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Increasing U.S. Mortality Due to Accidental Poisoning: The Role of the Baby Boom Cohort

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Abstract

Aims—In this study we examine whether the recent, sharp increase in mortality due to accidental poisoning since the year 2000 is the result of the aging of the baby boom cohort, or instead, a historical trend apparent among decedents of all ages.

Design—We conduct an age-period-cohort analysis using data from the U.S. Vital Statistics and the U.S. Census covering the period 1968–2007.

Setting and Participants—The United States population aged 15–64.

Findings—The increase in mortality due to accidental poisoning since the year 2000 stems primarily from a historical period effect across all ages for whites, but results in large part from a rate spike in the baby boom cohort among blacks. For all demographic groups baby boomers had higher odds of death due to accidental poisoning than the cohorts that came before them and after them. Historical influences acting across all ages led to an increase in accidental poisoning mortality that was almost tenfold for whites and threefold for blacks over the study period.

Conclusions—While the recent, sharp increase in accidental poisoning mortality stems in part from the aging of the baby boom cohort, substantially more of the increase results from influences unique to recent years that have affected all age groups. These results point to the need to bolster overdose prevention programs and policies as the historical increase in accidental poisoning mortality appears to continue unabated.

INTRODUCTION

Deaths due to accidental poisoning – which consists almost entirely of prescription and illegal drug overdoses [1] – have sharply increased in recent years. Between 1999 and 2005 the overall rate increased 62.5%, which includes a doubling of the rate among females [2]. Much remains unknown and unexamined about the forces responsible for this increase, despite the fact that deaths from accidental poisoning are substantial and rank second only to motor vehicle accidents among unintentional injury deaths [3].

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Conflict of interest statement:

None.

In this study we examine whether the recent increase in accidental poisoning mortality is a baby boom cohort effect or a historical period effect. The baby boom cohort, defined as people born between 1946 and 1964 [4], has used illegal drugs at rates substantially higher than the cohorts that came before it [5]. As the baby boom cohort ages it is expected that the associated problems of drug use will become more prevalent; for example, current estimates predict that the number of older adults needing treatment for a substance use problem will triple from 1.7 million in 2000 to 4.4 million in 2020 [6]. Because the peak age for drug overdose death is between age 35 and 54 [2] the recent increase in the national mortality rate due to accidental poisoning may be driven primarily by people from the baby boom cohort who are now entering this high risk age group in great numbers.

An alternative hypothesis is that the increase in accidental poisoning mortality since 2000 is the result of a historical period effect that affects all age groups, not just those in the baby boom cohort. The meaning of drug use and drug mores have changed in recent years. For example, in recent years the profile of cocaine users has shifted substantially so that users today as compared to the past have substantially lower socioeconomic status [7] and higher levels of psychopathology [8]. In addition, the increasing availability and abuse of prescription drugs [9,10] potentially puts the population at greater risk for a lethal overdose, especially to the extent that prescription drug use is included in polydrug combinations [10,11]. Any or all of these influences may be behind a historical increase in drug-related death across all ages that acts independent of birth cohorts, a trend observed in other countries [12].

To examine how the baby boom cohort affects rates of accidental poisoning mortality today, we look back at the historical record of drug-related deaths from 2007 to 1968. If the baby boom cohort is driving the increased mortality rate, then we expect to find a pattern we describe as a “pig in a python.” Just as a swallowed pig forms a hump in a snake that moves slowly through the snake’s body, we expect that the baby boom cohort would have elevated rates of accidental poisoning mortality that slowly moved to older ages with advancing historical years. Specifically, the baby boom cohort should have the highest rate of accidental poisoning among people age 20–24 starting in the late 1960s, the highest levels among those 25–29 five years later, and so on concluding with the highest levels among people age 50–54 in the present day. In contrast, if a historical period effect is at work, then we would expect that in recent years accidental poisoning rates have increased across all age groups.

METHODS

Data

The analysis uses U.S. Vital Statistics data from 1968 to 2007 to provide the number of U.S. deaths per year due to accidental poisoning (the numerator), and also uses data from the U.S. Census to provide the population size (the denominator).

