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Biodemography and Social Biology

Publication details, including instructions for authors and subscription information:

<http://www.tandfonline.com/loi/hsbi20>

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Available online: 23 Aug 2010

To cite this article: Isaac W. Eberstein, Charles B. Nam & Kathleen M. Heyman (2008): Causes of death and mortality crossovers by race, *Biodemography and Social Biology*, 54:2, 214-228

To link to this article: <http://dx.doi.org/10.1080/19485565.2008.9989143>

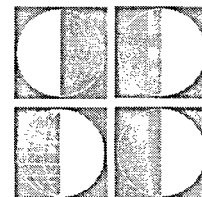
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Causes of Death and Mortality Crossovers by Race[†]



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ABSTRACT: The phenomenon of “mortality crossovers,” the intersection of age curves of mortality at older ages, has been observed in comparisons of various populations for some time. Some researchers have argued that crossovers are an artifact of deficient reporting of age that is greater for some populations than others. Other researchers attribute crossovers to selective processes by age that vary by group. We use mortality data from the National Center for Health Statistics for the U.S. at ages 55 and over, supplemented by comparable data from matched records of the National Health Interview Survey and National Death Index, to reexamine causes of death linked to mortality crossovers for Whites and Blacks in the U.S. Findings portray a more elaborate set of influences of causes of death than has been discovered heretofore; however, the major finding is that the mortality crossover for Whites and Blacks in the U. S. is real and, although observed for several causes of death, operates principally through varying trajectories of heart disease mortality.

The observed pattern of mortality crossovers – mortality rates higher for one population than another at younger ages becoming lower for that population at older ages – has perplexed demographers and health scientists for some time. Various representations of the phenomenon have been presented in the research literature over the years (e.g., Pearl, 1922; Spiegelman, 1948; Myers and Bayo, 1965; Coale and Kisker, 1986; Nam, 1995). Figure 1 is illustrative for White and Black populations in the U.S., based on the data for all causes of death for both sexes used in the present research (discussed below). However, a debate about the explanation for such

crossovers has ensued for a few decades, contestants essentially following one of two orientations. In this paper, we attempt to refine the discussion by focusing further attention on causes of death linked to mortality crossovers.

HISTORICAL BACKGROUND

As early as 1922, Raymond Pearl was intrigued by data showing the mortality crossover by race in the U.S. His initial reaction was that this was not a real phenomenon. On further thought, he surmised that it was consistent with the notion that an improved level of living “has saved for a time the lives of ever more and more babies and young people who formerly could not withstand the unfavorable conditions they met, and died in consequence rather promptly. But just because the process tends to preserve the weaklings, who were speedily eliminated under the rigorous action of unmitigated natural selection, there appear

Revised July 18, 2008.

[†]Revision of paper presented at the annual meeting of the Southern Demographic Association, November 3–5, 2005, Oxford, Mississippi. Portions of this work were supported by NIA grant R03 AG19335.

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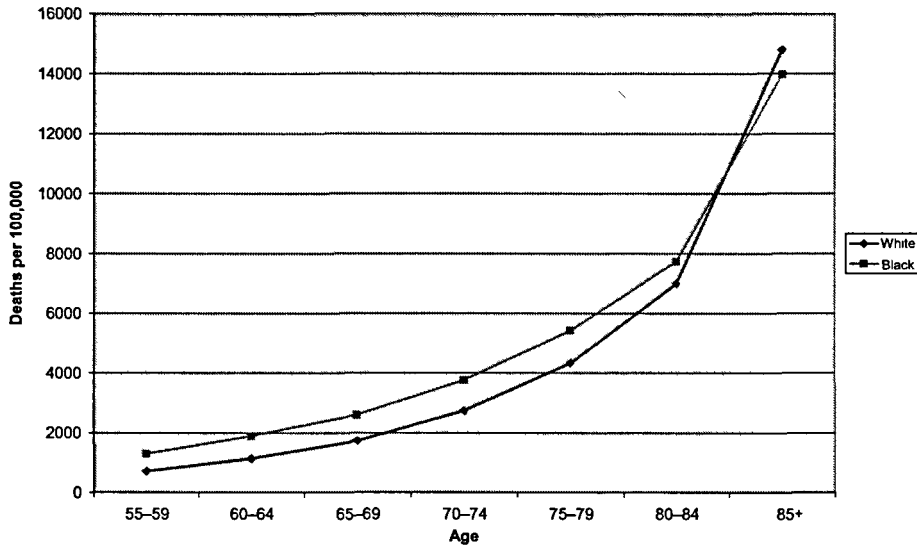


FIG. 1.—Death Rates by Age and Race, US: 2003.

now in the higher age groups of the population many weaker individuals than formerly ever got there.” (Pearl, 1922, p. 23)

Pearl’s idea of a selective process accounting for crossovers got little attention for a long while. At later times analyses identified the crossover and explained it in terms of selective processes (Sibley, 1930; Spiegelman, 1948; Myers and Bayo, 1965; Thornton and Nam, 1968).

Subsequently, when demographers were generally giving substantial attention to accuracy of reported demographic data, various analyses detected crossovers but attributed them to deficient data, especially misreporting of age by less-advantaged groups (Kitagawa and Hauser, 1973; Schoen, 1976; Rosenwaike, 1979). Coale and Kisker (1986) published a whole article on the issue and concluded that age misreporting was the explanation for crossovers.

Several responses to the “deficient data” hypothesis were then published. Manton et al. (1979) and Vaupel et al

(1979) highlighted the heterogeneity of frailty in populations and timing of the selection mechanisms. Manton and Stallard (1984) elaborated this view by stating that survivors represent a subgroup of the starting population who are constitutionally endowed for longer survival, where constitutional endowment is not restricted to genetic determinants of longevity but is taken to include the prenatal environment as well as a variety of life experiences that may influence heterogeneity in longevity.

