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Homicide Mortality in the United States, 1935–1994: Age, Period, and Cohort Effects

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The authors analyzed homicide mortality data for the United States from 1935 to 1994, to delineate temporal trends and birth cohort patterns. This study included 850,822 homicide-attributed deaths documented by the National Center for Health Statistics, and incorporated graphical presentation, median polish, and Poisson regression modeling in an age-period-cohort analysis. Death rates from homicide in the United States doubled in the past four decades, with most of the increase having occurred during the 1960s and early 1970s. Poisson regression models confirmed that the rise of homicide mortality in both men and women was largely attributable to a significant period effect between 1960 and 1974. No discernible cohort patterns were found among women. However, homicide rates for recent male birth cohorts appeared to peak at younger ages and at higher levels. A significant increase in homicide mortality risk beginning with males born around 1965 was found by examining the residuals of median polish, and the second-order changes in the regression coefficients from the age-period-cohort model. The hike of homicide mortality during 1985 and 1994 was explained by this cohort effect. Increased prevalence of substance abuse and availability of firearms are two likely factors underlying this disturbing cohort pattern. *Am J Epidemiol* 1999;150:1213–22.

cohort effect; homicide; models, statistical; mortality; Poisson distribution; wounds and injuries

The United States has the highest homicide rate among all Western industrialized countries (1). Homicide is the 14th leading cause of death in the United States, accounting for more than 20,000 deaths each year (2). For adolescents and young adults ages 15–24 years, homicide is the second leading cause of death. Over 80 percent of all homicide victims are under 44 years old (3). When considering the pattern of high incidence at younger ages, it is clear that homicide imposes a significant burden on society in terms of years of potential life lost.

Despite the importance of homicide as a public health issue, previous studies have been based primarily on cross-sectional analysis. In the present study, we compiled annual age- and sex-specific homicide and population data for the past 60 years and performed an age-period-cohort (APC) analysis to better understand the temporal trends and birth cohort patterns of death rates from homicide.

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Abbreviation: APC, age-period-cohort.

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MATERIALS AND METHODS

Homicide mortality and mid-year population data by age and sex were abstracted from annual *Mortality Statistics* and later *Vital Statistics of the United States* volumes published from 1910 to 1994 by the National Center for Health Statistics, Hyattsville, Maryland (4, 5). Homicide was defined initially by the *International List of Causes of Death*, and most recently by the *International Classification of Diseases and Related Health Problems* (ICD). These classification criteria were revised seven times between 1910 and 1994 (6). Categorization changes, however, have had virtually no effect on homicide reporting or classification, and vital statistics figures on homicide can be considered reliable with respect to case ascertainment (7–15). Excluded from this study were persons age 80 years and older because of insufficient numbers of homicide deaths and unavailable data for these individuals in early years. From 1900 until 1933, national mortality statistics were based on a death registration area, which continually added states until all 48 (excluding Alaska and Hawaii) were included in 1933. Previous research has indicated that the expanding registration area was not composed of a random group of states and that, initially, states with lower homicide rates were included (4, 16). To avoid bias related to uneven sampling, only data from 1935–1994 were included in the analysis.

Men and women were analyzed separately because of the considerable gender differences in homicide rates. This study included a total of 850,822 homicide deaths (661,423 men and 189,399 women).

Data analysis progressed from the classic graphical approach, to median polish analysis, to Poisson regression modeling. The data were tabulated into 16 5-year age groups (0–4 to 75–79 years), 12 5-year periods (1935–1939 to 1990–1994), and 27 overlapping 10-year birth cohorts (1855–1864 to 1985–1994), identified by central year of birth from 1860 to 1990. Age-adjusted mortality rates were computed by the direct method using the population from the 1940 census as the standard (17).

The graphical approach was used to visually detect age, period, and cohort effects. Age-specific rates were plotted by year of death (period), and year of birth-(cohort) specific rates were plotted by age group, for men and women separately. In order to facilitate easy interpretation while elucidating long-term patterns, certain age-specific (5–9 years, and every other age group beginning with 30–34 years) and year of birth-specific (every other cohort from 1860–1960) rates were not included in the plots. Age effects were identified whenever age-specific rates were consistently different for an age group over a range of periods or birth cohorts. Period effects were observed if rates for all age groups changed by period. Cohort effects were observed if age-specific rates were not parallel across periods, or were elevated for all ages of the same birth cohort.

The median polish method was used with the consideration that cohort effects are a type of age-period interaction. More specifically, we assume that cohort effects are the major component of the non-additive effects of mortality rates in a two-way contingency table. The median polish method is valuable because no assumptions about the distribution of the data are necessary to carry out the analysis, and it can be applied using rates or logarithms of the rates (18). Median polish analysis enables removal of the age (row) and period (column) influences by iteratively subtracting the median value of each row and column, leaving a residual value which reflects the non-additivity of the data. After several iterations, the residual values stabilized and were considered estimates of the cohort effect. This process also enabled comparison of actual rates with additive rates (i.e., with cohort effect being removed), to comprehensively examine the influences of the cohort effect over time (18). In the present study, the median polish analysis was performed using the logarithms of the homicide rates.

