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Temporal Changes in Education Gradients of 'Preventable' Mortality: A Test of Fundamental Cause Theory

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Abstract

Fundamental cause theory explains persisting associations between socioeconomic status and mortality in terms of personal resources such as knowledge, money, power, prestige, and social connections, as well as disparate social contexts related to these resources. We review evidence concerning fundamental cause theory and test three central claims using the National Health Interview Survey Linked Mortality Files 1986-2004. We then examine cohort-based variation in the associations between a fundamental social cause of disease, educational attainment, and mortality rates from heart disease, other "preventable" causes of death, and less preventable causes of death. We further explore race/ethnic and gender variation in these associations. Overall, findings are consistent with nearly all features of fundamental cause theory. Results show, first, larger education gradients in mortality risk for causes of death that are under greater human control than for less preventable causes of death, and, second, that these gradients grew more rapidly across successive cohorts than gradients for less preventable causes. Results also show that relative sizes and cohort-based changes in the education gradients vary substantially by race/ethnicity and gender.

Keywords

fundamental cause theory; mortality; United States; race/ethnicity; gender; trends

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INTRODUCTION

Fundamental cause theory (FCT) (1) is frequently used by researchers and policy-makers to inform analyses of U.S. health and mortality disparities. The theory explains persisting associations between socioeconomic status (SES) and mortality risk in terms of personal and flexible resources such as money, knowledge, power, prestige, and social connections. In addition, the theory also stresses the importance of broader social and environmental contexts related to SES (2, 3). First proposed in the mid-1990s by Link and Phelan (1995), FCT has become a leading medical sociological theory of health disparities and has been cited thousands of times in sociological, public health, and population science journals. Yet despite the theory's popularity only a handful of analyses have directly tested its central claims.

In this paper we review the foundations of FCT and evaluate extant evidence concerning its validity with respect to education differences in US adult mortality risks. We also discuss the conditions under which we ought to expect educational attainment to become more strongly associated with survival, with particular attention to race/ethnic and gendered contexts of the education-mortality association, as well as cohort-based changes in these contexts. We then use data from the National Health Interview Survey Linked Mortality Files for years 1986-2004 to test the theory's central claims by analyzing cohort-based trends in education gradients of U.S. adult mortality risk from “preventable” and less preventable causes of death.

According to FCT, socioeconomic gradients ought to be larger for causes of death under greater human control (4). This is because personal resources such as education, income, and social connections can be used to attain health-related knowledge, access helpful and/or needed services, and/or purchase preventative and curative technologies. Furthermore, such resources embed individuals in social contexts (e.g., workplace, neighborhood, peer networks) that might also contribute to differential exposures to both health-related threats (e.g., hazardous working conditions, higher rates of smoking among friends/coworkers) and protections (e.g., safer neighborhoods, increased health-related knowledge among friends). Conversely, these resources and social contexts should garner only minimal protection against causes of death that are highly random and/or less preventable or treatable. Consequently, according to FCT education differences in mortality rates should be greatest for causes of death that are more preventable and/or curable than deaths from causes under less human control(3, 4).

We directly test this claim, but also extend our analyses to test two key factors we believe affect the association between educational attainment and U.S. adult mortality risk. First, for both theoretical and empirical reasons we argue that the size of the education gradient in U.S. adult mortality from heart disease and other “preventable” causes should be growing larger across cohorts. Consistent with this position, recent findings have shown some education gaps in U.S. adult mortality to have widened across the 1990s and 2000s (5-7). Also, evidence suggests that changes in U.S. adult mortality rates exhibit strong cohort-based variation (8, 9). Second, we further argue that education gradients in US adult mortality and the rate at which they are changing across birth cohorts likely vary

substantially by race/ethnicity and gender. On the one hand, cohort trends in educational attainment – and other SES dimensions related to educational attainment such as employment and income – differ significantly by race/ethnicity and gender in the United States (10-12). On the other hand, the ability to transfer socioeconomic resources into better health and longer life has been shown to be significantly conditioned by race/ethnicity and gender (6, 12-16). Indeed, research continues to document persisting race/ethnic and gender differences in educational attainment (17), health outcomes and longevity (6, 12, 16, 18-20), and the mechanisms linking education and health and longevity (14, 18, 20, 21). Further, research has found evidence suggesting temporal changes in the education-mortality association differ by men and women and non-Hispanic blacks and non-Hispanic whites (8, 15, 22). For these reasons we believe FCT ought to be extended to explicitly incorporate how other fundamental social factors – such as gender and race – condition the effect of personal resources on health and mortality risk, and how these conditional effects might be changing across time and/or birth cohorts in the US population.

