Neighborhood Mortality and Age at First Intercourse among Chicago Adolescents

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ABSTRACT

This paper describes my efforts to test the hypothesis that adolescents initiate sexual intercourse at younger ages when they live in areas characterized by high young adult mortality rates. Using data from the Project on Human Development in Chicago Neighborhoods and other sources, I seek to isolate the effects of neighborhood mortality from the confounding influences of a wide range of individual-, family-, and neighborhood-level variables using a series of multilevel hazard models and stratification on a neighborhood-level generalized propensity score. I find that mortality among young adults in adolescents' neighborhoods has large gross impacts on age at first intercourse for both females and males, but adjustment for individual-, family-, and neighborhood-level variables substantially reduces those effects, often to the statistical null.

BACKGROUND

This paper describes my efforts to test the hypothesis that adolescents have their first experience of sexual intercourse at younger ages when they live in areas characterized by high young adult mortality rates. The work is motivated by recent scholarly interest in the influences of social contexts on human development and behavior, including sexual behaviors. The independent variable whose effect I seek to measure has seldom been considered in this literature. Yet there are compelling theoretical reasons to expect that it may have important influences on adolescent sexual behavior, including timing of first intercourse. I use data from Chicago, Illinois, during the 1990s to test this hypothesis. I devote considerable effort to trying to isolate the effects of young adult mortality rates from the confounding influences of a wide range of individual- and neighborhood-level variables.

Social scientific interest in age at first intercourse spans several decades and is motivated by at least three interrelated factors. The first is concern about teenage sexuality and its possible consequences, which include pregnancy and the repercussions thereof, as well as sexually transmitted infections. This concern is reflected not only in the publication of scholarly books (Hayes 1987; Maynard 1996) and articles (Brooks-Gunn and Furstenburg 1989) on the topic, but also in the agendas of several influential organizations, including the National Coalition to Prevent Teen and Unplanned Pregnancy (Kirby 2001). To be sure, the negative consequences of teenage sexuality may have been exaggerated in this literature. Rates of teenage childbearing have mostly fallen in United States since the 1950s (Ventura, Matthews, and Hamilton 2001), while the negative consequences of a teenage birth for the mother, child, and society as a whole are almost certainly smaller than was once believed (Geronimus and Korenman 1992). Similarly, although it is often claimed that more than half of all HIV infections in the United States occur among youth aged 13 through 24, the overall prevalence of HIV infection among teenagers and young adults remains quite low (Morris et al. 2006). Nevertheless, few would dispute the claim that teenage sexual activity does have some negative consequences.

The second impetus for research on this topic is the well-documented black-white inequality in average age at first intercourse in the United States. In 1971, for example, 57% of never-married African American 17-year-old females reported having had sexual intercourse, compared to only 22% of white females (Kantner and Zelnick 1972). This finding has been replicated in numerous surveys conducted in the 1970s, 1980s and 1990s (Santelli et al. 2000). For over twenty years researchers have been working to explain these variations in terms of socioeconomic and other variables (e.g., Furstenburg et al. 1987).

A third motivating factor is the growing popularity among social scientists of the life course as a conceptual framework for studying processes that link social environments to individual development and behavior. One paradigmatic principle of this framework is the principle of timing, which focuses attention on the developmental antecedents and consequences of the timing of significant life transitions (Elder, Johnson, and Crosnoe 2004). This framework is easily extended to encompass the study of sexuality, including the onset of sexual intercourse (Udry and Campbell 1994). Certainly one's first experience of sexual intercourse can be considered a significant life transition. Moreover, young age at first intercourse is empirically related to a range of prior and subsequent personality, behavioral, and environmental variables (Jessor et al. 1983). From this perspective, then, age at first intercourse takes on rich meaning as one point in a constellation of interrelated life course variables.

Investigators have used explanatory variables from numerous domains to account for variations in the timing of first intercourse among adolescents. During the 1980s, family structural and family process variables received considerable attention (Miller and Moore 1990). Several studies showed that adolescents from single-parent households began having sexual intercourse at younger ages than did adolescents from two-parent homes (Forste and Heaton 1987; Miller and Bingham 1989; Udry and Billy 1987). This pattern was sometimes interpreted as evidence of the importance of family social controls in adolescent sexuality. Other investigators documented associations between measures of mothers' youthful sexual behaviors and the early initiation of sexual intercourse by their adolescent children (Inazu and Fox 1980; Newcomer and Udry 1984; Thornton and Camburn 1987).

Other studies called attention to explanatory variables in other domains. Jessor and colleagues (1983) drew upon problem behavior theory to relate variations in age at first intercourse to a network of personality, attitudinal, environmental, and behavioral system variables. These included the value placed on academic achievement, the desire for independence, religiosity, parental controls, and perceived peer attitudes and behaviors. Udry and Billy (1987) presented a conceptual framework in which the timing of adolescents' transition to first intercourse is determined by the interaction of motivation (with both biological and social components), social controls, and personal attractiveness. Their panel data revealed substantial sex differences in the determinants of sexual initiation. They concluded that variation in the timing of sexual initiation is determined largely by biological factors for males, while social influences including parental controls play a more significant role for females (Udry and Billy 1987; Udry et al. 1985).

