State of birth and cardiovascular disease mortality: Multilevel analyses of the National Longitudinal Mortality Study

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ABSTRACT

Cardiovascular disease (CVD) is the leading contributor to mortality in the United States. Previous studies have linked early life individual and family factors, along with various contemporaneous place-based exposures to differential individual CVD mortality risk. However, the impacts of early life place exposures and how they compare to the effects of an individual's current place of residence on CVD mortality risk is not well understood. Using the National Longitudinal Mortality Study, this research examined the effects of both state of birth and state of residence on individual's risk of CVD mortality. We estimated individual mortality risk by estimating multi-level logistic regression models. We found that during a follow-up period of 11 years, 18,292 (4.2%) out of 433,345 participants died from CVD. The impact of state of birth on subsequent CVD mortality risk are greater than state of residence, even after adjusting for socio-demographic factors. Individuals who were born in certain states such as Tennessee, Kentucky, and Pennsylvania on average had higher CVD mortality risk. Conversely, those born in California, North Dakota, and Montana were found to have lower risk, no matter where they presently live. This study implies that early life state-level environments may be more prominent to individual's CVD mortality risk, compared to the state in which one lives. Future research should address specific mechanisms through which state of birth may affect people's risk of CVD mortality.

1. Introduction

Cardiovascular disease (CVD) is the number one underlying cause of death in the United States, directly contributing to almost 660,000 deaths in 2019 (Roth et al., 2018). Despite continued advances in public health and medicine, recent data suggest that the decline of CVD mortality may have plateaued in the US (Martinez et al., 2020; Wilmot et al., 2015). This stagnating decline in CVD mortality, rather than the rising "deaths of despair", has been credited as the main reason why life expectancy in the US has stalled/declined in recent years (Mehta et al., 2020). Reducing CVD mortality remains a significant public health challenge in the US.

Incorporating life course theories of health, a strand of research has linked early life conditions to CVD risk in later life. The famous "Barker hypothesis" posits that fetal and infant growth programs the development of CVD risk factors later in the life course (Barker, 1991, 1995; Paneth & Susser, 1995). Building on this hypothesis, research has examined the relationships between a wide range of early life factors and later CVD risk. For example, socioeconomic disadvantage in early life has been associated with worse CVD outcomes in later life (de Mestral & Stringhini, 2017; Pollitt et al., 2007). Research has also linked low birth weight to elevated risk of incident ischemic heart disease (Huxley et al., 2007), coronary heart disease and stroke (Lawlor et al., 2005) and CVD mortality (Risnes et al., 2011). In addition, early-life psychosocial factors may also influence cardiovascular risk later in life. For instance, positive childhood factors such as having a positive home environment, greater levels of cognition, and child attention regulation have been associated with favorable cardiovascular health in midlife (Appleton et al., 2013).

Another advance in our understandings of how disease and mortality risks vary across peoples and places is the growing attention to the effects of contextual factors. The "ecosocial theory" of health (Krieger, 2001) suggests that, in addition to individual level biological, behavioral and psychosocial factors, contextual factors operating at different geographic scales may independently affect our health. Moreover, these contextual factors may interact with individual factors to influence our health in complex and intricate ways. Built on this conceptual framework, empirical research has increasingly incorporated the

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characteristics of our proximate and distant environments (e.g., natural, built or social) in research on cardiovascular disease outcomes in recent decades. One example is the urbanicity/rurality of place of residence (Cross et al., 2020; Kulshreshtha et al., 2014). Recent research has shown that between 1999 and 2017, residents in rural areas in the US had greater CVD mortality rates among all subgroups defined by age, sex and race/ethnicity, compared to those residing in metropolitan areas (Cross et al., 2020). Area socioeconomic status (SES) is another example where deprivation has been associated with higher individual CVD mortality risk, net of individual biological, behavioral and SES factors (Ford & Highfield, 2016; Major et al., 2010; Ramsay et al., 2015; Sánchez-Santos et al., 2013; Smith et al., 1998). Other factors related to place of residence have been associated with differential risk of CVD mortality, including residential exposure to environmental hazards (e. g., noise, air pollution) (Huss et al., 2010; Miller & Newby, 2020; Pope et al., 2011; Rajagopalan et al., 2018), neighborhood social context (e.g., social cohesion) (LeClere et al., 1998; Tamura et al., 2019) and access to health care (Macinko & Elo, 2009).

