	0.912***
	(0.01)	(0.01)	(0.01)	(0.13)	(0.13)	(0.13)	(0.08)	(0.08)	(0.08)
log(GINI index)	−0.091***	−0.091***	−0.093***	1.156***	1.146***	1.180***	0.783***	0.793***	0.803***
	(0.01)	(0.01)	(0.01)	(0.18)	(0.18)	(0.18)	(0.10)	(0.10)	(0.10)
% blue collar	0.000*	0.000*	0.000*	0.008**	0.007**	0.008**	0.003*	0.003*	0.003*
	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
% urban population	−0.001***	−0.001***	−0.001***	0.010***	0.010***	0.010***	0.006***	0.006***	0.006***
	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
log(% w/out HS diploma)	0.004	0.005	0.004	−0.102	−0.109	−0.103	0.001	−0.004	−0.001
	(0.00)	(0.00)	(0.00)	(0.07)	(0.07)	(0.07)	(0.04)	(0.04)	(0.04)
Lagged indicators									
SHP indicator	−0.004	0.001	0.002	0.018	−0.014	−0.025	0.048	−0.002	−0.028*
	(0.00)	(0.00)	(0.00)	(0.05)	(0.03)	(0.02)	(0.03)	(0.02)	(0.01)
Median age	0.001	0.001	0.001	−0.005	−0.005	−0.004	−0.010*	−0.010	−0.010*
	(0.00)	(0.00)	(0.00)	(0.01)	(0.01)	(0.01)	(0.00)	(0.00)	(0.00)
log(gov't ideology)	0.005	0.002	0.003	−0.087	0.020	−0.066	−0.011	−0.063	0.018
	(0.01)	(0.01)	(0.01)	(0.09)	(0.10)	(0.09)	(0.05)	(0.05)	(0.05)
log(% in poor health)	−0.016	−0.014	−0.008	0.816***	0.785***	0.705***	−0.148	−0.156	−0.212
	(0.01)	(0.01)	(0.01)	(0.21)	(0.21)	(0.21)	(0.12)	(0.12)	(0.12)
log(% Black)	0.006***	0.005**	0.005**	−0.120***	−0.105***	−0.101***	−0.040**	−0.031*	−0.029*
	(0.00)	(0.00)	(0.00)	(0.03)	(0.03)	(0.03)	(0.01)	(0.01)	(0.01)
log(D index)	−0.002	−0.003	−0.002	0.054	0.065*	0.057	0.005	0.014	0.007
	(0.00)	(0.00)	(0.00)	(0.03)	(0.03)	(0.03)	(0.02)	(0.02)	(0.02)
log(med. HH income)	0.065***	0.062***	0.071***	−1.150***	−1.147***	−1.219***	−0.444***	−0.400***	−0.498***
	(0.01)	(0.01)	(0.01)	(0.20)	(0.20)	(0.20)	(0.11)	(0.11)	(0.11)
log(GINI index)	−0.136***	−0.142***	−0.152***	2.147***	2.148***	2.347***	0.946***	1.041***	1.095***
	(0.03)	(0.03)	(0.02)	(0.38)	(0.38)	(0.38)	(0.21)	(0.21)	(0.21)
% blue collar	−0.001**	−0.001**	−0.001**	0.011*	0.011*	0.012**	0.008**	0.008**	0.008**
	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
% urban population	0.000*	0.000*	0.000**	0.003	0.003	0.004*	0.004***	0.004***	0.004***
	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
log(% w/out HS diploma)	−0.029***	−0.028***	−0.028***	0.444***	0.413***	0.436***	0.230***	0.215***	0.216***
	(0.01)	(0.01)	(0.01)	(0.11)	(0.11)	(0.11)	(0.06)	(0.06)	(0.06)
λ	0.602***	0.609***	0.611***	0.622***	0.624***	0.629***	0.587***	0.599***	0.595***
	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)
RW test (p-value)	0.002	0.002	0.002	0.002	0.002	0.002	0.002	0.002	0.002
BSJK (C.2) test	3572***	1534***	1863***	1113***	3435***	3665***	1146***	3289***	1216***
Pseudo R^2	0.938	0.938	0.938	0.94	0.94	0.94	0.94	0.94	0.94
AIC	−31211	−31191	−31177	22509	22515	22537	10957	10993	10985
BIC	−13220	−13200	−13187	40500	40505	40528	28948	28984	28976

$^{*}p < 0.05$, $^{**}p < 0.01$, $^{***}p < 0.001$.

Table 4.12. Fully specified county group-level SDEM models estimating both-sex mortality indicators ($log(ASDR)$, $e(0)$, and $e(65)$) featuring various specifications of state health policy.

	$log(ASDR)$			$e(0)$			$e(65)$		
Variable	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$
SHP indicator	−0.003	−0.005**	−0.001	0.065	0.075*	0.007	0.015	0.040*	0.006
	(0.00)	(0.00)	(0.00)	(0.04)	(0.03)	(0.02)	(0.02)	(0.02)	(0.01)
Median age	−0.005***	−0.005***	−0.005***	0.047***	0.047***	0.048***	0.053***	0.054***	0.054***
	(0.00)	(0.00)	(0.00)	(0.01)	(0.01)	(0.01)	(0.00)	(0.00)	(0.00)
log(gov't ideology)	−0.012*	0.001	−0.013*	0.130	−0.052	0.155*	0.096*	0.035	0.100*
	(0.01)	(0.01)	(0.01)	(0.08)	(0.08)	(0.08)	(0.05)	(0.05)	(0.04)
log(% in poor health)	−0.065***	−0.066***	−0.061***	0.650***	0.664***	0.602***	0.688***	0.701***	0.662***
	(0.01)	(0.01)	(0.01)	(0.15)	(0.15)	(0.15)	(0.08)	(0.08)	(0.08)
log(% Black)	0.002*	0.001	0.001	−0.041**	−0.035*	−0.035*	−0.013	−0.010	−0.010
	(0.00)	(0.00)	(0.00)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)
log(D index)	−0.006***	−0.007***	−0.007***	0.104***	0.109***	0.107***	0.041***	0.044***	0.043***
	(0.00)	(0.00)	(0.00)	(0.02)	(0.02)	(0.02)	(0.01)	(0.01)	(0.01)
log(med. HH income)	−0.120***	−0.121***	−0.118***	1.818***	1.835***	1.800***	0.907***	0.928***	0.892***
	(0.01)	(0.01)	(0.01)	(0.13)	(0.13)	(0.13)	(0.07)	(0.07)	(0.07)
log(GINI index)	−0.113***	−0.113***	−0.118***	1.475***	1.461***	1.536***	0.953***	0.968***	0.997***
	(0.01)	(0.01)	(0.01)	(0.20)	(0.20)	(0.20)	(0.11)	(0.11)	(0.11)
% blue collar	−0.001***	−0.001***	−0.001***	0.011***	0.011***	0.011***	0.005**	0.005***	0.005***
	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
% urban population	−0.001***	−0.001***	−0.001***	0.012***	0.012***	0.013***	0.007***	0.007***	0.008***
	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
log(% w/out HS diploma)	0.001	0.002	0.002	−0.049	−0.072	−0.060	0.032	0.018	0.023
	(0.00)	(0.00)	(0.00)	(0.07)	(0.07)	(0.07)	(0.04)	(0.04)	(0.04)
Lagged indicators									
SHP indicator	−0.014***	−0.007**	0.003*	0.161**	0.103**	−0.033	0.122***	0.061**	−0.042**
	(0.00)	(0.00)	(0.00)	(0.05)	(0.04)	(0.03)	(0.03)	(0.02)	(0.01)
Median age	−0.003***	−0.003***	−0.003***	0.038**	0.038**	0.042**	0.030***	0.031***	0.032***
	(0.00)	(0.00)	(0.00)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)
log(gov't ideology)	0.002	0.009	−0.006	−0.082	−0.104	0.013	0.038	−0.109	0.108
	(0.01)	(0.01)	(0.01)	(0.10)	(0.11)	(0.10)	(0.06)	(0.06)	(0.06)
log(% in poor health)	−0.113***	−0.115***	−0.100***	2.404***	2.420***	2.216***	0.582***	0.623***	0.480**
	(0.02)	(0.02)	(0.02)	(0.30)	(0.30)	(0.31)	(0.16)	(0.16)	(0.16)
log(% Black)	0.010***	0.008**	0.008**	−0.183***	−0.156***	−0.156***	−0.067	−0.050*	−0.051*
	(0.00)	(0.00)	(0.00)	(0.04)	(0.04)	(0.04)	(0.02)	(0.02)	(0.02)
log(D index)	−0.007*	−0.009**	−0.008**	0.125**	0.147**	0.138**	0.039	0.054*	0.048
	(0.00)	(0.00)	(0.00)	(0.05)	(0.05)	(0.05)	(0.03)	(0.03)	(0.03)
log(med. HH income)	−0.009	−0.017	−0.001	−0.070	−0.012	−0.168	0.109	0.198	0.033
	(0.02)	(0.02)	(0.02)	(0.30)	(0.30)	(0.31)	(0.16)	(0.16)	(0.16)
log(GINI index)	−0.235***	−0.239***	−0.256***	3.414***	3.394***	3.691***	1.772***	1.844***	1.967***
	(0.04)	(0.04)	(0.04)	(0.55)	(0.55)	(0.56)	(0.30)	(0.30)	(0.30)
% blue collar	−0.002***	−0.002***	−0.002***	0.024***	0.025***	0.026***	0.014***	0.015***	0.015***
	(0.00)	(0.00)	(0.00)	(0.01)	(0.01)	(0.01)	(0.00)	(0.00)	(0.00)
% urban population	−0.001***	−0.001***	−0.001***	0.016***	0.016***	0.017***	0.011***	0.011***	0.012***
	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
log(% w/out HS diploma)	−0.033**	−0.026*	−0.029**	0.445**	0.350*	0.393*	0.278**	0.221*	0.238**
	(0.01)	(0.01)	(0.01)	(0.17)	(0.17)	(0.17)	(0.09)	(0.09)	(0.09)
ρ	0.606***	0.614***	0.618***	0.628***	0.630***	0.637***	0.591***	0.604***	0.601***
	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)
RW test (p-value)	0.002	0.002	0.002	0.002	0.002	0.002	0.002	0.002	0.002
BSJK (C.2) test	3572***	1534***	1863***	3605***	1263***	3665***	1146***	3289***	1216***
Pseudo R^2	0.913	0.912	0.911	0.913	0.913	0.911	0.918	0.916	0.917
AIC	31751	31771	31801	85479	85481	85520	73917	73953	73961
BIC	49742	49762	49791	103469	103472	103511	91907	91943	91951

$^{*}p < 0.05$, $^{**}p < 0.01$, $^{***}p < 0.001$.

Two additional sets of post-hoc diagnostics may help to more precisely identify the forms of spatial dependence and to illuminate the choices for the final model selection. The first is the Moran test on the model to verify the presence of residual spatial autocorrelation. This diagnostic procedure is equivalent between the regressions presented in this analysis and running of a linear regression with eigenvector spatial filtering applied to the residuals (Kim & Kim, 2019; Thayn, 2017). The results of non-spatial FE panel models for states and county groups for all year-sex combinations, as shown in Table 4.13, contained substantial amount of unabsorbed spatial variability. This is an expected outcome, as none of the non-spatial panel models allow to explicitly account for spatial structure. All spatial models for states performed considerably better than non-spatial FE panels, though SLX process has only marginally absorbed the spatial variation. However, considering county group models, the outcomes were stark. The most effective at mopping up the spatial autocorrelation in county group data were SAR, SDM and SDEM models for all sex-year-mortality indicators. The combination including the time-lagged I score and RFNPPPD predictors (not shown) followed an identical general pattern shown in Table 4.13 due to the closeness of fit between the model variants with three different SHP variables, as shown above. Interestingly, here too the SLX model largely failed to address the task of accounting for spatial autocorrelation that amounted in magnitude just under the non-spatial FE panel regression benchmark. This suggests that local dependence on the interaction between regional factors independently of other forms of spatial lag is not sufficient to explain the spatial variation in mortality and plays a fairly minor role in its diffusion. Relative to the panel SLX and FE models SEM spatial error component captured much, but not all spatial dependence in the county group set, leaving about one-seventh spatial autocorrelation as residual.

SAR and SDM models came out as clear winners in this test with negligible residual spatial autocorrelation left, although SDEM models followed closely with only a minor fraction of about 10% of the unaccounted spatial variation remaining, as compared to the non-spatial FE panel benchmark. These results confirm the conclusions of the earlier LM test indicating the preponderance of endogenous lag as the main driver of mortality pattern diffusion under the basic specification grounded in theory established in chapters 1 and 2. To a lesser degree, the process is dependent on the interaction of the regional contextual indicators that have greater leverage in isolated localities. While SDM is a less parsimonious model than SAR, its Durbin component incorporates an additional bit of flexibility to account for local spatial effects that SAR model does not. Indeed, likelihood ratio tests of SDM and SAR models pointed to the fact that a more general model attained significantly better fit for all geographic unit-sex-SHP-outcome combinations ($LR > 34.1$ (df 10) for states and $LR > 119.6$ (df 11) for county groups, significant at the 95% confidence level). The maximum likelihood estimation of SDM model is not much more computationally burdensome than that of SAR, since the additional Durbin term in SDM is efficiently estimated using the conventional OLS methods with little-to-none computational overhead. As such I deem SDM the model of choice to represent the spatial inter-dependence between states and county groups for predicting its mortality outcomes.

Prior to offering an interpretation of the results it is necessary to evaluate the amount of error involved in the models and how they compare in this regard. In the final diagnostic I assess the overall model fit with respect to the amount of total error using mean absolute

percent error (MAPE) metric. MAPE was chosen because it lends itself to a more intuitive interpretation as compared to other closely related metrics, such as mean absolute error (MAE) and root mean squared error (RMSE). Figure 4.9 summarizes the total MAPE in models by outcome, state and county group subset, the main SHP predictor, and by sex. Overall, the model fits were reasonably decent with relative amount of error within 1% for the most part, and often under 0.25% for the best fitting models. Before considering the model comparisons by sub-group, several patterns become immediately apparent. First, state models had approximately 50% lower relative error, as compared to its county group counterparts. This is a fairly obvious result, as the coarse state-level aggregation would offer stability to the indicators thus reducing the amount of variability around the sample means. Second, fitting the models to the logarithm of ASDR yielded the lowest error, followed by the fits to the life expectancy at birth, and then by the fits to the life expectancy at age 65. Again, this is unsurprising since log-transformed ASDR is a more direct mortality outcome than life expectancy measures. The life expectancy at age 65 has been more volatile as the outcome since there is greater variability in the remaining lifespan at older ages, given the assortment of accumulated health conditions over a lifespan and increased sensitivity of health at older ages to exposure to various concurrent context-specific risk factors. Third, in relative terms, MAPE in male models was consistently around 30% lower than in female models when log-transformed ASDR was the outcome, but about 10% greater when the outcome was either e(0) or e(65). However, the worst fits were for male county group models ($\approx 1.65\%$), while the best fit was found in male log-ASDR state-level model ($\approx 0.15\%$).

Final model selection: Spatial Durbin Model

Lastly, I review the side-by-side comparison of the models within each group in Figure 4.9. As expected, SDM, SAR, and SDEM models performed consistently better than the rest. SDM in particular had the lowest mean absolute percent error relative to all the other spatial and non-spatial panel fixed effects regressions. On the other hand, non-spatial fixed effects and SEM models performed consistently the worst. SEM had comparable or slightly higher overall error than the non-spatial FE. This underlines the contention that perhaps modeling unobserved spatial heterogeneity in an explicit way had virtually no added benefit over the simpler panel approach. Moreover, explicit spatial error component contained in SDEM specification sets it apart from a more parsimonious SLX model, yet it provided negligible advantage to SDEM model fit over that of SLX in terms of the relative error. Nevertheless, since the estimation of a more general model presented no additional significant computational constraints, I choose to review SDM and SDEM models as representatives of the two potential spatial dependence processes, albeit with an understanding that SDM had a clear advantage over SDEM as well as all the other models considered in the present analysis.

With the above selection in mind it is possible to examine the fit of panel fixed effects SDM and SDEM models spatially. Figures 4.10 and 4.11 demonstrate the distribution of model error across the state and county group samples for the natural logarithm of ASDR, broken down by sex, with I score as the main SHP predictor. Additional illustrations showing the model error distribution over other mortality outcomes-SHP predictor combinations in SDM and SDEM models may be found in Figures A.2.20–A.2.35 in the Appendices. Com-

paring the corresponding panels in the two figures, the overall visual level of error in SDM panels is somewhat lower than in SDEM panels, judging from the intensity of color shading. Interestingly, in terms of the proportional distribution of error across the map the two types of models were largely in agreement. States and county groups with smaller errors tended to be predominantly rural or suburban, yet larger errors were observed in places with a lot of heterogeneity (e.g., those with a large variation in the number, size/population density, and geographic dispersion of settlements). This included states, such as Virginia, South Dakota, Hawaii, Florida and California. Smaller errors were found preponderantly in the rural South and West (Oklahoma, Utah, Nevada, Mississippi, Alabama, Georgia, and South Carolina). The relative distribution of model error between state-level female and male mortality did not coincide in a number of cases, suggesting differential outcomes by sex under the same set of contextual factors. States with a diverging error pattern by sex were largely rural and suburban (e.g., Mississippi, Oklahoma, and Nevada). The error patterns in e(0) and e(65) models exhibited the same general arrangement, albeit with larger errors for the aforementioned reasons.

Shifting the attention to the comparison of the error distribution in the SDM and SDEM county group models, the situation was markedly different. Despite that the total MAPE error was arbitrarily larger than in the state sample (partially due to the limitations of the MAPE metric), the individual fit for most county groups was palpably more accurate. This is consistent with the prior expectation that contextual predictors in smaller, more homogeneous geographic units would more closely represent local outcomes. Larger errors in the county group models were observed in urbanized and densely populated suburban localities, whereas smaller values of model fit error were associated with the deeply rural regions. Just as in the state-level models, the lack of fit in the county group models was mostly associated with high local diversity of factors impacting the mortality/longevity response and with the omission of variables that could account for such variability.

Spatial Durbin Model: Interpretation

Having settled on the SDM fixed effects as the best fitting model for the SCM conceptual paradigm it is appropriate to offer an interpretation. I elaborate on the magnitude and directionality of the SHP-mortality association, all else being equal. Since SDM includes an endogenous feedback to the response under the lag form of spatial dependence it is not proper to interpret the model coefficients as presented in Table 4.7, for example. The spatial Durbin component channels indirect influences of regional contextual variables on the response in addition to a direct impact of an areal unit's own set of contextual attributes. To resolve the ambiguity due to the endogenous response feedback loop as well as due to the local contextual factor compounding, I run 1,000 Monte Carlo simulations and decompose the converged impacts into direct, indirect, and total components. Tables 4.14 and 4.15 summarize the simulated impact decomposition of SDM model coefficients for state and county group log-transformed ASDR, respectively, for each sex and by SHP indicator. I present an analogous output for e(0) and e(65) models in the Appendices. Here I elaborate on the key findings from the impact decomposition.

I first tackle the interpretation of the results from the state-level SDM models detailed in Tables 4.14, A.2.34, A.2.36. After applying the reverse variable transformations a few

Table 4.13. Post-regression residual Moran estimates for various models for the 3 mortality outcome indicators and I score as the state health policy indicator, by year and sex.

			States			County groups		
Model	Sex	Year	$log(ASDR)$	$e(0)$	$e(65)$	$log(ASDR)$	$e(0)$	$e(65)$
FE panel†	Both sexes	1990	0.142	0.100	0.218*	0.436***	0.462***	0.409***
		2000	0.225**	0.210*	0.245**	0.296***	0.286***	0.312***
		2010	0.211*	0.237*	0.095	0.346***	0.39***	0.265***
		2018	0.175	0.167	0.245**	0.344***	0.359***	0.357***
SAR	Both sexes	1990	−0.102	−0.108	−0.120	0.005	0.007	0.007
		2000	0.072	0.049	0.064	−0.011	−0.003	−0.008
		2010	0.067	0.100	0.008	0.019	0.027*	0.017
		2018	−0.161	−0.122	−0.207*	0	−0.002	0.002
	Female	1990	−0.056	−0.050	−0.082	0.004	0.004	0.007
		2000	0.047	0.048	0.021	−0.021	−0.013	−0.020
		2010	0.039	0.083	−0.043	0.014	0.018	0.015
		2018	−0.153	−0.105	−0.211*	0.004	0.001	0.009
	Male	1990	−0.095	−0.112	−0.096	0.008	0.012	0.013
		2000	0.082	0.033	0.086	−0.004	0.004	−0.001
		2010	0.084	0.100	0.066	0.024*	0.033**	0.022*
		2018	−0.130	−0.102	−0.139	0	−0.001	0.004
SLX	Both sexes	1990	0.159	0.145	0.219*	0.412***	0.444***	0.386***
		2000	0.176*	0.182*	0.136	0.323***	0.307***	0.339***
		2010	0.153	0.200*	0.081	0.329***	0.369***	0.262***
		2018	0.172	0.159*	0.183*	0.318***	0.339***	0.321***
	Female	1990	0.166*	0.143	0.235*	0.44***	0.467***	0.415***
		2000	0.127	0.124	0.131	0.372***	0.376***	0.371***
		2010	0.149	0.186*	0.098	0.349***	0.38***	0.297***
		2018	0.151	0.140	0.184*	0.337***	0.356***	0.327***
	Male	1990	0.064	0.104	0.06	0.372***	0.416***	0.34***
		2000	0.176*	0.193*	0.194*	0.265***	0.247***	0.289***
		2010	0.079	0.149	−0.002	0.296***	0.352***	0.227***
		2018	0.109	0.107	0.129	0.295***	0.319***	0.304***
SEM	Both sexes	1990	−0.115	−0.119	−0.136	0.052***	0.055***	0.056***
		2000	0.117	0.118	0.075	0.024	0.032**	0.028*
		2010	0.022	0.067	−0.020	0.067***	0.079***	0.063***
		2018	−0.129	−0.117	−0.159	0.030*	0.030**	0.037***
	Female	1990	−0.098	−0.093	−0.138	0.062***	0.060***	0.067***
		2000	0.061	0.082	0.021	0.025*	0.030**	0.024*
		2010	0.016	0.044	−0.040	0.07***	0.075***	0.070***
		2018	−0.108	−0.086	−0.153	0.045***	0.041***	0.054***
	Male	1990	−0.109	−0.118	−0.096	0.046***	0.053***	0.051***
		2000	0.177*	0.126	0.137	0.024*	0.035**	0.03**
		2010	0.056	0.094	0.012	0.058***	0.078***	0.054***
		2018	−0.128	−0.106	−0.146	0.021	0.026*	0.030**
SDM	Both sexes	1990	−0.001	−0.021	−0.005	0.003	0.004	0.006
		2000	0.096	0.061	0.088	−0.010	−0.001	−0.009
		2010	−0.108	−0.094	−0.109	0.021*	0.028**	0.019
		2018	−0.150	−0.103	−0.155	−0.003	−0.005	0.001
	Female	1990	0.040	0.036	0.021	0.002	0.001	0.005
		2000	0.031	0.041	0.006	−0.022*	−0.013	−0.022
		2010	−0.127	−0.117	−0.146	0.017	0.020	0.017
		2018	−0.168	−0.121	−0.192*	0.001	−0.002	0.007
	Male	1990	0.005	−0.037	0.030	0.006	0.007	0.013
		2000	0.173*	0.076	0.184*	−0.004	0.006	−0.002
		2010	−0.028	−0.046	0.007	0.027**	0.034**	0.025*
		2018	−0.102	−0.069	−0.066	−0.002	−0.003	0.003
SDEM	Both sexes	1990	−0.053	−0.068	−0.048	0.034***	0.032**	0.039***
		2000	0.061	0.045	0.053	0.021	0.030**	0.023
		2010	−0.121	−0.111	−0.107	0.058***	0.069***	0.052***
		2018	−0.165	−0.136	−0.164	0.01	0.008	0.019
	Female	1990	−0.010	−0.013	−0.019	0.041***	0.037***	0.045***
		2000	0.004	0.020	−0.022	0.015	0.023*	0.012
		2010	−0.143	−0.142	−0.155	0.058***	0.064***	0.055***
		2018	−0.178	−0.140	−0.200*	0.023*	0.019	0.032**
	Male	1990	−0.026	−0.067	0.002	0.030**	0.03**	0.039***
		2000	0.163	0.075	0.170*	0.024*	0.036***	0.027**
		2010	−0.043	−0.054	−0.006	0.056***	0.071***	0.05***
		2018	−0.113	−0.102	−0.072	0.006	0.008	0.016

$^{*}p < 0.05$, $^{**}p < 0.01$, $^{***}p < 0.001$.

† FE both-sex for benchmark comparison only.

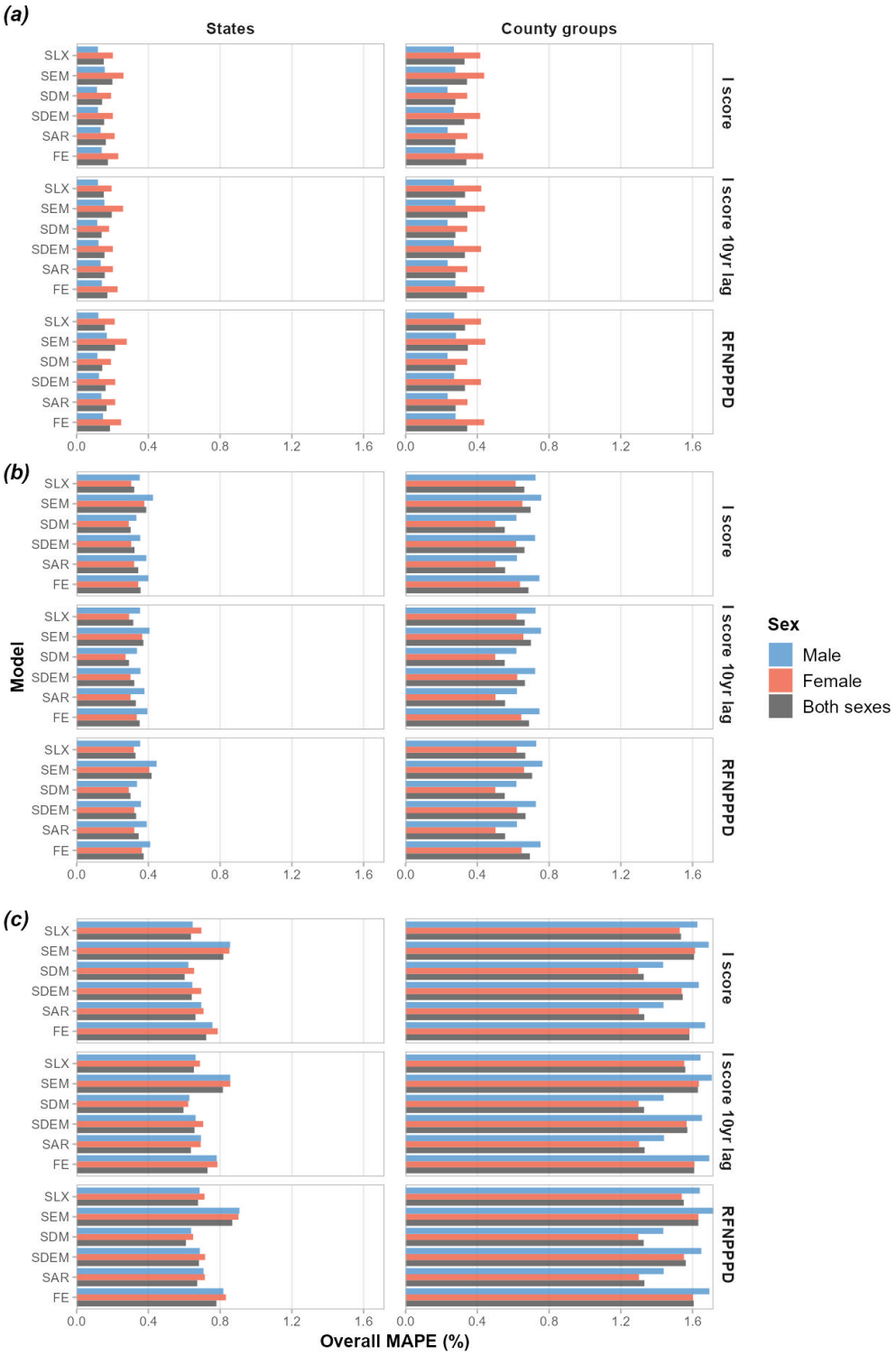


Figure 4.9. Comparison of state and county group fixed effects panel model fits for the three mortality indicators and three candidates for state health policy, by sex.

Note: the mean absolute percent error (MAPE) values are shown for (a) natural logarithm of the age-adjusted mortality rate, (b) life expectancy at birth, (c) life expectancy at age 65.

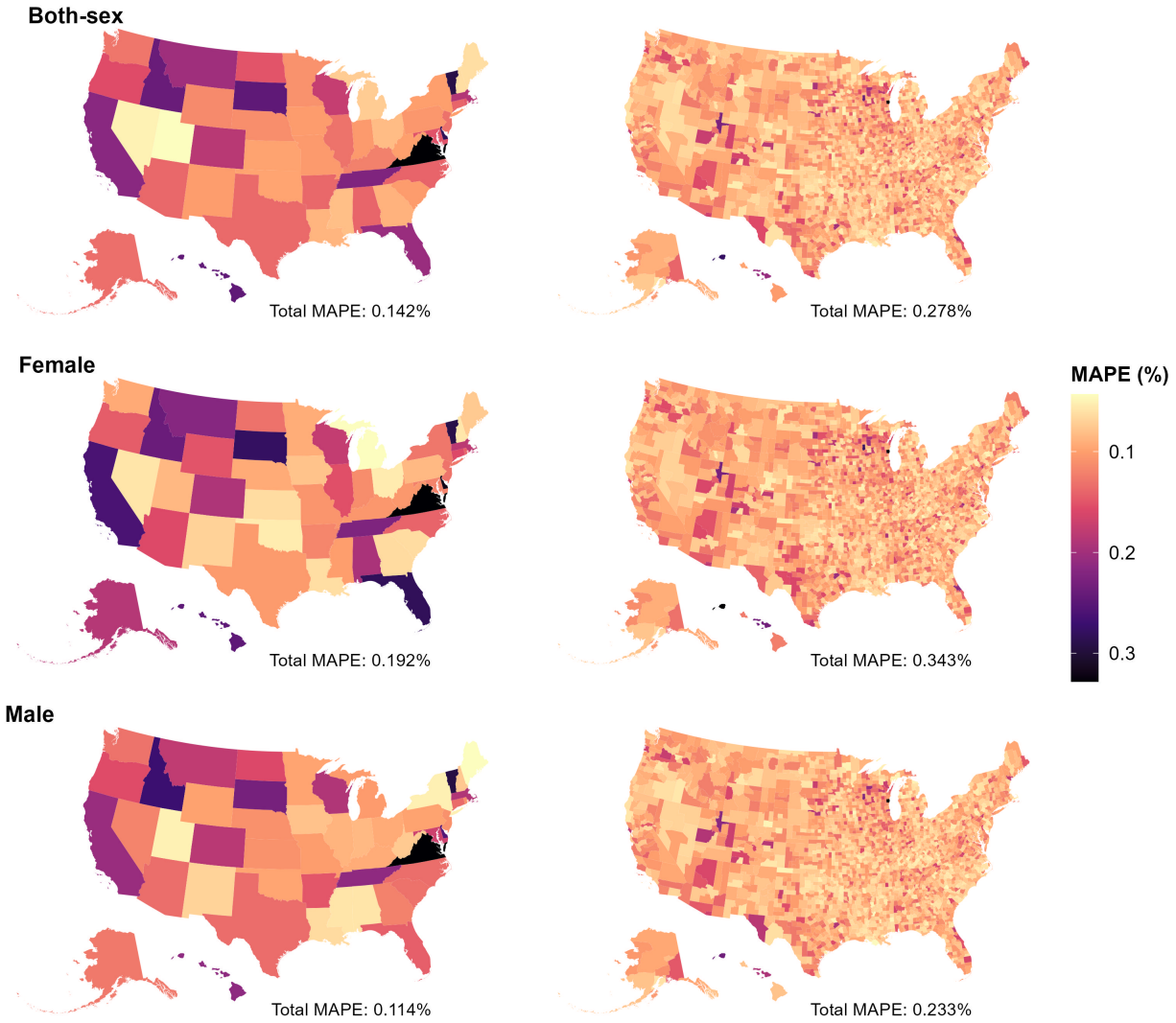


Figure 4.10. Spatial distribution of mean absolute percent error (MAPE) in fully adjusted spatial Durbin models (SDM) for the logarithm of the age-standardized mortality rate as the outcome and I score as the main state health policy predictor, by sex.

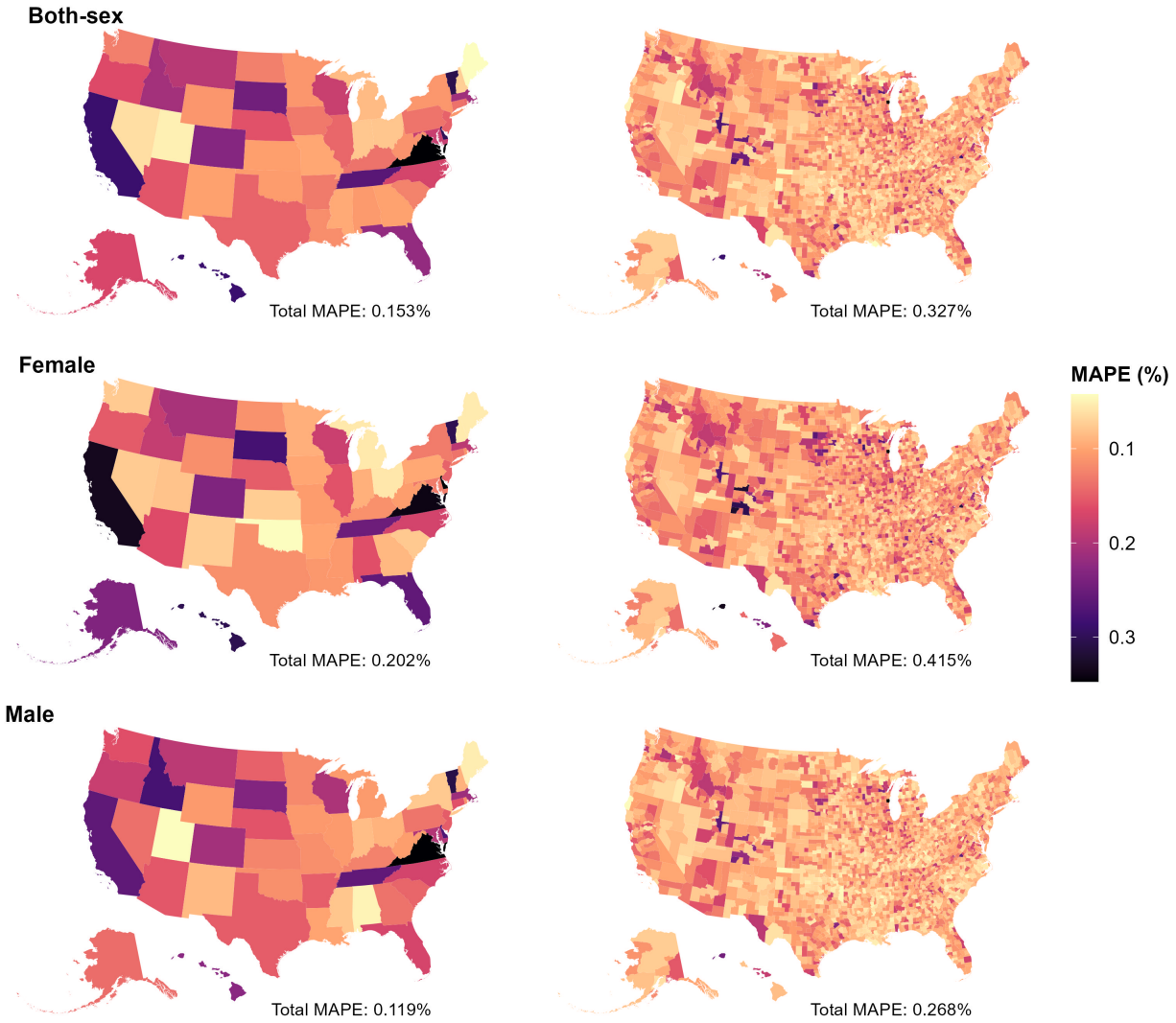


Figure 4.11. Spatial distribution of mean absolute percent error (MAPE) in fully adjusted spatial Durbin error models (SDEM) for the logarithm of the age-standardized mortality rate as the outcome and I score as the main state health policy predictor, by sex.

key findings emerge. First and foremost, the total impact of the I score and its 10-year lagged counterpart had a clear positive impact on mortality reduction. All else being equal, one additional standard deviation in I_Z or $I_{t-10,Z}$ carried the potential to reduce state-level age-standardized mortality rate by 2.1%–2.3%, for females, males, and both sexes combined. Assuming a normal distribution with minimum and maximum values representing $\pm$ 3 standard deviations around the mean, respectively, the I score measure could account for as much as 12.9% of the geographic gap in ASDR. At the same time, no significant impact was registered for the RFNPPPD measure. It remained insignificant across all the state SDM models, thus aligning with my prior expectations, as discussed in chapter 3 and parts of the present chapter.

When state e(0) was the outcome (see Table A.2.34) the results reciprocated those of the log-ASDR SDM model. One standard deviation increase on the I score spectrum was associated with a 0.35 year increase in state-level life expectancy at birth, while holding the other contextual attributes fixed. Applying the same distributional assumption as above, this translated to a difference of about 2.1 years between states with vastly different health policy priorities. Of interest was the difference between the male-female comparison for the time-lagged form of the I score. While both-sex and female coefficients were in agreement, the SHP indicator had a somewhat diminished effect on the male e(0), increasing the outcome by 0.28 years for every standard deviation increase in $I_{t-10,Z}$. Comparing this total impact coefficient with the total impact for male I_Z, it could suggest that the effect of introduction of new policies may have been less durable for the male population after a decade or more, even though the effect remained beneficial overall. For both log-ASDR and e(0) models, about two-thirds of the spatial effect of SHP on the outcome has been due to direct influences (i.e., state's own contextual factors), while one-third were due to a mixture of local and global influences (i.e., the neighboring unit spillovers and global endogenous diffusion).

Considering the impact of SHP on state e(65), the results in the Table A.2.36 show that both I_Z and $I_{t-10,Z}$ maintained significant predictive potential of the remaining longevity at older ages, while the RFNPPPD indicator stayed insignificant across all e(65)-sex combinations. The increase in one standard deviation in I_Z would have extended the lifespan by 0.19 years for both sexes combined, 0.2 years for females, and by 0.17 years for males. However, unlike I_Z the total impact of $I_{t-10,Z}$ was 13%, 15% and 23% lower for both sexes combined, females, and males, respectively. This could possibly be explained by the shorter lifespan over the age of 65 years such that death in the next decade would preclude taking advantage of the positive effects of health policies. This could also explain the reason behind the noticeably lower total impact of $I_{t-10,Z}$ on male e(65) than on female e(65).

Changing the focus to the impact decomposition results of the county group SDM models (see Tables 4.15, A.2.35, A.2.37), it is worth remarking the consistent insignificance of the I score and RFNPPPD indicators across all sex-outcome combinations, when controlling for the contextual attributes. At the same time, the decade-lagged form of the I score did, in fact, make an impact on mortality outcomes, albeit to a much lower degree. For instance, an upward change in one standard deviation on the $I_{t-10,Z}$ spectrum reduced age-adjusted death rate by approximately 1.2% for all sex categories. This is just slightly over a half of the effect that SHP had on the state-level ASDR outcome.

However, unlike the output of the state-level models, county group models featured

a lot of significant results for the contextual factors. This evidence can be indicative of the fact that increasingly localized mortality or longevity was more heavily dependent on the contextual aspects of a place. In other words, these are the elements that define the overall area character in terms of economic well-being, social structure, and deprivation. Nevertheless, most of these effects were of low magnitude. Curiously, holding all other contextual variables fixed, median age in the county group data manifested a minor, yet positive association with ASDR and life expectancy, particularly for the female population. This could possibly be explained as those with higher chances of survival are such because of the protective effects of age in areas represented by certain cohorts or a mixture of birth cohorts among the residents. A similar positive association could be observed for the fraction of the population in poor health. The magnitude of this effect was about 1.5 months of additional life expectancy per 10% change in the proportion of population in poor/fair health. Since the 10% represented about 2 standard deviations on either side of the mean for the indicator, this made poor health prevalence a weakly determining factor, albeit the one that likely reflected the effects of worse health in surviving older population groups with chronic health conditions. It was made more apparent by juxtaposing the results of the models for e(0) and e(65) with respect to the proportion of the population in poor health, where the latter set of models demonstrated a more pronounced effect.

County-level covariates such as the median household income and income inequality had a predictably stronger influence on mortality/longevity outcomes among the county groups. A 10% of additional median household income was associated with 0.42 additional years of life expectancy at birth for females and 0.49 additional years for males. Holding the contextual composition fixed, the same increment in median household income was associated with 0.20–0.22 years of additional life expectancy at age 65. The effects of area character were also evident in the county group models. The proportion of urban population and the prevalence of blue collar occupations were protective against mortality, approximately predicting about 0.02 years of increase in life expectancy at birth per one percentage point increase in these indicators. The effects of minority race (i.e., % African American in the racial composition) were significant in the state models, slightly increasing ASDR by 0.7% for every 10% increase in the proportion. Somewhat curiously, but after removing the variation from other factors, the minority race effects were not significant in the county group models, while the reverse was true about the multi-racial residential segregation. Contrary to some hypotheses, a 10% increase in residential segregation (as operationalized by the measure of distributional evenness, or the Divergence index) was actually associated with about 11 days of additional life expectancy at birth, and 4 additional days of life expectancy at age 65. While by no means substantial, this could be suggestive of a presence of protective communal effects, where ethno-racial enclaves fare better with family and communal support networks. As this effect was too small, it is hardly surprising it was readily wiped out in the state-level results. Next, contrary to the prevalent observation, local communities of comparable characteristics with higher percentage of high school dropouts do not necessarily perform worse in terms of its mortality and longevity indicators. In fact, for states 2% increase in the proportion of the adult population without a high school diploma was associated with an additional month of life expectancy at birth. This corroborates the notion that low educational attainment is not in and of itself a downward driver for longevity, but rather acts in conjunction with other

unfavorable contextual factors such as poverty to increase population mortality. Lastly, I would like to note that government ideology on its own was not directly associated with changes in mortality outcomes. This was to be expected, as it was intended to act as an auxiliary to inform the state health policy indicators, while having only a limited and indirect effect on proximal causes of death, as per the conceptual model proposed in chapter 2.

In wrapping up this results exposition, I would like to compare and highlight certain results for state and county group models by age and sex with respect to the significant SHP indicators. It is interesting to observe that nearly no difference in effect magnitude existed between females and males in terms of both log-ASDR and e(0) at the state level. However, the impact for the male e(65) was anywhere between 15%–40% lower, as compared to the female e(65), with a wider gap for the lagged I score indicator. There were negligible differences between sexes in the county group models for all mortality/longevity indicators, with the lagged I score being a sole, but weak significant predictor of mortality. To summarize, temporally proximal policies provided the greatest effect on state-level population with a differential impact favoring older-aged female population. The county group policy effect, though half as strong as the state effect, was mostly relevant only after a set period of time has elapsed, whereby sex differences were non-existent at all ages.

4.5 Discussion

The systematic approach I follow in the course of this analysis paints a compelling picture of the weight of legislative health initiatives in the progress toward the growth of population longevity. My work offers a number of worthwhile contributions and sets a foundation for research ramifications for the study of health policy influences on group-level sociodemographic phenomena. The advantage of the present approach lies in the fact that it embraces the breadth of policy initiatives from different time periods and states. It amalgamates individual pieces of health regulation in such a manner as to allow one to juxtapose the resultant indicator with summary indicators of area mortality and longevity on a level field. The I score indicators of state health policy I evaluate throughout this chapter reflect a plausible cumulative qualitative impact pattern on the sub-national population lifespan.

My analysis relies on many methodological decisions to enable a step-wise, systematic approach. The procedures I follow represent only one of many methods of attaining the result. Multiple aspects of my approach could be operationalized differently, thus changing the course and methods for subsequent parts of the analysis. I provide an overview of potential differences that may arise from application of different methodological solutions. I also detail the unique features that make the present set of methods stand out from the rest. To start off, this study adopts a frequentist paradigm to estimating mortality/longevity using the fixed effects spatial econometric modeling framework for states and county groups. While I try to account for the hierarchical structure of the data by state (i.e., group) clustering and correcting for heteroskedastic errors and serial autocorrelation, one could also consider alternate models that treat nested data hierarchy explicitly. Lacombe and McIntyre (2016) and Lacombe and McIntyre (2017) suggest some such spatial modeling approaches, albeit not within a panel setup. The technical implementation of hierarchical models is more challenging and often computationally burdensome when the number of observations is not small

Table 4.14. Spatial Durbin model (SDM) state-level impact decomposition for the natural logarithm of age-standardized death rate, by sex and SHP indicator.

		I_Z			$I_{t-10,Z}$			$\tilde{\pi}_{10,Z}$		
Sex	Regressor	Direct	Indirect	Total	Direct	Indirect	Total	Direct	Indirect	Total
Both sexes	SHP indicator	−0.015***	−0.008*	−0.023**	−0.013***	−0.008*	−0.021***	0.000	0.000	0.000
		(0.004)	(0.004)	(0.007)	(0.003)	(0.003)	(0.006)	(0.002)	(0.001)	(0.003)
	Median age	0.001	0.000	0.001	0.000	0.000	0.000	0.002	0.001	0.003
		(0.003)	(0.002)	(0.004)	(0.003)	(0.002)	(0.004)	(0.003)	(0.002)	(0.005)
	log(gov't ideology)	−0.010	−0.005	−0.015	0.017*	0.011	0.028*	−0.014	−0.010	−0.024
		(0.007)	(0.004)	(0.011)	(0.008)	(0.007)	(0.014)	(0.007)	(0.006)	(0.013)
	log(% in poor health)	0.025	0.014	0.039	−0.004	−0.003	−0.007	0.052	0.039	0.091
		(0.038)	(0.023)	(0.060)	(0.039)	(0.027)	(0.065)	(0.040)	(0.034)	(0.072)
	log(% Black)	0.038***	0.021*	0.059**	0.042***	0.028*	0.070***	0.037***	0.028*	0.065**
		(0.010)	(0.009)	(0.018)	(0.010)	(0.011)	(0.020)	(0.011)	(0.012)	(0.021)
	D index	−0.002	−0.001	−0.003	−0.002	−0.001	−0.003	−0.002	−0.001	−0.003
		(0.001)	(0.001)	(0.002)	(0.001)	(0.001)	(0.002)	(0.001)	(0.001)	(0.002)
	log(med. HH income)	0.033	0.018	0.050	0.032	0.021	0.053	0.053	0.039	0.092
		(0.052)	(0.030)	(0.080)	(0.054)	(0.039)	(0.091)	(0.055)	(0.045)	(0.099)
	% blue collar	−0.004*	−0.002	−0.006*	−0.004**	−0.003*	−0.007*	−0.005**	−0.004*	−0.008**
		(0.002)	(0.001)	(0.003)	(0.002)	(0.001)	(0.003)	(0.002)	(0.002)	(0.003)
	% urban population	−0.001	0.000	−0.001	−0.001	0.000	−0.001	−0.001	−0.001	−0.002
		(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.002)	(0.001)	(0.001)	(0.002)
	log(% w/out HS diploma)	−0.164***	−0.090*	−0.254***	−0.159***	−0.105**	−0.264***	−0.170***	−0.125**	−0.295***
		(0.033)	(0.037)	(0.063)	(0.034)	(0.040)	(0.068)	(0.036)	(0.047)	(0.075)
Female	SHP indicator	−0.016***	−0.008*	−0.024**	−0.014***	−0.009*	−0.023***	0.002	0.001	0.003
		(0.004)	(0.004)	(0.007)	(0.003)	(0.003)	(0.006)	(0.002)	(0.002)	(0.004)
	Median age	−0.002	−0.001	−0.002	−0.004	−0.002	−0.006	−0.003	−0.002	−0.005
		(0.003)	(0.001)	(0.004)	(0.002)	(0.002)	(0.004)	(0.003)	(0.002)	(0.005)
	log(gov't ideology)	−0.012	−0.006	−0.018	0.016	0.010	0.025	−0.018*	−0.013	−0.031*
		(0.008)	(0.004)	(0.012)	(0.009)	(0.006)	(0.014)	(0.008)	(0.007)	(0.014)
	log(% in poor health)	0.017	0.009	0.025	−0.042	−0.025	−0.067	0.021	0.015	0.036
		(0.044)	(0.024)	(0.067)	(0.043)	(0.029)	(0.071)	(0.047)	(0.036)	(0.082)
	log(% Black)	0.052***	0.026*	0.077***	0.057***	0.035**	0.093***	0.050***	0.036*	0.086***
		(0.012)	(0.011)	(0.020)	(0.011)	(0.014)	(0.022)	(0.013)	(0.014)	(0.025)
	D index	−0.002	−0.001	−0.003	−0.002	−0.001	−0.004	−0.002	−0.001	−0.003
		(0.001)	(0.001)	(0.002)	(0.001)	(0.001)	(0.002)	(0.001)	(0.001)	(0.002)
	log(med. HH income)	0.041	0.020	0.061	0.043	0.026	0.068	0.057	0.041	0.098
		(0.056)	(0.031)	(0.086)	(0.058)	(0.037)	(0.094)	(0.059)	(0.047)	(0.104)
	% blue collar	−0.005**	−0.003*	−0.008**	−0.006***	−0.004*	−0.010**	−0.007***	−0.005*	−0.012**
		(0.002)	(0.001)	(0.003)	(0.002)	(0.002)	(0.003)	(0.002)	(0.002)	(0.004)
	% urban population	−0.001	0.000	−0.001	−0.001	−0.001	−0.001	−0.001	−0.001	−0.003
		(0.001)	(0.001)	(0.002)	(0.001)	(0.001)	(0.002)	(0.001)	(0.001)	(0.002)
	log(% w/out HS diploma)	−0.143***	−0.070*	−0.213***	−0.133***	−0.082*	−0.215**	−0.149***	−0.107*	−0.255***
		(0.034)	(0.031)	(0.058)	(0.038)	(0.037)	(0.070)	(0.036)	(0.042)	(0.072)
Male	SHP indicator	−0.015***	−0.006*	−0.021***	−0.011***	−0.005*	−0.016***	0.000	0.000	0.000
		(0.004)	(0.003)	(0.006)	(0.003)	(0.002)	(0.004)	(0.002)	(0.001)	(0.003)
	Median age	0.000	0.000	0.000	0.000	0.000	0.000	0.001	0.001	0.002
		(0.003)	(0.001)	(0.004)	(0.003)	(0.001)	(0.004)	(0.003)	(0.002)	(0.004)
	log(gov't ideology)	−0.011	−0.004	−0.015	0.014*	0.006	0.021	−0.015*	−0.007	−0.022*
		(0.007)	(0.003)	(0.010)	(0.007)	(0.004)	(0.011)	(0.007)	(0.005)	(0.011)
	log(% in poor health)	−0.017	−0.007	−0.024	−0.010	−0.005	−0.015	0.013	0.007	0.020
		(0.032)	(0.014)	(0.045)	(0.033)	(0.016)	(0.048)	(0.033)	(0.018)	(0.050)
	log(% Black)	0.025*	0.010	0.035*	0.028**	0.013	0.041**	0.025*	0.013	0.038*
		(0.010)	(0.006)	(0.015)	(0.010)	(0.007)	(0.016)	(0.011)	(0.008)	(0.018)
	D index	−0.002	−0.001	−0.002	−0.002	−0.001	−0.002	−0.001	−0.001	−0.002
		(0.001)	(0.001)	(0.002)	(0.001)	(0.001)	(0.002)	(0.001)	(0.001)	(0.002)
	log(med. HH income)	0.002	0.001	0.003	0.008	0.004	0.012	0.028	0.015	0.043
		(0.049)	(0.021)	(0.069)	(0.052)	(0.025)	(0.076)	(0.051)	(0.029)	(0.078)
	% blue collar	−0.002	−0.001	−0.003	−0.003	−0.001	−0.004	−0.003	−0.002	−0.005
		(0.002)	(0.001)	(0.002)	(0.002)	(0.001)	(0.002)	(0.002)	(0.001)	(0.003)
	% urban population	−0.001	0.000	−0.002	−0.001	−0.001	−0.002	−0.001	−0.001	−0.002
		(0.001)	(0.000)	(0.001)	(0.001)	(0.000)	(0.001)	(0.001)	(0.001)	(0.001)
	log(% w/out HS diploma)	−0.174***	−0.068*	−0.241***	−0.178***	−0.081*	−0.258***	−0.176***	−0.090*	−0.266***
		(0.031)	(0.033)	(0.055)	(0.032)	(0.035)	(0.057)	(0.034)	(0.039)	(0.064)

$^{*}p < 0.05,\ ^{**}p < 0.01,\ ^{***}p < 0.001.$

Table 4.15. Spatial Durbin model (SDM) county-group level impact decomposition for the natural logarithm of age-standardized death rate, by sex and SHP indicator.

