

The Natural History of Neighborhood Violence

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Few studies have applied life course methods to understand the natural history of crime rates in neighborhoods or other small social areas. Recent research on neighborhood effects has produced evidence of small area variations in child development and maltreatment, teenage sexual behavior and childbearing, school dropout, home ownership, several indicia of health, suicide, drug use, and adolescent delinquency. However, fewer studies have examined neighborhood variation over time in rates of violence and injury. In this study, we estimate the effects of neighborhood disadvantage on cyclical and nonlinear patterns of violence in New York City from 1985 to 2000. The pattern of violence suggests a "slow epidemic," although with meaningful neighborhood differences in the onset, peak and decline of violence that vary according to neighborhood structure. Violence spreads and then contracts in a pattern similar to a contagious disease epidemic. Patterns of spread and change differ for gun violence compared to other forms of violence. The results illustrate the salience of a developmental perspective on neighborhoods, the unique conceptual meaning of gun violence, and the importance of modeling periods of decline as a unique phenomenon independent from the predictors of onset.

Keywords: *neighborhood; violence; injury; contagion; guns*

Until recently, research on neighborhood and community variation in crime and delinquency focused on identifying cross-sectional, between-area differences in rates of violence or property crime. Often constrained by data limitations, these studies have adopted a static view of com-

This research was supported by grant R49/CCR212753 from the Centers for Disease Control, grant 97-IJ-CX-0013 from the National Institute of Justice, and grant 044599 from the Investigator Awards in Health Policy Research Program of the Robert Wood Johnson Foundation. The authors are grateful to Dr. Susan Wilt and the Injury Prevention Program of the New York City Department of Health for their generous cooperation in this research. All opinions and mistakes

Journal of Contemporary Criminal Justice, Vol. 20 No. 2, May 2004 127-147

DOI: 10.1177/1043986204263769

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munity or neighborhood, assuming that differences in crime rates within neighborhoods were stable over time and that differences in crime rates between communities reflected variation in the characteristics of those communities that were invariant over time (see, for example, Bursik, 1984). Shaw and McKay (1942), for example, showed that crime rates were predictably higher in socially disorganized communities over time, independent of the residents of those areas. More recently, Land, McCall, and Cohen (1990) suggested that the social and economic correlates of crime were stable across time and different spatial aggregations.

More recent studies have adopted a dynamic, developmental perspective for the study of community and crime, as well as other social and economic behaviors. Recent interest in neighborhood effects has produced new research on small area variations in child development and child maltreatment, teenage sexual behavior and childbearing, school dropout, home ownership, several indicia of health, suicide, disorder, drug use, and adolescent delinquency (see, for example, Brooks-Gunn, Duncan, Klebanov, & Sealand, 1993; Coulton, Korbin, Su, & Chow, 1995; Miles-Doan, 1998; Crane, 1991; Gould, O'Carroll, & Mercy, 1990; Gould, Wallenstein, & Kleinman, 1990; Rowe & Rogers, 1994). These perspectives reflect a growing body of criminological research that recognizes that crime rates vary in communities over time and that there is a natural history of change in community crime rates that parallels life course studies of behavioral change among individuals (e.g., Piquero, Farrington, & Blumstein, 2003; Reiss, 1986; Schuerman & Korbin, 1986; Taylor & Covington, 1988). Moreover, evidence of the spread of social behaviors from one neighborhood to the next suggests that the element of social contagion may also explain variation in crime rates over time.

Despite these conceptual developments, few studies have recognized that neighborhoods (like people) are dynamic entities that change over time and that these transformations are likely to lead to complex outcomes of crime and other indicia of social and economic life (Sampson, Morenoff, & Gannon-Rowley, 2002). The few studies published thus far point to complex interactions and (nonrecursive) feedback processes between crime and the social dynamics and compositional characteristics of neighborhoods (Bellair, 2000).

