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Structural Racism and the Workplace: Impacts of Racial Diversity and Segregation on Cardiovascular Mortality in the United Autoworkers-General Motors Cohort

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Structural Racism and the Workplace: Impacts of Racial Diversity and Segregation on Cardiovascular Mortality in the United Autoworkers-General Motors Cohort

By

Hilary Lucinne Janjigian Colbeth

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requirements for the degree of
Doctor of Philosophy
in
Epidemiology
in the
Graduate Division
of the
University of California, Berkeley

Committee in Charge:

Professor Ellen A. Eisen, Chair
Professor Corinne Riddell
Professor Sadie Costello
Professor John R. Balmes

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Abstract

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The main objective of this dissertation was to investigate the cardiovascular disease (CVD) consequences of structural racism operating implicitly as discriminatory employment and labor policies and practices. Public health interventions targeting proximal risk factors such as health behavior have effectively reduced overall morbidity and mortality; yet, disparities in CVD mortality between Black and white populations persist even with a focus on working populations, which would presumably have similar baseline education, health, and healthcare access within skill levels.

The American Heart Association has stated that structural racism is a fundamental cause of disparities in CVD risk as it creates mutually reinforcing systems of unequal distribution of wealth, resources, opportunities, and power. First, we provide a background on the relationship between structural racism and cardiovascular health wherein ongoing interactions between macrosystems and institutions constrain the opportunities, resources, and power of socially racialized groups. This functions to prevent the elimination of Black-white cardiovascular health gaps. In the first chapter, we make explicit how structural racism is posited to function through the workplace in the form of labor and employment organizational practices and norms that impact CVD mortality. Next, we assess structural racism's influence on workforce policies and practices, particularly workplace racial diversity, which may present upstream targets for assessing and reducing racial health disparities in CVD. We evaluate whether a more racially balanced workforce would be protective against CVD mortality in a retrospective study of nearly 40,000 hourly Michigan autoworkers. We found that there was a protective effect of increasing exposure to racial diversity that was strongest and most consistent among Black workers. In this first chapter, our findings provided evidence that workplace practices encouraging diverse hiring and retention have the potential to improve all workers' health, particularly the socially racialized groups in that workforce.

Work as a social determinant of cardiovascular health and workplace exposures to particulate matter both remain understudied among Black working populations. In the next chapter, we

aimed to assess the risk of CVD mortality among Black autoworkers from the United Autoworkers – General Motors (GM) cohort study under several hypothetical scenarios: increasing exposure to workplace racial diversity, reduced metalworking fluid exposures, and both. Using longitudinal data from Black workers from the Detroit GM plant, we applied the parametric g-formula to assess the risk of CVD mortality under hypothetical scenarios with set values for plantwide racial diversity, and selected exposure limits for metalworking fluid, separately and jointly. Our findings were consistent with the hypothesis that interventions to increase racial diversity and decrease metalworking fluid would reduce CVD mortality risk. Our study underscored the importance of a racially diverse workplace in addition to reducing occupational hazard concerns for the prevention of CVD deaths among racialized populations.

Structural racism not only influences racial diversity, but also the ongoing racial segregation of Black Americans which has been associated with several cardiovascular outcomes. In recent decades, racial segregation has focused almost exclusively on measures of dissimilarity in residential spaces; however, little research has explored segregation beyond the residence. We present an analysis on the occupational structures and organization of work by examining the impact of job-based racial segregation and job mobility. This analysis used the index of dissimilarity to measure segregation and estimated exposure to cumulative job trajectories. We found that the relationship between increasing segregation and CVD mortality risk was highest among Black workers with lower job mobility. Black workers with higher job trajectories also experienced the harmful impacts of racial segregation, though at a lower magnitude. Among white workers, increasing racial segregation was harmful only for those with higher job mobility. Importantly, the higher-grade job assignments also have the most exposure to the GM occupational hazard of interest, metalworking fluid. Thus making our interaction between segregation and job mobility a cruicial method which can isolate pathways from segregation to CVD mortality via occupational hazards and psychosocial stress. Our findings provided evidence that workplace racial segregation may increase CVD mortality risk among Black workers, regardless of their career growth, and that addressing workplace racial segregation in concert with increasing opportunities for job mobility may be important to improving the long-term health of racialized working populations.

The hypotheses addressed in this dissertation are of public health importance considering the persistent racial disparities in CVD mortality and the gaps in the literature regarding the long-term health impacts of structural racism in the workplace. These studies provide information on the relationships between racial discrimination, occupational hazards, and CVD mortality, adding to the scientific literature that informs workplace policies and practices that aim to reduce racial disparities in preventable chronic disease.

To my mom, Denise, my dad, Richard, and my husband, Kristian, who always encouraged me to be curious and follow my own path.

&

This dissertation is dedicated to my family, friends, and colleagues, who lent me a great deal of support throughout graduate school.

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I dedicate this work to all of you.

Chapter 1. Background and Significance

This dissertation focuses on the association of workplace racial diversity and segregation in relation to cardiovascular disease (CVD) health and comprises five chapters. In this chapter, we summarize the current literature on workplace racial diversity and segregation and present hypotheses for how these group-level factors may impact cardiovascular disease outcomes in a working population. We also present a brief introduction to the study population, a subset of the United Autoworkers – General Motors (UAW-GM) cohort. In chapter 2, we present the first quantitative description of the UAW-GM cohort with known race (Black, white) and investigate impact of longitudinal exposure to racial diversity on CVD mortality and its subtypes, by race and sex. In chapter 3, we explore the associations between workplace racial diversity and the occupational hazard, metalworking fluid, separately and jointly on CVD mortality. We employ the parametric g-formula to investigate a spectrum of interventions on the diversity and metalworking fluid exposures. In chapter 4, we examine the relationship between workplace racial segregation and job mobility and CVD mortality. This work seeks to understand the importance segregation despite increasing diversity over time. Chapter 5 concludes the dissertation with a summary of our results, the strengths and limitations of the current work, and a discussion of possible next steps in occupational health disparities research.

Structural racism is defined by ongoing interactions between macrosystems and institutions to constrain the opportunities, resources, and power of marginalized racial groups.[1] Considered a fundamental cause of US racial health disparities,[2] structural racism influences a broad range of health-promoting resources that empower dominant social groups while simultaneously suppressing others. Based on Link and Phelan's fundamental cause theory,[3] a social cause of racial health disparities is comprised of four key constructs: (1) multiple disease outcomes are possible, (2) these outcomes are affected by multiple risk factors, (3) access to resources can mitigate disease sequalae or avoid risk all together, and (4) the health association can be reproduced through new mechanisms. Consistent with the theory, (1) Black-white racial disparities in CVD mortality have remained, independent of socioeconomic position (i.e., power, prestige, healthcare, economic, and neighborhood factors),[4] (2) these disparities in CVD have been shown to be driven by a variety of social, economic, environmental, psychological, behavioral, and physiological factors,[2] (3) quality health-promoting resources and opportunities that protect against CVD risk are often allocated by a racialized society (i.e., education, housing, job opportunities),[1] and (4) Black-white cardiovascular health gaps persist, even when medical care, treatment, and public health campaigns have dramatically improved population health.[5]

Based on this theory that structural racism is a fundamental cause of preventable chronic disease mortality, we will examine how its implicit influence on labor and employment practices via workplace organizational factors (racial diversity and segregation) impact racial disparities in CVD mortality in an occupational cohort. A primary motivation for studying workplace racial diversity and segregation is that societal hierarchies map onto workplace environments which perpetuate cardiovascular health inequities among working populations,[6] despite working populations being healthier at baseline than their general population counterparts.[7] Upstream socioeconomic and political contexts affect employment and labor practices, which translate to workplace hiring and retention.[8,9] These workplace policies and practices can affect job security,

work organization, and occupational health and safety, and workplace cultural norms, including interpersonal discrimination. Therefore, although racial discrimination in the workplace has been outlawed by Title VII of the Civil Rights Act of 1964, the effects of contemporary and historical structures of discrimination are still connected to cardiovascular health, to the workplace organization, and job trajectories.[10,11]

The potential links between workplace racial discrimination and cardiovascular outcomes are supported by strong theoretical frameworks and empirical evidence. Ecosocial theory, which posits that social and material contexts affect heath through pathways of embodiment,[12] provides a framework for understanding how the workplace may impact cardiovascular health.[8] These pathways become biologically embedded through physiological disruption that may alter multiple systems, including those that are metabolic and cardiovascular. The current literature has shown that perceived workplace discrimination, job strain, and low job control are linked to increased adverse cardiovascular outcomes.[13] Although work is regarded as a social determinant of health,[14] a major limitation of the existing epidemiologic literature is the lack of studies assessing the health consequences of racial diversity and racially motivated exclusion in the workplace. Moreover, there are few studies[15] that explore the cross-over between social and environmental exposures in occupational settings, much less with a focus on racialized populations.

The study population for this dissertation consists of 46,172 hourly workers who were ever employed for at least three years at three Michigan General Motors (GM) plants: Plant 1 in Detroit, Plant 2 in Ypsilanti, and Plant 3 in Saginaw. The study contract was originally awarded to a research team at Harvard in 1985, jointly funded by the United Autoworkers (UAW) union and the GM Corporation. It was initially designed to examine cancer risk in relation to workplace exposures to metalworking fluids.[16] Mortality follow-up begins in 1941 and the end of follow-up has been extended every 10 years from 1985 to 2015, with the support of the National Institute for Occupational Safety & Health (NIOSH).[17] The cohort currently includes 8,106 (20%) Black workers, most of whom worked at the Detroit plant.

Across the three GM plants and over the 75 years of follow-up there was a wide variation in racial composition (hereafter referred to as racial diversity). Black autoworkers made up anywhere from 5% to over 50% of the annual workforce in each of the plants over time and this variation provides the basis to assess whether plantwide racial diversity in the workplace contributes to improved CVD mortality rates among Black autoworkers. A racially diverse workplace may be segregated if members from different races work in isolation from each other. Therefore, we examined if racial segregation in the workplace functions as a pathway from structural racism to increased racial disparities in CVD mortality, a preventable cause of death. Our definitions of plantwide racial diversity and segregation by job are based on proportions of Black workers actively employed in each year and are both quantitative measures of structural racism that have not, to the best of our knowledge, been employed in occupational epidemiology. Our hypotheses are that increasing the racial diversity of the workforce to better reflect that of the surrounding county and decreasing segregation by job will improve long-term CVD outcomes for Black autoworkers.

This dissertation aims to address the gaps in the literature by (1) comparing the rates of CVD mortality in the GM workforce, the US, and state of Michigan, by race and over time, (2) examining the extent of a potential protective impact that increasing racial diversity in the GM workforce may have on CVD mortality, by race, (3) assessing the separate and joint impacts of social (diversity) and occupational (metalworking fluid) exposures on CVD mortality, and (4) understanding the magnitude of the harmful effects of racial segregation and low job trajectories may have on CVD mortality, while accounting for the racial diversity of the workforce. The results of this research can help advance our limited understanding of the mechanisms through which structural racism can operate in the workplace and its health consequences for non-racialized and racialized workers. This study may inform the implementation of anti-racism labor and employment policies, practices, and risk assessment strategies for upstream public health intervention. This research also has the potential to significantly contribute to reducing the persistent Black-white disparities in CVD mortality.

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Chapter 2. The Impact of Increasing Workforce Racial Diversity on Black-White Disparities in Cardiovascular Disease Mortality

2.1. Abstract

Structural racism's influence on workforce policies and practices presents possible upstream targets for assessing and reducing racial health disparities. This study is the first to examine workforce racial diversity in association with racial disparities in cardiovascular disease (CVD) outcomes. This retrospective cohort study of 39,693 hourly autoworkers from three Michigan automobile plants, includes 75 years of follow-up (1941-2015). Workforce racial diversity (percent Black autoworkers) was a plant and year level variable. Annual exposure was cumulated over each individual's working life and divided by time since hire. This time-varying measure was categorized into low, moderate, and high. We estimated age-standardized rates of CVD and cox proportional hazards ratios (HR) by race. CVD mortality per 100,000 person-years decreased among autoworkers over the study period; however, Black workers' rates remained higher than White workers. Among Black workers, we observed a strong protective association between greater workforce racial diversity and CVD mortality. For example, at the Detroit plant, the HR for moderate exposure to racial diversity was 0.94 (0.83, 1.08) and dropped to 0.78 (0.67, 0.90) in the highest level. Among White workers, results were mixed by plant, with protective effects in plants where less than 20% of workers were Black and null results where Black workers became the majority. Our findings provide evidence that workplace racial diversity may reduce CVD mortality risk among Black workers. Workplace practices encouraging diverse hiring and retention have potential to improve all workers' health; particularly the socially racialized groups in that workforce.

2.2. Introduction

Cardiovascular disease (CVD) remains a leading cause of death in the United States (US) despite advancements in CVD-related medical care, treatment, and public health campaigns.[1] The distribution of these benefits, however, has been far from equitable.[2] For example, racial inequities in age-adjusted heart disease mortality rates are persistently higher for Black Americans than any other racial/ethnic group.[3]

As a result, the American Heart Association has recently acknowledged structural racism, the totality of ways in which societies foster racial discrimination against marginalized groups,[4] as a fundamental cause of disparities in CVD risk.[5] Through mutually reinforcing systems, structural racism creates unequal distributions of resources, opportunities, power, and wealth.[4,6,7] In turn, these social and economic inequalities engender epidemiologic patterns of racial disparities in preventable diseases, such as CVD.[8] Some neighborhood studies have examined place-based social exclusion to identify the political, social, and economic systems that operationalize structural racism. By evaluating racialized socioeconomic indicators at the area-level, such as

historical redlining, researchers have revealed associations between systematic disinvestment in Black neighborhoods and adverse CVD outcomes.[9-11]

At a systemic level, structural racism can occur in contexts outside of the neighborhood, including workplaces. This can be evident in racialized workplace hiring and retention practices. We posit that the workplace offers opportunities to assess the cardiovascular consequences of structural racism because upstream socioeconomic and political context affects employment and labor practices;[12] thereby impacting job security, work organization, and occupational health and safety, including interpersonal discrimination. The connection between the workplace and CVD outcomes is also supported by strong theoretical frameworks, including Krieger's ecosocial theory.[13] This framework proposes that inequitable social and material contexts affect health via physiological pathways that embed repeated disruptions to metabolic and cardiovascular system functioning.

Although work is indeed recognized as a social determinant of health,[14] studies assessing the health consequences of racial diversity and racially motivated exclusion in the workplace are scarce.[15] The Sociology literature suggests that Black workers experience greater workforce retention, job satisfaction, perceived fairness, and decreased perceived race-based discrimination as the proportion of Black workers increased, though no health outcomes were examined.[16] To fill this research gap, we leverage longitudinal data (1941-2015) from the United Autoworkers – General Motors (UAW-GM) cohort, a mortality study of hourly automobile workers at three GM plants in Michigan. We hypothesized that exposure to an increasingly racially balanced workforce will improve long-term CVD outcomes for Black autoworkers.

2.3. Methods

Study Population

The UAW-GM cohort mortality study was originally designed in 1985 to assess the health implications of occupational exposures for Michigan workers employed by GM and has been described in detail previously.[17] The data includes birthdate, sex, race, and work history (until 1995) obtained from company records. This cohort includes all hourly workers identified through company records at three Michigan automobile manufacturing plants in Detroit, Ypsilanti, and Saginaw. This study population is composed of Black and White workers hired between January 1, 1938 and December 31, 1984. Details regarding ethnicity were not provided in the work records. Mortality follow-up begins three years after the subject's date of hire, to exclude short-term workers, and extends until 2015.

Outcome

Our outcomes of interest were cause-specific mortality attributed to overall cardiovascular disease, and separately, to subtypes cerebrovascular disease, ischemic heart disease (IHD), and acute myocardial infarction (AMI). Data on vital status were ascertained through the Social Security Administration, the National Death Index, plant records, and state mortality files. Cause of mortality was obtained from state vital records, death certificates, and the National Death Index. We used codes from the *International Classification of Diseases, Ninth and Tenth*

Revisions to define CVD (390-459, I00-78), cerebrovascular disease (430-438, I60-69), IHD (410, I21-22), and AMI (200-202, I21-22).

Exposure

Our primary exposure measure was annual average exposure to diversity (% Black workers). To measure the annual average diversity, first we constructed a time-varying plant-level variable measuring racial diversity (% Black workers) at each plant in each year (Figure 2.1). Each autoworker was thereby exposed to varying levels of racial diversity over time, which we cumulated during the employment and divided by the time since hire. Similar quantifications of average exposure are typically used in occupational studies.[18] We categorized plantwide racial diversity exposure into tertiles to avoid a linearity assumption.

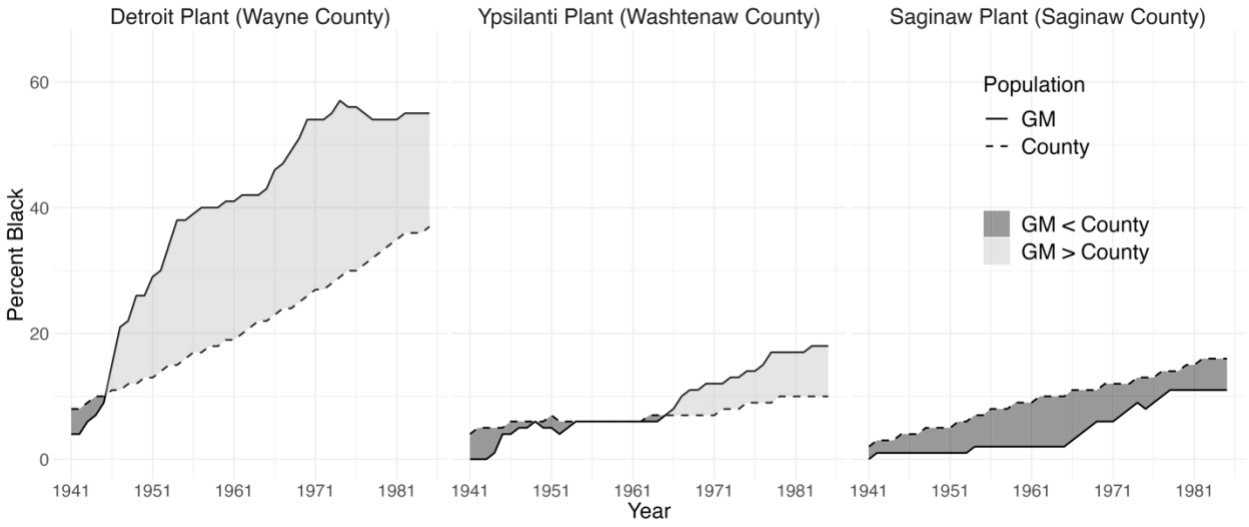

Figure 2.1. Percent Black active United-Autoworkers General Motors (UAW-GM) cohort workers in each year, by plant, and the percent Black residents in each year, by the Michigan county in which each plant was located (1941-1985). The percent Black workers at GM are represented by the solid lines and the percent Black of county residents are represented by the dashed lines. When the percent Black at GM was below the county, the difference is filled by dark grey and when GM percent Black workers is greater than the county, the difference is light grey.

Confounders

We used a directed acyclic graph (Figure 2.2) to identify confounders of the relationship between the exposure (% Black autoworkers), outcome (CVD mortality). The quantitative occupational exposure assessment for cumulative metalworking fluid (mg/m^3-year) has been described previously.[19,20] Year of hire was defined as a categorical variable with five-year bins, and calendar year was defined continuously. Annual county-level measures of the percent Black residents, percent below the poverty level, median household income, and percent with a Bachelor's degree or higher were extracted from the 1940-2000 US Censuses and the US Census Bureau's American Community Survey (ACS) from 2010-2015 in the plants' counties: Wayne

(Detroit plant), Washtenaw (Ypsilanti plant), and Saginaw (Saginaw plant). Annual county-level values between those reported by the US Census or ACS were linearly interpolated between the previous value and the next available value. We measured and adjusted for the racial diversity of the county where each plant was located in each year, to isolate the effect of exposure in the workplace from exposure in each worker's community. Information on age, sex (recorded as male or female), race (Black or White), plant location (Detroit, Ypsilanti, or Saginaw), birth date, and year of hire was obtained through employment records.