Weiss (1990) elaborated the Manton and Stallard formulation, focusing on biological processes. Kestenbaum (1992) used the best set of Social Security Administration’s Master Beneficiary Records to demonstrate that crossovers persisted when that dataset was used. Elo and Preston (1994) employed several techniques to gauge the age misreporting of African Americans as a means of reinforcing the “deficient data” hypothesis.

Nam (1995) reviewed the literature on the debate and pointed out that mortality crossovers were special cases of convergence of age-specific mortality rates. Even where crossovers of curves have not taken place, convergence would suggest that the same underlying phenomenon was occurring. He went on to argue that both data misreporting and selectivity were involved in explaining crossovers, but there was a question of which of the two was more critical in the explanation.

Rogers et al. (2000, p. 66) reported convergence across age groups of mortality rates between Black and White populations for the U.S., based on matched records of the National Health Interview Survey and the National Death Index for 1986–1994.

Most recently, attention in the crossover debate has been focused on patterns by cause of death, to see if particular causes account for the phenomenon. During the earlier period of the controversy about mortality crossovers, Nam et al. (1978) used *Cause of Death Life Tables for National Populations* by Preston et al. (1972) to examine the existence of crossovers for a large number of paired national populations at different dates, and to see how causes of death were related to the presence of crossovers. They found that crossovers were observed in only a third of the comparisons, and that group differences in the trend of cardiovascular diseases at the older ages was the principal medical cause of whether or not a crossover took place.

Corti et al (1999) looked at racial mortality crossovers by cause of death for some counties of North Carolina. They found crossover restricted to deaths from coronary heart disease. Hussey and Elo (1997), in a study of age misreporting by cause of death and race, determined that

“mortality crossovers are eliminated for most causes of death when more accurate age data are used.” Importantly, a crossover persisted for ischemic heart disease.

What follows is our analysis of comparisons of trends in age-specific mortality for adult Whites and Blacks in the U.S. by underlying cause-of-death category. Our purpose is to focus on the causes of death that contribute to overall mortality crossover.

DATA AND METHODS

Our analysis is based on two datasets. The principal data are statistics on mortality for 2003 by race, sex, age, and cause of death for persons 55 and over, as reported by the National Center for Health Statistics from vital registration reports (NCHS, 2006). We supplement those data with statistics derived from the matched records of the National Health Interview Survey (NHIS) from 1986–1994 and the National Death Index (NDI) through 1997 for comparable persons (NCHS, 1997, 2005; Rogers et al., 2000) because the latter dataset includes better age reporting.

The principal dataset uses complete death registration for 2003. Reports are derived from resident death certificates, as completed by funeral directors, attending physicians, medical examiners, and coroners. It includes the institutional population. The original records are filed in State registration offices, and statistical information is compiled into a national database through the Vital Statistics Cooperative Program of the Centers for Disease Control and Prevention’s National Center for Health Statistics (NCHS, 2006). Causes of death are processed in accordance with the International Classification of Diseases, Tenth Revision (ICD-10).

The supplementary dataset is derived from the matched records of the National Health Interview Survey 1986–1994 (NHIS) and the National Death Index (NDI) through 1997. The NHIS is an annual household survey of individuals designed to collect information on the health of the resident civilian non-institutional population of the U.S. The survey consists of two parts: (a) a basic health and demographic core questionnaire for each household member, and (b) special topic questionnaires that vary from year to year and are completed for representative subsamples of the population. (For further information about the survey sample, see Rogers et al., 2000.) NCHS links the NHIS survey sample to the annual death records of the NDI through computer matching of the two files using a probabilistic algorithm to determine which records in the NDI match records in the NHIS sample (Further details of the matching procedure are provided in Horm, 1996 and NCHS, 1997.) The NHIS-NDI matched file logically consists of a series of annual cohorts where cumulative mortality is estimated longitudinally as of a common date (1997 in our study), so that each annual NHIS sample has a different length of time since the initial interview (e.g., the 1986 sample has as many as twelve completed years by 1997 but the 1994 sample has four). As a result, when the NHIS-NDI cohorts are considered together, their cumulative mortality experiences may or may not reflect the mortality patterns of the US noninstitutional population if observed at a single point in time (cross-sectionally). For our purposes, jointly considering the annual mortality data from the vital registration system and the longitudinal mortality data from the NHIS-NDI matched files provides a useful perspective on the generality of racial

mortality crossovers. The cause-of-death data in these matched records are based on the International Classification of Diseases, Ninth Revision (ICD-9).

For both datasets, we calculate death rates by age group, race, sex, and cause-of-death category. Race is categorized as Black and White in the death registration data and as non-Hispanic White and non-Hispanic Black in the NHIS-NDI data, thereby giving some perspective on the generality of observed patterns. Age is classified in 5-year age groups (55–59, 60–64, 65–69, 70–74, 75–79, 80–84, and 85 and over) in the death registration analysis. It is classified in 10-year age groups 55 and over in the NHIS-NDI matched dataset because of sample size limitations. Racial differences are examined in the annual mortality data overall and controlling sex. We do not consider sex in the NHIS-NDI analysis because of sample size constraints.

As indicated below, we include some medical conditions that are less frequently considered to be underlying causes of death (e.g., hypertension, diabetes, influenza/pneumonia) but which are nonetheless important associated causes (cf. Eberstein et al., 2005) in order to examine the phenomenon of mortality crossovers more broadly than has heretofore been evident in the literature.