Deaths due to “accidental poisoning by drugs, medicaments, and biologicals” are coded in the U.S. Vital Statistics in all three versions of the mortality nosology used during the study period of this analysis. We code a death as the result of accidental poisoning if the

underlying cause of death is classified as E850-E859 in the ICDA-8 [used by the U.S. Vital Statistics from 1968 to 1978, 13], as codes E850-E858 in the ICD-9 [used from 1979 to 1998, 14], and codes X40-X49 in the ICD-10 [used from 1999 to 2006, 15]. In the year 1972 the released U.S. Vital Statistics are a 50% sample of the population, and therefore for this specific year the analysis adjusts the number of observed accidental poisoning deaths by doubling them.

The analysis ends with the year 2007 because it is the latest publicly released data. The analysis starts with the year 1968, when the baby boom cohort was in its teens, and does not go back further because of uncertain validity for mortality vital statistics with cause of death information in these earlier years [16].

The U.S. Vital Statistics data include information on decedents' race/ethnicity, gender, and age, which are provided by funeral directors and/or informants. On the basis of the mortality data, we code decedents as either black or white. We do not classify decedents on the basis of Hispanic ethnicity, which is not included in the mortality data before 1989.

The analysis uses data from the U.S. Census to provide estimates of the total population of the United States. While the U.S. Census provides yearly information on the U.S. residential population, since 1972 these estimates exclude people who are institutionalized, such as people incarcerated in prisons. Estimates for the total U.S. population that includes those living in institutionalized settings are only provided decennially. In this analysis we assumed linear changes in the institutionalized population between 1960, 1970, 1980, 1990 and 2000, and used a linear interpolation to add it to the residential population. This adjustment is typically small (less than 3%) except for young black males, for whom the institutionalized population adds more than 15% to the national population estimate for the group in their mid 20s.

Analytic Strategy

The research question of this study requires an age-period-cohort analysis, and we use the recently developed “intrinsic estimator” approach [17] to estimate the independent effects of each of these factors. While aging effects are not of central interest in this study, the analysis controls age to separate its influence from the cohort and historical period effects that are of interest.

Death due to accidental poisoning is expressed as the function:

$$\text{Logit}(Y_{ij}) = \alpha + C_k + P_j + A_i + e_{ij} \quad (1)$$

where the effect of the k-th cohort is given by C_k , the effect of the j-th period by P_j , and the effect of the i-th age group is given by A_i ; where α is a constant and e_{ij} is random disturbance. The age-period-cohort algorithm requires cells of equal time duration and in this study age, period, and cohort were each subdivided into five-year groups. Bookend periods and cohorts that were less than five years were estimated by imputing available information to a five-year period (e.g., estimates for the 1968–69 period come from using the rates for these two years to populate a cell that the algorithm treated internally as “1965–

1969”). Age consists of ten five-year groups starting with age 15–19 and ending with age 60–64, and cohort consists of 18 five-year periods starting at 1905. We follow demographic convention and each five-year cohort starts at a year that is a multiple of five. The baby-boom, which is defined as those born between 1946–1964 [4], fits closely into the five-year cohorts of 1945–49, 1950–54, 1955–59, and 1960–64.

The “intrinsic estimator” approach is a principle components estimator that provides a unique solution to equation (1) and thereby addresses issues of empirical identification that make it challenging to find a single solution for this model [18]. The method produces unbiased and efficient regression coefficients, and has performed well in simulation studies [17].

We use a stricter measure of statistical significance than the common $p < .05$ or $p < .01$ cutoff because the sample size is exceptionally large (e.g., 2.6 billion white males and 2.7 billion white females). Instead of using the standard t-values of 1.96 (for $p < .05$) and 2.58 (for $p < .01$), we use larger, more conservative t-values that take into account sample size [19]. Specifically, to denote “strong evidence” that an odds ratio does not include unity we use the t-value as calculated by:

$$t = \sqrt{\ln(\text{sample size}) + 6}$$

and to denote “very strong evidence” we calculate the t-value as:

$$t = \sqrt{\ln(\text{sample size}) + 10}$$

For “strong evidence” the t values come out as 5.26 for white men, 5.27 for white women, 5.06 for black men, and 5.07 for black women. For “very strong evidence” the t-values are 5.63 for white men and white women, 5.44 for black men, and 5.45 for black women.