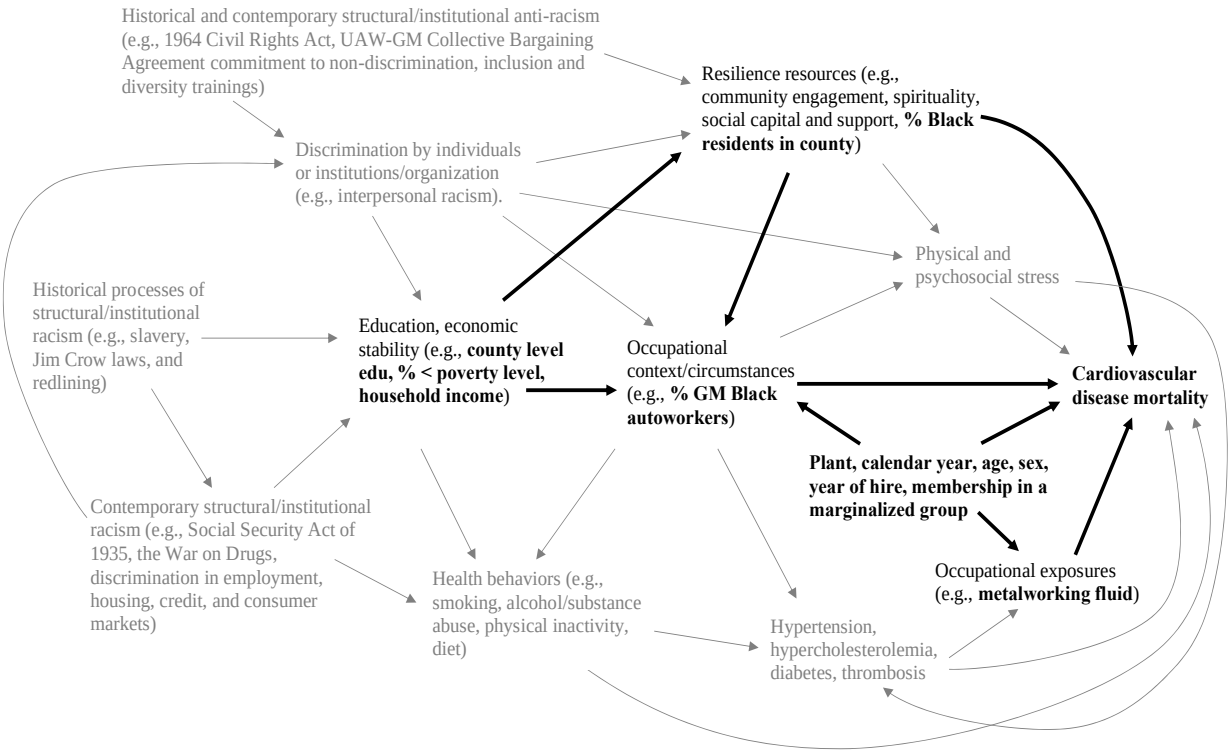

Figure 2.2. Directed acyclic graph (DAG) depicting the anticipated relationships between the exposure (% GM Black autoworkers) in each plant and year and the individual-level outcome (cardiovascular disease mortality), including the historical and contemporary processes that contribute to or push back against systemic racism, in all its forms. The pathways measured in the study are represented in bolded black arrows with this study's specifically measured variables in bolded black font. The contributing but unmeasured nodes and pathways are represented in grey arrows and font.

Statistical Analysis

We stratified the UAW-GM cohort by race and estimated age-adjusted CVD mortality rates among those of working age (24-65 years) from 1941-2015 by decade. Due to small numbers of CVD deaths at the beginning and end of follow-up of this open cohort, we combined 1941-1959 and 2000-2015. We calculated 95% confidence intervals for mortality rates using the gamma distribution for Poisson counts. Age-adjustment was performed via direct standardization based on the US 2000 population.[21]

We also calculated age-adjusted CVD mortality rates among those of working age (24-65 years) in the US and Michigan Black and White populations through the Centers for Disease Control and Prevention's (CDC) Wide-ranging Online Data for Epidemiologic Research (WONDER) database (https://wonder.cdc.gov/mortSQL.html). Using WONDER, we calculated annual rates over calendar year categories from 1968 (the earliest available year in WONDER) to 2015. As with the UAW-GM cohort rates, the annual rates from WONDER were age-standardized to the US 2000 population.

To examine associations between average exposure to percent Black workers and CVD mortality and its subtypes, we fit Cox proportional hazards models to estimate adjusted hazard ratios (HR) and 95% confidence intervals (CI).[22] We estimated cause-specific hazard ratios for the main analysis and, as a sensitivity analysis, sub-distribution hazard ratio for CVD mortality because other causes of death (e.g., cancers) may remove workers from the risk set before the event of interest is observed.[23] Cause-specific HRs were calculated representing the rate of CVD-related (or CVD subtype) deaths among workers with high levels of exposure (e.g., high plantwide diversity) relative to workers with low levels of exposure.[24,25] The subdistribution HRs were estimated using the Fine-Gray model.[23,24]

We ran the above models stratified by race given that the impact of plantwide racial diversity on cardiovascular disease mortality may differ by membership in a socially racialized group. Moreover, due to differences in both the percent and absolute numbers of Black autoworkers between plants (Figure 2.3), we avoid a positivity problem by also stratifying our models by plant. The Ypsilanti and Saginaw plants were combined which, comparatively to the Detroit plant, employed far fewer Black workers in any given year. As a secondary analysis, we categorized the exposure into quintiles in the Detroit plant and examined outcomes with the greatest number of cases, CVD and IHD.

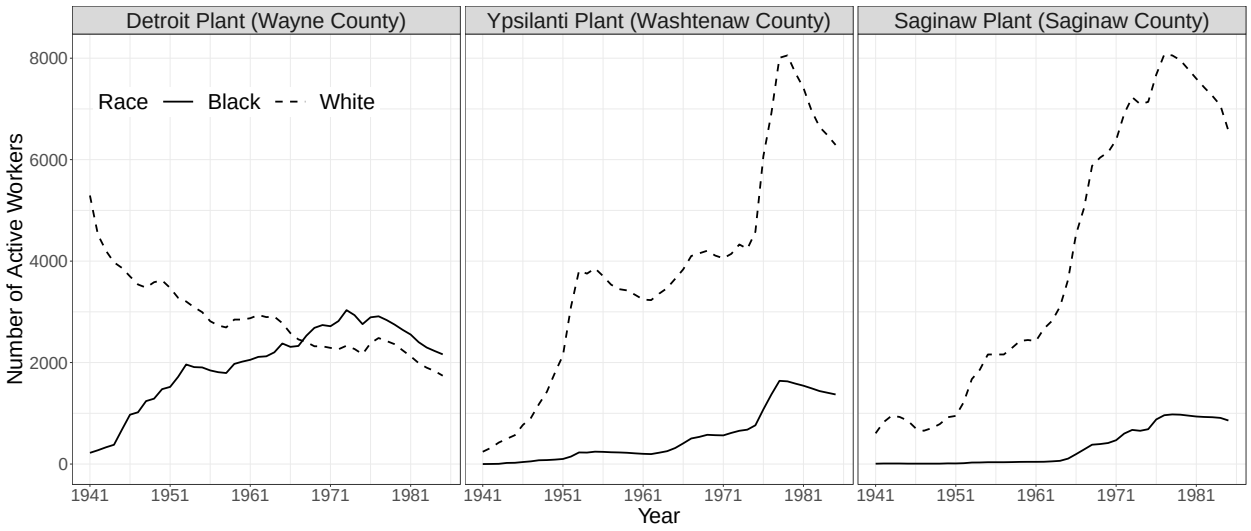

Figure 2.3. Number of active United Autoworkers – General Motors cohort workers in each year, by race and plant (1941-1985). Black autoworkers are represented by the solid lines and White workers are represented by the dashed lines.

All Cox models were adjusted for the following individual-level variables: plant (if the model is not restricted to one plant), sex, calendar year, year of hire, total cumulative metalworking fluid (mg/m^3-year), cumulative percent Black residents, percent below the poverty level, median household income, and percent with a Bachelor's degree or higher. The proportional hazards assumptions were assessed using a Wald test for the interaction between follow-up time and each covariate. Sensitivity analyses were conducted by restricting the main Cox models to males to examine the cardiovascular effects of an increasingly Black workforce on specific subgroups.

All analyses were conducted in SAS version 9.4 (SAS Institute Inc, Cary, North Carolina, USA). Data visualization was conducted in R version 12.0 (R Foundation for Statistical Computing, Vienna, Austria). This study was approved by the Committee for the Protection of Human Subjects at the University of California, Berkeley.

2.4. Results

Study sample characteristics

This UAW-GM cohort study included 39,693 autoworkers (>1 million person-years), of whom 8,106 (20%) were Black (Table 1). Far more Black autoworkers (62%) worked at the Detroit plant than at the other two locations (24% or lower). The distribution of White workers was more even across the three plants (29-37%). The proportion of CVD deaths among Black autoworkers due to IHD (48%) was lower than the proportion among White autoworkers (63%), as was the proportion of IHD deaths due to AMI (37% vs. 55% for Black and White autoworkers, respectively). Compared with White workers, Black workers experienced similar years of hire and years of employment; however, their median age at CVD death was younger and their average exposure to racial diversity was higher (20% vs. 6% Black autoworkers plantwide).

Table 2.1. Demographic Characteristics by Racial Category in the United Autoworkers – General Motors (UAW-GM) Cohort, 1941-2015

	Total Study Population		**Black Workers**		**White Workers**	
	N	%	N	%	N	%
Study Population (person-years)	39,693 (1,661,816)	100	8,106 (331,199)	100	31,587 (1,330,617)	100
Sex						
Male	35,341	89	6,811	84	28,530	90
Female	4,352	11	1,295	16	3,057	10
Plant						
Detroit Plant	14,637	37	5,047	62	9,590	30
Ypsilanti Plant	13,566	34	1,931	24	11,635	37
Saginaw Plant	11,490	29	1,128	14	10,362	33
All-cause mortality	22,932	58	4,572	56	18,360	58
Cardiovascular disease (CVD) [a]	9,868	43% of deaths	1,844	40% of deaths	8,024	44% of deaths
Cerebrovascular disease	1,312	13% of CVD	290	16% of CVD	1,022	13% of CVD
Ischemic heart disease (IHD)	5,969	60% of CVD	886	48% of CVD	5,083	63% of CVD
Acute myocardial infarction (AMI)	3,105	52% of IHD	330	37% of IHD	2,775	55% of IHD
	Median	**Q1, Q3**	**Median**	**Q1, Q3**	**Median**	**Q1, Q3**
Average annual racial diversity (% Black autoworkers)	7	4, 16	20	8, 38	6	4, 11
Detroit plant			34	22, 44	18	4, 32
Ypsilanti and Saginaw plants			8	5, 10	6	4, 8
Years of follow-up	38	33, 46	37	32, 44	38	33, 46
Year of hire	1967	1951, 1976	1970	1955, 1976	1965	1949, 1976
Years at work	19	13, 28	19	13, 27	19	13, 28

Cumulative metalworking fluid exposure (mg/m^3-year) [b]	8	4, 21	7	3, 18	8	4, 22
Year of death [c]	1992	1979, 2002	1996	1985, 2005	1990	1977, 2001
Age at death [c]	73	63, 81	69	59, 78	73	64, 82
County Level						
% Black residents	15	11, 12	23	12, 37	13	11, 19
% below the poverty level	14	12, 17	15	12, 17	13	12, 17
Median household income (USD)	22,382	10089, 38176	23267	11844, 38047	22,382	9904, 38176
% with a bachelor's degree or higher	20	9, 32	19	9, 27	21	7, 34

[a] Specific causes of death within are presented as proportions of a larger category of cases.
[b] Summary statistic calculated at the end of follow-up.
[c] Among those who died of a cardiovascular disease.

Age-standardized mortality rates

Race-stratified age-adjusted CVD mortality rates per 100,000 person-years for the GM cohort, the state of Michigan, and the US are presented in Figure 2.4. Over the entire follow-up period, the age-standardized CVD mortality rate (95% CI) for the GM cohort was 158.9 per 100,000 (95% CI: 152.8, 165.2) and was significantly higher among Black compared to White autoworkers, 203.3 (187.7, 219.9) and 147.9 (141.4, 154.7). From 1941 to 2015, age-standardized CVD mortality rates among Black workers decreased from 252.0 (175.5, 350.2) to 69.5 (59.8, 80.6) whereas White workers' rates declined from 234.0 (211.2, 258.9) to 40.1 (36.1, 44.4). Steady decreases and racial disparities were also reflected at the state and national level. Within racial categories, Michigan and US rates were comparable and nearly all state and national rates remained higher than GM rates over time. Among White populations, Michigan and US rates remained moderately above those of GM White workers. In contrast, the difference between Black Michigan and US rates and the Black GM population was consistently larger.

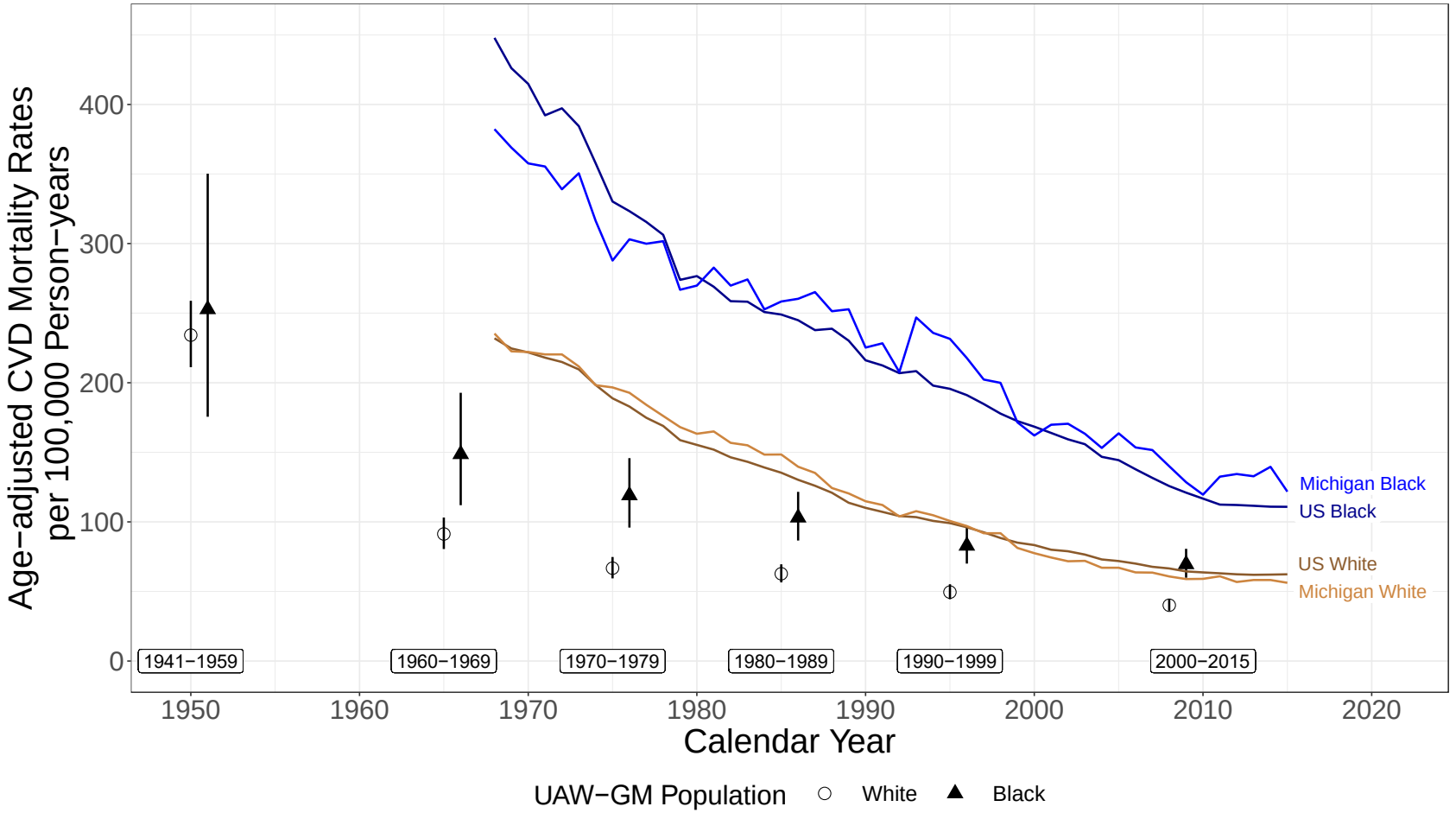

Figure 2.4. Age-adjusted cardiovascular disease mortality rates per 100,000 person-years among those 25-64 years with 95% confidence intervals among United Autoworkers – General Motors Black (triangles) and White (circles) workers, respectively. Rates for General Motors workers were estimated for time intervals 1941-1959, 1960-1969, 1970-1979, 1980-1989, 1990-1999, 2000-2015 and rates for the state of Michigan Black (light blue) and White (light brown) populations and the overall US Black (dark blue) and White (dark brown) populations from 1968-2015 were estimated annually. All rates are age-standardized to the US 2000 population.

Proportional hazards models for survival analysis

Among those in the Detroit plant, increasing exposure to annual average racial diversity at work was protective against CVD mortality for Black workers (HR=0.94 (95% CI: 0.83, 1.08) for moderate exposure; HR=0.78 (95% CI: 0.67, 0.90) for high exposure). Similar protective effects were estimated for the IHD and AMI outcomes among Black workers, with greater protection offered by the highest levels of exposure. For White workers, no protective effect against CVD mortality or its subtypes was found (Table 2.2).

Table 2.2. Adjusted hazard ratios restricted to the Detroit plant autoworkers and stratified by race, for cardiovascular disease and its subtypes in association with exposure to annual average racial diversity (percent Black) UAW-GM autoworkers (1941-2015).

Annual average racial diversity (% Black workers)	**Cardiovascular Disease**			**Cerebrovascular Disease**			**Ischemic Heart Disease**			**Acute Myocardial Infarction**		
	No. of cases	HR	95% CI	No. of cases	HR	95% CI	No. of cases	HR	95% CI	No. of cases	HR	95% CI
BLACK WORKERS [a]												
Low	484	1.00	-	76	1.00	-	229	1.00	-	91	1.00	-
Moderate	502	0.94	0.83, 1.08	85	1.09	0.78, 1.52	260	0.95	0.78, 1.15	78	0.87	0.63, 1.21
High	485	0.78	0.67, 0.90	72	0.90	0.61, 1.31	228	0.71	0.57, 0.88	108	0.79	0.57, 1.10
WHITE WORKERS [b]												
Low	1,299	1.00	-	178	1.00	-	872	1.00	-	568	1.00	-
Moderate	1,338	1.11	1.02, 1.21	201	1.17	0.92, 1.47	858	1.09	0.97, 1.21	507	1.01	0.88, 1.16
High	1,297	1.01	0.91, 1.11	157	0.97	0.74, 1.28	881	0.96	0.84, 1.08	588	1.01	0.87, 1.17

Abbreviations: United Autoworkers-General Motors (UAW-GM); hazard ratio (HR); confidence interval (CI).

[a] Low (0-19.08), moderate (19.09-28.85), and high (28.86-57.00); [b] low (0-3.42), moderate (3.43-19.41), and high (19.42-57.00). Cut-points for low, moderate, and high categories are based on the exposure distribution of the cardiovascular disease cases. Estimates of average intensity were calculated in each person-year as the cumulative exposure divided by time since first exposure. Cox models used age as the time scale and adjusted for sex, calendar year, year of hire (5-year bins), total cumulative metalworking fluid, and county levels variables cumulative % Black residents, percent below the poverty level, median household income, and percent with a Bachelor's degree or higher for each GM plant's county and year.

Our secondary analysis within the Detroit plant which categorized the exposure using quintiles (Figure 2.5) was reflective of the primary results.

