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Social Contagion of Violence

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CHAPTER 36

Social Contagion of Violence

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Introduction

Like many large American cities, New York City experienced a sudden and dramatic increase in homicides beginning in 1985. The homicide run-up was highest for adolescents, but rates increased quickly for older persons as well (Fsagan, Zimring, & Kim, 1998). Unlike many other cities, in which homicides declined gradually over the next decade yet remained above their pre-1985 levels, the increase in New York was followed by an even larger decline over the next 5 years. By 1995, homicide in New York City had dropped below its 1985 level; by 1996, it was lower than the 1985 rates; by 1998, homicide rates were 25% lower than the 1985 levels; and by 1998, homicide rates were lower than three decades earlier. The drop in crime generally, and not just homicide, was an order of magnitude greater than any observed in large American cities since the 1950s (Zimring, 2007).

Explanations of this roller-coaster pattern of violence beginning in the 1960s have tended to partition the periods of increase and decline as distinct phenomena with unique causes. Moreover, these causes are typically regarded as exogenous to the people or areas affected. For example, the onset and severity of the homicide trend were attributed to the sudden emergence of unstable street-level crack markets, with high levels of violence between sellers (Baumer, 1994; Baumer, Lauritsen, Rosenfeld, & Wright, 1998; Fagan & Chin, 1991; Fryer, Heaton, Levitt, & Murphy, 2005; Grogger & Willis, 2000). Others suggested that drug markets created a demand for guns that in turn trickled down from drug sellers into the hands of adolescents (Blumstein, 1995; Fagan, 1992). Structural theorists implicated race-specific economic deficits in inner cities (Krivo & Peterson, 2000; Peterson & Krivo, 1993) or racial residential segregation (Massey, 1995). There have been many claims regarding the sources of the decline, including changes in police strategy (Kelling & Cole, 1996), demographic changes (Cook & Laub, 1998; Eckberg, 1995), incarceration (Blumstein & Beck, 1999), and lower demand for illegal drugs (Curtis, 1998).

None of the popular explanations of either the increase or the decline is fully satisfying. Moreover, the gap between the scale of demographic and policy changes and the scale of the crime decline suggests that there are processes at work other than these usual suspects. Some have used the term "epidemic" metaphorically to describe the homicide run-up and decline, but with little precision and often conflating several features of epidemics, such as social concentration, spatial diffusion, and temporal spikes (see, for example, Bailey, 1975).

Epidemic is a term used widely in the popular and scientific literature to describe two quite separate components of a phenomenon: an elevated incidence of the phenomena and its rapid spread via a contagious process within a population in a short period of time. For example, Gladwell (2000) describes how the incidence of an ordinary and stable phenomenon such as a seasonal flu can become epidemic when its incidence increases in a very short time from a predictable base rate to an elevated rate of infections. Moreover, epidemics need not be contagious. Consider an outbreak of food poisoning from contaminated materials or a cancer cluster near a polluted water supply. These medical problems may occur at a rate well above an expected base rate, but are not spread from person to person through physical contact or an infectious process. In contrast, an outbreak of influenza, the adaptation of cultural fads, medical or industrial innovation, or changes in the rates of antisocial behavior all reflect spread through interpersonal exposure to an "infectious" agent.

Although disease spreads through a host and agent (Robertson, 1990; Rothman, 1986), social contagion involves the mutual influence of individuals within social networks who turn to each other for cues and behavioral tools that reflect the contingencies of specific situations (Bailey, 1967; Burt, 1987; Coleman, Katz, & Menzel, 1966). The contagious dimension is especially salient during the upswing of an epidemic, when physical or social contact is critical to spread pursuant to exposure. But epidemics also end, as the rate of new incidence of the phenomenon declines. This decline may occur because the density of contacts may decrease or because some form of resistance develops that reduces the odds of transmission from one person to the next, even in the presence of exogenous contributing factors (Bailey, 1967; Burt, 1992).

In this chapter, we assess whether the roller-coaster pattern of homicides in New York City beginning in 1985 fits a contagion model and identify mechanisms of social contagion that predict its spread across social and physical space. This framework for interpreting the homicide trends as an epidemic includes two perspectives. First, the sharp rise and fall are indicative of a nonlinear pattern in which the phenomenon spreads at a rate far beyond what would be predicted by exposure to some external factor and declines in a similar pattern in which the reduction from year to year exceeds what might be expected by linear regression trends. This leads to the second perspective: the factors leading to its spread are not exogenous factors, as in the case of contamination or disaster. Instead, the nonlinear increase and decline suggest that the phenomenon is endemic to the people and places where its occurrence is highest and that this behavior may be effectively passed from one person to another through some process of contact or interaction.

We assess the validity of these assumptions in three ways. In the section "Violence as Social Contagion," we introduce a framework of social contagion that informs this analysis. The diffusion and spread of social behaviors are recurring themes in both the scientific and popular literatures. But there has been little theorizing on the mechanisms of social diffusion generally and specifically on the social interactions that may qualify as a contagious process. This section reviews the literature generally on social contagion and then constructs an analytic framework to explain the spread of youth violence over time and space. In the section "The Epidemiology of Youth Homicide in New York City," we analyze Vital Statistics data from the New York City Department of Health and Mental Hygiene to construct simple time

series data that characterize the increase and decline in homicides from 1985–2000. We concentrate on homicides involving adolescents and young adults, populations who experienced the sharpest rise and decline in homicide, both in New York and nationally (Cook & Laub, 1998; Fagan & Davies, 2004; Fagan et al., 1998).

In the section "Neighborhood Effects on Social Contagion of Youth Homicide," we estimate models to identify the spatial and social trends in youth homicide. Using growth curve or hierarchical models, we disaggregate homicide and nonlethal injury data by neighborhoods over the 11-year period and fit models to demonstrate the spatial diffusion of youth homicide from one neighborhood to the next across New York City. By co-varying neighborhood social and economic characteristics with temporal homicide trends, we are able to show that the diffusion of homicide in this era was specific to the most socially isolated areas of the city. We isolate gun homicides as the contagious agent, showing that it is gun homicides that diffused across New York City neighborhoods and that gun homicides retreated just as quickly.

In the section "Violent Events, Social Networks, and Social Contagion," we present data from interviews with young males active in gun violence during this time. Their reports of the role of guns in violent events further specify how diffusion may in fact be the result of a dynamic process of social contagion. We conclude the chapter by integrating these perspectives into a unifying framework that links elements of models of infectious disease with social interactionist perspectives to explain the social contagion that contributed to New York City's homicide epidemic, which remains today as the source of contentious debates in public policy and social science.