		I_Z			$I_{t-10,Z}$			$\tilde{\pi}_{10,Z}$		
Sex	Regressor	Direct	Indirect	Total	Direct	Indirect	Total	Direct	Indirect	Total
Both sexes	SHP indicator	−0.002	−0.002	−0.004	−0.005*	−0.007*	−0.012*	−0.001	−0.002	−0.003
		(0.003)	(0.004)	(0.007)	(0.002)	(0.003)	(0.005)	(0.001)	(0.002)	(0.003)
	Median age	−0.005***	−0.006***	−0.011***	−0.005***	−0.006***	−0.011***	−0.005***	−0.007***	−0.011***
		(0.000)	(0.001)	(0.001)	(0.000)	(0.001)	(0.001)	(0.000)	(0.001)	(0.001)
	log(gov't ideology)	−0.009	−0.013	−0.022	0.002	0.003	0.004	−0.010	−0.014	−0.024
		(0.006)	(0.008)	(0.014)	(0.006)	(0.009)	(0.015)	(0.006)	(0.008)	(0.014)
	log(% in poor health)	−0.057***	−0.075***	−0.131***	−0.057***	−0.077***	−0.135***	−0.055***	−0.075***	−0.130***
		(0.011)	(0.015)	(0.026)	(0.011)	(0.015)	(0.026)	(0.011)	(0.016)	(0.026)
	log(% Black)	0.001	0.001	0.002	0.001	0.001	0.002	0.001	0.001	0.002
		(0.001)	(0.001)	(0.002)	(0.001)	(0.001)	(0.002)	(0.001)	(0.001)	(0.002)
	log(D index)	−0.007***	−0.009***	−0.015***	−0.007***	−0.009***	−0.016***	−0.007***	−0.009***	−0.016***
		(0.001)	(0.001)	(0.002)	(0.001)	(0.002)	(0.003)	(0.001)	(0.002)	(0.003)
	log(med. HH income)	−0.131***	−0.173***	−0.305***	−0.132***	−0.180***	−0.312***	−0.132***	−0.180***	−0.312***
		(0.010)	(0.015)	(0.024)	(0.010)	(0.015)	(0.024)	(0.010)	(0.016)	(0.025)
	log(GINI index)	−0.098***	−0.129***	−0.227***	−0.098***	−0.133***	−0.232***	−0.100***	−0.137***	−0.238***
		(0.013)	(0.019)	(0.032)	(0.013)	(0.019)	(0.031)	(0.013)	(0.019)	(0.032)
	% blue collar	0.000*	−0.001*	−0.001*	0.000*	−0.001*	−0.001*	0.000*	−0.001*	−0.001*
		(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
	% urban population	−0.001***	−0.001***	−0.002***	−0.001***	−0.001***	−0.002***	−0.001***	−0.001***	−0.002***
		(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
	log(% w/out HS diploma)	0.005	0.006	0.011	0.005	0.007	0.013	0.005	0.007	0.011
		(0.005)	(0.007)	(0.012)	(0.005)	(0.007)	(0.012)	(0.005)	(0.007)	(0.012)
Female	SHP indicator	−0.002	−0.002	−0.004	−0.005*	−0.007*	−0.011*	−0.001	−0.001	−0.002
		(0.003)	(0.004)	(0.008)	(0.002)	(0.003)	(0.006)	(0.001)	(0.002)	(0.004)
	Median age	−0.005***	−0.007***	−0.012***	−0.005***	−0.007***	−0.012***	−0.005***	−0.007***	−0.012***
		(0.000)	(0.001)	(0.001)	(0.000)	(0.001)	(0.001)	(0.000)	(0.001)	(0.001)
	log(gov't ideology)	−0.011	−0.016	−0.028	0.007	0.010	0.017	−0.012	−0.018	−0.030
		(0.006)	(0.009)	(0.015)	(0.007)	(0.010)	(0.017)	(0.006)	(0.009)	(0.015)
	log(% in poor health)	−0.072***	−0.105***	−0.177***	−0.073***	−0.111***	−0.184***	−0.071***	−0.106***	−0.177***
		(0.011)	(0.016)	(0.026)	(0.010)	(0.016)	(0.026)	(0.011)	(0.017)	(0.027)
	log(% Black)	0.001	0.002	0.003	0.001	0.002	0.003	0.001	0.002	0.003
		(0.001)	(0.001)	(0.002)	(0.001)	(0.001)	(0.002)	(0.001)	(0.002)	(0.003)
	log(D index)	−0.006***	−0.009***	−0.016***	−0.007***	−0.010***	−0.017***	−0.007***	−0.010***	−0.016***
		(0.001)	(0.002)	(0.003)	(0.001)	(0.002)	(0.003)	(0.001)	(0.002)	(0.003)
	log(med. HH income)	−0.115***	−0.167***	−0.282***	−0.116***	−0.175***	−0.291***	−0.115***	−0.172***	−0.287***
		(0.010)	(0.015)	(0.024)	(0.010)	(0.016)	(0.025)	(0.010)	(0.017)	(0.026)
	log(GINI index)	−0.101***	−0.146***	−0.247***	−0.102***	−0.154***	−0.256***	−0.103***	−0.154***	−0.257***
		(0.013)	(0.021)	(0.034)	(0.013)	(0.022)	(0.035)	(0.013)	(0.021)	(0.034)
	% blue collar	−0.001***	−0.001***	−0.002***	−0.001***	−0.001***	−0.002***	−0.001***	−0.001***	−0.002***
		(0.000)	(0.000)	(0.001)	(0.000)	(0.000)	(0.001)	(0.000)	(0.000)	(0.001)
	% urban population	−0.001***	−0.001***	−0.002***	−0.001***	−0.001***	−0.002***	−0.001***	−0.001***	−0.002***
		(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
	log(% w/out HS diploma)	0.004	0.005	0.009	0.005	0.007	0.011	0.004	0.006	0.010
		(0.005)	(0.007)	(0.012)	(0.005)	(0.008)	(0.013)	(0.005)	(0.008)	(0.013)
Male	SHP indicator	−0.003	−0.003	−0.006	−0.006**	−0.007**	−0.013**	−0.002	−0.002	−0.004
		(0.003)	(0.003)	(0.006)	(0.002)	(0.003)	(0.005)	(0.001)	(0.002)	(0.003)
	Median age	−0.003***	−0.004***	−0.007***	−0.003***	−0.004***	−0.007***	−0.003***	−0.004***	−0.007***
		(0.000)	(0.000)	(0.001)	(0.000)	(0.000)	(0.001)	(0.000)	(0.000)	(0.001)
	log(gov't ideology)	−0.007	−0.008	−0.015	−0.003	−0.004	−0.007	−0.007	−0.009	−0.016
		(0.006)	(0.007)	(0.013)	(0.006)	(0.008)	(0.014)	(0.006)	(0.007)	(0.013)
	log(% in poor health)	−0.024*	−0.028*	−0.051*	−0.023*	−0.028*	−0.051*	−0.022*	−0.026*	−0.048*
		(0.010)	(0.011)	(0.021)	(0.010)	(0.012)	(0.021)	(0.010)	(0.012)	(0.022)
	log(% Black)	0.001	0.001	0.002	0.001	0.001	0.002	0.001	0.001	0.002
		(0.001)	(0.001)	(0.002)	(0.001)	(0.001)	(0.002)	(0.001)	(0.001)	(0.002)
	log(D index)	−0.007***	−0.008***	−0.016***	−0.008***	−0.009***	−0.016***	−0.007***	−0.009***	−0.016***
		(0.001)	(0.001)	(0.002)	(0.001)	(0.001)	(0.002)	(0.001)	(0.001)	(0.002)
	log(med. HH income)	−0.132***	−0.153***	−0.285***	−0.133***	−0.156***	−0.288***	−0.132***	−0.159***	−0.291***
		(0.010)	(0.014)	(0.023)	(0.010)	(0.013)	(0.022)	(0.010)	(0.014)	(0.023)
	log(GINI index)	−0.102***	−0.118***	−0.220***	−0.102***	−0.120***	−0.222***	−0.104***	−0.125***	−0.229***
		(0.013)	(0.016)	(0.029)	(0.013)	(0.016)	(0.029)	(0.013)	(0.017)	(0.030)
	% blue collar	0.000*	−0.001*	−0.001*	0.000*	−0.001*	−0.001*	0.000*	−0.001*	−0.001*
		(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
	% urban population	−0.001***	−0.001***	−0.002***	−0.001***	−0.001***	−0.002***	−0.001***	−0.001***	−0.002***
		(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
	log(% w/out HS diploma)	0.003	0.003	0.006	0.004	0.004	0.008	0.003	0.004	0.007
		(0.005)	(0.006)	(0.011)	(0.005)	(0.006)	(0.011)	(0.005)	(0.006)	(0.011)

$^{*}p < 0.05$, $^{**}p < 0.01$, $^{***}p < 0.001$.

(Elhorst, 2014). Bayesian alternatives, such as spatial conditional autoregressive regressions (CAR), make a compelling case for the estimation of hierarchical phenomena as the geographical structure is well suited for a parameterization of multi-level data with well-defined likelihood functions (J. P. LeSage, 2014). A useful property of Bayesian models to pool strength from geographic areas with robust support to produce more reliable estimates has been been previously demonstrated by Besag et al. (1991). In fact, Besag-York-Mollié model has since been widely applied across the multidisciplinary spectrum. These types of models would be quite useful to analyze sub-county areas where data are normally sparse due to unavailability or suppression. Geographic regression discontinuity (GRD) designs offer still another alternative for causal inference of spatially linked data. Though technical challenges remain, GRD implementation allows for triangulation of results obtained via more conventional designs where implicit treatment characteristics of bordering units of analysis that set them apart from one another are attributed and modeled explicitly (Keele & Titiunik, 2015). State health policy is expected to be coincidental with state boundaries and pertaining only to pairs of counties that are separated by a common state border. However, despite its advantages, GRD approaches require a selection of a search bandwidth or adjacency that assumes no—or only localized—spillovers. All of these strategies have its own merits in particular circumstances. In this chapter I take a different route. I pursue a spatial econometric approach by which the entire country worth of sub-national units could be treated consistently and simultaneously, regardless of geographic scale. My approach allows for an explicit accounting of the state- and county-group global and local influences to which these areas could be subjected.

All of the aforementioned techniques perform differently based on the type, number, size, orientation and relationships between the geographic units. The benefit of using small-scale data relates to an ability to gauge the sensitivity of the effects of changes in spatial support. Point data, being the most useful type, can be aggregated into a variety of spatial "containers" that enable evaluation of robustness of models to changes in spatial units of analysis (Openshaw, 1984). For example, research has demonstrated the advantage of using data of very fine spatial granularity (Huie, 2001) to uncover the complexity of inter- and intra-neighborhood mixing of characteristics that vary with race and ethnic background (Catney, 2018). This kind of observations are particularly relevant in light of the modifiable areal unit problem (MAUP). It postulates that changes in borders and degree of aggregation lead to transformations in a spatial relationship structure between units as well as to changes in values of the observation attributes (Openshaw, 1984), ultimately leading to varying conclusions. In this chapter I attempt to navigate the pitfalls of MAUP by fixing the boundaries and spatial relationship structure between the units, and by adopting the same variable definitions for states and county groups. This does not completely eliminate bias, but doing so reduces it in its most predictable realizations. Both state and county-group level results with respect to the state health policy indicators are consistent with the prior expectations. The results demonstrate plausible outcomes and serve as benchmarks to assess sensitivity of the models to geographic scale. Future analyses of mortality in a local context employing any of the aforementioned techniques can thus greatly benefit from the application of fine-scale spatial data. Nevertheless, researchers should remain increasingly vigilant of the effects that MAUP may introduce to their analyses. High spatial granularity often leads to

discontinuities, sparseness and volatility in the estimates. Standardizing the geography (by fixing its spatial extent) to a single set of boundaries across several time points would be an additional safeguard to avoiding some of the dangers posed by MAUP, and ensuring consistency of the comparison and the estimates. In turn, Anselin (2002) warns of a related issue of ecological fallacy in spatial modeling. Aggregate data conceals a great deal of individual heterogeneity that may form sub-groups with distinct patterns from the one observed in an aggregate. Triangulating the findings across multiple geographic scales, time periods, and attribute categories may offer useful insights and a sense of reliability of conclusions drawn from such analyses. Lastly, if one's research depends on small area estimation in longer time series, one must be cognizant of the historical errors that, if not corrected, carry forward affecting the estimates and conclusions based on those results (Baker et al., 2013). This is not a particularly salient concern for the present study, as the data panel I analyze contains only 4 time points, with observation attributes measured using temporally consistent methods.

I would like to remark on several limitations and technical nuances. First, I expect some degree of artificially deflated variance in the results of my spatial panel models. I am currently unable to properly adjust for the clustering of the errors in the model, owing to the lack of software implementation of this procedure, which exists for most non-panel and non-spatial panel approaches (Primo et al., 2007). However, since the estimates of the coefficients of interest are on par with those of the properly adjusted-for non-spatial panel FE models and the low overall error, I conclude that the uncertainty in the final selection of spatial models is unlikely to be of major concern. Next, the endogeneity of the response variable in simultaneous autoregressive (SAR) and spatial Durbin (SDM) models induces a global propagation of mortality dependence. This raises theoretical questions as to the validity of such a process when mortality, say, in a county in California could, albeit to a limited extent, predict mortality in a county in Florida. However, this is not necessarily a problem. The impact of global diffusion effects is mitigated through a series of local spillovers in all the counties throughout the chain defined by the spatial weights matrix. In this sense, the global diffusion effects from any particular county would be strongest among the closest neighbors and would decay geometrically with each subsequent degree of separation. Given that most counties are small and of comparable size, a global spillover from a particular county will not propagate very far beyond its closest neighbors. However, it is important that it does propagate in the vicinity of a state or county group, thus ensuring stronger representation of regional interdependence of mortality patterns.

The stability of model coefficients across the models with each of the 3 proposed state health policy indicators and the associated goodness-of-fit statistics act in support of the validity of the presence and magnitude of an impact of these indicators on all mortality outcomes. Moreover, the low overall amount of error involved suggests that the chosen models are representative of the observed patterns of mortality, undergirded by the theoretical framework, paint a compelling result of the sought-after impact. I find that the cumulative state health policy indicators (the I score and the decade lagged I score indices) are the best two of the three state health policy conditional predictors of mortality outcomes. Assuming a 7-year gap in life expectancy at birth between the lowest and the highest values among states in 2018, each of the I score SHP measures could help to explain up to 30% of this difference. This is quite useful in a study of mortality differentials on a coarse sub-national

level. At the same time I find that mortality/longevity outcomes across counties are less susceptible to the effects of the parent and neighboring states' health policies, as much as they are dependent on local socio-economic and demographic context (e.g., various measures of deprivation). State health policy effects are only predictive of the county-group mortality after a significant time delay and percolate down in a rather attenuated form, as compared to the effect on the state-level outcomes. Therefore, I contend that studies of policy effects on the county or more granular geographies ought to shy away from encompassing summary measures and should incorporate isolated policies or thematic summary groups of policies that may be relevant to an area in question. Of course, smaller levels of aggregation would benefit from the inclusion of predictors related to socio-economic and social structure contexts, such as resource deprivation. In this, the results of the present study corroborate the preponderant findings of a growing body of literature on local causes and determinants of mortality (Centers for Disease Control and Prevention, 2013; Rodriguez et al., 2022; Singh & Siahpush, 2014).

In the course of the exposition of the results I demonstrate the mortality impact of three indicators representing state health policy. On the basis of this evidence I conclude that my prior expectations with respect to the validity of the indicators are largely corroborated. Much in line with my prior hypothesis, the I score and its time-lagged counterpart indeed represent the general outlook on the "positivity" of a state's health agenda. Conversely, the rate of passage of new policies has little-to-no significant bearing on the mortality outcomes considered here. The former two measures are rooted in the notion of cross-sectional and longitudinal cumulativity and confluence of qualitative benefits to population health over time, while encompassing a broad range of policies within different thematic categories and regulatory classes. The latter indicator is tied to an approach of acting-by-proxy for the rate but not the quality of health policy innovation. Though approaches of similar kind are popular in the research on the effects of general policy innovation and serve as a motivation for policy data gathering initiatives (Boehmke et al., 2018; Montez, 2022) those may not be particularly useful within the scope of a more narrow application that I pursue in the present work. Notwithstanding the aforementioned attributes, I cannot implement several additional features due to practical constraints. Arguably, such features may improve the quality of the SHP indicators. Many policies are strategic by design and require a certain time to elapse before the intended impact could be realized. To this end I include the policies that have been adopted starting in 1970, thus preceding the first time point in the data by two decades. Although both the I score indicator and its decade-lagged counterpart make full use of the 20-year "burn-in" period, it is likely that my elaboration on these indicators omits many more important health regulations that were set in motion prior to 1970. Its inclusion would most probably boost its impact on mortality and longevity, in the same directions as I show throughout the latter part of the results section. If the remaining indicators are known and kept the same, then the improvement in the quality of an SHP indicator could explain the mortality gap between the most proactive and the least proactive states, potentially above the reported 13%.

It is equally important to dedicate a few remarks to the model predictors of mortality that I feature throughout my analysis, because an inference is only as good as the ingredients that are involved in its process. I outline a number of such theoretical concepts in chapter 2

and use its operational data representations in the current chapter. Assuming the validity of a the structural causal model for the time being, the quality of the data and the alignment of the quantities with the concepts these data represent play an integral part in the model performance. Granted, it is difficult to characterize many concepts in an aggregate setting, which is the case here, while retaining a parsimonious and practically feasible set of variables. To supplement the SHP indicators I include a number of summary predictors, some drawn directly from secondary data sources and some derived independently on its basis. To put the results of my analysis in conversation with the past literature on predictors of mortality I discuss the most salient findings with respect to the impact of each such measure in the presence of the SHP indicators as controls.

First and foremost, in line with the background research I evaluate indicators that may characterize or align with areal socio-economic deprivation. These include educational attainment, racial minority composition, degree of residential segregation, occupational composition of the local population in the labor force, degree of urbanization, median household income and income inequality. When it comes to educational attainment it is often noted that a very low level of education in adults (e.g., below high school) is more deleterious to one's health than a college degree is beneficial (Barbieri, 2022; Montez et al., 2019). Although I dwell on this dynamic in both the state and county group data descriptively, I find that with the additional contextual factors the effect largely disappears. Indeed, this confirms that circumstances of a particular socio-economic environment exert a strong influence over the effect of education on mortality. Nevertheless, more attention should be paid to spatial disparities in mortality by educational attainment (Montez et al., 2019), as these differentials reinforce the divide in the progress of longevity occurring across multiple axes. Counties or small groups of counties may still be too coarse a level of aggregation for an analysis aimed at addressing this issue. The geographic scale of a county conceals a lot of interesting variation that stems from interaction of local contextual factors and educational attainment. Such an analysis is best carried out either on individuals or on a local neighborhood level. The same can be said with respect to the income and its inequality. As prior research reports for individual outcomes (Chetty et al., 2016) I too find that after accounting for the context higher income significantly improves longevity. However, since this effect is only notable at the level of individual counties and its groupings, but not at the level of states, I would argue that it is closely associated with poverty and resource deprivation as a fine-scale phenomenon.

Other aspects that are tangential but associated with resource deprivation have been extensively examined in sociological research studies. Of those, race, urbanization and segregation have a common role in identifying an area's character due to the associative properties, hence explaining its part in the present analysis. As structural racism and residential segregation continue to permeate parts of the US society to varying degrees so do the consequences for mortality attached to disadvantaged ethno-racial minorities (Brown & Homan, 2024). Segregation in particular diminishes the extent of mixing and integration of disadvantaged groups with the well-off groups within urban and rural communities (Brown & Homan, 2024; Massey & Fischer, 2000). Specifically, African American communities that form parts of inner city ghettos and southern rural enclaves demonstrate a long-standing association with socio-economic and longevity disadvantage, making the group focal in soci-

ological studies (Massey et al., 1987). I find that increasing African American character of an area follows a predictably positive association with increased mortality, albeit to a minor degree and only on the state level. At the same time I show that county group mortality is not sensitive to African American composition in the local population, whereas it is sensitive to residential segregation as such. Contrary to the conclusions from some prior studies, residential segregation may actually offer a weak protective effect to the local population of different ethnic and racial groups. The results of my analysis show that net of the effects of socio-economic environment, racially and ethnically homogeneous communities provide some form of social and material buffer in face of morbidity and risk factors associated with lower life expectancy at all ages. I anticipate that future studies investigating this dynamic on a more granular spatial scale may find stronger evidence for the health and mortality benefits of community shared resources and social capital. Moreover, urban character often dictates the conditions in which segregation may be either advantageous, innocuous, or else harmful to the area residents.

My findings also align with those of Hamidi et al. (2018) in that urban sprawl is associated with increased mortality across the US counties, though the association I demonstrate is weak, given a fairly naive definition of urbanicity I adopt along with the grouping of a sizable number of counties in the data. Ultimately, what qualitative attributes characterize an urban place is a challenging problem without defining a specific subject of a background (Balk et al., 2018). Notwithstanding the challenges in precise characterization of what an urban or a resource-deprived place may be, research shows that socio-economic disadvantage measured by the degree of rurality, residential segregation and poverty hinder the prevention of early mortality (Sparks et al., 2013). Future examination of how various forms of urban character may impact mortality or longevity at a city, county, and state levels would be a valuable contribution to the body of literature on geographic disparities.

I further examine factors that are more proximal to mortality and state health policy association, namely median age and poor health prevalence. These two indicators are important for several reasons. First, its inclusion in the models helps to remove the effects of population aging when making inferences about the impact of SHP on mortality/longevity. Secondly, the demographic processes underlying the final stage of the epidemiologic transition in the US presuppose that the population prevalence of chronic illnesses would continue to grow in the course of the lifespan extension (Olshansky & Ault, 1986). Incorporating these measures in an analysis of health policy thus ensures that the effects of health reform can be discussed within the scope of changing epidemiological landscape and that the conclusions would not misrepresent the true effect of the policies, particularly those targeting health issues in older adults. Lastly, state government ideology is a rather unique and peculiar facet of the analysis that does not weigh in on mortality outcomes per se, but is an indispensable control that places state policy decisions within the shifting political context which cannot be relegated to a nebulous fixed effect in a model. Additionally, government ideology accounts for the views and needs of the electoral base, which in turn is dictated by the generational shifts within it (Fisher, 2020). To understand the effect of a bundle of interdependent policies it is then necessary to disentangle complex legislative mechanisms situated at the heart of demographic, commercial and political environments at various levels of aggregation (Montez & Grumbach, 2023). I implement one particular measure of state

government ideology based on conservatism and liberalism of the states' political representation (Berry et al., 2010) to which the policy determinants are attached. However, more explicit measures of political influence may be considered in future studies, not limited to a group's declared political affinity on a ballot.

Interactions, moderators and mediators

As I point out in the discussion of the results, lower model errors in places with high mortality may be indicative of the stronger predictive potential of the SHP indicators alongside factors that determine socio-economic disadvantage and resource deprivation. On the other hand, the preponderant dynamics of urban places and wealthier areas comprise a greater variety of unobserved factors that may be responsible for the poorer fit. This underscores the importance of nuance when dealing with the spatially granular aggregate data, particularly county and below. This also sets the stage for the future work on the relationship between SHP and mortality, while accounting for characteristics of urbanicity, deprivation, and so forth, expressed through alternative definitions. A more detailed examination and comparison of interaction effects would be in order.

One study suggests that the progressively increasing variance in age at death is becoming more common in the US counties with high levels of socio-economic deprivation (Seaman et al., 2019). In the present study I only include median age as one area-level summary measure to account for the aging in the population. Perhaps introducing age quintiles instead and testing its interactions with measures of income or poverty, for example, may present valuable insights, particularly in the presence of relevant policy indicators. In fact, systematic treatment of distributional properties of key variables related to area socio-economic deprivation and its associations with mortality outcomes is an established practice in several works to date (e.g., see Dukhovnov and Barbieri (2022) and Singh (2003)).

Some literature suggests that the combination of income inequality and poverty has a multiplier effect on the association with mortality outcomes (Massey & Fischer, 2000). I do not find very strong evidence in support of this claim, given that income inequality is not associated with increases or decreases in mortality to the same extent as median household income. However, I recognize that compounding effects may be more pronounced, particularly in smaller counties and sub-county geography. The interaction between poverty and inequality in the scope of health policy analysis merits further investigation, specifically surrounding any state policies that are aimed at reducing the income inequality gap to improve population health outcomes.

Other two- and three-way interactions may prove beneficial for future analyses, namely surrounding the intersection of race, segregation and urbanicity. The concept of inner-city ghettos and aftereffects of historic redlining practices may provide additional insights into the changing urban landscape as well as the changing impact of institutionalized racism. Examining these interactions with an indicator of state health policy could inform future research on social determinants of health and mortality in a spatial context. Likewise, interactions between race and education would be interesting in relation to SHP and mortality analysis, since the minority races (aside from Asian) tend to gain increments of years of life with college degree or higher out of proportion relative to non-Hispanic Whites (Currie & Schwandt, 2016).

Contributions

From the above discussion it becomes evident that it might be premature to declare the discovery of the true causal effect of state health policy on mortality for a variety of important reasons. Some of these barriers involve the choice of variables to represent theoretical concepts including, but not limited to, overlooking key omitted variables, data measurement errors, model mis-specification and estimation inefficiency. Despite this seeming hindrance my step-wise analysis demonstrates the persistent sign of positive influence of state health policy on lowering mortality outcomes, particularly on the level of a state.

I nonetheless demonstrate that state health policy remains an effective predictor of the aggregate female, male, and both-sex period mortality and longevity measures when accounting for a range of socio-economic, political, and demographic factors. The effect is isolated in the process of mortality transition and changes in societal trends over the course of nearly 3 decades. I also show that temporally cumulative impact of state health policies is advantageous to mortality reduction, often long after policy proposals have been implemented. Indeed, the multi-level comparison of the outcome illustrates that county mortality only becomes sensitive to policy changes after a significant time delay. Mirroring the benefits of prevention as opposed to treatment on an individual level, this finding could serve as impetus for state governments to design greater number of preventive, rather than reactive health policies. Once effective policies stay in place for an extended period the lasting cumulative impact percolates down the levels of spatial hierarchy imposing health improvement and, as a result, mortality reduction in the target populations.

4.6 Conclusion

Health reform remains one of the most enduring, and yet ever-changing determinants of population health. Due to the nature of health policy complexity its inclusion into research analyses has seen limited application, mostly restricted to individual pieces of health regulation in isolated places. The conclusions reached in the present study represent only the first of many initial steps toward broad scale simultaneous analyses involving multiple health policies and localities. It is important to keep in mind that the conclusions I draw from the results presented in this study apply to population groups and not to individuals. Often policy work aims to address a particular health concern in a population, such that it could apply to some individuals but not to others. Likewise, certain policies are designed to act over a long period of time, whereas the other are more immediate in nature along with its consequences for the population health. A great many studies in public health, demography, epidemiology, political science and related fields clearly illustrate the impact of individual health regulations on outcomes in a particular population sub-group, be those related to traffic fatalities, cancers, or substance abuse. By contrast, I test a summary indicator for proactive legislative initiatives that serves as a gauge for the sub-national population health resilience potential, with outcomes measured by commonplace mortality/longevity summary indicators. The findings of this study show that greater degree of health positivity in a continuously expanding bundle of state health reforms, acting in unison and over extended time, has a substantial impact throughout the state populations and beyond. This effect

on mortality is not only attenuated, but is also time-lagged at lower geographic scales (e.g., counties) in favor of local socio-economic contextual factors that best describe the lived experience of communities on the ground. At the same time, the results of this study are a testament to the proposed idea that demographic studies of phenomena at most finer-scale geographies ought to be *spatially aware* (Matthews & Parker, 2013; Wachter, 2005) by nature of the place-specific social and economic interactions that give rise to said demographic outcomes. It is only a matter of *how* future study designs implement spatially-linked components to improve the estimation methodologies and refine the understanding of the type and magnitude of localized factors contributing to the progress in human longevity.

Conclusion

The past four decades in the United States mark the period of changing fortunes for the progress in mortality reduction. The downward divergence of the US longevity indicators from the OECD countries in the recent decades is a by-product of a multi-faceted interaction of structural, socio-economic, and racial inequalities (Barbieri, 2022; Masters et al., 2022; Rogers et al., 2020). Various aspects of health and mortality disparities between different places cannot be effectively explained by individual behavioral characteristics of the respective population groups. Situational background in a given place provides valuable information in the process of unraveling the reasons behind the population health inequalities (Kindig & Cheng, 2013). The analysis put forward in the current work thus heeds the advice of the recent research to identify additional mechanisms that lead to mortality disadvantage in various parts of the country and as a whole. Specifically, my analysis evinces the role of state health policy (SHP) as a structural factor mediating the relationship between longevity and socio-economic condition of the population in a sub-national setting.

Present contributions

I highlight two primary contributions of the present work to the literature on institutional determinants of mortality. First, I introduce a novel SHP index, or the I score. It features several important advantages for the study of area-level mortality and other phenomena. The I score index describes a collective action of state health policies accumulated over time. In addition, it is a thematically decomposable and numerically relative quantity with respect to cross-sectional maxima. Hence its qualitative component transposed onto a numeric scale represents the relative extent of a state's health promotion efforts, or "health positivity". The I score is not a mere tally or rate of policy introduction, as compared to the benchmark indicator of the relative fraction of new policies passed over a prior decade. Instead, it carries the memory of existing local policy context forward in time and relative to other states. Unlike analyses evaluating the impact of individual policies on an outcome, the use of I score is not restricted geographically because it relies on a synthesis of thematically harmonized regulations across all the US states over multiple decades. The capacity of the I score to serve as a summary measure in a study of population-level phenomena thus makes it a useful counterpart to many geographically linked social and demographic summary indicators.

The second salient contribution of the current dissertation is the application of spatial econometric models to the study of state health policy impacts on sub-national mortality.

The main benefit of this spatial approach is that it describes the magnitude and significance of spatial dependence and spillover effects surrounding the SHP-mortality association. At the state level, the indirect or spillover effects on mortality in neighboring states amount to as much as one-third of the explained variation. Specifically, of the tested models, spatial Durbin model offers the best fit in both state and county group levels. It describes the data generating process by which mortality in an area equals a sum of an endogenous spatially lagged mortality, spatially lagged as well as own exogenous predictors. The relationship between SHP and mortality is placed in a comparative context where the interplay of social, economic, demographic, and political influences explains the differences across the temporal and spatial dimensions.

Connections to the literature

Beyond its main contributions the current work engages various discussion points of research on causes and determinants of mortality. Recent studies report an increase in the number of deaths by cause that disproportionately affect the middle-aged segment of the US population (Dwyer-Lindgren et al., 2016). These causes resulted in a greater loss of person-years across the US states and counties, as compared to more old-age affine patterns of mortality in other developed nations. Among such direct and proximal causes are maternal deaths (Carroll, 2017; Kindig & Cheng, 2013), opioid and alcohol abuse, and suicide (Case & Deaton, 2015, 2017), deteriorating physical, economic and social living conditions precipitating in higher morbidity due to obesity, smoking, and mental health crises (Browning et al., 2011; Mueller & Tighe, 2007; Nandi & Kawachi, 2011). Many of these causes of death take root at the intersection of social inequalities, mounting economic pressures, and inadequate local public policy responses. While a number of research studies delve into examining socio-economic deprivation as a factor in the rise of mortality disparities, I choose to focus my attention on the role of public policy action in the same outcome. In the course of this work I demonstrate how positive long-term health policy influences convert into meaningful sub-national area longevity outcomes. Moreover, I note that policy effects are dependent on the choice of geographic scale and include sizable diffusion and spillover effects from the closest area neighbors. My findings highlight that up to 30% of the state-level geographic mortality disparities by sex could be attributed to the effects of local health legislation, of which approximately one-third is due to the health policies adopted by the closest state neighbors. With the change in spatial resolution to the level of a county, mortality disparities between 1990 and 2018 were influenced predominantly by socio-economic and demographic factors. The overall state health policy effect, though halved and delayed relative to the states, offered up to about 1 year of additional life expectancy at birth and up to half an additional year at age 65 for county residents in the most health-proactive states. This goes to show that the influence of local health regulation should not be dismissed as negligible in the context of sub-national analyses of mortality and its risk factors.

Due to the fact that health ordinance falls under the purview of the state governments the responses to public health follow the respective state legislative agenda. The weight of government initiatives and legal processes directed toward population health management cannot be undervalued. Research shows that local health departments prove effective com-

bating opioid overdose thanks to the knowledge of the local area specificity and efficiency in reaching the affected populations (Feuerstein-Simon et al., 2020). Likewise, aspects pertaining to urban planning infrastructure often reflect a substantial share of states' and local governments' long-term health development goals (Giles-Corti et al., 2016). The gradual replacement of lead water mains, passing asbestos regulation, instituting pollution caps and smoking bans, development of green spaces, regulating work safety environment and many other reforms deliver impacts to the common living conditions that reach beyond individual spheres of influence. Although the outlined aims of this study do not directly address these fine points of interest, I note that modeling the association between health policy and mortality in the presence of contextual factors typically yields poorer fits in dense urban clusters due to a greater variety of such unaccounted factors and interactions that could alter population outcomes.

Another key function of state governments is to design plans for and to respond to imminent public health crises. Such was the case during the recent COVID-19 pandemic when states issued own regulation in consultation with the federal Center for Disease Control and Prevention in an attempt to curb the spread of the virus. Unfortunately, this is where local political agenda got in the way of expedient and effective interventions. This partisan political phenomenon gained unprecedented notoriety by way of the media outlets. Despite the attention brought to the issue, much like socio-economic disadvantage (Dukhovnov & Barbieri, 2022) and partisan health politics (Rodriguez et al., 2022), mortality disparity mechanics did not change course with the onset of the pandemic. These processes were only an extension of the socio-economic and latent political ills that existed prior to 2020, many of which also gave rise to the peak of the opioid crisis in the 2010s (Case & Deaton, 2017).

By conducting my research within the framework of spatial analysis, I build on the work that draws on the evidence that contextual attributes of a place serve as a background to geographic health inequalities (Cummins et al., 2007). In fact, growing interest in topics surrounding spatial and contextual determinants of health led to the development of tools and resources for geographically linked data analysis (Dush et al., 2023). Notably, the contribution of political factors to local health becomes recognized by the research community with an increasing frequency. Indeed, as of the latest iteration (February, 2024), IPUMS Contextual Determinants Of Health series include variables on political participation of citizens in the 2020 election by state and county (Van Riper et al., 2024). Similar election participation data are made available via Social Explorer (Social Explorer, 2024). The descriptive measures of my analysis corroborate the motivation for such a push. The most recent decade has seen a deepening divide in the association between political representation and mortality outcomes on the ground. Ideological polarization intensifies geographic differences in health legislation (Oliver, 2006). This fault line has become increasingly evident in the recent decade insofar as the majority Republican and Democrat state governments respond differently to issues related to reproductive rights, firearm legislation, social safety net programs for economically vulnerable populations, and acute health shocks, such as the recent COVID-19 pandemic. In this sense, state health policy will likely stand out as an even more salient predictor in contemporary and future analyses on its effects on population health and mortality than I show in the course of the present work.

Future directions

The outcome of the present study answers the question of how impactful local health regulation has been for mortality across the US states and counties over the past 3 decades. On the other hand, the conclusions reached in this study reveal several new directions for future scientific inquiry into the role of sub-national legislation in health and mortality differentials.

On the substantive level, there is still a relative paucity of research using contextual data to estimate local and hyper-local demographic phenomena. Health regulation has been understudied relative to other socio-economic determinants of mortality and its inequality. To disentangle geographic health and mortality disparities in the United States it is imperative to have an understanding of what population-level measures are taken by local authorities to alleviate major health concerns among the residents. Most fine details of health policies are debated in state assemblies, signed off on by state governors, while the majority of health reform activism takes place in and around municipalities. Though important in many respects, concurrent national political ideology and legislative activities are not directly reflected in the healthcare administration, provisions, and regulatory measures that citizens experience in their day-to-day lives. In my work I attempt to synthesize universal measures of health policy that could used to compare the local governmental health interventions across the US. However, this comes at a cost of the loss of precision and detail for any individual geographic instance. An interesting avenue for future research to explore is then to look at grouped components of any such conceivable, all-encompassing measures of state health policy. An approach like that would strike a balance between generality of coarse summary measures and specificity of individual local regulatory actions. Although the reasons and benefits to evaluating the impact of individual pieces of health legislation are clear, individual institutional actions may not always convey important information about the associations those induce with the existing regulatory background. For this reason, some authors advocate for examining the legal and commercial determinants of health more holistically and in the presence of a place-specific context (Montez & Grumbach, 2023). One way to advance the understanding of how health reform shapes the outcomes in a locality, such as a county or smaller, is to examine a bundle of measures that are better adapted to that particular geographic area. Specifically, as disparate communities sharing a unique socio-demographic, economic, and epidemiologic environment these groups experience health and mortality shocks differently. In chapter 3 I propose a template for composite thematic and regulatory class-based measures of state health policy that could cast light on the effects of thematic policy bundles aimed at tackling health concerns in an area. Future developments in this direction would create a more nuanced approach to inferences for small areas, thus obviating the need for individual microdata. In fact, the analysis presented in chapter 4 characterizes the overall summary indicator of state health policy as a better predictor for state and regional populations, whereas the thematic group components of an indicator are hypothesized to be better fit for counties and smaller geography. For the latter method, incorporation of additional policy data will be necessary to avoid sparseness and discontinuities in the thematic component measures, as is the case in the present analysis of mere 110 harmonized policy groups.

In line with the above point, further work is needed to expand upon the wealth of

public policy data. Fortunately, a great number of new policy data bases have sprung up in the recent couple of decades, freely available to researchers in all fields of science. Of those, I use SPPD, SPID, and CDC repositories to produce the analysis in this study. The outcome of chapter 3 clearly demonstrates the value of combining policy metadata to advance the understanding of contemporary and lagged collective impacts on the aggregate mortality outcomes. Other sources of publicly available secondary data have now begun including additional proximal indicators relating to governance and politics at multiple scales of aggregation. Incorporating these associated elements may provide additional qualitative perspective on findings of policy related analyses.

I also find that the decade-lagged measure of SHP elicits a mortality/longevity response distinct from that of the contemporaneous measure, albeit only within counties, but not within states. I conclude that mortality in the county populations is more sensitive to the socio-economic context of an area, whereas only the effect of time-lagged state policy is significant, despite being smaller in magnitude than in states. This raises a question of whether or not the collective policy action takes more time to percolate down the levels of geography and come into force, or whether the effect is less important in view of more prevalent local determinants of mortality. This question merits further investigation, involving sensitivity testing of multiple SHP indicators (including contemporaneous) to various time lag bandwidths and geographic scales.

When it comes to mortality, delayed publication of vital registration data, individual anonymity and confidentiality concerns limit the data collection and its distribution, which adds to the methodological complexities in analyses that often produce imprecise results. In turn, this undermines the efforts of effective small-scale geographically targeted policy interventions. The complete and accurate assessment of population processes at the level of a county, city borough or a neighborhood is often desirable for effective policy engagement and forecasts. Various statistical techniques for small area estimation have been used to infer the missing information on larger sub-national units, such as states, counties or municipalities (National Center for Health Statistics (U.S.), 2018). Nevertheless, care ought to be taken to account for multiple sources of bias as well as for the area specifics. My work shows that finer levels of geographic aggregation are characterized by higher levels of heterogeneity and variance in the attribute components. Additionally, future studies should take care to properly define the scope of geographic units of analysis. What boundaries define a "place" in the context of health interventions and reforms may differ from the administrative boundaries (Macintyre et al., 2002). Compositional attributes of smaller areas are thus more sensitive to the definition.

Research involving spatial analyses should keep in mind the potential pitfalls of the modifiable areal unit problem (MAUP) (Openshaw, 1984). Changes in geographic scale, location, and orientation have important implications for the results-based inference. If there is no feasible alternative area or a geographic scale to check the robustness of the results, one could apply alternative methods that do not depend on the same set of geographic units and spatial relationships among them. For example, Skovron and Titiunik (2015) and Keele and Titiunik (2015) demonstrate how geographic discontinuity designs could be used to study causality in social phenomena, an approach that is methodologically distinct from spatial weights-based approaches (Kamenetsky et al., 2019; J. P. LeSage, 2014). In addition, in a

study of areal phenomena caution needs to be exercised to avoid ecological fallacy (Anselin, 2002). It is important to remember that inferences made about an area, no matter how large or small, do not represent its "average" inhabitant (or a small group), but rather describe the indivisible collective encompassed by the area boundaries.

Limitations

With due understanding that my analysis involves many theoretical and methodological decisions, it comes with several limitations. First, the qualitative model of state health policy impact on mortality may need revisions to components of the structural causal model. For example, breaking down the nodes representing such variables as age or residential segregation into more precise concepts may improve the understanding of what aspects of age and residential segregation impact other variables within the structure. It would also make finding more precise quantitative representations of these notional concepts easier. However, doing so would also complicate the model considerably, adding nodes and edges representing additional relationships. As a consequence, quantitative estimation of an effect may become unwieldy. At present, I have reservations about using a limited number of parent nodes to represent the directional effect on mortality emanating from population health risk factors. It will be necessary to devote more attention to separating out components that hold much weight in determining the strength of association with nodes proximal to mortality.

Second, when it comes to the estimation of mortality using spatial panel models, at present stage I am unable to fully adjust for all sources of heteroskedasticity and clustering within the spatial longitudinal panel setup, particularly for the county group subset. Future work is needed to improve the estimation of variance around the nested longitudinal data. To my knowledge some previous studies have explored this possibility with distance based Conley errors (Bester et al., 2011; Conley & Molinari, 2007). These methods could feasibly be extended to areal units with adjacency structure with open-source software implementations. An equally pertinent task is a re-evaluation of the fixed effects approaches to estimate causal average treatment effects, as I do in the present work. This cautionary matter emerged in light of the recent developments debating the commonplace application of models that violate fundamental assumption of the difference-in-differences without proper consideration or statistical adjustment (Goodman-Bacon, 2021).

Another important methodological point that future studies should address is migration. The estimates for a small area the size of a county or smaller would likely be quite sensitive to major or sudden population inflows or outflows. Depending on the scale of migration and the temporal frame of a study, this could lead to significant compositional changes and, consequently, to instability in areal demographic estimates. Therefore, factoring in migration in an analysis of a small area population would be a necessary prerequisite, unless it could be safely assumed as constant or otherwise inconsequential.

Finally, from a conceptual standpoint, I acknowledge the standing issue of population health selection. As I mention at the structural causal modeling stage, tracking decisions of a particular demographic to gravitate toward health-promoting areas is not a straightforward process. Ultimately, I choose to encapsulate this concept by unit-specific fixed effects in my mathematical models. Of course, this approach is only valid if the rate and manner

of population health selection into particular states or counties is indeed fixed throughout the period of observation. Although I assume this is the case for the sake of expediency, I recognize that a more nuanced approach to accounting for health selection into particular areas is necessary both at theoretical and applied analysis stages. Despite the drawback of the current study design I contend that this aspect is weakly dependent on SHP that is defined at the state level, and not necessarily in specific areas where healthier populations congregate. Therefore, it is unlikely that certain strata of the population choose a particular county within a state as a place of residence solely on the basis of that state's health policy. Or, perhaps they do, if a particular policy happens to apply to a specific municipality or district. This contention is likely untrue when it comes to selecting into a state of residence. Should inter-state health selection be sizable and the relationship between SHP and population health selection into particular states unstable over time, the model coefficients could be biased. In the meantime, I urge the reader to exercise caution in referring to the prediction estimates for individual areal units.

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Appendices

A.1 Appendix to Chapter 3

Table A.1.1. Description of policies used in the computation of the state health policy measures.

Abbreviation	Description	Data set	Assessed positivity	Category	Class
comsrv12	Mandated Community Service After 1St Or 2Nd Offense	SPID	1	1	1
dwi reform	State Reform For Driving Intoxication Limits	SPID	1	1	1
frontpass seatbelt	Requiring Front Passengers To Wear Seatbelts	SPID	1	1	1
mprisdm	Mandates Prison Time For First Dui	SPID	1	1	1
primaryenforce all	Police May Pull Over Drivers For Driving Without Seatbelt	SPID	1	1	1
primaryenforce minor	Police May Pull Over Drivers For Minors Riding Without Seatbelt	SPID	1	1	1
primseat	Primary Seat Belt Laws	SPID	1	1	1
damagereduce	Presence Of A Law That Decreases Monetary Awards For Injuries In Law Suits For Seatbelt Nonuse	SPID	1	1	2
allpass seatbelt	Requiring All Passengers To Wear A Seatbelt	SPID	1	1	4
bikehelmet	Requiring Helmet For Riding Bike	SPID	1	1	4
helmetlaw 17	Motorcyclists 17 And Under Required To Wear A Helmet	SPID	1	1	4
helmetlaw pass	Helmet Required For Motorcycle Passenger	SPID	1	1	4
concealed carry	State Permit For Conceal And Carry	SPID	0	2	1
guncontrl bc private handgu	Does The State Require Background Checks For Private Handgun Sales?	SPID	1	2	1
guncontrl bc private rifl	Does The State Require Background Checks For Private Rifle Sales?	SPID	1	2	1
guncontrl waitperiod handgu	Is There A Waiting Period After Handgun Sales?	SPID	1	2	1
guncontrol assaultweapon ba	Is There A Waiting Period After Assault Weapon Sales?	SPID	1	2	1
guncontrol licenses dealer	Does The State Have Any License Requirements For Manufacturers Or Dealers?	SPID	1	2	1
guncontrol opencarr	Does The State Have Open Carry Laws?	SPID	1	2	1
guncontrol satnightspecial ba	Does The State Ban “Saturday Night Special\ Handguns?"	SPID	1	2	1
w guncontrl bc dealer handgu	Does The State Require Background Checks For Buying Handguns From Dealers?	SPID	1	2	1
w guncontrl bc dealer rifl	Does The State Require Background Checks For Buying Rifles From Dealers?	SPID	1	2	1
w guncontrol registration requi	Does The State Have Registration Requirements For Rifles?	SPID	1	2	1
w guncontrol waitingperio	Does The State Have A Waiting Period For All Gun Purchases?	SPID	1	2	1
controlled substances act	Drug Policy Under Which The Manufacture, Importation, Possession, Use And Distribution Of Certain Substances Is Regulated	SPID	1	3	1
imitcontsub	Regulating Imitation Prescription Drugs	SPID	1	3	1
methpre	Restrictions On Otc Medications With Methamphetamine Precursors	SPID	1	3	1
salvia	Regulation Of Salvi Divinorum	SPID	1	3	1
drugs medical marijuan	Is It Legal To Use Marijuana For Medical Purposes?	SPID	1	3	3
medmar	Indicates whether medical marijuana was legal in a given year (0=no, 1=yes)	SPDD	1	3	3
recmar	Indicates whether recreational marijuana was legal in a given year (0=no, 1=yes)	SPDD	0	3	3
goodsam911	Good Samaritan Protection For Reporting Overdose	SPID	1	3	4
naloxone	Naloxone access laws that provide civil or criminal immunity to licensed health care clinicians or lay responders for administration of opioid antagonists, such as naloxone hydrochloride, to reverse overdose; 0 = no law in effect, 1= law in effect	SPDD	1	3	4
pdmp access	Prescription Drug Monitoring Program (PDMP) access laws provide access to the electronic database that tracks controlled substance prescriptions in a state; 0 = no law in effect, 1= law in effect. For more information, see the original data source	SPDD	1	3	4
pdmp mandate	Mandatory Prescription Drug Monitoring Programs (PDMPs) require prescribers under certain circumstances to access the PDMP database prior to prescribing opioids; 0 = no law in effect, 1= law in effect	SPDD	1	3	4
pillmill	Pain clinic laws that regulate the operation of pain clinics; 0 = no law in effect, 1= law in effect	SPDD	1	3	4

Abbreviation	Description	Data set	Assessed positivity	Category	Class
prescript	Prescription laws that impose limitations on the number of days that medical professionals dispense opioids for acute pain; 0 = no law in effect, 1= law in effect	SPDD	1	3	4
admittpriv	Requirement That Abortion Clinics Must Have Admitting Privileges	SPID	-1	4	1
clinic access	No-Protest Zone Around Abortion Clinic	SPID	1	4	1
foca	Codifies Roe V Wade In State	SPID	1	4	1
medicalrest	Restrictions On Medical Abortions	SPID	-1	4	1
parconsent	Requires Parental Consent For Abortions	SPID	0	4	1
physreq	Require A Licensed Physician For Abortion	SPID	1	4	1
refuseserv	Physicians Can Refuse To Do An Abortion	SPID	-1	4	1
sexselect	Prohibits Abortion On Sex-Discrimination	SPID	0	4	1
trap	Targeted Regulation Of Abortion Providers (Trap) Laws	SPID	-1	4	1
trap hosp	Requires Hospital Like Facilities Or Other Physical Plant Requirements At Abortion Clinics	SPID	1	4	1
trap lic	Requires Additional Licensure For Abortion Providers	SPID	1	4	1
waiting	Require Waiting Period For Abortions	SPID	-1	4	1
aborpreroe	Abortion Pre-Roe	SPID	1	4	3
fundslife	Bans Public Funding To Be Used For Abortion	SPID	-1	4	3
fundslifehea	Bans Public Funding For Abortion, But With More Exceptions	SPID	-1	4	3
ins stateexch	Bans Abortion Coverage In State Exchanges	SPID	-1	4	3
insprivate	Ban On Insurance Coverage By Private Insurance For Abortions	SPID	-1	4	3
inspublic	Ban On Insurance Coverage By Public Insurance For Abortions	SPID	-1	4	3
postvia	Ban On Post-Viability Abortion	SPID	-1	4	3
pre20wk	Pre-20 Week Abortion Ban	SPID	-1	4	3
pubemployees	Ban On Public Employees Conducting Abortions	SPID	-1	4	3
pubfacilities	Ban On Using Public Facilities For Abortions	SPID	-1	4	3
wk20via	Ban Abortions After 20 Weeks	SPID	-1	4	3
const protect	The State Constitution Protects Access To Abortion	SPID	1	4	4
continsurance	Mandates Contraception Coverage By Insurance	SPID	1	4	4
bar	Degree of indoor clean air smoking laws for bars. 0 = no restrictions, 1 = ban	SPDD	1	5	1
drugs boehmke zeroto	Does The State Have A Zero Tolerance Law For Blood Alcohol Levels <0.02 For Individuals Under Age 21?	SPID	1	5	1
dui08	.08 Per Se Penalty For Dui	SPID	1	5	1
hostliability	Laws Impose Liability Against Hosts Of Underage Drinking Events	SPID	1	5	1
restaurant	Degree of indoor clean air smoking laws for restaurants. 0 = no restrictions; 1 = ban	SPDD	1	5	1
workplace	Degree of indoor clean air smoking laws for workplaces. 0 = no restrictions; 1 = ban	SPDD	1	5	1
worksite-banstate	Smoking Ban In All Indoor Workplaces	SPID	1	5	1
beer offadv	The retail off-premises ad valorem excise tax (see Definitions) levied on beer of 5% ABV	SPDD	1	5	2
cigtax	Cigarette Tax	SPID	1	5	2
spirit offadv	The retail ad valorem tax is given for off-premises spirits of 40% ABV	SPDD	1	5	2
tobaccotax	State tax on cigarettes per pack, USD	SPDD	1	5	2
wine offadv	The retail off-premises ad valorem excise tax (see Definitions) levied on wine of 12% ABV	SPDD	1	5	2
advertban	Banning Of Cigarette Advertising	SPID	1	5	3
barbanstate	Full Smoking Ban In Bars	SPID	1	5	3
drugs smoking ban restaurant	Does The State Ban Smoking In Restaurants?	SPID	1	5	3
drugs smoking ban workplace	Does The State Ban Smoking In All Workplaces?	SPID	1	5	3
restaurant-banstate	Smoking Ban In All Restaurants In State	SPID	1	5	3
smokeban	Statewide Smoking Ban	SPID	1	5	3
sundayban	Banning Alcohol Sales On Sundays	SPID	1	5	3
hiport	Health Insurance Portability	SPID	1	6	1
hiprecon	Health Insurance Preexisting Conditions Limits	SPID	1	6	1
banfaninc	Ban On Financial Incentives For Doctors To Perform Less Costly Procedures/Prescribe Less Costly Drugs	SPID	1	6	3
bangag	Prohibits Agreements That Limits A Doctor'S Ability To Inform Patients Of All Treatment Options	SPID	1	6	3
gap	Pharmacists Are Not Allow To Deny Prescriptions	SPID	1	6	3
alc hlplncov	Indicates whether health plans must cover alcohol-related disorders, with specific limitations (0=no, 1=yes)	SPDD	1	6	4
alc hlplnofr	Indicates whether health plans must offer an option of coverage for treatment of alcohol-related disorders, but are not required to cover unless offer is accepted (0=no, 1=yes)	SPDD	1	6	4

Abbreviation	Description	Data set	Assessed positivity	Category	Class
higissue	Guaranteed Issue Of Health Insurance	SPID	1	6	4
higrenew	Guaranteed Renewal Of Health Insurance	SPID	1	6	4
medicaidexp	State implementation of Medicaid expansion	SPDD	1	6	4
boehmke snrpres	Does The State Provide Pharmaceutical Coverage Or Assistance For Seniors Who Do Not Qualify For Medicaid?	SPID	1	7	4
duration max	Maximum number of weeks of unemployment benefits in a calendar year	SPDD	1	7	4
eitc	State EITC Rate as Percentage of Federal Credit	SPDD	1	7	4
psl	Indicates whether a state had a paid sick leave (PSL) law in effect at any time during the calendar year (1=PSL law in effect, 0 otherwise)	SPDD	1	7	4
SNAP	Maximum monthly allotment Food Stamp / SNAP benefit for 2-person family, USD	SPDD	1	7	4
ui max	Maximum unemployment insurance benefit per calendar year. Calculated by taking the product of the wba max variable and the duration max variable	SPDD	1	7	4
wba max	Maximum Weekly Benefit Amount for unemployment, USD	SPDD	1	7	4
hmomod1	Health Maintenance Organization Model Act (First)	SPID	1	8	1
hmomod2	Health Maintenance Organization Model Act (Second)	SPID	1	8	1
anatomical gift (1968)	Sets Framework For The Donation Of Organs And Other Body Parts	SPID	1	8	4
anatomical gift act (1987)	Framework For Donation Of Organs Other Body Parts (1987 Version)	SPID	1	8	4
anatomical gift act (2006)	Framework For Donation Of Organs Other Body Parts (2006 Version)	SPID	1	8	4
breastdensity	Breast Density Notification Acts	SPID	1	8	4
colcanscr	Colorectal Cancer Screenings	SPID	1	8	4
medicaid stateadoptio	Does The State Have A Medicaid Program?	SPID	1	8	4
newbornheart-screen	Coverage For Newborn Screening For Critical Congenital Heart Disease	SPID	1	8	4
transboundary pollution reciprocal access act	Overcomes Procedural Obstacles That Prevent A Pollution Victim In One State Or Province From Seeking Enforceable Remedies In The State Or Province Where The Pollution Originated.	SPID	1	8	4
universal health care	Universal Healthcare Bill	SPID	1	8	4
w ec access expan	Has The State Expanded Access To Emergency Contraception?	SPID	1	8	4

Recoding non-binary SPDD variables to harmonize with the remainder of the data set

(beer offadv, wine offadv, spirit offadv) Beer, wine, spirit VAT (1 = above 10% of minimum hourly state wage, 0 = otherwise)

(beer stax, wine stax, spirit stax) Beer, wine, spirit per gallon tax (1 = above 25% of state's minimum hourly wage, 0 = otherwise)

(cig cost) Cost of cigarettes per pack (1 = above 50% of minimum hourly state wage, 0 = otherwise)

(SNAP) Supplementary Nutrition Assistance Program per 2-person family (1 = above a 15% of federal poverty line for the family of 2, 0 = otherwise)

(ui max) Maximum annual unemployment insurance amount (1 = above 100% of a state's annual minimum annual full-time equivalent wage, 0 = otherwise)

(EITC) State's Earned Income Tax Credit as a percentage of federal EITC (1 = 10% or more, 0 = otherwise)

Summary of correlations between various measures of mortality and State Health Policy, ANOVA quantile-quantile plots.