A few studies have looked at disparities in later CVD mortality risk based on place of birth. Researchers examined the "Southern-born effect" in the US where Americans born in the South had higher death rates from cancer, diabetes, heart and cerebrovascular diseases compared to persons born in other regions (Greenberg & Schneider, 1998; Schneider et al., 1997). Similar birthplace effects have been observed in other developed countries such as Finland (Valkonen, 1987) and the UK (Shaper & Elford, 1991). The results are suggestive that place of birth may have long lasting effects on circulatory diseases mortality regardless of place of residence at time of death. Thus, individual risk of CVD mortality may be better approached through a combination of the life course and contextual perspectives of health. Our study differs from previous ones in a few ways: first, we utilize a new dataset, the National Longitudinal Mortality Study (NLMS), that include information on both state of birth and state of residence to longitudinally examine their relationships with CVD mortality. Compared to studies carried out at more coarse geographies (e.g., census region) (Greenberg & Schneider, 1998; Mansfield et al., 1999; Schneider et al., 1997; Sorlie et al., 1995), our study may reveal the relationship between birthplace and CVD mortality at more granular geographic scales. Second, we estimate the effects of both state of birth and state of residence on CVD mortality risk at the individual level. Previous research on place of birth and CVD mortality in the US has mainly utilized area-based risk measures (e.g., regional death rates). Although these studies are indicative of the potential effects of place of birth on later CVD mortality, the results cannot be used to infer individual risk due to ecological fallacy (Piantadosi et al., 1988). Third, using multilevel regression modeling, our analytical approach accounts for the hierarchical nature of individuals nested within places. Within this structure, we estimate the effects of both state of birth and state of residence by assigning individuals multiple memberships according to their state of birth and state of residence. Specifically, we address the following questions: (1) between state of birth and residence, which explain a greater degree of variation in CVD mortality? (2) which states of birth and residence are associated with higher or lower CVD mortality risk?

2. Methods

This research utilized data from the public use file[1] of the National Longitudinal Mortality Study (NLMS). The NLMS is a nationally representative sample of non-institutionalized individuals in the United States established to examine social, economic, and demographic characteristics of the population (Sorlie et al., 1995). The public use file contains a subgroup of 11 of the 26 Current Population Survey (CPS) cohorts that are in the full NLMS. These 11 surveys, collected during the 1980s, were

combined together and re-weighted so they are representative of the United States population on April 1, 1983. For the individuals who are deceased, their records are linked with the National Death Index, the centralized mortality database (Curb et al., 1985). The third release of the NLMS public use file began to include state of residence and state of birth for the respondents for the first time. This allowed for the comparison of the importance of both state of birth and state of residence in predicting mortality risk. The analysis excluded respondents who had missing information regarding their state of birth, were foreign born, or were missing survey weights. Not every CPS collected place of birth information on survey participants. In our data, 96.6% of the records excluded were due to invalid place of birth information. Overall, the analytical sample is not a biased subsample of the full NLMS sample. The mean age (33.5) of those individuals excluded from the sample is similar to that of those included (31.2). The final analytic sample in total includes 433,345 individuals born in the United States.

2.1. Outcome measure

CVD Mortality. We used a binary variable to indicate whether or not a study participant died from CVD (0 = no, 1 = yes) during the 11-year follow up period. All causes of death were coded according to the International Classification of Diseases, ninth version (ICD-9) and diseases codes 390–459 were used to identify CVD deaths.

2.2. Place measures

State of Birth (SoB). State of birth was constructed from the Place of Birth (POB) variable in the initial study. The NLMS uses the National Death Index Geographic Codes for the participant state of birth. These state codes were then recoded according to the Federal Information Processing Standard (FIPS). Participants who were born outside of the contiguous US or had unknown place of birth were excluded.

State of Residence (SoR). State of residence was ascertained with State Recode (STATER), which was used to indicate the state of residence of the study participants at the time of their interview. The first digit of STATER in the NLMS is the Census Bureau division code, and the second digit is the specific state in each division. STATER was also recoded according to the state FIPS codes to ensure that both state of birth and state of residence conform to the same coding system.

