Shared Trends in Drug and Obesity-Related Mortality Among Black and White Americans

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1. Introduction

US life expectancy at birth declined again between 2016 and 2017, with death rates increasing for 7 of the 10 leading causes of death (Murphy et al. 2018), and significant mortality increases for those at midlife (Case and Deaton 2015, 2017; Meara and Skinner 2015; Masters, Tilstra, Simon 2017a, b). To date, attention has largely focused on deaths attributed to suicide and poisonings from drugs and alcohol (Hedegaard, Curtin, Warner 2018; Hedegaard, Minino, Warner 2018), especially among the white population (Case and Deaton 2015; Kochanek et al. 2017; Meara and Skinner 2015; Shiels et al. 2017). These popularly proclaimed, "deaths of despair" (i.e. chronic liver disease, suicide, and drug-overdoses) are said to reflect the combination of rising distress, economic insecurity, and chronic pain in the *white* population (Case and Deaton 2015, Goldman, Glei, and Weinstein 2018). However, empirical evidence has not supported the assertions that these "deaths of despair" are "symptoms of the same underlying epidemic" impacting recent cohorts of white Americans (Case and Deaton 2015). Instead, research shows inconsistencies across gender and cause of death and illustrates that the evidence is more consistent with period-based explanations coinciding with the increased availability of prescription drugs in the late 1990s (Masters, Tilstra, and Simon 2017a, b; Ruhm 2018). Another potential explanation for worsening US population health and recent mortality increases is the expanding obesogenic environment. Over a decade ago, researchers warned that the obesogenic environment could reverse life expectancy gains (Olshanky et al. 2005) and, indeed, recent trends among the white population show that declines in mortality from metabolic disease have slowed or stalled for men and women (Masters, Tilstra, and Simon 2017a, b). Yet, little research has explored the ways in which these two explanations (i.e. rising drug availability, obesogenic environment) might also be affecting US black mortality. While attention to the opioid epidemic and debates surrounding the underlying causes of recent mortality increases in the white population are important, it is imperative that researchers and policy-makers also consider black mortality from the same causes of death, as overall death rates among blacks remain *above* white Americans (Diez Roux 2017; Murphy et al. 2018). Further, reports suggest that the opioid crisis is getting worse for black Americans (Katz and Goodnough 2017), and in many areas of the United States, blacks experience opioid overdose rates higher than the general population (Bechteler and Kane-Willis 2017).

Thus, while recent increases in mortality among whites have garnered the bulk of the media (Cassidy 2015; Douhat 2015; Krugman 2015) and research attention, these reports shift the focus away from the persistently large health disparities between white and black Americans. In this study, we explore recent trends in cause-specific mortality for both white and black men and women. We analyze the two existing explanations for rising mortality rates, causes of death

commonly associated with "deaths of despair" and those related to an expanding obesogenic environment (i.e. metabolic-related causes). Although we find some differences between white and black Americans, our results show that the same trends in recent increases in mortality among white populations are also observed in the black population. We find period-based increases in drug-related mortality for both whites and blacks beginning in the late 1990s, which is most consistent with an availability and over- prescription of opioid painkillers explanation (Ruhm 2018; Masters, Tilstra, and Simon 2017a, b). This indicates that the opioid epidemic and subsequent increases in drug-related mortality are not exclusive to white Americans, further complicating the "despair" narrative. We also find that metabolic-related mortality has increased for *both* whites and blacks, beginning with cohorts born in the 1950s. Taken together, we argue that there are two current epidemics affecting both white and black Americans – the opioid and obesity epidemics. Researchers and policy-makers should not exclude either one in any explanation or intervention aimed at improving US population health.

2. Background

Two consecutive years of reductions in US life expectancy is particularly concerning given that a decline has not occurred since during the HIV/AIDS epidemic in 1993 and, prior to that, following a particularly deadly influenza year in 1980 (Xu et al. 2016). In fact, Xu et al. (2016) note that in contrast to previous declines, which had one clear underlying cause, these most recent reversals are particularly troubling given that mortality rates for more than half of the leading causes of death have also increased. This follows previous research documenting how Americans live shorter lives and experience poorer health across the life-course compared to their counterparts in other high-income countries (Woolf et al. 2013). One explanation points to higher rates of mortality in early-life through age 50 (Ho 2013), predominately driven by injury-related causes of death (Fenelon, Chen, and Baker 2016). Others have attributed declines in life expectancy to increased risk of death from accidental poisonings and homicide among US men, and a reduction in the mean age of death for most causes among women (Acciai and Firebaugh 2017). However, the most dominant narrative attributes recent increases in mortality to rising death rates from chronic liver disease, suicide, and poisonings from drugs and alcohol (Shiels et al. 2017; Stein et al. 2017).

The prevailing explanation for worsening population health among white Americans has focused on these "deaths of despair," which have been argued to be driven by a common set of societal forces afflicting a "lost generation" of white Americans (Case and Deaton 2015). In this way, Case and Deaton (2015) attribute the increased mortality among middle-aged whites to rising distress, economic insecurity, and chronic pain. The authors followed up their original study with a second study, citing that mortality increases were especially pronounced for those without a college degree (Case and Deaton 2017). As such, they argue that white Americans with low-levels of education experience cumulative disadvantages across cohorts. While some research does indicate that whites with low-socioeconomic standing report decreased well-being and increased distress (Goldman, Glei, and Weinstein 2018), issues have been raised surrounding the methods in Case and Deaton (2015) (see Gelman and Auerbach 2016), and others have pointed out that the "deaths of despair" narrative is not consistent across gender or cause of death (Masters, Tilstra, and Simon 2017a, b), which questions whether despair can be the underlying

cause. In contrast to the "deaths of despair" narrative, research has found that rising drug-related mortality is largely a period-based phenomenon that is most consistent with the increased availability of drugs, not economic distress (Masters, Tilstra, and Simon 2017 a, b; Ruhm 2018). For example, drug-related mortality doubled between 2000-2015, and opioid- related mortality rates have tripled in the same period (Rudd et al. 2016). To put this in perspective, findings suggest that drug-related poisonings alone have reduced life expectancy among US whites by 0.28 years between 2000-2015 (Dowell et al. 2017). While rising opioid and drug-related mortality among white Americans is undoubtedly a public health crisis, existing research has overwhelmingly focused on these trends within the white population, masking what is likely a problem among other race/ethnic groups too.

The white-washing of the opioid epidemic is particularly troubling given that African Americans experience overdose rates at levels higher the national average in several states in the Midwest. Additionally, in Wisconsin, Missouri, and West Virginia, opioid-related mortality rates in the black population are *higher* than those observed in the white population (Bechteler and Kane-Willis 2017). Other research has documented the relative consistency in opioid mortality trends for whites and blacks in recent years (Alexander et al. 2017). Moreover, between 2010-2015, increases in opioid mortality for both populations have been driven by heroin and synthetic opioids (e.g., fentanyl). This same research does report differences in the opioid mortality trends across race between 1993-2010, as whites experienced increases driven by natural and semi-synthetic opioids (e.g., oxycodone, hydrocodone, morphine). However, these differences may reflect racial bias in opioid prescriptions, as white patients are more likely to be prescribed opioids for all types of pain, with the disparity most pronounced for those with severe pain (Pletcher et al. 2008). Nonetheless, the few studies that include the black population clearly indicate that the opioid epidemic is not restricted to the white population.

While significant attention has been paid to rising mortality from drug-related poisonings and their role in recent reductions in US life expectancy, others have warned about obesity and the role that the increasing obesogenic environment has played in US population health. As early as 2005, researchers warned that the current levels of obesity in the United States may reverse the gains made in life expectancy over the previous two-centuries (Olshansky et al. 2005). In fact, researchers have documented that while the all-cause mortality rate has decreased over time, there has been a stalling in the rates of improvement for mortality from heart disease, stroke, and diabetes (Ma et al. 2015). This is in line with recent scholarship that finds that metabolic-related mortality exhibits strong cohort-based patterns in the white population (Masters, Tilstra, and Simon 2017 a, b). These troubling findings may also be observed in the black population, as black individuals are at higher risk of developing diabetes, hypertension, and stroke (Chang et al. 2017).