Poisson regression modeling was used to estimate the age, period, and cohort effects with the assumptions that the number of homicide deaths follows a Poisson distribution and the death rates are a multiplicative function of the included model parameters, making the logarithm of the rates an additive function of the parameters. For instance, the form of the age-period-cohort model was given by

$$\log(d_{ij}/p_{ij}) = \mu + \alpha_i + \beta_j + \gamma_k + \varepsilon_{ij},$$

where d_{ij} denotes the number of deaths in the ith age group and jth period; p_{ij}, the population at risk in the ith age group and jth period; α_i, the effect of the ith age group; β_j, the effect of the jth period category; and γ_k, the effect of the kth cohort category ($k = I - i + j$ when $i = 1, 2, ..., I$) (19–21). Parameter estimates given by two-factor models were interpreted as the log of the relative risk, adjusted for the other factor. In this study, the age group 40–44 years, period 1940–1944, and cohort with central year of birth 1900, were used as the reference categories. Dummy variables for age, period, and cohort, were used throughout the Poisson regression analysis to avoid assumptions concerning the type of association between mortality rates and the dependent variables (19, 20). A sequence of models was fitted separately for men and women, starting with the intercept-only model, progressing to the one-factor (age) model, to the two-factor (age-period and age-cohort) models, and to the three-factor (age-period-cohort (APC)) model. Two-factor models were fitted to provide quantitative estimates of the first-order effects of age, period, and cohort, unavailable from other types of analyses. Parameter estimates and their confidence intervals were generated by the maximum-likelihood method. The modeling was carried out using the GENMOD procedure of the Statistical Analysis Software (SAS) version 6.12 (22).

Recently, statistical fitting of regression models in APC analysis has been increasingly employed in research on cancer and other diseases (23–30). Inherent in the APC multi-classification model is the non-identifiability problem: parameters for age, period, and cohort are not uniquely estimable because of the exact linear dependence of the regressor variables (cohort = period – age) (19, 20, 31–35). Although several methods for dealing with the non-identifiability problem have been proposed, there is no consensus in the literature as to which method is optimal (19, 20, 23–36). In this study, the three-factor APC models were fitted by constraining the regression coefficients for the two extreme cohorts as zero, an approach commonly used in previous studies (31, 37). While this approach does not provide unique and interpretable first-order relative risk estimates, second-order changes in the slope of age, period, and cohort

effects, which are invariant to changes in the model constraints applied, can be estimated (31, 37, 38). Evaluation of the second-order changes was achieved by construction of identifiable linear contrasts between parameter effects. These contrasts followed the form

$$((\Pi_{h+2} - \Pi_h) - (\Pi_h - \Pi_{h-2})),$$

where Π_h is the hth period or cohort effect (31). Contrasts evaluated to detect statistically significant changes in period and cohort effects were selected from hypothesized instances of significant effects from graphical presentation, median polish analysis, and plots of parameter estimates from the three-factor APC models.

RESULTS

Over the last 40 years of the study period, age-adjusted annual death rates from homicide per 100,000 population doubled for both men and women, with most of the increases having occurred in the 1960s and early 1970s (figure 1). Overall, the age-adjusted death rate from homicide for men (12.0 per 100,000 per year) was 3.6 times that for women (3.3 per 100,000 per year).

Graphical presentation

Age-specific homicide rates by period were plotted separately for men (figure 2) and women (figure 3). The increase in death rates between 1960 and 1974 among all age groups for both sexes indicates a period effect, and the unparalleled rise since 1975 in males age 15–24 years indicates a cohort effect. When the death rates were displayed by year of birth and age, the cohort effect in men was more evident: homicide rates in recent generations appeared to peak at younger ages and at higher levels (figure 4). Homicide rates for women, however, did not show apparent cohort patterns (figure 5).

Median polish

Cohort effects were further examined by assessing the residuals from the median polish analysis. Considerable positive residuals were observed in the 1970–1990 birth cohorts, especially among men (figure 6). The magnitude of the cohort effect on homicide