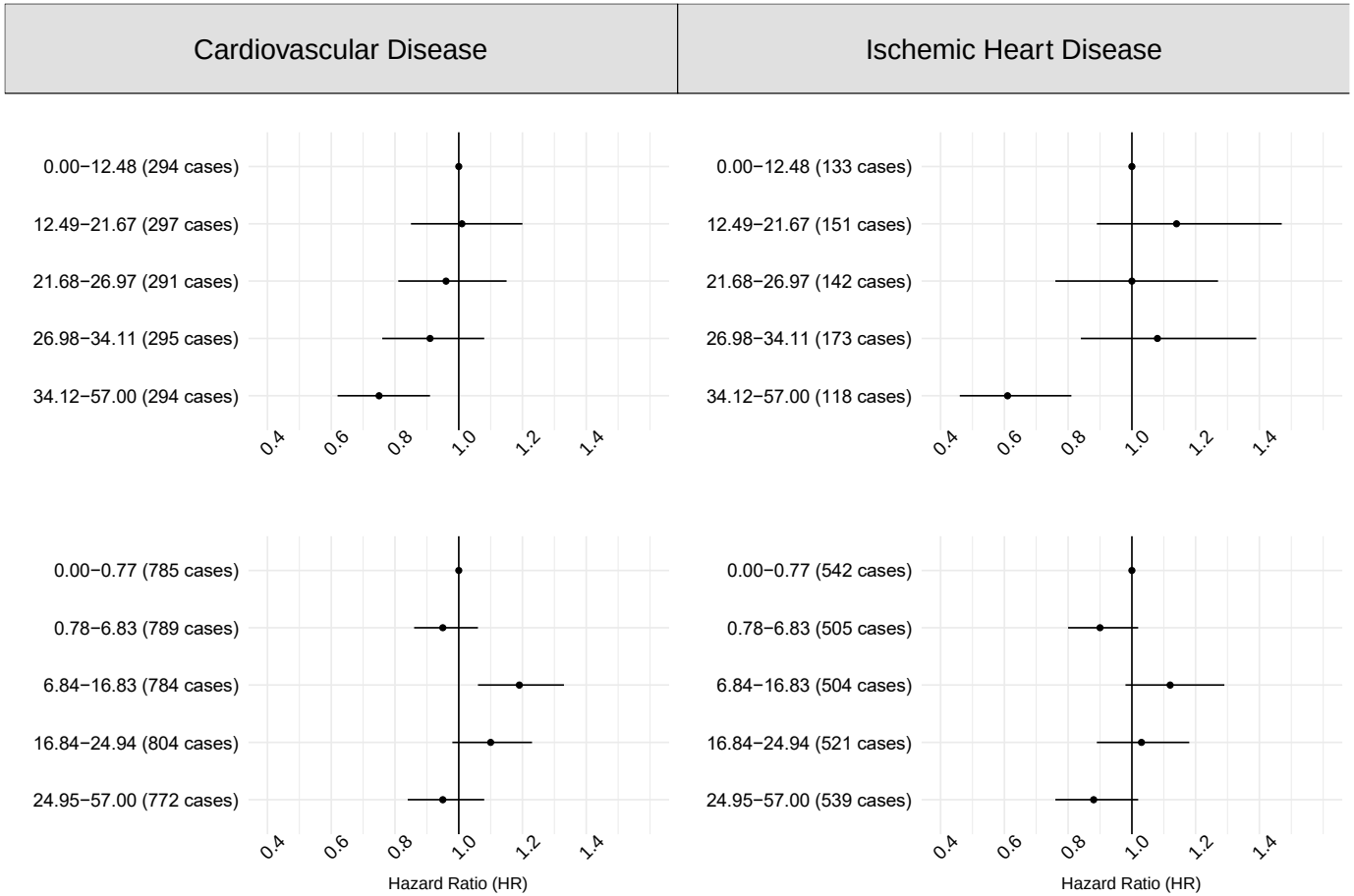

Figure 2.5. Adjusted hazard ratios restricted to the Detroit plant United Autoworkers – General Motors workers and stratified by race, for cardiovascular disease and ischemic heart disease in association with quintiles of annual average exposure to racial diversity (% Black) (1941-2015). All models were adjusted for the sex, calendar year, year of hire, total cumulative metalworking fluid (mg/m^3-year), cumulative percent Black residents, percent below the poverty level, median household income, and percent with a Bachelor's degree or higher.

Among autoworkers at Ypsilanti and Saginaw plants (Table 2.3), similar protective effects with increasing exposure to racial diversity were observed among Black workers. Based on 373 CVD deaths among Black workers, the HRs were strongly protective in the moderate (0.71 (95% CI: 0.54, 0.94)) and high (0.58, (95% CI: 0.43, 0.79)) exposure categories compared to the lowest level. We noted similar a protective exposure-response relationship for IHD, although the number of deaths were much lower for this subtype (n cases = 169). In contrast to the Detroit Plant, results among White workers at the Ypsilanti and Saginaw plants were protective with greater average diversity exposure (HR=0.69 (95% CI: 0.63, 0.79) for high exposure).

Table 2.3. Adjusted hazard ratios restricted to the Ypsilanti and Saginaw plant autoworkers and stratified by race, for cardiovascular disease and its subtypes in association with exposure to annual average racial diversity (percent Black) UAW-GM autoworkers (1941-2015).

Annual average racial diversity (% Black workers)	**Cardiovascular Disease**			**Cerebrovascular Disease**			**Ischemic Heart Disease**			**Acute Myocardial Infarction**		
	No. of cases	HR	95% CI	No. of cases	HR	95% CI	No. of cases	HR	95% CI	No. of cases	HR	95% CI
BLACK WORKERS [a]												
Low	121	1.00	-	19	1.00	-	61	1.00	-	20	1.00	-
Moderate	129	0.71	0.54, 0.94	16	0.80	0.39, 1.64	61	0.60	0.40, 0.90	13	0.43	0.20, 0.92
High	123	0.58	0.43, 0.79	22	1.03	0.48, 2.22	47	0.49	0.31, 0.77	20	0.57	0.27, 1.18
WHITE WORKERS [b]												
Low	1,349	1.00	-	170	1.00	-	798	1.00	-	369	1.00	-
Moderate	1,389	0.78	0.72, 0.85	168	0.75	0.59, 0.95	831	0.79	0.71, 0.89	316	0.83	0.70, 0.98
High	1,352	0.69	0.63, 0.79	148	0.69	0.52, 0.89	843	0.71	0.64, 0.80	427	0.84	0.70, 1.00

Abbreviations: United Autoworkers-General Motors (UAW-GM); hazard ratio (HR); confidence interval (CI).
[a] Low (0-3.68), moderate (3.69-5.93), and high (5.94-17.57); [b] low (0-2.81), moderate (2.82-4.94), and high (4.95-17.57).
Cut-points for low, moderate, and high categories are based on the exposure distribution of the cardiovascular disease cases.
Estimates of average intensity were calculated in each person-year as the cumulative exposure divided by time since first exposure.
Cox models used age as the time scale and adjusted for plant, sex, calendar year, year of hire (5-year bins), total cumulative metalworking fluid, and county levels variables cumulative % Black residents, percent below the poverty level, median household income, and percent with a Bachelor's degree or higher for each GM plant's county and year.

Sensitivity analyses

Sub-distribution HRs that accounted for competing risks also followed similar patterns as the main results (Tables 2.4 and 2.5).

Table 2.4. Subdistribution hazard ratios (sdHR) and 95% Confidence Intervals (CI) for cardiovascular disease in relation to levels of racial composition among UAW-GM autoworkers, by race and restricted to the Detroit plant (1941-2015)

Annual average racial diversity (% Black workers)	No. of cases	sdHR	95% CI
BLACK WORKERS [a]			
Low	484	1.00	-
Moderate	502	0.94	0.82, 1.08
High	485	0.77	0.66, 0.90
WHITE WORKERS [b]			
Low	1,299	1.00	-
Moderate	1,338	1.10	1.00, 1.20
High	1,297	1.09	1.00, 1.20

Abbreviations: United Autoworkers-General Motors (UAW-GM).
[a] Low (0-19.08), moderate (19.09-28.85), and high (28.86-57.00); [b] low (0-3.42), moderate (3.43-19.41), and high (19.42-57.00). Estimates of average annual racial diversity were calculated in each person-year as the cumulative exposure divided by time since hire. All models were adjusted for sex, calendar year, year of hire, total cumulative metalworking fluid (mg/m^3-year), cumulative percent Black residents, percent below the poverty level, median household income, and percent with a Bachelor's degree or higher.

Table 2.5. Subdistribution hazard ratios (sdHR) and 95% Confidence Intervals (CI) for cardiovascular disease in relation to levels of racial composition among UAW-GM autoworkers, by race and restricted to the Ypsilanti and Saginaw plants (1941-2015)

Annual average racial diversity (% Black workers)	No. of cases	sdHR	95% CI
BLACK WORKERS [a]			
Low	121	1.00	-
Moderate	129	0.71	0.53, 0.95
High	123	0.61	0.44, 0.84
WHITE WORKERS [b]			
Low	1,349	1.00	-
Moderate	1,389	0.78	0.72, 0.85
High	1,352	0.70	0.63, 0.77

Abbreviations: United Autoworkers-General Motors (UAW-GM).
[a] Low (0-4.94), moderate (4.94-8.82), and high (8.82-17.73); [b] (0-2.83), (2.83-5.98), and (5.98-17.89). All models were adjusted for plant, sex, calendar year, year of hire, total cumulative metalworking fluid (mg/m^3-year), cumulative percent Black residents, percent below the poverty level, median household income, and percent with a Bachelor's degree or higher.

Overall, the sensitivity analyses restricted to males found that the protective effects of higher racial diversity for black workers did not differ from the main results (Tables 2.6 and 2.7).

Table 2.6. Adjusted hazard ratios restricted to the Detroit plant male autoworkers, by race, for cardiovascular disease and its subtypes in association with exposure to average intensity percent Black UAW-GM autoworkers (1941-2015)

Annual average racial diversity (% Black workers)	**Cardiovascular Disease**		
	No. of cases	HR	95% CI
BLACK MALE WORKERS [a]			
Low	465	1.00	-
Moderate	479	0.95	0.82, 1.09
High	464	0.77	0.66, 0.89
WHITE MALE WORKERS [b]			
Low	1,294	1.00	-
Moderate	1,332	1.11	1.01, 1.21
High	1,292	1.00	0.91, 1.10

Abbreviations: United Autoworkers-General Motors (UAW-GM); hazard ratio (HR); confidence interval (CI). [a] Low (0-19.2), moderate (19.3-28.9), and high (29.0-57.0); [b] (0-3.4), (3.5-19.4), and (19.5-57). Estimates of average annual racial diversity were calculated in each person-year as the cumulative exposure divided by time since hire. All models were adjusted for calendar year, year of hire, total cumulative metalworking fluid (mg/m^3-year), cumulative percent Black residents, percent below the poverty level, median household income, and percent with a Bachelor's degree or higher.

Table 2.7. Adjusted hazard ratios restricted to the Ypsilanti and Saginaw male autoworkers, by race, for cardiovascular disease and its subtypes in association with exposure to average intensity percent Black UAW-GM autoworkers (1941-2015)

Annual average racial diversity (% Black workers)	Cardiovascular Disease		
	No. of cases	HR	95% CI
BLACK MALE WORKERS [a]			
Low	104	1.00	-
Moderate	107	0.69	0.52, 0.93
High	102	0.50	0.37, 0.70
WHITE MALE WORKERS [b]			
Low	1,198	1.00	-
Moderate	1,232	0.74	0.67, 0.81
High	1,196	0.65	0.59, 0.71

Abbreviations: United Autoworkers-General Motors (UAW-GM); hazard ratio (HR); confidence interval (CI). [a] Low (0-3.7), moderate (3.8-5.8), and high (5.9-17.6); [b] (0-2.9), (3.0-5.0), and (5.1-17.6). Estimates of average annual racial diversity were calculated in each person-year as the cumulative exposure divided by time since hire. All models were adjusted for plant, calendar year, year of hire, total cumulative metalworking fluid (mg/m^3-year), cumulative percent Black residents, percent below the poverty level, median household income, and percent with a Bachelor's degree or higher.

2.5. Discussion

Using an ecosocial framework to acknowledge the inextricably social nature of work and the biological embodiment of discrimination that can lead to poor health outcomes,[12] this study of hourly Michigan autoworkers from 1941-2015 aimed to capture the cumulative impact of structural racism using our metric of workplace racial diversity. Similar to trends in the US and Michigan, we observed decreasing rates of CVD mortality among Black and White autoworkers over time, though Black autoworkers' rates remained higher than White workers. Among Black autoworkers, we observed a strong protective association between greater workforce racial diversity and CVD mortality. For example, among Black workers at the Detroit plant, the HR dropped to 0.78 (0.67, 0.90) at the highest level of exposure to racial diversity.

Overall age-adjusted CVD mortality rates steadily decreased over time for both Black and White autoworkers. Compared to the state and national populations, CVD mortality rates among GM workers were consistently lower. This difference between the CVD mortality rates of the general and UAW-GM populations partially reflects the healthy hire effect, as the workers were all healthy enough to be hired and thus experience more favorable mortality outcomes than the population at large.[26] The difference was especially striking between the Black general and worker populations, suggesting that Black GM workers who were hired were an even more highly select group of healthy individuals and/or that a stable job with benefits offered them more protection from CVD mortality than the White workers.[27]

Overall, mortality rates of the US and Michigan White populations were consistently lower than the respective Black populations. These racial disparities are downstream of the high burden of CVD risk factors among Black US adults compared to their White counterparts.[28] Comparatively, at GM, we observed that the disparities between Black and White autoworkers begin with smaller differences that reduced over time. The smaller and narrowing gap in CVD mortality risk among the autoworkers underscores the important role of employment and labor practices in disrupting systems of discrimination that affect physical and psychosocial stress, which may translate into adverse cardiovascular health outcomes and related racial inequities. Often, research focused on closing racial gaps in the general population investigate the benefits of health behavior change or the built environment.[29] However, when examined within this socioeconomic strata of hourly autoworkers, the health consequences of structural racism are perhaps better explained by assessing the uniquely racialized workplace policies and practices that can be targets for intervention and reform.[30] Our primary analysis aimed to do so by studying the impacts of increasing workplace racial diversity by race and baseline demographics.

This study provides empirical support for an association between plantwide racial diversity and CVD mortality risk. Our primary results showed strong protective associations from CVD and the subtypes IHD and AMI among Black workers as racial diversity increased. These results were robust to sensitivity analyses among males only and in the models' handling of competing risks. As evidenced by our consistent results across plants, the protective effects among Black workers of increasing proportions of Black co-workers remained, regardless of the absolute number of Black workers employed at the plant. The lack of impact of the plant environments is supportive of research suggesting that those in socially racialized group(s) find moderate health benefits from social support networks and collective efficacy.[31,32] Moreover, our results are consistent with literature documenting that social environment indicators of support may have a strong effects on cardiovascular health in Black cohorts.[33,34]

By contrast, among White workers, our results differed by the number of Black workers in the plants, with null results where there were many Black workers (i.e., Detroit plant) and protective effects where their numbers were low (i.e., Ypsilanti and Saginaw plants). The non-symmetry hypothesis[35] proposes that socially racialized and dominant groups may experience workplace racial diversity thresholds differently. In the Ypsilanti and Saginaw plants, the absolute numbers of Black workers (a maximum of 1,640 and 978 in 1978, respectively (eFigure 3)) were dramatically lower than that of White workers. However, in the Detroit plant, White GM workers were no longer the majority after 1967. Research suggests that White workers in a majority non-White work environment may experience heightened anxiety or status threat because they are not used to occupying a numeric minority status.[35] Additionally, racial demographic shifts can be perceived as threatening, impacting the White groups' attitudes toward racialized groups.[36] Therefore, among White workers, we may have observed a threshold effect based on plantwide racial demographics. These findings highlight the importance of measuring and investigating structural and policy-level exposures that capture the compounding exposures faced unilaterally by Black workers.[4,37]

Our study has notable methodological considerations. One limitation is possible confounder misclassification of the percent Black population living in the county in each year. Home

addresses of the workers were unknown, suggesting residual confounding if the worker lived in a different county than they worked in.[38] Second, autoworkers comprising the categories "Black" and "White" may be heterogenous and ethnicity is unknown; therefore, we may be missing important subgroup specific effects. That said, census reports estimate that the percentage of other races and of ethnicities, such as Hispanic, was low in the Michigan counties Detroit, Ypsilanti, and Saginaw.[39] Our cohort involves long-term follow-up of a large group of Black and White workers with similar incomes, and health and pension benefits by mid-life. Finally, our results may suffer from the healthy worker hire effect because people hired into physically demanding jobs are healthier at baseline than the general US population. Therefore, the generalizability of these results should be limited to workers employed in similar occupations.

2.6. Conclusions

To our knowledge, this is the first study of the association between workplace racial diversity and CVD mortality. Our results suggest the workforce may be an upstream target for the reduction of racial health disparities[40] because hiring and retention practices and policies offer important opportunities to decrease racial inequities and subsequent health disparities. We advocate that developing interventions to encourage social support, representation, and inclusivity can reduce harms from racial exclusions and are key to the health of socially racialized groups in a workforce.

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Chapter 3. Workplace racial diversity, metalworking fluid exposure, and cardiovascular disease mortality among Black US autoworkers

3.1. Abstract

Work as a social determinant of cardiovascular disease (CVD) and occupational exposures to particulate matter and other cardiovascular hazards remain understudied in Black working populations. Our objective was to assess the risk of CVD mortality among Black autoworkers from the United Autoworkers-General Motors cohort study under several hypothetical scenarios: increased exposure to workplace racial diversity, reduced metalworking fluid exposure, and both. We analyzed longitudinal data on 5,043 Black workers from a Detroit General Motors plant (follow-up 1941-2015), with 1,458 observed CVD deaths. We applied the parametric g-formula to assess risk under hypothetical scenarios with set values for plantwide racial diversity and selected exposure limits for metalworking fluid, separately and jointly. When assessing the impact of increasing racial diversity we compared plantwide racial diversity set to the 10th percentile of the observed distribution (38% Black workers) to racial diversity set to the maximum observed value (64% Black workers). For this comparison, at age 80, the CVD mortality risk difference per 1000 was 29.7 (95% confidence interval: -8.6, 74.8) and the corresponding relative risk was 1.12 (95% confidence interval: 0.97, 1.32). We observed 6 to 7 additional deaths from CVD comparing higher to lower metalworking fluid limits based on the 0.5 mg/m^3 NIOSH recommended exposure limit. Our findings are consistent with the hypothesis that interventions to increase racial diversity and decrease metalworking fluid would reduce CVD mortality risk. Our study underscores the importance of a racially diverse workplace in the prevention of CVD deaths among racialized populations.

3.2. Introduction

An ongoing challenge for public health in the U.S. today is the persistence of racial and ethnic disparities in cardiovascular disease (CVD) mortality, despite substantial progress over the past 40 years.[1] The American Heart Association has pointed to structural racism as the cause of the higher burden of CVD risk factors and mortality among Black adults compared to their white counterparts.[2,3] In addition, researchers posit structural racism as the root of the disproportionately high exposure to airborne particulate matter among socially racialized communities.[4] Shown to impact intermediary points on the inflammatory and oxidative stress pathways and in autonomic function,[5] fine particulate matter is linked to increased risk of several cardiovascular outcomes such as myocardial infarction, stroke, and heart failure.[6]

Similar biologic pathways to cardiovascular disease among automobile workers may follow exposure to metalworking fluids (MWFs).[7-11] MWFs are widely used in machining operations as coolants and lubricants to lower high temperatures during grinding, cutting, and drilling processes. Previous research has conceptualized and measured aerosolized MWFs as a fine particulate matter exposure.[7,12-15] Given that occupational exposures often exceed ambient US air pollution levels by at least an order of magnitude,[8] studies of occupational settings present

valuable opportunities to investigate the long-term impacts of particulate matter. However, the limited evidence that occupational particulate matter exposure increases the risk of heart disease mortality comes primarily from studies of white men.[16] Furthermore, the chronic effects of psychosocial stress due to racial discrimination may exacerbate environmental impacts on cardiovascular health.[17] These gaps in the literature call for continued research on the causal impacts of occupational exposures among racialized populations, such as Black workers, who often experience an excess burden of adverse workplace exposures.[18]

Although there is a growing body of literature on racial disparities in fine particulate matter exposures[19,20] and on community and social conditions[21,22] as drivers of CVD health disparities, there are few cross-investigations between these research areas.[23] We are also guided by the ecosocial theory of disease distribution and its focus on how people biologically embody their societal context, thereby shaping population patterns of morbidity and mortality.[24,25] Environmental and social risks commonly co-occur among working populations, and the independent effects of one exposure may lead to underestimated or mis-specified associations with the outcome of interest.

Structural racism involves interconnected institutions both historically rooted and culturally reinforced that foster inequitable systems of racial discrimination.[26] Therefore, causal epidemiologic questions aiming to confront structural racism should operate above the individual level because structural change or collective action must occur at the systems level.[27] Scholars have theorized that social organizations, including the workplace, are racialized, in that they often maintain practices and policies that undermine diversity and inclusion and reinforce systemic inequalities.[28] This means that structural racism may be a critical mechanism behind workplace racial diversity and subsequent cardiovascular health risk. For these reasons, we assess the impacts of workplace racial diversity on a racialized population by focusing on Black workers at a Detroit automobile plant who likely experienced similar overall socioeconomic status and neighborhood contexts outside of work.