We run the age-period-cohort analysis in StataMP version 9.0 [20] using the publicly available add-on file for the “intrinsic estimator” algorithm [17].

RESULTS

Figure 1 graphically displays for whites the mortality rate due to accidental poisoning from 1968–2007 across four age groups. These results suggest that for whites a historical period effect and not a cohort effect is responsible for the increase in accidental poisoning mortality since 2000. The increase is present across all age groups and it is not driven solely by decedents from the baby boom cohort. Specifically, for both males and females the highest rates of accidental poisoning mortality ever recorded since 1968 occur after year 2000 for all age groups depicted. For example, white males age 20–24 in 2007 came from a cohort born many years after the baby boomers, and their mortality rate was more than three times higher than it ever had been when the baby boom cohort was this age (19.9 v. 6.32 per 100,000). These results suggest that for whites the recent increase in accidental poisoning mortality results from new influences that were not present in the past.

Figure 2 graphically displays for blacks the mortality rate due to accidental poisoning from 1968–2007 across four age groups. In contrast to the findings for whites, these results are consistent with a cohort effect and not a historical period effect. In all four black male age groups displayed in Figure 2 the baby boom cohort had a rate of accidental poisoning mortality that was higher than the cohort that came before it and/or the cohort that came after it. These results conform to the “pig in a python” pattern in which high rates of accidental poisoning mortality are concentrated in the baby boom cohort and follow it as it ages. For black females the baby boom cohort does not always have the highest rates of accidental poisoning mortality, but when it does not it is tied for the highest or close to it.

Table 1 presents results for the age-period-cohort analysis predicting accidental poisoning mortality. As detailed below, the results indicate that for both blacks and whites the baby boom cohort had rates of accidental poisoning mortality higher than the cohorts that came before it and after it. For whites these baby boom effects were overshadowed by a much stronger historical period effect, which gradually and steadily increased the accidental poisoning death rate about ninefold over the study period. For blacks these baby boom effects were less overwhelmed by the historical period effect, which for blacks increased the accidental poisoning death rate about threefold over the study period.

Table 1 presents unexponentiated coefficients that each sum to zero within the cohort, period, and age estimate groups. The null hypothesis of no cohort, period, or age effects would be indicated by uniform coefficients of zero within the cohort, period, or age groups. The positive coefficient of 0.694 for the 1955–59 cohort of white men indicates an increased odds of death due to accidental poisoning for this cohort. Specifically, in comparison to all cohorts combined, members of this cohort were two times ($2.00 = e^{.694}$) more likely to die of accidental poisoning. The odds of accidental poisoning death for a group of cohorts is indicated by the average of their coefficients; e.g., white men born between 1945 and 1964 had odds of death due to accidental poisoning that were about 62% higher than the odds of all cohorts combined, as calculated by exponentiating .483 (the average of .14, .596, .694, and .502). Finally, comparisons across groups are calculated by taking the difference between their coefficients; e.g., the odds of death due to accidental poisoning were about 3.22 times higher for white men in 1955–59 cohort as compared to the 1920–24 cohort, as calculated by exponentiating 1.169 (which is .694 - -.475).

The results in Table 1 indicate that members of the baby boom cohort had the highest odds of death due to accidental poisoning in comparison to other cohorts. For all demographic groups the largest coefficient is in one of the five-year cohorts of the baby boom from 1945–1964 (the one exception is white women, but the baby boom contains the five year group with the second largest coefficient). The baby boom cohort had higher odds of accidental poisoning death than the cohorts that came before it or after it, as indicated by a higher average coefficient for the baby boomers as compared to the average of the pre- and post-baby boom groups. The odds of accidental poisoning death for baby boomers as compared to all cohorts combined varied substantially across demographic groups, with a ceiling of 2.5. Specifically, the odds were 1.62 higher for white men, 1.09 higher for white women, 2.55 higher for black men, and 1.83 higher for black women (calculated by exponentiating the average of the coefficients for 1945–1964 period).

