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Original Contribution

Decomposing Black-White Disparities in Heart Disease Mortality in the United States, 1973–2010: An Age-Period-Cohort Analysis

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Against the backdrop of late 20th century declines in heart disease mortality in the United States, race-specific rates diverged because of slower declines among blacks compared with whites. To characterize the temporal dynamics of emerging black-white racial disparities in heart disease mortality, we decomposed race-sex–specific trends in an age-period-cohort (APC) analysis of US mortality data for all diseases of the heart among adults aged ≥35 years from 1973 to 2010. The black-white gap was largest among adults aged 35–59 years (rate ratios ranged from 1.2 to 2.7 for men and from 2.3 to 4.0 for women) and widened with successive birth cohorts, particularly for men. APC model estimates suggested strong independent trends across generations ("cohort effects") but only modest period changes. Among men, cohort-specific black-white racial differences emerged in the 1920–1960 birth cohorts. The apparent strength of the cohort trends raises questions about life-course inequalities in the social and health environments experienced by blacks and whites which could have affected their biomedical and behavioral risk factors for heart disease. The APC results suggest that the genesis of racial disparities is neither static nor restricted to a single time scale such as age or period, and they support the importance of equity in life-course exposures for reducing racial disparities in heart disease.

age-period-cohort models; blacks; health status disparities; heart diseases; United States; whites

Abbreviations: APC, age-period-cohort; CI, confidence interval; ICD, *International Classification of Diseases*.

Editor's note: *An invited commentary on this article appears on page 313, and the authors' response appears on page 318.*

The dramatic reduction in heart disease mortality in the United States—a 60% decline between 1950 and 1999—ranks among the Centers for Disease Control and Prevention's top 10 greatest public health achievements of the 20th century (1). All major racial and ethnic groups in the United States experienced declines, but the rate of decline for whites was relatively consistent and steep, while it has been slower for blacks, resulting in a widening relative gap between blacks and whites (2, 3). In 2010, the racial gap in heart disease mortality accounted for more than a quarter of the black-white life expectancy disparity (4).

In order to further develop sound hypotheses regarding the conditions contributing to the widening gap in heart disease mortality between blacks and whites, it is important to examine the dimensions of these trends from multiple angles. For example, are the growing racial disparities a function of differential rates by age group, differential exposures to contemporaneous period effects, or differential cohort experiences? It is not uncommon to attribute population-wide heart disease mortality declines in the United States to a combination of risk factor reduction and implementation of new effective secondary and tertiary medical and surgical interventions (5). However, age-period-cohort (APC) analyses, which decompose trends into component dimensions of time, suggest that the overall declines may be attributable primarily to generational cohort improvements (6)—a pattern mirrored in some other countries (7–9).

While APC analyses have been used in sex-stratified studies, this analytical approach has not (to our knowledge) been used to examine the emergence and persistence of racial disparities in heart disease mortality. Such results would have important implications for better understanding the determinants of the widening disparities and would have implications for prevention efforts. For instance, large period trends in emerging racial disparities would point to the relative importance of equitable access to advances in medical technology and health services as drivers of disparities, whereas a larger cohort trend would be consistent with racial differences in exposures during critical periods of early life or cumulatively across the life course.

To more fully characterize the temporal dynamics of the late 20th century emergence of black-white racial disparities in heart disease mortality, we carried out APC analysis of race- and sex-specific rates of heart disease mortality for black and white adults in the United States between 1973 and 2010.

METHODS

Data sources

Annual numbers of US heart disease deaths among people aged 35 years or older were obtained from the National Center for Health Statistics (10). We analyzed data from the period 1973–2010, as it represented the longest span of complete rather than sampled data. Deaths were defined according to the *International Classification of Diseases* (ICD) definition for "all diseases of the heart," which included the following codes: for the Eighth Revision of the ICD (1968–1978), codes 390–398, 402, 404, and 410–429; for the Ninth Revision (1979–1998), codes 390–398, 402, and 404–429; and for the Tenth Revision (1999–present), codes I00–I09, I11, I13, and I20–I51. This inclusive definition had the benefit of comparability ratios between ICD revisions that were approximately 1, indicating that temporal changes in the ICD codes introduced minimal bias into the study and no adjustments for ICD coding changes were necessary (11, 12).

Heart disease deaths and US Census Bureau midyear population estimates for persons aged ≥35 years (13) were aggregated by year of death (1973–2010), age at death, sex, and race (white or black). Year of birth was calculated as year of death minus age. Age, period, and cohort time scales were grouped into 5-year categories. Information on Hispanic ethnicity was not collected in the United States nationally on death records prior to 1999, and therefore data for Hispanics were not analyzed separately in this study.

Descriptive analysis

Data were arranged separately by race and sex into contingency tables permitting summarization in several ways. Age-specific rates can be compared along columns, period-specific rates can be compared across rows, and the experience of a birth cohort can be tracked along the diagonal as each group ages up to a subsequent period. Age × period and age × cohort plots of these rates permit visualization of trends. Age-specific plots of black-white mortality rate ratios visualize variation in the magnitude of the black-white disparity by age and cohort.

Statistical analysis

Formal testing of independent age, period, and cohort patterns was carried out using Poisson regression. We compared parameters and Akaike's Information Criterion values for a nested series of 1-, 2-, and 3-factor models including age alone, age + period, age + cohort, and age + period + cohort in order to assess whether additional time dimensions had independent associations with death rates and whether additional parameters significantly improved model fit. Reported race- and sex-specific estimates were derived from a 3-factor model with interactions between each time scale and race-sex group membership.

The challenges involved in fitting the full 3-factor APC model have been widely discussed in the statistical literature (14, 15). Fitting of such models is hindered by the inherent collinearity in the predictors: cohort + age = period (16). Several methods have been proposed to address this parameter identification problem, but none is without limitations. Following common practice, our primary analysis was a Poisson regression that constrained 2 parameters in 1 time scale (period categories 1973–1977 and 1978–1982) to be zero in order to make other parameters estimable (17). The final model specification is more fully described in the Web Appendix (available at http://aje.oxfordjournals.org/).