Also missing from much of the discussion on recent trends in US mortality is the fact that large disparities in all-cause mortality and life expectancy between white and black Americans persists. For example, life expectancy has generally increased for both groups across recent decades, but the all-cause mortality disparity was still 16-percent in 2015 (Cunningham et al. 2017). Diez Roux (2017) notes that while there have been reductions in black death rates over time, black mortality remains "unacceptably high" and recent research focusing on slight increases in the white population may only distract from the persistent health disparities across

race. Further, evidence at midlife suggests that black mortality has also increased for 17 major causes of death between 1999-2016, which is highly concerning given the already high baseline mortality rates (Woolf et al. 2018). We argue that the causes of death that are found to be the major drivers of worsening overall mortality rates for the white population are also worsening in the black population. Further, attention to these causes of death in the black population may have been neglected because research has focused much of its attention on overall black-white mortality disparities, with relative improvements in overall mortality for the black population. To address this gap, this study examines race- and sex-specific mortality from the "deaths of despair" said to be exclusive to the white population, as well as mortality from metabolic-related causes of death.

In the current study, we explore whether the "deaths of despair" and obesity related explanations are consistent across black and white Americans. If there is an underlying epidemic of pain, economic distress, and despair among more recent cohorts of white Americans (Case and Deaton 2015, 2017), we ought to find cohort-based variation in suicide, drug, and alcohol-related poisonings among white men and women. In contrast, if the underlying trends are a result of rising availability and over-prescription of opioids beginning in the late 1990s, we would expect period-based variation in mortality from drug-related poisonings for both white and black men and women. Finally, if mortality increases are a result of an increasing obesogenic environment, the results should show cohort-based mortality increases from metabolic-related causes in both groups as well. By examining yearly trends in age-standardized rates of suicide, drug- and alcohol-related deaths, and metabolic deaths for black and white Americans, separately by gender, and employing cause-specific Age-Period-Cohort (APC) models, we are able to ascertain if the trends in these causes of mortality are largely driven by period or cohort trends, as discussed above. Similar patterning across races and within gender would suggest that the trends and potential underlying causes are similar between black and white Americans.

3. Methods

3.1 Data

We use official death counts from the multiple cause of death mortality files as the numerator. These data come from the National Vital Statistics System (NVSS) Multiple Cause of Death files (NCHS 2016) and are compiled by NAPHSIS. We use official population counts from SEER as the denominator. Data are restricted to years 1990-2015, given that Hispanic ethnicity is specified in population counts beginning in 1990. We then restrict analyses to non-Hispanic white (white) and non-Hispanic black (black) men and women, ages 20-59. We restrict our sample to ages 20-59 because the effect of obesity on mortality increases with age (Masters, Powers, and Link 2013) and because our focus is on external causes of deaths, which are known to be the primary contributors to U.S. mortality prior to age 50 (Ho 2013). All ages are analyzed together, and also stratified into two groups – 20-39 and 40-59 – for several analyses. Foreign residents are excluded.

3.2 Outcomes

We examine four causes of death – metabolic, suicide, and drug- and alcohol-related deaths. Metabolic deaths include those related to diabetes, heart disease, hypertension, and obesity. In line with previous research we also create a category titled "deaths of despair" which includes deaths from suicide, drug- and alcohol- related mortality together (Case and Deaton 2015). Causes of death are coded using underlying cause of death codes from the International Classification of Disease (ICD). In 1999 ICD codes changes from version 9 to version 10, so we standardize across these two separate time periods. We mirror codes from Masters et al. (2017a, b) and use the following ICD codes: *metabolic* (ICD9: 250, 278, 390-398, 402-404, 410-417, 420-429; ICD10: E10-E14, E65-E67, I00-I13, I20-I51), *suicide* (ICD9: 950-959.9; ICD10: U03, X60-X84.9, Y87.0), *drug-related deaths* (ICD9: 304, 850-858, 962, 980-980.5; ICD10: F11-F16, F19, X40-X45, X85, Y10-Y15), and *alcohol-related deaths* (ICD9: 291, 303, 571; ICD10: F10, K70, K73-K74).

3.3 Methods

To find yearly age-standardized mortality rates, we use two separate methods. First, for "deaths of despair" causes, we calculate age-standardized mortality rates for each age, in each year. We then use the age distribution across the entire time period, 1990-2015, to determine an "exposure." Finally, we determine the year-specific age-standardized mortality rate by adding the product of each age-specific rate and exposure. We do this separately by race, sex, and cause of death. Second, for metabolic-related deaths, we fitted Poisson rate models to annual counts of death occurring in single-year ages among all black and white men and all white women aged 20-59 during the time period 1990-2015:

$$\log\left(Deaths_{ij}\right) = \log\left(E_{ij}\right) + \boldsymbol{age}_i'\boldsymbol{\beta} + \boldsymbol{year}_j'\boldsymbol{\gamma}$$

The hazard rate for the *i*th age in the *j*th year is expressed as a function of a set of *i* age groups, $\boldsymbol{age}_i'$ = ($age_1, age_2, \ldots, age_i$) and associated parameters, $\boldsymbol{\beta} = (\beta_1, \beta_2, \ldots, \beta_i)$, *j* time periods, $\boldsymbol{year}_j'$ = ($year_1, year_2, \ldots, year_j$) and associated parameters, $\boldsymbol{\gamma} = (\gamma_1, \gamma_2, \ldots, \gamma_j)$, and the log exposure, $\log\left(E_{ij}\right)$, is declared an offset term in the model where E_{ij} is the estimated July 1 population for age group *i* during time period *j*. Poisson models were fitted separately for black and white men and for women and separately by cause of death. The Poisson rate models estimate the conditional expectation (i.e., mean rate) of the death rate for a given age in a given year (Powers and Xie 2008). As such, fitting Poisson rate models to mortality trends that include single-age fixed effects is comparable to estimating age standardized mortality rates for the U.S. black and white men and women aged 20-59.

We also fitted Age-Period-Cohort (APC) models using the Intrinsic Estimator (IE) constraint (Yang and Land 2013). To improve reliability, we fitted two-year age groups and two-year periods for all models, except the model for black women and alcohol deaths was fitted using three-year age groups and two-year periods. The IE is an appropriate constraint for APC models fitted to tabular rate data (i.e., age-specific rates by time periods), in which birth cohort is perfectly linearly dependent of age and period (i.e., Period – Age = Cohort). The IE has many features that make it attractive for such analyses (see Fu 2016; Powers 2015; Yang et al. 2008; Yang and Land 2013). The IE's constraint is motivated by the fact that in a model with highly

collinear predictors, the use of dummy variable coding will estimate results that strongly privilege a particular solution in the solution space. Kupper and Janis (1980) noted that dummy variable designs impose "unnatural constraints" by setting age, period, and cohort parameters equal to one another (e.g., $\alpha_1 = \beta_1 = \gamma_1 = 0$), which "can produce misleading patterns in estimated coefficients" of APC variation (p. 818). Kupper and Janis (1980: 10) therefore reparameterized the APC model using ANOVA (i.e., mean-centered) effect coding "so as to deal directly with the constraint" that minimized APC parameter invariance. Their proposed solution, equations, and coding of the model design matrix became the foundation for the development of the IE (Fu 2000; Yang and Land 2013). Thus, at the center of the IE's constraint is a rescaling of the input variables to minimize APC parameter invariance in the solution space. It has been shown that the IE minimizes bias in APC estimates when the Age X Period design matrix is large (i.e., if one has many age and period categories, the APC estimates are consistent) (Fu 2016; Kupper et al. 1985; Masters et al. 2016).

4. Results

Age-standardized death rates for "deaths of despair" are shown in Table 1. We see three notable trends. First, we observe differences in cause-specific mortality by race and sex. For example, all race/sex groups experienced increases in drug-related mortality across the study window, although the mortality rate levels are higher among whites. Second, there are notable declines in suicide and alcohol-related deaths for black men and women, while there are increases in the same causes for white men and women. Third, the levels of cause-specific mortality within sex groups show no significant age patterns for suicide and drug-related mortality among whites; however, alcohol-related mortality does vary across age, with mortality rates in the 40-59 age category much higher than those observed in the 20-39 age category. Among blacks, the levels of suicide mortality show no age-specific patterns, but alcohol- and drug-related mortality does differ across age, with higher rates observed in the 40-59 age category than those observed in the 20-39 age category within each sex group.

Figure 1 shows the age-standardized cause-specific "deaths of despair" mortality rates for all ages, by race/sex group (left: white; right: black; top: women; bottom: men). Suicide rates have remained relatively stable for all four groups across the time analyzed, with slight increases among the white population in recent years. Drug-related deaths have increased across the time period analyzed for all four groups. However, these rates have fluctuated more over time for black men compared to the other groups. There have been significant declines in alcohol-related mortality rates for black men and women, while rates for white men and women have increased slightly.

