Does the union make us strong? Labor unions, health, and health inequities in the United States

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Abstract

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Recently, life expectancy in the U.S. has stagnated or declined for the poor and working classes and risen for the middle and upper classes. Declining labor union density – the percent of workers belonging to labor unions – has contributed to burgeoning income inequity. In this dissertation, we examined the relationship between unionism and health, and tested whether declining union density has exacerbated racial and educational mortality inequities.

In the first study, we analyzed the state-level relationships between union density, mortality, and mortality inequities from 1986-2016. State-level all-cause mortality and overdose/suicide mortality overall and by gender, gender-race, and gender-education came from CDC, while state-level union density came from the Current Population Survey. Using inverse-probability-of-treatment-weighted Poisson models with state and year fixed effects, we estimated

three-year-moving-average union-density's effects on the following year's mortality rates. Then, we tested for gender, gender-race, and gender-education effect-modification. Finally, we estimated how racial and educational all-cause mortality inequities would change if union density increased to baseline levels. Overall, a 10% increase in union density was associated with a 17% relative decrease in overdose/suicide mortality (95% CI: 0.70, 0.98), or 5.7 lives saved per 100,000 person-years (95% CI: -10.7, -0.7). Union density's absolute (lives-saved) effects on overdose/suicide mortality were stronger for men than women, but its relative effects were similar across genders. However, our estimates were sensitive to the analytic approach. Moreover, union density had little effect on all-cause mortality overall or across subgroups, and modeling suggested union-density increases would not affect mortality inequities.

In the second study, we analyzed the individual-level relationship between union membership, self-rated health (SRH), and moderate mental illness. The union membership and SRH analyses used data on 16,719 Panel Study of Income Dynamics (PSID) respondents followed between 1985 and 2017, while the union membership and mental-illness analyses included 5,813 PSID respondents followed between 2001 and 2017. Using the parametric g-formula, we contrasted cumulative incidence of the outcomes under two hypothetical scenarios, one in which we set all employed-person-years to union-member employed-person-years (union scenario), and one in which we set no employed-person-years to union-member employed-person-years (non-union scenario). We also examined whether the scenarios' effects varied by gender, gender-race, and gender-education in stratified models. Overall, the union scenario did not reduce incidence of poor/fair SRH (RR: 1.01, 95% CI: 0.95, 1.09; RD: 0.01, 95% CI: -0.03, 0.04) or moderate mental illness (RR: 1.02, 95% CI: 0.92, 1.12; RD: 0.01, 95% CI: -0.04, 0.06) relative to the non-union scenario. These effects largely did not vary by subgroup.

In the final study, we examined the individual-level relationship between union membership, mortality, and mortality inequities using data on 23,022 PSID respondents followed between 1979 and 2017. First, using the parametric g-formula, we contrasted cumulative incidence of mortality in the union and non-union scenarios. Next, we examined whether the scenarios' effects varied by race or education in stratified models. Finally, we estimated how racial and educational mortality inequities would change if union density had remained at 1979 levels throughout follow-up rather than that at 2015 levels. Overall, the union scenario modestly reduced mortality (RR: 0.90, 95% CI: 0.80, 0.99; RD per 1,000: -18.7, 95% CI: -36.5, -0.9) relative to the non-union scenario. However, the scenarios' effects largely did not vary by subgroup, and modeling did not suggest racial and educational mortality inequities would lessen if union density had remained at baseline levels.

Overall, we found little evidence that declining union density (at least as operationalized in this dissertation) explained changing racial and educational mortality inequities in the U.S. over the last several decades. However, our results did suggest that increasing state-level union density might decrease overdose/suicide mortality, and that increasing individual-level union membership might decrease all-cause mortality.

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Chapter 1. INTRODUCTION

Over the last several decades in the U.S., life expectancy has stagnated or declined for the poor and working class and risen for the middle and upper classes.[1] Declining life expectancy among those at the bottom of the class structure contributed to an overall decline in U.S. life expectancy annually from 2014-2017, a break with the 60 prior years of general improvement.[2] Increased mortality rates from drug poisonings and suicides among sections of the working class have contributed substantially to these trends.[1] Nonetheless, few studies have sought to explain the widening class inequities in longevity. Moreover, the existing empirical studies have focused on changes in the class distribution of “risk behaviors”, like smoking or drug use, which provide only partial explanations.[1] The concurrent rise of health and income inequities[3] suggests that structural factors – the economic, social, and political environments that "structure" social outcomes[4] – have exacerbated inequities across multiple domains. One such structural factor – declining labor union density – has precipitated burgeoning income inequity.[5] The disproportionate decline of union membership among less-educated, racialized, and blue-collar workers,[6] and the well-documented relationship between unions and wages, benefits, and occupational safety, suggests a connection between declining union density and growing health inequities,[7,8] an hypothesis this dissertation tested.

1.1 GROWING HEALTH AND INCOME INEQUITIES

Income inequity today in the U.S. is greater than any time since the Great Depression.[3] Since 1980, average pre-tax incomes of the bottom 50% of Americans have stagnated while incomes at the top have skyrocketed.[1] For example, in 1980, the top 1% of adults earned on average 27 times more than the bottom 50%, whereas in 2015, they earned 81 times more (pre-tax).[3] Moreover, the share of pre-tax income going to the wealthiest 10% increased from 34% in

1980 to 47% in 2015, while the share going to the bottom 50% halved.[9] Income inequities across social classes have also grown; one study estimated that the household-income gap between the large business owning class and the working class increased 2.5 fold from the 1970s to the 2000s.[10] Meanwhile, racial income inequities have remained largely unchanged since the late 1960s.[11]

Mortality inequities across socioeconomic groups – already substantial several decades ago – have also increased. For example, period life expectancy inequities between high and low socioeconomic groups (defined by income or education) have increased 1-2 years since the 1980s, due partially to increasing overdose and suicide rates among the marginalized.[1] Among certain demographics, inequities have increased more sharply. One study found that among white women, the age-25 life expectancy gap between those with a college degree and those without a high school degree increased by 2.2 years from 1991-2005; among those without a high school degree, age-25 life expectancy actually *declined* by 0.7 years (from 58.5 years to 57.8 years).[12] Cohort inequities (inequities between those born in the same year or era) have increased more substantially. The National Academies of Sciences, Engineering, and Medicine (NAS) projects that the age-50 life expectancy gap between those in the top and bottom lifetime-income quintiles will increase from 5.1 years among men born in 1930 to 12.7 years among men born in 1960. Among women, the gap will increase from 3.9 years to 13.6 years.[13] Moreover, NAS projects that age-50 life expectancy among those in the bottom income quintile will stagnate for men across the two cohorts and decrease for women.[13] Although racial disparities in survival have narrowed, substantial inequities persist.[14] For example, in 2015, life expectancy for white men was 4.1 years longer than life expectancy for Black men, while life expectancy for white women was 2.6 years long than life expectancy for Black women.[15] One study estimated that racial survival inequities precipitated 2.7 million excess Black deaths between 1970 and 2004.[16] Moreover, in 2016, mortality rates among Black men increased for the second consecutive year, eroding the inequity reductions of prior decades.[17]

Falling relative incomes among the poor and working class and a strengthening income-mortality relationship have contributed to the growing mortality inequities.[1] Social service cuts,[18] increasing costs of necessities like health care,[19] and mass incarceration[20] may have exacerbated the latter. Furthermore, income inequity may directly worsen health inequities by giving the wealthy more political power (who are less likely to support health-promoting policies than others) and by undermining social cohesion.[1] Among the disadvantaged, increasing rates of deaths due to drug overdoses, suicides, and alcohol-related liver disease may reflect a loss of social cohesion, as well as a loss of hope, given reduced economic mobility.[1,21]

1.2 Labor unions, social inequities, and health

From 1983-2017, the proportion of wage and salary workers covered by labor union contracts (union density) decreased from 23.2% to 11.9%.[22] This decrease was particularly sharp among Black and less-educated workers. For example, union density decreased from 31.7% to 13.7% among Black workers (versus from 22.2% to 12.2% among white workers) and from 23.4% to 5.9% among those with less than a high school education (versus from 22.7% to 13.7% among those with a college degree or more).[22] Union militancy has decreased alongside union density. Faced with increasingly hostile business and political opposition,[23] many union leaders have turned to partisan electoral politics and labor-management partnerships, eliding the rank-and-file organizing and direct action undergirding organized labor's historical successes.[24] For example, the number of strikes involving at least 1,000 workers decreased from 200-400 per year in the 1970s to fewer than 20 per year in the 2010s.[25] Declining militancy may have undermined union organizing for health-equity-promoting policies[24] and reduced salutary solidarity among union members.[26]

Multiple studies have connected declining union density to growing income inequity.[5,27–30] Low union density undermines worker power over working conditions and wages, which has spillover effects for prevailing standards in non-unionized workplaces,[5] and undermines union organizing for broad social benefits, such as a $15 minimum wage.[31] For example, one study estimated that declining union density from 1973-2007 explained a third of the rise of wage inequity among men and a fifth of the rise among women.[5] Another estimated that if union density had remained at 1973 levels, Black-white wage inequity in 2007 would have been 3%-10% lower among men and 13%-30% lower among women.[29]

Unions may also protect health and reduce health inequities, as the labor movement has struggled with worker health and safety since the beginning of the industrial revolution.[7,8] Labor organizing led to the enactment and enforcement of regulations that improved working conditions, such as child labor laws, restrictions on working hours, and occupational safety standards.[32,33] Overall, despite some conflicting tendencies in the union movement, union organizing contributed to the creation and preservation of salutary social programs like Medicare, Medicaid, and Social Security.[34,35] Moreover, although undermined in certain industries by racism, sexism, and red-baiting (the persecution of suspected socialists, communists, and anarchists),[36] unions helped institutionalize compensation and non-wage benefits in heavily-unionized industries, benefiting union and non-union workers alike.[7,8] Today, unionized workers enjoy better wages, benefits, and job security than non-unionized workers in the same occupational category,[7,8] particularly blue-collar workers, women, and people of color.[37,38] They also enjoy more job control and social support.[7,8] Higher wages, better working conditions, and increased control and security improve health and wellbeing.[39,40] Indeed, the society-wide health benefits of strong labor unions have been formally recognized by the American Public Health Association.[41,42]

Figure 1.1 displays the specific hypothesized pathways linking unions and health. At the workplace level, by regulating the balance of power between workers and management, unionization may improve worker health by reducing material deprivation (e.g., inadequate wage and non-wage benefits),[30,39] occupational hazards (e.g., toxic chemicals),[7,8] and stressors (e.g., job instability or a lack of job autonomy),[39] factors which can cause chronic disease and occupational injury, as well as mental illness, drug use, and their sequelae, like suicide and fatal overdose.[43] Similarly, at the societal level, by regulating the balance of power between the working class and owning class, unionism may improve working-class health by advancing economy-wide workplace compensation standards, labor rights, and progressively-funded social programs, reducing material deprivation and psychosocial stressors throughout the working class, union membership aside.[5,8,44,45] There are non-recursive relationships between unionism and many of the mediating pathways outlined in the model, since certain policies and economic conditions, like a low unemployment rate, may augment worker bargaining power and thus make it easier to unionize (or vice

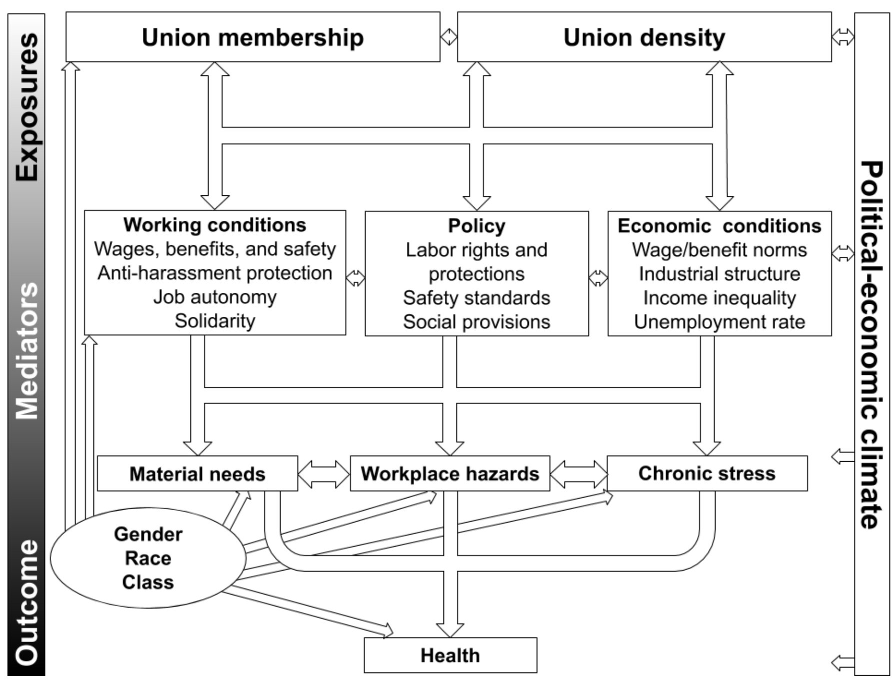

Figure 1.1. Conceptual model linking unionization and health.

versa).[46] Unionism's health benefits may be stronger for marginalized and minoritized groups than for others, suggesting a connection between unionism's decline and growing health inequities. For example, studies have found the union wage premium – typically 15-20% – is largest for less-educated, Black, and less-skilled workers, as is the benefit premium.[30] Moreover, union-supported policies, like a $15 minimum wage, often disproportionately affect the wellbeing of marginalized groups.[31]

Nonetheless, there are reasons for skepticism about unionism's salutary effects and the role of declining unionization in growing health inequities. First, although unionization can improve working conditions, its effects – like a 15-20% wage premium[30] – may be too weak to substantially improve health, particularly given weakening union power over the last several decades.[39] Second, social-class inequities in mortality, particularly those due to overdose or suicide, have primarily grown in the last several decades, well after union density began precipitously declining. Finally, empirical health research on labor unions is limited. Several ecological studies have identified protective associations between union density and rates of occupational fatalities[32,47,48] and fatal overdoses.[49] However, prior individual-level studies have found that unionization *increases* occupational-injury risk, although those findings may be due to unmeasured confounding.[50] Moreover, only two individual-level U.S.-based studies have analyzed the relationship between unionism and non-occupational health outcomes among working-age adults. A study by Reynolds et al.[39] identified a modest cross-sectional association between union membership and better self-rated health (SRH) among male, less-educated, and lower-income workers, while another by Waitzman[51] found that union contract-coverage was associated with lower mortality risk among a cohort of men in the 1960s-1970s.

1.3 AIMS

This dissertation analyzed the longitudinal relationships between unions, health, and health inequities. To our knowledge, it was the first to investigate whether unionism's decline has undermined health equity.

In Chapter 2, we analyzed the state-level relationship between union density, mortality, and racial and educational mortality inequities from 1986-2016. Using inverse-probability-of-treatment-weighted Poisson models with state and year fixed effects, we estimated three-year-moving-average union-density's effects on the following year's mortality rates. Then, we tested for gender, gender-race, and gender-education effect-modification. Finally, we estimated how racial and educational all-cause mortality inequities would change if union density increased to baseline levels.

In Chapter 3, we examined the individual-level relationship between union membership, self-rated health (SRH), and moderate mental illness from 1984-2017. Using the parametric g-formula and Panel Study of Income Dynamics (PSID) data, we contrasted cumulative incidence of poor/fair SRH and moderate mental illness under two hypothetical scenarios, one in which we set all employed-person-years to union-member employed-person-years (union scenario), and one in which we set no employed-person-years to union-member employed-person-years (non-union scenario). We also examined whether the scenarios' effects varied by gender, gender-race, or gender-education using stratified models.

In Chapter 4, we analyzed the individual-level relationship between union membership, mortality, and mortality inequities using PSID data from 1979-2017. First, using the parametric g-formula, we contrasted cumulative incidence of mortality in the union and non-union scenarios. Next, we examined whether the scenarios' effects varied by race or education using stratified models. Finally, we estimated how racial and educational mortality inequities would change if union-membership prevalence across racial and educational groups had remained at 1979 levels throughout follow-up rather than at 2015 levels

In Chapter 5, we summarized our findings and discussed their implications for future research.

Chapter 2. SOLIDARITY AND DISPARITY: DECLINING LABOR UNION DENISTY AND CHANGING RACIAL AND EDUCATIONAL MORTALITY INEQUITIES IN THE UNITED STATES

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Background: Recently, U.S. life expectancy has stagnated or declined for the poor and working class and risen for the middle and upper classes. Declining labor-union density – the percent of workers who are unionized – has precipitated burgeoning income inequity. We examined whether it has also exacerbated racial and educational mortality inequities.

Methods: From CDC, we obtained state-level all-cause and overdose/suicide mortality overall and by gender, gender-race, and gender-education from 1986-2016. State-level union density and demographic and economic confounders came from the Current Population Survey. State-level policy confounders included the minimum wage, AFDC/TANF generosity, and unemployment-insurance generosity. To model the exposure-outcome relationship, we used marginal-structural-modeling. Using state-level inverse-probability-of-treatment-weighted Poisson models with state and year fixed effects, we estimated three-year-moving-average union-density's effects on the following year's mortality rates. Then, we tested for gender, gender-race, and gender-education effect-modification. Finally, we estimated how racial and educational all-cause mortality inequities would change if union density increased to 1985 or 1988 levels respectively.

Results: Overall, a 10% increase in union density was associated with a 17% relative decrease in overdose/suicide mortality (95% CI: 0.70, 0.98), or 5.7 lives saved per 100,000 person-years (95% CI: -10.7, -0.7). Union density's absolute (lives-saved) effects on overdose/suicide mortality were stronger for men than women, but its relative effects were similar across genders. Union density had little effect on all-cause mortality overall or across subgroups, and modeling suggested union-density increases would not affect mortality inequities.

Conclusions: Declining union density (as operationalized in this study) may not explain all-cause mortality inequities, although increasing union density may reduce overdose/suicide mortality.

2.1 INTRODUCTION

Over the last several decades in the U.S., life expectancy has stagnated or declined for the poor and working class and risen for the middle and upper classes.[1] The decline among those at the bottom of the class structure has precipitated an overall decline in U.S. life expectancy each of the last three years,[52] eroding a century of general improvement and suggesting a fundamental fracturing of society. Increased mortality from drug overdoses and suicides (often called "deaths of despair") among the white working class has contributed to the growing socioeconomic inequities in mortality among white people, and fatal-overdose and suicide rates are increasing across racial groups.[1,53,54] However, despite the considerable amount of research documenting the widening inequities, few studies have sought to identify the widening's causes. Moreover, studies that have tend to focus on changes in the class distribution of "risk behaviors", like drug use, which provide only partial explanations.[1] The concurrent rise of health and income[3] inequities suggests that structural factors – the economic, social, and political environments that drive social outcomes[4] – have exacerbated inequities across multiple domains. One such structural factor – declining labor union density – has precipitated burgeoning income inequity.[30] Unionism's disproportionate decline among less-educated, racialized, and blue-collar workers, and the well-documented relationship between unions and wages, benefits, occupational safety, and protective policies suggests a connection with growing mortality inequities.[7,8] Thus, in this state-level study, we analyzed union density's relationship with all-cause mortality and overdose/suicide mortality, and tested whether declining union density has exacerbated racial and educational mortality inequities.

Today, income inequity is greater than any time since the Great Depression. In 1980, the average pretax earnings of the top 1% of Americans were 27 times more than those of the bottom

50%, whereas in 2015, they were 81 times more.[3] Income inequities across social classes have also grown: from the 1970s to the 2000s, the household-income gap between the large-business-owning class and the working class increased 2.5-fold.[10] Meanwhile, racial inequities in family incomes have remained largely unchanged since the late 1960s.[11]

Mortality inequities have grown alongside the income inequities. For example, period life expectancy inequities between high and low socioeconomic groups (defined by education or income) have increased 1-2 years since the 1980s, due in part to increases in overdose and suicide rates among the marginalized.[1] Among certain demographics, inequities have grown more sharply. One study estimated that among white women, the life-expectancy gap between those with a college degree and those without a high-school (HS) degree increased by 2.2 years from 1991 to 2005; among those without a HS degree, life expectancy *declined* by 0.7 years.[12] Cohort inequities have also increased. The National Academies of Sciences, Engineering, and Medicine predicts the age-50 life expectancy gap between those in the top and bottom lifetime-income quintiles will increase from 3.9 years among women born in 1930 to 13.6 years among women born in 1960, while among men, they predict the gap will increase from 5.1 years to 12.7 years.[13] Although racial mortality disparities have narrowed, substantial inequities persist.[55] For example, in 2015, the white-Black life expectancy gap was 2.6 years among women and 4.1 years among men.[15] One study estimated racial survival inequities precipitated 2.7 million excess Black deaths from 1970 to 2004.[16]

Falling relative incomes among the poor and working class and a strengthening income-mortality relationship have contributed to the growing SES-related mortality inequities.[1] Social-service cuts,[18] increasing costs of necessities like health care,[19] and mass incarceration[20] may have strengthened the income-mortality relationship. Furthermore, income inequity itself may worsen health inequities by undermining social cohesion among the marginalized and by giving the wealthy more political power.[1] For example, societies with greater income inequity tend to spend less on income redistribution, which may undermine the ability of those in the poor and working class to satisfy their material needs.[56,57]

The proportion of U.S. wage and salary workers ages 25-to-64 covered by labor union contracts (i.e., labor union density) decreased from 27.1% to 13.1% from 1983 to 2017, including from 36.3% to 14.9% among Black workers (compared with 25.9% to 13.5% among white workers) and from 29.7% to 11.4% among workers with a HS degree or less (compared with 24.1% to 13.9% among workers with more than a HS degree).[22] Several factors have precipitated this decline, including the hostile business and political opposition faced by unions following the 1970s and 1980 recessions.[23,24] For example, the number of unfair labor practices and illegal firings by employers doubled from 1970 to 1980.[23] In response to the opposition, many union leaders pursued labor-management partnerships, suppressing the workplace organizing and direct action undergirding organized labor's historical successes;[23,24,58] from the 1970s to the 2010s, the number of strikes involving at least 1,000 workers decreased from approximately 300 per year to fewer than 20.[25] In this context of declining density and militancy, unions have grown increasingly likely to grant concessions to employers like benefit cuts in exchange for promises of job security.[39,59]

Multiple studies have connected declining union density to growing income inequity.[30] Low union density undermines worker power over wages, which has spillover effects for prevailing norms in non-unionized workplaces,[5] and undermines union organizing for redistributive policies, like a higher minimum wage.[44] For example, one study estimated that declining union membership from 1973 to 2007 explained a third of the rise of wage inequity among men and a fifth of the rise among women,[5] an effect size similar to that found in more-recent research.[30,60] Declining union density has also exacerbated racial income inequities: Rosenfeld et al. estimated that Black-white wage inequity in 2007 would have been 3%-10% lower among men and 13%-30% lower among women if union membership had remained at 1973 levels.[29]

Unions may also protect health and reduce health inequities. At the workplace level, by regulating the balance of power between workers and management, unionization may reduce exposure to material deprivation (e.g., inadequate wage and non-wage benefits, including access to health insurance and drug treatment),[30,39] occupational hazards (e.g., toxic chemicals),[7,8] and stressors (e.g., job instability or a lack of job autonomy),[39] which can cause chronic disease and occupational injury, as well as mental illness, drug use, and their sequelae, like suicide and fatal overdose.[43] For example, Hagedorn et al. found that collectively-bargained, legally-binding union contracts tend to contain provisions promoting workers' income, benefits, working-time arrangements, safety, and decision-making power.[7] The workplace-level health benefits of unionization may be stronger for less-educated, racialized, and otherwise marginalized workers, a finding demonstrated for economic outcomes like the union wage premium.[30] Similarly, at the societal level, by regulating the balance of power between the working class and owning class, unionism may improve working-class health by advancing economy-wide compensation norms, labor rights, and progressive social protections and public-health programs, reducing material deprivation and psychosocial stressors throughout the working class, union membership aside.[5,8,44,45] For example, Feigenbaum et al. found that state-level right-to-work laws, which weaken unions by allowing workers in unionized businesses to opt out of paying union dues, reduce state-level policy progressivism, such as the strength of redistributive social programs.[44]

Additionally, by fostering solidarity among workers and their broader communities, unionism may reduce alienation and feelings of powerlessness, factors associated with mental illness and drug use.[49,61] For example, DeFina et al. found that higher state-level union density was associated with lower state-level overdose death rates from 1999-2016.[49]

Despite these potential mechanisms, few US-based studies aside from DeFina et al.'s have analyzed unionism's relationship with non-occupational health outcomes or inequities in those outcomes, although several have identified a protective effect of unionization on occupational fatalities.[32,47,48] Regarding non-occupational health outcomes, Reynolds et al. identified a cross-sectional association between union membership and better self-rated health among workers who were men, had less than a college degree, or had incomes below the 75th percentile,[39] while Waitzman found that union membership was associated with reduced mortality among men in the 1960s and 1970s.[51] Given the limited prior research, the relationship between union density and mortality, as well the role of declining union density in growing mortality inequities, remains understudied.

Using a longitudinal, ecological study of U.S. states, we tested the relationships between union density, mortality rates, and racial and educational mortality inequities. Our specific goals were to estimate state-level union density's effects on all-cause mortality rates and overdose/suicide ("despair") mortality rates from 1986 to 2016, and to test for effect modification of these estimates by gender, gender-race, and gender-education. Additionally, we estimated how racial and educational mortality inequities would change if union density increased from 2015 levels to 1985 or 1988 levels respectively. We hypothesized that increases in union density would have small protective effects on all-cause mortality and stronger protective effects on despair mortality (because despair mortality may be more immediately affected by changes in union density than other types of mortality). We also hypothesized that increases in union density would be more protective for men (given their stronger attachment to the waged labor force throughout the study period), as well as for Black and less-educated people (given findings from prior research on unionism and economic outcomes), than for women, white people, and more-educated people. Finally, because of union density's disproportionate decline among Black and less-educated workers, as well as our hypothesis that union density would be most protective for those groups, we hypothesized that racial and educational inequities would lessen if union density increased to earlier levels.

2.2 Methods

2.2.1 *Data*

2.2.1.1 Exposure

Beginning in 1983 (the first year union status was available), we calculated overall and demographic-specific (gender, gender-race, and gender-education) state-level union density among 25-to-64-year-olds using the Merged Outgoing Rotation Group files of the Current Population Survey (CPS) maintained by the Center for Economic Policy Research.[62] The Census Bureau cleans CPS data and imputes missingness. Using the year-specific estimates, we created a three-year moving average union density variable using a given state's union density in that year and each of the two preceding years. We used a moving average to reduce sampling error and because average union density over several years may be more mortality-relevant than union density in a single year. We excluded respondents ages 65 and older to focus on populations with high labor-force participation, as union density may be most relevant to their health. We excluded respondents below age 25 not only because of their lower labor-force participation, but also because education level at death, which we used in the gender-education effect-modification analyses, may not accurately proxy socioeconomic status in this group.

The overall state-level union density measure was the exposure for our primary analyses, while the gender, gender-race, and gender-education specific state-level union density measures were the exposures for each of the effect-modification analyses. In the effect-modification analyses, we defined race as white or Black (the only consistently-coded races in the outcome data) and education as ≤HS or >HS educated. Because decedents with < HS education are sometimes misclassified as having a HS degree on death certificates, our chosen categories minimized misclassification.[63]

2.2.1.2 Mortality counts

Annual overall, gender-, and gender-race-specific state-level all-cause mortality counts among 25-to-64-year-olds from 1986 to 2016 came from CDC's Compressed Mortality Files (CMF).[64] Due to small counts, CDC suppressed 307 Black-female-state-year observations and 231 Black-male-state-year observations, including all Black-state observations from MT, ND, SD, VT, and WY. Gender-education specific state-level all-cause mortality counts from 1989 to 2004 (1989 was the first year death certificates recorded education) came from CDC's publicly-available Multiple Cause of Death (MCD) files maintained by the National Bureau of Economic Research.[65] Counts from 2005 to 2016 came from CDC's restricted MCD files. Twenty-one states recorded no education information on their death certificates in at least one year; we excluded these state-year observations from our analyses. In other state-years, a fraction of deaths lacked education (the mean state-level proportion of deaths lacking education information was 3.8%). For these state-years, we used hot-deck imputation to impute missing values assuming the missingness was random conditional on state, year, gender, race, and age.[66]

Using the same sources, we also obtained annual overall and gender-specific state-level counts of deaths with underlying causes of drug and alcohol poisoning and suicide ("deaths of despair").[67,68] CDC suppressed 31 female-state-year observations due to small counts. We did not obtain gender-race- and gender-education-specific state-level despair-deaths due to small counts. Appendix A1 discusses the ICD codes used in these analyses.

2.2.1.3 Population denominators

For analyses of overall, gender-, and gender-race-specific state-level mortality, population denominators came from CDC WONDER, which provided estimates from the decennial census for 1990, 2000, and 2010 and interpolated estimates for intercensal years.[64] For analyses of gender-education-specific state-level mortality, we estimated the denominators using decennial census and American Community Survey (ACS) data maintained by IPUMS.[69]

2.2.1.4 Confounders

Potential time-varying state-level confounders identified *a priori* included: 1) the distribution of age, race, and education, 2) social/labor policies (the minimum wage, the AFDC/TANF-to-poverty ratio – an indicator of welfare generosity, and the unemployment insurance recipiency rate – an indicator of unemployment insurance generosity), 3) economic conditions (unemployment rate, mean hourly wage, and per-capita GDP), and 4) industrial structure (percent of workforce employed in: a) manufacturing, b) transportation and utilities, c) construction, and d) the public sector). State and year, which we modeled as fixed effects, were also confounders due to time-invariant differences across states in confounding factors (e.g., labor laws), as well as secular trends across states in such factors (e.g., recessions or the fatal-overdose epidemic). Table I displays how we specified the time-varying confounders; Appendix A2 displays their sources. In our primary analyses, we measured non-policy confounders at the state-level among 25-to-64-year-olds, while in our effect-modification analyses, we measured non-policy confounders at the state-demographic level among 25-to-64-year-olds. The confounders did not contain any missingness.

2.2.2 *Statistical methods*

We hypothesized prior union density affected prior socioeconomic conditions and policies (because unionization may immediately affect factors like wages), as well as current socioeconomic conditions and policies (because union organizing may take time to affect factors like policies), and mortality (Figure 2.1). In turn, current socioeconomic conditions and policies affected future union density (because certain conditions and policies may make it easier to unionize, or vice versa) and mortality. Thus, given that policies and socioeconomic conditions were confounders *and* mediators of the union density-mortality relationship, we used a marginal structural modeling (MSM) approach to estimate the relationship between state-level union density and state-level mortality rates.[70]

First, we calculated stabilized inverse-probability-of-treatment weights (IPTW) for each state-year observation. We defined the stabilized weight (SW) for state *i* at time *t* as:

$$SW_{it} = \frac{f_{E_{it}|I_i\,T_t}\left(E_{i,t}, I_i, T_t\right)}{f_{E_{it}|\,C_{i,t-1}, E_{i,t-3}, I_i, T_t}\left(E_{i,t,t-3}, C_{i,t-1}, I_i, T_t\right)}$$

where $f(\cdot)$ is the probability density function for the exposure *E*, *I* is state, *T* is year, and *C* is a vector of time-varying confounders. The weight denominator for state *i* in year *t* was the density of the exposure model at the observed exposure value E_{it} given prior time-varying confounders C_{it-1}, prior exposure E_{it-3}, state I_i, and year T_t. Using R code from Hazelbag et al.,[71] we estimated this probability-density function using a pooled gamma linear model that had three-year moving average state-level union density as the outcome and the aforementioned time-varying confounders (lagged one year), prior exposure (lagged three years to reduce collinearity), and state and year fixed effects as predictors. Not lagging the time-varying confounders did not meaningfully affect the estimated weights. To estimate the weight numerator, we used the same gamma linear model but included only state and year fixed effects as predictors. The final stabilized weight for each observation was the product of the state's weight in a given year and each of the two preceding years. This approach assumed that a state's probability of receiving its union density value in a given year was independent of exposure and confounder history beyond the two preceding years;[71] we were unable to include more years without the weight range growing large. Large estimated weights, which often result from non-positivity, make certain observations exert undue influence in the outcome model, causing imprecision and bias.[72] Following standard practice, we used weight truncation to ensure the weights had a mean of approximately 1 and a small range.[72] For the main analyses, we truncated the weights at the 5th and 95th percentiles (i.e., weights smaller than the 5th percentile or larger than the 95th percentile were set equal to the 5th percentile or 95th percentile weight values respectively). Appendix A3 shows the weight distribution.

Next, in Stata, we used log-linear Poisson models with IPTW weights, state and year fixed effects (modeled using dummy variables), an offset of log(population size), and state-level cluster-robust standard errors to estimate the effects of a 10% (interquartile range) increase in one-year lagged, three-year moving average state-level union density on the following year's state-level all-cause and despair mortality rates;[73,74] ordinary least squares models weighted by state-population size and the IPTW with mortality rates as the outcome yielded similar estimates (Appendix A4). Because state and year fixed effects were in the numerator and denominator of the IPTW, we included them in the outcome models to adjust for confounding by those factors.[72] The Poisson models directly estimated the relative effects of union density on mortality (i.e., risk ratios or RRs). We also estimated absolute effects (i.e., risk differences or RDs) using Stata's “margins” command.[75] We conducted the analyses on a sample of 1,581 observations (51 states – including D.C. – observed for 31 years each).

To test for effect modification of the union-density-mortality relationship by gender, we calculated IPTW using a similar approach. However, in addition to the aforementioned time-varying confounders, the denominator model included gender by state-fixed-effect and gender by year-fixed-effect interaction terms as predictors (i.e., gender-state and gender-year fixed effects), due to time-invariant state-level differences in gender disparities in union density and mortality, as well as strong gender-specific temporal trends in such factors across states. In addition, we included the preceding year’s union density exposure among the complementary gender (e.g., for a given woman-state-year observation, the preceding year’s union density exposure among men in that state). We included the complementary gender’s union density because it may affect the given gender’s propensity to unionize, as well as their access to health-promoting resources. The numerator model included gender-state and gender-year as predictors. We truncated the final weights at the 4th and 96th percentiles (Appendix A3). We then tested whether the relationship between union density and mortality varied by gender by including a union density by gender interaction term in the IPTW log-linear Poisson models, which also included gender-state and gender-year fixed effects. The models provided evidence about multiplicative effect modification directly. We tested for additive effect modification using “margins”. We conducted the analyses on a sample of 3,162 observations for all-cause mortality and 3,131 observations for despair mortality.

To test for effect modification of the union-density-mortality relationship by race, we first stratified the data by gender, due to gender differences in levels of union density and mortality, as well as in the hypothesized relationship between the variables. Next, we calculated stabilized IPTW. The denominator models included the time-varying confounders as predictors, as well as race-state and race-year fixed effects. We also included the union density exposures among the complementary gender-races. The numerator models included race-state and race-year as predictors. We truncated the final weights at the 4th and 96th percentiles for women and men (Appendix A3). We then tested whether the relationship between union density and all-cause mortality varied by race by including a union density by race interaction term in the IPTW log-linear Poisson models, which also included race-state and race-year fixed effects. To reduce exposure measurement error, we excluded gender-Black-state-year observations with fewer than 50 Black respondents surveyed about their union-contract coverage on average over the prior three years; increasing the cutoff to 100 did not change our effect estimates. We also excluded gender-race-state-year observations that had estimated complementary gender-race union densities of 0% or 100%; these values resulted from small cell sizes in the CPS and caused the IPTW models to fit poorly. Excluding the complementary gender-race union densities as predictors in the IPTW models did not affect the estimated weights. Overall, the exclusions reduced the sample size from 2,855 to 2,347 observations among women and from 2,931 to 2,279 observations among men. After the exclusions, certain states lacked Black observations throughout follow-up: AL, AZ, HI, ID, IA, ME, MT, NE, ND, NH, NM, OR, SD, UT, VT, WV, WY, and, among women, WA.

To test for effect modification of the union-density-mortality relationship by education, we used a similar approach to the racial effect-modification analyses. However, to reduce misclassification, we excluded state-year observations in which more than 20% of death certificates lacked education information (n=266); increasing the threshold to 30% did not meaningfully change our estimates, nor did excluding the 21 states that recorded no education information on their death certificates in at least one study year. We truncated the final weights at the 4^{th} and 96^{th} percentiles for women and men (Appendix A3). For each gender, we conducted the analyses on a sample of 2,590 observations.

Finally, to estimate counterfactual racial and educational mortality inequities, we used parameter estimates from the IPTW log-linear Poisson models to compare how state-level all-cause mortality rates would change for Black people versus white people and the less-educated versus the more-educated if three-year moving average union density increased from mean 2015 levels to mean 1985 or 1988 levels respectively for each group across all states. Regarding racial inequities, this corresponded to an increase in union density from 14.7% to 29.4% among Black women, 13.2% to 18.3% among white women, 17.2% to 38.4% among Black men, and 14.6% to 29.1% among white men). Regarding educational inequities, this corresponded to an increase in union density from 9.2% to 15.6% among less-educated women, 14.8% to 19.9% among more-educated women, 13.5% to 31.6% among less-educated men, and 14.4% to 21.4% among more-educated men. Our approach assumed union density's mortality effects did not change temporally.

2.2.3 *Sensitivity analyses*

We tested our results' robustness to three specific concerns.

First, we tested the sensitivity of our results to the estimated weights. In MSM analyses, obtaining asymptotically unbiased estimates requires correct specification of the weighting model.[76] In settings with continuous exposures, this can be complicated by the need to identify the exposure's correct distributional form.[76] Our primary analyses used gamma models to generate the weights rather than linear models because weighting by gamma IPTW may lead to less-biased estimates for skewed exposures.[76] We tested the sensitivity of our results to this choice by re-estimating the IPTW using linear models (with R code from Hernan et al.[77]) prior to fitting the outcome models.

Second, we tested whether lagging the exposure three years rather than one year affected our results. Although certain health-related factors may be immediately affected by changes in union density, others may remain unaffected for several years. Moreover, changes in such factors may take several years to affect health.

Finally, we tested for nonlinear union density-mortality relationships. Unions could plausibly have stronger mortality effects at higher densities if unions at lower densities lack the power to organize workers and change working conditions. Contrarily, unions could have stronger mortality effects at lower densities given diminishing returns to improving such factors. We tested for nonlinear relationships by modeling union density with 3-knot restricted cubic splines (with knots at the 10th, 50th, and 90th union-density percentiles) in the outcome analyses. We estimated RRs from the spline terms using Stata's "xblc" command.[78]

2.3 RESULTS

Three-year moving average union density among 25-to-64-year-olds decreased from 25.4% in 1985 to 13.6% in 2015 (Figure 2). This decrease did not distribute evenly across genders, races, or education groups, but rather concentrated among Black and less-educated workers, particularly Black and less-educated male workers (Figure 2.2). The decrease did not distribute evenly across states either; in New Hampshire, union density decreased from 15.4% to 12.8%, whereas in Michigan, it decreased from 38.0% to 18.5%, including from 72.3% to 28.0% among Black male workers and from 57.6% to 20.8% among ≤ HS-educated male workers. Compared to state-year observations with lower union density, state-year observations with higher union density tended to be less Black, more educated, and have higher mean wages, minimum wages, AFDC/TANF-to-poverty ratios, and unemployment insurance recipiency rates (Table 2.1).

A 10% increase in one-year lagged, three-year moving average union density was not associated with all-cause mortality (RR: 0.99 [95% CI: 0.91, 1.08]; RD per 100,000: -3.2 [95% CI: -36.3, 29.8]) (Table 2.2). However, it was associated with a 17% decrease in despair mortality (95% CI: 0.70, 0.98), which corresponded to a 5.7 per 100,000 person-years decrease in the outcome (95% CI: -10.7, -0.7). We found little evidence that union density's effect on all-cause mortality varied by gender on the additive or multiplicative scales (Table 2.2). However, although its effect on despair mortality did not vary by gender on the multiplicative scale, it did vary by gender on the additive scale (Table 2.2). Specifically, among women, a 10% increase in one-year lagged, three-year moving average union density was associated with a 2.3 per 100,000 person-years decrease in despair mortality (95% CI: -6.0, 1.3), while among men, it was associated with an 11.0 per 100,000 person-years decrease (95% CI: -16.8, -5.1); the difference in these RDs was 8.6 (95% CI: 3.5, 13.8). Finally, we found no evidence that union density's effect on all-cause mortality varied by gender-race or gender-education (Table 2.3). Rather, across genders, races, and education groups, its effects were null.

Modeling suggested that Black-white all-cause mortality inequities would remain largely unchanged if one-year lagged, three-year moving average union density increased to baseline levels. Specifically, among women, modeling suggested inequities would increase by 3% (95% CI: 0.95, 1.12), a 6.8 per 100,000 person-years increase in the inequity (95% CI: -22.9, 36.4). Among men, meanwhile, modeling suggested inequities would increase by 7% (95% CI: 0.92, 1.24), a 26.1 per 100,000 person-years increase in the inequity (95% CI: -82.5, 134.8) (Table 2.4).

Modeling also suggested educational inequities would be largely unaffected if union density increased to baseline levels. Specifically, among women, modeling suggested educational inequities would decrease by 1% (95% CI: 0.93, 1.06), an 8.8 per 100,000 person-years reduction in the inequity (95% CI: -38.9, 21.4), while among men, modeling suggested educational inequities would decrease by 3% (95% CI: 0.86, 1.09), a 44.1 per 100,000 person-years reduction in the inequity (95% CI: -135.3, 47.1) (Table 2.4).

Our results were somewhat sensitive to the estimated IPTW, particularly in despair-death analyses (Appendix A5). For example, in models weighted by linear IPTW, a 10% increase in one-year lagged, three-year moving average union density was associated with just an 11% decrease in despair mortality (95% CI: 0.72, 1.09), smaller than the estimate produced by models weighted by gamma IPTW. Nonetheless, lagging the exposure three years instead of one year did not meaningfully change most of the effect estimates, although increases in three-year lagged union density appeared to be protective for white men (Appendix A6). Finally, there were nonlinear relationships between union density and all-cause mortality for the overall and effect-modification analyses (Appendix A7). Specifically, increases in union density tended to be more protective at lower densities than at higher densities (although the estimates were imprecise), and increases in union density appeared to be most protective for white men. Nonetheless, modeling union density with a spline term did not appreciably change estimates from the counterfactual racial- and educational-inequity analyses. Meanwhile, for despair mortality, increases in union density tended to be more protective at higher densities than at lower densities.

2.4 DISCUSSION

In our primary analyses, we found that increases in union density had protective effects on despair mortality but little effect on all-cause mortality. Moreover, union density's effects did not vary substantially by gender, gender-race, or gender-education, and modeling did not suggest relative or absolute racial and educational all-cause mortality inequities would meaningfully change if union density increased to 1985 or 1988 levels. Across subgroups, weighted estimates tended to be smaller than unweighted estimates (sometimes considerably), suggesting that time-varying confounding of the union density-mortality relationship by economic, political, and demographic factors may have been considerable, even after adjusting for state and year fixed effects. Given the potential non-recursive relationship between union density and these time-varying confounders, our MSM approach to confounder-adjustment may have been less biased than traditional covariate-adjustment approaches. Although we did identify a protective effect of union density on all-cause mortality in certain sensitivity analyses – particularly among white men – we believe these estimates should be interpreted cautiously given the large number of statistical tests performed, generally small effect sizes, and our approach's limitations (described below).

Several factors may explain our largely null findings. First, while unionization can improve working conditions, its effects – like a 15-20% wage premium[30] – may be too weak to lower mortality rates, particularly given weakening union power. Second, SES inequities in mortality have primarily grown in recent decades, which is well after union density began precipitously declining,[1] suggesting other factors have played a larger role in the trend. That some of the largest inequity increases have occurred among women suggests that explanations focused solely on changes in the organization of wage labor, like declining union density, may be inadequate, particularly given patterns of sexism in the labor movement.[21] Third, although prior research has shown that the union wage and benefit premium is larger for Black workers than for white workers,[30] racism in the union movement[79,80] may undermine the health benefits of unionization for Black workers. Although many unions today do work to protect members against workplace harassment and discrimination, some have ignored or been hostile to such concerns, particularly unions dominated by white workers.[7] Fourth, because some union hierarchies have grown detached from their rank-and-file membership,[24,58] the solidarity-promoting and alienation-reducing effects of union membership and high union density may have weakened. Finally, we may not have identified the relevant etiologic period for the exposure. For example, average union density throughout one's lifetime may be more relevant to health than the short-term changes we analyzed.[81] Relatedly, certain outcomes that could be affected by union density, like stress-related cardiovascular disease, may not strike until older ages.[82]

To our knowledge, no prior research has analyzed the effects of union density on these mortality outcomes. A recent state-level instrumental-variable analysis that used right-to-work-laws as an instrument for union density and data from 1992 to 2016 found that a 1% increase in union density was associated with a 5% (95% CI: 0.93, 0.97) decrease in occupational fatalities.[48] Given that occupational fatalities constitute a small proportion of all-cause mortality, these findings do not contradict our own. Another recent study using data from 1999 to 2016 found that a 10% increase in union density was associated with a 2.7 per 100,000 person-years (95% CI: 0.5, 4.9) decrease in the overdose mortality rate.[49] Coupled with ours, these latter findings – and those of other studies[67] – point to the potential alleviation of overdose and suicide mortality with more robust social and economic protections for marginalized populations. Nonetheless, a recent study by Ruhm suggests that worsening economic conditions (and attendant increases in "despair") have played a relatively small role in the fatal-overdose epidemic compared with changes in the drug environment, namely the cost, composition, and availability of drugs.[83] Other researchers have also challenged the “deaths of despair” framing, arguing that rather than “despairing”, marginalized groups in the U.S. have long struggled against their oppression, a stressful coping process which itself can harm health.[53] Critics have further argued that excessive focus on “deaths of despair" has led researchers to overlook the primary causes of premature death among Black people and ignore other diseases contributing to widening SES inequities in mortality.[53] More research is needed to disentangle the relative contributions of various social and economic factors to these phenomena.

Our analyses had several limitations, including potential violations of the strong MSM assumptions, which are: 1) no unmeasured confounding, 2) positivity (non-zero probability of all exposure-confounder combinations), 3) counterfactual consistency (states’ counterfactual outcomes under their observed exposure histories equal their observed outcomes), 4) no interference (states’ potential outcomes do not depend on other states’ exposures), and 5) no model misspecification.[72,84] Regarding the no-unmeasured-confounding assumption, our estimates may have been biased by time-varying confounding due to the exposure-outcome relationship’s complexity and our inability to incorporate states’ complete confounding histories into the IPTW. One potential unmeasured confounder was an indicator of state-level health-insurance generosity, such as the percent insured, consistent data on which was not available for all study years. Although estimates from models adjusted for percent insured for the years a consistent measure was available (1988 to 2013 in the CPS) closely resembled estimates from models unadjusted for the variable (Appendix A8), there may be other unmeasured confounders. Regarding the positivity assumption, our analyses had no structural positivity violations, given that each observation could have theoretically received any value of the exposure and confounders. Nonetheless, random positivity violations can cause unstable weights, a problem we observed.[72] Although we used weight truncation to reduce bias and variance from weight variability, such truncation may have decreased control of confounding bias.[72] Regarding the consistency assumption, our analyses assumed union density's effects on mortality were independent of how union density changed. This assumption would be violated if, for example, increases in union density driven by rank-and-file organizing have different mortality effects than increases driven by labor-law changes. Nonetheless, we think meaningful consistency violations are unlikely given our largely null overall findings. Regarding the no-interference assumption, in preliminary analyses we found no evidence that union density in one state affected mortality rates in another state (Appendix A9), although more rigorous approaches are needed to rule interference out. Finally, regarding the no-model-misspecification assumption, our results' sensitivity to the choice of IPTW, as well as the large range of weights, raises concerns about possible misspecification of the weighting model, which could have biased our estimates.

In addition to potential violations of certain MSM assumptions, our analyses may have suffered from sampling error, particularly in the race by union density analyses, despite our use of moving averages and sample-size restrictions. For Black-state-year observations, we based the CPS-derived exposure and confounding variables on responses from just 241 Black women and 195 Black men on average per year. For approximately ¼ of Black-state-year observations, we based the exposure and confounding variables on responses from fewer than 100 Black respondents of each gender on average per year. These small sample sizes increased the risk of random error in our union-density estimates. Sample size aside, nondifferential exposure measurement error may have also biased our density estimates. For example, Card found that 2.5% of CPS respondents in 1977 misreported their union status.[85] Although sampling error had an unpredictable effect on our estimates, measurement error may have biased them away from the null.[86]

Despite these limitations, our analyses had several strengths. First, few studies have used MSM approaches in ecological settings with continuous exposures and extensive follow-up. We demonstrated the feasibility of using MSMs in such settings, as well as the potential pitfalls, like unstable weights. Second, our 30 years of follow-up was much longer than the few prior studies on union density and health.[47–49] Third, we collected comprehensive time-varying confounder data, which we drew from a variety of sources. Thus, we may have more thoroughly adjusted for time-varying confounding than prior studies on the topic, particularly because our MSM approach accommodated the non-recursive relationships between union-density and the time-varying confounders. Finally, to our knowledge, this is one of few empirical studies to examine the role of structural factors – rather than behavioral ones – in explaining recent increases in mortality inequities across socioeconomic groups.

2.5 Conclusion

In summary, we found that increases in state-level union density were associated with reductions in despair mortality. However, we found little consistent evidence that state-level union density affected state-level all-cause mortality rates overall or among subgroups.

Given the important roles of unions in wages, working conditions, and political-economic factors, our largely null findings raise questions that should be pursued in future research. First, researchers should examine these associations at the individual-level, including how individual-level union membership interacts with area- or industry-level union density to produce health outcomes. Individual-level data could also allow researchers to analyze interactions between union membership and relational social class measures based on property ownership and supervisory authority, which may more strongly interact with union membership than SES-based measures like education.[87] Second, researchers should test how long-term changes in union density affect health outcomes; states' cumulative union density over several decades may have stronger mortality effects than the short-term changes we analyzed. Finally, researchers should examine how alternative measures of labor-movement and working-class power, like the strike rate, affect health and health inequities, both independently and through their interactions with union density.