Violence As Social Contagion

Background

There are many examples of social contagion that inform the development of this research, especially among adolescents: trends in fashion and art (Gladwell, 2000), as well as problematic social behaviors, such as alcohol and drug use (Rowe & Rodgers, 1994), smoking (Rowe & Rodgers, 1991), teenage pregnancy (Crane, 1991), suicide (Berman, 1995; Gould, Wallenstein, & Kleinman, 1990; Gould, Wallenstein, Kleinman, O'Carroll, & Mercy, 1990), and delinquency (Jones, 1997). Common to each of these examples is the social structure of transmission. Thus, the fundamental *social* causes of disease – primarily social structural or social interactionist in nature – can be seen as pathways along which more micro-level causes can exert their effect (Farmer, 1999; Gostin, Burris, & Lazzarini, 1999, p. 74; Lynch et al., 1998; Tolnay & Beck, 1995; Tolnay, Deane, & Beck, 1996; Wilkinson & Fagan, 1996).

The spread of ideas, behaviors, and practices is contingent on the way in which social structure brings people together in close physical proximity within dense social networks (Burt, 1987, p. 1288). For example, Rowe and Rodgers (1994) show that 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 (see also Rodgers & Rowe, 1993). HIV transmission also has been modeled as a contagious epidemic (May, Anderson, & Blower, 1990). Through a process of mutual influence involving contact, communication, and competition, adoption of behaviors occurs when information about behaviors is transmitted in a way that communicates the substance of an innovation and the consequences of its adoption. The consequences can be socially rewarding or intrinsically pleasurable and may be reinforced through the benefits of a vicarious experience or a trial use. In addition, these behaviors acquire social meaning that is communicated through repeated interactions within social networks (Kahan, 1997; Lessig, 1995, p. 1947).

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 (Jones & Jones, 1994, 1995). 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 et al., 1999, p. 74). According to Gostin and colleagues (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.

Memetics provides a complementary framework for understanding how beliefs, ideas, and behaviors spread throughout society. Memes are singular ideas that evolve through a process of natural selection not unlike the evolution of genes in evolutionary biology (Balkin, 1998; Lynch, 1996). The principal law governing the birth and spread of memes is that of the "fittest ideas," defined as those ideas that are the best at self-replication rather than those that may be truest or have the greatest utilitarian value (Lynch, 1996). In the present analysis, violence may be the "fittest" behavior, even when it contradicts more socially useful normative values imported from the dominant society. Memes achieve high-level contagion through a variety of social interactions across social units, such as families and social networks, and each mode increases the "host" population for that meme. The meme is then reproduced within networks and transmitted across interstitial network boundaries.

Replicated memes become what Balkin (1998, pp. 42–57) refers to as "cultural software" that is expressed in language, behavior, and normative beliefs, creating a set of normative expectations or behavioral "scripts." (Abelson, 1976, 1981; Fagan, 1999). According to Abelson, the script framework is an event schema used to organize information about how people learn to understand and enact commonplace behavioral patterns. A "script" is a cognitive structure or framework that organizes a person's understanding of typical situations, allowing the person to have expectations and to make conclusions about the potential result of a set of events. Script theory has been used widely in social psychology to identify patterns of decision making and social interactions that persist among persons within social networks. Script theory can explain contagion in several ways: (1) Scripts are ways of organizing knowledge and behavioral choices; (2) individuals learn behavioral repertoires for different situations; (3) these repertoires are stored in memory as scripts and are elicited when cues are sensed in the environment; (4) the choice of scripts varies among individuals, and some individuals will have limited choices; (5) individuals are more likely to repeat scripted behaviors when the previous experience was considered successful; (6) scripted behavior may become "automatic" without much thought or weighing of consequences; and (7) scripts are acquired through social interactions among social network members (Abelson, 1976, 1981).

Accordingly, social contagion is the convergence of transmission of behaviors and of 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; Gostin, 1991; Rodgers & Rowe, 1993; Rowe & Rodgers 1991, 1994). It requires an interaction in which information, behavioral innovation, belief, or meme is transmitted across a social synapse. At its core, contagion occurs when two people interact where one has adopted a construct and the other has not. Contact, communication, and imitation are influential processes that make transmission possible (Burt, 1987, pp. 1288–1289). Synapses themselves are situated within social networks, and the adoption of an innovation or a meme triggers the adoption by another person. Burt (1987) suggests that adoption 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, street corner boys – than persons simply because they are closely bonded (Burt, 1987, p. 1291).

Within structurally equivalent networks, similarly situated people are likely to influence or adopt behaviors from one another that can make that person more attractive as a source of further relations. The importance of structural equivalence – or placement within a socially homogeneous interpersonal network – is that it fosters interconnected patterns of relationships that make contagion efficient.

In the remainder of this section, we show how transmission of violence occurs across neighborhoods whose social structures of densely packed networks are vulnerable to rapid contagion. Our previous work on urban youth violence has shown how the memes of toughness and the valued status from violence are the object of transmission and exchange among similarly situated male youth (Fagan & Wilkinson, 1998a,b; Wilkinson, 2003). The implications for a social influence model of contagion are discussed in the concluding section.

Guns and Social Contagion

Several processes have contributed to the epidemic of lethal violence. The growth in illegal markets heightens the demand for guns as basic tools that are associated with routine business activity in illegal markets (Blumstein, 1995; Johnson, Williams, Dei, & Sanabria, 1990). In turn, the increased presence of weapons and their diffusion into the general population change normative perceptions of the danger and lethality associated with everyday interpersonal disputes, giving rise to an "ecology of danger" (Fagan & Wilkinson, 1998a). Thus, we hypothesize that guns were initially an exogenous factor in launching an epidemic of gun homicide, but became endogenous to socially isolated neighborhoods and came to dominate social interactions (Wilkinson & Fagan, 1996). Everyday disputes, whether personal insults or retributional violence, in turn are more likely to be settled with potentially lethal violence (Fagan & Wilkinson, 1998a; Wilkinson, 2003).

Whether viewed in social, medical, or memetic frameworks, guns can be constructed as a primary agent of violence contagion over the most recent epidemic cycle. Guns are a form of social toxin (Delgado, 1985; Fagan & Wilkinson, 1998b) in everyday social interactions, altering the outcome of disputes and changing the developmental trajectories of young males whose adolescent development took place in the contexts of high rates of gun use and widely perceived danger, contributing to an ecology of danger that had profound developmental impacts on adolescents growing up in these settings (Bingenheimer, Brennan, & Earls, 2005; Fagan, 1999).

The development of such an *ecology of danger* reflects the confluence and interaction of several sources of contagion. First is the contagion of fear. Weapons serve as an environmental cue that in turn may increase aggressiveness (Slaby & Roedell, 1982). Adolescents presume that their counterparts are armed and, if not, could easily become armed. They also assume that other adolescents are willing to use guns, often at a low threshold of provocation.