The objective of this study was to estimate the individual and joint causal effects of plantwide racial diversity and occupational exposure to metalworking fluid on CVD mortality in the United Autoworkers-General Motors (UAW-GM) cohort. We hypothesize that the joint impacts of higher plantwide racial diversity and lower MWF exposure will reduce CVD mortality risk among Black workers more than the sum of the individual exposures. The study data are composed of extensive retrospective work and vital records from an iconic cohort of Black and white autoworkers in UAW-GM's Detroit plant from 1941 through 2015.

3.3. Methods

Study population

The UAW-GM cohort has been described in detail previously.[29-31] Briefly, the original study included all hourly workers hired between 1938 and 1982 who worked at least 3 years at one of three automobile manufacturing plants in Michigan, USA. This study includes 5,043 Black hourly workers employed at the Detroit plant who were hired no younger than age 16. Information on date of birth, year of hire, race, sex, and plant was ascertained from employment

records. Follow-up began three years after hire and ended at the time of death, age 80, or on December 31, 2015, whichever occurred first.

Outcome ascertainment

Data on vital status and cause of death were obtained through the Social Security Administration, the National Death Index, plant records, death certificates, and state mortality files.[29] The outcome of interest was mortality from CVD (*International Classification of Diseases, Ninth and Tenth Revision*s: 390-459, I00-78). We identified 1,458 CVD deaths among the workers during follow-up from 1941 through 2015.

Exposure assessments

Annual average exposure to racial diversity was calculated as the percent of Black workers actively working in each year. Each worker was thereby exposed to varying levels of racial diversity over time, which was cumulated during the course of employment.

Annual average daily exposure to MWF was estimated for each worker by combining detailed employment records with a time-varying job-exposure matrix. The matrix was based on an extensive retrospective exposure assessment.[31,32] MWF concentrations characterized by particle-size fraction were estimated for homogeneous exposure groups as an 8-hour time-weighted average (mg/m^3) based on measurements collected in 1985. Scale factors were developed to adjust MWF concentrations for temporal trends, including decreases in MWF exposure concentrations in the early 1970s following the passage of the Occupational Safety and Health Act.[32] This study uses total particulate matter to measure the combined exposure to MWF from all three types (straight, soluble, and synthetic), which deposit mostly in the tracheobronchial and alveolar regions of the lung. Annual average exposure to MWF was calculated for each worker by combining work histories with estimated exposure concentrations. Cumulative exposure is the sum of annual exposures. Gaps in work history information for individual workers were linearly interpolated by averaging exposures from previous and subsequent jobs.

Although follow-up extends to 2015, work history records were available only until December 31, 1994. For workers who were still employed when work records ended, we assumed that those workers stayed in their last job assignment and were exposed to the same level of MWF and racial diversity until reaching 55 years of age, as the age of eligibility for retirement with partial benefits was set to 55 years in 1964.[33] Alternatively, we conducted a sensitivity analysis which assumed their MWF and racial diversity exposures dropped to zero after 1994. We also conducted a sensitivity analysis in which racial diversity dropped to zero after 1985, when the original open cohort ended.

Statistical methods

The directed acyclic graph in Figure 3.1 depicts the assumed causal relationships between plantwide racial diversity, MWF exposure, and the outcome of interest, CVD mortality. The parametric g-formula is an approach used to quantify the expected distribution of outcomes under changes to the distribution of some exposure of interest. The method is essentially an

extension of standardization to a time-varying framework and allows estimation of population average risk under hypothetical exposure scenarios that differ from each other, and from the observed (counterfactuals).[34] Here, we are interested in quantifying the effects on mortality of hypothetical changes to plantwide racial diversity and to occupational MWF exposure limits over the course of follow-up. The parametric g-formula allows us to estimate the effects of such interventions, if identification assumptions (described later) are met.[34,35]

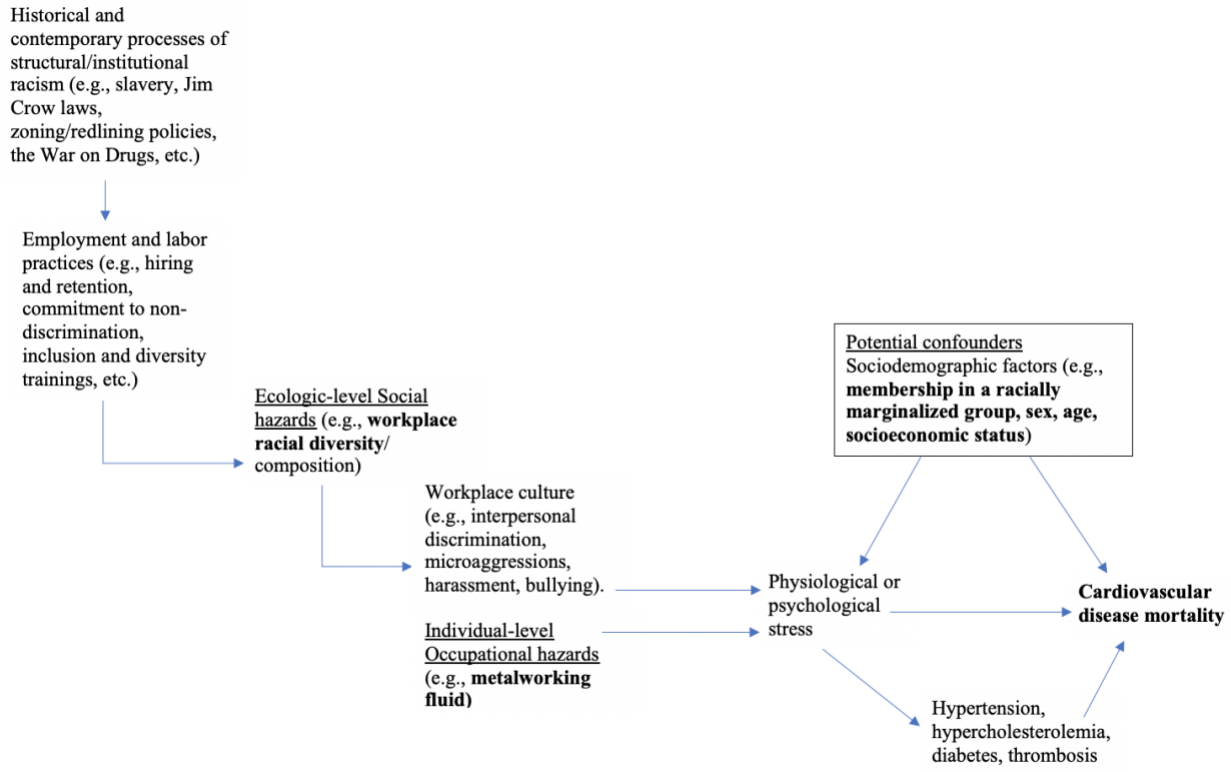

Figure 3.1. A directed acyclic graph for understanding the individual and joint contributions of workplace racial diversity and metalworking fluid exposures on cardiovascular disease mortality.

We applied the parametric g-formula to assess the risk of CVD mortality under a simulated natural course (i.e., what was observed) and under various hypothetical scenarios of changing exposures.[36] Counterfactual cumulative risk at age 80 years was estimated under the natural course and a series of hypothetical scenarios setting a plant-wide racial diversity (% Black) level based on the observed racial diversity distribution over all person-time. We set plantwide diversity in our scenarios using the maximum level and various percentiles. Then, scenarios limiting the maximum average daily MWF exposure levels based on the current National Institute for Occupational Safety and Health (NIOSH) recommended exposure limit for MWF aerosols of 0.50 mg/m^3 for total particulate mass.[37] We then estimated counterfactual risks with both racial diversity and MWF interventions together.

Details on the parametric g-formula can be found elsewhere,[34,36,38] and in the Supplementary Appendix S1, specifically for its application in the current study. In this study, we fit parametric models for the outcome (CVD mortality), competing events (non-CVD mortality), the two separate exposures (plant-wide racial diversity, MWF), and the time varying covariate employment status, based on the observed data. These models were all conditional on calendar year, age, prior exposure, and covariate histories as well as baseline covariates (sex and year of hire). Models were fit on the person-year level. Annual average daily exposure estimates were entered in all models for both exposures, while the outcome and competing events models also included terms for cumulative exposure up to the prior year. We used restricted cubic splines for MWF exposure variables as predictors because analyses based on conditional Cox proportional hazards models indicated nonlinearity of the exposure-response for cerebrovascular disease (a subtype of CVD), with an increasing trend at lower exposures and a decreasing trend at moderate to high exposures.[39]

Based on the observed distributions of the baseline covariates, we then generated a large pseudo-sample (n=100,000). In this pseudo-sample, we simulated exposure, covariate, and outcome values at each age ≤ 80 using the estimated parameters from the models above. For predicting the natural course, the simulation uses values for the exposure and time-varying covariates that are predicted from the models and then predicts the risk under those covariate and exposure histories. Our model specifications were selected with the goal of minimizing the difference between the observed and simulated natural course cumulative CVD mortality, a strategy for reducing model misspecification. We evaluated the predicted risk by comparing (1) the cumulative incidence of CVD mortality in the observed data and the simulated natural course, and (2) the cumulative risk of CVD mortality at age 80 years in the observed data to that simulated in the natural course. Assuming the natural course accurately represents the observed data, we can then 'intervene' in this pseudo-cohort to fix a new level of exposure.

For the hypothetical exposure scenarios, the exposure values were changed from the predicted values according to a fixed level (racial diversity) or set to a specific limit (MWF). For racial diversity, all exposures were set to the fixed level, regardless of an individual's observed value. Whereas, under a maximum hypothetical MWF exposure limit of 0.5 mg/m^3, all predicted MWF values above 0.5 mg/m^3 were replaced with 0.5 mg/m^3; otherwise, they remained unchanged. Values for all covariates at the subsequent time point, including probabilities of death due to competing risks and to CVD, were then predicted using the assigned exposure(s) and observed covariate values at each age and the estimated coefficients from the covariate, outcome, and competing risks models. The results for single interventions on racial diversity are adjusted for MWF as a time-varying covariate and, conversely, results for single interventions on MWF are adjusted for racial diversity.

We estimate cumulative incidence of mortality ("risk") from CVD. We estimated the counterfactual risk under each hypothetical exposure scenario described below and compared it to the risk under an "ideal" scenario (i.e., fixed intervention at the highest racial diversity exposure (64% Black workers); intervention at the lowest MWF exposure limit (0.05 mg/m^3)) on the ratio and difference scales. The standard deviation (SD) of estimates from 500 bootstrap

samples was used as an estimate of the standard error to generate 95% confidence intervals (CI).[40]

Our counterfactual risk scenarios for racial diversity were equal to the median (54% Black workers), 25th (43%), and 10th (38%) percentiles of the observed racial diversity exposure distribution during actively employed person-time. These percentiles were chosen to provide a range of interventions. The hypothetical MWF limits examined were a) 1.00 mg/m^3 (twice the NIOSH recommended exposure limit), b) 0.5 mg/m^3 (the NIOSH recommended exposure limit), and c) 0.10 mg/m^3 (one fifth the NIOSH recommendation). We estimated counterfactual risk per 1000 by age 80 years under interventions on racial diversity and MWF, separately and jointly. The joint interventions were a) 38% Black workers and a MWF limit of 1.00 mg/m^3, b) 54% Black workers and 0.5 mg/m^3 MWF, and c) 64% Black workers and 0.05 mg/m^3 MWF (the counterfactual).

The analysis was performed in SAS software V9.4 (SAS Institute, Cary, North Carolina, USA) based on GFORMULA macro available online (https://causalab.sph.harvard.edu/software/). Use of human subjects in this study was reviewed and approved by the Office for the Protection of Human Subjects at the University of California, Berkeley.

3.4. Results

Table 3.1 presents the characteristics of 5,043 Black workers, who accrued over 200,000 person-years. Over the follow-up there were 3,496 deaths, of which, 42% were attributed to CVD.

Table 3.1. Demographic Characteristics of Detroit plant Black workers in the United Autoworkers – General Motors (UAW-GM) Cohort, 1941-2015.

Characteristics	N	%	Median	Q1, Q3
Study Population (person-years)	5,043 (200,545)	100.0		
Sex				
Male	4,579	90.8		
Female	464	9.2		
All-cause mortality	2,861	56.7		
CVD mortality	1,166	23.1		
Cumulative metalworking fluid exposure (mg/m^3-year) [a]			11.9	5.4, 26.8
Average racial diversity exposure (% Black workers)			38.7	32.2, 45.0
Years of follow-up			38	29, 44
Year of birth			1929	1919, 1942
Year of hire			1962	1951, 1972
Age at hire			30	25, 37
Years at work			21.8	13.2, 28.5
Year of CVD death [b]			1991	1980, 2001
Age at CVD death [b]			66	58, 73

Abbreviations: CVD, cardiovascular disease. [a] Summary statistic calculated at the end of follow-up. [b] Among those who died of a cardiovascular disease.

Figure 3.2 presents the number of CVD deaths by age, and Figure 3.3 presents patterns of racial diversity and average MWF exposure by year. Among exposed workers, the mean annual daily average MWF exposure concentration was highest in 1941 and decreased markedly, especially after the Occupational Safety and Health Act of 1970, until work records ended at the end of 1994. On average, workers under follow-up were exposed to decreasing amounts of metalworking fluid and an increasingly diverse workforce.

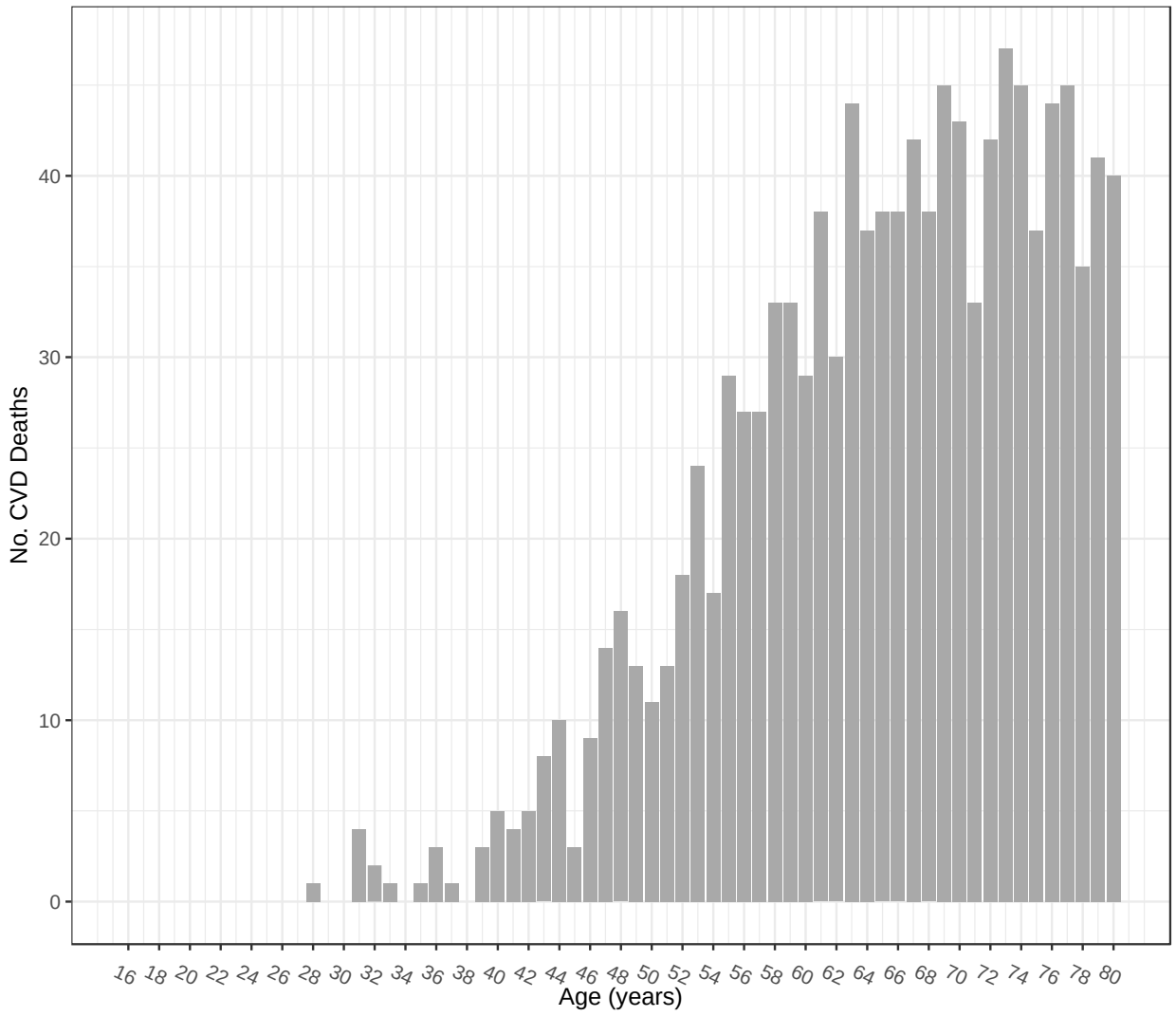

Figure 3.2. Number of cardiovascular disease (CVD) deaths by age among 5,043 Black autoworkers during the 200,545 person-years of follow-up between January 1, 1941 and December 31, 1994.

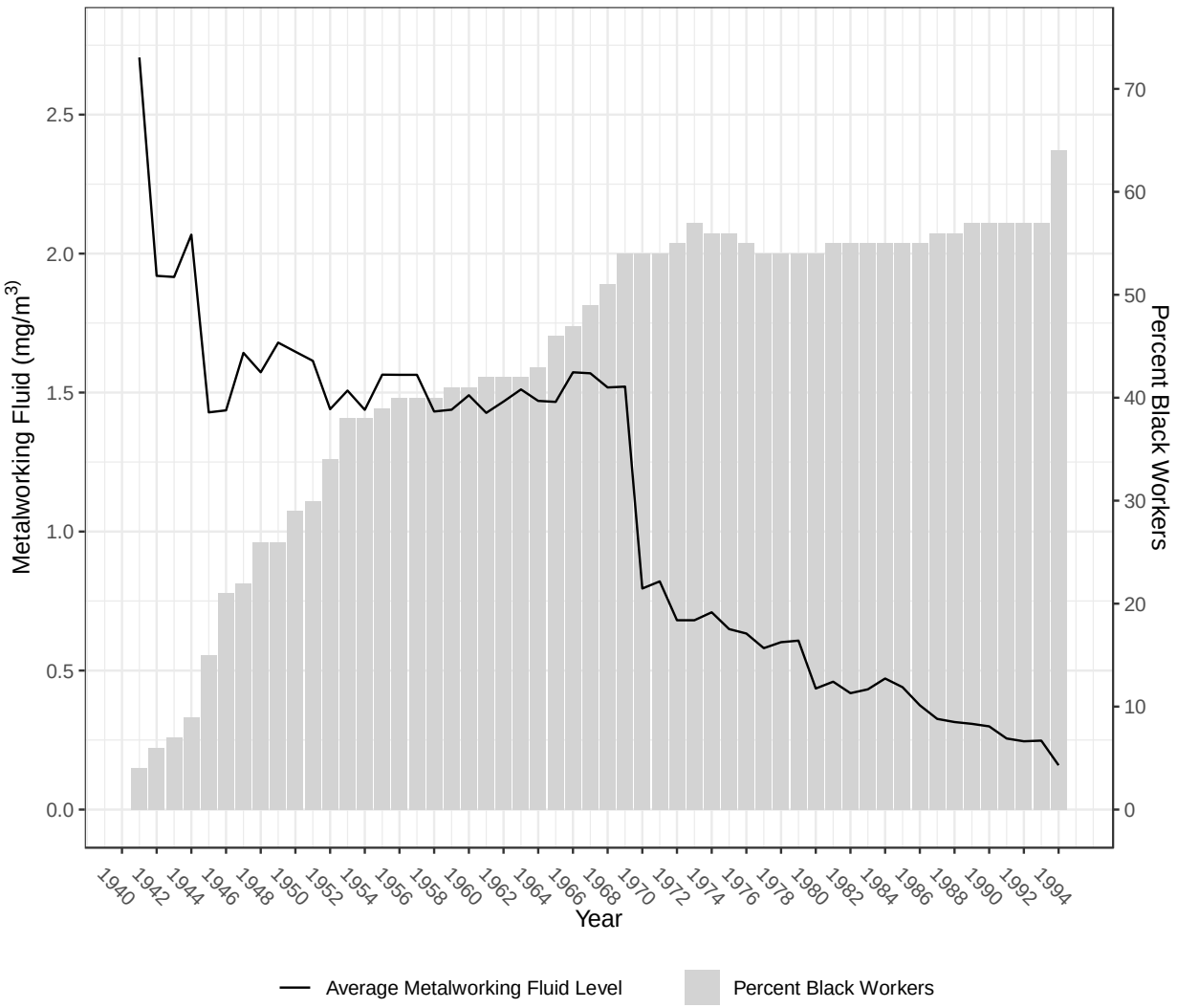

Figure 3.3. Percent Black workers actively employed (grey vertical bars) along with mean annual daily average metalworking fluid exposure concentration (solid line) for each calendar year in the United Autoworkers – General Motors cohort.