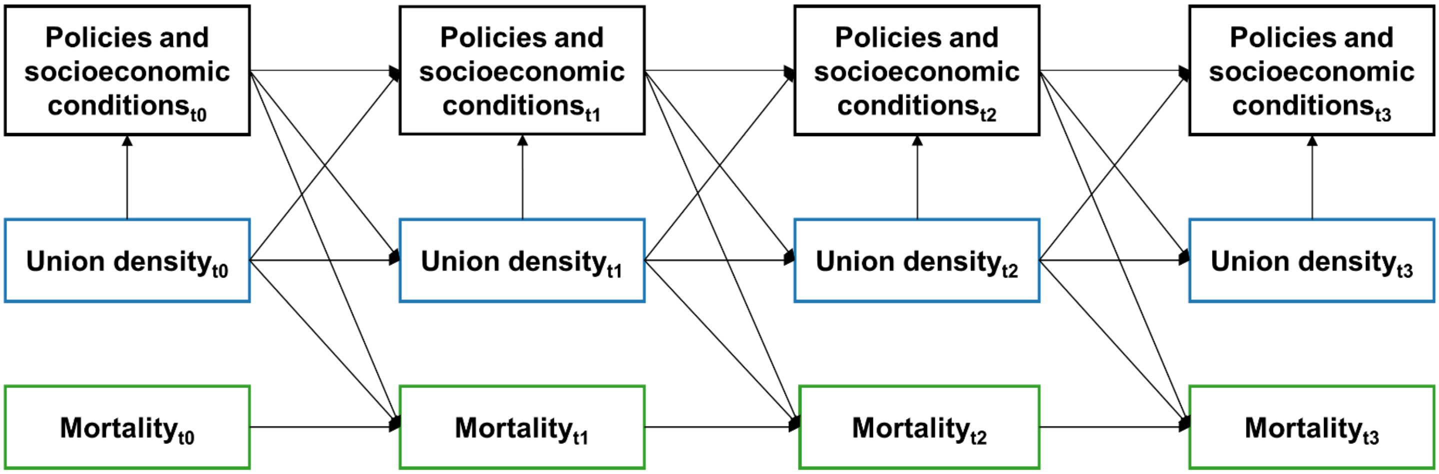

Figure 2.1. Directed acyclic graph depicting hypothesized relationship between union density, mortality, and time-varying confounders. Policies and socioeconomic conditions are confounders and mediators of the union density-mortality relationship.

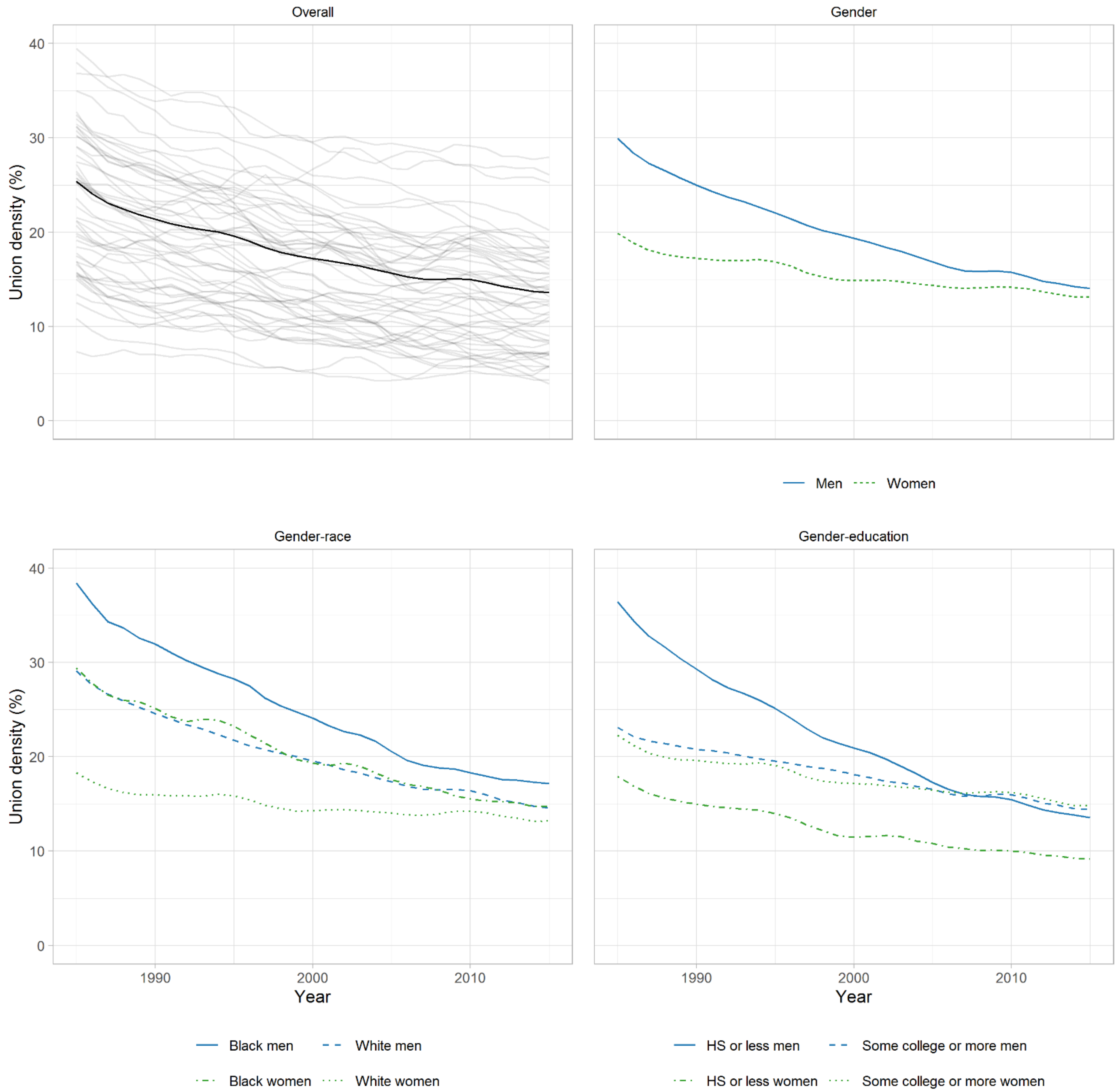

Figure 2.2. Three-year moving average union density in the United States from 1985 to 2015 among 25-to-64-year-old wage and salary workers overall (with state-specific trends shown in grey), as well as by gender, gender-race, and gender-education. **Note:** Union density is the proportion of wage and salary workers covered by a labor-union contract, which we estimated from the Current Population Survey's Merged Outgoing Rotation Group files maintained by the Center for Economic Policy Research.

Table 2.1. Mean (SD) of three-year moving average state-level variables, as well as state-level mortality rates, in 1985 (top) and 2015 (bottom) across year-specific union-density quartiles.[a]

			Quartile 1	Quartile 2	Quartile 3	Quartile 4
1985	Age	% 25-34 years old	0.35 (0.02)	0.36 (0.02)	0.35 (0.02)	0.35 (0.03)
		% 35-44 years old	0.27 (0.01)	0.26 (0.01)	0.26 (0.01)	0.27 (0.01)
		% 45-54 years old	0.19 (0.01)	0.19 (0.01)	0.19 (0.01)	0.19 (0.01)
		% 55-64 years old	0.19 (0.02)	0.18 (0.02)	0.19 (0.01)	0.19 (0.03)
	Gender	% women	0.51 (0.01)	0.51 (0.01)	0.51 (0.01)	0.51 (0.01)
	Race	% white	0.79 (0.11)	0.89 (0.08)	0.83 (0.20)	0.84 (0.17)
		% Black	0.11 (0.10)	0.07 (0.09)	0.11 (0.19)	0.06 (0.04)
		% other	0.09 (0.12)	0.04 (0.03)	0.06 (0.07)	0.10 (0.18)
	Education	% <HS	0.23 (0.06)	0.19 (0.08)	0.18 (0.03)	0.18 (0.06)
		% HS	0.36 (0.02)	0.39 (0.03)	0.38 (0.06)	0.41 (0.04)
		% some college	0.21 (0.04)	0.22 (0.05)	0.20 (0.04)	0.20 (0.05)
		% college	0.20 (0.03)	0.20 (0.04)	0.24 (0.05)	0.21 (0.04)
	Econ. conditions	% unemployed	0.06 (0.01)	0.06 (0.02)	0.06 (0.01)	0.08 (0.02)
		GDP per capita ($10k)[b]	3.40 (0.44)	3.71 (0.78)	4.28 (1.84)	4.26 (2.13)
		Mean wage ($)[b]	19.26 (1.82)	19.47 (1.85)	21.16 (2.31)	22.02 (3.00)
	Ind. structure	% agriculture	0.04 (0.02)	0.06 (0.05)	0.03 (0.03)	0.03 (0.02)
		% services	0.75 (0.06)	0.75 (0.04)	0.77 (0.09)	0.75 (0.07)
		% manufacturing	0.21 (0.07)	0.19 (0.06)	0.20 (0.09)	0.22 (0.08)
		% public sector	0.18 (0.04)	0.18 (0.02)	0.19 (0.07)	0.18 (0.04)
	Policies	State - federal MW ($)[c]	0.00 (0.00)	0.00 (0.00)	0.08 (0.27)	0.09 (0.33)
		AFDC/TANF:poverty[d]	0.40 (0.13)	0.48 (0.14)	0.73 (0.28)	0.72 (0.16)
		UI recipiency rate[e]	0.21 (0.04)	0.27 (0.08)	0.31 (0.06)	0.31 (0.09)
	Mortality	All-cause per 100k	441.75 (60.04)	405.08 (79.40)	447.53 (91.65)	416.24 (70.89)
		Despair per 100k	20.27 (3.00)	18.74 (3.84)	18.73 (6.21)	16.86 (3.85)
2015	Age	% 25-34 years old	0.27 (0.02)	0.27 (0.04)	0.24 (0.02)	0.26 (0.02)
		% 35-44 years old	0.25 (0.01)	0.24 (0.01)	0.23 (0.01)	0.24 (0.01)
		% 45-54 years old	0.25 (0.01)	0.25 (0.02)	0.27 (0.01)	0.26 (0.01)
		% 55-64 years old	0.23 (0.02)	0.24 (0.02)	0.27 (0.02)	0.24 (0.01)
	Gender	% women	0.51 (0.01)	0.50 (0.01)	0.50 (0.01)	0.50 (0.01)
	Race	% white	0.67 (0.13)	0.70 (0.16)	0.84 (0.12)	0.63 (0.18)
		% Black	0.16 (0.12)	0.12 (0.12)	0.07 (0.08)	0.08 (0.05)
		% other	0.17 (0.12)	0.18 (0.13)	0.08 (0.05)	0.29 (0.19)
	Education	% <HS	0.10 (0.03)	0.07 (0.02)	0.06 (0.02)	0.07 (0.02)
		% HS	0.31 (0.04)	0.30 (0.05)	0.33 (0.05)	0.28 (0.03)
		% some college	0.29 (0.03)	0.29 (0.06)	0.27 (0.04)	0.29 (0.04)
		% college	0.30 (0.04)	0.34 (0.10)	0.34 (0.07)	0.36 (0.05)
	Econ. conditions	% unemployed	0.05 (0.01)	0.05 (0.01)	0.04 (0.01)	0.06 (0.01)
		GDP per capita ($10k)[b]	4.90 (0.76)	6.66 (3.71)	5.26 (0.87)	6.13 (0.96)
		Mean wage ($)[b]	22.54 (2.18)	23.95 (3.48)	23.94 (2.75)	25.73 (1.98)
	Ind. structure	% agriculture	0.02 (0.02)	0.02 (0.02)	0.02 (0.02)	0.01 (0.01)
		% services	0.86 (0.03)	0.86 (0.06)	0.85 (0.05)	0.88 (0.05)
		% manufacturing	0.12 (0.02)	0.11 (0.05)	0.13 (0.04)	0.11 (0.05)
		% public sector	0.16 (0.02)	0.18 (0.05)	0.16 (0.03)	0.15 (0.04)
	Policies	State - federal MW ($)[c]	0.09 (0.23)	0.39 (0.62)	0.42 (0.53)	1.10 (0.54)
		AFDC/TANF:poverty[d]	0.10 (0.07)	0.22 (0.24)	0.27 (0.10)	0.33 (0.14)
		UI recipiency rate[e]	0.16 (0.04)	0.22 (0.08)	0.28 (0.07)	0.29 (0.07)
	Mortality	All-cause per 100k	437.23 (96.55)	433.29 (73.54)	425.74 (91.85)	349.85 (46.77)
		Despair per 100k	45.32 (10.51)	48.10 (11.41)	57.92 (15.29)	44.04 (11.07)

Notes: [a] Quartile ranges: 1985: 7.3-16.9%, 16.9-23.6%, 25.6-29.0%, 30.2-39.5%; 2015: 3.9-7.4%, 8.2-12.6%, 12.8-16.5%, 16.9-28.0%. [b] 2017 dollars. [b] MW is the minimum wage. [c] Proportion of families receiving AFDC/TANF benefits out of all impoverished families with children. [d] Share of unemployed workers receiving unemployment insurance (UI) benefits from regular state programs.

Table 2.2. Relationship between 10% increase in 1-year lagged, 3-year moving average state-level union density and state-level mortality rates from 1986 to 2016 overall and by gender.[a]

	Unweighted				**Weighted**[b]			
	RR	95% CI	RD	95% CI	RR	95% CI	RD	95 % CI
Overall								
All-cause mortality	0.92	0.84, 1.01	-30.7	-65.9, 4.5	0.99	0.91, 1.08	-3.2	-36.3, 29.8
Despair mortality	0.69	0.56, 0.85	-10.8	-17.1, -4.6	0.83	0.70, 0.98	-5.7	-10.7, -0.7
Gender								
All-cause mortality								
Women	0.89	0.79, 1.01	-32.1	-66.2, 1.9	0.96	0.81, 1.12	-13.2	-60.2, 33.8
Men	0.95	0.89, 1.03	-23.6	-60.2, 13.1	1.00	0.92, 1.08	-0.1	-41.8, 41.6
Interaction	0.93	0.83, 1.06	-8.6	-49.7, 32.6	0.96	0.78, 1.17	-13.1	-84.0, 57.9
Despair mortality								
Women	0.65	0.48, 0.89	-6.4	-10.7, -2.0	0.86	0.68, 1.09	-2.3	-6.0, 1.3
Men	0.78	0.68, 0.89	-11.5	-18.1, -4.9	0.79	0.70, 0.89	-11.0	-16.8, -5.1
Interaction	0.84	0.66, 1.07	5.2	0.4, 10.0	1.10	0.89, 1.34	8.6	3.5, 13.8

Notes:

[a] Risk ratio (RR) and risk difference (RD, per 100,000 person-years) estimates calculated using log-linear Poisson models with state-level cluster-robust standard errors. Overall models included state and year fixed effects, while gender models included union density*gender interaction terms and gender-state and gender-year fixed effects. RD is average marginal effect of union density on mortality.

[b] Models weighted by gamma inverse probability of treatment weights.

Table 2.3. Relationship between 10% increase in 1-year lagged, 3-year moving average state-level union density and state-level mortality rates from 1986 to 2016 by gender-race and from 1989 to 2016 by gender-education.[a]

	Unweighted				Weighted[b]			
	RR	95% CI	RD	95% CI	RR	95% CI	RD	95 % CI
Gender-race								
All-cause mortality								
Women								
Black	1.00	0.95, 1.04	-1.0	-20.6, 18.6	0.99	0.95, 1.04	-2.4	-21.0, 16.3
White	0.82	0.73, 0.92	-53.0	-82.0, -23.9	0.93	0.82, 1.05	-20.0	-52.4, 12.3
Interaction	1.22	1.09, 1.37	52.0	20.6, 83.4	1.07	0.95, 1.21	17.6	-17.2, 52.5
Men								
Black	1.00	0.94, 1.07	3.5	-42.6, 49.5	1.00	0.92, 1.08	-1.5	-59.4, 56.4
White	0.92	0.87, 0.97	-39.0	-63.9, -14.0	0.95	0.90, 1.01	-22.6	-47.8, 2.6
Interaction	1.09	1.03, 1.16	42.4	3.2, 81.6	1.05	0.98, 1.13	21.1	-30.3, 72.6
Gender-education								
All-cause mortality								
Women								
≤HS	0.89	0.78, 1.02	-50.5	-109.0, 8.1	0.96	0.86, 1.07	-18.6	-68.9, 31.7
>HS	0.91	0.85, 0.98	-16.7	-29.4, -3.9	0.96	0.90, 1.03	-6.4	-18.7, 5.9
Interaction	0.98	0.90, 1.08	-33.8	-83.4, 15.8	1.00	0.90, 1.10	-12.2	-57.7, 33.4
Men								
≤HS	0.94	0.88, 1.01	-44.1	-99.6, 11.4	0.96	0.90, 1.03	-30.9	-85.8, 24.1
>HS	0.93	0.87, 1.00	-19.3	-39.4, 0.8	0.95	0.87, 1.03	-15.3	-39.8, 9.3
Interaction	1.01	0.92, 1.11	-24.8	-80.3, 30.7	1.01	0.93, 1.11	-15.6	-67.2, 36.0

Notes:

[a] Risk ratio (RR) and risk difference (RD, per 100,000 person-years) estimates calculated using log-linear Poisson models with state-level cluster-robust standard errors. Gender-race models included union density*race interaction terms and race-state and race-year fixed effects, while gender-education models included union density*education interaction terms and education-state and education-year fixed effects. RD is average marginal effect of union density on mortality.

[b] Models weighted by gamma inverse probability of treatment weights.

Table 2.4. Change in state-level all-cause mortality rates among Black people versus white people and the less-educated versus the more-educated if 1-year lagged, 3-year moving average union density increased from 2015 levels to 1985 or 1988 levels respectively.[a]

	Women				**Men**			
	RR	95% CI	RD	95% CI	RR	95% CI	RD	95 % CI
Race[b]								
Black	0.99	0.93, 1.06	-3.5	-30.7, 23.7	1.00	0.84, 1.17	-6.8	-127.6, 114.0
White	0.96	0.90, 1.03	-10.3	-26.8, 6.3	0.93	0.86, 1.01	-33.0	-69.5, 3.5
Ratio/difference	1.03	0.95, 1.12	6.8	-22.9, 36.4	1.07	0.92, 1.24	26.1	-82.5, 134.8
Education[c]								
≤HS	0.97	0.91, 1.05	-12.0	-44.7, 20.6	0.93	0.82, 1.06	-54.8	-150.7, 41.1
>HS	0.98	0.95, 1.02	-3.2	-9.3, 2.9	0.96	0.91, 1.02	-10.7	-28.0, 6.5
Ratio/difference	0.99	0.93, 1.06	-8.8	-38.9, 21.4	0.97	0.86, 1.09	-44.1	-135.3, 47.1

Notes:

[a] Risk ratio (RR) and risk difference (RD, per 100,000 person-years) estimates calculated using linear combinations of parameters from inverse probability of treatment weighted log-linear Poisson models with state-level cluster-robust standard errors. Gender-race models included union density*race interaction terms and race-state and race-year fixed effects, while gender-education models included union density*education interaction terms and education-state and education-year fixed effects. RD is average marginal effect of union density on mortality.

[b] Models run on outcome data from 1986-2016. Counterfactual union density set to 1985 levels.

[c] Models run on outcome data from 1989-2016. Counterfactual union density set to 1988 levels.

Chapter 3. DOES THE UNION MAKE US STRONG? LABOR UNION MEMBERHIP, SELF-RATED HEALTH, AND MENTAL ILLNESS: A PARAMETRIC G-FORMULA APPROACH

Introduction: Union members enjoy better wages and benefits than non-members, which can improve health. However, the longitudinal relationship between unionism and health remains uncertain, partially because of healthy-worker bias, a bias that cannot be addressed without high-quality data and methods that account for time-varying confounding affected by prior exposure and structural non-positivity. Applying one such method, the parametric g-formula, to Panel Study of Income Dynamics data, we analyzed the longitudinal relationships between union membership, poor/fair self-rated health (SRH), and moderate mental illness (Kessler K6 $\geq$ 5).

Methods: Our union membership and SRH analyses included 16,719 respondents followed between 1985 and 2017, while our union membership and mental-illness analyses included 5,813 respondents followed between 2001 and 2017; all respondents were employed at baseline. Using the parametric g-formula, we contrasted cumulative incidence of the outcomes under two hypothetical scenarios, one in which we set all employed-person-years to union-member employed-person-years (union scenario), and one in which we set no employed-person-years to union-member employed-person-years (non-union scenario). We also examined whether the scenario's effects varied by gender, gender-race, or gender-education using stratified models.

Results: Overall, the union scenario did not reduce incidence of poor/fair SRH (RR: 1.01, 95% CI: 0.95, 1.09; RD: 0.01, 95% CI: -0.03, 0.04) or moderate mental illness (RR: 1.02, 95% CI: 0.92, 1.12; RD: 0.01, 95% CI: -0.04, 0.06) relative to the non-union scenario. These effects largely did not vary by subgroup.

Conclusion: We found little evidence that our union scenario would reduce incidence of poor/fair SRH or mental illness.

3.1 INTRODUCTION

The labor movement has struggled with worker health and safety since the beginning of the industrial revolution. In the 19th century, Marx and Engels decried the toxic working and living conditions faced by the burgeoning English working class and invoked nascent working-class organizations like labor unions as the means to overcome the deleterious conditions.[88–90] Concurrently, U.S. workers created informal labor organizations and guilds to respond to wage cuts and deadly working conditions.[91,92] In the early-to-mid-20th century, many of these organizations formalized into unions after workers won legal rights to unionize and collectively bargain.[91,92] Unions have since advanced occupational health and safety in the U.S.,[8] as the American Public Health Association has formally recognized.[41,42] Unions helped pass the Occupational Safety and Health Act of 1970 (OSHA),[91,93] and despite their declining power, remain critical in illuminating the dangers posed by occupational hazards,[94] in enforcing OSHA regulations,[95,96] and in protecting workers from workplace harassment and discrimination.[7]

In prior U.S.-based ecological studies, researchers have identified protective associations between union density (the proportion of workers who belong unions) and rates of occupational fatalities,[32,47,48] fatal overdoses,[49,97] and suicide,[97] hypothesizing union density influences structural factors like regulatory regimes, social policies, and working-class power. However, although many individual-level U.S.-based studies have analyzed the relationship between union membership and occupational injuries, few have analyzed the relationship between union membership and non-occupational health outcomes.[39,51,98] For example, a study by Reynolds et al.[39] identified a cross-sectional association between union membership and better self-rated health (SRH) among male, less-educated, and lower-income workers, while another by

Waitzman[51] found union contract-coverage was associated with lower mortality risk among a cohort of men in the 1960s-1970s.

Limited prior research aside, several mechanisms may link union membership to better SRH and mental health (the outcomes under study). First, by bolstering workers' bargaining power with management, unionization may protect workers from material deprivation (e.g., inadequate wages and benefits),[30,39,99] occupational hazards (e.g., toxic chemicals),[7,8] and stressors (e.g., job instability, lack of autonomy, or discrimination),[7,39] lowering their risk of chronic diseases and occupational injuries (and their sequelae, like poor SRH), as well as their risk of mental illness.[43,100,101] Additionally, by building solidarity among workers, unions may lessen feelings of alienation and powerlessness, factors which can worsen SRH and mental health.[61,102] Union membership's health benefits may be strongest for less-educated and racialized workers. For example, studies have found the union wage premium – typically 15-20% – is largest for less-educated, Black, and lower-skilled workers, as is the benefit premium.[30] Union membership's health benefits may also differ by gender, given gender differences in exposure to workplace harassment and discrimination, as well as in occupation and industry.[29]

One barrier to studying the union-health relationship at the individual-level has been healthy-worker survivor bias, a bias that occurs when prior exposure (e.g., union membership) affects current employment status (a confounder), current employment status affects current or future exposure, and employment status independently affects the outcome. In this study, we hypothesized 1) prior union membership affected current employment status (because union membership may affect employment stability), 2) current employment status affected current union membership (because only the employed were eligible to be union members), and 3) current employment status affected current and future health (because being employed may improve health). Relationship 2) is a feature of union membership, while relationships 1) and 3) have been identified in prior studies that used our data source;[103,104] we also identified them in our sample (see Appendix B1 for details). Thus, employment-status confounded *and* mediated the union-health relationship, a setting in which standard covariate-adjustment approaches cannot consistently estimate total exposure effects.[105–108] However, the parametric g-formula – a generalization of standardization – can consistently estimate total exposure effects in such settings, as well as avoid bias in settings with structural non-positivity, essential for this analysis because one cannot be simultaneously unemployed and a union member.[105–108]

Applying the parametric g-formula to data from the Panel Study of Income Dynamics (PSID), we estimated the longitudinal relationships between union membership, SRH, and mental illness. Specifically, our goals were: 1) to estimate how a hypothetical scenario setting all (versus none) of respondents' two-year-lagged employed-person-years to union-member employed-person-years would have affected incidence of poor/fair SRH and moderate mental illness, and 2) to examine whether the scenarios' effects varied by gender, gender-race, and gender-education.

3.2 METHODS

3.2.1 *Data*

The PSID is a panel survey conducted by the University of Michigan's Survey Research Center.[109] In 1968, PSID enrolled a nationally-representative probability sample of U.S. families.[109] PSID interviewed respondents in these "core" families and subsequent "split-off" families (those who moved out of core families to form new, economically-independent families) annually from 1969-1997 and biennially thereafter. Since 1972, most interviews have been conducted via telephone.[109] Wave-to-wave response rates have averaged 94% since the survey's inception.[110] Although attrition is greatest among less-educated, lower-income, and racialized respondents, research has found little evidence of attrition bias in parameter estimates from health analyses.[111] PSID has collected socioeconomic and demographic data since 1968.[109] Beginning in 1984, PSID asked proxy and non-proxy respondents to rate their general health status.[109] From 2001-2017, save 2005, PSID administered to non-proxy respondents the Kessler-K6 (K6),[109] a six-question scale developed to estimate the prevalence of serious mental illness.[112]

Our SRH and mental-illness analyses used data on family "heads" and heads' "partners" (non-heads/partners did not have data on all variables of interest) from survey waves in odd years from 1985-2017 and 2001-2017 respectively; we treated the survey as biennial to be consistent with the survey's post-1997 structure. We excluded respondents in PSID's 1990-1995 "Latin[x] sample" because of their short follow-up and extensive missingness on several variables of interest, as well as respondents ever employed in "military" occupations or industries (1%) due to the lack of labor-union membership in that sector.

3.2.2 *Exposure*

Each study year, PSID asked respondents who were employed by someone other than themselves whether they were covered by a labor-union contract, and if so, whether they were members of the union providing the contract (80-90% of those covered). We used the labor-union membership variable as our exposure rather than the labor-union contract variable because membership more strongly correlates with health-promoting factors like high wages than contract-coverage; the reason for the discrepancy is uncertain.[113] We lagged union membership two-years prior to outcome measurement to reduce the likelihood of reverse causation and because unionization's health benefits may not accrue immediately.

3.2.3 *Health outcomes*

We dichotomized SRH—measured using the standard question ("Would you say your health in general is…")—as poor/fair versus good/very-good/excellent to improve the measure's reliability.[114] We also dichotomized K6 (range 0-24) as <5 versus ≥5, which reliably distinguishes those with/without moderate mental illness, defined as mental illness requiring treatment and causing impaired function.[112] We did not use a cutoff of 13 (to distinguish those with/without serious mental illness[112]) because there were only 476 events overall, including <75 in each male-gender-education and male-gender-race subgroup).

3.2.4 *Confounders*

Baseline covariates identified as potential confounders included respondents' age, race (Black/other/white, unless otherwise noted), gender (female/male), education (<HS/HS/some college/≥college, unless otherwise noted), census division of residence (see Table 3.1), childhood socioeconomic status (poor/average/well-off), disability status (whether respondents had a disability that limited the amount or type of work they could do), year, and study-time (years since baseline).

Time-varying covariates identified as potential confounders included respondents' marital status (married or cohabiting/not married or cohabiting), employment status (employed/not employed), occupation, and industry. Regarding occupations, PSID used 1970 census codes from 1985-2001, 2000 codes from 2003-2015, and 2010 codes in 2017; after crosswalking the codes to create a consistent time series,[115,116] we categorized the occupations into seven categories (see Table 3.1). Regarding industries, PSID used 1970 census codes from 1977-2001, 2000 codes from 2003-2015, and 2012 codes in 2017; after crosswalking,[117] we categorized the industries into nine categories (see Table 3.1).

3.2.5 *Sample*

For analyses of union membership and SRH, respondents ages 25-64 entered our sample at the first wave they were employed by someone other than themselves; we excluded those reporting the outcome at that wave, as well as those only observed for one wave. Respondents remained in our sample until their first incident outcome or their last wave of follow-up, whichever came first. For analyses of union membership and mental illness, respondents only had K6 measurements in certain waves because PSID did not administer the K6 in 2005, while in other waves, PSID only administered the K6 to non-proxy respondents; we assumed 2005 respondents and proxy respondents did not have K6 values $\geq$5. Respondents ages 25-64 entered our sample at the first wave they were employed by someone other than themselves *and* had a valid K6 measurement; we excluded those reporting the outcome at that wave. Respondents remained in the sample until their last K6 measurement or their first incident outcome, whichever came first. We excluded respondents with fewer than two K6 measurements during follow-up. For both outcomes, we excluded respondents missing any outcome data during follow-up, and censored respondents who missed a wave of follow-up at their last contiguous wave. See Appendix B2 for flow diagrams.

3.2.6 *Statistical analyses*

3.2.6.1 Primary analyses

We conducted our parametric g-formula analyses using R's "gfoRmula" package; Appendix B3 contains code for implementing our approach.[118] The parametric g-formula can consistently estimate the mean potential outcome under a hypothetical exposure scenario, given assumptions of: 1) no unmeasured confounding, 2) counterfactual consistency (respondents' counterfactual outcomes under their observed exposure histories equal their observed outcomes), 3) no model misspecification, 4) no interference (respondents' potential outcomes do not depend upon other respondents' exposures), and 5) positivity (no exposure scenarios that require respondents be exposed within strata of a confounder in which the probability of exposure is zero).[106,107,119] The incidence of the outcome in each scenario is the weighted sum over the various exposure and covariate histories of the probability of the outcome conditional on exposure and covariates.[105] First, using the observed data, we fit pooled parametric models for time-varying exposure (lagged two-years), time-varying confounders (lagged two-years), and the outcome of interest, using logistic models for binary variables and multinomial logistic models for categorical variables.[120] In each wave, we assumed the following temporal ordering of time-varying exposure and confounders: 1) marital status, 2) employment status, 3) occupation, 4) industry, and 5) union membership (Figure 1). Time-varying variables at wave t_k were functions of baseline confounders, prior time-varying variables in t_k (if any), time-varying variables in t_{k-1}, follow-up time, and year. In all models, we specified categorical covariates as described in the "*Confounders"* sub-section and age as a 3-knot restricted cubic spline (via R's rms[121] package) to allow for nonlinear age-outcome relationships.[122] In most models, we specified follow-up time as a fixed effect, although we specified it as a restricted cubic spline in selected analyses to improve fit; year's specification varied more. See Appendix B4 for details.

Next, after fitting the parametric models, we randomly drew respondents with replacement from the original data to create a Monte Carlo pseudo-sample of 25,000.[120] We drew a sample size larger than the original cohort to minimize simulation error;[123] using an even larger sample size was not computationally feasible. Using the baseline observations of the pseudo-sample, we predicted observations in the second wave using parameters from the pooled models described in step one.[120] We then used predicted observations in the second wave to predict observations in the third wave, and so on, until the end of follow-up or the outcome of interest, whichever came first.[120] In the "natural course",[120] we left union-membership as predicted by the parametric models. In our first scenario, we set two-year-lagged union membership to "union" whenever respondents were employed (union scenario), while in our second scenario, we set two-year-lagged union membership to "non-union" whenever respondents were employed (non-union scenario). These scenarios avoided bias from non-positivity by only requiring workers to be eligible for union membership when employed.[119,124] In all scenarios, we eliminated censoring from administrative causes, loss to follow-up, and competing events (death) by simulating respondents' outcomes beyond the waves in which they were observed censored.[120] We assumed censoring was random within levels of measured confounders.[120]

Finally, using the simulations described above, we estimated the cumulative incidence of each outcome under each scenario through the end of follow-up (32 years for SRH and 16 years for mental illness).[120] We calculated risk ratios (RRs) and risk differences (RDs) for our contrast of interest (union versus non-union) by comparing the cumulative incidence in scenario one with the cumulative incidence in scenario two.[120] To calculate confidence intervals for the RRs and RDs, we repeated the g-formula algorithm on 250 bootstrap samples and based the lower and upper confidence-interval bounds on percentiles of the bootstrap distribution.[120] We tested for potential model misspecification by comparing the simulated variable distributions at each timepoint in the natural course with those in the observed data.[105–107,120]

3.2.6.2 Secondary analyses

In secondary analyses, we examined whether the effect estimates varied by gender (women/men), gender-race (person-of color/white, with most of the former composed of Black respondents), and gender-education ($\leq$HS/>HS) by running the approach outlined above in each subgroup.

3.2.6.3 Sensitivity analyses

First, to test the sensitivity of our results to the exposure lag, we used an unlagged exposure. Second, instead of treating death as a censoring event and estimating cause-specific risks of the outcomes of interest, we allowed for deaths during follow-up and estimated subdistribution risks, which may correspond more closely to the risks that would be observed in real-world scenarios;[125] see Appendix B7 for details. Third, due to difficulties accurately modeling time-varying occupation and industry, we examined whether treating the variables as time-invariant throughout the analyses affected our results. Fourth, we probed for residual geographic confounding by including baseline state of residence rather than baseline division of residence as a covariate in the exposure, time-varying confounder, and outcome models. We did not run this specification for the subgroup analyses because there were too few respondents in many states. Finally, to compare our g-formula estimates to estimates from a traditional approach, we ran confounder-adjusted Cox models using R's survival package.[126] These models had baseline union membership as the exposure, baseline confounders as covariates (with age and year as restricted cubic splines), and incident SRH or K6 as outcomes. As in the g-formula analyses, all respondents were employed at baseline.

3.2.7 *Missing data*

Our exposure, outcome, and confounders contained a small amount of missingness ($\leq$4%). To address missingness in baseline confounders, we carried respondents' observed values forwards (and backwards if necessary) when possible. To address remaining missingness in the confounders and exposure, we performed a single multivariate imputation by chained equations with 25 iterations using R's "mice" package.[127] The imputation models included as predictors all baseline confounders, time-varying exposure and confounders in t_k and t_{k-1} (or t_{k+1} in respondents' baseline wave), and time-varying SRH in t_k and t_{k-1} (or t_{k+1} in respondents' baseline wave). We did not use imputed SRH or K6 values in the outcome analyses, nor did we create multiple imputed datasets, as doing so in a parametric g-formula setting was not computationally feasible and methods for pooling estimates after imputation in such a setting have not been well-developed. Estimates from complete-case analyses were similar.

3.3 RESULTS

3.3.1 *Descriptives*

The SRH analyses used data on 16,719 respondents with 3,878 events and 87,422 observations, while the mental-illness analyses used data on 5,813 respondents with 1,981 events and 20,920 observations. At baseline in the SRH analyses, 16% of respondents were union workers (Table 3.1). Compared with non-union workers, union workers were more likely to be older, less-educated, persons of color, men, married/cohabiting, living outside the South, and to have grown up poor. Moreover, union workers more often worked in "operator, fabricator, and laborer" and "precision production, craft, and repair" occupations, as well as in "manufacturing" and "transportation, communications, and other public utilities" industries. Finally, union workers' median family incomes were 21% higher than non-union workers' median family incomes. Appendix B5 shows trends in union membership over the study period by demographic group, occupation, and industry.

3.3.2 *Union membership, self-rated health, and mental illness*

Overall, the cumulative incidence of poor/fair SRH by the end of follow-up in the natural course was 47% (Figure 3.2); the corresponding figure for moderate mental illness was 45% (Figure 3.3). Across subgroups, incidences of the outcomes were greater among women, people of color, and the less-educated than among men, white people, and the more-educated (Tables 2 and 3), although racial inequities were smaller for mental illness than for SRH. In all analyses, the simulated cumulative incidence in the natural course aligned with the observed cumulative incidence, as did the simulated distribution of union membership (Figures 2 and 3 and Appendices B12 and B13). However, although the simulated distribution of employment status aligned with the observed distribution, the simulated distributions of other time-varying confounders tended to differ from the observed distributions more considerably, particularly occupation and industry (Appendices B12 and B13).

In the SRH analyses in the full sample, approximately 9% of person-years in the union scenario were spent not employed, lower than the 12% in the non-union scenario. However, the union scenario did not meaningfully affect incidence of poor/fair SRH relative to the non-union scenario (RR: 1.01, 95% CI: 0.95, 1.09; RD: 0.01, 95% CI: -0.03, 0.04) (Table 3.2). This null effect largely remained across subgroups, although the union scenario was somewhat protective for men (RR: 0.94, 95% CI: 0.87, 1.02; RD: -0.03, 95% CI: -0.06, 0.01), particularly men of color (RR: 0.90, 95% CI: 0.79, 1.00; RD: -0.06, 95% CI: -0.12, 0.00), and somewhat harmful for women (RR: 1.10, 95% CI: 1.00, 1.19; RD: 0.05, 95% CI: 0.00, 0.08), particularly less-educated women (RR: 1.17, 95% CI: 1.05, 1.28; RD: 0.09, 95% CI: 0.03, 0.15).

In the mental-illness analyses in the full sample, approximately 7% of person-years in the union scenario were spent not employed, lower than the 8% in the non-union scenario. However, the union scenario did not meaningfully affect incidence of moderate mental illness relative to the non-union scenario (RR: 1.02, 95% CI: 0.92, 1.12; RD: 0.01, 95% CI: -0.04, 0.06) (Table 3.3). This null effect largely remained across subgroups, although the union scenario was somewhat protective for women of color (RR: 0.90, 95% CI: 0.74, 1.06; RD: -0.05, 95% CI: -0.15, 0.03), men of color (RR: 0.89, 95% CI: 0.66, 1.15; RD: -0.06, 95% CI: -0.17, 0.07), and more-educated men (RR: 0.90, 95% CI: 0.70, 1.11; RD: -0.04, 95% CI: -0.13, 0.05) and somewhat harmful for white women (RR: 1.17, 95% CI: 1.00, 1.37; RD: 0.08, 95% CI: 0.00, 0.18), less-educated women (RR: 1.10, 95% CI: 0.90, 1.31; RD: 0.05, 95% CI: -0.05, 0.16), and less-educated men (RR: 1.11, 95% CI: 0.89, 1.34; RD: 0.05, 95% CI: -0.05, 0.15).

3.3.3 *Sensitivity analyses*

Using an unlagged exposure did not meaningfully affect our estimates (Appendix B6). Estimating subdistribution risks (Appendix B7), treating occupation and industry as baseline variables (Appendix B8), and including state of residence (rather than division) as a covariate (Appendix B9) did not meaningfully affect our estimates either. However, although using confounder-adjusted Cox models did not meaningfully affect most of our estimates, union membership appeared somewhat more harmful for mental illness in certain subgroups (Appendix B10).

3.4 DISCUSSION

3.4.1 *Summary of results and comparison with prior research*

Using a parametric g-formula approach, we estimated how a hypothetical scenario setting all (versus none) of respondents' two-year-lagged employed person-years to union-member employed person-years would have affected incidence of poor/fair SRH and moderate mental illness in a sample of working-age adults with labor-force attachment. Contrary to our expectations, we found little evidence the scenario would have reduced incidence of the outcomes in the full sample. Moreover, although we also found evidence of larger benefits among certain marginalized subgroups, such as on SRH among men of color and on mental health among men and women color, we also had contradictory findings, such as potential harms on SRH among women of color and less-educated women.

To our knowledge, the Reynolds et al.[39] study is the only prior U.S.-based study of this age group on union membership and SRH. That study identified a modest protective association between union membership and SRH, particularly among male, less-educated, and lower-income workers. Nonetheless, the study is not directly comparable to ours given its cross-sectional design. We have not identified any prior U.S.-based studies of this age group on union membership and mental illness.

Our modest findings may be because union membership's salutary effects on working conditions, wages, and benefits[7,8,30,39] are too weak to measurably improve these health outcomes, particularly given diminishing union power over the study period.[24] Although the union wage premium remained largely unchanged,[30] certain union hierarchies grew detached from their membership,[24] suggesting unionism's solidarity-promoting and alienation-reducing effects may have weakened.

Our modest findings may also be due to bias. First, union membership may be misclassified. In a 1996 study, Card found 2.5% of respondents in the 1977 Current Population Survey misreported their union status, true union status aside.[85] At the average union-membership prevalence observed in our SRH analyses (14%), Card's misclassification rate would mean approximately 16% of workers classified as union were actually non-union (Appendix B11), causing bias towards the null. To our knowledge, there is no recent research on the accuracy of PSID's union-membership data. Second, prior research suggests pre-existing firm-level characteristics, like hazardous working conditions, may cause workers to unionize; this may partially explain why certain quantitative studies – conflicting historical and anecdotal evidence – have found unionization *increases* occupational injury risk.[50] Although we adjusted for respondent occupation and industry, we did not have access to firm-level data. Thus, unmeasured confounding by firm-level factors may have caused an underestimate of unionism's protective effects. Finally, respondents with similar objective health statuses may have differentially assessed their SRH and mental health depending on their union status. For example, respondents often compare themselves to peers when evaluating their health.[128] In our study, if these peers included respondents' coworkers (who were likely union-member coworkers for union-member respondents), union membership might not appear to improve health, effect of union membership on objective health status aside. Although this bias is unlikely to be severe, given that prior studies have identified substantial racial and SES disparities in these outcomes, it may have contributed to our findings.[129]

3.4.2 *Strengths and limitations*

Our study's primary strengths included first, a large sample with extensive follow-up and confounder data. To our knowledge, our study is the first since Waitzman's 1988 study, which only included men, to analyze the union-health relationship longitudinally among this age group. Second, few prior social epidemiological studies have used parametric g-formula approaches. Our study demonstrated the benefits of such an approach, including flexible estimation of scenario contrasts, as well as the drawbacks, including computational intensiveness (our SRH analyses took eight days to run in parallel on a Dell PowerEdge M620 Blade server with 16 2.00GHz Intel Xeon CPUs and 192GB of RAM). Finally, our parametric g-formula approach addressed potential bias from healthy-worker survivor effects and other forms of time-varying confounding. Moreover, unlike other approaches often used to address such biases, like marginal structural modeling, our approach allowed us to avoid bias from non-positivity by only requiring respondents to be eligible for union membership when employed.[119,124]

In addition to misclassification and firm-level confounding, our study limitations included potential violation of the no-model-misspecification assumption Although the simulated exposure and outcome distributions in the natural course resembled the observed distributions in most analyses, the simulated distributions of certain time-varying covariates – particularly occupation and industry – tended to differ from the observed distributions more considerably. Nonetheless, we do not think residual confounding by time-varying covariates had an undue influence given that: 1) we accurately modelled employment status, our most important confounder, and 2) our results were consistent across many modeling specifications. Moreover, traditional covariate-adjusted Cox models yielded similar estimates to our g-formula analyses, suggesting our null findings were not an artefact of our approach. Additional limitations include potential violations of the no-interference and consistency assumptions. Regarding interference, research suggests unions may improve health-related factors (e.g., wages) not only among union workers, but also among non-union workers by improving prevailing standards in non-union workplaces in similar regions and sectors.[5] Such spillovers could bias estimates of union-membership's health effects towards the null by making union and non-union workers appear spuriously similar. Such spillovers could be especially likely in this study because PSID recruited respondents through familial networks. Nonetheless, spillovers are unlikely to be strong given the small direct effects of union membership we identified. Regarding consistency, we assumed union-membership's effects did not vary by region, sector, or year, a strong assumption given the variability in union types (e.g., militant versus conservative) and declining union power over follow-up.[130] This heterogeneity would violate consistency, although our subgroup stratification may have proxied for sector and region. Moreover, the union-wage-premium's consistency over the last few decades suggests temporal changes in union-membership’s effects are modest.[30]

3.4.3 *Future directions*

Given union membership’s roles in wages, benefits, and occupational safety, our largely null findings raise questions that should be pursued in future research. For one, researchers could consider additional health outcomes, including mortality, which may be more reliable than SRH and mental illness. Researchers could also consider how area-level union density and other area-level labor-related factors, like right-to-work laws, interact with individual-level union membership to affect health.

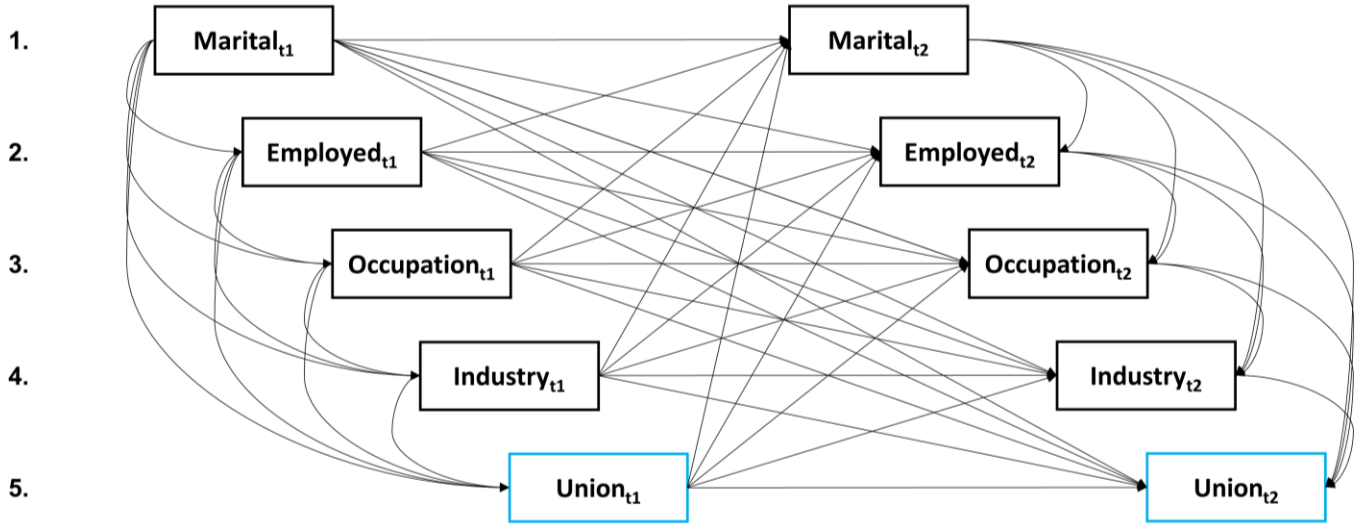

Figure 3.1. Hypothesized temporal ordering of time-varying variables. In parametric g-formula analyses, time-varying variables at wave t_k were functions of baseline confounders, prior time-varying variables in t_k (if any), time-varying variables in t_{k-1}, year, and follow-up time.

Table 3.1. Descriptive statistics of Panel Study of Income Dynamics sample at baseline in self-rated-health analyses stratified by two-year lagged union membership.

		Non-union	Union
N		14459	2260
Age (median [1st quartile, 3rd quartile])		29 [26, 37]	32 [27, 40]
Male (%)		6838 (47.3)	1356 (60.0)
Race (%)			
	Black	4300 (29.7)	798 (35.3)
	Other	1101 (7.6)	158 (7.0)
	White	9058 (62.6)	1304 (57.7)
Education (%)			
	<HS	2158 (14.9)	349 (15.4)
	HS	4568 (31.6)	882 (39.0)
	Some college	3900 (27.0)	546 (24.2)
	College+	3833 (26.5)	483 (21.4)
Married/permanently-cohabiting (%)		10484 (72.5)	1729 (76.5)
Childhood socioeconomic status (%)[a]			
	Poor	3829 (26.5)	726 (32.1)
	Average	6499 (44.9)	965 (42.7)
	Well-off	4131 (28.6)	569 (25.2)
Occupation (%)			
	Farming, forestry, and fishing	170 (1.2)	3 (0.1)
	Managerial	1410 (9.8)	60 (2.7)
	Operators, fabricators, and laborers	2039 (14.1)	669 (29.6)
	Professional specialty	2537 (17.5)	391 (17.3)
	Precision production, craft, and repair	1364 (9.4)	350 (15.5)
	Services	2529 (17.5)	349 (15.4)
	Technical, sales, and admin support	4410 (30.5)	438 (19.4)
Industry (%)			
	Agriculture, forestry, and fisheries	299 (2.1)	11 (0.5)
	Construction	766 (5.3)	132 (5.8)
	Finance, insurance, and real estate	991 (6.9)	24 (1.1)
	Manufacturing	2360 (16.3)	571 (25.3)
	Mining	97 (0.7)	11 (0.5)
	Public administration	696 (4.8)	213 (9.4)
	Services	5431 (37.6)	686 (30.4)
	Transport, communications, and other public utilities	918 (6.3)	433 (19.2)
	Wholesale and retail trade	2901 (20.1)	179 (7.9)
Division of residence (%)			
	East North Central	2200 (15.2)	488 (21.6)
	East South Central	1195 (8.3)	124 (5.5)
	Middle Atlantic	1471 (10.2)	447 (19.8)
	Mountain	793 (5.5)	55 (2.4)
	New England	488 (3.4)	83 (3.7)
	Pacific	1795 (12.4)	397 (17.6)
	South Atlantic	3615 (25.0)	360 (15.9)
	West North Central	1257 (8.7)	176 (7.8)
	West South Central	1645 (11.4)	130 (5.8)
Work disability (%)[b]		819 (5.7)	116 (5.1)
Family income (median [1st quartile, 3rd quartile])[c]		6.1 [3.7, 9.2]	7.4 [4.9, 10.3]

Notes:
[a] Childhood socioeconomic status when respondent was growing up.
[b] Respondent had disability that limited the type or amount of work they could do.
[c] Tens of thousands of family income in 2017 dollars.

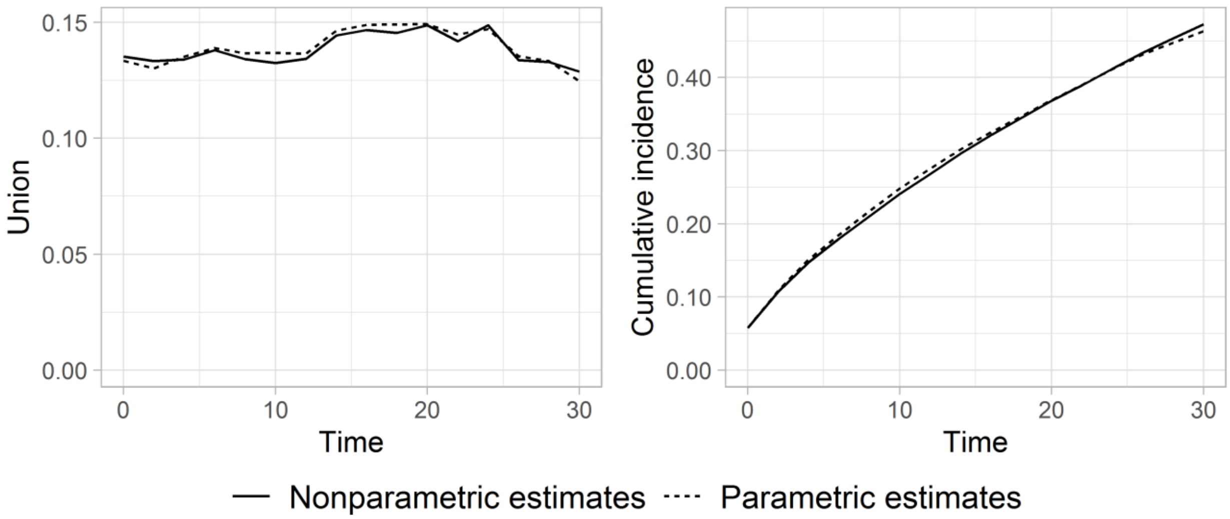

Figure 3.2. Simulated (parametric) probability of two-year-lagged union membership and cumulative incidence of poor/fair SRH during follow-up in the natural course compared with the observed (nonparametric) values.