The primary criticism of the constraint approach to estimating APC models is that parameter estimates may be sensitive to the arbitrary choice of constraints (14, 15, 18). Therefore, we implemented 2 alternative approaches—the intrinsic estimator and the median polish—as sensitivity analyses. The intrinsic estimator is also a constraint-based Poisson regression, but the constraint is imposed via a principal-components analysis in which the design matrix of age, period, and cohort indicators is weighted to adjust for linear dependency (19, 20). The median polish is a nonparametric means of characterizing cohort patterns as a nonlinear departure from the additive contributions of age and period (21, 22). Further details about the intrinsic estimator and the median polish are provided in the Web Appendix.

Descriptive and regression analyses were implemented in R 3.0 (R Foundation for Statistical Computing, Vienna, Austria), and the intrinsic estimator modeling was carried out using the apc_ie macro in Stata 12 (StataCorp LP, College Station, Texas) (20).

RESULTS

We analyzed 23.2 million deaths from all diseases of the heart among black and white adults in the United States, with individuals and populations at risk categorized into 11 age categories, 8 period categories, and 18 cohort categories. In 1973, white men had the highest age-adjusted rate of heart disease death, followed closely by black men (Table 1, Figure 1). Women had lower rates than men, but unlike men, in the early 1970s black women experienced higher mortality rates than white women. Heart disease mortality rates decreased for all groups between 1973 and 2010, but whites—particularly white men—saw steeper and more consistent declines than did blacks. Among men, black and white rates crossed over in the late 1970s as the rate of improvement for black men slowed between 1980 and 1990, producing an increasing black-white gap. Black

Table 1. Rates of Heart Disease Mortality in the United States, by Age, Period, Race, and Sex, 1973–2010

Race-Sex and Age Group, years	Heart Disease Mortality per 100,000 Persons							
	1973–1977	1978–1982	1983–1987	1988–1992	1993–1997	1998–2002	2003–2007	2008–2010
White men								
35–39	47.9	41.4	35.7	27.5	27.3	24.9	24.1	22.5
40–44	117.8	95.9	81.1	62.6	57.6	53.3	50.7	45.2
45–49	240.3	201.3	162.3	128.5	113.9	99.3	93.0	86.0
50–54	419.9	358.2	303.5	230.6	205.6	169.4	155.0	144.4
55–59	695.4	595.9	509.2	404.0	340.3	278.7	234.6	217.3
60–64	1,107.6	944.0	818.9	654.2	567.2	445.6	360.6	315.7
65–69	1,663.7	1,462.6	1,262.7	1,014.1	869.7	695.6	530.2	453.1
70–74	2,452.0	2,220.7	1,992.2	1,582.1	1,350.5	1,102.4	835.5	695.5
75–79	3,673.4	3,321.2	3,018.8	2,461.2	2,132.1	1,783.3	1,385.9	1,153.9
80–84	5,455.2	5,080.5	4,700.9	3,993.3	3,549.9	3,030.7	2,443.9	2,043.0
≥85	6,014.1	5,510.8	5,029.3	4,490.8	4,249.9	3,801.1	3,203.6	2,744.2
Black men								
35–39	104.4	93.3	85.0	74.8	63.3	54.2	53.4	50.3
40–44	197.5	186.5	172.4	142.8	126.8	104.9	92.4	83.4
45–49	370.4	335.9	292.9	271.7	241.5	194.3	172.0	145.1
50–54	596.6	549.0	510.2	439.4	406.8	325.7	295.4	245.4
55–59	854.0	844.5	757.4	698.3	618.1	529.9	441.1	385.5
60–64	1,253.9	1,185.6	1,184.6	1,034.9	929.8	769.7	647.3	550.7
65–69	1,663.3	1,585.0	1,570.3	1,459.1	1,247.6	1,066.4	870.6	774.8
70–74	2,482.7	2,229.8	2,277.9	2,033.3	1,905.8	1,570.2	1,254.7	1,046.7
75–79	3,138.6	2,993.9	2,949.2	2,786.4	2,502.5	2,233.7	1,847.4	1,566.1
80–84	4,310.6	4,483.4	4,548.5	4,065.4	3,770.1	3,274.0	2,784.9	2,340.7
≥85	3,748.9	3,813.8	3,785.7	3,805.0	3,529.0	3,277.0	2,906.4	2,506.0
White women								
35–39	13.3	11.8	10.0	8.4	9.6	10.2	9.8	9.5
40–44	28.8	25.5	21.5	16.5	17.1	17.7	18.7	17.6
45–49	55.4	50.5	43.9	34.9	32.9	30.1	32.0	31.5
50–54	105.4	95.4	87.8	70.7	64.4	55.6	50.8	50.2
55–59	198.6	179.0	163.8	139.2	122.3	104.0	85.1	76.1
60–64	370.0	336.2	308.3	255.0	230.5	189.3	151.7	129.3
65–69	648.8	592.5	540.6	443.0	390.4	331.7	252.8	211.1
70–74	1,168.7	1,054.0	962.6	791.9	690.7	577.8	453.6	368.2
75–79	2,112.8	1,866.3	1,693.0	1,413.2	1,236.3	1,049.1	831.5	691.1
80–84	3,668.0	3,395.8	3,093.0	2,637.7	2,343.5	2,025.0	1,632.0	1,343.4
≥85	4,381.1	3,974.1	3,568.3	3,120.7	2,864.0	2,532.3	2,087.0	1,753.8
Black women								
35–39	49.9	41.5	36.1	33.5	34.3	30.0	27.6	24.5
40–44	97.4	82.7	73.3	66.9	63.6	59.1	51.3	46.6
45–49	178.5	153.3	132.2	123.2	113.9	100.9	88.7	78.9
50–54	295.4	260.1	239.8	215.0	193.6	160.0	144.5	126.9
55–59	455.3	419.8	398.8	357.0	320.1	267.0	213.7	186.7
60–64	725.1	660.9	645.1	584.4	504.5	422.7	332.3	279.3
65–69	1,043.1	954.1	934.8	879.9	761.6	647.2	493.6	401.7
70–74	1,751.7	1,484.3	1,460.9	1,289.3	1,221.6	1,001.8	762.1	618.3
75–79	2,272.8	2,177.2	2,098.7	1,941.1	1,743.4	1,574.9	1,219.7	999.6
80–84	3,237.2	3,418.4	3,447.3	3,062.2	2,811.5	2,444.9	2,033.7	1,662.6
≥85	2,920.8	2,854.9	2,921.5	2,946.6	2,671.1	2,462.9	2,091.5	1,734.0