Underlying causes of death are grouped into eleven categories (ICD-10 codes) in the vital registration analysis – malignant neoplasms (C00-C97), diseases of heart (I00-I09, I11, I13, I20-I51), chronic lower respiratory diseases (J40-J47), diabetes mellitus (E10-E14), cerebrovascular diseases (I60-I69), septicemia (A40-A41), nephritis, nephritic syndrome & nephrosis (N00-N07, N17-N19, N25-N27), influenza & pneumonia (J10-J18), essential (primary) hypertension & hypertensive

renal disease (I10, I12), Alzheimer's disease (G30), and all remaining causes of death (residual). They are grouped into eight categories (ICD-9 codes) in the NHIS-NDI analysis – diseases of heart (390–398, 402, 404, 410–429), malignant neoplasms (140–165, 170–175, 179–208), diabetes (250), cerebrovascular diseases (430–438), respiratory disease (490–496), influenza & pneumonia (480–487), hypertension (401, 403), and all other causes (residual).

The vital registration data have strengths and weaknesses. Coverage of deaths is estimated to be 98 percent and the information is gathered by trained personnel. However, the denominators of death rates from this source are based on population estimates for 2003. In addition, reporting of race is slightly different on the death certificates and in census records. Moreover, some age-misreporting on the certificates and population denominators is probably present.

The NHIS-NDI matched dataset also has strengths and weaknesses for our purposes (see Rogers et al., 2000). The principal limitation for our study is that the NHIS sample excludes the institutional population, thereby omitting persons residing in nursing homes and prisons whose death rates are generally higher than for the sample and whose causes of death may differ to some extent. The institutional population makes up 6.5% of persons ages 55–64 and 8.4% of persons ages 75+ (U.S. Bureau of the Census, 2003), but data are not available to estimate the proportion of deaths by age contributed by this group.

The principal strength for our study is that the NHIS data are prospective to the time of death, such that reports of age and race might be expected to be higher in quality than would be afforded by death

certificates. As a consequence, age and race reporting can be regarded as superior in the NHIS-NDI matched dataset, as compared with the death registration/population estimates information. Manton et al. (1991) show that, in well-defined closed cohort populations that produce data on age at death and person-years consistent within the studies, errors in the numerators and denominators of the death rates compensate. The NHIS-NDI matched dataset provides that kind of consistency with regard to age and race.

Statistical significance is assessed for racial contrasts in the national mortality data using procedures recommended by NCHS (2006:115–119). Mortality rates from the NHIS-NDI are estimated using the annual weights from the NHIS, but significance tests are based on the unweighted sample to maintain appropriate statistical power.

FINDINGS

NCHS DEATH REGISTRATION DATA, 2003

Using our principal data, Table 1 presents deaths and death rates by age, sex, and race for eleven underlying cause-of-death categories for the U.S. in 2003. The purpose of our analysis is to examine death rates by race across age groups 55 and over by sex and then see to what extent the paired comparisons differ by cause-of-death category. Figure 1 shows the overall crossover pattern for Whites and Blacks in the U.S., based on our data. This pattern is consistent with earlier literature in that the mortality rate for Blacks is higher in the 55–64 age group, the lines for Whites and Blacks converge at older ages, and a crossover eventually takes place. The only difference is that the lines appear to cross over at a somewhat later age (85+) than in earlier studies (e.g., Thornton and Nam, 1968; Manton

TABLE 1
DEATH RATES PER 100,000 FOR UNDERLYING CAUSES OF DEATH (ICD-10) BY AGE, SEX, RACE: US, 2003^{a,b}

	AGE						
	55-59	60-64	65-69	70-74	75-79	80-84	85+
WHITE – Both Sexes^c							
All Causes of Death	710.6	1127.8	1739.2	2728.6	4329.5	6992.1	14792.3
Malignant Neoplasms	261.2	434.5	644.8	908.5	1195.1	1450.7	1701.4
Diseases of Heart	170.9	275.7	432.1	706.5	1199.6	2153.0	5363.5
Respiratory	28.0	67.3	129.5	223.2	347.2	478.9	664.5
Diabetes Mellitus	26.4	42.7	66.5	98.1	143.1	202.7	300.8
Cerebrovascular	22.6	38.3	70.8	140.3	283.2	565.9	1382.2
Septicemia	8.8	14.3	21.7	36.9	63.7	102.5	191.1
Nephritis	7.8	14.1	25.2	43.5	77.6	136.0	284.8
Influenza & Pneumonia	7.4	14.0	25.2	47.9	102.4	218.4	679.3
Hypertension & Renal	3.3	5.8	9.8	16.6	33.0	64.1	181.0
Alzheimer's disease	–	3.6	10.3	34.5	101.8	264.4	834.0
All other (residual)	174.2	217.5	303.3	472.6	782.8	1355.5	3209.7
<i>Number of Deaths</i>	<i>95720</i>	<i>11701</i>	<i>145645</i>	<i>204904</i>	<i>286819</i>	<i>340633</i>	<i>629695</i>
BLACK – Both Sexes^c							
All Causes of Death	1295.1	1889.9	2595.8	3754.9	5421.2	7745.3	13976.7
Malignant Neoplasms	382.6	589.1	802.7	1080.1	1385.5	1640.8	1853.1
Diseases of Heart	348.4	533.8	752.7	1115.2	1638.3	2445.2	4941.2
Respiratory	31.3	52.7	86.5	146.7	211.9	271.9	353.0
Diabetes Mellitus	69.8	106.9	156.1	221.2	300.0	389.9	527.5
Cerebrovascular	72.4	113.1	157.4	256.3	433.2	677.0	1348.7
Septicemia	25.3	41.5	61.0	88.0	133.4	193.4	372.0
Nephritis	34.3	54.2	83.8	127.8	171.0	260.7	440.5
Influenza & Pneumonia	15.7	27.1	41.6	70.1	122.5	205.6	525.4
Hypertension & Renal	20.5	29.0	42.9	68.9	102.1	145.4	301.1
Alzheimer's disease	–	–	–	30.2	91.4	207.9	583.8
All other (residual)	294.8	342.5	411.1	550.4	831.9	1307.5	2730.4
<i>Number of Deaths</i>	<i>20458</i>	<i>22365</i>	<i>24747</i>	<i>28381</i>	<i>31859</i>	<i>30513</i>	<i>47908</i>
WHITE – Males^d							
All Causes of Death	884.2	1390.6	2146.2	3359.5	5349.4	8522.6	16037.9
Malignant Neoplasms	285.4	494.7	763.2	1108.1	1518.9	1930.1	2412.1
Diseases of Heart	251.9	394.1	599.0	949.5	1575.7	2710.0	5747.2
Respiratory	29.4	71.5	139.3	252.4	406.9	585.6	894.4
Diabetes Mellitus	31.6	51.5	79.1	115.3	170.2	241.1	333.4
Cerebrovascular	24.7	44.5	81.8	158.5	310.0	590.4	1247.0
Septicemia	9.3	15.3	24.6	40.8	71.3	116.7	203.3
Nephritis	9.2	15.5	29.1	50.5	96.9	179.7	383.6
Influenza & Pneumonia	8.5	15.7	30.8	59.2	127.2	271.4	767.1
Hypertension & Renal	4.1	6.6	11.6	–	–	60.3	145.3
Alzheimer's disease	–	–	10.3	33.1	94.9	238.5	630.3
All other (residual)	230.1	281.2	377.4	592.1	977.4	1598.8	3274.2
<i>Number of Deaths</i>	<i>58348</i>	<i>69404</i>	<i>84313</i>	<i>113540</i>	<i>148708</i>	<i>158342</i>	<i>209575</i>
BLACK – Males^d							
All Causes of Death	1688.7	2454.5	3334.8	4843.9	6978.2	9827.8	14903.4
Malignant Neoplasms	464.8	747.3	1045.9	1455.8	1975.5	2417.1	2825.5
Diseases of Heart	481.2	718.0	997.2	1460.0	2083.2	2981.9	4850.3
Respiratory	38.0	67.7	112.0	212.5	326.8	471.8	601.6
Diabetes Mellitus	77.3	121.7	170.6	232.6	306.3	387.6	453.5
Cerebrovascular	90.6	141.7	184.9	307.4	503.0	727.9	1180.3