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In this article, we apply a developmental perspective to chart the natural history of interpersonal violence in New York City neighborhoods from 1985 through 2000. We assume that this natural history includes dynamic elements of change within neighborhoods and spread across areas. That is, just as there are neighborhood differences in risk and onset at the outset of the epidemic, there also are differences in the trajectories of the violence problems over time, notably in their rates of decline. The fact of declining rates of homicide and violence challenges theories that are built on cross-sectional, time-limited differences in violence rates from one area to the next (Blumstein & Wallman, 2000; Fagan, Zimring, & Kim, 1998). Even when grappling with change, most theories can explain ascension or stability but are silent on decline. These challenges raise questions about whether the predictors of onset can also explain differential rates of change over time and whether the predictors of increasing rates of violence can also predict differences in the trajectory of change. Accordingly, we conceptualize the rise, spread, and decline in violence rates over time as a process akin to a contagious disease epidemic and test a theoretical framework of neighborhood risk as an engine of social contagion within and between these small social areas.

We begin by developing a conceptual framework of where structural disadvantage compromises the resistance of neighborhoods to violence, making them susceptible to dynamics of social contagion. Next, we use mixed effects regression models to test for evidence of contagion in the natural history of violence across and within neighborhoods. We control for concentrated disadvantage (Bursik & Grasmick, 1993; Krivo & Peterson, 1996, 2000; Kubrin & Weitzer, 2003; Parker & Pruitt, 2000; Sampson & Wilson, 1995) to measure susceptibility and resilience across neighborhoods and over time. Finally, we isolate specific risks of violence such as the socially toxic effects of guns (Fagan & Wilkinson, 1998; Fagan et al., 1998; Wintemute, 2000).

CRIME AND NEIGHBORHOOD CHANGE

Natural History and Neighborhood Crime Rates

Interest in neighborhood change as a predictor of changing crime rates can be traced to the Chicago School traditions of studying “natural social areas” whose identities are the products of complex social and economic factors, sometimes endogenous (Park, 1916) and sometimes imposed from the outside by political economic dynamics (Logan & Molotch, 1988; Suttles, 1968). It is surprising that, despite this interest, there have been few longitudinal studies of neighborhood change and changes in crime rates.

The good news is that these few studies converge in several areas to shape theory. Physical and social deterioration is a persistent theme of neighborhood change in several studies (Harrell & Gouvis, 1994; Schuerman & Kobrin, 1986; Taub, Taylor, & Dunham, 1984). Deterioration often cued citizens to leave previously stable areas based on changes in their subjective evaluation of the likelihood of crime affecting them personally. A second thread in neighborhood change studies is the reciprocal influence of adjacent neighborhoods on crime rates. Taylor and Covington (1988), Morenoff and Sampson (1997), and Heitgerd and Bursik (1987) all identified dynamics where crime or violence in one area influenced homicide rates in adjacent areas over time. Taylor and Covington examined gentrification as a trigger for crime, whereas Heitgerd and Bursik used a similar strategy to show that even stable, well-organized communities can have high rates of delinquency when the adjacent neighborhoods experienced rapid racial change. A third area of research identifies turning points in neighborhoods that precede the onset or intensification of crime. Bursik and colleagues (Bursik, 1984; Bursik & Grasmick, 1992, 1993; Bursik & Webb, 1982) analyzed neighborhood change in Chicago's 74 planning areas to identify turning points in the natural history of neighborhood development to pinpoint when crime rates change and grow.

Each of these studies offers important clues about neighborhood change and crime but is also limited in some important ways. First, most of these studies have used census tracts to bound and characterize neighborhoods. The Chicago studies are an exception, but the 74 areas are large, heterogeneous aggregates of several smaller neighborhoods, a strategy that might mask important influences in smaller corners of these larger areas. For smaller areal units, there is no consensus whether census block groups or tracts or other boundaries are either socially meaningful or theoretically appropriate to study either community structure or social processes (see Bursik, 1988). Research with alternate social-spatial configurations may yield more accurate units to specify social processes, but these may run into other types of data problems and limit comparability between studies. In this study, we use boundaries that were drawn based on an integration of residents' perceptions of the natural boundaries of their neighborhoods, proscribed by their attribution of shared belonging among residents, with census and other administrative boundaries that provide data conveniences for consistent measurement and comparability across studies.

Second, because census data are collected decennially, researchers interested in neighborhood change have limited their study periods to these fixed 10-year intervals. Other studies use much shorter time windows, limiting their analyses to shorter periods where the window for estimating change

may be artifactually short. Yet, crime trends usually do not cooperate with the markers of the decennial censuses. Crime trends can be quite volatile within a decade or even span decades, and inferences about changes in crime rates at a decade apart can be quite misleading.¹ Nonlinear patterns in both neighborhood ecology and crime patterns demand more complex functional forms for analysis, including quadratic terms for time parameters to allow for curvilinear changes in violence rates as well as their predictors. We estimate models that include both linear and nonlinear terms for estimating time change. We also include a sufficiently long time period that provides observations over a cyclical change in crime rates. By including years from three different decades, we can specify the time frame to comport with the actual points of onset, peak, and decline in the crime cycle.