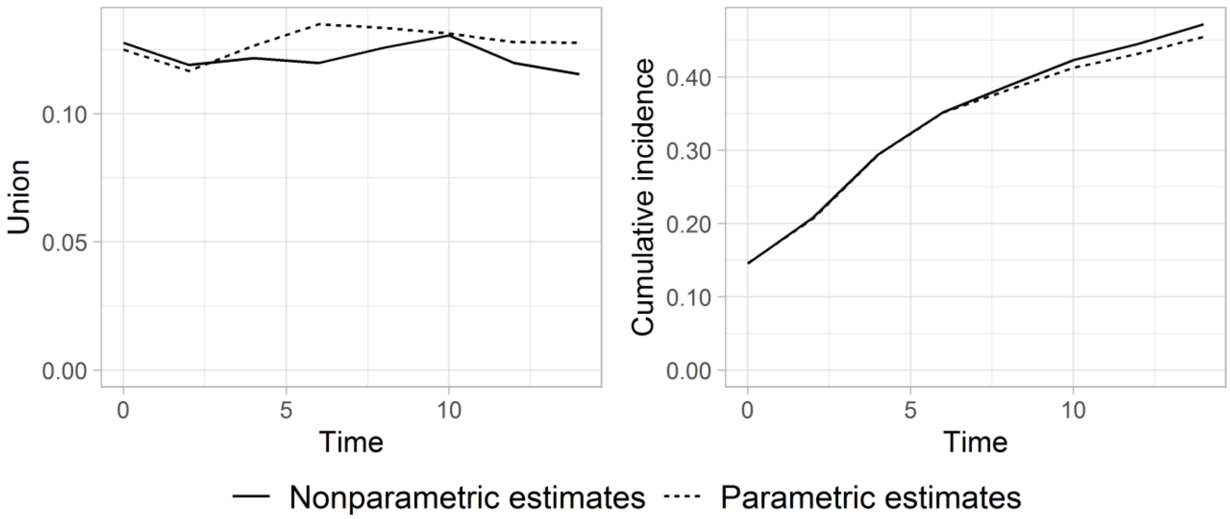

Figure 3.3. Simulated (parametric) probability of two-year-lagged union membership and cumulative incidence of moderate mental illness during follow-up in the natural course compared with the observed (nonparametric) values.

Table 3.2. Parametric g-formula estimates of 32-year risk (i.e., cumulative incidence) of poor/fair SRH in scenario (sc.) setting all two-year-lagged employed-person-years to union-member employed-person-years (scenario 1) relative to risk in scenario setting no two-year-lagged employed-person-years to union-member employed-person-years (scenario 2).

	Respondents[a]	Observations[a]	Sc.	Risk	RR	95% CI		RD	95% CI	
Overall	16,719	87,422	1	0.47	1.01	0.95	1.09	0.01	-0.03	0.04
			2	0.46						
Gender										
Women	8,525	45,288	1	0.50	1.10	1.00	1.19	0.05	0.00	0.08
			2	0.46						
Men	8,194	42,134	1	0.45	0.94	0.87	1.02	-0.03	-0.06	0.01
			2	0.48						
Gender-race										
Women of color[b]	3,351	15,557	1	0.63	1.10	0.99	1.20	0.06	-0.01	0.12
			2	0.58						
White women[c]	5,142	29,603	1	0.42	1.09	0.96	1.25	0.03	-0.02	0.09
			2	0.38						
Men of color	3,000	12,609	1	0.56	0.90	0.79	1.00	-0.06	-0.12	0.00
			2	0.62						
White men	5,194	29,525	1	0.38	0.98	0.86	1.11	-0.01	-0.05	0.04
			2	0.39						
Gender-education										
≤HS women[d]	3,903	19,586	1	0.64	1.17	1.05	1.28	0.09	0.03	0.15
			2	0.55						
>HS women[e]	4,563	25,249	1	0.39	1.07	0.90	1.23	0.02	-0.04	0.08
			2	0.36						
≤HS men	4,040	18,604	1	0.56	0.95	0.86	1.04	-0.03	-0.08	0.02
			2	0.60						
>HS men	4,154	23,530	1	0.32	0.96	0.80	1.14	-0.01	-0.07	0.04
			2	0.33						

Notes:
Risk ratio and risk difference estimates compare risk in scenario 1 relative to risk in scenario 2. Confidence intervals calculated from non-parametric bootstrap with 250 repetitions. Subgroup estimates produced from stratified models.

[a] Unique respondents and observations in original Panel Study of Income Dynamics dataset used to fit pooled time-varying exposure, confounder, and outcome models. Monte Carlo pseudo-sample used in simulations had 25,000 respondents.

[b] Analysis excluded 6 respondents ever employed in "mining" industry due to small counts in that stratum, which produced bootstrap samples with 0 mining-industry respondents.

[c] Analysis excluded 26 respondents ever employed in "mining" industry.

[d] Analysis excluded 14 respondents ever employed in "mining" industry.

[e] Analysis excluded 43 respondents ever employed in "mining" industry or "farming, forestry, and fishing' occupation.

Table 3.3. Parametric g-formula estimates of 16-year risk (i.e., cumulative incidence) of moderate mental illness (K6≥5) in scenario (sc.) setting all two-year-lagged employed-person-years to union-member employed-person-years (scenario 1) relative to risk in scenario setting no two-year-lagged employed-person-years to union-member employed-person-years (scenario 2).

	Respondents[a]	Observations[a]	Sc.	Risk	RR	95% CI		RD	95% CI	
Overall	5,813	20,920	1	0.46	1.02	0.91	1.12	0.01	-0.04	0.06
			2	0.45						
Gender										
Women	3,376	12,185	1	0.54	1.04	0.91	1.19	0.02	-0.04	0.10
			2	0.51						
Men	2,437	8,735	1	0.42	1.01	0.86	1.19	0.00	-0.06	0.08
			2	0.42						
Gender-race										
Women of color[b]	1,528	5,415	1	0.49	0.90	0.74	1.06	-0.05	-0.15	0.03
			2	0.55						
White women[c]	1,832	6,711	1	0.58	1.17	1.00	1.37	0.08	0.00	0.18
			2	0.49						
Men of color[d]	881	2,892	1	0.46	0.89	0.66	1.15	-0.06	-0.17	0.07
			2	0.52						
White men	1,553	5,835	1	0.44	1.07	0.85	1.27	0.03	-0.06	0.11
			2	0.41						
Gender-education										
≤HS women	1,340	4,777	1	0.58	1.10	0.90	1.31	0.05	-0.05	0.16
			2	0.53						
>HS women[e]	2,027	7,378	1	0.46	1.01	0.82	1.19	0.00	-0.08	0.08
			2	0.45						
≤HS men	979	3,323	1	0.54	1.11	0.89	1.34	0.05	-0.05	0.15
			2	0.49						
>HS men[f]	1,447	5,367	1	0.39	0.90	0.70	1.11	-0.04	-0.13	0.05
			2	0.43						

Notes:

Risk ratio and risk difference estimates compare risk in scenario 1 relative to risk in scenario 2. Confidence intervals calculated from non-parametric bootstrap with 250 repetitions. Subgroup estimates produced from stratified models.

[a] Unique respondents and observations in original Panel Study of Income Dynamics dataset used to fit pooled time-varying exposure, confounder, and outcome models. Monte Carlo pseudo-sample used in simulations had 25,000 respondents.

[b] Analysis excluded 2 respondents ever employed in "mining" industry due to small counts in that stratum, which produced bootstrap samples with 0 mining-industry respondents.

[c] Analysis excluded 14 respondents ever employed in "mining" industry or "farming, forestry, and fishing" occupation.

[d] Analysis excluded 3 respondents ever employed in "mining" industry.

[e] Analysis excluded 9 respondents ever employed in "mining" industry or "farming, forestry, and fishing" occupation.

[f] Analysis excluded 11 respondents ever employed in "farming, forestry, and fishing" occupation.

Chapter 4. UNION BURYING GROUND: SINKING LABOR UNION MEMBERSHIP AND CHANGING RACIAL AND EDUCATIONAL MORTALITY INEQUITIES IN THE UNITED STATES

Objective: Over the last several decades in the U.S., socioeconomic inequities in life expectancy have increased 1-2 years. Declining labor-union density has fueled growing class inequalities in income and exacerbated racial income inequalities. Using Panel Study of Income Dynamics (PSID) data, we examined the longitudinal union-mortality relationship and estimated whether declining union density has also exacerbated racial and educational mortality inequities.

Methods: Our sample included respondents ages 25-66 to the 1979-2015 PSID with mortality follow-up through 2017. To address healthy-worker survivor bias, we used the parametric g-formula. First, we estimated how a hypothetical scenario setting all (versus none) of respondents' employed-person-years to union-member employed-person-years would affect mortality incidence. Next, we examined whether the scenarios' effects varied by gender, race, and education. Finally, we estimated how racial and educational mortality inequities would change if union-membership prevalence had remained at 1979 levels throughout follow-up rather than at 2015 levels.

Results: In the full sample (respondents=23,022, observations=146,681), mortality was lower in the union scenario than in the non-union scenario (RR: 0.90, 95% CI: 0.80, 0.99; RD per 1,000: -18.7, 95% CI: -36.5, -0.9). This effect generally held across subgroups, although it was stronger among the more-educated. However, we found little evidence racial and educational mortality inequities would lessen if union membership had remained at 1979 levels.

Conclusion: To our knowledge, this was the first individual-level U.S.-based study with repeated union-membership measurements to analyze the union-mortality relationship. We estimated that increasing union membership may reduce mortality but would have little effect on mortality inequities.

4.1 INTRODUCTION

Socioeconomic inequities in U.S. life expectancy have grown 1-2 years since the 1980s.[1] Declining life expectancy among the poor and less-educated has fueled the growing inequities[1] and contributed to an overall decline in U.S. life expectancy annually from 2014-2017.[2] Meanwhile, racial inequities in life expectancy remain considerable despite narrowing over the last several decades; in 2015, the white-Black life expectancy disparity was 4.1 years among men and 2.6 years among women.[15] Class inequities in income have grown alongside the mortality inequities. For example, average pretax earnings of the top 1% of Americans increased from 27 times greater than the bottom 50%'s in 1980 to 81 times greater in 2015.[3] Additionally, racial inequities in income have remained largely unchanged since the 1960s. In 1968, the median Black family income was 57% of the median white family income; in 2016, the corresponding figure was 56%.[11]

Declining labor union density – the proportion of wage and salary workers belonging to labor unions – has exacerbated the income inequities.[30] From 1983 to 2017, union density decreased from 23.5% to 11.8% among 25-to-66-year-old workers, including from 31% to 13% among Black workers (compared with 22.5% to 12.2% among white workers) and from 26.7% to 10.4% among workers with a high-school (HS) degree or less (compared with 19.8% to 12.3% among those with some college or more).[22] Low union density undermines worker power over wages, including the wages of workers in non-unionized workplaces, and undermines union organizing for redistributive policies, like progressive taxation.[5,30,44] One study estimated declining union density from 1973-2007 explained a third of the rise in overall wage inequity among men and a fifth of the rise among women,[5] while another estimated that Black-white wage inequity in 2007 would have been 3%-10% lower among men and 13%-30% lower among women if union density had remained at 1973 levels.[29] Similar effects have been found in more recent research.[30,60]

Unionization may also reduce mortality and mortality inequities. For example, by augmenting worker power, unionization may allow workers to demand better wages[30] and benefits from their employers,[99] as well as stronger protections from occupational hazards,[7,8,96] layoffs,[103] and discrimination,[7] lowering their risk of fatal chronic diseases, occupational injuries, and mental illnesses.[43] Moreover, by promoting solidarity among workers, unionization may protect workers from feelings of alienation and powerlessness, reducing their risk of mental illnesses, drug use, and their sequelae, like suicide and fatal overdose.[49,61] Union membership's salutary effects may be greatest for Black and less-educated workers, a finding of prior studies on the union wage and benefit premium.[30]

Despite potential mechanisms linking unionization, mortality, and mortality inequities, few U.S.-based studies have examined the relationship empirically (or the relationship with other non-occupational health outcomes); those that have produced mixed findings. For example, although several ecological-level studies estimated protective associations between union density and occupational fatalities,[32,47,48] fatal overdoses,[49,97] and suicides,[97] another found no association between union density, all-cause mortality, and all-cause mortality inequities.[97] Individual-level studies have produced similarly mixed findings. For example, although a study by Waitzman[51] estimated a modest protective association between union contract-coverage and mortality risk among a male cohort in the 1960s-1970, a recent longitudinal study by Eisenberg-Guyot et al. found no association between union membership and SRH or mental illness.[131] Given the limited and contradictory prior research, the individual-level relationship between union membership, mortality, and mortality inequities remains uncertain.

Using Panel Study of Income Dynamics (PSID) data, we examined the longitudinal relationships between union membership, mortality, and racial and educational mortality inequities. Our specific aims were to: 1) estimate how a hypothetical scenario setting all (versus none) of respondents' employed-person-years to union-member employed-person-years would affect cumulative incidence of mortality, 2) examine whether the scenarios' effects varied by gender, race, and education, and 3) estimate how racial and educational mortality inequities would change if union-membership prevalence across racial and educational groups had remained at 1979 levels throughout follow-up rather than at 2015 levels.

4.2 Methods

4.2.1 *Data and sample*

The University of Michigan's Survey Research Center runs the PSID, which enrolled a nationally-representative probability sample of U.S. families in 1968.[109] PSID interviewed these "core" families and subsequent "split-off" families (families formed by persons who left core families to form new, economically-independent families) annually from 1969-1997 and biennially thereafter; since 1972, most interviews have been over telephone.[109]

Our analyses used data on family "heads" and heads' "partners" ages 25-66 from survey waves in odd years from 1979-2015 with mortality follow-up through 2017; we treated the survey as biennial to be consistent with the survey's post-1997 structure. Heads and partners entered our sample at the first wave they were employed by someone other than themselves and remained in our sample until death or their last wave of follow-up, whichever came first. We censored respondents who missed a wave of follow-up at their last contiguous wave. We excluded non-heads/non-partners because they did not have data on all variables of interest. We also excluded respondents in PSID's 1990-1995 "Latin[x] sample" because of their short follow-up and extensive missingness on several variables of interest, as well as respondents ever employed in "military" occupations or industries (1%) due to the lack of labor-union membership in that sector.

4.2.2 *Exposure*

Each wave from 1979-2015, PSID asked non-self-employed respondents whether they were covered by a labor-union contract (i.e., a collective bargaining agreement), and if so, whether they were members of the union providing the contract (80-90% of those covered). We used union membership as the exposure rather than union-contract coverage because membership more strongly correlates with health-promoting factors like high wages than contract-coverage.[113]

4.2.3 *Outcome*

Our outcome was all-cause mortality, a variable available for all respondents in PSID's restricted-use mortality file.[109] We assigned deaths occurring within two years of a survey wave to that survey wave (e.g., for respondents interviewed in 1985, we assigned 1985 and 1986 deaths to the 1985 wave, as well as 1987 deaths that occurred before the 1987 wave). In most instances, surviving household members reported death information about deceased respondents to survey administrators at the next survey wave.[132] For deceased respondents without surviving household members, death information came from several sources, including surviving non-household contact persons, administrators of decedents' estates, or the post office.[132] For 98.4% of deaths within our years and ages of interest, PSID provided the precise death year. For 1.2% of deaths, PSID provided a one- or two-year range for the death year (e.g., 1982-1983 or 1982-1984); for these deaths, we assigned the death year to the range's latter year. We excluded respondents associated with the remaining 0.4% of deaths, which were only known to have occurred within a three-plus-year range.

4.2.4 *Confounders*

Baseline (i.e., time-invariant) covariates identified as potential confounders included respondents' gender (female/male), race (Black/other/white, unless otherwise noted), age, education (<HS/HS/some college/≥college, unless otherwise noted), census region of residence (Midwest/Northeast/South/West), childhood socioeconomic status (poor/average/well-off), disability status (whether respondents had a disability that limited the amount or type of work they could do), year, and follow-up time (years since baseline).

Time-varying covariates identified as potential confounders included respondents' marital status (married or cohabiting/not married or cohabiting), employment status (employed/not employed), occupation, and industry. Regarding occupations, PSID used 1970 census codes from 1985-2001 and 2000 codes from 2003-2015; we categorized the occupations into seven categories (see Table 4.1) after crosswalking the codes to make them consistent across waves.[115,116] Regarding industries, PSID used 1970 census codes from 1977-2001 and 2000 codes from 2003-2015; we categorized the industries into nine categories (see Table 4.1) after crosswalking.[117]

4.2.5 *Statistical analyses*

4.2.5.1 Primary and subgroup analyses

We hypothesized that prior union membership affected current employment status (because union membership may increase employment stability[103,131]) and that current employment status affected current union membership (because only the employed were eligible can be union members) and future mortality (because being employed may improve health[104]) (see diagram in Appendix C1), a confounding structure that could cause healthy-worker survivor bias. In such a setting, standard covariate-adjustment approaches cannot consistently estimate mean potential outcomes under hypothetical exposure scenarios because: 1) employment-status confounds *and* mediates the union-health relationship, and 2) only the employed are eligible to be union members, creating structural non-positivity.[105–108] Nonetheless, parametric-g-formula approaches, which generalize standardization to settings with time-varying exposure-confounder feedback, can consistently estimate mean potential outcomes in such a setting, assuming: 1) no unmeasured confounding, 2) counterfactual consistency (respondents' counterfactual outcomes under their observed exposure histories equal their observed outcomes), 3) no model misspecification, 4) no interference (respondents' potential outcomes do not depend upon other respondents' exposures), and 5) positivity (no exposure scenarios that require respondents be exposed within strata of a confounder in which exposure is impossible).[106,107,119]

Appendix C2 contains code for implementing our parametric g-formula approach using R's "gfoRmula" package.[118] First, we fit pooled parametric models on the observed data for time-varying union membership, time-varying confounders, and mortality, using logistic models for binary variables and multinomial logistic models for categorical variables.[120] We assumed the following temporal ordering of time-varying exposure and confounders within each wave: 1) marital status, 2) employment status, 3) occupation, 4) industry, and 5) union membership (see diagram in Appendix C3). To predict time-varying variables in wave t_k, the pooled parametric models had predictors of baseline confounders, prior time-varying variables in t_k (if any), time-varying variables in t_{k-1}, follow-up time, and year. We specified categorical covariates as described in the "*Confounders"* sub-section and age as a 3-knot restricted cubic spline (using R's rms[121] package) to allow for nonlinear age-outcome relationships.[122] We specified follow-up time and year as 5-knot restricted cubic splines in most models, although we specified them differently in several models to improve fit; Appendix C4 contains details.

Next, we created a Monte-Carlo pseudo-sample of 25,000 by randomly drawing respondents with replacement from the observed data;[120] we drew a sample size larger than the original cohort to minimize simulation error.[123] We then predicted values of time-varying variables in respondents' second wave using their baseline pseudo-sample observations and parameters from the pooled parametric models described above.[120] We then used predicted values in respondents' second wave and parameters from the pooled parametric models to predict values in respondents' third wave, and so on, until mortality or the end of follow-up, whichever came first.[120] In our "natural course" scenario,[120] we left union-membership as predicted by the pooled parametric models. In our union scenario, we set union membership to "union" whenever respondents were employed. Finally, in our non-union scenario, we set union membership to "non-union" whenever respondents were employed. These scenarios avoided non-positivity bias by only allowing employed workers to be union-membership eligible.[119] In all scenarios, we eliminated administrative censoring and loss to follow-up, unbiased if censoring and loss to follow-up are random within levels of measured confounders.[120]

Finally, we calculated risk ratios (RRs) and risk differences (RDs) by contrasting the simulated cumulative incidence of mortality in the union scenario with the simulative cumulative incidence of mortality in the non-union scenario. We calculated confidence intervals for the RRs and RDs by repeating the g-formula algorithm on 200 bootstrap samples, with standard errors estimated as the standard deviations of the bootstrap distributions.[120] We probed for model misspecification by comparing the simulated exposure, time-varying confounder, and outcome distributions at each timepoint in the natural course with the distributions in the observed data.[105–107,120] We also examined whether the scenarios' effects varied by gender (women/men), race (Black/white), or education (≤HS/>HS) by running the approach outlined above in each subgroup.

4.2.5.2 Inequity analyses

In our inequity analyses, we used the sample and approach outlined above to contrast cumulative incidence of the outcomes for each racial and educational subgroup in two additional scenarios. In these scenarios, we randomly drew employed respondents' union membership values at each wave from binomial distributions with means equal to the probability of union membership observed for that subgroup in 1979 or 2015. In the 1979 scenario, this corresponded to a probability of union membership of 0.28 for Black respondents, 0.23 for white respondents, 0.28 for ≤HS respondents, 0.18 for >HS respondents. In the 2015 scenario, this corresponded to a probability of union membership of 0.14 for Black respondents, 0.12 for white respondents, 0.14 for ≤HS respondents, 0.12 for >HS respondents. A sharper mortality reduction in the 1979 scenario relative to the 2015 scenario for Black and less-educated respondents than for white and more-educated respondents would suggest declining union membership exacerbated racial and educational mortality inequities over the study period.

4.2.5.3 Sensitivity analyses

We conducted several sensitivity analyses. First, we tried lagging our exposure two years, as union membership may take time to affect mortality. Second, due to difficulties accurately modeling time-varying occupation and industry, we tested whether treating the variables as time-invariant throughout our analyses affected our estimates. Third, we tested whether additionally adjusting for baseline self-rated health (SRH, dichotomized as poor/fair vs good/very good/excellent to improve reliability[114]) affected our estimates, as selection into union jobs may vary by pre-existing health-status, a possibility not entirely accounted for by baseline-disability adjustment. We did not adjust for SRH in our primary analyses because SRH was not asked until 1984; thus, these analyses used imputed SRH values pre-1984. Fourth, we addressed possible residual geographic confounding by including baseline division of residence rather than baseline region of residence as a covariate in the pooled parametric models. We did not run this specification in subgroup analyses because there were few respondents in certain divisions. Finally, to compare our g-formula estimates to estimates from a traditional approach, we ran confounder-adjusted Cox models using R's survival package.[126] These models had baseline union membership as the exposure, baseline confounders as covariates (with age and year specified as 3- and 5-knot restricted cubic splines, respectively), and mortality as the outcome. As in the g-formula analyses, all respondents were employed at baseline. We calculated follow-up time for those who experienced an outcome during follow-up by subtracting their baseline year from their outcome year, while for those who remained outcome-free we calculated follow-up time by subtracting their baseline year from their final interview year.

4.2.5.4 Missing data

Our exposure and confounders contained a small amount of missingness ($\leq$4%). We addressed baseline-confounder missingness by carrying respondents' observed values forwards (or backwards if necessary) where possible. We addressed remaining missingness in the confounders and exposure using a single multivariate imputation by chained equations with 50 iterations using R's "mice" package.[127] As predictors, the imputation models included all baseline confounders, as well as time-varying exposure and confounders in t_k and t_{k-1} (or t_{k+1} in respondents' baseline wave). We did not create multiple imputed datasets because doing so in a parametric g-formula setting was computationally infeasible and methods for pooling parametric g-formula estimates have not been well-developed.

4.3 Results

4.3.1 *Descriptives*

Our overall sample included 23,022 respondents with 910 deaths and 146,681 observations. At baseline, 14% of respondents were union workers (Table 4.1). Union workers tended to be older and less educated than non-union workers, and were more likely to be persons of color, men, married/cohabiting, living outside the South, and to have grown up poor. Moreover, union workers more often had “operator, fabricator, and laborer” and “precision production, craft, and repair” occupations, as well as “manufacturing” and “transportation, communications, and other public utilities” industries. Finally, union workers had median family incomes 22% higher than non-union workers. Appendix C5 displays trends in union membership over the study period by demographic group, occupation, and industry.

4.3.2 *Primary and subgroup analyses*

Overall, the cumulative incidence of mortality by the end of follow-up in the natural course was 181.0 per 1,000 (Figure 4.1). Across subgroups, mortality incidence was greater among men, Black people, and the less-educated than among women, white people, and the more-educated (Table 4.2). In most analyses, the simulated mortality incidence in the natural course aligned with the observed cumulative incidence, as did the simulated distribution of union membership (Figure 4.1 and Appendix C12). However, although the simulated distribution of employment status generally aligned with the observed data, the simulated distributions of other time-varying confounders tended to differ from the observed data more considerably, particularly occupation and industry (Appendix C12).

Overall, approximately 20% of person-years in the union scenario were spent not employed, lower than the 23% in the non-union scenario. Moreover, mortality incidence was lower in the union scenario than in the non-union scenario (RR: 0.90, 95% CI: 0.80, 0.99; RD per 1,000: -18.7, 95% CI: -36.5, -0.9) (Table 4.2). This effect was largely consistent across subgroups, although the union scenario was much more protective among the more-educated (RR: 0.72, 95% CI: 0.57, 0.88; RD per 1,000: -34.9, 95% CI: -56.4, -13.4) than among the less-educated (RR: 0.95, 95% CI: 0.85, 1.06; RD per 1,000: -10.8, 95% CI: -36.1, 14.5).

4.3.3 *Inequity analyses*

We found little evidence that racial and educational mortality inequities would lessen if union-membership prevalence had remained at 1979 levels throughout follow-up rather than at 2015 levels. Specifically, modeling suggesting mortality among Black respondents would remain largely unchanged in the 2015 scenario relative to the 1979 scenario (RR: 0.99, 95% CI: 0.96, 1.01; RD per 1,000: -3.0, 95% CI: -7.2, 1.1), similar to the effect among white people (RR: 0.99, 95% CI: 0.97, 1.00; RD per 1,000: -2.0, 95% CI: -4.4, 0.3) (Table 4.3). Likewise, modeling suggesting mortality among less-educated respondents would remain largely unchanged in the 2015 scenario relative to the 1979 scenario (RR: 0.99, 95% CI: 0.97, 1.01; RD per 1,000: -2.4, 95% CI: -6.0, 1.2), similar to the effect among more-educated respondents (RR: 0.99, 95% CI: 0.98, 1.00; RD per 1,000: -1.2, 95% CI: -2.9, 0.5) (Table 4.3).

4.3.4 *Sensitivity analyses*

Lagging the exposure two years did not meaningfully affect our estimates (Appendix C6). Treating occupation and industry as baseline variables (Appendix C7), adjusting for baseline SRH (Appendix C8) or division of residence (Appendix C9), and using Cox models (Appendix C10) did not meaningfully affect our estimates either.

4.4 DISCUSSION

4.4.1 *Summary of results*

Using a parametric g-formula approach, we estimated how a hypothetical scenario setting all (versus none) of respondents' employed person-years to union-member employed person-years would affect cumulative incidence of mortality, and estimated how racial and educational mortality inequities would change if union-membership prevalence across racial and educational groups had remained at 1979 levels throughout follow-up rather than at 2015 levels. We found the union scenario might modestly reduce mortality incidence overall and in certain subgroups, particularly among the more-educated. However, contrary to expectations, we found little evidence that racial and educational mortality inequities would lessen if union-membership prevalence had remained at 1979 levels. If anything, effects on inequities in the actual U.S. population may be weaker than those estimated here due to our sample's artificially high labor-force attachment (as respondents only entered our sample upon employment).

Using a 1960s and 1970s male-only sample and baseline union-membership exposure, Waitzman also identified protective associations between union membership and mortality.[51] Although the protective effects estimated in his study were generally stronger than the effects estimated in our ours, the studies are not directly comparable given the different time periods, samples, and modeling approaches. For example, union-membership's beneficial effects may have weakened since Waitzman's study given the labor movement's diminishing power.[24]

Although we believe our results are plausible, several biases may have influenced our estimates. The biases' net effects are ambiguous. Regarding bias towards the null, research suggests that unmeasured workplace-level characteristics like hazardous working conditions may cause workers to unionize.[50] This partly explains why some quantitative studies have found unionization *increases* occupational injury risk, contradicting historical and anecdotal evidence.[50] Because we were unable to adjust for workplace-level confounders, unmeasured confounding by such factors may have resulted in an underestimate of unionism's protective effects. Unmeasured workplace-level confounding may be especially likely in this study because of the unexpectedly weak association between union membership and mortality among the less-educated, the workers most likely to be exposed to hazardous working conditions. Union-membership misclassification may have also biased our results towards the null. For example, Card found that 2.5% of 1977 Current Population Survey respondents misreported their union status.[85] This misclassification rate would mean approximately 17% of workers classified as union in our analyses were actually non-union (Appendix C11). Unfortunately, there is no research on the accuracy of PSID's union-membership data.

Regarding bias away from the null, union workers may differ from non-union workers in unmeasured skills.[113] For example, although contested in the literature, some authors argue that employers at unionized firms selectively hire highly-productive workers to offset the costs of the union wage and benefit premium.[113] These productivity-enhancing factors may also affect mortality. For example, in this study, if healthier respondents selected into union jobs because such jobs required physical exertion or workers who could endure more hazardous working conditions, union membership might spuriously appear to reduce mortality, true mortality effects aside. Although we adjusted for baseline disability status and SRH (to address potential health selection into union jobs), as well as broad occupation and industry categories (to, among other things, address potential differences in the physical attributes of workers required to perform various types of labor), we may not have completely blocked possible confounding pathways.

4.4.2 *Strengths and limitations*

Our study had several strengths. First, it is the only individual-level U.S.-based study to examine the union-mortality relationship using data with repeated union-membership measurements. Second, it is one of the first individual-level, longitudinal studies to examine the role of structural factors (like union density) rather than behavioral ones in contributing to recent changes in mortality inequities. Finally, unlike standard covariate-adjustment approaches, our parametric g-formula approach allowed us to flexibly estimate various scenario contrasts and address potential bias from time-varying exposure-confounder feedback and structural non-positivity.

Our study also had additional limitations beyond unmeasured confounding and union-membership misclassification. First, the natural-course distributions of occupation and industry differed somewhat from the observed distributions throughout our analyses, suggesting our models for those variables may have been misspecified. However, we do not think misspecification had an undue influence on our results because: 1) we accurately modeled the distributions of other time-varying variables in most analyses, and 2) our results were similar in traditional Cox analyses. Second, our sample had few deaths during follow-up, which prevented us from examining effect-modification or mortality inequities by gender-race or gender-education. Nonetheless, the relationship between union membership, mortality, and mortality inequities may vary by gender within racial and educational groups given the within-group gender differences in union-membership prevalence. Third, in the inequity analyses we assumed that setting union-membership prevalence to 1979 or 2015 levels would affect health and health inequities solely through the direct individual-level relationship between union membership and mortality. In reality, drastically increasing or decreasing union density may have many spillover and societal-level effects, given organized labor's role in shaping the U.S.'s social, political, and economic structures.[5,130,133] Spillover effects would violate the parametric g-formula's no-interference assumption, although such effects may not be strong given union-membership's modest direct effects on mortality. Finally, throughout our analyses we assumed union-membership's effects did not change temporally or vary by occupation, industry, or region, a strong assumption given changing union power over the study period and the many union organizing models (e.g., militant versus conservative).[130] This heterogeneity would violate the parametric g-formula's consistency assumption. Nonetheless, the union-wage-premium's consistency over follow-up suggests temporal changes in union-membership's effects may be modest.[30] Moreover, our stratification by gender, race, and education may have proxied for occupation, industry, and region.

4.5 Conclusion

In summary, we found that union membership may modestly protect against mortality among working-age adults. However, we found little evidence that increasing union membership would reduce racial and educational mortality inequities. In future studies, to address unmeasured confounding, researchers could consider quasi-experimental approaches such as regression-discontinuity and instrumental-variable designs,[48,96] although finding data to make such approaches viable is challenging. Additionally, to mitigate reductionism, researchers could consider multilevel approaches, such as examining how individual-level union membership interacts with area-level union density and other measures of working-class power, like the strike rate, to affect health and health inequities.

Table 4.1. Descriptive statistics of sample at baseline stratified by union membership.

	Non-union	Union
N	19656	3366
Age (median [1st quartile, 3rd quartile])	29 [26, 36]	31 [26, 41]
Male	9391 (47.8)	2092 (62.2)
Race (%)		
Black	6283 (32.0)	1318 (39.2)
Other	1698 (8.6)	258 (7.7)
White	11675 (59.4)	1790 (53.2)
Education (%)		
<HS	3734 (19.0)	704 (20.9)
HS	6626 (33.7)	1381 (41.0)
Some college	4857 (24.7)	726 (21.6)
College+	4439 (22.6)	555 (16.5)
Married/permanently-cohabiting (%)	14384 (73.2)	2630 (78.1)
Childhood socioeconomic status (%)[a]		
Poor	5906 (30.0)	1211 (36.0)
Average	8440 (42.9)	1360 (40.4)
Well-off	5310 (27.0)	795 (23.6)
Occupation (%)		
Farming, forestry, and fishing	280 (1.4)	9 (0.3)
Managerial	1721 (8.8)	64 (1.9)
Operators, fabricators, and laborers	3123 (15.9)	1170 (34.8)
Professional specialty	3069 (15.6)	471 (14.0)
Precision production, craft, and repair	1943 (9.9)	537 (16.0)
Services	3733 (19.0)	487 (14.5)
Technical, sales, and admin support	5787 (29.4)	628 (18.7)
Industry (%)		
Agriculture, forestry, and fisheries	424 (2.2)	16 (0.5)
Construction	1169 (5.9)	231 (6.9)
Finance, insurance, and real estate	1311 (6.7)	37 (1.1)
Manufacturing	3375 (17.2)	1061 (31.5)
Mining	119 (0.6)	17 (0.5)
Public administration	980 (5.0)	273 (8.1)
Services	7134 (36.3)	894 (26.6)
Transport, communications, and other public utilities	1183 (6.0)	573 (17.0)
Wholesale and retail trade	3961 (20.2)	264 (7.8)
Region of residence (%)		
Midwest	4509 (22.9)	1016 (30.2)
Northeast	2595 (13.2)	737 (21.9)
South	9074 (46.2)	976 (29.0)
West	3478 (17.7)	637 (18.9)
Work disability (%)[b]	1474 (7.5)	216 (6.4)
Family income (median [1st quartile, 3rd quartile])[c]	5.7 [3.5, 8.6]	7.0 [4.6, 9.8]

Notes:

[a] Childhood socioeconomic status when respondent was growing up.

[b] Respondent had disability that limited the type or amount of work they could do.

[c] Tens of thousands of family income in 2017 dollars.

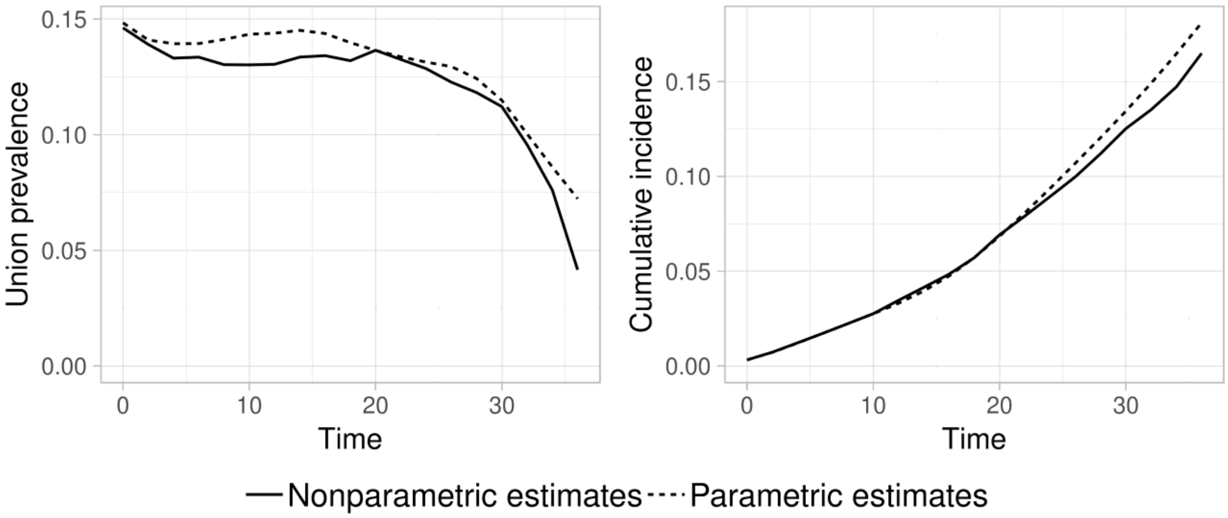

Figure 4.1. Simulated (parametric) probability of union membership and cumulative incidence of mortality in the natural course compared with the observed (nonparametric) values.

Table 4.2. Parametric g-formula estimates of risk of mortality by the end of follow-up (38 years) in a scenario (sc.) setting all of respondents' employed-person-years to union-member employed-person-years (scenario 1) relative to the risk in a scenario setting none of respondents' employed-person-years to union-member employed-person-years (scenario 2).

	Respondents[a]	Observations[a]	Sc.	Risk[b]	RR	95% CI		RD[b]	95% CI	
Overall	23,022	146,681	1	166.4	0.90	0.80	0.99	-18.7	-36.5	-0.9
			2	185.1						
Gender										
Women	11,539	76,285	1	128.2	0.87	0.71	1.03	-19.1	-43.8	5.5
			2	147.3						
Men	11,483	70,396	1	208.7	0.92	0.83	1.02	-17.9	-40.2	4.4
			2	226.6						
Race										
Black	7,601	43,785	1	183.7	0.89	0.75	1.03	-22.3	-51.4	6.8
			2	206.0						
White	13,465	92,844	1	139.9	0.90	0.77	1.03	-15.6	-35.6	4.4
			2	155.5						
Education										
≤HS	12,445	72,809	1	222.6	0.95	0.85	1.06	-10.8	-36.1	14.5
			2	233.4						
>HS	10,577	73,872	1	90.1	0.72	0.57	0.88	-34.9	-56.4	-13.4
			2	125.0						

Notes:

Risk ratio (RR) and risk difference (RD) estimates compare the risk (i.e., cumulative incidence) in scenario 1 relative to the risk in scenario 2. Subgroup estimates produced from stratified models. Confidence intervals calculated from non-parametric bootstrap with 200 repetitions.

[a] Unique respondents and observations in Panel Study of Income Dynamics dataset used to fit pooled time-varying exposure, confounder, and outcome models. Monte Carlo pseudo-sample used in simulations had 25,000 respondents.

[b] Per 1,000.

Table 4.3. Parametric g-formula estimates of risk of mortality by the end of follow-up (38 years) in a scenario (sc.) setting respondents' employed-person-years to union-member employed-person-years with the union-membership probability observed in 1979 (1979 scenario) relative to the risk in a scenario setting respondents' employed-person-years to union-member employed-person-years with the union-membership probability observed in 2015 (2015 scenario).

	Respondents[a]	Observations[a]	Sc.	Risk[b]	RR	95% CI		RD[b]	95% CI	
Race										
Black	7,601	43,785	1979	200.3	0.99	0.96	1.01	-3.0	-7.2	1.1
			2015	203.3						
White	13,465	92,844	1979	152.2	0.99	0.97	1.00	-2.0	-4.4	0.3
			2015	154.2						
Education										
≤HS	12,445	72,809	1979	230.2	0.99	0.97	1.01	-2.4	-6.0	1.2
			2015	232.6						
>HS	10,577	73,872	1979	118.9	0.99	0.98	1.00	-1.2	-2.9	0.5
			2015	120.1						

Notes:
Risk ratio (RR) and risk difference (RD) estimates compare the risk (i.e., cumulative incidence) in the 1979 scenario relative to the risk in the 2015 scenario. Subgroup estimates produced from stratified models. Confidence intervals calculated from non-parametric bootstrap with 200 repetitions.
[a] Unique respondents and observations in Panel Study of Income Dynamics dataset used to fit pooled time-varying exposure, confounder, and outcome models. Monte Carlo pseudo-sample used in simulations had 25,000 respondents.
[b] Per 1,000.

Chapter 5. CONCLUSION

5.1 SUMMARY OF FINDINGS

This dissertation analyzed the longitudinal relationships between unions, health, and health inequities. In Chapter 1, we described the changes in racial and educational mortality inequities over the last several decades, as well as the hypothesized mechanisms linking unions, health, and health inequities. In Chapter 2, we found that increases in state-level union density were associated with reductions in overdose and suicide mortality, although our estimates were sensitive to our approach. However, we found little evidence that increases in union density affected all-cause mortality or could reduce racial and educational mortality inequities. In Chapter 3, we identified no individual-level relationship between union membership and incidence of poor/fair SRH or moderate mental illness overall or across gender, gender-race, and gender-education subgroups. Finally, in Chapter 4, we found a modest protective association between union membership and all-cause mortality, which largely persisted across gender, race, and education subgroups. However, as in Chapter 2, we found little evidence that increases in union membership could reduce racial and educational mortality inequities.

5.2 EXPLANATION OF FINDINGS

The discrepancy between Chapter-2's and Chapter-4's all-cause-mortality findings may be due to random error in Chapter-2's union-density estimates, which could have masked underlying associations between unionism and mortality. The discrepancy may also be due to weaker than anticipated spillover effects of increases in union density on non-union-worker mortality, a plausible explanation given that spillover effects would likely be weaker than the modest direct effects we identified. Meanwhile, the discrepancy between Chapter-3's and Chapter-4's findings may be due to misclassification of respondents' SRH and mental illness, which are more difficult to measure accurately than mortality.[114,128,129]

Minor discrepancies across chapters aside, we generally identified small or null relationships between unionism, health, and health inequities. This may be due to a truly modest effect of unionism on the health outcomes we studied, a possibility we discussed in detail in prior chapters. Explanations for a modest effect include: 1) weakening union power over the study period, 2) racism and sexism in the union movement, 3) the detachment of certain union hierarchies from rank-and-file membership, and 4) difficulties identifying the relevant etiologic period for the unionism exposures. Bias may have also contributed to our modest findings. Potential sources of bias include: 1) misclassification of union membership and union density, 2) unmeasured confounding, and 3) the assumption that increasing union density to baseline levels would affect health and health inequities solely through the *direct* relationship between unionism and health.

5.3 Future Directions

Given the important roles of unions in working conditions, sociopolitical factors, and social inequality, we believe further research on the union-health relationship is warranted, particularly research which addresses the limitations of this dissertation. First, researchers could consider quasi-experimental approaches, which could address unmeasured confounding of the union-health relationship. For example, in prior epidemiological studies on unionism, researchers have used instrumental-variable approaches, instrumenting union density with state-level right-to-work laws.[48] However, because right-to-work laws cluster in the South and are often passed alongside other anti-labor reforms,[134] they may not be valid instruments. Other researchers, primarily in non-epidemiological studies, have used regression-discontinuity designs, comparing outcomes among workers in firms that narrowly vote for unionization with outcomes among workers in firms that narrowly vote against it.[96] However, such approaches require firm-level health data, which is rarely available. They also assume that employers (or workers) cannot precisely manipulate union-election results near the 50% threshold required for unionization,[96] a strong assumption given the frequency of unfair labor practices filed against employers during union elections.[135]

Second, researchers could consider how area-level union density, as well as how other area-level measures of working-class power and economic conditions, interact with individual-level union membership to affect health. For example, in our conceptual model (Figure 1.1), we hypothesized there were non-recursive relationships between union membership, area-level union density, and other structural factors that influence worker bargaining power, like the unemployment rate. Indeed, prior studies have found the national unemployment rate[136] and industry-level union density[113] modify the union wage premium. This suggests unionism's health effects may also vary across levels of such factors.

Finally, researchers could analyze interactions between union membership and relational social class measures based on property ownership and supervisory authority.[87] In this dissertation, education proxied respondents' social-class positions. However, education is an imperfect measure of social class. For example, a substantial proportion of those without a HS degree are managers, self-employed, or business owners, and thus ineligible for union membership.[137] Relational social-class measures directly separate "workers" (most of whom are eligible for union membership) from "managers" and "owners". Thus, they may more strongly interact with union membership than SES-based measures like education. This is especially likely because union membership may more strongly benefit those who lack workplace authority and class power (workers) than those who already have it (managers and owners).

5.4 Conclusion

Despite this dissertation's modest findings, we encourage public-health workers to pursue future research on and collaboration with the labor movement. The recent strikes by public educators,[134] women,[138] and "essential workers"[139] suggest that public-health resources like education, healthcare, and occupational safety will be key terrains of future class struggle.[140] Public-health workers committed to health equity should engage with these struggles.

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APPENDIX A

A1. ICD underlying cause of death codes used in analyses of fatal drug and alcohol poisoning and suicide (“deaths of despair”)

CDC’s Compressed Mortality Files used ICD-9 codes from 1986-1998 and ICD-10 codes from 1999-2016. Following Pierce et al.[1], who used the National Center for Health Statistics’(NCHS) ICD crosswalks,[2] we classified a death as due to drug poisoning if it had an ICD-9 code of E850-E858 or E980.0-E980.5 or an ICD-10 code of X40-X44 or Y10-Y14. Also following Pierce et al.,[1] we classified a death as due to suicide if it had an ICD-9 code of E950-E959 or an ICD-10 code of X60-X84, Y87.0, or U03. Finally, we classified a death as due to alcohol poisoning if it had an ICD-9 code of E860 or an ICD-10 code of X45. Our final despair-death outcome measure included deaths from any of these causes. Rates of our outcome measure did not change abruptly in 1999 upon the switch to ICD-10 (Figure A1), suggesting comparability of our measure over time.

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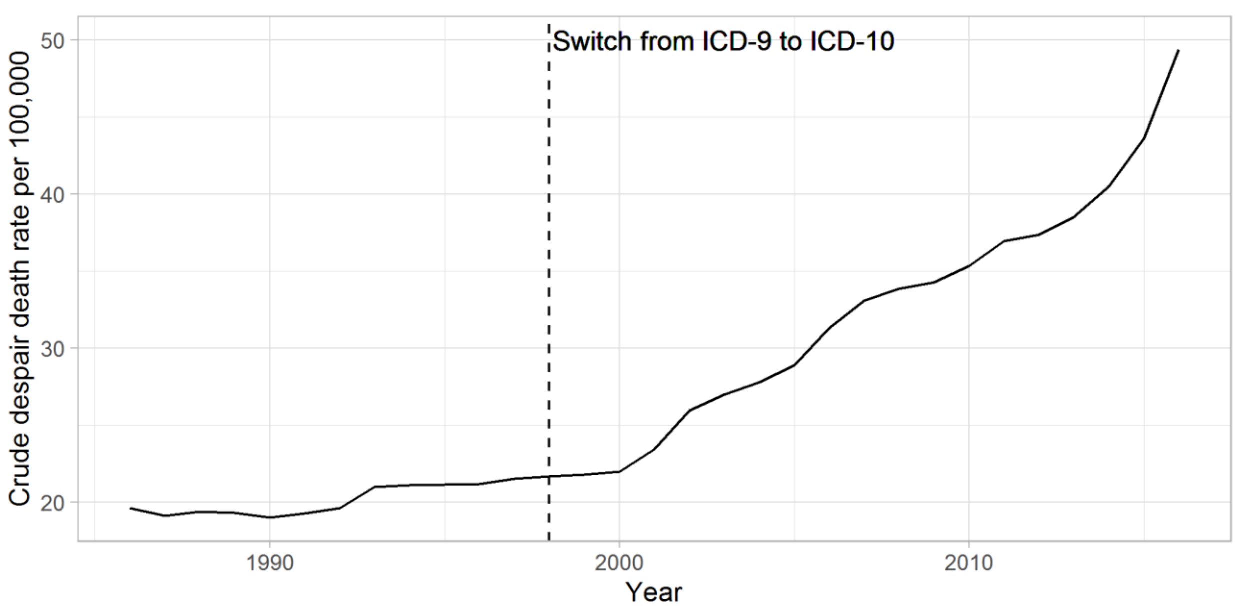


Figure A1. Crude despair death rate per 100,000 among those ages 25-64 from 1986-2016.

In some prior studies, researchers have included ICD-10 codes of Y15 in their measure of deaths due to alcohol poisoning ("poisoning by and exposure to alcohol, undetermined intent"). We did not include this code in our outcome measure because, to our knowledge, there are no directly comparable ICD-9 codes. However, including Y15 in our outcome measure did not appreciably change our regression estimates (Table A1) because there were at most 100 deaths per year nationwide with that code among those ages 25 to 64. Excluding deaths due to alcohol poisoning from our outcome measure entirely did not meaningfully change our estimates either (Table A1).

Table A1. Relationship between 10% increase in 1-year lagged, 3-year moving average state-level union density and state-level despair mortality rates from 1986 to 2016 overall.[a]

	Unweighted				**Weighted[b]**			
	RR	95% CI	RD	95% CI	RR	95% CI	RD	95 % CI
Primary estimates[c]	0.69	0.56, 0.85	-10.8	-17.1, -4.6	0.83	0.70, 0.98	-5.7	-10.7, -0.7
Including Y15[d]	0.69	0.56, 0.85	-10.9	-17.1, -4.6	0.83	0.70, 0.98	-5.7	-10.7, -0.7
Excluding alcohol poisoning[e]	0.69	0.56, 0.86	-10.6	-16.9, -4.3	0.83	0.70, 0.99	-5.3	-10.3, -0.3

Notes:
[a] Risk ratio (RR) and risk difference (RD, per 100,000 person-years) estimates calculated using log-linear Poisson models with state-level cluster-robust standard errors and state and year fixed effects. RD is average marginal effect of union density on mortality.
[b] Models weighted by gamma inverse probability of treatment weights.
[c] Estimates from primary models presented in main text. Outcome measure includes deaths due to drug and alcohol poisoning (ICD-9 codes: E850-E858, E860, E980.0-E980.5; ICD-10 codes: X40-45, Y10-14), as well as suicide (ICD-9 codes: E950-E959; ICD-10 codes: X60-84, Y87.0, U03).
[d] In addition to the ICD codes listed above, outcome measure includes ICD-10 code Y15 ("poisoning by and exposure to alcohol, undetermined intent").
[e] Outcome measure excludes deaths due to alcohol poisoning (ICD-9 codes: E860; ICD-10 codes: X45, Y15)

A2. Data sources for confounders.

Confounder	Source
Age distribution	Decennial census for 1990, 2000, and 2010; interpolation or ACS for intercensal years[a]
Gender distribution	Decennial census for 1990, 2000, and 2010; interpolation or ACS for intercensal years[a]
Racial distribution	Current Population Survey Merged Outgoing Rotation Group (CPS MORG)[a]
Education distribution	CPS MORG[a]
Economic conditions	
% unemployed	CPS MORG[a]
GDP per capita ($10000s)	Bureau of Economic Analysis
Mean wage ($)	CPS MORG[a]
Industrial structure	CPS MORG[a]
Policies	
State MW – federal MW ($)	Federal Reserve Economic Data
AFDC/TANF-to-poverty ratio	Center on Budget and Policy Priorities
UI recipiency rate	Numerator: Department of Labor Employment & Training Administration Claims and Payment Activities data (ETA 5159); Denominator: CPS MORG[a]

Notes:
[a] Data available from multiple sources.