The second source is the contagion of gun behaviors themselves. The use of guns has instrumental value that is communicated not only through urban "myths" but also through the incorporation of gun violence into the social discourse of everyday life among pre-adolescents and adolescents. Guns are widely available to adolescents (Cook & Ludwig, 2004), and when carried by adolescents, they are frequently displayed (Harcourt, 2006; Wilkinson, 2003).[1] They are salient symbols of power and status and strategic means of gaining status, domination, or material goods (Wilkinson, 2003). Wilkinson's interviews with adolescents and young adults in two New York City neighborhoods during the mid-1990s

show that guns are used in a myriad of different ways and for different purposes; some uses are seemingly more mild and symbolic than others, but may be the first steps or building blocks for later more serious use. For example, very first steps might be simply seeing someone or knowing someone who has a gun, then looking for a gun in one's own house, then maybe trying to get one, then just flashing one when trying to threaten/scare an opponent, then using the gun for pistol whipping, then firing the gun to scare someone but not aiming to hit them, then actually firing toward someone, then firing to injure but not kill, and then firing to kill. The socialization process into the urban youth gun world begins at a young age, with influences coming from family and peer networks (Wilkinson, 2003; Wilkinson & Fagan, 2001a). How these processes unfold at the event level is explored in Part IV of this chapter.

Third is the contagion of violent identities and the consequent eclipsing or devaluation of other identities in increasingly socially isolated neighborhoods. These identities reinforce the dominance hierarchy built on "toughness" and violence, and its salience devalues other identities. Those unwilling to adopt at least some dimensions of this identity are vulnerable to physical attack. Accordingly, violent identities are not simply affective styles and social choices, but instead are strategic necessities to navigate through everyday dangers (Wilkinson, 2003).[2]

Finally, when the group nature of youth violence is examined, diffusion and contagion of attitudes, scripts, and behaviors are clearly visible. The proximal link between one violent conflict and the next is startling. In addition, the social meanings of violent events reach a broader audience than those immediately present in a situation. Each violent event or potentially violent interaction provides a lesson for the participants, first-hand observers, vicarious observers, and others influenced by the communication of stories about the situation that may follow. Expectations, a violent status hierarchy, and norms of interpersonal conduct among groups of socially isolated young men work to hinder nonviolent conflict resolution. Conflict handling among youth who are affiliated with other youth at least in part to enhance their personal safety in a dangerous environment acts to increase the amount of violence that youth experience, rather than decrease it. We examine these issues empirically at the neighborhood level as well as at the micro-translational level in New York City.

The Micro-Processes of Social Contagion: Social Identity and Violence

Previously, Wilkinson (2003) and Fagan and Wilkinson (1998a) identified several processes operating at the event level that illustrate the spread of violence within social structures and that exert a contracting influence on social networks of adolescents: (1) Achieving a highly valued social identity; occurs through extreme displays of violence, (2) achieving a "safe" social identity may also require the use of extreme forms of violence, (3) the ready availability of guns clearly increases the stakes of how one achieves status; (4) much behavior is motivated by avoiding being a punk or "herb" (sucker or weakling); (5) identities can change from being a punk or herb into a more positive status such as "hold your own"; (6) guns equalize the odds for some smaller young men through the process of "showing nerve"; (7) one can feel like a punk for a specific situation but not take on a punk identity; and (8) one can feel like a "crazy" killer in a specific situation but not take on a "crazy" or killer identity. If "compulsive masculinity" or Anderson's (1994, 1999) "street orientation" is dominant in public spaces and personal safety as our data suggest, then those who do not conform will be victimized.

The maintenance and reinforcement of identities supportive of violence are made possible by an effective sociocultural dynamic that sets forth an age-grading pathway to manhood that includes both behaviors and the means of resolving violations of respect. Wilkinson (2003) described the strong influence of street codes similar to

the codes identified by Anderson (1999) or the code of honor described by Toch (1969), over the behaviors of young children, adolescents, and young adults. The absence of alternative means of attaining valued masculine identities further compounds the problem. The transmission of these social processes occurs on both the micro- and macro-levels. Children growing up in this environment learn these codes, or behavioral-affective systems, by navigating their way through interpersonal situations that often involve violent encounters.

The Epidemiology of Youth Homicide in New York City

Historical and Current Homicide Trends

The epidemic of youth violence in New York City since the mid-1980s is best understood in a social and historical context that spans nearly 35 years. Like the nation's largest cities, New York experienced a sharp increase in homicide and other violence rates beginning in the mid-1960s. The homicide rate rose from 4.7 per 100,000 population in 1960 to 31.0 in 1995. By 1996, the rate had receded to 13.9 per 100,000, a level unseen since 1968. Figure 36.1 shows the gun and nongun homicide rates for 1968–2000.

From 1900 through the beginning of the run-up in homicide in the mid-1960s, and with one exceptional era following the passage of the Volstead Act in 1919, homicide rates in New York City varied narrowly between 3.8 and 5.8 per 100,000 population (Monkonnen, 2001).[3] From 1965 to 1970, the average annual homicide rate rose from 7.6 to 12.6 and rose again to 21.7 by 1975. Thus, homicide in New York nearly tripled within a decade. The rates remained elevated above the 1968 rates until 1998. Accordingly, Figure 36.1 suggests that, for three decades, homicides in New York were normalized at an elevated rate and were for a long time characteristic of the city's social landscape. Thus, the escalation in killings until the 1990s was cumulative, with each new peak building on the elevation of the base rate established in the previous peak. One interpretation of the recent decline may simply be the recession of this longer-term social and historical trend.

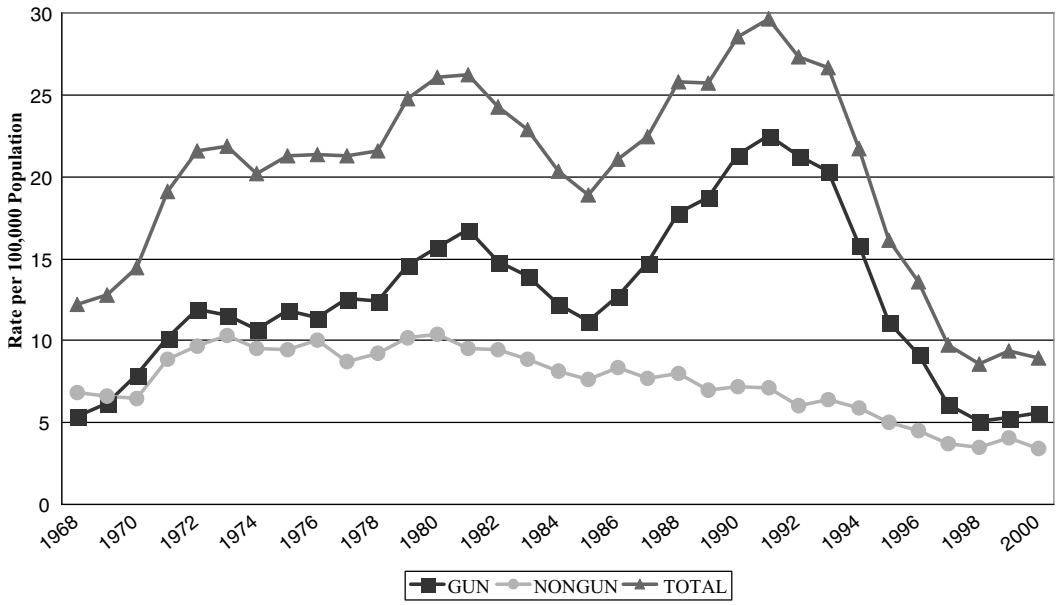

Figure 36.1. Gun and nongun homicide rates per 100,000 persons, 1968–2000, New York City. Source: Office of Vital Statistics and Epidemiology, New York City Department of Health and Mental Hygiene, various years.