2.3. Covariates

Age is a continuous variable, indicating the age of the study participants at the date of their interview for the NLMS. The variable is top coded at 90 years old, meaning that individuals who were any age above 90 were considered at baseline interview to be 90 years old. This covariate was used to adjust for the differential risks that come in older age with mortality from chronic illness such as CVD. Sex was recoded as 0 = male and 1 = female and was included in the regression models to account for the disparities in CVD mortality risk between men and women. Finally, race/ethnicity (non-Hispanic White, non-Hispanic Black, non-Hispanic Other, and Hispanic) was reconstructed from the original race and Hispanic origin variables and was used to account for the differential CVD mortality risk that exists between racial and ethnic groups. Participants without valid race or Hispanic origin information were excluded. Additional controls such as those relating to socioeconomic status were not included because those variables are conditioned upon state contexts. Including those variables could lead to the underestimating of the state effects on CVD mortality risk.

2.4. Statistical analysis

First, we summarized the descriptive statistics of the NLMS sample used in our study. The frequency distributions of study participants by sex race/ethnicity were calculated for the total sample and by mortality

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outcomes. For age, we calculated the mean, median and range of possible values. Next, a series of multi-level logistic regression models predicting individual CVD mortality risk were carried out. The first model included only state of residence random effects and the second model included only state of birth random effects. The third model included both state of birth and state of residence random effects in predicating CVD mortality risk. The fourth to sixth model were estimated in the similar way but all included sex, age and race/ethnicity covariates, accounting for differential CVD mortality risks associated with these factors. The fixed effects of demographic variables were expressed as odds ratios and 95% confidence intervals. State random effects were expressed as the variance of state-specific intercepts. We performed the statistical analysis using R (Version 4.0.3) (R Core Team, 2020).

To show the differences in CVD mortality risk by state of birth and state of residence, we created two maps showing the crude CVD mortality rates based on the two. We also created two figures ranking the state-specific intercepts on the odds scale from the fully adjusted model (model 6) to illustrate the heterogeneity in state-specific average CVD mortality risk, after adjusting for individual level covariates. Furthermore, we calculated the global Moran's I for SoB-specific intercepts from the fully adjusted multilevel model to explore whether state of birth effects on CVD mortality were clustered, dispersed or randomly distributed across the US states. We ran a Monte Carlo simulation based on 999 simulations to ensure the statistical stability of results. Following the global assessment, local Moran's I was also used to identify the locations of potential clusters of state of birth effects. When identifying clusters, neighboring states are defined by queen continuity, that is, states are considered neighbors if their boundaries share any common vertices. Global and Local Moran's Is were calculated using the *spdep* package (Bivand & Wong, 2018) in R.

3. Results

Descriptive statistics of the study sample are presented in Table 1. Among the 433,345 individuals in our sample, approximately 18,292 died from CVD. The average age at the interview of respondents who later died from CVD was 68.9 years old (SD = 12.6). Among those who died from CVD, 9561 (52.3%) were male, compared to 8731 (47.7%) who were female. For the racial breakdown, 16,037 (87.7%) were non-Hispanic white, whereas 1865 (10.2%) were non-Hispanic Black, and

Table 1
Descriptive statistics of the NLMS sample.

	Total (N = 433345)	Alive (N = 394068)	Death	
			CVD (N = 18292)	Other causes (N = 20985)
Sex				
Male	208376 (48.1%)	187154 (47.5%)	9561 (52.3%)	11661 (55.6%)
Female	224969 (51.9%)	206914 (52.5%)	8731 (47.7%)	9324 (44.4%)
Age (years)				
Mean (SD)	31.2 (21.0)	27.9 (18.4)	68.9 (12.6)	60.7 (17.8)
Median	28.0 [0, 90.0]	25.0 [0, 90.0]	70.0 [0, 90.0]	64.0 [0, 90.0]
[Min, Max]				
Race/ethnicity				
non-Hispanic White	359816 (83.0%)	325973 (82.7%)	16037 (87.7%)	17806 (84.9%)
non-Hispanic Black	46723 (10.8%)	42389 (10.8%)	1865 (10.2%)	2469 (11.8%)
non-Hispanic Other	9386 (2.2%)	8913 (2.3%)	178 (1.0%)	295 (1.4%)
Hispanic	17420 (4.0%)	16793 (4.3%)	212 (1.2%)	415 (2.0%)

the categories of non-Hispanic other and Hispanic made up 178 (1%) and 212 (1.2%), respectively.