Density plots for the exposure distributions for racial diversity and metalworking fluid among actively employed person-time are depicted in Figure 3.4. The racial diversity and MWF distributions were minimally correlated (Spearman's rank correlation coefficient = 0.16) and skewed in opposing directions.

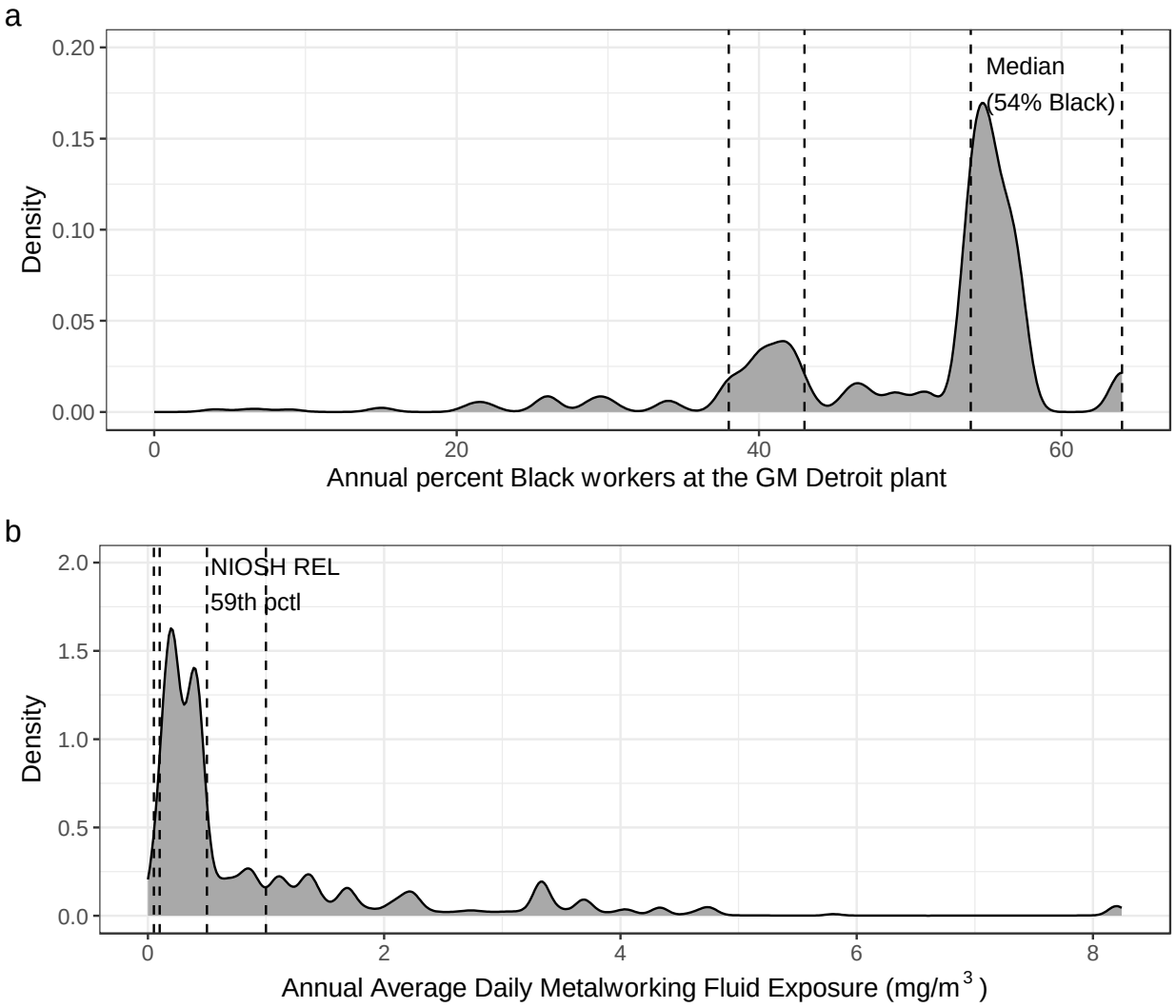

Figure 3.4. Density plots for the exposure distributions of racial diversity (annual % Black workers) and average annual daily metalworking fluid exposure (MWF, in mg/m^3) during active employment in a cohort of Detroit Black autoworkers in the United Autoworkers-General Motors (UAW-GM) cohort, 1941-1994. Hypothetical fixed intervention levels for racial diversity exposures (a) and hypothetical intervention maximum limits for MWF (b) applied in the parametric g-formula analyses are represented by the vertical dashed lines.

Parametric g-formula

Figure 3.5 displays comparisons of observed CVD mortality cumulative risk over age, and employment status over follow-up time, with the corresponding simulated values for the natural course designed to reproduce the observed course of events. The natural course simulation was consistent with the observed data. The cumulative incident CVD mortality under the natural course simulation (2.77%) was nearly identical to the observed cumulative risk (2.75%) by the end of follow-up at age 80.

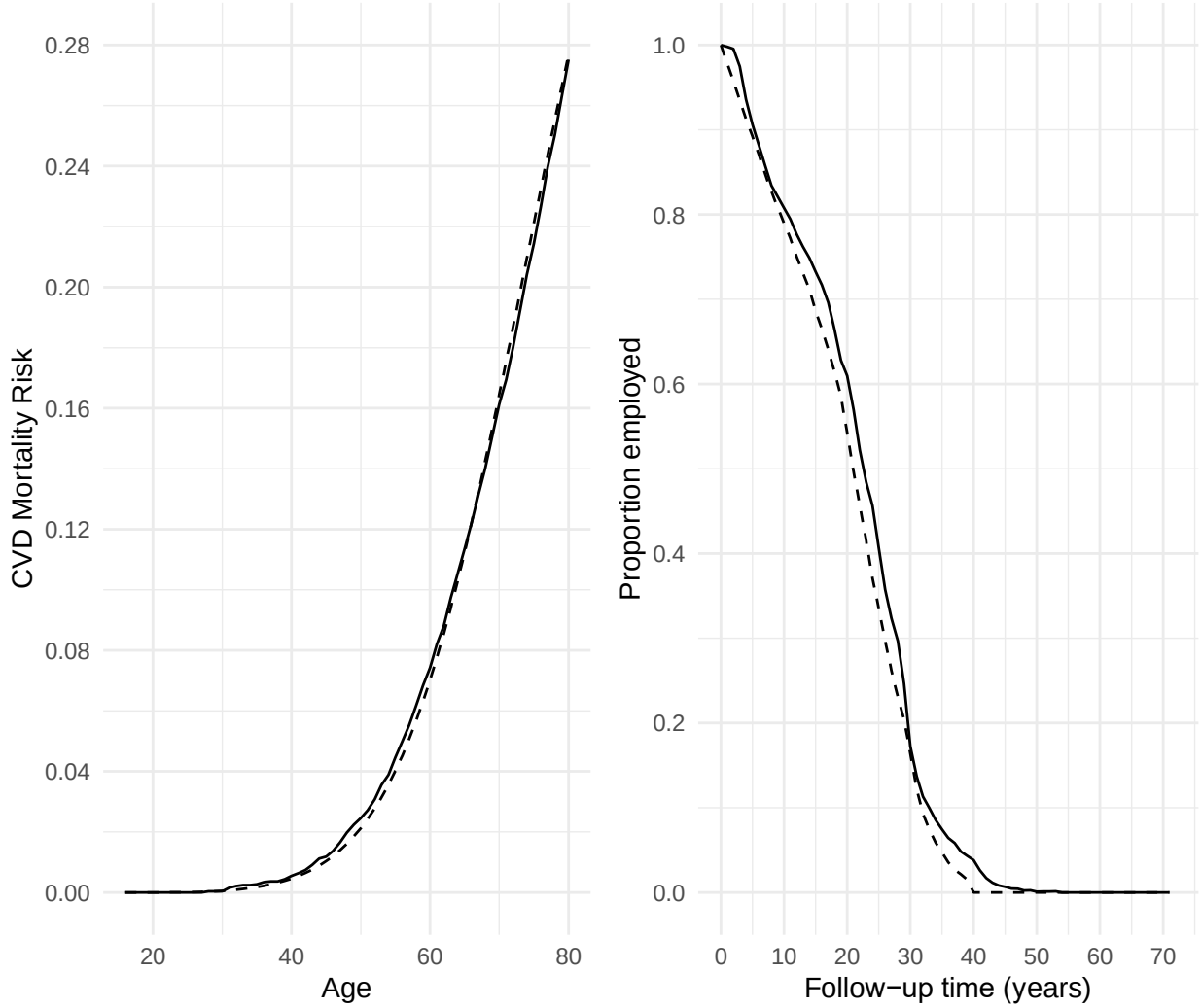

Figure 3.5. Observed (solid lines) cumulative risk of cardiovascular disease mortality over age, and employment status over follow-up time, among United Autoworkers-General Motors (UAW-GM) Black workers employed at the Detroit plant, 1941-2015, compared with simulated values (dashed lines) for the natural course.

The counterfactual risks at age 80 are summarized in Table 3.2 under fixed interventions on racial diversity, limit interventions on MWF, and joint interventions on both racial diversity and MWF.

Our results showed that as exposure to the percent Black autoworkers increased, the risk of CVD mortality progressively decreased. We observed monotonically decreasing risk ratios and excess risk comparing interventions with racial diversity fixed between 38% and 54% to racial diversity fixed at a maximum of 64%. For example, when the percent of Black autoworkers was 38% and 43%, there were 29 and 23 more CVD deaths per 1000 Black workers compared to when diversity reached 64% Black workers plantwide. While the confidence intervals of the racial diversity interventions crossed the null, most of the range of the confidence intervals was consistent with a harmful effect of reduced racial diversity on CVD mortality among Black autoworkers.

Next, we compared an intervention which set the average annual MWF limit to 10% of the NIOSH recommended exposure limit (0.05 mg/m^3) to interventions setting the MWF limit from 0.10 to 1.00 mg/m^3. Comparing the higher limits to the lowest limit, we observed only slightly higher relative risks and slightly more additional CVD deaths (between 6 and 7 additional deaths per 1000 workers for scenarios using the highest MWF exposure limits), although these results were imprecise with confidence intervals spanning the null.

Finally, we estimated decreases in CVD risk ratios and excess risks for joint interventions which progressively fixed racial diversity to higher levels and set lower limits on MWF in comparison to a counterfactual joint intervention in which diversity was set to its maximum level and MWF was set to its lowest limit. We observed an excess risk of 13.4 (95% CI: 1.1-27.0) comparing the joint intervention of racial diversity fixed at 54% Black workers and MWF at the NIOSH recommended exposure limit of 0.5 mg/m^3 to the joint intervention of 64% Black workers and 0.05 mg/m^3. Consistent with our hypothesis, this result was greater than the sum of the excess risks for the single interventions of racial diversity (54% vs. 64%) and MWF limits (0.50 mg/m^3 vs. 0.05 mg/m^3). Therefore, demonstrating a super-additive effect of the joint exposures on CVD mortality. Confidence intervals for the joint exposure at the 10^{th} percentile of the racial diversity exposure distribution and the 200^{th} percentile of the NIOSH recommended exposure limit were wide due to both exposure interventions being at the tail ends of their respective observed exposure distributions.

Table 3.2. Excess Risk of cardiovascular disease mortality among Black autoworkers in the UAW-GM Detroit plant at the select age of 80, with extended exposure up to age 55 years for those still at work at the end of 1994. (N=5,043)

Racial diversity fixed intervention [a]	Percent GM Active Black workers (per year)	CVD risk (per 1000)	RR (95% CI)	RD per 1000 (95% CI)
10th %-tile	38%	285.7 (253.0, 325.7)	1.12 (0.97, 1.32)	29.7 (-8.6, 74.8)
25th %-tile	43%	279.5 (246.3, 319.3)	1.09 (0.95, 1.29)	23.6 (-13.4, 69.2)
Median	54%	266.1 (239.3, 295.1)	1.04 (1.00, 1.08)	10.1 (-1.1, 20.4)
Maximum diversity	64%	255.9 (228.6, 285.1)	1.00	0.00
Metalworking fluid (MWF) limit intervention [b]	MWF (mg/m^3)			
200% of NIOSH REL	1.00	276.3 (251.3, 302.5)	1.03 (0.94, 1.10)	7.5 (-16.6, 24.8)
NIOSH REL	0.50	275.4 (251.0, 301.7)	1.02 (0.94, 1.10)	6.6 (-15.7, 24.8)
20% of NIOSH REL	0.10	270.4 (240.4, 299.7)	1.01 (0.99, 1.01)	1.6 (-1.5, 1.7)
10% of NIOSH REL	0.05	268.8 (240.7, 298.8)	1.00	0.00
Joint exposure (no. intervention)	Joint MWF & Racial Diversity			
10th %-tile racial diversity & 200% of NIOSH REL	38% & 1.00 mg/m^3	283.6 (250.7, 315.9)	1.13 (0.95, 1.33)	31.8 (-13.1, 74.3)
Median racial diversity & NIOSH REL	54% & 0.50 mg/m^3	265.1 (235.9, 296.7)	1.05 (1.00, 1.12)	13.4 (1.1, 27.0)
Maximum racial diversity & 10% of NIOSH REL	64% & 0.05 mg/m^3	251.8 (217.6, 287.1)	1.00	0.00

Abbreviations: CI, confidence interval; CVD, cardiovascular disease; NIOSH, National Institute for Occupational Safety and Health; REL, recommended exposure limit. [a] A fixed intervention to which all exposures are set while active. [b] A limit by which exposures can be no higher than the intervention level.

Across age, our results showed that with the joint interventions, CVD risk is reduced at ages over 60 years, with the greatest differences in risk at age 80. A graph of CVD mortality risk per 1000 across age under the three joint hypothetical scenarios is depicted in Figure 3.6.

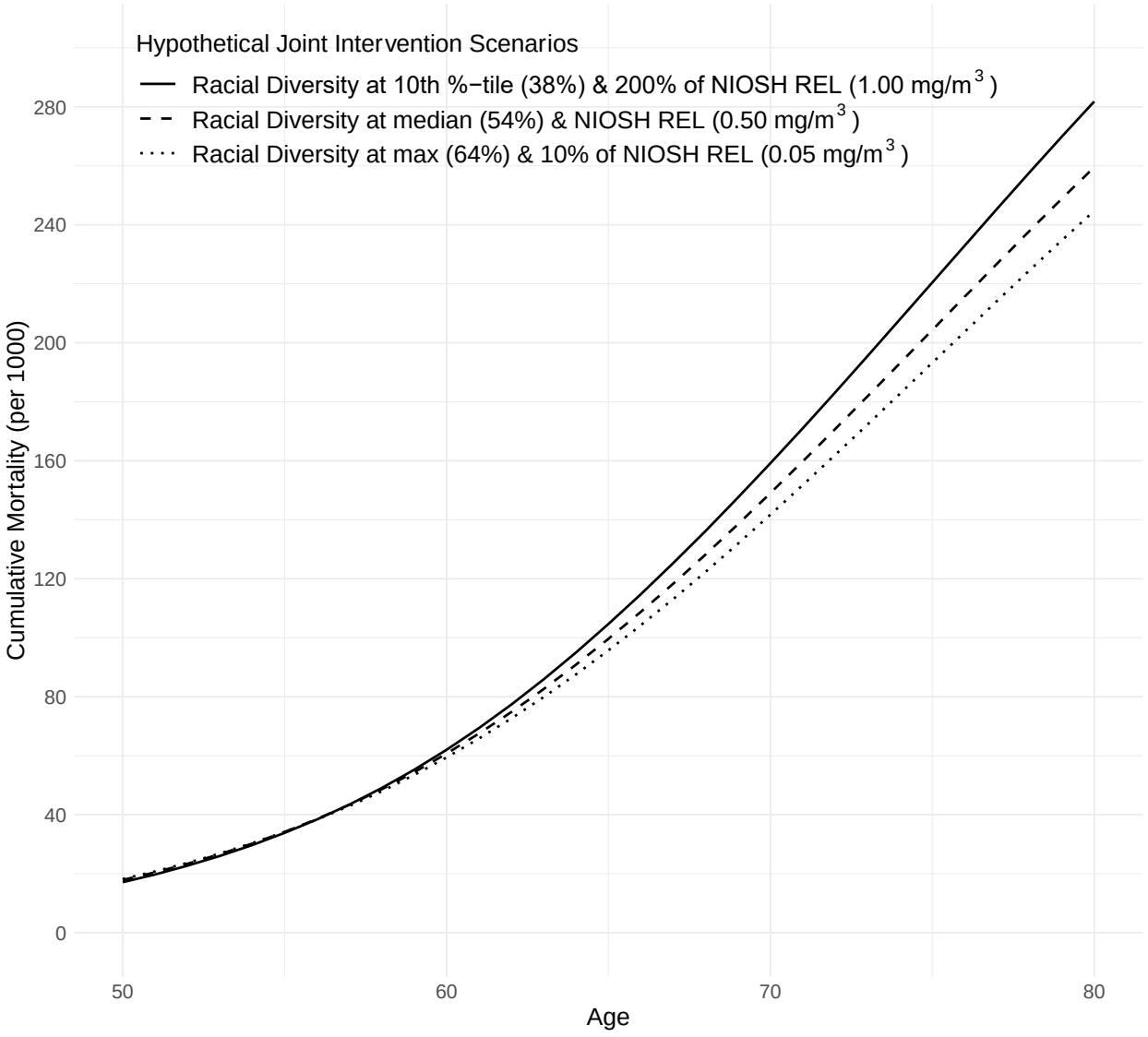

Figure 3.6. Risk of cardiovascular disease mortality among Black workers from the Detroit plant of the United Autoworkers-General Motors (UAW-GM) cohort, 1941-2015, under three scenarios of hypothetical exposure to annual plant-wide racial diversity and limits to annual MWF exposure. The x-axis of this graph is abbreviated to ages 50 years or older, when the majority of CVD deaths occurred. Abbreviations: NIOSH, National Institute for Occupational Safety and Health; REL, recommended exposure limit.

Results from the sensitivity analyses, one in which we assumed that workers still at work in 1994 received no exposure after work records ended in 1994 and one in which racial diversity exposure dropped to zero after the open cohort ended in 1985, were similar in patterns of CVD risk to the main analysis. They are summarized in Tables 3.3 and 3.4

Table 3.3. Excess Risk of Cardiovascular Disease Mortality among Black workers in the UAW-GM Detroit plant at the select age of 80. (N=5,043) [a]

Racial diversity static intervention [b]	Percent GM Active Black workers (per year)	CVD risk per 1000 (95% CI)	RR (95% CI)	RD per 1000 (95% CI)
10th %-tile	38%	279.0 (246.2, 316.7)	1.12 (0.97, 1.32)	29.1 (-8.1, 73.9)
25th%-tile	43%	277.3 (258.3, 313.8)	1.11 (0.97, 1.30)	27.4 (-7.6, 71.2)
Median	54%	260.3 (235.2, 288.8)	1.04 (1.00, 1.09)	10.4 (0.0, 20.6)
Maximum diversity	64%	249.9 (223.7, 279.4)	1.00	0.00
Metalworking fluid (MWF) limit intervention [c]	MWF (mg/m^3)			
200% of NIOSH REL	1.00	269.7 (243.5, 294.3)	1.02 (0.94, 1.10)	5.3 (-17.0, 23.9)
NIOSH REL	0.50	268.9 (244.4, 294.0)	1.02 (0.94, 1.10)	4.4 (-17.1, 24.6)
20% of NIOSH REL	0.10	265.0 (237.6, 292.7)	1.00 (0.99, 1.01)	0.6 (-1.6, 1.4)
10% of NIOSH REL	0.05	264.4 (237.2, 292.5)	1.00	0.00
Joint exposure	Joint MWF & Racial Diversity			
10th %-tile racial diversity & 200% of NIOSH REL	38% & 1.00 mg/m^3	276.7 (245.2, 308.2)	1.13 (0.95, 1.33)	32.0 (-12.7, 73.7)
Median racial diversity & NIOSH REL	54% & 0.50 mg/m^3	259.5 (233.8, 289.6)	1.06 (1.01, 1.12)	14.8 (1.4, 27.0)
Maximum racial diversity & 10% of NIOSH REL	64% & 0.05 mg/m^3	244.7 (210.9, 281.5)	1.00	0.00

Abbreviations: CI, confidence interval; CVD, cardiovascular disease; NIOSH, National Institute for Occupational Safety and Health; REL, recommended exposure limit. [a] When work records end in 1994, racial diversity and metalworking fluid exposure are set to 0. [b] A static intervention to which all exposures are set while active. [c] A limit by which exposures can be no higher than the intervention level. Cumulative CVD mortality by age 80 years for the: natural course: 0.27, observed: 0.28.

