

SEX MORTALITY DIFFERENCES IN THE UNITED STATES: THE ROLE OF COHORT SMOKING PATTERNS*

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This article demonstrates that over the period 1948–2003, sex differences in mortality in the age range 50–84 widened and then narrowed on a cohort basis rather than on a period basis. The cohort with the maximum excess of male mortality was born shortly after the turn of the century. Three separate data sources suggest that the turnaround in sex mortality differences is consistent with sex differences in cigarette smoking by cohort. An age-period-cohort model reveals a highly significant effect of smoking histories on men's and women's mortality. Combined with recent changes in smoking patterns, the model suggests that sex differences in mortality will narrow dramatically in coming decades.

ife expectancy for females in the United States has exceeded that of males whenever the mortality of the sexes has been compared (e.g., National Center for Health Statistics 2004). However, longevity differences in recent years have been narrowing. Female life expectancy at birth exceeded that of males by 7.7–7.8 years from 1972 to 1979, but by 2003, the difference had declined to only 5.3 years (National Center for Health Statistics 2004, 2005). The change in the trend of sex mortality differences has created major uncertainties for extrapolative mortality projections that are used to predict the fiscal burdens of an aging population (Wilmoth 2005).

Narrowing sex differences in mortality have also been observed in most European countries (e.g., Gjonca et al. 2005). The most commonly invoked explanation of the reduced differences is the different histories of cigarette smoking for men and women (Bongaarts 2006; Gjonca et al. 2005; Janssen et al. 2005; Pampel 2002; Valkonen and van Poppel 1997). In all countries where data exist, women's uptake of smoking has lagged behind that of men (Pampel 2002). Cigarette smoking was also implicated in earlier years when sex differences were widening rather than narrowing (Preston 1970; Retherford 1975). Smoking patterns are an obvious place to look for an explanation of sex mortality differences because the health risks of smoking are high and long-lasting, because large fractions of the population have engaged in the habit, and because smoking patterns have differed between the sexes (Waldron 1986). Although the health risks of cigarette smoking have been observed in large epidemiologic studies for a half-century, more recent studies using better research designs and more careful measurement have raised the estimated relative risk of death from current and past smoking (Rogers et al. 2005; Taylor et al. 2002; Thun et al. 1998).

In this article, we demonstrate that changes in sex mortality differences in the United States have been structured on a cohort basis rather than a period basis, a feature that has previously escaped attention. Furthermore, we show that the cohort imprint is closely

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related to a cohort's history of cigarette smoking. Rather than attempting to extrapolate from epidemiologic studies to the national level, as previous studies have done, we achieve these results through a difference-of-differences design that directly reveals the impact of smoking on mortality. The different smoking histories of women and men provide a telling vantage point from which to view the havoc that smoking has wrought on national mortality patterns.

DATA

For each sex, we reconstruct age-specific death rates from ages 50-54 to 80-84 for every fifth calendar year from 1948 to 2003. Using five-year age groups every fifth calendar year enables us to uniquely identify birth cohorts as they pass through life. Numerators of death rates are drawn from official vital statistics sources; denominators are drawn from U.S. census sources.¹

SEX DIFFERENCES IN RATES OF MORTALITY CHANGE

We begin by presenting the rates of mortality change for men and women separately. These changes reflect myriad factors, among which improvements in medical technology have probably played the most important role during the period under review (Cutler 2004; Tunstall-Pedoe et al. 2000). These improvements were deployed and diffused on a period-specific basis, probably accounting for the fact that demographers have noted a preponderance of period-specific influences on adult mortality during the period (Kannisto 1994; National Research Council 2000:149).

Table 1 shows the proportionate rates of change in men's mortality during five-year intervals. Rates of decline slower than the median value of -0.0658 are shaded. Clearly, the period 1948–1968 was one of relatively slow improvement, whereas the period since 1968 has shown persistently faster improvements at all ages except 80–84. Table 2 presents comparable data for women. The pattern is again organized primarily by rows (periods), but the periodicity is somewhat different. Like men's mortality, women's mortality improved relatively quickly from 1968 to 1978. Unlike men's, however, women's mortality improvement was unusually slow between 1978 and 1993 and rapid during 1948–1958.

When men's and women's rates of change are compared, a radically different pattern emerges. Table 3 presents the difference between rates of mortality change for men and women. When men's mortality rises relative to women's mortality (i.e., the difference between the rates of change for men and women is positive), the value is shaded. Clearly, the sex difference in rates of mortality change is organized diagonally. Above the diagonal line that is drawn on Table 3, all values are positive: men's mortality is increasing relative to women's within a five-year age-time block. Below the diagonal line, on the other hand, 38 of the 42 values are negative.