BACKGROUND

Existing Tests of Fundamental Cause Theory

Fundamental cause theory makes specific claims regarding the persistence of socioeconomic disparities in health, which, surprisingly, are frequently left untested by researchers when citing the theory (3, 23). Specifically, FCT involves four central features, all of which are amenable to empirical testing. First, the theory asserts that socioeconomic gradients exist across multiple health outcomes. Supporting this point, education differences have been found in self-rated health (10, 16, 24, 25), disability (26), use of health technologies and services (27, 28), and all-cause and cause-specific mortality risk (5, 6, 15, 29-31). Second, fundamental causes of these health differences are hypothesized to operate through multiple risk-factor mechanisms, including knowledge of health-related behaviors (e.g., diet, exercise, and use of tobacco), social support and psychosocial factors (32-34), and access to preventative and curative services and technologies (3, 27, 35). Third, fundamental causes of health are reliably reproduced through new intervening mechanisms (2, 22, 36). And lastly, the “essential” feature of fundamental social causes of health inequalities is that they involve access to flexible resources that can be used in different places and at different times to garner a health advantage. Consequently, fundamental causes affect health even when the profile of risk factors and diseases change radically (23).

These features of fundamental cause theory implicate dynamic social processes that are continuously shaping the relationship between socioeconomic status and multiple health outcomes, which should be observable and testable with respect to numerous conditions (2, 36). Indeed, a number of analyses have investigated specific components of FCT, primarily focusing on instances in which resources are used individually or collectively to garner and/or protect health advantages. For example, Chang and Lauderdale (2009) showed that the income gradient in US adult cholesterol levels was reversed in the post-statin era, a change the authors attribute to the income-dependence of the adoption of a new technology (i.e., statins used to control cholesterol levels) (27). Link (2008) showed the emergence of an education gradient in understanding the smoking-lung cancer association among the US

public as well as educational differences in US smoking rates between the 1950s and 2000s (36, 37). Lutfey and Freese (2005) highlighted multiple mechanisms behind socioeconomic differences in continuity of care at two diabetes clinics. Studies such as these show the significance of social processes shaping the effective deployment, dissemination, and adoption of health-relevant information, technologies, and behaviors (38). Collectively, Link and Phelan (2010) highlight three "facts" that studies have established supporting the tenets of FCT with respect to mortality risk (3). Admittedly, the first two sets of facts are not direct tests of the theory itself, but they do establish useful findings with which to gauge claims consistent or inconsistent with FCT. First, studies have repeatedly found that mortality rates from preventable causes of death have declined across time more rapidly than rates from less preventable causes (8, 9, 39-41). Second, and most commonly reported, evidence from multiple studies has demonstrated a strong and persistent SES gradient in mortality rates from preventable causes of death. Specifically, studies show an inverse association between socioeconomic resources and mortality risk from preventable causes of death (4, 8, 16, 21, 42-45). Third, evidence has also shown the association between SES and mortality risk to be stronger for more preventable causes of death than less preventable causes (4, 8, 16, 21, 42, 44).

In the present study, we explore the existence of a fourth "fact" concerning FCT by testing whether the education gradient in US adult mortality risk grew more rapidly for more preventable causes of death than did the gradient in mortality risk from less preventable causes during a time of significant reductions in US adult mortality. We first replicate findings to confirm all three sets of facts (4), but then move beyond them to incorporate a temporal dimension of the US education gradient in preventable mortality. If preventable mortality decreases more rapidly than mortality from less preventable causes of death, and the education gradient in mortality risk is more pronounced in more preventable causes of death, then we argue that the education gradient should be growing more rapidly across time for preventable mortality than for less preventable mortality. Based on recent evidence implicating cohort-based variation in both chronic disease incidence(9, 46, 47) and educational attainment in the United States (17, 48), we examine how educational differences in US adult mortality by preventable causes of death are changing across birth cohorts.

Race/Ethnic and Gendered Contexts

We also examine education gradients in US adult mortality risk by race/ethnicity and gender to explore variation in the gradients, as well as variation in temporal changes in the gradients. The effectiveness of education as a personal resource to positively affect longevity in America is highly conditioned by race/ethnicity and gender, a fact that FCT ought to directly engage and incorporate into its theoretical frame (8, 12, 14, 16, 18, 19, 31, 49). Thus, we push FCT to explicitly acknowledge how strongly race/ethnicity and gender affect the education-mortality association in the US population. In this regard, however, it is worth noting that Link and Phelan (2010) have outlined multiple instances in which fundamental cause theory will predict a small, absent, or inverse association between resources and a health outcome (3). It is important to keep in mind exceptions to FCT, as one must be mindful of the contexts in which resources are employed are highly variable (2,

14, 38, 51). It is not always the case that education, income, or other socioeconomic resources will garner a health advantage, and in those cases that they do we might hypothesize populations to derive unequal health benefits from such resources. Long term, cumulative stratification processes associated with gender and race/ethnicity can substantively affect the ability of personal resources to shape health in American society (12-14). In terms of basic gender differences in the education-mortality association, research has found evidence indicating a stronger education gradient in mortality among US men than among US women (6, 15, 16, 21, 30, 31). Likewise, research has repeatedly found strong evidence indicating a steeper education gradient for US non-Hispanic whites than for US non-Hispanic blacks (8, 15, 20, 21). Further, evidence indicates that temporal trends have led to increasingly larger education gradients in mortality among US white men and women than among US black men and women (6, 8, 15, 22).