Figure 36.1 also shows that this long-term trend involves three sub-epidemics. The first of these peaked in 1972, the second in 1981, and the third in 1991. Each coincided temporally with drug epidemics and the growth of retail drug markets: heroin in the early 1970s (Agar & Reisinger, 2002; Egan & Robinson, 1979; Hunt & Chambers, 1976; Inciardi, 1979), the emergence of urban street drug markets in the late 1970s where powdered cocaine was openly sold (Johnson et al., 1990; Johnston, 1987; Williams, 1989; Zimmer, 1987, 1990), and crack beginning in 1985 (Johnson et al., 1990). The successive epidemics were cumulative in their trends, not distinct. To re-introduce an idea from Part I, the pattern of killings in particular resembles a roller coaster, with an ascent through the late 1970s to a relatively low peak, a return to near the previous low point, and a sharp increase to a high peak in 1990 followed by a sharp drop.

Figure 36.1 shows the growing importance of guns in homicides in each of the three peaks. Increases in both gun and nongun homicides contributed to the tripling of homicide rates through 1972. In 1972, the ratio of gun to nongun homicides was 1.23. By the next peak in 1981, the 1,187 gun deaths were nearly 1.76 times greater than the 673 nongun homicides. In 1991, the modern peak, the 1,644 gun homicides were 3.16 times greater than the 519 nongun homicides. In addition to sharp increases in the number of gun homicides, the gun:nongun ratio also rose sharply because of a long-term decline in the number of nongun homicides. Since 1980, the number and rate of nongun homicides have declined by nearly 50%, from 735 to 335 nongun killings in 1996. There are thus two dynamic and different patterns in the data on homicide by weapon. Gun killings follow the roller-coaster pattern of steadily increasing peaks beginning in 1972. Nongun killings trend down from 1980 to rates unseen since 1960. This long-term secular trend in nongun killings is substantial, but it has not previously been noticed.

Figure 36.1 also shows that the recent cycle beginning in 1985 was qualitatively different from the preceding peaks in five important ways: (1) Its starting point was lower than the starting point for the previous (1981) peak, (2) its peak was about 15% higher than the preceding peak, (3) it had a far greater share of gun killings, (4) its decline was far steeper than any previous decline, and (5) homicides have remained at their low point far longer than in any of the previous three epidemics.

To illustrate the extent of the differences between the 1985–2000 cycle and its predecessors, Figure 36.2 presents the data from Figure 36.1 normed to the 1985 base.[4] Gun killings accounted for all of the increase in homicides since 1985 and most of the decline. Although the declines after 1992 in nongun killings are a continuation of the 8 years of previous decreases, the increase and decline in gun killings are evidence of a homicide spike that is unique from its predecessors. Nongun killings declined steadily since 1986 and by 1996 were about half the 1985 rate, suggesting a secular decline in nongun homicide that may be independent of the gun homicide epidemic.

The epidemic pattern was well-observed through homicide data, but public health data on nonlethal violence in part tell the same story. Data from the City's Injury Surveillance System provide information on hospitalizations for intentional injuries (NYC Department of Health and Mental Hygience, 1997, and various years). Figure 36.3 shows that the decline in homicides was accompanied by a general decline in nonfatal assaults. These data were available for analysis beginning in 1990, about the same time that the homicide epidemic reached its peak. Figure 36.3 shows that, beginning in 1990, nearly all the decline in nonfatal assaults were declines in gun assaults; assaults by other means, such as blunt instruments or cutting instruments (e.g., knives), declined at a much lower rate. So, the rise and fall in homicides did not necessarily reflect changes either in the lethality of gun violence or a change in the case-fatality rate. Rather, gun violence declined generally over time following increases that also were almost exclusively the result of gun violence.

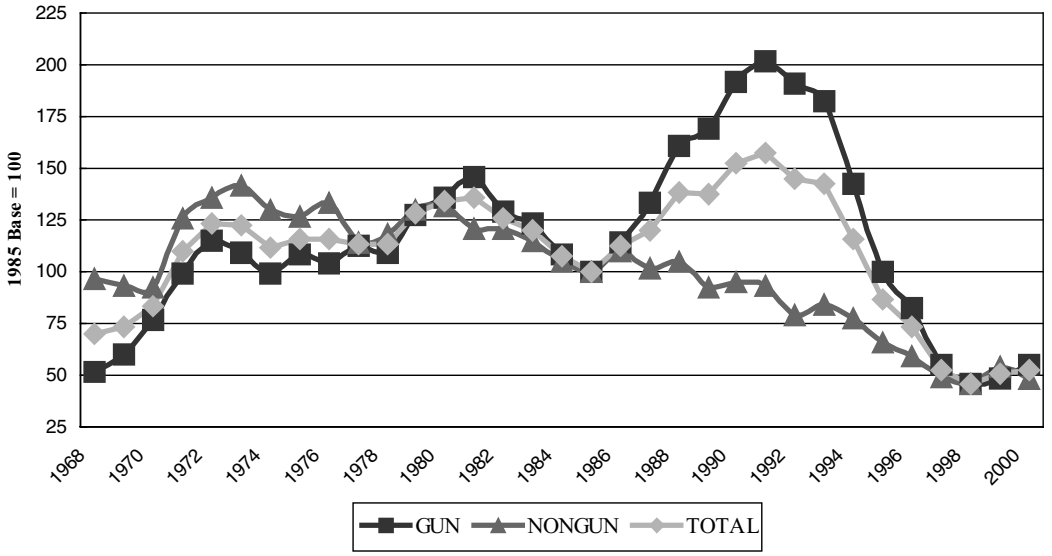

Figure 36.2. Gun and nongun homicide rates, 1968–2002, indexed to 1985 rates, New York City. Source: Office of Vital Statistics and Epidemiology, New York City Department of Health and Mental Hygiene, various years.

The importance of the most recent era of homicide rise and decline, not only in its epidemiological pattern but also in its influence on law and policy (Feld, 1999; Stuntz, 2001; Zimring, 1999), leads us to focus the analysis in the next part and also in the section titled "The Epidemiology of Youth Homicide in New York City" on the patterns of homicide and violence in this era, and especially on the period from 1985–1996, when the rise and fall in homicide were the most dramatic and acute.