(Continued)

TABLE 1
(Continued)

	Age						
	55-59	60-64	65-69	70-74	75-79	80-84	85+
Septicemia	31.8	46.1	74.9	100.2	160.2	235.5	404.5
Nephritis	43.3	62.7	94.1	155.7	191.7	333.7	518.2
Influenza & Pneumonia	21.3	39.7	61.6	100.5	177.6	285.0	641.2
Hypertension & Renal	24.3	36.2	50.0	74.6	102.2	147.7	274.2
Alzheimer's disease	-	-	-	32.5	93.1	206.0	459.8
All other (residual)	416.1	473.4	543.6	712.1	1058.6	1633.6	2694.3
<i>Number of Deaths</i>	<i>12040</i>	<i>12730</i>	<i>13544</i>	<i>14747</i>	<i>15288</i>	<i>13312</i>	<i>14294</i>
WHITE - Females^c							
All Causes of Death	543.9	884.5	1379.6	2212.3	3592.1	6048.6	14240.6
Malignant Neoplasms	237.8	378.9	540.3	745.2	961.0	1155.2	1386.5
Diseases of Heart	93.1	166.1	284.6	507.6	927.7	1809.7	5193.6
Respiratory	26.7	63.5	120.8	199.3	304.0	413.2	562.8
Diabetes Mellitus	21.4	34.5	55.4	84.0	123.5	179.1	286.3
Cerebrovascular	20.5	32.5	61.1	125.4	263.8	550.7	1442.1
Septicemia	8.2	13.4	19.2	33.7	58.2	93.7	185.8
Nephritis	6.5	12.8	21.8	37.7	63.6	109.1	241.0
Influenza & Pneumonia	6.3	12.4	20.2	38.6	84.5	185.7	640.4
Hypertension & Renal	2.4	5.0	8.2	15.5	33.2	66.5	196.8
Alzheimer's disease	-	4.2	10.3	35.7	106.8	280.3	924.3
All other (residual)	121.0	161.2	237.7	389.6	665.8	1205.4	3181.0
<i>Number of Deaths</i>	<i>37372</i>	<i>47697</i>	<i>61332</i>	<i>91364</i>	<i>138111</i>	<i>182291</i>	<i>420120</i>
BLACK - Females^c							
All Causes of Death	971.3	1449.4	2047.3	3020.5	4495.8	6654.1	13616.7
Malignant Neoplasms	314.9	465.7	622.1	826.8	1034.8	1234.0	1475.3
Diseases of Heart	239.2	390.1	571.3	882.6	1373.9	2164.0	4976.5
Respiratory	25.7	41.1	67.6	102.4	143.5	167.1	256.4
Diabetes Mellitus	63.6	95.4	145.3	213.6	296.3	391.1	556.2
Cerebrovascular	57.3	90.7	137.1	221.8	391.8	650.3	1414.2
Septicemia	20.0	37.9	50.8	79.8	117.5	171.4	359.3
Nephritis	26.9	47.5	76.2	109.0	158.7	222.4	410.4
Influenza & Pneumonia	11.1	17.3	26.9	49.6	89.8	164.0	480.4
Hypertension & Renal	17.4	23.3	37.6	65.1	102.0	144.3	311.5
Alzheimer's disease	-	-	10.1	28.6	90.3	208.9	631.9
All other (residual)	195.2	240.4	302.3	441.2	697.2	1136.6	2744.6
<i>Number of Deaths</i>	<i>8418</i>	<i>9635</i>	<i>11203</i>	<i>13634</i>	<i>16571</i>	<i>17201</i>	<i>33614</i>