In the mid-1990s, the range of potential explanatory variables expanded further to include measured characteristics of adolescents' residential neighborhoods. Brewster and her colleagues modeled age at first intercourse among female participants in Cycle III of the National Survey of Family Growth, appending information about respondents' residential counties and census tracts from several sources (Brewster 1994a, 1994b; Brewster, Billy, and Grady 1993). The results of their analyses were inconsistent. For white women, after controlling for a set of individual-level variables, tract-level indicators of residential mobility and marital instability increased the hazard of first intercourse, while the percentage black and the percentage foreign-born both decreased it. However, several other theoretically important tract- and county-level variables failed to achieve statistical significance in their model (Brewster, Billy, and Grady 1993). Moreover, for black women, no community-level variable had a statistically significant effect on age at first intercourse once individual-level variables were included in the model (Brewster 1994a). And for the combined sample, the only community-level variable with a statistically significant effect net of individual-level controls was a measure of female labor force opportunities, and its effect was in the opposite direction from what Brewster had hypothesized (Brewster 1994b).

In spite of these inconsistent results, these papers brought community-level determinants to the center of scholarly attention. Equally important, they laid out a compelling theoretical framework for understanding how neighborhood social conditions might influence adolescent sexual behavior. Specifically, Brewster and colleagues (1993) argued that "community characteristics define behavioral alternatives and their associated social, psychic, and economic costs, thereby shaping individual perceptions, attitudes, and values that ultimately guide young people's behaviors" (p. 715). Both the local opportunity structure and the community's normative climate may play important roles in this process.

Further research on the neighborhood-level determinants of age at first intercourse was done by teams working in Los Angeles, California, and Chicago, Illinios. In Los Angeles, Upchurch and colleagues found that adolescents' perceptions of ambient hazards in their neighborhoods (e.g., gangs, drug dealing, and drive-by shootings) increased the risk of sexual onset (Upchurch et al. 2001; Upchurch et al. 1999). In Chicago, Browning and his colleagues found that high levels of collective efficacy at the neighborhood level reduced the hazard of first intercourse during early adolescence for both males and females, especially among youth who received relatively little monitoring from their own parents (Browning, Leventhal, and Brooks-Gunn 2005). In other analyses, the inclusion of neighborhood-level variables in a model drove the effect of race/ethnicity to statistical insignificance, whereas controlling only for individual- and family-level variables failed to do so (Browning, Leventhal, and Brooks-Gunn 2004). Taken together, these findings are consistent with the view that social ecological characteristics of neighborhoods may influence the timing of first sexual intercourse, but they leave unresolved the question of exactly what neighborhood-level variables play the biggest roles in this process.

Arguably, although it has yet to be considered in this body of sociological literature, there are compelling reasons to believe that the mortality schedule facing young people in a local population may exert an important influence on the timing of first sexual intercourse. This view is broadly consistent with Brewster's theoretical framework (Brewster, Billy, and Grady 1993) in that the salience to adolescents of the risks associated with early intercourse – the possibility of pregnancy, the threat of HIV and other sexually transmitted infections – may depend substantially upon the local mortality environment. For

adolescents living in neighborhoods characterized by high levels of violent crime, for example, the possibility of eventual death from HIV infection may seem remote compared to the immediate threat of dying from a gunshot wound (see Dow, Philipson, & Sala-i-Martin 1999, p. 1369). Similar arguments have been forwarded to explain ongoing risk behaviors in the context of sub-Saharan Africa's severe HIV epidemics, where competing risks include not only the threat of violence but also hunger and a range of infectious diseases (Zwi & Cabral 1991; Caldwell, 2000). More generally, Wilson and Daly (1997) argue that "life expectancy itself may be a psychologically salient determinant of risk-taking" (p. 1271). Thus, if adolescents make inferences about their own likely survival based upon the mortality environment in their neighborhood, those inferences may in turn influence adolescents' motivation to delay the initiation of sexual intercourse.

Furthermore, the salience of the risks associated with youthful sexual intercourse may depend not only upon adolescents' own perceived mortality risk, but also the risks faced by their parents and other family members. Geronimus, Bound, and Waidman (1999), for example, argue that in impoverished areas of American cities, "early fertility may mitigate the threat to family economies and caretaking systems imposed by the heavy burden of chronic disease and premature death borne by young through middle aged adults" (p. 1633; see also Geronimus 1996). That is, when young women hope to rely upon their own mothers for assistance in caring for their children, their mothers' future health and survival becomes an important consideration in choosing when to bear children. In such a context, high neighborhood mortality would tend to undermine the salience of the thread of teenage pregnancy as a deterrent to youthful intercourse. Consistent with this view, Wilson and Daly (1997) conducted an ecological study of the 77 community areas of Chicago, Illinois, and found that teenage birth rate in the ten neighborhoods with the shortest life expectancy was over four times higher than in the ten neighborhoods with the longest life expectancy.

These arguments provide compelling reasons to believe that neighborhood mortality may influence the timing of first intercourse among adolescents. Yet there have been no serious efforts to date

to measure those hypothesized effects or to isolate them from the confounding influences of other individual-, family-, and neighborhood-level variables. Doing so is the purpose of this paper.