Third, studies of neighborhood change in crime rates vary in the specificity of the crime form and the theoretical linkages that would predict changes in specific types of crime. Some studies specify linkages to violence based on carefully specified theories, whereas others measure changes in more global measures of crime without disaggregating crime into dimensions that might be differentially predicted by alternate theories. Wilson and Kelling's (1982) theory of "Broken Windows" suggested that signs of disorder launched a contagious process that signaled the onset of higher crime rates but were not specific as to which crimes.² Subsequent empirical tests showed quite limited predictive power for this notion (Harcourt, 2000; Sampson & Raudenbush, 1999). In contrast, Taylor and Covington (1988) hypothesized and confirmed that the juxtaposition of contrasting trajectories of neighborhood change may accelerate violence by creating targets of robbery opportunity in newly gentrified areas adjacent to chronically poor ones, as well as resentments growing from the relative deprivation of the older, poorer areas. In this article, we estimate models of two different forms of violence and develop two related theoretical paths—structural and cultural attenuation, toxicity of gun violence, and social contagion—that each are specific to the spread of violence and, more directly, to its rise, spread, and decline over time.

Structural and Cultural Sources of Susceptibility

Whether called *concentrated disadvantage*, *concentration effects*, or *resource deprivation*, neighborhoods in this social position are vulnerable to weakened forms of formal and informal social control and, in turn, elevated rates of crime and violence (Bursik & Grasmick, 1993; Land et al., 1990; Morenoff & Sampson, 1997; Sampson & Lauritsen, 1994; Taylor & Covington, 1988; Williams & Flewelling, 1988; Wilson, 1987). Wilson (1987, 1991) refers to the concentration of these conditions of weak social

control within specific spatial areas as *social isolation*, a consequence of the concentration of poverty that resulted from deindustrialization. Social isolation suggests an ecological dynamic where the components of poverty, joblessness, and structural disadvantage are interconnected with the dynamics of social control and opportunity structures. Land et al. (1990) showed that the entrenchment of the socially isolated poor grew more severe throughout the 1970s and 1980s, in the period preceding the sharp increase in adolescent homicide rates. Fagan (1992) and Blumstein (1995) each link the increase in homicides among young people in this era to the rapid expansion of drug markets that lured young unskilled workers in poor neighborhoods with the promise of higher incomes, a rational choice given the alternative of below-market wages in the spot labor market or the legal informal economy. In turn, violence arose from the churning effects of external shocks to these areas: for example, conflicts in illegal markets (e.g., Fagan, 1992; Goldstein, Brownstein, Ryan, & Bellucci, 1989) or the growing presence of firearms that gave rise to an "ecology of danger" where lethal violence became part of everyday interpersonal disputes (Fagan & Wilkinson, 1998).

The concentration of poverty through the 1980s and the collaterally weak social institutions in the poorest neighborhoods of large cities also undermined the presence of and institutional support for conventional behaviors (Sampson & Wilson, 1995). In this context, conventional values and behaviors were attenuated because they were not salient and had little payoff for one's survival or status (Elliott et al., 1996; Wilson, 1987). These dynamics in turn attenuated neighborhood social organization, increasing the likelihood that illegitimate opportunity structures would emerge. These structures competed with declining legal work opportunities both as income sources and as sources for social status. As these networks flourished, the systems of peer and deviant social control replaced the controls of social institutions and conventional peer networks (Fagan, 1992).

Together, these processes suggest that violence and homicide are more likely to occur in an ecological context of weak social control, poorly supervised adolescent networks, active illegal markets where violence is the primary regulatory device, widespread perceptions of danger and the demand for lethal weapons, and the attenuation of outlets to resolve disputes without violence.

