Variations in Age-Specific Homicide Death Rates: A Cohort Explanation for Changes in the Age Distribution of Homicide Deaths

Robert M. O’Brien

Department of Sociology, University of Oregon

and

Jean Stockard

Department of Planning, Public Policy, and Management, University of Oregon

An age-period-cohort characteristic model previously used to explain age-period-specific rates of homicide arrests for those 15 to 49 from 1960 to 1995 is applied to measures of age-period-specific homicide deaths. The extension of this model to the examination of homicide victimization is significant because we are able to test the utility of the model across a longer time span (1930 to 1995) and a wider range of ages (10 to 79) and disaggregated by sex and race (Whites and non-Whites). Although the results indicate that past and recent shifts in age-period-specific rates of homicide deaths are associated with specific characteristics of cohorts, there are some important differences across race and sex groupings in the effects of these characteristics. The effects of the cohort variables examined in our model are independent of age and period, often substantively large, and last throughout the life course. The results are consistent with Durkheimian explanations of lethal violence, hypotheses from victimization theory, and basic tenets of cohort theory. © 2002 Elsevier Science (USA)

Recent articles by O’Brien et al. (1999) and Savolainen (2000) show how changes over time in the age distribution of arrests for homicide offending are associated with two characteristics of birth cohorts. Both studies find a weak effect for relative cohort size (RCS) and a more substantial effect for measures that tap the cohorts’ childhood family structure. Their results indicate that these two cohort characteristics almost completely account for variations over time in
age-period-specific rates of homicide offending. Others (e.g., O’Brien, 1989; and to some extent Steffensmeier et al., 1992), examining time periods before the most recent upsurge in youthful violence and who do not include family measures in their models, find a positive, though weak, relationship between homicide offending and relative cohort size.

While the works cited above dealt with age-period-specific patterns of homicide offending, this article examines age-period-specific patterns of homicide victimization or deaths. Specifically, we examine the extent to which cohort characteristics can account for variations in age-period-specific rates of homicide mortality and, thus, changing patterns in the age distribution of homicide victimization rates. This might seem like a minor difference in the choice of a dependent variable; however, using the homicide death rate series has a number of advantages. Specifically, in contrast to the work on offending cited above, use of the homicide mortality series allows us to (1) disaggregate our analyses by sex and race, (2) begin our series in 1930 rather than in 1960, and (3) extend the range of age groupings in our analyses (from 5-year age groupings between 15 and 49 to 5-year age groups between ages 10 and 79). Thus, we are able to replicate and significantly extend previous analyses.

Using an age-period-cohort characteristic model, our results indicate that, like homicide offending, age-period-specific rates of homicide victimization are associated with two cohort characteristics. Members of birth cohorts that are relatively large or that have larger numbers of nonmarital births are at a higher risk of dying from homicide throughout their life spans. These cohort characteristics are associated with the upturn in youth homicide deaths that occurred in the mid-1980s as well as variations in earlier years. In addition, the analyses demonstrate the utility of the model for explaining changes over time in homicide death rates for both the total population and race–sex subgroups, although there are some significant differences in these disaggregated analyses.

RELATED LITERATURE

This article draws on three traditions: (1) sociological analyses of crime within a broad Durkheimian tradition; (2) literature within the subfield of victimology, which has documented a strong relationship between the age of homicide offenders and the age of homicide victims; and (3) the tradition of cohort theory and research.

Durkheimian Analyses of Homicide

Those employing a cohort approach to examine shifts in the age distribution of homicide have used at least three different theoretical traditions that build on Durkheim: control theory, general strain theory, and social capital theory. Other theories might be used—but these theories predominate in the published literature. Advocates of these traditions have argued among themselves regarding which approach most truly follows in Durkheim’s footsteps (e.g., Hirschi, 1969;
Agnew, 1997), but in the literature we review each of these approaches leads to the same general hypothesis. 

**Control theory.** This tradition suggests that external and internal social controls are crucial to maintaining low levels of deviant behavior. The more social ties that individuals have with conforming others, the more likely they will be to conform. Strong social ties with parents in the formative years especially promote internal social control. Strong social ties throughout life also promote external social control; individuals who have more stable and positive relationships with others will be less likely to violate group norms.

Using Durkheimian concepts, one might say that social ties that provide high levels of integration and regulation, and thus lower levels of anomie, are crucial for the development of self-regulation as well as effective control by others within the environment. Travis Hirschi, a prominent contributor to control theory, explicitly recognizes Durkheim’s contribution to this tradition: “Durkheim’s theory is one of the purest examples of control theory: both anomie and egoism are conditions of ‘deregulation,’ and the ‘aberrant’ behavior that follows is an automatic consequence of such deregulation” (Hirschi, 1969, p. 3; see also Gottfredson and Hirschi, 1990; Stafford and Gibbs, 1993).

**Strain theory.** Robert Merton’s original development of strain theory (Merton, 1938, 1968) has become broadened and specified in recent years in a tradition known as “general strain theory” (e.g., Agnew, 1997). Merton suggested that deviant behavior can be seen as resulting from the conflict between culturally defined goals and the institutionalized means to obtain those goals and the particular situations in which individuals are prevented from achieving culturally defined goals through institutionalized means. These situations are anomic, or lacking in social regulation, and are more likely to promote some type of deviant behavior. As Durkheim noted, “To pursue a goal which is by definition unattainable is to condemn oneself to a state of perpetual unhappiness” (Durkheim, 1951, p. 248). Building on Durkheim, strain theorists suggest that the absence of societal regulation is a source of strain to the individual, which can, in some instances result in lethal violence. Whether this strain results in deviance directed against others or involves aggression against the self (as in suicide) depends on the extent to which individuals blame themselves or others for their inability to obtain the desired goals (Agnew, 1997, p. 44).

**Social capital theory.** The concept of social capital was popularized by James Coleman (1988, 1990; Coleman and Hoffer, 1987) and refers to the resources that individuals garner from their relationships with other people. Hagan and McCarthy, who have used the concept to analyze criminal behavior, note the following:

... social capital refers not to a single entity but to a variety of resources. These resources originate in the social structured relations that connect individuals to families and to aggregations of other individuals in neighborhoods, churches, schools, and so on. Social capital is therefore embodied in relations between people, and it includes the knowledge
and sense of obligations, expectations, trustworthiness, information channels, norms, and sanctions that these relations engender. (Hagan and McCarthy, 1997, p. 130)

To use the more traditional Durkheimian terms, individuals have abundant social capital when they are in situations that are highly integrated and regulated. Hagan and his associates have demonstrated how youth with abundant social capital are less likely to be involved in delinquent activities, while those who lack social capital are more likely to have such experiences (e.g., Hagan and McCarthy, 1997; see also Short, 1997, pp. 56–61, 122–126).

As we discuss below, the researchers who use cohort theory to explain shifts in the age distribution of homicides employ different combinations of these three theories to explain shifts in the age distribution of homicides. They all, however, lead to the same general hypothesis: that lethal violence will be more prevalent in situations with less integration and regulation. Both integration and regulation reflect the presence of strong social ties and supportive social networks, that is, situations that produce more social capital, less social strain, and more social control. To the extent that individuals interact in social systems that embody stronger social relationships, or have greater amounts of social capital, or have less social strain, these theories suggest that they will experience greater integration and regulation and will be less likely to experience any type of lethal violence—either as aggressor or as victim.