<Table 1 about here>
<Figure 1 about here>

In Figure 2, we see the change in age-standardized metabolic mortality rates for four age groups: 40-44, 45-49, 50-54, and 55-59, relative to 1990. For black men and women and white men, of all ages, we see declines in metabolic mortality rates until around 2011. For white women, there

have been less significant declines, and in fact, increases for ages 40-44 across the time period. Improvements in metabolic mortality are more consistent across age groups for black men and women. Nonetheless, this figure shows that for all race/sex and age groups, we see that rates of metabolic mortality begin to increase by 2011.

<Figure 2 about here>

Period and cohort results from drug-related APC models[1] are shown in Figures 3 and 4, respectively. We see in Figure 3 that rates of drug-related mortality have increased for all four race/sex groups, but are especially pronounced for whites. Rates take off beginning in the late 1990s, though at a steeper rate for whites than for blacks. By 2015, period drug-related mortality rates in white men and women far outpace their black counterparts. White women's rate is double that of black women, and the rate for white men is 1.5 times the rate for black men. In Figure 4, cohort trends in drug-related mortality show little variation among recent cohorts in drug-related mortality, for all four race/sex groups. In fact, rates among younger cohorts (late-1980s/early-1990s) are close in rates to earlier cohorts (early/mid-1930s). There is also a notable increase among cohorts 1945-1955, but this declines and stabilizes again by the 1965 cohort. This increase is most prominent for black men, who reached a peak of 31 deaths per 100,000 for men born in the 1951 cohort, but this rate declines to 13 deaths per 100,000 by the 1969 cohort.

<Figure 3 about here>
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In Figures 5 and 6, we depict period and cohort results from APC models for suicide mortality. In Figure 5, we see that beginning in the early 2000s, suicide mortality rates steadily increased for white men and women. However, we see that suicide rates for black men declined slightly over the study window and rates remained stable for black women. Additionally, across the entire study time period, rates of suicide remain lower for black men and women than their white counterparts. Cohort trends in suicide mortality, shown in Figure 6, show little variation across cohorts. Similar to cohort trends in drug-related mortality, there are slight increases in suicide mortality rates for white men and women across cohorts 1946-60, though the rates then stabilize and slightly decline in recent cohorts.

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Period and cohort results from alcohol-related APC models are shown in Figures 7 and 8, respectively. Figure 7 illustrates that across the time period analyzed, alcohol-related mortality rates for white men and women steadily increased. Conversely, mortality rates decreased for black men and women until 2007, when the rates began to increase for black women and stall for black men. By 2015, alcohol-related mortality rates for white women are greater than the 1990 rates for black women, and for white men, rates are approaching the level of black men's alcohol-related mortality in 1990. Moreover, during the same period in which blacks made

[1] All graphs from APC models are plotted at the means. It is possible that between group differences are a result of compositional differences between the groups.

drastic improvements in alcohol-related mortality, whites have experienced significant setbacks. In Figure 8, we show cohort trends in alcohol-related mortality. Rates steadily declined for white men and women across cohorts. There is a slight increase in the 1946-1955 cohorts for white men and women, and again for cohorts 1975-1985, but the magnitude of the mortality rates for these cohorts is still significantly lower than those for the earliest cohorts. For black men and women, alcohol-related mortality increased until the 1950 cohorts, when it began to steadily decline. Black men and women in the 1975-1985 cohorts also experienced the same increase observed among their white counterparts.

<Figure 7 about here>
<Figure 8 about here>

Finally, results from metabolic-related APC models are shown in Figures 9 and 10. Figure 9 plots period trends, with declines in metabolic mortality for all race/sex groups except white women. Moreover, we observe increases for white women from 1990 to the early 2000s, followed by a slight decline across the early 2000s. Further, all four race/sex groups show stalling and slight increases in period-based metabolic-related mortality rates beginning in 2011. It is also worth noting that although the trends are similar, the levels are not. Rates of metabolic mortality for black men are double that of white men, and black women have rates nearly triple that of white women. In Figure 10, we highlight cohort trends in metabolic-related mortality rates. As with period trends in metabolic mortality, the levels of cohort metabolic mortality are different by race, with black metabolic mortality nearly double that of whites. Nonetheless, results suggest that the trends follow a similar pattern. In this way, we see declines across cohorts 1931-1949, followed by a stalling in the rates, and slight increases among cohorts born in the late 1960s and early 1970s. These results echo warnings about an increasing obesogenic environment in the United States (Olshansky et al. 2005). The metabolic-related mortality results also highlight the persistent health inequalities across race.

<Figure 9 about here>
<Figure 10 about here>

5. Discussion

Recent decreases in US white life expectancy have drawn the attention of both researchers and the public. Research has noted that increases in suicide, drug- and alcohol-related mortality in midlife are likely major contributors to these overall life expectancy declines, and some scholars emphasize rising "despair" as the underlying driver of these changes (Case and Deaton 2015; Goldman, Glei, and Weinstein 2018). Other work has found that metabolic disease deaths are on the rise, especially among recent cohorts of white Americans, evidence for an increasing obesogenic environment in the United States (Masters, Tilstra, and Simon 2017b). Yet, in the pursuit of the underlying explanations for rising U.S. white mortality, researchers have likely overlooked black mortality and the persistently high black-white mortality gap (Cunningham et al. 2017; Diez Roux 2017; Muennig et al. 2018). Although there have been improvements in

overall black mortality (Cunningham et al. 2017), we argue that black Americans have experienced similar setbacks with regard to these rising causes of mortality.

We use age-standardized mortality rates and age-period-cohort (APC) analyses to analyze trends in US black and white suicide, drug- and alcohol-related, and metabolic mortality. We restrict analyses to years 1990-2015 and ages 20-59. With some notable exceptions, our findings generally show similar trends for blacks and whites across these causes of death. Key among the findings is, first, period-based rises in drug-related mortality are comparable for both black and white Americans. Second, we observe increases in cohort-based metabolic disease mortality for both black and white Americans. Third, and divergent from the pattern of similarity, period-based increases in suicide are more pronounced for white men and women than for black men and women. Taken together, our findings suggest that the underlying processes for rising drug-, alcohol-, and metabolic-related mortality are likely shared by black and white Americans – with additional research needed to investigate rising suicide mortality in the white population. We argue that the comparable trends for blacks and whites are further evidence against the narrative of white "despair" previously forwarded by Case and Deaton (2015), and that in fact shared trends in drug- and metabolic-related mortality provide support for the role that broader structural factors play in shaping recent increases in U.S. mortality.

First, period-based increases in drug-related mortality for whites and blacks provide additional evidence that the opioid epidemic extends beyond just the white population (Alexander, Kiang, and Barbieri 2018). Although research indicates that there are racial differences in the type of medical care received (Epstein and Ayanian 2001), and that blacks, on average, are prescribed fewer painkillers (Pletcher et al. 2008), our findings suggest that they are still affected by the rising opioid epidemic. As such, the consequences of the opioid epidemic span beyond just one race/ethnic group, and it is likely that the underlying mechanisms are likely to be structural shifts in the prescription and availability of opioids, as opposed to the previously forwarded "despair" narrative (Case and Deaton 2015, 2017). For example, Figure 3 demonstrates that both black and white Americans experienced period-based drug-related mortality increases which coincide with the change in policies and practices regarding the prescription of opioid-based painkillers in the medical industry (Dart et al. 2015; Manchikanti et al. 2012; Mularski et al 2006; Rudd et al. 2016; Volkow 2014). Simultaneously, aggressive marketing strategies were employed by the pharmaceutical industry for the use of OxyContin (Purdue Pharma), Vicodin, Percocet, and other painkillers as safe, effective, and non-addictive (Van Zee 2009). Yet, all evidence suggests that opiates are highly addictive, and predictably, with the increased prescription and availability, Americans' began to abuse the drug such that between 2002-2015, 25 million Americans had used opiates non-medically (Quinones 2015: 190). Taken together, these structural shifts are likely behind the opioid, and subsequent heroin and fentanyl epidemics (Dart et al. 2015), with shared impacts across race/ethnicity (Katz and Sanger-Katz 2018).