Notes: ^aDownloaded 4/6/06 from http://www.cdc.gov/nchs/data/dvs/lcwk2_2003.pdf.^bSee text for ICD-10 codes for each cause of death category.^cAll racial comparisons are statistically significant at $p < .001$, except for the following: $p < .05$: Respiratory at ages 55-59, Alzheimer's at ages 70-74 and 75-79, and residual causes at ages 80-84. $p > .05$ (ns): Cerebrovascular at ages 85+ and Influenza & Pneumonia at ages 80-84.^dAll racial comparisons are statistically significant at $p < .001$, except for the following: $p < .05$: Alzheimer's at ages 65-69, 70-74 and 80-84. $p > .05$ (ns): Respiratory at ages 60-64, Cerebrovascular at ages 85+, Influenza & Pneumonia at ages 80-84, and Alzheimer's at ages 75-79.^eAll racial comparisons are statistically significant at $p < .001$, except for the following: $p < .01$: Influenza & Pneumonia at ages 60-64, 70-74, and 80-84, Alzheimer's at ages 70-74 and 75-79, and residual causes at ages 80-84. $p < .05$: Influenza & Pneumonia at ages 55-59, Alzheimer's at ages 65-69, and residual causes at ages 75-79. $p > .05$ (ns): Respiratory at ages 50-59, Cerebrovascular at ages 85+, and Influenza & Pneumonia at ages 75-79.

and Stallard, 1984), probably a reflection of convergence of health conditions among the groups over time (Nam, 1995).

Because cardiovascular diseases were observed in two earlier studies to be

principally responsible for the crossover effect, we focus attention first on the findings for diseases of the heart (Table 1 and Figure 2). Consistent with the findings of Nam et al. (1978) and Corti et al. (1999),

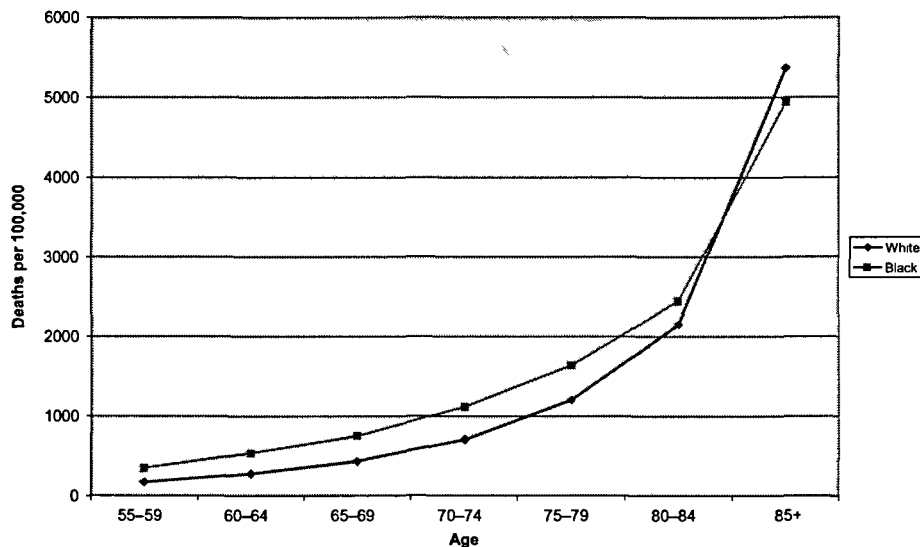


FIG 2—Death Rates from Heart Disease by Age and Race, US: 2003

the crossover pattern of heart diseases mirrors that for all causes combined.

However, Table 1 shows that mortality crossovers are also seen for other cause categories. For cerebrovascular diseases, influenza and pneumonia, and residual causes, crossovers take place but at an earlier age (around the 75–84 age group). In the case of chronic lower respiratory diseases, a crossover may have taken place before age 55 since the data reveal diverging curves of mortality (Whites having higher rates) after that age.

Most interesting and particularly important for the two interpretations that have been suggested for the crossover pattern (age misreporting and selective processes), no crossovers are observed for the remaining cause categories. For malignant neoplasms (Table 1 and Figure 3), the lines for Whites and Blacks do not cross, although there is some slight convergence by ages 85+. Neither crossover nor convergence appears for diabetes mellitus

(Table 1 and Figure 4), and for septicemia, nephritis, and hypertension and renal diseases. For each of these causes, the death rates for Blacks remains consistently high at all ages, perhaps reflecting known morbidity patterns for Blacks. The consistent disparity in mortality rates for respiratory diseases is noteworthy because Whites maintain a higher rate in all age categories.

For all of the above comparisons, consistent Black-White differences appear for both males and females within each cause-of-death category. That is, racial mortality crossovers are not due to differences between Whites and Blacks in changing sex composition by age. Because the conclusions are parallel, we do not discuss sex-specific rates.

Another way of looking at the racial mortality differential is evident in Table 2, where disparities at each age are separated into parts reflecting the separate contribution of deaths from the medical conditions. The racial differential in number