Social Contagion and Changing Crime Rates

The spread of violence both within and across social areas at times has been viewed as a process of contagion (Cork, 1999; Loftin, 1986; Sah, 1991). These perspectives comport well with more general theories and studies of

diffusion and contagion (Bovasso, 1996; Burt, 1987, p. 1288; Gladwell, 2000). Rowe and Rogers (1994) show that an epidemic model combining social contagion through social contacts among adolescents within a narrow age band explains the onset and desistance of adolescent sexual behavior. Through a process of mutual influence involving contact, communication, and competition, adoption of behaviors occurs when information is transmitted that communicates the substance of the innovation and the consequences of adoption. These behaviors acquire social meaning that is communicated through repeated interactions within social networks (Kahan, 1997; Lessig, 1995).

Contagious epidemics involve the transmission of an agent via a host through susceptible organisms whose resilience is weakened by other conditions or factors (Bailey, 1967). Susceptibility is critical to the ability of an agent to exert its process on a host. This medical rendering of contagion can be analogized to social contagion. Thus, the fundamental social causes of disease—primarily social structural or ecological—can be seen as pathways along which more microlevel causes can exert their effect (Gostin, Burris, & Lazzarini, 1999, p. 74; Morenoff, 2003). According to Gostin et al. (1999), these fundamental social causes reflect inequalities that work in two ways. First, these conditions increase exposure to the more proximal causes, whether microbic or behavioral. Second, they compromise the resistance or resilience of social groups to these proximal causes. That is, their exposure and their behavior in those structural circumstances both have social roots (Gostin et al., 1999).

Within social networks in these neighborhood contexts, a “cultural software” evolved that is expressed in language, behavior, and normative beliefs, creating a set of behavioral “scripts” (Abelson, 1976, 1981; Balkin, 1998; Cavalli-Sforza & Feldman, 1981; Fagan & Wilkinson, 1998). Accordingly, social contagion is convergence or transmission of behaviors and beliefs that motivate or sustain them. Social contagion arises from people in proximate social structures using one another to manage uncertainty of behavior (Burt, 1987, p. 1288). Burt (1987) suggests that adoption of behaviors or scripts has less to do with the cohesion of people within social structures, or networks, and more to do with the structural equivalence—the social homogeneity—of the network. That is, transmission is more likely to occur between similarly situated persons—siblings, fellow graduate students, streetcorner boys—than persons simply because they are closely bonded. Accordingly, social networks in homogeneous neighborhoods become hosts for the transmission of violence. In this article, we integrate the concepts of contagion and diffusion to show the mutual and reciprocal influence of adjacent neighborhoods to animate the rise and decline of neighborhood rates of violence.

METHOD

Variables and Measures

Neighborhood

We estimated models of fatal and nonfatal interpersonal injury for New York City neighborhoods from 1985 to 2000. Neighborhoods are made up of several census block groups—spatial units constructed by Jackson and Manbeck (1998) based on interviews with neighborhood residents and physical examination of naturally occurring neighborhood boundaries.³ These neighborhoods reflect small social areas where the effects of local social and economic contexts are influential both on social control and on crime opportunities. The final sample of neighborhoods is 285 after eliminating areas with no population, such as parks and heavily industrialized areas.

Neighborhood Ecology

The *ecology* variable is a latent construct representing the convergence of the components of concentrated disadvantage. Following Land et al. (1990), we selected 18 tract-level variables from the 1980-2000 Census (STF3A and 3C files) to characterize social areas. Table 1 shows the means and standard deviations of these indicators, which were sorted into seven dimensions—poverty, labor market, segregation, supervision, anonymity, immigration, and housing structure—that characterize the dimensions of concentrated disadvantage articulated in the theoretical and empirical literature linking neighborhood effects with indicia of social adversity and isolation (see, for example, Sampson et al., 2002). To more concisely represent neighborhood conditions, principal components analyses were used to construct a factor score for each dimension. Table 2 shows the item loads and factor scores for each dimension. Because these factors were designed to tap essential facets of neighborhoods, we then used a latent variable analysis to determine the relative weighting of each factor in an indicator for neighborhood ecology. In the final stage, each factor was multiplied by its corresponding weight and the sums used to produce a measure of ecology for each neighborhood.⁴ We estimated separate factors and ecology measures for each year, based on an imputation of census measures for each variable for the midcensus years.