Thus, the pattern of change in sex difference is tightly structured on a cohort basis. Relative to women, mortality grew worse for men through the cohort aged 40–44 in 1948. This cohort was born between 1903 and 1908. Sex differences in mortality began to narrow between this cohort and the cohort born in 1908–1913, and they continued to narrow from one cohort to the next all the way through the cohort born in 1948–1953. Taking a difference-of-differences approach removes the influence of period-specific factors that are common to both sexes and permits a striking cohort pattern to become visible.

^{1.} The numbers of deaths by age and sex are obtained from *Vital Statistics of the United States* for calendar years 1948, 1953, 1958, 1963, 1968, 1973, and 1978. Death rates from 1983 to 1998 are obtained online from the Web site of the National Center for Health Statistics, Centers for Disease Control and Prevention (www.cdc. gov/nchs). Unpublished death data for 2003 were supplied by the National Center for Health Statistics. The population at risk, by age and sex, between 1948 and 1978 is obtained from the U.S. Census Bureau, *Current Population Reports*, Series P-25, No. 311, No. 314, No. 519, and No. 870, and Series P-20, No. 441. Population estimates in 2003 are taken from the Web site of the U.S. Census Bureau (www.census.gov).

				Age Interval			
Period	50-54	55–59	60–64	65–69	70–74	75–79	80-84
1953–1948	-0.0715	-0.0606	-0.0232	-0.0005	-0.0338	-0.0625	0.0353
1958–1953	-0.0456	-0.0520	-0.0176	0.0091	-0.0052	-0.0131	-0.0195
1963–1958	-0.0071	0.0314	0.0010	0.0576	0.0421	0.0047	-0.0176
1968–1963	-0.0061	0.0064	0.0338	-0.0276	0.0645	0.0048	-0.0658
1973–1968	-0.0961	-0.0698	-0.0758	-0.0735	-0.0888	0.0184	0.0137
1978–1973	-0.1107	-0.1624	-0.1054	-0.1257	-0.1074	-0.0848	-0.0525
1983–1978	-0.1154	-0.0672	-0.1225	-0.0693	-0.0676	-0.0980	-0.0620
1988–1983	-0.0856	-0.0827	-0.0534	-0.0646	-0.0589	-0.0518	-0.0213
1993-1988	-0.0865	-0.1005	-0.0940	-0.0745	-0.1032	-0.0833	-0.0631
1998–1993	-0.1242	-0.1262	-0.1171	-0.1063	-0.0691	-0.0833	-0.0692
2003-1998	0.0339	-0.0610	-0.0926	-0.1067	-0.1064	-0.0704	-0.0850

 Table 1.
 Rates of Mortality Change for Men in the United States, by Age and Period, 1948–2003^a

Note: Shaded entries indicate rates of decline that are slower than the median value of -0.0658.

Sources: See footnote 1.

^aRates of mortality change for men are calculated as $\frac{M_i(t+5) - M_i(t)}{M_i(t)}$, where M_i = death rate for males in age interval *i*, year *t*.

	2005						
				Age Interval			
Period	50-54	55–59	60–64	65–69	70–74	75–79	80-84
1953–1948	-0.0937	-0.1235	-0.1123	-0.0771	-0.0964	-0.1028	-0.0277
1958–1953	-0.1172	-0.0931	-0.0663	-0.0621	-0.0770	-0.0584	-0.0231
1963–1958	-0.0313	-0.0265	-0.0258	-0.0240	-0.0424	-0.0580	-0.0392
1968–1963	-0.0091	-0.0115	-0.0443	-0.0311	-0.0249	-0.0624	-0.0924
1973–1968	-0.0769	-0.0398	-0.0715	-0.1381	-0.0940	-0.0269	-0.0844
1978–1973	-0.1059	-0.1299	-0.0579	-0.1139	-0.1365	-0.1173	-0.1000
1983–1978	-0.0793	-0.0471	-0.0686	-0.0065	-0.0451	-0.1382	-0.0841
1988–1983	-0.0611	-0.0293	-0.0183	-0.0120	-0.0174	-0.0719	-0.0219
1993–1988	-0.0836	-0.0693	-0.0501	-0.0366	-0.0420	-0.0413	-0.0601
1998–1993	-0.0859	-0.0845	-0.0667	-0.0448	-0.0214	-0.0324	-0.0130
2003-1998	0.0045	-0.0506	-0.0579	-0.0635	-0.0588	-0.0199	-0.0335

Table 2. Rates of Mortality Change for Women in the United States, by Age and Period, 1948–2003^a

Note: Shaded entries indicate rates of decline that are slower than the median value of -0.0584. *Sources:* See footnote 1.