**Homicide Offending and Victimization**

Researchers within the subfield of victimology have consistently found that homicide offenders and homicide victims tend to have similar demographic characteristics. For instance, the vast majority of murders occur within racial–ethnic groups; the majority of both offenders and victims are male; both offenders and victims have often had prior histories of violent and assaultive behavior; within the majority of cases offenders and victims know each other; and, most importantly for our work, offenders are about the same age or slightly younger than their victims (see Cook and Laub, 1998; Decker, 1993; Karmen, 1990; Luckenbill, 1977; Miethe and McCorkle, 1998; Prothrow-Stith, 1991, pp. 136–138; Short, 1997, pp. 42–44; Singer, 1981; Wolfgang, 1958, 1972). Homicide offenders and homicide victims also tend to share similar lifestyles and personality traits, including a propensity to engage in risky behavior such as frequenting public places, especially at night, and adopting interactive styles that can lead to aggressive interactions and outcomes (Gottfredson and Hirschi, 1990; Hindelang, Gottfredson, and Garofalo, 1978; Miethe and Meier, 1994; Short, 1997, pp. 139–140). Researchers in the victimology tradition stress that statistics regarding homicide offenders and homicide victims represent the outcome of social interactions—the “culmination of an intense interchange between an offender and victim” (Luckenbill, 1977, p. 177) and one in which “. . . it is extremely difficult to find instances of wholly ‘innocent’ and uninvolved victims” (Avison, 1973,
Given these similar demographic and behavioral characteristics of homicide offenders and victims it is not surprising that data at the macrolevel document a relationship between homicide offending and age that is similar to that between homicide victimization and age. Homicide offending has historically tended to peak in the early twenties, with lower levels of offending before and after that age (Gottfredson and Hirschi, 1990), while homicide victimization has tended to peak at a slightly older range (25–29 years of age). Importantly, trends in the age of homicide offenders and homicide victims have been very similar over time for both Blacks and Whites (Cook and Laub, 1998). As with homicide offenders, substantial recent increases occurred in the relative homicide death rates of those 15 to 19 compared to those in older age groups. Since the late 1980s both homicide victimization rates and homicide offending rates have increased dramatically for teenagers and young adults while declining for older age groups (Fox and Zawitz, 1998).

Based on these strong similarities in the characteristics of homicide offenders and victims as well as the strong similarities of macrolevel trends in the age distributions of homicide victims and offenders, we hypothesize that variables influencing the age distribution of homicide offenders also influence the age distribution of homicide victims.

It is important, however, to note some differences in the characteristics of victims and offenders as well as changes that have occurred over time. For instance, women traditionally have been much more likely to be killed by men, often their intimate partners, than to be killed by women. Men are far less likely to be killed by a member of the other sex or by their intimate partner. The number of deaths attributed to intimate partners has declined substantially since the 1970s, although this decline has been far less dramatic for White women than for White men or non-White men and women.1 There are also age differences in the

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1 From 1976 to 1999, for example, the number of males murdered by an intimate partner fell sharply from 1357 to 424 while the number of females murdered by an intimate partner fell less dramatically from 1600 to 1218 (the data reported in this footnote come from Bureau of Justice Statistics, 2001). The decrease in male victims of female partners is dramatic and represents a change in the percentage of male homicide deaths at the hands of an intimate offender from 9.6% in 1976 to 3.6% in 1999. The shift for female victims is much less dramatic: 34.9% in 1976 to 32.1% in 1999. These figures indicate another important difference in homicide victimizations involving men and women. Nearly one-third of all women victims are killed by an intimate, while less than 10% of men victims are killed by an intimate. The declines in intimate homicides impact age groups differently. Over the period 1976 to 1999 intimate violence accounted for 2% of the murders of males from 18 to 24 and 11% for males 45 to 49. For females 18 to 24, intimates murdered 29% of the homicide victims while 40% of those 45 to 49 were murdered by intimates. These trends are not the same across race: From 1976 to 1999 the number of intimate homicides involving White males fell from 493 to 221, while for non-Whites the drop was much more dramatic 864 to 211. For female victims the decline for Whites over the same periods was slight from 849 to 812, but for non-White females the decline was more substantial from 751 to 400. Most theorists would attribute many of these shifts to
trends of homicide victimization. Older people, when they are victims, are more likely than younger people to be killed by intimate partners and thus the decline for men and non-Whites in these deaths could affect these groups more than others. In addition, from the 1970s to the 1990s, young people have been much more likely to die from handguns and older victims less likely to die from this type of assault.\textsuperscript{2} Our analysis includes strong controls for age and period, and we disaggregate the data by race and sex to help control for some of these trends and differences between groups.

\textit{Cohort Theory and Research}

In their analysis of cohort variations in homicide offending O’Brien, Stockard, and Isaacson (1999) identify two major tenets of cohort theory and research: the “life stage principle” and the “lasting effects principle.” The “life stage principle” posits that the experiences of members of one cohort differ from those of another because they experience historical events at different ages or developmental periods. Infants experience historical events, such as the enfranchisement of women or the transition to a market economy differently than those who are 21, those middle aged, and those who have retired (Elder and Caspi, 1990; Elder, 1974; Elder, Modell, and Parke, 1993; Firebaugh and Chen, 1995). The “lasting effects principle” posits that certain events can produce lasting changes in the attitudes and behaviors of cohort members. These changes can last throughout the lives of cohort members. To demonstrate cohort effects, researchers must show that the effects associated with cohorts are distinct from those associated with age and period. In a statistical sense, the effects must appear after the effects of age and historical period are controlled. Showing a cohort effect exists, however, is different than explaining why such an effect occurs.

Two explanations of the effects of relative cohort size predominate in the literature investigating the age distribution of homicide. One, which builds most directly on Richard Easterlin’s (1987) classic discussion, focuses on economic disadvantages that may face members of larger birth cohorts. When members of larger cohorts reach adulthood, they face more competition for jobs and diminished chances of economic security, which, in concordance with strain theory, could promote higher levels of deviant behavior (see Savolainen, 2000, p. 118, greater economic independence of women that allows them to leave unhappy or violent relationships and perhaps to a cultural shift in the acceptability of violence in domestic situations (O’Brien, 1999; Rosenfeld, 1997; Rosenfeld, 2000).