Homicide and Violence

Data on homicides and nonfatal hospitalized injuries were obtained from the Vital Statistics records and hospital admissions registers of the New York

TABLE 1
Descriptive Statistics—Census Variables: New York City Neighborhoods, 1980-2000

Variable	1980		1990		2000	
	M	SD	M	SD	M	SD
% households with public assistance income	21.08	21.46	12.51	11.32	7.11	6.75
Gini for total household income	.39	.04	.37	.08	.38	.08
% households less than poverty level	17.70	12.99	16.64	13.11	18.81	12.13
% high school graduates—total—25+	60.83	15.23	70.62	13.99	74.05	13.58
% with managerial, professional, or technical jobs	26.93	13.85	33.52	14.82	36.70	15.81
Employment rate	92.51	3.68	91.18	4.65	90.39	6.10
Labor force participation rate	57.32	8.56	61.69	8.33	58.33	8.66
% non-White	42.31	32.97	50.30	33.02	58.87	31.33
Racial fragmentation index	.39	.20	.44	.18	.50	.16
% female-headed households with children younger than 18	9.72	8.32	6.45	7.32	10.44	8.16
Supervision ratio (25-64 by 5-24)	1.93	1.13	2.42	1.30	2.34	1.15
% youth population (5-15)	16.06	5.95	14.16	5.41	15.49	5.84
Residential mobility—same house as 5 years ago	59.94	11.17	63.02	8.21	60.39	8.95
Population	24,137	28,606	24,965	29,299	27,350	31,809
% foreign born	20.41	10.18	29.28	13.59	34.12	14.04
% linguistic isolation	6.38	6.39	10.46	8.60	12.35	9.25
% occupied units that are rentals	69.98	23.67	65.12	23.12	64.39	21.95
Vacancy rate	5.27	4.79	5.96	4.54	5.80	3.57

SOURCE: U.S. Census, STF 3-A, 3-C Files; New York City Department of City Planning; www.infoshare.org.

TABLE 2
Factor Composition: New York City Neighborhoods, 1980-2000

Factor	1980			1990			2000		
	Rotated Coefficient	Variance Explained	Eigenvalue	Rotated Coefficient	Variance Explained	Eigenvalue	Rotated Coefficient	Variance Explained	Eigenvalue
Poverty/inequality			2.61			2.78			2.78
% households less than poverty level	.975	87.0		.980	92.7		.982	92.6	
% households with public assistance income	.945			.969			.959		
Gini for total household income	.876		3.07	.939		3.06	.946		3.08
Labor market/human capital		76.7			76.4			76.8	
% high school graduates—total—25+	.955			.956			.944		
% technical, managerial, or professional	.886			.868			.887		
Employment rate	.802			.867			.819		
Labor force participation rate	.853			.799			.849		
Segregation		82.6	1.65		76.9	1.54		72.1	1.44
Racial fragmentation index	.909			.877			.849		
% non-White	.909			.877			.849		
Supervision		79.6	2.39		77.2	2.32		84.6	2.54
% youth population (5-15)	.963			.961			.969		
% female-headed households with children younger than 18	.857			.808			.899		
Supervision ratio (25-64 by 5-24)	-.853			-.860			-.890		
Anonymity		50.1	1.00		50.9	1.02		54.5	1.09
Population	.708			.714			.739		
Residential mobility—same house as 5 years ago	.708			.714			.739		
Immigration		77.5	1.55		91.1	1.82		88.5	1.77
% linguistic isolation	.880			.955			.941		
% foreign born	.880			.955			.941		
Housing structure		64.1	1.28		54.1	1.08		53.9	1.08
% occupied units that are rentals	.801			.735			.734		
Vacancy rate	.801			.735			.734		

City Department of Health and Mental Hygiene. Data included counts of both fatalities and nonfatal hospitalization cases, based on coding by the Office of the Medical Examiner (for deaths) and hospital staff (for nonfatal cases) using ICD-10 codes to classify injuries as either intentional or self-inflicted. Counts were developed for all cases, gun cases, and cases with African American victims. We included the latter given the disproportionate effect of the violence epidemic both in New York City (Fagan et al., 1998) and nationally (Cook & Laub, 1998, 2001; Eckberg, 1995) on African American victims. Neighborhood rates were estimated by aggregating from individual cases that were geocoded to the census tract. We geocoded cases using the residential address of the victim. Although this may distort the location estimates for violent events, we based this decision on prior work showing the close proximity of homicide events to the residences of victims.⁵