The second key finding presented in this study regards the rising rates of cohort metabolic mortality among blacks and whites, which together provides further evidence corroborating the warnings of a rising obesogenic environment in the United States (Masters, Tilstra, and Simon 2017b; Olshansky et al. 2005). Obesity rates continue to remain high for white and black Americans (Hales et al. 2017). Further, the prevalence of overweight and obese children remains high in recent years (Hales et al. 2017; Ogden et al. 2014), and research has shown that the

relationship between obesity and mortality increases with age (Masters, Powers, and Link 2013). That is to say, more recent cohorts of Americans are likely to continue to experience elevated risk of metabolic mortality. Our findings also suggest that although metabolic mortality rates are higher for the black population than the white population, the persistent obesogenic environment within the United States casts a wide net, one that is not confined to just one race.

Third, suicide is the only "deaths of despair" cause of death that differs drastically between blacks and whites. The "paradox" in suicide mortality – higher rates for white Americans compared to racial/ethnic minorities – is well documented (e.g., Rockett et al. 2010). Some research hypothesizes that the paradox might be attributable to death certificate misclassification, or not classifying a death as suicide that is indeed a suicide (Rockett et al. 2006, 2010), but it is not likely that potential misclassifications would account for the stark, persistent disparities between blacks and whites. Other research suggests that stronger social ties and religious connections might be contributing to lower rates of black suicide (Spates and Slatton 2017; Stack 1998). Yet, a clear theoretical explanation for the stark racial disparities in suicide mortality remains underdeveloped (Stack 2000). Further, recent increases in suicide mortality rates among the US population between 1999-2017 provide reason for concern (Hedegaard, Curtin, Warner 2018).

Among the black population, these concerning trends may have been masked by relative improvements in other areas such as smoking and early life mortality (Khan et al. 2018; National Research Council 2013). Although the overall improvements in black mortality should not be discounted, it is important to remember the persistently high black-white disparity in U.S. mortality (Cunningham et al. 2017; Diez Roux 2017; Muennig et al. 2018). Much of the research on recent increases in mortality neglects the experiences of black Americans by focusing exclusively on the worsening of white mortality (Case and Deaton 2015; Masters, Tilstra, and Simon 2017b) and relying heavily on the worsening mental health of white Americans as the potential explanation (Case and Deaton 2015; Goldman, Glei, and Weinstein 2018). Classifying mental health declines as "despair" and classifying suicide, drug- and alcohol-related mortality as "deaths of despair" simply misses the mark (Cherlin 2018; Muennig et al. 2018). Ultimately, our findings show that despair for disadvantaged whites is not the most likely explanation for the observed increases in these rising causes of death, given that black Americans are also experiencing increases for the same causes. As such, it is important that as we address increases in mortality, notably those associated with the opioid and obesity epidemics, that we focus our attention on the structural factors.

Our study is not without limitations. Notably, there is some debate about the use of Age-Period-Cohort analyses. Although results must be interpreted with these limitations in mind, our findings strongly suggest that there are similar underlying processes for black and white U.S. mortality, specifically for these rising causes of mortality: drug- and alcohol-related and metabolic disease. These patterns are important because they suggest that the explanation for increasing mortality rates is larger and potentially more structural than the explanations favoring worsening mental health and despair for white Americans. In light of these findings, we continue to advocate that scholars shift their attention away from narratives of "rising despair" and instead turn their attention to addressing the roots of recent mortality changes, especially the opioid and obesity epidemic.

6. References

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Table 1. Age-standardized "Deaths of Despair" Mortality Rates, 1990-2015. Mortality rates are per 100,000 person-years.

		White Women			**Black Women**			**White Men**			**Black Men**		
		1990	*1999*	*2015*	*1990*	*1999*	*2015*	*1990*	*1999*	*2015*	*1990*	*1999*	*2015*
DOD	All	11.6	14.7	39.1	19.0	15.5	18.4	38.2	46.5	85.7	59.9	50.8	42.4
	20-39	9.5	12.0	32.9	14.1	9.5	12.8	36.4	40.5	85.0	43.7	34.9	36.1
	40-59	19.6	22.1	58.6	32.7	27.4	33.2	55.1	66.2	114.2	103.5	89.3	70.3
Suicide	All	6.1	5.9	9.7	3.1	2.1	2.9	23.1	22.1	30.9	16.8	13.8	13.4
	20-39	6.2	5.6	8.8	3.6	2.2	3.3	26.8	24.7	31.8	20.4	17.9	16.1
	40-59	8.0	7.7	13.7	3.3	2.4	2.7	25.4	24.4	38.3	14.1	10.2	11.6
Alcohol	All	4.1	4.1	7.5	12.6	6.1	4.8	11.3	12.4	13.7	33.5	16.3	6.3
	20-39	1.7	1.7	2.6	6.6	2.1	1.8	4.4	3.4	4.5	12.9	3.5	1.6
	40-59	10.0	9.1	17.5	26.7	14.6	12.3	27.3	29.1	35.2	79.7	45.7	21.1
Drug	All	1.4	4.6	21.9	3.3	7.3	10.7	3.7	12.0	41.1	9.6	20.7	22.7
	20-39	1.6	4.6	21.6	4.0	5.2	7.8	5.2	12.4	48.7	10.3	13.5	18.4
	40-59	1.7	5.3	27.4	2.7	10.4	18.2	2.4	12.6	40.7	9.7	33.5	37.5

Figure 1. Age-standardized "Deaths of Despair" Mortality Rates, 1990-2015. Mortality rates are per 100,000 person-years.

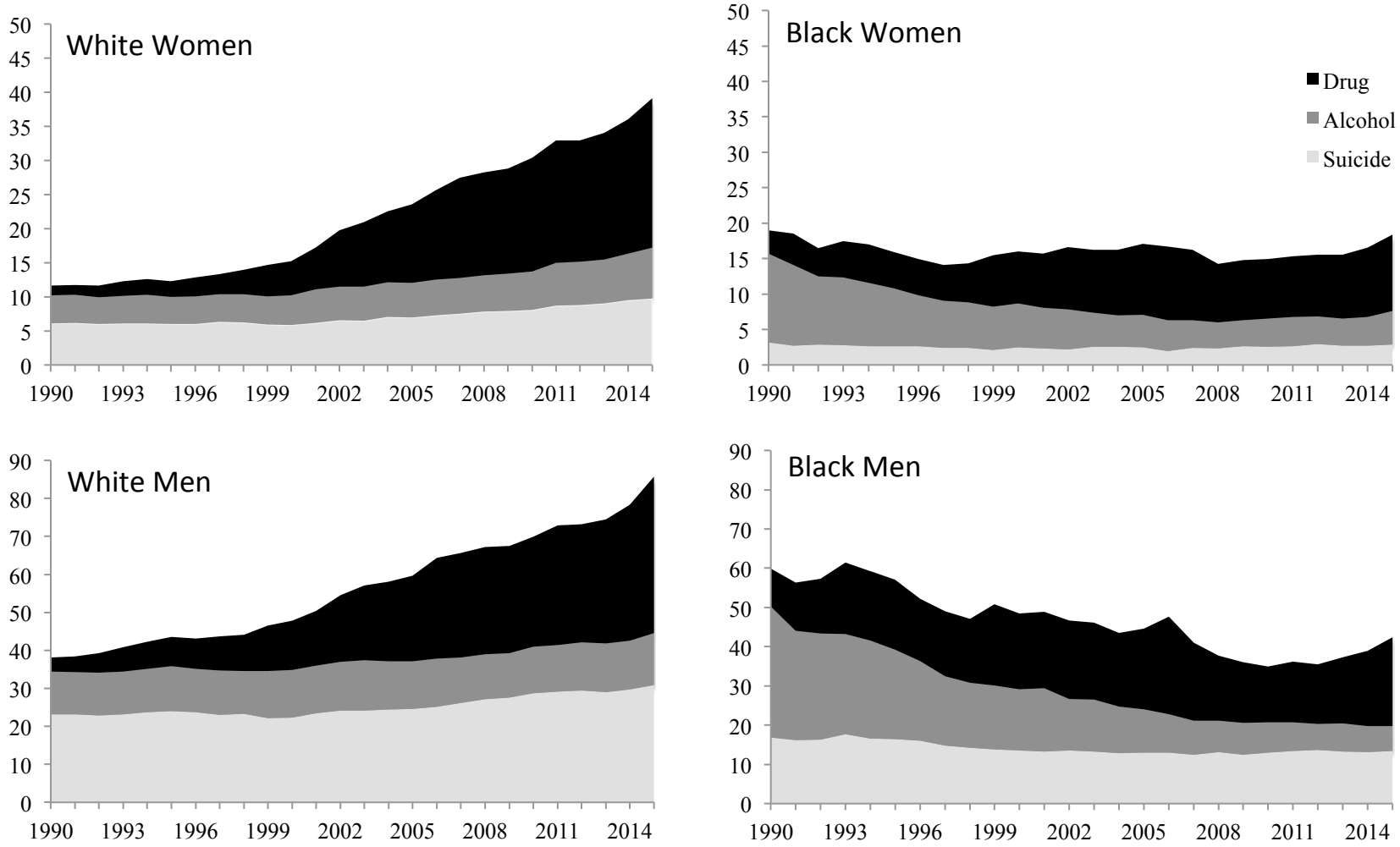

Figure 2. Absolute Changes in Age-standardized metabolic mortality rates, 1990-2015. Mortality rates are per 100,000 person-years.

