Article

The legacy of slavery and contemporary declines in heart disease mortality in the U.S. South

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ABSTRACT

Background: This study aims to characterize the role of county-specific legacy of slavery in patterning temporal (i.e., 1968–2014), and geographic (i.e., Southern counties) declines in heart disease mortality. In this context, the U.S. has witnessed dramatic declines in heart disease mortality since the 1960s, which have benefitted place and race groups unevenly, with slower declines in the South, especially for the Black population.

Methods: Age-adjusted race- and county-specific mortality rates from 1968–2014 for all diseases of the heart were calculated for all Southern U.S. counties. Candidate confounding and mediating covariates from 1860, 1930, and 1970, were combined with mortality data in multivariable regression models to estimate the ecological association between the concentration of slavery in1860 and declines in heart disease mortality from 1968–2014.

Results: Black populations, in counties with a history of highest versus lowest concentration of slavery, experienced a 17% slower decline in heart disease mortality. The association for Black populations varied by region (stronger in Deep South than Upper South states) and was partially explained by intervening socioeconomic factors. In models accounting for spatial autocorrelation, there was no association between slave concentration and heart disease mortality decline for Whites.

Conclusions: Nearly 50 years of declining heart disease mortality is a major public health success, but one marked by uneven progress by place and race. At the county level, progress in heart disease mortality reduction among Blacks is associated with place-based historical legacy of slavery. Effective and equitable public health prevention efforts should consider the historical context of place and the social and economic institutions that may play a role in facilitating or impeding diffusion of prevention efforts thereby producing heart healthy places and populations.

Introduction

Heart disease has remained the leading cause of death in the U.S. since 1950 (Heron and Anderson, 2016), despite substantial declines over 50 years (Cooper et al., 2000). This reduction is due, in part, to public health prevention and improved clinical treatment (Ford et al., 2007). Declines in heart disease mortality are evident across racial, gender, and geographic lines. For example, the average U.S. county experienced a 62% reduction in heart disease mortality rates between 1973 and 2010, but this varied dramatically by region, with many southern counties experiencing slower declines substantially less than 50%, and northern and western counties experiencing faster declines from 65–82%. (Casper et al., 2016). Furthermore, county-specific

declines were faster for Whites (63% on average) than for Blacks (54% on average) nationwide (Vaughan, Quick, Pathak, Kramer, & Casper, 2015). In this context of notable but uneven progress in addressing a complex chronic disease, heart disease represents both a success story for public health efforts, as well as an opportunity to learn lessons about the challenges of promoting equitable improvements in population health.

Unequal rates of decline

Characterizing inter-group differences in the rate of decline in heart disease mortality can be instructive for both etiologic and public health prevention purposes. Geoffrey Rose (1994, 2001) argued that the
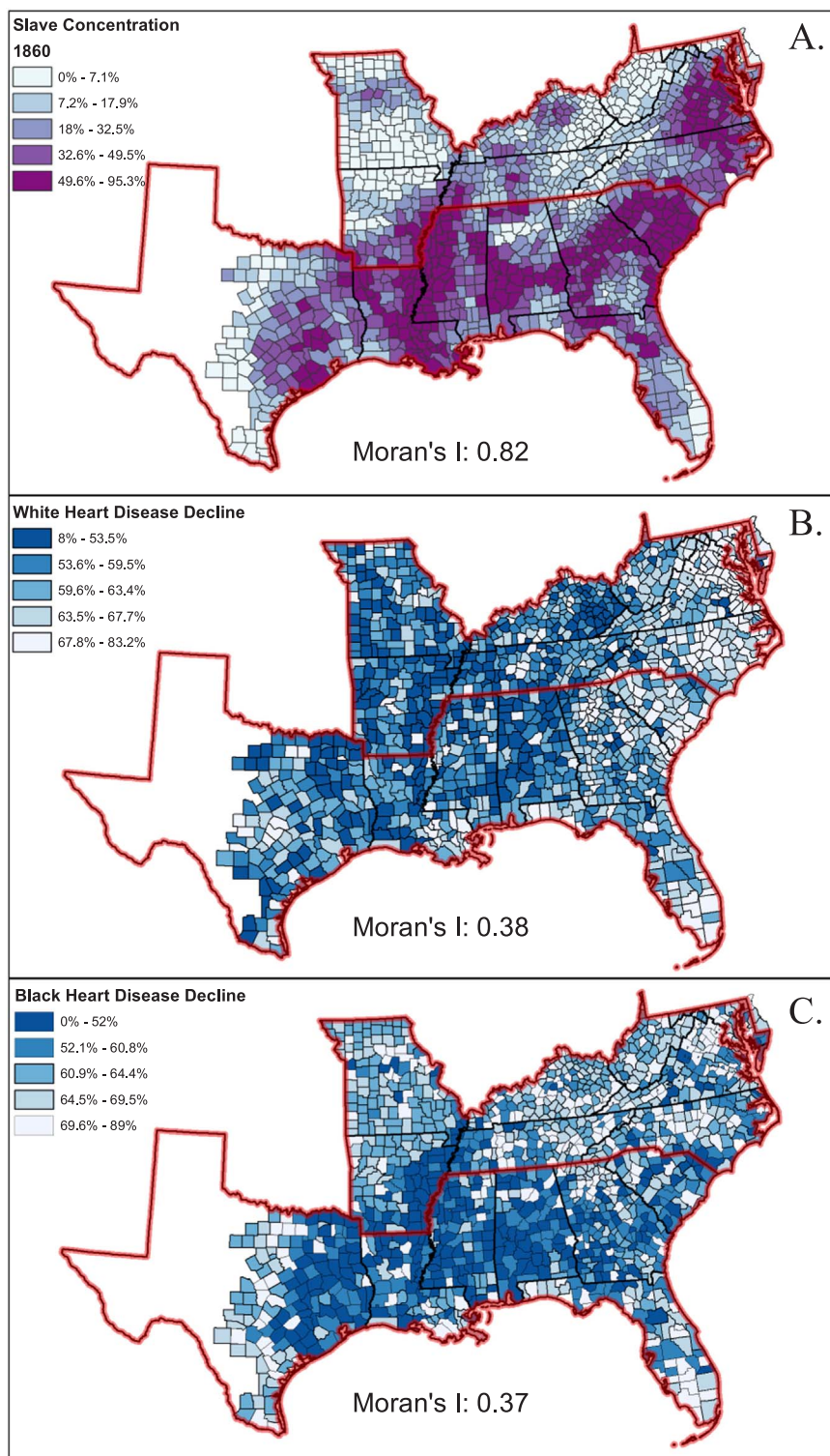