Table 1 indicates that for whites the odds of accidental poisoning mortality increased dramatically over historical time. Compared to 2005–07 the odds tripled as compared to 1995–99, specifically increasing by a factor of 2.97 for men ($e^{1.57-.483}$) and 3.87 ($e^{1.53-.178}$) for women. This increase since 1995–99 was part of a trend with a longer history, as indicated by period coefficients in Table 1 that grow steadily and consistently larger over time. In comparison to the most recent, 2005–07 period, since 1968–69 the accidental poisoning odds grew by a factor of 9.72 for white men ($e^{1.57-.705}$) and 9.20 for white women ($e^{1.53-.689}$).

Table 1 indicates that the long-term historical trends in accidental poisoning diverge considerably by race, although they are similar in more recent years. In the long term, the odds of accidental poisoning mortality in 2005–07 as compared to 1968–69 increased by a factor of about three (for men $e^{1.11-.0248} = 3.11$, and for women $e^{.904-.145} = 2.85$), which is considerably smaller than the nine-fold increase for whites during the same period. In the short term, trends for blacks and whites are more similar; from the most recent 2005–07 period in comparison to 1995–99 the odds increased by a factor of about two for both black men and women (for black men $e^{1.11-.368} = 2.10$ and for black women $e^{.904-.0914} = 2.25$), an increase close in magnitude to that experienced by whites during this time period.

Table 1 takes into account aging effects. The general pattern is that the association of age with accidental poisoning mortality followed an inverse U shape, in which odds were lowest at the youngest and oldest ages, and highest in the middle ages of this study.

DISCUSSION

This study set out to examine whether the recent increase in accidental poisoning mortality has been driven by the baby boom cohort or a historical period effect. To address this question the analysis includes an age-period-cohort analysis using the ‘intrinsic estimator’ algorithm and draws on U.S. Vital Statistics mortality data and the U.S. Census. To our knowledge this is the only study that examines the relative influence of age, period, and cohort effects on U.S. accidental poisoning mortality, and one of few on the topic of accidental poisoning to use national data.

The bivariate graphs in Figure 1 suggest that the influence of cohort and period effects varies by race. For whites, a historical period effect appears responsible for the recent increase in accidental poisoning mortality because across all age groups the highest rates were in the latest time period, after year 2000. For blacks, a cohort effect seemed at play because the highest rates followed the baby boom cohort as it aged, consistent with the “pig in a python” pattern of findings.

A multivariate age-period-cohort analysis provided more detailed information on these trends. It indicated, as expected, that the baby boom cohort had an elevated rate of death due to accidental poisoning. Both black and white baby boomers had higher odds of accidental poisoning mortality than the cohorts that came before them and after them. Baby boom membership had a moderately larger influence among blacks than it did among whites, and

led to about a doubling of the odds of accidental poisoning death among blacks and a 50% increase among whites.

The analysis indicated that the most substantial racial difference was a long-term, historical increase in accidental poisoning mortality that was substantially stronger for whites than it was for blacks. From the most recent period (2005–07) to the earliest (1968–69) the odds of death due to accidental poisoning increased almost tenfold among whites, while it increased about threefold among blacks. For whites this period effect was much stronger than the baby boom cohort effect, and the period effect dominates the bivariate graph of observed data in Figure 1; for blacks the baby boom effect is apparent in the bivariate graphs because it is not overshadowed by such a strong historical period effect.

It is not surprising that the baby boom cohort had elevated odds for accidental poisoning death in comparison to the cohorts that came before and after it. A high rate of illegal drug use among this cohort is well documented, and it follows that the odds of accidental poisoning should also be high, to the extent that it is a function of drug use prevalence. Baby boom membership has a somewhat higher impact among blacks than among whites, but this difference is much smaller than the race difference in the long-term historical period effects.