A3. Distribution of stabilized gamma inverse probability of treatment weights at various truncation percentiles used in overall and effect-modification analyses.

Trunc. percentiles	Mean	SD	Min	Pctl(25)	Median	Pctl(75)	Max
Overall							
0.0, 100.0	2.42	26.59	0.00	0.38	0.63	1.10	935.44
0.5, 99.5	1.44	3.57	0.00	0.38	0.63	1.10	31.63
1.0, 99.0	1.39	3.13	0.00	0.38	0.63	1.10	24.28
2.5, 97.5	1.12	1.57	0.01	0.38	0.63	1.10	8.02
4.0, 96.0	1.04	1.23	0.03	0.38	0.63	1.10	5.43
5.0, 95.0	1.00	1.11	0.03	0.38	0.63	1.10	4.67
Gender							
0.0, 100.0	1.73	13.99	0.00	0.43	0.65	1.01	710.82
0.5, 99.5	1.37	3.38	0.00	0.43	0.65	1.01	34.02
1.0, 99.0	1.27	2.60	0.00	0.43	0.65	1.01	20.34
2.5, 97.5	1.10	1.54	0.01	0.43	0.65	1.01	8.31
4.0, 96.0	0.98	1.08	0.03	0.43	0.65	1.01	4.75
5.0, 95.0	0.95	0.97	0.04	0.43	0.65	1.01	4.04
Gender-race							
Women							
0.0, 100.0	10.45	161.76	0.00	0.50	0.70	0.99	4,773.75
0.5, 99.5	2.13	10.39	0.01	0.50	0.70	0.99	125.36
1.0, 99.0	1.56	4.62	0.02	0.50	0.70	0.99	38.82
2.5, 97.5	1.11	1.63	0.04	0.50	0.70	0.99	9.39
4.0, 96.0	0.97	1.01	0.09	0.50	0.70	0.99	4.89
5.0, 95.0	0.91	0.79	0.11	0.50	0.70	0.99	3.52
Men							
0.0, 100.0	2.06	15.85	0.00	0.54	0.74	1.03	430.32
0.5, 99.5	1.38	3.85	0.01	0.54	0.74	1.03	43.56
1.0, 99.0	1.20	2.14	0.02	0.54	0.74	1.03	17.03
2.5, 97.5	1.03	1.15	0.06	0.54	0.74	1.03	6.53
4.0, 96.0	0.95	0.78	0.10	0.54	0.74	1.03	3.65
5.0, 95.0	0.92	0.69	0.13	0.54	0.74	1.03	3.04
Gender-education							
Women							
0.0, 100.0	35.80	1,390.21	0.00	0.52	0.72	1.05	69,112.55
0.5, 99.5	1.53	5.23	0.01	0.52	0.72	1.05	58.55
1.0, 99.0	1.19	2.02	0.01	0.52	0.72	1.05	15.70
2.5, 97.5	1.05	1.20	0.04	0.52	0.72	1.05	6.64
4.0, 96.0	0.97	0.88	0.07	0.52	0.72	1.05	4.11
5.0, 95.0	0.94	0.77	0.10	0.52	0.72	1.05	3.35
Men							
0, 100	3.46	35.31	0.00	0.44	0.65	0.96	1,208.83
0.5, 99.5	2.14	11.48	0.00	0.44	0.65	0.96	140.95
1.0, 99.0	1.37	3.51	0.01	0.44	0.65	0.96	29.93
2.5, 97.5	1.08	1.57	0.03	0.44	0.65	0.96	8.63
4.0, 96.0	0.96	1.09	0.05	0.44	0.65	0.96	5.15
5.0, 95.0	0.91	0.88	0.08	0.44	0.65	0.96	3.83

A4. Relationship between 10% increase in 1-year lagged, 3-year moving average state-level union density and state-level mortality rates from 1986 to 2016 overall.

	Unweighted				Weighted			
	RR	**CI**	**RD**	**CI**	**RR**	**CI**	**RD**	**CI**
Poisson models[a]								
All-cause mortality	0.92	0.84, 1.01	-30.7	-65.9, 4.5	0.99	0.91, 1.08	-3.2	-36.3, 29.8
Despair mortality	0.69	0.56, 0.85	-10.8	-17.1, -4.6	0.83	0.70, 0.98	-5.7	-10.7, -0.7
OLS models I[b,c]								
All-cause mortality	0.92	0.83, 1.01	-34.5	-72.6, 3.5	0.98	0.90, 1.07	-7.7	-40.7, 25.4
Despair mortality	0.70	0.57, 0.87	-10.8	-18.0, -3.7	0.81	0.67, 0.97	-6.2	-11.5, -0.8
OLS models II[b,d]								
All-cause mortality	0.92	0.84, 1.00	-32.3	-69.4, 4.7	0.99	0.91, 1.07	-3.5	-37.7, 30.6
Despair mortality	0.70	0.54, 0.86	-11.1	-19.0, -3.1	0.86	0.68, 1.05	-4.2	-10.8, 2.4

[a] Estimates calculated using log-linear Poisson models with an offset of log(state-population size), state-level cluster-robust standard errors, and state and year fixed effects. Weighted models weighted by stabilized gamma IPTW.
[b] Estimates calculated using ordinary least squares (OLS) models with state-level cluster-robust standard errors and state and year fixed effects. Unweighted models weighted by state-population size; weighted models weighted by the product of the state-population size weights and the stabilized gamma IPTW.
[c] Outcome log-transformed. Risk difference is average marginal effect of union density on mortality.
[d] Outcome not log-transformed. Risk ratio (RR) calculated using nonlinear combinations of parameters.

A5a. Relationship between 10% increase in 1-year lagged, 3-year moving average state-level union density and state-level mortality rates from 1986 to 2016 overall and by gender.[a] Weighted models weighted by linear inverse probability of treatment weights (IPTW) rather than gamma IPTW; results from models weighted by gamma IPTW are presented in the main text.

	Unweighted				**Weighted[b]**			
	RR	95% CI	RD	95% CI	RR	95% CI	RD	95 % CI
Overall								
All-cause mortality	0.92	0.84, 1.01	-30.7	-65.9, 4.5	0.97	0.85, 1.09	-13.7	-63.4, 35.9
Despair mortality	0.69	0.56, 0.85	-10.8	-17.1, -4.6	0.89	0.72, 1.09	-3.5	-9.5, 2.5
Gender								
All-cause mortality								
Women	0.89	0.79, 1.01	-32.1	-66.2, 1.9	0.95	0.88, 1.04	-13.6	-37.5, 10.4
Men	0.95	0.89, 1.03	-23.6	-60.2, 13.1	1.04	0.94, 1.14	17.2	-31.9, 66.3
Interaction	0.93	0.83, 1.06	-8.6	-49.7, 32.6	0.92	0.81, 1.04	-30.8	-83.6, 22.1
Despair mortality								
Women	0.65	0.48, 0.89	-6.4	-10.7, -2.0	0.91	0.76, 1.09	-1.5	-4.3, 1.3
Men	0.78	0.68, 0.89	-11.5	-18.1, -4.9	0.92	0.81, 1.04	-3.7	-9.2, 1.8
Interaction	0.84	0.66, 1.07	5.2	0.4, 10.0	0.99	0.82, 1.19	2.2	-3.1, 7.5

Notes:

[a] Risk ratio (RR) and risk difference (RD, per 100,000 person-years) estimates calculated using log-linear Poisson models with state-level cluster-robust standard errors, state and year fixed effects, and where appropriate, union density*gender interaction terms. RD is average marginal effect of union density on mortality.

[b] Models weighted by linear IPTW.

A5b. Relationship between 10% increase in 1-year lagged, 3-year moving average state-level union density and state-level mortality rates from 1986 to 2016 by gender-race and from 1989 to 2016 by gender-education.[a] Weighted models weighted by linear inverse probability of treatment weights (IPTW) rather than gamma IPTW; results from models weighted by gamma IPTW are presented in the main text.

	Unweighted				**Weighted[b]**			
	RR	95% CI	RD	95% CI	RR	95% CI	RD	95 % CI
Gender-race								
All-cause mortality								
Women								
Black	1.00	0.95, 1.04	-1.0	-20.6, 18.6	1.00	0.96, 1.04	-1.2	-17.2, 14.9
White	0.82	0.73, 0.92	-53.0	-82.0, -23.9	0.85	0.77, 0.95	-40.8	-66.9, -14.7
Interaction	1.22	1.09, 1.37	52.0	20.6, 83.4	1.17	1.05, 1.30	39.6	12.4, 66.8
Men								
Black	1.00	0.94, 1.07	3.5	-42.6, 49.5	0.96	0.92, 1.01	-29.0	-63.1, 5.0
White	0.92	0.87, 0.97	-39.0	-63.9, -14.0	0.95	0.91, 1.00	-21.8	-44.6, 1.0
Interaction	1.09	1.03, 1.16	42.4	3.2, 81.6	1.01	0.95, 1.07	-7.3	-43.9, 29.4
Gender-education								
All-cause mortality								
Women								
≤HS	0.89	0.78, 1.02	-50.5	-109.0, 8.1	0.99	0.92, 1.06	-5.5	-38.9, 27.8
>HS	0.91	0.85, 0.98	-16.7	-29.4, -3.9	0.97	0.92, 1.03	-4.6	-14.2, 5.1
Interaction	0.98	0.90, 1.08	-33.8	-83.4, 15.8	1.01	0.94, 1.09	-1.0	-32.6, 30.7
Men								
≤HS	0.94	0.88, 1.01	-44.1	-99.6, 11.4	1.02	0.96, 1.10	19.4	-35.3, 74.1
>HS	0.93	0.87, 1.00	-19.3	-39.4, 0.8	1.01	0.94, 1.09	2.7	-19.0, 24.5
Interaction	1.01	0.92, 1.11	-24.8	-80.3, 30.7	1.02	0.91, 1.14	16.7	-46.4, 79.8

Notes:

[a] Risk ratio (RR) and risk difference (RD, per 100,000 person-years) estimates calculated using log-linear Poisson models with state-level cluster-robust standard errors, state and year fixed effects, and union density*race or union density*education interaction terms. RD is average marginal effect of union density on mortality.

[b] Models weighted by linear IPTW.

A5c. Change in state-level all-cause mortality rates among Black people versus white people and the less-educated versus the more-educated if 1-year lagged, 3-year moving average union density increased from 2015 levels to 1985 or 1988 levels respectively.[a] Models weighted by linear inverse probability of treatment weights (IPTW) rather than gamma IPTW; results from models weighted by gamma IPTW are presented in the main text.

	Women				Men			
	RR	95% CI	RD	95% CI	RR	95% CI	RD	95 % CI
Race[b]								
Black	1.00	0.93, 1.06	-1.6	-29.3, 26.1	0.99	0.84, 1.16	-6.8	-127.6, 114.0
White	0.96	0.90, 1.02	-11.3	-27.6, 5.0	0.93	0.86, 1.01	-33.0	-69.5, 3.5
Ratio/difference	1.04	0.96, 1.13	9.7	-20.2, 39.6	1.06	0.92, 1.23	26.1	-82.5, 134.8
Education[c]								
≤HS	0.99	0.95, 1.04	-3.6	-25.2, 18.1	1.05	0.92, 1.19	35.4	-65.5, 136.3
>HS	0.99	0.96, 1.02	-2.3	-7.2, 2.5	1.01	0.96, 1.06	1.9	-13.4, 17.2
Ratio/difference	1.01	0.96, 1.05	-1.3	-21.9, 19.4	1.04	0.90, 1.20	33.5	-71.8, 138.8

Notes:
[a] Risk ratio (RR) and risk difference (RD, per 100,000 person-years) estimates calculated using linear combinations of parameters from IPTW log-linear Poisson models with state-level cluster-robust standard errors, state and year fixed effects, and union density*race or union density*education interaction terms. RD is average marginal effect of union density on mortality.
[b] Models run on outcome data from 1986-2016. Counterfactual union density set to 1985 levels.
[c] Models run on outcome data from 1989-2016. Counterfactual union density set to 1988 levels.

A6a. Relationship between 10% increase in 3-year lagged, 3-year moving average state-level union density and state-level mortality rates from 1986 to 2016 overall and by gender.[a] Results from models with 1-year lagged, 3-year moving average state-level union density are presented in the main text.

	Unweighted				Weighted[b]			
	RR	95% CI	RD	95% CI	RR	95% CI	RD	95 % CI
Overall								
All-cause mortality	0.91	0.83, 1.00	-36.8	-72.0, -1.6	0.97	0.88, 1.06	-12.6	-50.9, 25.7
Despair mortality	0.69	0.55, 0.86	-11.1	-17.8, -4.5	0.81	0.68, 0.98	-6.4	-12.1, -0.6
Gender								
All-cause mortality								
Women	0.89	0.79, 1.01	-31.6	-65.0, 1.8	0.97	0.83, 1.13	-10.2	-54.8, 34.5
Men	0.94	0.87, 1.01	-32.6	-69.6, 4.4	0.97	0.89, 1.13	-14.8	-61.0, 31.4
Interaction	0.95	0.84, 1.08	1.0	-40.2, 42.3	0.99	0.78, 1.17	4.6	-71.9, 81.0
Despair mortality								
Women	0.67	0.47, 0.95	-6.2	-11.1, -1.2	0.83	0.64, 1.06	-3.0	-6.9, 0.9
Men	0.77	0.67, 0.89	-12.1	-19.1, -5.0	0.80	0.70, 0.91	-10.7	-17.4, -4.0
Interaction	0.87	0.65, 1.15	5.9	0.2, 11.6	1.04	0.82, 1.31	7.6	1.4, 13.9

Notes:

[a] Risk ratio (RR) and risk difference (RD, per 100,000 person-years) estimates calculated using log-linear Poisson models with state-level cluster-robust standard errors, state and year fixed effects, and where appropriate, union density*gender interaction terms. RD is average marginal effect of union density on mortality.

[b] Models weighted by gamma inverse probability of treatment weights.

A6b. Relationship between 10% increase in 3-year lagged, 3-year moving average state-level union density and state-level mortality rates from 1986 to 2016 by gender-race and from 1989 to 2016 by gender-education.[a] Results from models with 1-year lagged, 3-year moving average state-level union density are presented in the main text.

	Unweighted				**Weighted[b]**			
	RR	95% CI	RD	95% CI	RR	95% CI	RD	95 % CI
Gender-race								
All-cause mortality								
Women								
Black	1.00	0.96, 1.04	-0.5	-18.6, 17.6	1.01	0.96, 1.06	3.4	-16.1, 22.9
White	0.82	0.73, 0.92	-52.0	-80.2, -23.8	0.92	0.82, 1.04	-21.9	-52.6, 8.8
Interaction	1.22	1.09, 1.37	51.5	21.5, 81.5	1.09	0.97, 1.24	25.3	-9.3, 59.9
Men								
Black	1.00	0.94, 1.06	-1.4	-46.4, 43.7	0.98	0.91, 1.06	-14.0	-69.2, 41.2
White	0.90	0.85, 0.95	-48.8	-74.2, -23.4	0.92	0.87, 0.98	-36.2	-64.0, -8.4
Interaction	1.11	1.05, 1.17	47.4	10.9, 83.9	1.06	0.99, 1.14	22.1	-26.8, 71.0
Gender-education								
All-cause mortality								
Women								
≤HS	0.92	0.81, 1.03	-39.9	-92.0, 12.3	0.97	0.88, 1.07	-13.0	-57.2, 31.2
>HS	0.91	0.83, 0.99	-17.3	-32.4, -2.2	0.98	0.91, 1.06	-2.9	-15.8, 10.0
Interaction	1.01	0.92, 1.11	-22.6	-67.8, 22.6	0.99	0.89, 1.10	-10.1	-53.8, 33.6
Men								
≤HS	0.95	0.89, 1.02	-40.3	-94.8, 14.3	0.96	0.89, 1.02	-36.3	-90.7, 18.2
>HS	0.96	0.87, 1.05	-12.4	-39.6, 14.7	0.96	0.86, 1.07	-12.8	-44.5, 18.9
Interaction	0.99	0.89, 1.11	-27.9	-86.3, 30.6	1.00	0.89, 1.12	-23.4	-79.2, 32.4

Notes:

[a] Risk ratio (RR) and risk difference (RD, per 100,000 person-years) estimates calculated using log-linear Poisson models with state-level cluster-robust standard errors, state and year fixed effects, and union density*race or union density*education interaction terms. RD is average marginal effect of union density on mortality.

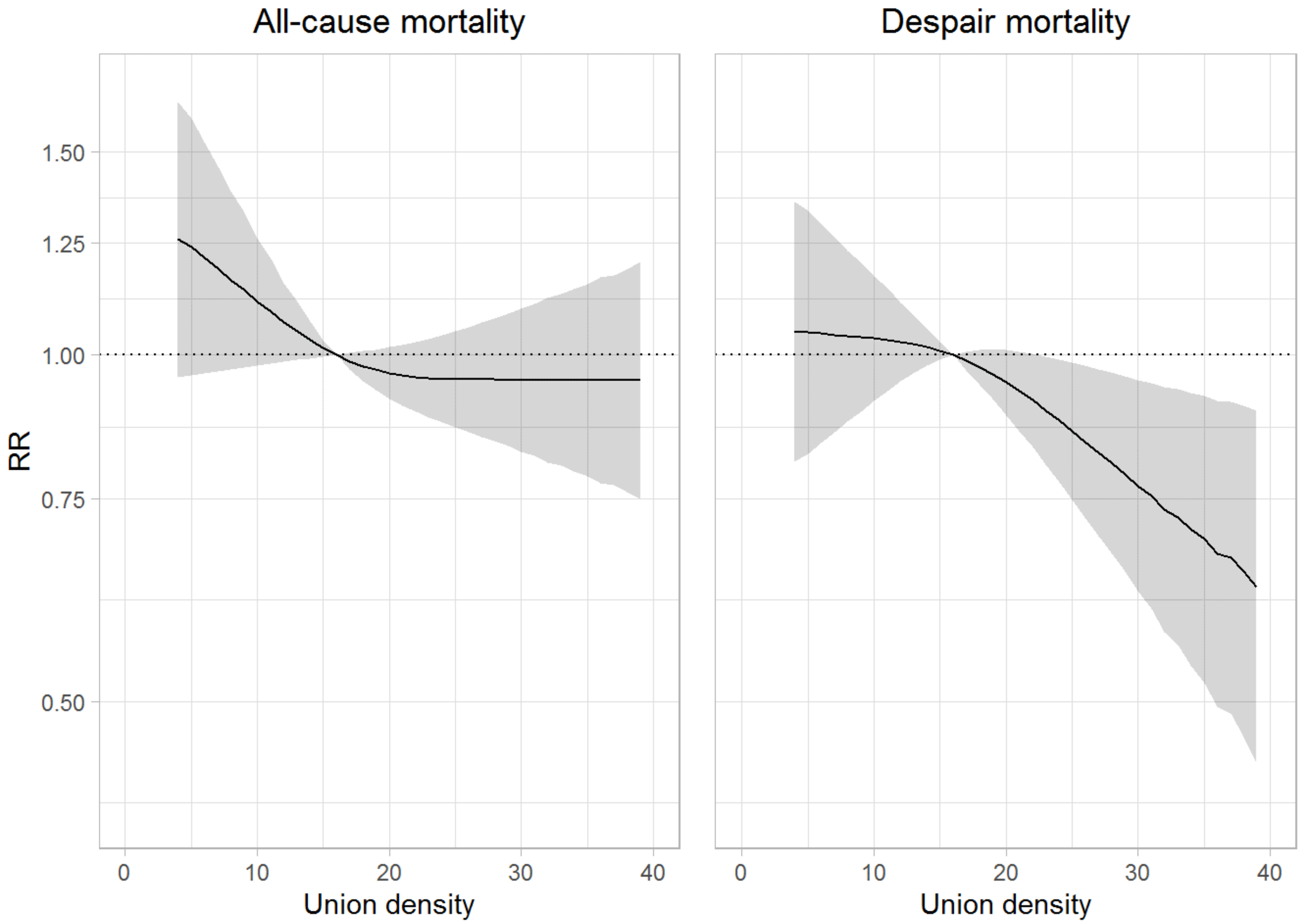
[b] Models weighted by gamma inverse probability of treatment weights.

A6c. Change in state-level all-cause mortality rates among Black people versus white people and the less-educated versus the more-educated if 3-year lagged, 3-year moving average union density increased from 2015 levels to 1985 or 1988 levels respectively.[a] Results from models with 1-year lagged, 3-year moving average state-level union density are presented in the main text.

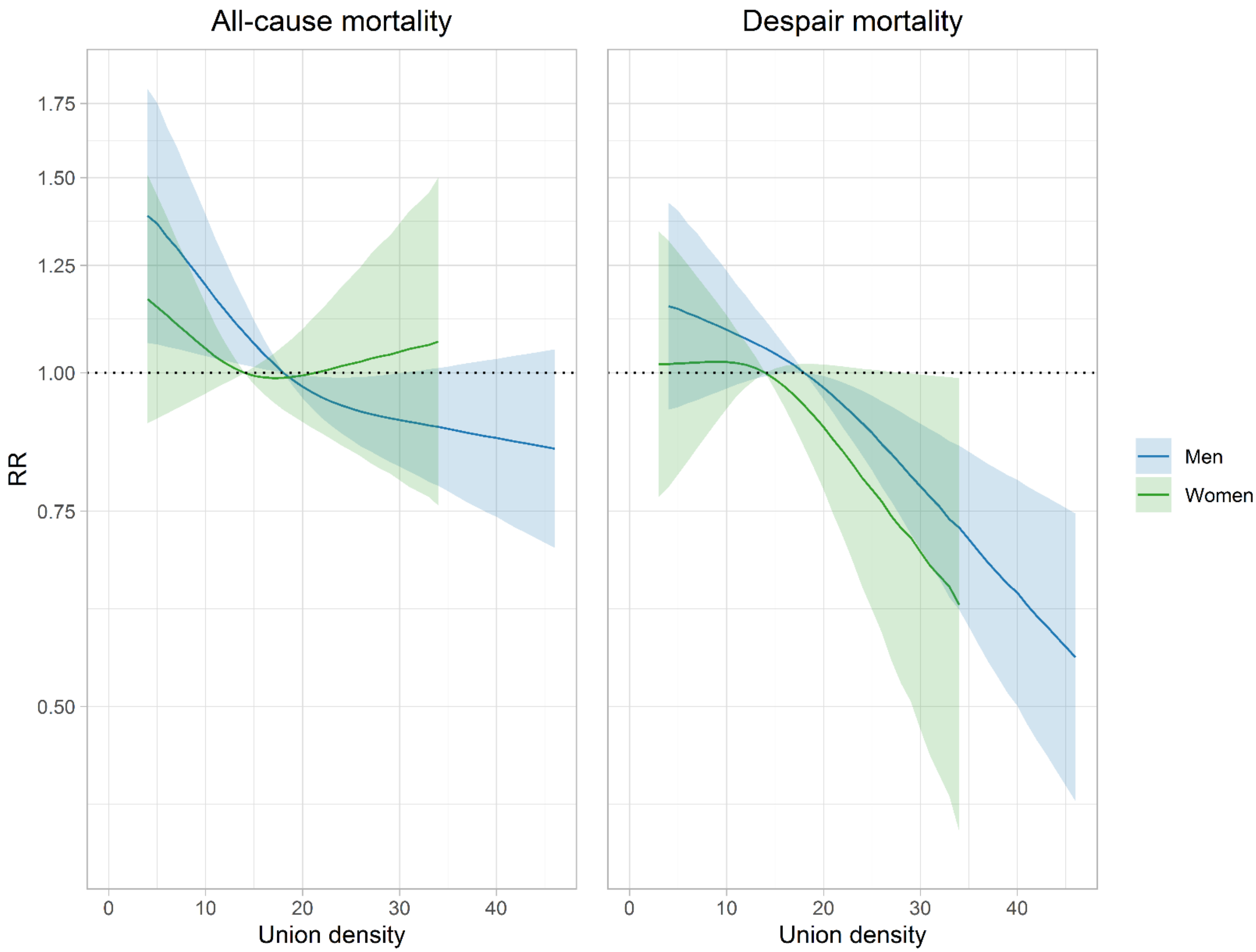
	Women				**Men**			
	RR	95% CI	RD	95% CI	RR	95% CI	RD	95 % CI
Race[b]								
Black	0.97	0.87, 1.08	-20.7	-101.6, 60.1	0.96	0.82, 1.12	-29.7	-144.4, 84.9
White	0.96	0.93, 0.99	-19.5	-34.3, -4.8	0.89	0.82, 0.97	-53.0	-91.2, -14.8
Ratio/difference	1.01	0.92, 1.12	-1.2	-76.3, 73.9	1.08	0.94, 1.24	23.3	-79.5, 126.0
Education[c]								
≤HS	0.97	0.93, 1.01	-24.1	-61.5, 13.2	0.92	0.81, 1.04	-64.5	-159.9, 30.9
>HS	0.98	0.92, 1.03	-6.6	-23.1, 9.9	0.97	0.90, 1.05	-9.1	-31.6, 13.4
Ratio/difference	0.99	0.93, 1.06	-17.5	-54.4, 19.4	0.95	0.84, 1.08	-55.4	-147.9, 37.0

Notes:
[a] Risk ratio (RR) and risk difference (RD, per 100,000 person-years) estimates calculated using linear combinations of parameters from inverse probability of treatment weighted log-linear Poisson models with state-level cluster-robust standard errors, state and year fixed effects, and union density*race or union density*education interaction terms. RD is average marginal effect of union density on mortality.
[b] Models run on outcome data from 1986-2016. Counterfactual union density set to 1985 levels.
[c] Models run on outcome data from 1989-2016. Counterfactual union density set to 1988 levels.

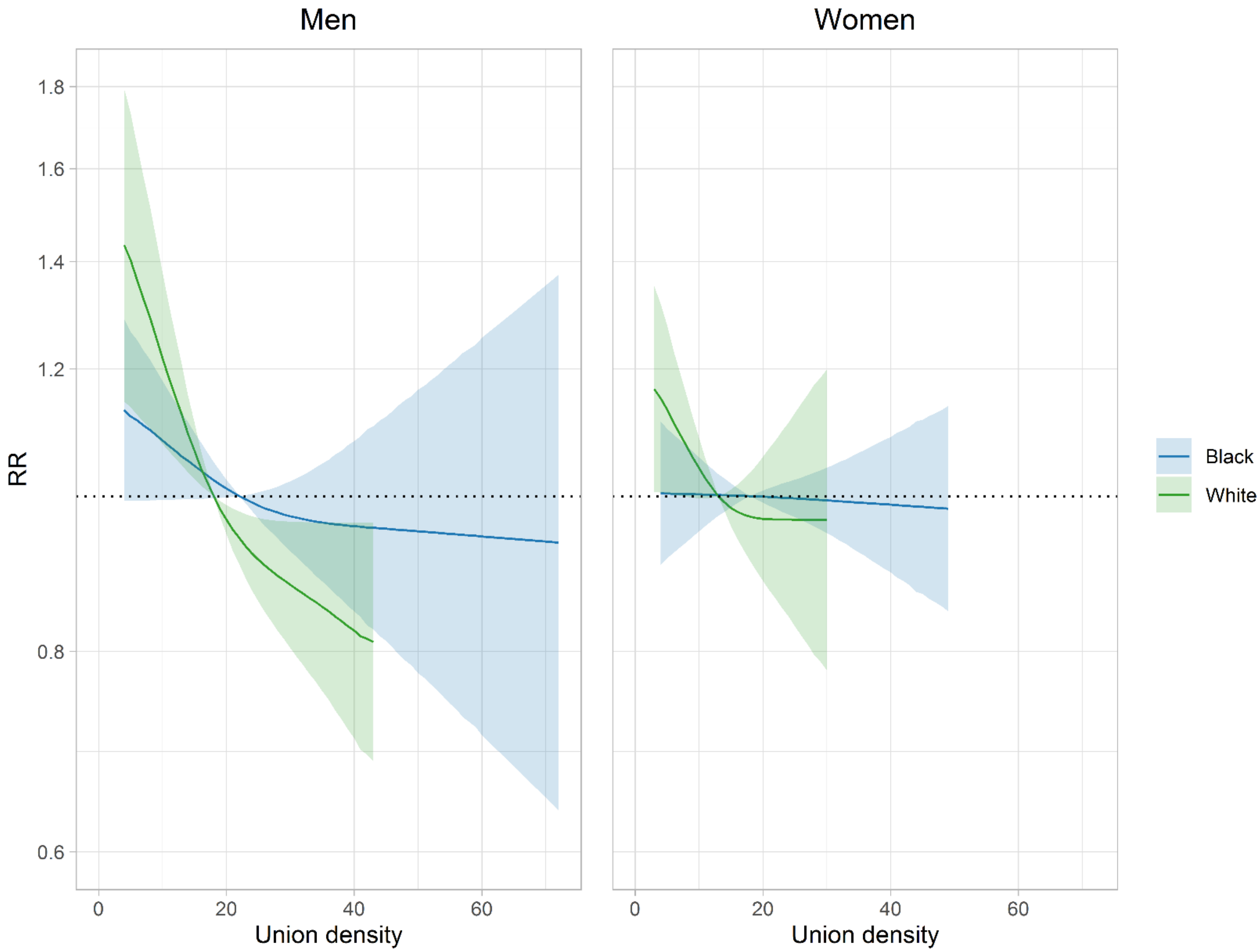
A7a. Relationship between state-level union density – modeled with a 3-knot restricted cubic spline – and state-level mortality rates from 1986 to 2016. Estimates from log-linear Poisson models weighted by gamma inverse probability of treatment weights with state-level cluster-robust standard errors and state and year fixed effects. Reference level is median state-level union density (16%).



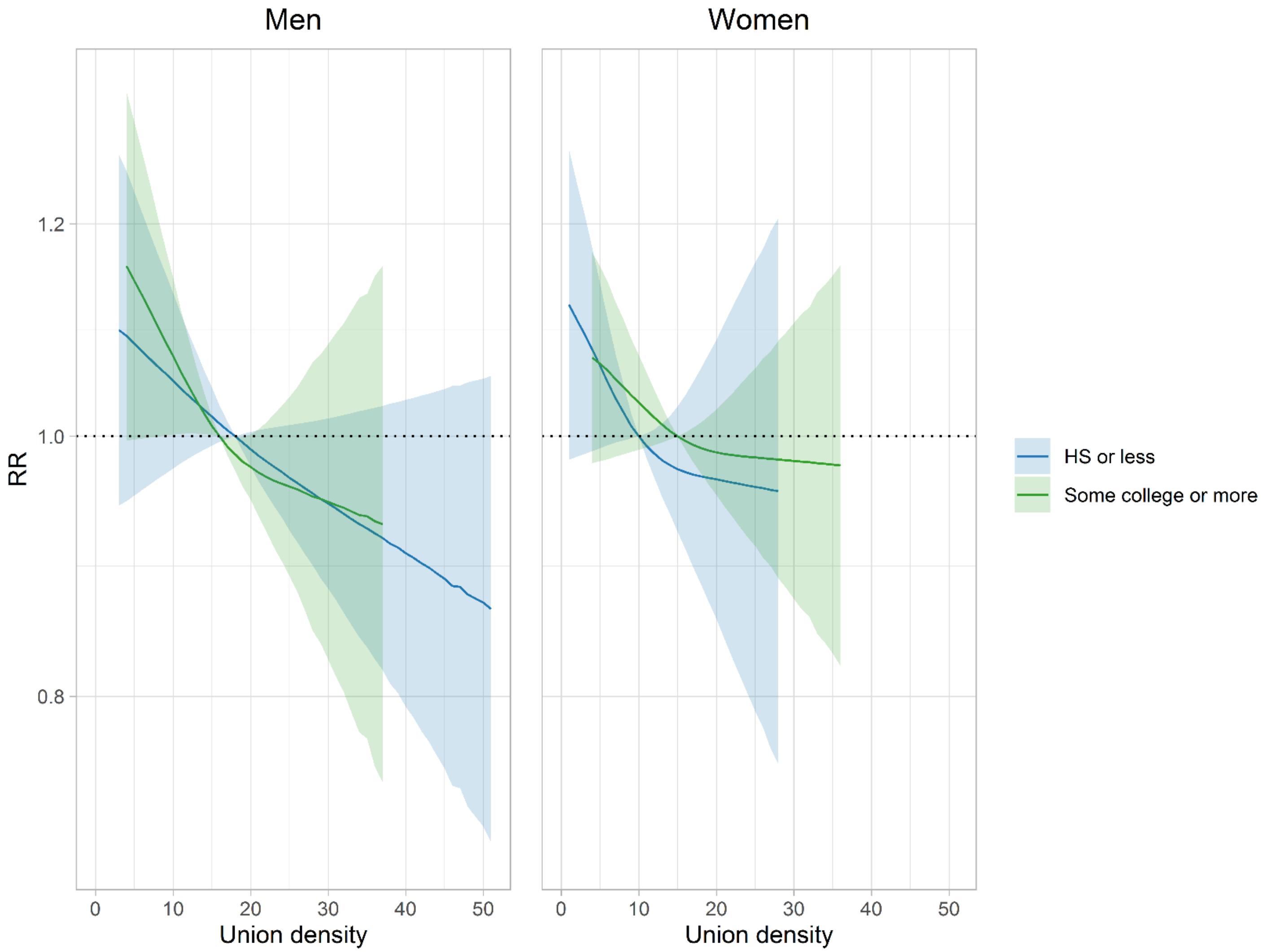
A7b. Relationship between state-level union density – modeled with a 3-knot restricted cubic spline – and state-level mortality rates from 1986 to 2016 by gender. Estimates from log-linear Poisson models weighted by gamma inverse probability of treatment weights with state-level cluster-robust standard errors, state and year fixed effects, and union density*gender interaction terms. Reference level is median gender-specific state-level union density (18% among men and 14% among women).



A7c. Relationship between state-level union density – modeled with a 3-knot restricted cubic spline – and state-level all-cause mortality rates from 1986 to 2016 by gender-race. Estimates from log-linear Poisson models weighted by gamma inverse probability of treatment weights with state-level cluster-robust standard errors, state and year fixed effects, and union density*race interaction terms. Reference level is median gender-race-specific state-level union density (22% among Black men, 18% among white men, 18% among Black women, and 13% among white women).



A7d. Relationship between state-level union density – modeled with a 3-knot restricted cubic spline – and state-level all-cause mortality rates from 1989 to 2016 by gender-education. Estimates from log-linear Poisson models weighted by gamma inverse probability of treatment weights with state-level cluster-robust standard errors, state and year fixed effects, and union density*education interaction terms. Reference level is median gender-education-specific state-level union density (18% among less-educated men, 16% among more-educated men, 10% among less-educated women, and 15% among more-educated women).



A8. Relationship between 10% increase in 1-year lagged, 3-year moving average state-level union density and state-level mortality rates from 1990-2015. Estimates on left adjusted for confounders described in main text using IPTW, whereas estimates on right adjusted for confounders described in main text plus the proportion of the population ages 25 to 64 with health insurance.

	No adjustment for HI				**Adjustment for HI**			
	RR	95% CI	RD	95% CI	RR	95% CI	RD	95 % CI
All-cause mortality	0.93	0.85, 1.02	-27.7	-64.0, 8.6	0.93	0.85, 1.01	-30.0	-65.8, 5.8
Despair mortality	0.86	0.72, 1.04	-4.5	-10.0, 1.1	0.85	0.71, 1.03	-4.7	-10.3, 0.9

Notes:
Risk ratio (RR) and risk difference (RD, per 100,000) estimates calculated using IPTW-weighted log-linear Poisson models with state-level cluster-robust standard errors and state and year fixed effects. RD is average marginal effect of union density on mortality.

A9. Testing for interference

If union density in one state affected mortality rates in another state, the no-interference assumption of MSM's would be violated.[84] This could occur through two primary mechanisms. First, union density in one state could directly affect mortality rates in a bordering state. For example, if people live in one state and work in another state, higher union density in their state of employment may affect their mortality risk in their state of residence (or vice versa). Second, union density in one state could modify union density's effects in another state. For example, high union density in a given region may augment worker bargaining power in states within that region, increasing the efficacy of union organizing. To reduce labor costs, an employer in a highly-unionized state may want to move their factory to a less-unionized state, or to replace union labor with non-union labor. However, in a highly-unionized region, the employer may be unable to easily move or replace workers. Without the threat of factory relocation or replacement, workers in that firm may be able to make stronger health-promoting demands of their employer.

Unfortunately, to our knowledge, methods for testing for interference or estimating causal effects in the presence of interference in settings such as ours – a continuous exposure and possible general interference (i.e., interference within groups (e.g., regions), as well as across groups) – have not been well-developed. Nonetheless, we performed preliminary analyses to test whether union density in one state directly affected all-cause or despair mortality rates in a bordering state. To this end, we randomly sampled 51 state/bordering-state pairs with replacement from the data. Using observations from this sample from 1986 to 2016, we estimated unweighted log-linear Poisson models with a given state's mortality count as the outcome and a given state's union density as an exposure, as well as the bordering state's union density, state fixed effects, and year fixed effects as covariates, and log(population) as an offset. We used cluster bootstrapping with 1,000 replications to generate confidence intervals.

Results are shown in Table A6 below. The coefficient of the bordering state's union density is null for analyses of all-cause mortality and for analyses of despair mortality, which is what we expected if interference were minimal.

Table A9. Relationships between 10% increase in 1-year lagged, 3-year moving average state-level union density among state of interest, as well as random bordering state, and state-level mortality rates from 1986 to 2016.

	All-cause mortality		Despair mortality	
	RR	95% CI	RR	95% CI
State	0.94	0.83, 1.05	0.71	0.56, 0.90
Bordering state	0.98	0.84, 1.15	0.97	0.77, 1.24

Notes:
Estimates calculated using non-IPTW-weighted log-linear Poisson models with state and year fixed effects. Standard errors calculated via state-level cluster bootstrap.

APPENDIX B

B1. Healthy-worker survivor bias.

For healthy-worker survivor bias to be a potential issue in this study, three relationships should hold in the data: 1) prior union membership should be associated with current employment status, 2) current employment status should be associated with current union membership, and 3) current employment status should be associated with current or future health.[1] The simplified directed acyclic graph below shows how we conceptualized these relationships in our study (Figure B1).

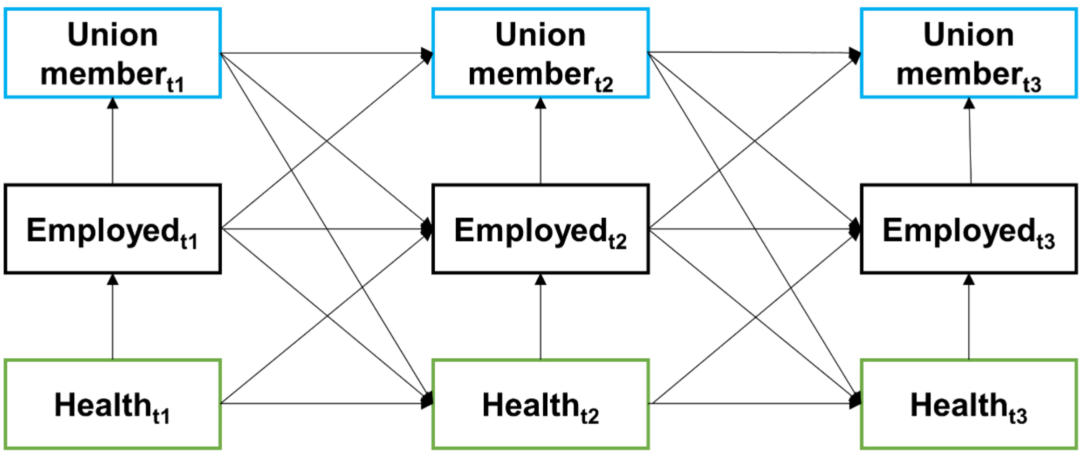


Figure B1. Simplified directed acyclic graph depicting time-varying confounding by employment status.

Following prior research on healthy-worker survivor bias and the parametric g-formula,[2,3] we tested for relationships 1) and 3) in our sample. Relationship 2) is a feature of union membership and thus did not require testing.

a. Prior union membership and current employment status

To test whether prior union membership was associated with current employment status, we used a Cox model with two-year-lagged union membership as the exposure, incident non-employment as the outcome, and covariates (measured at baseline unless otherwise noted) of gender, race, division of residence, education, childhood socioeconomic status, disability status, time-varying age (as a 3-knot restricted cubic spline [RCS]), time-varying year (as a 5-knot RCS), time-varying two-year-lagged occupation, time-varying two-year-lagged industry, and time-varying two-year-lagged marital status.

Respondents ages 25-64 entered the sample used to fit this model at the first wave they were employed by someone other than themselves and remained in the sample until their first bout of non-employment or censoring, whichever came first.

b. Current employment status and future health

To test whether current employment status was associated with future health, we used Cox models with two-year-lagged employment status (employed/not employed) as the exposure, incident poor/fair SRH or moderate mental illness as the outcome, and covariates (measured at baseline unless otherwise noted) of gender, race, division of residence, education, childhood socioeconomic status, disability status, time-varying age (as a 3-knot RCS), time-varying year (as a 3-knot or 5-knot RCS in the mental illness and SRH analyses respectively), and time-varying two-year-lagged marital status. In sensitivity analyses, we additionally adjusted for four-year-lagged union membership.

For analyses of employment status and SRH, respondents ages 25-64 entered the sample at the first wave they were observed in the data; we excluded those reporting the outcome at that wave. Respondents remained in the sample until their first incident outcome or censoring, whichever came first. For analyses of employment status and moderate mental illness, respondents only had K6 measurements in certain waves because PSID did not administer the K6 in 2005 and only administered the scale to non-proxy respondents in other waves. We assumed 2005 respondents and proxy respondents did not have K6 values $\geq$5 because they did not have K6 measurements. Respondents ages 25-64 entered the sample at the first wave they were employed by someone other than themselves *and* had a valid K6 measurement; we excluded those reporting the outcome at that wave. Respondents remained in the sample until their last K6 measurement or their first incident outcome, whichever came first. We excluded respondents with <2 K6 measurements during follow-up.

c. Results

The Cox models supported our hypothesis about potential healthy-worker survivor bias in this study (Table B1), suggesting our parametric g-formula approach was warranted. First, regarding the association between prior union membership and current employment status, two-year-lagged union membership was associated with a 14% lower (95% CI: 0.80, 0.92) hazard of non-employment than two-year-lagged union non-membership. Second, regarding the association between current employment status and future SRH, two-year-lagged employment was associated with a 31% lower (95% CI: 0.63, 0.75) hazard of poor/fair SRH than two-year lagged non-employment. Finally, regarding the association between current employment status and future SRH, two-year-lagged employment was associated with an 18% lower (95% CI: 0.74, 0.91) hazard of moderate mental illness than two-year lagged non-employment. Adjustment for four-year-lagged union membership did not meaningfully affect the latter two estimates.

Table B1. Estimates from Cox models used to assess potential healthy-worker survivor bias.

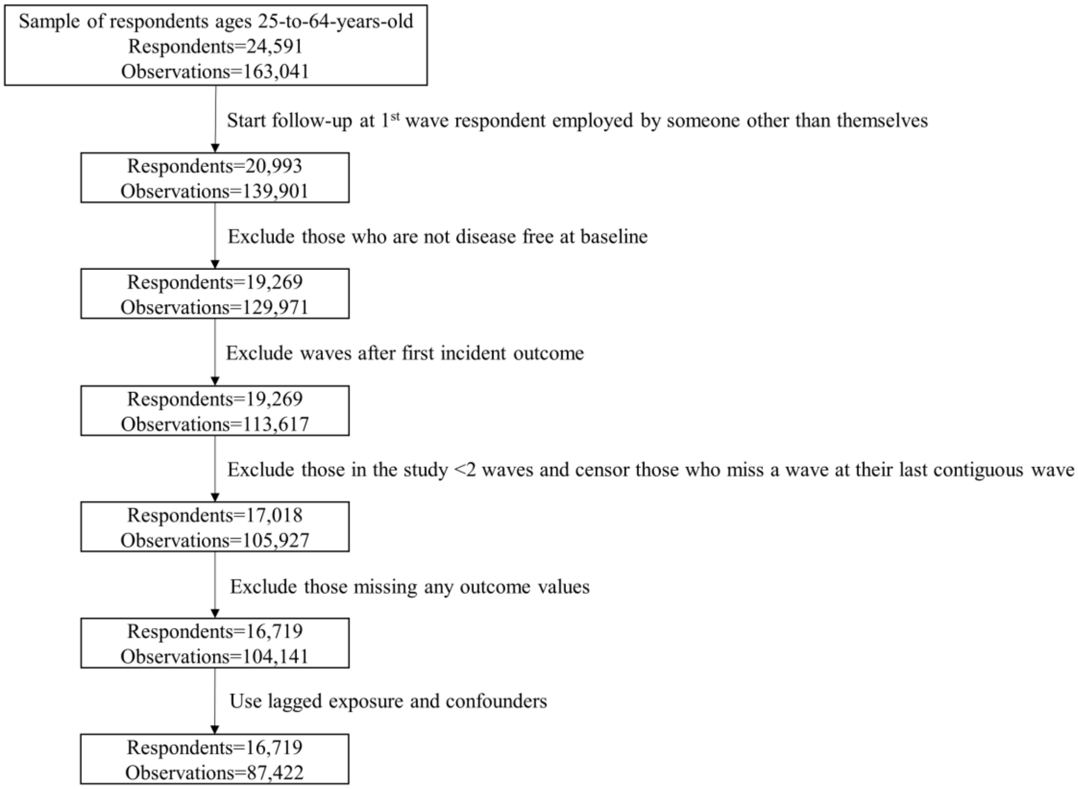
	Model 1			**Model 2**		
	HR	95% CI		HR	95% CI	
2-year-lagged union and non-employment (ref: non-union)[a]	0.86	0.80	0.92	-	-	-
2-year-lagged employment and poor/fair SRH (ref: non-employment)[b]	0.69	0.63	0.75	0.71	0.64	0.79
2-year-lagged employment and mental illness (ref: non-employment)[c]	0.82	0.74	0.91	0.77	0.67	0.87

Notes:
Confidence intervals for all models calculated using robust standard errors.
[a] Model used data on 18,463 respondents with 83,157 observations. Estimate adjusted for covariates described above.
[b] Model used data on 13,194 respondents with 62,172 observations. Estimate adjusted for covariates described above. Estimate from model 2 additionally adjusted for four-year-lagged union membership.
[c] Model used data on 6,595 respondents with 25,773 observations. Estimate adjusted for covariates described above. Estimate from model 2 additionally adjusted for four-year-lagged union membership.

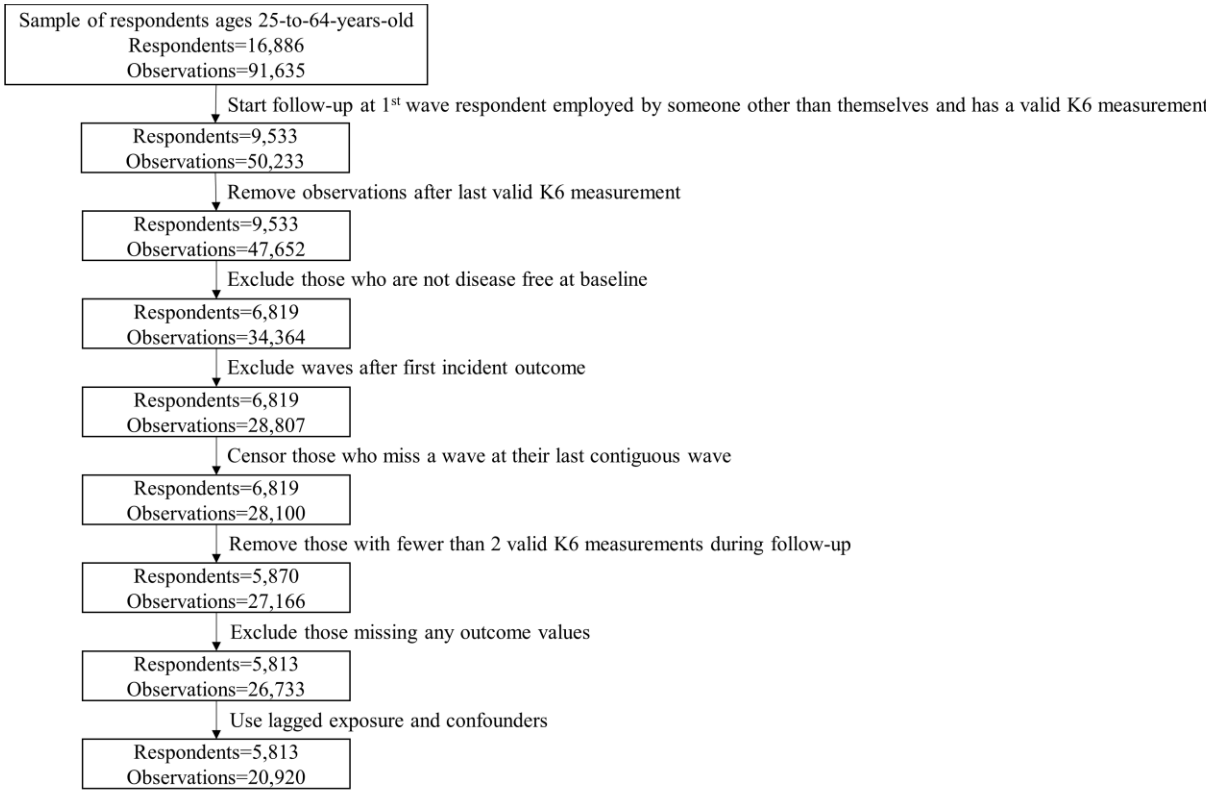
d. *References*

1. Naimi AI, Cole SR, Hudgens MG, Brookhart MA, Richardson, DB. Assessing the component associations of the healthy worker survivor bias: occupational asbestos exposure and lung cancer mortality. *Ann Epidemiol*. 2013;23(6):334-341. doi:10.1016/j.annepidem.2013.03.013
2. Keil AP, Richardson DB, Westreich D, Steenland K. Estimating the impact of changes to occupational standards for silica exposure on lung cancer mortality. *Epidemiology*. 2018;29(5):658-665. doi:10.1097/EDE.0000000000000867
3. Neophytou AM, Picciotto S, Costello S, Eisen EA. Occupational diesel exposure, duration of employment, and lung cancer: an application of the parametric g-formula. *Epidemiology*. 2016;27(1):21-28. doi:10.1097/eDe.0000000000000389

B2a. Flow diagram for SRH analyses.



B2b. Flow diagram for mental-illness analyses.