Fig. 1. Geographic variation in 1860 slave concentration, and relative decline in heart disease mortality by race, 1968–2014. Regions (Upper South & Deep South) outlined with red border. Panel A: Slave concentration (% of total population who are slaves) as measured in 1860 Census normalized to 2010 county and county-equivalent boundaries. Source: National Historic Geographic Information System (NHGIS). Panels B & C: Age adjusted, percent decline in mortality from all diseases of the heart between 1968 and 2014 for Whites and Blacks respectively. Source: National Center for Health Statistics. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

causes of sick individuals (within-population variation in disease *cases*) are not necessarily the same as the causes of sick populations (between-population variation in disease *incidence rates*) (Schwartz & Diez-Roux, 2001). The etiologies of cases and rates could differ for several reasons. For example, population group level processes may be direct upstream determinants of individual level risk behavior, such as when state and local smoke-free legislation, tobacco marketing, and taxation affect individual tobacco accessibility and affordability, resulting in variation in area-based smoking prevalence (Dwyer-Lindgren et al., 2014). Alternatively, contextual effects of local areas may indirectly affect health

behaviors through broad social norms, micro-level social network influences, or exposure of individuals to health-relevant socioeconomic opportunities and constraints embedded in places and institutions (Osypuk, 2013; Yusuf & Anand, 2010).

Fundamental social causes of disease (FSCD) theory suggests that inequities in disease rates between socially stratified groups reflect the groups' differential access to the technology, knowledge, and resources necessary to prevent or treat disease (Phelan, Link, & Tehranifar, 2010). For instance, in the absence of effective prevention or treatment, variation in disease rates is likely to be random; it is the unequal access to,

or adoption of, effective health promotion strategies, and the unequal exposure to health-detrimental factors that produces population differences in incidence and mortality rates. However, health relevant knowledge, technologies, and resources are not static over time: new understanding about individual and environmental determinants of health, changing social norms, and new diagnostic and treatment modalities for disease emerge more or less continually. Because early adopters of new health-relevant technology tend to be members of social groups with greater social, economic and political capital, health improvements tend to diffuse unevenly through populations (Phelan & Link, 2005).

Frequently, geographic places – specifically states, counties, and neighborhoods – are simplistically conceived as containers of populations, affecting health through trickle-down allocation and constraint of individual-level resources and exposures (Entwisle, 2007). However, theoretical literature and increasingly empirical work underscore the *relational* aspects of 'place', which emphasizes that the interaction between populations and places is multilayered, bidirectional, and historically contingent (Cummins, Curtis, Diez-Roux, & Macintyre, 2007; Macintyre, Ellaway, & Cummins, 2002; Matthews, 2008). Counties, for example, are important sub-national regions integrally involved in local governance, delivery of social welfare, taxation, regulation and delivery of health services, and oversight of political processes (Lobao, 2005; Lobao & Hooks, 2007). In this paper, we combine FSCD theory with a relational perspective about place to examine the degree to which inter-county variations in the rate of decline in heart disease mortality for Blacks and Whites in the U.S. South is a function of the county's history of chattel slavery.

The place-based legacy of slavery

History is embedded in places through the ongoing interaction of actors and institutions, making places 'durably distinct' from one another (Motlotch, Freudenburg, & Paulsen, 2000). An emerging body of literature documents the contemporary social, economic, and political legacy of one particular historical 'initial condition' of places: the concentration of slavery and a corollary dependence on slave labor in the U.S. South prior to the Civil War. A persistent legacy of slavery could be manifested through its effects on individuals or on places (O'Connell, 2012). For instance, individual descendants of slaves or slave owners might transmit cultural values, and human and economic capital across generations, producing and reproducing racial hierarchy well after legal emancipation. However, studies of Blacks remaining in, versus migrating out of, the South show that residence in places where slavery flourished have an independent effect on economic and social outcomes, above and beyond the ancestral connection to the experience of enslavement (Ruef & Fletcher, 2003; Vandiver, Giacopassi, & Lofquist, 2007).