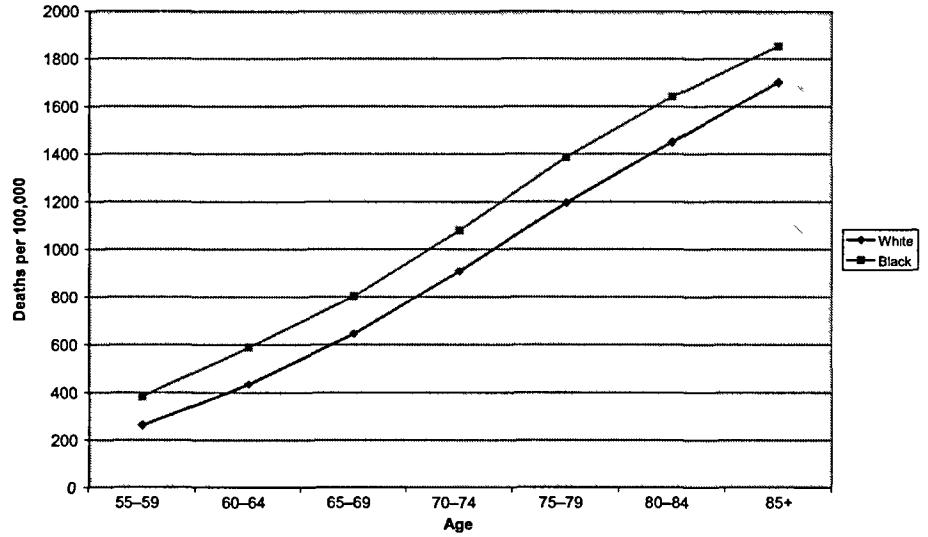


FIG 3 —Death Rates from Malignant Neoplasms by Age and Race, US: 2003

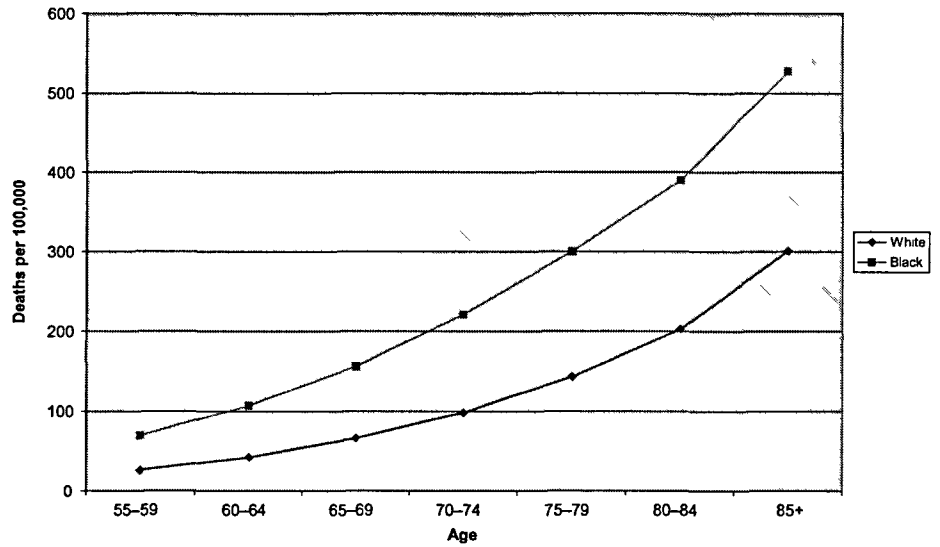


FIG. 4 —Death Rates from Diabetes Mellitus by Age and Race, US: 2003.

of deaths per 100,000 person years (Black-White) was computed for both sexes, males, and females from the data in Table 1. The algebraic percent of this differential associated with each cause of

death is shown for each age in Table 2. Negative percentages refer to racial differentials for specific causes that are opposite the differential of the age group overall. Not surprisingly, deaths from heart

TABLE 2
DECOMPOSITION OF THE BLACK-WHITE MORTALITY DIFFERENTIAL BY AGE INTO UNDERLYING
CAUSES OF DEATH, BY SEX. (PERCENT)^a

	AGE						
	55-59	60-64	65-69	70-74	75-79	80-84	85+
Both Sexes							
All Causes of Death	100.0	100.0	100.0	100.0	100.0	100.0	100.0
Malignant Neoplasms	20.8	20.3	18.4	16.7	17.4	25.2	-18.6
Diseases of Heart	30.4	33.9	37.4	39.8	40.2	38.8	51.8
Respiratory	0.6	-1.9	-5.0	-7.5	-12.4	-27.5	38.2
Diabetes Mellitus	7.4	8.4	10.5	12.0	14.4	24.9	-27.8
Cerebrovascular	8.5	9.8	10.1	11.3	13.7	14.8	4.1
Septicemia	2.8	3.6	4.6	5.0	6.4	12.1	-22.2
Nephritis	4.5	5.3	6.8	8.2	8.6	16.6	-19.1
Influenza & Pneumonia	1.4	1.7	1.9	2.2	1.8	-1.7	18.9
Hypertension & Renal	2.9	3.0	3.9	5.1	6.3	10.8	-14.7
Alzheimer's disease	0.0	-0.5	-1.2	-0.4	-1.0	-7.5	30.7
All other (residual)	20.6	16.4	12.6	7.6	4.5	-6.4	58.8
Males							
All Causes of Death	100.0	100.0	100.0	100.0	100.0	100.0	100.0
Malignant Neoplasms	22.3	23.7	23.8	23.4	28.0	37.3	-36.4
Diseases of Heart	28.5	30.4	33.5	34.4	31.2	20.8	79.1
Respiratory	1.1	-0.4	-2.3	-2.7	-4.9	-8.7	25.8
Diabetes Mellitus	5.7	6.6	7.7	7.9	8.4	11.2	-10.6
Cerebrovascular	8.2	9.1	8.7	10.0	11.8	10.5	5.9
Septicemia	2.8	2.9	4.2	4.0	5.5	9.1	-17.7
Nephritis	4.2	4.4	5.5	7.1	5.8	11.8	-11.9
Influenza & Pneumonia	1.6	2.3	2.6	2.8	3.1	1.0	11.1
Hypertension & Renal	2.5	2.8	3.2	5.0	6.3	6.7	-11.4
Alzheimer's disease	0.0	0.0	-0.9	0.0	-0.1	-2.5	15.0
All other (residual)	23.1	18.1	14.0	8.1	5.0	2.7	51.1
Females							
All Causes of Death	100.0	100.0	100.0	100.0	100.0	100.0	100.0
Malignant Neoplasms	18.0	15.4	12.3	10.1	8.2	13.0	-14.2
Diseases of Heart	34.2	39.7	42.9	46.4	49.4	58.5	34.8
Respiratory	-0.2	-4.0	-8.0	-12.0	-17.8	-40.6	49.1
Diabetes Mellitus	9.9	10.8	13.5	16.0	19.1	35.0	-43.3
Cerebrovascular	8.6	10.3	11.4	11.9	14.2	16.4	4.5
Septicemia	2.8	4.3	4.7	5.7	6.6	12.8	-27.8
Nephritis	4.8	6.1	8.1	8.8	10.5	18.7	-27.2
Influenza & Pneumonia	1.1	0.9	1.0	1.4	0.6	-3.6	25.6
Hypertension & Renal	3.5	3.2	4.4	6.1	7.6	12.8	-18.4
Alzheimer's disease	0.0	-0.7	0.0	-0.9	-1.8	-11.8	46.9
All other (residual)	17.4	14.0	9.7	6.4	3.5	-11.4	69.9