There are at least three generic ways by which the education-mortality relationship likely varies for US men and women and for US race/ethnic populations. If we assume that people desire to transfer their personal resources, such as education, into improved health outcomes, we surmise that this transferability might be blocked at three levels. First, “accumulation of resources” may be blocked whereby, for example, educational attainment among some populations is less likely to confer subsequent employment, income, prestige, power, and/or beneficial connections than among other populations. Second, “effective deployment of resources” can be blocked, whereby quality of schooling and/or institutional and interpersonal discrimination makes the health-related application of education more difficult to achieve for women and/or race/ethnic minorities (12, 21, 52). And third, “contextual contingency” can affect if and how one draws on one's education to improve and/or maintain health. For example, the propensity for highly educated US black women to experience multiple caregiving roles among extended kin networks has been argued to be a potential factor in limiting potential health returns of higher education and personal resources among this population (53, 54). Also useful here is Rieker and Bird's (2008) notion of “constrained choice” in understanding gender differences in health behaviors, whereby competing demands on resources, time, and relationships can differentially affect men's and women's cumulative biological risks of health outcomes (55), and the potential for education to affect these risks.

In short, gender- and race/ethnic-based factors in the US population continuously reshape the context in which education is attained, reshape the life course processes affecting health and longevity, and reshape various mechanisms linking education and mortality risk across the life course. While FCT maintains that education-mortality association will persist despite changing mechanisms, it should also consider how various contexts and other fundamental social causes of health and longevity – race/ethnicity and gender – in America shape the education-mortality association.

In this paper, we test central claims of fundamental cause theory and also test new hypotheses consistent with FCT by investigating the size and stability of education gradients in US adult mortality risks from preventable and less preventable causes of death across cohorts of non-Hispanic black and white men and women. In doing, we replicate analyses showing evidence for the existence of three sets of “facts” regarding the claims made by

fundament cause theory (4). That is, we test (1) whether mortality rates from preventable causes of death declined more rapidly between 1986 and 2006 than rates from less preventable causes of death, (2) whether or not education gradients exist in mortality risk from preventable causes of death, and (3) whether the education gradients in mortality are larger for preventable causes of death as compared to less preventable causes of death. Beyond these points our data permit us to additionally test (4) if the education gradients in US adult mortality risk from preventable causes of death have *changed* more rapidly than the education gradients in less preventable deaths across a recent period of time, and (5) if the gradients and cohort-based changes in these gradients significantly vary by race/ethnicity and gender.

ANALYTICAL STRATEGY

Data

We use 19 continual waves of the National Health Interview Survey (NHIS) 1986-2004 linked to official death records up to December 31st, 2006 at the National Death Index (NDI). The resulting National Health Interview Survey-Linked Mortality Files (NHIS-LMF) provide annual individual-level cause-specific mortality status for eligible respondents between survey quarter-year and December 31st, 2006. Stata 12's *svy* command was used to account for the NHIS's clustered sampling design, and analyses were weighted to make results representative of the noninstitutionalized U.S. adult population between years 1986 and 2006.

Samples

Analyses were stratified by race/ethnicity and gender in order to explore variations in the way education is associated with mortality risk across age, period, and cohort. NHIS respondents self-identified as non-Hispanic black and white male and female subsamples were created. Due to small samples – especially at older ages and older cohorts – respondents not identifying as non-Hispanic black or non-Hispanic white were excluded from analyses. The resulting samples were composed of 306,306 non-Hispanic white males; 335,584 non-Hispanic white females; 42,430 non-Hispanic black males; and 60,853 non-Hispanic black females. Table 1 presents descriptive statistics of person-level samples.

Outcomes

Respondents' individual-level survival was observed across all ages between time of survey and during the period of mortality follow-up to December 31, 2006. An event of mortality was classified as non-heart disease "preventable" if the underlying cause of death listed on the death certificate had a value higher than or equal to 3.0 on Phelan et al.'s (2004) scale of preventability in their Appendix A, but the underlying cause was not due to heart disease. The Phelan et al. (2004) rating scale ranged from 1, virtually impossible to prevent death from this disease/cause, to 5, virtually all deaths preventable. Those deaths attributable to underlying causes of death with values less than midpoint of 3.0 were designated as "low in preventability". Deaths attributable to heart disease were classified separately from other "preventable" causes and were classified as deaths coded 53 through 68 in the National Center for Health Statistic's 113 cause of death variable. We classified heart disease deaths

separately from other “preventable” causes for two primary reasons. First, heart disease has been and remains the leading cause of death in the US adult population, as well as one of the most modifiable causes of death in US society (39). Second, pertinent to testing fundamental cause theory, Capewell and Graham (2010) highlight cardiovascular disease prevention as a case in which we are likely to observe growing inequalities in the aftermath of “agentic” interventions (51). This is because the most commonly employed cardiovascular disease preventions are usually “mediated through individual-level change in knowledge, motivation, and behavior,” and thus are highly susceptible to personal resources such as education (Capwell and Graham 2010: 1).

Covariates

The primary variable of interest is respondents’ individual-level educational attainment. Time-invariant levels of educational attainment were self-reported by respondents at time of survey. One's highest degree obtained and years of completed schooling were both used to create four categories of educational attainment: less than high school, high school degree or GED, some college, and bachelor degree or higher (reference).