METHODS

Sample and Outcome Data

Most of the data used in this study come from the longitudinal cohort study of youth conducted by the Project on Human Development in Chicago Neighborhoods (PHDCN). The sample was obtained using a two-stage stratified design. First, Chicago was divided into 343 neighborhood clusters (NCs), the NCs were stratified according to race/ethnic and socioeconomic composition, and 80 NCs were sampled from these strata (for more details see Sampson, Raudenbush, and Earls, 1997). In the second stage, study personnel screened over 40,000 households within the sampled NCs and identified 1,103 people within six months of their twelfth birthday, and 972 within six months of their fifteen month birthdays. These individuals were eligible for entry into the 12- and 15-year-old cohorts, respectively (other cohorts not considered here included a birth cohort as well as 3-, 6-, 9-, and 18-year-old cohorts). The majority (N = 1,517) agreed to participate and completed the first round of interviews, for a response rate of 73.1%. Wave 1 interviews were conducted between November 1994 and June 1997, with over 75% of interviews occurring in 1996. All individual- and family-level control variables, described in detail below, were drawn from this first round of interviews. Select characteristics of the sample are presented in Table 1.

[TABLE 1 ABOUT HERE]

The dependent variable in this study is age at first sexual intercourse, assessed in Waves 2 and 3 of the PHDCN cohort study. Wave 2 interviews were conducted between January 1997 and February 2000. Wave 3 interviews were conduced between January 2000 and January 2002. In the middle of these interviews, respondents were asked to self-administer a pencil-and-paper questionnaire covering aspects of their relationships and sexual behaviors. After several questions about dating, attitudes toward sex and

childbearing, and the sexual and childbearing behaviors of their peers, respondents were asked, "Have you ever had sexual intercourse?" Those answering this question affirmatively were they asked, "How old were you when you first had sexual intercourse?" Of the respondents interviewed at Wave 1, n=1266 (83.5%) provided some information about their sexual initiation status at one or both of these follow-up interviews.

Mortality During Young Adulthood

The key independent variable is the rate of mortality among young adults in respondents' residential neighborhoods in 1993. I chose this year because it falls after the 1990 U.S. Census of the Population (from which several neighborhood-level control variables are derived) and just before the first PHDCN Wave 1 interviews. Numerous measures of mortality are available. I chose to use the probability that a neighborhood resident who is alive at her or his 15th birthday will die before reaching age 45. I computed these probabilities separately for males and females, and used the female probability of dying in models of female sexual initiation and the male probability of dying in models of male sexual initiation.

The basic requirement for computing these probabilities is the set of age-specific mortality rates for each sex in each neighborhood (Preston, Heuveline, and Guillot 2001). Denote these as $\mu_{fj}(t)$ and $\mu_{mj}(t)$, where the subscripts *f* and *m* indicate female and male, the subscript *j* indexes the NC, and *t* measures age in years and takes integer values from 15 through 44. For each of the 343 NCs I obtained counts of deaths by sex and single-year-of-age from an electronic database of death certificate data provided by the Chicago Department of Public Health. I summarized these across the calendar years 1992 to 1994, and denote them as $D_{mj}(t)$ and $D_{fj}(t)$. I obtained measures of female and male person-years at risk of dying in each single-year-of-age category and each NC by linear interpolation between the 1990 and 2000 counts by the U.S. Census of the Population. I multiplied these by three because the count of deaths covers a three-year period, and denote the resulting values as $N_{fj}(t)$ and $N_{mj}(t)$.

The standard approach to computing the age-specific mortality rate is simply to divide the number of deaths by the number of person-years at risk. In this application, however, the denominators of those quotients are in many cases quite small, leading to unstable estimates of $\mu_{fj}(t)$ and $\mu_{mj}(t)$. Moreover, the standard procedure ignores the well-established fact that mortality tends to vary smoothly as a function of age. I therefore used a combination of a seventh-degree polynomial and empirical Bayes estimation to obtain more stable estimates. Specifically, for females, I estimated the following generalized linear multilevel model (Raudenbush and Bryk 2002), using age groups 10 through 50:

$$D_{fj}(t) \mid N_{fj}(t), \mu_{fj}(t) \sim \text{Poisson}[\mu_{fj}(t) \cdot N_{fj}(t)]$$

$$\ln[\mu_{fj}(t)] = \eta_{fj}(t),$$

$$\eta_{fj}(t) = \gamma_{0j} + \gamma_{1j}*(t - 30) + \gamma_{2j}*(t - 30)^2 + \ldots + \gamma_{7j}*(t - 30)^7.$$

Using empirical Bayes estimates of the coefficients γ_{0j} through γ_{7j} derived from this model, I computed estimates of the age-specific mortality rates for ages 15 through 44 in each neighborhood. From these, in turn, I computed the probability of survival to age 45 conditional upon survival to age 15 for each neighborhood using the standard formula:

$$\hat{P}_{fj} = 1 - \exp\left\{-\sum_{t=15}^{44} \hat{\mu}_{fj}(t)\right\}.$$

I used an identical procedure for males. This approach provides both flexibility and stability in the estimation of mortality rates.

One further complication in the measurement of the independent variable must be noted. Overall, the hypothesis to be tested is that neighborhood-level young adult mortality accelerates the initiation of sexual intercourse by adolescents. One might reasonably expect to observe the opposite effect, however,

for mortality due specifically to HIV infection. That is, as HIV prevalence and related mortality increases in a neighborhood, young people might respond to this by taking a variety of precautionary measures, including delaying the onset of sexual intercourse. For this reason, rather than defining $D_{fj}(t)$ and $D_{mj}(t)$ as all deaths regardless of cause,I define them instead as all deaths for which HIV infection was not identified as the underlying cause. This was possible because the death certificates included cause of death codes. The resulting estimated probabilities of dying by age 45 therefore are properly interpreted as hypothetical probabilities that would apply if HIV were eliminated as a cause of death in these neighborhoods (Preston, Heuveline, and Guillot 2001). All analyses described in this paper were conducted using both standard and HIV-elimination estimated probabilities of dying, and produced substantially identical results in all cases. I report only results based on the HIV-elimination probabilities of dying, as these may be preferable on theoretical grounds.