In 1860, across counties in the U.S. South, there was substantial geographic variation in slave concentration (Fig. 1A). Moreover, southern counties with higher versus lower concentration of slaves in 1860 experienced slower 20th century economic development (Nunn, 2007); larger late-20th century Black-White disparities in poverty (O'Connell, 2012), educational attainment (Bertocchi & Dimico, 2012, 2014), and public school participation (Reece & O'Connell, 2015); and higher rates of violence (Gouda & Rigterink, 2015). Slavery is also associated with greater racial resentment and conservative political opinions among Whites (Acharya, Blackwell, & Sen, 2016). Building on this literature, we conceive of the legacy of slavery as a place-based attribute indicative of local institutional dependence on historical initial conditions. Hence, the institution of slavery was imprinted in the social, political, and economic institutions and norms across the US South. These enduring social products, and their differential distribution by race, are plausibly among the fundamental social determinants of both the pace of diffusion and the population-wide adoption of health-promoting knowledge and behaviors (Phelan & Link, 2005).

FSCD theory, thus, leads us to expect that the concentration of county-level slavery in 1860 will predict the rate of decline in heart disease mortality among Blacks and Whites, between 1968 and 2014; and the association will be stronger for Blacks than Whites. Furthermore, previously described socioeconomic consequences of the legacy of slavery will explain a portion of the hypothesized association.

Methods

Study design and population

In this ecological study, the unit of analysis is the county or county equivalent with all data normalized to 2010 Census boundary definitions. All counties in states where slavery was legal in 1860 were included in the analyses. For each county and year from 1968 to 2014, we extracted annual counts and age-adjusted rates of heart disease mortality stratified by race (Black and White) from National Center for Health Statistics data (Centers for Disease Control and Prevention (CDC), 2000a, 2000b, 2016). These years represent the longest series of high quality cause- and race-specific mortality data publicly available. Deaths were defined among adults 35+ according to the International Classification of Diseases (ICD) definition for "all diseases of the heart," which included the following codes: for ICD-8 (1968–1978), codes 390–398, 402, 404, and 410–429; ICD-9 (1979–1998), codes 390–398, 402, and 404–429; and ICD-10 (1999–present), codes I00–I09, I11, I13, and I20–I51. This inclusive definition of heart disease mortality has the benefit of substantial stability in coding across ICD versions with comparability ratios of approximately 1 (Anderson, Miniño, Hoyert, & Rosenberg, 2001; Klebba & Scott, 1980). To estimate stable county-level rates we pooled data into three-year periods (e.g. 1968–70; 2012–14) and then further stabilized small area estimates using empirical Bayesian techniques (Rao, 2003). We then calculated the relative change in mortality rates from 1968–70 to 2012–14 for each county and race group.

Data on slavery

The 1860 U.S. Census tabulated the total population, and also classified persons who were slaves; we used these variables to calculate the percent of each county population that was enslaved. Our analyses focus on places rather than individuals. Hence, county-level slave concentration represents an antebellum county attribute indicating the geographically varying dependence on a slave labor force as an economic system. This and subsequent county-level data was retrieved from the National Historic Geographic Information System Archive (Minnesota Population Center, 2011).

Covariates

Empirically linking the historical institution of slavery to contemporary population health dynamics is challenging. It requires comparing and contrasting county context and processes during the antebellum, as well as during the intervening 150 years. This task is made more challenging by the limited availability of historical data at the county level. Two broad concerns structure our selection of additional county measures. First, any observed association between slave concentration and heart disease decline could be confounded if a third factor was a determinant of each. For example, the location and density of plantations, and therefore demand for slave labor, might be affected by physical features such as land quality, proximity to a river, or potential for infrastructure development. Such geographic features could also influence 20th century economic development, resulting in places with more or less healthy populations, thereby inducing a spurious association between slave concentration and contemporary indices of population health. Second, the place-based legacy of slavery thesis argues that historical institutions influence contemporary populations

through their imprint on social processes inhering in contemporary local institutions. This usually takes the form of reproducing or modifying social processes leading to racial and economic inequality in social and economic opportunity (Reece & O'Connell, 2015). Because both individual and aggregate socioeconomic status determines health, these inequality-producing processes could mediate the legacy of slavery on racially distinct patterns of decline in heart disease mortality.

Candidate confounding variables, reflecting processes that precede and potentially determine the geographic variation in slave concentration, are drawn from the 1860 census. The total population count for each county indicates settlement patterns, distinguishing place based potential for growth and development. To measure the size and diversity of county economies we captured the average value of farmland and buildings per acre, and annual economic output from manufacturing in 1860.

Historic and contemporary racial inequality are evident in a wide range of Black-White disparities in social and economic indicators across the U.S. South. However, the local imprint of a legacy of slavery could be conceived of as geographic regulator of the magnitude of these racial disparities. To account for inter-county differences in educational opportunity and attainment, we measure Black-White disparities in illiteracy in 1930, and in college attainment in 1970. We measure inequality in economic opportunity by including variables for Black-White disparities in unemployment and median home value in 1930, and poverty rates in 1970. Measures from the 1930 census correspond to the approximate mid-point between the Civil War and our study period, and also encompass the early life course of adults most at risk for heart disease mortality during our study period. Measures from 1970 are closest to the beginning of our 46-year study period. A final candidate mediator of the legacy of slavery is the number of lynchings in each county between 1877 and 1950, which was tabulated by the Equal Justice Initiaitve (2015). Lynchings are an extreme form of political violence, the intent of which was to reinforce racial hierarchy and halt Black economic and political advancement (Beck & Tolnay, 1995).