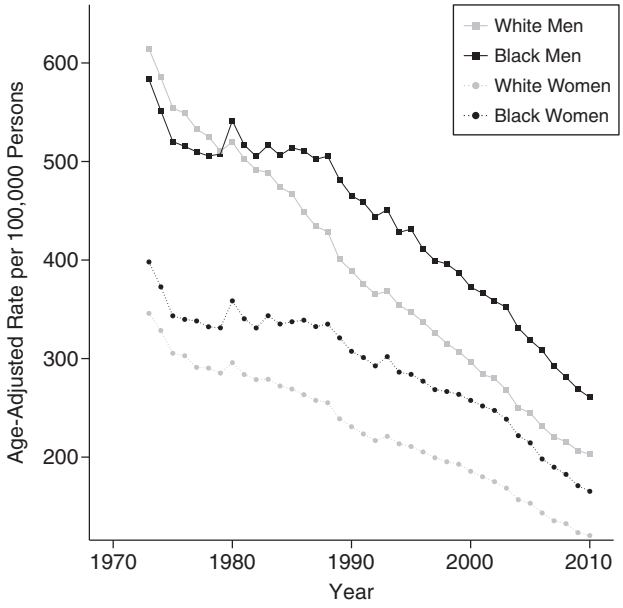

Figure 1. Age-adjusted rates of heart disease mortality per 100,000 persons, by race and sex, United States, 1973–2010. Rates were age-adjusted to the 2000 US Standard Population.

women also experienced slower rates of decline than white women. The disparities produced by these differential rates of decline are plotted in Figure 2, where the age-adjusted racial gap for both men and women increases until the early 2000s and then declines slightly.

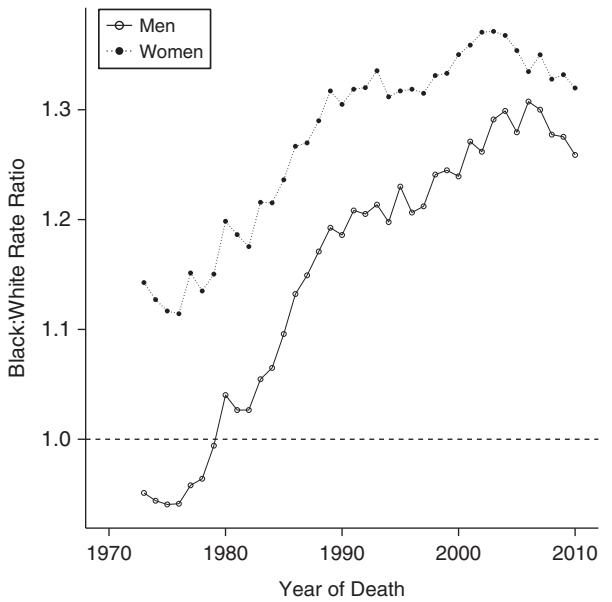

Figure 2. Age-adjusted black-white heart disease mortality rate ratios, by sex, United States, 1973–2010. Rates were age-adjusted to the 2000 US Standard Population.

The race- and sex-specific age trends by period and cohort are displayed in Figures 3 and 4. The decline in heart disease mortality across successive periods is apparent for all groups, but the relative compression of the period-specific lines for black men and women (Figures 3B and 3D, respectively) illustrates the smaller improvement for blacks in comparison with whites. Figure 4 displays the longitudinal experience of specific birth cohorts across their observed life courses. Each generation experienced lower rates than the generation preceding it, although the cohort lines were most spread out for white men (Figure 4A) and most compressed for black women (Figure 4D).

In order to examine the evolution of racial disparities across generations, in Figure 5 we graphed the black-white heart disease mortality rate ratio for birth cohorts (rate ratios and 95% confidence intervals in Web Table 1). For each birth cohort, the magnitude of the racial gap is inversely related to age, with large disparities (black-white rate ratios of 1.2–2.7 for men and 2.3–4.0 for women) among adults aged 35–59 years and little-to-no disparity at the oldest ages. Comparing one generation with the next, patterns vary by sex. Among women, the age-specific disparities are relatively consistent across generations (Figure 5B). However, among men, the black-white gap has grown with each successive generation, with the possible exception of men born in the 1950s and 1960s, for whom disparities were similar to or even slightly smaller than those of preceding cohorts (Figure 5A).

Table 2 shows the Akaike's Information Criterion model fit statistics from 1-, 2-, and 3-factor Poisson models. For each sex-race group separately and for comprehensive models with race-sex interaction terms, each time scale was independently associated with heart disease death rates, and the 3-factor constrained model had the best fit, as evidenced by the smaller fit statistic.

In 3-factor interaction models, there was significant statistical interaction of each time scale (age, period, and cohort) by race and sex ($P < 0.001$ for all tests; data not shown). We summarized results from the interaction model in 2 complementary ways. In Table 3 and Web Figure 1, within-group contrasts are reported separately for each race-sex group. Controlling for period and cohort, the patterns of heart disease mortality rate ratios by age (referent group, age 65–70 years) are similar across race-sex groups, with the exception of white women, who have lower relative risks at the younger ages but higher relative risks at the oldest ages. Within-race and -sex group cohort patterns are characterized by declining relative rates with successive generations born from the late 19th century to the mid-20th century, with a slowdown in change for the latter generations of the 20th century (referent birth cohort, 1913–1922). Black women and white women have similar relative cohort patterns, but among men, blacks have more modest relative generational improvements than whites. The period trends—conditional on age and cohort—are modest, with little race or sex difference.