It was not anticipated to find such substantial race differences in the overall historical increase in the odds of accidental poisoning mortality. The study results suggest that over the past four decades an as-yet-unspecified factor (or factors) has influenced whites much more than blacks and has substantially increased whites' odds of death due to accidental poisoning. Proper identification of this factor most likely warrants a separate study to rigorously test competing hypotheses, a study that would require more information than is available from the death certificate data used in this study.

The rise in pharmaceutical drug use is a strong candidate to explain the increase in accidental poisoning mortality among whites. From 1991 to 2007 the number of U.S. opiate prescriptions (including hydrocone and oxycodone products) increased more than fourfold from 40 million to 180 million, while stimulant prescriptions increased sevenfold from 5 million to nearly 35 million [21]. In part because of this greater prevalence and availability of pharmaceuticals, nonmedical use of prescription drugs now ranks second behind marijuana among the most common forms of U.S. illicit drug use [22]. In addition, nonmedical use of prescription opioids has grown to such an extent that by the year 2002 they surpassed heroin and cocaine as the most common cause of fatal drug poisoning in the United States [23].

This increase in nonmedical use of prescription drugs has been more acute for whites than it has been for blacks. A substantial body of research indicates that whites are at greater risk for nonmedical use of prescription drugs than blacks [24–26], with the most recent national report indicating that lifetime, nonmedical use of a pain reliever is about 50% higher for whites v. blacks [14.1% v. 9.8%, Table 1. 57B, 27]. One reason for this racial disparity is that blacks are less likely than whites to be prescribed pain relievers by doctors [28], and another reason is a substantial skepticism among blacks about the efficacy and side effects of psychotropic medication [29].

The study has limitations. First, vital statistics data underestimate the true number of deaths that are due to accidental poisoning because some deaths that are a result of accidental poisoning are not recorded as such. To the extent that the underestimation is constant it does not threaten the substantive conclusions of this study, which focuses on trends over time and not the true level of accidental poisoning mortality in any specific year.

A second limitation is that the death classification system changed over the course of the study period, as noted in the Methods section. Fortunately these changes in and of themselves appear to have little effect on the accidental poisoning rate, as evidenced in Figure 1 by the lack of any sudden spike or decrease in the accidental poisoning mortality rate in the years when the coding system changed (i.e., in 1978–79 and in 1998–99).

A third limitation is that the code “accidental poisoning” is not purely a function of substance use and misuse. It is a broad category that in addition to drug overdose also includes unintentional poisonings due to factors such as pesticides and gases. However, in light of the fact that the proportion of poisonings due to factors other than substances is low (<5% in recent years) [1], it is unlikely that they account for substantial increases in accidental poisoning mortality over the study period, such as the nearly tenfold increase among whites.

A final limitation is that the data do not allow us to separate white and Hispanic decedents, who are combined into one group in this analysis. We expect that the nearly tenfold increase in accidental poisoning mortality for whites would have been even higher if Hispanics were separated out from the analysis pool, given evidence that the increase has been smaller for Hispanics than for whites, at least in recent years [2].

CONCLUSION

Accidental poisoning mortality has increased dramatically in the past decade, and this increase is not isolated to the baby boom cohort. In recent years the increase is present among all age and demographic groups, and has been particularly acute among whites. These results point to the need to bolster overdose prevention programs and policies [e.g., 30] as the historical increase in accidental poisoning appears to continue unabated.

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White Males, Age 50-54

White Females, Age 50-54

White Males, age 40-44

White Females, age 40-44

White Males, Age 35-39

White Females, Age 35-39

White Males, Age 20-24

White Females, Age 20-24

Figure 1.
U.S. Mortality Rate Due to Accidental Poisoning for Whites from 1968 to 2007. Hollow Circles Indicate Members of the Baby Boom Cohort.

Black Males, Age 50-54

Black Females, Age 50-54

Black Males, Age 40-44

Black Females, Age 40-44

Black Males, Age 35-39

Black Females, Age 35-39

Black Males, Age 20-24

Black Females, age 20-24

Figure 2.
U.S. Mortality Rate Due to Accidental Poisoning for Blacks from 1968 to 2007. Hollow Circles Indicate Members of the Baby Boom Cohort.