B3. R code used to estimate cumulative incidence of poor/fair SRH by the end of follow-up (32 years) in a scenario setting all of respondents' two-year-lagged employed-person-years to union-member employed-person-years relative to the cumulative incidence in a scenario setting none of respondents' two-year-lagged employed-person-year to union-member employed-person-year.

```
library(gfoRmula) #development version on GitHub as of 1/25/20 needed for control
argument in "covparams"
library(dplyr)
library(here)
library(data.table)
library(knitr)
library(kableExtra)
library(rms)
library(ggplot2)
library(cowplot)
library(gridExtra)

######read data

dat <- fread(here('srh_dat.csv'))

######set number of cores and bootstrap samples for future parallelization, as well as
Monte Carlo sample size

ncores <- parallel::detectCores() - 1
nsamples <- 250
nsimul <- 25000
seed <- 2345

######define parameters for gformula_survival call

id <- 'unique_id'
time_points <- 16
time_name <- 'time'

#time-varying covariates (TVC)
covnames <- c('marital_bin', 'employ_bin', 'occupation', 'industry', 'union_bin')

#baseline covariates
basecovs <- c('baseline_age', 'year', "gender", "race", "baseline_division",
"baseline_education", "baseline_parents_poor", "baseline_disability")

#outcome is already led two-years
outcome_name <- 'srh_bin_lead'

#TVC types
covtypes <- c('binary', 'binary', 'categorical', 'categorical', 'binary')

#type of lag variables for TVC
histories <- c(lagged)

#lag variables needed
histvars <- list(c('marital_bin', 'employ_bin', 'occupation', 'industry', 'union_bin'))

#models for TVC
#rms::rcs() can be used to model confounder with a restricted cubic spline
covmodels = c("marital_bin ~
               lag1_employ_bin +
               lag1_marital_bin +
```

```
                lag1_union_bin +
                lag1_occupation +
                lag1_industry +
                rms::rcs(baseline_age, c(28, 43, 59))  +
                baseline_division +
                race +
                gender +
                baseline_education +
                baseline_parents_poor +
                baseline_disability +
                as.factor(time) +
                year",
             "employ_bin ~
               marital_bin +
               lag1_employ_bin +
               lag1_marital_bin +
               lag1_union_bin +
               lag1_occupation +
               lag1_industry +
               rms::rcs(baseline_age, c(28, 43, 59))  +
               baseline_division +
               race +
               gender +
               baseline_education +
               baseline_parents_poor +
               baseline_disability +
               as.factor(time) +
               year",
             "occupation ~
               employ_bin +
               marital_bin +
               lag1_employ_bin +
               lag1_marital_bin +
               lag1_union_bin +
               lag1_occupation +
               lag1_industry +
               rms::rcs(baseline_age, c(28, 43, 59))  +
               baseline_division +
               race +
               gender +
               baseline_education +
               baseline_parents_poor +
               baseline_disability +
               as.factor(time) +
               year",
             "industry ~
               employ_bin +
               marital_bin +
               occupation +
               lag1_employ_bin +
               lag1_marital_bin +
               lag1_union_bin +
               lag1_occupation +
               lag1_industry +
               rms::rcs(baseline_age, c(28, 43, 59))  +
               baseline_division +
               race +
               gender +
               baseline_education +
               baseline_parents_poor +
               baseline_disability +
```

```
              as.factor(time) +
              year",
            "union_bin ~
              employ_bin +
              occupation +
              industry +
              marital_bin +
              lag1_employ_bin +
              lag1_marital_bin +
              lag1_union_bin +
              lag1_occupation +
              lag1_industry +
              rms::rcs(baseline_age, c(28, 43, 59)) +
              baseline_division +
              race +
              gender +
              baseline_education +
              baseline_parents_poor +
              baseline_disability +
              as.factor(time) +
              year")

#increase maxit for multinom models so they converge
covparams <- list(covmodels = lapply(covmodels, function (x) as.formula(x)),
                  control=c(NA, NA, list(maxit=10000), list(maxit=10000), NA))

#outcome model
ymodel <- srh_bin_lead ~
  employ_bin +
  occupation +
  industry +
  union_bin +
  marital_bin +
  lag1_employ_bin +
  lag1_occupation +
  lag1_industry +
  lag1_union_bin +
  lag1_marital_bin +
  rms::rcs(baseline_age, c(28, 43, 59)) +
  baseline_division +
  gender +
  race +
  baseline_education +
  baseline_parents_poor +
  baseline_disability +
  as.factor(time) +
  rms::rcs(year, c(1988, 1994, 2001, 2008, 2014))

#define exposure variable and scenario names
intvars <- list('union_bin', 'union_bin')
int_descript <- c('Always union if employed', "Never union if employed")

#define custom scenario: if employed (employ_bin > intval provided below), set intvar
(union_bin) to 1 (union member); if not employed (employ_bin < intval provided below),
set intvar (union_bin) to 0 (not union)
example_intervention <- function(newdf, pool, intvar, intvals, time_name, t){
  newdf[,(intvar) := 0]
  newdf[employ_bin > intvals[[1]], (intvar) := 1]
}
```

```
#Package also allows for incorporating deterministic knowledge about relationships
between time-varying covariates via the "restrictions" argument. For example, whenever
respondents are predicted to be "not employed", we could use the "restrictions" argument
to set occupation and industry to "not employed". However, even without using that
argument, no simulated observations (returned when "sim_data_b" argument set to "TRUE")
are predicted to have logical mismatches between variables (e.g., employ_bin=="employed"
but occupation=="not employed"), as such observations don't exist in the observed data.

######gformula survival function call

gform_basic <- gformula_survival(obs_data = dat_sub_first_worker_SRH,
                                 id = id,
                                 time_points = time_points,
                                 time_name = time_name,
                                 covnames = covnames,
                                 outcome_name = outcome_name,
                                 covtypes = covtypes,
                                 covparams = covparams,
                                 ymodel = ymodel,
                                 intvars = intvars,
                                 interventions = list(list(c(example_intervention,
0.99)), list(c(example_intervention, 1.01))), #in always-union scenario, if employ_bin is
greater than 0.99 (1=employed), union_bin is set to 1 (union); in never-union scenario,
union_bin is always set to 0 (non-union) because employ_bin is never greater than 1.01_
                                 int_descript = int_descript,
                                 histories = histories,
                                 histvars = histvars,
                                 basecovs = basecovs,
                                 nsimul=nsimul,
                                 seed=seed,
                                 nsamples=nsamples,
                                 parallel=TRUE,
                                 ncores=ncores,
                                 ref_int = 2)

######intervention results

results <- gform_basic$result[46:48,c(2,3,4,6,7,8,10,11,12,14,15)]
kable(results, digits=2) %>%
  kable_styling("striped")

######plotting functions (can also got default plots using plot(gform_basic)

###function for continuous variable

#variable names
cont_opts <- c("Marital", "Employment", "Union")

plot_cont <- function(var, num){

  out <- data.frame(gform_basic$dt_cov_plot[var])
  names(out) <- c('time', 'cov', 'legend')

  ggplot(out, aes(x=time*2, y=cov, group=legend, lty=legend)) +
    geom_line() +
    theme_light() +
    ylab(cont_opts[num]) +
    xlab('Time') +
    scale_y_continuous(limits=c(0, max(out$cov)),
labels=scales::number_format(accuracy=0.01))
```

```
}

###function for categorical variable

#variable names
cat_opts <- c("Occupation", "Industry")

plot_cat <- function(var, num){

  out <- data.frame(gform_basic$dt_cov_plot[var])
  names(out) <- c("t0", "V1", "var", "legend")

  out %>%
    group_by(t0, legend) %>%
    mutate(prop=V1 / sum(V1)) -> out

  ggplot(subset(out, c(t0==1 | t0==8 | t0==15)), aes(x=t0*2, y=prop,
group=interaction(var, legend))) +
    geom_bar(aes(lty=legend), stat="identity", position='dodge', fill='white',
color="black") +
    theme_light() +
    ylab(cat_opts[num]) +
    xlab('Time')+
    scale_x_discrete(limits=c(2, 16, 30)) +
    scale_y_continuous(labels=scales::number_format(accuracy=0.01))

}

###function for outcome variable

plot_out <- function(var){

  out <- data.frame(gform_basic[var])
  names(out) <- c('time', 'risk', 'survival', 'legend')

  ggplot(out, aes(x=time*2, y=risk, group=legend, lty=legend)) +
    geom_line() +
    theme_light() +
    theme(legend.title=element_blank(), legend.position='bottom',
legend.text=element_text(size=12)) +
    ylab('Cumulative incidence') +
    xlab('Time') +
    scale_y_continuous(limits=c(0, max(out$risk)),
labels=scales::number_format(accuracy=0.01)) +
    scale_linetype_discrete(labels=c("Nonparametric estimates", "Parametric estimates"))

}

######plot

mar <- plot_cont('marital_status_num', 1)
empl <- plot_cont('employ_num_bin', 2)
occ <- plot_cat('occ_broad_compl', 1)
ind <- plot_cat('ind_broad_compl', 2)
uni <- plot_cont('union_num_compl', 3)
out <- plot_out('dt_out_plot')
my_legend <- get_legend(out)

grid.arrange(arrangeGrob(mar + theme(legend.position = "none"),
                         empl + theme(legend.position = 'none'),
                         occ + theme(legend.position = 'none'),
```

```
                         ind + theme(legend.position = 'none'),
                         uni + theme(legend.position = 'none'),
                         out + theme(legend.position = 'none'),
                         nrow=3),
             my_legend, nrow=2, heights=c(10,1))
```

B4a. Specification of follow-up time and year in pooled parametric models fit in step 1 of parametric g-formula analyses of union and SRH.

		Marital	Employment	Occupation	Industry	Union	SRH
Overall	**Time**	FE[a]	FE	FE	FE	FE	FE
	Year	Linear	Linear	Linear	Linear	Binary	5-knot RCS[b]
Gender							
Women	**Time**	FE	FE	FE	FE	FE	FE
	Year	Linear	Linear	Linear	Linear	Linear	5-knot RCS
Men	**Time**	FE	FE	FE	FE	FE	FE
	Year	Linear	Linear	Linear	Linear	None	5-knot RCS
Gender-race							
Women of color	**Time**	FE	FE	FE	FE	FE	FE
	Year	5-knot RCS	5-knot RCS	5-knot RCS	5-knot RCS	5-knot RCS	5-knot RCS
White women	**Time**	FE	FE	FE	FE	FE	FE
	Year	Linear	Linear	Linear	Linear	Linear	5-knot RCS
Men of color	**Time**	FE	FE	FE	FE	3-knot RCS	FE
	Year	Linear	Linear	Linear	Linear	Binary	5-knot RCS
White men	**Time**	FE	FE	FE	FE	FE	FE
	Year	Linear	Linear	Linear	Linear	Binary	5-knot RCS
Gender-education							
≤HS women	**Time**	FE	FE	FE	FE	FE	FE
	Year	Linear	Linear	Linear	Linear	Linear	5-knot RCS
>HS women	**Time**	FE	FE	FE	FE	FE	FE
	Year	Linear	Linear	Linear	Linear	Linear	5-knot RCS
≤HS men	**Time**	FE	FE	FE	FE	FE	FE
	Year	Linear	Linear	Linear	Linear	None	5-knot RCS
>HS men	**Time**	FE	FE	FE	FE	FE	FE
	Year	Linear	Linear	Linear	Linear	Binary	5-knot RCS

Notes:
[a] Fixed effect.
[b] Restricted cubic spline.

B4b. Specification of follow-up time and year in pooled parametric models fit in step 1 of parametric g-formula analyses of union and mental illness.

		Marital	Employment	Occupation	Industry	Union	K6
Overall	**Time**	FE[a]	FE	FE	FE	FE	FE
	Year	FE	FE	FE	FE	Lin. + cat.[b]	Binary
Gender							
Women	**Time**	FE	FE	FE	FE	3-knot RCS[c] + binary	FE
	Year	FE	FE	FE	FE	Binary	Linear
Men	**Time**	FE	FE	FE	FE	FE	FE
	Year	FE	FE	FE	FE	Linear	Binary
Gender-race							
Women of color	**Time**	FE	FE	FE	FE	FE	FE
	Year	FE	FE	FE	FE	Linear	Linear
White women	**Time**	FE	FE	FE	FE	FE	FE
	Year	FE	FE	FE	FE	3-knot RCS	Linear
Men of color	**Time**	FE	FE	FE	FE	FE	FE
	Year	FE	FE	FE	FE	Lin. + quad.[d]	Binary
White men	**Time**	FE	FE	FE	FE	FE	FE
	Year	FE	FE	FE	FE	Lin. + quad.	Linear
Gender-education							
≤HS women	**Time**	FE	FE	FE	FE	FE	FE
	Year	FE	FE	FE	FE	Lin. + quad.	Linear
>HS women	**Time**	FE	FE	FE	FE	3-knot RCS	FE
	Year	FE	FE	FE	FE	FE	Binary
≤HS men	**Time**	FE	FE	FE	FE	FE	FE
	Year	FE	FE	FE	FE	3-knot RCS	Binary
>HS men	**Time**	FE	FE	FE	FE	FE	FE
	Year	FE	FE	FE	FE	Linear	Binary

Notes:

[a] Fixed effect.

[b] Linear term and 5-level categorical term.

[c] Restricted cubic spline.

[d] Linear and quadratic terms.

B5. Trends in prevalence of union membership among employed respondents in SRH sample over study period by demographic group, occupation, and industry.

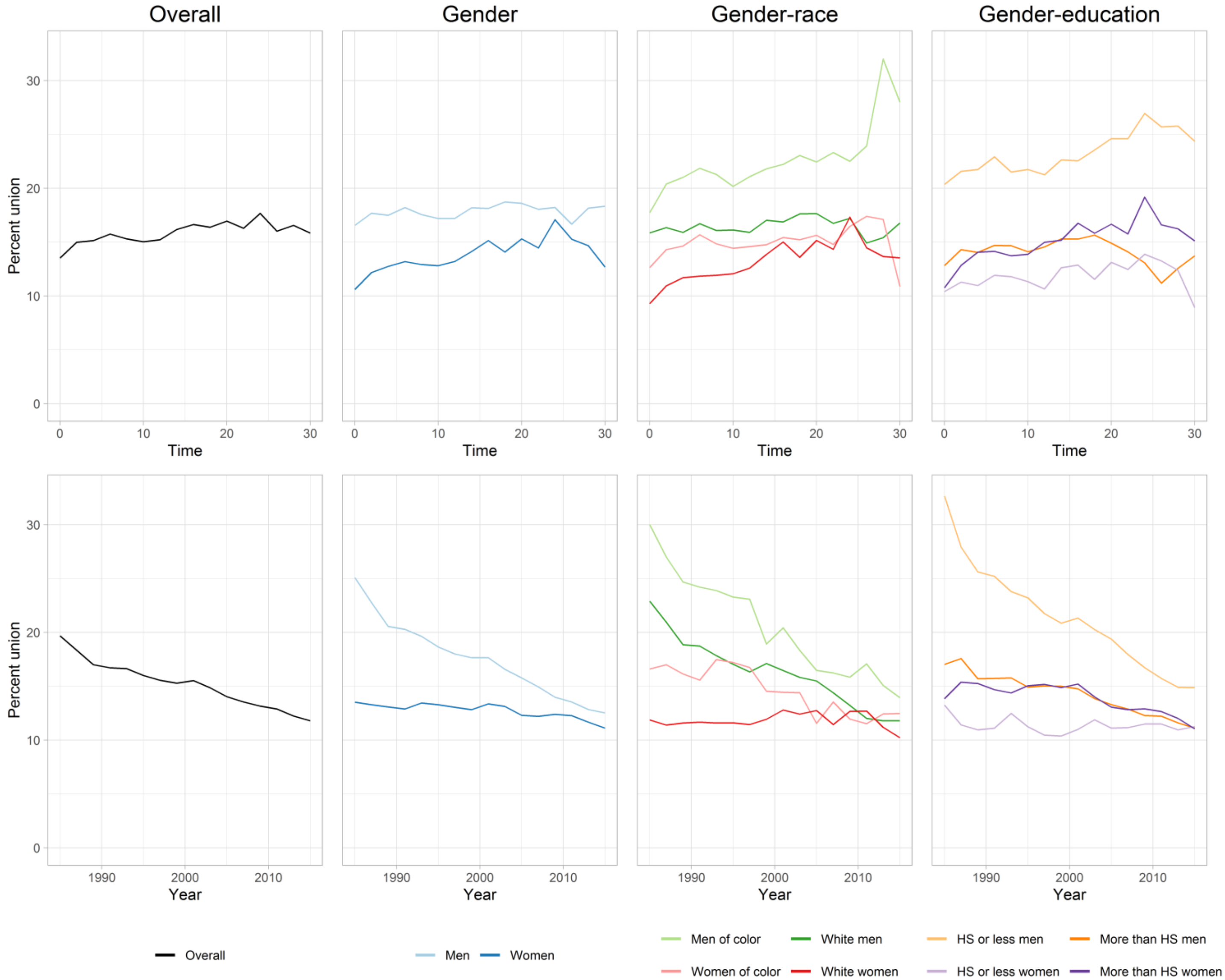


Figure B5a. Trends in prevalence of union membership among employed respondents in SRH sample by follow-up time (top row) and calendar year (bottom row) overall and in each gender, gender-race, and gender-education subgroup.

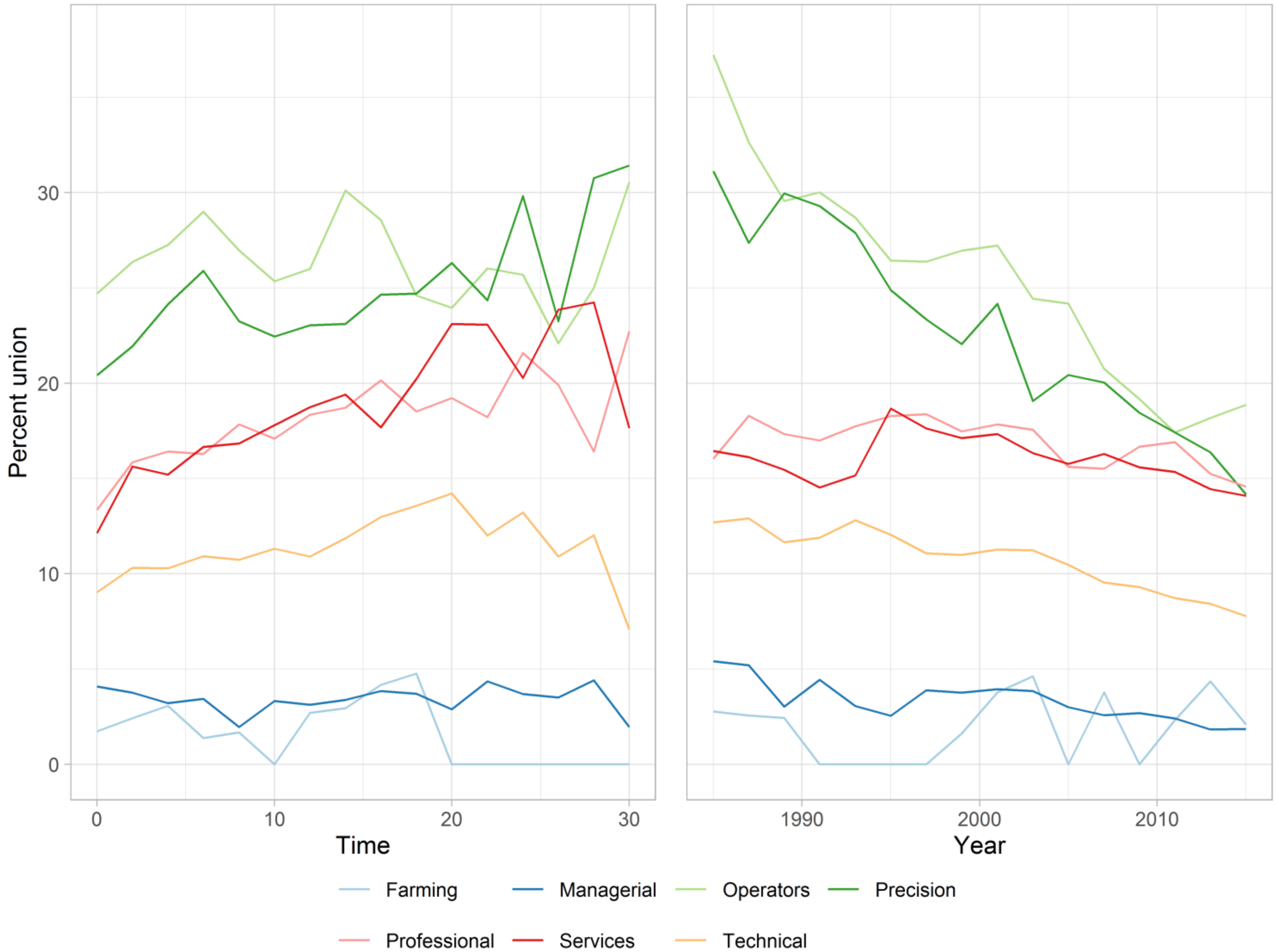


Figure B5b. Trends in prevalence of union membership among employed respondents in SRH sample by follow-up time (left) and calendar year (right) in each occupation.

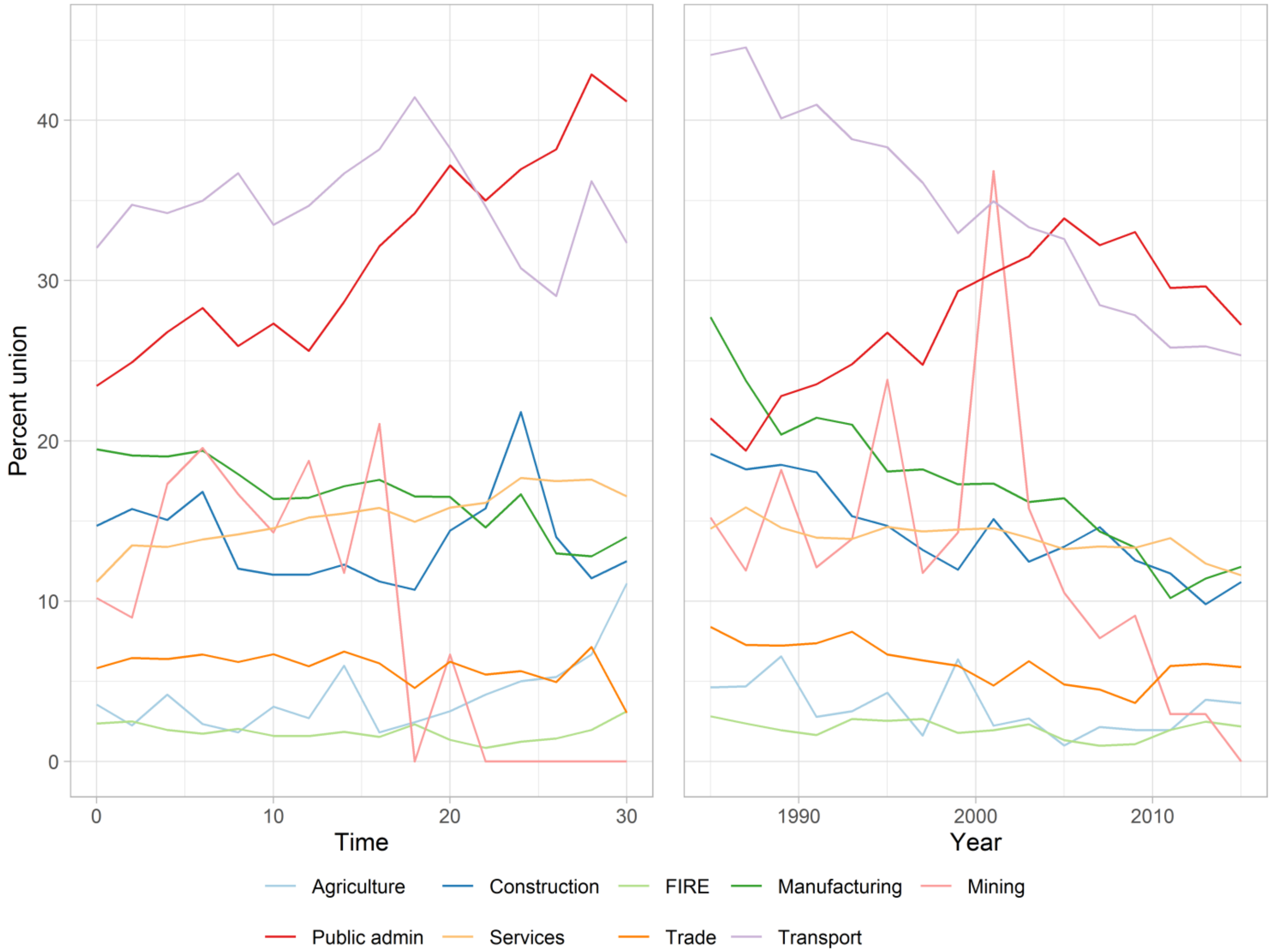


Figure B5c. Trends in prevalence of union membership among employed respondents in SRH sample by follow-up time (left) and calendar year (right) in each industry.

B6a. Parametric g-formula estimate of 32-year risk of poor/fair SRH in scenario (sc.) setting all employed-person-years to union-member employed-person-years (scenario 1) relative to 32-year risk in scenario setting no employed-person-years to union-member employed-person-years (scenario 2). Unlike in analyses presented in main text, union membership was unlagged.

	Respondents[a]	Observations[a]	Sc.	Risk	RR	95% CI		RD	95% CI	
Overall	16,719	104,141	1	0.48	1.00	0.94	1.07	0.00	-0.03	0.03
			2	0.48						
Gender										
Women	8,525	53,813	1	0.51	1.07	0.98	1.15	0.03	-0.01	0.07
			2	0.48						
Men	8,194	50,328	1	0.46	0.95	0.88	1.01	-0.03	-0.06	0.01
			2	0.48						
Gender-race										
Women of color[b]	3,349	18,904	1	0.61	1.03	0.92	1.14	0.02	-0.04	0.08
			2	0.59						
White women[c]	5,138	34,625	1	0.46	1.11	0.99	1.25	0.05	0.00	0.10
			2	0.41						
Men of color	3,000	15,609	1	0.58	0.92	0.83	1.01	-0.05	-0.11	0.01
			2	0.63						
White men	5,194	34,719	1	0.38	0.96	0.85	1.06	-0.02	-0.06	0.02
			2	0.39						
Gender-education										
≤HS women[d]	3,900	23,469	1	0.65	1.09	0.97	1.20	0.05	-0.02	0.12
			2	0.59						
>HS women[e]	4,555	29,761	1	0.40	1.08	0.95	1.24	0.03	-0.02	0.08
			2	0.37						
≤HS men	4,040	22,644	1	0.58	0.96	0.89	1.04	-0.02	-0.07	0.02
			2	0.61						
>HS men	4,154	27,684	1	0.30	0.93	0.75	1.11	-0.02	-0.08	0.03
			2	0.32						

Notes:

Risk ratio and risk difference estimates compare risk in scenario 1 relative to risk in scenario 2. Confidence intervals calculated from non-parametric bootstrap with 250 repetitions. Subgroup estimates produced from stratified models.

[a] Unique respondents and observations in original Panel Study of Income Dynamics dataset used to fit pooled time-varying exposure, confounder, and outcome models. Monte Carlo pseudo-sample used in simulations had 25,000 respondents.

[b] Analysis excluded 8 respondents ever employed in “mining” industry due to small counts in that stratum, which produced bootstrap samples with 0 mining-industry respondents.

[c] Analysis excluded 30 respondents ever employed in “mining” industry.

[d] Analysis excluded 17 respondents ever employed in “mining” industry.

[e] Analysis excluded 53 respondents ever employed in “mining” industry or “farming, forestry, and fishing’ occupation.

B6b. Parametric g-formula estimate of 16-year risk of moderate mental illness (K6≥5) in scenario (sc.) setting all employed-person-years to union-member employed-person-years (scenario 1) relative to 16-year risk in scenario setting no employed-person-years to union-member employed-person-years (scenario 2). Unlike in analyses presented in main text, union membership was unlagged.

	Respondents[a]	Observations[a]	Sc.	Risk	RR	95% CI		RD	95% CI	
Overall	5,813	26,733	1	0.44	1.06	0.96	1.17	0.03	-0.02	0.07
			2	0.42						
Gender										
Women[b]	3,369	15,519	1	0.55	1.09	0.97	1.23	0.05	-0.01	0.11
			2	0.51						
Men	2,437	11,172	1	0.38	1.02	0.88	1.20	0.01	-0.05	0.07
			2	0.38						
Gender-race										
Women of color[c]	1,528	6,943	1	0.55	1.02	0.86	1.18	0.01	-0.08	0.10
			2	0.54						
White women[d]	1,829	8,528	1	0.57	1.16	0.98	1.35	0.08	-0.01	0.17
			2	0.49						
Men of color[e]	880	3,771	1	0.38	0.87	0.67	1.13	-0.06	-0.17	0.06
			2	0.44						
White men	1,553	7,388	1	0.44	1.10	0.87	1.33	0.04	-0.05	0.12
			2	0.40						
Gender-education										
≤HS women[f]	1,337	6,093	1	0.56	1.03	0.84	1.21	0.02	-0.09	0.11
			2	0.54						
>HS women[g]	2,025	9,397	1	0.47	1.13	0.96	1.34	0.05	-0.02	0.14
			2	0.41						
≤HS men	979	4,302	1	0.46	1.10	0.88	1.33	0.04	-0.05	0.13
			2	0.42						
>HS men[h]	1,446	6,812	1	0.39	0.94	0.72	1.15	-0.03	-0.13	0.06
			2	0.42						

Notes:
Risk ratio (RR) and risk difference (RD) estimates compare risk in scenario 1 relative to risk in scenario 2. Confidence intervals calculated from non-parametric bootstrap with 250 repetitions. Subgroup estimates produced from stratified models.
[a] Unique respondents and observations in original Panel Study of Income Dynamics dataset used to fit pooled time-varying exposure, confounder, and outcome models. Monte Carlo pseudo-sample used in simulations had 25,000 respondents.
[b] Analysis excluded 7 respondents ever employed in “mining” industry due to small counts in that stratum, which produced bootstrap samples with 0 mining-industry respondents.
[c] Analysis excluded 2 respondents ever employed in “mining” industry.
[d] Analysis excluded 17 respondents ever employed in “mining” industry or “farming, forestry and fishing” occupation.
[e] Analysis excluded 4 respondents ever employed in “mining” industry.
[f] Analysis excluded 3 respondents ever employed in “mining” industry.
[g] Analysis excluded 11 respondents ever employed in “farming, forestry, and fishing” occupation.
[h] Analysis excluded 12 respondents ever employed in “farming, forestry, and fishing” occupation.

B7. Estimating sub-distribution risks rather than treating death as a censoring event.

In our primary analyses, we treated death as a censoring event. Consequently, because our parametric g-formula approach eliminated censoring, our analyses estimated the effects of scenarios in a hypothetical cohort in which death were impossible. In a real-world cohort, certain respondents may have died before developing our outcomes of interest. Thus, the cumulative incidences of the outcomes in our hypothetical cohort are likely greater than what would be observed in a real-world cohort.[1,2] Although unrealistic, this may be useful for etiologic research such as ours, which sought to elucidate the direct health effects of a novel exposure.[2]

An alternative approach to treating death as a censoring event (and thus to estimating "cause-specific risks") is to estimate the subdistribution risks of the outcomes of interest in the presence of death.[2] This allows for the estimation of cumulative incidences in a more realistic hypothetical cohort in which a competing event like mortality is present *and* may be affected by exposure.[2,3] Effect estimates in such a setting capture both direct health effects of the exposure, as well as indirect health effects that operate through the competing event (e.g., if union membership has no direct effect on SRH, it may still indirectly increase risk of poor/fair SRH if it decreases risk of death, as certain respondents would have otherwise died before developing the outcome of interest).[2] The cause-specific and subdistribution approaches are only equivalent when the risk of the competing event is zero, although the approaches may approximate each other when the risk of the competing event is low.[2]

To examine the extent to which treating death as a censoring event affected our estimates, we re-ran our unstratified analyses estimating subdistribution risks. To do so, we followed the same approach described in the main text, but in the model-fitting stage, we fit an additional pooled logistic model for death, which we then used in the simulation stage to predict death at each wave.[4] This model was specified similarly to the pooled logistic models we used to predict SRH or mental illness. However, in the K6 analyses, we did not include lagged values of time-varying covariates, nor did we adjust for baseline disability status or childhood socioeconomic status, because the small number of deaths during follow-up prevented fully-adjusted models from converging.

The estimates from the sub-distribution analyses were similar to those from the primary analyses (Table B7), partially because death was rare during follow-up (unsurprising given our cohort's 25-64-year-old age range). Specifically, in the SRH analyses, only 191 respondents died before developing poor/fair SRH, being lost to follow-up, or being administratively censored (compared with 3,878 incident cases of poor/fair SRH), while in the mental-illness analyses, only 45 respondents died before developing moderate mental illness, being lost to follow-up, or being administratively censored (compared with 1,981 incident cases of moderate mental illness).

Table B7. Parametric g-formula estimates of risk of poor/fair SRH and moderate mental illness by the end of follow-up (32 years and 16 years respectively) in scenario (sc.) setting all two-year-lagged employed-person-years to union-member employed-person-years (scenario 1) relative to risk in scenario setting no two-year-lagged employed-person-years to union-member employed-person-years (scenario 2). Analyses estimated subdistribution risks rather than treated death as a censoring event.

	Respondents[a]	**Observations[a]**	**Sc.**	**Risk**	**RR**	**95% CI**		**RD**	**95% CI**	
Overall										
SRH	16,717	87,606	1	0.46	1.03	0.97	1.09	0.01	-0.02	0.04
			2	0.45						
Mental illness[b]	5,774	20,791	1	0.47	1.03	0.95	1.14	0.01	-0.02	0.06
			2	0.45						

Notes:
Risk ratio (RR) and risk difference (RD) estimates compare risk in scenario 1 relative to risk in scenario 2. Confidence intervals calculated from non-parametric bootstrap with 250 repetitions.
[a] Unique respondents and observations in original Panel Study of Income Dynamics dataset used to fit pooled time-varying exposure, confounder, outcome, and competing event models. Dataset excluded 0.4% of respondents in PSID's mortality file whose deaths were only known to have occurred within a three-plus-year range. Monte Carlo pseudo-sample used in simulations had 25,000 respondents.
[b] Analysis excluded 38 respondents ever employed in "mining" industry because there 0 deaths in that stratum.

References

1. Westreich, Daniel. From exposures to population interventions: pregnancy and response to HIV therapy. *Am J Epidemiol*. 2014;178(7):797-806. doi:10.1093/aje/kwt328
2. Lau B, Cole SR, Gange SJ. Competing risk regression models for epidemiologic data. *Am J Epidemiol*. 2009;170(2):244-256. doi:10.1093/aje/kwp107
3. Neophytou AM, Picciotto S, Brown DM, Gallagher LE, Checkoway H, Eisen EA, Costello S. Estimating counterfactual risk under hypothetical interventions in the presence of competing events:

crystalline silica exposure and mortality from 2 causes of death. *Am J Epidemiol*. 2018;187(9):1942-1950. doi: 10.1093/aje/kwy077.

4. Lin V, McGrath S, Zhang Z, et al. gfoRmula: an R package for estimating effects of general time-varying treatment interventions via the parametric g-formula. 2019. http://arxiv.org/abs/1908.07072.

B8a. Parametric g-formula estimates of 32-year risk (i.e., cumulative incidence) of poor/fair SRH in scenario (sc.) setting all two-year-lagged employed-person-years to union-member employed-person-years (scenario 1) relative to risk in scenario setting no two-year-lagged employed-person-years to union-member employed-person-years (scenario 2). Unlike in analyses presented in main text, occupation and industry treated as baseline covariates.

	Respondents[a]	Observations[a]	Sc.	Risk	RR	95% CI		RD	95% CI	
Overall	16,719	87,422	1	0.47	1.01	0.95	1.08	0.00	-0.03	0.04
			2	0.46						
Gender										
Women	8,525	45,288	1	0.50	1.10	1.00	1.18	0.05	0.00	0.08
			2	0.45						
Men	8,194	42,134	1	0.45	0.94	0.87	1.02	-0.03	-0.07	0.01
			2	0.48						
Gender-race										
Women of color[b]	3,354	15,586	1	0.62	1.09	0.97	1.22	0.05	-0.02	0.12
			2	0.57						
White women[c]	5,155	29,622	1	0.42	1.09	0.93	1.25	0.03	-0.03	0.09
			2	0.38						
Men of color	3,000	12,609	1	0.55	0.89	0.79	0.98	-0.07	-0.13	-0.01
			2	0.62						
White men	5,194	29,525	1	0.39	0.99	0.88	1.11	-0.01	-0.05	0.04
			2	0.39						
Gender-education										
≤HS women[d]	3,911	19,646	1	0.63	1.17	1.05	1.27	0.09	0.03	0.15
			2	0.54						
>HS women[e]	4,592	25,538	1	0.39	1.08	0.93	1.22	0.03	-0.03	0.08
			2	0.36						
≤HS men	4,040	18,604	1	0.56	0.95	0.86	1.04	-0.03	-0.08	0.03
			2	0.59						
>HS men	4,154	23,530	1	0.32	0.95	0.81	1.11	-0.02	-0.07	0.04
			2	0.34						

Notes:

Risk ratio and risk difference estimates compare risk in scenario 1 relative to risk in scenario 2. Confidence intervals calculated from non-parametric bootstrap with 250 repetitions. Subgroup estimates produced from stratified models.

[a] Unique respondents and observations in original Panel Study of Income Dynamics dataset used to fit pooled time-varying exposure, confounder, and outcome models. Monte Carlo pseudo-sample used in simulations had 25,000 respondents.

[b] Analysis excluded 3 respondents employed in “mining” industry at baseline due to small counts in that stratum, which produced bootstrap samples with 0 mining-industry respondents.

[c] Analysis excluded 13 respondents employed in “mining” industry.

[d] Analysis excluded 6 respondents employed in “mining” industry.

[e] Analysis excluded 16 respondents employed in “mining” industry or “farming, forestry, and fishing’ occupation.

B8b. Parametric g-formula estimate of 16-year risk of moderate mental illness (K6≥5) in scenario (sc.) setting all two-year-lagged employed-person-years to union-member employed-person-years (scenario 1) relative to 16-year risk in scenario setting no two-year-lagged employed-person-years to union-member employed-person-years (scenario 2). Unlike in analyses presented in text, occupation and industry treated as baseline covariates.

	Respondents[a]	Observations[a]	Sc.	Risk	RR	95% CI		RD	95% CI	
Overall	5,813	20,920	1	0.46	1.02	0.92	1.13	0.01	-0.04	0.06
			2	0.45						
Gender										
Women	3,376	12,185	1	0.54	1.05	0.92	1.19	0.03	-0.04	0.09
			2	0.52						
Men	2,437	8,735	1	0.41	0.98	0.83	1.15	-0.01	-0.07	0.06
			2	0.42						
Gender-race										
Women of color[b]	1,529	5,420	1	0.51	0.92	0.75	1.09	-0.04	-0.14	0.05
			2	0.55						
White women[c]	1,838	6,743	1	0.58	1.17	0.99	1.35	0.09	-0.01	0.17
			2	0.49						
Men of color[d]	871	2,872	1	0.49	0.88	0.68	1.08	-0.07	-0.18	0.04
			2	0.56						
White men	1,553	5,835	1	0.44	1.07	0.85	1.29	0.03	-0.07	0.12
			2	0.41						
Gender-education										
≤HS women[e]	1,339	4,769	1	0.59	1.10	0.87	1.29	0.06	-0.07	0.15
			2	0.54						
>HS women[f]	2,032	7,400	1	0.46	1.01	0.85	1.19	0.01	-0.07	0.08
			2	0.46						
≤HS men[g]	972	3,302	1	0.54	1.11	0.89	1.33	0.06	-0.05	0.16
			2	0.49						
>HS men[h]	1,443	5,369	1	0.39	0.90	0.65	1.14	-0.04	-0.14	0.05
			2	0.43						

Notes:
Risk ratio (RR) and risk difference (RD) estimates compare risk in scenario 1 relative to risk in scenario 2. Confidence intervals calculated from non-parametric bootstrap with 250 repetitions. Subgroup estimates produced from stratified models.
[a] Unique respondents and observations in original Panel Study of Income Dynamics dataset used to fit pooled time-varying exposure, confounder, and outcome models. Monte Carlo pseudo-sample used in simulations had 25,000 respondents.
[b] Analysis excluded 1 respondent employed in “mining” industry at baseline due to small counts in that stratum, which produced bootstrap samples with 0 mining-industry respondents.
[c] Analysis excluded 8 respondents employed in “mining” industry or “farming, forestry, and fishing” occupation.
[d] Analysis excluded 13 respondents employed in “mining” industry or “farming, forestry, and fishing” occupation.
[e] Analysis excluded 1 respondents employed in “mining” industry.
[f] Analysis excluded 4 respondents employed in “mining” industry or “farming, forestry, and fishing” occupation.
[g] Analysis excluded 7 respondents employed in “mining” industry or “farming, forestry, and fishing” occupation.
[h] Analysis excluded 11 respondents employed in “mining” industry or “farming, forestry, and fishing” occupation.

B9. Parametric g-formula estimates of risk of poor/fair SRH and moderate mental illness by the end of follow-up (32 years and 16 years respectively) in scenario (sc.) setting all two-year-lagged employed-person-years to union-member employed-person-years (scenario 1) relative to cumulative incidence in scenario setting no two-year-lagged employed-person-years to union-member employed-person-years (scenario 2). Unlike in analyses presented in main text, the exposure, time-varying confounder, and outcome models include baseline state of residence as a covariate rather than baseline division of residence.

	Respondents[a]	Observations[a]	Sc.	Risk	RR	95% CI		RD	95% CI	
Overall										
SRH	16,719	87,422	1	0.47	1.01	0.94	1.08	0.00	-0.03	0.04
			2	0.46						
Mental illness[b]	5,746	20,716	1	0.46	1.01	0.90	1.12	0.00	-0.05	0.05
			2	0.45						

Notes:
Risk ratio and risk difference estimates compare risk in scenario 1 relative to risk in scenario 2. Confidence intervals calculated from non-parametric bootstrap with 250 repetitions.
[a] Unique respondents and observations in original Panel Study of Income Dynamics dataset used to fit pooled time-varying exposure, confounder, and outcome models. Monte Carlo pseudo-sample used in simulations had 25,000 respondents.
[b] Analysis excluded the 67 respondents living in AK, DE, HI, MT, NH, NM, ND, RI, VT, WV, or WY at baseline because there were too few respondents in those strata, which produced bootstrap samples with 0 respondents from those states.

B10. Hazard of poor/fair SRH or moderate mental illness (K6≥5) among respondents who were union workers at baseline relative to the hazard among respondents who were non-union workers at baseline from Cox proportional hazards models. Subgroup estimates produced from stratified models.

	SRH				Mental illness			
	N	**HR**	**95% CI**		**N**	**HR**	**95% CI**	
Overall	16,719	1.02	0.93	1.12	5,813	1.12	0.97	1.28
Gender								
Women	8,525	1.06	0.92	1.22	2,437	1.14	0.95	1.36
Men	8,194	0.99	0.87	1.12	3,376	1.11	0.88	1.39
Gender-race								
Women of color	3,357	1.01	0.84	1.22	1,530	0.95	0.75	1.22
White women	5,168	1.09	0.87	1.37	1,846	1.34	1.03	1.75
Men of color	3,000	0.95	0.78	1.15	884	1.03	0.72	1.48
White men	5,194	1.02	0.86	1.21	1,553	1.19	0.89	1.60
Gender-education								
≤HS women	3,917	1.04	0.87	1.25	1,340	1.32	1.00	1.73
>HS women	4,608	1.11	0.89	1.39	2,036	1.04	0.82	1.32
≤HS men	4,040	0.94	0.81	1.10	979	1.16	0.84	1.61
>HS men	4,154	1.07	0.86	1.34	1,458	1.06	0.77	1.47

Notes:
Estimates from Cox proportional hazards models adjusted for confounders described in main text. Confidence intervals calculated using robust standard errors.

B11. Union membership misclassification.

Card (1996) found that approximately 2.5% of Current Population Survey respondents in 1977 misreported their union status, true union status aside. Given a true union prevalence TU and a misclassification rate MR, we can calculate the proportion of "union" respondents who are truly non-union at an observed union prevalence OU using the following equations:

- Non-union respondents misclassified as union (NUU) = (1 – TU) * MR
- Union respondents misclassified as non-union (UNU) = TU * MR
- OU = TU + NUU – UNU
- Proportion of "union" respondents who are truly non-union = NUU / OU

In our study, given Card's 2.5% misclassification rate and an observed union-membership prevalence of 14% (the prevalence in our SRH analyses), approximately 16% of workers classified as union would be truly non-union. The proportion of respondents misclassified would be even higher at lower union-membership prevalences, such as the prevalence observed in our mental-illness analyses (12%).

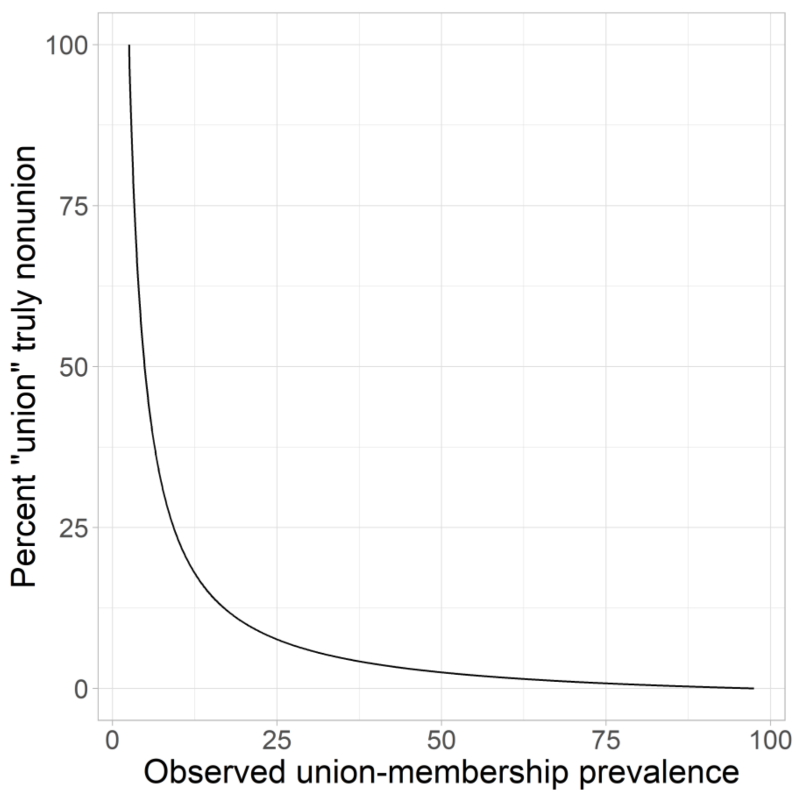
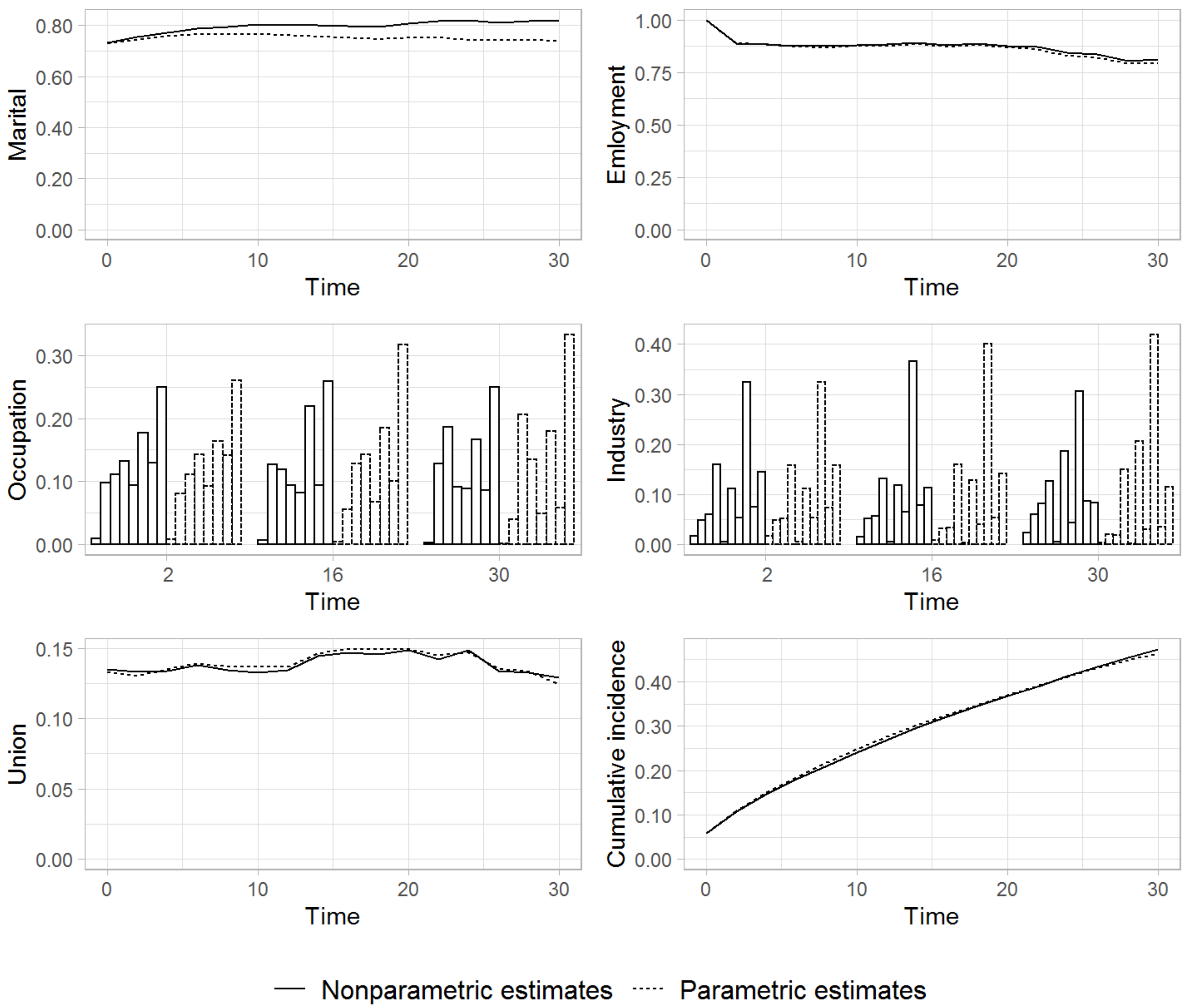


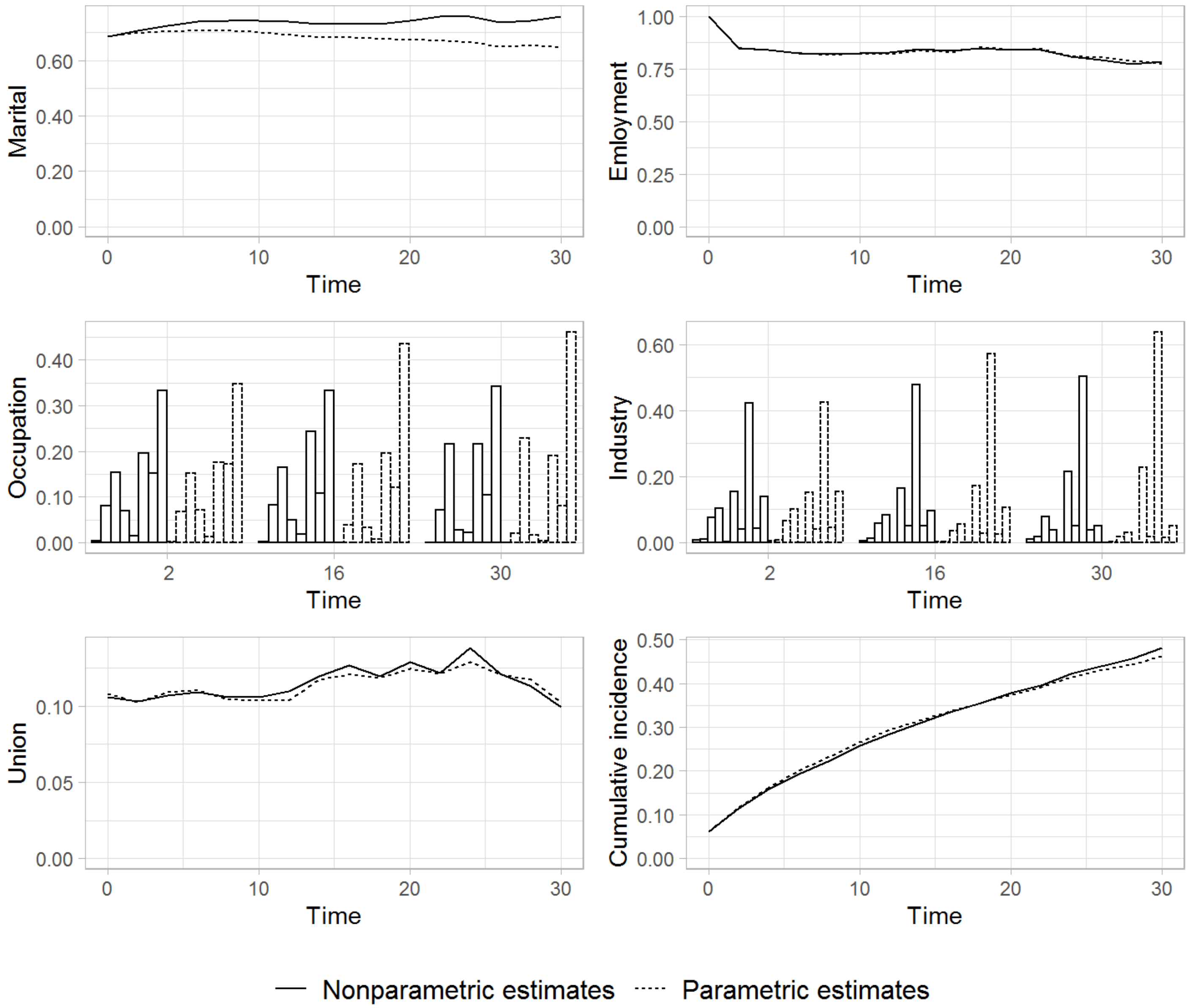
Figure B11. Proportion of respondents misclassified as "union" who were truly "non-union" at various union-membership prevalences, assuming a misclassification rate of 2.5%.