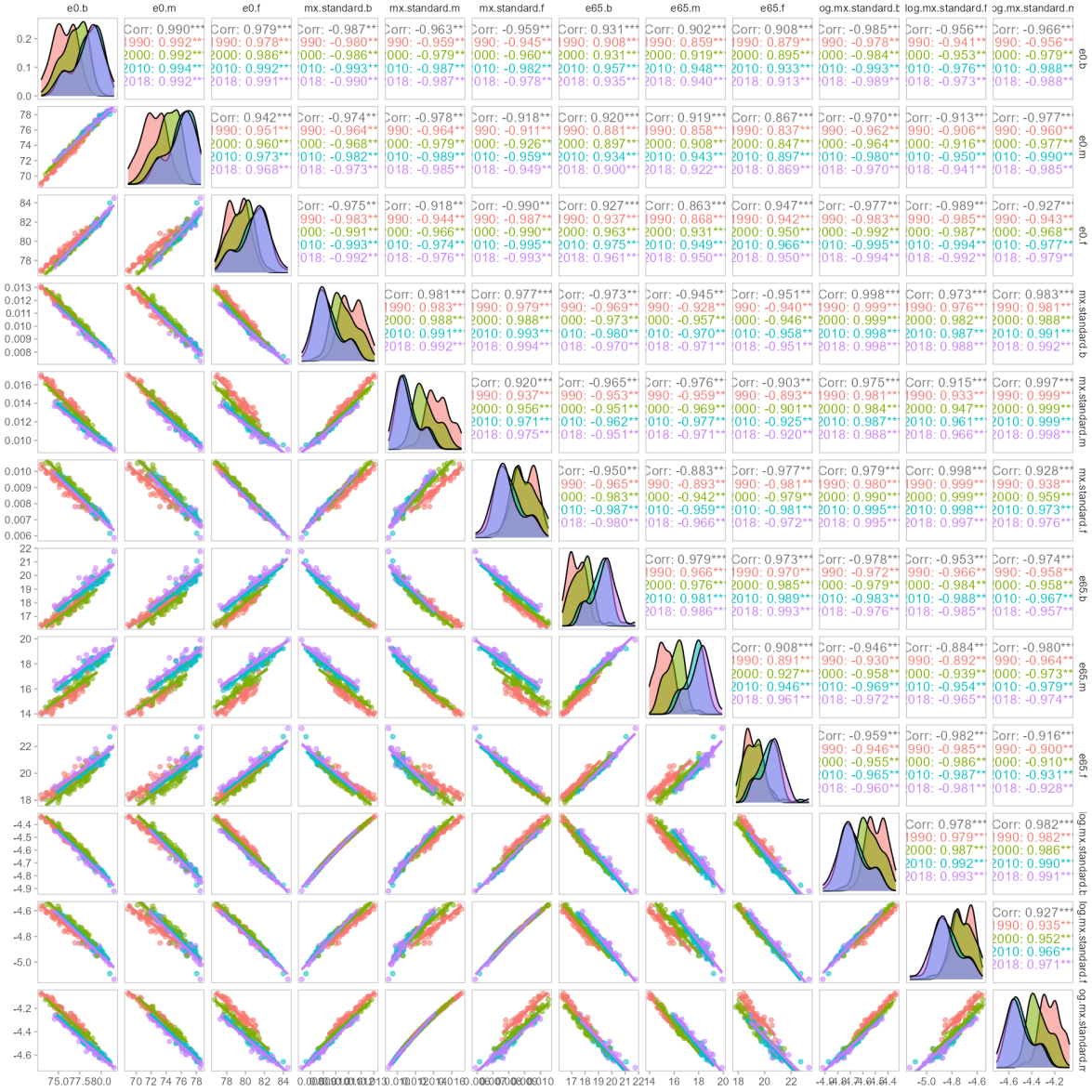


Figure A.1.1. State-level correlation summary and plots for various measures of mortality, by year (1990–2018).

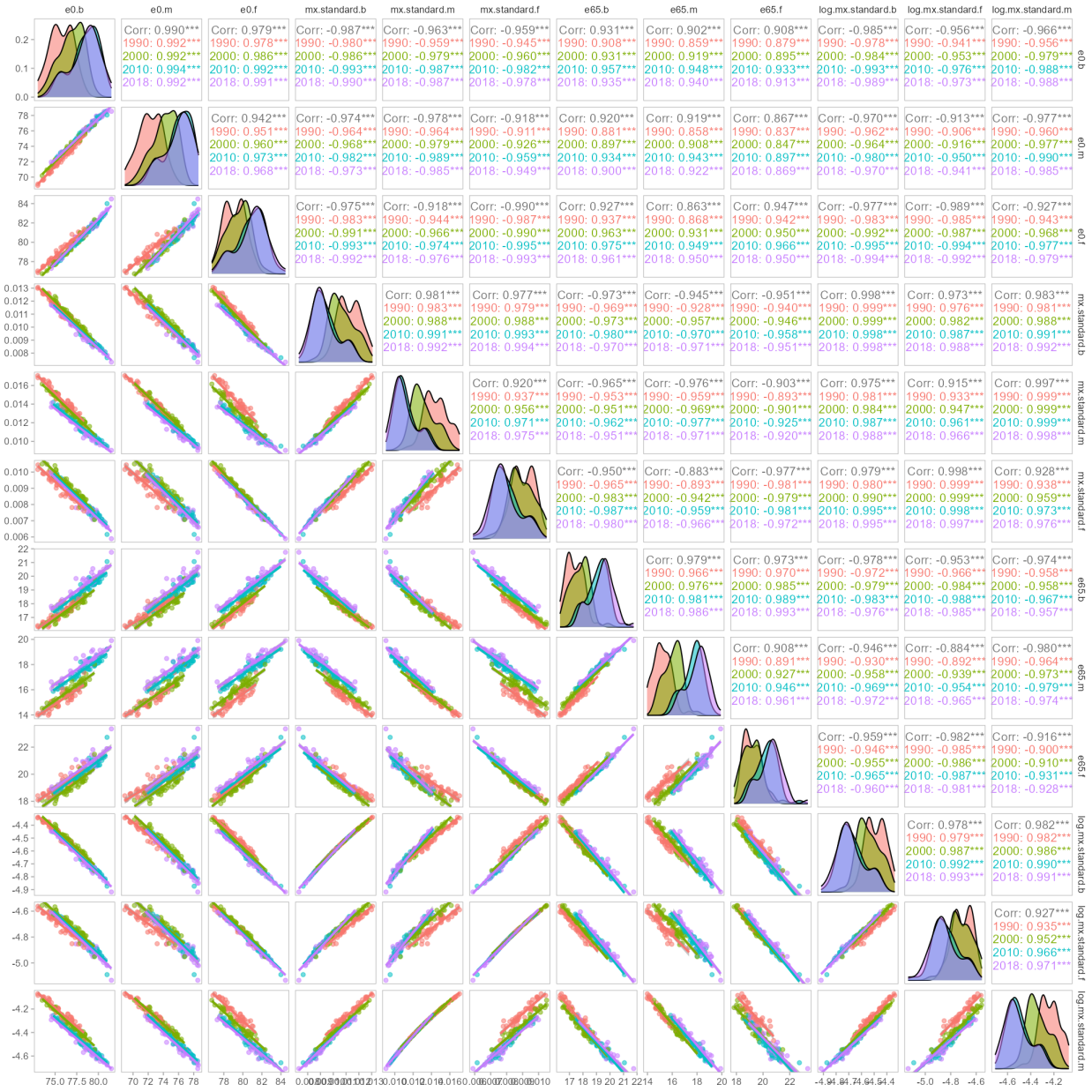


Figure A.1.2. County group-level correlation summary and plots for various measures of mortality, by year (1990–2018).

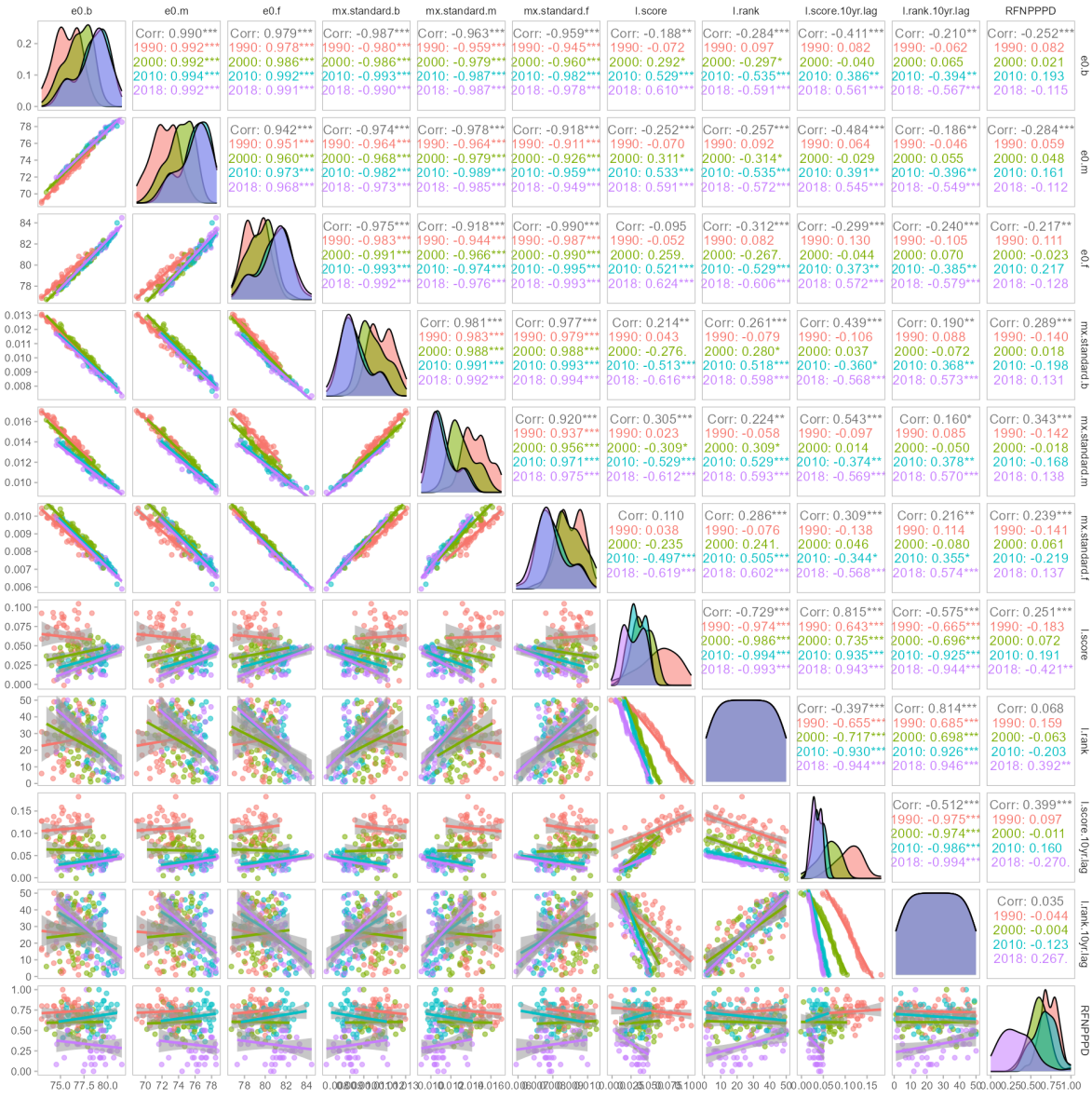


Figure A.1.3. State-level correlation summary and plots for select measures of mortality and indicators of state health policy, by year (1990–2018).

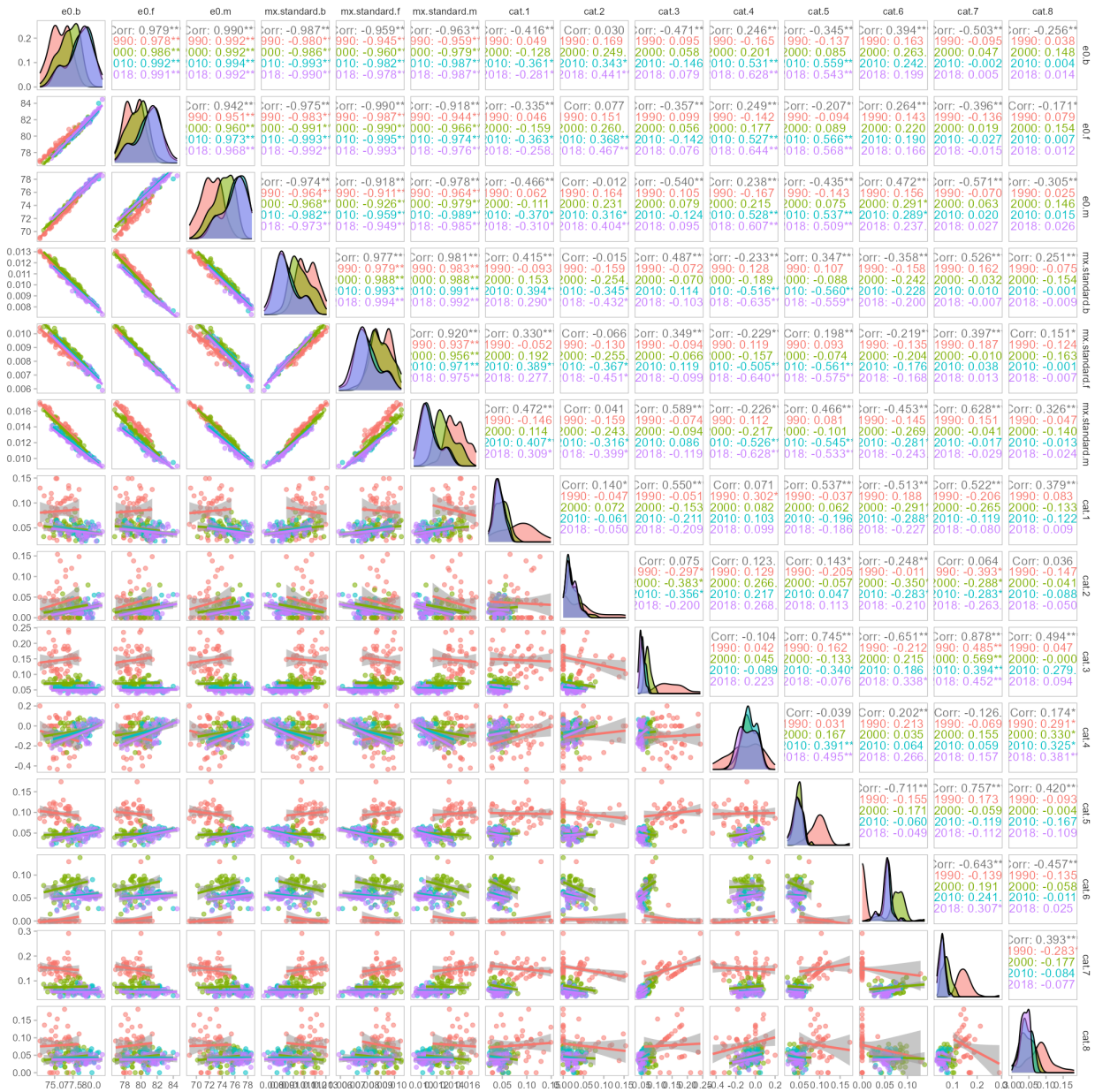


Figure A.1.4. State-level correlation summary and plots for select measures of mortality and thematic components of the state health policy score (I_g), by year (1990–2018).

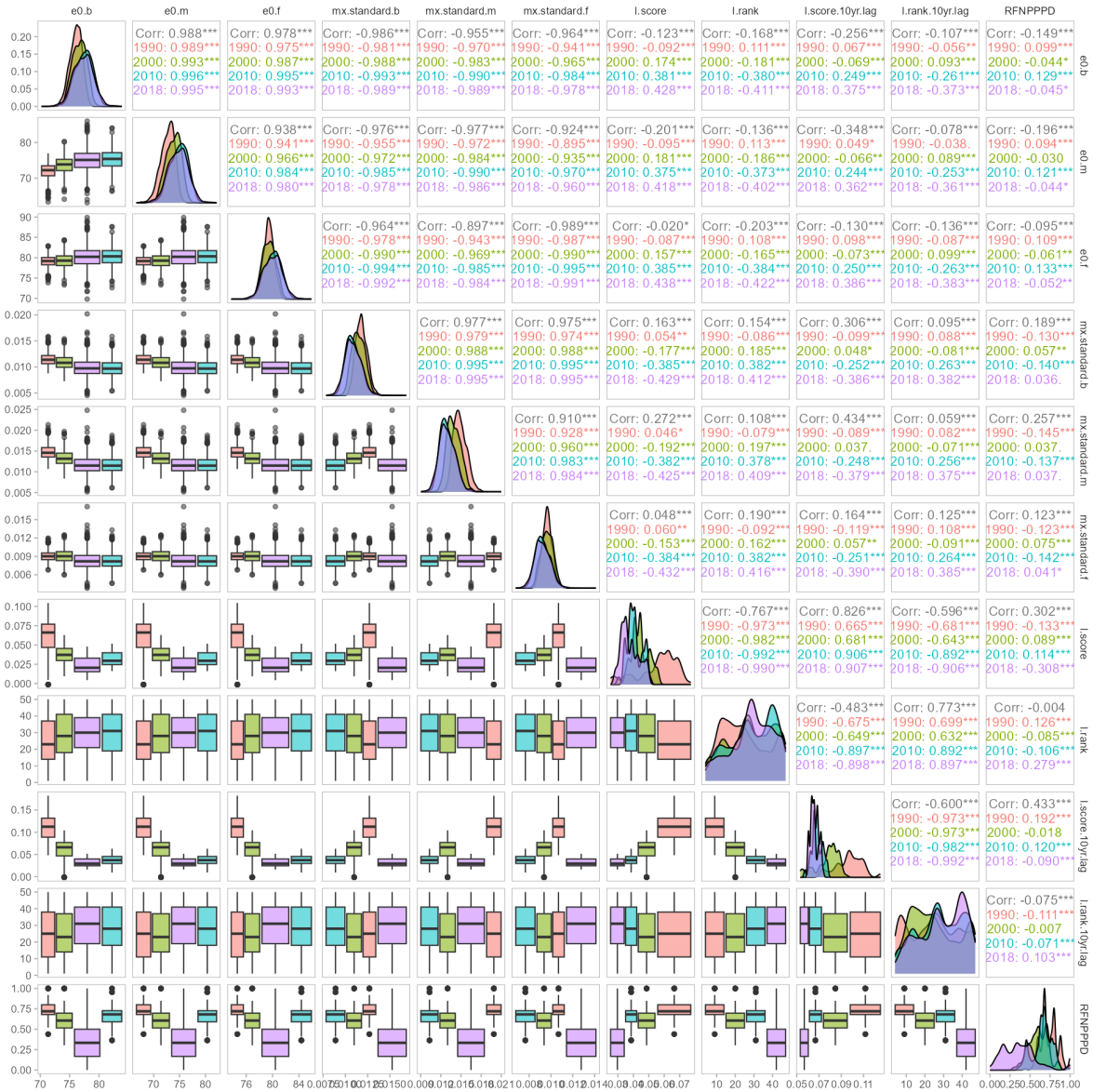


Figure A.1.5. County group-level correlation summary and plots for select measures of mortality and indicators of state health policy, by year (1990–2018).

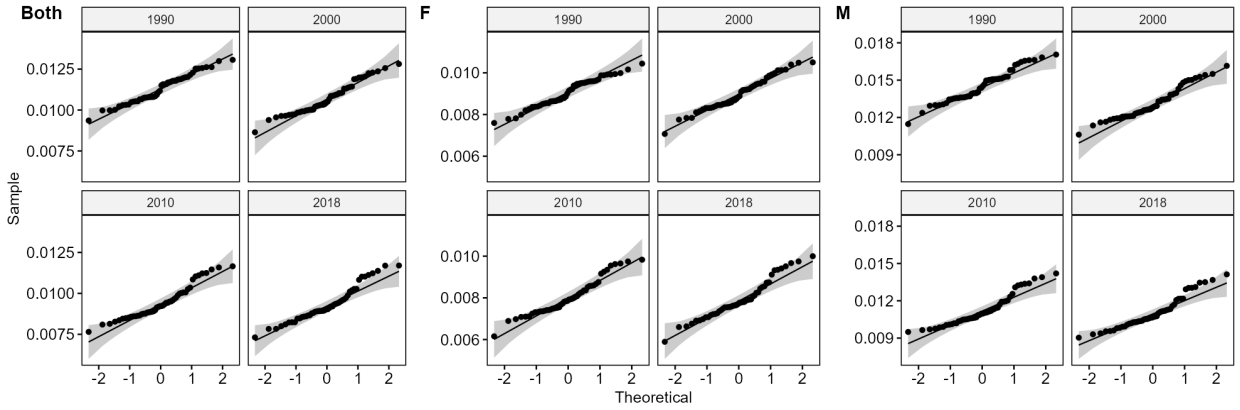


Figure A.1.6. State-level Q-Q plots for log-transformed age-standardized mortality rate, by year and sex.

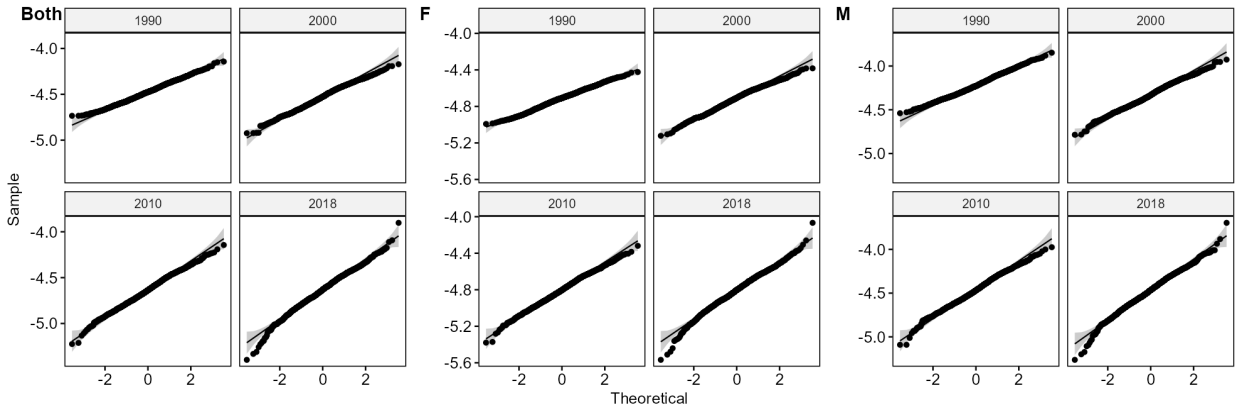


Figure A.1.7. County group level Q-Q plots for log-transformed age-standardized mortality rate, by year and sex.

A.2 Appendix to Chapter 4

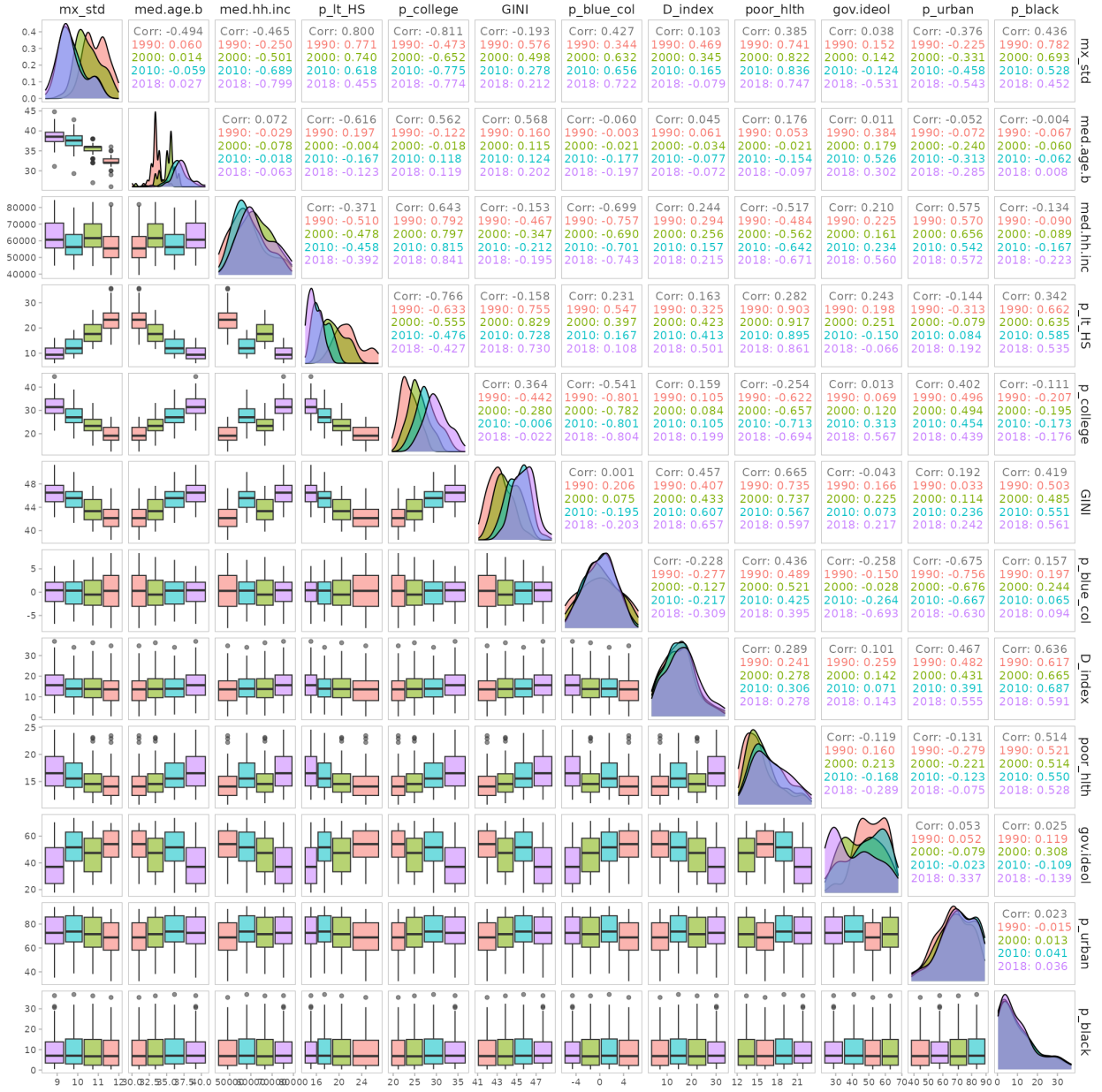


Figure A.2.8. Pearson correlation matrix and association box plots for select indicators in state data set, by year.

Note: for a succinct presentation and for illustrative purposes only both-sex indicators are shown (where applicable); age-standardized mortality rate is included in the matrix as the only mortality indicator to avoid redundancy, as previous analyses have shown high correlation between all mortality measures.

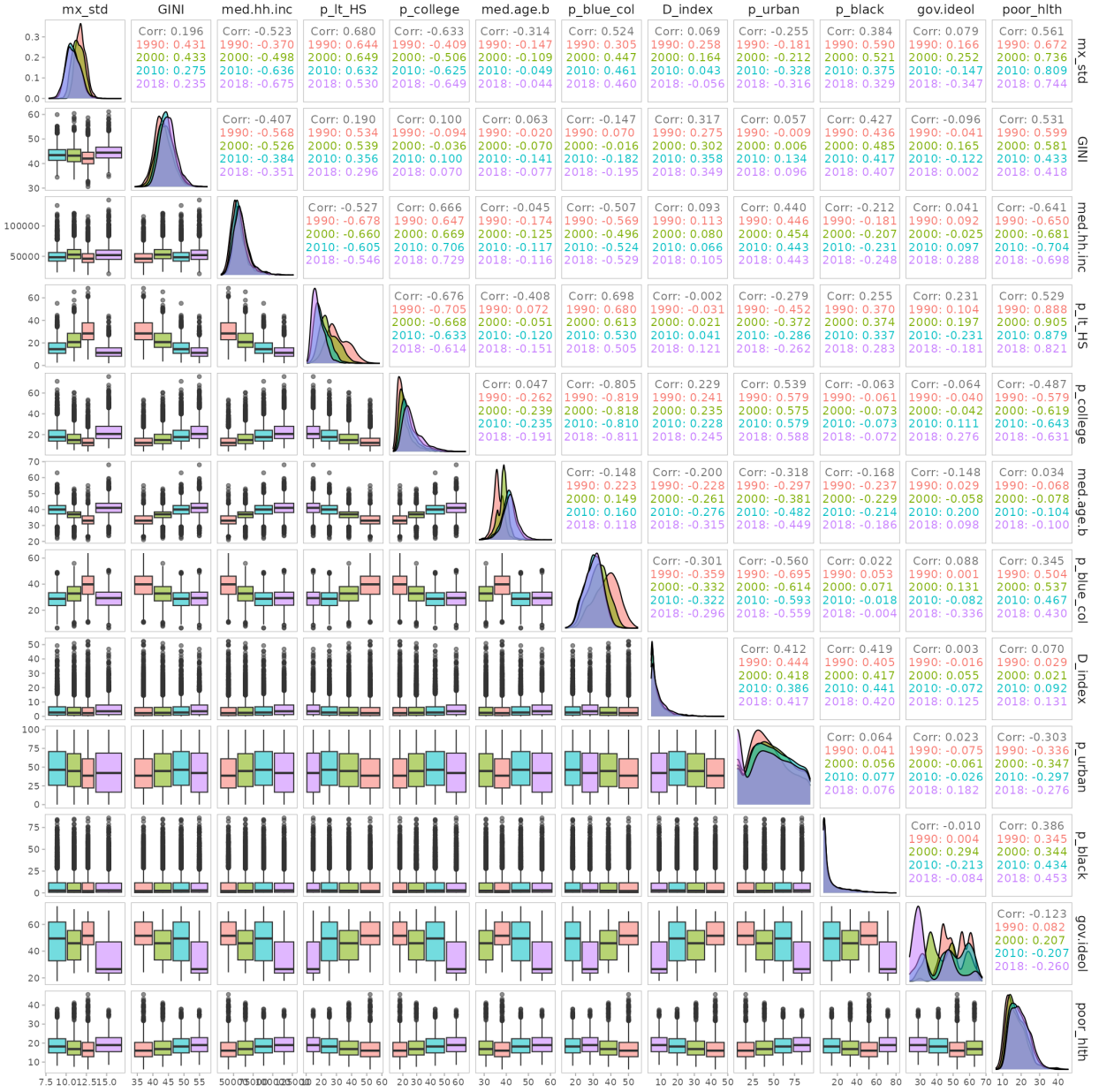


Figure A.2.9. Pearson correlation matrix and association box plots for select indicators in county group data set, by year.

Note: for a succinct presentation and for illustrative purposes only both-sex indicators are shown (where applicable); age-standardized mortality rate is included in the matrix as the only mortality indicator to avoid redundancy, as previous analyses have shown high correlation between all mortality measures.

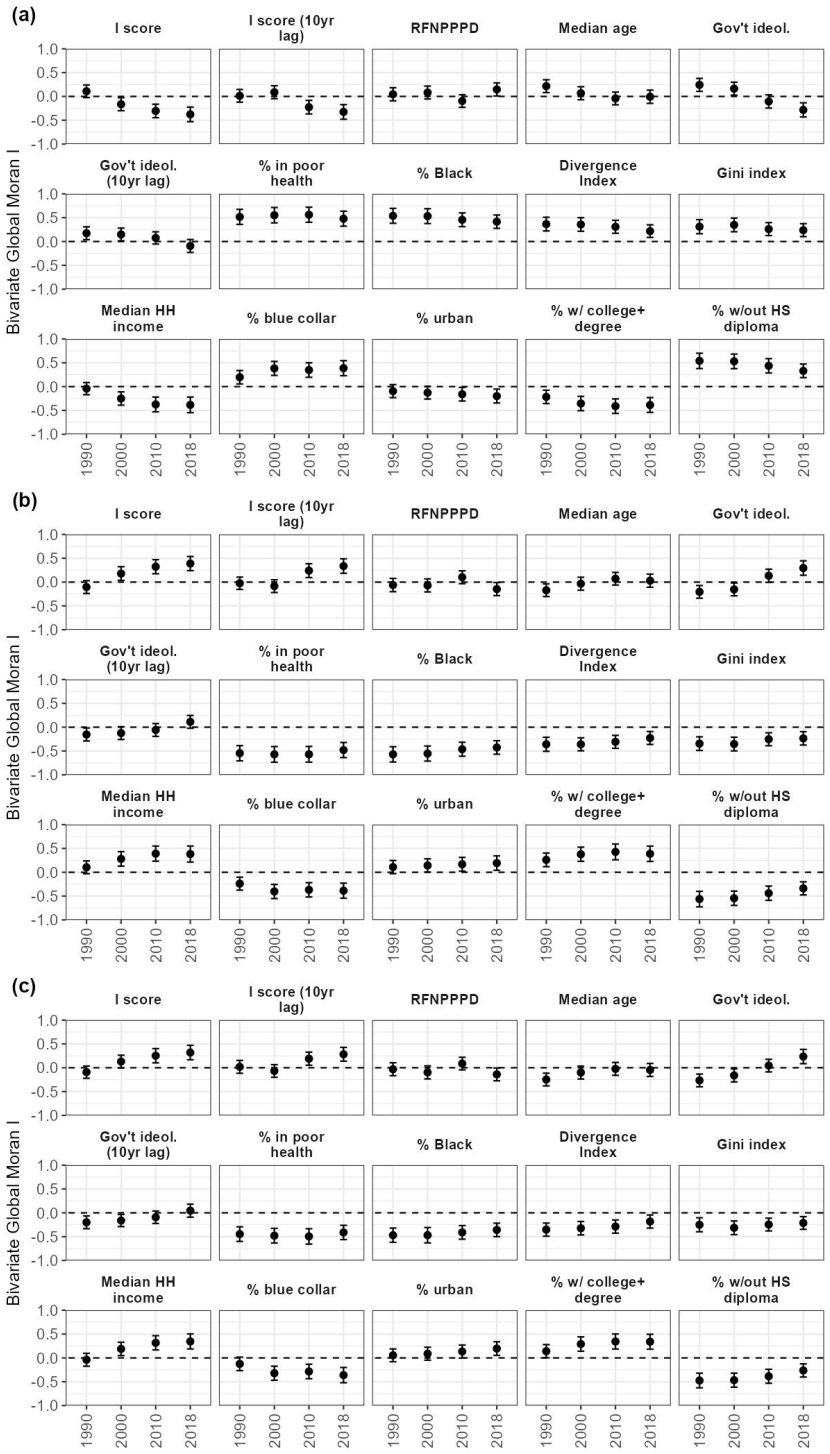


Figure A.2.10. State bivariate Moran statistic for pairwise combinations of 3 mortality indicators and various independent variables, by year (females).

Note: (a) age-standardized mortality rate, (b) life expectancy at birth, (c) life expectancy at age 65

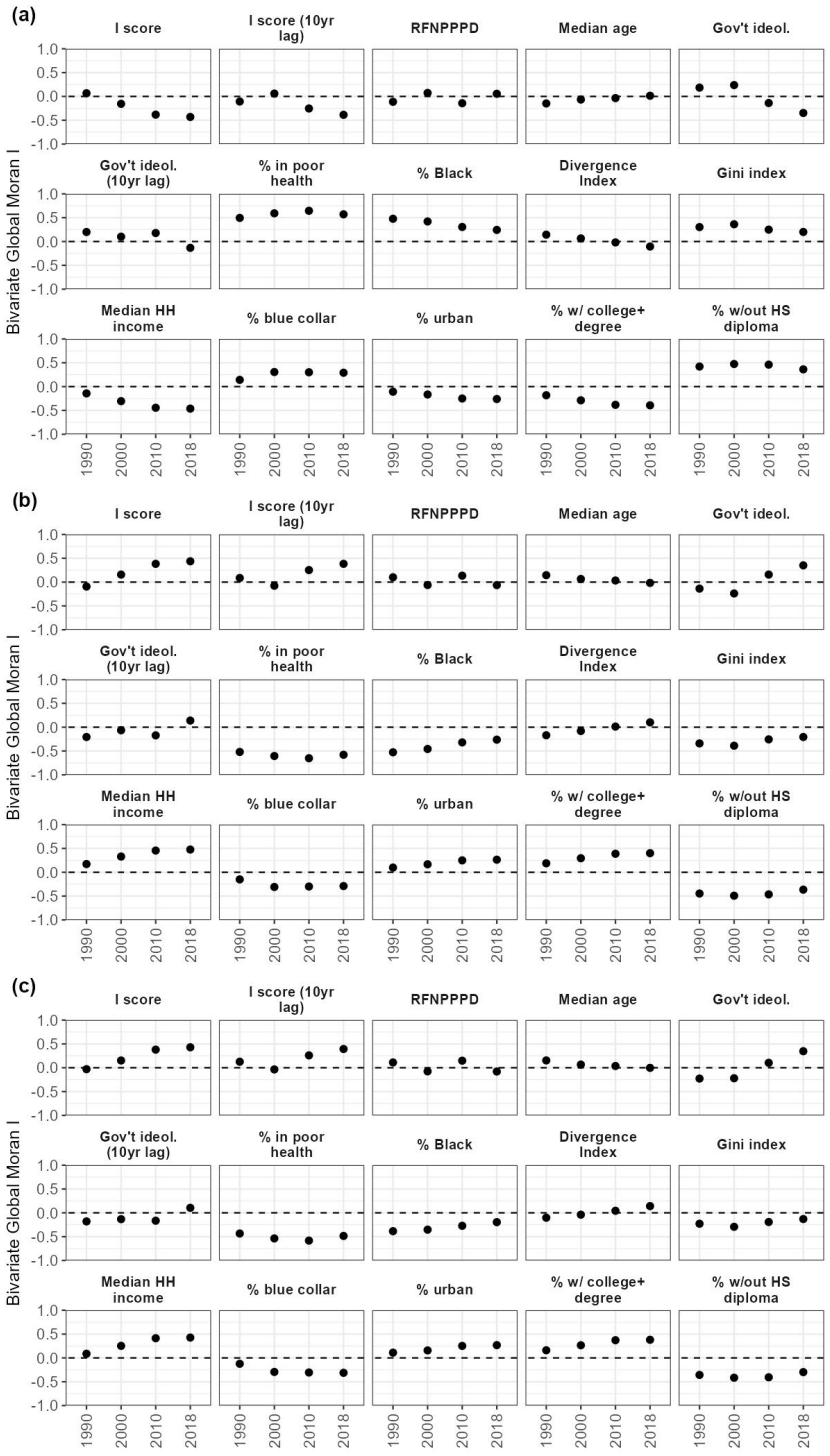


Figure A.2.11. County group bivariate Moran statistic for pairwise combinations of 3 mortality indicators and various independent variables, by year (females).

Note: (a) age-standardized mortality rate, (b) life expectancy at birth, (c) life expectancy at age 65

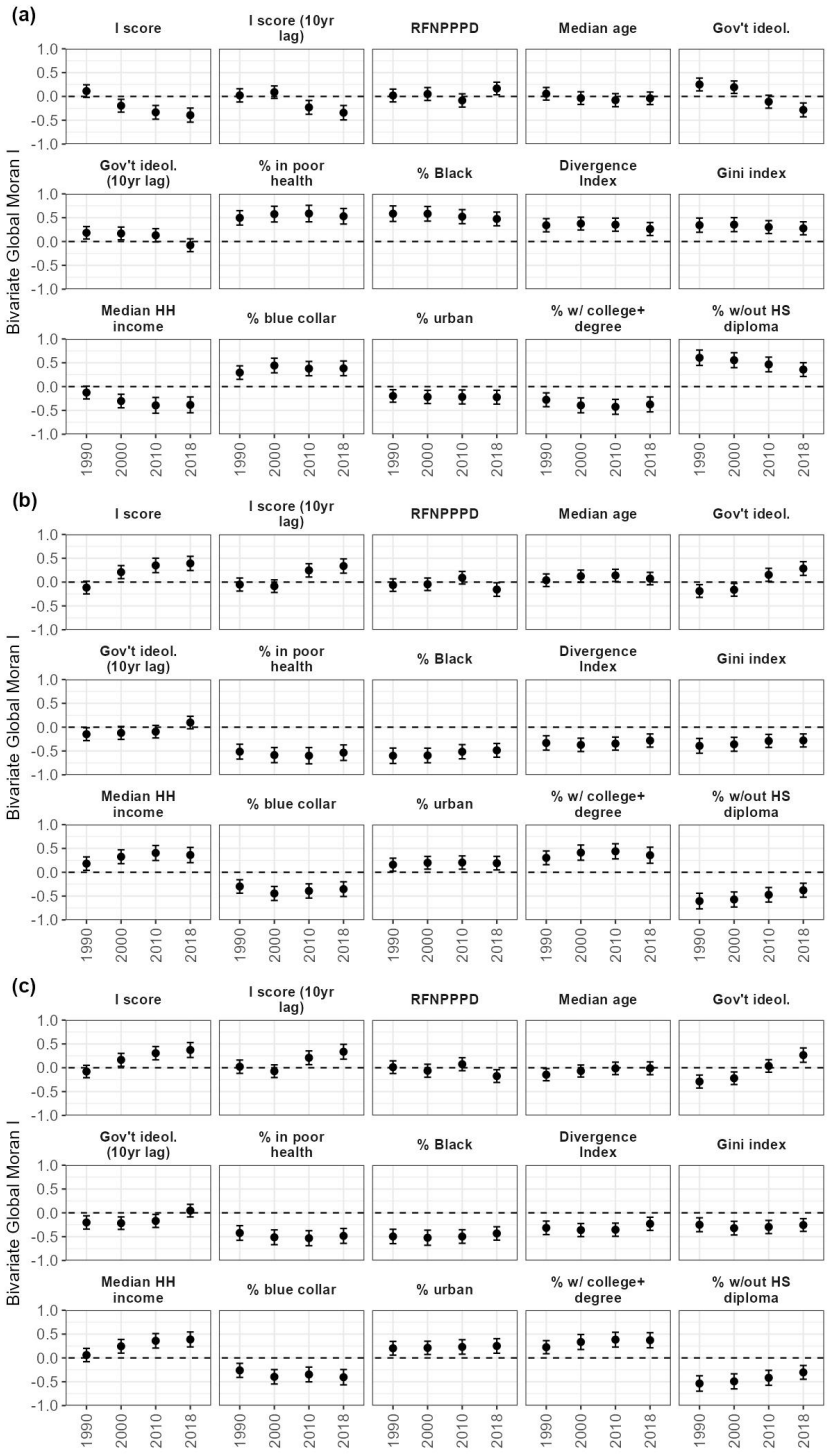


Figure A.2.12. State bivariate Moran statistic for pairwise combinations of 3 mortality indicators and various independent variables, by year (males).

Note: (a) age-standardized mortality rate, (b) life expectancy at birth, (c) life expectancy at age 65

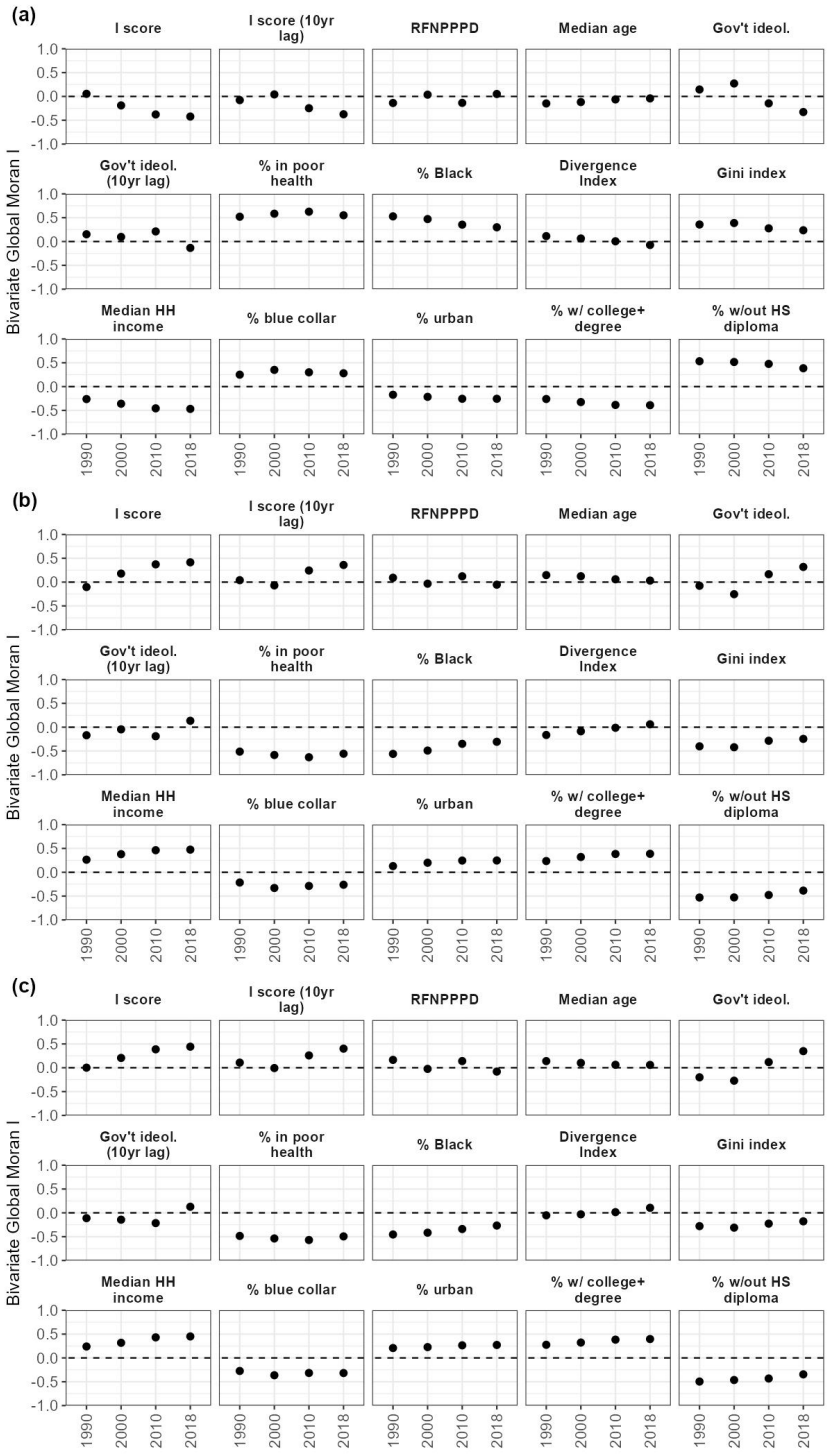


Figure A.2.13. County group bivariate Moran statistic for pairwise combinations of 3 mortality indicators and various independent variables, by year (males).

Note: (a) age-standardized mortality rate, (b) life expectancy at birth, (c) life expectancy at age 65

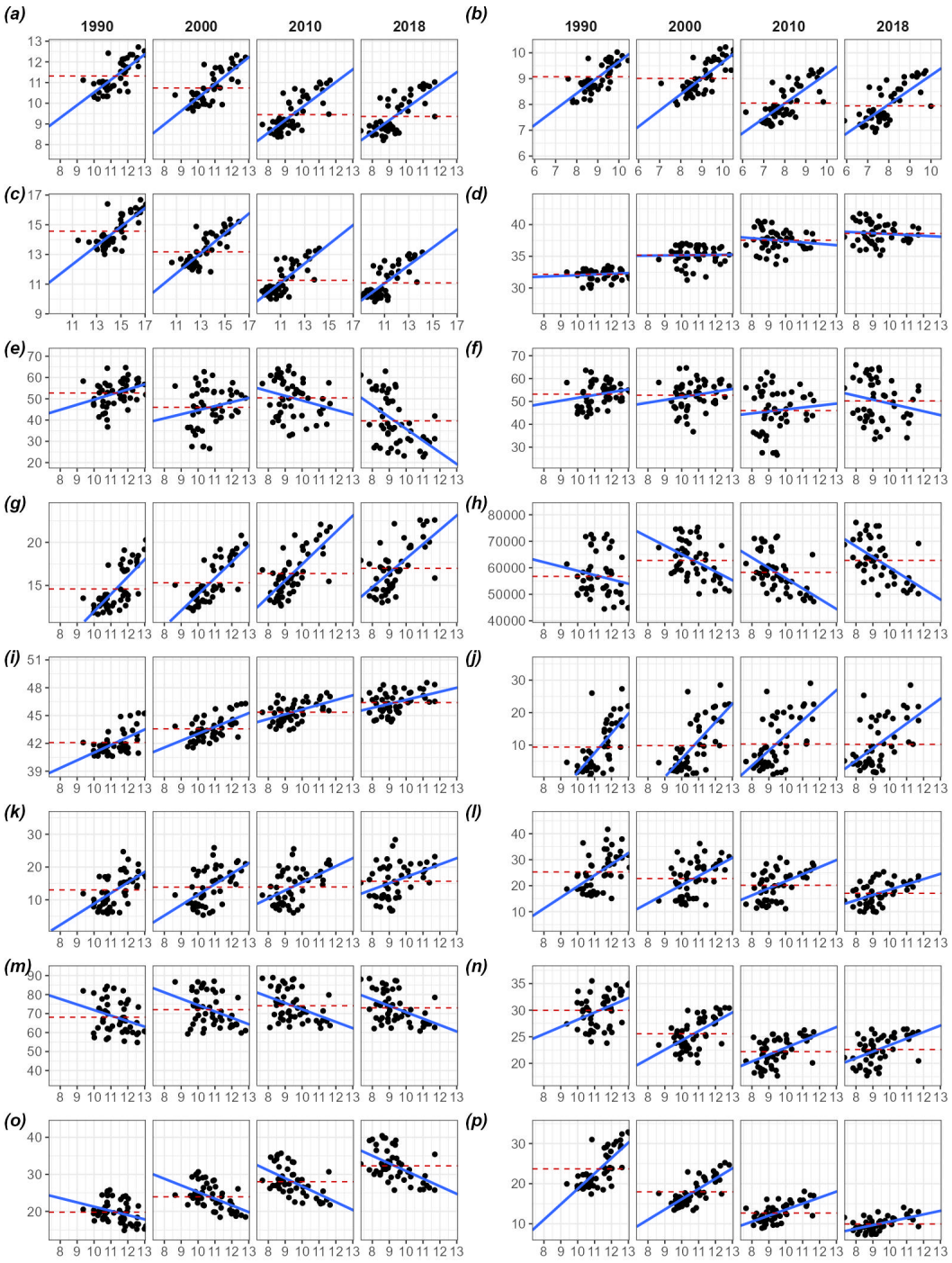


Figure A.2.14. State bivariate Moran plots for pairwise combinations of both-sex age-standardized mortality rate (ASDR) and various independent variables, by year.

Secondary variables: (a) both-sex ASDR (univariate Moran), (b) female ASDR, (c) male ASDR, (d) both-sex median age, (e) government ideology, (f) government ideology 10 years prior, (g) poor health prevalence, (h) median household income, (i) Gini index, (j) % Black population, (k) Divergence D index, (l) Information Theory H index, (m) % urban population, (n) % blue collar occupations, (o) % w/ college education or above, (p) % w/out a HS diploma.

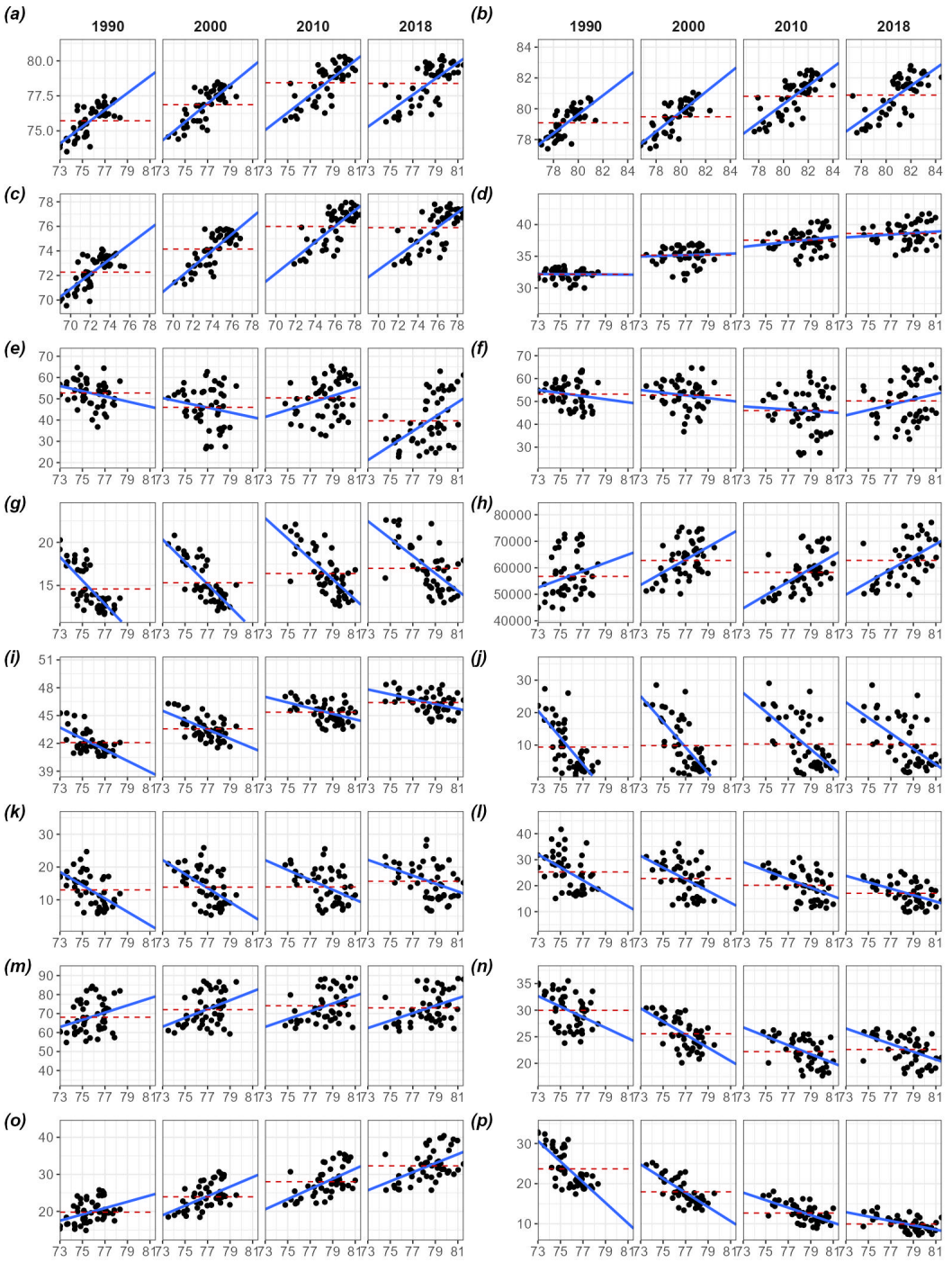


Figure A.2.15. State bivariate Moran plots for pairwise combinations of both-sex life expectancy at birth ($e(0)$) and various independent variables, by year.

Secondary variables: (a) both-sex $e(0)$ (univariate Moran), (b) female $e(0)$, (c) male $e(0)$, (d) both-sex median age, (e) government ideology, (f) government ideology 10 years prior, (g) poor health prevalence, (h) median household income, (i) Gini index, (j) % Black population, (k) Divergence D index, (l) Information Theory H index, (m) % urban population, (n) % blue collar occupations, (o) % w/ college education or above, (p) % w/out a HS diploma.