Table 1

Results From Intrinsic Estimator Analysis of Cohort Membership, Year, and Age on Accidental Poisoning Mortality by Demographic Groups, 1968–2007 (95% Confidence Intervals in Parentheses)

	Unexponentiated Coefficients			
	White Men (n=2,685,044,496)	White Women (n=2,711,294,267)	Black Men (n=341,121,011)	Black Women (n=366,536,752)
Birth Cohort				
1905–09	–0.0415 (–0.288 – 0.205)	0.401 (0.212 – 0.59)	–0.582 (–1.51 – 0.344)	0.043 (–0.889 – 0.975)
1910–14	0.0108 (–0.115 – 0.137)	0.232 (0.132 – 0.332)	–0.379 (–0.762 – 0.00338)	0.259 (–0.101 – 0.619)
1915–19	–0.181 (–0.285 – –0.0781)	0.0421 (–0.0376 – 0.122)	–0.224 (–0.543 – 0.0958)	–0.0695 (–0.387 – 0.248)
1920–24	–0.475** (–0.567 – –0.382)	–0.0989 (–0.168 – –0.0297)	–0.449 (–0.701 – –0.197)	–0.00994 (–0.275 – 0.255)
1925–29	–0.436** (–0.511 – –0.362)	–0.133 (–0.194 – –0.0712)	–0.157 (–0.355 – 0.0396)	–0.257 (–0.477 – –0.0366)
1930–34	–0.447** (–0.511 – –0.383)	–0.183** (–0.24 – –0.126)	0.0174 (–0.146 – 0.181)	–0.190 (–0.376 – –0.00463)
1935–39	–0.409** (–0.463 – –0.355)	–0.301** (–0.355 – –0.247)	0.180 (0.0428 – 0.318)	–0.206 (–0.368 – –0.0445)
1940–44	–0.205** (–0.246 – –0.163)	–0.387** (–0.432 – –0.342)	0.575** (0.464 – 0.686)	–0.119 (–0.251 – 0.0139)
1945–49 [†]	0.140** (0.109 – 0.171)	–0.257** (–0.292 – –0.222)	0.891** (0.803 – 0.979)	0.266 (0.163 – 0.369)
1950–54 [†]	0.596** (0.572 – 0.62)	–0.0125 (–0.0423 – 0.0172)	1.20** (1.13 – 1.27)	0.703** (0.622 – 0.784)
1955–59 [†]	0.694** (0.674 – 0.713)	0.286** (0.261 – 0.312)	1.03** (0.972 – 1.08)	0.808** (0.742 – 0.874)
1960–64 [†]	0.502** (0.486 – 0.518)	0.335** (0.311 – 0.358)	0.622** (0.579 – 0.665)	0.650** (0.592 – 0.708)
1965–69	0.218** (0.202 – 0.234)	0.211** (0.186 – 0.236)	0.242** (0.2 – 0.284)	0.386** (0.326 – 0.445)
1970–74	–0.0636** (–0.0817 – –0.0456)	0.0105 (–0.0191 – 0.0401)	–0.097 (–0.148 – –0.0458)	0.0658 (–0.00765 – 0.139)
1975–79	–0.124** (–0.146 – –0.103)	–0.0510 (–0.0857 – –0.0164)	–0.312** (–0.379 – –0.245)	–0.287** (–0.388 – –0.187)
1980–84	0.00965 (–0.0151 – 0.0344)	0.0463 (0.00825 – 0.0843)	–0.606** (–0.694 – –0.518)	–0.557** (–0.687 – –0.427)
1985–89	0.111** (0.0796 – 0.143)	0.0190 (–0.0294 – 0.0674)	–0.936** (–1.06 – –0.813)	–0.515** (–0.68 – –0.351)
1990–92	0.101 (0.0415 – 0.161)	–0.159 (–0.258 – –0.0594)	–1.01** (–1.28 – –0.754)	–0.970* (–1.34 – –0.602)
Period				
1968–69	–0.705** (–0.754 – –0.657)	–0.689** (–0.75 – –0.629)	–0.0248 (–0.125 – 0.0753)	–0.145 (–0.286 – –0.0049)
1970–74	–0.521** (–0.551 – –0.491)	–0.385** (–0.422 – –0.347)	–0.105 (–0.176 – –0.0343)	0.0338 (–0.0558 – 0.123)
1975–79	–0.727** (–0.755 – –0.7)	–0.353** (–0.389 – –0.318)	–0.816** (–0.881 – –0.75)	–0.351** (–0.434 – –0.269)
1980–84	–0.761** (–0.785 – –0.736)	–0.500** (–0.535 – –0.465)	–0.947** (–1 – –0.892)	–0.525** (–0.599 – –0.452)
1985–89	–0.388** (–0.407 – –0.369)	–0.463** (–0.495 – –0.431)	–0.344** (–0.381 – –0.308)	–0.372** (–0.431 – –0.314)
1990–94	–0.0262 (–0.0426 – –0.00973)	–0.312** (–0.341 – –0.284)	0.139** (0.105 – 0.174)	–0.0961 (–0.147 – –0.0448)
1995–99	0.483** (0.467 – 0.499)	0.178** (0.155 – 0.201)	0.368** (0.323 – 0.413)	0.0914 (0.0356 – 0.147)
2000–04	1.08** (1.06 – 1.1)	0.996** (0.977 – 1.02)	0.617** (0.556 – 0.679)	0.461** (0.395 – 0.528)
2005–07	1.57** (1.54 – 1.59)	1.53** (1.51 – 1.55)	1.11** (1.03 – 1.19)	0.904** (0.82 – 0.988)
Age				
15–19	–0.828** (–0.864 – –0.792)	–1.15** (–1.2 – –1.1)	–1.01** (–1.11 – –0.899)	–1.06** (–1.19 – –0.932)