Notes: ^aComputed from Table 1. Figures are Black mortality rates minus White rates, by cause of death, for each sex and age, expressed as a percent of the total racial differential (all causes of death) for each age.

disease consistently make up the largest proportion of total racial disparities in all age groups. Conditions less frequently cited as underlying causes of death, like influenza and pneumonia, are not very

important for overall racial inequalities. Respiratory disease mortality, where Whites have higher mortality than Blacks, contributes to a reduction in aggregate racial differentials except at the oldest

ages, where total mortality rates have crossed and White mortality is higher than for Blacks. In contrast, diabetes, where Black mortality is highest, generally contributes to the widening of racial differences.

NHIS-NDI MATCHED RECORDS DATA, 1986–1997

The same type of analysis was performed using the sample survey data from longitudinal matched records of the NHIS 1986–1994 and NDI through 1997. These data are subject to sampling variability not found in the registration data, omit the institutional population, and are derived from respondent reports on the survey. Further, only non-Hispanic Whites and non-Hispanic Blacks are compared in the analysis. The results do, nevertheless, provide an alternative source for assessing the cause of death contributions to racial mortality crossover effects, and one with superior age reporting.

Findings from this source shown in Table 3 are only summarized in order to conserve space. It is clear that the results from the NHIS-NDI matched data are consistent with the results from the registration data. We find racial mortality crossovers by cause occurring in generally the same manner in both sources. Although the curves from the NHIS-NDI dataset are less stable, due to sampling variability, the effects are parallel.

Table 4 shows for which causes of death mortality crossovers appear, comparing data from four datasets – Nam, Weatherby, and Ockay; Hussey and Elo, NCHS analysis presented here, and NHIS-NDI analysis given here. Because some cause-of-death categories are not shown for some of the sources, we look for consistent and inconsistent patterns of

crossover among those sources for which the causes are presented.

Crossovers are lacking for malignant neoplasms (cancers) in all four sources, and for diabetes, septicemia, nephritis, hypertension, Alzheimer's, and diarrhea, where only one source designates the cause category, and for certain degenerative diseases where two sources report the category.

Crossovers are observed for diseases of the heart for three sources; however, the fourth source (Hussey and Elo) reports a crossover for ischemic heart disease, which is not specified in the other sources. Since ischemic heart disease accounts for over two-thirds of all deaths due to diseases of the heart, the findings for diseases of the heart appear to be generally consistent for the four sources.

Crossovers appear in both the NCHS and NHIS-NDI data for cerebrovascular disease (stroke) and influenza/pneumonia, both of which were not specified in the Nam, Weatherby, Ockay and Hussey-Elo data. They are found for residual causes in all but the Hussey-Elo statistics.

DISCUSSION

As seen in earlier studies, the pattern of mortality rates for Whites and Blacks in the U.S. by age in our data reveals a mortality crossover at older ages. This finding is based on death rates by race, sex, and age, as calculated from published death registration/population estimates information and from matched records of the NHIS sample survey and NDI. The latter data contain prospective reports of age and race so that, although the data are not corrected for possible age misreporting, this problem would be expected to be a minimum.

TABLE 3
DEATH RATES PER 100,000 FOR UNDERLYING CAUSES OF DEATH (ICD-9) BY RACE AND AGE, NHIS-NDI LINKED
MORTALITY FILE: 1986-1997^a

UNDERLYING CAUSE	55-64		65-74		75-84		85+	
	RATE	N DEATHS	RATE	N DEATHS	RATE	N DEATHS	RATE	N DEATHS
<i>Heart</i>								
White, Non-Hispanic	630.06	1840	848.73	4048	1605.34	4754	2132.22	2082
Black, Non-Hispanic	1042.00	511	1013.76	719	1715.89	658	1747.28	220
Z		8.51***		4.11***		1.56 ^{ns}		-3.04***
<i>Cancer</i>								
White, Non-Hispanic	911.53	2662	792.33	3779	812.13	2405	589.89	576
Black, Non-Hispanic	1101.13	540	834.70	592	912.71	350	587.72	74
Z		3.75***		1.16 ^{ns}		1.95*		-0.03 ^{ns}
<i>Stroke</i>								
White, Non-Hispanic	90.40	264	156.41	746	335.32	993	450.61	440
Black, Non-Hispanic	161.09	79	194.57	138	310.32	119	405.05	51
Z		3.73***		2.18*		-0.82 ^{ns}		-0.75 ^{ns}
<i>Respiratory</i>								
White, Non-Hispanic	124.30	363	159.56	761	204.97	607	132.11	129
Black, Non-Hispanic	97.88	48	101.52	72	132.99	51	95.31	12
Z		-1.70 ^{ns}		-4.37***		-3.53***		-1.23 ^{ns}
<i>Influenza/Pneumonia</i>								
White, Non-Hispanic	27.39	80	71.29	340	168.50	499	301.09	294
Black, Non-Hispanic	53.02	26	81.78	58	153.86	59	198.55	25
Z		2.36*		0.92 ^{ns}		-0.68 ^{ns}		-2.36*
<i>Diabetes</i>								
White, Non-Hispanic	64.03	187	69.19	330	71.93	213	57.35	56
Black, Non-Hispanic	171.29	84	128.31	91	143.43	55	127.07	16
Z		5.57***		4.23***		3.58***		2.13*
<i>Hypertension</i>								
White, Non-Hispanic	8.22	24	11.53	55	21.61	64	32.77	32
Black, Non-Hispanic	12.23	6	21.15	15	70.41	27	63.54	8
Z		0.76 ^{ns}		1.69 ^{ns}		3.53***		1.33 ^{ns}
<i>Residual</i>								
White, Non-Hispanic	433.85	1267	532.97	2542	947.54	2806	1250.45	1221
Black, Non-Hispanic	668.83	328	585.13	415	944.00	362	1151.62	145
Z		6.04***		1.70 ^{ns}		-0.06 ^{ns}		-0.97 ^{ns}
<i>Total</i>								
White, Non-Hispanic	2289.79	6687	2642.02	12601	4167.34	12341	4946.52	4830
Black, Non-Hispanic	3307.47	1622	2960.92	2100	4383.60	1681	4376.14	551
Z		11.73***		4.64***		1.91 ^{ns}		-2.86**