Spatial interpolation

Some county boundaries have changed over 150 years, and new counties have been formed. We used spatial interpolation of variables based on the county boundaries from the year of source data to allow for comparison of counties through time. This technique allowed us to produce estimates aligned to the 2010 Census county boundary definitions. Interpolation methods are explained in more detail in the online supplement.

Statistical analysis

To answer the primary question of whether the concentration of slavery in 1860 is associated with the rate of decline in heart disease mortality, we fit unadjusted and confounder-adjusted race-stratified linear regression models with percent change in heart disease mortality between the 1968-70 and 2012-14 periods as the dependent variable, and concentration of slavery as the primary independent variable. To explore whether hypothesized intervening social and economic consequences of the legacy of slavery attenuated the slavery-heart disease association, a third model adjusting for confounders and intermediates was fit. All intervening measures of Black-White disparity were modeled as log-ratios representing excess Black 'risk' (e.g. ratio for poverty rates is Black/White, whereas ratio for median home value is White/Black) so that the sign of coefficients could be consistently interpreted.

Two spatial aspects of slave concentration, one substantive and one statistical, warrant elaboration. First, states permitting slavery in 1860 varied in their historic reliance on the slave trade, importance of large plantations, and role in the original Confederacy. Following Reece and

O'Connell (2015), we tested for effect modification by region, distinguishing the Deep South (Alabama, Florida, Georgia, Louisiana, Mississippi, South Carolina, and Texas) from the Upper South (Arkansas, Delaware, Kentucky, North Carolina, Maryland, Missouri, Tennessee, Virginia, and West Virginia). Second, because there is notable spatial clustering of slave concentration and heart disease decline, it is possible that our regression estimates are biased due to residual spatial autocorrelation. Moran's I for global spatial autocorrelation tests the null hypothesis that the residuals from conventional regression are spatially randomly distributed (Waller & Gotway, 2004). Rejection of this null suggests spatial autocorrelation or clustering of residuals and possibly biased regression coefficients. Moran's I statistics were calculated from residuals of all models, and when indicated selected spatial error or spatial lag econometric models were fit (Anselin & Rey, 2014). Spatial error models are appropriate when spatial autocorrelation is a nuisance arising from missing variables or spatially correlated predictors. In contrast, spatial lag models are indicated when the spatial structure is induced by contagion, diffusion, or spatial spillover processes from one county unit to the next. Lagrange multiplier tests provide guidance in selecting between error and lag model specifications (Anselin, Bera, Florax, & Yoon, 1996).

All maps were produced using ArcGIS 10.4 (ESRI, Redlands, CA) and all statistical analyses were carried out in R (R Core Team, Vienna, Austria).

Results

In 1860 there were 1100 counties within the boundaries of states legally permitting slavery (Upper South: 621; Deep South: 479), and in 2010 these same states consisted of 1,347 counties or county equivalents (716 and 624 for the Upper and Deep South, respectively). Spatial interpolation produced estimates of all variables in 2010 county boundaries, which were highly consistent with source geography (e.g. see supplement Figure S1). In 1860, slavery varied greatly throughout the South, ranging from 0–95% of the total population (mean 29%, median 25.4%) (see supplement, Figure S2).

To better understand the relation between 1860 slave concentration and both confounding and intervening variables, Table 1 summarizes all covariates across quintiles of slave concentration. Counties with higher slave concentrations were characterized by larger populations, higher land value and higher manufacturing output in 1860; higher Black, but not White, illiteracy in 1930; larger Black-White gaps in median home value in 1930; higher Black poverty in 1970, and a higher number of cumulative lynchings between 1877 and 1950. The proportion of Blacks in each county in 1930 and 1970 is highly correlated with the 1860 slave proportion (r = 0.88 and 0.82 respectively).

The spatial distribution of slavery and the relative decline in heart disease mortality by race are displayed in Fig. 1. There are slower declines for both Blacks and Whites in counties with higher rates of slavery particularly in the Deep South states of Alabama, Mississippi, and Louisiana; there is less apparent association in North and South Carolina and Virginia.

Table 2 summarizes conventional multivariable (but aspatial) modeled association between slavery and heart disease mortality decline. Because the dependent variable is percent decline, the interpretation of the coefficients is the change in the magnitude of decline (i.e., the 'pace of progress') in heart disease mortality due to each contrast in an explanatory variable. For example, in unadjusted models, Blacks in counties with the highest 1860 slave concentration experienced a 21% slower rate of decline than Blacks in counties with the lowest 1860 slave concentration (β -0.21, SE 0.014). With adjustment for confounders, this association is attenuated to 17.4% slower decline for Blacks in highest versus lowest slave concentration counties. For Whites, the association between slavery and heart disease decline is also significant, but much smaller in magnitude and in the opposite direction. For instance in the confounder adjusted model, contemporary

Table 1
Distribution of 1860, 1930, and 1970 covariates by quintile of 1860 slave concentration.