^aRates of mortality change for women are calculated as $\frac{F_i(t+5) - F_i(t)}{F_i(t)}$, where F_i = death rate for females in age interval *i*, year *t*.

				Age Interval	1		
Period	50-54	55–59	60–64	65–69	70–74	75–79	80-84
1953–1948	0.0221	0.0629	0.0891	0.0765	0.0625	0.0404	0.0630
1958–1953	0.0716	0.0410	0.0487	0.0712	0.0718	0.0452	0.0036
1963–1958	0.0243	0.0579	0.0269	0.0816	0.0844	0.0627	0.0216
1968–1963	0.0029	0.0179	0.0781	0.0035	0.0894	0.0672	0.0265
1973–1968	-0.0192	-0.0299	-0.0043	0.0646	0.0052	0.0453	0.0981
1978–1973	-0.0048	-0.0325	-0.0475	-0.0118	0.0291	0.0324	0.0475
1983–1978	-0.0361	-0.0201	-0.0540	-0.0628	-0.0224	0.0402	0.0220
1988–1983	-0.0245	-0.0534	-0.0350	-0.0526	-0.0415	-0.0339	0.0006
1993–1988	-0.0029	-0.0312	-0.0438	-0.0378	-0.0612	-0.0419	-0.0030
1998–1993	-0.0383	-0.0418	-0.0504	-0.0615	-0.0478	-0.0509	-0.0562
2003-1998	0.0294	-0.0104	-0.0347	-0.0431	-0.0476	-0.0505	-0.0515

Table 3.Sex Differences in Rates of Mortality Change in the United States, by Age and Period,
1948–2003^a

Note: Shaded entries indicate positive values, indicating that men's mortality rose relative to women's.

Sources: See footnote 1.

^aSex differences in rates of mortality change are calculated as

$$\frac{M_{i}(t+5) - M_{i}(t)}{M_{i}(t)} - \frac{F_{i}(t+5) - F_{i}(t)}{F_{i}(t)}$$

where M_i = death rate for males in age interval *i*, year *t*. F_i = death rate for females in age interval *i*, year *t*.

Can smoking patterns account for the change in direction of sex mortality differences across these cohorts? Three sources of information, independent of one another, can help answer this question. The first national sample survey of smoking behavior was conducted by the U.S. Census Bureau for the National Cancer Institute in 1955 (Haenszel, Shimkin, and Miller 1956). A question was asked about the age at which someone had become a "regular cigarette smoker," and the results were tabulated by birth cohort. No allowance was made for differential mortality by smoking status. Table 4 shows the percentage who reported that they had become regular cigarette smokers by age 35. Both men's and women's smoking prevalence continued to increase through cohorts born in the 1920s, but the sex difference in smoking behavior peaked at 44%–45% among the cohorts born in the 1890s and 1900s.

A careful and detailed reconstruction of smoking histories was made by Burns et al. (1998a). They employed a total of 15 National Health Interview Surveys conducted between 1965 and 1991 to estimate cohort smoking histories. The reliability of estimates is increased by virtue of the multiple observations available on the same cohort. The authors used estimates of differential mortality by smoking status to translate current reports by the living into past behavior by the living and dead.² David Burns supplied us with updated,

^{2.} Estimates were not available in this source for black cohorts born before 1900. We accounted for blacks in the three earliest national cohorts by fitting a linear trend line to the relationship between national smoking prevalence and white smoking prevalence for cohorts born 1900–1904 to 1950–1954. This line was extrapolated backward in time, and actual white cohort values were used to predict national prevalence. The disparity between white values and national values was always very small.

	1955 Survey: Cumulative % Who Had Become Regular Cigarette Smokers by Age 35ª			National Health Interview Survey Data Since 1964: Estimated Number of Years Spent as Current Smoker Before Age 40 per Member of Cohort ^b			
Year of Birth	Men	Women	Difference	Men	Women	Difference	
1885–1889	28.1°	1.7 ^c	26.4°	11.6	0.8	10.7	
1890–1894	51 ((1	45 1	12.9	1.4	11.5	
1895–1899	51.6	6.1	45.1	15.8	2.4	13.5	
1900–1904]	<i></i>	10.5	44.2	16.6	3.2	13.4	
1905–1909	62.7	18.5	44.2	17.5	5.3	12.3	
1910–1914]	<i>i</i>	67.3 33.8	33.5	17.9	7.5	10.4	
1915–1919	6/.3			17.8	8.9	9.0	
1920–1924]	(0 /	(2.0	20 (17.7	9.3	8.3	
1925–1929	68.4	42.0	28.4	17.3	10.1	7.2	
1930–1934				16.4	10.3	6.1	
1935–1939				15.1	10.5	4.7	
1940–1944				14.4	10.5	4.0	
1945–1949				12.5	9.2	3.3	
1950–1954				10.7	8.5	2.3	

Table 4.	Two Estimates of the Prevalence of Smoking Within Birth Cohorts

^aSource is Haenszel et al. (1956:56).