Respondents’ ages at time of survey (centered on age 50 years) were included as a linear term to control for age-related survey selection bias, and aging effects were absorbed into the time-metric of the survival models. Time-varying period was controlled for with three-year period dummy variables (1986-1988 through 2004-2006), and mortality variation across birth cohort was controlled for by including five-year cohort dummy variables (1901-1905 to 1961-1965) in exploratory analyses, and as linear terms in models testing cohort-based variation in the education-mortality association. Respondents’ residential region of country at time of survey (Northeast, Midwest, South, West) was also included to account for residual geographical variation in mortality risk (87).

Methods

Educational differences in individual-level survival were assessed using Cox regression and Royston-Parmar survival models. All models were stratified by respondents’ sex (male and female) and race/ethnicity (non-Hispanic black and non-Hispanic white) and the complex sampling design of the NHIS was accounted for using Stata 12's svy prefix command. To test the first claim of FCT we examined temporal trends in US adult mortality by preventability of cause of death (heart disease, non-heart disease “preventable” causes, less preventable causes) by fitting Cox survival models that tested period-based variation in mortality hazards. These models included three-year time-varying period effects, which we found could be accurately approximated with a linear term. For ease of comparison across preventability of death and race/ethnic-sex subsample, we present only results from the models fitted with the linear terms. We also fitted models including five-year fixed cohort effects, which we also found could be approximated with linear terms.

The second and third claims of fundamental cause theory – that education gradients in mortality exist for “preventable” causes of death, and these gradients are significantly larger than those observed for less preventable causes of death – were also tested using Cox regression survival models using age as the underlying time metric. All models were

stratified by race/ethnicity and sex, and included age at time of survey, region, and five-year fixed effects of birth cohort (beyond age and cohort effects, period-based changes in the associations were not found to be significant in these models). Relative differences in hazards by educational attainment were assessed by including less than high school, high school, and some college (college education or higher was the reference).

Finally, to test the new fourth claim of fundamental cause theory – that cohort-based changes in the education gradients of mortality from "preventable" causes of death are larger than cohort-based changes in the education gradients of mortality from less preventable causes of death – we fitted Royston-Parmar survival models with age as the underlying time metric. Using restricted cubic spline function of ln(*t*) with knots k_0, written as $s\{\ln(t)|\gamma, k_0\}$, Royston-Parmar models were estimated on the baseline log cumulative hazard scale. We incorporated age-varying effects of a less than high school education using interactions of the form $\sum_{j=1}^{1} s\{ln(t|\delta_j)\} z_{ij}$, resulting in the following model for the log cumulative hazard:

$$ln\{H_i(t|z_i, x_i)\} = s\{ln(t)|\gamma, k_0\} + \sum_{j=1}^{1} s\{ln(t)|\delta_k, k_j\} z_{ij} + x_i\beta, \quad (1)$$

for *j*=1 education level (less than high school) interacting with the spline terms using k_j number of knots for the *j*th covariate (see Lambert and Royston 2009 for a thorough discussion (88)). Models were stratified by sex and race/ethnicity and estimated using Stata 12's *stpm2* program, using sample weights that make results representative of the U.S. noninstitutionalized population. To conserve space, we present results from models that compare only the less than high school sample to the sample with a college degree or more. We restrict our analyses to these samples to keep the tests and discussion of the tests as simple as possible (results from additional models available upon request). The final models included a linear cohort term and a two-way interaction between the cohort term and less than high school education.

RESULTS

Test 1

Our first test is whether mortality rates from preventable causes of death declined more rapidly between 1986 and 2006 than rates from less preventable causes of death. The logic is simple – as death from a disease becomes preventable, age adjusted death rates from that disease are expected to decline. If death from a disease is less preventable, there is less expectation that purposive human action will produce declining death rates. Both the cohort and period results in Table 2 are entirely consistent with this reasoning

Table 2 presents period- and cohort-based trends in U.S. adult mortality risk by race/ethnicity, gender, and preventability of death. Three patterns are especially worth noting. First, for all groups we find no evidence suggesting period- or cohort-based declines in US adult mortality rates from less preventable causes of death. In fact, for white women we find evidence suggesting cohort-based increases in mortality risk from less preventable causes of

death (b=.014, SE=.002). Second, we find evidence suggesting period-based declines in heart disease mortality rates for all groups except black women. We also find evidence of strong cohort-based declines in heart disease mortality for all groups. In terms of non-heart disease preventable mortality, we find significant cohort-based reductions for both black and white men (but not for women) and significant period-based reductions for white men and women (but not for black men and women). Thus, overall, we find evidence consistent with FCT suggesting greater reductions in mortality rates from more preventable causes of death than from causes of death that are less preventable. However, and to the second pattern found in these results, we find strong gender differences in these mortality trends. While cohort-based reductions are observed in men's mortality risk from both heart disease and other “preventable” causes of death, irrespective of race, we find no significant cohort-based reductions in women's mortality from non-heart disease preventable causes of death (black women b=.004, SE=.002; white women b=.002, SE=.001).These trends might reflect gender differences in cohort-based smoking patterns in the US population (76-78) and the corresponding cohort-based trends in lung cancer mortality risk (39, 89, 90), as well as differential cohort-based prevalence of obesity, which is significantly greater among US women, especially non-Hispanic black women, than among US men (79, 91). Third, race/ethnic differences are apparent in period-based variation in US adult mortality risk from preventable causes of death. Except among US black men's mortality risk from heart disease (b=-.009 SE=.005), we find no evidence of period-based reductions among US black mortality from preventable causes of death between 1986 and 2006. Conversely, significant period-based reductions were found in both US white men's and women's mortality from heart disease and non-heart disease preventable causes of death. Taken together, we observe a general pattern supporting fundamental cause theory's claim that reductions in US adult mortality – both period-based and cohort-based – were larger for more preventable causes of death than for less preventable causes of death, but race/ethnic and gender variation in this overall pattern is substantial.