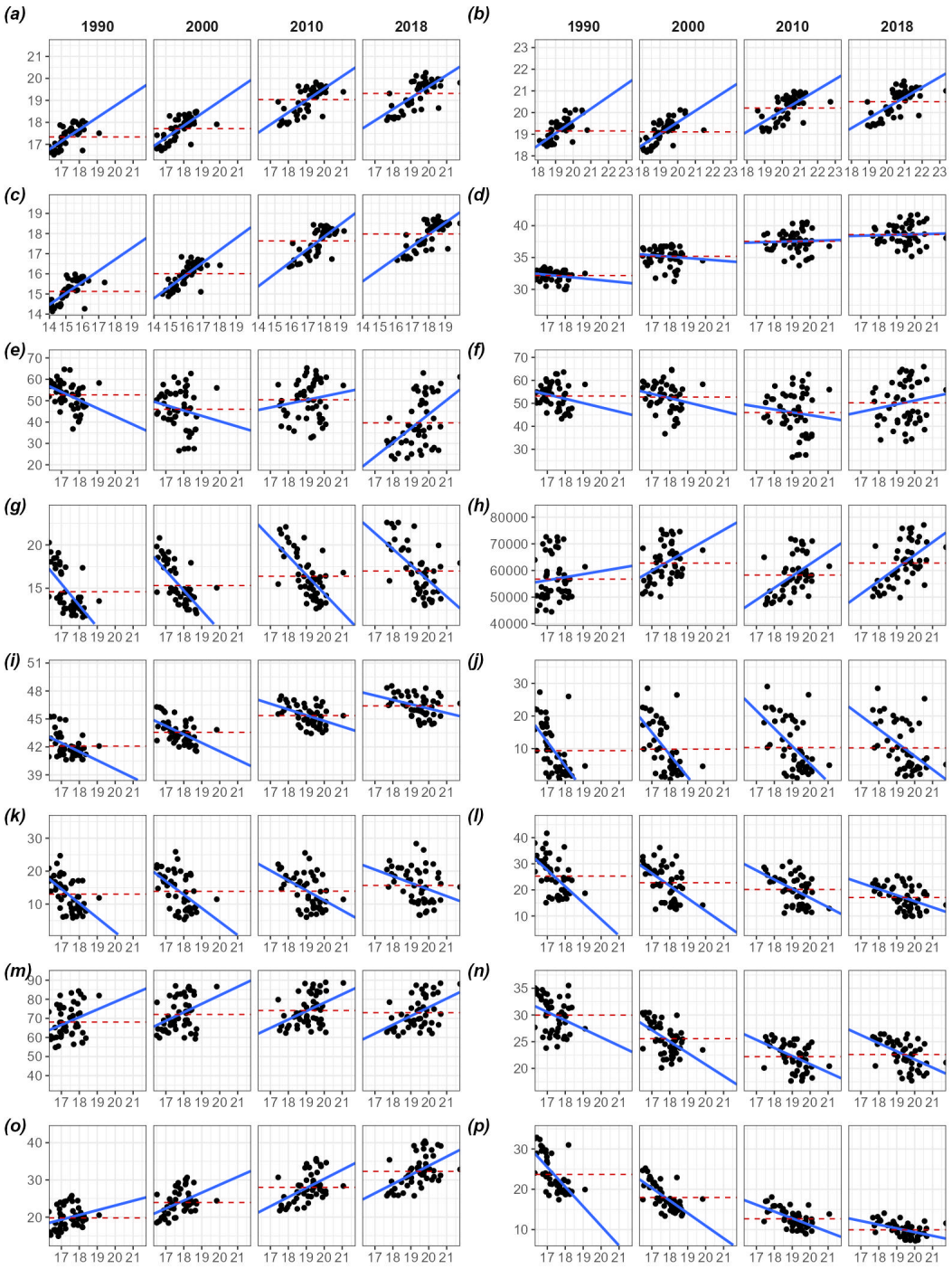


Figure A.2.16. State bivariate Moran plots for pairwise combinations of both-sex life expectancy at age ($e(65)$) and various independent variables, by year.

Secondary variables: (a) both-sex $e(65)$ (univariate Moran), (b) female $e(65)$, (c) male $e(65)$, (d) both-sex median age, (e) government ideology, (f) government ideology 10 years prior, (g) poor health prevalence, (h) median household income, (i) Gini index, (j) % Black population, (k) Divergence D index, (l) Information Theory H index, (m) % urban population, (n) % blue collar occupations, (o) % w/ college education or above, (p) % w/out a HS diploma.

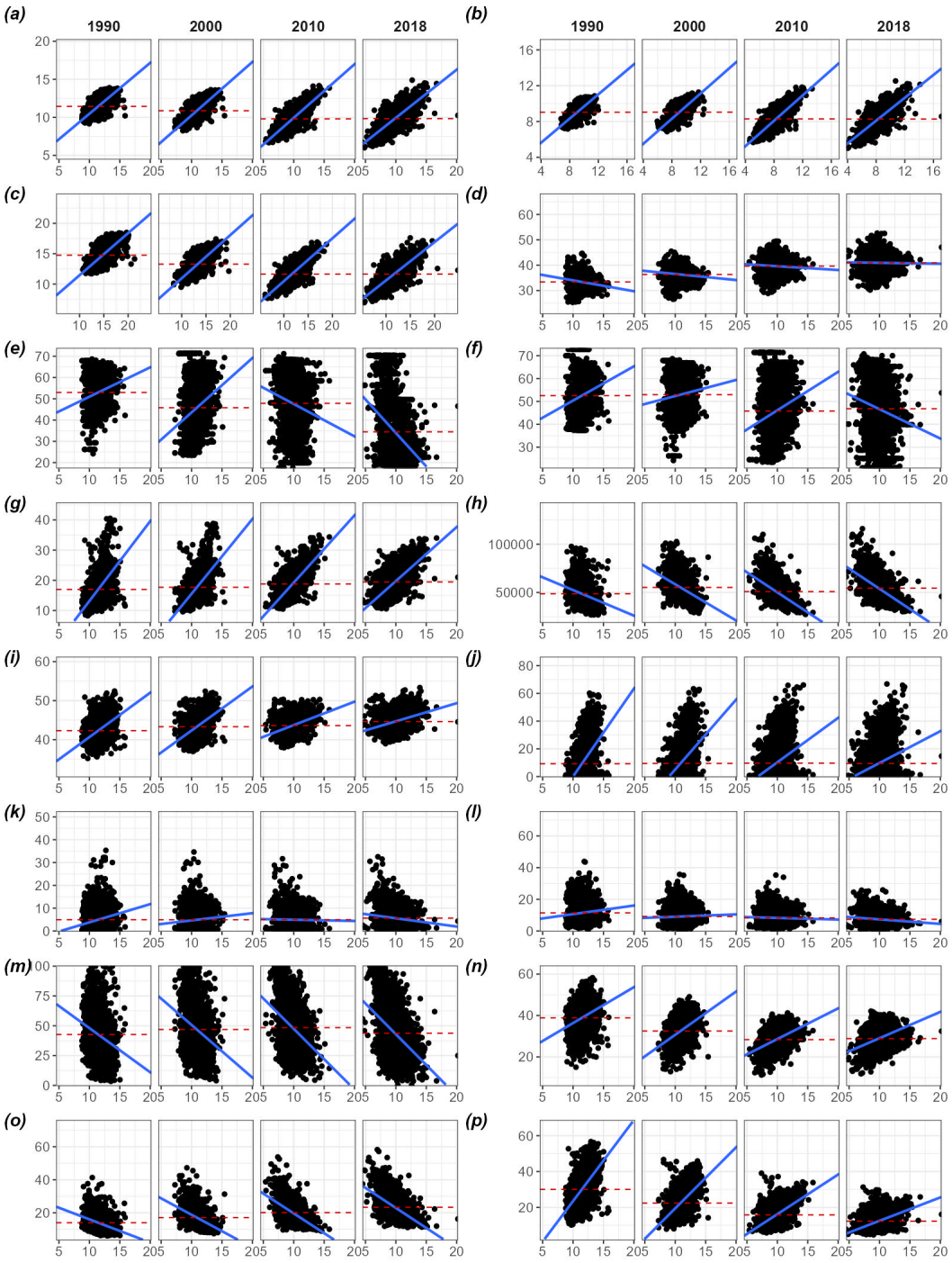


Figure A.2.17. County group bivariate Moran plots for pairwise combinations of both-sex age-standardized mortality rate (ASDR) and various independent variables, by year.

Secondary variables: (a) both-sex ASDR (univariate Moran), (b) female ASDR, (c) male ASDR, (d) both-sex median age, (e) government ideology, (f) government ideology 10 years prior, (g) poor health prevalence, (h) median household income, (i) Gini index, (j) % Black population, (k) Divergence D index, (l) Information Theory H index, (m) % urban population, (n) % blue collar occupations, (o) % w/ college education or above, (p) % w/out a HS diploma.

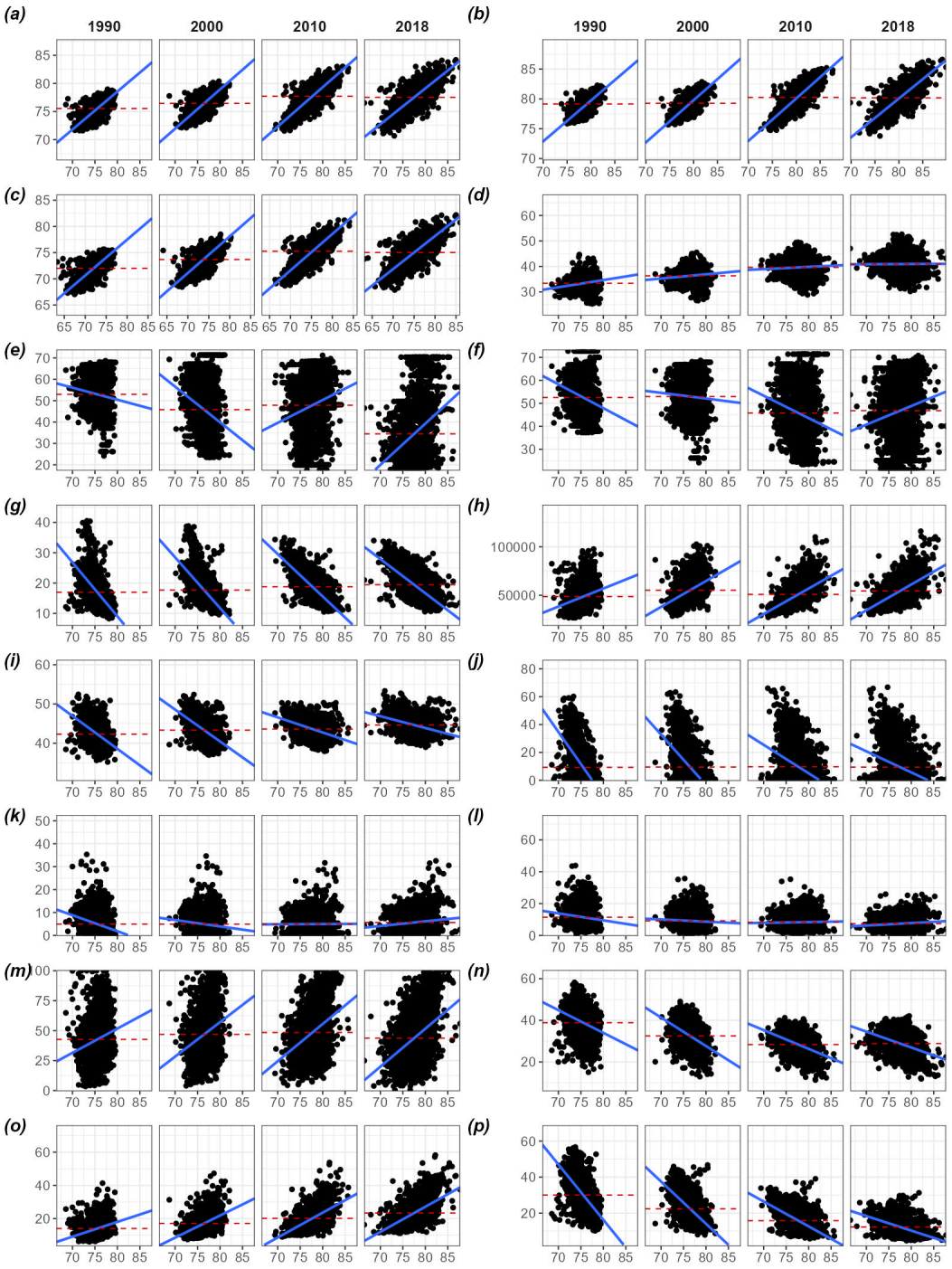


Figure A.2.18. County group bivariate Moran plots for pairwise combinations of both-sex life expectancy at birth ($e(0)$) and various independent variables, by year.

Secondary variables: (a) both-sex $e(0)$ (univariate Moran), (b) female $e(0)$, (c) male $e(0)$, (d) both-sex median age, (e) government ideology, (f) government ideology 10 years prior, (g) poor health prevalence, (h) median household income, (i) Gini index, (j) % Black population, (k) Divergence D index, (l) Information Theory H index, (m) % urban population, (n) % blue collar occupations, (o) % w/ college education or above, (p) % w/out a HS diploma.

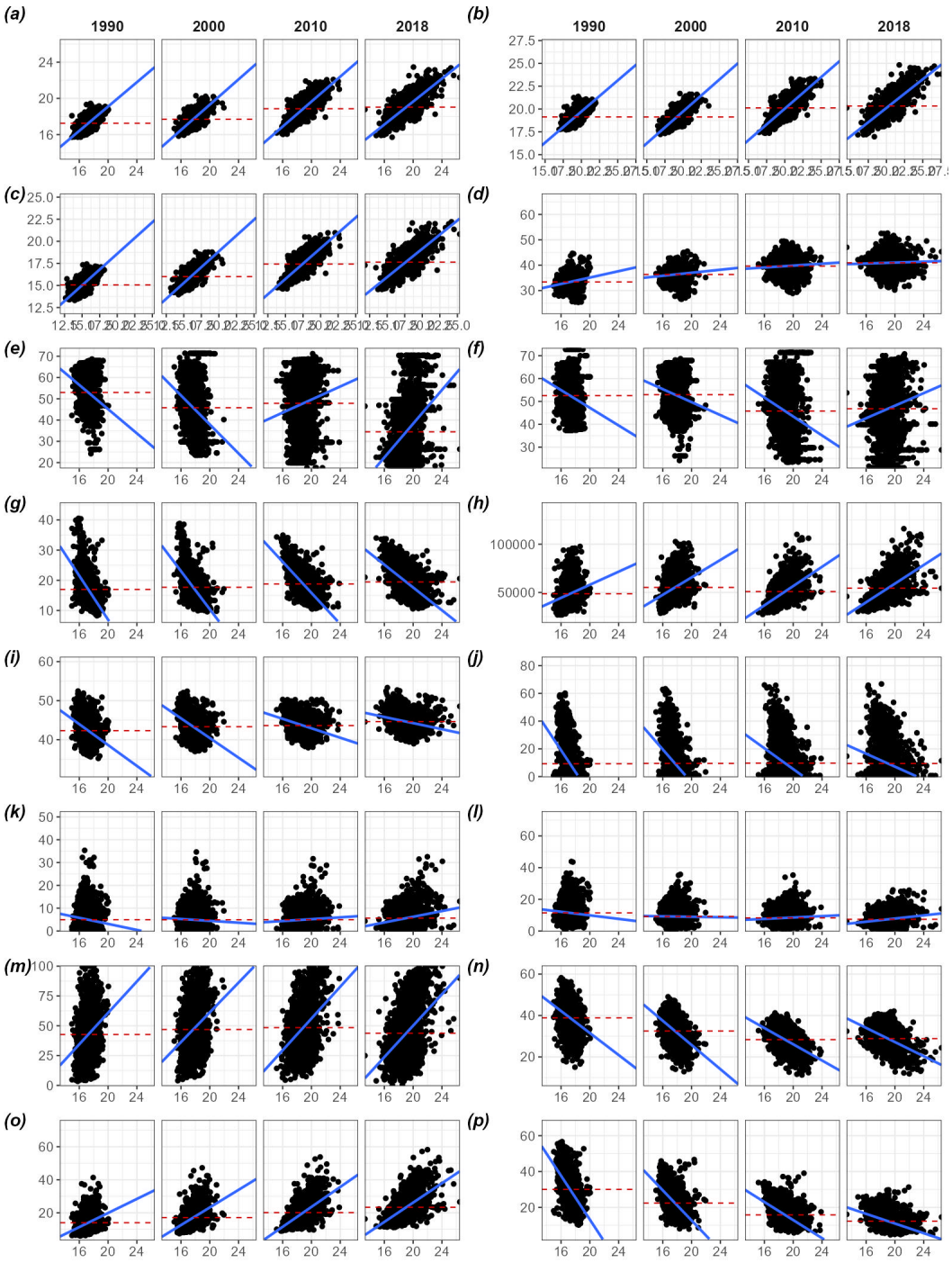


Figure A.2.19. County group bivariate Moran plots for pairwise combinations of both-sex life expectancy at age 65 ($e(65)$) and various independent variables, by year.

Secondary variables: (a) both-sex $e(65)$ (univariate Moran), (b) female $e(65)$, (c) male $e(65)$, (d) both-sex median age, (e) government ideology, (f) government ideology 10 years prior, (g) poor health prevalence, (h) median household income, (i) Gini index, (j) % Black population, (k) Divergence D index, (l) Information Theory H index, (m) % urban population, (n) % blue collar occupations, (o) % w/ college education or above, (p) % w/out a HS diploma.

Table A.2.2. Fully specified state-level fixed effects models estimating female mortality indicators ($log(ASDR)$, $e(0)$, and $e(65)$) featuring various specifications of state health policy.

	$log(ASDR)$			$e(0)$			$e(65)$		
Variable	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$
SHP indicator	−0.026***	−0.023***	0.003	0.360***	0.337***	−0.028	0.187***	0.168***	−0.035
	(0.01)	(0.00)	(0.00)	(0.08)	(0.06)	(0.04)	(0.04)	(0.03)	(0.02)
Median age	−0.007*	−0.009	−0.010**	0.085	0.098*	0.115*	0.069**	0.081**	0.085**
	(0.00)	(0.00)	(0.00)	(0.05)	(0.05)	(0.05)	(0.03)	(0.03)	(0.03)
log(gov't ideology)	−0.023*	0.006	−0.037***	0.246	−0.103	0.441**	0.215**	−0.041	0.316***
	(0.01)	(0.01)	(0.01)	(0.15)	(0.16)	(0.15)	(0.08)	(0.09)	(0.08)
log(% in poor health)	0.051	0.014	0.072	−0.258	0.320	−0.566	−0.619	−0.362	−0.753
	(0.06)	(0.06)	(0.06)	(0.83)	(0.84)	(0.89)	(0.46)	(0.48)	(0.49)
log(% Black)	0.033*	0.032*	0.020	−0.426*	−0.424*	−0.261	−0.337**	−0.331**	−0.239*
	(0.01)	(0.01)	(0.01)	(0.19)	(0.19)	(0.20)	(0.11)	(0.11)	(0.11)
D index	−0.005**	−0.005**	−0.004	0.069**	0.070**	0.055*	0.025	0.027	0.016
	(0.00)	(0.00)	(0.00)	(0.02)	(0.02)	(0.03)	(0.01)	(0.01)	(0.01)
log(med. HH income)	0.138*	0.132*	0.167*	−1.815	−1.775	−2.178*	−1.383**	−1.315*	−1.620**
	(0.07)	(0.07)	(0.07)	(0.94)	(0.92)	(1.01)	(0.52)	(0.53)	(0.55)
% blue collar	−0.005*	−0.007**	−0.008**	0.064	0.086*	0.099**	0.047*	0.062**	0.067**
	(0.00)	(0.00)	(0.00)	(0.03)	(0.03)	(0.04)	(0.02)	(0.02)	(0.02)
% urban population	−0.002	−0.002	−0.003*	0.034	0.036*	0.053**	0.016	0.018	0.027*
	(0.00)	(0.00)	(0.00)	(0.02)	(0.02)	(0.02)	(0.01)	(0.01)	(0.01)
log(% w/out HS diploma)	−0.085*	−0.061	−0.095*	1.322*	0.935	1.471*	0.621	0.470	0.693
	(0.04)	(0.04)	(0.05)	(0.61)	(0.61)	(0.65)	(0.34)	(0.35)	(0.36)
Joint Wald test	105***	100***	74***	92***	99***	64***	116***	104***	91***
df	10	10	10	10	10	10	10	10	10
Wooldridge test	82.85***	78.36***	94.6***	94.16***	85.16***	122***	55.06***	46.36***	59.78***
Hausman test	18***	16***	19***	18***	15***	19***	15***	15***	18***
LM Breusch-Pagan test	16	66***	32***	94***	105***	191***	24**	2	69***
Individual eff. F test	9.12***	9.27***	8.3***	8.28***	8.85***	7.47***	10.21***	10.23***	9.49***
Time eff. F test	36.09***	35.9***	35.34***	34.36***	33.8***	33.61***	37.15***	37.21***	36.77***
Two-way eff. F test	12.32***	12***	10.88***	9.76***	10.03***	8.49***	17.7***	16.65***	16.37***
R^2 (overall)	0.433	0.423	0.35	0.402	0.42	0.317	0.458	0.433	0.4
Adj. R^2 (within)	0.176	0.162	0.056	0.131	0.158	0.008	0.213	0.176	0.129
AIC	−783	−780	−756	280	274	306	44	54	65
BIC	−575	−572	−548	488	481	514	252	261	273

$^{*}p < 0.05$, $^{**}p < 0.01$, $^{***}p < 0.001$.

Table A.2.3. Fully specified state-level fixed effects models estimating male mortality indicators ($log(ASDR)$, $e(0)$, and $e(65)$) featuring various specifications of state health policy.

	$log(ASDR)$			$e(0)$			$e(65)$		
Variable	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$
SHP indicator	−0.022***	−0.018***	0.000	0.336***	0.318***	0.019	0.155***	0.126***	−0.011
	(0.01)	(0.00)	(0.00)	(0.09)	(0.06)	(0.04)	(0.04)	(0.03)	(0.02)
Median age	−0.004	−0.004	−0.003	0.020	0.010	0.008	0.048	0.050	0.043
	(0.00)	(0.00)	(0.00)	(0.07)	(0.07)	(0.07)	(0.03)	(0.03)	(0.03)
log(gov't ideology)	−0.016	0.010	−0.027**	0.154	−0.143	0.328*	0.150*	−0.040	0.231
	(0.01)	(0.01)	(0.01)	(0.16)	(0.18)	(0.16)	(0.07)	(0.08)	(0.07)
log(% in poor health)	−0.004	0.014	0.051	0.725	0.617	−0.154	−0.270	−0.422	−0.634
	(0.05)	(0.04)	(0.05)	(0.80)	(0.77)	(0.81)	(0.33)	(0.33)	(0.34)
log(% Black)	0.010	0.008	0.000	−0.242	−0.219	−0.095	−0.065	−0.046	0.014
	(0.01)	(0.01)	(0.01)	(0.21)	(0.20)	(0.22)	(0.09)	(0.09)	(0.09)
D index	−0.004**	−0.004**	−0.004*	0.067*	0.065*	0.059*	0.020	0.020	0.015
	(0.00)	(0.00)	(0.00)	(0.03)	(0.03)	(0.03)	(0.01)	(0.01)	(0.01)
log(med. HH income)	0.127	0.130*	0.171*	−1.808	−1.906	−2.465*	−1.098*	−1.105*	−1.430**
	(0.06)	(0.06)	(0.07)	(1.15)	(1.10)	(1.19)	(0.48)	(0.48)	(0.50)
% blue collar	−0.002	−0.004	−0.004	0.017	0.034	0.048	0.022	0.034*	0.037*
	(0.00)	(0.00)	(0.00)	(0.04)	(0.04)	(0.04)	(0.02)	(0.02)	(0.02)
% urban population	−0.002*	−0.002*	−0.003**	0.038	0.040*	0.050*	0.018*	0.020*	0.025**
	(0.00)	(0.00)	(0.00)	(0.02)	(0.02)	(0.02)	(0.01)	(0.01)	(0.01)
log(% w/out HS diploma)	−0.065	−0.059	−0.077	1.848**	1.616*	2.026**	0.245	0.219	0.322
	(0.04)	(0.04)	(0.04)	(0.65)	(0.64)	(0.68)	(0.27)	(0.28)	(0.29)
Joint Wald test	74***	72***	49***	68***	80***	50***	76***	66***	53***
df	10	10	10	10	10	10	10	10	10
Wooldridge test	78.66***	75.98***	132.22***	117.89***	124.81***	202.57***	54.71***	51.42***	70.83***
Hausman test	16***	18***	19***	21***	18***	22***	13***	17***	16***
LM Breusch-Pagan test	124***	86***	20*	109***	113***	141***	73***	47***	59***
Individual eff. F test	7.7***	7.49***	7.28***	6.81***	7.03***	6.31***	9.28***	8.72***	9.17***
Time eff. F test	55.76***	54.06***	54.84***	42.46***	41.34***	43.53***	62.33***	60.6***	59.34***
Two-way eff. F test	14.24***	13.76***	12.64***	12.76***	13.33***	11.27***	20.37***	18.77***	18.93***
R^2 (overall)	0.351	0.345	0.265	0.333	0.367	0.268	0.358	0.327	0.28
Adj. R^2 (within)	0.057	0.049	−0.067	0.031	0.081	−0.064	0.067	0.022	−0.045
AIC	−834	−832	−809	317	307	336	−35	−26	−12
BIC	−626	−625	−602	525	515	544	173	182	195

$^{*}p < 0.05$, $^{**}p < 0.01$, $^{***}p < 0.001$.

Table A.2.4. Fully specified county group-level fixed effects models estimating female mortality indicators ($log(ASDR)$, $e(0)$, and $e(65)$) featuring various specifications of state health policy.

	$log(ASDR)$			$e(0)$			$e(65)$		
Variable	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$
SHP indicator†	−0.019***	−0.015***	0.004***	0.249***	0.208***	−0.042***	0.149***	0.120***	−0.044***
	(0.00)	(0.00)	(0.00)	(0.02)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)
Median age	−0.007***	−0.007***	−0.007***	0.077***	0.078***	0.081***	0.068***	0.070***	0.071***
	(0.00)	(0.00)	(0.00)	(0.01)	(0.01)	(0.01)	(0.00)	(0.00)	(0.00)
log(gov't ideology)†	−0.023***	0.007*	−0.034***	0.273***	−0.109**	0.410***	0.223***	−0.062*	0.306***
	(0.00)	(0.00)	(0.00)	(0.04)	(0.04)	(0.03)	(0.02)	(0.02)	(0.02)
log(% in poor health)	−0.110***	−0.121***	−0.100***	1.577***	1.718***	1.439***	0.877***	0.977***	0.801***
	(0.01)	(0.01)	(0.01)	(0.14)	(0.14)	(0.14)	(0.09)	(0.09)	(0.09)
log(% Black)	0.005***	0.004**	0.004**	−0.084***	−0.060***	−0.062***	−0.051***	−0.037***	−0.038***
	(0.00)	(0.00)	(0.00)	(0.02)	(0.02)	(0.02)	(0.01)	(0.01)	(0.01)
log(D index)	−0.007***	−0.008***	−0.008***	0.120***	0.136***	0.131***	0.039**	0.049***	0.046***
	(0.00)	(0.00)	(0.00)	(0.02)	(0.02)	(0.02)	(0.01)	(0.01)	(0.01)
log(med. HH income)	−0.077***	−0.092***	−0.069***	1.071***	1.262***	0.967***	0.538***	0.676***	0.464***
	(0.01)	(0.01)	(0.01)	(0.14)	(0.14)	(0.15)	(0.09)	(0.09)	(0.09)
log(GINI index)	−0.197***	−0.209***	−0.219***	2.754***	2.870***	3.050***	1.501***	1.617***	1.679***
	(0.02)	(0.02)	(0.02)	(0.24)	(0.25)	(0.25)	(0.15)	(0.15)	(0.15)
% blue collar	−0.002***	−0.002***	−0.002***	0.030***	0.032***	0.032***	0.016***	0.018***	0.017***
	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
% urban population	−0.001***	−0.001***	−0.001***	0.018***	0.018***	0.020***	0.011***	0.011***	0.012***
	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
log(% w/out HS diploma)	−0.025***	−0.018**	−0.020***	0.273***	0.176*	0.212**	0.298***	0.241***	0.253***
	(0.01)	(0.01)	(0.01)	(0.08)	(0.08)	(0.08)	(0.05)	(0.05)	(0.05)
Joint Wald test	1585***	1391***	1361***	1416***	1295***	1201***	1610***	1381***	1442***
df	11	11	11	11	11	11	11	11	11
Wooldridge test	1348.18***	1296.26***	1342.68***	1534.9***	1452.55***	1542.78***	1035.32***	999.63***	1020.58***
Hausman test	705***	705***	695***	744***	728***	736***	675***	679***	664***
LM Breusch-Pagan test	276***	902***	1773***	544***	502***	2144***	1563***	2395***	1064***
Individual eff. F test	6.84***	6.39***	6.94***	6.74***	6.38***	6.78***	6.99***	6.58***	7.18***
Time eff. F test	494.18***	442.72***	505.73***	381.34***	338.8***	389.46***	650.29***	593.77***	669.56***
Two-way eff. F test	7.97***	7.53***	7.95***	7.41***	7.13***	7.34***	9***	8.41***	9.09***
R^2 (overall)	0.176	0.158	0.155	0.161	0.149	0.14	0.179	0.157	0.163
Adj. R^2 (within)	−0.1	−0.124	−0.128	−0.121	−0.137	−0.149	−0.097	−0.126	−0.118
AIC	−27046	−26830	−26797	25254	25390	25498	15410	15664	15596
BIC	−9149	−8933	−8900	43151	43287	43395	33307	33561	33493

$^*p < 0.05$, $^{**}p < 0.01$, $^{***}p < 0.001$.

Table A.2.5. Fully specified county group-level fixed effects models estimating male mortality indicators ($log(ASDR)$, $e(0)$, and $e(65)$) featuring various specifications of state health policy.

	$log(ASDR)$			$e(0)$			$e(65)$		
Variable	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$
SHP indicator†	−0.016***	−0.014***	0.001*	0.226***	0.210***	−0.011	0.136***	0.115***	−0.018***
	(0.00)	(0.00)	(0.00)	(0.02)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)
Median age	−0.005***	−0.005***	−0.005***	0.047***	0.048***	0.051***	0.055***	0.056***	0.057***
	(0.00)	(0.00)	(0.00)	(0.01)	(0.01)	(0.01)	(0.00)	(0.00)	(0.00)
log(gov't ideology)†	−0.012***	0.006*	−0.021***	0.094*	−0.117**	0.215***	0.146***	−0.021	0.220***
	(0.00)	(0.00)	(0.00)	(0.04)	(0.04)	(0.04)	(0.02)	(0.02)	(0.02)
log(% in poor health)	−0.105***	−0.103***	−0.093***	1.876***	1.855***	1.707***	0.755***	0.750***	0.661***
	(0.01)	(0.01)	(0.01)	(0.14)	(0.14)	(0.14)	(0.07)	(0.07)	(0.07)
log(% Black)	0.004***	0.003*	0.003*	−0.109***	−0.086***	−0.089***	−0.020*	−0.007	−0.009
	(0.00)	(0.00)	(0.00)	(0.02)	(0.02)	(0.02)	(0.01)	(0.01)	(0.01)
log(D index)	−0.008***	−0.009***	−0.009***	0.140***	0.155***	0.152***	0.048***	0.057***	0.055***
	(0.00)	(0.00)	(0.00)	(0.02)	(0.02)	(0.02)	(0.01)	(0.01)	(0.01)
log(med. HH income)	−0.097***	−0.107***	−0.090***	1.591***	1.692***	1.489***	0.669***	0.765***	0.602***
	(0.01)	(0.01)	(0.01)	(0.16)	(0.16)	(0.16)	(0.08)	(0.08)	(0.08)
log(GINI index)	−0.185***	−0.189***	−0.205***	2.479***	2.459***	2.756***	1.593***	1.658***	1.760***
	(0.02)	(0.02)	(0.02)	(0.27)	(0.27)	(0.27)	(0.13)	(0.13)	(0.13)
% blue collar	−0.001***	−0.001***	−0.001***	0.008*	0.009*	0.010**	0.008***	0.009***	0.009***
	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
% urban population	−0.001***	−0.001***	−0.001***	0.019***	0.018***	0.020***	0.011***	0.011***	0.012***
	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
log(% w/out HS diploma)	−0.004	0.000	−0.001	0.184*	0.114	0.143	0.000	−0.035	−0.030
	(0.01)	(0.01)	(0.01)	(0.09)	(0.09)	(0.09)	(0.04)	(0.04)	(0.04)
Joint Wald test	1171***	1132***	960***	875***	915***	727***	1436***	1330***	1210***
df	11	11	11	11	11	11	11	11	11
Wooldridge test	1184.88***	1158.59***	1278.11***	1440.52***	1404.92***	1527.77***	856.01***	837.72***	934.45***
Hausman test	718***	707***	726***	750***	745***	757***	704***	708***	716***
LM Breusch-Pagan test	2304***	2988***	2241***	3005***	72***	2950***	1701***	2196***	1680***
Individual eff. F test	8.12***	7.76***	8.38***	7.86***	7.66***	8***	8.58***	8.18***	8.97***
Time eff. F test	973.48***	856.77***	935.37***	791.53***	706.89***	767.27***	1144.23***	1014.98***	1101.53***
Two-way eff. F test	11.44***	10.97***	11.42***	10.54***	10.34***	10.47***	13.2***	12.51***	13.31***
R^2 (overall)	0.137	0.133	0.115	0.106	0.11	0.089	0.162	0.152	0.14
Adj. R^2 (within)	−0.153	−0.159	−0.182	−0.195	−0.189	−0.216	−0.119	−0.132	−0.148
AIC	−28113	−28068	−27865	27259	27212	27438	13065	13184	13321
BIC	−10216	−10171	−9968	45156	45109	45335	30962	31081	31218

$^*p < 0.05$, $^{**}p < 0.01$, $^{***}p < 0.001$.

Additional spatial panel fixed effects models

Table A.2.6. Fully specified state-level fixed effects SAR models estimating both-sex mortality indicators ($log(ASDR)$, $e(0)$, and $e(65)$) featuring various specifications of state health policy.

	$log(ASDR)$			$e(0)$			$e(65)$		
Variable	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$
SHP indicator	-0.019^{***}	-0.017^{***}	0.000	0.293^{***}	0.289^{***}	0.012	0.130^{***}	0.112^{***}	-0.018
	(0.00)	(0.00)	(0.00)	(0.07)	(0.05)	(0.03)	(0.03)	(0.02)	(0.01)
Median age	-0.003	-0.002	-0.002	0.024	0.010	0.022	0.033	0.034	0.032
	(0.00)	(0.00)	(0.00)	(0.04)	(0.04)	(0.05)	(0.02)	(0.02)	(0.02)
log(gov't ideology)	-0.012	0.013	-0.021^{**}	0.113	-0.179	0.252^{*}	0.125^{*}	-0.082	0.189^{***}
	(0.01)	(0.01)	(0.01)	(0.12)	(0.13)	(0.12)	(0.05)	(0.06)	(0.05)
log(% in poor health)	0.030	0.015	0.066	-0.057	0.282	-0.704	-0.410	-0.343	-0.610^{*}
	(0.04)	(0.04)	(0.04)	(0.68)	(0.66)	(0.69)	(0.30)	(0.30)	(0.31)
log(% Black)	0.023^{*}	0.022^{*}	0.015	-0.360^{*}	-0.348^{*}	-0.240	-0.202^{**}	-0.188^{**}	-0.133
	(0.01)	(0.01)	(0.01)	(0.15)	(0.15)	(0.16)	(0.07)	(0.07)	(0.07)
D index	-0.004^{**}	-0.004^{**}	-0.003^{*}	0.063^{**}	0.061^{**}	0.054^{**}	0.014	0.014	0.009
	(0.00)	(0.00)	(0.00)	(0.02)	(0.02)	(0.02)	(0.01)	(0.01)	(0.01)
log(med. HH income)	0.082	0.082	0.104^{*}	-1.391	-1.481	-1.711^{*}	-0.765^{*}	-0.729^{*}	-0.951^{*}
	(0.05)	(0.05)	(0.05)	(0.81)	(0.78)	(0.85)	(0.36)	(0.36)	(0.37)
% blue collar	-0.004^{*}	-0.004^{**}	-0.005^{**}	0.039	0.049	0.063^{*}	0.035^{**}	0.043^{***}	0.047^{***}
	(0.00)	(0.00)	(0.00)	(0.03)	(0.03)	(0.03)	(0.01)	(0.01)	(0.01)
% urban population	-0.001	-0.001	-0.002^{*}	0.025	0.024	0.035^{*}	0.008	0.007	0.013
	(0.00)	(0.00)	(0.00)	(0.01)	(0.01)	(0.02)	(0.01)	(0.01)	(0.01)
log(% w/out HS diploma)	-0.095^{**}	-0.081^{**}	-0.107^{***}	1.796^{***}	1.440^{**}	1.990^{***}	0.608^{**}	0.548^{*}	0.685^{**}
	(0.03)	(0.03)	(0.03)	(0.51)	(0.50)	(0.53)	(0.22)	(0.23)	(0.23)
λ	0.391^{***}	0.434^{***}	0.450^{***}	0.316^{***}	0.339^{***}	0.370^{***}	0.439^{***}	0.480^{***}	0.486^{***}
	(0.07)	(0.07)	(0.07)	(0.08)	(0.07)	(0.08)	(0.07)	(0.06)	(0.06)
RW test (p-value)	0.5475	0.1938	0.1818	0.3377	0.0599	0.0859	0.3576	0.3656	0.2398
BSJK (C.2) test	16^{***}	18^{***}	16^{***}	16^{***}	20^{***}	15^{***}	17^{***}	18^{***}	17^{***}
Pseudo R^2	0.973	0.974	0.971	0.967	0.969	0.964	0.982	0.982	0.981
AIC	-846	-850	-825	277	264	295	-47	-42	-30
BIC	-632	-636	-611	491	479	510	168	172	185

$^{*}p < 0.05$, $^{**}p < 0.01$, $^{***}p < 0.001$.

Table A.2.7. Fully specified state-level fixed effects SAR models estimating female mortality indicators ($log(ASDR)$, $e(0)$, and $e(65)$) featuring various specifications of state health policy.

	$log(ASDR)$			$e(0)$			$e(65)$		
Variable	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$
SHP indicator	−0.020***	−0.018***	0.002	0.290***	0.278***	−0.011	0.146***	0.133***	−0.027
	(0.00)	(0.00)	(0.00)	(0.06)	(0.04)	(0.03)	(0.03)	(0.02)	(0.02)
Median age	−0.004	−0.005	−0.005	0.040	0.048	0.057	0.039*	0.047*	0.048*
	(0.00)	(0.00)	(0.00)	(0.04)	(0.04)	(0.04)	(0.02)	(0.02)	(0.02)
log(gov't ideology)	−0.015*	0.014	−0.025**	0.151	−0.190	0.289*	0.160**	−0.095	0.233***
	(0.01)	(0.01)	(0.01)	(0.11)	(0.12)	(0.11)	(0.06)	(0.07)	(0.06)
log(% in poor health)	0.026	−0.006	0.039	−0.061	0.436	−0.289	−0.345	−0.118	−0.424
	(0.04)	(0.04)	(0.05)	(0.64)	(0.63)	(0.66)	(0.34)	(0.35)	(0.35)
log(% Black)	0.035***	0.035***	0.026*	−0.456**	−0.459**	−0.338*	−0.321***	−0.316***	−0.245**
	(0.01)	(0.01)	(0.01)	(0.15)	(0.14)	(0.15)	(0.08)	(0.08)	(0.08)
D index	−0.004**	−0.004**	−0.003*	0.062***	0.061***	0.051**	0.018	0.019	0.011
	(0.00)	(0.00)	(0.00)	(0.02)	(0.02)	(0.02)	(0.01)	(0.01)	(0.01)
log(med. HH income)	0.088	0.081	0.103*	−1.234	−1.189	−1.414	−0.965*	−0.880*	−1.112**
	(0.05)	(0.05)	(0.05)	(0.72)	(0.70)	(0.76)	(0.39)	(0.39)	(0.40)
% blue collar	−0.005*	−0.006**	−0.006***	0.052	0.065*	0.077**	0.041**	0.050***	0.055***
	(0.00)	(0.00)	(0.00)	(0.03)	(0.03)	(0.03)	(0.01)	(0.01)	(0.01)
% urban population	−0.001	−0.001	−0.002	0.020	0.020	0.032*	0.006	0.005	0.013
	(0.00)	(0.00)	(0.00)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)
log(% w/out HS diploma)	−0.090**	−0.073*	−0.099**	1.342**	1.031*	1.463**	0.678**	0.576*	0.737**
	(0.03)	(0.03)	(0.03)	(0.47)	(0.46)	(0.49)	(0.25)	(0.26)	(0.26)
λ	0.435***	0.471***	0.485***	0.400***	0.423***	0.452***	0.443***	0.479***	0.480***
	(0.07)	(0.06)	(0.06)	(0.07)	(0.07)	(0.07)	(0.06)	(0.06)	(0.06)
RW test (p-value)	0.8651	0.1259	0.2617	0.7053	0.1878	0.3816	0.7992	0.0659	0.4835
BSJK (C.2) test	19***	33***	19***	47***	43***	19***	18***	20***	18***
Pseudo R^2	0.962	0.963	0.958	0.957	0.96	0.953	0.969	0.968	0.966
AIC	−813	−816	−792	258	247	278	12	15	27
BIC	−598	−602	−578	472	461	493	226	229	241

$^{*}p < 0.05$, $^{**}p < 0.01$, $^{***}p < 0.001$.

Table A.2.8. Fully specified state-level fixed effects SAR models estimating male mortality indicators ($log(ASDR)$, $e(0)$, and $e(65)$) featuring various specifications of state health policy.

	$log(ASDR)$			$e(0)$			$e(65)$		
Variable	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$
SHP indicator	−0.018***	−0.016***	−0.001	0.309***	0.297***	0.029	0.118***	0.096***	−0.007
	(0.00)	(0.00)	(0.00)	(0.07)	(0.05)	(0.04)	(0.03)	(0.02)	(0.01)
Median age	−0.001	−0.001	0.000	−0.009	−0.021	−0.026	0.023	0.025	0.017
	(0.00)	(0.00)	(0.00)	(0.05)	(0.05)	(0.06)	(0.02)	(0.02)	(0.02)
log(gov't ideology)	−0.012	0.012	−0.021**	0.115	−0.167	0.265*	0.116*	−0.058	0.172***
	(0.01)	(0.01)	(0.01)	(0.13)	(0.14)	(0.13)	(0.05)	(0.06)	(0.05)
log(% in poor health)	−0.024	−0.012	0.018	0.856	0.782	0.058	−0.012	−0.113	−0.251
	(0.04)	(0.03)	(0.04)	(0.65)	(0.62)	(0.65)	(0.25)	(0.25)	(0.25)
log(% Black)	0.013	0.011	0.005	−0.269	−0.250	−0.147	−0.076	−0.062	−0.020
	(0.01)	(0.01)	(0.01)	(0.17)	(0.16)	(0.17)	(0.07)	(0.07)	(0.07)
D index	−0.004**	−0.004**	−0.003**	0.066**	0.064**	0.059**	0.015	0.015	0.011
	(0.00)	(0.00)	(0.00)	(0.02)	(0.02)	(0.02)	(0.01)	(0.01)	(0.01)
log(med. HH income)	0.095	0.095	0.124*	−1.546	−1.642	−2.077*	−0.758*	−0.738*	−0.963**
	(0.05)	(0.05)	(0.05)	(0.93)	(0.89)	(0.95)	(0.36)	(0.36)	(0.37)
% blue collar	−0.002	−0.003	−0.003	0.009	0.023	0.035	0.017	0.025*	0.028*
	(0.00)	(0.00)	(0.00)	(0.03)	(0.03)	(0.03)	(0.01)	(0.01)	(0.01)
% urban population	−0.002	−0.002	−0.002*	0.029	0.030	0.038*	0.010	0.011	0.014*
	(0.00)	(0.00)	(0.00)	(0.02)	(0.02)	(0.02)	(0.01)	(0.01)	(0.01)
log(% w/out HS diploma)	−0.071*	−0.066*	−0.081**	1.803***	1.577**	1.959***	0.359	0.355	0.428*
	(0.03)	(0.03)	(0.03)	(0.54)	(0.52)	(0.55)	(0.20)	(0.21)	(0.21)
λ	0.340***	0.372***	0.401***	0.247**	0.256**	0.291***	0.435***	0.461***	0.485***
	(0.08)	(0.07)	(0.07)	(0.08)	(0.08)	(0.08)	(0.07)	(0.07)	(0.07)
RW test (p-value)	0.2198	0.2557	0.044	0.8272	0.3077	0.5175	0.9391	0.2717	0.3996
BSJK (C.2) test	15***	16***	15***	16***	18***	15***	16***	15***	16***
Pseudo R^2	0.981	0.981	0.979	0.972	0.974	0.97	0.989	0.989	0.988
AIC	−846	−848	−827	314	302	330	−61	−55	−45
BIC	−631	−633	−612	528	517	545	154	160	170

$^{*}p < 0.05$, $^{**}p < 0.01$, $^{***}p < 0.001$.

Table A.2.9. Fully specified state-level fixed effects SEM models estimating both-sex mortality indicators ($log(ASDR)$, $e(0)$, and $e(65)$) featuring various specifications of state health policy.

	$log(ASDR)$			$e(0)$			$e(65)$		
Variable	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$
SHP indicator	−0.016***	−0.013***	0.001	0.280***	0.242***	0.007	0.119***	0.082***	−0.015
	(0.00)	(0.00)	(0.00)	(0.07)	(0.05)	(0.03)	(0.03)	(0.02)	(0.01)
Median age	0.001	−0.001	0.001	−0.026	−0.008	−0.035	0.012	0.023	0.008
	(0.00)	(0.00)	(0.00)	(0.05)	(0.04)	(0.05)	(0.02)	(0.02)	(0.02)
log(gov't ideology)	−0.010	0.018*	−0.016*	0.073	−0.266*	0.186	0.101*	−0.107	0.153**
	(0.01)	(0.01)	(0.01)	(0.12)	(0.12)	(0.12)	(0.05)	(0.05)	(0.05)
log(% in poor health)	−0.006	−0.024	0.016	0.512	0.805	0.053	−0.173	−0.080	−0.317
	(0.04)	(0.04)	(0.04)	(0.64)	(0.64)	(0.65)	(0.28)	(0.29)	(0.28)
log(% Black)	0.030**	0.035***	0.030**	−0.459**	−0.497**	−0.466**	−0.230**	−0.271***	−0.220**
	(0.01)	(0.01)	(0.01)	(0.16)	(0.16)	(0.17)	(0.07)	(0.07)	(0.08)
D index	−0.002*	−0.003**	−0.002	0.046*	0.057**	0.043*	0.007	0.012	0.005
	(0.00)	(0.00)	(0.00)	(0.02)	(0.02)	(0.02)	(0.01)	(0.01)	(0.01)
log(med. HH income)	0.048	0.056	0.051	−0.863	−1.111	−0.919	−0.779*	−0.820*	−0.823*
	(0.05)	(0.05)	(0.05)	(0.86)	(0.84)	(0.89)	(0.38)	(0.39)	(0.39)
% blue collar	−0.003*	−0.004*	−0.004*	0.035	0.045	0.050	0.037**	0.041**	0.044***
	(0.00)	(0.00)	(0.00)	(0.03)	(0.03)	(0.03)	(0.01)	(0.01)	(0.01)
% urban population	0.000	0.000	0.000	0.003	0.010	0.007	−0.002	0.001	0.001
	(0.00)	(0.00)	(0.00)	(0.02)	(0.01)	(0.02)	(0.01)	(0.01)	(0.01)
log(% w/out HS diploma)	−0.146***	−0.118***	−0.150***	2.510***	1.955***	2.579***	0.911***	0.736**	0.944***
	(0.03)	(0.03)	(0.03)	(0.55)	(0.55)	(0.57)	(0.25)	(0.26)	(0.25)
ρ	0.642***	0.643***	0.682***	0.571***	0.543***	0.614***	0.658***	0.659***	0.686***
	(0.06)	(0.06)	(0.05)	(0.07)	(0.07)	(0.06)	(0.06)	(0.06)	(0.05)
RW test (p-value)	0.5475	0.1938	0.1818	0.3377	0.0599	0.0859	0.3576	0.3656	0.2398
BSJK (C.2) test	16***	18***	16***	16***	20***	15***	17***	18***	17***
Pseudo R^2	0.961	0.961	0.954	0.958	0.961	0.952	0.972	0.971	0.968
AIC	−360	−362	−344	761	753	778	440	448	455
BIC	−146	−147	−130	976	968	993	655	662	669

$^{*}p < 0.05$, $^{**}p < 0.01$, $^{***}p < 0.001$.

Table A.2.10. Fully specified state-level fixed effects SEM models estimating female mortality indicators ($log(ASDR)$, $e(0)$, and $e(65)$) featuring various specifications of state health policy.

	$log(ASDR)$			$e(0)$			$e(65)$		
Variable	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$
SHP indicator	−0.018***	−0.013***	0.002	0.263***	0.223***	−0.014	0.131***	0.094***	−0.024
	(0.00)	(0.00)	(0.00)	(0.06)	(0.04)	(0.03)	(0.03)	(0.02)	(0.01)
Median age	−0.001	−0.003	−0.002	0.005	0.036	0.020	0.019	0.037	0.028
	(0.00)	(0.00)	(0.00)	(0.04)	(0.04)	(0.04)	(0.02)	(0.02)	(0.02)
log(gov't ideology)	−0.012	0.021**	−0.019*	0.109	−0.298*	0.211	0.127*	−0.137*	0.185**
	(0.01)	(0.01)	(0.01)	(0.11)	(0.12)	(0.11)	(0.06)	(0.06)	(0.06)
log(% in poor health)	−0.003	−0.033	0.008	0.434	0.879	0.233	−0.162	0.037	−0.237
	(0.04)	(0.04)	(0.04)	(0.64)	(0.63)	(0.66)	(0.34)	(0.35)	(0.35)
log(% Black)	0.042***	0.049***	0.042***	−0.530	−0.603***	−0.539**	−0.362***	−0.416***	−0.350***
	(0.01)	(0.01)	(0.01)	(0.16)	(0.16)	(0.17)	(0.09)	(0.09)	(0.09)
D index	−0.003*	−0.003**	−0.002	0.045*	0.056**	0.041*	0.009	0.016	0.007
	(0.00)	(0.00)	(0.00)	(0.02)	(0.02)	(0.02)	(0.01)	(0.01)	(0.01)
log(med. HH income)	0.055	0.057	0.043	−0.803	−0.899	−0.616	−0.878*	−0.893*	−0.817
	(0.06)	(0.06)	(0.06)	(0.81)	(0.79)	(0.84)	(0.44)	(0.44)	(0.45)
% blue collar	−0.005*	−0.005**	−0.006**	0.052	0.061*	0.067*	0.043**	0.046**	0.051***
	(0.00)	(0.00)	(0.00)	(0.03)	(0.03)	(0.03)	(0.01)	(0.01)	(0.01)
% urban population	0.000	0.000	0.000	0.004	0.010	0.010	−0.004	−0.001	0.000
	(0.00)	(0.00)	(0.00)	(0.01)	(0.01)	(0.02)	(0.01)	(0.01)	(0.01)
log(% w/out HS diploma)	−0.133***	−0.107**	−0.141***	1.894***	1.456**	2.006***	0.966***	0.782**	1.032***
	(0.04)	(0.04)	(0.04)	(0.52)	(0.52)	(0.54)	(0.28)	(0.29)	(0.29)
ρ	0.624***	0.641***	0.666***	0.576***	0.580***	0.626***	0.630***	0.637***	0.657***
	(0.06)	(0.06)	(0.06)	(0.07)	(0.07)	(0.06)	(0.06)	(0.06)	(0.06)
RW test (p-value)	0.8651	0.1259	0.2617	0.7053	0.1878	0.3816	0.7992	0.0659	0.4835
BSJK (C.2) test	19***	33***	19***	47***	43***	19***	18***	20***	18***
Pseudo R^2	0.942	0.941	0.934	0.941	0.943	0.932	0.953	0.951	0.948
AIC	−320	−322	−306	751	742	766	504	509	515
BIC	−105	−107	−92	965	957	981	718	723	729

$^{*}p < 0.05$, $^{**}p < 0.01$, $^{***}p < 0.001$.

Table A.2.11. Fully specified state-level fixed effects SEM models estimating male mortality indicators ($log(ASDR)$, $e(0)$, and $e(65)$) featuring various specifications of state health policy.

	$log(ASDR)$			$e(0)$			$e(65)$		
Variable	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$
SHP indicator	−0.016***	−0.012***	0.000	0.307***	0.257***	0.021	0.112***	0.072***	−0.006
	(0.00)	(0.00)	(0.00)	(0.07)	(0.05)	(0.03)	(0.03)	(0.02)	(0.01)
Median age	0.001	0.000	0.002	−0.049	−0.031	−0.067	0.017	0.026	0.010
	(0.00)	(0.00)	(0.00)	(0.05)	(0.05)	(0.05)	(0.02)	(0.02)	(0.02)
log(gov't ideology)	−0.009	0.016*	−0.015*	0.052	−0.239	0.178	0.084	−0.082	0.132**
	(0.01)	(0.01)	(0.01)	(0.13)	(0.14)	(0.13)	(0.05)	(0.05)	(0.05)
log(% in poor health)	−0.042	−0.037	−0.014	1.158	1.031	0.552	0.094	0.042	−0.093
	(0.03)	(0.03)	(0.03)	(0.60)	(0.59)	(0.61)	(0.23)	(0.23)	(0.23)
log(% Black)	0.018	0.021*	0.018	−0.385*	−0.389*	−0.387*	−0.086	−0.116	−0.081
	(0.01)	(0.01)	(0.01)	(0.18)	(0.18)	(0.19)	(0.07)	(0.07)	(0.07)
D index	−0.003*	−0.003**	−0.003*	0.049*	0.059**	0.047*	0.009	0.014	0.008
	(0.00)	(0.00)	(0.00)	(0.02)	(0.02)	(0.02)	(0.01)	(0.01)	(0.01)
log(med. HH income)	0.037	0.050	0.045	−0.607	−1.036	−0.840	−0.601	−0.671	−0.685
	(0.05)	(0.05)	(0.05)	(0.94)	(0.92)	(0.97)	(0.37)	(0.37)	(0.38)
% blue collar	−0.001	−0.002	−0.002	0.004	0.019	0.020	0.018	0.023	0.024
	(0.00)	(0.00)	(0.00)	(0.03)	(0.03)	(0.03)	(0.01)	(0.01)	(0.01)
% urban population	0.000	−0.001	0.000	0.007	0.017	0.011	0.004	0.006	0.006
	(0.00)	(0.00)	(0.00)	(0.02)	(0.02)	(0.02)	(0.01)	(0.01)	(0.01)
log(% w/out HS diploma)	−0.147***	−0.125***	−0.152***	2.850***	2.295***	2.900***	0.895***	0.762**	0.928***
	(0.03)	(0.03)	(0.03)	(0.59)	(0.58)	(0.61)	(0.23)	(0.24)	(0.24)
ρ	0.609***	0.598***	0.652***	0.535***	0.472***	0.569***	0.642***	0.638***	0.672***
	(0.06)	(0.06)	(0.06)	(0.07)	(0.08)	(0.07)	(0.06)	(0.06)	(0.06)
RW test (p-value)	0.2198	0.2557	0.044	0.8272	0.3077	0.5175	0.9391	0.2717	0.3996
BSJK (C.2) test	15***	16***	15***	16***	18***	15***	16***	15***	16***
Pseudo R^2	0.974	0.975	0.97	0.967	0.97	0.963	0.983	0.983	0.981
AIC	−362	−362	−346	796	789	812	425	433	440
BIC	−148	−147	−132	1010	1004	1026	640	648	654

$^{*}p < 0.05$, $^{**}p < 0.01$, $^{***}p < 0.001$.