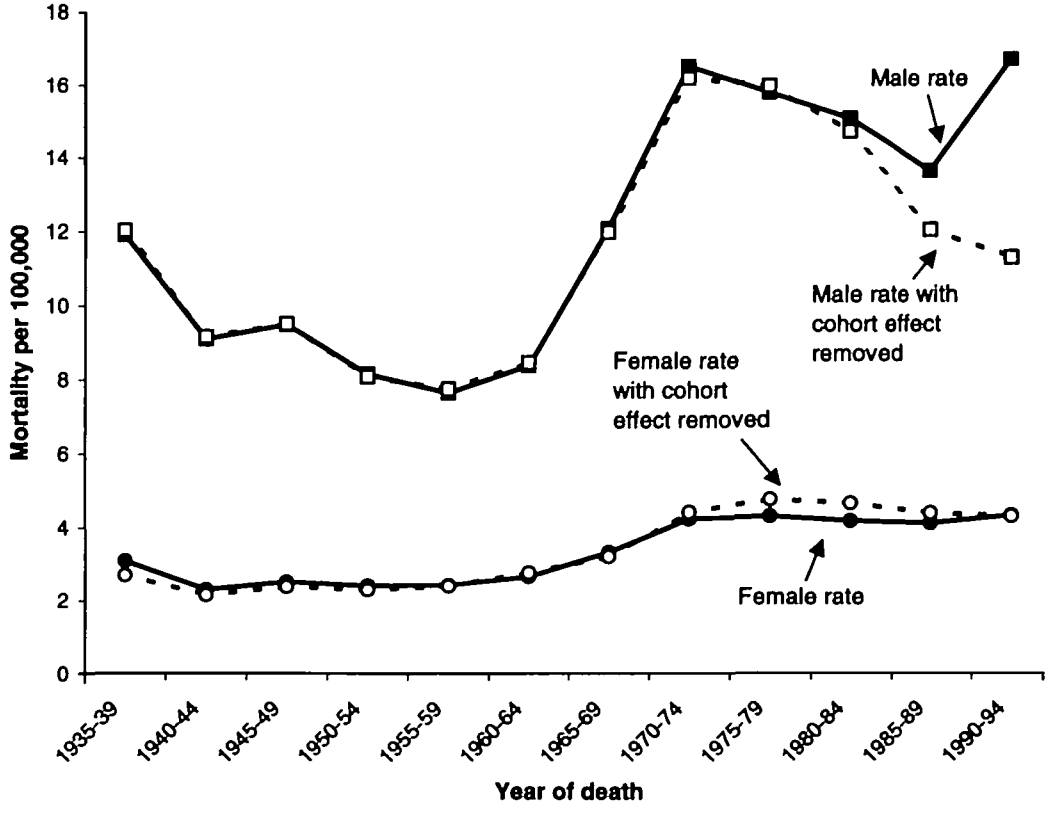

FIGURE 1. Age-adjusted homicide rates with and without cohort effect, by sex and year of death, United States, 1935–1994.

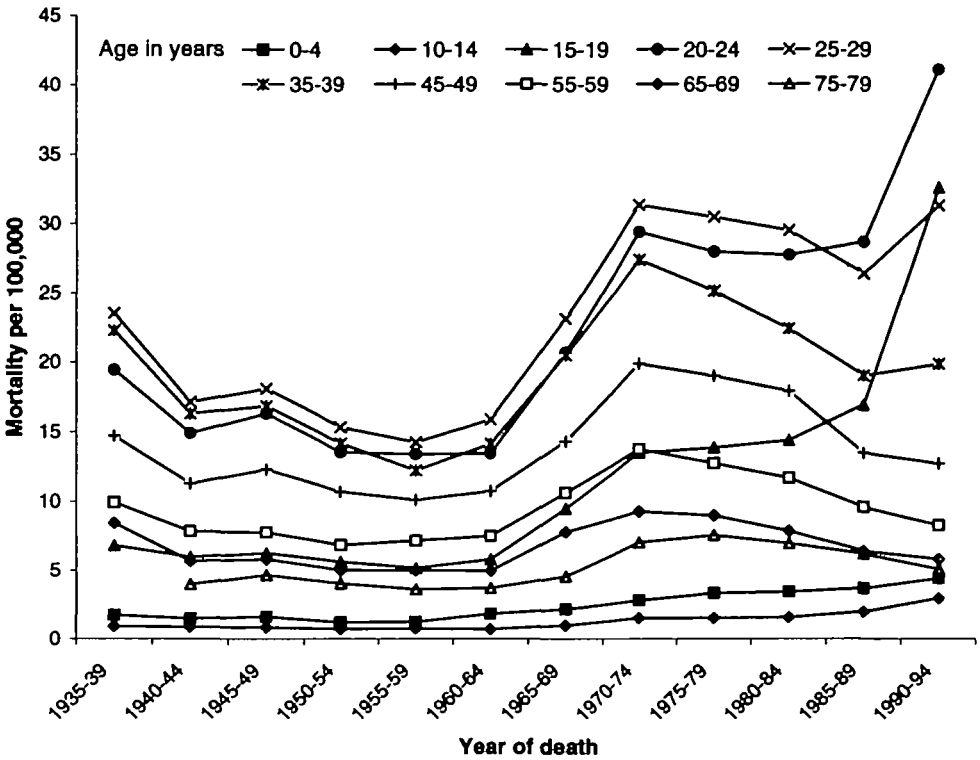

FIGURE 2. Age-specific homicide mortality rates among males aged 0–79 years, by year of death, United States, 1935–1994.