B12. Simulated (parametric) versus observed (nonparametric) distributions of exposure and time-varying covariates for overall SRH analyses and for subgroup SRH analyses.

Overall



Women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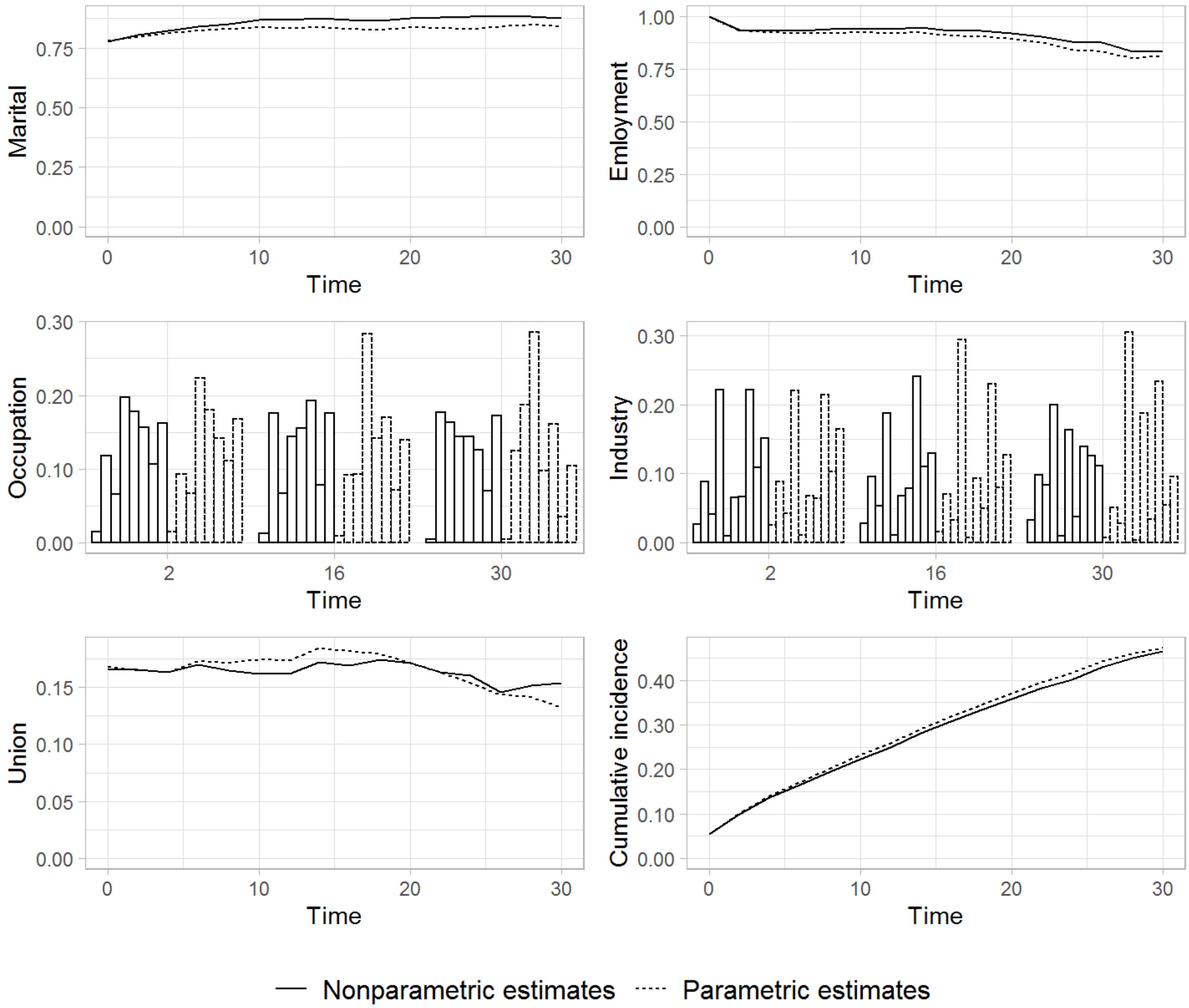
Time
Nonparametric estimates
Parametric estimates

Men



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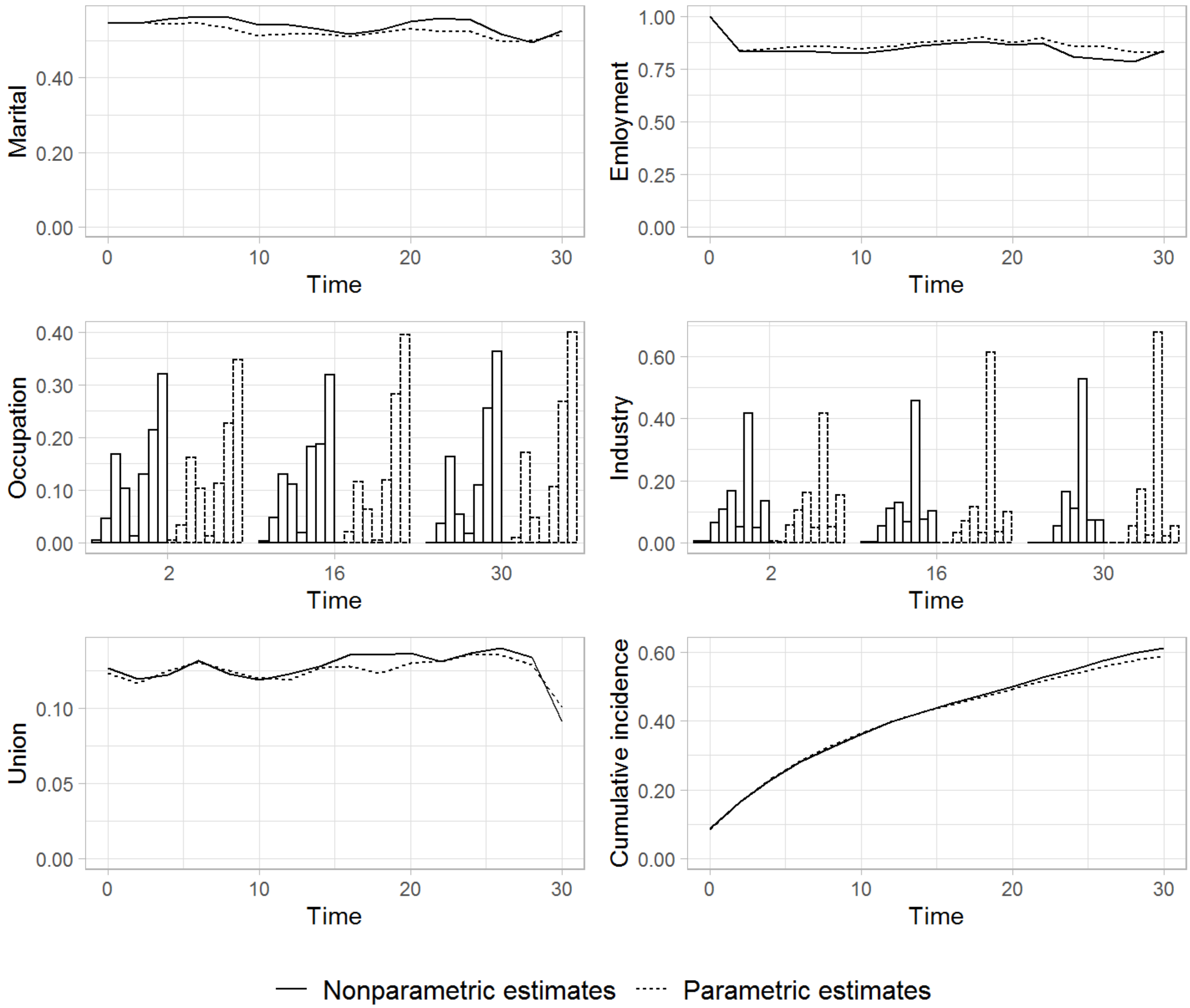
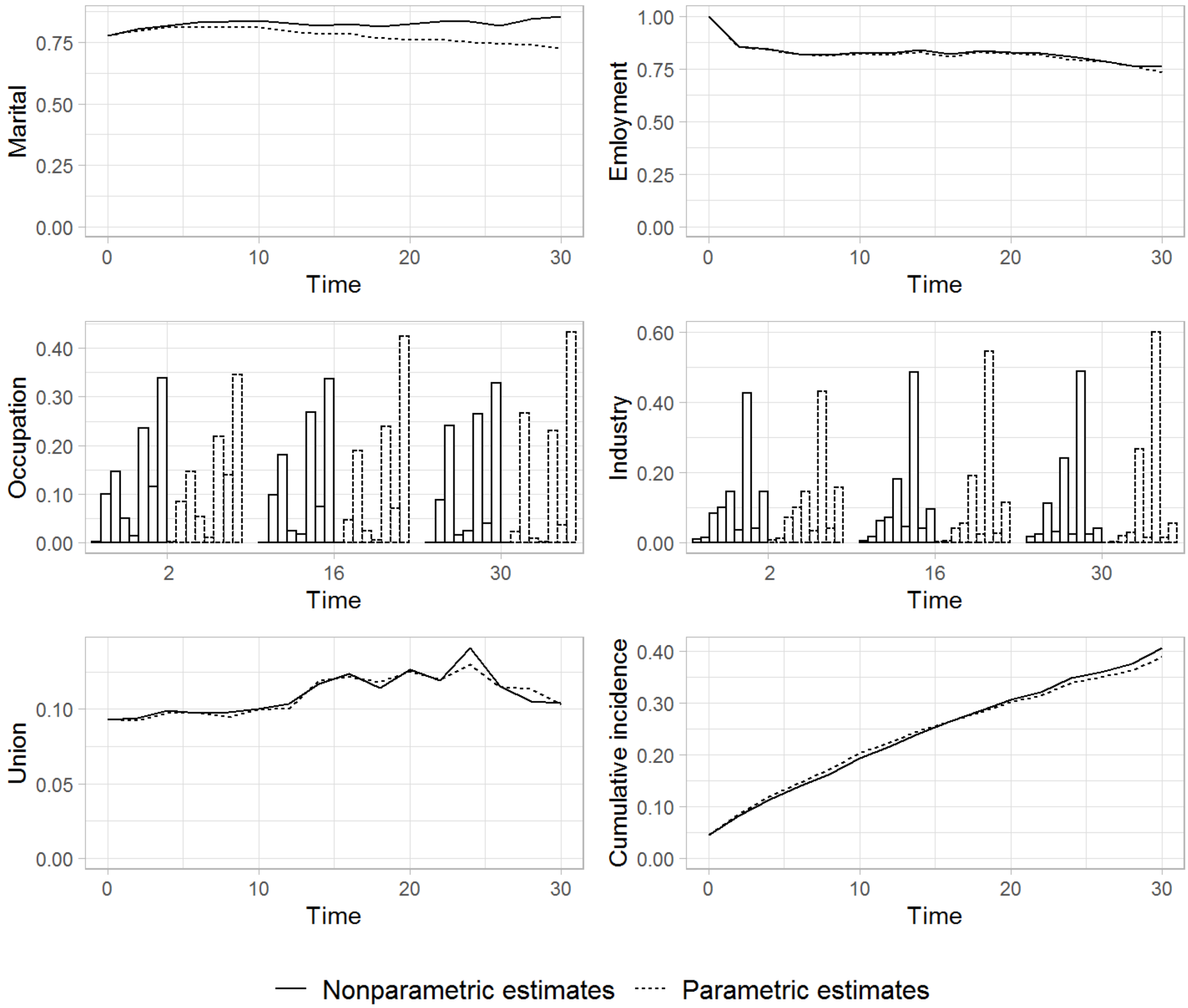
Time
Nonparametric estimates
Parametric estimates

Women of color



White women



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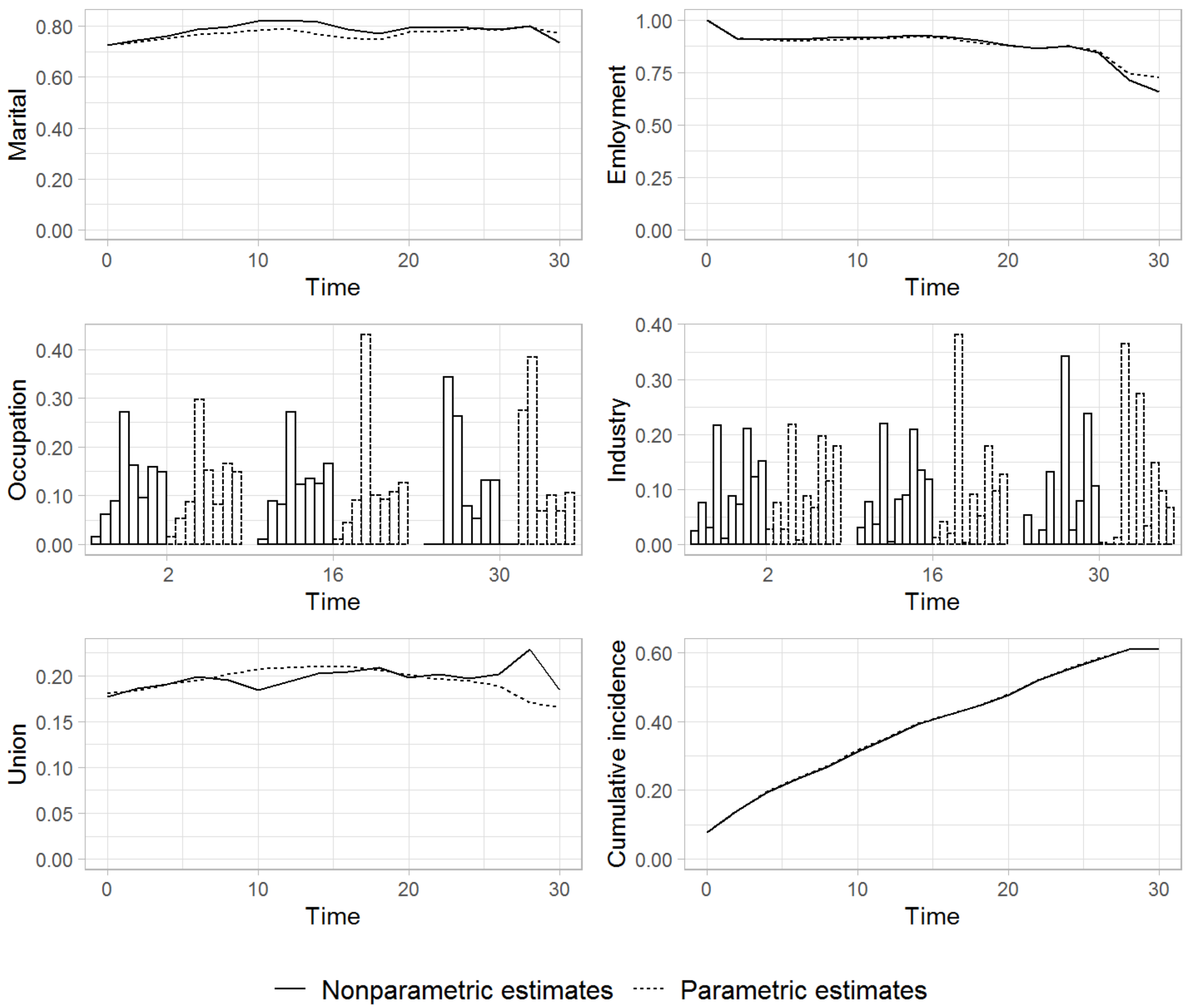
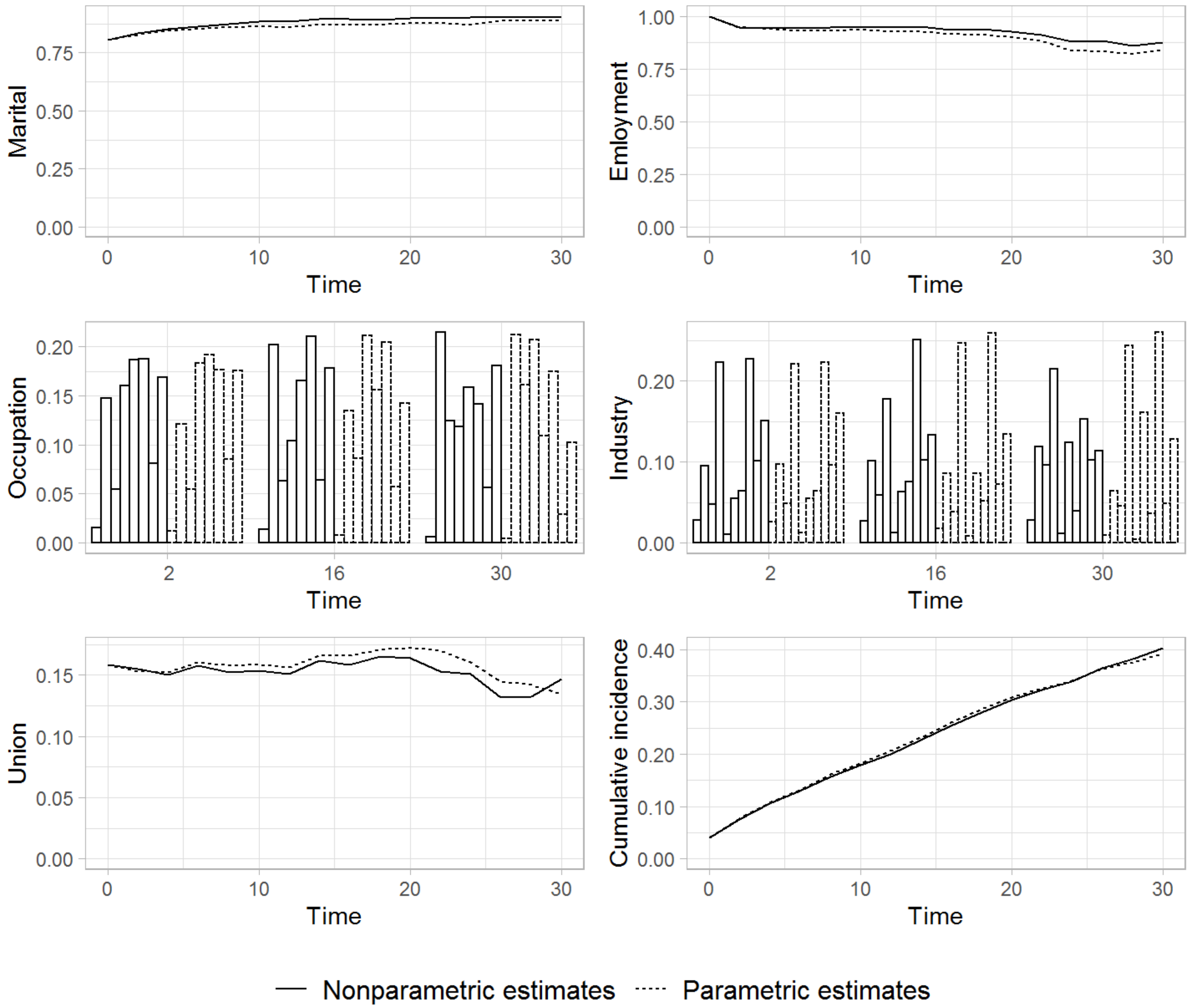
Nonparametric estimates
Parametric estimates

Men of color

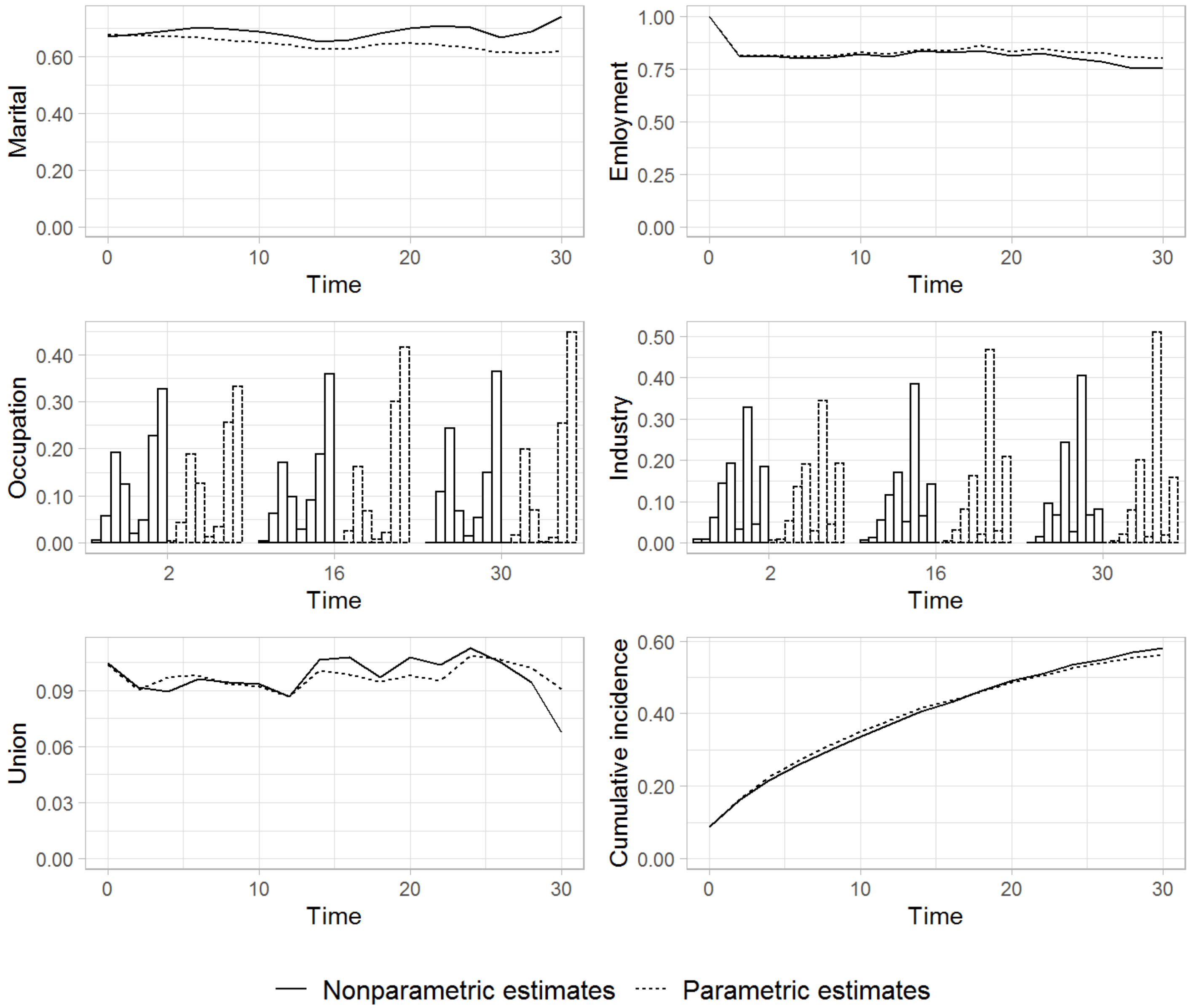


Marital
Time
Emloyment
Time
Occupation
Time
Industry
Time
Union
Time
Cumulative incidence
Time
Nonparametric estimates
Parametric estimates

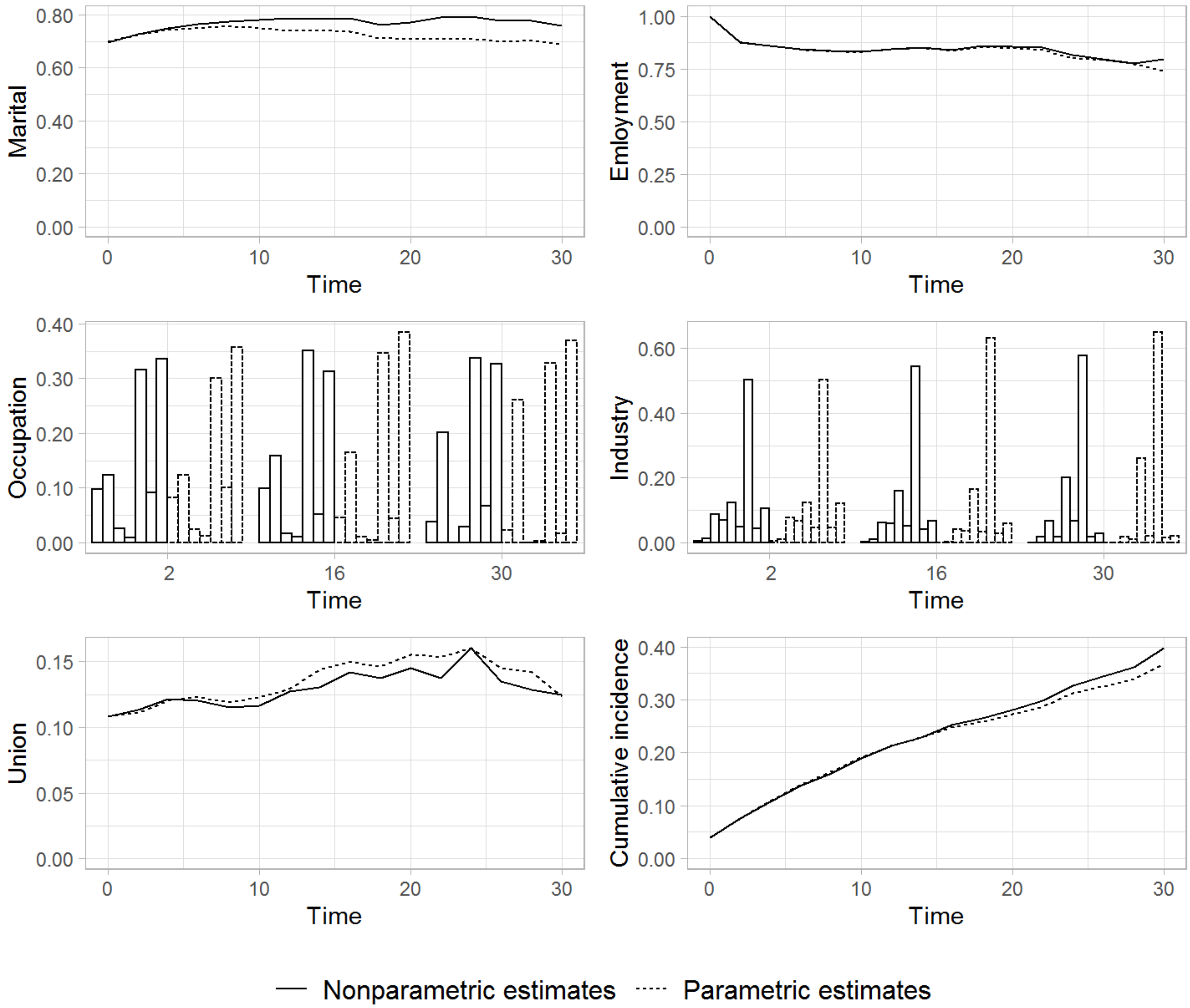
White men


Marital
0.75
0.50
0.25
0.00
0
10
20
30
Time
Emloyment
1.00
0.75
0.50
0.25
0.00
Occupation
0.20
0.15
0.10
0.05
2
16
Industry
Union
0.15
0.10
0.05
Cumulative incidence
0.40
0.30
0.20
Nonparametric estimates
Parametric estimates

≤HS women

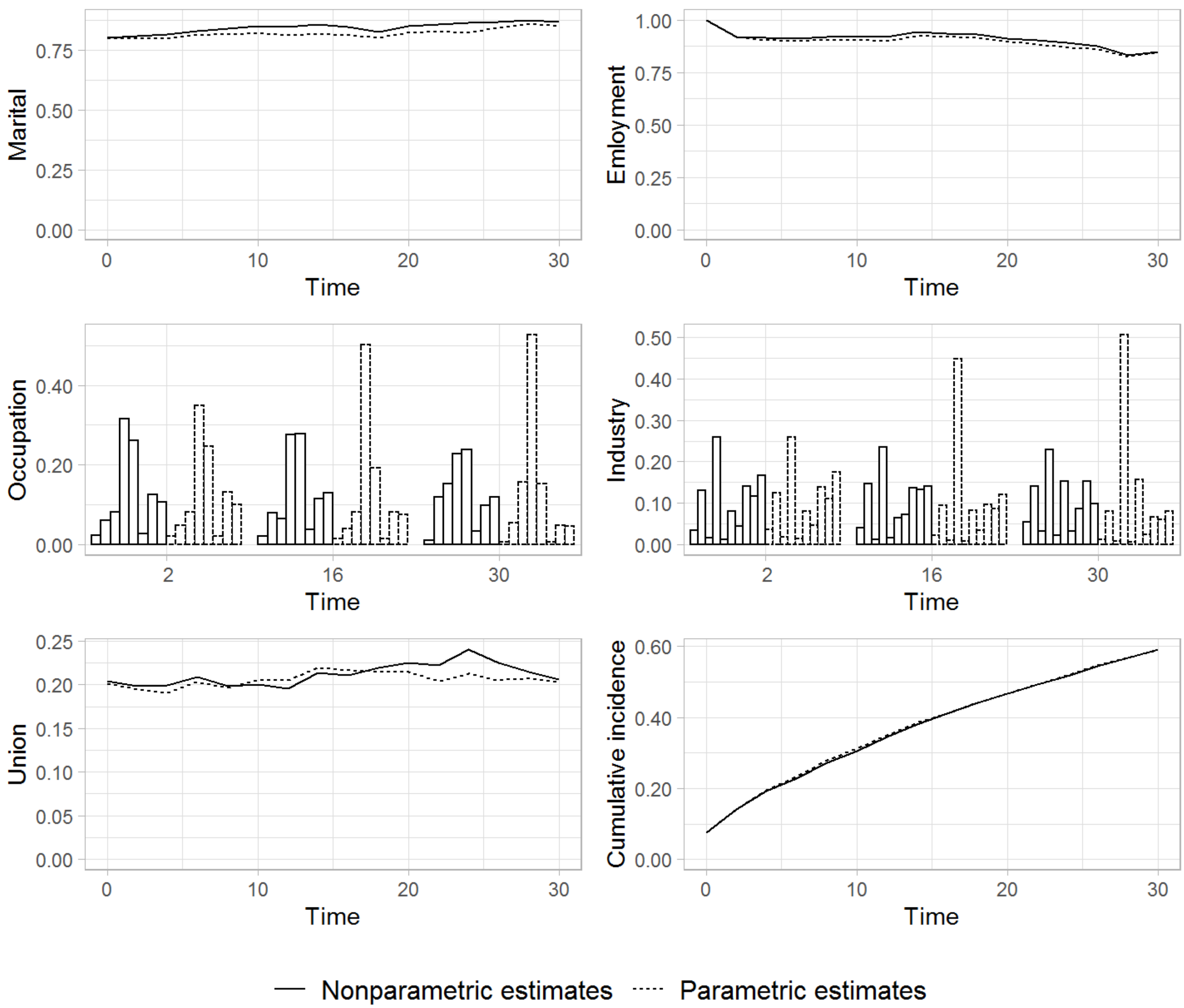

Marital
Emloyment
Occupation
Industry
Union
Cumulative incidence
Time
Nonparametric estimates
Parametric estimates

>HS women



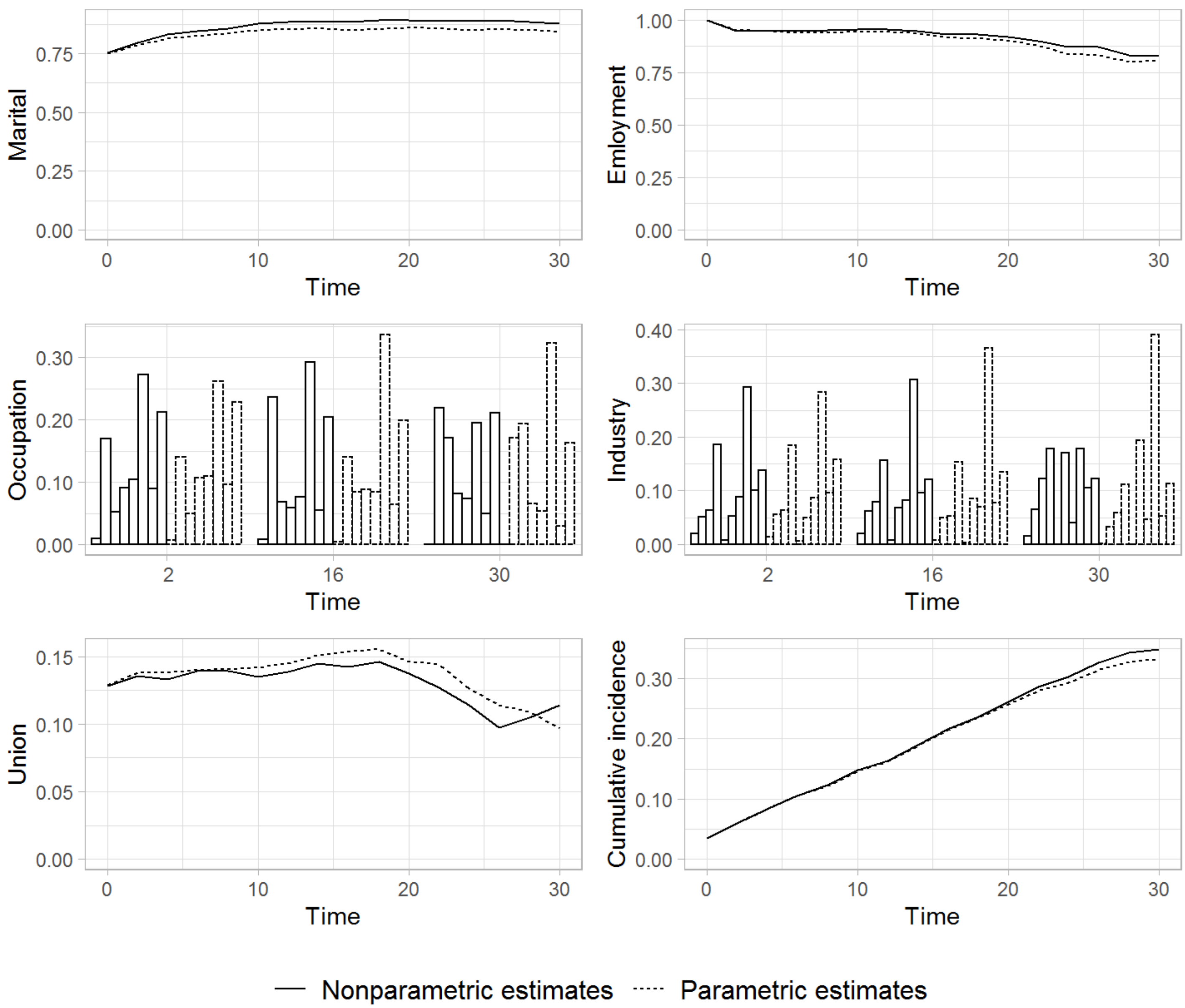
Marital
0.80
0.60
0.40
0.20
0.00
0
10
20
30
Time
Emloyment
1.00
0.75
0.50
0.25
0.00
0
10
20
30
Time
Occupation
0.40
0.30
0.20
0.10
0.00
2
16
30
Time
Industry
0.60
0.40
0.20
0.00
2
16
30
Time
Union
0.15
0.10
0.05
0.00
0
10
20
30
Time
Cumulative incidence
0.40
0.30
0.20
0.10
0.00
0
10
20
30
Time
Nonparametric estimates
Parametric estimates

≤HS men



Marital
Emloyment
Occupation
Industry
Union
Cumulative incidence
Time
Nonparametric estimates
Parametric estimates

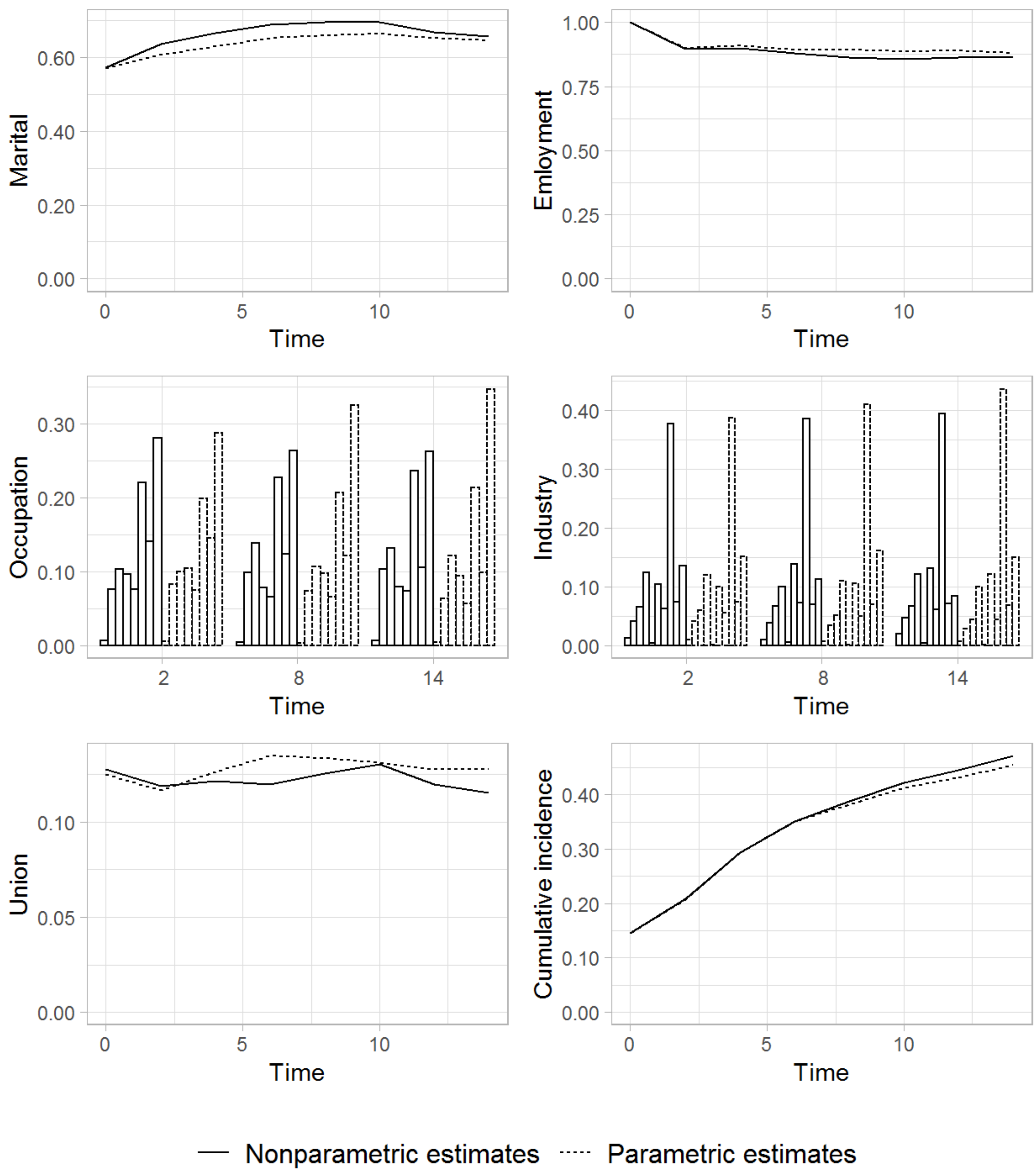
>HS men



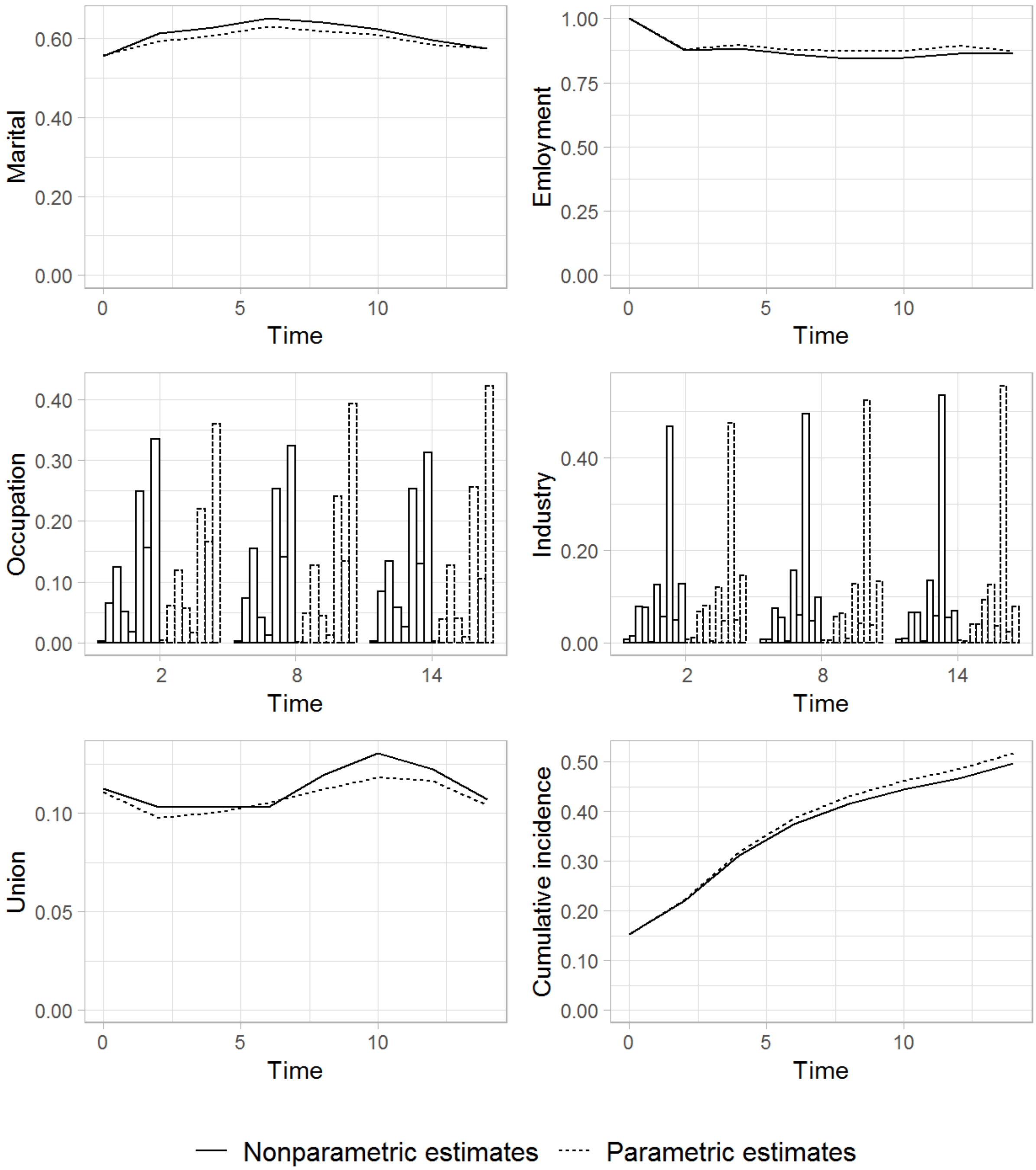
Marital
0.00
0.25
0.50
0.75
0
10
20
30
Time
Emloyment
0.00
0.25
0.50
0.75
1.00
0
10
20
30
Time
Occupation
0.00
0.10
0.20
0.30
2
16
30
Time
Industry
0.00
0.10
0.20
0.30
0.40
2
16
30
Time
Union
0.00
0.05
0.10
0.15
0
10
20
30
Time
Cumulative incidence
0.00
0.10
0.20
0.30
0
10
20
30
Time
Nonparametric estimates
Parametric estimates

B13. Simulated (parametric) versus observed (nonparametric) distributions of exposure and time-varying covariates for overall mental-illness analyses and for subgroup mental-illness analyses.