A complementary perspective on the results for interaction of race-sex groups with each of the 3 time scales is to focus on the contrast of rates between blacks and whites within each time category. Figure 6 and Web Table 2 depict such black-white contrasts from the same 3-factor constrained Poisson interaction model, showing important differences by sex.

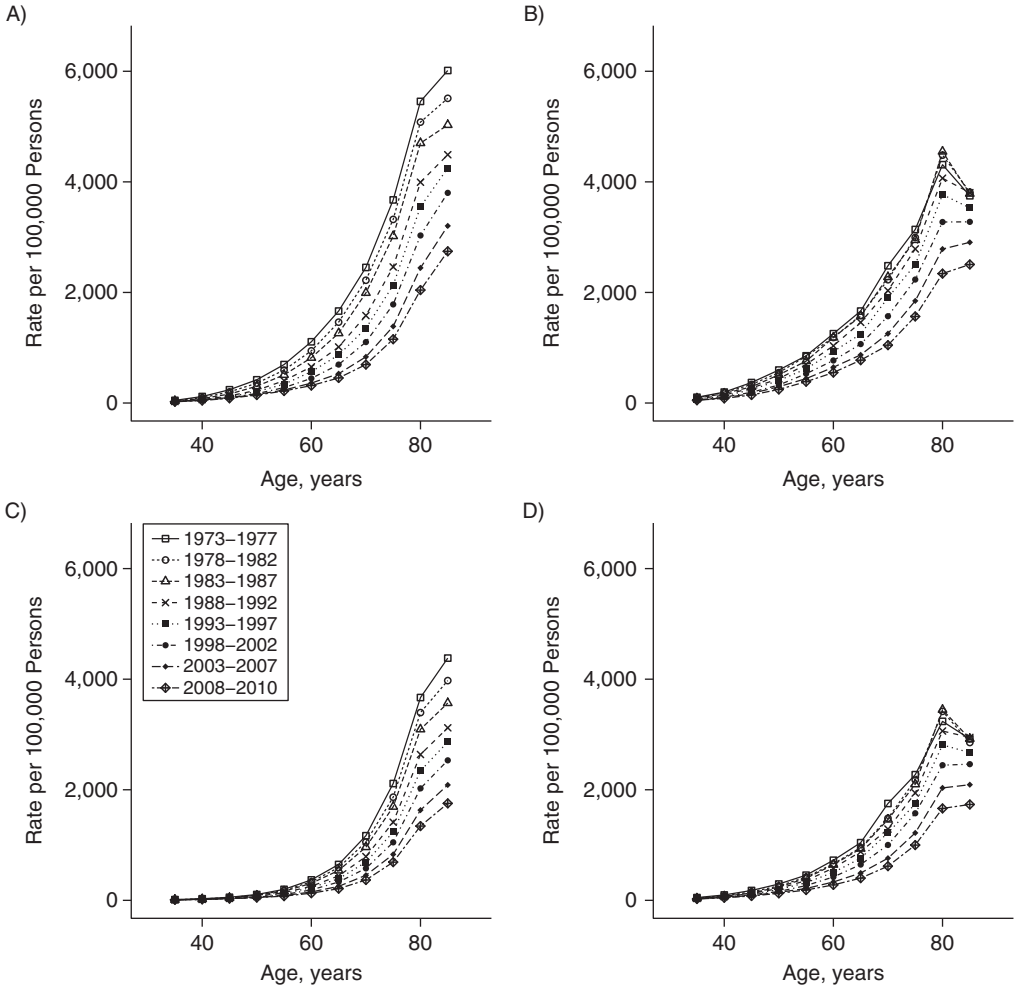

Figure 3. Age-specific heart disease mortality rates for 5-year periods between 1973 and 2010, by sex and race, United States. A) White men; B) black men; C) white women; D) black women.

This disparities perspective suggests that among women, racial disparities vary most on the age scale, where black-white gaps are largest for young women but decline to equality by older ages (Figure 6A). Period-specific black-white rate ratios among women increased from approximately 1.6 to 1.9 between 1973 and 1983 and then plateaued through 2010 (Figure 6B). For women, black-white rate ratios within each generational cohort were relatively constant at approximately 1.6 from 1900 to around 1960, when they began to decline (Figure 6C).

For men, age-specific disparities were more consistent, but generational cohort differences changed dramatically. Conditional on age and period, black men born before 1913 had lower heart disease mortality than white men of the same generations, but from approximately 1917 to the late 1950s, the black-white gap grew until black men experienced approximately twice the heart disease mortality as white men of the same birth cohort. The black-white disparities among men along the period time scale were small and declined from the late 1980s to 2010.

As a sensitivity analysis, we analyzed the same data with 2 alternative methods. The general patterns of the intrinsic estimator results (Web Table 3 and Web Figure 2) were consistent with the Poisson constraint-based model. Age was the dominant time scale, followed by birth cohort, with modest period trends. Racial differences in cohort patterns emerged for early 20th century cohorts of both sexes but declined by 1960. The median polish detected cohort trends as a departure from additive age and period associations and was consistent with greater racial differences in cohort trends among men than among women, particularly for mid-20th century cohorts (Web Table 4 and Web Figure 3).