Fig. 1 illustrates the cross-state differences in CVD mortality based on state of birth and state of residence. For state of birth, the highest crude CVD mortality rates were in Kentucky (66.7 per 1000 persons), Missouri (62.8) and Mississippi (61.5) while the lowest rates were in Nevada (7.9), Arizona (9.3) and California (10.2). For state of residence, the highest crude CVD mortality rates were in Florida (61.9), Missouri (57.2) and Kentucky (53.5) while the lowest rates were in Alaska (14.1), Hawaii (23.3) and Utah (26.1). One can see that differences in crude CVD mortality rate based on state of birth were greater than those based on state of residence. The state gaps in CVD mortality rate were 58.8 and 35.8 per 1000 persons for state of birth and state of residence, respectively.

Results from multilevel logistic regression models are presented in Table 2. Model 1–3 include only the random effects of state of birth and/or state of residence in predicting individual CVD mortality risk. In model 3 which includes both random effects, the variance of state of birth-specific intercepts (0.34) was almost five time of that of state of residence-specific intercepts (0.07). It means that a greater proportion of the variability in individual CVD mortality risk can be explained by state of birth than state of residence. Adjusting for age, sex and race/ethnicity fixed effects improved model performance in terms of predicting CVD mortality but reduced much of the state random effects, as demonstrated by results from model 4–6. However, model 6 showed that state of birth still had a larger effect on CVD mortality risk. The variance for state of birth (0.009) is nearly double that of state of residence (0.005), reinforcing the findings in model 3 that state of birth was a stronger predictor of CVD mortality than state of residence. These findings are consistent with those of previous work on the relationships between states of birth and residence and all-cause mortality (Xu et al., 2020). We further restricted our analytical sample to individuals aged 40 years and over. Results from the fully adjusted model (not presented) indicated that the random effects of state of birth continue to be greater than those of state of residence.

To better illustrate state differences, we rank state-specific intercepts from model 6 on the odds scale in Fig. 2 to show the deviation of state-specific average risks of CVD mortality from the national average. One can see that individuals born in Tennessee, Kentucky and Pennsylvania had the highest average CVD death risks, whereas those born in California, North Dakota and Montana had the lowest average risks. For state of residence, Michigan has the highest likelihood of mortality risk of CVD, along with several other midwestern states clustered towards the top. Contrarily, the state of Maryland shows the lowest CVD death risk. The greater variability in state of birth-specific intercepts also confirms the stronger effects of state of birth on CVD mortality.

To further explore the geographic patterning of state of birth effects on CVD mortality, we mapped state of birth intercepts from Model 6 as shown in Fig. 3(A). The results suggest a West-East division where Mountain and Western states of birth generally had lower average CVD mortality risks and those states in the Midwest, South and East had higher risks. The global Moran's I was significantly positive (Moran's I = 0.48, two-sided *p*-value < 0.01), suggesting that high and low state of birth intercepts were spatially clustered. Results from cluster analysis, as shown in Fig. 3(B), indicate two distinct clusters of state of birth intercepts. The cluster of high average state of birth CVD mortality risk consists of Kentucky, Tennessee, Virginia, West Virginia, and Pennsylvania while the low-risk cluster includes majority of states in the West census region.