\textsuperscript{2} Disaggregated data from the Bureau of Justice Statistics (2001) show important trends in the type of weapon used in homicides. Although handguns were the predominant weapons used in homicides throughout the period from 1976 to 1999 an upsurge in the use of handguns occurred from the mid-1980s through the early 1990s and this upsurge was greatest among youth 14 to 24. These shifts have been dramatic. For example, in 1976 57\% of the homicides with victims between the ages of 14 and 24 involved a handgun while 69\% of those with victims 25 or over involved a handgun; those figures escalated to 77\% for those 14 to 24 and dropped to 60\% for those 25 and over by 1993. Since that time they have declined to 71\% for youth and to 56\% for those 25 and over.
who cite Menard and Elliot, 1990). The second explanation is more directly tied to the traditions of control theory and social capital theory. It suggests that members of larger cohorts may overload institutions of social control simply because they do not have (per person) as many adults figures in their lives, such as parents, teachers, school counselors, or ministers, as smaller cohorts have. As a result, they may experience less social regulation and integration, less social capital, and thus higher levels of deviant and criminal behavior (O’Brien et al., 1999; O’Brien, 1989; Steffensmeier et al., 1992).

Explanations of how the family structure influences cohort variations in deviant behavior also have drawn on theories within the Durkheimian tradition. These explanations focus on the disadvantages that face children who are born out of wedlock, experience family disruption, or who grow up in single-parent families. For instance, Savolainen notes that childhood family structure is a major correlate of “economic disadvantage, which is the critical variable in the strain-theoretical explanation of crime” (Savolainen, 2000, p. 123). O’Brien et al. (1999) also note these financial disadvantages, but extend their argument to encompass the central role of family structure in ensuring that children experience monitoring and supervision, key elements in both the development of internal social control (self-control) as well as a major source of external social control. In line with social capital theory they also describe the ways in which family structure influences the social networks in which children are embedded and especially the likelihood that they will experience social closure and thus more social integration and regulation.

While Easterlin’s discussion of relative cohort size and its effects on cohorts could serve as an exemplar of a social science theory framed at the aggregate level, many discussions of the influence of family structure are couched at the individual level. The effects of changes in family structure, however, are not limited to children growing up in the affected families. O’Brien et al. (1999) note that while part of the influence of more single-parent families on cohorts comes from the simple aggregation of effects that occur within such families, another part of the influence involves factors that are transmitted across such family groupings. For example, not only are two-parent families more likely to be able to supervise and monitor their children (ceteris paribus), they also are more likely to contain an adult who knows one or more parents of their children’s friends and who has the time and opportunity to be acquainted with teachers. Thus, two-parent families increase the potential range of network ties and closure with others in the community. Cohorts with more two-parent families are more likely to be parts of “closed systems” (Coleman, 1990). O’Brien et al. (1999) also note that this conceptualization is consistent with Sampson (1987), who emphasizes the importance of community social organization and notes that increased family disruption decreases the effectiveness of both formal and informal social controls within communities. Importantly, all children, whether or not they are members of a disrupted family, are influenced by the decreased social control or
“social disorganization” that appears in environments with a greater proportion of disrupted families (Sampson and Wilson, 1995, p. 44).

Based on the linkages developed in the victimization literature between criminal offending and victimization, we hypothesize that, just as relative cohort size and changes in the family structure of cohorts’ childhood families are related to cohort variations in age-specific homicide offending, they will also be related to cohort variations in age-specific homicide deaths. We note again that the various explanations of this relationship in the literature can be derived from Durkheim. The different explanations in this literature suggest that these relationships arise because members of larger cohorts and cohorts with larger proportions of “disrupted families” during childhood are more likely to experience structural strain, lower levels of social control, and diminished levels of social capital.

We predict that the relationships of relative cohort size and childhood family structure to age-period-specific homicide rates will hold for the population as a whole and for separate race and sex groups. These population specific analyses provide “replications” and extensions of previous work in the area. The extensions are important not only because of the greater age range and time span covered, but also because homicide victimization rates differ markedly between the sex groups and racial–ethnic groups. Men’s homicide death rates are much higher than women’s rates and non-Whites have higher rates than Whites. We expect, however, that variations across time in the age-period-specific homicide rates of each of these four groups (White men, White women, non-White men, and non-White women) will be related in similar ways to variations in cohort characteristics.

Given these theoretical formulations and the results of past studies involving homicide offenses, we expect these cohort characteristics will be related to homicide victimization rates throughout the life span of the cohorts. Some authors (Kahn and Mason, 1987; and Steffensmeier et al., 1992), however, argue that the effects of relative cohort size should be especially pronounced for those who are young, when they are most in need of support from families and communities and perhaps more “vulnerable to social and interpersonal adversities” (Diekstra, 1995, p. 236). At the same time, Pampel (1998) notes that larger cohorts may have more political clout when they age and thus secure advantages that would make their situation better in relatively large older age groups. In addition, larger cohorts may provide more social and same-age peer support in older years than smaller cohorts do (see also McCall and Land, 1994). We therefore test the possibility that the effect of cohort size varies at both younger and older age ranges.

**METHODOLOGY**

We draw our data from U.S. government publications. These sources provide information on cohort characteristics and homicide death rates for cohorts born
between 1915 and 1985 and periods ranging from 1930 through 1995. We analyze results for both the total population and separately for White and non-White males and females and, except in the analyses of the total population, employ race-specific measures for the cohort characteristics and age-period-race-sex specific measures of homicide victimization.

Measures

To represent cohorts, we use two variables: relative cohort size (RCS) and, as a measure of family structure, the percentage of nonmarital births within a birth cohort (NMB). The operationalizations of these cohort characteristics are similar to those of O’Brien et al. (1999), but we are able to disaggregate these indicators by race.

Relative cohort size is operationalized as the percentage of the population ages 15 to 64 in the cohort when the cohort is ages 15 to 19. This measure corresponds closely with those used by Savolainen (2000) and Steffensmeier et al. (1992) and is the same as that used by O’Brien et al. (1999).3 The Current Population Surveys: Series P-25 (United States Bureau of the Census, various dates) supplies the data for calculating RCS.4 Again, we calculated these percentages for the total population and for Whites and for non-Whites separately.

Our measure of the family structure of cohorts’ childhood families is the percentage of nonmarital births associated with a birth cohort, the measure used by O’Brien et al. (1999). Although it differs from the measure used by Savolainen (2000), it is very highly correlated with that measure.5 Importantly,

3 Steffensmeier et al. (1992) define RCS as the percentage of the population from 15–64 who are 18 when the cohort is 18. This corresponds to their use of data on homicide arrests that have been interpolated from five-year age groupings for those 25 to 49 (see our footnote 7). Nevertheless, Steffensmeier et al.’s (1992) definition of RCS is conceptually close to the one we use for five-year age groupings as is that used by Savolainen. Savolainen’s (2000, p. 125) measures RCS as the “number of people in ages 15 to 19 divided by the number of those in ages 20 to 64 in the year when the cohort members were 15 to 19 years old.” Savolainen (2000) uses an odds ratio approach to the measurement of RCS, while O’Brien et al. (1999) use a percentage approach. Other than that, the two measures are equivalent. If we let p represent the percent measure used by O’Brien et al. (1999) and r represent the ratio measure used by Savolainen (2000), then we can easily transform p into r using the following formula: \( r = p/(100 - p) \). The transformation is nonlinear, so regression results using one or the other measure might differ. In the analyses that we perform in this article no substantively important differences occur based on the use of r rather than p. We report results based on p.

4 RCS for the youngest cohort in our analysis (those ages 10–14 in 1995) was estimated by calculating the percentage of the population ages 10–59 that was 10–14 in 1995.

