Health and Economic Geography in the US

Policy Interventions for Public Health

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A thesis presented for the degree of
Doctor of Philosophy

Department of Economics
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Greece

2023

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Abstract

Despite the fact that health and wealth are very much related, health policies tend to ignore the social and economic mechanisms behind health disparities. In this thesis I examine two subjects: premature mortality, and COVID-19 government interventions. To elaborate, I first study whether premature mortality, measured by the years of potential life lost (*YPLL*), converges among the U.S. states, by gender and by race. Based on these results, I also examine which mortalities, as well as health spending components, might have led to divergence. A novel convergence methodology is employed to this end for the years 1979–2017. Findings suggest that for males and blacks, all U.S. states converge to a steady-state, while for females, whites, and total population, the states form convergence clubs. These clubs differ mainly in infant, cardiovascular, and unintentional injury mortalities, with the ones with the lesser *YPLL* located mainly on the west and east coast. In conclusion, preventable deaths seem to be the main driver of premature mortality and spending on health does not appear to play a major role.

Second, I study the association between the COVID-19 pandemic government responses and the equity-efficiency relation. More specifically, wage inequality is the inequality in question. Cross-sectional data from the contiguous US states for the year 2020 and a spatial econometric model specification were the data and the method used for the analysis, respectively. The main finding is that the association of State government responses to COVID-19 with the relation depends on the per capita income of the States. Additionally, an inverted-U relationship between wage inequality and efficiency was found. These heterogenous effects may play a role into regional integration.

Dedication

To Taba, long may you run

Declaration

I declare that the thesis has been composed by myself and that the work has not been submitted for any other degree or professional qualification. I confirm that the work submitted is my own, except where work which has formed part of jointly-authored publications has been included. My contribution and those of the other authors to this work have been explicitly indicated below. I confirm that appropriate credit has been given within this thesis where reference has been made to the work of others.

The work presented in Chapter 2 was previously published in *Social Science & Medicine* as 'Premature mortality in the US: A convergence study' by Christopoulos K. (thesis author) and Eleftheriou K., the supervisor of the thesis. This study was conceived by all of the authors. I carried out the conceptualization, investigation, data curation, writing the original draft, reviewing and editing of the final manuscript. The work presented in Chapter 3 was previously published in *Regional studies* as 'The Equity-Efficiency Trade-off and the Intensity of COVID-19 Pandemic Government Responses: Evidence from US States' by Christopoulos K., Eleftheriou K. and Nijkamp P. This study was conceived by all authors. I carried out the conceptualization, investigation, writing the original draft, reviewing and editing of the final manuscript.

Acknowledgements

I want to thank all those who contributed directly or indirectly for the completion of this thesis. First, my supervisor, associate professor Konstantinos Eleftheriou with whom I achieved academic goals once thought impossible given the circumstances. Next, I want to thank my uncle, professor emeritus Christos Skourlas who supported and guided me throughout this academic odyssey. My parents, Panos and Eleni, who remained supportive despite not being fond of the idea of me as a PhD student, with zero income for at least another 3 years, and my other family Evi, Kostakis and Josephine for showing me the light when I only saw darkness. Last but not least, I want to thank Spyros Kapitsinas for saving my academic career at a time when nothing seemed to be heading to where I am now. I also owe a massive thanks to the research community responsible for free resources, software development, and the support of its users.

Contents

List of Figures

List of Tables

Chapter 1

Introduction

A positive association between wealth and health is well established at the micro and macro-level (Pollack et al., 2007). So much so, that a potential redistribution of income could blunt health disparities without us interfering at all with health policy (Deaton, 2002). Although some studies disprove this last hypothesis (Semyonov et al., 2013), others have shown that income inequality is a predictor of health outcomes, regardless of the individuals' socioeconomic status (Kennedy et al., 1998).[1] Nevertheless, some pathologies known as 'diseases of the affluence'[2] (e.g., cancer, type 2 diabetes, cardiovascular disease) is proof that this is not a simple monotonic relationship, but one with intricacies that require special attention.

Although micro evidence is always more preferable for health outcomes—so that ecological fallacies can be avoided—policy decisions are taken on a macro level. At the regional level, disparities in wealth and health cannot be regarded as independent. Disparities can be found when studying the health geography of macro and micro regional units. These are usually the result of socioeconomic and cultural factors that have operated over the years, and thus created divergence in population health outcomes.

Access to healthcare is perhaps another regional determinant of health dispar-

[1] See Subramanian and Kawachi (2004) for a review on income inequality and health.

[2] The term 'diseases of the affluence' gradually loses its validity since most of the products detrimental to health have already penetrated the markets of developing countries and are heavily marketed toward vulnerable populations. See Gómez (2021) for a discussion of the case of Latin America.

ities (Brezzi and Luongo, 2016). Of course, access to care itself is determined by other regional characteristics such as income and unemployment. Another possible mechanism through which geography could influence health disparities is the quality of care. If this mechanism is in place, then regardless of access to care, health disparities will be formed (Chandra and Skinner, 2003). It is therefore evident that the economic situation of a region can affect health via multiple pathways.

The hypothesised relations between these three variables, namely, health, equity, and efficiency are depicted in the form of a Directed Acyclic Graph.[3]

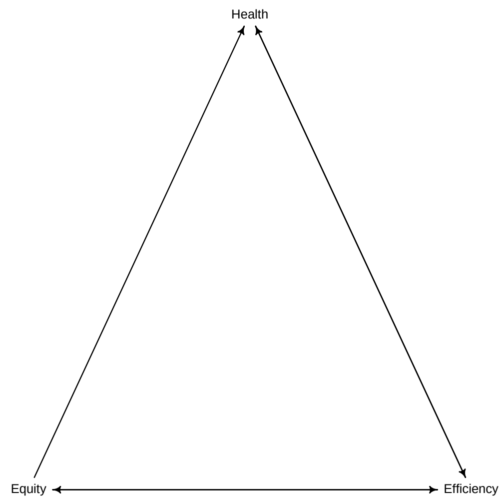


Figure 1.1: Hypothesized relations between health, equity, and efficiency

This thesis is going to cover mainly two subjects; one focusing on the health geography, and one focusing on the economic geography of US states. As previously mentioned, these two subjects are not independent, but rather entangled with complex mechanisms. The health geography part focuses on premature mortality—measured by the years of potential life lost—and how different mortalities and health spending affect the trends. The economic geography part focuses on the equity-efficiency relation and how this was affected by the recent COVID-19 government interventions. The setting, as already mentioned, is the US states plus the District of Columbia (DC). The study of the premature mortality is a panel data analysis for the years 1979–2017 and the study of the equity-efficiency relation is a cross-sectional study

[3]The graph was created with the *R* package *'daggitty'*. For a detailed explanation of these graphs McElreath (see 2020, Chapter 6).

for the year 2020. The convergence analysis was performed using the Phillips and Sul (2007) and Phillips and Sul (2009) methodology, while for the cross-sectional analysis a spatial econometrics approach was utilized.

The aforementioned can be summarized in the following research hypotheses/questions: For the first part,

1. Is premature mortality converging across all US states for different genders and races, and if not which are the convergent clubs?

2. Which of the main mortalities are behind the disparities in premature mortality?

3. Is there a difference in health spending among convergent clubs?

and for the second part,

1. Is there a modification effect on the equity-efficiency relation by the COVID-19 government responses?

2. Are the COVID-19 government responses associated with wage inequality?

Premature mortality is an important public health indicator with ramifications to social and economic outcomes. This first part aims to aid regional and national public health policy in the effort to reduce premature mortality and to identify whether interstate disparities between sexes and races exist. Moreover, the assessment of the relation between health spending and premature mortality will provide insights regarding value in health. The relation between equity and efficiency remains at the heart of regional policy. The second part aims to provide valuable information on how the distorting force of the governments' interventions can affect the equity-efficiency relation.

The US setting was preferred since there exists large within-country variation in health and economic outcomes. The peculiarities of the American healthcare system create mechanisms which amplify the disparities in health when inequalities in wealth exist, making US an especially interesting case (Subramanian and Kawachi, 2004).[4] Secondarily, data availability is ample. US states were preferred to a more disaggregate unit of analysis (e.g., counties) due to data censoring issues that would lead to unreliable estimates for premature mortality in the first part of the thesis, and due to data availability issues for the second part. The particularity of this geographical choice of course limits the transportability of the findings to mostly developed countries with large health and economic disparities within.

For the health geography part, the focus on premature mortality rather than plain mortality was in order to better capture the burden of deaths for the society and economy. The years of potential life lost were preferred to better quantify premature mortality and the time period was selected so as to properly conduct a convergence analysis. For economic geography part, the year 2020 was chosen since it was the first year of the pandemic and 2021 data or later, were not available.

The rest of the thesis has the following structure: Chapter 2 studies the convergence patterns and drivers of premature mortality across US states. In Chapter 3 the association between of the COVID-19 government responses and the equity-efficiency relation is examined. The final chapter draws some final conclusions from the previous two chapters.

[4] See Semyonov et al. (2013) for some empirical evidence on this matter.

Chapter 2

Disparities and trends of premature mortality between US states

2.1 Introduction

US premature mortality rates have been declining in the previous decades as a result of advances in medicine and hygiene (Krieger et al., 2008). Premature mortality is defined as the number of deaths occurring before a certain age limit. The age limit most frequently chosen is 75 years but other limits such as life expectancy at birth can be used. Its difference with standard mortality rates is that premature mortality places emphasis on deaths that occur at younger ages. Therefore, it provides information that is not dominated by the mortality of the elderly and can be used as a measure of the burden of a disease. It is also a valuable tool for public health policy makers since it enables them not to just monitor but also intervene and prevent premature deaths. From a socioeconomic perspective, premature mortality affects negatively the age-distribution of the population which in result leads to less productivity, decreased output and fiscal strains (Alkire et al., 2018).

In the United States, the main contributing mortalities in 2017 according to *CDC's* (Centers for Disease Control and Prevention) *WISQARS* (Web-based Injury Statistics Query and Reporting System), were the malignant neoplasms (cancers) followed by the unintentional injuries, cardiovascular deaths, suicide deaths, and deaths in perinatal period. Of these cause-specific mortalities, perinatal death is the main contributor to premature mortality and the number one priority for health policy, especially in countries where infant mortality rates remain high. In developed countries, policy focuses mainly on preventing and treating cardiovascular and cancer pathologies. Although a health issue, premature mortality has deep social and economic roots (Doubeni et al., 2012).

The importance of studying the phenomenon of premature mortality was acknowledged by the *CDC* in 1982, when it started to include in its tables the potential years of life lost (*YPLL*) as a measure of premature mortality (Gardner and Sanborn, 1990). The *YPLL* quantify the burden of loss by weighting deaths that occur at the earlier stages of life more (McDonnell et al., 1998). Consequently, infant and perinatal mortality contribute the most years. The *YPLL* can be used also as a measure of health performance as well as a health spending efficiency index (Christopoulos and Eleftheriou, 2020b).

The concept of convergence is popular in economics but recent studies have applied econometric convergence methodologies to study health indicators as well (Christopoulos and Eleftheriou, 2020a; Christopoulos et al., 2022; Duncan and Toledo, 2019; González-Álvarez et al., 2020; López-Mendoza et al., 2021). These panel data methods are more robust than the traditional trend analyses found in epidemiological studies which compare cross-sections temporaly. Converge analysis is an important tool for regional policy due to its ability to identify divergent entities that either excel, and therefore can be used as a good example, or entities that are left behind and are in need of integration policies in order to converge. At this point, I should note that the convergence of a variable is neither an improvement nor a deterioration per se, but an indicator that a situation is becoming similar cross-sectionally across time.[1]

In this chapter I study the convergence of premature mortality, measured by *YPLL*, for the U.S. states as a total, by gender and by race (black, white and

[1] Extra attention is need when interpreting convergence results from an entities' prespective. For example, consider an imaginary health indicator 'H'. If we have convergence in H some entities are probably improving, while others are probably worsening. There is also the case that some entities are converging toward a steady trend, either desirable or not.

Hispanic). I use the Phillips and Sul (2007) and Phillips and Sul (2009) approach which is a state-of-art econometric methodology for analysing convergence trends and identifying clubs. I also examine various mortality factors, as well as health spending indicators, that may drive the results for the occasions where there is no full sample convergence. This chapter contributes to the literature since it is the first study, to the best of my knowledge, that examines the convergence of a weighted premature mortality indicator (i.e., *YPLL*) in the United States examining data on a disaggregated level (U.S. states) not only for the total population but also for different demographic groups.

The rest of the chapter is organised as follows: The next section provides a literature review of studies that have applied econometric methods to study health indicators as well as studies that address the issue of premature mortality in the United States. Section 2.3 describes the material and the methods of the study including the *YPLL* calculation and the convergence methodology. Section 2.4 presents the results and Section 2.5 discusses them. In the final section I draw my conclusions.

2.2 Literature review

2.2.1 Convergence of health outcomes

The convergence of health outcomes has been widely examined in the literature. In the United States, studies on specific health indicators such as the mortality of African Americans (Naghshpour and Sameem, 2019) and suicide rates (Kitenge et al., 2019) have found convergence across all states with the use of β-convergence. On a global scale, Clark (2011) examined the convergence in world health as a result of economic growth, using life expectancy and infant mortality as health indicators, with mixed results. Duncan and Toledo (2019) also found that countries converge in clubs in terms of the body mass index (*BMI*), while for children, González-Álvarez et al. (2020) found convergence in overweight but not in obesity prevalence.