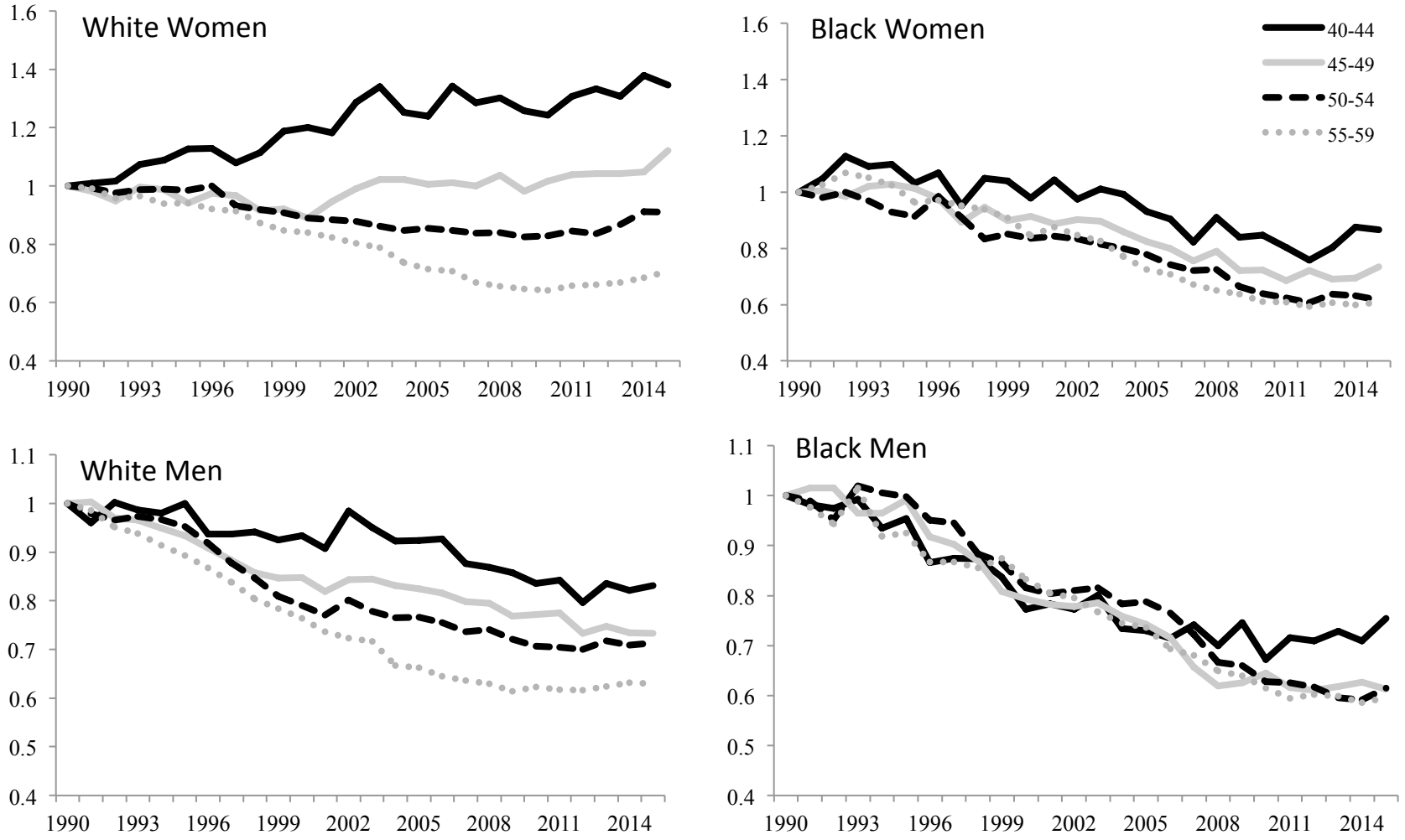

Figure 3. Period-based Variation in Drug-related Mortality, 1990-2015. Estimates are 3-year moving averages, and gray areas indicate upper and lower bounds of the 95% confidence interval. Estimates are mortality rates per 100,000 person-years.

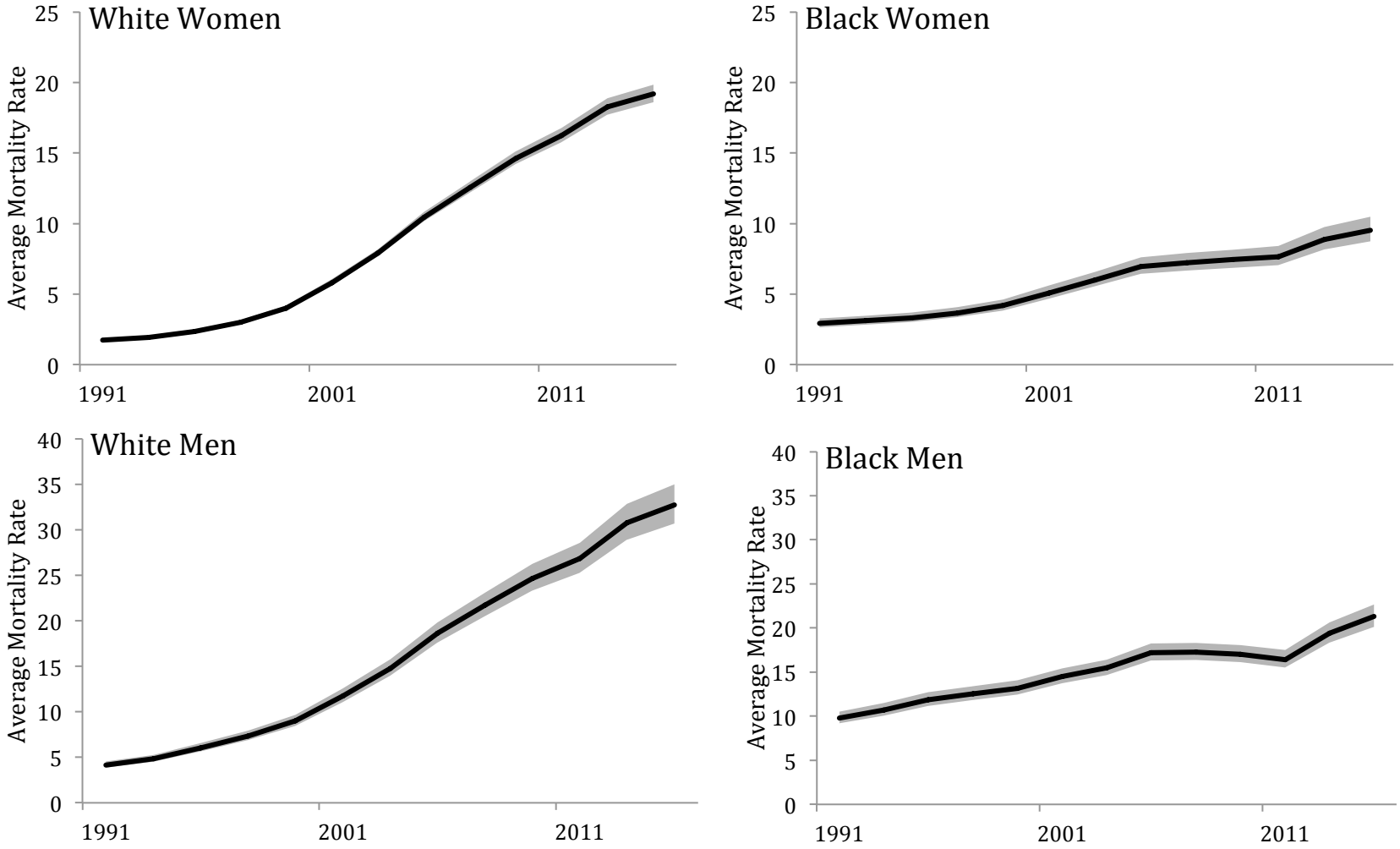

Figure 4. Cohort-based Variation in Drug-related Mortality, Cohorts 1931-1995. Estimates are 3-year moving averages, and gray areas indicate upper and lower bounds of the 95% confidence interval. Estimates are mortality rates per 100,000 person-years.