5 Savolainen (2000) used Public Use Micro Sample (PUMS) census data for the percentage of those in 5-year birth cohorts who lived in single parent families when they were ages 5 to 9. Because these data are only available for the years 1910, 1940, 1960, 1970, 1980, and 1990 he estimated this cohort characteristic for the years 1915–1939, 1941–1959, and for single years between the other decennial censuses. He then aggregated these estimates into figures for 5-year birth cohorts. The interpolated data that he used were not broken down by race. The correlation of our measure of NMB for the total group with Savolainen’s measure is .98. The correlation between the first differences of these two measures is .90.
using this operationalization, data are available for each of the birth cohorts disaggregated for Whites and non-Whites for this relatively long time span. Two different volumes of Vital Statistics of the United States (United States Bureau of the Census 1946, 1990) supply data for NMB (the number of births to unwed mothers per 100 live births). The 1946 volume provides data for the years 1917–1940 while data for the remaining years were drawn from the 1990 volume. To obtain the NMB for each cohort, we summed the appropriate percentages and divided by the number of years. For example, to obtain the NMB for those 10–15 in 1950, we summed the percentage of nonmarital births for the years 1935 through 1939 and divided by 5. We calculated the NMB for the cohort born between 1915 and 1919 by summing the NMB for 1917, 1918, and 1919 and dividing by 3 because data on the percentage of nonmarital births were not available for those born in 1915 and 1916. We calculated all other NMB values based on 5 years of data.

Age-period-specific homicide death rates per 100,000 were calculated from homicide death data [U.S. Department of Health, Education, and Welfare (various years)] and population data [U.S. Bureau of the Census (various years)]. We use data from 1930 to the present for 5-year age groups from 10 to 79 for the total population, White males, White females, non-White males, and non-White females. The series begins in 1930 for the age group of 10 to 14 years. This corresponds with the availability of data on nonmarital births, which first became available for the cohort born between 1915 and 1919 in 1930 when it was 10–14 years of age.

Analysis

Figure 1 contains the data used in our analysis of the age-period-specific homicide death rates for the total population. The matrix is triangular because data on nonmarital births are not available for cohorts born before 1915–1919. The rows and columns indicate period and age while each cell contains the age-period-specific homicide victimization rate.

While the rows represent periods and the columns represent ages, the cohorts are represented by the diagonals that run from the upper left to lower right. Cohort 1 was born between 1915 and 1919, cohort 2 between 1920 and 1924, and

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6 Not until the 1970s does Vital Statistics of the United States provide a breakdown of nonmarital births that distinguishes African Americans from others in the non-White category. If we insisted on examining the relationship between nonmarital births and homicides deaths for African Americans, we would need to begin our analysis with the 1970–1974 birth cohort rather than the 1915–1919 birth cohort.

7 Steffensmeier et al. (1992) analyze single-year cohorts rather than multiyear cohorts. This makes it necessary for them to interpolate single-year arrest rates for those 25 and older from the UCR arrest data, which do not report single year rates for those 25 and older. Our use of multiple year cohorts not only avoids such interpolations, but also more closely conforms with Easterlin’s contention that the analysis of cohort effects should involve the grouping of individuals born in more than a single year (see O’Brien et al., 1999, p. 1068; Easterlin, 1987, p. 7)
so on. The last cohort in our analysis (cohort 14) was born between 1980 and 1984. The uppermost diagonal (with 14 entries) represents cohort 1; the second diagonal (just below the diagonal that represents cohort 1) contains the entries for cohort 2; the bottom left-hand cell represents the single entry for cohort 14. The marginal at the bottom of the table contains two values that remain the same for each cohort over time. The two entries represent, respectively, the relative cohort size when the cohort members were 15–19 (top) and the percentage of the cohort members who were born to unwed mothers (bottom).

In addition to these data, we analyze data for non-White women, non-White men, White women, and White men. For each of these five data sets, we analyze

### FIG. 1

The age-period-specific homicide rate per 100,000 appear in each cell. The bottom marginal gives the data for each cohort (bolded middle number), with relative cohort size at the top and the percentage of nonmarital births at the bottom.
all of the data in the “triangular” data matrix for each of these groups, with 1 case from 1930 and 14 cases from 1995. These analyses include all of the cases for which we have the appropriate available data for the United States. Because there are so few cases in the older ages and the earlier periods, we also conduct analyses on a subset of each of these data sets to examine the periods after 1945 and the age groups under 60. This provides a test of the sensitivity of our results to periods and ages with so few cases. In another set of analyses we eliminate data for the 1990 and 1995 periods and examine models using data prior to the “epidemic of youth violence.”

In order to control for age effects and period effects and to then identify the factors associated with cohort differences in age-period-specific homicide death rates, we use an age-period-cohort characteristic (APCC) model to analyze these data. The model has been recently refined and extended in the work of O’Brien et al. (1999) and O’Brien (2000), but derives from the work of Mason, Mason, Winsborough, and Poole (1973).

The natural log of the age-period-specific homicide victimization rates serves as the dependent variable in all analyses. We logged the homicide death rates in part because these rates are positively skewed, but we also logged them for theoretical reasons. Our interest focuses on proportional changes in homicide death rates. For example, we are as interested in the doubling of the homicide death rates for those 25 to 29 as for those 55 to 59. Yet, because rates for those in the 25 to 29 age group are often much higher in magnitude, examining the raw data would not allow us to capture this similarity. Given that we control for the effects of age and period in the analyses, the conditional effect in logged form of a doubling of the raw rates is of the same magnitude whether the rate increases from 3.5 to 7.0 or from 10 to 20 per 100,000.

Thus in our analyses the log of the age-period-specific homicide death rate appearing in each cell of the triangular matrix constitutes the dependent variable. This dependent variable is regressed on the time periods, age groupings, and the measures of RCS and NMB associated with that cell. For an analysis involving age-group dummy variables, period dummy variables, relative cohort size, and the percentage of nonmarital births there are \( (14 - 1) + (14 - 1) + 1 + 1 = 28 \) independent variables. These are used to predict \( (14 \times 15) / 2 = 105 \) logged age-period-specific homicide death rates. We first test our model with these observations. When we test our model eliminating observations prior to 1950 and for ages 60 and above (leaving 85 observed cases), it allows us to see if the small number of cases that happen to fall in these periods and age groups affects our results. Finally we test our model after eliminating the periods 1990 and 1995 from the full data set. This permits us to examine how the model behaves when

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8 Including variables that measure cohort characteristics rather than dummy variables for each cohort avoids the problem of linear dependency that would occur if one included dummy variables for \( P - 1 \) time periods, \( A - 1 \) age groupings, and dummy variables for the \( C - 1 \) cohorts, a problem noted by Mason, Mason, Winsborough, and Poole (1973).
the years in which the epidemic of youth homicide occurred are eliminated from the analysis. This analysis has 78 observations.