For the European Union, Nixon et al. (2000) first studied the convergence of infant mortality and life expectancy along with that of healthcare expenditure and more recently, Weber and Clerc (2017) studied the convergence of deaths amenable to healthcare with both studies showing convergence. Panopoulou and Pantelidis (2012) studied the convergence of healthcare expenditure and health outcomes including *YPLL* for 19 *OECD* countries between 1972 and 2006 and found that countries converge into different clubs depending on the variable under consideration. Recent studies had their focus on COVID-19 deaths and cases (Christopoulos et al., 2022; López-Mendoza et al., 2021).

2.2.2 Premature mortality in the US

The effect of several mortalities and health factors on premature mortality has been previously studied; from cancer (Song et al., 2021a) to cardiovascular outcomes, such as myocardial infraction (Dani et al., 2022) and stroke (Song et al., 2021b). In an earlier study (Rockett and Smith, 1987) had highlighted the importance of injury deaths in the US premature mortality, and more recently, (Shiels et al., 2019) outline the contribution of deaths from drug poisonings. Mental health outcomes such as schizophrenia (Olfson et al., 2015) have also been the subject of investigation. Regarding risk factors, (Hirko et al., 2015) studied the effects of obesity and being overweight.

While health outcome and risk factor studies have their fair share, studies on the socioeconomic determinants of premature mortality are the most popular. Several studies point to income inequality as the main determinant at the city (Cooper et al., 2016; Ronzio et al., 2004) and county level (Cheng and Kindig, 2012; Song et al., 2021a; Song et al., 2020). Other socioeconomic factors such as education have been also found to influence premature mortality (Ma et al., 2022; Mansfield et al., 1999; Roy et al., 2020; Song et al., 2021a). Finally, Song et al. (2021a) point also to an association with the unemployment rate at the county level.

Research has been performed also for racial determinants. Although these predictors are not independent of the socioeconomic and health status, several studies reveal large disparities between black, hispanic and white populations (Cullen et al., 2012; Iribarren et al., 2005; Kiang et al., 2019; Krieger et al., 2014; Mansfield et al., 1999). Historical factors such as segregation laws have been pointed as a culprit for the disparities in premature mortality of the black population (Cooper et al., 2016; Krieger et al., 2014). Cullen et al. (2012) reveal that the survival probability from birth to age 70 is lower for the black population regardless of sex. This result of increased risk for premature mortality is very much consistent in every research that addresses the subject. On the bright side, health outcomes for some chronic diseases have been improving for black and hispanic populations (Chen et al., 2019). Ma et al. (2022) also found recently that disparities in premature mortality between whites and blacks have decreased. Best et al. (2018) predict that premature mortality from cancer and CVD will decrease for the black and hispanic population, while deaths from suicide and injuries are expected to rise for the entire US population.

Disparities in premature mortality exist also between sexes (Iribarren et al., 2005). Despite the fact the women have a higher life expectancy than men, evidence shows that females are at higher risk. Cullen et al. (2012) again calculated smaller survival probabilities for white females, while Mansfield et al. (1999) found that female-headed household have increased *YPLL*. Nevertheless, demographic factors were not a major predictor of premature mortality.

Geographic variation and spatial patterns have been observed across states and counties (Dani et al., 2022; Kiang et al., 2019; Mansfield et al., 1999). The emerging pattern has South US as more prone to premature deaths, while states in the West and Northeast appear to have better outcomes (Dani et al., 2022; Mansfield et al., 1999). Rurality, is a factor that appears to influence differently premature mortality depending on the cause of death. Nevertheless, disparities are present between urban and rural populations (Ma et al., 2022). The connection of this geography with inequality, either in the form of economic or social, may explain these spatial patterns in US premature mortality.

Last but not least, there are deep concerns regarding the effect of the climate change on premature mortality. The numerous mechanisms via which human health is affected by climate change[2] will inevitably result in increased mortality, especially for vulnerable populations (Benevolenza and DeRigne, 2019). So far studies have focused on the increased air pollution, in the form of particulate matter and ozone, from the climate change (Dedoussi et al., 2020; Fang et al., 2013; Neumann et al., 2021; Silva et al., 2013). While air pollution has been linked to increase mortality and morbidity, several other aspects of the climate change that are expected to impact premature mortality in the future remain understudied.

Given the prior research on the premature mortality in the US, this chapter expands the existing knowledge by studying the convergence patterns of premature mortality with a measure that captures the burden of the death (*YPLL*). This will help identify the states which are in need of intervention. By gender and race analysis will aid in deciding whether discrimination, and therefore disparities, is a thing of the past. Studying the main mortalities that create divergent clubs and whether health spending and funding has any effect will assist in focusing on specific causes of death and resource allocation.

2.3 Material & Methods

2.3.1 Data

The variable I use to conduct the convergence analysis is the potential years of life lost. It was calculated from the mortality data of the *CDC's WONDER* database (CDC, 2018) for the years 1979 to 2017.[3] *YPLL* for the total population is calculated by aggregating the difference between a selected age limit and the age of every premature death. Though they are many options to choose for the age limit, 75 years were chosen as the cut-off value because this value is the most commonly used by CDC and other organizations such as the OECD for the calculation of $YPLL$.

[2]See Kim et al. (2014) for a review on the effect of climate change on human health.

[3]Data for Hispanic population -which is independent of the other racial categories- were available only between 1999 and 2017.

As a consequence, all deaths occurring at the age of 75 or after did not contribute to this index. In order to ensure the comparability of our data, the *YPLL* were age-adjusted (using 2000 as the base year) to account for different age structures in the populations and are expressed as *YPLL* per 100,000 inhabitants in order to account for differences in the population size. The same procedure was followed for the gender and race specific *YPLL*. The reason for not including other races in our analysis apart from black, white and Hispanic is the fact that small population numbers lead to unreliable mortality rates, and therefore unreliable *YPLL* estimates.

In order to analyze which mortalities have led to the formation of the clubs, the following variables, also taken from *CDC's WONDER* database, were used: cancer mortality; infant mortality; cardiovascular mortality; suicide mortality, and the deaths by unintentional injury. These are the mortalities that had the most contribution to *YPLL* in the past years. ICD-9 and ICD-10 codes[4] were used in the extraction of the data for the years before and after (including) 1999, respectively. It should be noted that the comparability ratios between ICD-9 and ICD-10 classification systems for the mortality variables used in our analysis are close to one; this implies that the change in coding practices did not affect the comparability of the data across time and between states.[5] All variables are age-adjusted and in per 100,000 rates for cross-sectional and cross-trend comparison except for infant mortality which cannot be age-adjusted and it was expressed in per 1,000 live births. All calculated *YPLL* and mortality rates are described in Table 2.1.

Additionally, to test whether there is any statistical difference in health spending, data for the mean per capita real public health funding (PHF) and the mean per capita real health care expenditure (HCE) were extracted. For PHF from the Trust for America's Health Rankings for the period 2007-2017 (AHR, 2020), while for HCE from the Centers for Medicare and Medicaid Services for the period 1991-2014 (CMMS, 2018). The Consumer Price Index, used for the conversion to real

[4]International Statistical Classification of Diseases and Related Health Problems *(ICD)* code is a medical classification by the World Health Organization.

[5]For more information regarding comparability ratios, see https://www.cdc.gov/nchs/data/nvsr/nvsr49/nvsr49_02.pdf

terms, was retrieved from the US Bureau of Labor Statistics (BLS, 2020). The selection of time periods was based on data availability. The District of Columbia was not included due to missing data.

Table 2.1: Descriptive statistics for YPLL, cause-specific mortality rates and health spending

Variables	*N*	*Mean*	*S.D.*	*Min.*	*Max.*
$YPLL_{total}$	1989	7533.07	1723.24	4601.86	18 576.75
$YPLL_{male}$	1989	9675.70	2476.45	5599.44	27 292.63
$YPLL_{female}$	1989	5382.40	1098.13	3040.08	11 159.43
$YPLL_{white}$	1989	6977.52	1244.23	2733.88	10 857.78
$YPLL_{black}$	1989	13 318.96	10 714.17	0.00	121 496.10
$YPLL_{Hispanic}$	969	5705.44	4224.34	1736.21	45 036.77
$Cancer_{total}$	1989	197.58	24.92	124.90	295.80
$Cancer_{female}$	1989	163.51	18.17	107.10	235.80
$Cancer_{white}$	1989	194.24	22.15	94.40	246.40
$Cardio_{total}$	1989	270.92	82.50	123.10	487.40
$Cardio_{female}$	1989	214.83	64.38	93.80	391.20
$Cardio_{white}$	1989	266.07	81.62	89.70	486.10
$Injury_{total}$	1989	42.18	11.06	18.10	110.70
$Injury_{female}$	1989	26.19	7.09	10.70	66.70
$Injury_{white}$	1989	41.77	10.82	12.20	101.40
$Infant_{total}$	1989	8.30	2.68	3.10	25.10
$Infant_{female}$	1989	7.37	2.44	1.63	24.30
$Infant_{white}$	1989	7.01	2.02	2.03	18.84
$Suicide_{total}$	1989	13.27	3.79	3.80	29.60
$Suicide_{female}$	1989	5.33	1.85	1.21	14.70
$Suicide_{white}$	1989	14.11	3.74	2.42	30.20
HCE	1200	4260.34	2242.51	137.15	11 050.89
PHF	550	81.83	40.31	26.74	306.12

Notes: *YPLL* = Age-adjusted years of potential life lost per 100,000 inhabitants; *Cancer* = Age-adjusted cancer mortality per 100,000 inhabitants; *Cardio* = Age-adjusted cardiovascular mortality per 100,000 inhabitants; *Injury* = Age-adjusted unintentional injury deaths per 100,000 inhabitants; *Infant* = Infant mortality (age < 1) per 1,000 live births; *Suicide* = Age-adjusted suicide deaths per 100,000 inhabitants. *HCE = Mean per capita real healthcare expenditure in US dollars; PHF = Mean per capita real public health funding in US dollars.*

2.3.2 Methodology

YPLL calculation

Calculation of the *YPLL* was initially conducted for ten-year age groups which were then age-adjusted and aggregated for each state and year. Specifically, due to the structure of the data, from the year 1999 and onwards the age groups were the following: <1 year, 1-4 years, 5-14 years, 15-24 years, 25-34 years, 35-44 years, 45-54 years, 55-64 years and 65-74 years. From 1998 and backwards the groups were the following: <1 year, 1-4 years, 5-9 years, 10-14 years, 15-19 years, 19-24 years, 25-34 years, 35-44 years, 45-54 years, 55-64 years and 65-74 years. Groups from the 1979-1998 series were appropriately merged so that they match the age groups in the 1999-2017 series which were used in our analysis.

The *YPLL* for a specific state i in a denoted time t was calculated using the following equation:

$$YPLL = \sum_{g=1}^{G}(75 - \bar{\omega}_g)\left(\frac{D_{itg}}{P_{itg}}\right)\left(\frac{P_{base_g}}{P_{base}}\right)100,000, \tag{2.1}$$

where g denotes the age group, G is the total number of age groups and $\bar{\omega}$ is the median age of each age group.

The mortality rate, which is the second term in the parenthesis, is calculated by dividing the number of deaths D_{itg} by the total population P_{itg}.[6] This crude mortality rate is then age-adjusted by multiplying with the appropriate weight, the third term in the parenthesis. Different weights were used for each gender and race to account for the gender/race specific age distributions. These weights are obtained by dividing the base population (year 2000) of each age group P_{base_g} by the total population at base year P_{base}. Lastly, the value is multiplied by 100,000 to get a per 100,000 value.

Convergence analysis

The methodology used to examine the convergence process is the one developed by Phillips and Sul (2007) and Phillips and Sul (2009). The superiority of the Phillips and Sul (*PS*) methodology over the classic β and σ-convergence lies in the fact that it uses a time-varying factor model that allows for individual and transitional heterogeneity in order to identify convergence clubs. Additionally, the

[6] Due to data use restriction for deaths less than 10, the mean (5) was used.

test for convergence does not impose any particular assumption concerning trend stationarity or stochastic non-stationarity since it is robust to heterogeneity and to the stationarity properties of the series (Sichera and Pizzuto, 2019).

The panel data of interest are the different *YPLL* which are represented by X_{it} where i is the cross-section dimension (i.e., the 50 U.S. states and the District of Columbia) and t the time parameter. X_{it} consists of two components as presented in Equation (2.2).

$$X_{it} = \delta_{it}\mu_t. \tag{2.2}$$

The μ_t is the common component while δ_{it} is the idiosyncratic component, with δ_{it} measuring the deviation of each state (i) from the common trend μ_t. The idiosyncratic component is described by the following equation:

$$\delta_{it} = \delta_i + \sigma_i \xi_{it} L(t)^{-1} t^{-a}, \tag{2.3}$$

where δ_i is a time-invariant fixed value, σ_i are idiosyncratic scale parameters, ξ_{it} is an independent and identically distributed random variable across i (with zero mean and unit variance) but weakly dependent over t and $L(t)$ is a slow varying function for which $L(t) \to \infty$ when $t \to \infty$.[7]

The aim of the *PS* methodology is to test if all U.S. states converge to a steady-state or multiple ones. The null hypothesis, $\mathcal{H}_0 : \delta_i = \delta$ and $a \geqslant 0$ of convergence of all i versus the alternative, $\mathcal{H}_A : \delta_i \neq \delta$ or $a < 0$ of non-convergence for some i, can be tested through Equation (2.4).

$$\log\left(\frac{H_1}{H_t}\right) - 2\log L(t) = \hat{c} + \hat{b}\log t + \hat{u}_t, t = [rT], [rT]+1, ..., T, \tag{2.4}$$

where $r = 0.3$, $L(t) = \log(t)$, $\hat{b} = 2\hat{a}$ where $\hat{a}$ is the least squares estimate of a under the null hypothesis and u_t are zero mean, weakly dependent errors.[8] For the

[7] For more details, see Phillips and Sul (2007), pp. 1772-1773.