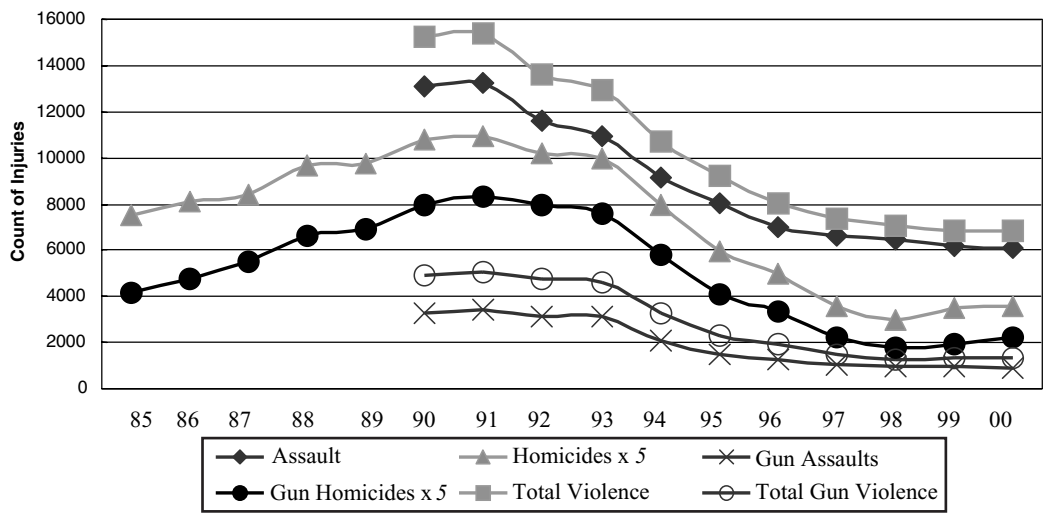

Figure 36.3. Gun and nongun homicide and assault by firearm, 1985–2000. Source: Office of Vital Statistics and Epidemiology, New York City Department of Health and Mental Hygiene, various years; Injury Prevention Bureau, New York City Department of Health and Mental Hygiene, various years.

The Social Structure of Homicide

The demographic patterns of gun and nongun homicide victimization during this period tell a series of interesting stories, some predictable and others surprising. First, the homicide trends for women differ from the patterns for men. Any benefits of the end of the violence epidemic accrued to men; women's risk of violent victimization remained stable at a level far lower than that of men. Second, changes in adolescent homicide rates were accompanied by parallel but less dramatic changes among older populations. This trend varies from the national picture of steadily declining rates among older groups. Third, as we saw earlier, the homicide run-up and decline were concentrated in gun killings. Fourth, the homicide epidemic was concentrated among non-Whites. We observed these trends for both homicide victims and offenders, which are discussed below. The data are available at: http://www2.law.columbia.edu/fagan/researchdata/contagion.

GENDER

Nearly all the increase and decline in killings from 1985–1995 were gun homicides of males. The rate of gun killings among males doubled from 21.8 per 100,000 in 1985 to 44.5 in 1991. Nongun killings of males declined steadily throughout this period and by 1995 were less than half the 1985 rate. Killings of males were increasingly gun events: the ratio of gun to nongun homicide victimizations of males increased from about 1.5:1 in 1985 to 3.23:1 in 1995.

The temporal patterns were similar for females. The rate of gun homicides of females peaked in 1991, the same year as males, and sustained their peak rate for approximately 3 years before dropping sharply in 1994. By 1995, gun homicides for females had dropped 5% below their 1985 levels. Nongun homicides of women declined steadily throughout this period, from 4.7 per 100,000 in 1985 to 3.8 per 100,000 in 1995. But unlike males, the rates of nongun homicides of females were higher than the rates of gun homicides. Throughout the period, the changes in rates for females were quite small, and not far from the expected rates historically. The same is true of male nongun killings. Accordingly, this epidemic is confined to gun killings among males.

AGE

Much of public and scholarly attention on violence in the past decade has focused on the increase in gun homicides by adolescents (Blumstein, 1995; Cook & Laub, 1998). Trends nationwide show that gun homicide rates for adolescents increased during this period while gun homicide rates for persons over 25 years of age were declining. In New York City, homicides were not confined to younger age groups, but were a serious problem across a wide age range from 15 to 34 years of age. Table 36.1 shows that gun homicide rates were higher than nongun rates for all age groups. For each year, gun homicide rates were highest for persons aged 20 to 24 in all years. Gun homicides by adolescents aged 15 to 19 rose more sharply over this period than other older population groups. Nevertheless, although adolescent participation in gun homicide rose sharply from 1985–1991, rates for other age groups also continued to rise during this period.

Gun homicide rates declined sharply for all three age groups from 1992–1995, to about 50% of their peak rates in 1991, and were about the same as their 1985 rates. Although the post-1991 decline was precipitous for adolescents aged 15 to 19, their 1995 gun homicide rates remained 25% above their 1985 base rate. Table 36.1 also shows that nongun homicide rates declined steadily for all age groups and by 1995 were 50% or more lower than their 1985 rates (NYC Department of Health and Mental Hygience, 1997). Similar to gun homicide rates, the nongun homicide rates were highest for persons aged 20 to 24.

RACE

Nationally, virtually all increases in homicide rates from 1985 to 1990 among people 10 to

Table 36.1: Adolescent gun and nongun homicide rates by age, New York City, 1985–1995

Year	*<15*	*15–19*	*20–24*	*25–34*	*≥35*
Gun Homicides					
1985	0.6	20.8	34.1	21.1	7.1
1986	1.1	19.9	44.1	25.6	6.9
1987	1.2	33.1	50.6	25.3	8.5
1988	1.5	45.5	56.8	33.5	9.2
1989	1.3	43.1	60.0	37.9	9.0
1990	2.2	50.9	66.3	42.2	10.8
1991	1.0	57.4	66.3	42.2	12.6
1992	1.3	48.3	68.1	42.8	11.0
1993	1.6	48.5	63.8	37.5	11.7
1994	0.7	39.6	47.5	30.0	8.3
1995	0.8	26.0	34.7	18.1	6.9
Nongun Homicides					
1985	2.9	10.4	15.7	12.2	9.4
1986	3.4	8.0	17.7	14.1	8.4
1987	1.8	6.3	14.1	12.7	7.8
1988	3.0	6.1	14.6	12.3	7.9
1989	2.6	6.5	12.3	13.4	7.0
1990	3.4	12.8	15.5	14.7	7.4
1991	3.1	8.0	11.1	10.9	6.5
1992	2.4	6.9	8.6	7.9	6.2
1993	4.1	3.9	7.0	8.9	6.5
1994	3.1	6.1	7.0	8.8	5.7
1995	2.6	5.2	6.8	6.6	5.1

34 years of age were due to deaths of African American males. Most of these were firearm fatalities that were overwhelmingly concentrated demographically and spatially among African American males in urban areas (Fingerhut, Ingram, & Feldman, 1992a,b.) Table 36.2 shows that the trends in New York mirror these national trends. Unfortunately, none of the data sources permitted detailed disaggregation of the homicide trends by ethnicity over the entire 1985–1995 period. Detailed data were available only for African Americans; Whites and Hispanics were not distinguished in the police or Vital Statistics data until after 1990. Therefore, our analysis is limited to comparisons between whites and non-Whites; non-Whites are primarily persons of African descent, including some Hispanics.