[FIGURE 1 ABOUT HERE]

Figure 1 shows the distribution of the neighborhood-specific probabilities of dying before age 45, given survival to age 15, for females and males derived according to the procedures described above. Not surprisingly, males are much more likely than females to die in this age range. In the average neighborhood, the probability that a male alive at his 15th birthday will die before his 45th birthday is .111, whereas the corresponding probability for females is .043. Most importantly, these probabilities of dying show substantial within-sex variation across neighborhoods. For males, the range is .023 to .376, with a standard deviation of .069; for females the range is .011 to .155, with a standard deviation of .024. These variations are large enough to be of practical significance. A 15-year-old male with a 20% chance of dying before age 45, for example, may have a very different outlook on his future than a male with only a 3% chance of dying in that age range.

Other Variables

Measures of numerous background characteristics of respondents and their families were compiled from information obtained during the Wave 1 interviews with respondents and their primary caregivers. These variables were chosen based upon prior research on the correlates of adolescent age at

first intercourse. Information from interviews with primary caregivers was used to construct variables representing race/ethnicity (Hispanic, non-Hispanic Black, non-Hispanic White, and Other), immigrant generation status (first, second, and third or higher generation, plus a category for those with missing or inconsistent information), household composition (2 biological parents present versus other configurations), household size (the total number of people residing in the household), and family socioeconomic status (an index based on several education, occupation, and income variables). Primary caregivers also completed several standardized assessments, including the Child Behavior Checklist (Achenbach 1991), which provided measures of respondents aggressive and delinquent behaviors; the EASI Temperament Survey (Buss & Plumin 1975), which provided a measure of respondents' sociability; and the PHDCN Homelife Interview (Leventhal et al. 2004), which provided a measure of parental monitoring.

Respondents also completed several standardized assessments at Wave 1. These included the Provision of Social Relations instrument (Turner, Frankel, & Levin 1983), which was used to obtain measures of social support received from family and from peers; the Self-Report of Offending (Loeber et al. 1989), which provided a scale measuring property crime; the Wide Range Achievement Test (Wilkinson 1993), which provided a measure of reading proficiency; and a measure of pubertal development (Robertson et al. 1987). They also answered several questions about the behaviors of their peers (Huizinga, Esbenson, and Weihar 1991). From these I derived scales measuring peer property crime and peer aggression, as well as indicators of peer drug use, peer drug selling, and peer sexual experience.

Naturally there was some missing data at the item and scale levels. I used a combination of procedures to complete the Wave 1 dataset. First, using a total of 78 continuous covariates (only some of which are described above), I applied the expectation-maximization algorithm to impute values (Graham et al. 1997). Next, for the binary, nominal, and ordinal covariates, I used model-based imputation procedures with random draws. Further details on the Wave 1 interviews, scale construction, and

imputation procedures have been published elsewhere (Bingenheimer, Brennan, and Earls 2005) and are available from the author upon request.

Finally, I used four additional neighborhood-level scales. Three of these – concentrated disadvantage, immigrant concentration, and residential stability – were derived from a factor analysis of ten variables from the 1990 Census of the Population (Sampson, Raudenbush, and Earls 1997). Variables with standardized factor loadings in excess of 0.70 on the concentrated disadvantage included percent below the poverty line, percent black, percent below 18 years of age, percent unemployed, percent female-headed households, and percent on public assistance. Percent Latino and percent foreign born loaded heavily on the immigrant concentration factor, and percent in same house as in 1985 and percent in owner-occupied housing loaded on the residential stability factor. These scale have been used widely in analyses of PHDCN data. The last neighborhood level scale is called "intergenerationally-oriented collective efficacy." It was derived by aggregating responses to ten questionnaire items by samples of adult residents of the Chicago neighborhoods (Browning et al. 2004, 2005). Items included "Parents in this neighborhood know their children's friends," and "Adults in this neighborhood know who the local children are." These other neighborhood-level scales are highly correlated with neighborhood-level mortality. Concentrated disadvantage, immigrant concentration, and residential stability respectively are correlated at 0.74, -0.48, and 0.09 with female mortality and at 0.83, -0.47, and 0.13 with male mortality, leading to variance inflation factors of 2.9 for females and 4.7 for males.

Data Analysis

Age at first sexual intercourse is a right-censored variable. Over 42% of respondents with usable outcome data reported that they had never had sexual intercourse. As a result, their age at first intercourse is unobserved; we know only that it is greater than their age at the time of the interview. Discrete-time hazard models are appropriate for this type of data, and these models have recently been extended to facilitate multilevel applications (Barber et al., 2000). The unit of analysis for this model is the person-period rather than the person. I therefore used information provided by respondents during their Wave 2 and 3 interviews to construct a person-year dataset covering ages 10 through 18. Each respondent could

contribute up to nine person-years to the dataset. For each person-year, respondents received a zero if they had not had sexual intercourse prior to the corresponding year and did not have sexual intercourse during that year; a one if they had not initiated sexual intercourse prior to that year but did initiate it during that year; and a nine if they had initiated sexual intercourse prior to that year or if their status during that year could not be determined. Person-years receiving a code of 9 were then eliminated from the dataset, leaving a total of 7484 person-years representing n=1236 respondents. I divided the dataset into male and female subsets for model fitting. Discrete-time multilevel hazard models are estimated by fitting logistic regression models with age effects and random effects of neighborhoods to these person-period datasets.