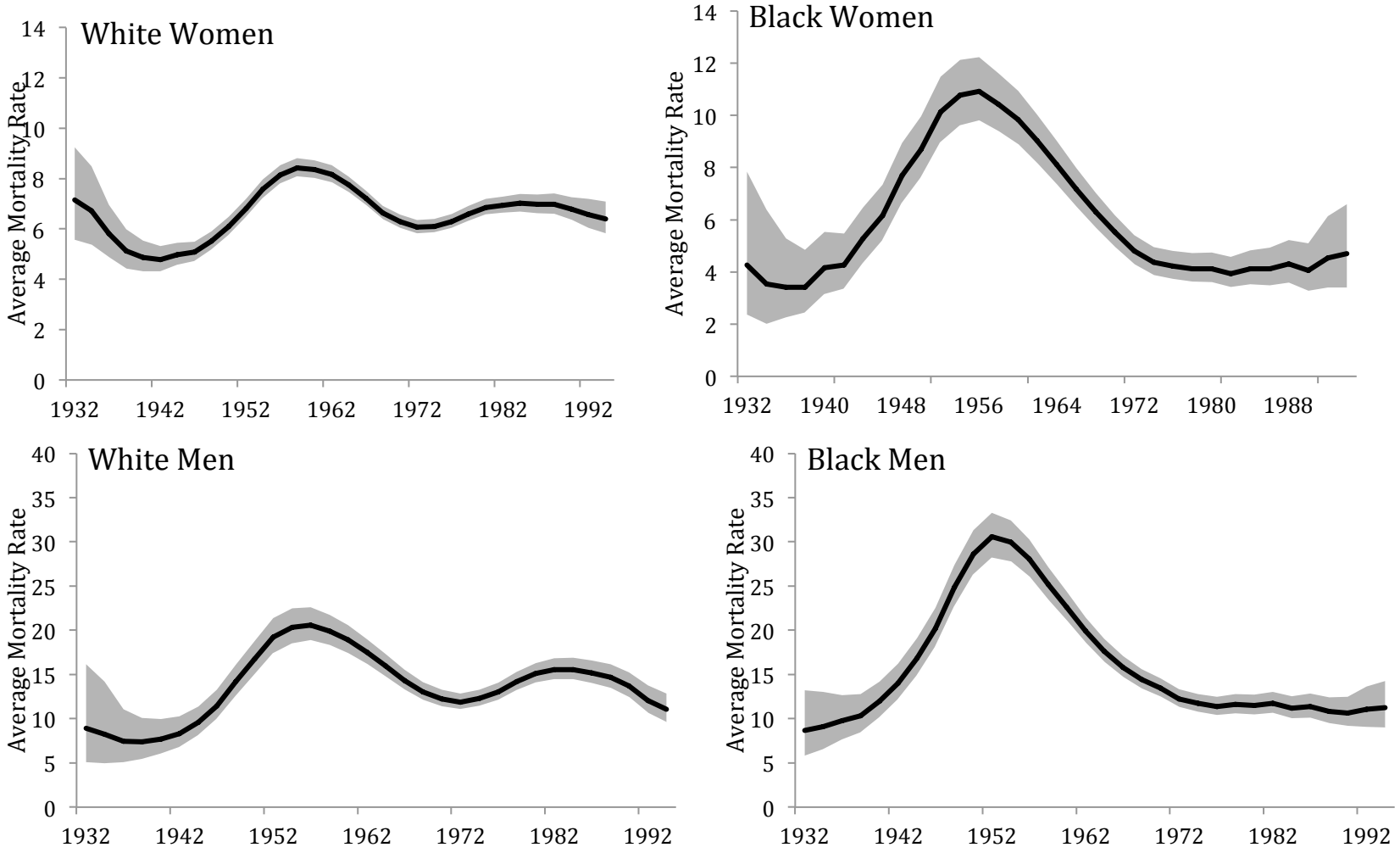

Figure 5. Period-based Variation in Suicide Mortality, 1990-2015. Estimates are 3-year moving averages, and gray areas indicate upper and lower bounds of the 95% confidence interval. Estimates are mortality rates per 100,000 person-years.

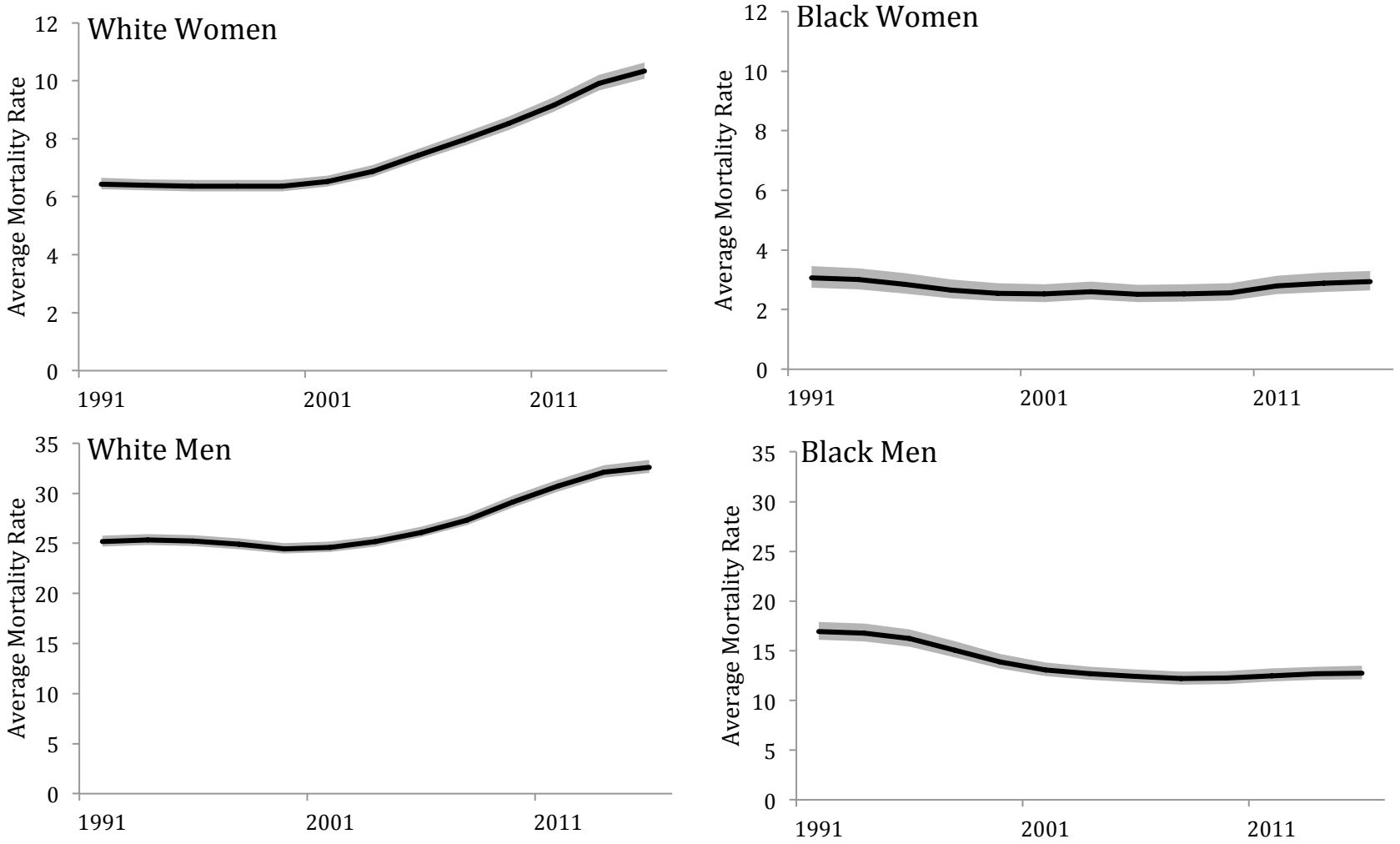

Figure 6. Cohort-based Variation in Suicide Mortality, Cohorts 1931-1995. Estimates are 3-year moving averages, and gray areas indicate upper and lower bounds of the 95% confidence interval. Estimates are mortality rates per 100,000 person-years.