The within-race ratio of gun to nongun homicide rates for each year in the 1985–1995 period illustrates the concentration of the homicide epidemic in gun homicides among non-Whites. For Whites, the ratio rises from 1.26:1 in 1985 to a peak of 3.23:1 in 1992, before receding to 2.1:1 in 1995. For non-Whites, the ratio rises from 1.20:1 in 1985 to a peak of 4.05:1 in 1992 and recedes to 2.32:1 in 1995. However, the narrow difference between Whites and non-Whites may reflect the inclusion of Hispanics among the Whites in the population and homicide counts. The extent of this bias can be seen in 1993 data from the New York City Department of Health injury surveillance system. The mortality and morbidity rates of gunshot wounds for Hispanics are 228 per 100,000 persons, compared to 302 for African Americans and 60 for Whites in that period (NYC Department of Health and Mental Hygiene, 1992; New York State Department of Health, 1994).

Table 36.2: Gun and nongun homicide rates per 100,000 persons by race, 1985–1995

	Gun		*Nongun*	
Year	*White*	*Non-White*	*White*	*Non-White*
1985	10.1	12.8	8.0	10.6
1986	10.9	15.2	7.3	11.3
1987	9.8	20.7	6.1	10.1
1988	13.2	23.6	6.4	10.1
1989	15.1	23.2	6.7	8.9
1990	18.8	25.1	8.9	9.0
1991	19.4	26.6	6.4	7.8
1992	19.3	24.3	6.0	6.0
1993	17.2	24.5	5.5	7.2
1994	12.8	18.9	5.7	5.8
1995	9.5	12.8	4.5	5.5

VICTIM-OFFENDER HOMOGENEITY

Most homicides are within-group events, especially with respect to gender, race, and ethnicity (Cook & Laub, 1998; Sampson & Lauritsen, 1994).[5] We analyzed data from police reports on the within-age distribution of homicide events for each year in the recent homicide cycle to estimate the proportion of homicides in which victims

and offenders both came from their own age group. From 1985–1995, we observed within-group homogeneity with respect to our limited categories of race, a trend evident also in national data (Cook & Laub, 1998). Age homogeneity was more varied and depended on the method of homicide. Age homogeneity for gun homicides was highest for homicide offenders aged 25 to 34 and lowest for 20- to 24- year-olds. The low rates for the group aged 20 to 24 reflects their age status between the two other groups and the higher likelihood of cross-age interactions.

Age differences widened during the homicide cycle beginning in 1985. At the outset of the homicide run-up in 1985, age homogeneity for gun and nongun homicides was low: about one gun homicide in four involved persons within the same age categories. For adults aged 25 to 34, about 4 in 10 gun homicides were within-age killings. A year later, both age homogeneity and homicide rates increased. Within-age gun homicides for young adults aged 20 to 24 rose from 22.9% of gun homicides in 1985 to 35.8% in 1990; for offenders aged 25 to 34 years, within-age group homicides rose from 38.5% in 1985 to 54.9% in 1989. Even with these increases, however, the majority of gun killings involved persons from different age groups. During the same period, within-age nongun homicide rates varied from year to year in an inconsistent pattern.

The results are not surprising: age stratification of peer groups has traditionally created age-specific social networks. Age grading is a hallmark of street gangs (Klein, 1995) and adolescent cliques (Schwendinger & Schwendinger, 1985). These rigid age boundaries offered few opportunities for cross-age social interactions among delinquent groups. But contextual changes in street corner life in inner cities, where homicides were concentrated throughout this period, contributed to a breakdown of traditional age grading. The emergence of street drug markets and dense street corner groups of males not in the workforce contributed to a mixing of the ages on the street. Among adolescents and young adults, competition for street status through violence contributes to a process of "status forcing" that promotes cross-age interactions (Wilkinson, 2003).

CONTEXTUAL EFFECTS

Both popular and social science explanations of the homicide epidemic in New York and elsewhere have focused on social trends, particularly changes in drug markets (Baumer, 1994; Baumer et al., 1998; Blumstein, 1995; Cork, 1999; Grogger & Willis, 2000). Fagan et al. (1998) discuss the appeal of these explanations. First, homicide and drug epidemics have been closely phased, both temporally and spatially, in New York and nationwide, for nearly 30 years (Fagan, 1990; Fagan & Wilkinson, 1997). Homicide peaks in 1972, 1979, and 1991 mirror three drug epidemics: heroin, cocaine hydrochloride (powder), and crack cocaine. These long-term trends predict that trends in drug use would occur contemporaneously with trends in homicide. Second, the emergence of volatile crack markets in 1985 is cited as one of the primary contextual factors that have driven up homicide rates in New York (Bourgois, 1995; Goldstein, Brownstein, Ryan, & Bellucci, 1989; Johnson et al., 1990). Competition between sellers, conflicts between buyers and sellers, and intraorganizational conflict were all contributors to lethal violence within crack markets (Fagan & Chin, 1989, 1991; Hamid, 1990). Crack also is implicated in the decline of homicide since 1991 (Curtis, 1998).

Figure 36.4 compares trends in gun homicides for three age groups with trends in drug overdose deaths. Drug overdose deaths follow a pattern of short cycles, with relatively brief periods of increase and decline. The rates increase from 1986 to 1988, decline through 1990, and increase again for 3 years before leveling off. The run-up of gun homicide rates in 1985 to 1988 matches an increase in drug overdose deaths, but homicides continued to increase even as drug overdose deaths declined. Drug overdose death rates increase from 1992 to 1994, even as gun homicide rates decline. Accordingly, there appears to be little mutual influence of drug overdose deaths and gun