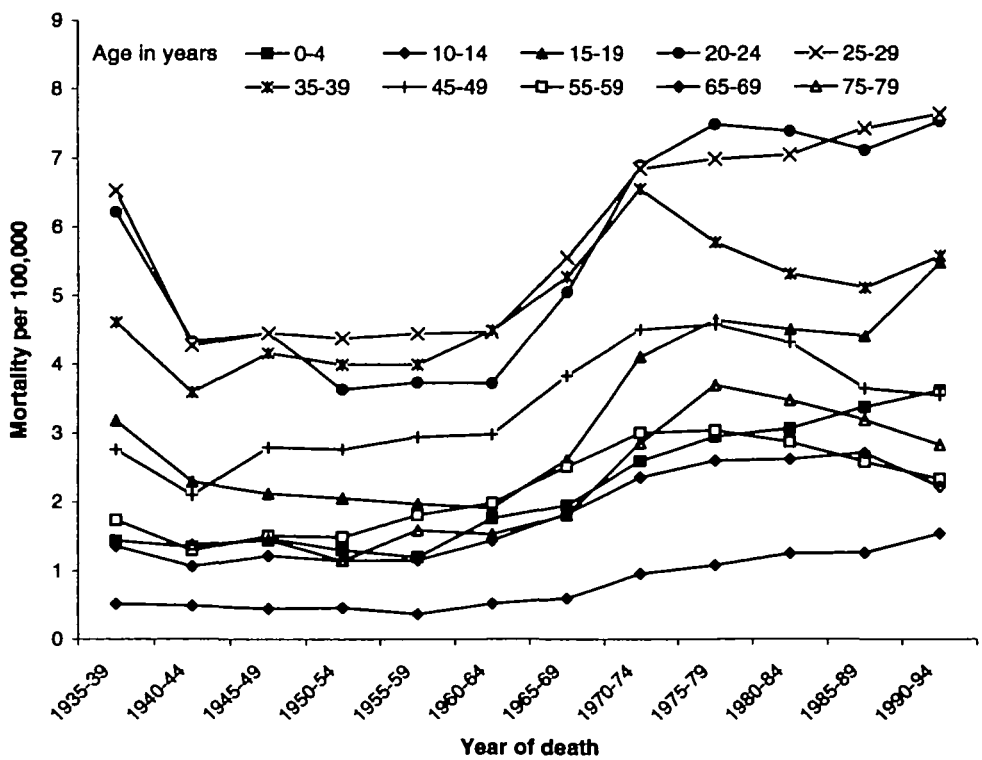

FIGURE 3. Age-specific homicide mortality rates among females aged 0–79 years, by year of death, United States, 1935–1994.

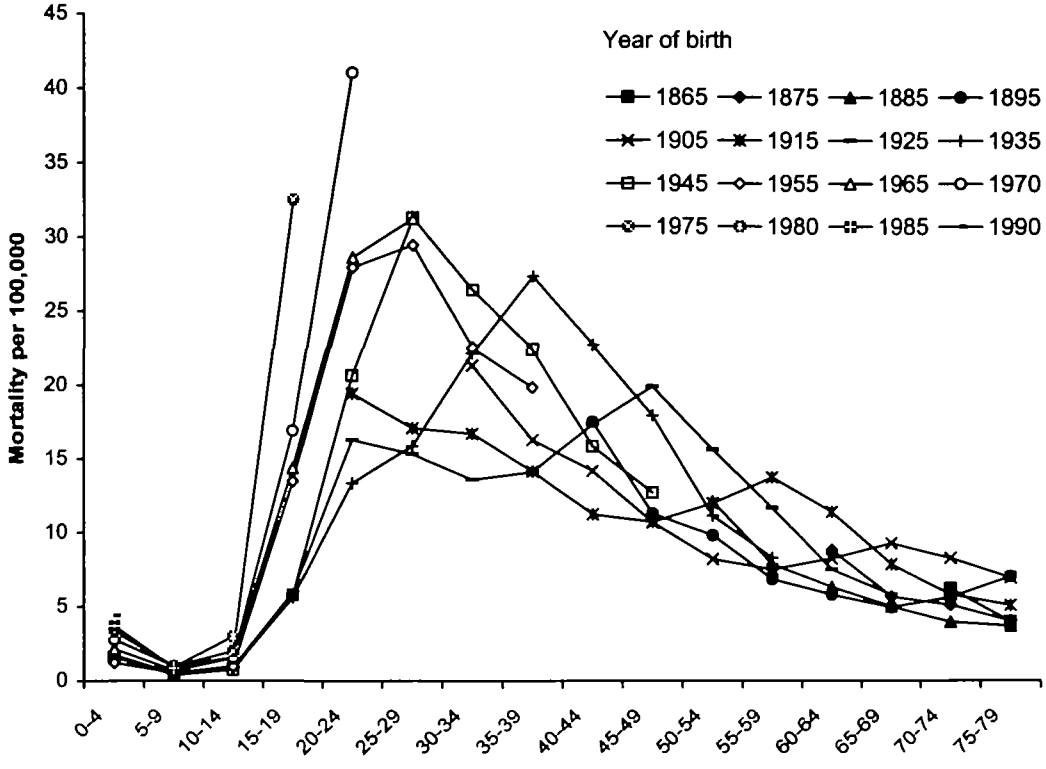

FIGURE 4. Year of birth-specific homicide mortality rates among males aged 0–79 years, by age, United States, 1935–1994.