Model Estimation

We developed individual growth curve models to neighborhoods using SAS PROC MIXED (Singer, 1998). We first estimate an unconditional growth curve model, then conduct a second estimation with covariates representing susceptibility. We estimate models with time-varying covariates where both slopes and intercepts vary, and residual observations within neighborhoods are correlated through the within-tract error-covariance matrix. We include a measure to account for the endogeneity of crime and social disadvantage, which is the predicted value from a Poisson regression for the count in the initial year in each series (1985 for homicide, 1990 for violence) predicted from the ecology measure. We include a *contagion* parameter that refers to the event count (homicide or nonfatal injury) in the base neighborhood in the preceding year. We lag homicides and assault rates by 1 year in the estimate of diffusion to surrounding areas and a *spatial lag* parameter to account for the event count in the adjacent neighborhoods in the preceding year.⁶ We specify separate models for homicide and nonfatal assaults and disaggregate by total events, gun events, and violent events with African American victims. Population is the logged count of the model-specific population at risk.⁷

RESULTS

For four decades, homicide rates exhibited a pattern of “roller coaster regression” in most cities, with increases in three distinct periods in most cities followed by sharp declines after each successive peak (Fagan et al., 1998). In this article, we focus on the period beginning in 1985—the most recent low point prior to the record high rate of homicide in 1991 and the subsequent

decline over the following decade. Because most of the increase and decline in both homicides and other violence in this period was due to guns and victimization fell primarily on African Americans (Cook & Laub, 1998, 2002), we compute separate rates and estimate separate models for gun violence and for African American victimization.

Table 3 shows the means and standard deviations for four time points in the series and an average over all years. The trends for total violence mirror the trends for gun violence. All rates peaked in 1991 (Fagan et al., 1998), and the decline by 1995 was more than 50% for all but one measure of violence. The trend in New York City and elsewhere suggests a slow epidemic, with lethal violence spreading over both time and space rapidly, reaching a peak, and then declining over time as rates returned to their preincrease levels (Fagan et al., 1998).

Tables 4a and 4b show separate model estimates for homicide and total violence and for victimization of African Americans. Table 4a shows that neighborhood ecology is a significant predictor of total homicide, gun homicide, and homicides with African American victims.⁸ It is not surprising that neighborhood disadvantage predicts homicide rates over time. Interactions with time suggest that neighborhood ecology is not a significant predictor of the declining homicide rates for total and gun homicide, but it is a positive and significant predictor of homicide victimization rates for African Americans. Even in an era of declining homicide, neighborhood disadvantage continues to pose elevated homicide risks for African Americans.

Contagion measures are inconsistent predictors of homicide victimization over time. For total and gun homicides, a neighborhood's homicide rate is a significant and positive predictor of homicide rates 1 year later in the surrounding neighborhoods—a sign of diffusion from area to adjacent areas. The significant negative coefficient for the interaction of time with contagion suggests that homicide contracts over time. As the overall epidemic receded, homicide rates in each neighborhood were significantly less likely to influence homicide rates in the surrounding neighborhoods.

But these contagion effects are not present for African American homicide victimization. In Table 4a, contagion is not a significant predictor of either total or gun homicide rates for African Americans. The interaction of contagion with time is significant for total African American homicide victimization but not for gun homicides. Even when significant, the effect size (i.e., the exponentiated coefficient) is relatively small. At the same time, the endogeneity control is significant for African American homicide victimization rates but not for total homicides or total guns. Thus, differences in homicide victimization rates for African Americans at the outset of the time series remain significant over time, differences that seem to be largely a function of structural disadvantages in neighborhoods characterized by high percent-

TABLE 3
Descriptive Statistics—Homicide and Nonfatal Assault Rates (per 100,000 persons): New York City Neighborhoods, 1985-2000

	1985		1990		1995		2000		All Years— Average	
	M	SD	M	SD	M	SD	M	SD	M	SD
Total homicide	1.57	2.00	2.64	3.80	1.17	1.82	.80	1.60	1.55	2.73
Gun homicide	.84	1.52	1.87	3.24	.73	1.06	.53	1.49	1.06	2.33
Black homicide	2.41	6.57	4.00	7.12	1.93	7.66	1.16	2.97	2.36	6.21
Black gun homicide	1.14	4.02	2.83	5.87	1.09	3.65	.66	1.30	1.58	4.93
Total violence ^a			16.75	23.01	10.72	11.38	7.88	8.14	11.95	14.75
Gun violence ^a			5.05	6.55	2.39	2.83	1.48	2.40	3.19	4.84
Black violence ^a			65.17	323.41	21.89	42.30	23.02	59.60	36.39	214.97
Black gun violence ^a			11.99	25.96	4.94	17.72	4.84	24.22	7.96	25.97

a. Nonfatal assault data are available only for 1990-2000.