4. Discussion

Most research regarding differential CVD mortality risk has emphasized individual level factors. Research examining effects of contextual factors on CVD mortality is growing in recent decades; however, they have mainly employed contemporaneous place-based exposures,
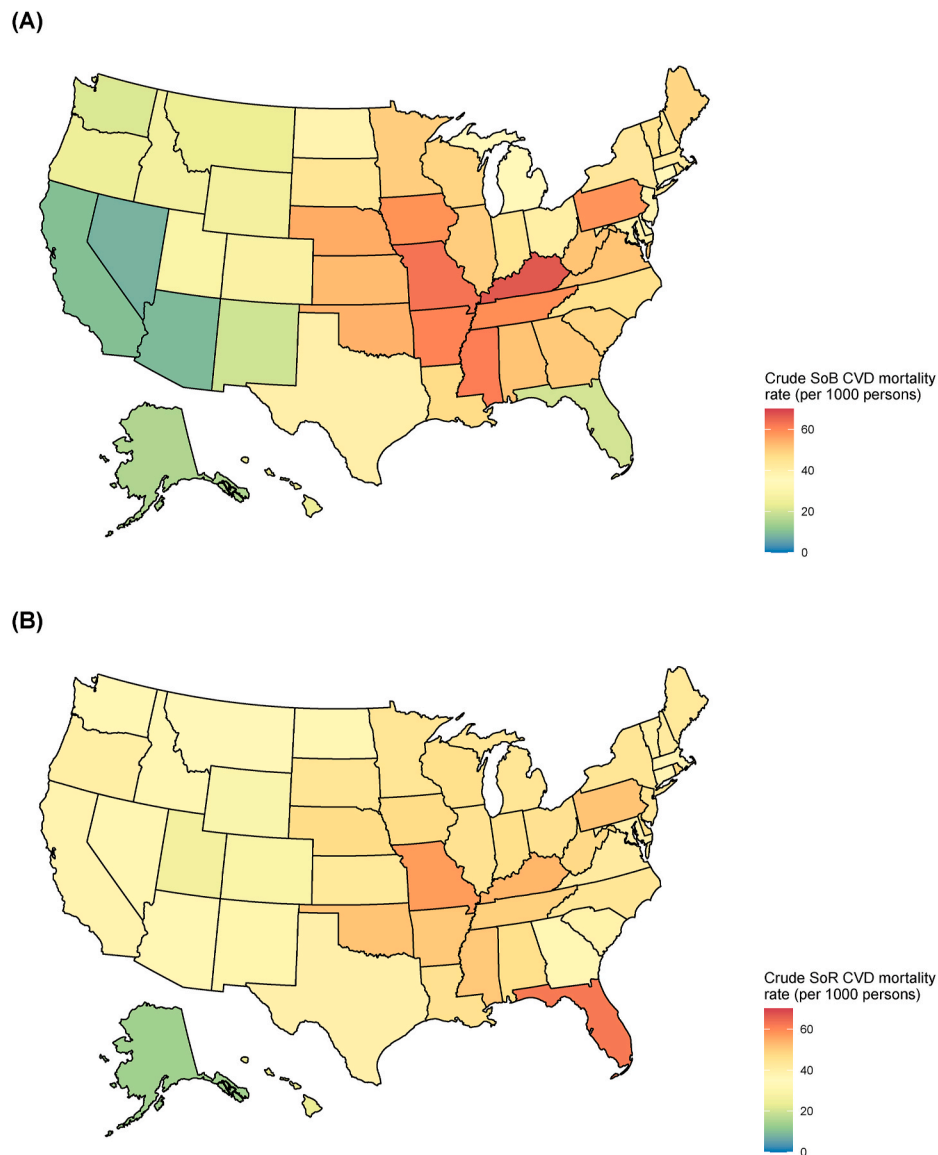
(A)

(B)

Fig. 1. Crude cardiovascular disease mortality rates based on (A) state of birth (SoB) and (B) state of residence (SoR).

Table 2
Multilevel logistic regression models predicting the risk of CVD death.

	Model 1 [a]	Model 2 [a]	Model 3 [a]	Model 4 [b]	Model 5 [b]	Model 6 [b]
Fixed effects						
Age				1.12*** (1.12, 1.12)	1.12*** (1.12, 1.12)	1.12*** (1.12, 1.12)
Sex (ref: Male)						
Female				0.57*** (0.55, 0.59)	0.57*** (0.55, 0.59)	0.57*** (0.55, 0.59)
Race/ethnicity (ref: Non-Hispanic white)						
Non-Hispanic black				1.23*** (1.16, 1.31)	1.22*** (1.14, 1.30)	1.22*** (1.14, 1.30)
Non-Hispanic other				0.98 (0.81, 1.17)	0.98 (0.82, 1.19)	0.99 (0.83, 1.20)
Hispanic				0.82*** (0.70, 0.96)	0.86 (0.73, 1.01)	0.85 (0.72, 1.01)
Random effects (variance)						
State of Birth		0.23	0.34		0.013	0.009
State of Residence	0.05		0.07	0.010		0.005
Diagnostics						
AIC	151111.9	149359.3	148690.0	88705.3	88692.1	88684.3
BIC	151133.9	149381.3	148722.9	88782.1	88768.9	88772.1
LL	−75554.0	−74677.7	−74342.0	−44345.6	−44339.0	−44334.2

Note: LL = Log Likelihood; AIC = Akaike information criterion; BIC = Bayesian information criterion.