[8] For details, see Phillips and Sul (2007), pp. 1788-1789 and Phillips and Sul (2009), p. 1168.

choice of r, extensive Monte Carlo simulations conducted by PS, show that this value of r (0.3) gives satisfactory results in terms of both the size and the power properties of the test (Phillips and Sul, 2007, pp. 1802-1803). For the choice of $L(t)$, PS recommend $\log(t)$ since it works well in simulations and has good asymptotic power. H_t appears on Equation (2.5) and the relative transition component h_{it} in Equation (2.6).

$$H_t = \frac{1}{N}\sum_{i=1}^{N}(h_{it}-1)^2, \tag{2.5}$$

$$h_{it} = \frac{X_{it}}{N^{-1}\sum_{i=1}^{N} X_{it}} = \frac{\delta_{it}}{N^{-1}\sum_{i=1}^{N} \delta_{it}}. \tag{2.6}$$

The convergence hypothesis is tested by an autocorrelation and heteroskedasticity robust one-sided t-test (Heteroskedasticity and Autocorrelation Consistent (HAC) standard errors are used) and is rejected at the 5% level when $t_{\hat{b}} < -1.65$. In the case of no full sample convergence, Phillips and Sul (2007, pp. 1800-1801) propose a procedure that identifies convergence clubs. This procedure has the following 4 steps:

i First, we sort the last period *YPLL* values of the N states in descending order.

ii Secondly, we form all the possible core clubs of the U.S. states. Starting from the first k highest-ordered states ($2 \leqslant k \leqslant N$), we calculate the convergence t-statistic (t_k) using Equation (2.4). We determine the size of the club k^* by maximizing the t-statistic of the $\log t$ regressions for $t_k > -1.65$.

iii Next, from the remaining states we add one state at a time to the main core clubs and rerun Equation (2.4) including the new state in the convergence club if the respective t-statistic is greater than zero.

iv Finally, we form another club from the states that failed the $\log t$-test in the previous step. Again we run Equation (2.4) for this club. If the convergence criterion is not satisfied, we repeat the previous steps in order to determine if that club can be further divided into convergence clubs. If there are no states left which satisfy the convergence criterion in the second step, these states diverge.

Due to the fact that the use of a conservative sieve criterion about the control value of the t-statistic in step (iii) may lead to an over-estimation in the number of clubs, we perform club merging tests using Equation (2.4) as recommend by Phillips and Sul (2009) and we also calculate the corresponding transition paths.

Finally, to examine the driving factors of the club formations, a t-test is employed to test for the equality of means of the average values of the mortality rates for each club. Club average values were calculated using the entire available time-series for each state of the club.

2.4 Results

In this section, we provide the results of the convergence tests for the gender and race groups as well as for the total population. Moreover, results from the driving factors that have led states to diverge from a single equilibrium when that was the case are also presented. The results for gender and race specific *YPLL* are presented first in order to better understand the dynamics of the total population. The convergence results are presented in Table 2.2. It is important to note here that the higher the number of the club, the lower the $YPLL$ average for each population group.

The results are considerably different between genders, with the male population converging into a single steady-state and the female population forming three clubs which could not be merged further. More specifically, for the female population, Club 1 has the highest average *YPLL* and consists of 10 contingent states that start from West Virginia and go south-southwest without major geographic gaps, with the exception of New Mexico which also shares a small border with Oklahoma. District of Columbia belongs also in that group though not directly contingent. Club 3, the club with the lowest average *YPLL*, is an augmented version of Club 2 of the total population consisting not only of coastal states but also of states located in the middle and north (see Figure 2.1). We can also observe spatial clustering in all three clubs of the female population which implies commonalities among these states in factors that affect premature mortality.

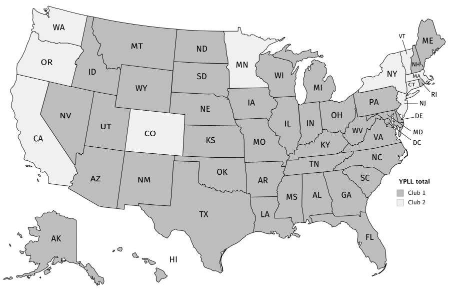
The racial analysis for the black, white and Hispanic populations also show disparities. For the black population all states converge into a single equilibrium as was the case for the male population. The Hispanic population also had all states converging with the exception of Vermont which diverted. The population with the largest variation is definitely the white population. With eleven initial clubs and two divergent states and six clubs plus the group of divergent states after the merging tests, the white population does not seem to follow the full convergence pattern the two other populations had. Washington and West Virginia are the two divergent states while Alabama, Kentucky, and Mississippi form the club with the highest average *YPLL*; and DC and Minnesota the one with the lowest.

The main question, whether the *YPLL* of the total population among the U.S. states converges, is answered negatively. The -3.851 value of the t-statistic leads to rejection the null hypothesis of convergence. Instead, we have the formation of three convergence clubs which remain two after the merge of Clubs 1 and 2. The first club consists of 41 states while the second club of the remaining 10 states.

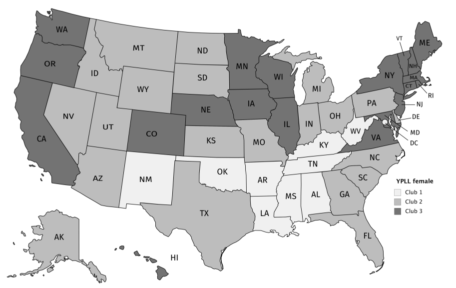
Table 2.2: Club convergence results for total, male, female, white, black and hispanic YPLL

Years of Potential Life Lost (*YPLL*)	Panel A: Phillips and Sul (2007)		Panel B: Phillips and Sul (2009)		
	log t	t-stat	Final club	log t	t-stat
Total					
Full sample	-0.302(0.078)	-3.851**			
Club 1 [AL, AR, DC, KY, LA, MS, NM, OH, OK, TN, WV, ME, MD, MI]	2.257(0.142)	15.839	*Club* 1 [AL, AK, AZ, AR, DE, DC, FL, GA, HI, ID, IL, IN, IA, KS, KY, LA, MS, MO, MT, NE, NV, NH, NM, NC, ND, OH, OK, PA, RI, SC, SD, TN, TX, UT, WV, WI, WY]	0.113(0.084)	1.346
Club 2 [AK, AZ, DE, FL, GA, HI, ID, IL, IN, IA, KS, ME, MD, MI, MO, MT, NE, NV, NH, NC, ND, PA,] RI, SC, SD, TX, UT, VA, WI, WY]	-0.135(0.090)	-1.499	*Club* 2 [CA, CO, CT, MA, MN, NJ, NY, OR, VT, WA]	0.919(0.272)	3.380
Club 3 [CA, CO, CT, MA, MN, NJ, NY, OR, VT, WA]	0.919(0.272)	3.380			
Male					
Full sample	0.043(0.083)	0.514			
Female					
Full sample	-0.954 (0.079)	-12.130**			
Club 1 [AL, AK, DC, KY, LA, MS, NM, OK, TN, WV]	l 1.333(0.255)	5.237	*Club* 1 [AL, AK, DC, KY, LA, MS, NM, OK, TN, WV]	1.333(0.255)	5.237
Club 2 [AK, AZ, DE, FL, GA, ID, IN, KS, MI, MO, MT, NV, NC, ND, OH, PA, SC, SD, TX, UT, WY]	-0.173(0.132)	-1.304	*Club* 2 [AK, AZ, DE, FL, GA, ID, IN, KS, MI, MO, MT, NV, NC, ND, OH, PA, SC, SD, TX, UT, WY]	-0.173(0.132)	-1.304
Club 3 [CA, CO,CT, HI, IL, IA, ME, MD, MA, MN, NE, NH, NJ, NY, OR, RI, VT, VA, WA, WI]	-0.172(0.109)	-1.570	*Club* 3 [CA, CO,CT, HI, IL, IA, ME, MD, MA, MN, NE, NH, NJ, NY, OR, RI, VT, VA, WA, WI]	-0.172(0.109)	1.570
White			*Club*1 [AL, KY, MS]	0.836(0.975)	0.857
Full sample	-1.978(0.085)	-23.361**	*Club* 2 [AR, OK, TN]	l 0.066(0.816)	0.081
Club 1 [AL, KY, MS]	0.836(0.975)	0.857	*Club* 3 [IN, LA, MO, NV, NM, OH, PA, SC]	-0.370(0.265)	-1.394
Club 2 [AR, OK, TN]	0.066(0.816)	0.081	*Club* 4 [AZ, DE, FL, GA, HI, ID, KS, ME, MI, MT, NH, NC, ND, TX, UT, WY]	-0.207(0.286)	-0.723
Club 3 [IN, LA, NV, NM, OH, SC]	0.080(0.362)	0.220	*Club* 5 [AK,CA, CO, CT, IL, IA, MD, MA, NE, NJ, NY, OR, RI, SD, VT]	-0.139(0.329)	-0.424
Club 4 [MO, PA]	-0.311(0.894)	-0.348	*Club* 6 [DC, MN]	-1.035(3.291)	-0.315
Club 5 [AZ, DE, FL, GA, HI, ID, KS, ME, MI, MT, NH, NC, ND, TX, UT, WY]	-0.207(0.286)	-0.723	*Group* 7 (*not convergent group*) [WA, WV]	-2.671 (-)	-20.111**
Club 6 [CA, IL, IA, MD, OR, RI]	0.239(0.588)	0.407			
Club 7 [AK, VT]	-0.440(1.619)	-0.272			
Club 8 [MA, NY, VA]	0.755(0.859)	0.878			
Club 9 [DC, NE, SD, WI]	0.484(0.444)	1.092			
Club 10 [CT, NJ]	1.522(0.642)	2.369			
Club 11 [DC, MN]	-1.035(3.291)	-0.315			
Group 12 (*not convergent group*) [WA, WV]	-2.671 (-)	-20.111**			
Black					
Full sample	2.094(0.137)	15.325			
Hispanic					
Full sample	-0.483(0.133)	-3.631**			
Club 1 [AK, AZ, CO, HI, IN, KS, LA, ME, MA, MI, MT, NH, NM, ND, OH, OK, PA, SD, TX, UT, WA, WV, WI, WY]	0.224(0.170)	1.314	*Club* 1 [all states apart from VT]	-0.145(0.137)	-1.054
Club 2 [AL, AR, CA, CT, DE, DC, FL, GA, ID, IL, IA, KY, MD, MN, MS, MO, NE, NV, NJ, NY, NC, OR, RI, SC, TN, VA]	0.691(0.263)	2.628	*Group* 2 (*not convergent group*) [VT]	-	-
Group 3 (*not convergent group*) [VT]	-	-			

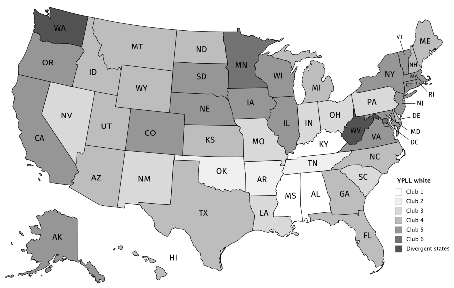
Notes: Standard errors are in parentheses. The abbreviations in square brackets denote the states included in each club (see Appendix A). The term log t denotes the convergence coefficient, while t-stat is the convergence test statistic. The latter is distributed as a simple one-sided t-test with a critical value of -1.65. ** denotes rejection of the null hypothesis (convergence) at 5% level of statistical significance. The results were estimated using the Stata codes of Du (2017). The higher the number of the club, the lower the *YPLL* average for each population group.