^aSee text for description of study population and ICD-9 codes. Rates are based on weighted deaths and significance tests on unweighted deaths. Symbols: ***p < .001, **p < .01, *p < .05, ^{ns}p > .05.

Age trends of mortality rates for Whites and Blacks in the U.S. vary considerably by underlying cause-of-death category. Crossovers are observed for heart diseases, cerebrovascular diseases, influenza/pneumonia, and other (residual) causes of death. There is some convergence but not crossover for cancers. No

TABLE 4
COMPARISON OF MORTALITY CROSSOVERS BY CAUSE OF DEATH FROM FOUR SOURCES

CAUSE	N/W/O	H/E	NCHS	NHIS-NDI
Malignant neoplasms	NC	NC	NC	NC
Diseases of heart	C	NC	C	C
Ischemic heart disease		C		
Other cardiovascular		NC		
Respiratory diseases			NC	NC
Diabetes			NC	
Cerebrovascular diseases		NC	C	C
Septicemia			NC	
Nephritis			NC	
Influenza & pneumonia	NC		C	C
Hypertension			NC	
Alzheimer's			NC	
Certain degenerative diseases	NC		NC	
Diarrhea	NC			
All other causes	C	NC	C	C

LEGEND:
N/W/O Nam, Weatherby, Ockay.
H/E Hussey, Elo.
NCHS National Center for Health Statistics.
NHIS-NDI Survey-death match.
C—crossover
NC—no crossover.
blank—not available.

crossover is seen for diabetes, septicemia, nephritis, and hypertension and renal diseases. Blacks maintain a higher mortality rate at all ages when causes of death are diabetes, septicemia, nephritis, or hypertension/renal, while Whites maintain a higher mortality rate at all ages when respiratory diseases are specified.

Although age misreporting probably contributes to some of the discrepancy in age trends of mortality rates for Whites and Blacks (less so based on the NHIS-NDI dataset), it seems unlikely that it can be a major contributor to mortality crossovers. That would necessitate an age-misreporting pattern that varied by cause of death in a peculiar manner.

The same pattern of racial differentials is seen for both males and females across the causes of death. Similarly, parallel findings are evident for all Blacks and Whites in the vital registration data and when attention is limited to non-Hispanic

Whites and non-Hispanic Blacks in the NHIS-NDI. In each case, diseases of the heart seem to account principally for the overall mortality crossover.

Convergence, crossover, and non-crossover patterns by cause of death suggest that the principal contributor to these patterns are selective processes whereby survival to older age is predicated on the relative health of groups indicated by the incidence of certain medical conditions and the physiological ability of those groups to cope with those conditions. The central mechanism for the patterns seems to be heterogeneity in frailty, or susceptibility to the risk of death, among individuals, along with variation in the consequent distributions of more or less frail individuals across groups who have survived to given points in the life course. Various factors have been considered to account for heterogeneity in frailty, and from Pearl's early reference to "weaklings"

(1922), there has been a biological dimension to discussions of the concept (e.g., Manton and Stallard, 1984; Weiss, 1990).

However, other factors are important for differential adult mortality risks by medical cause of death, including intrinsic health status, early life conditions, health risk behaviors, and social/environmental health insults or protective mechanisms (e.g., social support). Elo and Preston (1992) described various pathways through which childhood disease environments might affect adult mortality, but, although later research has demonstrated that mortality risks across the life course are positively correlated, the underlying mechanisms remain ambiguous (Hayward and Gorman, 2004; Preston et al., 1998). Crimmins (2005) describes how differential mortality selection by socioeconomic status leads to the convergence and even crossover of mortality rates across status groups at older ages for medical conditions associated with the risk of death. Dupre et al. (2006) consider racial mortality crossovers from the point of view of religious attendance, which is interpreted as an important source of heterogeneity in frailty even apart from

direct measures of important risk and protective behaviors. Manton and Stallard (1997) and Johnson (2000) show how crossovers are reflected in morbidity data at prior ages as well as in mortality data at later ages. Corti et al. (1999) discuss differential access and effectiveness of health care across racial groups as possibly having an important role in mortality selection and crossover. Others are considering more biological mechanisms (e.g., Ferrucci et al., 2005; Crimmins and Seeman, 2004), although in ways far removed from the negative overtones of Pearl's early formulation. Finally, Liu et al (2008) provide a mathematical basis for observing mortality crossovers in the context of long term trends in mortality convergence at older ages, a pattern consistent with multiple factors. Clearly, further research is necessary to integrate the wide range of demographic, socioeconomic, behavioral, and biomedical variables affecting mortality risks into a larger and more comprehensive model that describes the process of selective mortality and more particularly mortality crossovers across important social groups.

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