Table A.2.12. Fully specified state-level fixed effects SLX models estimating both-sex mortality indicators ($log(ASDR)$, $e(0)$, and $e(65)$) featuring various specifications of state health policy.

	$log(ASDR)$			$e(0)$			$e(65)$		
Variable	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$
SHP indicator	−0.016**	−0.014***	0.001	0.240**	0.224***	0.013	0.125**	0.094**	−0.020
	(0.01)	(0.00)	(0.00)	(0.09)	(0.06)	(0.04)	(0.04)	(0.03)	(0.02)
Median age†	0.000	−0.001	0.000	−0.028	−0.015	−0.033	0.022	0.030	0.022
	(0.00)	(0.00)	(0.00)	(0.06)	(0.05)	(0.06)	(0.03)	(0.03)	(0.03)
log(gov't ideology)*	−0.011	0.013	−0.016	0.095	−0.163	0.165	0.112	−0.080	0.154*
	(0.01)	(0.01)	(0.01)	(0.15)	(0.16)	(0.15)	(0.07)	(0.08)	(0.07)
log(% in poor health)	0.038	0.007	0.069	−0.054	0.562	−0.584	−0.578	−0.418	−0.775
	(0.05)	(0.05)	(0.05)	(0.83)	(0.84)	(0.86)	(0.38)	(0.41)	(0.40)
log(% Black)	0.036**	0.037**	0.032*	−0.525*	−0.543**	−0.513*	−0.297**	−0.303**	−0.248*
	(0.01)	(0.01)	(0.01)	(0.21)	(0.20)	(0.23)	(0.10)	(0.10)	(0.11)
D index	−0.002	−0.002	−0.002	0.033	0.030	0.029	0.007	0.004	0.000
	(0.00)	(0.00)	(0.00)	(0.02)	(0.02)	(0.03)	(0.01)	(0.01)	(0.01)
log(med. HH income)	0.043	0.044	0.081	−0.628	−0.568	−1.093	−0.634	−0.676	−0.939
	(0.07)	(0.07)	(0.07)	(1.12)	(1.10)	(1.15)	(0.51)	(0.54)	(0.54)
% blue collar	−0.004	−0.005*	−0.005*	0.045	0.052	0.060	0.039*	0.045**	0.049**
	(0.00)	(0.00)	(0.00)	(0.03)	(0.03)	(0.04)	(0.02)	(0.02)	(0.02)
% urban population	−0.001	−0.001	−0.002	0.018	0.019	0.026	0.010	0.010	0.014
	(0.00)	(0.00)	(0.00)	(0.02)	(0.02)	(0.02)	(0.01)	(0.01)	(0.01)
log(% w/out HS diploma)	−0.142	−0.139**	−0.139**	2.600***	2.426	2.550***	0.879**	0.877*	0.862*
	(0.04)	(0.04)	(0.05)	(0.70)	(0.72)	(0.73)	(0.32)	(0.35)	(0.34)
Lagged indicators									
SHP indicator	−0.023*	−0.022**	0.001	0.181	0.286*	0.003	0.206**	0.171**	−0.037
	(0.01)	(0.01)	(0.01)	(0.16)	(0.13)	(0.08)	(0.07)	(0.06)	(0.04)
Median age†	−0.014*	−0.009	−0.016*	0.222*	0.143	0.242*	0.094*	0.072	0.108*
	(0.01)	(0.01)	(0.01)	(0.10)	(0.10)	(0.11)	(0.05)	(0.05)	(0.05)
log(gov't ideology)*	−0.012	−0.024	−0.022	0.152	0.427	0.247	0.133	0.158	0.205
	(0.02)	(0.02)	(0.02)	(0.27)	(0.31)	(0.28)	(0.12)	(0.15)	(0.13)
log(% in poor health)	0.149	0.175	0.237*	−2.244	−2.168	−3.210	−1.360	−1.645*	−2.063**
	(0.10)	(0.10)	(0.10)	(1.63)	(1.63)	(1.66)	(0.74)	(0.79)	(0.78)
log(% Black)	−0.021	−0.035	−0.069*	0.506	0.496	1.087*	−0.036	0.142	0.351
	(0.03)	(0.03)	(0.03)	(0.45)	(0.43)	(0.43)	(0.20)	(0.21)	(0.20)
D index	−0.001	0.003	0.002	0.016	−0.042	−0.014	0.013	−0.016	−0.018
	(0.00)	(0.00)	(0.00)	(0.05)	(0.05)	(0.05)	(0.02)	(0.02)	(0.03)
log(med. HH income)	−0.070	−0.008	0.024	0.519	−0.051	−0.357	1.404	0.951	0.421
	(0.12)	(0.12)	(0.13)	(1.98)	(1.90)	(2.07)	(0.90)	(0.93)	(0.97)
% blue collar	0.007	0.001	0.004	−0.097	−0.036	−0.075	−0.059	−0.007	−0.023
	(0.00)	(0.00)	(0.00)	(0.07)	(0.07)	(0.07)	(0.03)	(0.03)	(0.03)
% urban population	−0.003	−0.002	−0.005*	0.063	0.044	0.080*	0.021	0.019	0.038*
	(0.00)	(0.00)	(0.00)	(0.04)	(0.04)	(0.04)	(0.02)	(0.02)	(0.02)
log(% w/out HS diploma)	0.005	0.044	−0.078	0.168	−0.699	1.311	0.038	−0.111	0.588
	(0.08)	(0.09)	(0.08)	(1.25)	(1.38)	(1.28)	(0.57)	(0.67)	(0.60)
Joint Wald test	166***	163***	129***	136***	151***	116***	181***	157***	139***
df	20	20	20	20	20	20	20	20	20
Wooldridge test	120.42***	80.66***	130.78***	187.93***	157.31***	225.15***	43.07***	21.84***	54.96***
Hausman test	20***	16***	18***	22***	20***	20***	18***	14***	17***
LM Breusch-Pagan test	66***	43**	131***	513***	181***	231***	178***	228***	552***
Individual eff. F test	6.36***	6.74***	6.32***	6.13***	6.62***	5.69***	7.88***	7.97***	8.67***
Time eff. F test	14.4***	14.94***	16.35***	11.34***	11.12***	12.2***	18.31***	18.74***	21.75***
Two-way eff. F test	7.78***	8.09***	7.59***	6.52***	7.18***	6.19***	12.39***	11.6***	12.46***
R^2 (overall)	0.566	0.562	0.504	0.517	0.543	0.477	0.587	0.552	0.523
Adj. R^2 (within)	0.32	0.314	0.222	0.243	0.284	0.181	0.354	0.298	0.253
AIC	−867	−865	−840	257	246	273	−58	−42	−29
BIC	−626	−624	−599	498	487	514	183	199	212

$^{*}p < 0.05$, $^{**}p < 0.01$, $^{***}p < 0.001$.

Table A.2.13. Fully specified state-level fixed effects SLX models estimating female mortality indicators ($log(ASDR)$, $e(0)$, and $e(65)$) featuring various specifications of state health policy.

	$log(ASDR)$			$e(0)$			$e(65)$		
Variable	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$
SHP indicator	−0.018**	−0.016***	0.002	0.253**	0.240***	−0.010	0.135**	0.110**	−0.027
	(0.01)	(0.00)	(0.00)	(0.08)	(0.06)	(0.04)	(0.05)	(0.03)	(0.02)
Median age†	−0.003	−0.005	−0.004	0.022	0.046	0.043	0.032	0.047	0.046
	(0.00)	(0.00)	(0.00)	(0.05)	(0.05)	(0.05)	(0.03)	(0.03)	(0.03)
log(gov't ideology)*	−0.015	0.009	−0.022*	0.140	−0.129	0.235	0.155	−0.045	0.212*
	(0.01)	(0.01)	(0.01)	(0.14)	(0.15)	(0.15)	(0.08)	(0.09)	(0.08)
log(% in poor health)	0.025	−0.044	0.025	0.140	1.139	0.058	−0.440	0.043	−0.423
	(0.06)	(0.06)	(0.06)	(0.83)	(0.83)	(0.88)	(0.46)	(0.49)	(0.49)
log(% Black)	0.050***	0.052***	0.045**	−0.636**	−0.659**	−0.589*	−0.427***	−0.453***	−0.373**
	(0.01)	(0.01)	(0.02)	(0.21)	(0.20)	(0.23)	(0.12)	(0.12)	(0.13)
D index	−0.002	−0.002	−0.001	0.038	0.034	0.026	0.009	0.007	0.000
	(0.00)	(0.00)	(0.00)	(0.02)	(0.02)	(0.03)	(0.01)	(0.01)	(0.01)
log(med. HH income)	0.056	0.060	0.091	−0.641	−0.648	−1.084	−0.768	−0.824	−1.021
	(0.07)	(0.07)	(0.08)	(1.07)	(1.06)	(1.13)	(0.59)	(0.62)	(0.63)
% blue collar	−0.006*	−0.007**	−0.007**	0.067*	0.080*	0.091*	0.048*	0.057**	0.062**
	(0.00)	(0.00)	(0.00)	(0.03)	(0.03)	(0.04)	(0.02)	(0.02)	(0.02)
% urban population	−0.001	−0.002	−0.002	0.023	0.025	0.036	0.009	0.011	0.016
	(0.00)	(0.00)	(0.00)	(0.02)	(0.02)	(0.02)	(0.01)	(0.01)	(0.01)
log(% w/out HS diploma)	−0.120**	−0.108*	−0.113*	1.833**	1.611*	1.722*	0.853*	0.759	0.819*
	(0.05)	(0.05)	(0.05)	(0.66)	(0.69)	(0.71)	(0.37)	(0.40)	(0.39)
Lagged indicators									
SHP indicator	−0.030**	−0.024**	0.002	0.344*	0.317*	−0.022	0.225*	0.170*	−0.033
	(0.01)	(0.01)	(0.01)	(0.16)	(0.13)	(0.08)	(0.09)	(0.07)	(0.05)
Median age†	−0.018**	−0.015*	−0.024***	0.246*	0.188*	0.333***	0.122*	0.115*	0.171**
	(0.01)	(0.01)	(0.01)	(0.09)	(0.09)	(0.10)	(0.05)	(0.06)	(0.05)
log(gov't ideology)*	−0.010	−0.047*	−0.018	0.128	0.685*	0.221	0.112	0.333	0.156
	(0.02)	(0.02)	(0.02)	(0.26)	(0.29)	(0.27)	(0.14)	(0.17)	(0.15)
log(% in poor health)	0.075	0.049	0.061	−1.292	−0.928	−1.090	−0.669	−0.471	−0.598
	(0.10)	(0.10)	(0.11)	(1.52)	(1.47)	(1.62)	(0.84)	(0.86)	(0.90)
log(% Black)	−0.023	−0.033	−0.074*	0.318	0.327	0.997*	0.000	0.129	0.376
	(0.03)	(0.03)	(0.03)	(0.43)	(0.42)	(0.43)	(0.24)	(0.25)	(0.24)
D index	−0.004	0.002	0.000	0.049	−0.032	−0.001	0.032	−0.006	−0.003
	(0.00)	(0.00)	(0.00)	(0.05)	(0.05)	(0.05)	(0.03)	(0.03)	(0.03)
log(med. HH income)	0.005	0.038	0.123	−0.119	−0.481	−1.569	0.459	0.261	−0.608
	(0.12)	(0.12)	(0.13)	(1.77)	(1.72)	(1.91)	(0.98)	(1.01)	(1.06)
% blue collar	0.006	−0.003	0.000	−0.077	0.027	0.001	−0.050	0.022	0.005
	(0.01)	(0.00)	(0.01)	(0.07)	(0.07)	(0.08)	(0.04)	(0.04)	(0.04)
% urban population	−0.002	−0.002	−0.005	0.043	0.033	0.073*	0.027	0.028	0.047*
	(0.00)	(0.00)	(0.00)	(0.03)	(0.04)	(0.04)	(0.02)	(0.02)	(0.02)
log(% w/out HS diploma)	−0.006	0.070	−0.041	0.181	−0.972	0.729	0.116	−0.346	0.289
	(0.08)	(0.09)	(0.09)	(1.23)	(1.33)	(1.32)	(0.68)	(0.78)	(0.73)
Joint Wald test	178***	177***	136***	151***	167***	118***	179***	163***	144***
df	20	20	20	20	20	20	20	20	20
Wooldridge test	119.06***	72.88***	115.66***	129.79***	96.94***	143.98***	60.45***	30.01***	67.75***
Hausman test	18***	14***	20***	20***	17***	21***	17***	13***	15***
LM Breusch-Pagan test	565***	2	299***	256***	159***	174***	2463***	116***	8100***
Individual eff. F test	6.83***	7.73***	7.67***	5.72***	6.61***	6.1***	8.58***	9.33***	10.14***
Time eff. F test	15.4***	16.39***	17.88***	12.75***	13.02***	14.52***	19.04***	19.38***	22.54***
Two-way eff. F test	8.14***	8.48***	8.38***	6.11***	6.77***	6.19***	12.01***	11.59***	12.67***
R^2 (overall)	0.583	0.583	0.517	0.543	0.568	0.482	0.585	0.563	0.532
Adj. R^2 (within)	0.347	0.346	0.244	0.283	0.322	0.189	0.349	0.315	0.266
AIC	−825	−824	−795	246	235	271	11	22	35
BIC	−584	−584	−555	487	476	512	252	262	276

$^{*}p < 0.05$, $^{**}p < 0.01$, $^{***}p < 0.001$.

Table A.2.14. Fully specified state-level fixed effects SLX models estimating male mortality indicators ($log(ASDR)$, $e(0)$, and $e(65)$) featuring various specifications of state health policy.

	$log(ASDR)$			$e(0)$			$e(65)$		
Variable	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$
SHP indicator	−0.016**	−0.011**	0.000	0.246**	0.201**	0.024	0.127***	0.081**	−0.015
	(0.00)	(0.00)	(0.00)	(0.09)	(0.06)	(0.04)	(0.04)	(0.03)	(0.02)
Median age†	−0.001	−0.001	0.000	−0.035	−0.035	−0.053	0.033	0.031	0.027
	(0.00)	(0.00)	(0.00)	(0.06)	(0.06)	(0.07)	(0.02)	(0.03)	(0.03)
log(gov't ideology)*	−0.011	0.013	−0.016	0.083	−0.148	0.159	0.104	−0.069	0.145*
	(0.01)	(0.01)	(0.01)	(0.16)	(0.17)	(0.16)	(0.06)	(0.07)	(0.07)
log(% in poor health)	−0.014	−0.001	0.023	0.731	0.737	0.157	−0.132	−0.280	−0.387
	(0.04)	(0.04)	(0.04)	(0.76)	(0.75)	(0.76)	(0.30)	(0.31)	(0.31)
log(% Black)	0.025	0.025*	0.022	−0.461*	−0.474*	−0.480*	−0.162	−0.158	−0.122
	(0.01)	(0.01)	(0.01)	(0.23)	(0.22)	(0.24)	(0.09)	(0.09)	(0.10)
D index	−0.002	−0.001	−0.001	0.026	0.024	0.025	0.002	−0.002	−0.003
	(0.00)	(0.00)	(0.00)	(0.03)	(0.03)	(0.03)	(0.01)	(0.01)	(0.01)
log(med. HH income)	0.009	0.015	0.045	−0.051	−0.052	−0.472	−0.331	−0.395	−0.626
	(0.07)	(0.07)	(0.07)	(1.20)	(1.18)	(1.22)	(0.47)	(0.49)	(0.50)
% blue collar	−0.002	−0.003	−0.003	0.019	0.027	0.034	0.020	0.027	0.031
	(0.00)	(0.00)	(0.00)	(0.04)	(0.04)	(0.04)	(0.01)	(0.02)	(0.02)
% urban population	−0.002	−0.002	−0.002	0.024	0.025	0.031	0.014	0.014	0.019*
	(0.00)	(0.00)	(0.00)	(0.02)	(0.02)	(0.02)	(0.01)	(0.01)	(0.01)
log(% w/out HS diploma)	−0.157***	−0.165***	−0.154***	3.153***	3.114***	3.095***	0.948**	1.041**	0.925**
	(0.04)	(0.04)	(0.04)	(0.74)	(0.76)	(0.76)	(0.29)	(0.31)	(0.31)
Lagged indicators									
SHP indicator	−0.014	−0.021**	0.000	−0.020	0.251	0.028	0.149*	0.183**	−0.037
	(0.01)	(0.01)	(0.00)	(0.18)	(0.13)	(0.09)	(0.07)	(0.06)	(0.04)
Median age†	−0.015*	−0.011	−0.015*	0.251	0.193	0.260	0.087	0.071	0.095
	(0.01)	(0.01)	(0.01)	(0.13)	(0.13)	(0.13)	(0.05)	(0.05)	(0.05)
log(gov't ideology)*	−0.019	−0.013	−0.026	0.259	0.315	0.272	0.212	0.092	0.259*
	(0.02)	(0.02)	(0.02)	(0.29)	(0.33)	(0.28)	(0.11)	(0.14)	(0.11)
log(% in poor health)	0.120	0.135	0.204*	−1.874	−1.277	−2.466	−1.076	−1.251	−1.807**
	(0.09)	(0.09)	(0.09)	(1.64)	(1.59)	(1.57)	(0.64)	(0.66)	(0.64)
log(% Black)	−0.012	−0.023	−0.048	0.679	0.566	1.043*	−0.134	−0.009	0.177
	(0.03)	(0.02)	(0.02)	(0.47)	(0.44)	(0.44)	(0.18)	(0.18)	(0.18)
D index	0.001	0.004	0.003	−0.002	−0.042	−0.015	−0.004	−0.025	−0.030
	(0.00)	(0.00)	(0.00)	(0.06)	(0.05)	(0.06)	(0.02)	(0.02)	(0.02)
log(med. HH income)	−0.027	0.017	0.053	−0.598	−0.634	−0.999	1.061	0.711	0.132
	(0.12)	(0.12)	(0.12)	(2.21)	(2.12)	(2.24)	(0.86)	(0.88)	(0.91)
% blue collar	0.003	−0.001	0.001	−0.056	−0.028	−0.056	−0.026	0.018	0.007
	(0.00)	(0.00)	(0.00)	(0.08)	(0.07)	(0.08)	(0.03)	(0.03)	(0.03)
% urban population	−0.003	−0.002	−0.004	0.069	0.046	0.074	0.014	0.011	0.028
	(0.00)	(0.00)	(0.00)	(0.04)	(0.04)	(0.04)	(0.01)	(0.02)	(0.02)
log(% w/out HS diploma)	0.137*	0.172*	0.078	−1.200	−1.983	−0.443	−1.305**	−1.516**	−0.900
	(0.07)	(0.07)	(0.07)	(1.21)	(1.33)	(1.22)	(0.47)	(0.55)	(0.50)
Joint Wald test	148***	146***	121***	120***	130***	107***	161***	140***	126***
df	20	20	20	20	20	20	20	20	20
Wooldridge test	73.4***	66.54***	101.48***	150.64***	141.2***	191.15***	21.85***	18.32***	37.88***
Hausman test	16***	16***	17***	18***	19***	19***	13***	15***	17***
LM Breusch-Pagan test	28	183***	269***	25	114***	204***	92***	310***	254***
Individual eff. F test	7.12***	6.68***	6.88***	7.73***	7.67***	7.1***	8.07***	7.06***	8.8***
Time eff. F test	11.84***	11***	12.53***	11.25***	10.71***	11.71***	14.35***	13.17***	16.03***
Two-way eff. F test	8.14***	8.16***	7.85***	7.91***	8.28***	7.38***	11.63***	10.61***	11.76***
R^2 (overall)	0.538	0.535	0.487	0.486	0.506	0.458	0.559	0.525	0.498
Adj. R^2 (within)	0.276	0.271	0.196	0.194	0.226	0.151	0.31	0.255	0.214
AIC	−882	−881	−861	285	277	296	−91	−75	−65
BIC	−641	−640	−621	526	518	537	150	165	176

*$p < 0.05$, **$p < 0.01$, ***$p < 0.001$.

Table A.2.15. Fully specified state-level fixed effects SDM models estimating female mortality indicators ($log(ASDR)$, $e(0)$, and $e(65)$) featuring various specifications of state health policy.

	$log(ASDR)$			$e(0)$			$e(65)$		
Variable	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$
SHP indicator	−0.001	−0.004*	−0.001	0.043	0.058*	0.008	0.001	0.031	0.005
	(0.00)	(0.00)	(0.00)	(0.04)	(0.03)	(0.02)	(0.02)	(0.02)	(0.01)
Median age†	−0.004***	−0.004***	−0.004***	0.046***	0.047***	0.046***	0.047***	0.047***	0.047***
	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
log(gov't ideology)*	−0.010	0.006	−0.011	0.122	−0.127	0.135	0.079	0.003	0.078
	(0.01)	(0.01)	(0.01)	(0.08)	(0.08)	(0.08)	(0.05)	(0.05)	(0.05)
log(% in poor health)	−0.066***	−0.067***	−0.065***	0.698***	0.709***	0.679***	0.625***	0.630***	0.615***
	(0.01)	(0.01)	(0.01)	(0.13)	(0.13)	(0.13)	(0.08)	(0.08)	(0.08)
log(% Black)	0.001	0.001	0.001	−0.020	−0.018	−0.017	−0.012	−0.011	−0.011
	(0.00)	(0.00)	(0.00)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)
log(D index)	−0.006***	−0.006***	−0.006***	0.091***	0.093***	0.092***	0.037***	0.038***	0.037***
	(0.00)	(0.00)	(0.00)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)
log(med. HH income)	−0.105***	−0.106***	−0.105***	1.537***	1.542***	1.535***	0.769***	0.775***	0.765***
	(0.01)	(0.01)	(0.01)	(0.12)	(0.12)	(0.12)	(0.08)	(0.08)	(0.08)
log(GINI index)	−0.092***	−0.093***	−0.094***	1.176***	1.178***	1.198***	0.765***	0.779***	0.785***
	(0.01)	(0.01)	(0.01)	(0.17)	(0.17)	(0.17)	(0.11)	(0.11)	(0.11)
% blue collar	−0.001***	−0.001***	−0.001***	0.011***	0.011***	0.011***	0.006***	0.006***	0.006***
	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
% urban population	−0.001***	−0.001***	−0.001***	0.009***	0.009***	0.010***	0.006***	0.006***	0.006***
	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
log(% w/out HS diploma)	0.003	0.004	0.004	−0.098	−0.105	−0.099	0.022	0.016	0.019
	(0.00)	(0.00)	(0.00)	(0.06)	(0.06)	(0.06)	(0.04)	(0.04)	(0.04)
Lagged indicators									
SHP indicator	−0.004	0.001	0.002	0.025	−0.015	−0.024	0.050	−0.003	−0.026*
	(0.00)	(0.00)	(0.00)	(0.04)	(0.03)	(0.02)	(0.03)	(0.02)	(0.01)
Median age†	0.001*	0.001	0.001	−0.010	−0.009	−0.009	−0.011*	−0.010*	−0.010*
	(0.00)	(0.00)	(0.00)	(0.01)	(0.01)	(0.01)	(0.00)	(0.00)	(0.00)
log(gov't ideology)*	0.005	−0.003	0.002	−0.070	0.088	−0.050	−0.011	−0.033	0.016
	(0.01)	(0.01)	(0.01)	(0.08)	(0.09)	(0.08)	(0.05)	(0.06)	(0.05)
log(% in poor health)	0.024	0.023	0.030*	−0.037	−0.028	−0.105	−0.296*	−0.278*	−0.341**
	(0.01)	(0.01)	(0.01)	(0.18)	(0.18)	(0.18)	(0.12)	(0.12)	(0.12)
log(% Black)	0.006***	0.005**	0.005**	−0.094***	−0.082***	−0.078	−0.053***	−0.044**	−0.042**
	(0.00)	(0.00)	(0.00)	(0.02)	(0.02)	(0.02)	(0.02)	(0.02)	(0.02)
log(D index)	−0.003	−0.004	−0.003	0.051	0.060*	0.053	0.017	0.025	0.019
	(0.00)	(0.00)	(0.00)	(0.03)	(0.03)	(0.03)	(0.02)	(0.02)	(0.02)
log(med. HH income)	0.066***	0.061***	0.071***	−1.074***	−1.021***	−1.129***	−0.441***	−0.378**	−0.488***
	(0.01)	(0.01)	(0.01)	(0.18)	(0.18)	(0.18)	(0.12)	(0.12)	(0.12)
log(GINI index)	−0.142***	−0.152***	−0.157***	2.085***	2.174***	2.259***	1.006***	1.134***	1.155***
	(0.03)	(0.03)	(0.03)	(0.35)	(0.35)	(0.35)	(0.22)	(0.22)	(0.22)
% blue collar	−0.001***	−0.001***	−0.001***	0.016***	0.017***	0.017***	0.010***	0.010***	0.010***
	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
% urban population	0.000**	0.000**	0.000***	0.004*	0.004*	0.004**	0.004***	0.004***	0.005***
	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
log(% w/out HS diploma)	−0.037***	−0.034***	−0.035***	0.492***	0.463***	0.476***	0.315***	0.293***	0.296***
	(0.01)	(0.01)	(0.01)	(0.10)	(0.10)	(0.10)	(0.07)	(0.07)	(0.07)
λ	0.627***	0.637***	0.634***	0.646***	0.652***	0.652***	0.607***	0.620***	0.612***
	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)
RW test (p-value)	0.002	0.002	0.002	0.002	0.002	0.002	0.002	0.002	0.002
BSJK (C.2) test	1188***	2963***	3270***	1276***	1644***	3401***	3109***	1699***	3152***
Pseudo R^2	0.924	0.924	0.924	0.928	0.928	0.928	0.92	0.92	0.92
AIC	−30812	−30779	−30787	21106	21124	21129	12021	12067	12038
BIC	−12821	−12788	−12796	39096	39114	39119	30011	30058	30028

$^{*}p < 0.05$, $^{**}p < 0.01$, $^{***}p < 0.001$.

Table A.2.16. Fully specified state-level fixed effects SDM models estimating male mortality indicators ($log(ASDR)$, $e(0)$, and $e(65)$) featuring various specifications of state health policy.

	$log(ASDR)$			$e(0)$			$e(65)$		
Variable	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$
SHP indicator	−0.015***	−0.011***	0.000	0.235***	0.192***	0.025	0.114***	0.070***	−0.013
	(0.00)	(0.00)	(0.00)	(0.07)	(0.05)	(0.03)	(0.03)	(0.02)	(0.01)
Median age	0.000	0.000	0.001	−0.055	−0.049	−0.071	0.026	0.026	0.018
	(0.00)	(0.00)	(0.00)	(0.05)	(0.05)	(0.05)	(0.02)	(0.02)	(0.02)
log(gov't ideology)	−0.010	0.014*	−0.015*	0.060	−0.189	0.132	0.095	−0.075	0.130**
	(0.01)	(0.01)	(0.01)	(0.12)	(0.13)	(0.13)	(0.05)	(0.05)	(0.05)
log(% in poor health)	−0.017	−0.010	0.013	0.707	0.750	0.186	−0.059	−0.147	−0.245
	(0.03)	(0.03)	(0.03)	(0.59)	(0.58)	(0.59)	(0.23)	(0.23)	(0.23)
log(% Black)	0.025**	0.027**	0.024*	−0.485**	−0.513**	−0.519**	−0.160*	−0.171*	−0.134
	(0.01)	(0.01)	(0.01)	(0.18)	(0.17)	(0.19)	(0.07)	(0.07)	(0.07)
D index	−0.002	−0.001	−0.001	0.024	0.026	0.025	0.002	0.001	−0.002
	(0.00)	(0.00)	(0.00)	(0.02)	(0.02)	(0.02)	(0.01)	(0.01)	(0.01)
log(med. HH income)	0.002	0.006	0.027	0.138	0.087	−0.197	−0.330	−0.385	−0.539
	(0.05)	(0.05)	(0.05)	(0.93)	(0.91)	(0.94)	(0.36)	(0.37)	(0.37)
% blue collar	−0.002	−0.002	−0.003	0.015	0.022	0.028	0.019	0.025*	0.028*
	(0.00)	(0.00)	(0.00)	(0.03)	(0.03)	(0.03)	(0.01)	(0.01)	(0.01)
% urban population	−0.001	−0.001	−0.001	0.015	0.018	0.021	0.010	0.011	0.013*
	(0.00)	(0.00)	(0.00)	(0.02)	(0.02)	(0.02)	(0.01)	(0.01)	(0.01)
log(% w/out HS diploma)	−0.169***	−0.173***	−0.170***	3.425***	3.272***	3.366***	1.030***	1.081***	1.036***
	(0.03)	(0.03)	(0.03)	(0.57)	(0.59)	(0.58)	(0.22)	(0.24)	(0.23)
Lagged indicators									
SHP indicator	−0.006	−0.015*	0.000	−0.128	0.158	0.014	0.075	0.120**	−0.033
	(0.01)	(0.01)	(0.00)	(0.14)	(0.11)	(0.07)	(0.06)	(0.04)	(0.03)
Median age	−0.010	−0.006	−0.010	0.178	0.131	0.191	0.053	0.033	0.054
	(0.01)	(0.01)	(0.01)	(0.10)	(0.10)	(0.10)	(0.04)	(0.04)	(0.04)
log(gov't ideology)	−0.014	−0.014	−0.015	0.187	0.308	0.143	0.153	0.097	0.141
	(0.01)	(0.01)	(0.01)	(0.22)	(0.25)	(0.22)	(0.09)	(0.10)	(0.09)
log(% in poor health)	0.113	0.120	0.159*	−2.207	−1.654	−2.412*	−0.790	−0.854	−1.110*
	(0.07)	(0.07)	(0.07)	(1.26)	(1.23)	(1.21)	(0.50)	(0.50)	(0.48)
log(% Black)	−0.024	−0.031	−0.051**	0.847*	0.710*	1.107**	−0.005	0.104	0.234
	(0.02)	(0.02)	(0.02)	(0.36)	(0.34)	(0.34)	(0.14)	(0.14)	(0.13)
D index	0.002	0.005*	0.004	−0.030	−0.060	−0.034	−0.011	−0.027	−0.030
	(0.00)	(0.00)	(0.00)	(0.04)	(0.04)	(0.04)	(0.02)	(0.02)	(0.02)
log(med. HH income)	−0.006	0.020	0.049	−1.101	−0.909	−1.227	0.972	0.799	0.341
	(0.09)	(0.09)	(0.09)	(1.70)	(1.64)	(1.73)	(0.66)	(0.66)	(0.68)
% blue collar	0.004	0.001	0.002	−0.057	−0.046	−0.069	−0.029	−0.001	−0.010
	(0.00)	(0.00)	(0.00)	(0.06)	(0.05)	(0.06)	(0.02)	(0.02)	(0.02)
% urban population	−0.002	−0.001	−0.003	0.060*	0.036	0.058*	0.012	0.007	0.019
	(0.00)	(0.00)	(0.00)	(0.03)	(0.03)	(0.03)	(0.01)	(0.01)	(0.01)
log(% w/out HS diploma)	0.156**	0.182**	0.118*	−1.907*	−2.384*	−1.341	−1.331***	−1.455***	−1.091**
	(0.05)	(0.06)	(0.05)	(0.94)	(1.03)	(0.96)	(0.36)	(0.41)	(0.37)
λ	0.286**	0.321***	0.350***	0.321***	0.297***	0.321***	0.321***	0.378***	0.406***
	(0.09)	(0.08)	(0.08)	(0.09)	(0.09)	(0.09)	(0.08)	(0.08)	(0.08)
RW test (p-value)	0.7013	0.2777	0.3017	0.8172	0.4735	0.0579	0.2018	0.1998	0.2138
BSJK (C.2) test	45***	34***	51***	49***	43***	50***	26***	20***	34***
Pseudo R^2	0.986	0.986	0.985	0.979	0.98	0.978	0.992	0.991	0.991
AIC	−887	−889	−871	279	272	289	−98	−89	−81
BIC	−639	−641	−624	526	520	536	150	158	166

$^{*}p < 0.05$, $^{**}p < 0.01$, $^{***}p < 0.001$.

Table A.2.17. Fully specified state-level fixed effects SDEM models estimating female mortality indicators ($log(ASDR)$, $e(0)$, and $e(65)$) featuring various specifications of state health policy.

	$log(ASDR)$			$e(0)$			$e(65)$		
Variable	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$
SHP indicator	−0.002	−0.005*	0.000	0.052	0.066*	0.001	0.012	0.037*	−0.001
	(0.00)	(0.00)	(0.00)	(0.04)	(0.03)	(0.02)	(0.02)	(0.02)	(0.01)
Median age†	−0.005***	−0.005***	−0.005***	0.051***	0.051***	0.052***	0.051***	0.051***	0.051***
	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
log(gov't ideology)*	−0.014**	0.005	−0.015**	0.166*	−0.100	0.188*	0.114*	0.008	0.119*
	(0.01)	(0.01)	(0.01)	(0.07)	(0.08)	(0.07)	(0.05)	(0.05)	(0.05)
log(% in poor health)	−0.073***	−0.075***	−0.070***	0.840***	0.873***	0.807***	0.660***	0.683***	0.642***
	(0.01)	(0.01)	(0.01)	(0.13)	(0.13)	(0.13)	(0.08)	(0.08)	(0.08)
log(% Black)	0.002*	0.002*	0.002*	−0.036**	−0.030*	−0.030*	−0.022**	−0.019*	−0.019*
	(0.00)	(0.00)	(0.00)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)
log(D index)	−0.006***	−0.007***	−0.006***	0.098***	0.103***	0.101***	0.038***	0.041***	0.040***
	(0.00)	(0.00)	(0.00)	(0.02)	(0.02)	(0.02)	(0.01)	(0.01)	(0.01)
log(med. HH income)	−0.102***	−0.104***	−0.100***	1.462***	1.490***	1.448***	0.746***	0.771***	0.732***
	(0.01)	(0.01)	(0.01)	(0.12)	(0.12)	(0.12)	(0.08)	(0.08)	(0.08)
log(GINI index)	−0.117***	−0.118***	−0.122***	1.516***	1.520***	1.570***	0.951***	0.972***	0.993***
	(0.01)	(0.01)	(0.01)	(0.18)	(0.18)	(0.18)	(0.12)	(0.12)	(0.12)
% blue collar	−0.001***	−0.001***	−0.001***	0.016***	0.016***	0.016***	0.008***	0.008***	0.008***
	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
% urban population	−0.001***	−0.001***	−0.001***	0.011***	0.011***	0.012***	0.007***	0.007***	0.007***
	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
log(% w/out HS diploma)	−0.002	0.000	0.000	−0.037	−0.062	−0.050	0.073	0.056	0.063
	(0.00)	(0.00)	(0.00)	(0.06)	(0.06)	(0.06)	(0.04)	(0.04)	(0.04)
Lagged indicators									
SHP indicator	−0.014***	−0.007**	0.004*	0.171***	0.103**	−0.040	0.127***	0.062**	−0.046**
	(0.00)	(0.00)	(0.00)	(0.05)	(0.04)	(0.02)	(0.03)	(0.02)	(0.01)
Median age†	−0.003***	−0.003***	−0.003***	0.035**	0.036**	0.039***	0.028***	0.030***	0.031***
	(0.00)	(0.00)	(0.00)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)
log(gov't ideology)*	−0.002	0.005	−0.010	0.002	−0.041	0.099	0.060	−0.090	0.130*
	(0.01)	(0.01)	(0.01)	(0.10)	(0.11)	(0.10)	(0.06)	(0.06)	(0.06)
log(% in poor health)	−0.060**	−0.068***	−0.050*	1.208***	1.305***	1.073***	0.348*	0.433*	0.267
	(0.02)	(0.02)	(0.02)	(0.28)	(0.28)	(0.28)	(0.17)	(0.17)	(0.17)
log(% Black)	0.010***	0.008	0.009***	−0.149***	−0.124***	−0.125***	−0.089***	−0.072**	−0.073***
	(0.00)	(0.00)	(0.00)	(0.04)	(0.04)	(0.04)	(0.02)	(0.02)	(0.02)
log(D index)	−0.007*	−0.009**	−0.008*	0.117**	0.136**	0.128**	0.043	0.057*	0.051
	(0.00)	(0.00)	(0.00)	(0.04)	(0.04)	(0.04)	(0.03)	(0.03)	(0.03)
log(med. HH income)	0.005	−0.006	0.012	−0.227	−0.101	−0.308	0.002	0.114	−0.070
	(0.02)	(0.02)	(0.02)	(0.29)	(0.29)	(0.29)	(0.17)	(0.18)	(0.18)
log(GINI index)	−0.253***	−0.260***	−0.272***	3.480***	3.530***	3.717***	1.900***	1.998***	2.087***
	(0.04)	(0.04)	(0.04)	(0.52)	(0.53)	(0.53)	(0.32)	(0.33)	(0.32)
% blue collar	−0.002***	−0.002***	−0.003***	0.035***	0.036***	0.036***	0.019***	0.019***	0.019***
	(0.00)	(0.00)	(0.00)	(0.01)	(0.01)	(0.01)	(0.00)	(0.00)	(0.00)
% urban population	−0.001***	−0.001***	−0.001***	0.016***	0.016***	0.018***	0.012***	0.011***	0.013***
	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
log(% w/out HS diploma)	−0.043***	−0.035**	−0.038**	0.505**	0.399*	0.443**	0.415***	0.345***	0.368***
	(0.01)	(0.01)	(0.01)	(0.16)	(0.16)	(0.16)	(0.10)	(0.10)	(0.10)
ρ	0.633***	0.644***	0.642***	0.654***	0.660***	0.662***	0.612***	0.626***	0.618***
	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)
RW test (p-value)	0.002	0.002	0.002	0.002	0.002	0.002	0.002	0.002	0.002
BSJK (C.2) test	1188***	2963***	3270***	3401***	1838***	3401***	3109***	1699***	3152***
Pseudo R^2	0.888	0.885	0.886	0.89	0.888	0.888	0.887	0.884	0.886
AIC	32179	32214	32220	84103	84122	84142	75005	75051	75035
BIC	50169	50205	50210	102093	102113	102132	92995	93042	93026

$^{*}p < 0.05$, $^{**}p < 0.01$, $^{***}p < 0.001$.

Table A.2.18. Fully specified state-level fixed effects SDEM models estimating male mortality indicators ($log(ASDR)$, $e(0)$, and $e(65)$) featuring various specifications of state health policy.

	$log(ASDR)$			$e(0)$			$e(65)$		
Variable	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$
SHP indicator	−0.015***	−0.012***	0.000	0.236***	0.214***	0.024	0.121***	0.085***	−0.016
	(0.00)	(0.00)	(0.00)	(0.07)	(0.05)	(0.03)	(0.03)	(0.02)	(0.01)
Median age	0.000	0.000	0.001	−0.047	−0.050	−0.063	0.030	0.027	0.022
	(0.00)	(0.00)	(0.00)	(0.05)	(0.05)	(0.05)	(0.02)	(0.02)	(0.02)
log(gov't ideology)	−0.012	0.014	−0.017*	0.091	−0.184	0.165	0.110*	−0.080	0.155**
	(0.01)	(0.01)	(0.01)	(0.13)	(0.14)	(0.13)	(0.05)	(0.06)	(0.05)
log(% in poor health)	−0.009	0.002	0.027	0.543	0.548	−0.023	−0.136	−0.255	−0.365
	(0.03)	(0.03)	(0.03)	(0.60)	(0.58)	(0.59)	(0.23)	(0.24)	(0.24)
log(% Black)	0.023*	0.023*	0.019	−0.430*	−0.460**	−0.436*	−0.150*	−0.146*	−0.101
	(0.01)	(0.01)	(0.01)	(0.18)	(0.17)	(0.18)	(0.07)	(0.07)	(0.07)
D index	−0.002	−0.001	−0.001	0.023	0.021	0.023	0.002	−0.002	−0.004
	(0.00)	(0.00)	(0.00)	(0.02)	(0.02)	(0.02)	(0.01)	(0.01)	(0.01)
log(med. HH income)	0.011	0.022	0.046	−0.083	−0.188	−0.526	−0.357	−0.470	−0.662
	(0.05)	(0.05)	(0.05)	(0.92)	(0.89)	(0.94)	(0.36)	(0.36)	(0.38)
% blue collar	−0.002	−0.002	−0.003	0.014	0.020	0.029	0.019	0.025*	0.029*
	(0.00)	(0.00)	(0.00)	(0.03)	(0.03)	(0.03)	(0.01)	(0.01)	(0.01)
% urban population	−0.001	−0.001	−0.002*	0.019	0.021	0.027	0.013*	0.012*	0.017**
	(0.00)	(0.00)	(0.00)	(0.02)	(0.02)	(0.02)	(0.01)	(0.01)	(0.01)
log(% w/out HS diploma)	−0.161***	−0.170***	−0.161***	3.292***	3.234***	3.249***	0.961***	1.047***	0.955***
	(0.03)	(0.03)	(0.03)	(0.55)	(0.57)	(0.57)	(0.22)	(0.23)	(0.23)
Lagged indicators									
SHP indicator	−0.014	−0.024***	0.000	0.023	0.315**	0.026	0.140*	0.193***	−0.033
	(0.01)	(0.01)	(0.00)	(0.16)	(0.11)	(0.07)	(0.06)	(0.05)	(0.03)
Median age	−0.011	−0.005	−0.011	0.169	0.092	0.187	0.068	0.040	0.072
	(0.01)	(0.01)	(0.01)	(0.11)	(0.11)	(0.12)	(0.04)	(0.05)	(0.05)
log(gov't ideology)	−0.017	−0.008	−0.023	0.201	0.242	0.217	0.182	0.048	0.215*
	(0.01)	(0.02)	(0.01)	(0.25)	(0.28)	(0.24)	(0.10)	(0.11)	(0.10)
log(% in poor health)	0.114	0.138	0.175*	−1.921	−1.755	−2.439	−0.982	−1.178*	−1.425
	(0.07)	(0.07)	(0.08)	(1.39)	(1.35)	(1.36)	(0.54)	(0.56)	(0.55)
log(% Black)	−0.018	−0.027	−0.050*	0.739	0.656	1.124**	−0.065	0.074	0.218
	(0.02)	(0.02)	(0.02)	(0.40)	(0.39)	(0.39)	(0.16)	(0.16)	(0.16)
D index	0.002	0.005	0.004	−0.028	−0.068	−0.037	−0.012	−0.034	−0.032
	(0.00)	(0.00)	(0.00)	(0.05)	(0.04)	(0.05)	(0.02)	(0.02)	(0.02)
log(med. HH income)	0.041	0.079	0.128	−2.116	−1.926	−2.524	0.654	0.347	−0.278
	(0.10)	(0.10)	(0.11)	(1.92)	(1.85)	(1.93)	(0.74)	(0.77)	(0.79)
% blue collar	0.002	−0.001	0.000	−0.047	−0.036	−0.046	−0.020	0.012	0.011
	(0.00)	(0.00)	(0.00)	(0.07)	(0.06)	(0.07)	(0.03)	(0.03)	(0.03)
% urban population	−0.003	−0.002	−0.005**	0.079*	0.050	0.084*	0.018	0.011	0.031*
	(0.00)	(0.00)	(0.00)	(0.03)	(0.03)	(0.03)	(0.01)	(0.01)	(0.01)
log(% w/out HS diploma)	0.142**	0.167**	0.093	−1.382	−1.911	−0.626	−1.278**	−1.359**	−0.948*
	(0.05)	(0.06)	(0.06)	(1.02)	(1.08)	(1.03)	(0.39)	(0.45)	(0.42)
ρ	0.266**	0.369***	0.328***	0.326***	0.360***	0.323***	0.273**	0.412***	0.365***
	(0.09)	(0.08)	(0.09)	(0.09)	(0.09)	(0.09)	(0.09)	(0.08)	(0.09)
RW test (p-value)	0.7013	0.2777	0.3017	0.8172	0.4735	0.0579	0.2018	0.1998	0.2138
BSJK (C.2) test	45***	34***	51***	49***	43***	50***	26***	20***	34***
Pseudo R^2	0.985	0.985	0.983	0.977	0.978	0.976	0.991	0.99	0.989
AIC	−392	−397	−375	773	763	783	400	404	419
BIC	−144	−149	−127	1020	1010	1031	647	651	666

$^{*}p < 0.05$, $^{**}p < 0.01$, $^{***}p < 0.001$.

Table A.2.19. Fully specified county group-level fixed effects SAR models estimating both-sex mortality indicators ($log(ASDR)$, $e(0)$, and $e(65)$) featuring various specifications of state health policy.

	$log(ASDR)$			$e(0)$			$e(65)$		
Variable	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$
SHP indicator	−0.006***	−0.005***	0.001	0.071***	0.069***	−0.006	0.050***	0.042***	−0.012**
	(0.00)	(0.00)	(0.00)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.00)
Median age†	−0.004***	−0.004***	−0.004***	0.046***	0.046***	0.047***	0.049***	0.049***	0.049***
	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
log(gov't ideology)*	−0.004*	0.004*	−0.007***	0.026	−0.066*	0.062*	0.055***	−0.028	0.079***
	(0.00)	(0.00)	(0.00)	(0.03)	(0.03)	(0.03)	(0.01)	(0.02)	(0.01)
log(% in poor health)	−0.071***	−0.072***	−0.066***	0.953***	0.971***	0.883***	0.633***	0.645***	0.593***
	(0.01)	(0.01)	(0.01)	(0.11)	(0.11)	(0.11)	(0.06)	(0.06)	(0.06)
log(% Black)	0.003***	0.002**	0.002**	−0.055***	−0.047***	−0.048***	−0.020**	−0.015*	−0.016*
	(0.00)	(0.00)	(0.00)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)
log(D index)	−0.006***	−0.006***	−0.006***	0.096***	0.101***	0.099***	0.037***	0.041***	0.040***
	(0.00)	(0.00)	(0.00)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)
log(med. HH income)	−0.091***	−0.094***	−0.088***	1.308***	1.341***	1.275***	0.712***	0.748***	0.686***
	(0.01)	(0.01)	(0.01)	(0.11)	(0.11)	(0.11)	(0.06)	(0.06)	(0.06)
log(GINI index)	−0.103***	−0.104***	−0.108***	1.328***	1.316***	1.396***	0.887***	0.899***	0.934***
	(0.01)	(0.01)	(0.01)	(0.18)	(0.18)	(0.18)	(0.10)	(0.10)	(0.10)
% blue collar	0.000*	0.000**	0.000**	0.006*	0.006*	0.006*	0.004**	0.004**	0.004**
	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
% urban population	−0.001***	−0.001***	−0.001***	0.012***	0.012***	0.012***	0.007***	0.007***	0.007***
	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
log(% w/out HS diploma)	−0.005	−0.003	−0.004	0.025	−0.004	0.010	0.082*	0.064	0.069*
	(0.00)	(0.00)	(0.00)	(0.06)	(0.06)	(0.06)	(0.03)	(0.03)	(0.03)
λ	0.627***	0.631***	0.638***	0.652***	0.651***	0.660***	0.608***	0.617***	0.619***
	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)
RW test (p-value)	0.002	0.002	0.002	0.002	0.002	0.002	0.002	0.002	0.002
BSJK (C.2) test	401***	1861***	3752***	3224***	811***	764***	2748***	458***	3733***
Pseudo R^2	0.937	0.937	0.937	0.939	0.939	0.939	0.939	0.939	0.939
AIC	−31027	−31027	−30989	22762	22749	22791	11082	11101	11120
BIC	−13116	−13115	−13077	40673	40660	40703	28993	29013	29031

$^{*}p < 0.05$, $^{**}p < 0.01$, $^{***}p < 0.001$.

Table A.2.20. Fully specified county group-level fixed effects SAR models estimating female mortality indicators ($log(ASDR)$, $e(0)$, and $e(65)$) featuring various specifications of state health policy.

	$log(ASDR)$			$e(0)$			$e(65)$		
Variable	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$
SHP indicator	−0.005***	−0.005***	0.001*	0.066***	0.060***	−0.011	0.046***	0.038***	−0.016***
	(0.00)	(0.00)	(0.00)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.00)
Median age†	−0.004***	−0.004***	−0.004***	0.046***	0.046***	0.047***	0.045***	0.045***	0.046***
	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
log(gov't ideology)*	−0.006***	0.004*	−0.009***	0.061*	−0.061*	0.094***	0.066***	−0.033	0.090***
	(0.00)	(0.00)	(0.00)	(0.02)	(0.03)	(0.02)	(0.02)	(0.02)	(0.01)
log(% in poor health)	−0.062***	−0.065***	−0.059***	0.791***	0.828***	0.747***	0.542***	0.569***	0.516***
	(0.01)	(0.01)	(0.01)	(0.10)	(0.10)	(0.10)	(0.06)	(0.06)	(0.06)
log(% Black)	0.003***	0.003**	0.003**	−0.047***	−0.040***	−0.041***	−0.028***	−0.024**	−0.024
	(0.00)	(0.00)	(0.00)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)
log(D index)	−0.006***	−0.006***	−0.006***	0.093***	0.097***	0.095***	0.036***	0.039***	0.038***
	(0.00)	(0.00)	(0.00)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)
log(med. HH income)	−0.073***	−0.077***	−0.071***	1.016***	1.060***	0.988***	0.558***	0.598***	0.534***
	(0.01)	(0.01)	(0.01)	(0.10)	(0.10)	(0.10)	(0.06)	(0.06)	(0.06)
log(GINI index)	−0.105***	−0.107***	−0.110***	1.358***	1.365***	1.417***	0.865***	0.884***	0.911***
	(0.01)	(0.01)	(0.01)	(0.17)	(0.17)	(0.17)	(0.11)	(0.11)	(0.11)
% blue collar	−0.001***	−0.001***	−0.001***	0.012***	0.012***	0.012***	0.007***	0.007***	0.007***
	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
% urban population	−0.001***	−0.001***	−0.001***	0.011***	0.011***	0.011***	0.007***	0.007***	0.007***
	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
log(% w/out HS diploma)	−0.008*	−0.006	−0.007	0.054	0.023	0.036	0.132***	0.111**	0.116***
	(0.00)	(0.00)	(0.00)	(0.05)	(0.05)	(0.05)	(0.03)	(0.03)	(0.03)
λ	0.655***	0.662***	0.664***	0.676***	0.679***	0.684***	0.633***	0.643***	0.640***
	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)
RW test (p-value)	0.002	0.002	0.002	0.002	0.002	0.002	0.002	0.002	0.002
BSJK (C.2) test	2274***	3138***	717***	3503***	3325***	3520***	3147***	764***	3228***
Pseudo R^2	0.923	0.923	0.923	0.927	0.927	0.927	0.92	0.92	0.92
AIC	−30631	−30616	−30601	21318	21320	21345	12167	12194	12188
BIC	−12720	−12705	−12690	39229	39231	39257	30078	30105	30100

$^{*}p < 0.05$, $^{**}p < 0.01$, $^{***}p < 0.001$.

Table A.2.21. Fully specified county group-level fixed effects SAR models estimating male mortality indicators ($log(ASDR)$, $e(0)$, and $e(65)$) featuring various specifications of state health policy.

	$log(ASDR)$			$e(0)$			$e(65)$		
Variable	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$
SHP indicator	−0.006***	−0.005***	0.000	0.072***	0.076***	0.001	0.050***	0.045***	−0.006
	(0.00)	(0.00)	(0.00)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.00)
Median age†	−0.003***	−0.003***	−0.003***	0.032***	0.032***	0.033***	0.039***	0.039***	0.040***
	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
log(gov't ideology)*	−0.003	0.003	−0.006***	0.005	−0.062	0.042	0.047	−0.015	0.071***
	(0.00)	(0.00)	(0.00)	(0.03)	(0.03)	(0.03)	(0.01)	(0.02)	(0.01)
log(% in poor health)	−0.054***	−0.054***	−0.049***	0.810***	0.809***	0.741***	0.439***	0.435***	0.398***
	(0.01)	(0.01)	(0.01)	(0.10)	(0.10)	(0.10)	(0.05)	(0.05)	(0.05)
log(% Black)	0.003**	0.002**	0.002**	−0.060***	−0.053***	−0.054***	−0.017*	−0.012	−0.013
	(0.00)	(0.00)	(0.00)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)
log(D index)	−0.007***	−0.007***	−0.007***	0.115***	0.121***	0.119***	0.043***	0.046***	0.045***
	(0.00)	(0.00)	(0.00)	(0.02)	(0.02)	(0.02)	(0.01)	(0.01)	(0.01)
log(med. HH income)	−0.088***	−0.091***	−0.086***	1.408***	1.433***	1.375***	0.652***	0.685***	0.628***
	(0.01)	(0.01)	(0.01)	(0.12)	(0.12)	(0.12)	(0.06)	(0.06)	(0.06)
log(GINI index)	−0.111***	−0.110***	−0.116***	1.318***	1.287***	1.389***	0.994***	1.001***	1.039***
	(0.01)	(0.01)	(0.01)	(0.20)	(0.19)	(0.19)	(0.10)	(0.10)	(0.10)
% blue collar	0.000	0.000	0.000	0.002	0.002	0.003	0.003**	0.004**	0.004**
	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
% urban population	−0.001***	−0.001***	−0.001***	0.012***	0.012***	0.013***	0.007***	0.007***	0.008***
	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
log(% w/out HS diploma)	−0.002	0.000	−0.001	0.035	0.010	0.022	0.012	−0.002	0.002
	(0.00)	(0.00)	(0.00)	(0.06)	(0.06)	(0.06)	(0.03)	(0.03)	(0.03)
λ	0.591***	0.593***	0.604***	0.626***	0.623***	0.634***	0.579***	0.584***	0.592***
	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)
RW test (p-value)	0.002	0.002	0.002	0.002	0.002	0.002	0.002	0.002	0.002
BSJK (C.2) test	3532***	3501***	3744***	3466***	3413***	642***	3598***	3622***	2025***
Pseudo R^2	0.951	0.951	0.951	0.948	0.948	0.948	0.958	0.958	0.958
AIC	−30590	−30601	−30548	24341	24320	24368	10704	10710	10750
BIC	−12679	−12690	−12637	42253	42231	42279	28615	28621	28662

$^{*}p < 0.05$, $^{**}p < 0.01$, $^{***}p < 0.001$.