An important strength of the APCC model is the inclusion of dummy variables for both age groups and periods. By using dummy variables these controls do not assume that the effects of age or period are linear, quadratic, or some other function, but instead control for the effects of age and period as completely as possible given the design we employ. In addition, the dummy variables for period control for the effects of many variables not explicitly included in our model. To the extent that the effects of variables such as news coverage surrounding homicides, changes in medical technology, changes in law enforcement strategies, and downturns in the economy are relatively constant across age groups, then the effects of these variables are controlled for by the period dummy variables. The same argument applies to the age-group dummy variables, which control for factors associated with age. Thus, including these controls provides a clearer indication of the effect of cohort related variables.

Unlike previous analyses of homicide offending, our focus on homicide deaths allows us to disaggregate our analyses by race (White vs non-White) and sex. We do this by running separate analyses for the four possible combinations of race and sex, thus “replicating” our analysis on different demographic groups. For each of these groups the age distribution of homicide deaths has changed over time. If our model is robust and includes appropriate variables, it should be able to account for these changes using variations in the cohort characteristics associated with these groups.

Since each cohort’s death rates “move diagonally” in Fig. 1 (across the two dimensional space of time and age) and are based on (mainly) the same group of people, there is a possibility that the residuals of these observations are not independent. For example, cohort 6 in Fig. 1 contributes nine observations to the

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9 More technically, we control for variables associated with period that are constant across age groups and variables associated with age that are constant across periods. To the extent that some of these variables are only relatively constant, the control is incomplete. But the control extends to variables associated with period and age that are not explicitly included in the equation.

10 We could have represented age as a continuous variable with, for example, the mean age of each age group. If we thought the effect of age were curvilinear, we might have included a quadratic age component. This would save degrees of freedom in comparison with our dummy variable representation. Similarly, one can represent periods with linear or higher order polynomial terms. But the most complete control for the main effects of age and period are obtained by our dummy variable representation. The importance of controlling for the main effects of age and period when examining cohort effects is highlighted in recent exchanges on cohort effects and changes in vocabulary scores over time (Wilson and Gove 1999a, 1999b; Glenn 1999; Alwin and McCammon 1999).
data: the first in 1955 when it was age 10 to 14 and the final one in 1995 when it was age 50 to 54. Differences in cohorts that affect their age-period-specific homicide death rates that are not predicted by the independent variables in our model will result in residuals within the cohorts that exhibit a systematic relationship to each other. Anything about a particular cohort that makes its age-period-specific rates higher (or lower) than expected, given the independent variables, will result in its residuals being positive (or negative).

O’Brien et al. (1999) developed a test for “autocorrelation due to cohorts” to detect this nonindependence. They also suggest a method for ameliorating this problem that involves inspecting the pattern of residuals within cohorts and adding a dummy variable for the cohort contributing most to the nonindependence. Because this procedure is ad hoc, we propose a different method to deal with nonindependence within cohorts. We use the “cluster” option in STATA (StataCorp, 1997) that allows us to specify the cohorts as clusters. This option allows the analyst to conduct an OLS regression analysis without requiring the observations to be independent within the cohorts. The regression coefficients are the same as with a regular OLS analysis, but the standard errors of the coefficients are corrected for nonindependence within the cohorts.

RESULTS

Descriptive Statistics

Table 1 provides descriptive statistics for each of the variables in our analyses for the total group and each race–sex subgroup. Each of our measures shows substantial variation. For the total population the homicide death rates vary from 0.5 for those 10–14 in 1960 to 22.5 for those 20–24 in 1990. The range is greater for non-White males (148.40) and non-White females (27.66) and less for White males (19.54) and White females (5.18). Non-White males have the highest average homicide death rate across the periods and ages in the sample (62.65 per 100,000), while White females have the lowest (2.47). Comparable figures for non-White females and White males are 13.22 and 7.94 per 100,000.11

Examining the percentage of nonmarital births, we see that the range for the total population is 2.1% for cohort 1 to 19.6% for cohort 14 (see Fig. 1). It is important to note, however, that the increase in this variable is not linear or even monotonic. The cohort born between 1935 and 1939 (cohort 5) has a value higher than any of the cohorts born between 1940 and 1954 (cohorts 6, 7, and 8). In addition, the percentage of nonmarital births differs greatly between Whites and non-Whites. The percentages for Whites range from 1.30 to 12.18 and for non-Whites from 11.86 to 49.28. Even with these differences, the pattern of fluctuations in the percentage of nonmarital births over time is similar across these groups.

11 All means and standard deviations are based on unweighted data, for example, the means and standard deviations of the marginal values at the bottom of Fig. 1 for RCS or for NMB.
Relative cohort size for the total population varies from 10.42 to 15.33. Cohorts 12, 13, and 14 (born in the 1970s and early 1980s) and cohorts 4 and 5 (born in the depression years of 1930 to 1939) have the smallest relative cohort sizes. The post-World War II baby boom cohorts (cohorts 7, 8, and 9), born between 1945 and 1959, have the largest relative sizes. The relative cohort sizes for non-Whites (with a range from 12.10 to 18.60) tend to be higher than those of Whites (with a range from 10.10 to 14.90). This reflects a slightly higher birth rate and also higher death rates for non-Whites, both of which reduce the proportion of older adults within the population. Again, the pattern of fluctuation of relative cohort size over time for Whites and non-Whites is quite similar.

Figure 2 presents the age distribution of homicide deaths for the age groups we use in our analyses. The rates for the age groups were calculated from the data in Fig. 1. Thus the rate for ages 10–14 is the average of the 14 rates in Fig. 1 and that for ages 75–79 is based on only a single observation. The resulting age distribution is not unlike the age distribution reported for those arrested for homicide offenses except that the mode is 25–29 for homicide deaths and is typically 20–24 for homicide offenders. If the age distribution of homicide deaths were “invariant,” the level of homicide deaths would shift from period to

---

**TABLE 1**

<table>
<thead>
<tr>
<th>Variables</th>
<th>Total</th>
<th>White</th>
<th></th>
<th>Non-White</th>
<th></th>
</tr>
</thead>
<tbody>
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<td></td>
<td></td>
<td>Male</td>
<td>Female</td>
<td>Male</td>
<td>Female</td>
</tr>
<tr>
<td>Homicide rate</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean</td>
<td>9.24</td>
<td>7.94</td>
<td>2.47</td>
<td>62.65</td>
<td>13.22</td>
</tr>
<tr>
<td>SD</td>
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<td>5.04</td>
<td>1.24</td>
<td>39.97</td>
<td>7.81</td>
</tr>
<tr>
<td>Range</td>
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<td>.36 – 19.90</td>
<td>.22 – 5.40</td>
<td>2.00 – 150.40</td>
<td>.74 – 28.40</td>
</tr>
<tr>
<td>Nonmarital birth rate</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean</td>
<td>4.34</td>
<td>2.26</td>
<td></td>
<td>17.70</td>
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</tr>
<tr>
<td>SD</td>
<td>2.96</td>
<td>1.61</td>
<td></td>
<td>7.57</td>
<td></td>
</tr>
<tr>
<td>Range</td>
<td>2.10 – 19.60</td>
<td>1.30 – 12.18</td>
<td></td>
<td>11.86 – 49.28</td>
<td></td>
</tr>
<tr>
<td>Relative cohort size</td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Mean</td>
<td>12.93</td>
<td>12.64</td>
<td></td>
<td>15.03</td>
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<tr>
<td>SD</td>
<td>1.57</td>
<td>1.58</td>
<td></td>
<td>1.76</td>
<td></td>
</tr>
<tr>
<td>Range</td>
<td>10.42 – 15.33</td>
<td>10.10 – 14.90</td>
<td></td>
<td>12.10 – 18.60</td>
<td></td>
</tr>
</tbody>
</table>

*a The age-period-specific death rates are based on the 105 cell entries (e.g., see Fig. 1 for the 105 cell values for the total population). For RCS and NMB the summary statistics are based on the 14 cohorts in our analysis.