Test 2 and Test 3

Table 3 presents estimates of education gradients in U.S. adult mortality risk, 1986-2006, by race/ethnicity, gender, and preventability of death. These results show estimated hazard ratios (HR) for the less than high school, high school, and some college populations relative to the college-educated population (reference).

Consistent with fundamental cause theory, the results show that recent education gradients in U.S. adult mortality risk are generally larger for causes of death that are under greater human control (both heart disease and non-heart disease “preventable” causes) than for less preventable causes of death. Thus, on balance, the results support the second and third claims of fundamental cause theory. However, there are substantive race/ethnic and gender differences in the education gradients of US adult mortality by preventability of cause. For example, we find no significant differences in the education gradients in non-Hispanic black women's mortality by preventability of cause. The estimated HR for less than high school education among black women are 1.81 for heart disease mortality, 1.97 for non-heart disease preventable mortality, and 1.61 for less preventable mortality, which are not significantly different from one another. Similarly, results suggest that the education

gradient in non-Hispanic black men's mortality from heart disease (e.g., <HS HR=1.68, SE=.084) does not differ from the education gradient in mortality from less preventable causes (e.g., <HS HR=1.64, SE=.084). Conversely, for non-Hispanic white men and women we see significantly larger education gradients in mortality risks for preventable causes of death than for less preventable causes of death. For example, among non-Hispanic white men the HRs associated with a less than high school education are 1.94 for heart disease mortality and 2.20 for non-heart disease preventable mortality, which are significantly larger than the 1.61 HR for less preventable causes of death. Thus, our findings indicate that the degree to which education differences in US adult mortality risk vary by the preventability of cause depends significantly on race/ethnicity and its interaction with gender.

Test 4

Table 4 presents estimated coefficients of cohort-based changes in US adult mortality risk from preventable and less preventable causes of death by race/ethnicity and sex. The cohort term indicates the estimated cohort-based variation in mortality for the college educated population, and the two-way interaction between <HS and cohort is the estimated adjustment to the cohort-based variation in mortality for the less than high school population.

In all population groups analyzed, the education gradient in US adult mortality from preventable causes of death grew across birth cohorts during the time period 1986 to 2006. Furthermore, in all population groups we also see that education gradients in heart disease mortality grew across cohorts as well. For example, among non-Hispanic white women we observe that the log(Mx) from heart disease decreased by .032 (b=-.032 SE=.006) across cohorts among those with a college degree, but decreased by only .013 (-.032+.019, SE=.006) among those with less than a high school education. The patterns behind the growing education gradients, however, differ considerably by race/ethnicity, gender, and cause of death. For example, in trends of heart disease mortality we find cohort-based reductions in both the college educated and less than high school educated populations, with significantly greater rates of cohort-based reductions among the college educated populations than among the population with less than high school education, irrespective of race/ethnicity or gender. In short, we see reductions across cohorts for both education groups, but the more highly educated experienced greater reductions. For non-heart disease “preventable” mortality, however, we observe a different trend. Here, we see cohort-based reductions in mortality for the college educated population in all groups, but stalling or even increasing rates of “preventable” mortality for the less than high school populations. That is, the two-way interaction effects between <HS*cohort are such that the reductions are not being experienced (as in the case in the men's populations) or are significantly greater than the main cohort effect such that the less than high school populations are experiencing cohort-based increases in non-heart disease “preventable” mortality (as is the case in the women's populations). This gender difference in cohort trends of “preventable” mortality is significant only in the non-Hispanic white population, and likely reflects gender differences in cohort-based smoking patterns, drug-related accidents, obesity patterns, as well as differential survival into older ages (7, 93).

We find some evidence suggesting educational differences in cohort-based trends of US adult mortality from less preventable causes of death in the non-Hispanic white population. Finding among non-Hispanic black men and women are not significant. Specifically, we find evidence suggesting significant cohort-based increases in mortality hazards from less preventable causes of death among the less than high school white population, and no significant cohort trends in the non-Hispanic black population, irrespective of educational attainment. These findings are consistent with recent evidence suggesting substantive declines in US longevity among the relatively less educated white population (6-8, 18).

Overall, these findings provide evidence supportive of FCT, in that cohort-based reductions in mortality are larger for more preventable causes of death, and that the greatest reductions in these deaths have occurred among the more highly educated populations. Indeed, we see that the greatest cohort-based reductions in mortality for the college educated populations were in heart disease-related mortality, followed by non-heart disease “preventable” mortality, with no significant cohort-based reductions in less preventable mortality except among college educated white men. Among those with a less than high school education, irrespective of race/ethnicity or gender, we observe smaller cohort-based reductions in heart disease mortality and mortality from other preventable causes of death.