^bSource is Burns et al. (1998a); updated estimates supplied by David M. Burns, June 29, 2005.

^cBorn before 1890.

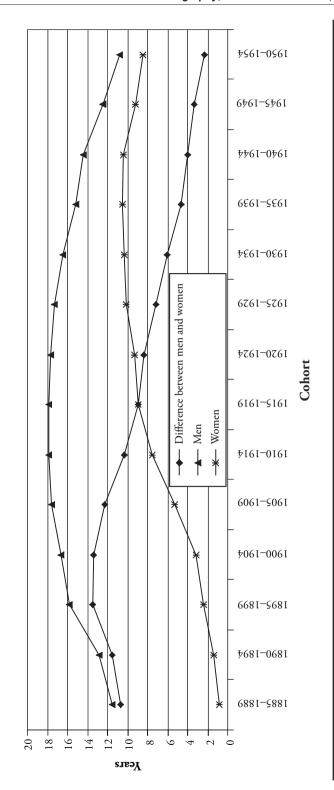
unpublished estimates using the same methodology. These incorporated data from three additional National Health Interview Surveys through 2001.

We converted these data into an estimate of the average number of years spent as a current smoker before the age of 40. This value is derived by summing across ages between 0 and 39 the proportion of cohort members who were estimated to be current cigarette smokers. Table 4 shows that this series has the same general conformation as that drawn from the 1955 survey. The peak difference between the prevalence of smoking among women and men occurs in the 1895–1899 and 1900–1904 cohorts (see also Figure 1). This latter cohort overlaps with the 1903–1908 cohort in which sex mortality differences peak.

Lung cancer death rates are often used as a proxy for cigarette smoking prevalence because such a high fraction of deaths from lung cancer are attributable to smoking (Pampel 2002; Peto et al. 1994). We reconstructed lung cancer death rates for the same ages and periods shown in Table 3.³ Table 5 presents the difference between men's and women's lung

^{3.} The number of deaths from malignant neoplasm of trachea, bronchus, and lung are drawn from the same sources as deaths from all causes combined (see footnote 1). For 1948, we combine two categories from the published data, cancer of trachea and cancer of bronchus and lung; for data between 1952 and 1963, we combine code 162 (malignant neoplasm of respiratory system of trachea, and of bronchus and lung specified as primary) and code 163 (malignant neoplasm of lung and bronchus, unspecified as primary or secondary). Between 1968 and 1978, data

Average Number of Years Spent as a Cigarette Smoker Before Age 40 Among Men and Women in Different Birth Cohorts Figure 1.



	population	011)						
	Age Interval							
Year	50–54	55–59	60–64	65–69	70–74	75–79	80-84	
1948	33.7	56.0	67.4	64.3	53.9	43.8	34.5	
1953	48.3	80.6	106.4	109.8	94.3	78.7	62.1	
1958	56.0	92.5	142.0	163.9	145.5	116.1	88.7	
1963	58.7	105.7	161.7	219.4	215.9	182.1	137.3	
1968	64.1	117.6	191.7	248.1	306.2	261.5	187.4	
1973	63.2	116.4	192.4	270.1	331.5	344.2	282.6	
1978	63.1	110.5	188.9	263.9	361.0	396.3	382.7	
1983	51.2	101.0	161.9	247.3	338.6	404.8	411.0	
1988	44.4	90.5	156.6	225.7	307.9	381.5	421.1	
1993	31.9	72.2	132.9	204.6	257.7	320.2	385.9	
1998	21.2	50.6	91.4	154.6	229.5	273.0	311.9	
2003	18.4	38.0	65.6	114.4	173.4	233.8	262.5	

Table 5. Differences in Lung Cancer Death Rates Between Men and Women (deaths per 100,000 population)

Note: Numbers in boldface type indicate the peak in sex differences in lung cancer mortality for a particular age interval. *Sources:* National Center for Health Statistics, U.S. Department of Vital Statistics, U.S. Census Bureau.

cancer death rates for these groups.⁴ In four out of seven age groups, the sex difference in lung cancer death rates peaks in the cohort born in 1903–1908, the same cohort identified earlier as having the highest sex mortality difference for all causes combined. In two other age groups, the peak is displaced by only five years from this cohort.