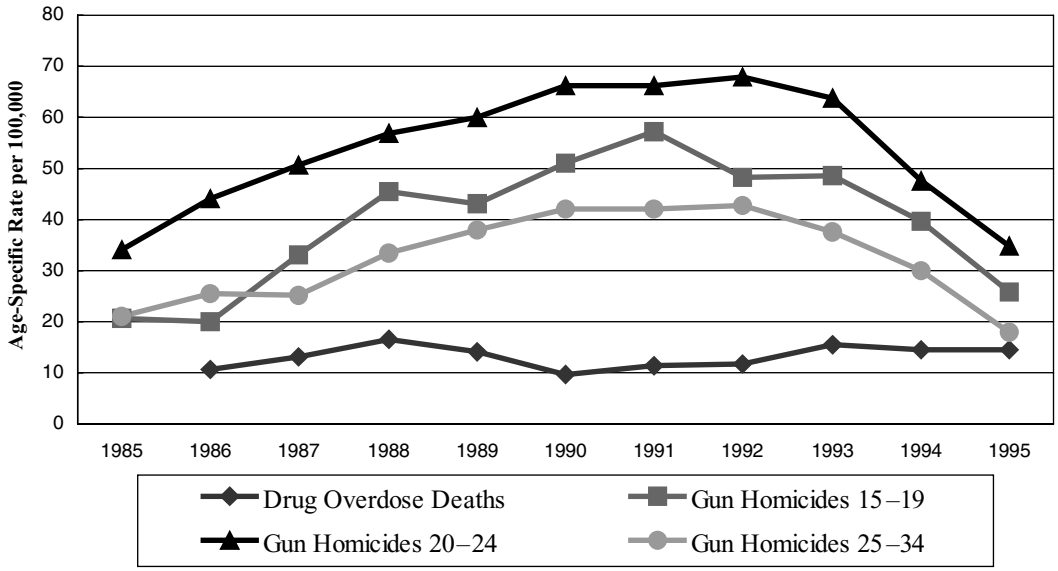

Figure 36.4. Drug overdose death and age-specific gun homicide victimization rate, 1985–1995. Source: Office of Vital Statistics and Epidemiology, New York City Department of Health and Mental Hygiene, various years.

homicide trends for any of the three age groups.[6] Changes in drug use patterns may explain this disjuncture, with drugs such as heroin returning as the favored street drug and displacing crack and crack markets (Curtis, 1998). These drugs are more likely to cause overdose deaths.

An alternative indicator of drug market activity is drug arrests. The size, location, and intensity of drug markets can be approximated by drug arrest rates (Cork, 1999; Rosenfeld & Decker, 1999). Accordingly, drug arrests reflect both strategic decisions by police and drug market characteristics. In conjunction with other indicators, arrests are a useful marker of drug trends. However, the trend lines in Figure 36.5 for age-specific homicide victimization rates and felony drug arrest rates show little relationship between gun homicides and drug arrests. Both drug arrests and gun homicide rates increase from 1986 through 1989, but the trend lines move in different directions after that. Homicides increase through 1991 for adolescents and 1992 for young adults. Drug arrests decline from 1990 through 1993 and begin to rise again in 1994. Most of these felony drug arrests were for sale or possession with intent to sell, and most were either crack or cocaine arrests, the two drugs that were traded most actively in street markets. The portion of felony drug arrests that involved crack or cocaine rose from 57% in 1986 to 64% in 1988 and declined steadily to 48% in 1995.

These figures show that neither drug selling activity nor increases in problematic drug consumption adequately explain the run-up and decline in gun homicides. Violence associated with drug use remained relatively infrequent during the onset of the crack crisis (Fagan, 1992; Fagan & Chin, 1989, 1991). Moreover, the share of homicides due to drug selling did not rise during the homicide run-up (Goldstein et al., 1989). Drug selling accounts for an unknown proportion of homicides, with estimates ranging from about 10% nationwide in the FBI's Supplemental Homicide Reports (FBI, various years) to 50% in local studies in New York (Goldstein et al., 1989) or Los Angeles (Klein, Maxson, & Cunningham, 1991). Thus, a decline in street-level drug selling activity may have reduced, to some unknown extent, the types of social interactions that lead to gun killings. But drug

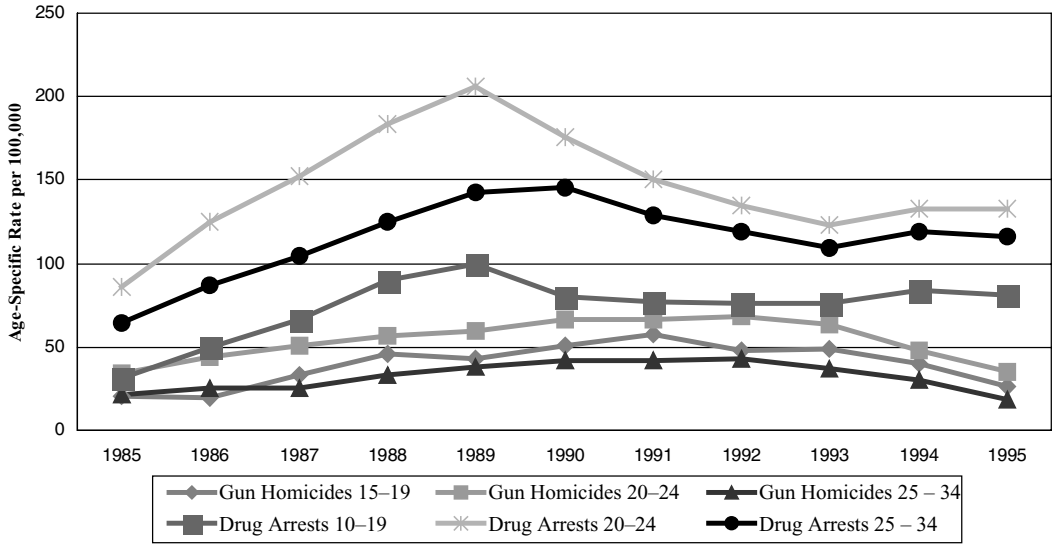

Figure 36.5. Age-specific felony drug arrest rates and gun homicide victimization rates, 1985–1995. Source: Office of Vital Statistics and Epidemiology, New York City Department of Health and Mental Hygiene, various years; New York City Police Department, various years.

selling alone is unlikely to have produced the unprecedented run-up or decline in gun killings so consistently across time, social groups, and areas.

Finally, demographic changes also offer limited explanations for either the homicide increase or decline. The population for the highest risk groups, non-White males aged 15 to 29, declined by about 10% from 1985–1995 (Fagan et al., 1998), a far smaller scale of change than the change that could produce the observed declines in gun homicides. Although it is tempting to dismiss demography as a correlate of the homicide decline, the relationship of population to a changing behavioral pattern may be nonlinear (Gladwell, 2000). In other words, did the population decline reach a threshold where it could lead to a decline in the incidence of gun homicides? According to Burt (1987), network density promotes social contagion by increasing exponentially the extent of contact between persons within groups is nonlinear. Perhaps population density among the highest risk groups rose during the run-up in violence and reached a threshold or tipping point at which behavioral change accelerated and spread through a population before beginning its process of decline (see, for example, Crane, 1991; Gladwell, 2000). As the population declined, so too did the rate of contacts. This is a plausible but unlikely explanation. First, the age decline was small: less than 10% in the highest risk groups. Second, the breakdown in age grading of violence during this period may have mitigated cohort effects and diffused behaviors broadly across age groups. Like the effects of declining drug markets, the contraction in the highest risk population is a potentially important influence in the decline in firearm homicides from 1992–1996, one that may contain the mechanisms of decline. We turn to these mechanisms in the next sections.