		Median value of county-specific variables by quintile of slaveholding in 1860				
		Q1 - lowest	Q2	Q3	Q4	Q5 - highest
N counties in 2010		269	270	269	270	269
1860	Total population count, 1860	5035	6748	7649	7864	10,849
	% Enslaved population	3%	12%	25%	41%	61%
	Average value of farmland and buildings per acre ($)	$5.3	$7.0	$7.5	$8.0	$11.2
	Annual value of manufacturing products ($)	$36,200	$73,845	$111,063	$100,120	$107,652
1930	Total population count, 1930	15,591	17,563	18,499	20,688	21,008
	% Black	1%	6%	19%	36%	54%
	% Whites over age 10 and illiterate	3%	3%	3%	3%	2%
	% Blacks over age 10 and illiterate	11%	12%	14%	15%	18%
	Illiteracy racial disparity (Black/White)[b]	3.24	3.52	4.61	5.51	7.29
	% Whites unemployed	5%	4%	4%	4%	4%
	% Blacks unemployed	6%	5%	4%	4%	3%
	Unemployment racial disparity (Black/White)[b]	1.20	1.31	1.23	1.06	0.86
	Median Home Value, Whites ($)	$1,945	$2,315	$2,590	$2,831	$3,370
	Median Home Value, Blacks ($)	$1,030	$1,028	$1,007	$900	$900
	Median Home Value disparity (White/Black)[c]	1.81	2.25	2.54	3.01	3.49
1970	Total population count, 1970	15,371	18,350	20,541	21,090	19,654
	% Black	1%	4%	14%	27%	42%
	Poverty rate, Whites, 1970	23%	17%	15%	15%	13%
	Poverty rate, Blacks, 1970	35%	38%	42%	49%	53%
	Poverty racial disparity (Black/White)[b]	2.00	2.32	2.88	3.15	3.98
	% White adults, bachelor degree	5%	5%	6%	7%	8%
	% Black adults, bachelor degree	1%	2%	3%	3%	3%
	Adults with a bachelor degree, racial disparity (White/Black)[c]	1.38	1.56	1.93	2.05	2.36
	Physicians per 10,000 population	4.0	4.6	4.7	4.7	4.2
	Rural/Urban					
	Metropolitan area	14%	23%	24%	26%	20%
	Non-metro town	43%	53%	53%	53%	57%
	Rural	43%	24%	23%	21%	23%
1877–1950	Cumulative number of documented lynchings[a]					
	0	82%	52%	31%	26%	14%
	1–10	14%	45%	63%	61%	66%
	11–20	0%	1%	3%	10%	16%
	> 20	0%	1%	1%	3%	4%

[a] Cumulative percent of counties by lynching category within each quintile of 1860 slaveholding may not sum to 100% due to rounding error.
[b] Racial disparity ratios represented as Black/White so that higher value represented larger White advantage.
[c] Racial disparity ratios represented as White/Black so that higher value represented larger White advantage.

declines in heart disease mortality were 5.2% faster (β 0.052, SE 0.014) for Whites in counties with a history of highest compared to lowest slave concentration. Finally, in models adjusting for hypothesized inequality-generating processes including Black-White disparities in educational and economic opportunity, and county cumulative incidence of lynchings, the association between slave concentration and heart disease mortality decline was approximately halved, although still significant, for Blacks; it was attenuated somewhat less for Whites.

There was evidence of significant autocorrelation of residuals in all models (Moran's I range from 0.19 to 0.33, p < 0.001), and Lagrange multiplier tests supported spatial lag econometric models as the best fitting solution. Two additional modifications were made to models reported in Table 3. First, based on significant interaction terms, we stratified on region (Deep South versus Upper South) to further address geographic heterogeneity. Second, while the slavery-heart disease association was linear in original models, after geographic stratification there was a significant quadratic relationship between slave concentration and heart disease decline. Therefore, coefficients for slave concentration and slave concentration squared are reported in Table 3. However, to facilitate interpretation, model-predicted and confounder-adjusted percent decline by region and race are graphically summarized in Fig. 2.

In both the Deep and Upper South regions, Blacks in counties with the lowest 1860 slave concentration actually experienced faster mean decline in heat disease mortality than Whites in those same counties (Fig. 2). However, as 1860 slave concentration increased, the rate of decline slows substantially for Blacks, but for Whites is either flat (Deep

South) or accelerated (Upper South).

Adjusting for region and accounting for spatial autocorrelation with spatial lag models, the relationships between slave concentration and declines in heart disease mortality persists for Blacks but not Whites (Table 3). For Blacks, the confounder adjusted association (β -0.17, SE 0.028) is consistent with conventional regression, although the confounder and intermediate adjusted association is not attenuated as much in spatial as compared to aspatial models (β -0.13, SE 0.033). In contrast, for Whites there is no significant association between 1860 slave concentration and contemporary heart disease decline after accounting for spatial dependencies in the data. It is important to note that the interpretation of spatial lag econometric coefficients is somewhat distinct from conventional regression. Because these models include a spatially weighted average value of the *dependent variable*, heart disease decline, as a predictor variable, they are commonly interpreted as evidence of contagion or spread of health or health behaviors from one county to its neighbors (Voss, Long, Hammer, & Friedman, 2006). The degree to which a given county's heart disease decline is predicted by neighboring counties heart disease decline is captured in the rho spatial lag coefficient reported in the last row of Table 3.

Discussion

In the last five decades, heart disease mortality has declined dramatically across the United States, but the rate of decline for both Whites and Blacks has been slower in the South compared to other regions of the country (Casper et al., 2016; Vaughan et al., 2015).

Table 2

Aspatial multivariable regression of 1860 slave concentration and percent decline in heart disease mortality, 1968–2014, by race.