Table 3.4. Excess Risk of Cardiovascular Disease Mortality among Black workers in the UAW-GM Detroit plant at the select age of 80. (N=5,043) [a]

Racial diversity static intervention [b]	Percent GM Active Black workers (per year)	CVD risk per 1000 (95% CI)	RR (95% CI)	RD per 1000 (95% CI)
10th %-tile	38%	272.4 (241.2, 308.8)	1.04 (0.93, 1.28)	10.1 (-20.5, 61.5)
25th%-tile	41%	274.4 (219.9, 306.3)	1.05 (0.98, 1.12)	12.1 (-4.8, 29.6)
Median	54%	265.5 (217.5, 290.9)	1.01 (1.00, 1.03)	3.2 (-0.3, 6.6)
Maximum diversity	57%	262.3 (215.1, 287.6)	1.00	0.00
Metalworking fluid (MWF) limit intervention [c]	MWF (mg/m^3)			
200% of NIOSH REL	1.00	278.3 (235.6, 305.5)	1.02 (0.94, 1.09)	4.9 (16.8, 22.3)
NIOSH REL	0.5	277.5 (236.4, 305.7)	1.01 (0.94, 1.09)	4.1 (-16.8, 22.2)
20% of NIOSH REL	0.10	275.4 (224.4, 306.6)	1.01 (0.99, 1.01)	1.9 (-0.3.4, 2.6)
10% of NIOSH REL	0.05	273.5 (224.7, 306.8)	1.00	0.00
Joint exposure	Joint MWF & Racial Diversity			
10th %-tile racial diversity & two-fold NIOSH REL	38% & 1.00 mg/m^3	283.5 (252.4, 315.3)	1.11 (0.94, 1.40)	27.8 (-17.7, 81.5)
Median racial diversity & NIOSH REL	54% & 0.50 mg/m^3	265.4 (214.3, 292.2)	1.04 (0.97, 1.11)	9.7 (-7.6, 24.0)
Maximum racial diversity & 10th of NIOSH REL	57% & 0.05 mg/m^3	255.8 (198.5, 289.8)	1.00	0.00

Abbreviations: CI, confidence interval; CVD, cardiovascular disease; NIOSH, National Institute for Occupational Safety and Health; REL, recommended exposure limit. [a] Racial diversity work records were dropped to zero in 1986. [b] A static intervention to which all exposures are set while active. [c] A limit by which exposures can be no higher than the intervention level. Cumulative CVD mortality by age 80 years for the: natural course: 0.27, observed: 0.28.

Figure 3.7 presents CVD risk in relation to the single racial diversity intervention by age.

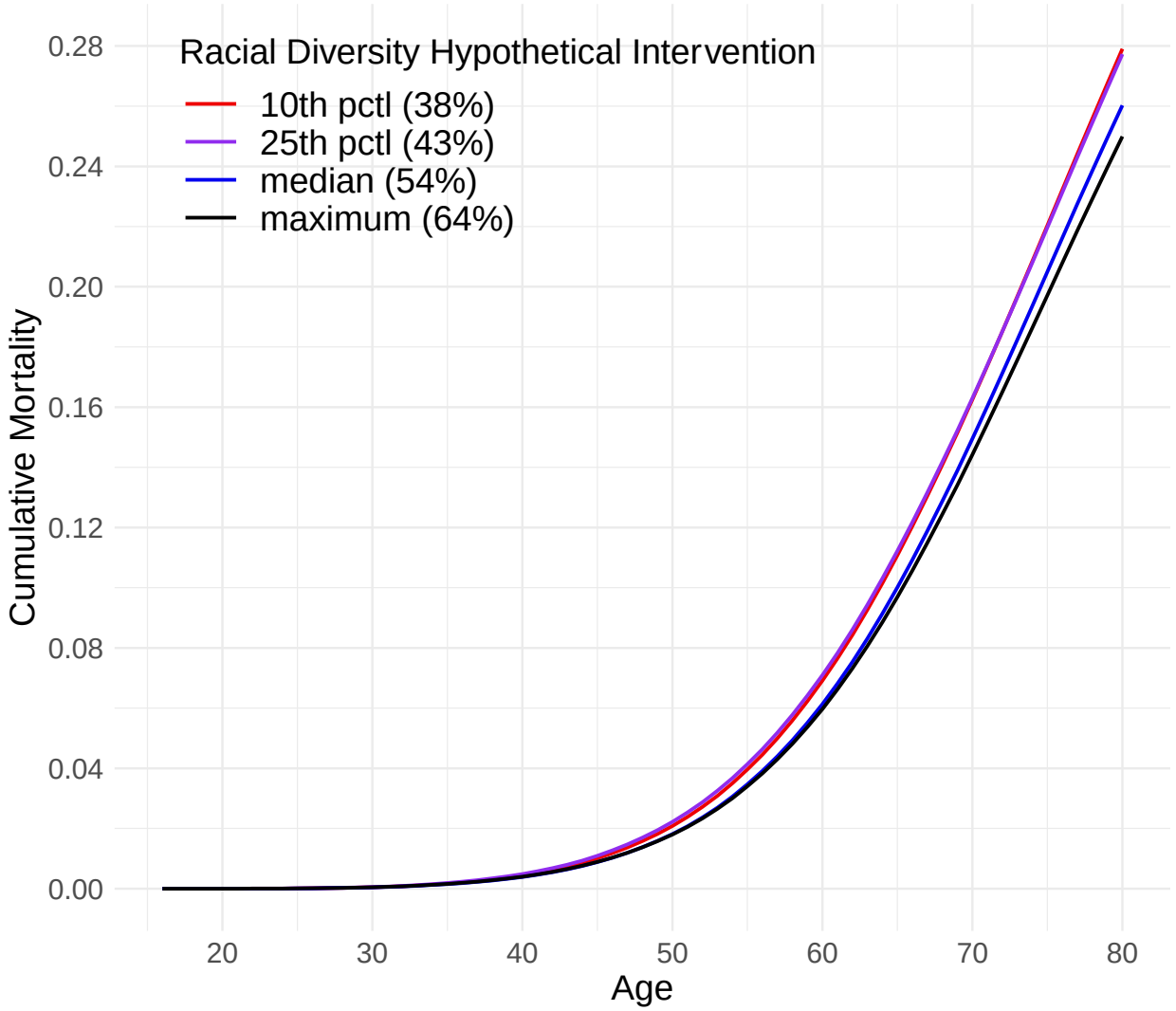

Figure 3.7. Cumulative incidence of cardiovascular disease mortality among Black autoworkers from the Detroit plant of the United Autoworkers-General Motors (UAW-GM) cohort, 1941-2015, under four scenarios of hypothetical exposure to plantwide racial diversity (% Black workers).

3.5. Discussion

This is the first study to jointly examine CVD mortality risk with respect to plantwide racial diversity and workplace chemical exposures in a cohort of Black workers. We used the parametric g-formula to allow for the use of a potential-outcomes framework for social variables.[41] We estimated CVD mortality risk on the multiplicative and additive scales under hypothetical scenarios based on observed plantwide racial diversity and on existing NIOSH guidelines for metalworking fluid exposures. Among the single interventions, we found that while lowering the MWF limit from 1.00 to 0.05 mg/m^3 was associated with 7 fewer CVD

deaths per 1000 at age 80, increasing racial diversity by 26% led to a much larger decrease – 29 fewer deaths per 1000 autoworkers. Although these reductions in exposure cannot be directly compared, our results suggest that increasing racial diversity is a potentially effective intervention for preventing CVD deaths among Black autoworkers. In addition, some joint interventions to progressively increase racial diversity exposure and lower MWF limits resulted in greater risk reductions than the sum of the single interventions on both the additive and multiplicative scales. These estimated joint effects support a growing literature considering the potential synergies between social and environmental exposures.

Although there have been earlier studies of heart disease in the UAW-GM cohort, ours is the first to examine the causal impacts of MWF exposure on CVD mortality in this longitudinal cohort of Black workers.[7-9,11,39] We observed that reductions to MWF exposure alone moderately reduced CVD mortality among GM Black workers. Our modest results may be due to the changing composition of MWFs, such as the introduction of severe hydrotreating to reduce polycyclic aromatic hydrocarbons (PAHs) in the 1980s, a potential causative agent of CVD.[42] PAHs exist in bulk mineral oil-based metalworking fluid,[42] which was the most common type of fluid until around the mid-1940s when production steadily decreased to <20% of all MWF types in the 1980s.[43] Meanwhile, the median year of hire among our cohort of Black workers was 1962, a period of transition to highly refined base oils with lower PAH content and that generated lower concentrations of particulate matter when aerosolized. In addition, the water-based MWFs increasingly common after the 1970s have not yet been linked to cardiovascular disease among autoworkers.[11] In this study, we acknowledge that our results between exposure to MWF and CVD mortality will be dominated by exposure to water-based fluids. We combined the three types of MWFs because we were interested in any mechanism of exposure, whether through the ambient air which could contain a confluence of MWF types or by working directly with the machines.

Our findings demonstrate the importance of workplace racial composition on cardiovascular disease mortality in a cohort of racialized workers. Previous studies have evaluated social hazards in an occupational setting,[44-46] such as reported experiences of racial discrimination and unfair treatment among Black working professionals in association with increases in systolic blood pressure.[47] Furthermore, one study of Black US workers examining workplace racial composition found that workers with >75% of Black co-workers experienced significantly less psychological distress than workers with lower percentages of Black co-workers.[48] Our research expands the literature on both workplace racial composition (diversity) and the joint effects of social and occupational stressors by presenting results from a causal perspective, in relation to cardiovascular disease mortality, and on both the additive and multiplicative scales. We argue that in addition to the allocation of wages, healthcare, and other job-related rewards, workplace practices and policies play a fundamental role in the cardiovascular health outcomes of workers by structuring the racial diversity of jobs. Racial diversity may then shape social relationships, workplace culture (discrimination, microaggressions),[49] and the power and relational dynamics afforded to various racial groups, thereby impacting physiological and psychological stress responses of racialized workers.[24,25,48] Although our findings were somewhat imprecise, which may be explained by our limited sample size, we encourage future researchers with larger cohorts of racialized workers to replicate this analysis.

The parametric g-formula is one of several "g-methods" which, under certain assumptions, allows adequate control for time-varying confounding affected by prior exposure. However, it is predicated on correctly specified models. In our study, we fit parametric models for employment status, MWF and racial diversity exposures, death due to a competing risk, and death due to CVD. Under assumptions of conditional exchangeability, consistency, no information bias, and correct model specification, g-methods estimate causal effects of time-varying exposures.[34] The assumption of conditional exchangeability (or no unmeasured confounding) requires that counterfactual outcomes are statistically independent of observed exposures, given measured covariates. A limitation pertaining to this assumption was the lack of information on smoking and potential employment at other factory jobs, which prevented us from accounting for smoking and other high-risk occupations as confounders of the MWF and CVD mortality relationship. However, several prior studies of lung cancer incidence and mortality in this UAW-GM cohort reported null associations with MWF,[50-52] indicating that smoking is probably uncommon in the GM cohort and unlikely to confound our results because if smoking was prevelant we would likely have observed an association due to unmeasured confounding from smoking-related lung cancer. We also rely on a job exposure matrix and employment histories to assign our exposures of interest, allowing potential exposure misclassification. However, information bias of this kind is likely nondifferential with regards to CVD mortality.

Although the features of the parametric g-formula render it particularly useful for analyses of risk on occupational epidemiology,[38,53] there have been fewer applications of the g-formula method in social epidemiology.[54,55] It has been debated that social processes, often with multiple interacting pathways and long causal chains, are not easily amenable to evaluations of causal effects.[56] Yet, over the past 30 years, social epidemiologists have capitalized on natural experiments and developed a range of causal inference approaches to complex longitudinal studies.[56,57] The continual use of causal methods, including g-methods, is necessary to guide policy-related action to improve cardiovascular health among racialized workers. Often when using causal frameworks to address social exposures, the consistency assumption is threatened.[58] Here, we attempt to avoid this and focus on a definable intervention under specific circumstances by explicitly conceptualizing structural racism's impact on labor and employment as a process that influences hiring and retention practices which affect the workforce's racial composition over time.

Regarding the counterfactual risk estimates in the current study, we assumed that risk prediction based on the observed exposure ranges is representative of risks that could occur through some intervention. We assessed risk under exposure scenarios that compared a range of racial diversity exposures to the maximum observed, thus allowing us to estimate bounds for risk reduction that could be achieved while in compliance with our observed data of the percent Black workers at the Detroit GM plant. However, we acknowledge that the type of intervention by which racial diversity exposure increased would likely affect exposure distributions differently. For example, an intervention where racial diversity was increased solely through retention of currently employed workers would only affect those who are already employed. Regarding MWFs, we considered an intervention in which all subjects were always unexposed (0 mg/m^3) to be theoretically infeasible. Since workers were exposed either directly, or indirectly to aerosolized MWF, a non-zero level of MWF exposure likely exists in all large metal machining operations. Analyses for interventions setting exposures to zero may also suffer from non-positivity as no

actively employed person-time was unexposed to plantwide racial diversity or MWF and we would have had to rely on extrapolation from the models.

3.6. Conclusions

In addition to lowering occupational environmental exposures, increasing the diversity of the workplace may prevent CVD mortality deaths among Black workers. Our results suggest that workplace equity policies such as Affirmative Action, which are meant to support equal access to workplaces, can lead to health benefits among Black working populations. Recently, CVD was found to be the largest contributor to the Black-white life expectancy gap among men,[59] underscoring the importance of research addressing disparities in such preventable diseases. We maintain that policy initiatives focused on diversifying workforces in emerging sectors of manufacturing and research, such as the 2022 CHIPS and Science Act, have the potential to not only advance workplace equity, but to improve health outcomes of racialized workers. However, the effectiveness of these initiatives hinges, in part, on whether historical inequalities in inclusion, hiring, and retention are specifically addressed in the workplace, and whether investments are continually made toward the long-term health of racialized workers.

3.7. References

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3.8. Appendix

The generalize computation algorithm formula (g-formula) describes the relationships between exposure, confounders, and potential outcomes (e.g., the expected cardiovascular disease (CVD) mortality at age 80 under no exposure). The g-formula links to the observed data via the causal identification assumptions. An assumption of the parametric g-formula is correct model specification. The relationships among the observed data can be modeled using parametric modeling, in which case the method is referred to as the parametric g-formula. However, baseline variables did not require parametric models as these values were drawn from the empirical distribution. While we do intervene upon the exposure variables in the simulated datasets, we do not intervene on all the exposures all of the time. For some interventions, exposure variables are treated as time-varying covariates. Thus, we assumed correct specifications of the parametric models for employment status, death due to a competing risk, death due to CVD, and for racial diversity and metalworking (MWF) exposures, including both annual and cumulative models. As an informal check of this assumption, we compared the cumulative CVD mortality in the observed data to the cumulative CVD mortality in the simulated natural course at age 80 years. Age 80 was selected as a salient age for which the cohort has adequate data, and at which cumulative risk for CVD mortality would not be 100% but most exposure effects from occupational metalworking fluid and workplace racial diversity were hypothesized to have occurred. Specification of the models described below was done with the goal of minimizing the difference between the observed and natural course cumulative CVD mortality. The observed cumulative CVD mortality was 2.70%. The final models, as described below, achieved the same with the natural course cumulative CVD mortality of 2.75%.

The parameters of the parametric g-formula can be estimated using the combination of a set of pooled logistic models for the joint distribution of the data, followed by a Monte Carlo algorithm. The modeling step can be performed using separate models for exposure, all time-varying covariates, and all outcomes of interest. The Monte Carlo step uses parameter estimates from the modeling step to simulate the target population under one or more intervention distributions for exposure. The simulations output discrete hazard estimates for each outcome yearly for each subject under each intervention which are combined to estimate the risk for the outcomes of interest under each intervention under a modified Kaplan-Meier algorithm. The risk difference comparing two interventions at a given age is the difference between the risk functions. Interval estimates for the risk functions and the risk difference estimates are obtained using a non-parametric bootstrap, whereby all steps of the parametric g-formula are repeated on random samples (with replacement) of individuals from the original data.

Baseline covariates were entered in all models as follows: indicators for variables for sex (male, female); linear term for year of hire; categorical variable for calendar year with cut points every five years starting with 1950 through 2000; categorical variable for age with a level per decade starting with the 30s. Calendar year and age were simulated as time-varying covariates that increased by one year for each person-year record after baseline. Age was the time-scale of interest, indexed a=16 to 80. All models were conditional on prior survival.

The parametric models were fit as follows:

1. A logistic model for the probability of employment termination at age *a*, restricted to records where prior employment termination status = 0 (not terminated). As an independent variable for succeeding covariate models, the prior value of employment termination was included as an indicator variable.

2. A linear model predicted the natural log of the MWF exposure level at age *a*. In records in which employment termination status = 1 (terminated), exposure was set to 0. As an independent variable for succeeding covariate models, the prior value of MWF exposure was included as a categorical variables with cut points at the quartiles of annual exposure among all observed person-time: 0.222, 0.4027, and 1.1955 mg/m^3.

3. A model accumulating annual average daily MWF exposure to predict cumulative MWF exposure at age *a*. Values of cumulative MWF exposure were not used as independent variables in covariate models. These were only used as independent variables in the models for CVD death and non-CVD death, specifically the prior value of cumulative MWF exposure was included as restricted cubic splines with knots at the quintiles of cumulative exposure among observed CVD deaths: 6.3193, 11.5894, 21.6802, 43.5315 mg/m^3-years.

4. A linear model predicted the natural log of the racial diversity exposure level at ag *a*. In records in which employment termination status = 1 (terminated), exposure was set to 0. As an independent variable for succeeding covariate models, the prior value of racial diversity exposure was included as a categorical variables with cut points at the quintiles of annual exposure among all observed person-time: 42, 46, 50, and 54 percent Black.

5. A model accumulating annual racial diversity exposure to predict cumulative racial diversity exposure at age *a*. Values of cumulative racial diversity exposure were not used as independent variables in covariate models. These were only used as independent variables in the models for CVD death and non-CVD death, specifically the prior value of cumulative racial diversity exposure was included as restricted cubic splines with knots at the octiles of cumulative exposure among observed CVD deaths: 383, 498, 829, 1009, 1207, 1406, 1480 percent Black-years.

6. A logistic model for the probability of death due to causes other than CVD at age *a*.

7. A logistic model for the probability of CVD death at age *a*.

Chapter 4. Workplace racial segregation and its joint effects with job mobility on cardiovascular disease mortality among US autoworkers

4.1. Abstract

Residential racial segregation has been associated with racial health inequalities in cardiovascular disease (CVD) mortality; however, there is little research on the health impacts of racial segregation in non-residential spaces, such as the workplace. This study aimed to assess associations between Black-white occupational racial segregation and its joint effects with job mobility and CVD mortality risk among a cohort of Detroit automobile plant workers from the United Autoworkers – General Motors (UAW-GM) cohort. This retrospective cohort study included 14,637 workers employed for at least three years, with follow-up from 1941 to 2015. Segregation (index of dissimilarity) was a year level variable. Annual exposure to segregation was cumulated over working life and divided by time since hire. We then calculated each worker's time-varying cumulative area under the curve of their job trajectory, which provided a measure of job mobility. In this study, higher job mobility indicated greater exposure to the occupational hazard, metalworking fluid. We used Cox models, by race, to examine the impact of segregation, job mobility, and the interaction between segregation and job mobility on CVD mortality. We observed a strong positive association between greater workforce racial segregation and CVD mortality among Black workers and smaller positive effects among white workers. Comparing moderate or high segregation exposure to low exposure, CVD mortality risk was high among Black workers with lower job mobility. Black workers with higher job mobility experienced smaller monotonic increases in CVD risk with increasing segregation. Among white workers with higher job mobility, there was a positive impact of segregation on CVD risk, which was not observed among those with lower job mobility. Our findings provide evidence that workplace racial segregation may increase CVD mortality risk among all workers, with especially strong impacts on racialized workers. We observed that the physiological harm of racial segregation to racialized workers operates in the workplace regardless of job advancements that provide better wages, mentorship, and social capital.