[a] *p* < 0.05, **p* < 0.01, ***p* < 0.001. Fixed effects are presented in odds ratios.

[a] Models 1 to 3 estimated state of birth and state of residence random effects. The first model includes only state of residence, the second state of birth, and the third both state of birth and residence random effects.

[b] Models 4 to 6 are estimated in a similar way to the first three models, but include fixed effects of sex, age, and race/ethnicity covariates.
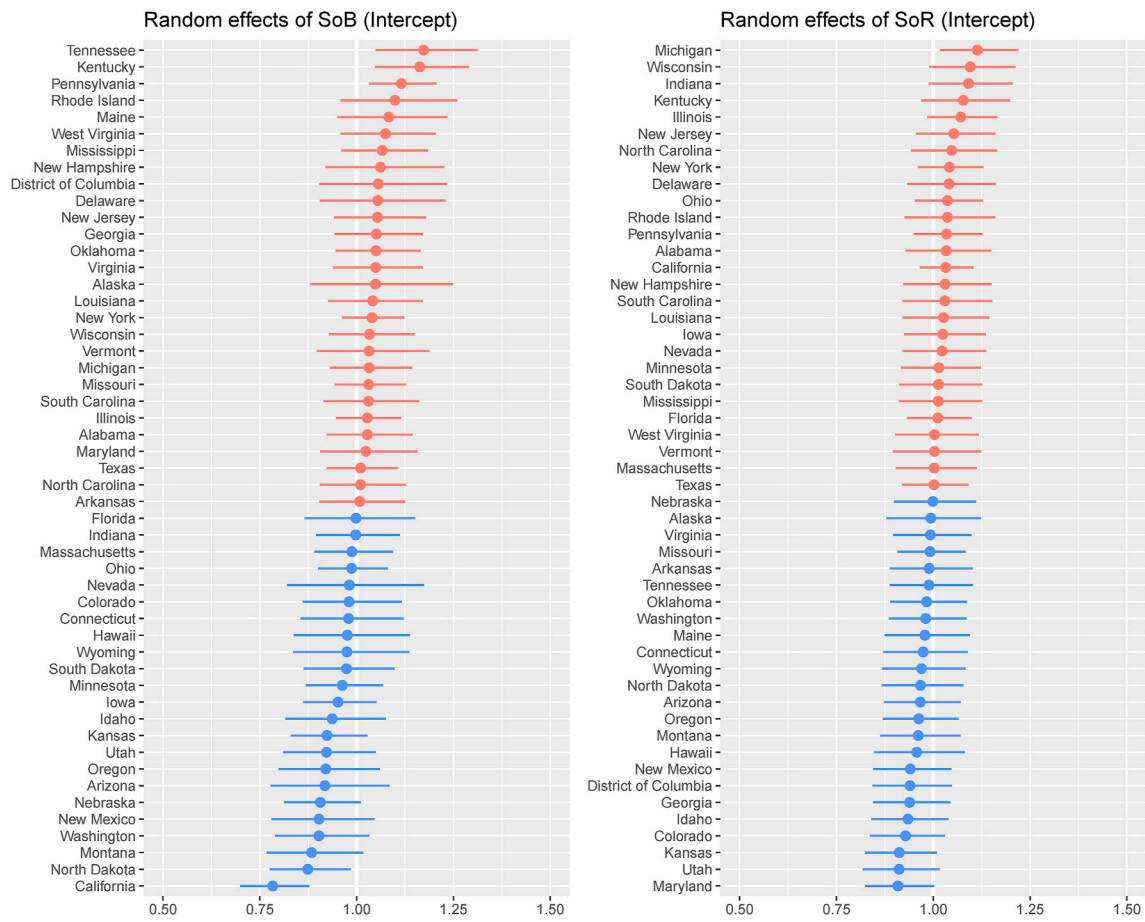
Fig. 2. Ranking of state-specific intercepts, representing state average CVD mortality risk on the odds scale. Intercepts are from logistic regression models with state of birth (SoB) and state of residence (SoR) random effects, adjusted for age, sex and race/ethnicity fixed effects.

ignoring the potential far-reaching effects of early life environments. Building from both the lifecourse and ecosocial theories of health, our study examines the relationship between early life place (i.e. state of birth), in addition to place of current residency (i.e. state of residence), and individual CVD mortality risk. The findings in this paper suggest several takeaways. First, we further the understanding of differential CVD mortality by including both state of birth and state of residence indicators. Regression diagnostics indicate that the models in which both state of birth and state of residence random effects are included better predict individual CVD mortality risk, compared to those that include state of residence alone. This suggests that state of birth may independently affect individual's risk of CVD mortality, regardless of where they currently live.