Thus, three independent tests support the plausibility of cigarette smoking patterns as the source of the widening and then contracting of sex mortality differences. It is reasonable to ask whether lung cancer is *solely* responsible for the diagonalized pattern of change in sex mortality differences shown in Table 3. That would be surprising in view of the fact that lung cancer accounts for only about 14%–28% of the excess deaths from smoking in the United States, depending on the study (Thun et al. 1998:328). To investigate whether lung cancer is exclusively responsible for the pattern of change in sex mortality differences, we subtracted lung cancer death rates from all-cause mortality and repeated the tabulation shown in Table 3. The result (not shown) is little altered: 33 of 35 observations above the diagonal remain positive, and 35 of 42 below the line remain negative. When lung cancer deaths are removed from Table 3, the difference between the mean values of observations above and below the diagonal declines only from .0815 to .0680. Clearly, other causes of death must also be implicated in this structure. Epidemiologic studies suggest that, in addition to lung cancer, the main causes of death responsible for the excess mortality

are coded according to the Eighth Revision, International Classification of Diseases, where malignant neoplasm of trachea, bronchus, and lung is coded 162. Between 1983 and 1998, the Ninth Revision is used, wherein malignant neoplasm of trachea, bronchus, and lung is also coded 162. Data from 2003 employ the Tenth Revision, in which malignant neoplasm of trachea, bronchus, and lung is coded as C33–C34.

^{4.} The sex difference in death rates is preferred to the ratio for this comparison because the difference should be linearly related to the difference in smoking prevalence between the sexes, assuming a linear relation between smoking and mortality for each sex.

of smokers are, in order, coronary heart disease, chronic obstructive lung disease, other smoking-related cancers, and stroke (Thun et al. 1998).

AGE-PERIOD-COHORT ANALYSIS OF MORTALITY TRENDS

Cohort influences on mortality have been recognized since the pioneering work of Kermack, McKendrick, and McKinlay (1934). Most of the successful studies, like theirs, used graphical methods to demonstrate that age patterns of mortality by cohort were very different from those arranged by period and to argue that the cohort patterns reflected genuine and persistent influences embedded in cohorts.

Less successful have been statistical efforts to disentangle age, period, and cohort effects in an accounting framework using dummy variables. Because each variable is a linear combination of the other two variables, some restriction must be imposed for the effects of ages, periods, and cohorts to be identified. These restrictions are often arbitrary, and results can be highly sensitive to the restriction employed because of the correlation among variables (Mason and Smith 1985). When nonlinear terms for cohort and period are introduced along with a common linear drift term, the typical result across countries is that the linear drift term explains the great majority of variation in all-cause mortality (Janssen and Kunst 2005).

In our case, it is not necessary to study cohort effects by employing a set of dummy variables to represent cohort membership because we have a hypothesis about cohort influences: that a cohort's smoking history affects its level of mortality. We will represent that history by using the variable introduced earlier, the mean number of years that members of a cohort smoked cigarettes before age 40. The value of this variable differs between men and women in the same cohort, reflecting their different smoking histories. While the variable is an indicator of only one of the two relevant dimensions of smoking, duration and intensity, all relevant studies of lung cancer mortality have concluded that the proportionate impact of duration is far greater than that of intensity (e.g., Knoke et al. 2004).⁵

We model age and period effects through a series of dummy variables. Our model includes both men and women, but we allow for well-known sex differences in the level and age pattern of mortality through a set of age-sex interaction dummy variables. We also allow for sex differences in the effect of smoking by constructing a sex-smoking interactive variable. We consider this model to be the simplest defensible age-period-cohort model of the effect of smoking. Adding complexity, for example, in the form of nonlinear smoking effects, age-period interactions, or sex-period interactions, would doubtless change the results. But with relatively few observations available and high colinearity among independent variables, the case for simplicity is strong.

We model the mortality process by using negative binomial regression. We initially used Poisson regression, but the hypothesis that the data were Poisson-distributed was decisively rejected: the amount of dispersion in outcomes was significantly underestimated by the Poisson model. Our model is⁶

^{5.} The use of mortality differences by smoking status to convert retrospective reports on smoking prevalence into estimates of actual prevalence in the past raises issues of circularity in the statistical estimation. However, there is no reason to believe that the adjustments made to the smoking prevalence estimates introduce bias in estimated coefficients. If no such adjustments were made, the estimated prevalence of smoking in the past would be lower than that shown in Table 2, and the smoking series would increase too rapidly with birth year. If the differential mortality assumed between smokers and nonsmokers were too great, the estimated prevalence of smoking shown in Table 2 would be too high, and the series would rise too slowly with birth year. Both circumstances would introduce error into the estimated smoking series and accordingly bias the smoking coefficient downward. The key is not whether differential mortality by smoking status is introduced into the estimation but whether it is introduced correctly.