TABLE 4a
Mixed Effects Poisson Regression of Homicide Contagion: New York City, 1985-2000

	<i>Total Homicide Victimization</i>		<i>Gun Homicide Victimization</i>		<i>African American Victimization</i>		<i>African American Gun Victimization</i>	
	Exp B	t	Exp B	t	Exp B	t	Exp B	t
Effects								
Intercept	.002	-12.39	.001	-13.66	.000	-27.92	.000	-32.31
Time	1.307*	7.10	1.493	9.22	1.631	10.81	2.011	13.82
Quadratic time	.956	-5.11	.930	-7.28	.911	-8.70	.869	-11.68
Contagion	1.002	3.11	1.003	2.87	1.001	.87	1.000	.28
Spatial lag	1.093	49.39	1.124	48.88	1.116	44.27	1.150	42.38
Ecology	1.499	12.29	1.635	13.77	1.210	7.10	1.207	7.73
Endogeneity	1.001	.33	.998	-.65	.996	-2.84	.993	-3.73
Population	2.057	15.57	2.203	15.97	2.501	31.43	2.643	35.03
Time interactions								
Contagion	1.000	-2.47	1.000	-2.08	1.000	-1.98	1.000	-1.13
Spatial lag	.996	-10.88	.994	-10.02	.992	-12.00	.989	-12.14
Ecology	1.002	.55	1.001	.36	1.014	3.33	1.018	3.85
-2 log likelihood	3596.2		5100.9		7671.5		8957.3	

* $p(t) < .05$ shown in bold.

ages of African American population.⁹ Diffusion seems not to be part of the animating dynamic of African American homicides, whereas endogeneity of homicide seems to sustain over time to perpetuate elevated homicide rates within these areas. Diffusion is evident, however, for total homicides and gun homicides, whereas endogeneity is not. One conclusion from these results is that there may be separate spatial and temporal dynamics of urban homicide, one for African Americans that is characterized by concentrated disadvantage and isolation, and another for other patterns of homicide victimization that is characterized by diffusion from one neighborhood to the next.

There is little evidence of diffusion of assault. Table 4b shows that the contagion predictor is not significant in any of the four models, either in the main effects or in its interaction with time (i.e., its slope). Ecology is significant only for the total violence and gun violence models and for gun victimization of African Americans, both at the intercepts and for the slopes. But the interaction of time and ecology for African American victimization is negative, suggesting that African American violence victimization rates were declining more slowly in neighborhoods with concentrated disadvantage. Endogeneity is significant only in the total violence model, but the effect size

TABLE 4b
Mixed Effects Poisson Regression of Violence Contagion: New York City, 1985-2000

	<i>Total Violence Victimization</i>		<i>Gun Violence Victimization</i>		<i>African American Victimization</i>		<i>African American Gun Victimization</i>	
	Exp B	t	Exp B	t	Exp B	t	Exp B	t
Effects								
Intercept	.031	-6.61	.002	-12.10	.025	-9.90	.004	-21.20
Time	.869*	-26.67	.792	-28.73	.836	-24.90	.792	-25.75
Quadratic time	1.005	12.69	1.008	11.30	1.007	12.46	1.010	11.05
Contagion	1.000	.88	1.000	1.12	1.000	-.94	1.000	.80
Spatial lag	1.008	29.74	1.017	19.87	1.010	19.95	1.019	16.90
Ecology	1.267	7.16	1.696	14.41	1.008	.21	1.151	5.34
Endogeneity	1.000	1.95	1.000	-.44	1.000	.42	1.000	-.47
Population	1.993	14.08	2.343	17.84	2.169	18.82	2.389	31.07
Time interactions								
Contagion	1.000	-.11	1.000	-.42	1.000	.98	1.000	.00
Spatial lag	1.001	20.28	1.005	21.16	1.002	19.72	1.007	20.17
Ecology	.994	-3.19	.998	-.91	.984	-5.33	.991	-2.96
-2 log likelihood	-1777.3		1839.8		1201.2		3905.9	