DISCUSSION

Enthusiasm about the dramatic overall reductions in heart disease mortality in the United States since 1970 is dampened somewhat by the emergence and persistence of black-white racial disparities. To more fully describe the temporal dynamics of this disparity, we applied an APC analysis to race- and

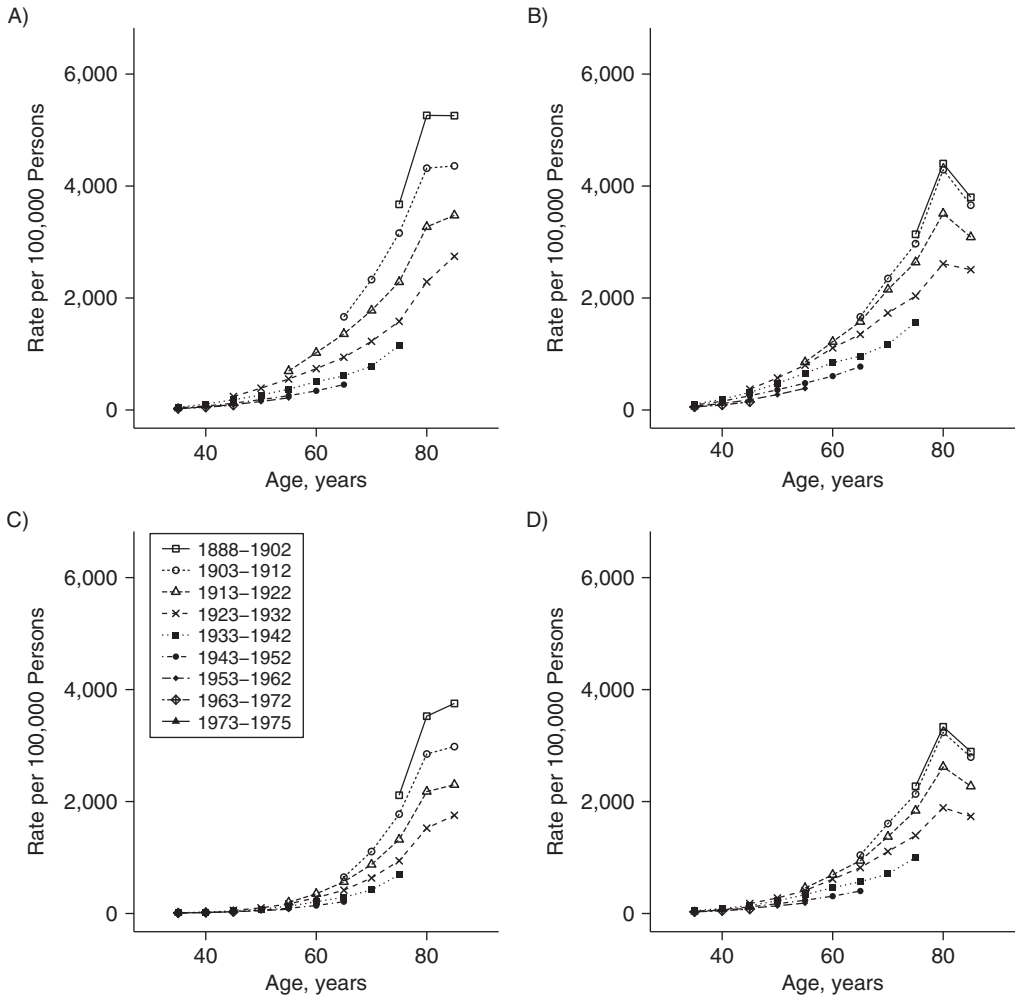

Figure 4. Age-specific heart disease mortality rates for 10-year birth cohorts born between 1888 and 1975, by sex and race, United States. A) White men; B) black men; C) white women; D) black women. Each line connects rates for a common 10-year birth cohort at each observed age.

sex-specific trends. Three patterns were evident. First, the magnitude of racial disparities in heart disease mortality varied substantially by age, particularly among women, with the largest disparities being observed among the youngest adults. Second, the change in period-specific disparities between 1973 and 2010 was modest for both men and women. Finally, cohort trends indicative of risk differences between generations were notable, with growing black-white disparities, particularly for men born between 1920 and 1960, suggesting exposures with impacts over the life course that slowed the rate of decline for black men relative to white men born during the mid-20th century.

The observation that racial disparities in heart disease mortality are greatest among young-to-middle-aged adults is not novel (23) but is important given the years of life lost and the impact on life expectancy gaps (24). Common use of age-adjusted summary rates for surveillance purposes masks this important source of disparity (25). Such patterns could arise from racial differences in age-specific risk factor profiles or age at onset of comorbidity. For example, there are racial differences in early-onset hypertension among blacks, as well as lower rates of hypertension control (26–29). The "John Henryism" hypothesis of stress among low-income blacks resulting from high-effort coping—trying to advance economically in the face of structural discrimination—has been posited as one explanation for earlier and more aggressive hypertension among young blacks (30, 31). Whether through a stress pathway or some other pathway, there is also growing evidence for contributions of residential segregation, neighborhood deprivation, and unhealthy built environments to racial disparities in cardiovascular disease risk factors and outcomes, and these processes particularly impact young and middle-aged adults (32–36). The smaller sex gap among blacks as compared with whites generally, along with differences in risk factor burden and secondary prevention, particularly among black women, probably explain some of the sex differences in age-specific disparities (37, 38). APC analyses typically treat the age dimension as a purely biological aspect of disease risk, but in the case of