I fit a series of multilevel discrete-time hazard models to these data. I began with models that included linear and squared terms for age, plus an effect of the sex-specific young adult mortality rate (Model 1). I standardized the young adult mortality variables, so the corresponding coefficients are interpreted as the age-adjusted effects of a one standard deviation increase in the neighborhood-level mortality rate. This should be borne in mind when comparing the coefficients from female models to those from male models, because a standard deviation for males is nearly three times as large as a standard deviation for females.

I then began adding individual-level covariates to the model to see how this would alter the estimated effects of young adult mortality on the hazard of first intercourse. This process was guided by the general principal that one should adjust for confounders but not for mediators. This simple guideline is difficult to implement in practice when some covariates cannot easily be classified as pure confounders or pure mediators (Bingenheimer and Raudenbush 2004). I therefore introduced groups of covariates sequentially, beginning with those I deemed most likely to be confounders and least likely to be mediators in this application. These were race/ethnicity and immigrant generation, both of which may be important determinants of the neighborhood or type of neighborhood in which a person lives, but which are unlikely to be affected by neighborhood characteristics (Model 2). I next added three family demographic variables: household composition (two biological parents versus other configurations), household size,

and the family SES index (Model 3). Although these variables could be influenced by neighborhood characteristics, they probably play a stronger role in sorting families into neighborhoods. I then added family supervision and social support received from the family (Model 4); all of the peer group variables (Model 5); and several measures of respondents' behaviors as well as their pubertal development (Model 6). Of the individual-level covariates, I consider those added in Model 6 to be most likely to be mediators of the effects of neighborhood characteristics, including young adult mortality, on age of sexual onset.

In the next series of models I removed all individual-level covariates but added neighborhood-level variables. First I added concentrated disadvantage (Model 7); then immigrant concentration, residential stability (Model 8), and then intergenerationally-oriented collective efficacy (Model 9). Finally, I estimated models with both individual- and neighborhood-level covariates. The first of these models includes race/ethnicity and immigrant generation at the individual level, and concentrated disadvantage, immigrant concentration, residential stability, and collective efficacy at the neighborhood level (Model 10). The second includes all individual- and neighborhood-level covariates (Model 11). In all of these models, all individual- and neighborhood-level quantitative independent variables are standardized, so that coefficients may be interpreted as effects on the log-odds of sexual initiation of one standard deviation increases in the corresponding variables, holding all other covariates constant. All qualitative variables are represented by dummy variables.

As a final approach to isolating the effect of neighborhood-level mortality on age at first intercourse I conducted a generalized propensity score analysis (Imai and van Dyk 2004) at the neighborhood level. To do this, I modeled the female (male) 1993 probability of dying during young adulthood as a function of concentrated disadvantage, immigrant concentration, residential stability, and female (male) 1990 probability of dying during young adulthood. Models for each sex included linear and squared terms for all variables, plus all two-way interactions between the linear terms. The probabilities of dying were transformed onto the log-odds scale. For females (males), the propensity model explained 71% (85%) of the between-neighborhood variation in the young adult mortality. I then ran, for each sex, a multilevel discrete-time hazard model of age at first intercourse in relation to

mortality, controlling for decile in the propensity score distribution by means of nine dummy variables (Model 12).

RESULTS

The first set of models (Model 1) revealed statistically significant effects of neighborhood-level mortality on age at first intercourse for both females ($b = 0.227$, $p < .001$) and males ($b = 0.464$, $p < 0.001$). In order to facilitate the interpretation of these coefficients, I graphed the cumulative probability of first intercourse as a function of age, sex, and neighborhood-level mortality, as implied by these models. Figure 2 shows that the effects are very large for both males and females. For both sexes living in low mortality neighborhoods, the median age at first intercourse is around 18.5 years. In contrast, for females (males) in high mortality neighborhoods, the median age at first intercourse is 15.7 years (14.8 years). Thus, living in a low instead of a high mortality neighborhood is associated with a 2.7 year delay of first intercourse for females, and a 3.6 year delay for males.

[FIGURE 2 ABOUT HERE]

[TABLE 2 ABOUT HERE]

To what extent can these large differences be attributed to neighborhood-level mortality rather than to confounding effects of other individual- or neighborhood-level variables? Table 2 shows results of select models (Models 2 and 6) that control for minimal and maximal sets of individual-level covariates. (For both males and females, the effects of neighborhood-level mortality and of all individual-level covariates obtained in Models 3 through 5 were similar to those presented for Model 6.) For females, controlling for race/ethnicity and immigrant generation reduces the effect of mortality by approximately 50%, but the effect remains statistically significant at the $\alpha = 0.10$ level. Controlling for additional individual-level covariates reduces the estimate further and renders it statistically insignificant, but the point estimates continue to be in the hypothesized direction. For males, controlling for race/ethnicity and immigrant generation reduces the effect of mortality by approximately 60%, but the

effect remains statistically significant. Controlling for additional individual-level variables further reduces the point estimate for males, but the effects remain statistically significant at the $\alpha = 0.10$ level, or marginally so, in most models.