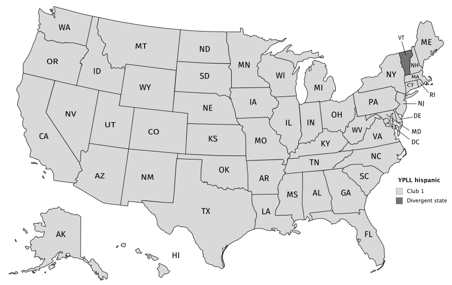
(a) Clubs for total population



(b) Clubs for female population



(c) Clubs for white population



(d) Clubs for Hispanic population

Figure 2.1: Estimated convergent clubs

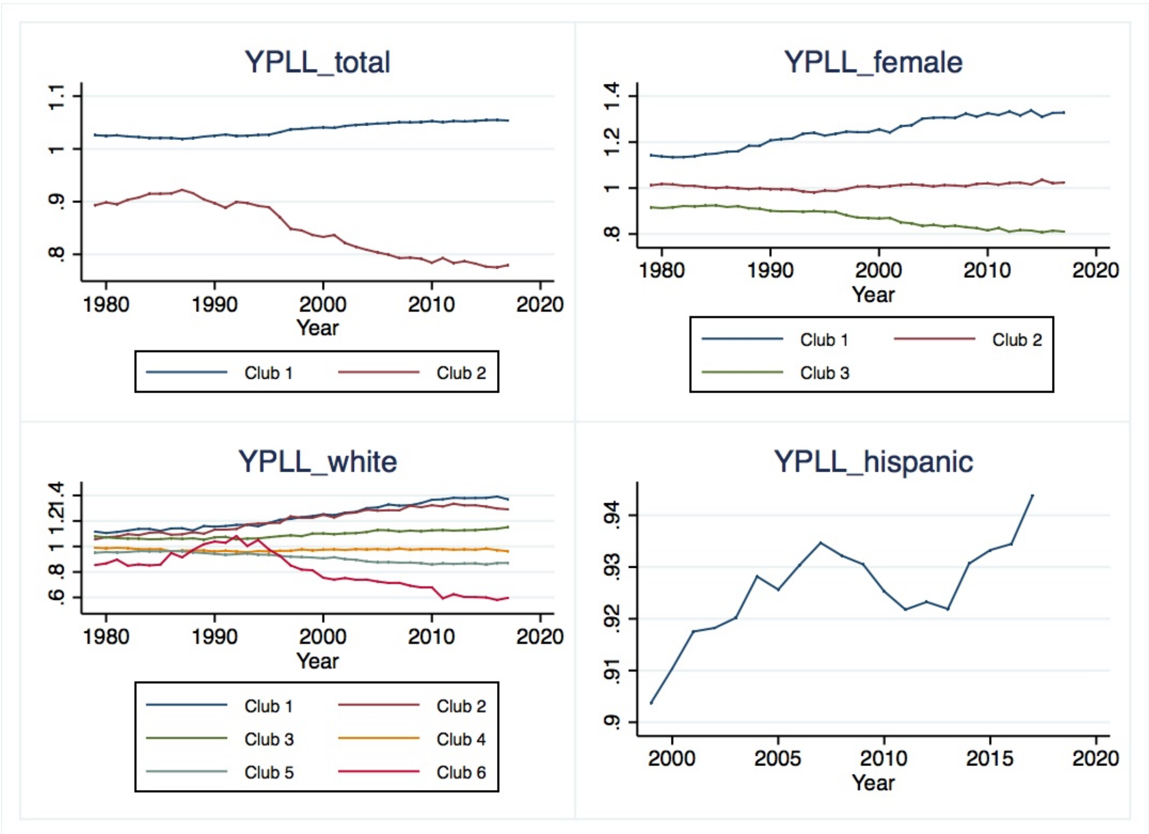


Figure 2.2: Transition paths for divergent clubs

The transition paths illustrated in Figure 2.2 are in line with the convergence test results. The total population exhibits a small tendency to converge in the first years but the transition curves of the two clubs begin to widen from the late 80s until our latest available year (2017) as the components of the total population begin to diverge. Indeed, for the female population, Club 1 never seems to converge while Clubs 2 and 3 exhibit a tendency to converge until the mid 90s when they begin to diverge. For the white population, there is no coherent pattern until the mid-90s when clubs begin to diverge. It appears that Clubs 1 to 3 will converge in the future and the same can be told for Clubs 4 and 5 but not for Club 6.

The differences in the means of the variables we chose as the main components of premature mortality that drive the *YPLL* show that for the female population, deaths by unintentional injury and cardiovascular mortality are the main separating factors between the two Clubs with lower average *YPLL* from the one with the highest. Moreover, Club 2 has significantly less cancer mortality rates while Club 3 has significantly less infant mortality rates compared to Club 1. For the white population, the two clubs with the highest average *YPLL* (Clubs 1 and 2) have no significant difference in all the reported driving factors. For the rest of the clubs, infant and cardiovascular mortality rates were different in Clubs 3 to 6, deaths by unintentional injury in Clubs 4 to 6, suicide rates in Clubs 5 and 6 and cancer mortality only in Club 6. The total population shows differences in means in infant mortality and deaths by unintentional injury but there is no significant difference in cardiovascular mortality as opposed to the female and white population.

Lastly, for the total *YPLL*, Club 2 had higher PHF per capita (\$87.15) compared to Club 1 (\$80.50) but there was no statistical difference at the 5% level (p-value=0.619 and p-value=0.556 when allowing for homogeneous and heterogeneous covariance matrices across groups, respectively). On the other hand, the mean per capita real HCE is found to be marginally statistically different between the two clubs at the 5% level under homogeneous (p-value=0.045) but not heterogeneous (p-value=0.118) covariance matrices across groups. The mean HCE for Club 1 was

\$4186.14 and for Club 2=\$4557.14.

Table 2.3: Mortality rates driving the *YPLL* divergence

YPLL	Club	*Unintetional injury mortality*	*Cancer mortality*	*Infant mortality*	*Cardiovascular mortality*	*Suicide mortality*
Total	1	**43.946**	198.839	**8.631**	275.332	13.666
	2	**34.937**	192.407	**6.955**	252.841	11.640
Female	1	31.310	169.539	8.882	245.900	5.301
	2	**27.831**	**160.324**	7.454	**210.808**	5.866
	3	**21.899**	163.839	**6.529**	**203.509**	4.784
White	1	54.991	207.731	7.906	314.304	15.112
	2	50.887	203.672	7.892	301.365	15.687
	3	45.493	200.393	**7.293**	**283.795**	15.792
	4	**42.096**	187.630	**6.937**	**252.952**	15.299
	5	**36.617**	194.374	**6.603**	**259.601**	**12.232**
	6	**29.758**	**177.183**	**6.801**	**214.503**	**9.514**

Notes: The numbers in the above Table are the average values of the corresponding risk factor for each club. The bold values for the *Female* and *White* clubs indicate that the club average for the respective risk factor is not included in the 95% confidence interval of the club with the highest *YPLL*. The bold values for the *Total* clubs indicate that the null hypothesis of the equality of means is rejected at a 0.05 significance level. The test for the equality of means is conducted allowing for both homogeneous and heterogeneous covariance matrices across groups. The test produces similar results under both options.

2.5 Discussion

The convergence analysis showed that the *YPLL* of males and blacks converge for all states. The same applies for the hispanic population with the exception of Vermont.[9] On the other hand, for females and whites inequalities appear from the formation of 3 and 6 groups of states by the clustering algorithm, repsectively. For the entire population, *YPLL* follow two paths. The main mortalities that drove the formation of clubs were infant, and unintentional injury mortality for the total *YPLL*, while for females, differences in cancer and cardiovascular mortality were also significant. Suicides appear to drive *YPLL* differences only in white population. Per capital public health funding and healthcare expenditure for the total population

[9] The Hispanic population of Vermont is a very small fraction of a very small population in general and given the reduced period of the available data, the question whether the premature mortality of the Hispanics of Vermont is actually diverging should be further studied.

were higher for the club with the less *YPLL* though not statistically significant at the 5% level.

While there are several studies that have associated black population with increased risk of premature mortality (Cooper et al., 2016; Cullen et al., 2012; Krieger et al., 2014; Mansfield et al., 1999), this research shows that this risk is or becoming similar at least at the state level. As Naghshpour and Sameem (2019) had previously found convergence for black mortality rates, their premature mortality appears also to converge. The improvements in chronic disease outcomes such as CVD for black (and also Hispanics) are probably the reason behind the full sample convergence and the reduction of the health 'gap' with whites (Chen et al., 2019; Ma et al., 2022). On the other hand, risk factors such as inequality and segregation may be in place regardless of the state of residence of black populations (Cooper et al., 2016; Krieger et al., 2014), and thus disparities between racial groups persist.

Despite the fact that the health of the white population has reached a plateau and further improvements are not easily achievable (Chen et al., 2019), the 6 resulting clubs signify large geographic variation and disparities between states. With more *YPLL* lost in Southern states and less in Northeast, Midwest, and West coast states, the results are in accordance with previous research on the subject (Dani et al., 2022; Mansfield et al., 1999). Once again, income inequality is higher in states with high premature mortality and that affects also the white population. It remains to be seen if predictions for injury and suicide death increase will affect white populations disproportionately.

The full sample convergence of the male population is an unexpected result. For women, *YPLL* follows the same geographical patterns as for the white population, albeit with half the numbers of divergent clubs. These disparities in female premature mortality are perhaps a result of an interaction between income inequality and the presence of more female-headed households in these states, as literature suggests (Mansfield et al., 1999). The combination of low income and the absence of a husband potentially affects the health of women negatively resulting in increase premature deaths.

As a result of the previous disaggregated populations, premature mortality for the total population converges for most of the states while there are 10 states, 8 of which are in the West and Northeast coast, that converge to an equilibrium with less *YPLL*, meaning less premature mortality. It is no surprise that the two geographical exceptions, Colorado and Minnesota, belong to the 'healthier' club despite being central and north in the map as shown in Figure 2.1a since their per capita GDP is above average. It is evident from the convergence analysis of the gender and race groups that the two convergent clubs in the total population are mainly a consequence of diverging patterns in the female and white population. In general, both coasts as well as middle and northern states appear to have less premature deaths as illustrated in Figure 2.1a.

Infant mortality plays a key role in premature mortality not only due to the maximum years added to *YPLL* but also due to the fact that medicine has come a long way in the prevention of most these deaths. The differences between clubs were approximately 1-2 deaths per 1,000 live births. In less aggregated entities the difference is expected to be larger. This disparity implies that the gap in infant deaths is possibly created by non-medical preventable deaths. Deaths by unintentional fatal injuries, given their non-medical nature, contribute severely to the *YPLL* since they include a high number death causes but the vast differences in club means (10-25 per 100,000) imply that states can actually intervene and influence this rates with the right policies. The small differences in suicide rates between clubs suggest that in order to reduce these rates a more radical approach should be taken and state policies might not be sufficient. Instead, a government initiative might be more appropriate to tackle effectively these issues. Intervention becomes more urgent for the last two mortalities since studies show that these death rate are expected to rise in the future (Shiels et al., 2017).

Cancer and CVD mortality are a major public health issue in developed countries. Although, rates and disparities are reducing the results show still large differences between clubs, especially for CVD mortality. The club means of the cardiovascular mortality rates in whites have differences up to a third, which can be attributed to lifestyle factors such as nutrition and exercise, as well as lack of access to quality health care or insurance due to income restrictions.

Differences in HCE and PHF bordered on statistical significance. While higher spending in healthcare, in either per capita healthcare expenditure or just public health funding, is related to lower *YPLL*, the state's differences in $YPLL$ are probably driven mostly by individuals' behavioral aspects and socioeconomic inequality. Mansfield et al. (1999) earlier work on *YPLL* also suggests that availability of medical care is not that important of a factor. This in turn highlights once more the importance of allocating public funds on preventive policies that modify behavior and health consciousness as well as welfare and redistribution programs in order to blunt inequality.

This research is limited in the following ways. First, the analysis in performed on an ecological level using aggregate data at the state level. Secondly, not all races were examined due to $CDC's$ data censorship policy which made *YPLL* calculation unreliable. This does not mean that these minority populations are not important and should not be studied. The mortalities examined are also a subspace of the total mortality which I found to have the most contribution to premature mortality. Finally, the expenditure data (PHF and HCE) are mere proxies for healthcare access, quality, and utilization.

2.6 Conclusions

The aim of this chapter was to examine the convergence patterns of premature mortality across the U.S. states and identify the mortality components behind them. By applying the Phillips and Sul (2007) and Phillips and Sul (2009) methodology, the conclusion came that premature mortality for males and blacks converged to unity while for the total, female and white population, convergence clubs were identified. The cross-sectional differences, driven mainly by unintentional injury, cardiovascular, and infant deaths as well as the unclear association between health spending and premature mortality, lead to the conclusion that prevention and equity are a key factors in narrowing the health gap between states. Public health policy makers should seriously address these issues since the role of prevention is unique to them. Interventions that aim at the socioeconomic level will probably have the most effect in *YPLL* but political will and public health are not always in accord. Populations with absolute convergence can be approached with a single strategy, while for converging clubs the proper policy can improve premature mortality for each one.

This chapter fills an existing gap in the literature since it studies the convergence of a health indicator in the U.S. states for a period of 39 years on multiple populations. Future research should focus on examining the effects of climate change on premature mortality and to try disentangle the effect of the underlying mechanisms that affect very much the same population that already is at higher risk of a premature death.

Chapter 3

The association between the COVID-19 pandemic US state government responses and the equity-efficiency relation

3.1 Introduction

Equity and efficiency are thought be two opposite forces in the mainstream economic thought. Whether this belief has political origins, or it is driven by the inability to justify the continuous rise in inequality in capitalist societies, matters not. What matters is more fundamental questions regarding policies and how can these prioritize the society and environment rather than the rat race of unconditional growth, which has become an end in itself in the minds of modern day economists.

The equity-efficiency trade-off was originally a theoretical construct in microeconomics, where, supposedly, an economy had to sacrifice social equality in order to achieve higher market efficiency. As described by Okun in the seventies, the transfer of wealth from one group to another resembles a leaky bucket, where efficiency 'leaks' because of the efficiency costs of taxation and transfers, as well as some often minuscule administrative or transaction costs. The main questions that arise therefore are: How much the bucket leaks and how much people (or elected politicians) valuate equality in a society?

Kuznets (1955) first described several problems that arise when studying this subject. For example, because the subject is rather political, ideologies often dominate when redistributive policies are discussed or proposed. Additionally, the complexity of the matter and the ambiguity of the semantics and definitions of the involved terms (i.e., inequality, efficiency) render the equity-efficiency research open to many—sometimes contradictory—interpretations. In order to investigate this relation, one has to clearly define what is the equity of interest and how it will be measured. The same applies to efficiency, although the options are more limited. Clearly, there are several approaches in the literature. More specifically, wealth, income and wage inequality are the most prevalent choices for assessing equity, while economic efficiency is usually measured as GDP growth or GDP per capita. Of course, the trade-off can also be examined outside of its traditional economic context; see Reidpath et al. (2012) for an example in healthcare.

This chapter aims to examine the associations between the State's government pandemic responses and the efficiency-equity trade-off in the US states during the COVID-19 pandemic. Additionally, the association between the State's pandemic responses and regional wage equality is also examined. To do so, I employ a 2020 cross-section analysis of US state level data, and use various econometric techniques in an attempt to capture the effect of this exogenous shock (i.e., the Sars-CoV-2 pandemic and its subsequent responses) on the efficiency and equity relation, at the subnational level. To the best of my knowledge, the effect of the government pandemic responses on the equity-efficiency relation and wage inequality have hardly been studied in the literature, due to the unavailability of recent inequality data. Since we know that efficiency and equity cannot remain unaffected by the corona situation, the findings or this research create new socioeconomic and equity knowledge, with important policy implications.