Table A.2.22. Fully specified county group-level fixed effects SEM models estimating both-sex mortality indicators ($log(ASDR)$, $e(0)$, and $e(65)$) featuring various specifications of state health policy.

	$log(ASDR)$			$e(0)$			$e(65)$		
Variable	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$
SHP indicator	−0.011***	−0.011***	0.001	0.150***	0.145***	−0.006	0.098***	0.090***	−0.015
	(0.00)	(0.00)	(0.00)	(0.03)	(0.02)	(0.01)	(0.01)	(0.01)	(0.01)
Median age†	−0.005***	−0.005***	−0.005***	0.048***	0.048***	0.048***	0.053***	0.053***	0.054***
	(0.00)	(0.00)	(0.00)	(0.01)	(0.01)	(0.01)	(0.00)	(0.00)	(0.00)
log(gov't ideology)*	−0.013***	0.006	−0.019***	0.126*	−0.109	0.196***	0.138***	−0.023	0.185***
	(0.00)	(0.00)	(0.00)	(0.06)	(0.06)	(0.06)	(0.03)	(0.03)	(0.03)
log(% in poor health)	−0.075***	−0.077***	−0.072***	0.759***	0.789***	0.713***	0.778***	0.796***	0.752***
	(0.01)	(0.01)	(0.01)	(0.15)	(0.15)	(0.15)	(0.08)	(0.08)	(0.08)
log(% Black)	0.000	0.000	0.000	−0.012	−0.009	−0.009	−0.003	−0.001	−0.001
	(0.00)	(0.00)	(0.00)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)
log(D index)	−0.006***	−0.006***	−0.006***	0.087***	0.089***	0.089***	0.036***	0.037***	0.037***
	(0.00)	(0.00)	(0.00)	(0.02)	(0.02)	(0.02)	(0.01)	(0.01)	(0.01)
log(med. HH income)	−0.116***	−0.118***	−0.116***	1.785***	1.813***	1.786***	0.865***	0.893***	0.863***
	(0.01)	(0.01)	(0.01)	(0.13)	(0.13)	(0.13)	(0.07)	(0.07)	(0.07)
log(GINI index)	−0.086***	−0.085***	−0.087***	1.044***	1.035***	1.053***	0.774***	0.766***	0.782***
	(0.01)	(0.01)	(0.01)	(0.18)	(0.18)	(0.18)	(0.10)	(0.10)	(0.10)
% blue collar	0.000*	0.000*	0.000*	0.006*	0.006*	0.007*	0.004*	0.004*	0.004*
	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
% urban population	−0.001***	−0.001***	−0.001***	0.011***	0.010***	0.011***	0.006***	0.006***	0.006***
	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
log(% w/out HS diploma)	0.006	0.007	0.006	−0.125	−0.136	−0.131	−0.005	−0.014	−0.011
	(0.00)	(0.00)	(0.00)	(0.07)	(0.07)	(0.07)	(0.04)	(0.04)	(0.04)
ρ	0.656***	0.659***	0.667***	0.681***	0.680***	0.689***	0.637***	0.645***	0.649***
	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)
RW test (p-value)	0.002	0.002	0.002	0.002	0.002	0.002	0.002	0.002	0.002
BSJK (C.2) test	608***	597***	3752***	3655***	3557***	3737***	3557***	3467***	3733***
Pseudo R^2	0.904	0.903	0.901	0.902	0.902	0.899	0.91	0.909	0.908
AIC	31986	31989	32020	85737	85731	85763	74121	74137	74158
BIC	49897	49900	49932	103648	103642	103675	92033	92048	92070

$^{*}p < 0.05$, $^{**}p < 0.01$, $^{***}p < 0.001$.

Table A.2.23. Fully specified county group-level fixed effects SEM models estimating female mortality indicators ($log(ASDR)$, $e(0)$, and $e(65)$) featuring various specifications of state health policy.

	$log(ASDR)$			$e(0)$			$e(65)$		
Variable	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$
SHP indicator	−0.011***	−0.010***	0.002	0.148***	0.136***	−0.015	0.092***	0.087***	−0.022**
	(0.00)	(0.00)	(0.00)	(0.03)	(0.02)	(0.01)	(0.02)	(0.01)	(0.01)
Median age†	−0.005***	−0.005***	−0.005***	0.050***	0.050***	0.050***	0.050***	0.050***	0.050***
	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
log(gov't ideology)*	−0.017***	0.007	−0.023***	0.193***	−0.123*	0.262***	0.163***	−0.040	0.209***
	(0.00)	(0.00)	(0.00)	(0.06)	(0.06)	(0.06)	(0.03)	(0.04)	(0.03)
log(% in poor health)	−0.078***	−0.081***	−0.076***	0.891***	0.922***	0.856***	0.718***	0.743***	0.699***
	(0.01)	(0.01)	(0.01)	(0.13)	(0.13)	(0.13)	(0.08)	(0.08)	(0.08)
log(% Black)	0.001	0.000	0.000	−0.010	−0.007	−0.008	−0.009	−0.007	−0.007
	(0.00)	(0.00)	(0.00)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)
log(D index)	−0.005***	−0.005***	−0.005***	0.081***	0.083***	0.082***	0.032***	0.033***	0.033***
	(0.00)	(0.00)	(0.00)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)
log(med. HH income)	−0.099***	−0.102***	−0.100***	1.461***	1.489***	1.463***	0.714***	0.740***	0.712***
	(0.01)	(0.01)	(0.01)	(0.12)	(0.12)	(0.12)	(0.08)	(0.08)	(0.08)
log(GINI index)	−0.086***	−0.085***	−0.086***	1.060***	1.050***	1.065***	0.736***	0.729***	0.744***
	(0.01)	(0.01)	(0.01)	(0.17)	(0.17)	(0.17)	(0.11)	(0.11)	(0.11)
% blue collar	−0.001***	−0.001***	−0.001***	0.010***	0.010***	0.011***	0.006***	0.006***	0.006***
	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
% urban population	−0.001***	−0.001***	−0.001***	0.009***	0.009***	0.010***	0.006***	0.006***	0.006***
	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
log(% w/out HS diploma)	0.004	0.005	0.004	−0.110	−0.121	−0.116	0.028	0.018	0.022
	(0.00)	(0.00)	(0.00)	(0.06)	(0.06)	(0.06)	(0.04)	(0.04)	(0.04)
ρ	0.685***	0.691***	0.694***	0.704***	0.708***	0.712***	0.662***	0.670***	0.670***
	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)
RW test (p-value)	0.002	0.002	0.002	0.002	0.002	0.002	0.002	0.002	0.002
BSJK (C.2) test	3321***	3138***	645***	3503***	3325***	913***	3147***	3022***	493***
Pseudo R^2	0.873	0.871	0.87	0.875	0.873	0.871	0.874	0.872	0.872
AIC	32402	32417	32430	84330	84337	84357	75216	75236	75239
BIC	50314	50328	50342	102241	102248	102268	93127	93147	93151

$^{*}p < 0.05$, $^{**}p < 0.01$, $^{***}p < 0.001$.

Table A.2.24. Fully specified county group-level fixed effects SEM models estimating male mortality indicators ($log(ASDR)$, $e(0)$, and $e(65)$) featuring various specifications of state health policy.

	$log(ASDR)$			$e(0)$			$e(65)$		
Variable	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$
SHP indicator†	−0.011***	−0.011***	0.000	0.149***	0.153***	0.004	0.100***	0.093***	−0.004
	(0.00)	(0.00)	(0.00)	(0.03)	(0.02)	(0.02)	(0.01)	(0.01)	(0.01)
Median age	−0.004***	−0.004***	−0.004***	0.032***	0.032***	0.032***	0.042***	0.042***	0.041***
	(0.00)	(0.00)	(0.00)	(0.01)	(0.01)	(0.01)	(0.00)	(0.00)	(0.00)
log(gov't ideology)†	−0.010**	0.003	−0.015***	0.070	−0.089	0.139*	0.108***	0.004	0.154***
	(0.00)	(0.00)	(0.00)	(0.06)	(0.07)	(0.06)	(0.03)	(0.03)	(0.03)
log(% in poor health)	−0.051***	−0.052***	−0.047***	0.511***	0.529***	0.461	0.514***	0.518***	0.482***
	(0.01)	(0.01)	(0.01)	(0.14)	(0.14)	(0.14)	(0.07)	(0.07)	(0.07)
log(% Black)	0.001	0.000	0.000	−0.015	−0.011	−0.012	−0.003	0.000	−0.001
	(0.00)	(0.00)	(0.00)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)
log(D index)	−0.006***	−0.007***	−0.006***	0.107***	0.109***	0.109***	0.041***	0.042***	0.042***
	(0.00)	(0.00)	(0.00)	(0.02)	(0.02)	(0.02)	(0.01)	(0.01)	(0.01)
log(med. HH income)	−0.115***	−0.117***	−0.114***	1.953***	1.978***	1.954***	0.818***	0.847***	0.817***
	(0.01)	(0.01)	(0.01)	(0.14)	(0.14)	(0.14)	(0.07)	(0.07)	(0.07)
log(GINI index)	−0.094***	−0.093***	−0.095***	1.019***	1.011***	1.033***	0.880***	0.872***	0.887***
	(0.01)	(0.01)	(0.01)	(0.20)	(0.20)	(0.20)	(0.10)	(0.10)	(0.10)
% blue collar	0.000*	0.000*	0.000*	0.004	0.005	0.005	0.004**	0.004**	0.004**
	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
% urban population	−0.001***	−0.001***	−0.001***	0.011***	0.010***	0.011***	0.007***	0.006***	0.007***
	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
log(% w/out HS diploma)	0.006	0.007	0.006	−0.092	−0.101	−0.097	−0.046	−0.053	−0.051
	(0.00)	(0.00)	(0.00)	(0.07)	(0.07)	(0.07)	(0.04)	(0.04)	(0.04)
ρ	0.615***	0.616***	0.629***	0.653***	0.650***	0.660***	0.602***	0.607***	0.618***
	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)
RW test (p-value)	0.002	0.002	0.002	0.002	0.002	0.002	0.002	0.002	0.002
BSJK (C.2) test	906***	3501***	1274***	3466***	3413***	439***	3598***	3622***	431***
Pseudo R^2	0.93	0.93	0.928	0.921	0.921	0.919	0.941	0.941	0.94
AIC	32404	32394	32443	87280	87265	87305	73738	73740	73786
BIC	50315	50305	50355	105191	105176	105216	91649	91651	91697

$^*p < 0.05$, $^{**}p < 0.01$, $^{***}p < 0.001$.

Table A.2.25. Fully specified county group-level fixed effects SLX models estimating both-sex mortality indicators ($log(ASDR)$, $e(0)$, and $e(65)$) featuring various specifications of state health policy.

	$log(ASDR)$			$e(0)$			$e(65)$		
Variable	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$
SHP indicator†	−0.001	−0.004	−0.002	0.057	0.057	0.025	−0.004	0.026	0.020
	(0.00)	(0.00)	(0.00)	(0.06)	(0.04)	(0.03)	(0.03)	(0.02)	(0.01)
Median age	−0.005***	−0.005***	−0.005***	0.045***	0.046***	0.046***	0.052***	0.053***	0.052***
	(0.00)	(0.00)	(0.00)	(0.01)	(0.01)	(0.01)	(0.00)	(0.00)	(0.00)
log(gov't ideology)†	−0.006	0.001	−0.006	0.060	−0.060	0.068	0.043	0.044	0.032
	(0.01)	(0.01)	(0.01)	(0.12)	(0.12)	(0.12)	(0.06)	(0.07)	(0.06)
log(% in poor health)	−0.061***	−0.061***	−0.056***	0.590**	0.597**	0.524*	0.659***	0.659***	0.624***
	(0.01)	(0.01)	(0.01)	(0.21)	(0.21)	(0.22)	(0.12)	(0.12)	(0.12)
log(% Black)	0.003*	0.002	0.002	−0.055**	−0.049**	−0.048	−0.017	−0.014	−0.012
	(0.00)	(0.00)	(0.00)	(0.02)	(0.02)	(0.02)	(0.01)	(0.01)	(0.01)
log(D index)	−0.007***	−0.008***	−0.007***	0.117***	0.123***	0.121***	0.043***	0.047***	0.045***
	(0.00)	(0.00)	(0.00)	(0.02)	(0.02)	(0.02)	(0.01)	(0.01)	(0.01)
log(med. HH income)	−0.125***	−0.127***	−0.124***	1.930***	1.944***	1.911***	0.946***	0.963***	0.932***
	(0.01)	(0.01)	(0.01)	(0.19)	(0.19)	(0.19)	(0.10)	(0.10)	(0.10)
log(GINI index)	−0.123***	−0.126***	−0.130***	1.693***	1.696***	1.791***	1.003***	1.050***	1.064***
	(0.02)	(0.02)	(0.02)	(0.26)	(0.26)	(0.26)	(0.14)	(0.14)	(0.14)
% blue collar	−0.001**	−0.001**	−0.001**	0.012**	0.012**	0.012**	0.006**	0.006**	0.006**
	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
% urban population	−0.001***	−0.001***	−0.001***	0.012***	0.012***	0.013***	0.008***	0.008***	0.008***
	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
log(% w/out HS diploma)	0.000	0.001	0.000	−0.036	−0.057	−0.040	0.033	0.022	0.029
	(0.01)	(0.01)	(0.01)	(0.10)	(0.10)	(0.10)	(0.05)	(0.05)	(0.05)
Lagged indicators									
SHP indicator†	−0.015***	−0.007*	0.005**	0.170**	0.109*	−0.058	0.140***	0.065*	−0.060***
	(0.00)	(0.00)	(0.00)	(0.06)	(0.05)	(0.03)	(0.03)	(0.03)	(0.02)
Median age	−0.004***	−0.005***	−0.005***	0.053***	0.055***	0.059***	0.043***	0.048***	0.047***
	(0.00)	(0.00)	(0.00)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)
log(gov't ideology)†	−0.006	0.007	−0.015	0.027	−0.065	0.137	0.118	−0.107	0.201**
	(0.01)	(0.01)	(0.01)	(0.13)	(0.13)	(0.12)	(0.07)	(0.07)	(0.07)
log(% in poor health)	−0.088***	−0.088***	−0.068***	2.110***	2.075***	1.818***	0.368*	0.384*	0.213
	(0.02)	(0.02)	(0.02)	(0.29)	(0.29)	(0.29)	(0.16)	(0.16)	(0.16)
log(% Black)	0.014***	0.011***	0.010***	−0.286***	−0.243***	−0.233***	−0.080***	−0.057**	−0.050*
	(0.00)	(0.00)	(0.00)	(0.04)	(0.04)	(0.04)	(0.02)	(0.02)	(0.02)
log(D index)	−0.010***	−0.013***	−0.011***	0.220***	0.257***	0.235***	0.036	0.061*	0.044
	(0.00)	(0.00)	(0.00)	(0.04)	(0.04)	(0.04)	(0.02)	(0.02)	(0.02)
log(med. HH income)	−0.006	−0.019	0.007	−0.120	−0.036	−0.282	0.076	0.241	−0.041
	(0.02)	(0.02)	(0.02)	(0.28)	(0.27)	(0.28)	(0.15)	(0.15)	(0.15)
log(GINI index)	−0.392***	−0.420***	−0.448***	6.187***	6.298***	6.961***	2.832***	3.196***	3.298***
	(0.03)	(0.03)	(0.03)	(0.52)	(0.52)	(0.52)	(0.28)	(0.28)	(0.28)
% blue collar	−0.002***	−0.002***	−0.003***	0.031***	0.032***	0.034***	0.019***	0.021***	0.021***
	(0.00)	(0.00)	(0.00)	(0.01)	(0.01)	(0.01)	(0.00)	(0.00)	(0.00)
% urban population	−0.001***	−0.001***	−0.002***	0.019***	0.020***	0.023***	0.014***	0.014***	0.016***
	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
log(% w/out HS diploma)	−0.059***	−0.055***	−0.058***	0.886***	0.807***	0.885***	0.496***	0.473***	0.474***
	(0.01)	(0.01)	(0.01)	(0.15)	(0.16)	(0.16)	(0.08)	(0.09)	(0.09)
Joint Wald test	2157***	2025***	1978***	1923***	1875***	1769***	2288***	2082***	2141***
df	22	22	22	22	22	22	22	22	22
Wooldridge test	1001.94***	948.32***	986.79***	1165.79***	1109.77***	1156.6***	751.51***	711.87***	730.11***
Hausman test	679***	670***	667***	726***	718***	717***	654***	646***	636***
LM Breusch-Pagan test	2406***	3448***	1322***	3499***	18147***	2760***	1651***	2177***	2301***
Individual eff. F test	7.28***	6.84***	7.45***	7.26***	6.96***	7.35***	7.59***	7.04***	7.85***
Time eff. F test	633.65***	545.09***	617.07***	555.04***	482.51***	538.84***	711.77***	612.32***	703.91***
Two-way eff. F test	8.55***	8.14***	8.55***	8.36***	8.11***	8.31***	9.29***	8.69***	9.4***
R^2 (overall)	0.226	0.215	0.211	0.206	0.202	0.193	0.236	0.220	0.225
Adj. R^2 (within)	−0.035	−0.05	−0.055	−0.061	−0.067	−0.079	−0.021	−0.044	−0.037
AIC	−28684	−28546	−28497	25344	25395	25509	13286	13499	13438
BIC	−10708	−10570	−10521	43320	43371	43485	31262	31475	31414

$^{*}p < 0.05$, $^{**}p < 0.01$, $^{***}p < 0.001$.

Table A.2.26. Fully specified county group-level fixed effects SLX models estimating female mortality indicators ($log(ASDR)$, $e(0)$, and $e(65)$) featuring various specifications of state health policy.

	$log(ASDR)$			$e(0)$			$e(65)$		
Variable	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$
SHP indicator	−0.001	−0.003	−0.002	0.037	0.046	0.024	−0.009	0.020	0.015
	(0.00)	(0.00)	(0.00)	(0.05)	(0.04)	(0.03)	(0.03)	(0.02)	(0.02)
Median age	−0.005***	−0.005***	−0.005***	0.052***	0.052***	0.052***	0.051***	0.051***	0.051***
	(0.00)	(0.00)	(0.00)	(0.01)	(0.01)	(0.01)	(0.00)	(0.00)	(0.00)
log(gov't ideology)	−0.008	0.005	−0.007	0.083	−0.120	0.084	0.054	0.019	0.043
	(0.01)	(0.01)	(0.01)	(0.11)	(0.12)	(0.11)	(0.07)	(0.07)	(0.07)
log(% in poor health)	−0.069***	−0.069***	−0.065***	0.781***	0.789***	0.732***	0.629***	0.630***	0.603***
	(0.01)	(0.01)	(0.01)	(0.19)	(0.19)	(0.19)	(0.12)	(0.12)	(0.12)
log(% Black)	0.003*	0.003*	0.003*	−0.049**	−0.044*	−0.043*	−0.027*	−0.025*	−0.023*
	(0.00)	(0.00)	(0.00)	(0.02)	(0.02)	(0.02)	(0.01)	(0.01)	(0.01)
log(D index)	−0.007***	−0.008***	−0.008***	0.114***	0.121***	0.118***	0.045***	0.049***	0.046***
	(0.00)	(0.00)	(0.00)	(0.02)	(0.02)	(0.02)	(0.01)	(0.01)	(0.01)
log(med. HH income)	−0.106***	−0.108***	−0.105***	1.539***	1.560***	1.526***	0.772***	0.791***	0.760***
	(0.01)	(0.01)	(0.01)	(0.18)	(0.18)	(0.18)	(0.11)	(0.11)	(0.11)
log(GINI index)	−0.129***	−0.134***	−0.136***	1.752***	1.801***	1.846***	1.001***	1.065***	1.062***
	(0.02)	(0.02)	(0.02)	(0.24)	(0.24)	(0.24)	(0.15)	(0.15)	(0.15)
% blue collar	−0.001***	−0.001***	−0.001***	0.018***	0.018***	0.018***	0.009***	0.009***	0.009***
	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
% urban population	−0.001***	−0.001***	−0.001***	0.012***	0.012***	0.012***	0.007***	0.007***	0.008***
	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
log(% w/out HS diploma)	−0.002	−0.001	−0.002	−0.018	−0.038	−0.024	0.070	0.059	0.065
	(0.01)	(0.01)	(0.01)	(0.09)	(0.09)	(0.09)	(0.06)	(0.06)	(0.06)
Lagged indicators									
SHP indicator	−0.016***	−0.007*	0.006**	0.183**	0.103*	−0.071*	0.148***	0.065*	−0.066***
	(0.00)	(0.00)	(0.00)	(0.06)	(0.04)	(0.03)	(0.04)	(0.03)	(0.02)
Median age	−0.004***	−0.005***	−0.005***	0.053***	0.058***	0.059***	0.041***	0.047***	0.045***
	(0.00)	(0.00)	(0.00)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)
log(gov't ideology)	−0.012	0.002	−0.021*	0.127	0.019	0.241*	0.149*	−0.086	0.234**
	(0.01)	(0.01)	(0.01)	(0.12)	(0.13)	(0.12)	(0.07)	(0.08)	(0.07)
log(% in poor health)	−0.027	−0.036	−0.012	0.785**	0.888***	0.579*	0.081	0.171	−0.038
	(0.02)	(0.02)	(0.02)	(0.26)	(0.26)	(0.26)	(0.16)	(0.16)	(0.16)
log(% Black)	0.015***	0.012***	0.011***	−0.232***	−0.197***	−0.183***	−0.118***	−0.097***	−0.089***
	(0.00)	(0.00)	(0.00)	(0.03)	(0.03)	(0.03)	(0.02)	(0.02)	(0.02)
log(D index)	−0.013***	−0.016***	−0.014***	0.224***	0.263***	0.239***	0.063*	0.091***	0.072**
	(0.00)	(0.00)	(0.00)	(0.04)	(0.04)	(0.04)	(0.03)	(0.03)	(0.03)
log(med. HH income)	0.014	−0.007	0.026	−0.355	−0.125	−0.501	−0.085	0.131	−0.200
	(0.02)	(0.02)	(0.02)	(0.26)	(0.26)	(0.26)	(0.16)	(0.16)	(0.16)
log(GINI index)	−0.428***	−0.473***	−0.483***	6.423***	6.851***	7.148***	3.004***	3.487***	3.466***
	(0.04)	(0.04)	(0.04)	(0.49)	(0.50)	(0.49)	(0.30)	(0.31)	(0.30)
% blue collar	−0.004***	−0.004***	−0.004***	0.053***	0.055***	0.055***	0.028***	0.030***	0.029***
	(0.00)	(0.00)	(0.00)	(0.01)	(0.01)	(0.01)	(0.00)	(0.00)	(0.00)
% urban population	−0.002***	−0.002***	−0.002***	0.020***	0.021***	0.023***	0.015***	0.016***	0.017***
	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
log(% w/out HS diploma)	−0.080***	−0.075***	−0.077***	1.050***	0.975***	1.025***	0.733***	0.700***	0.697***
	(0.01)	(0.01)	(0.01)	(0.15)	(0.15)	(0.15)	(0.09)	(0.09)	(0.09)
Joint Wald test	2411***	2195***	2256***	2294***	2153***	2142***	2351***	2090***	2245***
df	22	22	22	22	22	22	22	22	22
Wooldridge test	922.84***	872.69***	876.91***	1043.04***	981.42***	1001***	726.52***	689.27***	682.06***
Hausman test	647***	635***	628***	685***	666***	668***	616***	613***	595***
LM Breusch-Pagan test	1700***	2160***	2623***	1843***	2443***	4651***	1314***	1421***	1376***
Individual eff. F test	6.7***	6.28***	6.87***	6.6***	6.28***	6.72***	6.92***	6.47***	7.17***
Time eff. F test	482.64***	429.86***	488.2***	384.12***	341.18***	385.66***	591.61***	528.89***	606.51***
Two-way eff. F test	7.72***	7.32***	7.77***	7.33***	7.07***	7.34***	8.46***	7.9***	8.59***
R^2 (overall)	0.246	0.229	0.234	0.237	0.226	0.225	0.241	0.22	0.233
Adj. R^2 (within)	−0.009	−0.031	−0.025	−0.021	−0.036	−0.037	−0.015	−0.043	−0.026
AIC	−27898	−27676	−27739	24334	24478	24491	14647	14916	14755
BIC	−9922	−9700	−9763	42310	42455	42467	32623	32892	32731

$^{*}p < 0.05$, $^{**}p < 0.01$, $^{***}p < 0.001$.

Table A.2.27. Fully specified county group-level fixed effects SLX models estimating male mortality indicators ($log(ASDR)$, $e(0)$, and $e(65)$) featuring various specifications of state health policy.

	$log(ASDR)$			$e(0)$			$e(65)$		
Variable	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$
SHP indicator	−0.002	−0.005	−0.002	0.080	0.065	0.021	0.008	0.034	0.023
	(0.00)	(0.00)	(0.00)	(0.06)	(0.04)	(0.03)	(0.03)	(0.02)	(0.01)
Median age	−0.003***	−0.003***	−0.003***	0.028***	0.028***	0.028***	0.040***	0.040***	0.040***
	(0.00)	(0.00)	(0.00)	(0.01)	(0.01)	(0.01)	(0.00)	(0.00)	(0.00)
log(gov't ideology)	−0.005	−0.004	−0.004	0.040	−0.005	0.060	0.024	0.074	0.016
	(0.01)	(0.01)	(0.01)	(0.12)	(0.13)	(0.12)	(0.06)	(0.06)	(0.06)
log(% in poor health)	−0.029*	−0.030*	−0.026*	0.180	0.187	0.133	0.369***	0.372***	0.345***
	(0.01)	(0.01)	(0.01)	(0.20)	(0.20)	(0.20)	(0.10)	(0.10)	(0.10)
log(% Black)	0.002*	0.002	0.002	−0.056**	−0.049*	−0.049*	−0.013	−0.010	−0.010
	(0.00)	(0.00)	(0.00)	(0.02)	(0.02)	(0.02)	(0.01)	(0.01)	(0.01)
log(D index)	−0.008***	−0.008***	−0.008***	0.137***	0.143***	0.141***	0.048***	0.051***	0.049***
	(0.00)	(0.00)	(0.00)	(0.02)	(0.02)	(0.02)	(0.01)	(0.01)	(0.01)
log(med. HH income)	−0.127***	−0.129***	−0.126***	2.138***	2.150***	2.124***	0.936***	0.952***	0.925***
	(0.01)	(0.01)	(0.01)	(0.20)	(0.20)	(0.20)	(0.10)	(0.10)	(0.10)
log(GINI index)	−0.125***	−0.126***	−0.131***	1.634***	1.609***	1.722***	1.080***	1.111***	1.131***
	(0.02)	(0.02)	(0.02)	(0.27)	(0.27)	(0.27)	(0.13)	(0.13)	(0.13)
% blue collar	−0.001*	−0.001*	−0.001*	0.009*	0.009*	0.010*	0.006**	0.006**	0.006**
	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
% urban population	−0.001***	−0.001***	−0.001***	0.012***	0.012***	0.012***	0.008***	0.008***	0.008***
	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
log(% w/out HS diploma)	0.000	0.001	0.000	0.020	0.001	0.014	−0.001	−0.010	−0.005
	(0.01)	(0.01)	(0.01)	(0.10)	(0.10)	(0.10)	(0.05)	(0.05)	(0.05)
Lagged indicators									
SHP indicator	−0.013**	−0.006*	0.004*	0.132*	0.102*	−0.034	0.108***	0.054*	−0.046**
	(0.00)	(0.00)	(0.00)	(0.07)	(0.05)	(0.03)	(0.03)	(0.02)	(0.02)
Median age	−0.003***	−0.003***	−0.003***	0.033*	0.034**	0.039**	0.037***	0.041***	0.040***
	(0.00)	(0.00)	(0.00)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)
log(gov't ideology)	−0.004	0.011	−0.012	−0.016	−0.114	0.072	0.110	−0.117	0.177**
	(0.01)	(0.01)	(0.01)	(0.13)	(0.14)	(0.13)	(0.07)	(0.07)	(0.07)
log(% in poor health)	−0.098***	−0.091***	−0.080***	2.412***	2.306***	2.155***	0.408**	0.369**	0.280*
	(0.02)	(0.02)	(0.02)	(0.26)	(0.26)	(0.27)	(0.13)	(0.13)	(0.13)
log(% Black)	0.012***	0.010***	0.009***	−0.308***	−0.268***	−0.262***	−0.056**	−0.037*	−0.032
	(0.00)	(0.00)	(0.00)	(0.04)	(0.04)	(0.04)	(0.02)	(0.02)	(0.02)
log(D index)	−0.011***	−0.013***	−0.011***	0.230***	0.264***	0.244***	0.039	0.061**	0.047*
	(0.00)	(0.00)	(0.00)	(0.05)	(0.05)	(0.05)	(0.02)	(0.02)	(0.02)
log(med. HH income)	0.005	−0.002	0.016	−0.211	−0.212	−0.361	−0.060	0.060	−0.157
	(0.02)	(0.02)	(0.02)	(0.29)	(0.29)	(0.30)	(0.14)	(0.14)	(0.15)
log(GINI index)	−0.377***	−0.393***	−0.428***	6.138***	6.048***	6.858***	2.875***	3.123***	3.274***
	(0.03)	(0.03)	(0.03)	(0.55)	(0.55)	(0.55)	(0.27)	(0.27)	(0.27)
% blue collar	−0.001**	−0.001***	−0.001***	0.012	0.014*	0.016*	0.011**	0.013***	0.012***
	(0.00)	(0.00)	(0.00)	(0.01)	(0.01)	(0.01)	(0.00)	(0.00)	(0.00)
% urban population	−0.001***	−0.001***	−0.002***	0.019***	0.019***	0.022***	0.014***	0.014***	0.015***
	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
log(% w/out HS diploma)	−0.034***	−0.031**	−0.033	0.782***	0.703***	0.777***	0.201*	0.186*	0.183*
	(0.01)	(0.01)	(0.01)	(0.16)	(0.16)	(0.16)	(0.08)	(0.08)	(0.08)
Joint Wald test	1708***	1648***	1555***	1525***	1524***	1403***	1948***	1836***	1812***
df	22	22	22	22	22	22	22	22	22
Wooldridge test	979.21***	946.76***	1001.72***	1149.91***	1135.52***	1174.7***	714.64***	679.63***	727.59***
Hausman test	681***	676***	684***	716***	713***	719***	665***	660***	665***
LM Breusch-Pagan test	2483***	17462***	2018***	2670***	1569***	2526***	1437***	3187***	525***
Individual eff. F test	7.94***	7.48***	8.16***	7.94***	7.66***	8.06***	8.37***	7.79***	8.73***
Time eff. F test	733.91***	617.14***	680.19***	627.8***	542.79***	586.69***	798.35***	665.21***	746.29***
Two-way eff. F test	10.03***	9.57***	10.07***	9.8***	9.54***	9.77***	10.99***	10.33***	11.17***
R^2 (overall)	0.188	0.182	0.174	0.171	0.171	0.16	0.209	0.199	0.197
Adj. R^2 (within)	−0.087	−0.094	−0.105	−0.109	−0.109	−0.124	−0.059	−0.072	−0.074
AIC	−28693	−28629	−28526	26531	26533	26668	12527	12646	12672
BIC	−10717	−10653	−10550	44507	44509	44644	30504	30622	30648

$^{*}p < 0.05$, $^{**}p < 0.01$, $^{***}p < 0.001$.

Table A.2.28. Fully specified county group-level fixed effects SDM models estimating female mortality indicators ($log(ASDR)$, $e(0)$, and $e(65)$) featuring various specifications of state health policy.

	$log(ASDR)$			$e(0)$			$e(65)$		
Variable	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$
SHP indicator	−0.001	−0.004*	−0.001	0.043	0.058*	0.008	0.001	0.031	0.005
	(0.00)	(0.00)	(0.00)	(0.04)	(0.03)	(0.02)	(0.02)	(0.02)	(0.01)
Median age	−0.004***	−0.004***	−0.004***	0.046***	0.047***	0.046***	0.047***	0.047***	0.047***
	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
log(gov't ideology)	−0.010	0.006	−0.011	0.122	−0.127	0.135	0.079	0.003	0.078
	(0.01)	(0.01)	(0.01)	(0.08)	(0.08)	(0.08)	(0.05)	(0.05)	(0.05)
log(% in poor health)	−0.066***	−0.067***	−0.065***	0.698***	0.709***	0.679***	0.625***	0.630***	0.615***
	(0.01)	(0.01)	(0.01)	(0.13)	(0.13)	(0.13)	(0.08)	(0.08)	(0.08)
log(% Black)	0.001	0.001	0.001	−0.020	−0.018	−0.017	−0.012	−0.011	−0.011
	(0.00)	(0.00)	(0.00)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)
log(D index)	−0.006***	−0.006***	−0.006***	0.091***	0.093***	0.092***	0.037***	0.038***	0.037***
	(0.00)	(0.00)	(0.00)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)
log(med. HH income)	−0.105***	−0.106***	−0.105***	1.537***	1.542***	1.535***	0.769***	0.775***	0.765***
	(0.01)	(0.01)	(0.01)	(0.12)	(0.12)	(0.12)	(0.08)	(0.08)	(0.08)
log(GINI index)	−0.092***	−0.093***	−0.094***	1.176***	1.178***	1.198***	0.765***	0.779***	0.785***
	(0.01)	(0.01)	(0.01)	(0.17)	(0.17)	(0.17)	(0.11)	(0.11)	(0.11)
% blue collar	−0.001***	−0.001***	−0.001***	0.011***	0.011***	0.011***	0.006***	0.006***	0.006***
	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
% urban population	−0.001***	−0.001***	−0.001***	0.009***	0.009***	0.010***	0.006***	0.006***	0.006***
	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
log(% w/out HS diploma)	0.003	0.004	0.004	−0.098	−0.105	−0.099	0.022	0.016	0.019
	(0.00)	(0.00)	(0.00)	(0.06)	(0.06)	(0.06)	(0.04)	(0.04)	(0.04)
Lagged indicators									
SHP indicator	−0.004	0.001	0.002	0.025	−0.015	−0.024	0.050	−0.003	−0.026*
	(0.00)	(0.00)	(0.00)	(0.04)	(0.03)	(0.02)	(0.03)	(0.02)	(0.01)
Median age	0.001*	0.001	0.001	−0.010	−0.009	−0.009	−0.011*	−0.010*	−0.010*
	(0.00)	(0.00)	(0.00)	(0.01)	(0.01)	(0.01)	(0.00)	(0.00)	(0.00)
log(gov't ideology)	0.005	−0.003	0.002	−0.070	0.088	−0.050	−0.011	−0.033	0.016
	(0.01)	(0.01)	(0.01)	(0.08)	(0.09)	(0.08)	(0.05)	(0.06)	(0.05)
log(% in poor health)	0.024	0.023	0.030*	−0.037	−0.028	−0.105	−0.296*	−0.278*	−0.341**
	(0.01)	(0.01)	(0.01)	(0.18)	(0.18)	(0.18)	(0.12)	(0.12)	(0.12)
log(% Black)	0.006***	0.005**	0.005**	−0.094***	−0.082***	−0.078	−0.053***	−0.044**	−0.042**
	(0.00)	(0.00)	(0.00)	(0.02)	(0.02)	(0.02)	(0.02)	(0.02)	(0.02)
log(D index)	−0.003	−0.004	−0.003	0.051	0.060*	0.053	0.017	0.025	0.019
	(0.00)	(0.00)	(0.00)	(0.03)	(0.03)	(0.03)	(0.02)	(0.02)	(0.02)
log(med. HH income)	0.066***	0.061***	0.071***	−1.074***	−1.021***	−1.129***	−0.441***	−0.378**	−0.488***
	(0.01)	(0.01)	(0.01)	(0.18)	(0.18)	(0.18)	(0.12)	(0.12)	(0.12)
log(GINI index)	−0.142***	−0.152***	−0.157***	2.085***	2.174***	2.259***	1.006***	1.134***	1.155***
	(0.03)	(0.03)	(0.03)	(0.35)	(0.35)	(0.35)	(0.22)	(0.22)	(0.22)
% blue collar	−0.001***	−0.001***	−0.001***	0.016***	0.017***	0.017***	0.010***	0.010***	0.010***
	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
% urban population	0.000**	0.000**	0.000***	0.004*	0.004*	0.004**	0.004***	0.004***	0.005***
	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
log(% w/out HS diploma)	−0.037***	−0.034***	−0.035***	0.492***	0.463***	0.476***	0.315***	0.293***	0.296***
	(0.01)	(0.01)	(0.01)	(0.10)	(0.10)	(0.10)	(0.07)	(0.07)	(0.07)
λ	0.627***	0.637***	0.634***	0.646***	0.652***	0.652***	0.607***	0.620***	0.612***
	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)
RW test (p-value)	0.002	0.002	0.002	0.002	0.002	0.002	0.002	0.002	0.002
BSJK (C.2) test	1188***	2963***	3270***	1276***	1644***	3401***	3109***	1699***	3152***
Pseudo R^2	0.924	0.924	0.924	0.928	0.928	0.928	0.92	0.92	0.92
AIC	−30812	−30779	−30787	21106	21124	21129	12021	12067	12038
BIC	−12821	−12788	−12796	39096	39114	39119	30011	30058	30028

$^{*}p < 0.05$, $^{**}p < 0.01$, $^{***}p < 0.001$.

Table A.2.29. Fully specified county group-level fixed effects SDM models estimating male mortality indicators ($log(ASDR)$, $e(0)$, and $e(65)$) featuring various specifications of state health policy.

	$log(ASDR)$			$e(0)$			$e(65)$		
Variable	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$
SHP indicator	−0.002	−0.005**	−0.002	0.075	0.074*	0.013	0.013	0.041*	0.017
	(0.00)	(0.00)	(0.00)	(0.05)	(0.03)	(0.02)	(0.02)	(0.02)	(0.01)
Median age	−0.003***	−0.003***	−0.003***	0.026***	0.026***	0.026***	0.038***	0.038***	0.038***
	(0.00)	(0.00)	(0.00)	(0.01)	(0.01)	(0.01)	(0.00)	(0.00)	(0.00)
log(gov't ideology)	−0.007	−0.003	−0.007	0.068	−0.018	0.092	0.041	0.068	0.040
	(0.01)	(0.01)	(0.01)	(0.09)	(0.10)	(0.09)	(0.05)	(0.05)	(0.05)
log(% in poor health)	−0.022*	−0.022*	−0.020*	−0.020	−0.015	−0.041	0.339***	0.341***	0.329***
	(0.01)	(0.01)	(0.01)	(0.15)	(0.15)	(0.15)	(0.08)	(0.07)	(0.08)
log(% Black)	0.001	0.001	0.001	−0.022	−0.019	−0.020	−0.006	−0.004	−0.004
	(0.00)	(0.00)	(0.00)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)
log(D index)	−0.007***	−0.007***	−0.007***	0.118***	0.120***	0.119***	0.043***	0.044***	0.043***
	(0.00)	(0.00)	(0.00)	(0.02)	(0.02)	(0.02)	(0.01)	(0.01)	(0.01)
log(med. HH income)	−0.124***	−0.124***	−0.123***	2.088***	2.092***	2.086***	0.905***	0.912***	0.901***
	(0.01)	(0.01)	(0.01)	(0.15)	(0.15)	(0.15)	(0.07)	(0.07)	(0.07)
log(GINI index)	−0.096***	−0.095***	−0.097***	1.120***	1.101***	1.143***	0.865***	0.871***	0.881***
	(0.01)	(0.01)	(0.01)	(0.20)	(0.20)	(0.20)	(0.10)	(0.10)	(0.10)
% blue collar	0.000*	0.000*	0.000*	0.007*	0.007*	0.007*	0.004**	0.004**	0.004**
	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
% urban population	−0.001***	−0.001***	−0.001***	0.010***	0.010***	0.010***	0.006***	0.006***	0.006***
	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
log(% w/out HS diploma)	0.003	0.003	0.003	−0.048	−0.054	−0.050	−0.018	−0.022	−0.020
	(0.00)	(0.00)	(0.00)	(0.08)	(0.08)	(0.08)	(0.04)	(0.04)	(0.04)
Lagged indicators									
SHP indicator	−0.004	0.001	0.002	0.000	−0.017	−0.018	0.035	−0.006	−0.027*
	(0.00)	(0.00)	(0.00)	(0.05)	(0.04)	(0.02)	(0.02)	(0.02)	(0.01)
Median age	0.000	0.000	0.000	−0.003	−0.003	−0.001	−0.004	−0.003	−0.004
	(0.00)	(0.00)	(0.00)	(0.01)	(0.01)	(0.01)	(0.00)	(0.00)	(0.00)
log(gov't ideology)	0.004	0.007	0.001	−0.083	−0.036	−0.070	0.008	−0.094	0.033
	(0.01)	(0.01)	(0.01)	(0.10)	(0.11)	(0.10)	(0.05)	(0.05)	(0.05)
log(% in poor health)	−0.041***	−0.038**	−0.033**	1.221***	1.175***	1.117***	0.056	0.033	−0.001
	(0.01)	(0.01)	(0.01)	(0.20)	(0.20)	(0.20)	(0.10)	(0.10)	(0.10)
log(% Black)	0.006***	0.005**	0.005**	−0.137***	−0.122***	−0.119***	−0.032*	−0.023	−0.022
	(0.00)	(0.00)	(0.00)	(0.03)	(0.03)	(0.03)	(0.01)	(0.01)	(0.01)
log(D index)	−0.003	−0.003	−0.003	0.057	0.069*	0.060	0.007	0.016	0.010
	(0.00)	(0.00)	(0.00)	(0.03)	(0.03)	(0.03)	(0.02)	(0.02)	(0.02)
log(med. HH income)	0.067***	0.066***	0.072***	−1.278***	−1.303***	−1.346***	−0.475***	−0.440***	−0.524***
	(0.01)	(0.01)	(0.01)	(0.22)	(0.22)	(0.22)	(0.11)	(0.11)	(0.11)
log(GINI index)	−0.140***	−0.143***	−0.155***	2.311***	2.248***	2.514***	1.006***	1.075***	1.137***
	(0.03)	(0.03)	(0.03)	(0.41)	(0.41)	(0.41)	(0.21)	(0.21)	(0.21)
% blue collar	0.000	−0.001	−0.001	0.004	0.005	0.005	0.004	0.005	0.005
	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
% urban population	0.000**	0.000**	0.000***	0.004*	0.004*	0.005*	0.004***	0.004***	0.005***
	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
log(% w/out HS diploma)	−0.019*	−0.017*	−0.018*	0.385**	0.350**	0.376**	0.110	0.100	0.099
	(0.01)	(0.01)	(0.01)	(0.12)	(0.12)	(0.12)	(0.06)	(0.06)	(0.06)
λ	0.566***	0.570***	0.576***	0.596***	0.596***	0.603***	0.555***	0.563***	0.564***
	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)
RW test (p-value)	0.002	0.002	0.002	0.002	0.002	0.002	0.002	0.002	0.002
BSJK (C.2) test	1291***	3508***	3809***	3588***	3485***	3692***	3056***	1518***	3947***
Pseudo R^2	0.951	0.951	0.951	0.949	0.949	0.949	0.958	0.958	0.958
AIC	−30765	−30759	−30730	24063	24062	24090	10595	10612	10626
BIC	−12775	−12768	−12740	42054	42052	42080	28586	28602	28616

$^{*}p < 0.05$, $^{**}p < 0.01$, $^{***}p < 0.001$.

Table A.2.30. Fully specified county group-level fixed effects SDEM models estimating female mortality indicators ($log(ASDR)$, $e(0)$, and $e(65)$) featuring various specifications of state health policy.

	$log(ASDR)$			$e(0)$			$e(65)$		
Variable	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$
SHP indicator	−0.002	−0.005*	0.000	0.052	0.066*	0.001	0.012	0.037*	−0.001
	(0.00)	(0.00)	(0.00)	(0.04)	(0.03)	(0.02)	(0.02)	(0.02)	(0.01)
Median age	−0.005***	−0.005***	−0.005***	0.051***	0.051***	0.052***	0.051***	0.051***	0.051***
	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
log(gov't ideology)	−0.014**	0.005	−0.015**	0.166*	−0.100	0.188*	0.114*	0.008	0.119*
	(0.01)	(0.01)	(0.01)	(0.07)	(0.08)	(0.07)	(0.05)	(0.05)	(0.05)
log(% in poor health)	−0.073***	−0.075***	−0.070***	0.840***	0.873***	0.807***	0.660***	0.683***	0.642***
	(0.01)	(0.01)	(0.01)	(0.13)	(0.13)	(0.13)	(0.08)	(0.08)	(0.08)
log(% Black)	0.002*	0.002*	0.002*	−0.036**	−0.030*	−0.030*	−0.022**	−0.019*	−0.019*
	(0.00)	(0.00)	(0.00)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)
log(D index)	−0.006***	−0.007***	−0.006***	0.098***	0.103***	0.101***	0.038***	0.041***	0.040***
	(0.00)	(0.00)	(0.00)	(0.02)	(0.02)	(0.02)	(0.01)	(0.01)	(0.01)
log(med. HH income)	−0.102***	−0.104***	−0.100***	1.462***	1.490***	1.448***	0.746***	0.771***	0.732***
	(0.01)	(0.01)	(0.01)	(0.12)	(0.12)	(0.12)	(0.08)	(0.08)	(0.08)
log(GINI index)	−0.117***	−0.118***	−0.122***	1.516***	1.520***	1.570***	0.951***	0.972***	0.993***
	(0.01)	(0.01)	(0.01)	(0.18)	(0.18)	(0.18)	(0.12)	(0.12)	(0.12)
% blue collar	−0.001***	−0.001***	−0.001***	0.016***	0.016***	0.016***	0.008***	0.008***	0.008***
	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
% urban population	−0.001***	−0.001***	−0.001***	0.011***	0.011***	0.012***	0.007***	0.007***	0.007***
	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
log(% w/out HS diploma)	−0.002	0.000	0.000	−0.037	−0.062	−0.050	0.073	0.056	0.063
	(0.00)	(0.00)	(0.00)	(0.06)	(0.06)	(0.06)	(0.04)	(0.04)	(0.04)
Lagged indicators									
SHP indicator	−0.014***	−0.007**	0.004*	0.171***	0.103**	−0.040	0.127***	0.062**	−0.046**
	(0.00)	(0.00)	(0.00)	(0.05)	(0.04)	(0.02)	(0.03)	(0.02)	(0.01)
Median age	−0.003***	−0.003***	−0.003***	0.035**	0.036**	0.039***	0.028***	0.030***	0.031***
	(0.00)	(0.00)	(0.00)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)
log(gov't ideology)	−0.002	0.005	−0.010	0.002	−0.041	0.099	0.060	−0.090	0.130*
	(0.01)	(0.01)	(0.01)	(0.10)	(0.11)	(0.10)	(0.06)	(0.06)	(0.06)
log(% in poor health)	−0.060**	−0.068***	−0.050*	1.208***	1.305***	1.073***	0.348*	0.433*	0.267
	(0.02)	(0.02)	(0.02)	(0.28)	(0.28)	(0.28)	(0.17)	(0.17)	(0.17)
log(% Black)	0.010***	0.008	0.009***	−0.149***	−0.124***	−0.125***	−0.089***	−0.072**	−0.073***
	(0.00)	(0.00)	(0.00)	(0.04)	(0.04)	(0.04)	(0.02)	(0.02)	(0.02)
log(D index)	−0.007*	−0.009**	−0.008*	0.117**	0.136**	0.128**	0.043	0.057*	0.051
	(0.00)	(0.00)	(0.00)	(0.04)	(0.04)	(0.04)	(0.03)	(0.03)	(0.03)
log(med. HH income)	0.005	−0.006	0.012	−0.227	−0.101	−0.308	0.002	0.114	−0.070
	(0.02)	(0.02)	(0.02)	(0.29)	(0.29)	(0.29)	(0.17)	(0.18)	(0.18)
log(GINI index)	−0.253***	−0.260***	−0.272***	3.480***	3.530***	3.717***	1.900***	1.998***	2.087***
	(0.04)	(0.04)	(0.04)	(0.52)	(0.53)	(0.53)	(0.32)	(0.33)	(0.32)
% blue collar	−0.002***	−0.002***	−0.003***	0.035***	0.036***	0.036***	0.019***	0.019***	0.019***
	(0.00)	(0.00)	(0.00)	(0.01)	(0.01)	(0.01)	(0.00)	(0.00)	(0.00)
% urban population	−0.001***	−0.001***	−0.001***	0.016***	0.016***	0.018***	0.012***	0.011***	0.013***
	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
log(% w/out HS diploma)	−0.043***	−0.035**	−0.038**	0.505**	0.399*	0.443**	0.415***	0.345***	0.368***
	(0.01)	(0.01)	(0.01)	(0.16)	(0.16)	(0.16)	(0.10)	(0.10)	(0.10)
ρ	0.633***	0.644***	0.642***	0.654***	0.660***	0.662***	0.612***	0.626***	0.618***
	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)
RW test (p-value)	0.002	0.002	0.002	0.002	0.002	0.002	0.002	0.002	0.002
BSJK (C.2) test	1188***	2963***	3270***	3401***	1838***	3401***	3109***	1699***	3152***
Pseudo R^2	0.888	0.885	0.886	0.89	0.888	0.888	0.887	0.884	0.886
AIC	32179	32214	32220	84103	84122	84142	75005	75051	75035
BIC	50169	50205	50210	102093	102113	102132	92995	93042	93026

$^{*}p < 0.05$, $^{**}p < 0.01$, $^{***}p < 0.001$.

Table A.2.31. Fully specified county group-level fixed effects SDEM models estimating male mortality indicators ($log(ASDR)$, $e(0)$, and $e(65)$) featuring various specifications of state health policy.

	$log(ASDR)$			$e(0)$			$e(65)$		
Variable	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$	I_Z	$I_{t-10,Z}$	$\tilde{\pi}_{10,Z}$
SHP indicator	−0.003	−0.006**	−0.001	0.079	0.082**	0.010	0.019	0.045**	0.014
	(0.00)	(0.00)	(0.00)	(0.04)	(0.03)	(0.02)	(0.02)	(0.02)	(0.01)
Median age	−0.003***	−0.003***	−0.003***	0.029***	0.029***	0.030***	0.041***	0.041***	0.041***
	(0.00)	(0.00)	(0.00)	(0.01)	(0.01)	(0.01)	(0.00)	(0.00)	(0.00)
log(gov't ideology)	−0.009	−0.004	−0.010	0.102	0.001	0.131	0.068	0.074	0.072
	(0.01)	(0.01)	(0.01)	(0.09)	(0.09)	(0.09)	(0.04)	(0.05)	(0.04)
log(% in poor health)	−0.032***	−0.032***	−0.029***	0.236	0.236	0.196	0.387***	0.388***	0.364***
	(0.01)	(0.01)	(0.01)	(0.14)	(0.14)	(0.14)	(0.07)	(0.07)	(0.07)
log(% Black)	0.002*	0.001	0.001	−0.044**	−0.037*	−0.038*	−0.011	−0.008	−0.008
	(0.00)	(0.00)	(0.00)	(0.02)	(0.02)	(0.02)	(0.01)	(0.01)	(0.01)
log(D index)	−0.007***	−0.008***	−0.008***	0.127***	0.133***	0.131***	0.047***	0.050***	0.049***
	(0.00)	(0.00)	(0.00)	(0.02)	(0.02)	(0.02)	(0.01)	(0.01)	(0.01)
log(med. HH income)	−0.120***	−0.121***	−0.119***	2.001***	2.013***	1.985***	0.875***	0.894***	0.862***
	(0.01)	(0.01)	(0.01)	(0.14)	(0.14)	(0.14)	(0.07)	(0.07)	(0.07)
log(GINI index)	−0.117***	−0.117***	−0.122***	1.439***	1.413***	1.501***	1.038***	1.049***	1.079***
	(0.01)	(0.01)	(0.01)	(0.21)	(0.21)	(0.21)	(0.11)	(0.11)	(0.11)
% blue collar	−0.001**	−0.001**	−0.001**	0.008**	0.008**	0.009**	0.005***	0.006***	0.006***
	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
% urban population	−0.001***	−0.001***	−0.001***	0.012***	0.012***	0.012***	0.007***	0.007***	0.008***
	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
log(% w/out HS diploma)	0.001	0.002	0.001	0.004	−0.015	−0.006	−0.011	−0.021	−0.018
	(0.00)	(0.00)	(0.00)	(0.07)	(0.07)	(0.07)	(0.04)	(0.04)	(0.04)
Lagged indicators									
SHP indicator	−0.012***	−0.006*	0.003	0.132*	0.092*	−0.019	0.103***	0.052*	−0.034*
	(0.00)	(0.00)	(0.00)	(0.05)	(0.04)	(0.03)	(0.03)	(0.02)	(0.01)
Median age†	−0.002*	−0.002*	−0.002**	0.021	0.022	0.025	0.025***	0.026***	0.027***
	(0.00)	(0.00)	(0.00)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)
log(gov't ideology)*	0.003	0.013	−0.004	−0.111	−0.148	−0.031	0.041	−0.127*	0.102
	(0.01)	(0.01)	(0.01)	(0.11)	(0.12)	(0.11)	(0.05)	(0.06)	(0.05)
log(% in poor health)	−0.111***	−0.108***	−0.099***	2.519***	2.472***	2.346***	0.545***	0.534***	0.453***
	(0.02)	(0.02)	(0.02)	(0.27)	(0.27)	(0.27)	(0.13)	(0.13)	(0.13)
log(% Black)	0.010***	0.008**	0.008**	−0.204***	−0.178***	−0.178***	−0.055**	−0.039*	−0.040*
	(0.00)	(0.00)	(0.00)	(0.04)	(0.04)	(0.04)	(0.02)	(0.02)	(0.02)
log(D index)	−0.008**	−0.010***	−0.009**	0.145**	0.168***	0.158**	0.051*	0.066**	0.060*
	(0.00)	(0.00)	(0.00)	(0.05)	(0.05)	(0.05)	(0.02)	(0.02)	(0.02)
log(med. HH income)	0.000	−0.005	0.008	−0.201	−0.187	−0.298	0.003	0.071	−0.065
	(0.02)	(0.02)	(0.02)	(0.32)	(0.32)	(0.32)	(0.15)	(0.15)	(0.16)
log(GINI index)	−0.237***	−0.240***	−0.259***	3.525***	3.452***	3.816***	1.853***	1.911***	2.042***
	(0.04)	(0.04)	(0.04)	(0.59)	(0.59)	(0.59)	(0.29)	(0.29)	(0.29)
% blue collar	−0.001*	−0.001*	−0.001*	0.012	0.013	0.014	0.009*	0.010**	0.010**
	(0.00)	(0.00)	(0.00)	(0.01)	(0.01)	(0.01)	(0.00)	(0.00)	(0.00)
% urban population	−0.001***	−0.001***	−0.001***	0.016***	0.016***	0.017***	0.011***	0.011***	0.012***
	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
log(% w/out HS diploma)	−0.021	−0.015	−0.017	0.427*	0.346*	0.379*	0.108	0.069	0.075
	(0.01)	(0.01)	(0.01)	(0.18)	(0.18)	(0.18)	(0.09)	(0.09)	(0.09)
ρ	0.569***	0.574***	0.581***	0.601***	0.601***	0.609***	0.558***	0.567***	0.568***
	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)
RW test (p-value)	0.002	0.002	0.002	0.002	0.002	0.002	0.002	0.002	0.002
BSJK (C.2) test	1291***	3508***	3809***	1400***	1322***	2054***	3056***	1518***	3947***
Pseudo R^2	0.936	0.935	0.935	0.929	0.929	0.928	0.946	0.945	0.945
AIC	32179	32185	32229	87018	87012	87055	73543	73560	73591
BIC	50170	50175	50219	105009	105003	105046	91534	91550	91581

$^{*}p < 0.05$, $^{**}p < 0.01$, $^{***}p < 0.001$.