---

12 Alternatively, we could have calculated this age distribution from a rectangular matrix of the rates for each age across all periods.
period up or down in a proportional manner. The work of Gottfredson and Hirschi (1990) and Hirschi and Gottfredson (1983) can be interpreted as implying this sort of invariance for homicide offenses, suggesting that the age distribution of homicide offending does not change its shape over time. Such a situation would imply that there were age differences and period differences, but no cohort differences. The age distribution of homicide offending, however, dramatically changed shape during the period from the 1980s to the 1990s (Maquire and Pastore, 1998, Table 3.137). This shift in the age distribution of homicide often is referred to as the “epidemic of youth violence.”

The age distribution of homicide deaths, like that for homicide offenses, has changed over time. Figure 3 shows the age distribution of homicide deaths in 1980 and 1995 again using data from Fig. 1. The age distribution in 1980 shows modes in the age group 25–29; in contrast the age distribution in 1995 has its mode at 20–24 and much higher relative rates at 15–19 than was the case in 1980. Just as homicide offenses became relatively more common among younger people during this time period, homicide deaths also became relatively more common among the young. Our analysis tests the hypothesis that changes in the age distribution of homicide deaths, like those shown in Fig. 3, as well as changes involving data disaggregated by race and sex, and for other periods not shown in the figure, are associated with cohort characteristics that are theoretically related to social integration and social regulation.

The Relationship of Age and Period to Homicide Death Rates

We began our analysis by regressing the log of age-period-specific homicide death rates on the age and period dummy variables. The $R^2$ for the analyses...
appear in Table 2. For each set of rates, age and period explain much of the variation. The $R^2$ measures range from .969 for White males to .930 for non-White females. The age dummy variables explain more variance than the period dummy variables: when the period dummy variables are entered into the equation first they explain from 11% (non-White females) to 38% (White females) of the variance in age-period-specific homicide victimization rates.

The Influence of Cohort Characteristics

One way of viewing the age-period model (the model with only these dummy variable sets) is that it constrains the age distribution across different periods to have the same form (although they may differ in levels). We add cohort characteristics to the model to examine whether we can explain changes in the shape of the age distributions over periods, such as the shifts illustrated in Fig. 3. The results from our APCC analyses for the total population and for each race–sex group suggest that the cohort characteristics in our model can help explain these shifts.

When the cohort characteristics are added to the model, the $R^2$ ranges from .993 for the total sample to .972 for White females. The change in $R^2$ when the two cohort characteristics are added to the analyses that contain the age and period dummy variables results in statistically significant ($p < .001$) increments in the variance explained in each of the five analyses. In each analysis NMB is significantly ($p < .001$) and positively related to age-period-specific homicide death rates after controlling for the effects of age groups, periods, and relative cohort size. The results for relative cohort size are not as strong. In each of the five analyses the coefficients associated with RCS are positive, but in two of the analyses, those for White males and White females, they are not statistically
TABLE 2
Logged Age-Period-Specific Homicide Victimization Rates Regressed on Age Dummies, Period Dummies, Relative Cohort Size, and the Percentage of Nonmarital Births and Corrected for Cohort Heterogeneity

<table>
<thead>
<tr>
<th>Variables</th>
<th>Total</th>
<th>White</th>
<th>Non-White</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$b$</td>
<td>$t$</td>
<td>$b$</td>
</tr>
<tr>
<td>Intercept</td>
<td>−.761</td>
<td>−5.045</td>
<td>−.588</td>
</tr>
<tr>
<td>1930</td>
<td>.000*</td>
<td>.000*</td>
<td>.000*</td>
</tr>
<tr>
<td>1935</td>
<td>−.263</td>
<td>−5.167</td>
<td>−.309</td>
</tr>
<tr>
<td>1945</td>
<td>−.572</td>
<td>−11.856</td>
<td>−.452</td>
</tr>
</tbody>
</table>

Age
20 – 24 2.885 56.092 2.717 29.097 1.815 17.890 3.663 45.502 2.938 29.117
25 – 29 3.023 60.136 2.789 31.922 1.843 27.311 3.927 46.136 3.263 33.455
35 – 39 2.918 46.127 2.726 29.732 1.797 20.189 3.899 46.981 3.320 27.360
40 – 44 2.804 41.252 2.610 29.171 1.754 17.695 3.843 47.166 3.183 26.556

RCS* .032 3.347 .011 1.079 .017 1.274 .041 5.897 .059 3.503
NMB* .114 7.873 .133 5.907 .078 3.783 .070 23.331 .069 8.255
N 105 105 105 105 105 105
SE .0923 .1333 .1371 .1238 .1593
$R^2$ Age and Period .964 .969 .962 .956 .930
$R^2$ full model .993 .986 .972 .991 .977
$R^2$ Adjusted .990 .981 .961 .988 .969

* Dummy variable omitted for purposes of estimation.
* These measures are based on the total population, whites, and nonwhites depending on the dependent variable.
significant at the .05 level. This finding shows the importance of being able to disaggregate our analyses by race and gender. Without disaggregated data, we would only be able to interpret the results from the total population in Table 2. Our conclusions would have been similar to those reached by O’Brien et al. (1999). Based on their analyses using homicide arrest data, they concluded that the effects of RCS and NMB on age-period-specific homicide rates were both statistically significant and noted that the relationship of NMB was stronger than that of RCS. Here, we conclude that the relationship between RCS and homicide rates is not significant for either White males or females (though it is in the predicted direction). The relationship of RCS to the age-period-specific homicide rates for both non-White males and females is highly significant ($p < .001$) and for these two groups its importance approaches that of NMB.