The remaining part of the chapter has the following structure: The next section entails a literature review on the equity-efficiency research. Section 3.3 provides the theoretical framework that the research idea is based upon. Section 3.4 describes the data and methods used in the analyses and Section 3.5 presents the empirical results. Finally, Section 3.6 discusses them and in Section 3.7, the final conclusions are drawn.

3.2 Literature review

Although a microeconomic concept, empirical testing has utilized predominately macro-data due to measurement difficulties on the individual level.[1] The use of macro-data implies some degree of aggregation and a geographic level of analysis. In cross-country comparisons, early research by Persson and Tabellini (1992) and Alesina and Rodrik (1994) showed that wealth inequality has a negative effect on economic growth, with Alesina and Rodrik (1994) emphasizing the political aspect of this relation (i.e., it holds true for democracies). A few years later, Li and Zou (1998) and Forbes (2000) using panel data from heterogeneous countries presented evidence of the opposite, that is, inequality is good for growth. Next up, Barro (2000) divided his sample countries into rich and poor, and found that the trade-off was present for the rich countries, but not for the poor ones. Banerjee and Duflo (2003) argue subsequently that an inverted U-shaped curve—as proposed by Kuznets (1955)—is the main driver behind the contradictory results in the literature and that changes in equity tend to affect negatively economic growth in the subsequent period, *irrespective* of their direction.

The measurement error found when measuring inequality, especially in developing countries, led Knowles (2005) to use expenditure data to enhance the comparability. His study of developing countries found again that for this group, there is a positive correlation between equity and growth. In more recent years, Fawaz et al. (2014), considering potential endogeneity issues, employed a generalized method of moments (GMM) estimator to find different relationships between inequality and growth for high- and low-income countries, with results similar to Barro (2000). Finally, a recent study by Andersen and Maibom (2020) used stochastic frontier

[1] For a micro-data approach see Browning and Johnson (1984).

analysis on the basis of the fact that not all countries start at the same point. This novel approach appeared to indicate the existence of a trade-off.

Within-country analyses were not in favour in the past but are beginning to increase in popularity in recent years. Using panel data from US states, Frank (2009) points towards a negative relationship between equity and growth in the long term due to wealth concentration among the richest 10%. Alexiadis and Eleftheriou (2011), on the other hand, using time-series analysis of US data, found that efficiency and equity can co-exist and that the relation is also affected by policies of the governing party. Due to the existence of geographical interdependencies, spatial econometric approaches were also employed to analyze the relation. Ezcurra (2007) examined the relation between inequality (measured by income dispersion) and regional growth to find a negative association. There are also studies that explore the effect of endogenous factors to assess the relation. For example, Kim (2016) finds that financial accessibility has the potential to change the negative relationship between inequality and growth into a positive one, especially for fragile countries.

The conclusion from the empirics discussed above is that the lack of consensus on the relation between equity and efficiency is caused several factors including: different estimation techniques, structural assumptions of the authors (e.g., linearity), measurement errors of the variables in question, as well as unobservable confounding factors that make a statistical identification all the more difficult (Banerjee and Duflo, 2003). In addition, empirical evidence seems to be sensitive to the time horizon (i.e., short vs. long-term effect) and the starting point, that is, the development phase in the case of cross-country comparisons (Neves and Silva, 2014). It is important to note that both De Dominicis et al. (2008) and Neves and Silva (2014)—who reviewed intensively the empirical literature—accord with the idea that it is better to study one country at subnational level (i.e., regionally) than more heterogeneous national units. The reasoning behind this claim is that studying regional units can limit unobservable factors and measurement errors in inequality, that might affect the relation. Hence, more transferable conclusions for policy can be drawn. Accordingly, this study focuses on regional inequalities within a given country, viz. the USA.

3.3 Theoretical framework

Recent COVID-19 government interventions may have had an influence on the equity-efficiency dilemma at regional scale. This did not held true just for the sparse available public health resources, but for the operation of the economy—or at least parts of it. Previous research has shown that pandemics had a negative effect on equity, affecting mainly socioeconomically vulnerable individuals via financial and epidemiological mechanisms (Furceri et al., 2022). Therefore, it is safe to assume that given the magnitude and universality of the COVID-19 pandemic, both equity and efficiency suffered.

What previous pandemics were lacking (at least at this magnitude) were government responses. These responses which aimed to mitigate the spread of the virus, as well as to provide some economic support, may were more detrimental to the efficiency and equity. Mandates on teleworking and the operation of essential goods stores might have led to increases in income/wage inequality, as a result of those who were unable to work from home (Christopoulos et al., 2022). Health-wise, individuals employed as essential workers had increased exposure to the virus. Furceri et al. (2021) argue that government economic interventions helped mitigated the rise in inequality in previous pandemics, therefore the economic support given in this pandemic might had the same effect.

The outbreak of COVID-19 has affected US states in different ways since the dispersion of the corona virus has been geographically uneven. The government responses to COVID-19 have also shown remarkable differences at state level as well (Hallas et al., 2021). It not clear though if the State's government responses to the pandemic altered the relation between equity and efficiency, and is therefore pertinent to examine the equity-efficiency dilemma in corona times.

3.4 Data

This section entails information about the variables used to measure equity and efficiency as well as the COVID-19 pandemic government responses for 49 US states and the District of Columbia (DC). Due to the spatial analysis and following the no 'island' rule, Alaska and Hawaii were excluded from the sample. The descriptive statistics of all variables included in the analysis are presented in Table 3.1.

Table 3.1: Descriptive statistics

Variables	*Obs.*	*Mean*	*S.D.*	*Min.*	*Max.*
Gini	49	.497	.020	.457	.543
GDPpc	49	54619.29	19836.08	33518.95	173027
GRI	49	41.44	7.42	22.67	60.21
SI	49	42.04	8.26	18.38	60.82
CHI	49	42.44	6.92	22.94	57.48
ESI	49	34.46	16.77	3.01	79.37
Deaths	49	1079.14	440.76	232.38	2143.84

Notes: *Gini*=Gini index of wage inequality; ; *GDPpc*= 2020 real GDP per capita, *GRI*=Mean government response index for 2020; *SI*=Mean stringency index for 2020; *CHI*=Mean containment and health index for 2020, *ESI*=mean economic support index for 2020, *Deaths*= 2020 COVID-19 deaths per million inhabitants.

3.4.1 Data for equity and efficiency

In general, inequality is not easy to measure. More so in times of crisis, like the COVID-19 pandemic, as this could lead to additional measurement errors (Banerjee and Duflo, 2003). The wage inequality data, namely the Gini index [2] for the year 2020 were retrieved from Gambau Suelves et al. (2021). This variable was estimated by Gambau Suelves et al. (2021) with the use of the Lockdown Working Ability index (Dingel and Neiman, 2020) so as to incorporate changes in the distribution of wages, reflecting in a way the essentiality of occupations during the COVID-19 pandemic. The focus is only on wage and not income inequality data, since these

[2] The values of the Gini index range from 0 to 1, where 0 indicates that the income distribution is equal (everyone earns the same income) and 1 denotes perfect inequality (one person/household earns all the income).

were the only available and reliable data at that moment. The Gini index of wage inequality will be the variable used as the regressand.

Economic efficiency is measured by the real 2020 GDP per capita (in chained 2012 dollars), extracted from the Bureau of Economic Analysis (BEA, 2020). Since this is a cross-sectional study and growth rates are expected to be negative in all US states, real GDP was preferred. The natural logarithm of real per capita GDP was used in the statistical analysis for normality reasons.

3.4.2 COVID-19 government response data

For the measurement of COVID-19 government responses, I use the overall Government Response Index (GRI) and its components: the Stringency Index (SI), the Containment and Health Index (CHI), and the Economic Support Index (ESI). All indices were retrieved from Oxford COVID-19 Government Response Tracker (Hale et al., 2020). The indices at the State level for US do not include Federal policies that apply to the country as a whole. However, these indices may reflect a mixture of Federal government and State government responses since Federal government gave recommendations which may have been followed.

The SI measures mainly the severity of closure policies, designed to restrict human mobility and social behavior. CHI additionally measures the degree of testing policies, contact tracing, and investments in healthcare (including vaccines). On the other hand ESI was designed to measure income support and debt relief policies for households. Finally, GRI contains an aggregate measure of government responses computed from the above components. For more details on government response indices, see Hale et al. (2020). These variables range from 0 to 100 according to the severity of the government intervention. To calculate an annual variable, each of these indices was averaged for its 2020 values for each US state. The calculated variables were dichotomized based on the sample mean for use in the empirical analysis. Specifically, for each index, I created a dummy variable taking the value of one if the corresponding averaged index is above the sample mean, and zero otherwise.

3.4.3 Instrumental variables

Several candidate instrumental variables were used to address potential endogeneity issues. The corresponding data were retrieved from the following sources: Labor union membership from Hirsch and Macpherson (2003); Human capital and Environmental organizations from the US Census Bureau;[3] and Adolescent fertility from America's Health Rankings analysis of CDC WONDER.[4]

3.5 Methodology

This subsection entails details on the econometric methods used for the analysis of the data as well as various statistical tests.

3.5.1 Endogeneity and candidate instruments

Since GDP might be an endogenous regressor, parameter estimates might be biased and inconsistent. In order to examine whether endogeneity—that arises from bidirectional causality between GDP and the Gini index or omitted variables—is present, a series of tests based on the results of two-stage least squares regressions were performed, based on IV (instrumental variable) analysis. Finding valid and strong instruments for this relationship borders on impossibility but the best efforts were made. It worth noting the most of the literature ignores this potential problem.

Four instruments were required for the (over)identification of the model (presented later in this subsection). The four candidate instruments used were: human capital, measured as % of individuals over the age of 25 with bachelors' degree or higher; unionization as % of employed workers who are members of a labor union;

[3]Available from: https://data.census.gov/cedsci/

[4]Available from: https://www.americashealthrankings.org/explore/annual/measure/TeenBirth_MCH.

environmental organizations per million inhabitants; and adolescent fertility, measured as births per 1,000 female aged 15-19.

For the instruments to carry the 3 properties required for the identification of the model[5] a theoretical approach is often required. As education is a proxy for human capital—whose positive correlation between human capital and growth is well documented in the literature (Barro, 2001)—but correlation with wage inequality at the state level is also probable—but not as clear as the correlation at the individual level—the validity of this instrument remains theoretically unclear. Labor unionization has also been associated with economic growth (Hirsch, 1997; Kim, 2005), but a negative association with wage inequality is very probable since one of the main purposes of labor unions is to tackle wage inequity.

On the bright side, the number of environmental organizations which serves as a proxy for environmental protection performs better as an instrument (at least theoretically). According to Inglehart (1995), wealthier individuals who have satisfied their basic needs are more likely to develop ecological consciousness, therefore an association with per capita GDP exists. The link between ecological valuation and income has also been proven empirically by Gelissen (2007). It is theoretically unclear whether wage inequality could be associated with the number of environmental organizations. Furthermore, adolescent fertility is likely also to be negatively correlated with per capita GDP (Santelli et al., 2017), and not correlated with wage inequality at the state level. In sum, the four candidate instruments used are relevant but their validity remains dubious at best. The magnitude of the correlation with GDP also suggests that we are dealing with weak instruments.

Aside from the literature suggesting an association between the aforementioned candidate instruments and GDP, the validity of statistical tests for instrument validity is very limited. In the case of overidentified models one can perform a test of overindentifying restrictions, although whether or not the overidentifying restrictions are valid gives little information on whether the instruments are correlated with

[5]See Cameron and Trivedi (2005, page 100) for details.

the errors of the model, and on whether parameters of interest can be successfully identified, regardless of the sample size (Parente and Santos Silva, 2012). Therefore, there is no clear answer as to whether a candidate instrument is exogenous.

Nevertheless, overidentifying assumptions for each specification (one for every government response index) was tested with the Hansen J test (Hansen, 1982), under the null hypothesis that , with the corresponding p-values ranging from 0.0813 to 0.6492. The endogeneity tests were performed using the C statistic (difference-in-Sargan) test under the null hypothesis that GDP is exogenous (see Baum et al., 2003) with the corresponding p-values ranging from 0.1584 to 0.6348. For all the aforementioned reasons IV methods were not considered further for the final analysis of the data.

3.5.2 Cross-sectional dependence

Despite the potentially limited regional variation at the state level, inequality and growth tend to exhibit spatial clustering. In such cases, failure to account for the spatial dependence will produce biased estimates (Elhorst, 2014). Examples of spatial heterogeneity in inequality can be found in Hortas-Rico and Rios (2019) for Spain, and for growth, in López-Bazo et al. (2004) for European regions. To test for cross-sectional dependence in the sample states I conduct the Moran's I test (where the null hypothesis is the absence of spatial dependence) for the Gini index and the natural logarithm of GDP per capita. The results of the test are a p-value equal to 0.011 and 0.041, respectively. These verify the existence of spatial dependence in the above two variables. A depiction and description of this dependence is illustrated in Section 3.6.1.

3.5.3 Model specification

Four models, one for each government response indicator (i.e., GRI, SI, CHI, ESI), were used in the analysis. To test for a potential additive effect modification of the government responses on the equity-efficiency relation an interaction term between the dummy variable of the response index and the natural logarithm of real per capita GDP we included in our model. Moreover, to test the non-linearity hypothesis, a quadric term of the natural logarithm of real per capita GDP was also included. Due to limited observations no other control variables were used.