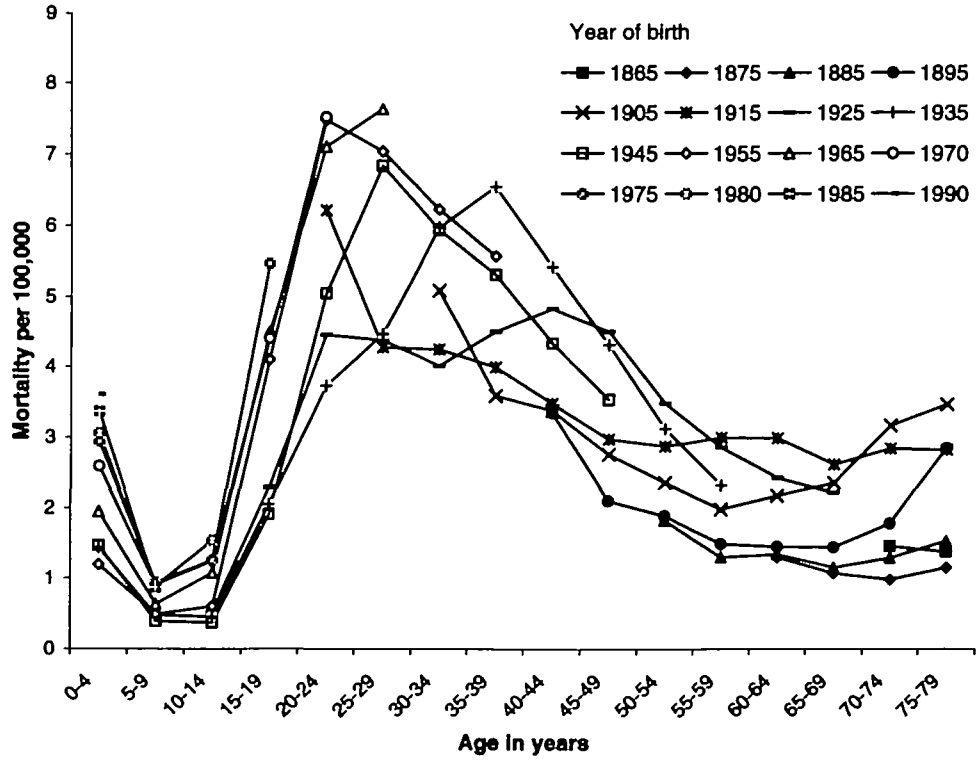

FIGURE 5. Year of birth-specific homicide mortality rates among females aged 0–79 years, by age, United States, 1935–1994.

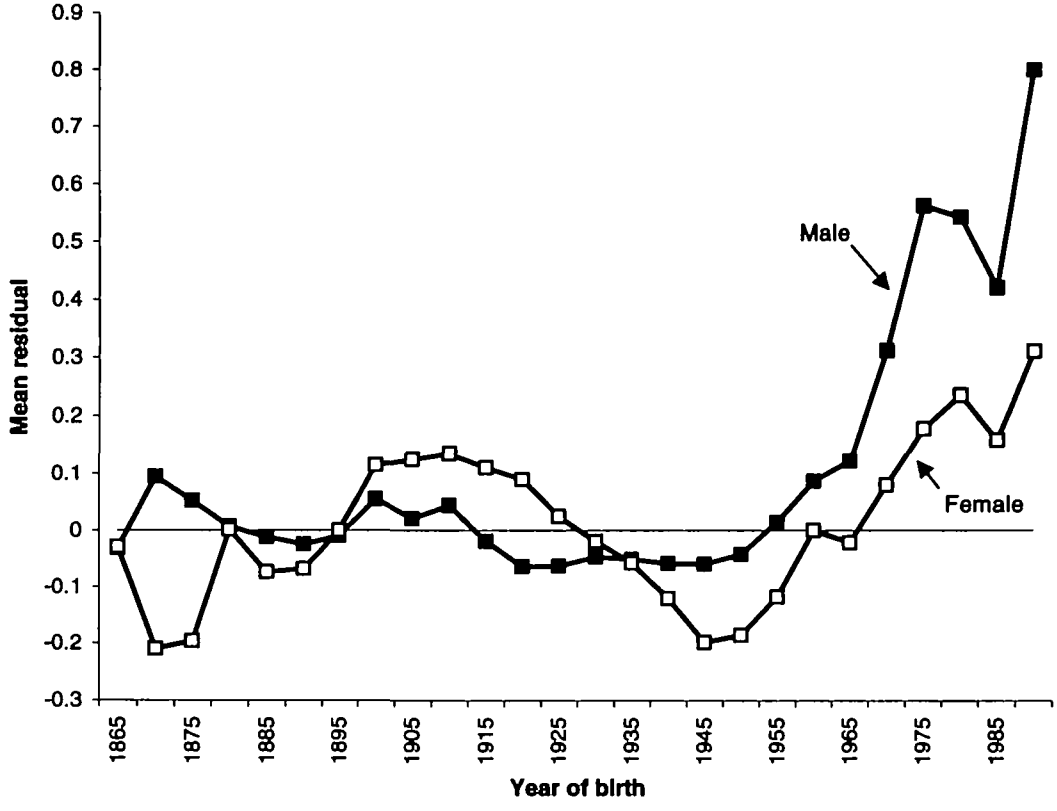

FIGURE 6. Mean residuals from the median polish method, by sex and year of birth, homicide mortality data, United States, 1935–1994.

mortality was gauged by comparing the actual homicide rates with the rates consisting of only the additive age and period effects. For all ages combined, the cohort effect was negligible among women but pronounced among men from calendar period 1985–1994 (figure 1). Specifically, removal of the cohort effect would result in a 26 percent reduction in the death rates among men aged 15–29 years from 1975 to 1994 (figure 7).

The goodness-of-fit of various Poisson regression models is summarized in table 1. Two-factor (age-period and age-cohort) modeling significantly improved the fit over the one-factor and intercept-only models. Tests for the period effect (likelihood ratio statistic (LRS) = 142.92, degrees of freedom (df) = 11, $p < 0.001$), and cohort effect (LRS = 106.65, df = 25, $p < 0.01$), when adjusting for each other and age, were significant for males but not for females (table 1).

When adjusting for year of death, men aged 20–34 years were at significantly increased risk of homicide (table 2). For men, homicide risk during the period 1965–1994 was significantly higher than in the 1940–1944 time span. The period effect exhibited a similar pattern among women. When adjusting for cohort, significantly elevated relative risks were observed only for men aged 25–29 years (table 3). Increase in the relative risk of homicide among men became significant with the 1950 cohort, continuously rising until the 1975 cohort. Among women, relative risk increased continuously since the 1870 birth cohort. Because the results in table 3 were adjusted only for age, the relative risks for birth cohorts were likely to be seriously confounded by period effects.