When we compare the coefficients for RCS and for NMB from one group to another to see if the differences between them are statistically significant, we find that for RCS the coefficient associated with White males is significantly smaller ($p < .05$, for a two-tailed test) than the coefficients for non-White males and non-White females. None of the other RCS coefficients are statistically significantly different from each other. In the case of NMB, the White male coefficient is significantly larger ($p < .05$) than those for each of the other groups. The coefficients for the other groups (White females, non-White males, and non-White females) are very similar. Thus, for White males RCS has less impact (and is not statistically significant) and NMB seems to have a greater impact, on the age distribution than these two cohort characteristics do for the other groups. We

---

13 To assess the effects of multicollinearity in the analyses in Table 2, we calculated Variance Inflation Factors (VIF). Rawlings (1988) and Chatterjee and Price (1991) suggest that serious collinearity problems do not occur with VIF values less than 10. This same rule of thumb is used in the Stata Manual (StataCorp, 1997, p. 391), which notes that some authors suggest a value of 30. Snee and Marquardt (1984) show that there is no practical difference between a VIF of 10 and the conditioning number of 30, suggested as a criterion by Belsley, Kuh, and Welsch (1980). Still some suggest a lower figure, since a variance inflation factor of 10 indicates that the standard error of the regression coefficient is ($\sqrt{10} = 3.16$) 3.16 time greater than it would be if all independent variables were orthogonal to the independent variable under consideration. The VIF value associated with RCS was 1.8 for non-Whites and NMB was 7.3 for non-Whites. In the case of Whites, the VIF values were 3.5 for NMB and 1.6 for RCS. By almost any criteria the VIF values for Whites are not problematic. Importantly, the standard errors for the coefficients of NMB are much smaller for non-Whites than Whites. This results from the far greater variance in NMB for non-Whites than for Whites (see Table 1). Thus, even in the face of inflated standard errors due to multicollinearity, the standard errors of the coefficients associated with NMB for non-Whites are smaller than those associated with NMB for Whites. Further, the variance explained by our models is always in excess of .97, and this reduces the size of all of our standard errors. This fact is not irrelevant to the rule of thumb one adopts for the size of VIF (Freund and Wilson, 1998). Given the size of the VIF values, the consistency for the coefficients involving NMB across our analyses (especially for non-Whites where the VIF value of NMB is high), we feel confident interpreting the coefficients associated with the coefficients for RCS and NMB.

14 We use the standard $t$ test for the difference between regression coefficients from independent samples that treats the estimates of the standard errors as separate estimates rather than pooling them.
had not predicted differences in the impact of these variables across the different race–gender groups, but being able to disaggregate these data allowed us to discover these differences.

**Interactions**

Because several authors have suggested that the effects of relative cohort size on an outcome may differ for cohort members of different ages (Kahn and Mason, 1987; Pampel, 1996; Steffensmeier et al., 1992), we checked for interactions between age and relative cohort size for both the younger age groups and the older age groups. To do this we created product terms based on the dummy variable for an appropriate age group times the relevant race-specific relative cohort size measure for that cohort when it was that age. For example, for the total population and the age group 15 to 19 in 1950 (members of cohort 4), we multiplied the relative cohort size for their cohort (10.80) times 1. For those 15 to 19 in 1955 (members of cohort 5), we multiplied 10.87 times 1. This process was continued for all of the observations in the age group 15 to 19. In this way we developed an interaction term for the interaction of the age group 15 to 19 with relative cohort size. We then ran the full regression models presented in Table 2 with interaction terms for age groups with relative cohort size for the three age groups from 15 to 29. In a second regression analysis we included these three interaction terms and added four more for the oldest age groups: those 60 and above.\(^{15}\)

The major finding is that none of the sets of interactions, when added to the full models for each sex–race group and for the total population, was statistically significant, although in a few instances an individual interaction term reached conventional levels of statistical significance. Here, we follow Cohen and Cohen’s (1983) “protected \(t\) test” strategy and do not examine the individual coefficients unless they are significant as a set. Not surprisingly, adding the seven interaction terms of age with RCS increases the multicollinearity and reduces the statistical significance of the main effect of RCS in our analyses.\(^{16}\) Still in all cases the coefficients for the main effect of RCS and NMB remained positive and statistically significant for the same analyses for which they were significant in Table 2 (\(p < .05\)). We seem only to be adding multicollinearity to the analysis through this procedure, and our substantive conclusions do not change.

\(^{15}\) Since there was only one observation for those 75 to 79, this case was perfectly predicted by our model without an interaction term. With an interaction term the two observations in the 70 to 74 age group were perfectly predicted.

\(^{16}\) Adding the age by RCS interactions to the full models in Table 2 creates extremely high VIFs for both the interactions and the age groups involved in the interactions. Some of these VIF values exceeded 1000.
We examine whether the effects of having so few cases in the periods before 1950 and in the age groups 60 to 64 and above might exert a strong influence on the results. For example, there is only one case in the age category 75 to 79 and one case in the period 1930. To examine this we eliminated cases before 1950 and cases in age groups 60 to 64 and older.

Results from applying our APCC model to this more limited data set are displayed in Table 3. The results are very similar to those found with the full data set. The coefficients associated with the two cohort characteristics are all in the hypothesized direction, and three of the five coefficients associated with RCS (for the same groups as in Table 2) are statistically significant. Those associated with the measure of nonmarital births are again statistically significant for the total group and all race-sex groups. The magnitudes of the coefficients are very similar to those of the coefficients reported in Table 2, as are the $R^2$ values.

As in the analyses reported in Table 2, the coefficient for NMB for White males is larger than those for each of the other race-sex groups, but none of the other NMB coefficients differs significantly ($p < .05$) from each other. Once again the effect of RCS on the age-period-specific homicide rates for white males and females is not statistically significant. The results comparing the coefficients for RCS in Table 3 are somewhat different than those for the analyses in Table 2. Here, the relationship is significantly different between White males and non-White males and between White females and non-White males ($p < .05$; two-tailed test). None of the other differences is statistically significant.

---

### Table 3

Logged Age-Period-Specific Homicide Death Rates Regressed on Age Dummies, Period Dummies, Relative Cohort Size, and the Percentage of Nonmarital Births

<table>
<thead>
<tr>
<th>Variables</th>
<th>Total</th>
<th>White</th>
<th>Non-White</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$b$</td>
<td>$t$</td>
<td>$b$</td>
</tr>
<tr>
<td>RCS$^a$</td>
<td>.034</td>
<td>3.733</td>
<td>.015</td>
</tr>
<tr>
<td>NMB$^a$</td>
<td>.116</td>
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</tr>
<tr>
<td>$N$</td>
<td>85</td>
<td>85</td>
<td>85</td>
</tr>
<tr>
<td>SE</td>
<td>.0947</td>
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<td>.1252</td>
</tr>
<tr>
<td>$R^2$</td>
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<td>.984</td>
<td>.972</td>
</tr>
<tr>
<td>$R^2_{Adjusted}$</td>
<td>.988</td>
<td>.979</td>
<td>.964</td>
</tr>
</tbody>
</table>

**Note.** The data are truncated to exclude the periods before 1950 and the age groups 60 and above and corrected for cohort heterogeneity.  
$^a$ These measures are based on the total population, Whites, and non-Whites depending on the dependent variable.

---

We have not reported the coefficients associated with age and period dummy variables in order to save space and to ease reading of the table; these coefficients are available on request.
O’Brien et al. (1999), using a model similar to the full model of Table 2, demonstrated that the model held for homicide offending for the total population of the United States for the period from 1960 to 1995 as well as for the period 1960 to 1985. They did this to show that RCS and NMB could account not only for the dramatic shift in the age distribution of homicide that accompanied the epidemic of youth homicide, but could account for the less dramatic shift before that epidemic. To see if our model holds for the periods before the epidemic of youth homicide, we analyze the data on homicide deaths for the total population and for the separate race–gender groups with the two periods after 1985 removed from the analysis. The results are reported in Table 4.