Overall

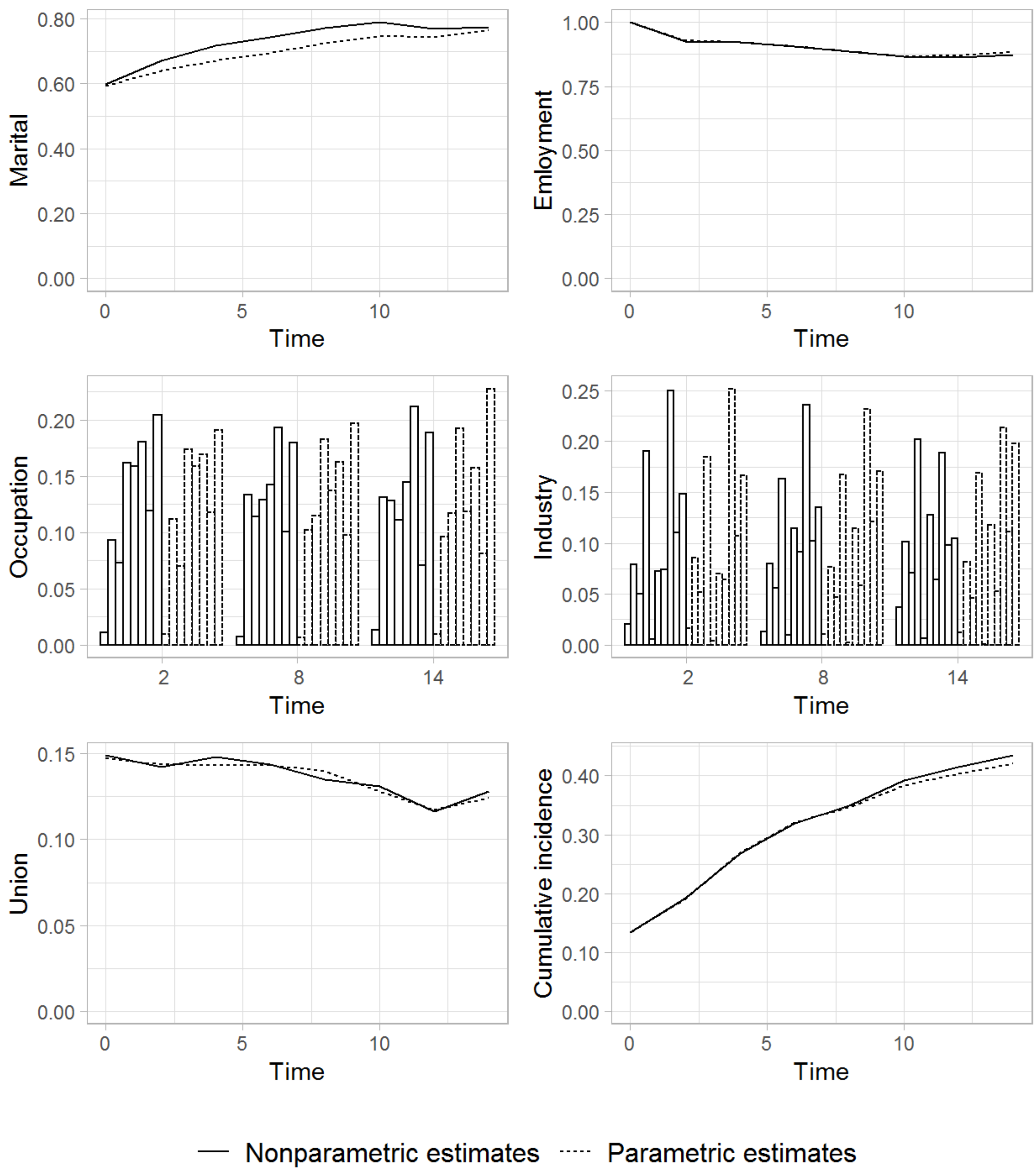


Women

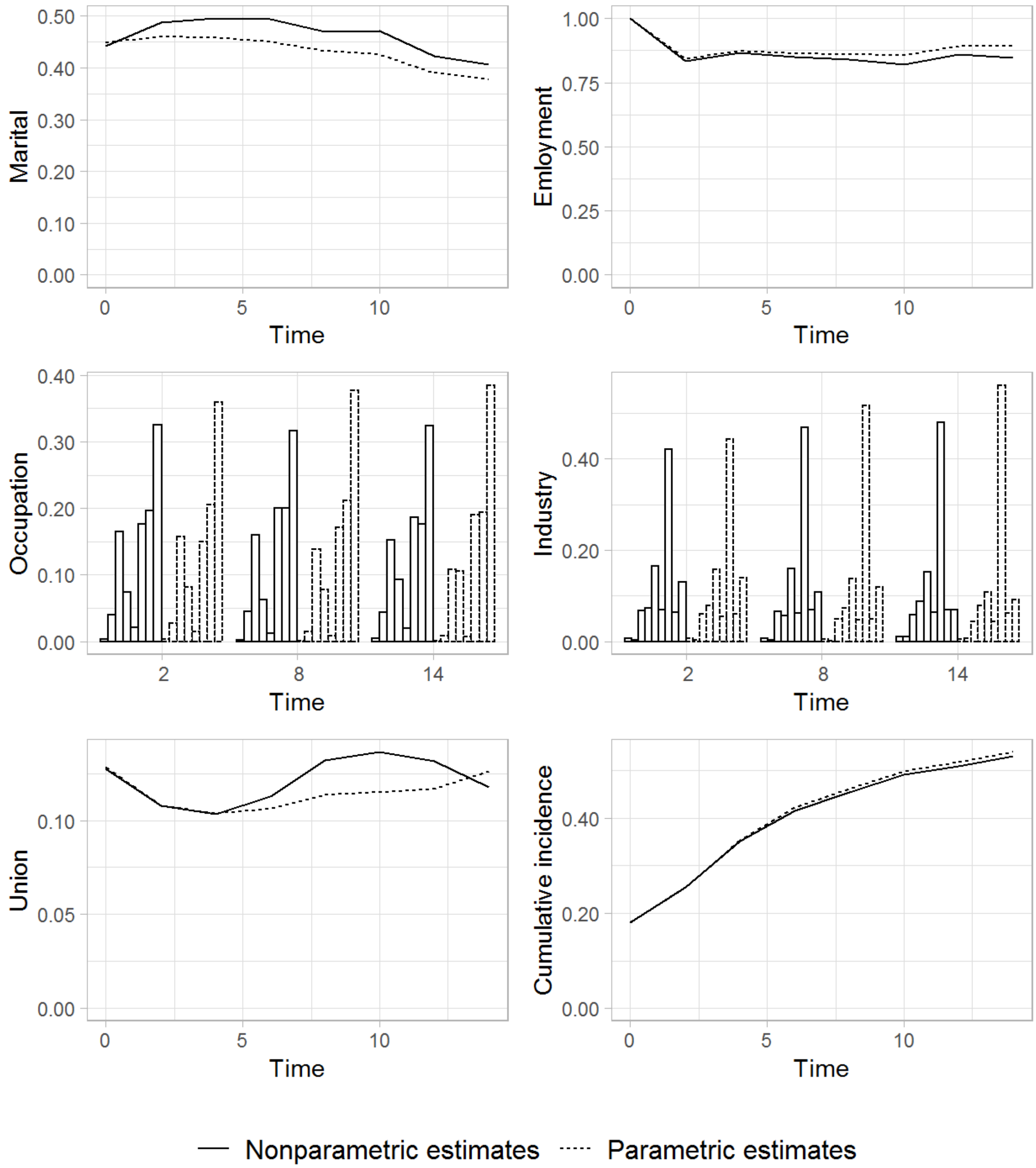


Marital
0.60
0.40
0.20
0.00
0
5
10
Time
Emloyment
1.00
0.75
0.50
0.25
0.00
0
5
10
Time
Occupation
0.40
0.30
0.20
0.10
0.00
2
8
14
Time
Industry
0.40
0.20
0.00
2
8
14
Time
Union
0.10
0.05
0.00
0
5
10
Time
Cumulative incidence
0.50
0.40
0.30
0.20
0.10
0.00
0
5
10
Time
Nonparametric estimates
Parametric estimates

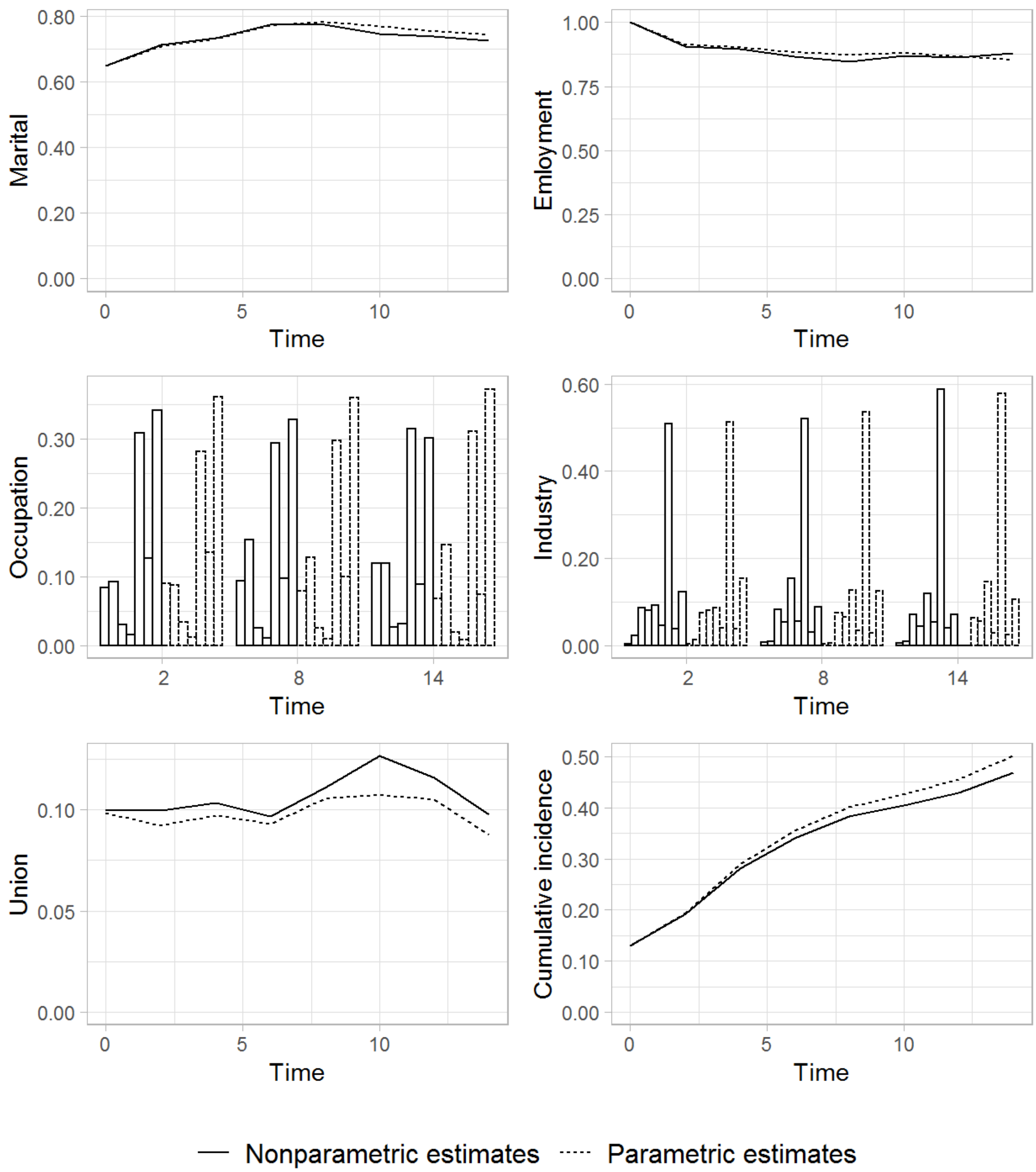
Men


Marital
0.80
0.60
0.40
0.20
0.00
0
5
10
Time
Emloyment
1.00
0.75
0.50
0.25
0.00
0
5
10
Time
Occupation
0.20
0.15
0.10
0.05
0.00
2
8
14
Time
Industry
0.25
0.20
0.15
0.10
0.05
0.00
2
8
14
Time
Union
0.15
0.10
0.05
0.00
0
5
10
Time
Cumulative incidence
0.40
0.30
0.20
0.10
0.00
0
5
10
Time
Nonparametric estimates
Parametric estimates

Women of color

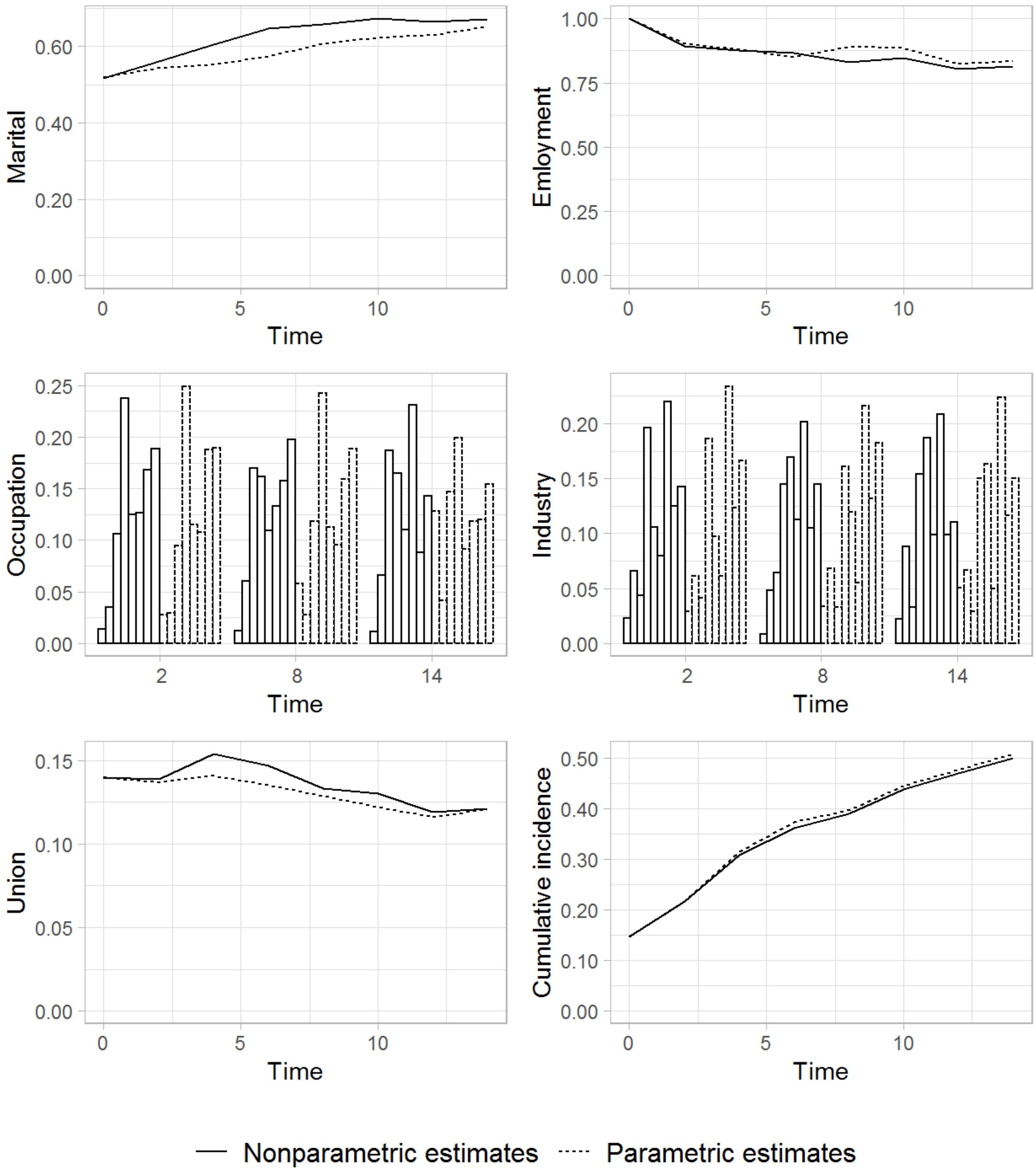


White women

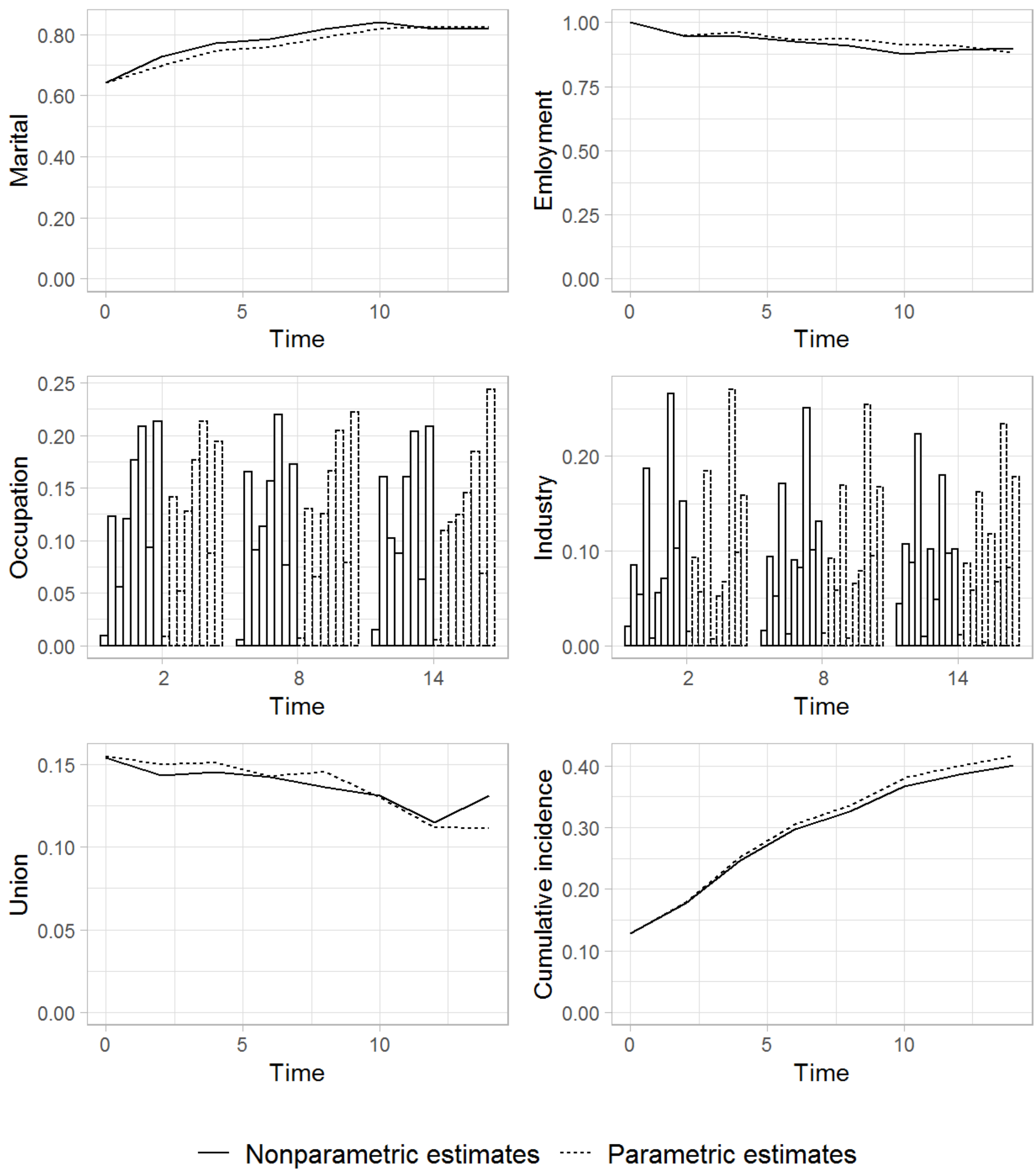


Marital
0.80
0.60
0.40
0.20
0.00
0
5
10
Time
Emloyment
1.00
0.75
0.50
0.25
0.00
0
5
10
Time
Occupation
0.30
0.20
0.10
0.00
2
8
14
Time
Industry
0.60
0.40
0.20
0.00
2
8
14
Time
Union
0.10
0.05
0.00
0
5
10
Time
Cumulative incidence
0.50
0.40
0.30
0.20
0.10
0.00
0
5
10
Time
Nonparametric estimates
Parametric estimates

Men of color

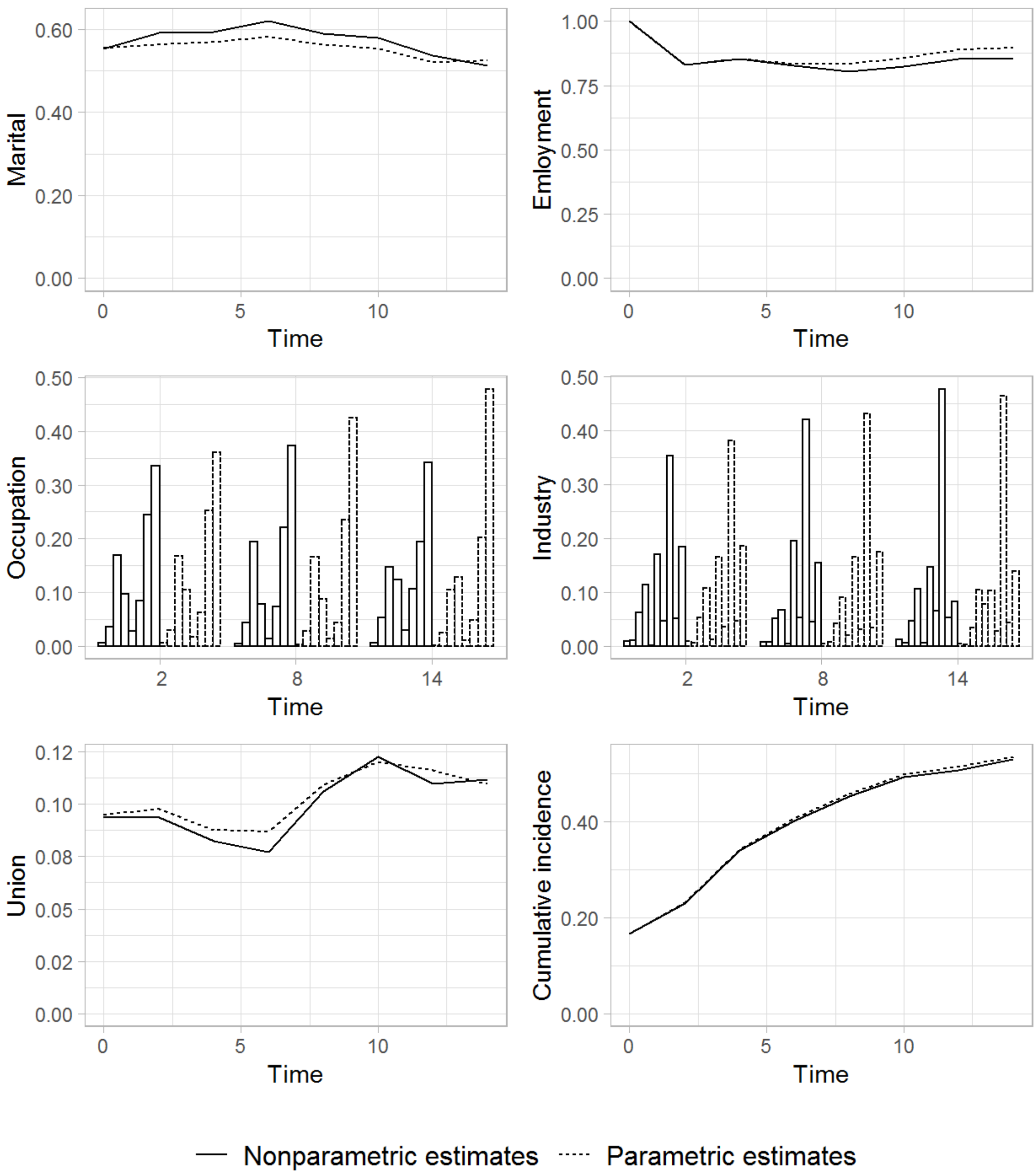

Marital
0.00
0.20
0.40
0.60
0
5
10
Time
Emloyment
0.00
0.25
0.50
0.75
1.00
0
5
10
Time
Occupation
0.00
0.05
0.10
0.15
0.20
0.25
2
8
14
Time
Industry
0.00
0.05
0.10
0.15
0.20
2
8
14
Time
Union
0.00
0.05
0.10
0.15
0
5
10
Time
Cumulative incidence
0.00
0.10
0.20
0.30
0.40
0.50
0
5
10
Time
Nonparametric estimates
Parametric estimates

White men



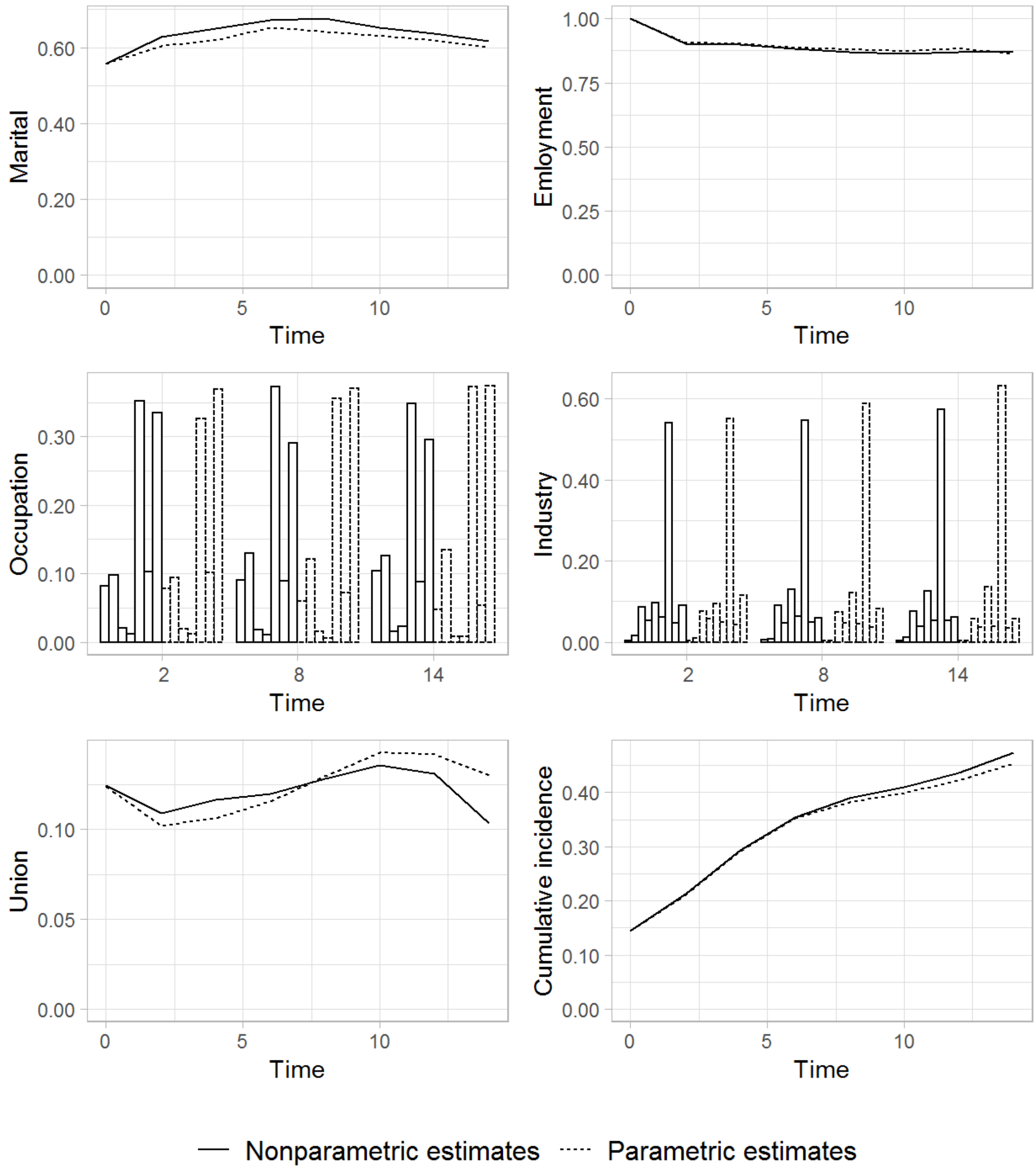
Marital
0.80
0.60
0.40
0.20
0.00
0
5
10
Time
Emloyment
1.00
0.75
0.50
0.25
0.00
0
5
10
Time
Occupation
0.25
0.20
0.15
0.10
0.05
0.00
2
8
14
Time
Industry
0.20
0.10
0.00
2
8
14
Time
Union
0.15
0.10
0.05
0.00
0
5
10
Time
Cumulative incidence
0.40
0.30
0.20
0.10
0.00
0
5
10
Time
Nonparametric estimates
Parametric estimates

≤HS women



Marital
0.60
0.40
0.20
0.00
0
5
10
Time
Emloyment
1.00
0.75
0.50
0.25
0.00
0
5
10
Time
Occupation
0.50
0.40
0.30
0.20
0.10
0.00
2
8
14
Time
Industry
0.50
0.40
0.30
0.20
0.10
0.00
2
8
14
Time
Union
0.12
0.10
0.08
0.05
0.02
0.00
0
5
10
Time
Cumulative incidence
0.40
0.20
0.00
0
5
10
Time
Nonparametric estimates
Parametric estimates

>HS women



Marital
0.00
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0.60
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10
Time
Emloyment
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0.25
0.50
0.75
1.00
0
5
10
Time
Occupation
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0.20
0.30
2
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14
Time
Industry
0.00
0.20
0.40
0.60
2
8
14
Time
Union
0.00
0.05
0.10
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5
10
Time
Cumulative incidence
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0.10
0.20
0.30
0.40
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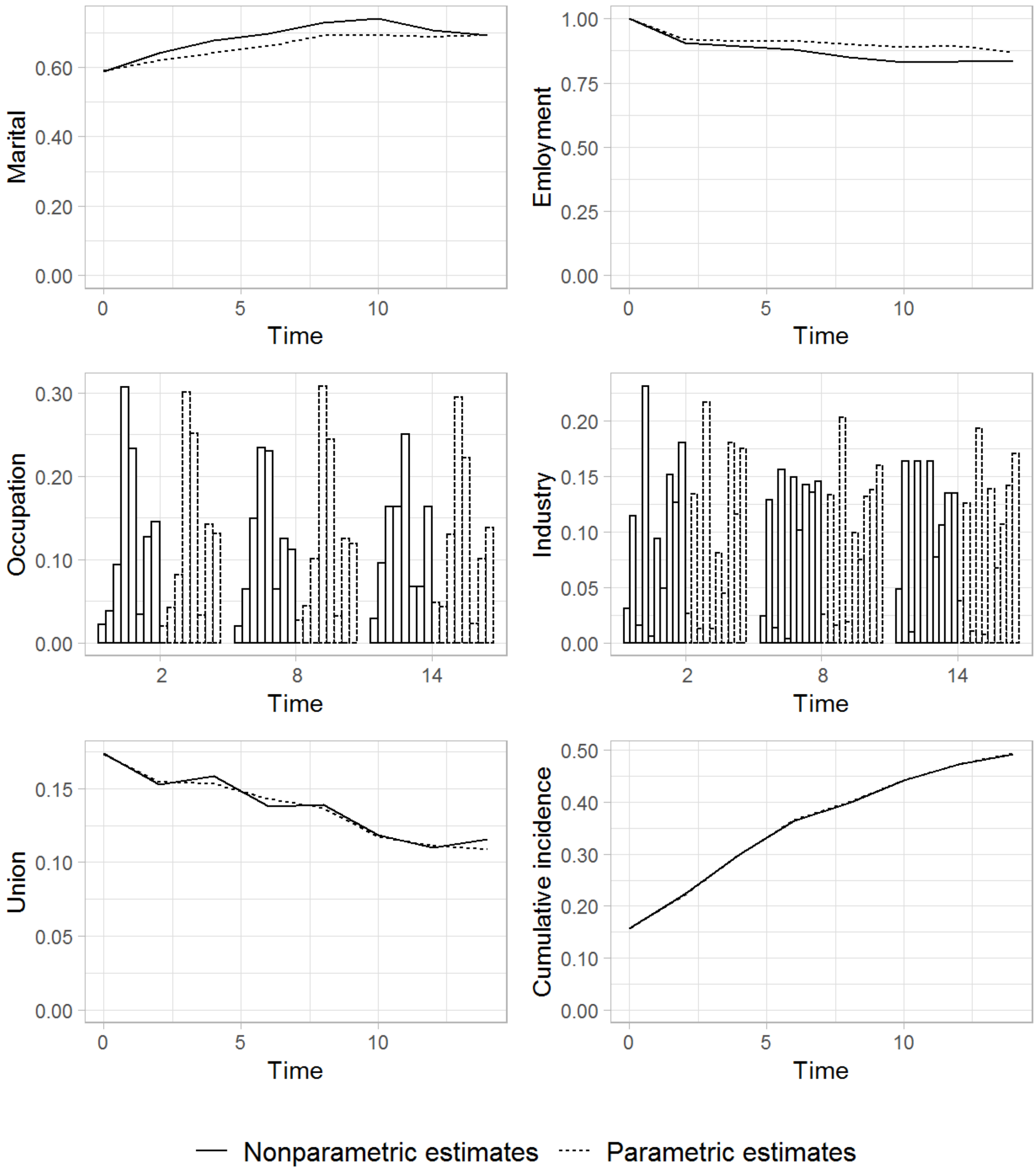
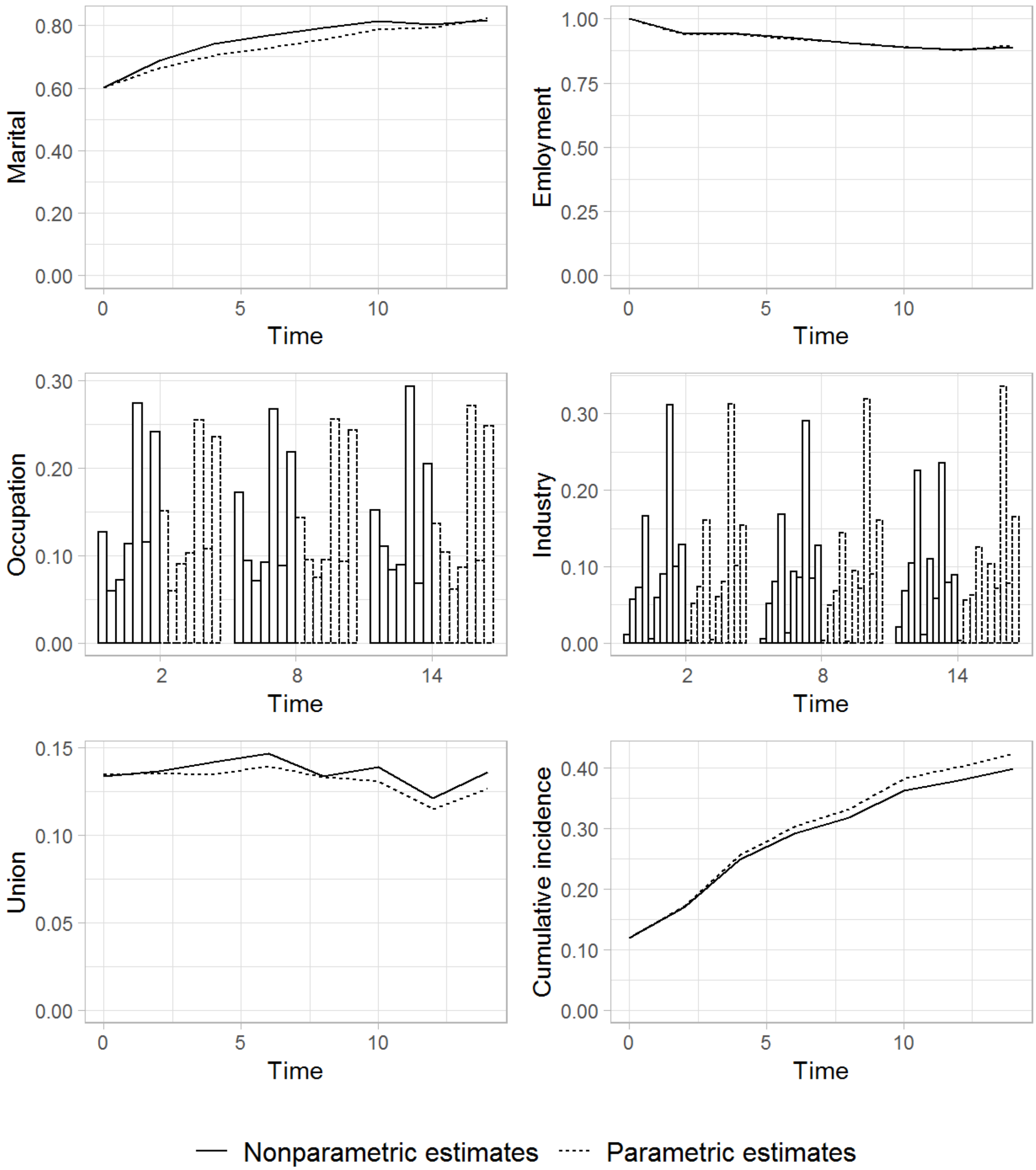
5
10
Time
Nonparametric estimates
Parametric estimates

≤HS men



Marital
0.60
0.40
0.20
0.00
0
5
10
Time
Emloyment
1.00
0.75
0.50
0.25
0.00
0
5
10
Time
Occupation
0.30
0.20
0.10
0.00
2
8
14
Time
Industry
0.20
0.15
0.10
0.05
0.00
2
8
14
Time
Union
0.15
0.10
0.05
0.00
0
5
10
Time
Cumulative incidence
0.50
0.40
0.30
0.20
0.10
0.00
0
5
10
Time
Nonparametric estimates
Parametric estimates

>HS men


Marital
0.80
0.60
0.40
0.20
0.00
0
5
10
Time
Emloyment
1.00
0.75
0.50
0.25
0.00
0
5
10
Time
Occupation
0.30
0.20
0.10
0.00
2
8
14
Time
Industry
0.30
0.20
0.10
0.00
2
8
14
Time
Union
0.15
0.10
0.05
0.00
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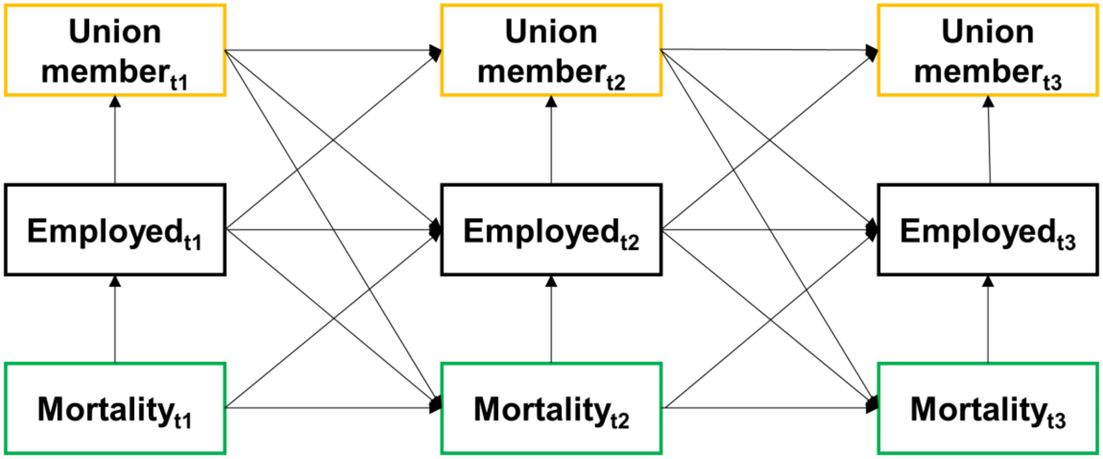
10
Time
Cumulative incidence
0.40
0.30
0.20
0.10
0.00
0
5
10
Time
Nonparametric estimates
Parametric estimates

APPENDIX C



C1. Directed acyclic graph depicting time-varying confounding by employment status.

C2. R code used to estimate cumulative incidence of mortality by the end of follow-up (38 years) in a scenario (sc.) setting all of respondents' employed-person-years to union-member employed-person-years (scenario 1) relative to the risk in a scenario setting none of respondents' employed-person-years to union-member employed-person-years (scenario 2).

```
library(gfoRmula) #development version on GitHub as of 1/25/20 needed for control
argument in "covparams"
library(dplyr)
library(here)
library(data.table)
library(knitr)
library(kableExtra)
library(rms)
library(ggplot2)
library(cowplot)
library(gridExtra)

#######read data

dat <- fread(here('dat.csv'))

#######set number of cores and bootstrap samples for future parallelization, as well as
Monte Carlo sample size
ncores <- parallel::detectCores() - 1
nsamples <- 200
nsimul <- 25000
seed <- 2345

#######define parameters for gformula_survival call
id <- 'unique_id'
time_points <- 19
time_name <- 'time'

#time-varying covariates (TVC)
covnames <- c('marital_status_num', 'employ_num_bin', 'occ_broad_compl',
'ind_broad_compl', 'union_num_compl')

#baseline covariates
basecovs <- c('baseline_age', 'year', "gender", "race", "baseline_region_num",
"baseline_division", "baseline_education", "baseline_parents_poor",
"baseline_disabl_limits_work", "baseline_srh_bin", 'baseline_year')

#outcome
outcome_name <- 'dead'

#TVC types
covtypes <- c( 'binary', 'binary', 'categorical', 'categorical', 'binary')

#type of lag variable for TVC
histories <- c(lagged)

#lag variables needed
histvars <- list(c('marital_status_num', 'employ_num_bin', 'occ_broad_compl',
'ind_broad_compl', 'union_num_compl'))

#models for TVC
#rms::rcs() can be used to model confounder with a restricted cubic spline
covmodels = c("marital_status_num ~
              lag1_marital_status_num +
```

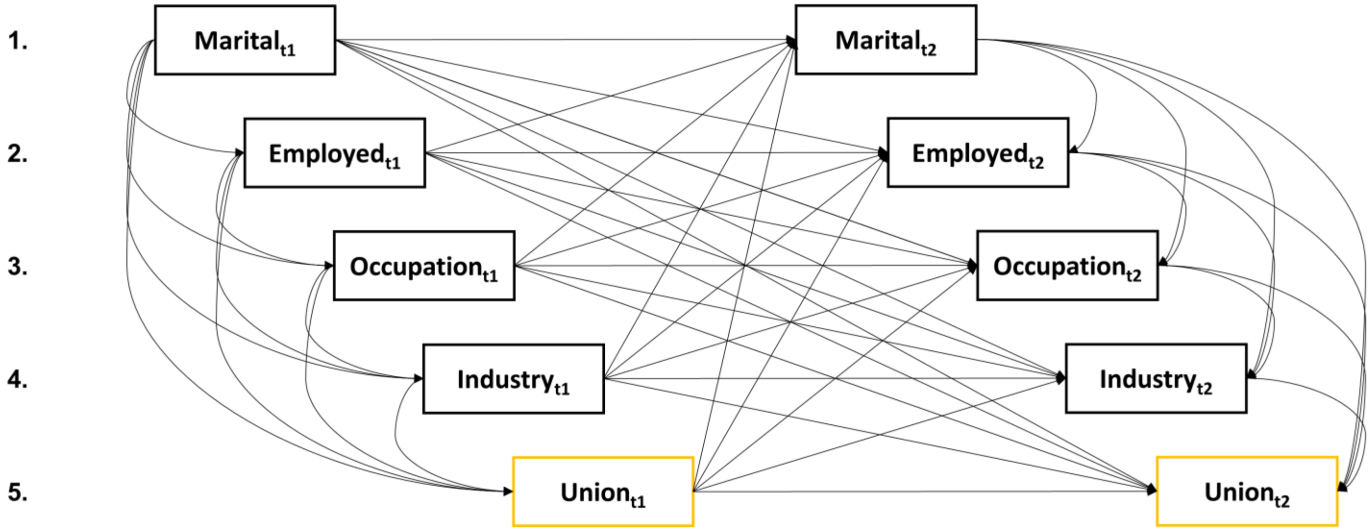
```
              lag1_occ_broad_compl +
              lag1_ind_broad_compl +
              lag1_employ_num_bin +
              lag1_union_num_compl +
              rms::rcs(baseline_age, c(30, 46, 62)) +
              baseline_region_num +
              gender +
              race +
              baseline_education +
              baseline_parents_poor +
              baseline_disabl_limits_work +
              rms::rcs(time, c(3, 6, 9, 12, 15)) +
              rms::rcs(year, c(1984, 1991, 1997, 2003, 2010))",
            "employ_num_bin ~
              marital_status_num +
              lag1_marital_status_num +
              lag1_occ_broad_compl +
              lag1_ind_broad_compl +
              lag1_employ_num_bin +
              lag1_union_num_compl +
               rms::rcs(baseline_age, c(30, 46, 62)) +
              baseline_region_num +
              gender +
              race +
              baseline_education +
              baseline_parents_poor +
              baseline_disabl_limits_work +
              rms::rcs(time, c(3, 6, 9, 12, 15)) +
              rms::rcs(year, c(1984, 1991, 1997, 2003, 2010))",
            "occ_broad_compl ~
              employ_num_bin +
              marital_status_num +
              lag1_marital_status_num +
              lag1_occ_broad_compl +
              lag1_ind_broad_compl +
              lag1_employ_num_bin +
              lag1_union_num_compl +
              rms::rcs(baseline_age, c(30, 46, 62)) +
              baseline_region_num +
              gender +
              race +
              baseline_education +
              baseline_parents_poor +
              baseline_disabl_limits_work +
              rms::rcs(time, c(3, 6, 9, 12, 15)) +
              rms::rcs(year, c(1984, 1991, 1997, 2003, 2010))",
            "ind_broad_compl ~
              occ_broad_compl +
              employ_num_bin +
              marital_status_num +
              lag1_marital_status_num +
              lag1_occ_broad_compl +
              lag1_ind_broad_compl +
              lag1_employ_num_bin +
              lag1_union_num_compl +
              rms::rcs(baseline_age, c(30, 46, 62)) +
              baseline_region_num +
              gender +
              race +
              baseline_education +
              baseline_parents_poor +
```

```
               baseline_disabl_limits_work +
               rms::rcs(time, c(3, 6, 9, 12, 15)) +
               rms::rcs(year, c(1984, 1991, 1997, 2003, 2010))",
             "union_num_compl ~
               occ_broad_compl +
               ind_broad_compl +
               employ_num_bin +
               marital_status_num +
               lag1_marital_status_num +
               lag1_occ_broad_compl +
               lag1_ind_broad_compl +
               lag1_employ_num_bin +
               lag1_union_num_compl +
               rms::rcs(baseline_age, c(30, 46, 62)) +
               baseline_region_num +
               gender +
               race +
               baseline_education +
               baseline_parents_poor +
               baseline_disabl_limits_work +
               rms::rcs(time, c(3, 6, 9, 12, 15)) +
               year")

#increase maxit for multinom models so they converge
covparams <- list(covmodels = lapply(covmodels, function (x) as.formula(x)),
                  control=c(NA, NA, list(maxit=10000), list(maxit=10000), NA))

#outcome model
ymodel <- dead ~
  occ_broad_compl +
  ind_broad_compl +
  employ_num_bin +
  union_num_compl +
  marital_status_num +
  lag1_marital_status_num +
  lag1_occ_broad_compl +
  lag1_ind_broad_compl +
  lag1_employ_num_bin +
  lag1_union_num_compl +
  rms::rcs(baseline_age, c(30, 46, 62)) +
  baseline_region_num +
  gender +
  race +
  baseline_education +
  baseline_parents_poor +
  baseline_disabl_limits_work +
  rms::rcs(time, c(3, 6, 9, 12, 15)) +
  baseline_year

#define exposure variable and scenario names
intvars <- list('union_bin', 'union_bin')
int_descript <- c('Always union if employed', "Never union if employed")

#define custom scenario: if employed (employ_bin > intval provided below), set intvar
(union_bin) to 1 (union member); if not employed (employ_bin < intval provided below),
set intvar (union_bin) to 0 (not union)
example_intervention <- function(newdf, pool, intvar, intvals, time_name, t){
  newdf[,(intvar) := 0]
  newdf[employ_bin > intvals[[1]], (intvar) := 1]
}
```

```
#Package also allows for incorporating deterministic knowledge about relationships
between time-varying covariates via the "restrictions" argument. For example, whenever
respondents are predicted to be "not employed", we could use the "restrictions" argument
to set occupation and industry to "not employed". However, even without using that
argument, no simulated observations (returned when "sim_data_b" argument set to "TRUE")
are predicted to have logical mismatches between variables (e.g., employ_bin=="employed"
but occupation=="not employed"), as such observations don't exist in the observed data.

#######gformula survival function call

gform_basic <- gformula_survival(obs_data = dat_sub_first_worker_dead,
                                 id = id,
                                 time_points = time_points,
                                 time_name = time_name,
                                 covnames = covnames,
                                 outcome_name = outcome_name,
                                 covtypes = covtypes,
                                 covparams = covparams,
                                 ymodel = ymodel,
                                 intvars = intvars,
                                 interventions = list(list(c(example_intervention,
0.99)), list(c(example_intervention, 1.01))), #in always-union scenario, if employ_bin is
greater than 0.99 (1=employed), union_bin is set to 1 (union); in never-union scenario,
union_bin is always set to 0 (non-union) because employ_bin is never greater than 1.01_
                                 int_descript = int_descript,
                                 histories = histories,
                                 histvars = histvars,
                                 basecovs = basecovs,
                                 seed=seed,
                                 nsimul=nsimul,
                                 parallel=TRUE,
                                 ncores=ncores,
                                 nsamples=nsamples,
                                 ci_method="normal",
                                 ref_int = 2)

#######intervention results

results <- gform_basic$result[55:57,c(2,3,4,6,7,8,10,11,12,14,15)]
kable(x, digits=2) %>%
  kable_styling("striped")

#######plotting functions (can also get basic plots using plot(gform_basic)

#plot for continuous variables

cont_opts <- c("Marital", "Employment", "Union")

plot_cont <- function(var, num){

  out <- data.frame(gform_basic$dt_cov_plot[var])
  names(out) <- c("time", "cov", "legend")

  ggplot(out, aes(x=time*2, y=cov, group=legend, lty=legend)) +
    geom_line() +
    theme_light() +
    ylab(cont_opts[num]) +
    xlab('Time') +
    scale_y_continuous(limits=c(0, max(out$cov)),
labels=scales::number_format(accuracy=0.01))
}
```

```
#plot for categorical variables

cat_opts <- c("Occupation", "Industry")

plot_cat <- function(var, num){

  out <- data.frame(gform_basic$dt_cov_plot[var])
  names(out) <- c("t0", "V1", "var", "legend")

  out %>%
    group_by(t0, legend) %>%
    mutate(prop=V1 / sum(V1)) -> out

  ggplot(subset(out, c(t0==1 | t0==9 | t0==17)), aes(x=t0*2, y=prop,
group=interaction(var, legend))) +
    geom_bar(aes(lty=legend), stat='identity', position='dodge', fill='white',
color='black') +
    theme_light() +
    ylab(cat_opts[num]) +
    xlab('Time') +
    scale_x_discrete(limits=c(2, 18, 34)) +
    scale_y_continuous(labels=scales::number_format(accuracy=0.01))
}

#plot for outcome variable

plot_out <- function(var){

  out <- data.frame(gform_basic[var])
  names(out) <- c("time", "risk", "survival", "legend")

  ggplot(out, aes(x=time*2, y=risk, group=legend, lty=legend)) +
    geom_line() +
    theme_light() +
    theme(legend.title=element_blank(), legend.position='bottom',
legend.text=element_text(size=12)) +
    ylab('Cumulative incidence') +
    xlab('Time') +
    scale_y_continuous(limits=c(0, max(out$risk))) +
    scale_linetype_discrete(labels=c("Nonparametric estimates", "Parametric estimates"))
}

#######plot

mar <- plot_cont('marital_status_num', 1)
empl <- plot_cont('employ_num_bin', 2)
occ <- plot_cat('occ_broad_compl', 1)
ind <- plot_cat('ind_broad_compl', 2)
uni <- plot_cont('union_num_compl', 3)
out <- plot_out('dt_out_plot')
my_legend <- get_legend(out)

grid.arrange(arrangeGrob(mar + theme(legend.position = "none"),
                         empl + theme(legend.position = 'none'),
                         occ + theme(legend.position = 'none'),
                         ind + theme(legend.position = 'none'),
                         uni + theme(legend.position = 'none'),
                         out + theme(legend.position = 'none'),
                               nrow=3),
```

```
                  my_legend, nrow=2, heights=c(10,1))
```



C3. Hypothesized temporal ordering of time-varying variables. In parametric g-formula analyses, time-varying variables at wave t_k were functions of baseline confounders, prior time-varying variables in t_k (if any), time-varying variables in t_{k-1}, year, and follow-up time.

C4. Specification of follow-up time and year in pooled parametric models fit in step 1 of parametric g-formula analyses.

		Marital	Employment	Occupation	Industry	Union	Mortality
Overall	**Time**	5-knot RCS[a]	5-knot RCS	5-knot RCS	5-knot RCS	5-knot RCS	5-knot RCS
	Year	5-knot RCS	5-knot RCS	5-knot RCS	5-knot RCS	4-level cat.[b]	Linear
Gender							
Women	**Time**	5-knot RCS	5-knot RCS	5-knot RCS	5-knot RCS	5-knot RCS	5-knot RCS
	Year	5-knot RCS	5-knot RCS	5-knot RCS	5-knot RCS	Linear	Lin.+quad[c]
Men	**Time**	5-knot RCS	5-knot LSP[d]	5-knot RCS	5-knot RCS	5-knot RCS	4-level cat.
	Year	5-knot RCS	Linear	5-knot RCS	5-knot RCS	Linear	Linear
Race							
Black	**Time**	5-knot RCS	5-knot RCS	5-knot RCS	5-knot RCS	5-knot RCS	5-knot RCS+bin.[e]
	Year	5-knot RCS	5-knot RCS	5-knot RCS	5-knot RCS	Linear	Linear
White	**Time**	5-knot RCS	5-knot RCS	5-knot RCS	5-knot RCS	5-knot RCS	4-level cat.
	Year	5-knot RCS	5-knot RCS	5-knot RCS	5-knot RCS	Linear	Linear
Education							
≤HS	**Time**	5-knot RCS	5-knot RCS	5-knot RCS	5-knot RCS	5-knot RCS	5-knot RCS
	Year	5-knot RCS	5-knot RCS	5-knot RCS	5-knot RCS	Linear	Linear
>HS	**Time**	5-knot RCS	5-knot RCS	5-knot RCS	5-knot RCS	5-knot RCS	5-knot RCS
	Year	5-knot RCS	5-knot RCS	5-knot RCS	5-knot RCS	Linear	Linear

Notes:
[a] 5-knot restricted cubic spline.
[b] 4-level categorical term.
[c] Linear and quadratic terms.
[d] 5-knot linear spline.
[e] 5-knot restricted cubic spline and binary terms.