[TABLE 3 ABOUT HERE]

Table 3 shows the results of select models (Models 7 and 9) that control for neighborhood-level covariates. (For both males and females, the effects of mortality and of all neighborhood-level covariates obtained in Model 8 were very similar to those presented for Model 9.) For both sexes, controlling for concentrated disadvantage barely attenuates the effects of neighborhood mortality. The coefficients remain large and statistically significant in both of those models. Moreover, the effects of concentrated disadvantage, controlling for mortality, are small and not statistically significant for either sex. The addition of further neighborhood-level control variables, however, reverses this result. In Model 9 (and in Model 8) the effects of concentrated disadvantage become large and statistically significant, while the effects of neighborhood mortality become small and statistically null; in fact, the point estimates are in the opposite of the hypothesized direction for both sexes.

In Models 10 and 11, which include both individual- and neighborhood-level covariates, the effects of mortality are in the opposite of the hypothesized direction for sexes, but remain statistically null in all cases. In Model 10, the estimated effects of neighborhood mortality for females and males, respectively, were -0.053 (ns) and -0.75 (ns); in Model 11 these estimates were -0.145 (ns) and -0.021 (ns). Likewise, the propensity stratified models (Model 12) yielded null results. For females, the estimated effect of neighborhood mortality was 0.066 (ns); for males it was -0.081 (ns). To summarize the results, Figure 3 presents point estimates and 95% confidence intervals for the effects of neighborhood mortality on age at first intercourse for all 12 models and for both sexes. Full results for all untabulated models are available from the author upon request.

[FIGURE 3 ABOUT HERE]

DISCUSSION

The most reasonable interpretation of these results is that the effects of neighborhood young adult mortality rates on adolescents' age of first sexual intercourse, if any, cannot be isolated from the confounding influences of other individual- and especially neighborhood-level variables. An argument could be made for preferring Model 2 to Model 6 on theoretical grounds, thereby making a case for accepting statistically significant estimates over null results. That argument would emphasize that some of the covariates in Model 6 should be regarded as mediators, implying that the estimate from that model is biased toward the null. An equally compelling case, however, could be mobilized in favor of Model 2 over Model 6, this one emphasizing the likelihood that important confounding variables have been omitted from Model 2. My view is that both arguments have merit, meaning that estimates from both models are potentially problematic. The fact that the intermediate Models 2, 4, and 5 give results more similar to those obtained in Model 6 may add weight in favor of that model, but this is hardly decisive. In any case, if we ignore neighborhood-level covariates, it would be tempting to take the estimates from Model 2 as an upper bound on the true effect of neighborhood mortality on adolescent sexual initiation, and to take zero as the lower bound for that effect for both sexes.

Yet neighborhood-level covariates should not be ignored. The results of Models 8 through 11 for both sexes provide reason to reject zero as a lower bound for the effects of neighborhood mortality on adolescent sexual initiation. All of these models yield point estimates in the direction opposite to that hypothesized, and in the female version of Model 11 the estimate, although statistically null, is rather large in magnitude.

Perhaps the most bewildering result involves the contrast between Models 7 and 9 (or 8). In Model 7, for females and males, the effect of neighborhood mortality is large and statistically significant, while the effect of concentrated disadvantage is small and statistically null. The inclusion of other neighborhood-level covariates in Models 8 and 9 reverses this outcome. Neighborhood mortality and concentrated disadvantage are highly correlated for both females ($r = 0.74$) and males ($r = 0.83$). It is not surprising, therefore, that these variables would "compete" to explain variation in adolescent sexual

initiation. What is surprising is how dramatically the outcome of that competition depends upon the inclusion or omission of other neighborhood-level variables from the model. This volatility in the estimated effects of neighborhood mortality and concentrated disadvantage should give us pause, as it may be indicative of serious problems with the model, including off-support inference.

In this regard, the results of the propensity-stratified analyses (Model 12) are somewhat reassuring. One advantage of propensity stratification (or matching) is that it protects the analyst from unwittingly making off-support inferences (Oakes and Johnson 2006; Rubin 1997). The null results obtained in those models may therefore bolster confidence in the choice of zero as a lower bound for the effects of neighborhood mortality on adolescent sexual initiation.

A number of limitations of this study should be noted. Certainly the most serious of these is the fact that neighborhood mortality during young adulthood is highly correlated with other neighborhood as well as individual characteristics. As discussed at length above, this makes it extremely difficult to isolate the unique contribution of neighborhood mortality to variation in the timing of first sexual intercourse. Previous discussions of the threats to valid causal inference in observational studies of neighborhood effects have generally emphasized the threat of confounding by individual- and family-level variables that are involved in the sorting of people into neighborhoods (Oakes 2004). Less attention has been given to the high correlations among many neighborhood-level variables, and the implications thereof. The results presented here, however, suggest that confounding by neighborhood-level covariates can be even more severe than confounding by individual- or family-level variables.

Another limitation involves the measurement of age at first intercourse. Self-report measures of sexual behaviors are subject to considerable error, as epidemiologists widely recognize (e.g., Aral and Peterman 1996). Indeed, some investigators have reported substantial inconsistency between two waves of a longitudinal survey in adolescent self-reports of age at first intercourse (Upchurch et al. 2002). Although biomarkers can be used to check the validity of self-reports for some sexual behaviors (e.g., Gallo et al. 2006), it is not obvious how such an approach could be used in practice to establish the timing of first sexual intercourse in a large-scale survey such as this one.