In order to select the appropriate specification, the residuals of the simple Ordinary Least Squares (OLS) model were tested for spatial autocorrelation. The results indicated the existence of spatial autocorrelation (p-value = 0.0311) and hence the use of a spatial model may be justified. The traditional way of choosing between spatial specifications are data-driven model comparison techniques. Like all data driven approaches, they ignore any theoretical knowledge and rely on strong assumption about the data generating process.[6] While the aforementioned approach pointed into a Spatial Durbin Model (SDM), the use of spatial lags for the interaction and quadratic term does not have any theoretical reasoning and leads to overparameterization which, given the tiny sample, would create more severe problems than that of spatial autocorrelation.

Instead, and in order to control for the spatial dependence of the Gini index, a spatial lag was added to the models resulting in the classic Spatial Autoregressive Model (SAR). Additionally, another set of models that contain a spatial lag for the per capita GDP as well, were also used. The contiguity matrix $\mathbf{W}$ used was a binary row standardized matrix where the elements w_{ij} are

$$w_{ij} = \begin{cases} w_{ij} = 0, \text{if } i = j \\ w_{ij} = 1, \text{if } i \text{ and } j \text{ are contiguous} \\ w_{ij} = 0, \text{if } i \text{ and } j \text{ are not contiguous} \end{cases}$$

where i and j refer to US states.

[6]For a discussion on the limitations of these approaches on the spatial context see Gibbons and Overman (2012).

The two following spatial autoregressive models were estimated:

$$\begin{aligned} Gini_i &= \beta_0 + \beta_1 ln(GDP_i) + \beta_2[ln(GDP_i)]^2 + \beta_3 index_i + \beta_4[ln(GDP_i) \times index_i] \\ &\quad + \lambda \sum_{j=1}^{N} \tilde{w_{ij}} Gini_j + u_i \qquad (3.1) \end{aligned}$$

$$\begin{aligned} Gini_i &= \beta_0 + \beta_1 ln(GDP_i) + \beta_2[ln(GDP_i)]^2 + \beta_3 index_i + \beta_4[ln(GDP_i) \times index_i] \\ &\quad + \lambda \sum_{j=1}^{N} \tilde{w_{ij}} Gini_j + \gamma \sum_{j=1}^{N} \tilde{w_{ij}} ln(GDP_j) + u_i \qquad (3.2) \end{aligned}$$

where i refers to a given US state, $Gini$ denotes the Gini index, GDP is the real per capita GDP, $index$ is a dummy variable taking the value of 1 if the corresponding government response index (i.e., SI, ESI, CHI, GRI) of US state i is above the sample mean and 0 otherwise, u_i is the standard error term, and $\tilde{w_{ij}}$ is the ijth element of the previously described row standardized contiguity matrix $\mathbf{W}$ with $\tilde{w_{ij}} = \frac{w_{ij}}{\sum_j w_{ij}}$ and $\sum_j w_{ij} = 1$.

Maximum Likelihood Estimator (MLE) was used to estimate the above two specifications. According to Anselin et al. (2001), if the spatially lagged dependent variable is the only endogenous variable, then MLE is considered to be the proper estimation method. Lastly, heteroskedasticity robust standard errors were used throughout the analysis.

3.6 Results

This section presents the empirical results starting from a description of the spatial patters and continuing with the models estimates.

3.6.1 Spatial patterns

This subsection provides a visual representation of the spatial patterns in wage inequality and GDP per capita, as well as of government responses (GRI) and COVID-

19 deaths with the use of choropleth maps.[7] Variables were divided into quartiles.

Starting with the Gini index of wage inequality the clustering of higher wage inequality is more evident on the East, South, and New Jersey-New York region; while for lower inequality on the Midwest. These patterns appears on Figure 3.1. On the next map, Figure 3.2, appears the spatial clustering of per capita GDP.

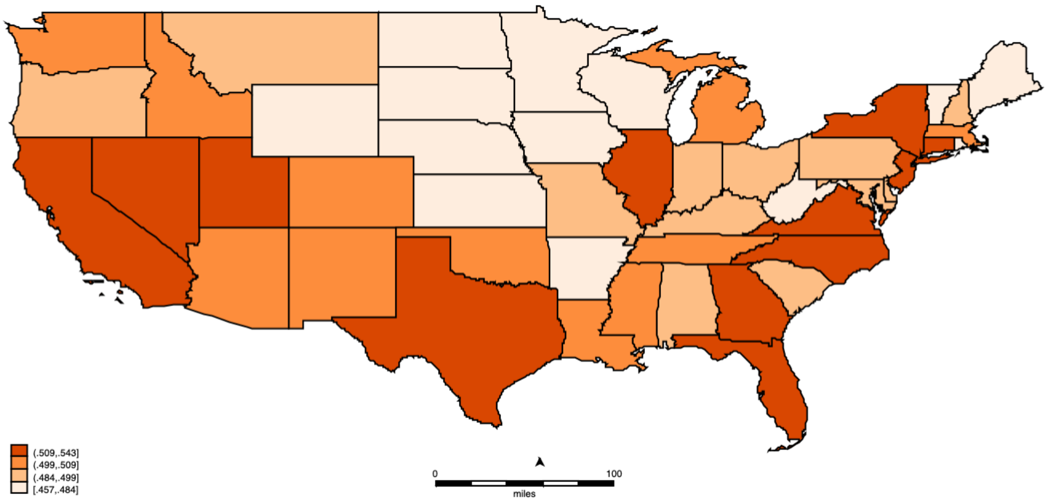


Figure 3.1: Gini index of wage inequality choropleth map

High levels of per capita GDP can be seen on the West, Midwest, and Northeast; while a lower GDP per capita levels appear Southeast. On Figure 3.3 we observe

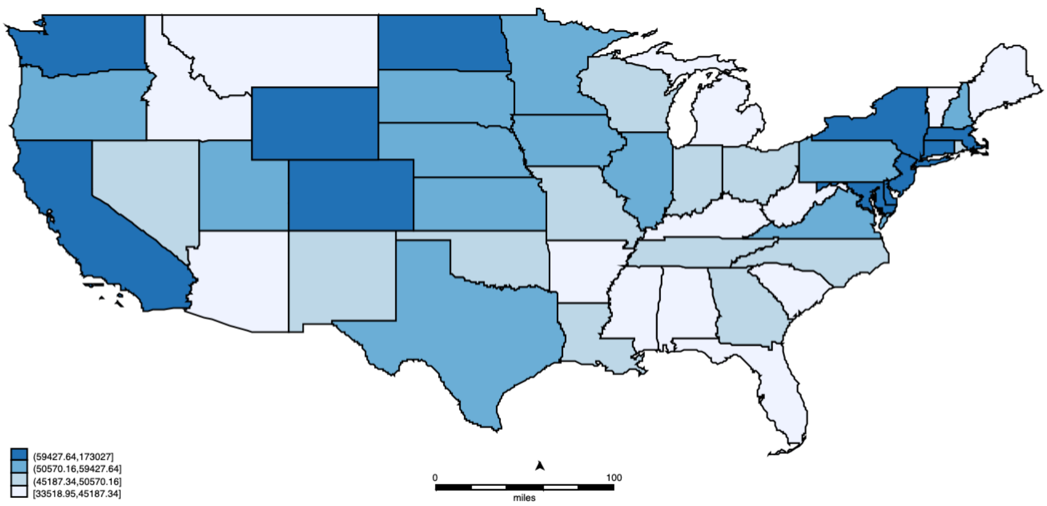


Figure 3.2: Per capita GDP choropleth map

more intense government responses Northeast and less intense in the Midwest and Southern states. On the other hand, according to Figure 3.4, COVID-19 deaths

[7] The choropleth maps were created used the -spmat- command (Pisati, 2018).

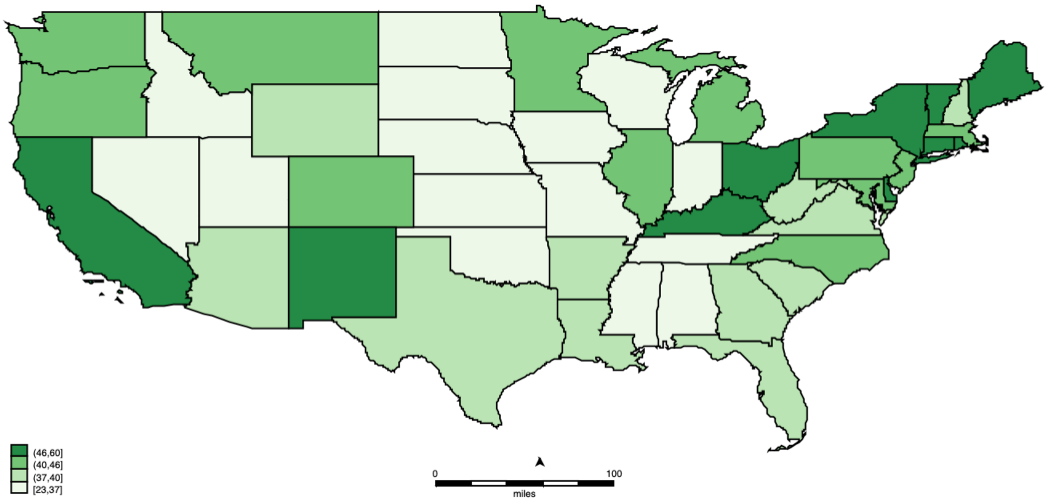


Figure 3.3: Mean Government Response Index choropleth map

exhibit higher values Midwest and South, and lower values on the West and Mid-Atlantic region.

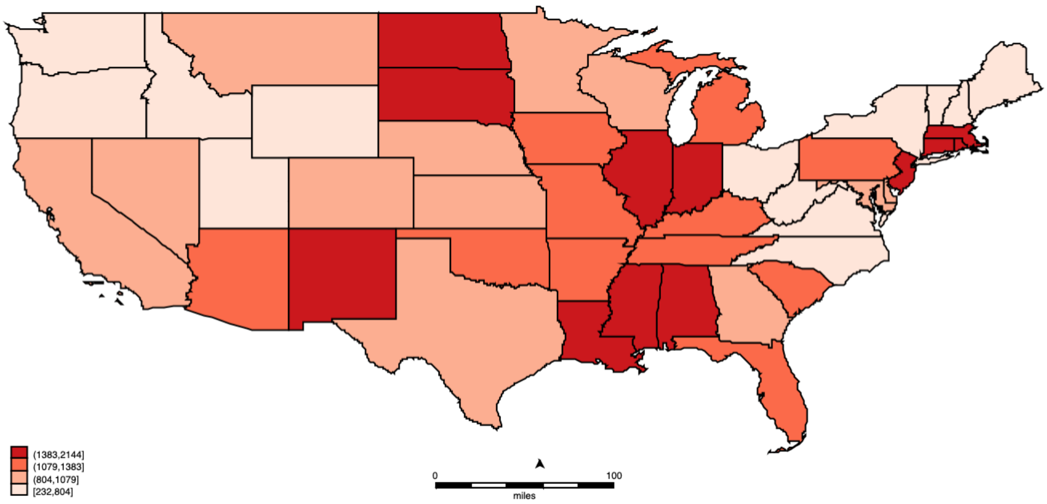


Figure 3.4: COVID-19 deaths per million inhabitants choropleth map

3.6.2 Model estimates

All eight in total specifications (with and without spatial lag for per capita GDP) show an inverted U-shaped relationship between the Gini index and GDP per capita—very much like the one Kuznets (1955) proposed—as it is evident from the negative statistically significant quadratic term as can be seen in Table 3.2. This points towards the co-existence of a trade-off and a complementarity depending of the level of income. The interaction term with any of the State's response indices was positive and statistically significant. This implies that the intensity of the government responses amplifies the trade-off for states with above the mean government response indices. On the other hand, all State responses are negatively associated with wage inequality, a finding that is also statistically significant. Nevertheless, since every interaction is a two-way interaction, this association depends also on the interaction term.

The spatial lags for the Gini index and the per capita GDP were both significant, and positive and negative, respectively. Since the coefficients of special autoregressive models do not represent marginal effects, the direct, indirect and total effects were calculated post-estimation.[8] The direct effects were statistically significant in all specifications. The indirect effects, on the other hand, were not statistically significant with the exception of the ESI specification where they were significant at the 10% level only.

Here I present only the results for the GRI specification (Table 3.2) which is the composite index. The empirical results for its components (i.e., SI, CHI, and ESI) can be found in Appendix B.

3.6.3 Sensitivity analysis

As a sensitivity analysis, I also performed the analysis on a more disaggregate level, namely on a subset of Metropolitan Statistical Areas (MSAs; N=326)[9] using 2020 data. Since the Gini index for wage inequality was not available, a decile dispersion ratio, namely the ratio between the median wage and the lower 10th percentile of wages (D5/D1),[10] was used as the dependent variable. This ratio captures the lower-

[8]The direct effects are the average impact of a regressor from a state on the dependent variable of that state. The indirect effects are the impact of a regressor of all other neighbor states on the dependent variable of an individual state, averaged for all states. The total effects equal the direct and indirect effects.

[9]From a total of 384 MSAs, 18 were removed due to missing values and 40 due to the spatial specification as 'islands'.

[10]D5/D1 had also a strong positive correlation with another widely used measure of inequality, that of D9/D1 (the ratio between the upper 10th and the lower 10th percentile of wages) in our data.