		Unadjusted				M1 - Confounder Adjusted				M2 -Confounder + Intermediate adjusted			
		Black		White		Black		White		Black		White	
		β	SE	β	SE	β	SE	β	SE	β	SE	β	SE
1860	Slave Concentration	**-0.211**	**0.014**	**0.045**	**0.011**	**-0.174**	**0.017**	**0.052**	**0.014**	**-0.096**	**0.024**	**0.038**	**0.018**
	Population count (log)	**-0.010**	**0.003**	**0.005**	**0.002**	**-0.007**	**0.003**	-0.004	0.002	**-0.011**	**0.004**	-0.005	0.003
	Average value of farmland and buildings per acre ($)	0.001	0.004	**0.018**	**0.003**	**0.009**	**0.004**	0.005	0.004	**0.012**	**0.005**	0.001	0.004
	Annual value of manufacturing products ($)	0.000	0.002	**0.012**	**0.002**	0.001	0.002	**0.008**	**0.002**	0.002	0.003	0.004	0.002
1930	Illiteracy racial disparity log(Black/White)	**-0.042**	**0.005**	**0.013**	**0.004**					**-0.016**	**0.006**	-0.008	0.005
	Unemployment racial disparity log (Black/White)	**0.043**	**0.006**	**0.022**	**0.005**					0.011	0.007	**0.014**	**0.005**
	Median Home Value disparity log (White/Black)	**-0.049**	**0.007**	**0.039**	**0.005**					-0.005	0.009	**0.023**	**0.007**
1970	Poverty racial disparity log(Black/White)	**-0.070**	**0.007**	**0.039**	**0.005**					-0.021	0.011	**0.031**	**0.008**
	Adults with bachelors degree, racial disparity log(White/Black)	**-0.036**	**0.006**	**0.032**	**0.005**					0.006	0.007	**0.018**	**0.006**
	Physicians per 1000 population	0.008	0.005	**0.021**	**0.004**					0.003	0.005	**0.01**	**0.00**
	Rural/Urban												
	Metropolitan area	Ref		Ref		Ref		Ref		Ref		Ref	
	Non-metro town	**-0.042**	**0.008**	**-0.040**	**0.006**	**-0.036**	**0.008**	**-0.032**	**0.006**	**-0.040**	**0.009**	**-0.023**	**0.007**
	Rural	-0.013	0.010	**-0.030**	**0.007**	**-0.024**	**0.009**	**-0.018**	**0.007**	**-0.036**	**0.011**	0.006	0.008
1877–1950	Cumulative number of documented lynchings[a]												
	0	Ref		Ref		Ref		Ref		Ref		Ref	
	1–10	**-0.065**	**0.007**	-0.006	0.005					**-0.023**	**0.008**	**-0.019**	**0.006**
	11–20	**-0.143**	**0.014**	**-0.028**	**0.011**					**-0.067**	**0.015**	**-0.046**	**0.011**
	> 20	**-0.097**	**0.026**	-0.007	0.020					-0.032	0.026	**-0.046**	**0.020**
Region													
	Deep South	Ref		Ref		Ref		Ref		Ref		Ref	
	Upper South	**0.074**	**0.006**	**0.012**	**0.005**	**0.041**	**0.008**	**0.016**	**0.006**	**0.028**	**0.009**	**0.017**	**0.007**
Moran's I for residuals						0.220	p < 0.001	0.330	p < 0.001	0.19	p < 0.001	0.27	p < 0.001

p < 0.05 for bolded coefficients

Table 3

Spatial lag econometric regression of slave concentration and heart disease decline from 1968–2014, by race.

|		Confounder adjusted				Confounder + intermediate adjusted			
		Black		White		Black		White	
		β	SE	β	SE	β	SE	β	SE
1860	Slave Concentration	**-0.169**	**0.028**	-0.015	0.020	**-0.125**	**0.033**	-0.016	0.024
	(Slave Concentration)^2	**0.144**	**0.068**	0.085	0.050	0.133	0.072	0.081	0.052
	Population count (log)	-0.002	0.003	0.002	0.002	-0.007	0.004	-0.001	0.003
	Average value of farmland and buildings per acre ($)	0.006	0.004	0.003	0.003	**0.009**	**0.005**	0.000	0.003
	Annual value of manufacturing products ($)	0.001	0.002	**0.004**	**0.002**	0.002	0.002	0.002	0.002
1930	Illiteracy racial disparity log(Black/White)					-0.010	0.006	-0.003	0.004
	Unemployment racial disparity log(Black/White)					0.005	0.007	0.009	0.005
	Median Home Value disparity log(White/Black)					-0.011	0.008	0.011	0.006
1970	Poverty racial disparity log(Black/White)					-0.005	0.010	**0.020**	**0.007**
	Adults with bachelors degree, racial disparity log(White/Black)					0.004	0.007	**0.019**	**0.005**
	Physicians per 1,000 population					0.001	0.005	0.003	0.003
	Rural/Urban								
	Metropolitan area	Ref		Ref		Ref			
	Non-metro town	**-0.026**	**0.008**	**-0.020**	**0.006**	**-0.028**	**0.008**	**-0.016**	**0.006**
	Rural	-0.016	0.009	-0.008	0.006	**-0.025**	**0.010**	0.006	0.007
1877–1950	Cumulative number of documented lynchings[a]								
	0					ref			
	1–10					-0.011	0.007	-0.006	0.005
	11–20					**-0.039**	**0.014**	**-0.019**	**0.010**
	> 20					-0.008	0.024	-0.018	0.017
Region									
	Deep South	Ref		Ref		Ref			
	Upper South	**0.018**	**0.007**	0.004	0.005	0.014	0.008	0.010	0.006
Slave Concentration * Region		**0.069**	**0.034**	**0.052**	**0.025**	**0.071**	**0.035**	0.026	0.026
ρ (lag coefficient)		0.410		0.542		0.400		0.513	