^{6.} This specification assumes that a particular history of smoking will have the same proportionate effect on mortality rates from ages 50 to 85. While this is a common starting point in cohort analysis, there are many reasons why it may not be accurate. These include the possibility of age-cohort interactions and of turnover of cohort membership through death and immigration. In the present case, differential mortality by smoking status can be

$$D_{iiks} = \exp\{\ln N_{iiks} + B_i X_i + B_j X_i + B_c C_{ks} + B_s X_s + B_{is} X_{is} + B_{cs} X_{cs} + V_{iiks}\},\$$

where D_{ijks} equals the number of deaths in age group *i*, period *j*, cohort *k*, and sex *s*; N_{ijks} equals the number of person-years of exposure at age *i*, period *j*, cohort *k*, and sex *s*; X_i is a dummy variable signifying membership in age group *i*; X_j is a dummy variable signifying observation pertained to period *j*; X_s is a dummy variable signifying observation applied to sex *s*; C_{ks} equals the average number of years spent as a current smoker prior to age 40 by members of cohort *k* and sex *s*; X_{is} is an interactive dummy variable indicating observation pertained both to age *i* and sex *s*; X_{cs} is an interactive variable that indicates that the smoking variable pertained to sex *s*; V_{ijks} is an error term whose exponential is gamma distributed; and B_i , B_j , B_c , B_s , B_{is} , and B_{cs} are coefficients indicating estimated effect of the variable on mortality.

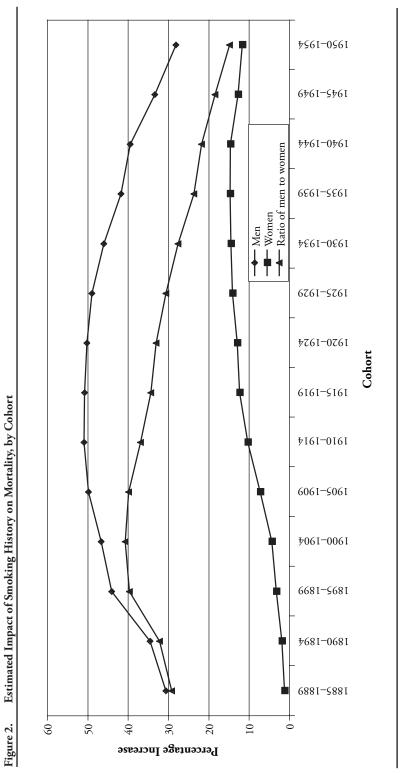
Coefficients of this model are estimated using STATA and are presented in Appendix Table A1. The coefficient of the cohort-sex smoking variable is 0.0230, with a standard error of 0.0022 (p < .001). The coefficient implies that a cohort's death rates will rise by 2.33% for every one-year increase in average smoking duration by the cohort. The sex-smoking interaction term has a significant (p < .001) coefficient of -0.0100, indicating that a particular level of smoking prevalence in a cohort has a smaller proportionate effect on women's mortality than on men's, perhaps because women smokers on average consume fewer cigarettes per day, inhale less frequently, and smoke cigarettes lower in tar content (Thun et al. 1998:311–15).

The ratio of the female-to-male relative risks is 0.0130 / 0.0230 = 0.57. This ratio is roughly consistent with sex disparities in the risk from smoking recorded in large epidemiologic studies. The first large study, the American Cancer Society Cancer Prevention Study I, conducted between 1959 and 1972, found a ratio of the mortality of current smokers to never-smokers at ages 40–84 of 1.91 for white men and 1.46 for white women, implying that the excess risk for women was 0.51 of that for men (Burns et al. 1998b:232, 292). The later Cancer Prevention Study II estimated the ratio to be 2.3 for men and 1.9 for women between 1982 and 1988, suggesting that the relative risks from smoking have risen for both sexes and have risen more quickly for women (Thun et al. 1998). The mean of the excess risks from these two studies, which span the 1970s midpoint of our own study, is 0.68 for women and 1.10 for men. The sex ratio of mean excess risk in these studies is thus 0.62, close to our estimate of 0.57.

The regression results, combined with the smoking data shown in Table 4, enable us to address the question of how much variability smoking has introduced into sex mortality differences. Figure 2 shows for men and women the estimated percentage excess in mortality rates by cohort that is attributable to smoking. The impact is clearly higher among men than among women, both because more men have smoked and because smoking increased mortality rates for men than for women. The estimated smoking-induced elevation of 51% in mortality rates for the male cohort born 1910–1914 may seem implausibly high, but remember that smoking has increased men's mortality risks by a factor of 1.7–3.5 (depending on the study) and that the proportion of this cohort who were current smokers at any one time reached 77% (Burns et al. 1998a).