Second, we find that the random effects of state of birth on individual CVD mortality risk are greater than those of state of residence. This difference, although diminished, persists even after accounting for individual sociodemographic factors in the models. These results imply that an individual's place-based exposures during critical developmental periods (i.e., in utero, infant, childhood) may be more important to their CVD mortality risk at later life than where they currently live. The state environments people were born into may have long-lasting and irreversible effects on later CVD mortality risk.

Third, we display the intercepts of state of residence and state of birth to visualize how the states rank on the odds scale with regard to their average CVD death risk. Here, we show that regardless of where they currently live, persons who were born in certain states such as Kentucky, Mississippi and Tennessee have higher average CVD mortality risk than those born in other states, even after individual level factors are accounted for. In contrast, people born in California, Montana and

Washington had the lowest average risk. Our results are consistent with the "Southern-born effects" found in previous studies where Americans born in the South were burdened with elevated mortality risk of circulatory diseases (including cardiovascular diseases and stroke) (Glymour at al., 2009; Schneider et al., 1997) and cancer (Greenberg & Schneider, 1995). It is unclear why being born in states such as Kentucky, Mississippi and Tennessee is associated with higher CVD mortality as the mechanisms may be multidimensional. However, it has been suggested that income inequality, education, and racial composition of one's early state environment may be associated with later life ischemic heart disease and hypertension (Rehkopf et al., 2015). Other factors such as exposure to poverty in early childhood, differential dietary practices, access to medical care, exposure to environmental hazards, social norms affecting health behaviors, and social organization of local communities (Glymour et al., 2009; Greenberg & Schneider, 1995) may be also contributing to the observed differences. Considering the "southern-born effects" have been identified for a range of adverse health outcomes, it is reasonable to think that more structural factors may be driving the patterns.

This research has some limitations. First, the main geographic unit of analysis is state. We were unable to examine the heterogeneity of individuals' place of birth or residence at different sub-state levels (e.g., county, neighborhood). Given the within-state variability of various environmental factors, two individuals who were born and lived in the same state at the interview could have different exposures to more proximate places. While our work indicates elevated CVD mortality risk associated with being born in specific states/regions in the US, future research may examine place of birth (and/or residence) at more refined geographical scales to better pinpoint areas with the strongest birthplace

(A)