p < 0.05 for bolded coefficients.
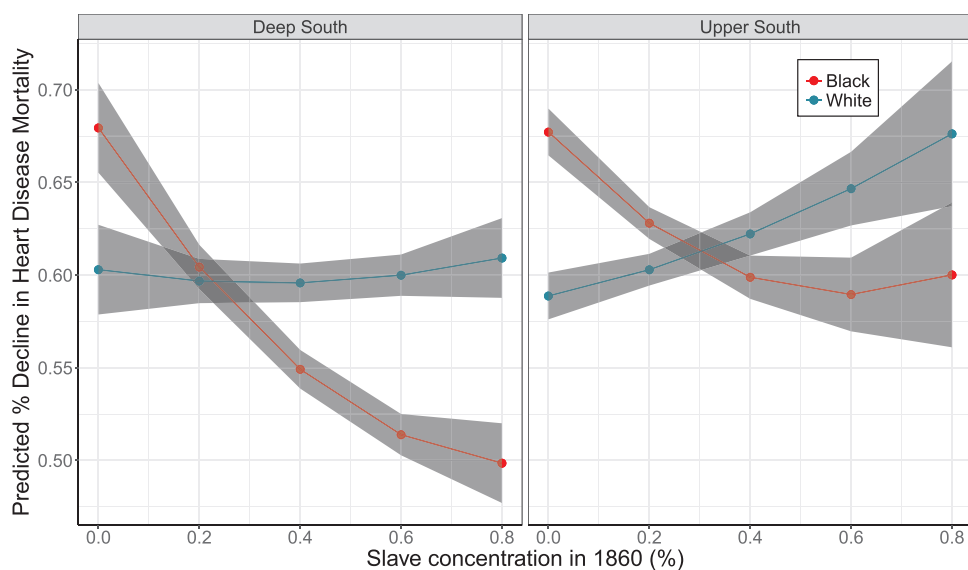
Fig. 2. Model-predicted association between 1860 county slave concentration and percent decline in heart disease mortality from 1968–2014, by region and race. Predicted percent decline and 95% confidence ribbons from models adjusted for log(1860 population count); 1860 average farm and building value per acre; 1860 average manufacturing output; and 1970 rural/urban status.

However, the South is not a monolithic region. The South has notable variation in heart disease mortality decline, including distinct patterns by race and place. The magnitude of reduction in heart disease mortality—the pace of public health progress—in counties at the end of the 20th century was associated with the concentration of slavery in 1860 for Blacks, but not Whites. Between 1968 and 2014, Blacks in counties with the highest 1860 concentration of slavery experienced slower rates of mortality declines compared to Blacks in counties with a history of lower slave concentration. Nearly half of this association is explained by inter-county 20th century differences in educational and economic racial inequalities, and the cumulative use of lynchings to enforce Black subordination.

Places and populations, not persons

This ecologic study uses places (counties) and populations (county-race groups) rather than individual persons as the object of study and the target of inference. Instead of being a limitation, this focus is important for at least two reasons. First, since the Civil War, there has been substantial Black migration out of, and return migration to, the South (Frey, 2004; Wilkerson, 2010). Therefore, without extensive genealogical data, we cannot determine whether contemporary populations are direct ancestors of slaves, or slave owners, in specific places. While most African-Americans in the U.S. descended from slaves in the South, the average county-level rates of decline in heart disease mortality for the Black population outside the South were substantially faster than the decline for the Black population within the South (Vaughan et al., 2015). We posit that the presence of slower declines for Blacks in—as opposed to outside—the South, and slower declines for Blacks within southern counties with historically more dependence on slave labor, suggests that it is the place and its associated institutions and social norms that transmit health consequences, independent of the inter-generational transmission of the trauma of slavery (Sotero, 2006).

Second, conventional explanation of inter-group differences in point-in-time rates of disease might include characterizing the static (or possibly time-lagged) 'risk profile' of aggregated individuals. However, by modeling rates of change, we capture the pace of progress (at least in the case of declining trends), and necessarily shift the focus from the health status of a group of individuals, to the spatiotemporally dynamic diffusion of heart-healthy factors through populations within places.

The diffusion of progress

Declining rates of heart disease mortality are reflective of enhanced biomedical knowledge and technology, evolving knowledge and norms around health risk behaviors such as smoking, and also the social, economic, and historical dynamics that influence population risk accrual across the life course (Harper, Lynch, & Smith, 2011). Age-period-cohort analyses suggest that the Black-White disparities in heart disease mortality in the U.S. were larger across the life course of cohorts born from 1920–1950 (Kramer, Valderrama, & Casper, 2015), and were narrowed as a result of abolition of Jim Crow laws in the South (Krieger et al., 2014).