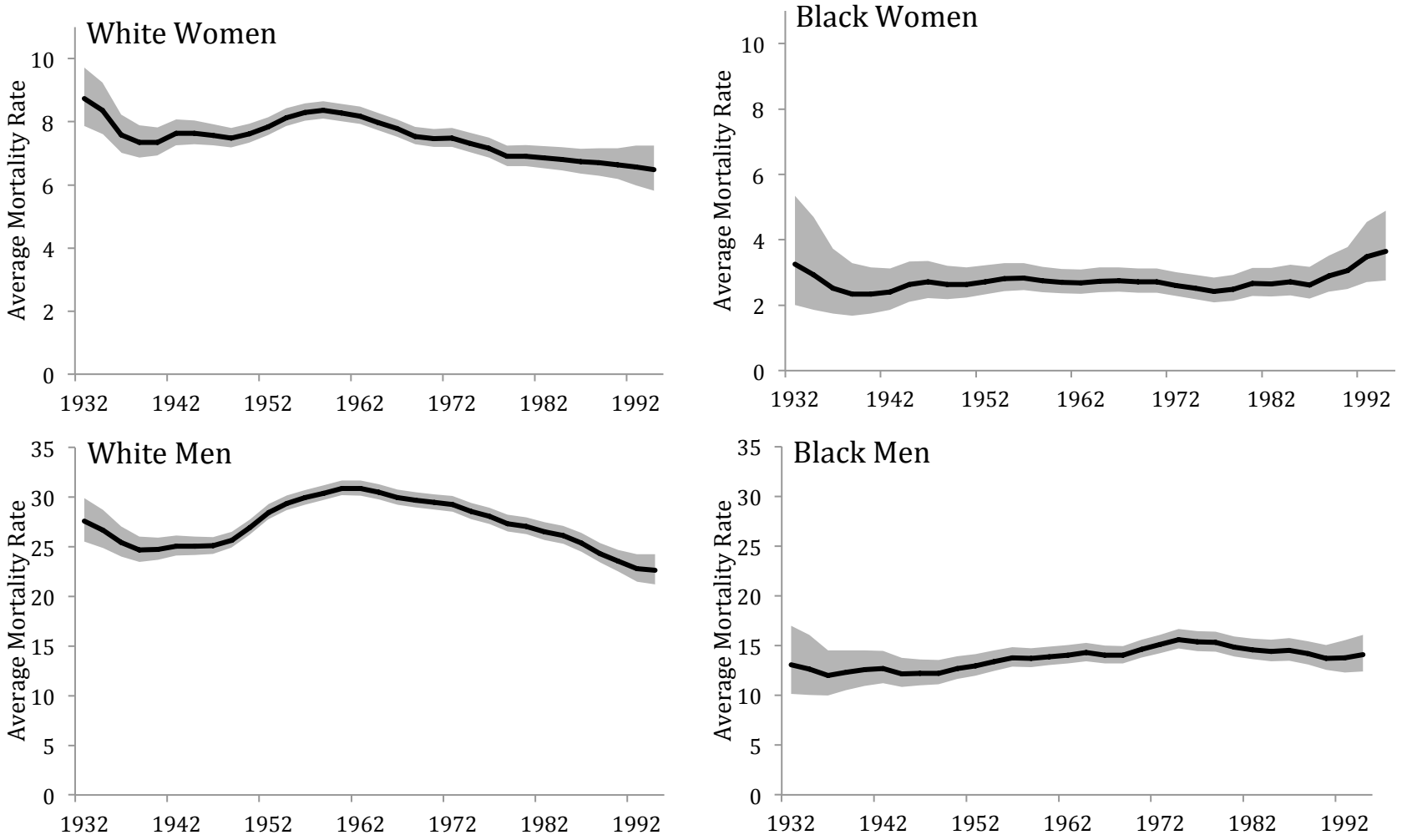

Figure 7. Period-based Variation in Alcohol-related Mortality, 1990-2015. Estimates are 3-year moving averages, and gray areas indicate upper and lower bounds of the 95% confidence interval. Estimates are mortality rates per 100,000 person-years.

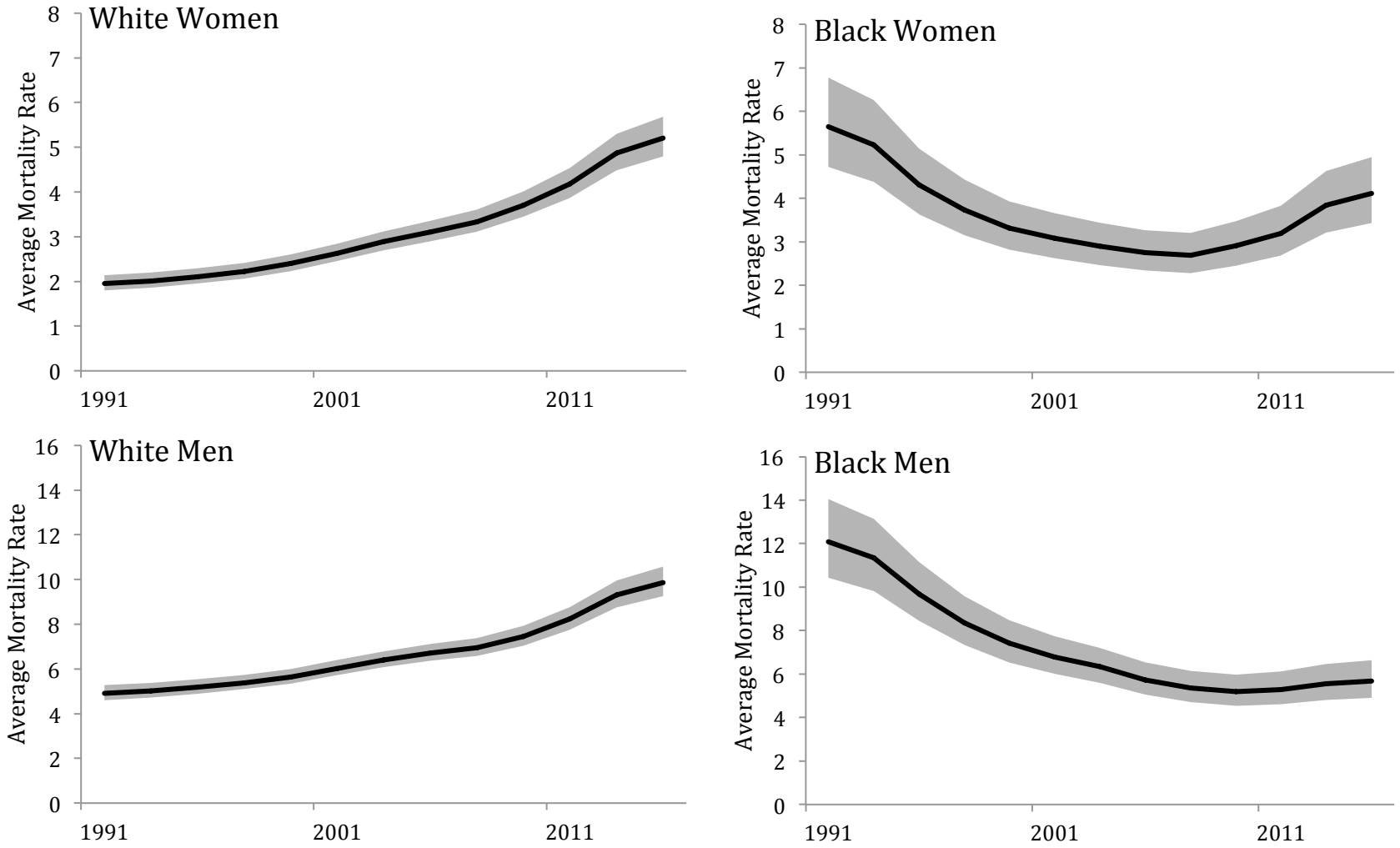

Figure 8. Cohort-based Variation in Alcohol-Related Mortality, Cohorts 1931-1995. Estimates are 3-year moving averages, and gray areas indicate upper and lower bounds of the 95% confidence interval. Estimates are mortality rates per 100,000 person-years.