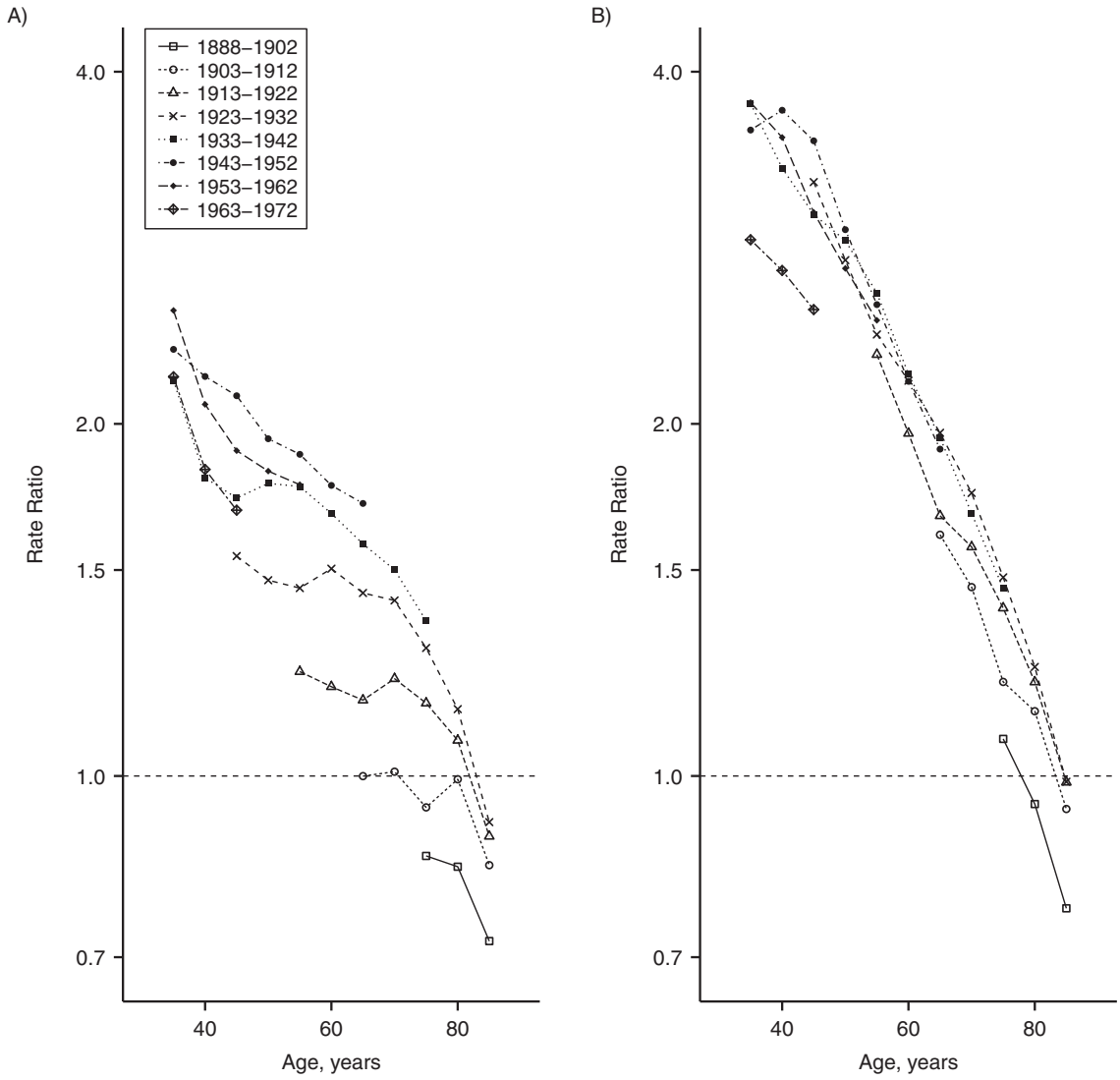

Figure 5. Black-white heart disease mortality rate ratios among men (A) and women (B) in the United States, by age and birth cohort, 1973–2010. Each line connects rate ratios for a common 10-year birth cohort at each observed age. Rate ratios with 95% confidence intervals are shown in Web Table 1.

Table 2. Model Fit Statistics for 1-, 2-, and 3-Factor Age-Period-Cohort Models of Racial Disparities in Heart Disease Mortality, United States, 1973–2010

Model	Degrees of Freedom	Akaike's Information Criterion				
		Whites		Blacks		Race × Sex Interaction[a]
		Men	Women	Men	Women	
Intercept only	87	18,811,486	19,164,309	1,640,338	1,871,111	41,487,245
Age, years	77	1,466,678	876,091	85,073	81,906	2,509,749
Age-period	71	53,774	18,063	4,455	5,109	101,333
Age-cohort	60	9,383	10,547	3,966	4,658	28,554
Age-period-cohort (constrained)	54	6,646	4,983	1,706	1,647	14,982

[a] The "intercept only" model in the "Race × Sex Interaction" column included race and sex; other models included race × sex × time interactions, where "time" was age, period, or cohort as indicated.

Table 3. Race- and Sex-Specific Rate Ratios for Heart Disease Mortality From a 3-Factor Constrained Age-Period-Cohort Model, United States, 1973–2010