	Unexponentiated Coefficients			
	White Men (n=2,685,044,496)	White Women (n=2,711,294,267)	Black Men (n=341,121,011)	Black Women (n=366,536,752)
20–24	0.249** (0.224 – 0.273)	–0.365** (–0.397 – –0.332)	–0.0752 (–0.151 – 0.00111)	–0.370** (–0.461 – –0.279)
25–29	0.474** (0.455 – 0.494)	–0.0483 (–0.0766 – –0.0199)	0.214** (0.155 – 0.272)	–0.0336 (–0.106 – 0.0392)
30–34	0.529** (0.513 – 0.545)	0.144** (0.118 – 0.169)	0.300** (0.256 – 0.343)	0.231** (0.174 – 0.288)
35–39	0.499** (0.485 – 0.513)	0.333** (0.311 – 0.355)	0.403** (0.372 – 0.434)	0.393** (0.348 – 0.439)
40–44	0.399** (0.385 – 0.413)	0.428** (0.407 – 0.449)	0.347** (0.317 – 0.378)	0.468** (0.423 – 0.512)
45–49	0.156** (0.138 – 0.173)	0.412** (0.389 – 0.435)	0.247** (0.206 – 0.288)	0.375** (0.319 – 0.431)
50–54	–0.206** (–0.229 – –0.183)	0.285** (0.259 – 0.312)	0.0734 (0.0149 – 0.132)	0.264** (0.19 – 0.337)
55–59	–0.544** (–0.574 – –0.514)	0.109** (0.0778 – 0.141)	–0.184 (–0.263 – –0.105)	–0.0638 (–0.162 – 0.0346)
60–64	–0.727** (–0.766 – –0.689)	–0.149** (–0.185 – –0.112)	–0.319** (–0.421 – –0.218)	–0.204 (–0.329 – –0.0791)
Intercept	–10.5** (–10.5 – –10.5)	–11.1** (–11.1 – –11)	–10.1** (–10.2 – –10.0)	–10.8** (–10.9 – –10.7)

[†]These birth cohorts include baby boomers, who were born between 1946 and 1964 [4]

* Indicates “strong evidence” that coefficient differs from zero (a more conservative test than $p < .01$, see Methods section).

** Indicates “very strong evidence” that coefficient differs from zero.