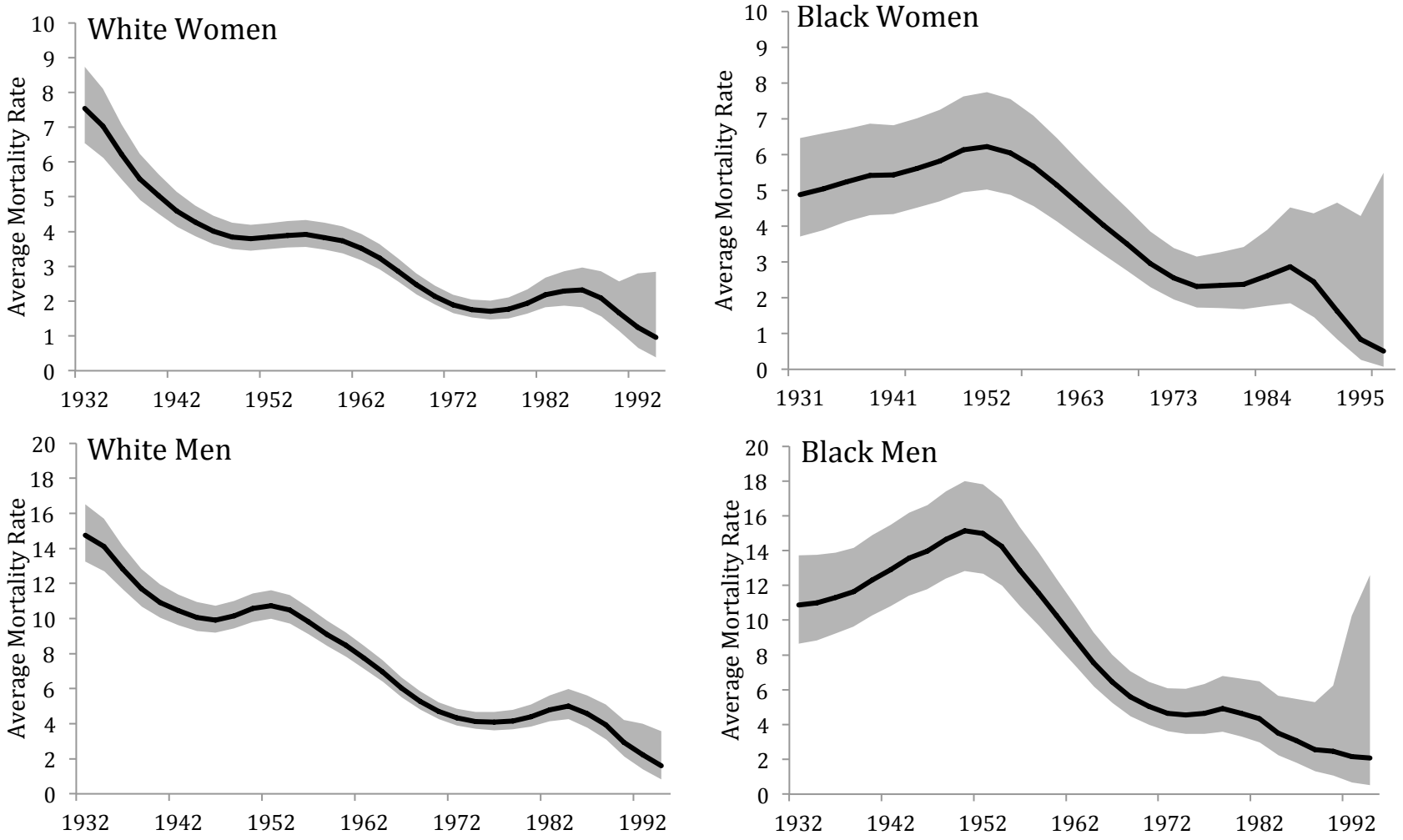

Figure 9. Period-based Variation in Metabolic-related Mortality, 1990-2015. Estimates are 3-year moving averages, and gray areas indicate upper and lower bounds of the 95% confidence interval. Estimates are mortality rates per 100,000 person-years.

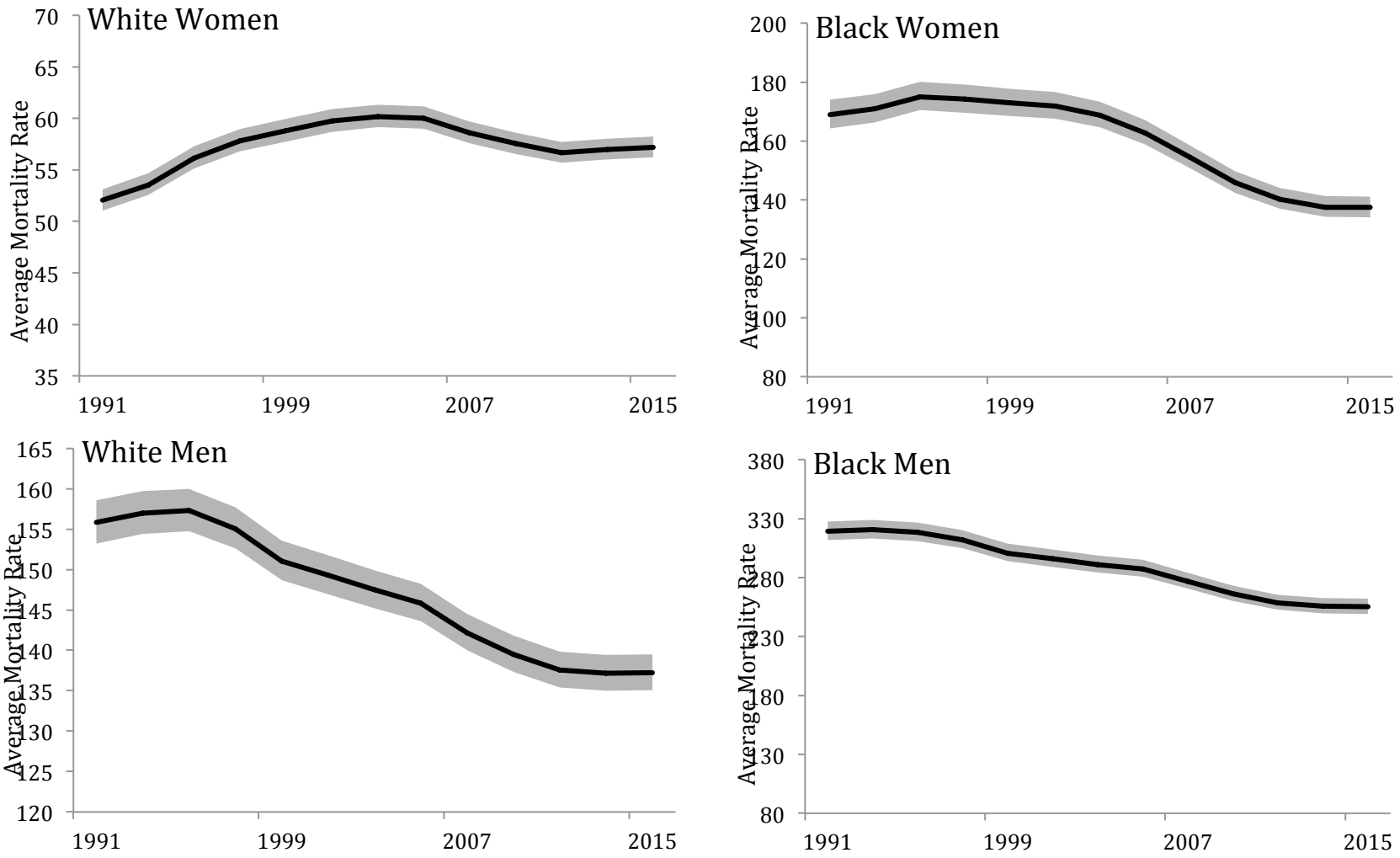

Figure 10. Cohort-based Variation in Metabolic-related Mortality, Cohorts 1931-1995. Estimates are 3-year moving averages, and gray areas indicate upper and lower bounds of the 95% confidence interval. Estimates are mortality rates per 100,000 person-years.