Table 3.2: Estimates for the GRI specification

	Coefficient	*Direct effect*	*Indirect effect*	*Total effect*
		Model 3.1		
ln(GDP)	1.637***	1.685***	0.754	2.438***
	(0.438)	(0.447)	(0.526)	(0.803)
$[\ln(\text{GDP})]^2$	-0.077***	-0.079***	-0.035	-0.114***
	(0.020)	(0.021)	(0.025)	(0.037)
GRI	-0.958***	-0.986**	-0.441	-1.428***
	(0.323)	(0.331)	(0.319)	(0.549)
ln(GDP)×GRI	0.089***	0.092***	0.041	0.133***
	(0.030)	(0.031)	(0.030)	(0.051)
W×Gini	0.329**			
	(0.155)			
Constant	-8.394***			
	(2.359)			
Observations	49			
Pseudo R^2	0.260			
Log-Likelihood	131.6			
		Model 3.2		
ln(GDP)	1.772***	1.835***	0.892	2.727***
	(0.388)	(0.404)	(0.617)	(0.870)
$[\ln(\text{GDP})]^2$	-0.082***	-0.086***	-0.046	-0.132***
	(0.018)	(0.019)	(0.030)	(0.041)
GRI	-0.889***	-0.924***	-0.500	-1.424***
	(0.287)	(0.296)	(0.329)	(0.535)
ln(GDP)×GRI	0.083***	0.087***	0.047	0.133***
	(0.027)	(0.027)	(0.031)	(0.050)
W×Gini	0.376**			
	(0.149)			
W×ln(GDP)	-0.070***			
	(0.020)			
Constant	-8.478***			
	(2.081)			
Observations	49			
Pseudo R^2	0.444			
Log-Likelihood	137.2			

Notes: Dependent variable (Gini) = Gini index; ln(GDP)= natural logarithm of real per capita GDP; GRI = dummy variable indicating whether the Government Response Index of a US state is above (=1) or below (=0) the sample mean; **W** the contiguity matrix. Robust standard errors are reported in parentheses. ***, ** and * indicate significance at the 1%, 5% and 10% level, respectively.

tail wage inequality—which probably entails the individuals mostly affected by the pandemic interventions—and is frequently used as a measure of inequality in the US (Autor et al., 2008).

The data on D5/D1 were extracted and computed from the Bureau of Labor

Statistics, while the data on GDP were obtained from the Bureau of Economic Analysis as in the State-level analysis and were adjusted using Regional Price Parities (RPP) to capture the difference in price levels across MSAs. The descriptive statistics of the above variables are presented in Table 3.3.

Table 3.3: Descriptive statistics for Metropolitan Statistical Areas

Variables	*Obs.*	*Mean*	*S.D.*	*Min.*	*Max.*
D5/D1	326	1.805	0.189	1.250	2.654
GDP	326	55.641	14.155	28.084	163.120

Notes: *D5/D1* = The ratio between the median wage and the lower 10th percentile of wages; *GDP*= natural logarithm of per capita GDP (in thousands USD) in regional price parities.

The government response indices are only available at the State level, hence new dummies were created with the method described in Section 3.4.2 for the MSAs that belong to a single State. In the case of multi-state MSAs, the mean of these States was used. We estimate the specification where both the inequality index and the income variable are spatially lagged (see Model 3.2 in Section 3.5.3) using a row standardized contiguity matrix. The corresponding estimation results for GRI are presented in Table 3.4 and for the rest of the indices in Appendix B. Overall, the results are similar between indices and aggregation level and corroborate the main findings of the state-level analysis.

3.7 Discussion

The discussion that follows is based on the wage inequality, as calculated by Gambau Suelves et al. (2021), using the Lockdown Working Ability index. This is important, because these estimates already account for a potential new distribution of wages during the COVID-19 pandemic.

In this chapter I focused on the effect of COVID-19 pandemic State interventions on the equity-efficiency relation. The findings indicate an inverted U-shaped relation between equity and efficiency. The inverted U-shape indicates that depending on the

Table 3.4: Model 2 estimates for the GRI specification at the MSA level

	Coefficient	*Direct effect*	*Indirect effect*	*Total effect*
ln(GDP)	1.368**	1.513**	0.801*	2.313**
	(0.589)	(0.666)	(0.443)	(1.101)
$[\ln(\text{GDP})]^2$	-0.130*	-0.147*	-0.0958*	-0.243*
	(0.0737)	(0.0835)	(0.0558)	(0.138)
GRI	-1.007***	-1.141***	-0.743***	-1.884***
	(0.255)	(0.288)	(0.214)	(0.489)
ln(GDP)×GRI	0.237***	0.268***	0.174***	0.443***
	(0.0636)	(0.0720)	(0.0528)	(0.122)
W×(D5/D1)	0.465***			
	(0.0408)			
W×ln(GDP)	-0.131***			
	(0.0405)			
Constant	-1.872			
	(1.169)			
Observations	326			
Pseudo R^2	0.388			
Log-Likelihood	206.8			

Notes: Dependent variable (D5/D1) = The ratio between the median wage and the lower 10th percentile of wages; $ln(GDP)$ = natural logarithm of per capita GDP (in thousands USD) in regional price parities; GRI = dummy variable indicating whether the Stringency Index of an MSA is above (=1) or below (=0) the sample mean. Heteroskedasticity robust standard errors are reported in parentheses. ***, ** and * indicate significance at the 1%, 5% and 10% level, respectively.

position on the curve, efficiency can raise or reduce equity in a State. This finding is in accordance with the literature which claims that there is no single pattern in the relation.

At first glance, it appears that all State's government pandemic responses intensify the equity-efficiency trade-off at the state level creating an additive quantitative modification effect. This is conditionally true. Due to the complexity of our model specification let us use Figure 3.5 to better interpret the results of Tables 2–5. The purpose of Figure 3.5 is to aid in the analysis, and not to provide exact empirical predictions for the Gini index.

Looking at Figure 3.5, we observe that in all specifications, States exhibit a trade-off or complementarity depending on their per capita GDP levels. Moreover, State interventions appear to shift the curve to the right. For low levels of per capita GDP, this translates to States with above mean interventions (dashed line) exhibiting a lower wage inequality compared to States with below the mean response

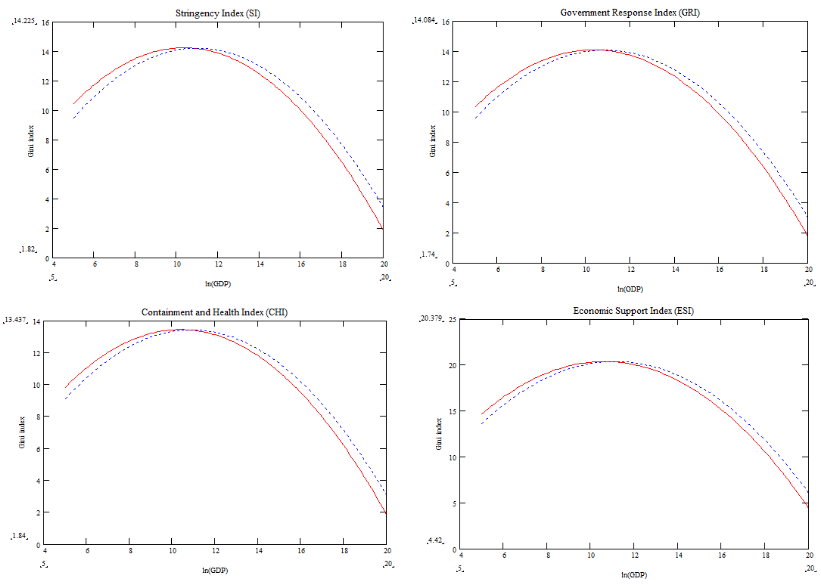


Figure 3.5: Graphical representation of the equity-efficiency relation for states with above mean (dashed blue) and below mean (red) government responses. Graphs were produced using the estimated total effects from Model (2)

indices (solid line). Contrariwise, for high levels of per capita GDP, we observed higher levels on wage inequality for States with above mean interventions.

The impact of response indices on wage inequality is twofold in our model; at each per capita GDP level, wage inequality is negatively affected by more intense government responses (i.e., a negative total effect of the indicator variable), and positively affected by the fact that more intense responses augment the negative impact of per capita GDP on equity (i.e., a positive total effect of the interaction term). Figure 3.5 demonstrates that the latter effect— the increase in wage inequality for above the mean interventions—dominates the former, for States with higher per capita GDP. Moreover, the above composite effect of COVID-19 State responses leads to another interesting result; States with below the mean response indices require lower per capita GDP levels to move from the efficiency-equity trade-off to the efficiency-equity complementarity.

In sum, State's interventions might have been beneficial for States with lower income, reducing the trade-off between wage inequality and per capita GDP, but not for States with higher per capita GDP whose complementarity relation lessened.

Policy-wise, reducing equity-efficiency complementarity in richer States in order to blunt the trade-off in poorer States is a sound policy for regional integration. Nevertheless, with the exception of the economic support, these measures were taken for the containment of the pandemic and not for exercising socioeconomic policy.

The higher position of the efficiency-equity curve for those States with more intense COVID-19 State government responses in the downward-sloping part of the curve maybe be the result of pre-pandemic levels of wage inequality. These levels might have played a role in the State's decision making for COVID-19 interventions. For example, States with high wage inequality before the pandemic, may had to step in more intensively because of worst epidemiological outcomes and the need of additional economic support for workers. Indeed, income inequality before the pandemic did affect the COVID-19 cases and deaths in US counties (Tan et al., 2021) and states (Oronce et al., 2020). In other words, more intensive intervention occurred to States that faced more severe pandemic effects. It is also logical that regions with higher stringency will probably need higher economic support in the future.

A difficulty lies in interpreting why the trade-off effect intensifies due to State government interventions during the pandemic (positive total effect of the interaction term) in the case of State economic support (ESI). A possible explanation is that lockdown measures introduce a force that distorts economic efficiency and at the same time raises inequality, namely wage inequality, since not all workers have the ability to work from home. For essential workers the wage may remain intact, but for non-essential workers the wage might become even zero. The above effect may increase the structural unemployment (preference towards teleworking) for the States with more intense COVID-19 government interventions[11] and in turn lead to a more intense efficiency-equity trade-off.

The insignificant indirect effects demonstrate that spillovers were limited. ESI being weakly significant indicates that the trade-off of neighbors is intensified and

[11] The level of economic support provided during the pandemic may increase moral hazard in a similar way as the level of unemployment insurance benefits do.

their inequality is reduced because of the economic support a State receives. This spillover effect may arise from stronger economic links between neighboring States, but further research is needed to test this hypothesis.

This research has clearly some limitations. First and foremost, our cross-sectional static design limits the quality of evidence since the direction of the association cannot be determined and no causal claims can be made. This issues can be overcome when post-pandemic data become available and a treatment evaluation approach can be performed. A second limitation is the level of aggregation. This level of analysis assumes homogeneity of exposure to interventions within states, ignoring intrastate variation. Despite the fact that federal government and states dictated the responses, there was variation at the local level regarding the responses and the level of enforcement and compliance to them.[12] Unfortunately, pandemic responses indices are not available at a more disaggregate level (e.g., county) so even if the analysis was performed at that level—as was in the sensitivity analysis—the homogeneity assumption within state would have to be made. Consequently, the cross-sectional design and the aggregate level of the analysis make the study prone to the reverse causality and ecological fallacy scenario.

All indices used for the analysis have several components where when studied one their own could yield different results with important policy implications. Nevertheless, since the total number of components is 41, future research will have to address the effect of individual components. Similarly, and given that the focus of this research was an advanced economy at the state level, an analysis at a different level of territorial disaggregation or of a different, e.g. developing, economy may yield different results. Therefore, the transportability of the results is limited.

Moreover, the wage inequality data come from estimated data and not a proper population-level survey. Another major drawback is the lack of control variables. Despite the fact that R^2 and pseudo-R^2 were high in OLS and MLE models for the cross-sectional data analyzed, this is no guarantee for correct specification and

[12]This can lead to erroneous measurement of the exposure to the intervention, which in turn may lead to over or underestimation of the association.

omitted variable bias is bound to exist in observational studies. The decision to not include additional controls was not made due to the lack of data, but due to the small number of observations which limits how many predictors can be used in our models. The fact that we study entities within a country somewhat mitigates the omitted variable bias, since some of the inequality determinants on cross-sections as described by Furceri and Ostry (2019) (i.e., the level of development, financial globalization, international trade, technology) may remain constant within the country. Finally, a decomposition analysis of the inequality, or further examination on whether the business cycle position during the pandemic would alter the results was not performed.

3.8 Conclusions

The aim of this chapter was to study the effect of the government responses on the equity-efficiency relation during the COVID-19 pandemic. Using a cross-section of 48 US states and the District of Columbia for the year 2020, the study found an inverted U-shape relation, where more intense government responses increase wage inequality in States with higher per capita GDP levels, but decrease it in States with lower per capita GDP levels. This provides evidence that the effect of spatially differentiated non-pharmaceutical interventions at the State level, such as lockdown restrictions and closures as well as income support, depends on the level of income.

Since no single pattern was observed, state policy design should consider its position on the curve when addressing the equity-efficiency trade-off during the pandemic. Alterations in the equity-efficiency relation during the pandemic might affect the efficacy of past, current, or future regional interventions. Their effect cannot be determined *a priori* but researchers and policymakers are advised to take it into account when studying similar subjects. To better assess the effect of the pandemic on the equity-efficiency trade-off, as well as the effect of government responses on the relation between efficiency and inequality, future research should employ a before and after methodology when more appropriate data become available.

In similar future crises one may expect more severe measures in regions with more inequality. Similar shocks may lead to a quantitative, or even qualitative effect modification on the equity and efficiency relation.

Chapter 4

Concluding remarks

This thesis aimed at examining relations between the health and economic geography of US states. The two parts were examined somewhat separately but with their interrelation always in mind. In general, a divide exists between Southern states and the rest of US in both dimensions.

Regarding health, disparities in premature mortality come mainly from preventable causes of death and health spending, either in public health or as personal expenditure, does not seem to be the answer. Christopoulos and Eleftheriou (2020b) have shown that spending more for healthcare does not do the trick for premature mortality, at least for developed countries. Perhaps interventions that aim at improving the socioeconomic status of underprivileged individuals might serve as a better counter to premature mortality. Of course, since the US case is a category of its own, a natural experiment in the form of a regional policy intervention is necessary for hard evidence.