Among women, the impact of smoking has been smaller. Nevertheless, the rise in smoking prevalence between the cohorts of 1885–1889 and the peak cohort of 1940–1944 is estimated to have increased women's mortality by 13.4%.

expected to selectively reduce the proportion of ever-smokers or current smokers in a cohort as it ages. In view of this compositional change, a constant proportional effect of smoking on a cohort's mortality could be expected only if the risk from smoking increases with age, for example, because smoking behavior is correlated across the life cycle, and its effects cumulate.



Consistent with earlier data and discussion, the sex difference in the estimated impact of smoking peaks in the cohorts born around the turn of the century. Our estimates suggest that smoking raised the sex ratio of death rates for the cohort born 1900–1904 by 41%. For the cohort born 1945–1949, the estimated impact is only 18%. Thus, changes in smoking patterns account for a reduction of 23% in the sex difference in mortality across these birth cohorts. The hypothesis that smoking is principally responsible for change in the pattern of sex mortality differences is strongly supported by this analysis.

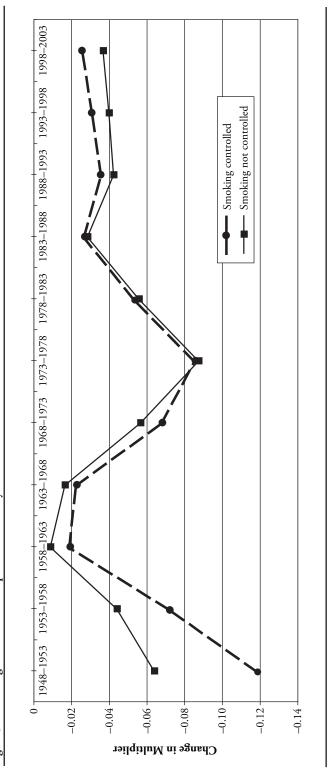
Figure 3 presents the estimated changes in "period effects" on mortality (i.e., first differences in the exponentiated period coefficients in Appendix Table A1). When smoking is controlled, as in our basic model, the declines in mortality tend to grow smaller over time. However, when smoking is not controlled, the series is essentially trendless, with a reduction in mortality averaging approximately 4% during each five-year period. The implication is that the upsurge in smoking shortly after World War II has partially obscured the major reductions in mortality that would otherwise have occurred during that period, and the recession in smoking over the entire period is to have reduced the amount of decline in mortality. When we control for smoking histories, mortality levels are reduced by 56% during this period. If smoking is not controlled, the estimated period decline in mortality would have been only 48%. Since most descriptive accounts of mortality decline during this period omit the primarily obstructive role of smoking, they provide an overly pessimistic view of the period-specific progress that has been made in extending longevity.

We have demonstrated that a cohort's smoking history prior to age 40 has a powerful impact on the cohort's subsequent mortality. To some extent, its power reflects a positive correlation in smoking propensities across the life cycle, including smoking beyond age 40. But it also reflects the enduring impact of early smoking behavior on health and mortality at later ages. Recent studies that more carefully measured smoking histories found larger impacts of smoking at younger ages than did earlier studies. For example, using follow-up data from Cancer Prevention Study II, Taylor et al. (2002) found that former smokers aged 60–69 at baseline who had quit smoking 11–15 years earlier had a risk of death relative to lifetime nonsmokers of 1.75 (for men) and 1.59 (for women) during the period 1982–1996, that is, an average of 20 years after they stopped smoking.

There may also be period-specific influences on smoking behavior that our cohort smoking coefficients would not reflect. One possibility is that the U.S. Surgeon General's first report describing the dangers of smoking (Department of Health, Education, and Welfare 1964) and a subsequent national antismoking advertising campaign during 1967–1970 may have produced a reduction in smoking propensities across all cohorts (Burns et al. 1998a:30; Tolley et al. 1991:85–86). If so, these changes would be reflected in period coefficients. Our period coefficients do show an unusually rapid reduction in mortality between 1968 and 1973, although a rapid diffusion of antihypertensive drugs has also been identified as an important factor in mortality during this period (Sytkowski et al. 1996). Whatever period-specific influences on smoking behavior are present, they clearly do not erase the statistical impact of a cohort's early smoking behavior on its subsequent mortality.