4.2. Introduction

Structural racism constitutes the ways in which societies perpetuate discrimination through interconnected inequitable systems. These systems, in turn, reinforce discriminatory beliefs and the unequal distribution of resources, which collectively affect the risk of adverse health outcomes, such as cardiovascular disease (CVD).[1,2] One example of structural racism pertains to the ongoing residential segregation of Black Americans, which is associated with several cardiovascular outcomes including increases in systolic blood pressure,[3] incident CVD,[4] and mortality.[5]

In recent decades, research on the spatial distribution of racialized groups have focused extensively on measures such as dissimilarity and isolation indices,[6] to explore residential

unevenness and segregation exposure. However, there is comparatively less research on other spatial contexts, such as the workplace - a crucial non-home activity space.[7] Workplace segregation, racial diversity, and the resources provided or denied certain groups are shaped by a multitude of historical and contemporary structural and societal factors,[8] including cultural practices and national and local economies. Therefore, without an explicit focus on workplace segregation as a pillar that maintains structural racism, efforts to increase diversity and eliminate racial health disparities in chronic disease, will have limited effects.

It is imperative to continue investigations on how the conditions of work may elicit stress responses in workers and cause physiological harm to the cardiovascular system. Thus far, occupational epidemiology has focused on psychosocial stressors such as job control, strain, and social support in association with stress-related adverse health outcomes.[9] However, less attention has been given to the influence of occupational structures and organization, such as the distribution of job rewards, access to technical expertise, job mobility (e.g., promotions), or racial segregation by job assignment.[9,10] Our study aims to address this gap in the literature by investigating the impact of workplace racial segregation and job mobility on long-term chronic disease, specifically CVD mortality.

Despite the outlawing of employment discrimination 60 years ago, racial segregation in the US workforce persists and can minimize Black workers' agency.[11] Ray (2019) argues that racialized organizations can reproduce inequalities by mapping onto societal racial stratification with organization procedures such as job sorting, used to slot racialized workers into lower job assignments compared to white workers, thereby actively maintaining societal racial hierarchies and inequalities.[8] For example, over the last two decades, occupations predominately comprising white workers averaged an annual income of approximately $120,000 (over two-fold the national average), compared to approximately $31,000 in occupations predominately comprising Black workers.[11,12] Furthermore, studies of Black professional workers have found various forms of racial discrimination that likely influence workplace racial segregation and career advancement, including selectively enforced rules,[13] limited advancement opportunities, diminished credibility,[14] differential treatment by managers, and fewer mentorship opportunities.[15] Workplace racial segregation may also reshape behavioral norms and affect peer networks,[16] given collective exposure to segregation-induced inequalities. These disparities in wages, resources, and opportunity are significant since heightened stress due to income insecurity, perceived social status, and workplace discrimination may directly promote the "biological embedding" of disease, including CVD, through pathogenic metabolic and inflammatory pathways.[17,18]

In the present study, we take advantage of retrospective quantitative work records to examine the implications of racial segregation on cardiovascular disease (CVD) mortality, by race, among Detroit autoworkers from the United Autoworkers – General Motors mortality cohort (UAW-GM). Our study had two primary scientific objectives. First, we examined the risk of CVD mortality as a function of exposure to racial segregation into differing job assignments. We hypothesized that the risk of CVD mortality would be increased with increasing levels of exposure to workplace racial segregation. Second, we examined the interaction between racial segregation and job mobility (i.e., promotions) and CVD mortality risk. Our consideration of

these joint effects may elucidate the potential harm of racial segregation among Black workers, regardless of career advancement.

4.3. Methods

Study population

The United Autoworkers-General Motors cohort was originally designed in 1985 and included 46,316 hourly workers from 3 General Motors plants in Michigan (Detroit, Ypsilanti, and Saginaw) who were exposed to metalworking fluid. This cohort has been described in detail previously.[19] The data includes birthdate, sex, race, and work history (until 1995) obtained from company records. All hourly employees who had worked at least 3 years before January 1, 1985, were included in the cohort. The population for this study was further restricted to Black and white autoworkers from the Detroit plant - details regarding ethnicity were not provided in the work records. The final analytic population comprised 14,637 workers. Mortality follow-up begins three years after the subject's date of hire, to minimize potential confounding introduced by short-term or transient workers,[20] and extends until death or the end of 2015.

Outcome

Our outcome of interest was cause-specific mortality attributed to overall cardiovascular disease mortality. Data on vital status were ascertained through the Social Security Administration, the National Death Index, plant records, and state mortality files. Cause of mortality was obtained from state vital records, death certificates, and the National Death Index. We defined cardiovascular disease using codes 390-459 and I00-78 from the International Classification of Diseases, Ninth Revision.

Exposure assessment

Our primary exposure measure was annual average exposure to racial segregation. Racial segregation in each year from 1941 to 1985 was measured by the dissimilarity index, a commonly used measure to describe evenness of racial distributions.[21] In this study, the dissimilarity index conceptually measured evenness across the following GM job assignments: assembly, machining, and grinding. The index measures the percentage of a group's population that would have to change jobs for each job assignment to have the same percentage of that group as is in the overall Detroit plant population. The dissimilarity index ranges from 0 to 1, with 0 as an indicator of complete integration and 1 as an indicator of complete segregation. Since we measured the dissimilarity index in each year, each worker was thereby exposed to varying levels of racial segregation over time, which we cumulated during their employment and divided by the time since hire. This time-varying calculation represented their average annual exposure to racial segregation. We categorized the racial segregation exposure into tertiles based on the exposure distribution of the CVD cases to avoid a linearity assumption.

Our secondary measure of exposure was of job mobility. At GM, assembly work involved the least amount of contact with the occupational hazard, metalworking fluid (generally only through ambient air), while machining and grinding work involved direct contact with the fluids. As

documented across the automobile industry in Michigan,[22] assembly jobs were the lowest-skilled jobs, machining jobs were moderately skilled, and grinding jobs required the highest level of skill. Wages were positively correlated with the level of skill requisite for each role.[22] This ordering of the job assignments was a key part of our calculations for job mobility. During a worker's active employment, for each person-year, we calculated the area under the curve of their job assignment level (assembly=1, machining=2, grinding=3) using the trapezoidal method.[23] We then cumulated the areas and the final measure was a time-varying year-level measure of cumulative area under the curve (hereafter, job mobility), which remained at its last value after leaving work. We binarized job mobility into higher and lower trajectories based on the exposure distribution of the CVD cases. Figure 4.1 shows four random examples from the analytic cohort of each workers' job trajectory (encompasses job mobility and stability) over their working life, the calculated area under the curve in each year of work, and their cumulative area under the curve for each year of their follow-up.

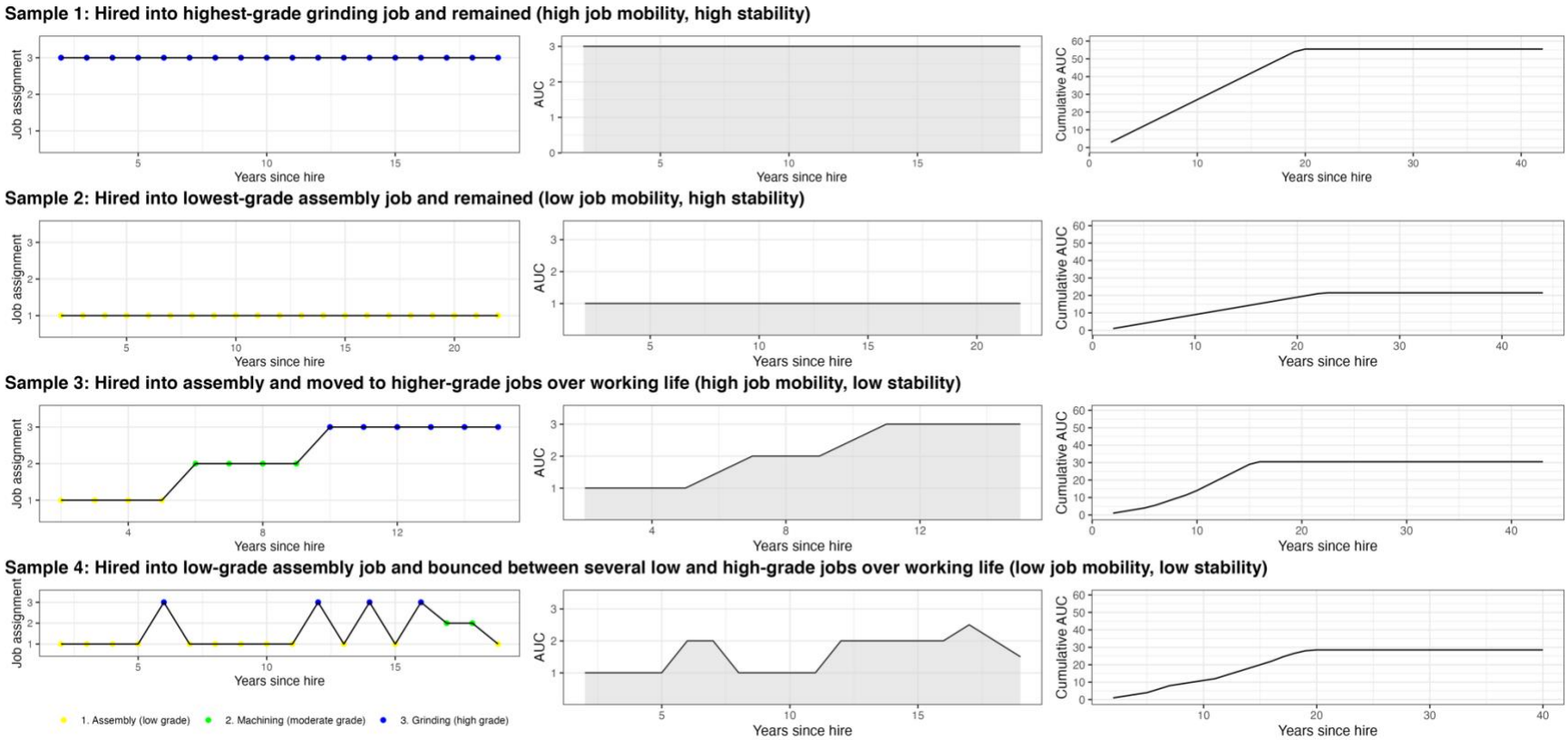

Figure 4.1. Four random scenarios of job trajectory (job mobility and stability) and its translation to area under the curve in each year of work since hire and cumulative area under the curve in each year of follow-up since hire.

Confounders

We used a directed acyclic graph (Figure 4.2) to identify confounders of the relationship between the exposure (racial segregation), interaction (job mobility), and outcome (CVD mortality). Year of hire was defined as a categorical variable with five-year bins, and calendar year was defined continuously.

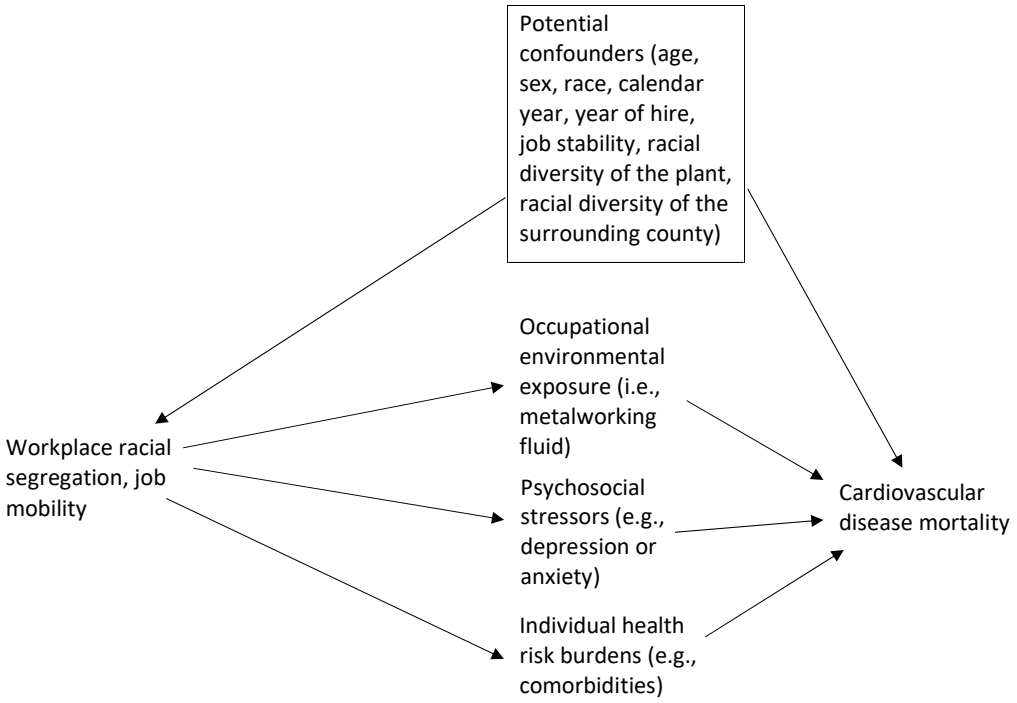

Figure 4.2. Directed acyclic graph (DAG) depicting the anticipated relationships between the exposures (workplace racial segregation and job mobility) in each year and the outcome (cardiovascular disease mortality).

We constructed a time-varying individual-level measure of job stability by measuring the absolute value of the difference in job assignments from one active year to the next for each worker. This measure was cumulated and divided by the time since hire in each year of follow-up. We also accounted for racial diversity in the workplace by measuring the annual average exposure to plantwide diversity (percent Black workers). We constructed a time-varying plant-level variable measuring racial diversity in each year, which was cumulated during the worker's employment and divided by the time since hire.

Finally, we measured and adjusted for the racial diversity of Wayne county (in which the Detroit plant was located) in each year, to account for each worker's exposure to racial diversity in their community. Annual county-level measures of the percent Black residents were extracted from the 1940-2000 US Censuses and the US Census Bureau's American Community Survey (ACS) from 2010-2015. Annual county-level values between those reported by the US Census or ACS were linearly interpolated between the previous value and the next available value.

Statistical methods

To examine associations between average exposure to racial segregation and CVD mortality, we fit Cox proportional hazards models to estimate adjusted hazard ratios (HR) and 95% confidence intervals (CI).[24] We estimated cause-specific HRs for the main analysis and, as a sensitivity analysis, sub-distribution HRs for CVD mortality because other causes of death (e.g., cancers) may remove workers from the risk set before the event of interest is observed.[25] Cause-specific HRs were calculated representing the rate of CVD-related deaths among workers with high levels of exposure (e.g., high racial segregation) relative to workers with low levels of exposure. The sub-distribution HRs were estimated using the Fine-Gray model.[25,26]

For our secondary analysis, first, cause-specific hazard ratios were measured for job mobility in relation to CVD mortality. Then, interactions between racial segregation and job mobility were assessed on the multiplicative scale. We evaluated multiplicative interaction between racial segregation and job mobility using likelihood ratio tests (LRT) which were conducted to compare the nested models with and without the multiplicative term.[27] Given low statistical power for interaction, an a priori alpha of 0.10 was used as the significance threshold. Workers who did not have available occupational exposure information and therefore no information on their job assignment were excluded from the analyses with job mobility (n=496 (3%)).

We stratified all the models by race given that the respective impacts of racial segregation and job mobility on CVD mortality may differ by membership in a socially racialized group. All Cox models used age as the time scale and adjusted for sex, calendar year, year of hire, cumulative percent Black residents, and average exposure to plantwide racial diversity. Models with job mobility additionally adjusted for annual average job instability. The proportional hazards assumptions were assessed using a Wald test for the interaction between follow-up time and each covariate.

All analyses were conducted in SAS version 9.4 (SAS Institute Inc, Cary, North Carolina, USA). Data visualization was conducted in R version 12.0 (R Foundation for Statistical Computing,

Vienna, Austria). This study was approved by the Committee for the Protection of Human Subjects at the University of California, Berkeley.

4.4. Results

Study sample characteristics

Table 4.1 lists demographic and exposure characteristics of the 14,637 autoworkers from the Detroit plant, of whom 37% died from CVD and 34% were Black. The median annual average racial segregation exposure is higher among white workers, likely because they were hired earlier when less Black workers were employed. While the median of job mobility is similar in both racial groups, median job instability is higher among Black workers indicated more movement into different jobs throughout their working life. Black workers also have higher median year of hire, years of follow-up and year of death, although their median age at death is slightly younger.

Table 4.1. Demographic Characteristics by Racial Category in the United Autoworkers – General Motors (UAW-GM) Cohort Detroit plant automobile workers, 1941-2015

	Total Study Population		**Black Workers**		**White Workers**	
	N	%	N	%	N	%
Study Population (person-years)	14,637 (570,236)	100	5,047 (207,625)	100	9,590 (362,611)	100
Sex						
Male	14,081	96	4,583	91	9,498	99
Female	556	4	464	9	92	1
All-cause mortality	11,461	78	3,536	70	7,925	83
Cardiovascular disease	5,405	37	1,471	29	3,934	41
	Median	**Q1, Q3**	**Median**	**Q1, Q3**	**Median**	**Q1, Q3**
Average annual segregation (index of dissimilarity: % evenness across jobs) [a]	17	14, 20	16	14, 18	20	15, 21
Job mobility (cumulative area under the curve) [b]	21	9, 38	20.5	9.5, 36	21	9, 39
Average annual job instability (job changes) [b]	0.11	0, 0.27	0.14	0, 0.30	0.08	0, 0.25
Average annual racial diversity (% Black autoworkers) [a]	40	25, 49	43	36, 52	31	17, 46
Years of follow-up	37	27, 46	39	29, 46	36	26, 46
Year of hire	1947	1933, 1963	1959	1948, 1969	1934	1929, 1959
Age at hire	27	22, 35	27	22, 34	27	22, 35
Years at work	21	13, 29	22	13, 29	20	12, 30
Year of death [c]	1983	1970, 1996	1994	1982, 2004	1979	1967, 1991
Age at death [c]	73	63, 81	70	61, 79	73	64, 82

[a] Among active workers. [b] Among active workers with available job assignment information.
[c] Among those who died of a cardiovascular disease.

Figure 4.3 presents the percent Black autoworkers in each job assignment, by year.

Figure 4.3. Percent Black workers in the United Autoworkers – General Motors cohort working in each broad job category, by year (1950-1985). The solid line represents the percent Black workers in assembly, the dashed line represents machining, and the dotted line represents grinding.

The plant racial diversity (percent Black workers) and racial segregation (as measured by evenness) by year are presented in Figure 4.4. As racial diversity increased over time, there is a slight decrease in segregation. For example, in 1950, the Detroit plant comprised 29% Black autoworkers and the segregation was 14%. Meaning, 14% of Black workers would have had to move job assignments to match the percentage of Black workers in the overall plant. In 1985, the percent Black workers increased to 55% and segregation had decreased slightly to 11%.