There are numerous other, less serious, limitations. One is the omission of potentially important variables, such as religiosity, from the analysis. Another is the possibility that many of the individual- and family-level covariates, as well as collective efficacy at the neighborhood level, may be measured with a nontrivial amount of error. Yet it seems unlikely that the elimination of measurement error or the inclusion of omitted covariates would have much impact on the results of the multivariate models presented here, except perhaps to move the point estimates even closer to the null or to make the standard errors even larger. The substantive conclusion would almost certainly remain the same. Although neighborhood-level young adult mortality has large gross effects in the hypothesized direction on the timing of adolescent sexual initiation, the unique effects of this variable cannot be isolated from the confounding influences of numerous individual- and neighborhood-level variables in these data. Rather, it appears that young adult mortality is deeply embedded within a constellation of strongly interrelated individual, family, and neighborhood variables. As such, it may be most fruitful to treat it as a part of that network, perhaps by using it as an additional indicator of concentrated disadvantage, rather than attempting to isolate its unique effects in future research.

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Table 1. Select Characteristics of Respondents, Wave 1.

Variable	% of Respondents
Male	49.1
Race/Ethnicity	
Hispanic	44.8
Black	36.7
White	14.3
Other	1.9
Immigrant Generation	
First	13.8
Second	29.7
Third or Higher	53.1
Unknown	3.4
Live with Both Biological Parents	43.4
Number of Peers Using Drugs	
None	80.2
Some	15.0
All	1.5
Number of Peers	
None	57.5
Some	34.8
All	7.7

Table 2. Results from Select Multilevel Hazard Models of Sexual Initiation with Individual-Level Controls.

	Model 2 (Females)		Model 6 (Females)		Model 2 (Males)		Model 6 (Males)	
	b	SE	b	SE	b	SE	b	SE
Mortality	0.114*	0.060	0.067	0.069	0.185*	0.075	0.151*	0.084
Hispanic	0.096	0.266	-0.042	0.292	0.247	0.221	0.118	0.238
Black	0.188	0.219	-0.055	0.264	0.873***	0.239	0.726**	0.243
Other Race/Ethnicity	-0.267	0.474	-0.541	0.496	-0.177	0.482	-0.082	0.505
First Generation Immigrant	-0.632*	0.290	-0.445	0.273	-0.427*	0.232	-0.140	0.264
Second Generation Immigrant	-0.351*	0.185	-0.252	0.194	-0.197	0.195	0.046	0.205
Unknown Generation Immigrant	-0.338	0.247	-0.272	0.267	0.069	0.301	-0.032	0.311
Two Biological Parents			-0.268*	0.135			-0.502***	0.142
Family SES			-0.111	0.072			0.004	0.074
Household Size			0.022	0.074			0.046	0.083
Monitoring			-0.015	0.072			0.001	0.066
Family Support			-0.202*	0.083			-0.179*	0.075
Peer Support			0.207*	0.102			0.155*	0.078
Peer Drug Use 1			-0.241	0.190			-0.220**	0.085
Peer Drug Use 2			-0.377	0.574			0.091	0.077
Peer Drug Selling			0.005	0.094			0.097	0.183
Peer Sex 1			0.254	0.175			-1.961*	0.767
Peer Sex 2			0.686*	0.319			0.142	0.116
Peer Property Crime			0.041	0.087			0.156	0.122
Peer Aggression			0.211*	0.111			0.890**	0.276
Pubertal Development			-0.071	0.070			-0.070	0.057
Property Crime			0.075	0.084			0.098	0.065
Aggression			-0.039	0.112			0.061	0.088
Delinquency			0.279**	0.106			0.169	0.114
Sociability			0.148*	0.066			0.046	0.066
Reading Achievement			-0.071	0.075			-0.007	0.078
Age	2.053***	0.180	2.185***	0.188	1.759***	0.140	1.868***	0.156
Age2	-0.361***	0.078	-0.354***	0.081	-0.424***	0.068	-0.429***	0.071
Intercept	-3.231***	0.214	-3.289***	0.263	-2.869***	0.199	-2.847***	0.210

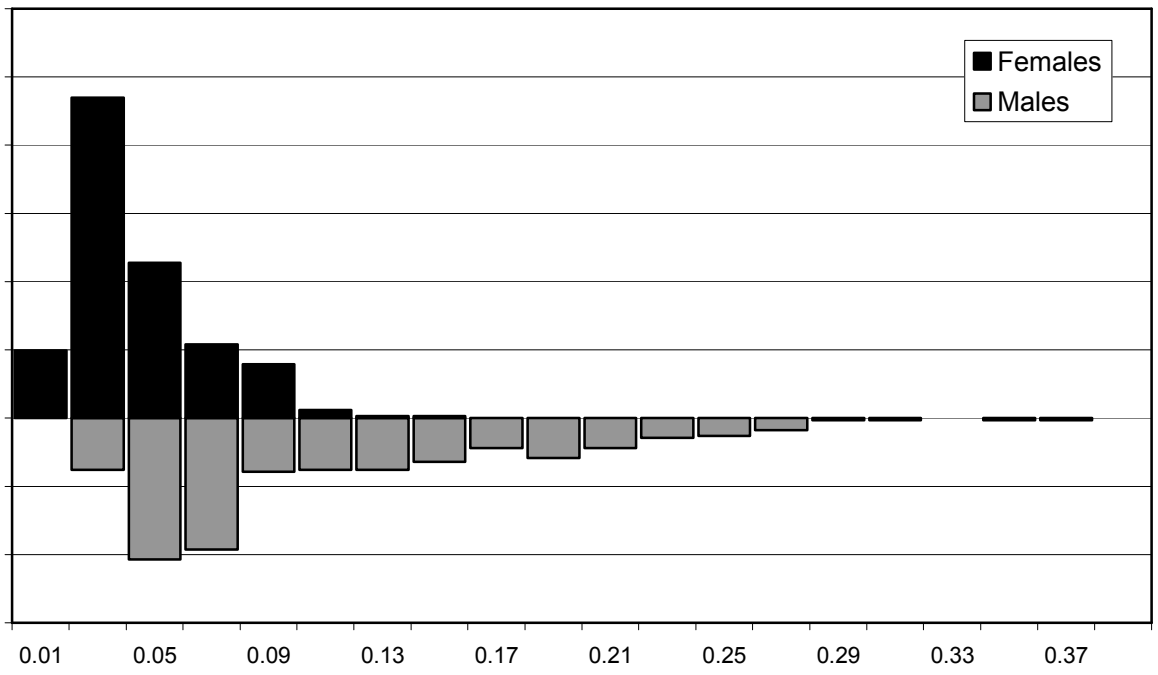
* $p < 0.10$; ** $p < 0.01$; *** $p < 0.001$