Table A.2.32. Post-regression residual Moran estimates for various models for the 3 mortality outcome indicators and 10-year lagged I score as the state health policy indicator, by year and sex.

			States			County groups		
Model	Sex	Year	$log(ASDR)$	$e(0)$	$e(65)$	$log(ASDR)$	$e(0)$	$e(65)$
FE panel†	Both sexes	1990	0.240**	0.152	0.317***	0.467***	0.477***	0.449***
		2000	0.217**	0.206*	0.213*	0.276***	0.274***	0.290***
		2010	0.169*	0.193*	0.047	0.343***	0.387***	0.268***
		2018	0.183*	0.149	0.262**	0.352***	0.359***	0.373***
SAR	Both sexes	1990	−0.053	−0.064	−0.075	0.004	0.006	0.008
		2000	0.150	0.113	0.131	−0.013	−0.005	−0.011
		2010	0.031	0.069	−0.007	0.021	0.028**	0.020
		2018	−0.141	−0.107	−0.189*	0	−0.003	0.004
	Female	1990	−0.004	−0.001	−0.029	0.004	0.002	0.007
		2000	0.093	0.098	0.054	−0.022	−0.015	−0.022
		2010	0.036	0.085	−0.045	0.016	0.019	0.018
		2018	−0.131	−0.087	−0.192*	0.005	0.001	0.010
	Male	1990	−0.061	−0.080	−0.074	0.007	0.011	0.013
		2000	0.186*	0.099	0.190*	−0.006	0.003	−0.004
		2010	0.015	0.039	0.019	0.027*	0.035***	0.026*
		2018	−0.117	−0.092	−0.132	0.001	−0.001	0.006
SLX	Both sexes	1990	0.305**	0.223*	0.398***	0.439***	0.459***	0.418***
		2000	0.318**	0.239**	0.308**	0.310***	0.298***	0.325***
		2010	0.085	0.130	0.077	0.333***	0.372***	0.271***
		2018	0.224**	0.202*	0.239**	0.324***	0.336***	0.337***
	Female	1990	0.302**	0.258*	0.354***	0.468***	0.487***	0.446***
		2000	0.286**	0.229*	0.297**	0.359***	0.365***	0.36***
		2010	0.054	0.088	0.039	0.354***	0.382***	0.306***
		2018	0.179*	0.163*	0.206*	0.355***	0.366***	0.352***
	Male	1990	0.186*	0.147	0.241*	0.393***	0.425***	0.367***
		2000	0.227*	0.165*	0.233**	0.254***	0.241***	0.274***
		2010	0.002	0.089	−0.018	0.297***	0.355***	0.23***
		2018	0.166*	0.150	0.203*	0.293***	0.312***	0.312***
SEM	Both sexes	1990	−0.068	−0.078	−0.083	0.051***	0.050***	0.057***
		2000	0.126	0.140	0.081	0.020	0.026*	0.025*
		2010	−0.007	0.031	−0.014	0.071***	0.079***	0.07***
		2018	−0.115	−0.102	−0.145	0.032*	0.028*	0.042***
	Female	1990	−0.046	−0.042	−0.087	0.062***	0.057***	0.07***
		2000	0.044	0.074	0.005	0.023*	0.025*	0.023*
		2010	0.018	0.043	−0.023	0.075***	0.077***	0.077***
		2018	−0.096	−0.072	−0.144	0.048***	0.041***	0.059***
	Male	1990	−0.072	−0.086	−0.050	0.044***	0.048***	0.051***
		2000	0.217*	0.157	0.172*	0.020	0.030**	0.025*
		2010	−0.012	0.028	−0.025	0.063***	0.078***	0.06***
		2018	−0.117	−0.096	−0.135	0.022*	0.023*	0.034**
SDM	Both sexes	1990	−0.007	−0.025	−0.003	0.002	0.003	0.007
		2000	0.080	0.035	0.079	−0.012	−0.002	−0.011
		2010	−0.108	−0.102	−0.072	0.023*	0.029*	0.023*
		2018	−0.117	−0.078	−0.144	−0.002	−0.004	0.003
	Female	1990	0.026	0.015	0.028	0.002	0	0.006
		2000	−0.016	−0.005	−0.027	−0.023*	−0.015	−0.023*
		2010	−0.089	−0.080	−0.102	0.019	0.021	0.020
		2018	−0.111	−0.068	−0.158	0.003	−0.001	0.009
	Male	1990	0.001	−0.039	0.015	0.005	0.006	0.013
		2000	0.164	0.055	0.174*	−0.005	0.005	−0.004
		2010	−0.065	−0.072	0.004	0.030**	0.035**	0.029*
		2018	−0.105	−0.066	−0.103	−0.001	−0.003	0.005
SDEM	Both sexes	1990	−0.075	−0.075	−0.070	0.036**	0.030**	0.046***
		2000	0.023	0.001	0.033	0.020	0.027*	0.022*
		2010	−0.145	−0.140	−0.103	0.065***	0.072***	0.063***
		2018	−0.107	−0.083	−0.132	0.013	0.007	0.025*
	Female	1990	−0.041	−0.039	−0.036	0.045***	0.037***	0.053***
		2000	−0.042	−0.027	−0.049	0.015	0.020	0.014
		2010	−0.126	−0.127	−0.131	0.066***	0.068***	0.066***
		2018	−0.095	−0.058	−0.146	0.027*	0.020	0.039***
	Male	1990	−0.042	−0.073	−0.028	0.031**	0.028*	0.044**
		2000	0.130	0.033	0.137	0.023	0.034**	0.025*
		2010	−0.099	−0.097	−0.028	0.063***	0.073***	0.059***
		2018	−0.106	−0.081	−0.103	0.008	0.007	0.021*

$^{*}p < 0.05$, $^{**}p < 0.01$, $^{***}p < 0.001$.
† FE panel both-sex for benchmark comparison only.

Table A.2.33. Post-regression residual Moran estimates for various models for the 3 mortality outcome indicators and the relative fraction of policies passed in prior decade as the state health policy indicator, by year and sex.

			States			County groups		
Model	Sex	Year	$log(ASDR)$	$e(0)$	$e(65)$	$log(ASDR)$	$e(0)$	$e(65)$
FE panel†	Both sexes	1990	0.239*	0.158	0.314**	0.475***	0.491***	0.449***
		2000	0.257**	0.226*	0.252**	0.283***	0.276***	0.299***
		2010	0.260**	0.262**	0.168	0.386***	0.423***	0.309***
		2018	0.149*	0.143	0.219*	0.333***	0.351***	0.346***
SAR	Both sexes	1990	−0.109	−0.118	−0.125	0.004	0.007	0.007
		2000	0.087	0.057	0.096	−0.013	−0.005	−0.010
		2010	−0.030	0.035	−0.078	0.017	0.025*	0.014
		2018	−0.095	−0.065	−0.138	0.001	−0.001	0.004
	Female	1990	−0.064	−0.063	−0.085	0.003	0.003	0.005
		2000	0.079	0.074	0.064	−0.022	−0.015	−0.021
		2010	−0.054	0.024	−0.124	0.013	0.017	0.014
		2018	−0.081	−0.043	−0.134	0.006	0.003	0.010
	Male	1990	−0.100	−0.125	−0.096	0.008	0.012	0.013
		2000	0.097	0.040	0.111	−0.006	0.002	−0.003
		2010	0.008	0.036	0.003	0.022	0.032**	0.019
		2018	−0.075	−0.056	−0.090	0.002	0.001	0.006
SLX	Both sexes	1990	0.251**	0.186*	0.326**	0.439***	0.464***	0.411***
		2000	0.334***	0.262**	0.309***	0.316***	0.300***	0.334***
		2010	0.157	0.185*	0.137	0.361***	0.395***	0.297***
		2018	0.164	0.151	0.180*	0.308***	0.331***	0.309***
	Female	1990	0.255**	0.215**	0.307***	0.461***	0.486***	0.434***
		2000	0.323**	0.26**	0.316**	0.367***	0.372***	0.368***
		2010	0.139	0.166*	0.113	0.384***	0.410***	0.335***
		2018	0.159	0.138	0.183*	0.322***	0.344***	0.310***
	Male	1990	0.133	0.117	0.160*	0.396***	0.432***	0.364***
		2000	0.254**	0.212**	0.261**	0.259***	0.241***	0.283***
		2010	0.095	0.136	0.099	0.321***	0.372***	0.251***
		2018	0.118	0.111	0.154	0.288***	0.314***	0.298***
SEM	Both sexes	1990	−0.091	−0.097	−0.115	0.053***	0.057***	0.056***
		2000	0.116	0.118	0.084	0.025*	0.032**	0.029**
		2010	−0.070	−0.019	−0.100	0.067***	0.080***	0.062***
		2018	−0.085	−0.070	−0.112	0.035**	0.036***	0.042***
	Female	1990	−0.075	−0.070	−0.122	0.062***	0.061***	0.066***
		2000	0.077	0.095	0.044	0.027*	0.032**	0.026**
		2010	−0.064	−0.024	−0.114	0.071***	0.077***	0.069***
		2018	−0.072	−0.050	−0.109	0.050***	0.047***	0.057***
	Male	1990	−0.083	−0.098	−0.065	0.047***	0.054***	0.052***
		2000	0.177*	0.131	0.141	0.024*	0.035**	0.029*
		2010	−0.026	0.016	−0.055	0.058***	0.079***	0.053***
		2018	−0.088	−0.066	−0.112	0.027*	0.031**	0.036**
SDM	Both sexes	1990	−0.020	−0.039	−0.021	0.002	0.004	0.006
		2000	0.098	0.056	0.109	−0.013	−0.003	−0.011
		2010	−0.112	−0.076	−0.132	0.020	0.027**	0.018
		2018	−0.081	−0.052	−0.112	−0.002	−0.004	0.002
	Female	1990	0.033	0.018	0.028	0.001	0	0.004
		2000	0.044	0.052	0.029	−0.023*	−0.015	−0.023
		2010	−0.126	−0.090	−0.168	0.016	0.019	0.017
		2018	−0.097	−0.056	−0.143	0.003	−0.001	0.008
	Male	1990	−0.015	−0.051	0	0.005	0.007	0.013
		2000	0.183	0.070	0.221*	−0.006	0.003	−0.004
		2010	−0.026	−0.026	−0.012	0.025*	0.033**	0.023*
		2018	−0.051	−0.041	−0.046	−0.001	−0.002	0.004
SDEM	Both sexes	1990	−0.079	−0.084	−0.084	0.032**	0.031**	0.038**
		2000	0.059	0.040	0.075	0.017	0.026*	0.020
		2010	−0.111	−0.088	−0.113	0.056***	0.068***	0.050***
		2018	−0.061	−0.053	−0.084	0.013	0.011	0.021
	Female	1990	−0.026	−0.031	−0.031	0.040***	0.037***	0.044**
		2000	0.008	0.026	−0.005	0.014	0.021	0.012
		2010	−0.123	−0.101	−0.163	0.058***	0.064***	0.055***
		2018	−0.066	−0.036	−0.113	0.026*	0.023*	0.034***
	Male	1990	−0.051	−0.080	−0.041	0.029**	0.029*	0.039***
		2000	0.170*	0.067	0.204*	0.019	0.030**	0.022
		2010	−0.038	−0.034	−0.018	0.053***	0.069***	0.046***
		2018	−0.040	−0.053	−0.028	0.009	0.011	0.018

$^{*}p < 0.05$, $^{**}p < 0.01$, $^{***}p < 0.001$.
† FE panel both-sex for benchmark comparison only.

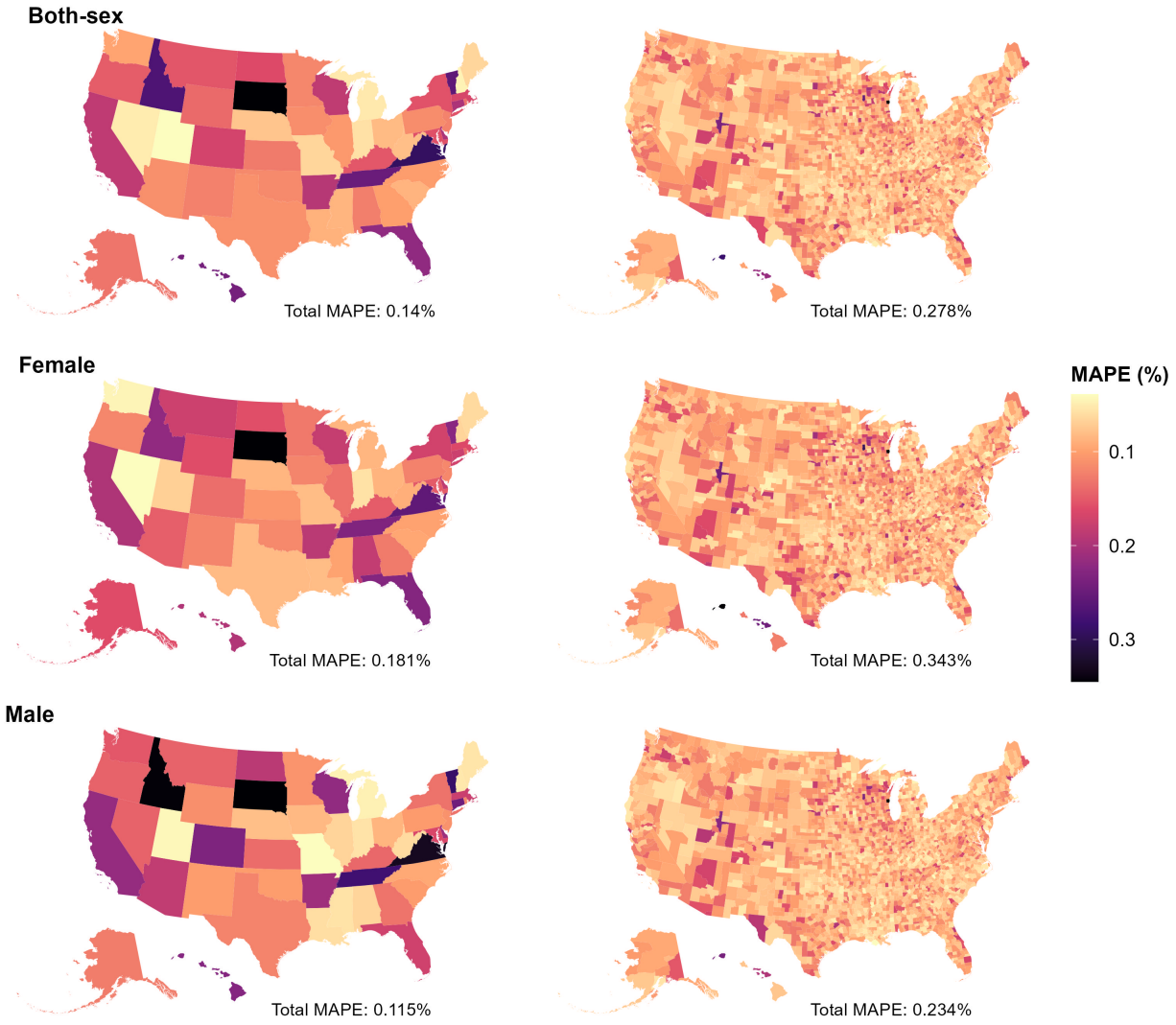


Figure A.2.20. Spatial distribution of mean absolute percent error (MAPE) in fully adjusted spatial Durbin models (SDM) for the logarithm of the age-standardized mortality rate as the outcome and the 10-year lagged form of I score as the main state health policy predictor, by sex.

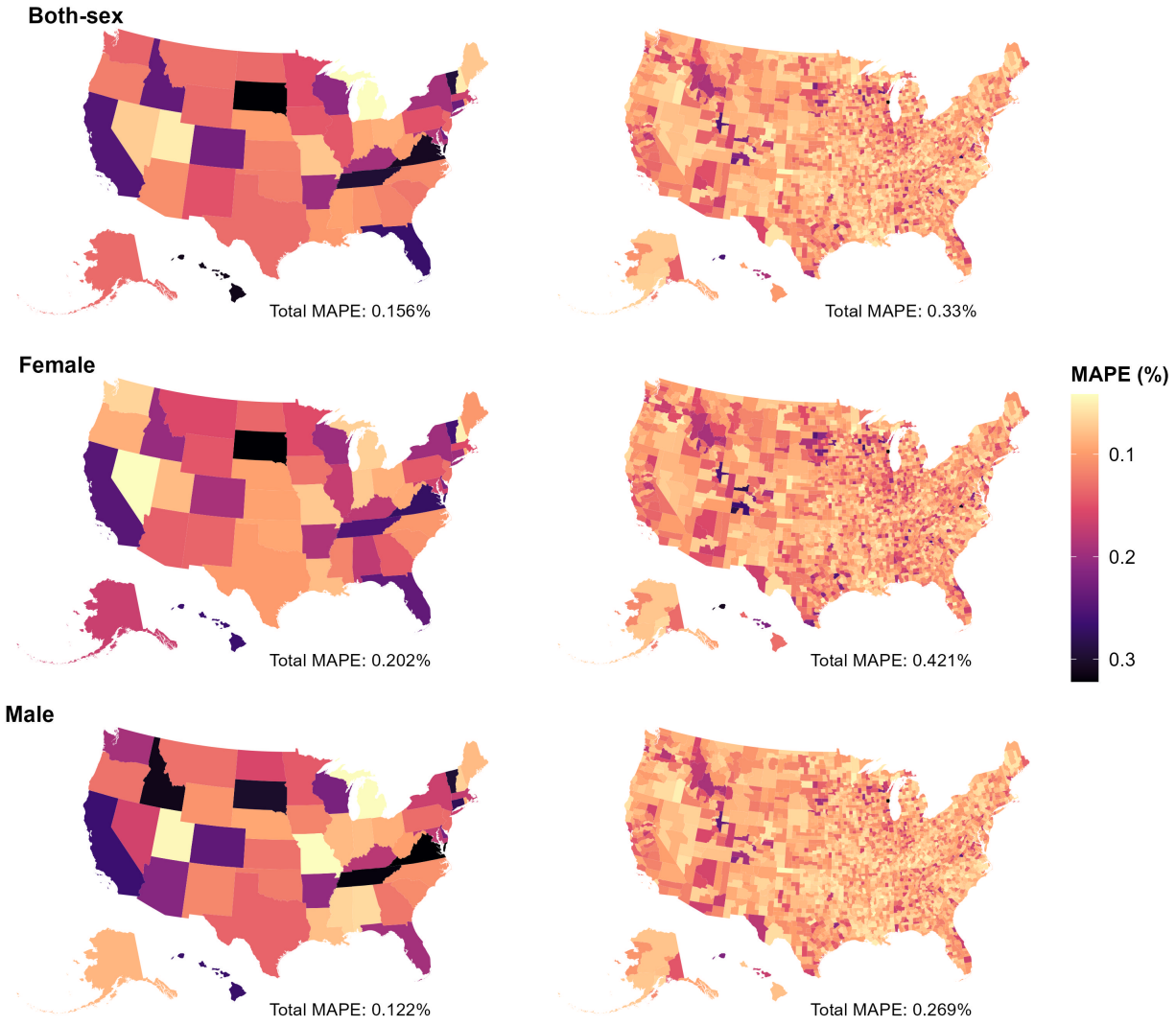


Figure A.2.21. Spatial distribution of mean absolute percent error (MAPE) in fully adjusted spatial Durbin error models (SDEM) for the logarithm of the age-standardized mortality rate as the outcome and the 10-year lagged for of I score as the main state health policy predictor, by sex.

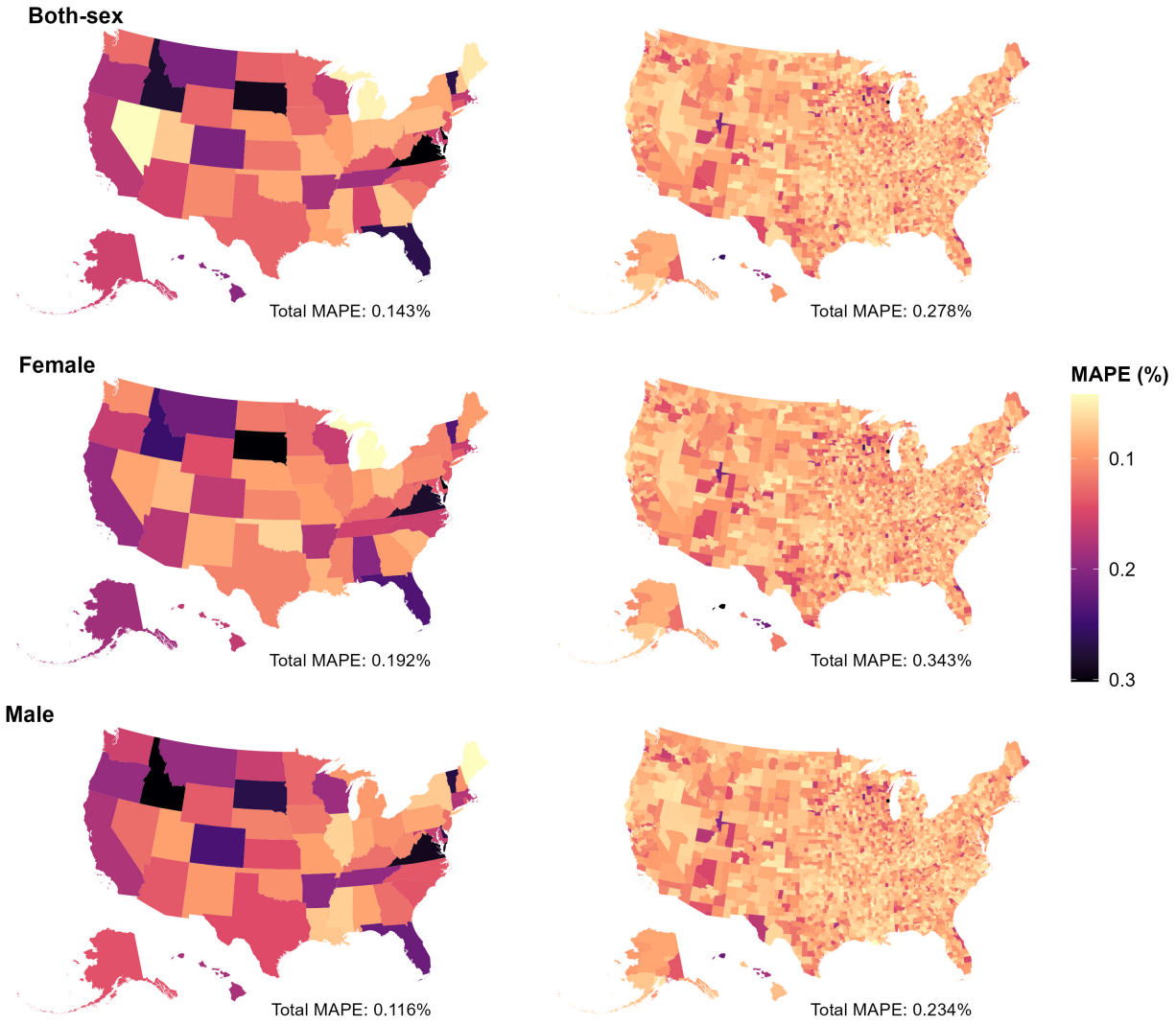


Figure A.2.22. Spatial distribution of mean absolute percent error (MAPE) in fully adjusted spatial Durbin models (SDM) for the logarithm of the age-standardized mortality rate as the outcome and the relative fraction of new policies passed in prior decade as the main state health policy predictor, by sex.

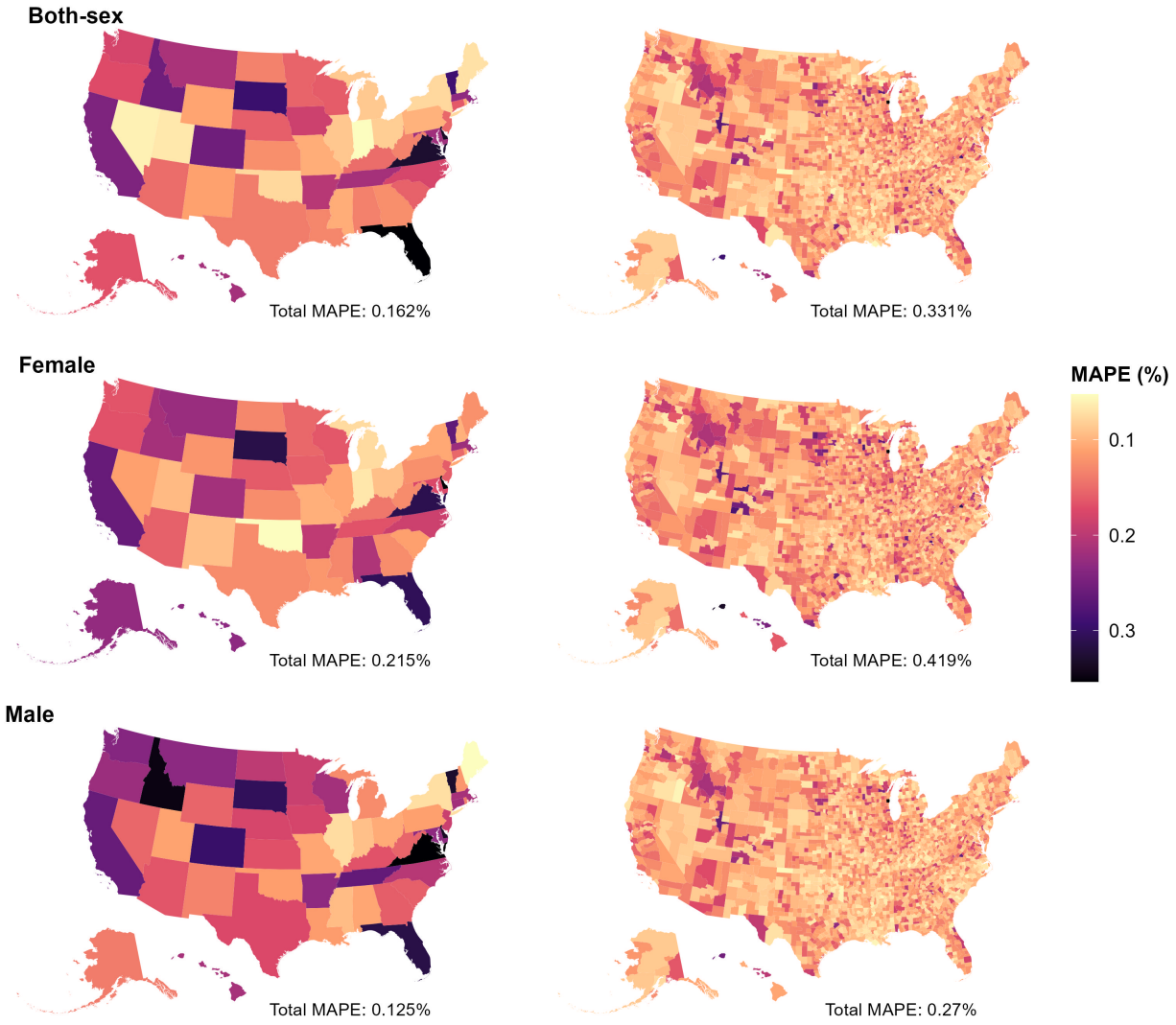


Figure A.2.23. Spatial distribution of mean absolute percent error (MAPE) in fully adjusted spatial Durbin error models (SDEM) for the logarithm of the age-standardized mortality rate as the outcome and the relative fraction of new policies passed in prior decade as the main state health policy predictor, by sex.

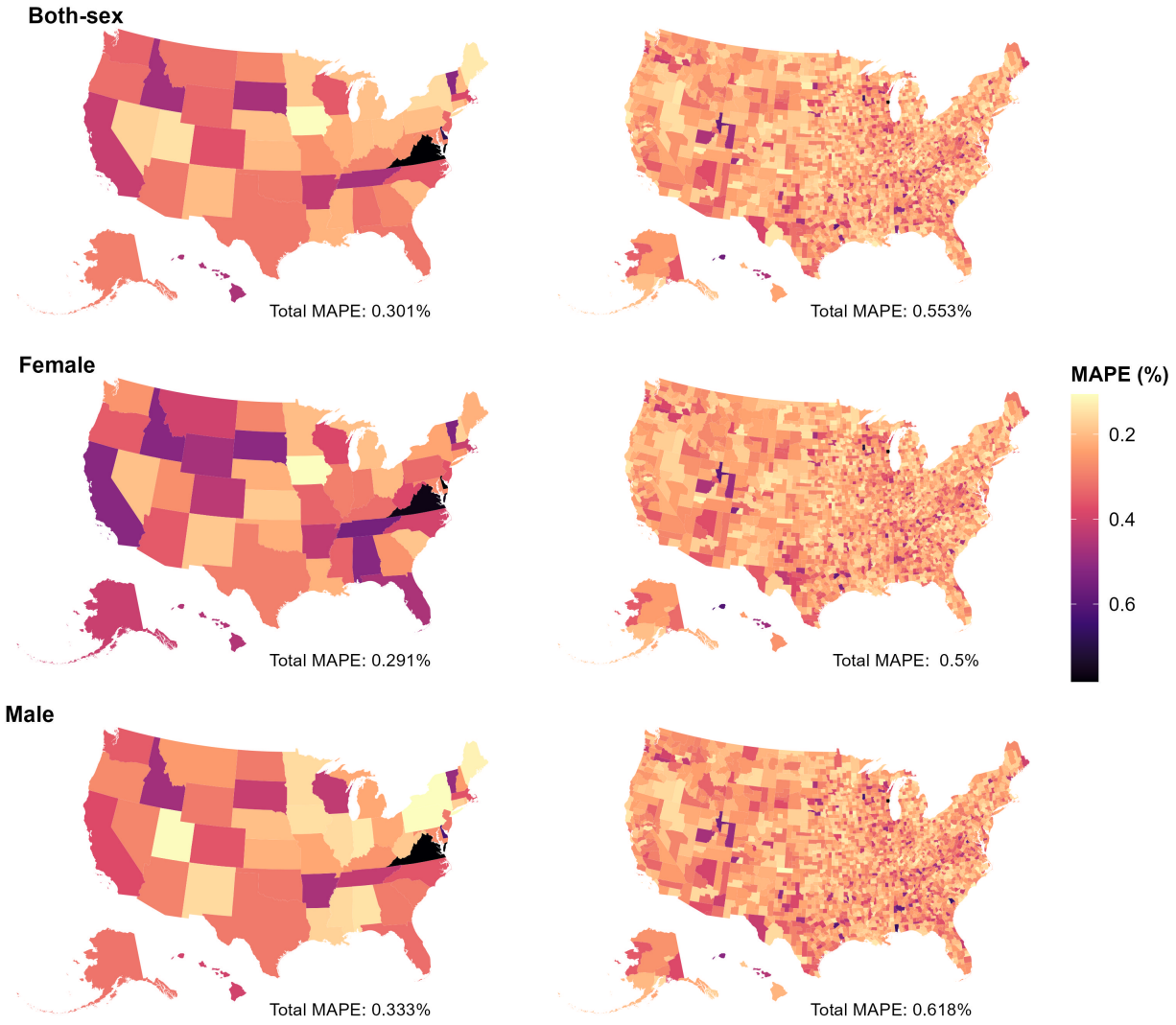


Figure A.2.24. Spatial distribution of mean absolute percent error (MAPE) in fully adjusted spatial Durbin models (SDM) for the life expectancy at birth as the outcome and I score as the main state health policy predictor, by sex.

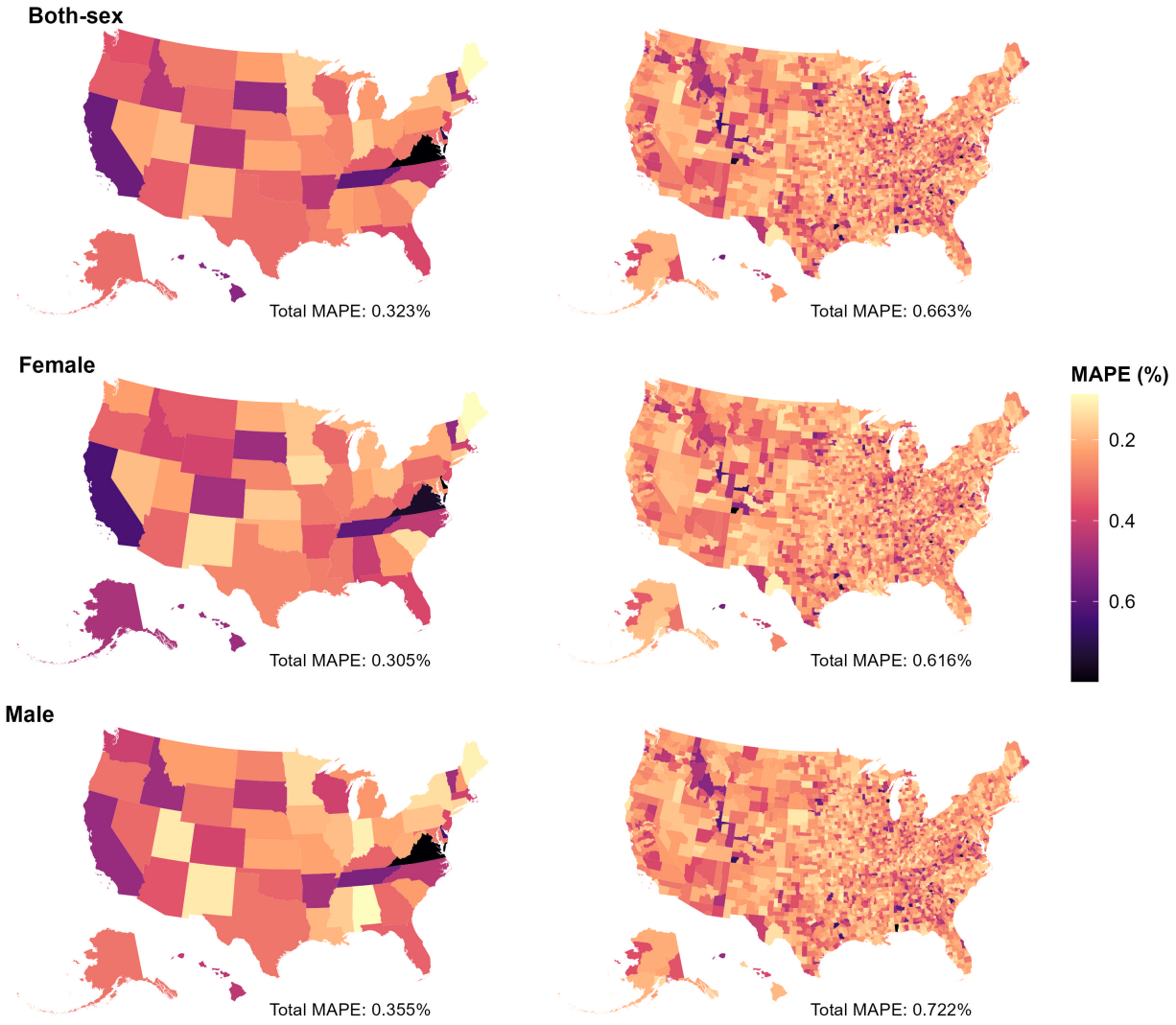


Figure A.2.25. Spatial distribution of mean absolute percent error (MAPE) in fully adjusted spatial Durbin error models (SDEM) for the life expectancy at birth as the outcome and I score as the main state health policy predictor, by sex.

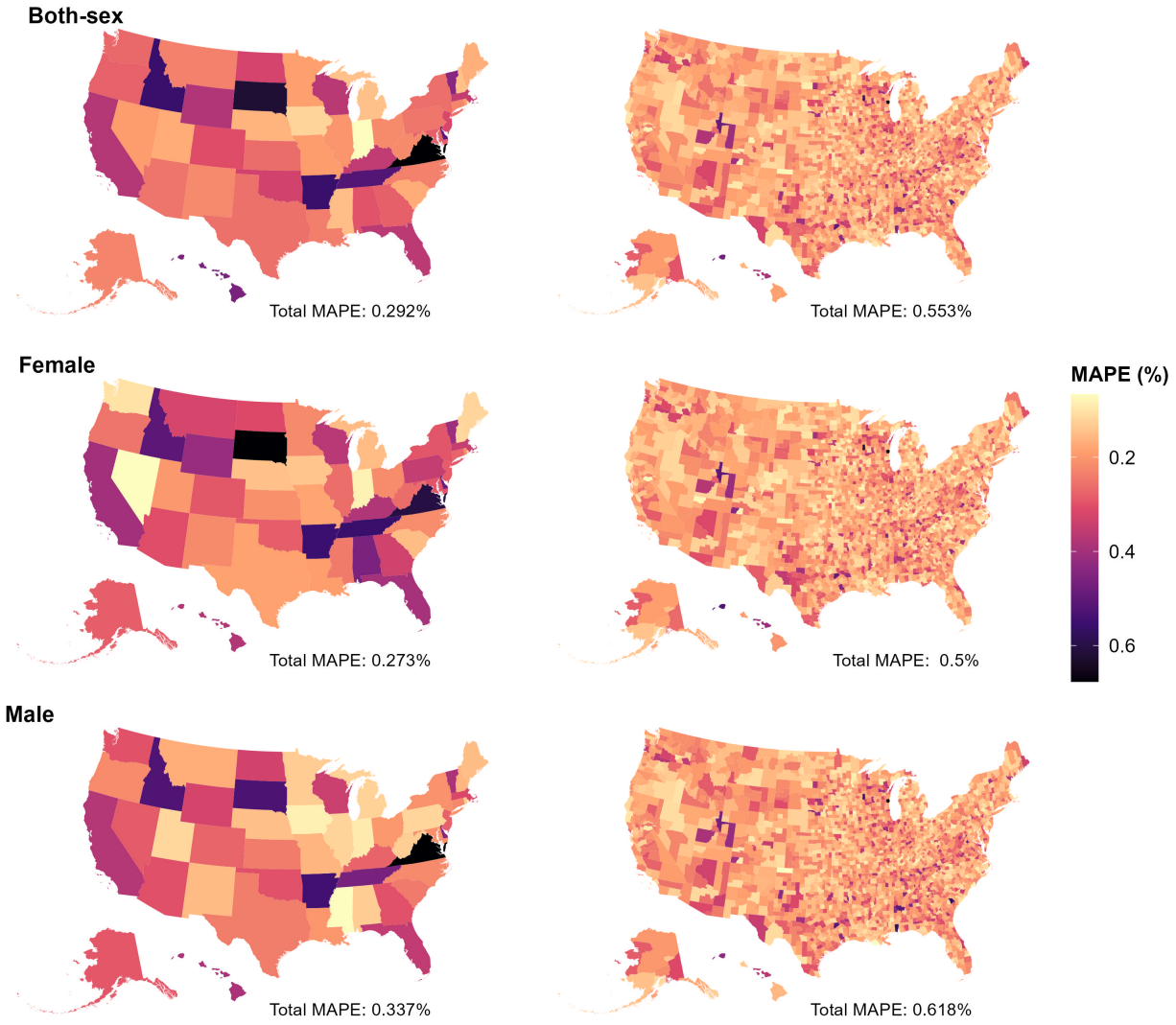


Figure A.2.26. Spatial distribution of mean absolute percent error (MAPE) in fully adjusted spatial Durbin models (SDM) for the life expectancy at birth as the outcome and the 10-year lagged form of I score as the main state health policy predictor, by sex.

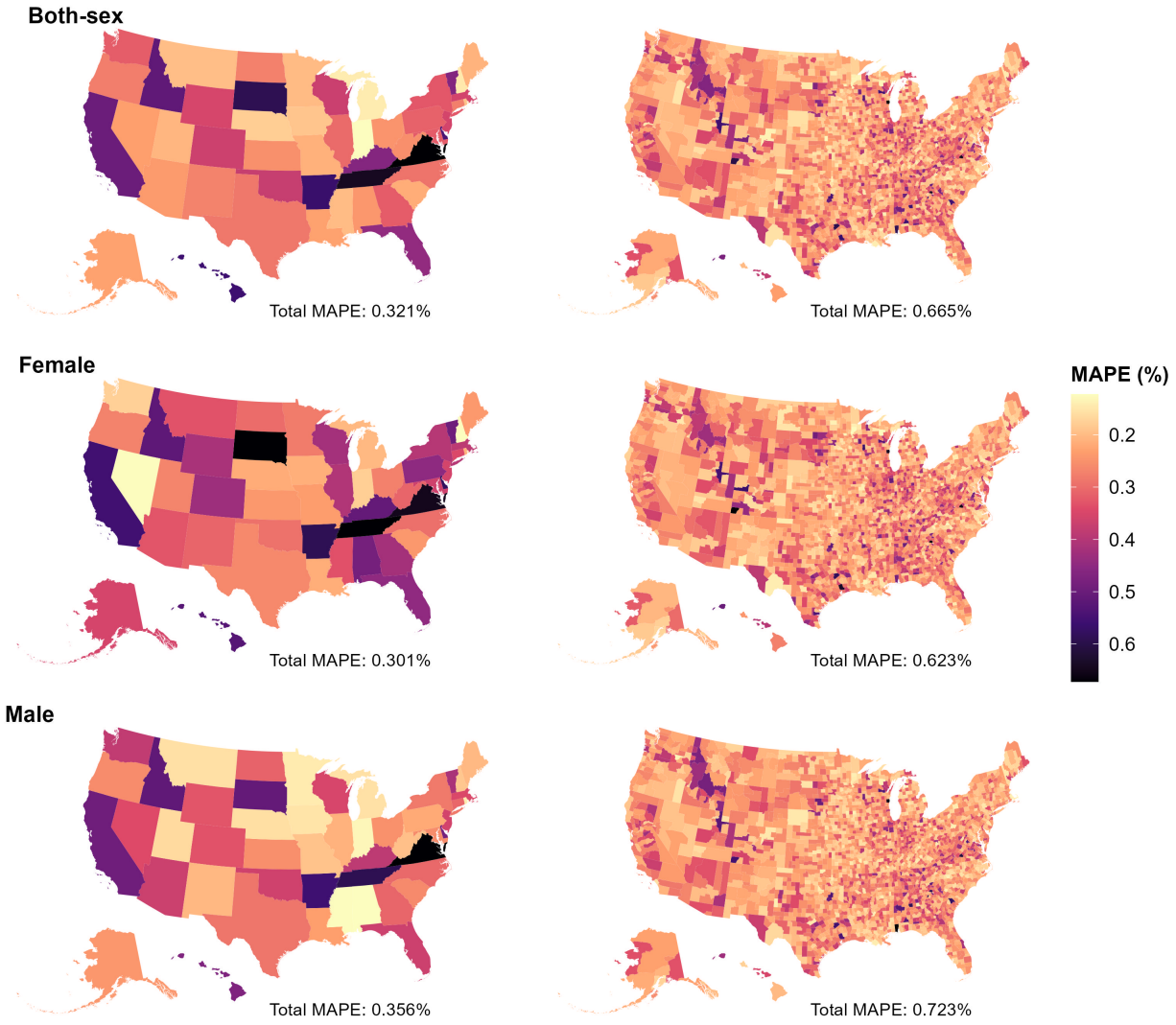


Figure A.2.27. Spatial distribution of mean absolute percent error (MAPE) in fully adjusted spatial Durbin error models (SDEM) for the life expectancy at birth as the outcome and the 10-year lagged form of I score as the main state health policy predictor, by sex.

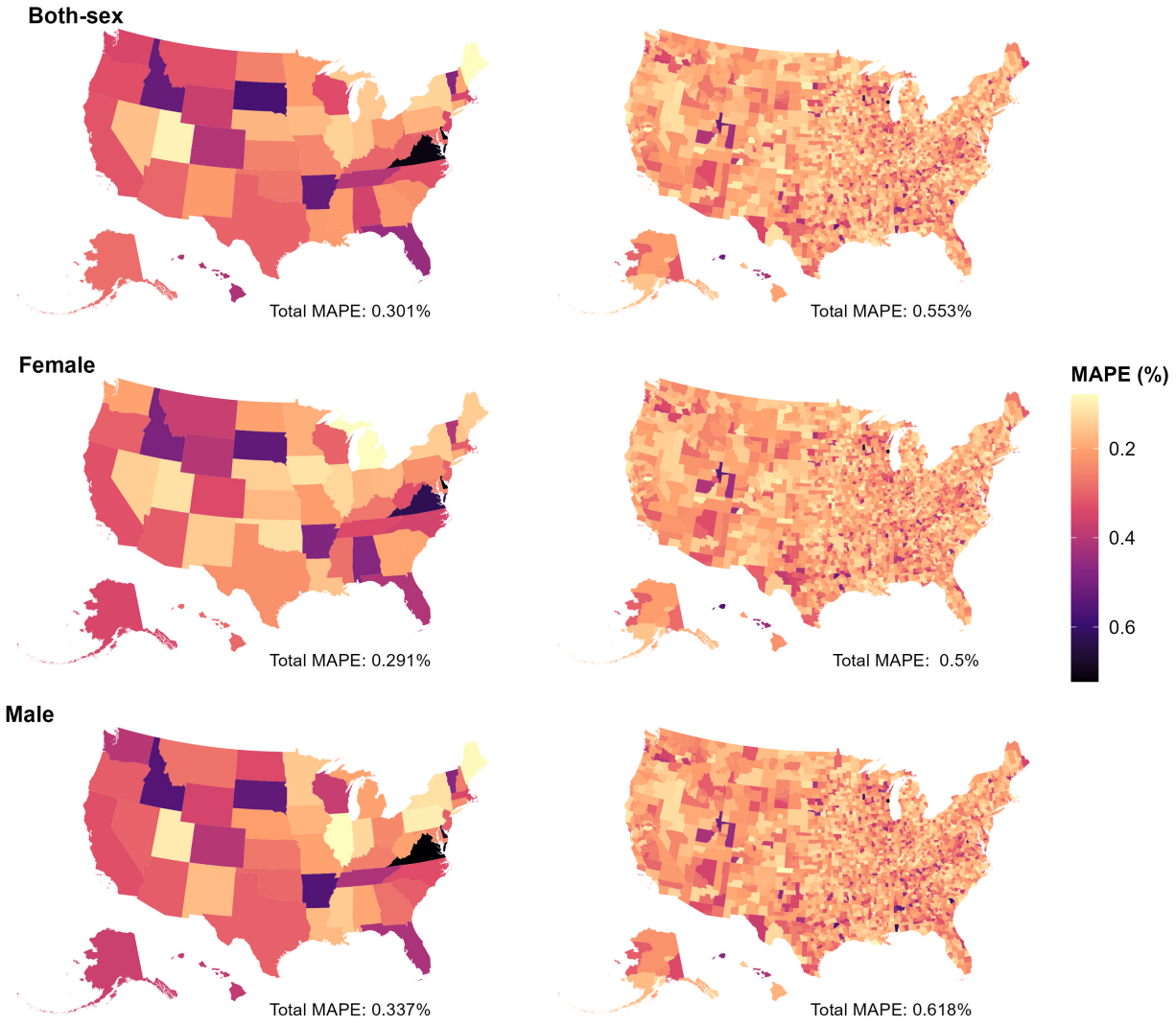


Figure A.2.28. Spatial distribution of mean absolute percent error (MAPE) in fully adjusted spatial Durbin models (SDM) for the life expectancy at birth as the outcome and the relative fraction of new policies passed in prior decade as the main state health policy predictor, by sex.

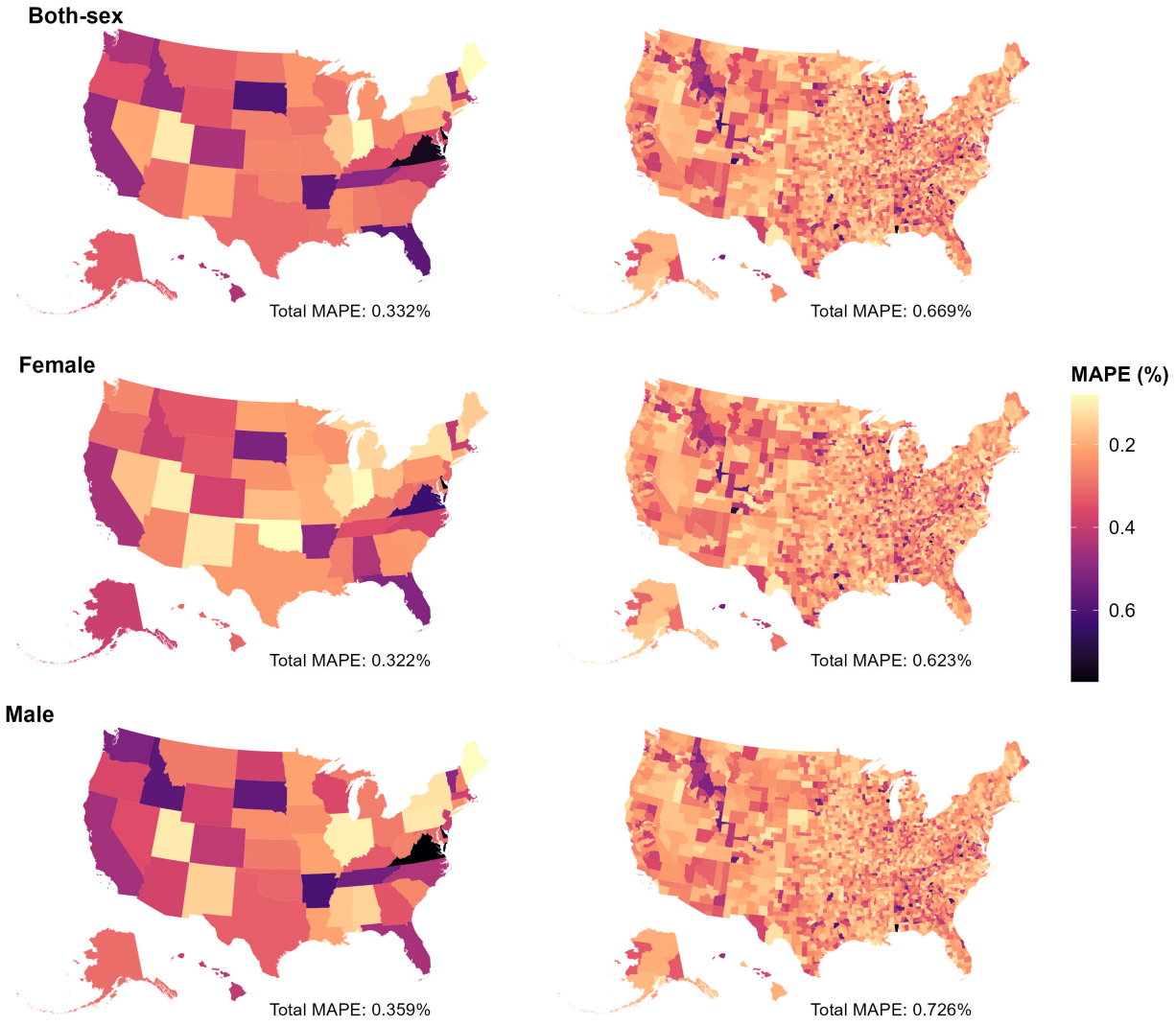


Figure A.2.29. Spatial distribution of mean absolute percent error (MAPE) in fully adjusted spatial Durbin error models (SDEM) for the life expectancy at birth as the outcome and the relative fraction of new policies passed in prior decade as the main state health policy predictor, by sex.

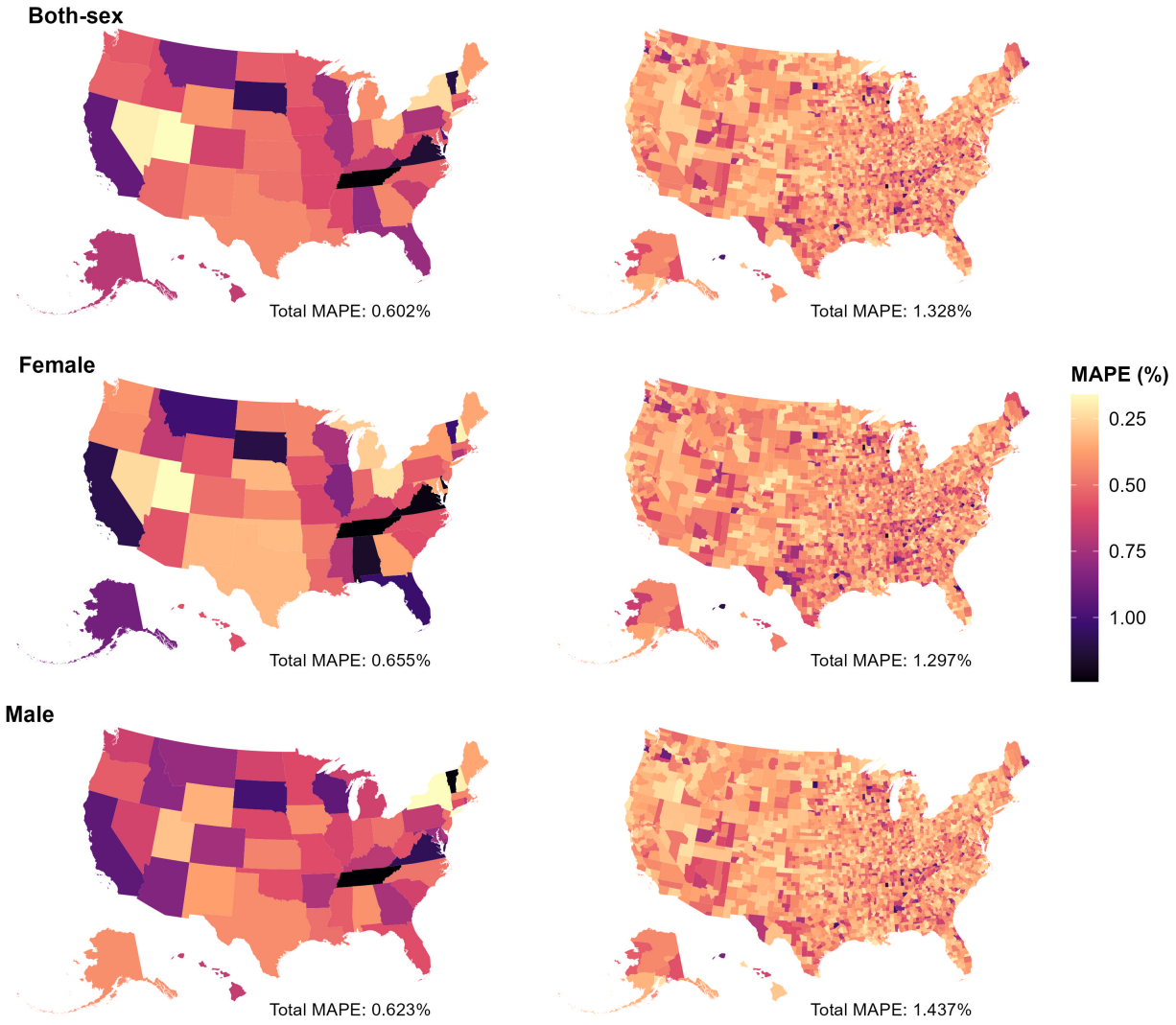


Figure A.2.30. Spatial distribution of mean absolute percent error (MAPE) in fully adjusted spatial Durbin models (SDM) for the life expectancy at age 65 as the outcome and I score as the main state health policy predictor, by sex.