(B)
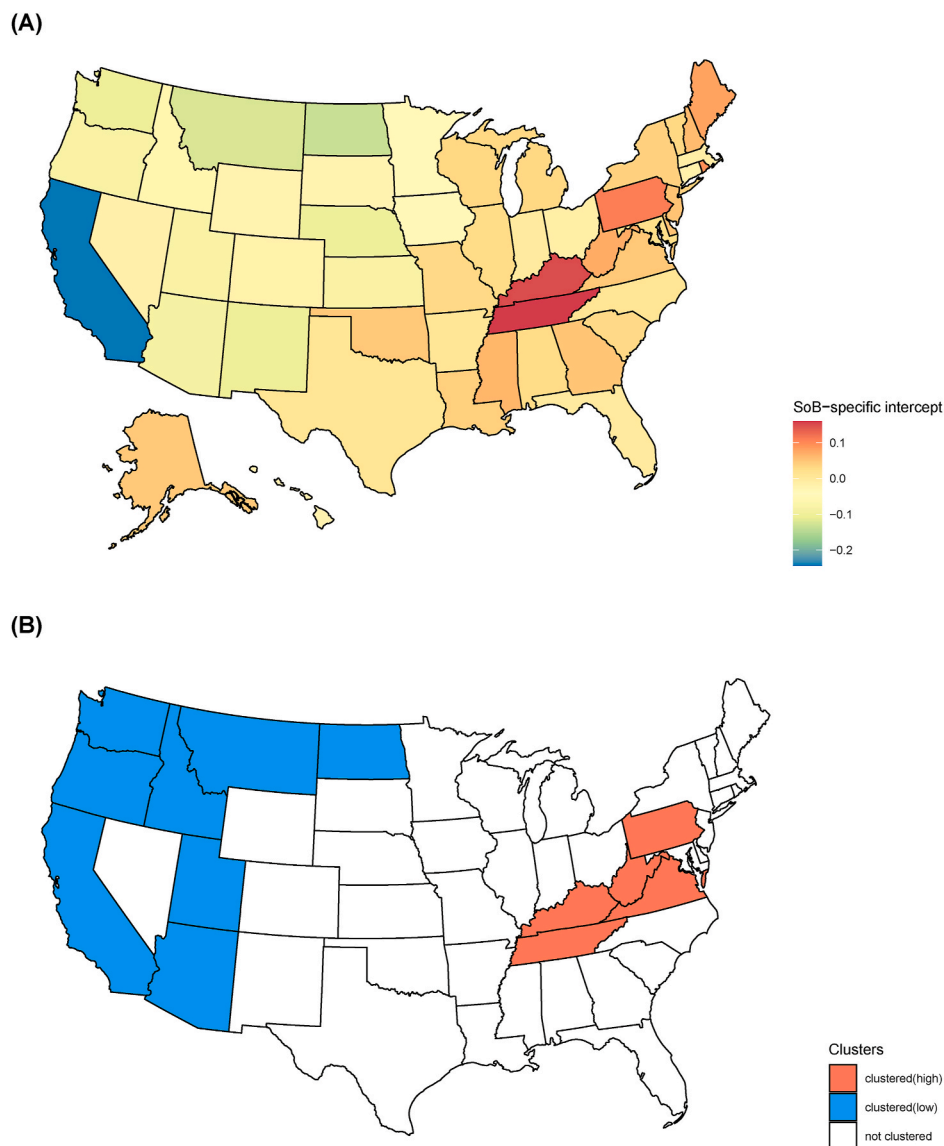
Fig. 3. Geographical patterns of state of birth (SoB) effects. (A) Maps of SoB-specific intercepts and (B) significant geographical clusters of states with high or low SoB-specific intercepts, based on local Moran's *I* statistics. We excluded Alaska and Hawaii in the calculation of local Moran's *I* because these two states are not spatial neighbors to other states.

effects. Second, this data utilized in this study does not include information on the timing of moving between states among movers and whether those stayers lived in other states for a substantial period of time. Therefore, the effects of state of birth on mortality later in life could have either a greater or lesser impact depending on when the individual moved, if so, from their state of birth. Incorporating durations of residency in both state of birth and state of residence will help us better understand the relative importance of each on later CVD mortality risk. Third, since we did not include any contextual variables for state of birth and state of residence (other than their random effects) in our models, we were unable to investigate the specific mechanisms in which both state of birth and state of residence may affect CVD mortality risk at later life as well as the reasons why state of birth may be more salient for CVD mortality risk compared to state of residence. Characteristics of state of birth may be directly associated with an individual's CVD mortality risk. For example, in utero exposure to environmental hazards such as air pollution may program poor fetal development and through various mechanisms induce adverse cardiovascular effects later in life (Burris & Baccarelli, 2017; Chin, 2015). State of birth may also indirectly affect CVD mortality risk through individual socioeconomic,

behavioral, and psychosocial pathways. Future research, incorporating more individual and state (of birth and residence) level factors, is needed to further our understanding of *how* early life place-based exposures are linked to later CVD mortality.

This research is one of the first to specifically link both state of birth and state of residence with CVD mortality. The results are indicative of the significance of early life place-based exposures, in addition to those in later life, in explaining the variability of individual CVD mortality risk. We show that the effects of state of birth are greater than those of state of residence, even after adjusting for individual socio-demographic factors. Future research should aim to examine whether the effects of early-life and contemporaneous place-based exposures persist for other major causes of death and explore whether these effects vary across population subgroups defined by sex, race and ethnicity. More importantly, future studies should also investigate the specific mechanisms through which state of birth, in addition to state of residence, may influence individual risk of CVD mortality. Evidence-based public health interventions that address early life place factors may help reduce CVD mortality at the population level and extend the overall life expectancy in the US.

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Contributors

WX and JF conceptualized the study. WX and JF were responsible for support in data collection activities. WX conducted the statistical analysis. MT and WX drafted the manuscript. WX, MT and JF contributed to interpretation of the findings and critically revised the manuscript. All authors approved the final version of the paper.

Declaration of competing interest

None declared.

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