Results from the three-factor APC models are presented in figure 8. Because first-order measures of the magnitude of the period and cohort effects vary with the model constraint chosen, only invariant second-order changes in the slope of period and cohort effects were examined. For both sexes, the slope of the period effects increased around the period 1940–1944 and rose continually from 1955 to 1969, then decreased after the 1970–1974 period (figure 8). A rise in the slope of the period effect among men was observed around period 1985–1989. The slope of cohort effects for males increased between the 1940 and 1970 cohorts, followed by decline in the slope after the 1975 cohort, then increasing again until the 1985 cohort (figure 8). Among females, the slope of the cohort effect decreased around the 1880, 1960, and 1975 cohorts and increased between the 1940 and 1955

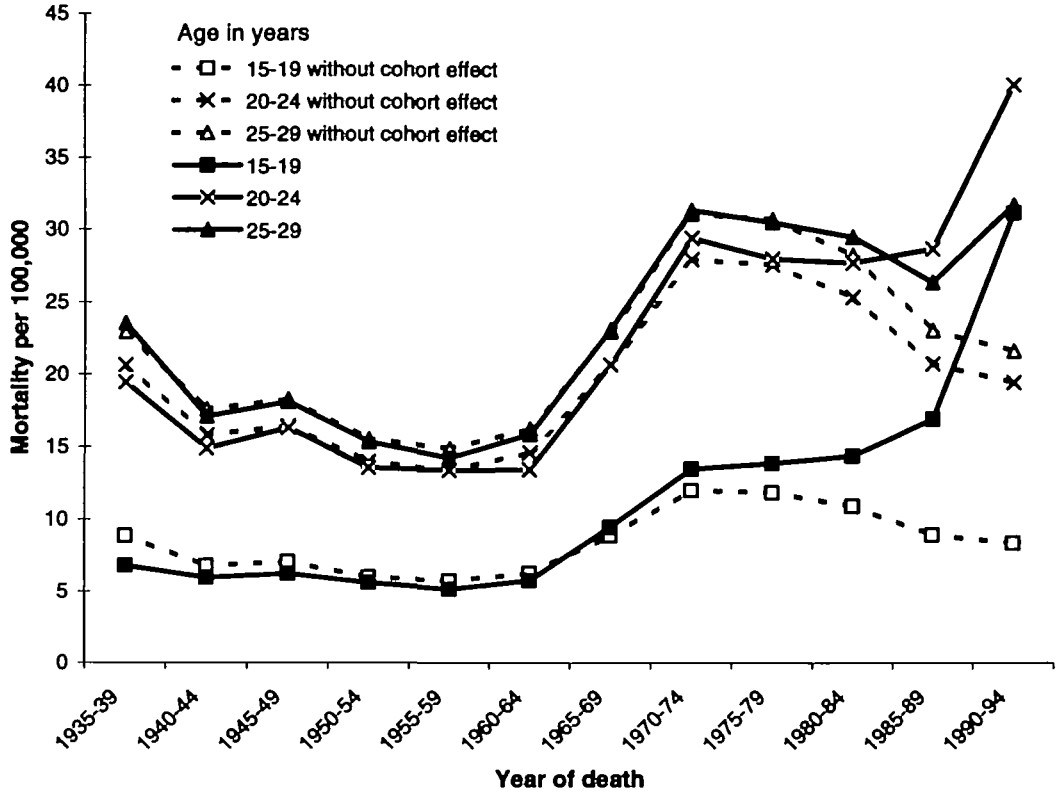

FIGURE 7. Age-specific mortality rates with and without cohort effect, males aged 15–29 years, by year of death, United States, 1935–1994.

cohorts (figure 8). Linear contrasts for men revealed a significant increase in the period slope beginning in 1960–1964 ($p < 0.01$), followed by a significant decrease in the period slope beginning in 1970–1974 ($p < 0.01$). These contrasts indicate an increase in homicide mortality risk for males beginning in calendar period 1960–1964 and continuing until 1970–1974, after which there was a decrease in mortality risk associated with period effects. A significant increase in the cohort effect among men was found beginning with the 1965 cohort ($p < 0.05$). This contrast indicates an increase in homicide mortality risk beginning with male cohorts born around 1965. None of the changes in the slopes of period and cohort effects for women was statistically significant.

DISCUSSION

Estimation of age, period, and cohort effects using full three-factor models has been a subject of debate among researchers. With each proposed solution come several refutations as to why such a solution is ambiguous, uninterpretable, or statistically unsound (19, 20, 32, 39–42). Avoiding these problems, this study incorporated graphical presentation, median polish, and Poisson regression modeling to analyze historical homicide data.