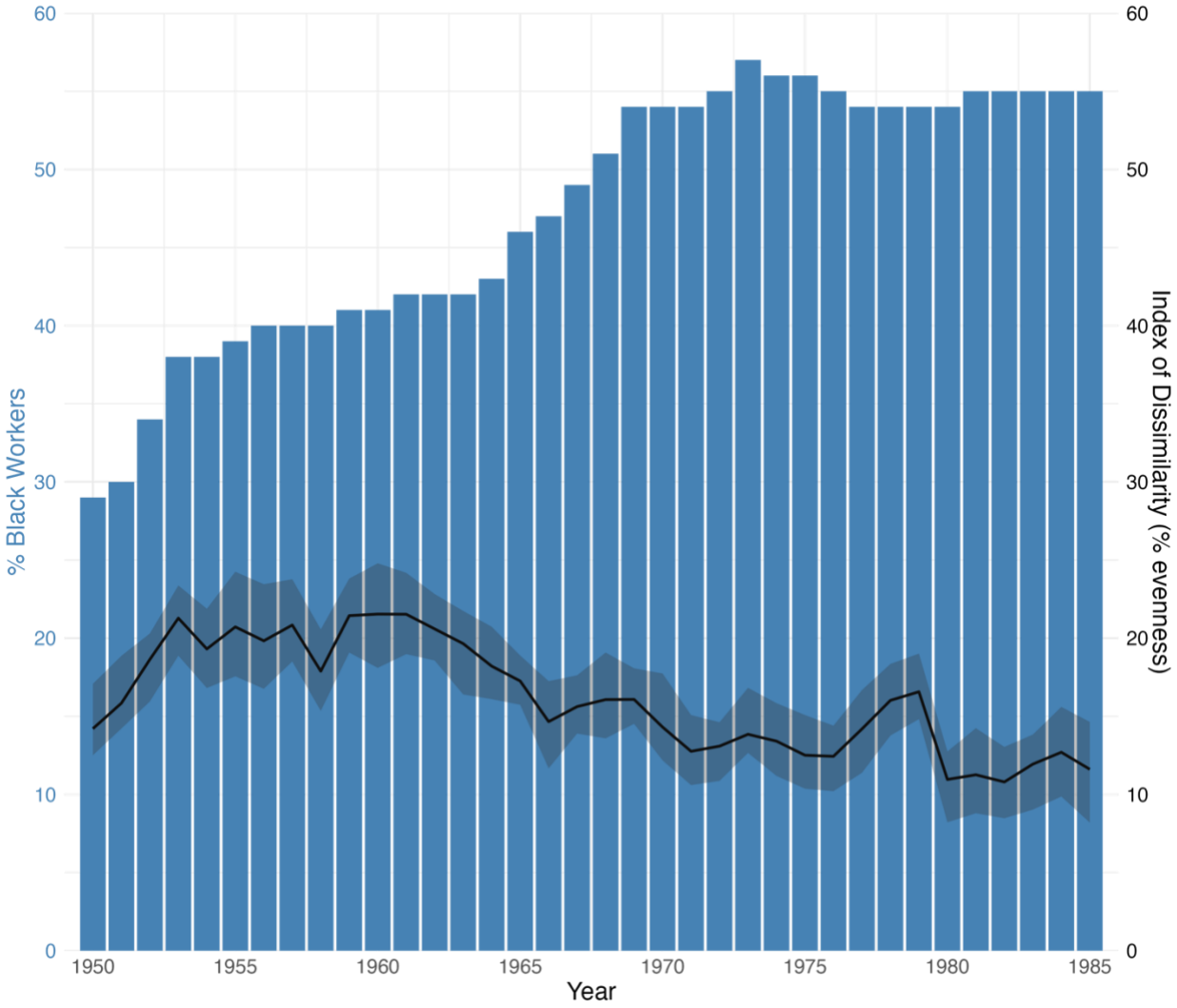

Figure 4.4. The percent Black workers in the Detroit plant from the United Autoworkers – General Motors cohort (blue bars) and the index of dissimilarity value (solid line), by year. The gray ribbons represent the 95% confidence bands for the index of dissimilarity as estimated from 500 bootstraps. The index of dissimilarity is an estimate which measures segregation of a population group by conceptualizing deviations from evenness.[21]

Proportional hazards models for survival analysis

Results from Table 4.2 show that, among Black workers, annual average exposure to segregation was associated with monotonically increasing risk for CVD mortality up to the highest category (HR: 1.61, 95% CI: 1.18, 2.19) compared to workers who experienced the lowest levels of racial segregation. Although the relationship between segregation and CVD mortality was also elevated among white workers, the magnitude of the associations were lower and we did not observe a monotonic response.

Table 4.2. Adjusted cause-specific hazard ratios for cardiovascular disease mortality among Detroit plant UAW-GM autoworkers, by race, in association with average exposure to racial segregation (1941-2015)

Annual average exposure to racial segregation [a]	BLACK WORKERS (n=5,047)		
	No. of cases	HR	95% CI
0.00 - 0.07	484	1.00	-
0.08 - 0.11	501	1.50	1.23, 1.83
0.12 - 0.54	486	1.61	1.18, 2.19
	WHITE WORKERS (n=9,590)		
	No. of cases	HR	95% CI
0.00 - 0.05	1,298	1.00	-
0.06 - 0.11	1,342	1.20	1.07, 1.34
0.12 - 0.54	1,294	1.12	0.93, 1.35

All cox models used age as the time scale and are adjusted for year, sex, year at hire in 5-year bins, average intensity of racial diversity, and cumulative exposure to the racial diversity of the plant's surrounding county. Calculations of average intensity to racial segregation and diversity are time-varying measures calculated by cumulative exposure to the variable of interest divided by the years since hire. [a] Measured by the index of dissimilarity which ranges from 0 to 1, wherein 0 indicates complete integration and 1 indicates complete segregation.

Results from the sensitivity analysis using the sub-distribution HRs were similar and presented in table 4.3.

Table 4.3. Adjusted sub-distribution hazard ratios for cardiovascular disease mortality among Detroit plant UAW-GM autoworkers, by race, in association with average exposure to racial segregation (1941-2015)

Annual average exposure to racial segregation [a]	BLACK WORKERS (n=5,047)		
	No. of cases	HR	95% CI
0.00 - 0.07	484	1.00	-
0.08 - 0.11	501	1.49	1.23, 1.82
0.12 - 0.54	486	1.60	1.18, 2.17
	WHITE WORKERS (n=9,590)		
	No. of cases	HR	95% CI
0.00 - 0.05	1,298	1.00	-
0.06 - 0.11	1,342	1.21	1.08, 1.35
0.12 - 0.54	1,294	1.14	0.94, 1.38

All cox models used age as the time scale and are adjusted for year, sex, year at hire in 5-year bins, average intensity of racial diversity, and cumulative exposure to the racial diversity of the plant's surrounding county. Calculations of average intensity to racial segregation and diversity are time-varying measures calculated by cumulative exposure to the variable of interest divided by the years since hire. [a] Measured by the index of dissimilarity which ranges from 0 to 1, wherein 0 indicates complete integration and 1 indicates complete segregation.

The relationship between job mobility, as represented by the cumulative area under the curve, and CVD mortality among Black and white workers, respectively, are shown in Table 4.4. Among Black workers, the relationship was null. However, among white workers, a higher job mobility was associated with an increased risk of CVD mortality (HR: 1.17, 95% CI: 1.06, 1.29). We observed that for Black workers, within the person-years spent with lower job mobility, 56% of those years were spent in assembly work and within the person-years spent with higher job mobility, 79% of those years were spent in either machining or grinding. For white workers, 58% of the years spent with lower job mobility was in assembly, and 85% of the years with higher job mobility was in either machining or grinding.

Table 4.4. Adjusted hazard ratios for cardiovascular disease mortality among Detroit plant UAW-GM autoworkers, by race, in association with cumulative area under the curve for job mobility (1941-2015) [a]

Job mobility [b]	BLACK WORKERS (n=4,903)		
	No. of cases	HR	95% CI
Lower AUC: 0.50 - 35.50	727	1.00	-
Higher AUC: 35.60 - 130.5	712	1.01	0.88, 1.16
	WHITE WORKERS (n=9,238)		
	No. of cases	HR	95% CI
Lower AUC: 0.50 - 25.00	2,030	1.00	-
Higher AUC: 25.10 - 131.50	1,773	1.17	1.06, 1.29

Abbreviations: Area under the curve (AUC), cardiovascular disease (CVD). All cox models used age as the time scale and are adjusted for year, sex, year at hire in 5-year bins, average intensity of racial diversity, average intensity of job change, and cumulative exposure to the racial diversity of the plant's surrounding county. Calculations of average intensity to diversity and job change are time-varying measures calculated by cumulative exposure to the variable of interest divided by the years since hire. [a] Excludes workers who had missing metalworking fluid exposure information and were therefore unable to be placed into a job assignment category (n=144 Black workers; n=352 white workers). [b] The AUC in each person-year was calculated using the trapezoidal method[23] and cumulated for each year of follow-up. After work termination, the cumulative AUC remains at its last value until the end of follow-up.

The multiplicative interaction results between racial segregation and job mobility are shown in Table 4.5. Among Black workers with higher job mobility, those who experienced increasing levels of racial segregation were at increasing risk of CVD mortality, compared to those who experienced low levels of racial segregation. Among Black workers with low job mobility, the impact of racial segregation was more strongly elevated above the null, but the effect did not vary from moderate to high segregation compared to low segregation (HRs = 1.61).

Contrastingly, among white workers, the relationship between job mobility and CVD mortality varied by job mobility category. Among white workers with higher mobility, the results were above the null and non-monotonic. Within the lower job mobility category, the relationship between segregation and CVD mortality was close to the null. Overall, we observed that the effects of racial segregation on CVD mortality within the lower mobility categories, where workers had less exposure to metalworking fluid, were strikingly different among Black workers and white workers.

Table 4.5. Adjusted hazard ratios and 95% confidence intervals for the multiplicative interaction between segregation and job mobility and the risk of CVD mortality in a cohort of Detroit autoworkers, 1941-2015 [a]

Job mobility [b]	**CVD HR (95% CI)**							
Model	**Black workers (N=4,903)**						LRT χ^2 [c]	*P*
	n cases	Low Segregation (0-6%)	n cases	Moderate Segregation (8-11%)	n cases	High Segregation (12-54%)		
Higher cumulative AUC (35.6-130.5)	95	1.00	333	1.39 (1.07, 1.82)	284	1.50 (1.06, 2.13)		
Lower cumulative AUC (0.5-35.5)	383	1.00	163	1.61 (1.27, 2.03)	182	1.61 (1.14, 2.25)	0.9	0.60
Model	**White workers (N=9,238)**							
	n cases	Low Segregation (0-5%)	n cases	Moderate Segregation (6-11%)	n cases	High Segregation (12-54%)		
Higher cumulative AUC (25.1-131.5)	32	1.00	829	1.52 (1.04, 2.23)	912	1.40 (0.93, 2.13)		
Lower cumulative AUC (0.5-25.0)	1220	1.00	476	1.05 (0.92, 1.19)	334	0.89 (0.74, 1.09)	5.4	0.07

Abbreviations: Area under the curve (AUC), cardiovascular disease (CVD). [a] Excludes workers with missing metalworking fluid exposure information and were therefore unable to be placed into a job assignment category (n=144 Black workers; n=352 white workers).[b] Time-varying calculation of the AUC used job assignments categorized ordinally. The AUC in each person-year was calculated using the trapezoidal method[23] and cumulated for each year of follow-up. After work termination, the cumulative AUC remains at its last value until the end of follow-up. [c] Likelihood ratio test (LRT) χ^2 calculated using nested models for the multiplicative interaction.

4.5. Discussion

In this retrospective cohort study of Detroit autoworkers, we observed strong increases in the risk of CVD mortality with increasing exposure to racial segregation among Black and white workers; although, the effect estimates were lower and non-monotonic among white workers. Comparatively, there was a null relationship between job mobility and CVD risk among Black workers, while white workers demonstrated slightly increased risk of mortality with higher job mobility. In addition, we are the first to report an interaction between racial segregation and job mobility in relation to CVD mortality risk. We observed that both Black and white workers exposed to a higher job mobility level had an increased risk of CVD mortality. With lower job mobility, Black workers experienced a 61% increased risk of CVD comparing moderate and high to low segregation exposure, while white workers' results were near the null.

We note that being transfered to machining and grinding jobs from assembly equates to moving up the job ladder with respect to skill and wages. At the same time, however, these higher skilled jobs involve higher concentrations of aerosolized metalworking fluid, which have been moderately associated with ischemic heart disease [28,29] and ischemic stroke.[30] Therefore the effect of racial segregation on CVD mortality may be partially explained because metalworking fluid exposure is on the pathway. However, our interaction analysis is critical to remove most of the exposure to the fluids because we can examine the exposure-outcome relationship within the lower job mobility category for Black and white workers. In this category, we observed that, on average, workers spent more than half their time in assembly work where there is little fluid exposure. Yet Black workers still experienced cardiovascular harm from segregation, meaning the risk of CVD mortality from racial segregation may be operating through the psychosocial stress pathway.[17,18]

Additionally, Black workers did not experience CVD risk in relation to higher job mobility (Table 4.4), indicating that the observed interactions among Black workers experiencing higher job mobility (Table 4.5) are likely also operating through a psychosocial stress pathway. These results are elevated but slightly lower in magnitude compared to the results in the lower job mobility category, likely due to the benefits of higher wages and social status conferred from a higher-grade job.[31] In direct contrast, white workers did experience increased risk with higher job mobility; we believe this difference is because Black workers who were hired into GM were an even more highly select group of healthy individuals and/or that a stable job with benefits offered them more protection from CVD mortality than their white counterparts.[32,33] Overall, our interaction results underscore the importance of considering the organizational structure of a workforce, such as racial segregation by job, in association with CVD mortality risk for the benefit of all workers, especially those that are racialized.

The strong positive association between racial segregation and CVD mortality risk among Black workers in this study is similar to associations reported among the few studies assessing occupational segregation and racial health inequities.[34,35] One study, which simulated the US occupational segregation in the US healthcare workforce,[34] found an increased risk in hypertension in the white workforce when they were allocated to job assignments based on race-independent models; whereas there was a decrease in hypertension in the Black workforce. Similarly, our results found that increasing segregation lowered the magnitude of the CVD

mortality risk among the white GM workers, while it increased the risk for Black workers. W.E.B. Du Bois's *Black Reconstruction in America* (1935) famously argues that whiteness served as a "public and psychological wage", which characterized the benefits that the white working-class were afforded due to their racial status in a racialized society, despite having a similar economic experience as Black workers.[36] Our study assumes that all GM workers likely experienced similar socioeconomic status and educational achievement at their year of hire. However, once a part of the workforce, white workers appeared to be systematically slotted into machining or grinding jobs which likely afforded them more opportunities for mentorship and guidance to work with the machines and higher wages,[22] and in reference to our results, lower risk of CVD mortality.

Evidence suggests that the 1960s saw gains in Black workers entering higher level positions in occupational structures; however, this trend began to stagnate in the 1980s as political pressures and enforcement waned.[37-39] As a result, racial inequalities in access to job authority and promotions persist. Our study expands on this research by revealing the potential long-term health consequences of racialized job stagnation, calling back to the Inverse Hazard Law which states that hazards accumulate inversely with power and resources in US workplaces. We do acknowledge that at GM, higher-paid machining and grinding jobs expose workers more to metalworking fluids which have been modestly associated cardiovascular harm.[28-30] However, focusing on these environmental exposures alone have drawn attention away from the organization of labor and the reasons specific sub-groups systematically occupy specific job assignments. Thus, allowing occupational epidemiology to ignore how disease burdens may be exacerbated by political and social inequalities. We believe this investigation is part of a growing body of literature investigating work itself as a social determinant of health[40,41] and encourage more epidemiologic studies to continue uncovering the health impacts of racial segregation in occupational spaces.

Finally, no previous US study has reported the potential interaction between racial segregation and job mobility and CVD mortality risk. Indeed, consideration of an interaction may be preferable, given that a ratio measure permits only one type of relation between the two exposures and the outcome, whereas an interaction is more flexible. Generally, we observed a greater increase in CVD risk with increasing segregation regardless of job mobility among Black workers, whereas the relationship varied with job mobility among white workers. These findings reiterate that racial segregation in the workplace plays a defining role in increasing the risk of a preventable chronic disease specifically among racialized workers. Furthermore, our results of reduced CVD mortality risk with higher job mobility, emphasize that workers in lower socioeconomic status positions are disproportionately exposed to low job control, social support, and effort-reward imbalances,[33] all risk factors for CVD.[42]

Our study has several limitations. First, the index of dissimilarity is only an indicator of one dimension of segregation, evenness, which may not capture the entirety of the exposure.[43] In addition, autoworkers comprising the categories "Black" and "white" may be heterogenous and ethnicity is unknown. This limitation in our records may conceal important opportunities to investigate multiple racial and/or ethnic pairings using the segregation index. Despite these drawbacks, the index is not sensitive to group size, and has an intuitive interpretation. Second, the analysis presented here is constrained by the study data, in that employment records end in

1994, although the Detroit plant continued to operate into the 2000s and follow-up continued until 2015. Third, our GM work records did not provide wage information to guide our categorization of job mobility, though we were able to validate our assumptions that assembly offered the lowest wages and grinding the highest through wage information provided by the Bureau of Labor Statistics.[22] Finally, the generalizability of our study results may be limited to Black and white working populations in manufacturing industries in the United States. However, our study strengths include a long follow-up of a large group of Black and white workers with similar incomes, and health and pension benefits by mid-life. Future research among cohorts with greater power to observe workplace segregation exposure-outcome relationships among female workers specifically should continue this work with an intersectional lens. After all, occupational literature has argued for years that the conditions of visibility in the workplace are both gendered and racialized because workplace hierarchies reinforce deep societal structures of race, gender, and power that are inextricably linked to career trajectories and health.[44]

4.6. Conclusions

We observed that both Black and white workers who experienced high levels of workplace racial segregation had an increased risk of CVD mortality and that this increased risk was consistent among Black workers, regardless of job mobility. Our results suggest that reducing job-based racial segregation in the workplace and increasing opportunities for career advancement, may improve cardiovascular health among racialized workers and therefore reduce workplace racial inequalities in CVD mortality.

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Chapter 5. Conclusions and future research

Structural racism is widely considered a fundamental cause of US racial health disparities in CVD. A large body of public health research examining the link between racism and CVD risk factors and mortality have applied a social health disparities and stress lens to understand how racial discrimination is embodied. The effects of structural racism, however, are not adequately captured without consideration of long-term social exposures outside of the neighborhood—in particular, the workplace.

Targeting institutional policies that reinforce structural-level discrimination which impact Black-white disparities in morbidity and mortality can be a considerable challenge for public health researchers. There are mutually reinforcing systems across the life course that contribute to the accumulation of stress due to structural racism. Within such a complex system, researchers must therefore find innovative methods to conceptualize and isolate specific aspects of structural racism. We found that the workplace offers an upstream source of racial health disparities. Moroever, the workplace provides a potential lever for improving health equity via employment policies that promote racial diversity and reduce segregation of the workforce. Because hourly workers within a specific skill-level and job assignment have similar benefits, education, and socioeconomic status, researchers may be better able to isolate and assess the impacts of structural racism on health.

This research investigated the potential contribution of workplace policies and practices that may reduce racial health disparities in CVD mortality. In addition to lowering hazardous workplace exposures, increasing the diversity of the workplace, and reducing job-based segregation may prevent CVD mortality among Black workers. Workplace equity policies like Affirmative Action have been established to support equal access to the workplace, and our results suggest that such efforts may lead to health benefits for racialized workers. Initiatives focused on diversifying the workforce in emerging sectors of manufacturing and research, such as the 2022 CHIPS and Science Act, can not only advance workplace equity, but improve health outcomes. Still, implementation alone is not enough, policies and practices that aim to improve inclusion, equitable support, hiring, and retention (to name a few), must be informed by historical and contemporary structures that have enforced racial discrimination, be evaluated for their effectiveness, and continuously updated with new research and input from stakeholders.

Future research should focus on the improvement in assessing the long-term impacts of structural racism in the workplace, specifically on the development of standardized measures that capture cumulative exposure to racial and ethnic diversity, job instability, and job mobility over time. Epidemiologic study designs should continue to measure workplace racial diversity and segregation in multiple occupational populations, racial and ethnic groups, geographic locations, and historical periods to build our knowledge of structural racism in the workplace and its impact on preventable chronic disease. Moreover, public health researchers can measure the effectiveness of labor laws and workplaces practices that claim to increase diversity and inclusion by using empirical data to measure to what extent such support is improving job and health outcomes among marginalized groups.

Finally, epidemiologic evidence shows that racial health disparities persist, indicating it remains a pressing public health burden despite widespread awareness. Understanding the negative health consequences of structural-level racial inequality requires epidemiologic approaches that encompass the fundamental cause and ecosocial frameworks. Both use a life-course perspective which is an important lens through which epidemiologic focus can shift from individual experiences to identifying structures and systems as the sources of power for racialization and racial health inequalities. Future peer-reviewed studies of the long-term health consequences of workplace policies and practices will provide the evidence needed to promote those that eliminate racial disparities in working populations and improve health overall.