Time Scale	White Men		Black Men		White Women		Black Women	
	RR	95% CI	RR	95% CI	RR	95% CI	RR	95% CI
Age, years								
35–39	0.08	0.08, 0.08	0.09	0.09, 0.10	0.04	0.04, 0.04	0.09	0.08, 0.09
40–44	0.16	0.16, 0.16	0.17	0.16, 0.17	0.08	0.08, 0.08	0.16	0.15, 0.16
45–49	0.27	0.27, 0.28	0.28	0.27, 0.29	0.14	0.14, 0.14	0.26	0.25, 0.26
50–54	0.41	0.41, 0.42	0.43	0.42, 0.44	0.24	0.24, 0.24	0.38	0.37, 0.39
55–59	0.58	0.57, 0.58	0.59	0.58, 0.60	0.40	0.40, 0.40	0.55	0.54, 0.55
60–64	0.77	0.77, 0.78	0.80	0.79, 0.81	0.65	0.65, 0.66	0.76	0.76, 0.77
65–69	1.00	Referent	1.00	Referent	1.00	Referent	1.00	Referent
70–74	1.32	1.31, 1.32	1.34	1.33, 1.35	1.58	1.57, 1.58	1.38	1.37, 1.40
75–79	1.77	1.76, 1.78	1.70	1.68, 1.73	2.51	2.50, 2.53	1.83	1.80, 1.86
80–84	2.51	2.49, 2.52	2.39	2.34, 2.44	4.18	4.14, 4.21	2.65	2.59, 2.72
≥85	2.62	2.60, 2.64	2.15	2.09, 2.21	4.56	4.51, 4.60	2.32	2.26, 2.40
Period								
Pre-1983	1.00	Referent	1.00	Referent	1.00	Referent	1.00	Referent
1983–1987	1.01	1.01, 1.02	1.04	1.02, 1.05	1.00	1.00, 1.01	1.06	1.05, 1.08
1988–1992	0.96	0.95, 0.96	1.01	0.99, 1.03	0.95	0.94, 0.95	1.08	1.06, 1.11
1993–1997	0.98	0.97, 0.99	0.99	0.96, 1.01	0.95	0.94, 0.96	1.10	1.07, 1.13
1998–2002	0.97	0.96, 0.98	0.92	0.89, 0.94	0.92	0.91, 0.93	1.07	1.04, 1.11
2003–2007	0.93	0.92, 0.94	0.85	0.82, 0.88	0.85	0.83, 0.86	0.98	0.94, 1.02
2008–2010	0.94	0.93, 0.96	0.80	0.76, 0.83	0.80	0.79, 0.81	0.91	0.87, 0.96
Cohort								
1888–1892	1.57	1.55, 1.58	1.08	1.04, 1.12	1.61	1.59, 1.63	1.29	1.24, 1.34
1893–1897	1.47	1.45, 1.48	1.11	1.08, 1.15	1.47	1.45, 1.48	1.26	1.22, 1.30
1898–1902	1.37	1.36, 1.38	1.13	1.10, 1.16	1.36	1.34, 1.37	1.27	1.24, 1.30
1903–1907	1.27	1.26, 1.27	1.12	1.10, 1.14	1.23	1.23, 1.24	1.25	1.22, 1.27
1908–1912	1.14	1.14, 1.15	1.04	1.03, 1.05	1.12	1.11, 1.12	1.09	1.08, 1.10
1913–1917	1.00	Referent	1.00	Referent	1.00	Referent	1.00	Referent
1918–1922	0.85	0.85, 0.85	0.93	0.92, 0.94	0.89	0.88, 0.89	0.90	0.89, 0.90
1923–1927	0.72	0.71, 0.72	0.89	0.87, 0.90	0.78	0.77, 0.78	0.82	0.81, 0.83
1928–1932	0.59	0.59, 0.60	0.79	0.77, 0.81	0.67	0.67, 0.68	0.71	0.69, 0.72
1933–1937	0.49	0.48, 0.49	0.72	0.70, 0.74	0.59	0.59, 0.60	0.61	0.59, 0.63
1938–1942	0.40	0.39, 0.40	0.64	0.62, 0.67	0.51	0.51, 0.52	0.53	0.51, 0.55
1943–1947	0.34	0.33, 0.34	0.60	0.58, 0.63	0.46	0.45, 0.47	0.46	0.44, 0.48
1948–1952	0.29	0.29, 0.30	0.53	0.51, 0.56	0.42	0.41, 0.42	0.41	0.39, 0.43
1953–1957	0.26	0.26, 0.27	0.49	0.47, 0.52	0.40	0.39, 0.41	0.38	0.36, 0.41
1958–1962	0.25	0.24, 0.25	0.44	0.41, 0.47	0.44	0.43, 0.45	0.37	0.34, 0.39
1963–1967	0.23	0.22, 0.23	0.40	0.37, 0.43	0.47	0.46, 0.49	0.34	0.32, 0.37
1968–1972	0.21	0.21, 0.22	0.40	0.37, 0.43	0.48	0.47, 0.50	0.33	0.30, 0.36
1973–1975	0.20	0.20, 0.21	0.41	0.38, 0.46	0.50	0.47, 0.53	0.31	0.28, 0.35

Abbreviations: CI, confidence interval; RR, rate ratio.

age-varying racial disparities, age may reflect social as well as biological factors.

The results observed on the period time scale were most notable for their modest contribution to race-sex–specific heart disease mortality trends. Independent period contributions are those which similarly affect all age groups during a given period of time. For instance, the advent of a medical or surgical intervention which enhanced survival across the age spectrum would produce a period-specific decline in mortality. As much as 50% of overall declines in heart disease mortality