DISCUSSION

Fundamental cause theory has been a leading perspective used by sociologists, epidemiologists, and demographers to explain socioeconomic inequalities in health. While the theory is cited often when discussing persisting associations between social factors and health outcomes, the central claims of fundamental cause theory are often left untested. Yet the fundamental causality of social factors in shaping health outcomes can and should be directly engaged empirically with specific tests. In the current analyses we sought to test the claims of fundamental cause theory as they apply to US mortality differentials by educational attainment, as well as expand the theory's application to more seriously consider variations in the link between personal resources and survival. Specifically, we argued that FCT ought to be expanded to consider more seriously gender- and race/ethnic-based variation in the association between a fundamental cause of health, education, and mortality risk. We also argued that FCT ought to more explicitly make claims regarding temporal trends in this association, moving beyond the expectation that such associations simply persist despite changing mechanisms.

By using the distinction between causes of death that are more or less preventable and by embedding these concepts in a data structure that allows the examination of cohort processes across race and gender groupings we were able to produce multiple tests of predictions from fundamental cause theory. Results from our analyses are largely supportive of FCT. Overall, evidence indicates that education gradients in US adult mortality risk among non-Hispanic black and white men and women are largest for heart disease and other “preventable” causes of death. Further, education gradients in US adult mortality grew larger across cohorts, and more so for preventable causes of death than for less preventable causes of death. Findings further suggest that the theoretical framework of FCT ought to be broadened to more seriously consider race/ethnicity and gender variations in the associations between

socioeconomic resources and mortality risk, and temporal changes therein (2). Indeed, on the one hand, the link between socioeconomic resources and mortality risk in the United States is significantly conditioned by both race/ethnicity and gender. Thus, while personal resources remain the essential component of FCT, the theory must be more mindful that the ability to transfer resources into good health and long life is highly contextualized in American society by one's race/ethnicity and gender (12-14, 58). On the other hand, the links between educational attainment and mortality risk from heart disease and other "preventable" causes of death has changed considerably across birth cohorts, and these changes have occurred differently in the US non-Hispanic black and non-Hispanic white populations (23). In general, evidence here suggests that the education gradients in U.S. adult mortality risk have grown wider across cohorts, and more so for "preventable" causes of death than less preventable causes of death. However, some evidence indicates persistent or even narrowing education gradients for some population groups. While fundamental cause theory is most often used to explain the *persistence* of socioeconomic gradients in health and mortality, researchers ought to directly integrate a temporal dimension into the theory in order to account for changes in health disparities (8, 22, 48).

To consider possible explanations of race/ethnic and gender differences in the US education-mortality association, as well as cohort-based trends in these associations, we briefly entertain how the following mechanisms might affect the three levels by which education can be effectively deployed for good health and longevity: (1) biological differences in disease susceptibility and longevity, (2) structural and interpersonal discrimination, (3) differences in early-life conditions and subsequent life course processes of adult disease and mortality, (4) differences in health risk behaviors, and (5) differential exposure and effects of cumulative stress processes. In terms of biological differences in disease susceptibility and longevity, no strong evidence implicates variation in gene frequencies as causal factors of race-based differences in US adult health and longevity (12, 56, 57). On the contrary, arbitrary racial classifications stem from "systems of stratification, power, and ideology" (58), and the persisting gaps in health and longevity between US race/ethnic groups overwhelmingly reflect a host of socioeconomic, political, and behavioral factors (20, 49). Thus, while some race-based disparities in US health may reflect differences in gene expression, the evidence increasingly shows such expressions are likely triggered by differential exposure to adverse social and environmental factors (57, 59, 60). Conversely, some research has suggested biological factors are implicated in some of the health and longevity differences between men and women (19). For instance, Yang and Kozloski (2011) provide evidence indicating strong biological bases for sex differences in age patterns of inflammation and allostatic load (61), and Crimmins et al. (2012) demonstrate striking consistency in gender differences in health, disability, and longevity in US and European populations (62). Thus, gender differences in the association between education and US adult mortality risk might partially be affected by men's and women's different age-specific susceptibility to mortality risk across the life course, and gender differences in education levels across these aging cohorts.

When considering the role of discrimination in blocking the acquirement and deployment of health-related resources, it is important to note that income has been shown to be an

increasingly important mediator of the education-health association in the US population (24). In a context of increasing income inequality (11), widening health disparities across socioeconomic class (6-8, 15, 22), disparate employment opportunities and wage earnings for non-Hispanic black and white men and women (63-66), and rising race/ethnic inequalities in incarceration (67-69), the strength of the US education-mortality association is likely substantively affected by impediments to transfer higher educational attainment into additional personal resources. As such, gender and race/ethnic variation in education gradients of US adult mortality risk, especially for more preventable causes of death, likely stems in part from discrimination and disparate abilities to effectively deploy educational attainment for health (58).