TABLE 1. Goodness of fit of Poisson regression models on homicide mortality in the United States, 1935–1994

Model	Degrees of freedom	Males			Females		
		Deviance	*p* value	R^2	Deviance	*p* value	R^2
Intercept	191	1,202.86			215.29		
Age	175	195.75	0.13	0.84	46.46	1	0.78
Age-period	164	52.83	1	0.96	9.55	1	0.96
Age-cohort	150	89.10	1	0.93	13.48	1	0.94
Age-period-cohort	140	7.77	1	0.99	2.07	1	0.99

TABLE 2. Relative risks and 95% confidence intervals (CI) of homicide mortality by sex, age, and year of death, from two-factor age-period models, United States, 1935–1994

Variable	Males		Females	
	Relative risk	95% CI	Relative risk	95% CI
Age (years)				
0–4	0.15	0.10, 0.22	0.52	0.33, 0.84
5–9	0.04	0.02, 0.08	0.15	0.07, 0.32
10–14	0.08	0.05, 0.13	0.19	0.10, 0.38
15–19	0.69	0.56, 0.86	0.79	0.52, 1.20
20–24	1.36	1.13, 1.63	1.35	0.94, 1.95
25–29	1.41	1.17, 1.69	1.40	0.98, 2.02
30–34	1.27	1.05, 1.53	1.28	0.88, 1.86
35–39	1.17	0.97, 1.42	1.17	0.80, 1.71
40–44	1.00*		1.00*	
45–49	0.85	0.69, 1.04	0.82	0.54, 1.24
50–54	0.70	0.57, 0.88	0.63	0.40, 0.99
55–59	0.58	0.46, 0.73	0.52	0.33, 0.84
60–64	0.48	0.38, 0.62	0.47	0.29, 0.76
65–69	0.41	0.32, 0.53	0.44	0.26, 0.72
70–74	0.35	0.27, 0.46	0.46	0.28, 0.76
75–79	0.32	0.24, 0.43	0.54	0.33, 0.87
Year of death				
1935–1939	1.31	1.05, 1.64	1.34	0.85, 2.12
1940–1944	1.00*		1.00*	
1945–1949	1.04	0.83, 1.32	1.11	0.69, 1.78
1950–1954	0.90	0.70, 1.14	1.08	0.67, 1.74
1955–1959	0.85	0.66, 1.08	1.10	0.69, 1.77
1960–1964	0.92	0.72, 1.17	1.21	0.76, 1.93
1965–1969	1.31	1.05, 1.64	1.50	0.97, 2.34
1970–1974	1.79	1.46, 2.21	1.92	1.26, 2.93
1975–1979	1.72	1.39, 2.12	1.98	1.30, 3.01
1980–1984	1.62	1.31, 2.01	1.89	1.24, 2.89
1985–1989	1.43	1.15, 1.78	1.85	1.21, 2.83
1990–1994	1.66	1.34, 2.05	1.88	1.23, 2.88

* Reference group.

TABLE 3. Relative risks and 95% confidence intervals (CI) of homicide mortality by sex, age, and year of death, from two-factor age-cohort models, United States, 1935–1994

Variable	Males		Females	
	Relative risk	95% CI	Relative risk	95% CI
Age (years)				
0–4	0.07	0.04, 0.11	0.29	0.16, 0.51
5–9	0.02	0.01, 0.04	0.09	0.04, 0.20
10–14	0.04	0.03, 0.08	0.13	0.06, 0.26
15–19	0.45	0.35, 0.58	0.57	0.36, 0.89
20–24	1.06	0.87, 1.30	1.07	0.73, 1.58
25–29	1.21	1.01, 1.46	1.18	0.81, 1.72
30–34	1.16	0.96, 1.41	1.14	0.78, 1.66
35–39	1.12	0.92, 1.36	1.10	0.75, 1.61
40–44	1.00*		1.00*	
45–49	0.89	0.72, 1.10	0.87	0.57, 1.31
50–54	0.77	0.62, 0.96	0.71	0.45, 1.12
55–59	0.66	0.52, 0.83	0.62	0.38, 1.01
60–64	0.57	0.44, 0.73	0.60	0.36, 0.99
65–69	0.49	0.37, 0.64	0.60	0.35, 1.01
70–74	0.42	0.31, 0.57	0.68	0.40, 1.15
75–79	0.39	0.28, 0.53	0.84	0.50, 1.41
Year of birth				
1865	0.85	0.43, 1.66	0.54	0.15, 1.91
1870	0.94	0.56, 1.58	0.48	0.15, 1.51
1875	0.85	0.54, 1.35	0.48	0.17, 1.33
1880	0.80	0.52, 1.21	0.62	0.26, 1.44
1885	0.79	0.54, 1.16	0.60	0.27, 1.33
1890	0.82	0.58, 1.15	0.67	0.33, 1.37
1895	0.89	0.65, 1.22	0.80	0.43, 1.50
1900	1.00*		1.00*	
1905	1.00	0.76, 1.31	1.08	0.63, 1.84
1910	1.05	0.80, 1.36	1.16	0.69, 1.95
1915	1.00	0.77, 1.30	1.18	0.71, 1.95
1920	0.98	0.75, 1.28	1.21	0.73, 2.01
1925	1.03	0.79, 1.34	1.22	0.73, 2.05
1930	1.09	0.84, 1.42	1.27	0.75, 2.13
1935	1.17	0.90, 1.53	1.33	0.79, 2.25
1940	1.23	0.94, 1.61	1.33	0.78, 2.26
1945	1.32	1.00, 1.73	1.37	0.80, 2.36
1950	1.44	1.09, 1.91	1.49	0.86, 2.59
1955	1.51	1.13, 2.02	1.70	0.97, 2.99
1960	1.61	1.18, 2.18	1.92	1.08, 3.44
1965	1.84	1.33, 2.55	2.00	1.07, 3.72
1970	2.58	1.81, 3.66	2.26	1.14, 4.46
1975	4.46	2.91, 6.82	2.87	1.29, 6.37
1980	3.69	1.61, 8.49	3.18	1.14, 8.86
1985	3.44	1.22, 9.66	3.26	1.04, 10.22
1990	4.37	1.51, 12.67	3.65	1.07, 12.47

* Reference group.