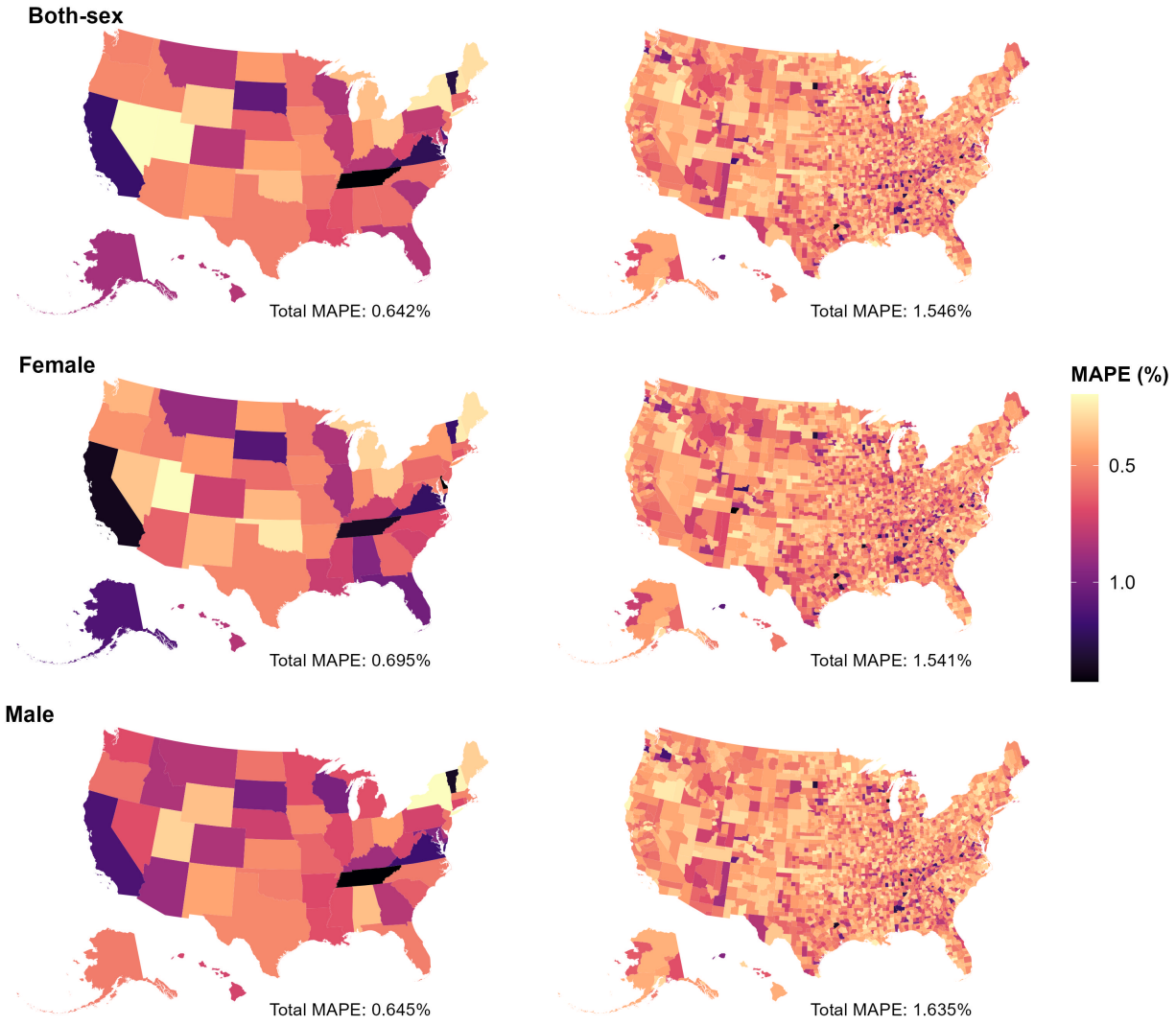


Figure A.2.31. Spatial distribution of mean absolute percent error (MAPE) in fully adjusted spatial Durbin error models (SDEM) for the life expectancy at age 65 as the outcome and I score as the main state health policy predictor, by sex.

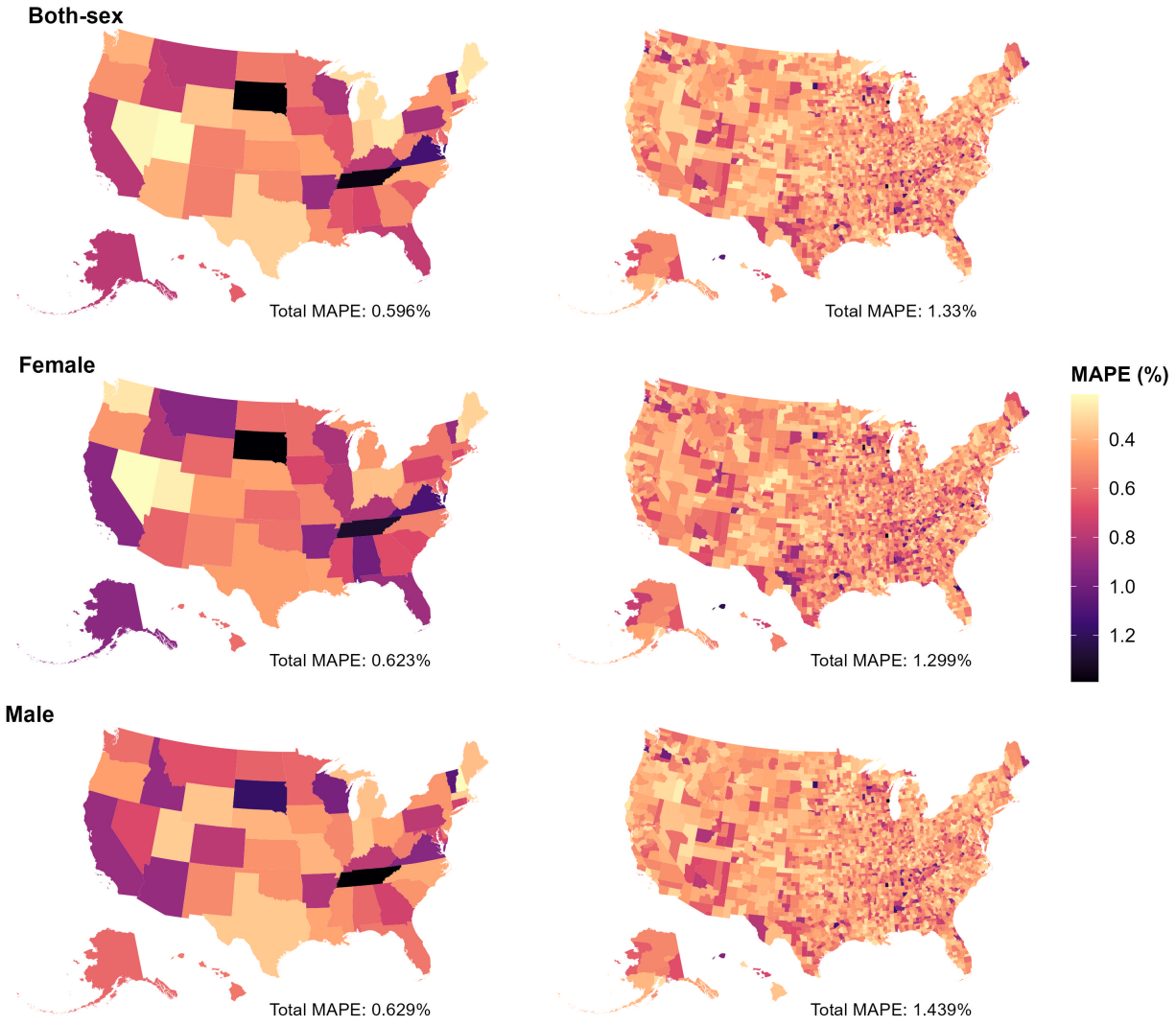


Figure A.2.32. Spatial distribution of mean absolute percent error (MAPE) in fully adjusted spatial Durbin models (SDM) for the life expectancy at age 65 as the outcome and the 10-year lagged form of I score as the main state health policy predictor, by sex.

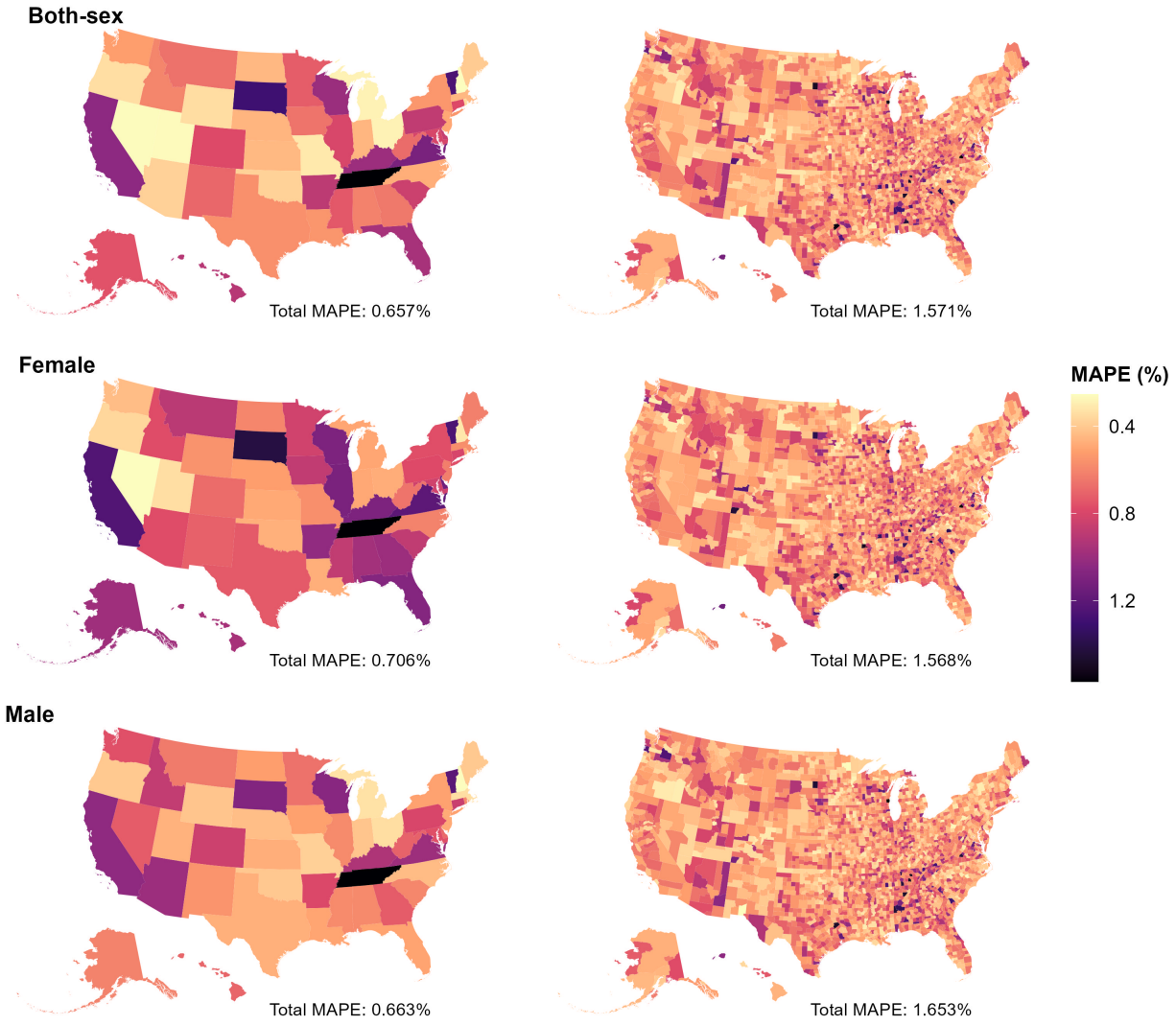


Figure A.2.33. Spatial distribution of mean absolute percent error (MAPE) in fully adjusted spatial Durbin error models (SDEM) for the life expectancy at age 65 as the outcome and the 10-year lagged form of I score as the main state health policy predictor, by sex.

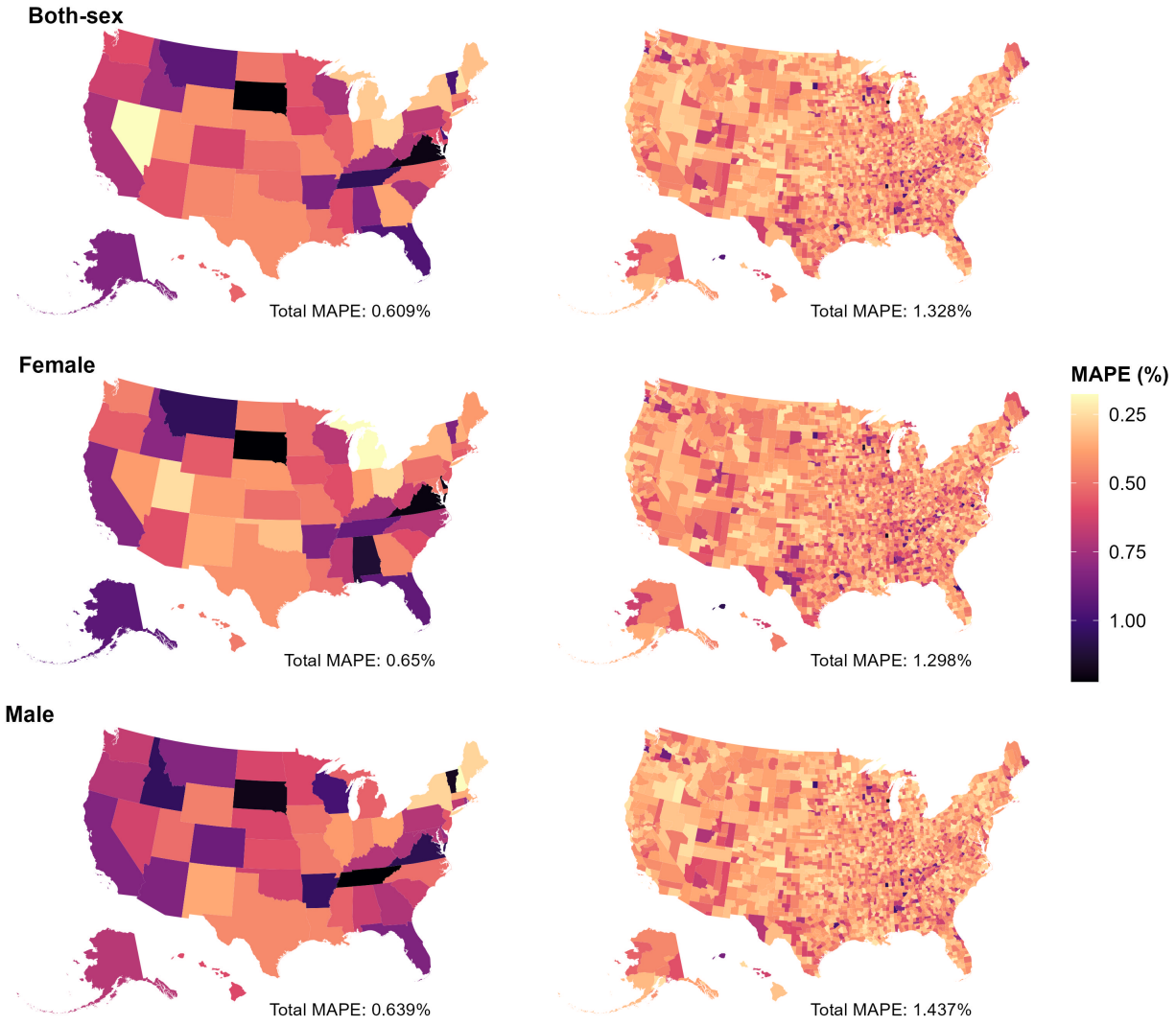


Figure A.2.34. Spatial distribution of mean absolute percent error (MAPE) in fully adjusted spatial Durbin models (SDM) for the life expectancy at age 65 as the outcome and the relative fraction of new policies passed in prior decade as the main state health policy predictor, by sex.

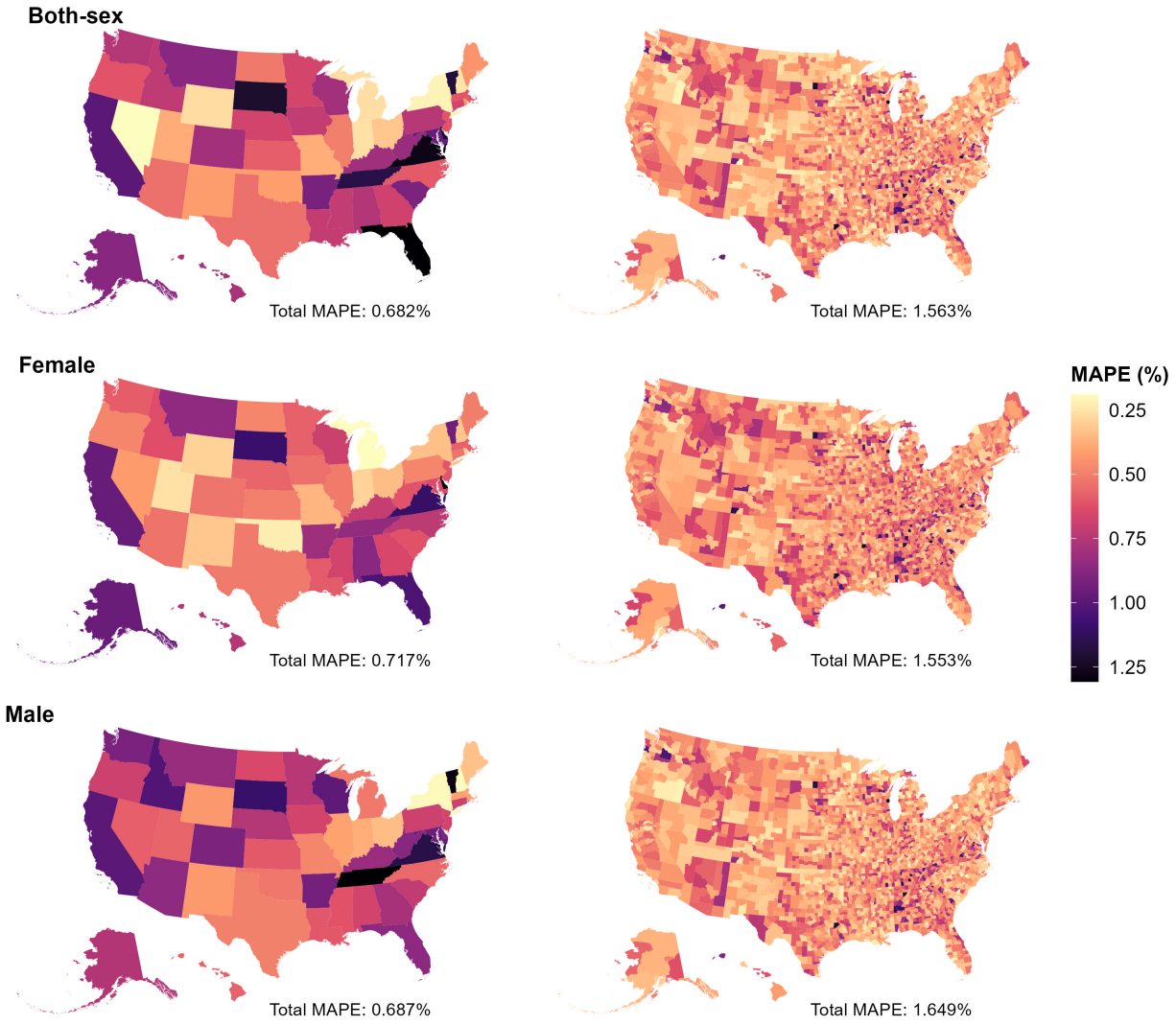


Figure A.2.35. Spatial distribution of mean absolute percent error (MAPE) in fully adjusted spatial Durbin error models (SDEM) for the life expectancy at age 65 as the outcome and the relative fraction of new policies passed in prior decade as the main state health policy predictor, by sex.

Table A.2.34. Spatial Durbin model (SDM) state-level impact decomposition for life expectancy at birth, by sex and SHP indicator.

		I_Z			$I_{t-10,Z}$			$\tilde{\pi}_{10,Z}$		
Sex	Regressor	Direct	Indirect	Total	Direct	Indirect	Total	Direct	Indirect	Total
Both sexes	SHP indicator	0.230***	0.123*	0.353**	0.214***	0.114*	0.327***	0.014	0.008	0.022
		(0.068)	(0.061)	(0.118)	(0.048)	(0.049)	(0.087)	(0.030)	(0.019)	(0.048)
	Median age	−0.045	−0.024	−0.069	−0.029	−0.015	−0.044	−0.051	−0.031	−0.082
		(0.043)	(0.026)	(0.067)	(0.041)	(0.024)	(0.064)	(0.044)	(0.030)	(0.072)
	log(gov't ideology)	0.065	0.034	0.099	−0.227	−0.122	−0.349	0.140	0.085	0.224
		(0.115)	(0.064)	(0.176)	(0.123)	(0.082)	(0.196)	(0.121)	(0.082)	(0.198)
	log(% in poor health)	0.040	0.023	0.063	0.644	0.344	0.988	−0.427	−0.253	−0.680
		(0.622)	(0.362)	(0.971)	(0.651)	(0.397)	(1.022)	(0.657)	(0.417)	(1.059)
	log(% Black)	−0.565***	−0.301*	−0.867**	−0.604***	−0.321*	−0.925***	−0.581***	−0.348*	−0.929**
		(0.164)	(0.145)	(0.282)	(0.154)	(0.143)	(0.267)	(0.176)	(0.160)	(0.309)
	D index	0.031	0.017	0.048	0.032	0.017	0.049	0.029	0.017	0.046
		(0.019)	(0.013)	(0.030)	(0.019)	(0.012)	(0.030)	(0.020)	(0.014)	(0.032)
	log(med. HH income)	−0.403	−0.210	−0.613	−0.372	−0.203	−0.575	−0.716	−0.442	−1.157
		(0.863)	(0.486)	(1.327)	(0.841)	(0.491)	(1.312)	(0.882)	(0.582)	(1.439)
	% blue collar	0.042	0.022	0.064	0.047	0.025	0.072	0.055	0.033	0.088
		(0.028)	(0.017)	(0.043)	(0.026)	(0.017)	(0.042)	(0.029)	(0.022)	(0.049)
	% urban population	0.010	0.005	0.015	0.012	0.006	0.018	0.017	0.010	0.027
		(0.015)	(0.008)	(0.023)	(0.015)	(0.008)	(0.022)	(0.015)	(0.010)	(0.025)
	log(% w/out HS diploma)	2.983***	1.590*	4.573***	2.709***	1.442*	4.151***	2.955***	1.760**	4.715***
		(0.544)	(0.652)	(1.046)	(0.559)	(0.606)	(1.026)	(0.559)	(0.677)	(1.084)
Female	SHP indicator	0.239***	0.111*	0.350**	0.225***	0.116*	0.341***	−0.009	−0.006	−0.015
		(0.066)	(0.053)	(0.106)	(0.046)	(0.048)	(0.082)	(0.030)	(0.020)	(0.049)
	Median age	0.009	0.004	0.013	0.033	0.017	0.050	0.023	0.014	0.037
		(0.038)	(0.019)	(0.056)	(0.037)	(0.021)	(0.056)	(0.038)	(0.025)	(0.062)
	log(gov't ideology)	0.115	0.054	0.170	−0.215	−0.111	−0.326	0.194	0.122	0.316
		(0.113)	(0.060)	(0.168)	(0.119)	(0.077)	(0.187)	(0.122)	(0.090)	(0.206)
	log(% in poor health)	0.174	0.082	0.256	1.065	0.552	1.617	0.042	0.021	0.064
		(0.656)	(0.332)	(0.972)	(0.645)	(0.410)	(1.012)	(0.687)	(0.442)	(1.118)
	log(% Black)	−0.657***	−0.304*	−0.960***	−0.699***	−0.361*	−1.059***	−0.644***	−0.402*	−1.045***
		(0.164)	(0.142)	(0.271)	(0.148)	(0.154)	(0.266)	(0.172)	(0.176)	(0.317)
	D index	0.035	0.017	0.052	0.036*	0.019	0.055	0.027	0.017	0.044
		(0.019)	(0.012)	(0.029)	(0.018)	(0.013)	(0.029)	(0.020)	(0.014)	(0.033)
	log(med. HH income)	−0.402	−0.183	−0.584	−0.416	−0.216	−0.632	−0.617	−0.390	−1.007
		(0.808)	(0.411)	(1.200)	(0.804)	(0.454)	(1.240)	(0.842)	(0.563)	(1.385)
	% blue collar	0.064*	0.030	0.094*	0.073**	0.038	0.111*	0.082**	0.051*	0.134**
		(0.027)	(0.018)	(0.042)	(0.027)	(0.021)	(0.044)	(0.028)	(0.025)	(0.050)
	% urban population	0.016	0.007	0.022	0.016	0.008	0.024	0.024	0.015	0.040
		(0.014)	(0.007)	(0.021)	(0.015)	(0.009)	(0.023)	(0.016)	(0.012)	(0.027)
	log(% w/out HS diploma)	2.146***	0.993*	3.139***	1.935***	0.997*	2.932**	2.177***	1.352*	3.529***
		(0.511)	(0.454)	(0.851)	(0.536)	(0.459)	(0.900)	(0.540)	(0.558)	(0.992)
Male	SHP indicator	0.240**	0.110	0.350**	0.197***	0.081*	0.278***	0.024	0.011	0.035
		(0.076)	(0.059)	(0.122)	(0.049)	(0.039)	(0.077)	(0.032)	(0.016)	(0.047)
	Median age	−0.056	−0.026	−0.081	−0.050	−0.021	−0.070	−0.073	−0.033	−0.105
		(0.050)	(0.027)	(0.075)	(0.049)	(0.024)	(0.070)	(0.050)	(0.027)	(0.075)
	log(gov't ideology)	0.056	0.025	0.080	−0.198	−0.082	−0.280	0.139	0.064	0.203
		(0.124)	(0.060)	(0.181)	(0.133)	(0.068)	(0.193)	(0.128)	(0.067)	(0.190)
	log(% in poor health)	0.706	0.326	1.031	0.789	0.322	1.111	0.210	0.095	0.305
		(0.573)	(0.324)	(0.866)	(0.581)	(0.286)	(0.837)	(0.585)	(0.283)	(0.855)
	log(% Black)	−0.498**	−0.228	−0.726**	−0.523**	−0.214	−0.737**	−0.531**	−0.240	−0.771**
		(0.177)	(0.127)	(0.280)	(0.171)	(0.116)	(0.259)	(0.187)	(0.129)	(0.291)
	D index	0.024	0.011	0.035	0.026	0.010	0.036	0.025	0.011	0.036
		(0.021)	(0.011)	(0.030)	(0.021)	(0.010)	(0.029)	(0.021)	(0.011)	(0.031)
	log(med. HH income)	0.137	0.058	0.194	0.101	0.045	0.146	−0.207	−0.089	−0.296
		(0.936)	(0.457)	(1.371)	(0.929)	(0.422)	(1.329)	(0.958)	(0.465)	(1.405)
	% blue collar	0.016	0.008	0.024	0.023	0.009	0.032	0.028	0.013	0.041
		(0.030)	(0.015)	(0.044)	(0.030)	(0.014)	(0.043)	(0.032)	(0.016)	(0.047)
	% urban population	0.016	0.007	0.023	0.018	0.007	0.026	0.022	0.010	0.032
		(0.016)	(0.008)	(0.024)	(0.016)	(0.007)	(0.022)	(0.016)	(0.009)	(0.024)
	log(% w/out HS diploma)	3.507***	1.602*	5.109***	3.348***	1.382*	4.730***	3.427***	1.541*	4.969***
		(0.563)	(0.680)	(1.054)	(0.596)	(0.646)	(1.064)	(0.594)	(0.654)	(1.066)

$^{*}p < 0.05$, $^{**}p < 0.01$, $^{***}p < 0.001$.

Table A.2.35. Spatial Durbin model (SDM) county-group level impact decomposition for for life expectancy at birth, by sex and SHP indicator.

		I_Z			$I_{t-10,Z}$			$\tilde{\pi}_{10,Z}$		
Sex	Regressor	Direct	Indirect	Total	Direct	Indirect	Total	Direct	Indirect	Total
Both sexes	SHP indicator	0.062	0.088	0.150	0.072*	0.104*	0.176*	0.013	0.020	0.033
		(0.045)	(0.064)	(0.108)	(0.034)	(0.049)	(0.083)	(0.021)	(0.031)	(0.052)
	Median age	0.046***	0.066***	0.111***	0.046***	0.066***	0.112***	0.046***	0.067***	0.113***
		(0.006)	(0.009)	(0.014)	(0.005)	(0.009)	(0.014)	(0.005)	(0.009)	(0.014)
	log(gov't ideology)	0.098	0.139	0.237	−0.075	−0.108	−0.184	0.125	0.183	0.308
		(0.088)	(0.126)	(0.214)	(0.092)	(0.133)	(0.225)	(0.087)	(0.129)	(0.216)
	log(% in poor health)	0.397*	0.567*	0.963*	0.405*	0.583*	0.988*	0.367*	0.540*	0.907*
		(0.169)	(0.244)	(0.413)	(0.170)	(0.247)	(0.417)	(0.166)	(0.247)	(0.413)
	log(% Black)	−0.024	−0.034	−0.058	−0.021	−0.030	−0.051	−0.021	−0.030	−0.051
		(0.015)	(0.021)	(0.036)	(0.015)	(0.022)	(0.036)	(0.015)	(0.022)	(0.037)
	log(D index)	0.106***	0.151***	0.257***	0.108***	0.156***	0.264***	0.107***	0.157***	0.264***
		(0.017)	(0.024)	(0.041)	(0.017)	(0.026)	(0.042)	(0.017)	(0.026)	(0.043)
	log(med. HH income)	2.047***	2.923***	4.970***	2.047***	2.950***	4.997***	2.049***	3.014***	5.063***
		(0.146)	(0.245)	(0.380)	(0.145)	(0.247)	(0.380)	(0.144)	(0.251)	(0.384)
	log(GINI index)	1.262***	1.803***	3.065***	1.250***	1.802***	3.052***	1.292***	1.901***	3.194***
		(0.201)	(0.300)	(0.498)	(0.196)	(0.295)	(0.488)	(0.204)	(0.313)	(0.513)
	% blue collar	0.008**	0.012*	0.020**	0.008**	0.012**	0.020**	0.008**	0.012**	0.021**
		(0.003)	(0.005)	(0.008)	(0.003)	(0.004)	(0.008)	(0.003)	(0.005)	(0.008)
	% urban population	0.011***	0.016***	0.027***	0.011***	0.016***	0.027***	0.011***	0.017***	0.028***
		(0.001)	(0.002)	(0.003)	(0.001)	(0.002)	(0.003)	(0.001)	(0.002)	(0.003)
	log(% w/out HS diploma)	−0.112	−0.159	−0.271	−0.118	−0.171	−0.289	−0.114	−0.168	−0.282
		(0.078)	(0.111)	(0.189)	(0.074)	(0.108)	(0.182)	(0.078)	(0.115)	(0.192)
Female	SHP indicator	0.046	0.072	0.117	0.064*	0.102*	0.166*	0.008	0.014	0.022
		(0.042)	(0.066)	(0.108)	(0.032)	(0.051)	(0.082)	(0.020)	(0.032)	(0.051)
	Median age	0.051***	0.080***	0.131***	0.051***	0.083***	0.134***	0.051***	0.082***	0.133***
		(0.005)	(0.008)	(0.013)	(0.005)	(0.008)	(0.013)	(0.005)	(0.009)	(0.013)
	log(gov't ideology)	0.130	0.203	0.333	−0.143	−0.230	−0.374	0.152	0.245	0.397
		(0.082)	(0.130)	(0.212)	(0.087)	(0.140)	(0.227)	(0.081)	(0.132)	(0.213)
	log(% in poor health)	0.772***	1.212***	1.984***	0.786***	1.261***	2.047***	0.751***	1.208***	1.960***
		(0.145)	(0.234)	(0.377)	(0.145)	(0.240)	(0.383)	(0.152)	(0.251)	(0.401)
	log(% Black)	−0.022	−0.034	−0.056	−0.020	−0.032	−0.052	−0.020	−0.032	−0.052
		(0.014)	(0.022)	(0.035)	(0.014)	(0.022)	(0.036)	(0.014)	(0.022)	(0.036)
	log(D index)	0.101***	0.158***	0.259***	0.103***	0.166***	0.269***	0.102***	0.164***	0.266***
		(0.016)	(0.025)	(0.040)	(0.016)	(0.026)	(0.042)	(0.016)	(0.026)	(0.042)
	log(med. HH income)	1.691***	2.653***	4.344***	1.700***	2.729***	4.429***	1.697***	2.729***	4.426***
		(0.133)	(0.236)	(0.360)	(0.134)	(0.244)	(0.369)	(0.131)	(0.242)	(0.364)
	log(GINI index)	1.294***	2.030***	3.325***	1.301***	2.088***	3.389***	1.320***	2.123***	3.443***
		(0.184)	(0.304)	(0.484)	(0.185)	(0.311)	(0.492)	(0.185)	(0.309)	(0.489)
	% blue collar	0.012***	0.019***	0.031***	0.012***	0.019***	0.031***	0.012***	0.020***	0.032***
		(0.003)	(0.005)	(0.007)	(0.003)	(0.005)	(0.008)	(0.003)	(0.005)	(0.008)
	% urban population	0.010***	0.016***	0.026***	0.010***	0.017***	0.027***	0.011***	0.017***	0.028***
		(0.001)	(0.002)	(0.002)	(0.001)	(0.002)	(0.003)	(0.001)	(0.002)	(0.003)
	log(% w/out HS diploma)	−0.109	−0.170	−0.279	−0.115	−0.185	−0.301	−0.110	−0.177	−0.288
		(0.072)	(0.114)	(0.186)	(0.071)	(0.115)	(0.185)	(0.071)	(0.115)	(0.186)
Male	SHP indicator	0.084	0.108	0.192	0.080*	0.103*	0.183*	0.013	0.018	0.031
		(0.048)	(0.062)	(0.109)	(0.036)	(0.047)	(0.083)	(0.023)	(0.030)	(0.053)
	Median age	0.029***	0.037***	0.065***	0.028***	0.037***	0.065***	0.028***	0.037***	0.066***
		(0.006)	(0.008)	(0.013)	(0.006)	(0.007)	(0.013)	(0.006)	(0.008)	(0.014)
	log(gov't ideology)	0.069	0.089	0.159	−0.023	−0.029	−0.052	0.102	0.136	0.238
		(0.095)	(0.123)	(0.217)	(0.099)	(0.129)	(0.229)	(0.093)	(0.124)	(0.217)
	log(% in poor health)	−0.027	−0.034	−0.061	−0.013	−0.016	−0.029	−0.049	−0.065	−0.115
		(0.160)	(0.207)	(0.366)	(0.159)	(0.206)	(0.364)	(0.157)	(0.210)	(0.367)
	log(% Black)	−0.025	−0.032	−0.057	−0.022	−0.028	−0.050	−0.021	−0.028	−0.049
		(0.016)	(0.020)	(0.036)	(0.015)	(0.020)	(0.035)	(0.016)	(0.021)	(0.037)
	log(D index)	0.128***	0.165***	0.293***	0.130***	0.169***	0.299***	0.130***	0.172***	0.302***
		(0.018)	(0.024)	(0.042)	(0.018)	(0.025)	(0.042)	(0.018)	(0.025)	(0.042)
	log(med. HH income)	2.253***	2.912***	5.164***	2.260***	2.925***	5.185***	2.257***	3.000***	5.257***
		(0.156)	(0.244)	(0.387)	(0.155)	(0.242)	(0.384)	(0.154)	(0.246)	(0.387)
	log(GINI index)	1.209***	1.563***	2.771***	1.192***	1.543***	2.735***	1.240***	1.649***	2.889***
		(0.214)	(0.288)	(0.499)	(0.217)	(0.290)	(0.504)	(0.219)	(0.302)	(0.518)
	% blue collar	0.007*	0.009*	0.016*	0.007*	0.009*	0.016*	0.007*	0.010*	0.017*
		(0.003)	(0.004)	(0.008)	(0.003)	(0.004)	(0.008)	(0.003)	(0.005)	(0.008)
	% urban population	0.011***	0.014***	0.025***	0.011***	0.014***	0.025***	0.011***	0.015***	0.026***
		(0.001)	(0.002)	(0.003)	(0.001)	(0.001)	(0.002)	(0.001)	(0.002)	(0.003)
	log(% w/out HS diploma)	−0.053	−0.068	−0.121	−0.057	−0.073	−0.130	−0.056	−0.074	−0.130
		(0.082)	(0.106)	(0.188)	(0.083)	(0.108)	(0.191)	(0.081)	(0.108)	(0.190)

$^{*}p < 0.05$, $^{**}p < 0.01$, $^{***}p < 0.001$.

Table A.2.36. Spatial Durbin model (SDM) state-level impact decomposition for the life expectancy at age 65, by sex and SHP indicator.

		I_Z			$I_{t-10,Z}$			$\tilde{\pi}_{10,Z}$		
Sex	Regressor	Direct	Indirect	Total	Direct	Indirect	Total	Direct	Indirect	Total
Both sexes	SHP indicator	0.115***	0.072*	0.187***	0.084***	0.066*	0.150***	−0.017	−0.015	−0.032
		(0.031)	(0.030)	(0.056)	(0.022)	(0.027)	(0.045)	(0.014)	(0.013)	(0.026)
	Median age	0.011	0.007	0.018	0.020	0.016	0.035	0.009	0.008	0.017
		(0.020)	(0.013)	(0.033)	(0.020)	(0.017)	(0.036)	(0.020)	(0.018)	(0.038)
	log(gov't ideology)	0.100	0.062	0.162	−0.109	−0.086	−0.195	0.146**	0.122*	0.268*
		(0.054)	(0.040)	(0.091)	(0.060)	(0.055)	(0.112)	(0.054)	(0.059)	(0.107)
	log(% in poor health)	−0.430	−0.269	−0.699	−0.249	−0.196	−0.446	−0.561	−0.466	−1.027
		(0.291)	(0.213)	(0.490)	(0.310)	(0.266)	(0.568)	(0.303)	(0.294)	(0.578)
	log(% Black)	−0.303***	−0.189*	−0.492***	−0.335***	−0.264**	−0.599***	−0.282***	−0.236*	−0.518**
		(0.076)	(0.078)	(0.141)	(0.074)	(0.095)	(0.155)	(0.084)	(0.100)	(0.173)
	D index	0.005	0.003	0.007	0.005	0.004	0.009	0.000	0.000	0.000
		(0.008)	(0.006)	(0.014)	(0.009)	(0.008)	(0.016)	(0.009)	(0.008)	(0.017)
	log(med. HH income)	−0.650	−0.406	−1.056	−0.688	−0.543	−1.231	−0.852*	−0.709	−1.562*
		(0.386)	(0.292)	(0.657)	(0.400)	(0.366)	(0.744)	(0.405)	(0.402)	(0.779)
	% blue collar	0.039**	0.024*	0.064**	0.044***	0.034*	0.078**	0.048***	0.040*	0.088**
		(0.012)	(0.011)	(0.022)	(0.013)	(0.014)	(0.025)	(0.014)	(0.017)	(0.029)
	% urban population	0.005	0.003	0.008	0.005	0.004	0.010	0.008	0.007	0.015
		(0.007)	(0.004)	(0.011)	(0.007)	(0.006)	(0.013)	(0.007)	(0.006)	(0.013)
	log(% w/out HS diploma)	1.030***	0.641*	1.671***	1.010***	0.794*	1.804***	1.058***	0.884**	1.942***
		(0.250)	(0.258)	(0.462)	(0.265)	(0.310)	(0.531)	(0.258)	(0.335)	(0.548)
Female	SHP indicator	0.127***	0.077*	0.204**	0.099***	0.074*	0.173***	−0.024	−0.019	−0.044
		(0.035)	(0.033)	(0.062)	(0.026)	(0.030)	(0.052)	(0.016)	(0.014)	(0.030)
	Median age	0.022	0.014	0.036	0.037	0.027	0.064	0.032	0.026	0.058
		(0.021)	(0.014)	(0.034)	(0.021)	(0.019)	(0.039)	(0.021)	(0.019)	(0.039)
	log(gov't ideology)	0.139*	0.084	0.223*	−0.103	−0.076	−0.178	0.189**	0.150*	0.338**
		(0.061)	(0.048)	(0.104)	(0.068)	(0.056)	(0.121)	(0.064)	(0.069)	(0.126)
	log(% in poor health)	−0.360	−0.220	−0.580	0.036	0.024	0.060	−0.379	−0.301	−0.680
		(0.362)	(0.248)	(0.599)	(0.371)	(0.289)	(0.653)	(0.377)	(0.328)	(0.695)
	log(% Black)	−0.438***	−0.267*	−0.705***	−0.490***	−0.363**	−0.853***	−0.412***	−0.325**	−0.738***
		(0.093)	(0.105)	(0.178)	(0.090)	(0.128)	(0.195)	(0.096)	(0.123)	(0.200)
	D index	0.007	0.004	0.012	0.009	0.007	0.015	0.001	0.001	0.001
		(0.010)	(0.007)	(0.016)	(0.010)	(0.008)	(0.018)	(0.010)	(0.009)	(0.019)
	log(med. HH income)	−0.745	−0.452	−1.196	−0.780	−0.582	−1.362	−0.870	−0.685	−1.554
		(0.441)	(0.315)	(0.729)	(0.469)	(0.411)	(0.856)	(0.461)	(0.423)	(0.855)
	% blue collar	0.048**	0.029*	0.076**	0.053***	0.039*	0.092**	0.059***	0.047*	0.106***
		(0.015)	(0.014)	(0.027)	(0.015)	(0.016)	(0.029)	(0.015)	(0.018)	(0.031)
	% urban population	0.003	0.002	0.006	0.004	0.003	0.008	0.009	0.007	0.015
		(0.008)	(0.005)	(0.013)	(0.008)	(0.007)	(0.015)	(0.008)	(0.007)	(0.015)
	log(% w/out HS diploma)	1.052***	0.642*	1.694***	0.997**	0.740*	1.736**	1.100***	0.871*	1.971**
		(0.282)	(0.276)	(0.512)	(0.308)	(0.323)	(0.592)	(0.297)	(0.353)	(0.606)
Male	SHP indicator	0.118***	0.054*	0.172***	0.073***	0.042*	0.115***	−0.015	−0.009	−0.024
		(0.029)	(0.025)	(0.048)	(0.020)	(0.018)	(0.035)	(0.013)	(0.009)	(0.021)
	Median age	0.026	0.012	0.039	0.026	0.015	0.041	0.019	0.012	0.031
		(0.020)	(0.011)	(0.030)	(0.019)	(0.013)	(0.031)	(0.020)	(0.014)	(0.034)
	log(gov't ideology)	0.095	0.044	0.139	−0.080	−0.045	−0.125	0.138**	0.088*	0.227*
		(0.051)	(0.031)	(0.077)	(0.055)	(0.036)	(0.088)	(0.050)	(0.044)	(0.089)
	log(% in poor health)	−0.068	−0.029	−0.097	−0.144	−0.081	−0.224	−0.247	−0.156	−0.403
		(0.223)	(0.109)	(0.327)	(0.236)	(0.144)	(0.375)	(0.234)	(0.163)	(0.389)
	log(% Black)	−0.163*	−0.076	−0.239*	−0.175*	−0.099	−0.274*	−0.140	−0.089	−0.228
		(0.071)	(0.046)	(0.110)	(0.072)	(0.053)	(0.119)	(0.076)	(0.058)	(0.129)
	D index	0.002	0.001	0.003	0.001	0.000	0.001	−0.002	−0.002	−0.004
		(0.008)	(0.004)	(0.012)	(0.008)	(0.005)	(0.013)	(0.008)	(0.006)	(0.014)
	log(med. HH income)	−0.341	−0.158	−0.499	−0.401	−0.226	−0.626	−0.564	−0.356	−0.920
		(0.364)	(0.187)	(0.537)	(0.366)	(0.228)	(0.581)	(0.383)	(0.280)	(0.644)
	% blue collar	0.020	0.009	0.029	0.026*	0.014	0.040*	0.028*	0.018	0.047*
		(0.012)	(0.007)	(0.018)	(0.012)	(0.009)	(0.020)	(0.013)	(0.011)	(0.022)
	% urban population	0.011	0.005	0.016	0.011	0.006	0.017	0.014*	0.009	0.023*
		(0.006)	(0.004)	(0.010)	(0.007)	(0.004)	(0.011)	(0.007)	(0.005)	(0.011)
	log(% w/out HS diploma)	1.054***	0.487*	1.541***	1.118***	0.637*	1.755***	1.070***	0.680*	1.750***
		(0.219)	(0.219)	(0.382)	(0.248)	(0.263)	(0.458)	(0.238)	(0.267)	(0.457)

$^{*}p < 0.05$, $^{**}p < 0.01$, $^{***}p < 0.001$.

Table A.2.37. Spatial Durbin model (SDM) county-group level impact decomposition for the life expectancy at age 65, by sex and SHP indicator.

		I_Z			$I_{t-10,Z}$			$\tilde{\pi}_{10,Z}$		
Sex	Regressor	Direct	Indirect	Total	Direct	Indirect	Total	Direct	Indirect	Total
Both sexes	SHP indicator	0.005	0.006	0.011	0.037*	0.048*	0.086*	0.012	0.016	0.028
		(0.025)	(0.031)	(0.056)	(0.019)	(0.024)	(0.043)	(0.012)	(0.015)	(0.027)
	Median age	0.053***	0.067***	0.120***	0.054***	0.070***	0.124***	0.054***	0.069***	0.123***
		(0.003)	(0.005)	(0.008)	(0.003)	(0.005)	(0.008)	(0.003)	(0.005)	(0.008)
	log(gov't ideology)	0.069	0.087	0.156	0.036	0.047	0.083	0.070	0.090	0.160
		(0.049)	(0.061)	(0.110)	(0.051)	(0.067)	(0.118)	(0.048)	(0.063)	(0.111)
	log(% in poor health)	0.672***	0.840***	1.512***	0.677***	0.883***	1.560***	0.657***	0.848***	1.505***
		(0.094)	(0.122)	(0.214)	(0.095)	(0.130)	(0.222)	(0.092)	(0.124)	(0.213)
	log(% Black)	−0.006	−0.008	−0.015	−0.005	−0.007	−0.012	−0.005	−0.006	−0.012
		(0.008)	(0.010)	(0.019)	(0.008)	(0.011)	(0.019)	(0.008)	(0.010)	(0.018)
	log(D index)	0.041***	0.052***	0.093***	0.043***	0.056***	0.100***	0.042***	0.054***	0.097***
		(0.009)	(0.011)	(0.020)	(0.009)	(0.012)	(0.021)	(0.009)	(0.012)	(0.021)
	log(med. HH income)	0.988***	1.235***	2.222***	0.995***	1.299***	2.294***	0.985***	1.271***	2.256***
		(0.081)	(0.115)	(0.190)	(0.080)	(0.120)	(0.195)	(0.080)	(0.119)	(0.194)
	log(GINI index)	0.845***	1.056***	1.901***	0.857***	1.119***	1.976***	0.868***	1.121***	1.989***
		(0.111)	(0.149)	(0.257)	(0.109)	(0.150)	(0.256)	(0.113)	(0.154)	(0.264)
	% blue collar	0.004*	0.004*	0.008*	0.004*	0.005*	0.008*	0.004*	0.005*	0.008*
		(0.002)	(0.002)	(0.004)	(0.002)	(0.002)	(0.004)	(0.002)	(0.002)	(0.004)
	% urban population	0.007***	0.008***	0.015***	0.007***	0.009***	0.015***	0.007***	0.009***	0.016***
		(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)
	log(% w/out HS diploma)	0.000	0.000	0.001	−0.004	−0.005	−0.009	−0.002	−0.003	−0.005
		(0.043)	(0.054)	(0.097)	(0.041)	(0.054)	(0.095)	(0.043)	(0.056)	(0.099)
Female	SHP indicator	0.000	0.000	0.001	0.034	0.049	0.083	0.005	0.007	0.012
		(0.026)	(0.035)	(0.061)	(0.020)	(0.028)	(0.048)	(0.012)	(0.017)	(0.029)
	Median age	0.051***	0.069***	0.120***	0.052***	0.073***	0.125***	0.051***	0.071***	0.122***
		(0.003)	(0.005)	(0.008)	(0.003)	(0.005)	(0.008)	(0.003)	(0.005)	(0.007)
	log(gov't ideology)	0.083	0.113	0.196	0.002	0.003	0.004	0.086	0.119	0.205
		(0.052)	(0.070)	(0.121)	(0.054)	(0.077)	(0.132)	(0.051)	(0.070)	(0.121)
	log(% in poor health)	0.680***	0.917***	1.597***	0.688***	0.977***	1.665***	0.670***	0.922***	1.592***
		(0.091)	(0.127)	(0.216)	(0.091)	(0.135)	(0.223)	(0.092)	(0.131)	(0.221)
	log(% Black)	−0.013	−0.018	−0.031	−0.012	−0.017	−0.029	−0.012	−0.016	−0.027
		(0.008)	(0.011)	(0.020)	(0.009)	(0.012)	(0.021)	(0.009)	(0.012)	(0.020)
	log(D index)	0.040***	0.053***	0.093***	0.041***	0.059***	0.100***	0.040***	0.056***	0.096***
		(0.010)	(0.013)	(0.022)	(0.010)	(0.014)	(0.023)	(0.009)	(0.013)	(0.023)
	log(med. HH income)	0.834***	1.125***	1.959***	0.844***	1.199***	2.043***	0.833***	1.147***	1.979***
		(0.083)	(0.124)	(0.203)	(0.084)	(0.132)	(0.212)	(0.085)	(0.128)	(0.210)
	log(GINI index)	0.829***	1.119***	1.948***	0.849***	1.205***	2.054***	0.852***	1.173***	2.025***
		(0.115)	(0.162)	(0.275)	(0.116)	(0.172)	(0.284)	(0.116)	(0.170)	(0.282)
	% blue collar	0.006***	0.009***	0.015***	0.006***	0.009***	0.015***	0.007***	0.009***	0.015***
		(0.002)	(0.003)	(0.004)	(0.002)	(0.003)	(0.004)	(0.002)	(0.003)	(0.004)
	% urban population	0.006***	0.008***	0.015***	0.006***	0.009***	0.015***	0.006***	0.009***	0.015***
		(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.002)	(0.001)	(0.001)	(0.001)
	log(% w/out HS diploma)	0.023	0.031	0.054	0.018	0.025	0.043	0.020	0.028	0.048
		(0.045)	(0.061)	(0.106)	(0.044)	(0.063)	(0.108)	(0.043)	(0.059)	(0.102)
Male	SHP indicator	0.014	0.015	0.029	0.043*	0.050*	0.093*	0.018	0.021	0.039
		(0.024)	(0.027)	(0.052)	(0.018)	(0.021)	(0.039)	(0.011)	(0.013)	(0.025)
	Median age	0.040***	0.045***	0.085***	0.041***	0.046***	0.087***	0.040***	0.046***	0.087***
		(0.003)	(0.004)	(0.007)	(0.003)	(0.004)	(0.006)	(0.003)	(0.004)	(0.007)
	log(gov't ideology)	0.042	0.047	0.089	0.071	0.082	0.153	0.044	0.051	0.095
		(0.048)	(0.053)	(0.100)	(0.050)	(0.057)	(0.107)	(0.046)	(0.054)	(0.100)
	log(% in poor health)	0.360***	0.399***	0.759***	0.368***	0.420***	0.788***	0.348***	0.400***	0.748***
		(0.080)	(0.090)	(0.169)	(0.079)	(0.093)	(0.171)	(0.079)	(0.092)	(0.170)
	log(% Black)	−0.006	−0.007	−0.014	−0.005	−0.005	−0.010	−0.005	−0.006	−0.010
		(0.008)	(0.009)	(0.016)	(0.008)	(0.009)	(0.017)	(0.008)	(0.009)	(0.016)
	log(D index)	0.046***	0.051***	0.096***	0.047***	0.054***	0.101***	0.046***	0.053***	0.099***
		(0.009)	(0.010)	(0.018)	(0.009)	(0.010)	(0.019)	(0.009)	(0.010)	(0.019)
	log(med. HH income)	0.963***	1.069***	2.032***	0.974***	1.112***	2.086***	0.963***	1.106***	2.068***
		(0.078)	(0.101)	(0.174)	(0.077)	(0.102)	(0.174)	(0.077)	(0.103)	(0.175)
	log(GINI index)	0.923***	1.024***	1.947***	0.930***	1.062***	1.992***	0.943***	1.083***	2.027***
		(0.110)	(0.132)	(0.238)	(0.106)	(0.130)	(0.232)	(0.110)	(0.136)	(0.242)
	% blue collar	0.004**	0.005*	0.009**	0.005**	0.005**	0.010**	0.005**	0.005**	0.010**
		(0.002)	(0.002)	(0.004)	(0.002)	(0.002)	(0.004)	(0.002)	(0.002)	(0.004)
	% urban population	0.007***	0.007***	0.014***	0.007***	0.008***	0.014***	0.007***	0.008***	0.015***
		(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)
	log(% w/out HS diploma)	−0.020	−0.023	−0.043	−0.023	−0.026	−0.049	−0.022	−0.026	−0.048
		(0.041)	(0.045)	(0.086)	(0.041)	(0.047)	(0.089)	(0.041)	(0.047)	(0.088)

$^{*}p < 0.05$, $^{**}p < 0.01$, $^{***}p < 0.001$.