Table 3. Results from Select Multilevel Hazard Models of Sexual Initiation with Neighborhood-Level Controls.

	Model 7 (Females)		Model 9 (Females)		Model 7 (Males)		Model 9 (Males)	
	b	SE	b	SE	b	SE	b	SE
Mortality	0.212**	0.073	-0.050	0.081	0.409**	0.132	-0.059	0.129
Concentrated Disadvantage	0.032	0.109	0.288**	0.101	0.087	0.150	0.493**	0.180
Immigrant Cencentration			-0.236***	0.065			-0.313***	0.075
Residential Stability			-0.053	0.070			0.196**	0.067
Collective Efficacy			0.092*	0.054			-0.148*	0.078
Age	2.052***	0.178	2.062***	0.178	1.720***	0.137	1.737***	0.139
Age Squared	-0.374***	0.077	-0.364***	0.076	-0.413***	0.066	-0.413***	0.067
Intercept	-3.280***	0.112	-3.217***	0.121	-2.516***	0.096	-2.430***	0.088

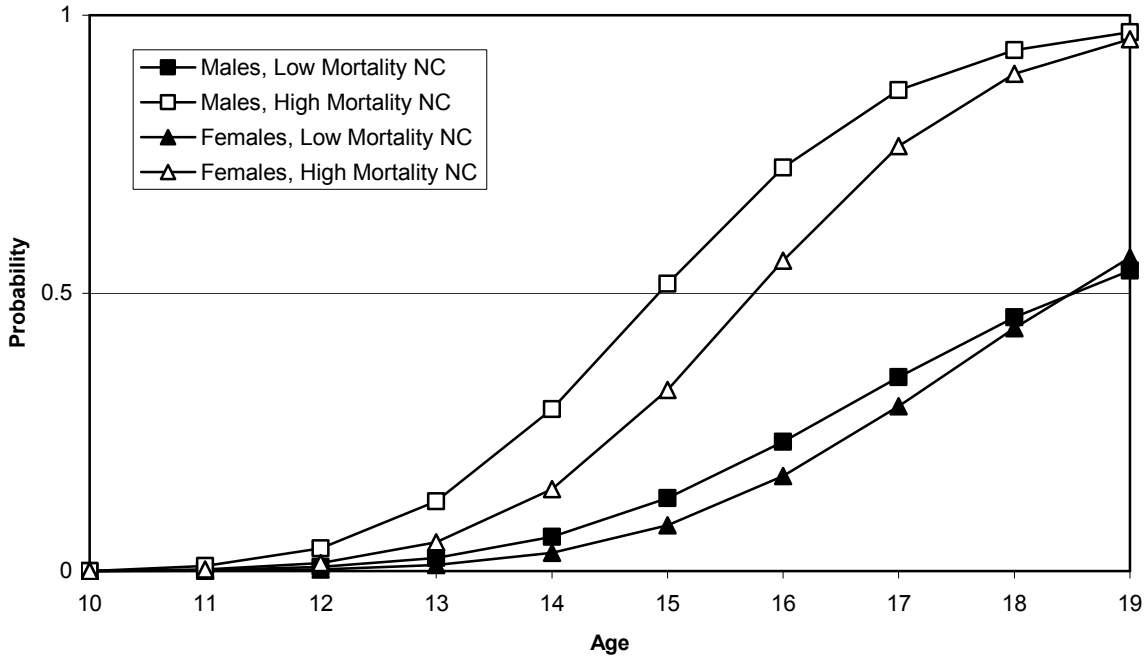
* $p < 0.10$; ** $p < 0.01$; *** $p < 0.001$

Figure 1. Distribution of HIV-Eliminated Probabilities of Dying Before Age 45, Given Alive at Age 15, Chicago Neighborhoods, 1993.



NOTE: Labels on the horizontal axis give the midpoint of the range represented by corresponding bars on the graph. Thus the first bar covers 0.00 to 0.02, the second covers 0.02 to 0.04, and so on.

Figure 2. Cumulative Probabilities of First Sexual Intercourse for Females and Males in Low and High Mortality Neighborhood Clusters.



NOTE: Low (high) mortality neighborhood clusters are defined as those as the 20th (80th) percentile of the distributions of the probabilities of dying during young adulthood. These probabilities are .023 (.060) for females and .052 (.172) for males.

Figure 3. Summary of Results from Multilevel Discrete-Time Hazard Models of First Sexual Intercourse.