C5. Trends in prevalence of union membership among employed respondents in sample over study period by demographic group, occupation, and industry.

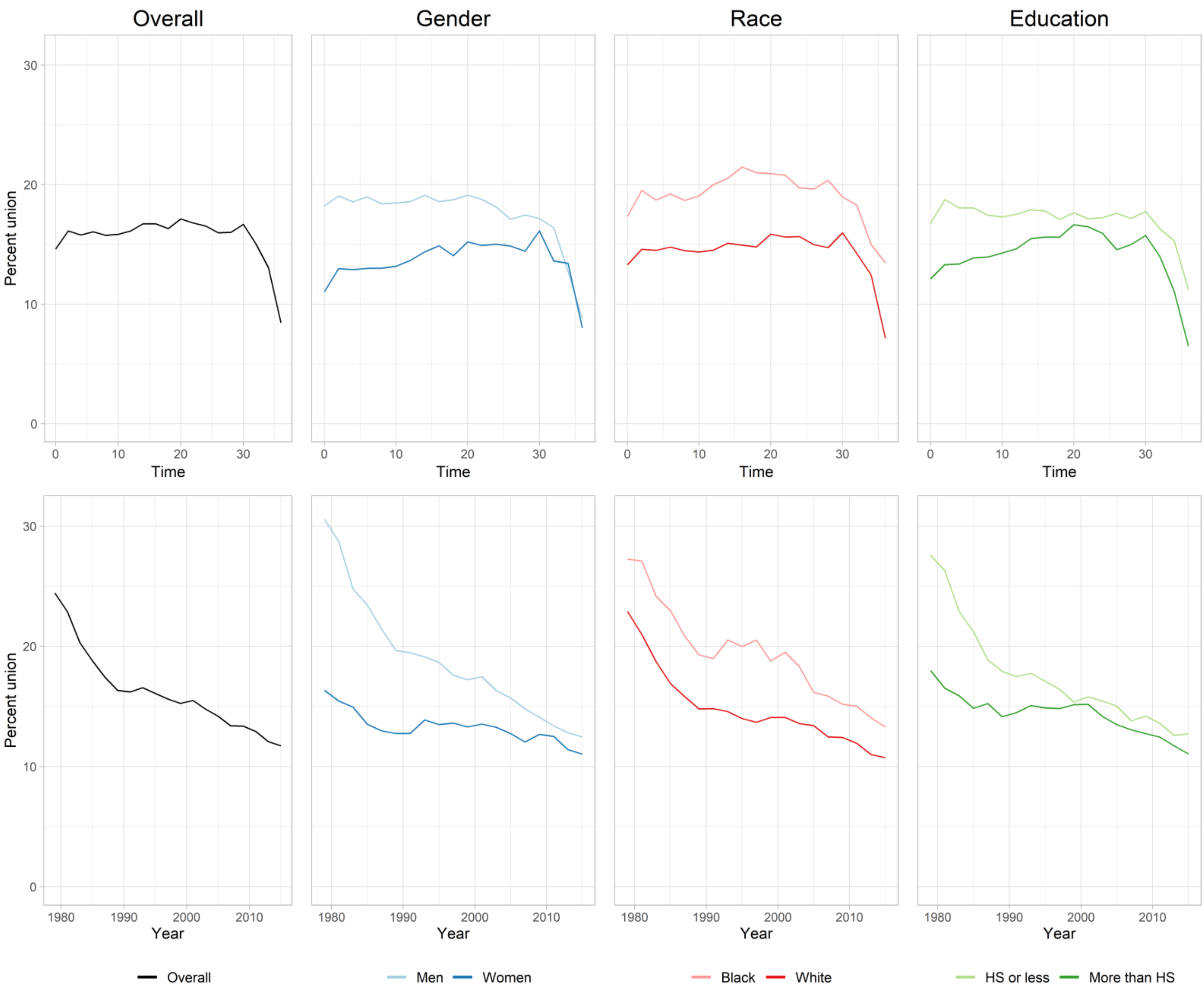


Figure C5a. Trends in prevalence of union membership among employed respondents in sample by follow-up time (top row) and calendar year (bottom row) overall and in each gender, gender-race, and gender-education subgroup.

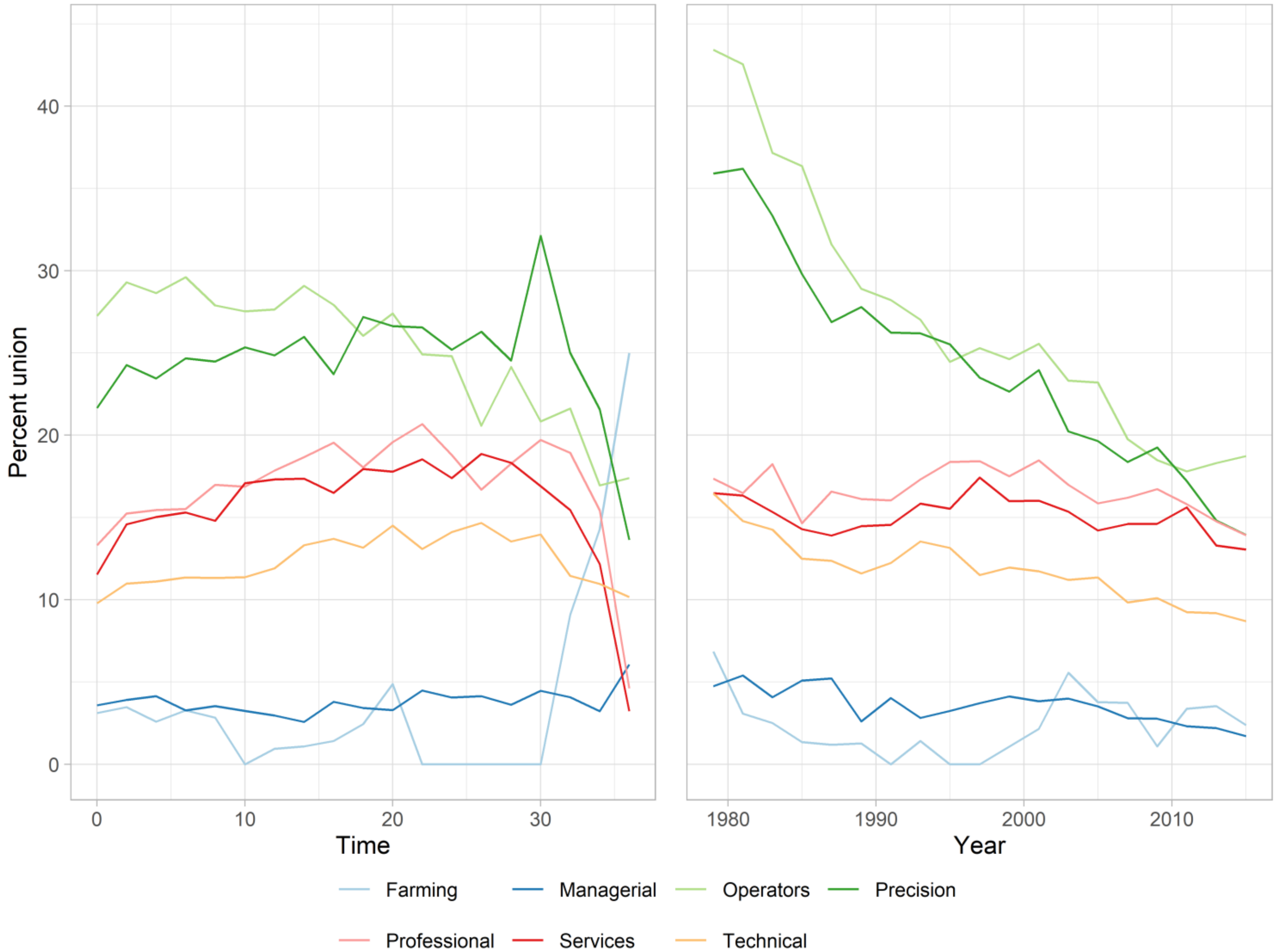


Figure C5b. Trends in prevalence of union membership among employed respondents in sample by follow-up time (left) and calendar year (right) in each occupation.

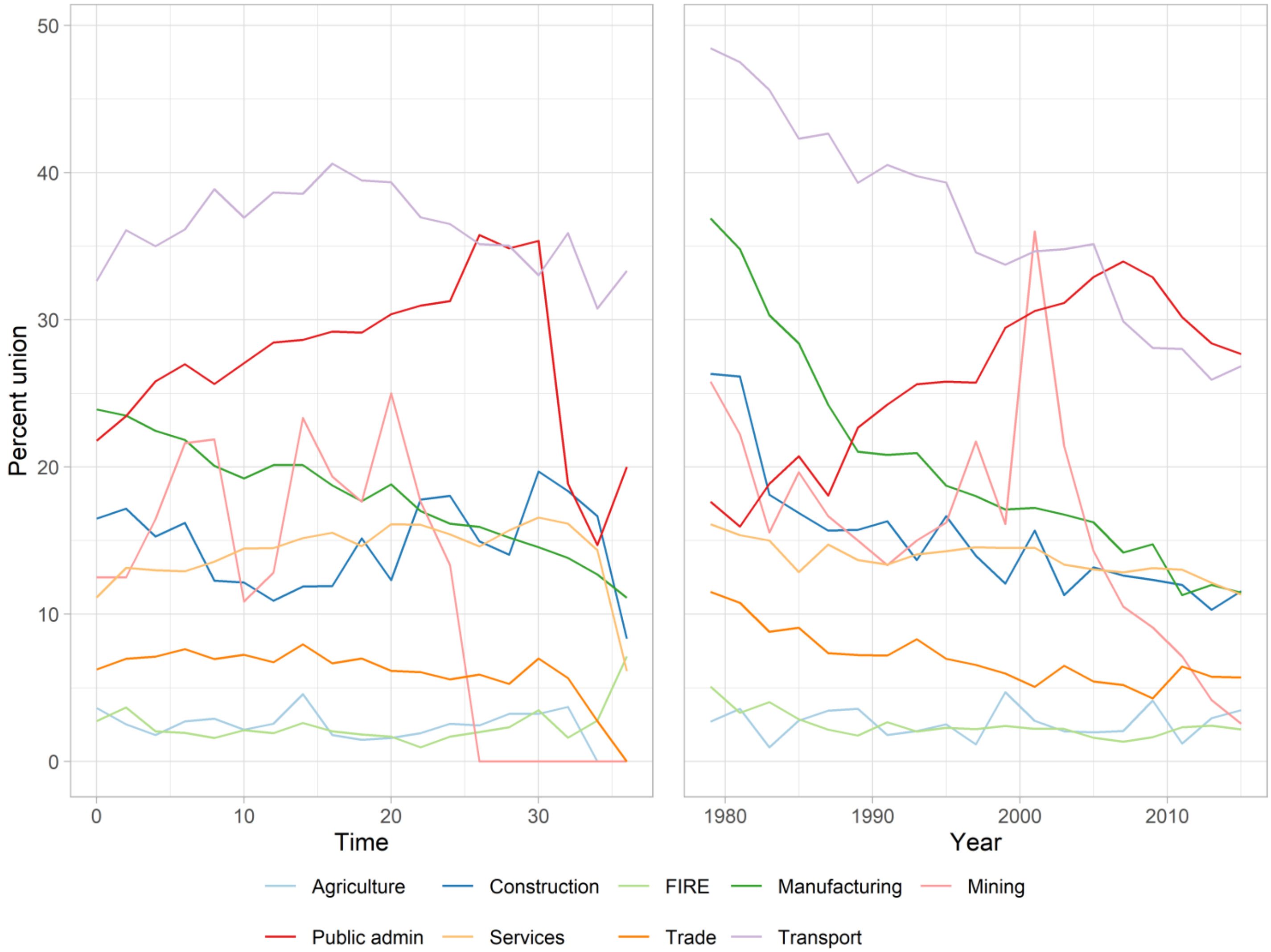


Figure C5c. Trends in prevalence of union membership among employed respondents in sample by follow-up time (left) and calendar year (right) in each industry.

C6. Parametric g-formula estimates of risk of mortality by the end of follow-up (38 years) in a scenario (sc.) setting all of respondents' two-year-lagged employed-person-years to union-member employed-person-years (scenario 1) relative to the risk in a scenario setting none of respondents' two-year-lagged employed-person-years to union-member employed-person-years (scenario 2).

	Respondents[a]	Observations[a]	Sc.	Risk[b]	RR	95% CI		RD[b]	95% CI	
Overall	19,176	123,659	1	168.5	0.89	0.79	0.99	-21.2	-40.1	-2.0
			2	189.6						
Gender										
Women	9,775	64,746	1	124.2	0.84	0.66	1.02	-23.7	-51.6	4.1
			2	147.9						
Men	9,401	58,913	1	206.9	0.90	0.81	1.00	-21.9	-43.0	-0.8
			2	228.8						
Race										
Black	6,103	36,184	1	186.5	0.88	0.72	1.04	-24.9	-59.3	9.5
			2	211.4						
White	11,489	79,379	1	137.7	0.89	0.75	1.04	-16.6	-39.5	6.3
			2	154.3						
Education										
≤HS	10,293	65,852	1	219.6	0.93	0.81	1.05	-16.9	-46.4	12.6
			2	236.5						
>HS	8,883	57,807	1	95.1	0.77	0.55	0.99	-27.8	-56.6	1.0
			2	122.9						

Notes:
Risk ratio (RR) and risk difference (RD) estimates compare the risk (i.e., cumulative incidence) in scenario 1 relative to the risk in scenario 2. Subgroup estimates produced from stratified models. Confidence intervals calculated from non-parametric bootstrap with 200 repetitions.
[a] Unique respondents and observations in Panel Study of Income Dynamics dataset used to fit pooled time-varying exposure, confounder, and outcome models. Monte Carlo pseudo-sample used in simulations had 25,000 respondents.
[b] Per 1,000.

C7. Parametric g-formula estimates of risk of mortality by the end of follow-up (38 years) in a scenario (sc.) setting all of respondents' employed-person-years to union-member employed-person-years (scenario 1) relative to the risk in a scenario setting none of respondents' employed-person-years to union-member employed-person-years (scenario 2). Unlike in analyses presented in main text, occupation and industry treated as baseline covariates.

	Respondents[a]	Observations[a]	Sc.	Risk[b]	RR	95% CI		RD[b]	95% CI	
Overall	23,022	146,681	1	161.8	0.86	0.77	0.95	-25.8	-42.9	-8.8
			2	187.7						
Gender										
Women	11,539	76,285	1	129.5	0.87	0.69	1.04	-20.0	-46.1	6.1
			2	149.5						
Men	11,483	70,396	1	214.9	0.88	0.79	0.98	-28.0	-51.1	-4.8
			2	242.8						
Race										
Black	7,601	43,785	1	175.4	0.84	0.71	0.98	-32.3	-61.3	-3.2
			2	207.7						
White	13,465	92,844	1	159.4	0.88	0.76	0.99	-22.6	-44.3	-0.9
			2	182.0						
Education										
≤HS	12,445	72,809	1	215.4	0.92	0.81	1.02	-19.2	-44.1	5.7
			2	234.6						
>HS	10,577	73,872	1	86.1	0.70	0.56	0.85	-37.1	-57.6	-16.6
			2	124.3						

Notes:
Risk ratio (RR) and risk difference (RD) estimates compare the risk (i.e., cumulative incidence) in scenario 1 relative to the risk in scenario 2. Subgroup estimates produced from stratified models. Confidence intervals calculated from non-parametric bootstrap with 200 repetitions.
[a] Unique respondents and observations in Panel Study of Income Dynamics dataset used to fit pooled time-varying exposure, confounder, and outcome models. Monte Carlo pseudo-sample used in simulations had 25,000 respondents.
[b] Per 1,000.

C8. Parametric g-formula estimates of risk of mortality by the end of follow-up (38 years) in a scenario (sc.) setting all of respondents' employed-person-years to union-member employed-person-years (scenario 1) relative to the risk in a scenario setting none of respondents' employed-person-years to union-member employed-person-years (scenario 2). Unlike in analyses presented in main text, baseline self-rated health included as a covariate.

	Respondents[a]	Observations[a]	Sc.	Risk[b]	RR	95% CI		RD[b]	95% CI	
Overall	23,022	146,681	1	164.7	0.89	0.79	0.99	-19.6	-38.2	-1.0
			2	184.3						
Gender										
Women	11,539	76,285	1	128.3	0.87	0.69	1.05	-19.2	-46.7	8.3
			2	147.4						
Men	11,483	70,396	1	207.9	0.91	0.82	1.01	-20.0	-42.3	2.3
			2	228.0						
Race										
Black	7,601	43,785	1	184.1	0.89	0.75	1.03	-23.0	-52.1	6.1
			2	207.1						
White	13,465	92,844	1	140.2	0.90	0.78	1.03	-14.9	-34.8	5.0
			2	155.1						
Education										
≤HS	12,445	72,809	1	221.6	0.95	0.84	1.06	-11.2	-36.2	13.7
			2	232.9						
>HS	10,577	73,872	1	90.0	0.72	0.56	0.88	-35.1	-56.6	-13.5
			2	125.0						

Notes:
Risk ratio (RR) and risk difference (RD) estimates compare the risk (i.e., cumulative incidence) in scenario 1 relative to the risk in scenario 2. Subgroup estimates produced from stratified models. Confidence intervals calculated from non-parametric bootstrap with 200 repetitions.
[a] Unique respondents and observations in Panel Study of Income Dynamics dataset used to fit pooled time-varying exposure, confounder, and outcome models. Monte Carlo pseudo-sample used in simulations had 25,000 respondents.
[b] Per 1,000.

C9. Parametric g-formula estimates of risk of mortality by the end of follow-up (38 years) in a scenario (sc.) setting all of respondents' employed-person-years to union-member employed-person-years (scenario 1) relative to the risk in a scenario setting none of respondents' employed-person-years to union-member employed-person-years (scenario 2). Unlike in analyses presented in main text, the exposure, time-varying confounder, and outcome models include baseline division of residence[a] as a covariate rather than baseline region of residence.

	Respondents[b]	Observations[b]	Sc.	Risk[c]	RR	95% CI		RD[b]	95% CI	
Overall	23,022	146,681	1	164.7	0.89	0.80	0.99	-20.2	-38.1	-2.3
			2	184.8						

Notes:
Risk ratio (RR) and risk difference (RD) estimates compare the risk (i.e., cumulative incidence) in scenario 1 relative to the risk in scenario 2. Subgroup estimates produced from stratified models. Confidence intervals calculated from non-parametric bootstrap with 200 repetitions.
[a] The divisions are: East North Central, East South Central, Middle Atlantic, Mountain, New England, Pacific, South Atlantic, West North Central, West South Central.
[b] Unique respondents and observations in Panel Study of Income Dynamics dataset used to fit pooled time-varying exposure, confounder, and outcome models. Monte Carlo pseudo-sample used in simulations had 25,000 respondents.
[c] Per 1,000.

C10. Hazard of mortality among respondents who were union workers at baseline relative to the hazard among respondents who were non-union workers at baseline from Cox proportional hazards models. Subgroup estimates produced from stratified models.

	Respondents	HR	95% CI
Overall	23,022	0.87	0.72, 1.05
Gender			
Women	11,539	0.99	0.70, 1.40
Men	11,483	0.82	0.66, 1.02
Race			
Black	7,061	0.84	0.63, 1.12
White	13,465	0.84	0.65, 1.08
Education			
≤HS	12,445	0.88	0.71, 1.10
>HS	10,577	0.81	0.54, 1.20

Notes:
Estimates from Cox proportional hazards models adjusted for confounders described in main text. Confidence intervals calculated using robust standard errors.

C11. Union membership misclassification.

Card (1996) found that approximately 2.5% of Current Population Survey respondents in 1977 misreported their union status, true union status aside. Given a true union prevalence TU and a misclassification rate MR, we can calculate the proportion of "union" respondents who are truly non-union at an observed union prevalence OU using the following equations:

- Non-union respondents misclassified as union (NUU) = (1 – TU) * MR
- Union respondents misclassified as non-union (UNU) = TU * MR
- OU = TU + NUU – UNU
- Proportion of "union" respondents who are truly non-union = NUU / OU

Assuming Card's 2.5% misclassification rate and an observed union-membership prevalence of 13% (the prevalence in our primary sample), approximately 17% of workers in our analyses who were classified as union were truly non-union.

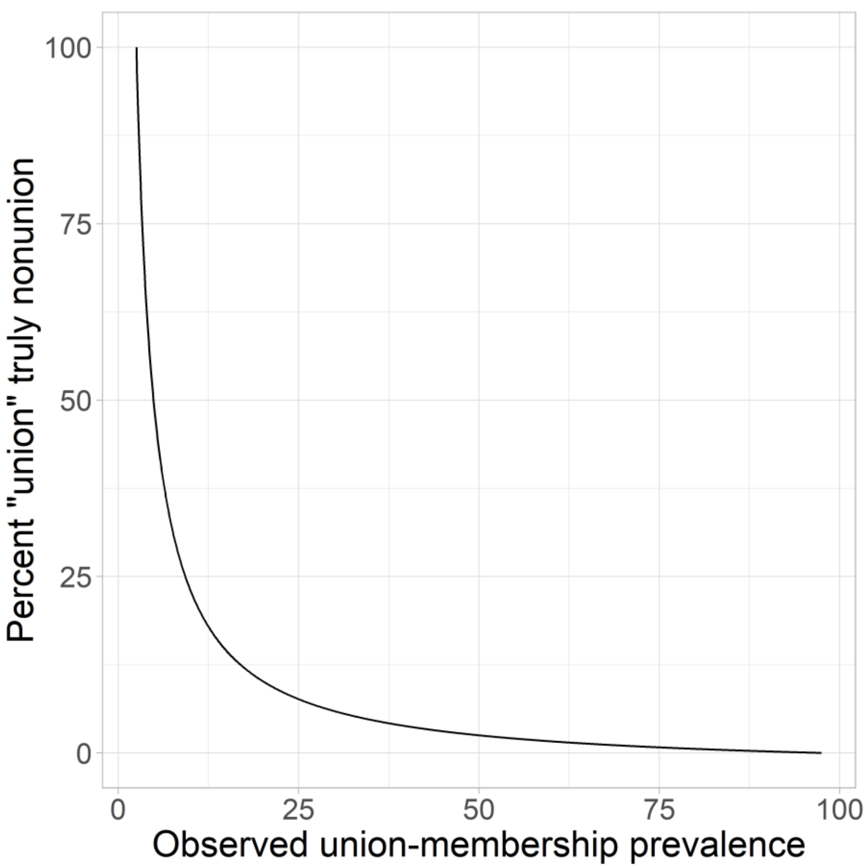
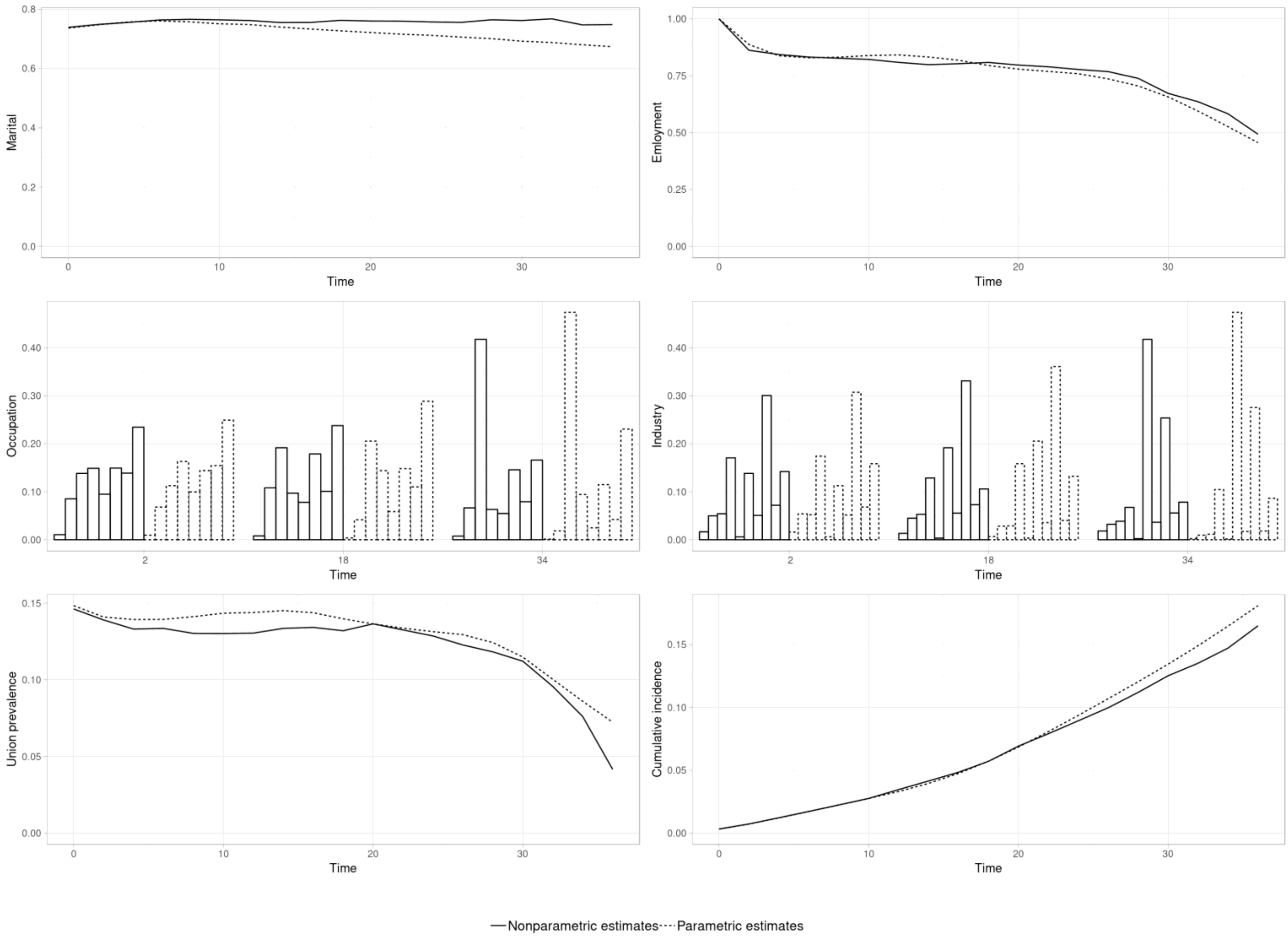


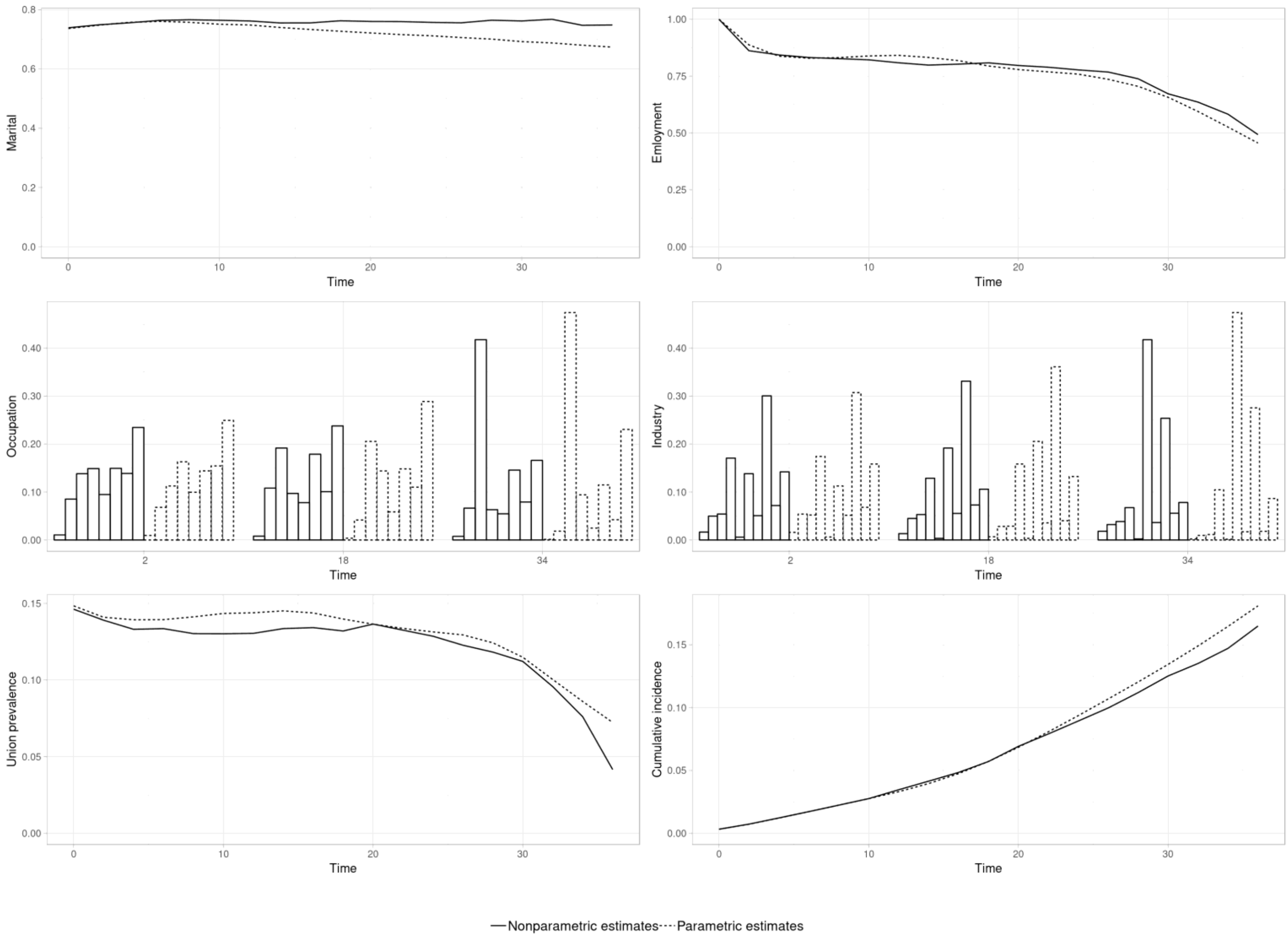
Figure C11. Proportion of respondents misclassified as "union" who were truly "non-union" at various union-membership prevalences, assuming a misclassification rate of 2.5%.

C12. Simulated (parametric) versus observed (nonparametric) distributions of exposure and time-varying covariates overall and in each subgroup.

Overall

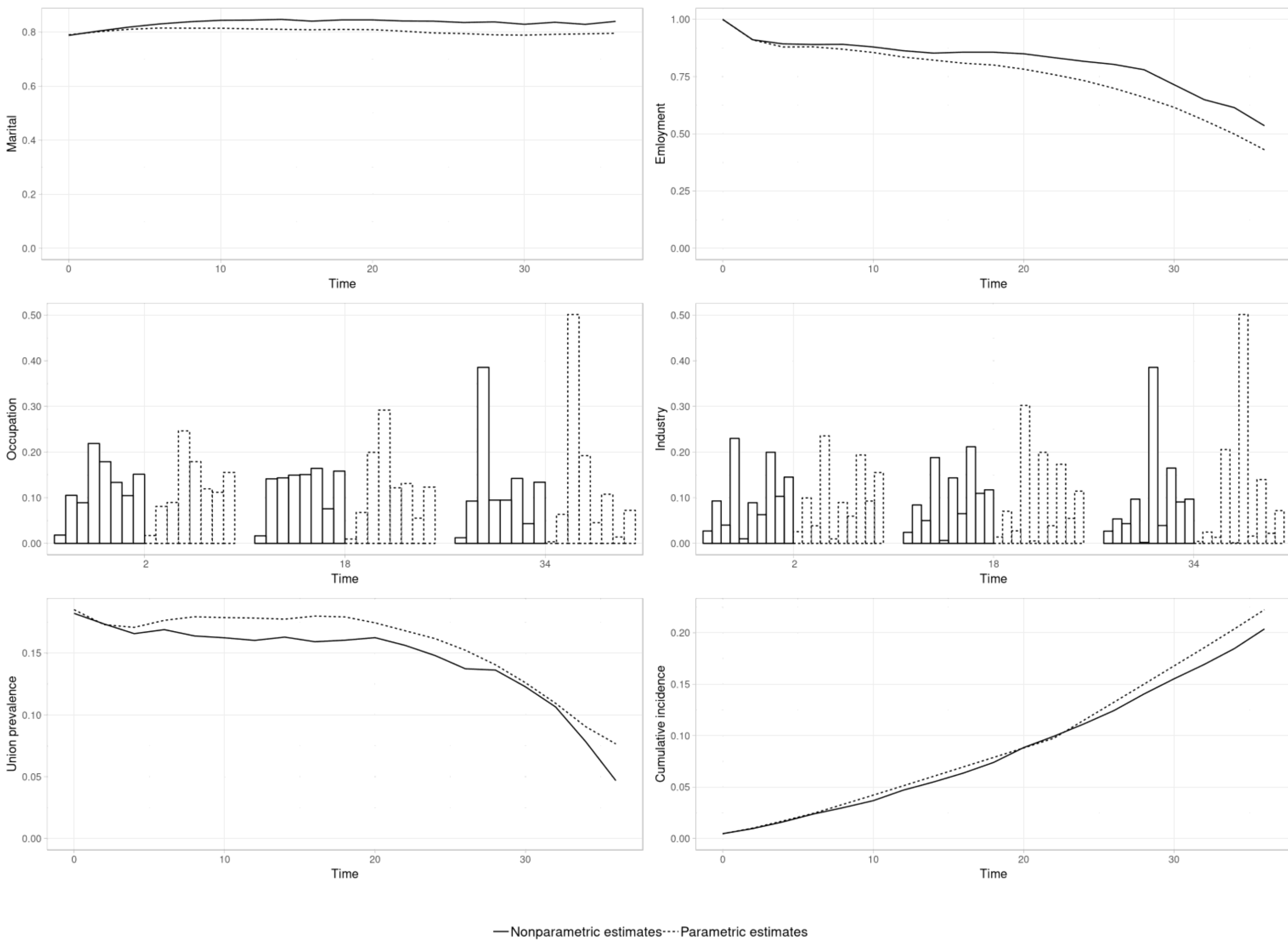


Women



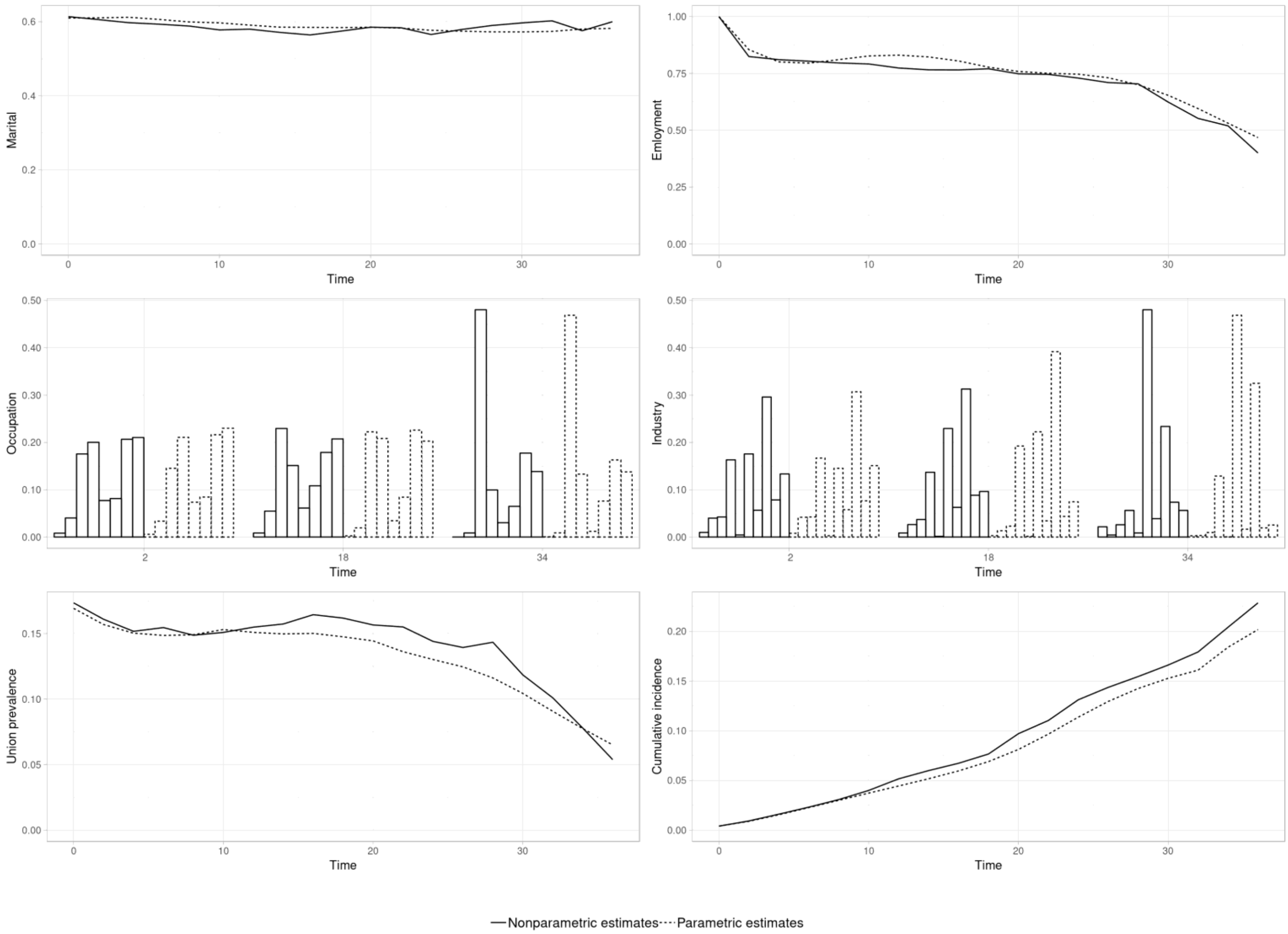
Marital
Emloyment
Occupation
Industry
Union prevalence
Cumulative incidence
Time
Nonparametric estimates
Parametric estimates

Men

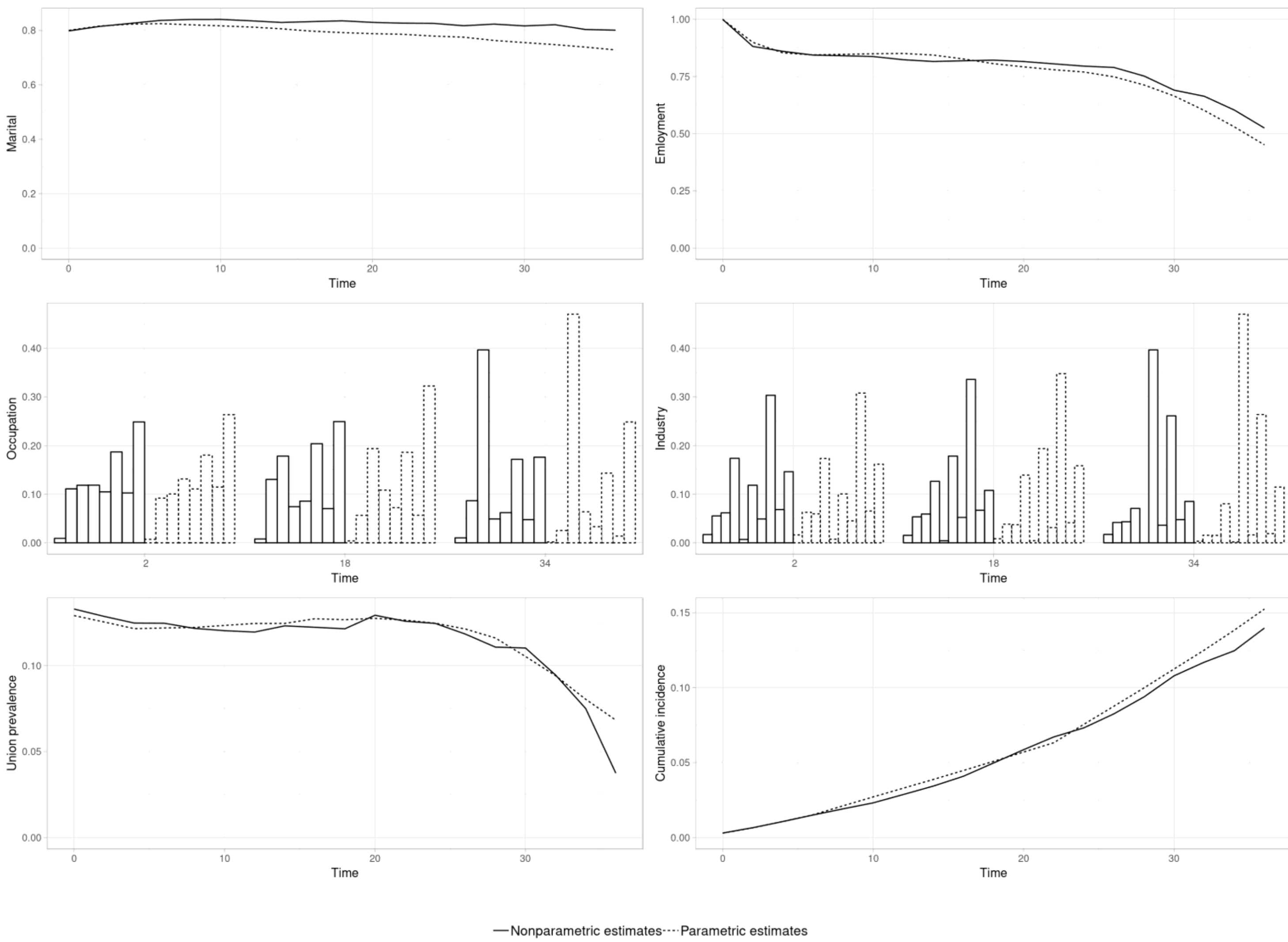


Marital
Time
Emloyment
Time
Occupation
Time
Industry
Time
Union prevalence
Time
Cumulative incidence
Time
Nonparametric estimates
Parametric estimates

Black

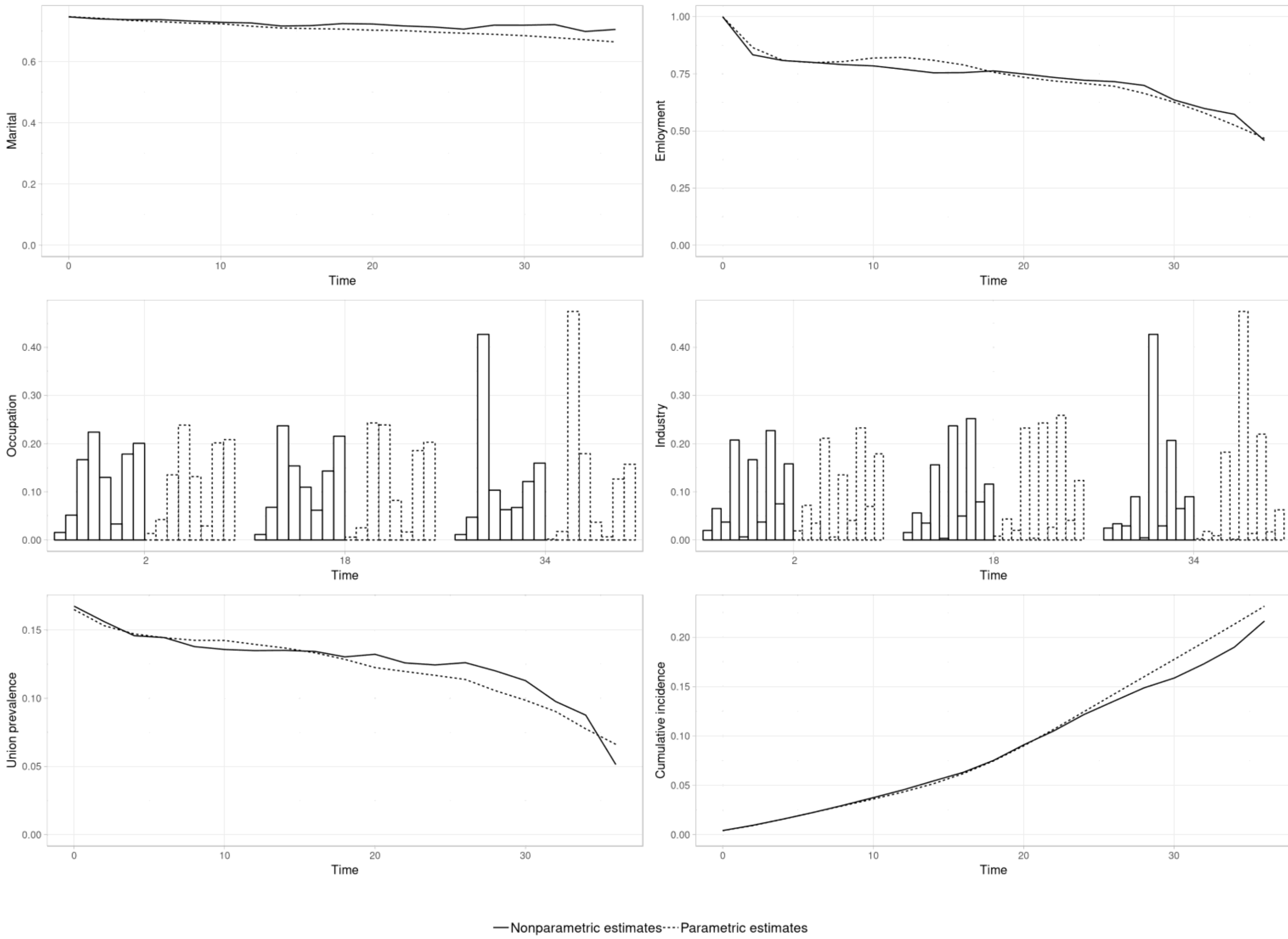

Marital
Time
Emloyment
Time
Occupation
Time
Industry
Time
Union prevalence
Time
Cumulative incidence
Time
Nonparametric estimates
Parametric estimates

White



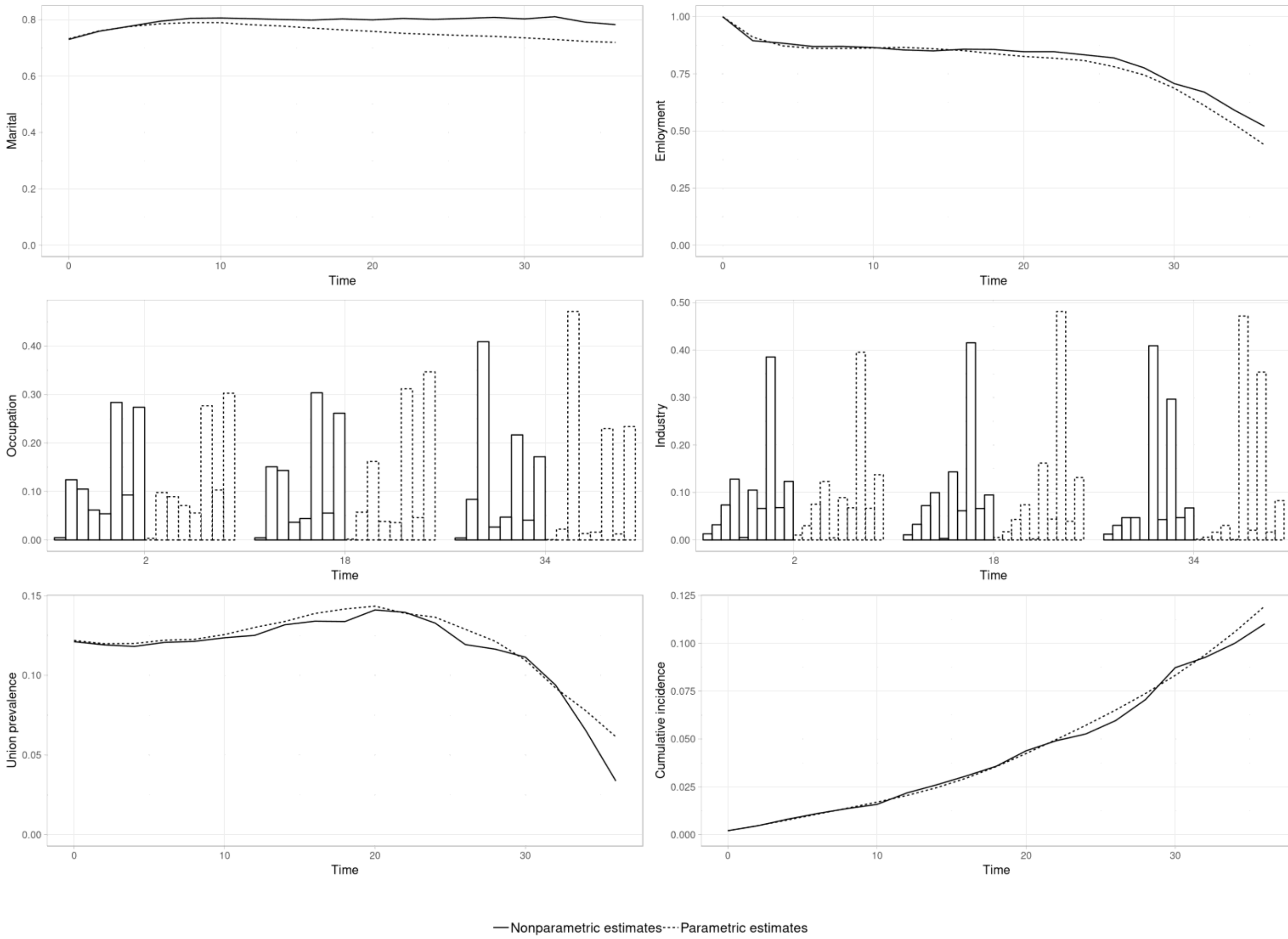
Marital
Time
Emloyment
Time
Occupation
Time
Industry
Time
Union prevalence
Time
Cumulative incidence
Time
Nonparametric estimates
Parametric estimates

≤HS



Marital
Time
Emloyment
Time
Occupation
Time
Industry
Time
Union prevalence
Time
Cumulative incidence
Time
Nonparametric estimates
Parametric estimates

>HS



Marital
Time
Emloyment
Time
Occupation
Time
Industry
Time
Union prevalence
Time
Cumulative incidence
Time
Nonparametric estimates
Parametric estimates

VITA

Jerzy Eisenberg-Guyot completed his PhD in Epidemiology at the University of Washington in June 2020. Jerzy completed his BA in Community Health and International Relations and his MPH in Epidemiology and Biostatistics at Tufts University. Jerzy's work focuses on political epidemiology and the political economy of health. In September 2020, Jerzy is starting a postdoctoral fellowship in the Psychiatric Epidemiology Training Program at Columbia University.