For the economy part, the finding that the effect of COVID-19 pandemic State responses (mainly responses to secure public health) on the equity-efficiency relation is conditional on the income also highlights the importance of socioeconomic disparities when formulating policy. Public health interventions with the potential to seriously affect regional economic inequalities will in turn affect other (than the COVID-19 pandemic) public health outcomes. Failure to account for the long-term effects of these interventions can lead to devastating results in both economy and health.

The recent epidemic trends in infectious diseases indicate that it will not be long until the next public health crisis arrives. That is not to say that public health was safe before the COVID-19 pandemic, since that number one public health enemy, corporations, continue to operate very much unopposed.[1] Taking also into account the myriad health effects of the climate change, we have an explosive mix that threatens public health, and especially the most vulnerable populations. Therefore, urgent action is necessary in order to reduce health disparities and support those in need. Whether this support will be best delivered through economic or healthcare mechanisms is something that only, once again, a natural experiment can answer at the ecological level. Either way serious government intervention is needed in many levels, and ideally from a government for the people.

Several future topic research topics arise from the previous analysis. The ideal of natural experiments requires exploiting opportunities and is not always possible. Nevertheless, robust research is needed to assess the role of the access to, and quality of, healthcare in relation to premature mortality. Additionally, aside from studying for the health effects of the climate change, future research should focus on the aftermath of the COVID-19 regarding changes in wealth distributions as well as health outcomes in the short and long-run.

[1]Hastings (2012) explores the understudied corporate impact on public health, which functions with the blessing of the government agencies and the current economic system. Of course several books exist on the topic as well.

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Appendix A

Chapter 2 supplementary material

Table A.1: State abbreviations

State	*Abbreviation*	*State*	*Abbreviation*	*State*	*Abbreviation*
Alabama	AL	Kentucky	KY	North Dakota	ND
Alaska	AK	Louisiana	LA	Ohio	OH
Arizona	AZ	Maine	ME	Oklahoma	OK
Arkansas	AR	Maryland	MD	Oregon	OR
California	CA	Massachussetts	MA	Pennsylvania	PA
Colorado	CO	Michigan	MI	Rhode Island	RI
Connecticut	CT	Minnesota	MN	South Carolina	SC
Delaware	DE	Mississippi	MS	South Dakota	SD
District of Columbia	DC	Missouri	MO	Tennessee	TN
Florida	FL	Montana	MT	Texas	TX
Georgia	GA	Nebraska	NE	Utah	UT
Hawaii	HI	Nevada	NV	Vermont	VT
Idaho	ID	New Hampshire	NH	Virginia	VA
Illinois	IL	New Jersey	NJ	Washington	WA
Indiana	IN	New Mexico	NM	West Virginia	WV
Iowa	IA	New York	NY	Wisconsin	WI
Kansas	KS	North Carolina	NC	Wyoming	WY

Table A.2: ICD codes

Mortality Variable	*ICD-9 (1979-1998)*	*ICD-10 (1999-2017)*
Cancer	140-239	C00-D48
Cardio	393-398, 402, 410-414, 420-429	I00-I52
Injury	E800-E869, E880-E929	V01-X59, Y85-Y86
Infant	All codes	All codes
Suicide	E950-E959	X60-X84

Appendix B

Chapter 3 supplementary material

This Appendix presents the additional empirical results of Chapter 3 for the three components of GRI: ESI, CHI, and SI, first for the State level and next for the MSA level.

Table B.1: Estimates for the ESI specification

	Coefficient	*Direct effect*	*Indirect effect*	*Total effect*
		Model 3.1		
ln(GDP)	1.999***	2.134***	1.642*	3.775***
	(0.450)	(0.488)	(0.977)	(1.307)
$[\ln(GDP)]^2$	-0.093***	-0.099***	-0.076*	-0.175***
	(0.021)	(0.023)	(0.045)	(0.061)
ESI	-1.076***	-1.148***	-0.883	-2.031**
	(0.312)	(0.337)	(0.553)	(0.801)
ln(GDP)×ESI	0.099***	0.105***	0.081	0.186**
	(0.029)	(0.031)	(0.051)	(0.074)
W×Gini	0.470***			
	(0.142)			
Constant	-10.52***			
	(2.435)			
Observations	49			
Pseudo R^2	0.192			
Log-Likelihood	132			
		Model 3.2		
ln(GDP)	2.031***	2.170***	1.671*	3.841***
	(0.425)	(0.467)	(1.000)	(1.321)
$[\ln(GDP)]^2$	-0.094***	-0.100***	-0.811*	-0.181***
	(0.020)	(0.022)	(0.047)	(0.062)
ESI	-0.997***	-1.068***	-0.863*	-1.931**
	(0.297)	(0.320)	(0.519)	(0.753)
ln(GDP)×ESI	0.092***	0.098***	0.794*	0.178**
	(0.027)	(0.029)	(0.048)	(0.069)
W×Gini	0.484***			
	(0.137)			
W×ln(GDP)	-0.049**			
	(0.021)			
Constant	-10.22***			
	(2.309)			
Observations	49			
Pseudo R^2	0.270			
Log-Likelihood	134.7			

Notes: Dependent variable (Gini) = Gini index; ln(GDP)= natural logarithm of real per capita GDP; ESI = dummy variable indicating whether the Economic Support Index of a US state is above (=1) or below (=0) the sample mean; **W** the contiguity matrix. Robust standard errors are reported in parentheses. ***, ** and * indicate significance at the 1%, 5% and 10% level, respectively.

Table B.2: Estimates for the SI specification

	Coefficient	*Direct effect*	*Indirect effect*	*Total effect*
		Model 3.1		
ln(GDP)	1.811***	1.856***	0.745	2.601***
	(0.387)	(0.395)	(0.524)	(0.749)
$[\ln(\text{GDP})]^2$	-0.086***	-0.088***	-0.035	-0.123**
	(0.018)	(0.018)	(0.025)	(0.035)
SI	-1.327***	-1.359***	-0.545	-1.905***
	(0.297)	(0.303)	(0.383)	(0.558)
ln(GDP)×SI	0.123***	0.126***	0.051	0.177***
	(0.028)	(0.028)	(0.036)	(0.052)
W×Gini	0.303**			
	(0.151)			
Constant	-9.123***			
	(2.088)			
Observations	49			
Pseudo R^2	0.388			
Log-Likelihood	135.2			
		Model 3.2		
ln(GDP)	1.860***	1.915***	0.836	2.751***
	(0.356)	(0.368)	(0.577)	(0.791)
$[\ln(\text{GDP})]^2$	-0.087***	-0.090***	-0.043	-0.133***
	(0.017)	(0.017)	(0.028)	(0.037)
SI	-1.188***	-1.227***	-0.584	-1.811***
	(0.279)	(0.284)	(0.372)	(0.536)
ln(GDP)×SI	0.111***	0.114***	0.054	0.168***
	(0.026)	(0.026)	(0.035)	(0.050)
W×Gini	0.344**			
	(0.147)			
W×ln(GDP)	-0.055***			
	(0.019)			
Constant	-8.997***			
	(1.922)			
Observations	49			
Pseudo R^2	0.485			
Log-Likelihood	139.1			

Notes: Dependent variable (Gini) = Gini index; ln(GDP)= natural logarithm of real per capita GDP; SI = dummy variable indicating whether the Stringency Index of a US state is above (=1) or below (=0) the sample mean; **W** the contiguity matrix. Robust standard errors are reported in parentheses. ***, ** and * indicate significance at the 1%, 5% and 10% level, respectively.

Table B.3: Estimates for the CHI specification

	Coefficient	*Direct effect*	*Indirect effect*	*Total effect*
		Model 3.1		
ln(GDP)	1.649***	1.689***	0.664	2.353***
	(0.423)	(0.429)	(0.502)	(0.758)
$[\ln(\text{GDP})]^2$	-0.077***	-0.079***	-0.031	-0.110***
	(0.020)	(0.020)	(0.024)	(0.035)
CHI	-1.011***	-1.035***	-0.407	-1.442***
	(0.314)	(0.319)	(0.311)	(0.519)
ln(GDP)×CHI	0.094***	0.096***	0.038	0.134***
	(0.029)	(0.029)	(0.029)	(0.048)
W×Gini	0.299*			
	(0.161)			
Constant	-8.436***			
	(2.277)			
Observations	49			
Pseudo R^2	0.282			
Log-Likelihood	131.2			
		Model 3.2		
ln(GDP)	1.779***	1.829***	0.763	2.592***
	(0.385)	(0.397)	(0.573)	(0.809)
$[\ln(\text{GDP})]^2$	-0.083***	-0.085***	-0.397	-0.125***
	(0.018)	(0.018)	(0.027)	(0.038)
CHI	-0.926***	-0.955***	-0.445	-1.400***
	(0.289)	(0.294)	(0.306)	(0.496)
ln(GDP)×CHI	0.086***	0.089***	0.041	0.130***
	(0.027)	(0.027)	(0.029)	(0.046)
W×Gini	0.338**			
	(0.156)			
W×ln(GDP)	-00.64***			
	(0.021)			
Constant	-8.551***			
	(2.066)			
Observations	49			
Pseudo R^2	0.421			
Log-Likelihood	135.6			

Notes: Dependent variable (Gini) = Gini index; ln(GDP)= natural logarithm of real per capita GDP; CHI = dummy variable indicating whether the Containment and Health Index of a US state is above (=1) or below (=0) the sample mean; **W** the contiguity matrix. Robust standard errors are reported in parentheses. ***, ** and * indicate significance at the 1%, 5% and 10% level, respectively.

Table B.4: Model 2 estimates for the SI specification at the MSA level

	Coefficient	*Direct effect*	*Indirect effect*	*Total effect*
ln(GDP)	1.310**	1.451**	0.774*	2.225**
	(0.592)	(0.672)	(0.453)	(1.117)
$[\ln(\text{GDP})]^2$	-0.121	-0.138	-0.091	-0.229
	(0.074)	(0.084)	(0.057)	(0.141)
SI	-0.878***	-0.998***	-0.662***	-1.660***
	(0.254)	(0.289)	(0.211)	(0.490)
ln(GDP)×SI	0.205***	0.233***	0.155***	0.388***
	(0.064)	(0.072)	(0.052)	(0.122)
W×(D5/D1)	0.471***			
	(0.041)			
W×ln(GDP)	-0.133***			
	(0.041)			
Constant	-1.785			
	(1.175)			
Observations	326			
Pseudo R^2	0.370			
Log-Likelihood	204.1			

Notes: Dependent variable (D5/D1) = The ratio between the median wage and the lower 10th percentile of wages; $ln(GDP)$ = natural logarithm of per capita GDP (in thousands USD) in regional price parities; SI = dummy variable indicating whether the Stringency Index of an MSA is above (=1) or below (=0) the sample mean. Heteroskedasticity robust standard errors are reported in parentheses. ***, ** and * indicate significance at the 1%, 5% and 10% level, respectively.

Table B.5: Model 2 estimates for the CHI specification at the MSA level

	Coefficient	*Direct effect*	*Indirect effect*	*Total effect*
ln(GDP)	1.456**	1.626**	0.927**	2.552**
	(0.589)	(0.670)	(0.464)	(1.125)
$[\ln(\mathrm{GDP})]^2$	-0.141*	-0.161*	-0.108*	-0.268*
	(0.074)	(0.084)	(0.058)	(0.141)
CHI	-1.056***	-1.204***	-0.809***	-2.013***
	(0.259)	(0.295)	(0.228)	(0.510)
ln(GDP)×CHI	0.248***	0.282***	0.190***	0.472***
	(0.065)	(0.074)	(0.056)	(0.127)
W×(D5/D1)	0.476***			
	(0.040)			
W×ln(GDP)	-0.117***			
	(0.041)			
Constant	-2.123*			
	(1.170)			
Observations	326			
Pseudo R^2	0.371			
Log-Likelihood	208			

Notes: Dependent variable (D5/D1) = The ratio between the median wage and the lower 10th percentile of wages; $ln(GDP)$ = natural logarithm of per capita GDP (in thousands USD) in regional price parities; CHI = dummy variable indicating whether the Stringency Index of an MSA is above (=1) or below (=0) the sample mean. Heteroskedasticity robust standard errors are reported in parentheses. ***, ** and * indicate significance at the 1%, 5% and 10% level, respectively.

Table B.6: Model 2 estimates for the ESI specification at the MSA level

	Coefficient	*Direct effect*	*Indirect effect*	*Total effect*
ln(GDP)	1.181*	1.292*	0.631	1.923*
	(0.610)	(0.684)	(0.431)	(1.109)
$[\ln(\text{GDP})]^2$	-0.101	-0.114	-0.071	-0.185
	(0.077)	(0.086)	(0.055)	(0.140)
ESI	-0.656**	-0.738**	-0.463**	-1.201**
	(0.259)	(0.291)	(0.193)	(0.478)
ln(GDP)×ESI	0.148**	0.167**	0.105**	0.272**
	(0.065)	(0.073)	(0.048)	(0.119)
W×(D5/D1)	0.453***			
	(0.041)			
W*ln(GDP)	-0.130***			
	(0.042)			
Constant	-1.567			
	(1.201)			
Observations	326			
Pseudo R^2	0.396			
Log-Likelihood	202.4			

Notes: Dependent variable (D5/D1) = The ratio between the median wage and the lower 10th percentile of wages; $ln(GDP)$ = natural logarithm of per capita GDP (in thousands USD) in regional price parities; ESI = dummy variable indicating whether the Stringency Index of an MSA is above (=1) or below (=0) the sample mean. Heteroskedasticity robust standard errors are reported in parentheses. ***, ** and * indicate significance at the 1%, 5% and 10% level, respectively.