* $p(t) < .05$ shown in bold. Violence includes both homicide and assault. Nonfatal assault data are available only for 1990-2000.

is very small. Neighborhoods marked by disadvantage have higher risks for all forms of violence. Compared to homicide, there is no evidence that violence diffuses to surrounding areas and less evidence of its endogeneity compared to homicide. Evidently, the natural history of nonlethal violence differs from the patterns for homicide. Although these differences may reflect the salience of homicide in social interactions (Fagan & Wilkinson, 1998), the results may be an artifact of the truncated time series for nonlethal violence that began in 1990, 5 years later than the violence series and close to the peak of the homicide epidemic in 1991.

CONCLUSIONS

There are several elements to a natural history of violence, including its onset and rise, its persistence over time, the endogeneity of violence and structural disadvantage within some neighborhoods, and the diffusion of violence from one area to the next. During the homicide epidemic in New York City beginning in 1985, we find differences in the natural history of homicide but fewer differences when we include nonlethal violence. We also find dif-

ferences in race-specific victimization patterns. The dynamics of the rise, spread, and fall of homicide differ for African Americans, the group that suffered disproportionately during this epidemic. Patterns of racial residential segregation and its collateral concentration of disadvantage in neighborhoods with high African American populations combined to isolate homicides and minimize the spread to surrounding areas. At the same time, these same patterns sustained homicide at a higher rate in these areas, even as homicide rates were declining in other areas.

Accordingly, the structural position of a neighborhood, its social and spatial relationships and connectedness to its surrounding areas, and specific forms of violence are important for understanding the patterns of violence over time. Concentrated disadvantage is a consistent factor that makes neighborhoods susceptible to elevated rates of homicide and violence, but segregation may sharply limit the spread of violence to surrounding neighborhoods. Focusing on a single neighborhood may mask potentially important contextual dynamics from the broader social environment that bear on the natural history of violence.

Perhaps most important, we find that gun violence is significantly likely to spread across neighborhoods. The role of guns in homicide may explain why gun homicides are more likely to show a contagious pattern than violence, generally. Gun homicides account for more than 75% of all homicides during much of this 17-year period, but guns were involved in a far smaller and variable percentage of nonlethal violent events during the same period (Fagan et al., 1998). Gun violence may have a churning effect on social norms that explains its salience as a predictor of the spread of gun violence (Anderson, 1999; Canada, 1995; Fagan & Wilkinson, 1998).

Finally, guns are an agent in the transmission of violence and a cancer on social norms. Because the recent epidemic cycle of violence was in reality a gun homicide epidemic, the case for gun-oriented policing strategies is much stronger than practices based on the more diffuse and unsupported theory of disorder control and order-maintenance strategies (see, for example, Kelling & Coles, 1996). Whereas disorder embraces orderliness, cleanliness, and sobriety (Harcourt, 2000), violence appears to travel on vectors quite unrelated to that particular set of social norms. Its natural history suggests that more complex and disaggregated strategies are needed to respond to violence epidemics.

NOTES

1. See, for example, Fagan, Zimring, and Kim (1998) on the "roller coaster" of crime rates in New York City throughout the 1980s.
2. See, also, Skogan (1990).

3. The neighborhood boundaries are available at www.infoshare.org, for neighborhood indicators and boundary maps depicting these relatively new spatial units.
4. Data are not shown and are available from the authors.
5. See, for example, Fagan (1999), Fagan and Wilkinson (1998), and Fagan, Medina-Ariza, and Wilt (2003).
6. Models with 2-year time lags produced results very similar to those reported here. Thus, the results as reported do not appear to be an artifact of the lag time chosen.
7. We used model-specific population counts to estimate rates. These were either the total population in the adjacent neighborhood or the Black-only population.
8. We also estimated all models with the separate components of the ecology variable and obtained nearly identical results. Data are not shown but are available from the authors.
9. Recall that we use the African American population as the population predictor in the models for African American homicide victimization. Patterns of racial residential segregation suggest that neighborhoods with high rates of African American homicide victimization also are areas with a high concentration of African American population and little racial heterogeneity.

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