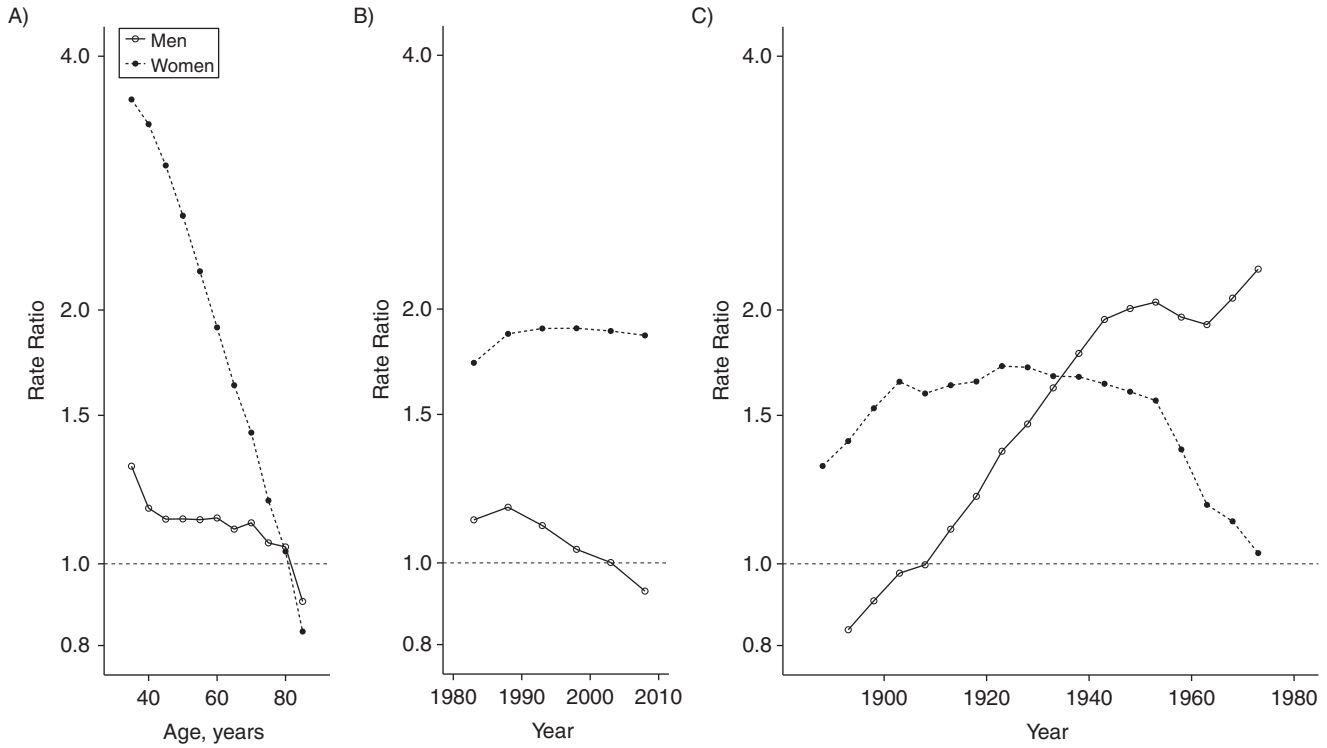

Figure 6. Black-white heart disease mortality rate ratios on age (A), period (B), and cohort (C) time scales from a 3-factor constrained Poisson model, by sex, United States, 1973–2010. The model was fitted by constraining the first 2 period categories (1973–1977 and 1978–1982) to be zero. Rate ratios represent the contrast of black mortality with white mortality (referent) for each time value and sex. See Web Table 2 for point estimates and 95% confidence intervals.

have been attributed to medical and technological advancements made since 1980 (5). The modest declines in racial disparities among men since the early 1990s may reflect some reduction in the racial gap in health care among men, but persistent period-specific disparities among women suggest that the intersection of sex and race complicates interpretation (39). Regardless, it is not apparent from our analysis that a differential distribution of medical or technological advances along the period time scale is a primary driver of observed trends in racial disparities.

The dominance of cohort contributions over period contributions to heart disease mortality in the general population has been previously reported in the United States (6), but the identification in our study of racial and sex differences in the magnitude and timing of cohort changes is a novel finding, as far as we know. Cohort trends—with the connotation of life-course and generational differences—point toward exposure histories that are unique to specific generations due to the social systems and environments into which they were born or in which they experienced critical life-stage benchmarks (21).

The cohort patterns described do not arise simply from a generational difference in risk for a single older age group. Instead differences are seen across the age spectrum, producing patterns that can be understood through the lens of life-course processes for chronic disease etiology (40, 41). Evidence for cohort differences in accumulated exposure to major risk factors comes in part from other APC analyses. An APC analysis of midlife smoking prevalence found that smoking peaked for both black and white birth cohorts born between 1925 and 1935, while subsequent generations had lower smoking prevalences, with similar rates of decline by race and sex (42–44). Therefore, population shifts in smoking patterns correlate roughly with overall cohort improvements in heart disease death rates but probably do not explain racial differences in improvement. In a separate APC analysis of systolic and diastolic blood pressure, there was little independent cohort effect on trends, but for every generation blacks had higher mean blood pressure than whites, and the racial gap widened over time, particularly for men born after 1920 (45). Thus, changes in blood pressure probably do not explain the overall cohort patterns in heart disease for all race-sex groups, but they may partially explain the racial gap in improvement among men born midcentury.

While the distribution of well-established cardiovascular disease risk factors among individuals is important, the presence of both strong cohort patterns and racial disparities in heart disease mortality raises questions about the "causes of causes," including social determinants of health (46, 47). The 20th century brought broad improvements in nutrition, health technology, and socioeconomic status, but diffusion of such benefits has been socially patterned, thereby giving rise to health disparities (48). Blacks in the United States have a higher prevalence of heart disease than blacks in the Caribbean,

independent of socioeconomic status and health behaviors (49), and efforts to reduce socioeconomic barriers to advanced treatment for heart disease by providing universal access to health care have thus far not reduced racial disparities (50). Together these findings raise questions about other aspects of the sociohistorical context in which racial disparities arise in the United States. For example, Krieger et al. (51) reported an association of discriminatory "Jim Crow" laws in the southern United States with cohort and period trends for black premature mortality but not white premature mortality. The sex differences in both age and cohort trends in racial disparities suggest that changing social and health environments may affect the life-course health of men and women differently.

APC decomposition of surveillance data has several limitations. APC models do not describe causal mechanics of disease incidence, but instead provide an exploratory descriptive tool for examining population health patterns. No statistical method fully solves the fundamental nonidentifiability problem of full APC modeling, but the relative consistency in patterns between the constrained Poisson, intrinsic estimator, and median polish approaches lends cautious support to the current findings. Our choice to examine all diseases of the heart may have masked differences in age, period, or cohort patterns of subtypes of heart disease mortality, but it offered the advantage of reduced bias from time-varying misclassification over several revisions of the ICD. Because information on ethnicity was not routinely recorded on US death records until 1999, we could not further separate race and Hispanic ethnicity for the study period. While Hispanic whites have lower rates of heart disease than non-Hispanic whites, the trends are virtually identical (Web Figure 4).

In conclusion, this APC analysis identified modest period contributions but larger age and cohort contributions to race- and sex-specific trends in heart disease mortality. Racial disparities among men may arise from differences in birth cohorts born in the mid-20th century, whereas disparities for women may be a combination of age-specific advantages for white women and modest period-specific disadvantages for black women. These findings point to the significance of a large racial disparity in heart disease mortality at younger ages and the importance of life-course determinants of this racial disparity in heart disease mortality. In addition to ensuring equitable access to secondary prevention for heart disease mortality, further investigations aimed at identifying levers for reduction of black-white disparities in heart disease mortality should consider racial differences in early life and cumulative exposures which have slowed improvements in the burden of heart disease mortality among black Americans compared with white Americans.

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