Coherent results are obtained on comparison of results obtained from different methods. Generally, second-order changes in effects obtained uniquely via full APC models corresponded with changes in the mean residuals from the median polish analysis. Full APC modeling also supported the trends in effect estimates obtained from two-factor models. Overall, the analyses indicate that significant period and cohort effects exist in male homicide mortality. Most important is the finding of a significant increase in homicide mortality risk beginning with the men born around 1965. This increase is responsible for a large portion of the overall increase in homicide mortality between 1985 and 1994. Graphical presentation also revealed that death rates from homicide in recent male birth cohorts tend to peak at younger ages and at higher levels.

Although this study cannot establish any causal association of risk factors with homicide, it provides valuable information for better understanding the temporal trends and for generating hypotheses. Cohort effects usually result from environmental and societal changes (43). The significant increase in homicide risk for male cohorts born after 1965 suggests that this generation is exposed to an environment with unique or intensified risk factors. Abuse of alcohol and other drugs is a well established risk factor for homicide (44), and male birth cohorts born after 1965 have

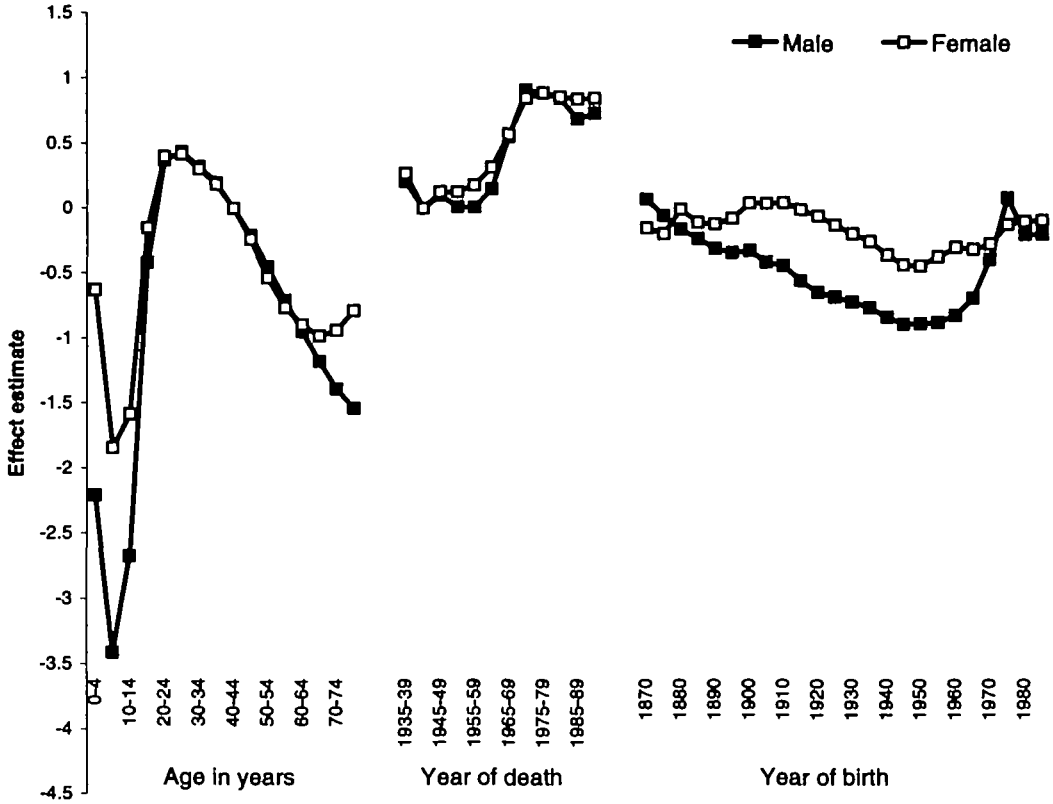

FIGURE 8. Estimated age, period, and cohort effects of homicide mortality from age-period-cohort model, by sex, United States, 1935–1994.

experienced an extremely high prevalence of substance abuse in the late 1980s and early 1990s (45). Coinciding with the prevalent substance abuse in these birth cohorts is the increased exposure to violence (46, 47) and acceptance of a violent life-style such as carrying and using lethal weapons (48, 49). Such increases in the prevalence of known risk factors for homicide in recent male cohorts may be major contributors to the observed cohort effect.

Firearm-related mortality is the most important component of homicide, accounting for over 70 percent of all homicide deaths (50). Although homicide rates associated with knives, ropes, and other methods have remained constant over the past 60 years, firearm-related homicide rates have fluctuated greatly over time, primarily responsible for the temporal variations of the overall homicide rates (50, 51). Therefore, it is conceivable that most of the period and cohort effects in homicide mortality found in this study were due to firearm-related violence.

Male cohorts born after 1965 have experienced unprecedented high homicide rates during their childhood and adolescence. Unless effective interventions are implemented to target this special population group, this generation of men, who currently are adolescents and young adults, are likely to be consistently at greater risk of homicide throughout the rest of their life span than were earlier generations.

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