Beyond its negative effects on resource deployment, discrimination has also been shown to be strongly associated with adverse mental health outcomes, and has been directly implicated in various stress processes that negatively affect physiological health as well (52, 53, 70). Discrimination-related stress has been argued to be a salient outcome of residential, educational, and occupational segregation experienced by African Americans (71-74). An emerging body of evidence has indicated higher allostatic load among the US black population as a product of such stressors (75), and that black women in particular might suffer accelerated biological aging as a result of prolonged exposure to stressful living conditions (53).

Large gender and race/ethnic differences are observed in both the prevalence and incidence of leading behavioral risk factors associated with early death in the United States (e.g., smoking and obesity rates). Smoking prevalence is much higher among men than among women, and cohort-based differences between men and men and between US blacks and whites in both incidence and quitting rates are striking (76-78). Specifically, Ho and Elo (2013) report negligible race-based differences in the likelihood of ever having smoked cigarettes among the US adult population, but very strong race-based differences in quitting rates and strong cohort-based differences between black men and white men in current rates of smoking (76). The differences in smoking rates account for very little of the US black-white mortality gap among women, but a sizeable proportion of the US gap among men. Such differences in smoking patterns and trends likely affect trends in US mortality rates by gender and race/ethnicity, and likely confound the education-mortality association across cohorts in different ways for US black and white men and women. Obesity prevalence, an additional risk factor for preventable diseases and mortality risk, also exhibits strong cohort, gender, and race/ethnic variation in the US population. More recent US cohorts, irrespective of gender or race/ethnicity, are estimated to have higher rates of obesity (79), although strong gender and race/ethnic differences in obesity prevalence exist in the US population. Overall, women are more likely to be obese than are men, especially at high levels of body mass index (BMI ≥ 35.0) and at older ages (80). Further, large race/ethnic differences in obesity prevalence exist among US women, but much less so among US men, and socioeconomic gradients in obesity prevalence differ significantly across race/ethnic groups (81).

Finally, evidence suggests substantive variation in life course processes of US adult disease, disability, and mortality risk between men and women and between race/ethnic populations

(82). Yet as Colen (2011: 90) stresses, there is a “propensity of extant frameworks to emphasize linear, cumulative pathways while ignoring critical interactions—both over time and across stratification processes—[which] severely limits their ability to reveal the social processes necessary to maintain health disparities” (13) We believe tests of fundamental cause theory must be attentive to these long-term stratification processes that shape the ways by which black and white men and women differentially can and do draw on education to affect adult health outcomes. For example, evidence indicates that adverse conditions in early life may have stronger detrimental effects on women's later-life health and mortality risk than on men's (82, 83). Further, research has implicated strong intergenerational effects on poverty, educational opportunities, and mobility among some segments of the US black population (84, 85), suggesting entirely different life course processes of status attainment and adult health for some populations than for others. Also, some work has argued that the association between early-life adverse conditions and later-life mortality risk is likely changing across US cohorts, but the changes have unfolded differentially for men and women and for US blacks and whites (8, 86).

CONCLUSION

What we found is both strongly supportive of the theory and simultaneously powerfully challenging to it. The storyline behind the support for the theory begins with the observation that the prediction of sharper gradients for causes of death that are more rather than less preventable is one that FCT makes that other explanations for disparities do not. Stress theory has no basis for predicting that SES disparities should be stronger for more rather than less preventable causes of death nor any basis for predicting that these disparities would emerge and change over time. Similarly a selection theory that posits illness associated disability as the reason for SES gradients (reverse causation) has no basis for predicting that disability would be a stronger determinant of SES for diseases for which death can be prevented as opposed to diseases for which death is much less preventable. Because the prediction is novel, evidence supportive of the prediction is particularly important for the theory, forming something close to a lynchpin test of the theory. In light of this consideration our results were quite supportive of the theory. In the vast majority of tests across the multiple groups and with and without applying the cohort lens, the prediction of stronger education effects for more rather than less preventable causes of death were found.

Although supportive of the theory our results also proved challenging to it. The data structure we exploited allowed an examination of the generality of the education effects in distinct gender and racial groupings. Relying on extant literature we formed an expectation that education differences in mortality rates from more and less preventable causes of death might vary across these very powerful social fault lines and we found that they in fact did so. The reason this challenges FCT is that the theory as currently proffered has no explanation for such variation. Why should the versatile, flexible resources it champions work more effectively in some groups and in some contexts than in others? Two responses from FCT are possible. As we have indicated, one is to elaborate the theory with the specific goal of identifying the circumstances that ameliorate or enhance the effective deployment of SES-related resources of knowledge, money, power, prestige and beneficial social connections. Elaborating and then testing predictions based on the elaborations might extend

the theory making it more general. A second response is to step back from strong claims about the theory's generality. In such a response one might claim that while the theory points to processes that exist and exert substantial impact, the forces its points to inevitably collide with other factors and these other factors have their own powerful impacts that dilute the effect of the processes specified in FCT. In keeping with this latter notion Link and Phelan (3) have suggested that FCT is appropriately designated a theory of the “middle range” (92) that must join other such middle range theories if more complete explanations are to be derived. The task associated with this response is to specify what the other middle range theories are and point to the ways they intersect with FCT. In light of the results we generated we expect some combination of these two responses is appropriate and that both FCT and our understanding of health disparities will be enhanced if each is taken up in the time ahead.