All of the coefficients are again in the predicted direction: cohorts with higher levels of RCS and NMB have relatively higher rates of age-period-specific homicide deaths. If we compare these results to those in Table 2, however, we see some interesting differences. Perhaps the most notable difference in the coefficients between Tables 2 and 4 is that the NMB coefficient for White males is not statistically significant for these earlier periods. For all of the other groups the NMB coefficients are statistically significant ($p < .001$). As before, the RCS coefficient for White males is not statistically significant, but unlike the previous analyses, the coefficient for RCS for White females is statistically significant. Again, if we only had available data for the total population, our results would be similar to those of O’Brien et al. (1999) analysis based on homicide offenses: Both RCS and NMB are statistically significant for the total population, though the relationship of NMB to age-period-specific homicide rates is much stronger than that of RCS.

### Table 4

<table>
<thead>
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<th>Variable</th>
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<th>Female</th>
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<td>$t$</td>
<td>$b$</td>
<td>$t$</td>
<td>$b$</td>
<td>$t$</td>
</tr>
<tr>
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<td>.006</td>
<td>.612</td>
<td>.026</td>
<td>1.957</td>
</tr>
<tr>
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<td>.116</td>
<td>3.450</td>
</tr>
<tr>
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</tr>
<tr>
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</tr>
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</tr>
<tr>
<td>$R^2$ Adjusted</td>
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<td>.986</td>
<td>.962</td>
<td>.992</td>
<td>.977</td>
<td></td>
</tr>
</tbody>
</table>

*Note.* The data are truncated to exclude the periods after 1985 and corrected for cohort heterogeneity.

*These measures are based on the total population, Whites, and non-Whites depending on the dependent variable.

---

### Analysis for Data Excluding Periods after 1985

O’Brien et al. (1999), using a model similar to the full model of Table 2, demonstrated that the model held for homicide offending for the total population of the United States for the period from 1960 to 1995 as well as for the period 1960 to 1985. They did this to show that RCS and NMB could account not only for the dramatic shift in the age distribution of homicide that accompanied the epidemic of youth homicide, but could account for the less dramatic shift before that epidemic. To see if our model holds for the periods before the epidemic of youth homicide, we analyze the data on homicide deaths for the total population and for the separate race–gender groups with the two periods after 1985 removed from the analysis. The results are reported in Table 4.
In Table 4, the RCS coefficient for White males is statistically significantly different from those for both non-White males and non-White females. The NMB coefficients for White females and non-White males differ, as do the coefficients for non-White males and non-White females \((p < .05)\). None of the other coefficients are statistically significantly different.\(^{18}\)

**DISCUSSION AND SUMMARY**

By using homicide death rates calculated from vital statistics rather than homicide arrest rates derived from law enforcement data, we were able to examine relationships between theoretically important characteristics of cohorts and their age-period-specific homicide victimization rates over an extended period of time (1930 to 1995) and ages (10–15 to 75–79) and across race–sex subgroups. Previous researchers, who studied homicide offenders in the United States, were limited to a shorter time period and narrower age ranges and did not have available data disaggregated by sex and race over a long period of time. Overall the results from our analysis of homicide death rates parallel earlier applications of the APCC model to variations in homicide arrest rates (O’Brien et al., 1999; Savolainen, 2000).

Our strongest results involve the relationships between nonmarital births and homicide deaths, which are statistically significant and substantively strong for all analyses except for White males in the period before 1985. In contrast, the relationship between relative cohort size and homicide deaths is statistically significant and substantively important only for analyses involving non-White males and females. Because of the strength of the relationship between RCS and homicide rates in these two groups, this relationship is significant in the total population. In one analysis, in the period from 1930 to 1985, RCS is statistically significantly related to homicide deaths for White females. Although not always the case, the RCS coefficients for nonwhites are often statistically significantly stronger than those for whites.

We are unsure why these differences occur, but they illustrate Pampel’s (2001) conclusion that the influences of demographic and nondemographic forces, such as relative cohort size, on social outcomes is contingent in nature and that demographic and nondemographic forces combine to influence outcomes. As a reviewer of this article suggested, the negative or positive effects that result from demographic characteristics, such as relative cohort size or nonmarital births, may not fall equitably on all individuals and, in a stratified society, might fall more heavily on subordinate groups. In the present case, the data suggest that larger cohort sizes have a more substantial effect on homicide victimization within subordinate racial groups than among the White majority. As shown in Table 1, relative cohort size has been substantially larger for non-Whites than for

\(^{18}\) Note that the \(t\) values do not agree in this case with the absolute difference in the point estimates of the coefficients.
Whites for all of the cohorts in our analyses. Thus, with no demographic changes
the non-White community has had to cope with a smaller adult–child ratio and
in this sense less support for children. At the same time, the non-White com-
munity has fewer resources with which to purchase supports to compensate for
this demographic situation or to cope with increases in relative cohort size when
they occur.

Our results (especially those for nonmarital births) are generally consistent
with explanations of lethal violence that fall within a Durkheimian framework.
These explanations suggest that members of cohorts that are relative large and
cohorts with a high percentage of “disrupted families” while the cohort members
are still young have less social integration and regulation and lower levels of
social capital. The results also are consistent with explanations that stress the
important impact of lower levels of financial resources that are available to
members of cohorts that are relatively large and that have a large number of
single-parent families. These results hold for the analyses involving the entire
time period from 1930 to 1995 and for the analyses of the time period prior to the
epidemic of youth homicide. They also hold when we truncate the data by
eliminating periods before 1950 and age groups 60 and above.

Our results provide support for hypotheses from the victimization literature
that suggests that characteristics of offenders and victims are similar. The
findings using homicide deaths replicate the results of O’Brien, Stockard, and
Isaacson’s (1999) study of homicide offenders quite closely when based on the
total population. For this population, birth cohorts that have relatively high rates
of homicide offenses also have relatively high rates of homicide deaths and the
same cohort variables can explain variations in both sets of rates.

Finally, our results support the importance of a cohort-based approach. It is
possible to consider both changes in relative cohort size (size of the youth
population in comparison with the adult population) and the rate of nonmarital
births as an historical or “period effect.” No doubt, all members of a society are
affected by such changing demographic patterns. Yet, our results indicate that
these demographic changes have a special effect on individual birth cohorts,
independent of their period effects.

These findings greatly extend the more limited research on the relationship
between cohort membership and homicide: albeit, we shift the focus from
homicide offending to homicide victimization. Rather than using data from 1960
to 1995, we use data from 1930 to 1995. Rather than examining data for those 15
to 49 years old, we examine data for those 10 to 79 years old. Rather than
examining data on the total population, we examine data from the total popula-
tion and for non-White males, non-White females, White males, and White
females. In each of our analyses the relationship between nonmarital births and
the logged homicide death rate is stronger than that between relative cohort size
and the logged homicide death rate. There are some important differences among
our disaggregated groups. The most important of these is the diminished, and
statistically insignificant, impact of relative cohort size on homicide rates for most analyses involving White groups.

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