IMPENDING SMOKING-RELATED CHANGES IN FUTURE MORTALITY

Just as mortality improvements at older ages in the past half-century have been inhibited by increases in smoking, so should mortality declines in the future be accelerated by reductions in smoking. Even with no subsequent changes in smoking behavior, current age-specific smoking behavior implies that members of future cohorts reaching age 50 will have accumulated fewer years of smoking than cohorts who are currently in this age range. To illustrate this effect, we have created a synthetic cohort whose smoking





	Probability of Surviving From Age 50 to 85			
	Men	Women	Women / Men	
U.S. Life Table of 2003 ^a	.302	.464	1.536	
Age-Period-Cohort Model 2003 predictions with actual smoking histories	.304	.468	1.539	
2003 predictions with 2000 current smoking behavior	.384	.479	1.247	
2003 predictions with no smoking	.464	.519	1.119	

Table 6. Estimated Changes in Probabilities of Surviving From Age 50 to Age 85 if Smoking Were Reduced or Eliminated Reduced or Eliminated

^aData come from the National Center for Health Statistics (2005).

prevalence is the same at each age as the prevalence recorded at that age in 2000. Cumulating these values to age 40 gives an expectation of 8.40 years as a smoker for men and of 7.58 years for women. Substituting these values for the actual cohort-specific values in 2003 indicates how much improvement in mortality can be expected simply if current behavior continues.

Table 6 shows the result of this exercise in the form of probabilities of survival from age 50 to age 85. Note first that our age-period-cohort model comes close to replicating the actual survival probability in the official U.S. life table for 2003. Substituting the smoking values calculated for the synthetic cohort for those values actually observed in 2003 suggests that men's mortality will benefit enormously from reductions in smoking that have already occurred. Men's survival probability is estimated to increase from .307 to .377, or by 23%. The expected improvement for women is much lower at only 2%. The main reason for this disparity is that current female smoking patterns do not differ radically from those of the past, whereas male smoking patterns have shown large reductions. As a result, it is extremely likely that sex mortality differences will continue to narrow. Pampel (2005) reached a similar conclusion for the United States and other countries by projecting forward period changes in smoking behavior.

What if smoking were eliminated altogether? Table 6 shows that another large improvement in mortality could be expected. Both sexes would share in this improvement, but the survival enhancement once again would be larger for men. The combined effect of these reductions in smoking on sex differences in mortality would be enormous. Currently, women have a 54% higher probability of surviving from age 50 to age 85 than men, whether estimated from the official U.S. life table or from our model. With no smoking by either sex, our model suggests that the difference would be only 12%. With no smoking by men, their probability of survival would be identical to the actual female survival probability in 2003.

Thus, there is considerable potential for major reductions in mortality from a recession in smoking. Large reductions for men seem not only possible but very likely based on changes in smoking behavior that have already occurred. It is likely that these reductions will affect mortality in a manner that is organized by birth cohort. National mortality projections, all major versions of which are currently based upon extrapolations of period trends in mortality, would be well advised to take account of these powerful cohort effects.

Regression								
Covariates	Coefficients	SE	Z	P > z				
Age Group								
50–54 (ref.)								
55–59	0.4297	0.0122	35.13	0.000				
60–64	0.8674	0.0126	68.99	0.000				
65–69	1.2756	0.0129	98.51	0.000				
70–74	1.6908	0.0134	126.24	0.000				
75–79	2.1012	0.0139	150.96	0.000				
80-84	2.5357	0.0146	174.05	0.000				
Period								
1948 (ref.)								
1953	-0.1265	0.0196	-6.45	0.000				
1958	-0.2118	0.0188	-11.25	0.000				
1963	-0.2360	0.0185	-12.78	0.000				
1968	-0.2652	0.0183	-14.46	0.000				
1973	-0.3583	0.0183	-19.53	0.000				
1978	-0.4890	0.0188	-26.04	0.000				
1983	-0.5802	0.0191	-30.32	0.000				
1988	-0.6296	0.0194	-32.45	0.000				
1993	-0.6997	0.0196	-35.78	0.000				
1998	-0.7650	0.0196	-39.12	0.000				
2003	-0.8231	0.0194	-42.51	0.000				
Number of Years as a Current								
Smoker Before Age 40	0.0230	0.0022	10.45	0.000				
Female	-0.3297	0.0370	-8.90	0.000				
Female × Number of Years as a Current Smoker Before Age 40	-0.0100	0.0025	-4.05	0.000				
Sex × Age Interactions								
Female × Age 55–59	-0.0061	0.0174	-0.35	0.727				
Female × Age 60–64	-0.0032	0.0178	-0.18	0.856				
Female × Age 65–69	0.0166	0.0183	0.91	0.365				
Female × Age 70–74	0.0686	0.0189	3.63	0.000				
Female × Age 75–79	0.1424	0.0197	7.24	0.000				
Female × Age 80–84	0.2265	0.0206	11.00	0.000				
Constant	-4.5574	0.0362	-125.77	0.000				

Appendix Table A1. Estimates of Coefficients and Standard Errors of Negative Binomial Regression

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