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RESEARCH HIGHLIGHTS

- We review evidence concerning fundamental cause theory and test its three central claims.
- We extend fundamental cause theory to consider race/ethnicity and gender.
- We extend fundamental cause theory to consider birth cohort.
- We examine education gradients in US adult mortality by “preventability” of death.

Table 1

Descriptive Statistics of non-Hispanic black and non-Hispanic white Male and Female NHIS-LMF Samples 1986-2004

	Women				Men			
	Black		White		Black		White	
	Mean	S.D.	Mean	S.D.	Mean	S.D.	Mean	S.D.
Age at Survey	47.1	15.4	49.5	16.2	46.8	14.9	48.2	15.3
Age at Exit	58.9	14.9	61.5	15.5	58.1	14.2	59.9	14.4
Survey Year	1994.1	5.3	1994.0	5.3	1994.2	5.3	1994.0	5.3
Birth Year	1946.6	16.0	1944.0	16.6	1946.9	15.6	1945.4	15.8
Died before 2007	*15.3	36.0	14.8	35.5	20.3	40.2	17.0	37.6
Preventable Causes	39.0	48.8	33.5	47.2	37.9	48.5	32.1	46.7
Heart Disease Mortality	31.1	46.3	29.5	45.6	30.6	46.1	32.0	46.6
Less Preventable Causes	29.9	45.8	37.0	48.3	31.6	46.5	35.9	48.0
Educational Attainment								
< H.S.	28.5	45.2	15.4	36.1	29.9	45.8	15.7	36.4
H.S. Graduate	37.1	48.3	39.9	49.0	37.7	48.5	35.1	47.7
Some College	22.0	41.4	23.3	42.3	20.7	40.5	21.8	41.3
B.A.+	12.4	32.9	21.5	41.1	11.7	32.2	27.4	44.6
N	77,665		407,162		53,734		366,690	

*All numbers listed below line are percentages.

Table 2

U.S. Men's and Women's Period and Cohort Variation in Mortality Risk by Preventability

	Black Men		White Men		Black Women		White Women	
	b	**se**	**b**	**se**	**b**	**se**	**b**	**se**
Period Trends								
All-cause Mortality	−.002	.003	−.005	.001***	.004	.002	−.004	.001**
Heart Disease	−.009	.005*	−.012	.002***	.005	.005	−.012	.002***
Preventable, No HD	−.002	.004	−.004	.002*	.001	.004	−.005	.002**
Less Preventable	.003	.005	.000	.002	.005	.004	.001	.002
Cohort Trends								
All-cause Mortality	−.013	.003***	−.015	.001***	−.006	.002**	−.003	.001**
Heart Disease	−.023	.004***	−.034	.002***	−.024	.004***	−.028	.002***
Preventable, No HD	−.010	.004**	−.009	.002**	.002	.004	.001	.002
Less Preventable	−.007	.004	−.003	.002	.003	.005	.014	.002***

* $p < .05$

** $p < .01$

*** $p < .001$

Table 3

U.S. Men's and Women's Education Gradients in Mortality Risk by Cause of Death

	Black Men		White Men		Black Women		White Women	
	b	**se**	**b**	**se**	**b**	**se**	**b**	**se**
Heart Disease								
<HS	1.68	.084	1.94	.026***	1.81	.086	1.89	.033***
HS	1.32	.093	1.50	.025**	1.40	.091	1.40	.033*
SC	1.38	.099	1.32	.028	1.20	.100	1.27	.039*
Preventable, No Heart Disease								
<HS	2.11	.072*	2.20	.024***	1.97	.074	1.81	.031***
HS	1.64	.068*	1.66	.024***	1.54	.076	1.42	.028**
SC	1.43	.080	1.50	.026**	1.42	.082	1.30	.033**
Less Preventable								
<HS	1.64	.082	1.61	.022	1.80	.090	1.53	.026
HS	1.35	.082	1.38	.021	1.43	.092	1.28	.023
SC	1.21	.099	1.35	.023	1.26	.106	1.16	.029

One-tail test of difference

Test of Education Gradient in Heat Disease Mortality and Preventable Mortality vs. Education Gradient in Less Preventable Mortality.

*p < .05

**p < .01

***p < .001

Table 4

U.S. Men's and Women's Cohort Trends in Education Gradients in Mortality Risk by Preventability

	Black Men		White Men		Black Women		White Women	
	b	**se**	**b**	**se**	**b**	**se**	**b**	**se**
Heart Disease								
Cohort	−.031	.008***	−.043	.004***	−.033	.007***	−.032	.006***
<HS*Cohort	.013	.006*	.022	.005***	.028	.005***	.019	.006**
Preventable, No HD								
Cohort	−.017	.007*	−.011	.004**	−.007	.006	−.013	.005**
<HS*Cohort	.021	.005***	.017	.004***	.026	.004***	.034	.005***
Less Preventable								
Cohort	−.008	.013	−.007	.004*	.009	.014	.005	.005
<HS*Cohort	.004	.014	.013	.004**	.011	.014	.027	.005***

Sample composed only of respondents with educational attainment <H.S. or ≥B.A.

*p < .05

**p < .01

***p < .001