The uneven allocation or ineffective diffusion of public health prevention, education, and interventions may occur for many reasons. In general, if there were no effective means of preventing a chronic disease, variation in incidence might be attributable to fixed features of the physical environment (e.g. potential nutrition), or host susceptibility (e.g. genetics). However, given effective approaches to heart disease prevention—for instance, healthy diet and food environments (Penney, Brown, Maguire, Kuhn, & Monsivais, 2015), physical activity and built environments (Chaix, 2009), community-level smoking regulation (Fichtenberg & Glantz, 2000), education, employment and economic opportunity (Armstrong, Strogatz, & Wang, 2004; Masters, Link, & Phelan, 2015), and medical screening and therapy (Ford et al., 2007)—the relative pace of progress in reducing mortality is determined by the individual and collective resources available to equitably implement these efforts (Phelan et al., 2010). Thus, the legacy of slavery, which is associated with economic investment in collective resources, including public education and social welfare (Engerman & Sokoloff, 2002), could also yield uneven diffusion of risk amelioration strategies. In addition, the coercive and sometimes violent social control systems that arose after slavery—sharecropping, lynchings, convict leasing, Jim Crow laws, and mass incarceration (Alexander, 2012; Krieger et al., 2014; O'Connell, 2012)—further separate Blacks from available resources, contributing to the accumulation of experienced racism, stress, and allostatic load which are associated with cardiovascular risk across the life course (Lewis, Cogburn, & Williams, 2015).

Determining trajectories of places

Nevertheless, history is not fully deterministic of destiny. We observed stronger negative impacts of the legacy of slavery on Blacks in the Deep South compared to counties in the Upper South. Compared to the Deep South, the Upper South region had stark variation in the rates of slavery but experienced relatively faster declines in heart disease mortality for both Blacks and Whites. These regional differences could

reflect the impact of distinct political and environmental ecologies of places on post-Reconstruction trajectories, including differences in geographic and environmental resources, economic development, and Black political power and social capital (Acemoglu & Robinson, 2013; Mayer, 1996). This may also reflect differences in perceived racial threat, racial residential composition, and variation in regulating the racial social hierarchy (Tomaskovic-Devey & Roscigno, 1996).

The magnitude and significance of the spatial lag coefficient is conventionally interpreted as evidence for contagion or diffusion of the dependent variable, in this case heart disease decline. While heart disease itself is not 'contagious', the norms and behaviors (e.g. smoking, dietary habits, physical activity) that drive it could be (Ball, Jeffery, Abbott, McNaughton, & Crawford, 2010; Luke & Stamatakis, 2012). Beyond the individual-level diffusion of behavior, it is also possible that the spatial scale of social, economic, and health service contexts of local areas are not aligned with county boundaries, producing an apparent spillover effect across adjacent counties (Voss et al., 2006).

Limitations

We rely heavily on death records, which offer a wealth of valuable data, but do not characterize whether differences in cardiovascular disease incidence or treatment resulted in heart-disease specific mortality. It is possible that the observed declines in heart disease mortality represent a mix of primary prevention of cardiovascular disease—perhaps through alterations in life course smoking prevalence—and secondary prevention of heart disease mortality through behavioral and medical control of risk factors such as hypertension and diabetes. This distinction is important for strengthening future prevention efforts. While our outcome definition of all disease of the heart aimed to optimize validity of cause-of-death coding through time, there is little known about geographic variation in cause-of-death coding. If quality of death certificate coding were related to slavery, or other related characteristics, our estimates could be biased.

Our reliance on only selected covariates partially reflects limited county-level measures of socioeconomic processes over the 150-year study period. There may be important covariates missing from our analysis. However, the impact of missing variables must be considered in the context of the conceptual distinction between variables. Failure to adjust for factors, which could plausibly confound the observed association (e.g. variables which precede both the distribution of slavery and heart disease decline), will result in biased estimates of the association of interest. However, missing variables that are intermediates between slavery and the rate of heart disease decline constitute missed opportunities to fully investigate pathways through which historical slavery influences contemporary health, but they do not threaten the validity of the estimate of the core parameter of interest.

Finally, in order to analyze historical variables for a common set of analytic units (counties), we used spatial areal interpolation techniques to normalize all data to 2010 county boundaries. Most county boundaries were largely unchanged, and visual inspection of all interpolated data increased our confidence that the underlying spatial structure of each covariate was reliably translated to 2010 boundaries (Figure S1). Heart disease mortality rate estimates are unstable in sparsely populated counties; this risks generating biased estimates of rate of decline, and possibly induces spurious spatial autocorrelation in rural regions. However aspatial empirical Bayes small area estimates produced robust, small-count rates, and the use of formal spatial regression models accounted for autocorrelation, with findings for Blacks unchanged, and those for Whites attenuated to null.

Conclusions

The dramatic decline in heart disease mortality is a public health success story, but now public health researchers and practitioners have a new challenge: to further examine and tackle the emergence of racial and geographic disparities in heart disease mortality. This research informs a strategy for new investigation, and future public health practice. In this ecological study, the legacy of slavery across counties in the South is associated with the magnitude of decline in heart disease mortality between 1968 and 2014 for the Black, but not the White, population. This association is partially explained by adjustment for hypothesized intervening variables, including racial gaps in education and economic attainment and historical, county-level variations in lynchings. These results are consistent with the hypothesis that intergroup and inter-place differences in mortality reductions result from uneven diffusion of effective prevention approaches, and/or uneven distribution of deleterious exposures within places and between races. Social, economic, and political structures, that have deep roots in the institution and legacy of slavery may contribute to racial differences in local opportunity structures that, in turn, influence heart health. While it is critical that contemporary prevention efforts target individual risk factors, public health experts must also consider, and engage with, local structural conditions that continue to impede such efforts.

Ethical statement

Author agreement/declaration

All authors contributed to the study reported in this manuscript, and have approved the submitted version.

Conflict of interest

No conflicts of interest from any authors.

Permission note

Does not apply.

Appendix A. Supporting information

Supplementary data associated with this article can be found in the online version at http://dx.doi.org/10.1016/j.ssmph.2017.07.004.

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