Neighborhood Effects on Social Contagion of Youth Homicide

We begin with a set of analyses that estimate the probabilities of the diffusion of behaviors across social areas or neighborhoods. We use the census tract as the boundary for "neighborhood," based on the size (area) of tracts in New York and their

isomorphism with important social units, such as public housing developments and feeder school patterns. Tracts are commonly used to represent neighborhoods in sociological research because of their size and robustness in predicting variation in a variety of social interactions (see, for example, Land, McCall, & Cohen, 1990). Other studies have estimated diffusion across similarly small areas comprised of a few tracts that represent neighborhoods with meaningful social boundaries (e.g., Crane, 1991; Fagan, West, & Holland, 2003; Morenoff, Sampson, & Raudenbush, 2001). These analyses set the stage for the analysis in Part V of microsocial interactions in which social interactions animate dynamics of social contagion that diffuse violence across groups and places.

Susceptibility: Neighborhood Risk

We draw on the literature of neighborhoods and violence to construct a framework of structural risk that simultaneously compromises resilience against transmission while increasing susceptibility. Both theory and empirical research suggest that neighborhoods are susceptible to the spread of violence when structurally weakened (Massey, 1995; Patterson, 1991; Roncek & Maier, 1991; Rose & McClain, 1990; Taylor & Covington, 1988). For example, recent studies suggest that violence shares several explanatory variables with concentrated poverty (Sampson & Lauritsen, 1994; Wilson, 1987, 1991), resource deprivation (Land et al., 1990; Williams & Flewelling, 1988), and inequality (Messner & Tardiff, 1986; Morenoff & Tienda, 1997; Sampson, 1987; Shihadeh & Steffensmeier, 1994). These constructs describe the lack of sufficient means, including income poverty and inequality, to sustain informal social control (Sampson, 1993; Sampson & Wilson, 1995).

Wilson (1987) argues that there has been both an economic and a social transformation of the inner city, in which the exodus of manufacturing jobs beginning in the 1970's has changed the social and economic composition of inner cities, leading to a concentration of resource deprivation. He suggests that the concentration of resource deprivation in specific areas led to dynamic changes in the processes of socialization and social control in those areas. As middle- and working class African American families moved away from the inner cities when their jobs left, there remained behind a disproportionate concentration of the most disadvantaged segments of the urban populations: poor female-headed households with children and chronically unemployed males with low job skills. The secondary effects of this exodus created conditions that were conducive to rising teenage violence: the weakness of mediating social institutions (e.g., churches, schools), and the absence of informal social controls to supervise and mentor youths.[7] Wilson (1987) refers to these conditions of weak social control as social isolation.

The concept of social isolation suggests an ecological dynamic in which the components of poverty and structural disadvantage are interconnected with the dynamics of social control and opportunity structures. The decline of manufacturing jobs increased unemployment among adult males, primarily African Americans, whose lack of technical skills and deep human capital limited them to low-wage and short-term unskilled labor positions. Other economic transformations, including the rise of service and technical jobs outside central cities, motivated the exodus of middle-class families to the outer rings and suburbs surrounding the inner cities. Remaining within the abandoned central cities were unskilled males whose "marriage capital" was low, giving rise to an increasing divorce rate and declining marriage rate.

Changes in the composition of central city neighborhoods also weakened the social institutions that were critical to the informal social control and collective supervision of youths. The weakening of social controls had their strongest effects in transactional settings of neighborhoods and in places like schools and church where adolescent development takes place. And, the exodus of middle-class families from inner cities weakened the political strength of the remaining families leading to physical deterioration

(Wallace, 1991), lower housing values, and in turn increased residential (spatial) segregation (Massey & Denton, 1993).

In turn, the social isolation of people and families was extended to institutions (Wacquant & Wilson, 1989). The rise in poverty and weakening of social institutions also undermined the presence of and institutional support for conventional behaviors. As a result, 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; Tienda, 1991; Wilson, 1987). These dynamics in turn attenuated neighborhood social organization, increasing the likelihood that illegitimate opportunity structures would emerge: illegal economies including drug distribution or extortion, gangs (Fagan, 1989, 1993; Brotherton et al., 2004), and social networks to support them. 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, 1993).

Accordingly, violence and homicide are more likely to occur in an ecological context of weak social control, poorly supervised adolescent networks, widespread perceptions of danger and the demand for lethal weapons, and the attenuation of outlets to resolve disputes without violence. It is in this ecology of danger that violence becomes transmittable through weapons and their impact on perception and decision making in social interactions.

Analytic Models: Diffusion and Contagion

We estimated models of contagion of gun violence (homicide and assault) and its diffusion across New York City neighborhoods from 1985–2000. Although the sharpest changes in violence rates occurred from 1991–1995, we took advantage of data through 2000 to chart the continuing pattern of decline over the succeeding 5 years. We tested two distinct conceptual models for the spread of gun violence from one neighborhood to the next. An outward contagion model posits that adolescent violence spreads out from a central census tract (T) or the immediate neighborhood to adjacent census tracts (X, Y, and Z) or the surrounding community. In this model, the incidence and prevalence of adolescent homicide or assault violence in a given neighborhood exert a significant influence over the incidence and prevalence of adolescent violence rates in the adjacent community.

This influence is hypothesized to operate in at least two different ways. First, a threshold effect is expected concerning adolescent homicide counts, such that the presence of at least one adolescent homicide in a given neighborhood will substantially increase the probability of experiencing at least one adolescent homicide in the surrounding community. Second, with respect to adolescent homicide rates more precisely, positive covariation is anticipated whereby increases or decreases in the adolescent homicide rate of violence in a neighborhood are reflected in concomitant increases or decreases in the surrounding community's adolescent homicide violence rate.

It is also possible that the contagion effect of adolescent violence is reversed. Accordingly, the inward contagion model asserts that the level of adolescent violence in an immediate neighborhood is at least partially contingent on the level of adolescent violence in its broader community. Again, the two distinct relationship forms (threshold effect and positive co-variation) are possible. By considering the simultaneous influence of adjacent spaces, we address the problem of spatial autocorrelation by effectively controlling for mutual influences within and over time.

In addition to corresponding adolescent violence rates, both the outward and inward contagion models incorporate relevant structural and demographic features of neighborhoods and communities as key explanatory constructs. Thus, for the full outward contagion model, the presence and rate of the adolescent homicide rate or assault rate in the surrounding community are a function of the relevant characteristics of the

community, as well as the presence or rate of adolescent homicide violence in the neighborhood. In contrast, the full inward contagion model suggests that both relevant neighborhood features and the presence or rate of adolescent homicide in the community predict the presence or rate of adolescent violence in the neighborhood. Although these factors are presumed to play a significant, independent role in the prediction of adolescent violence rates, it is nonetheless hypothesized that effects of homicide violence rates as independent variables will remain substantial, even once controls for relevant neighborhood and community characteristics have been introduced.

Models were estimated using mixed effects regression models. Mixed effects regression models can be used to approximate multilevel models in which data are hierarchically organized. For example, this class of models is useful in such cases as estimating the simultaneous effects of school climate and individual student family background on standardized test scores, or neighborhood characteristics and household composition on crime rates (Bryk & Raudenbush, 1992; Singer & Willett, 2003; Snijders & Bosker, 1994). Mixed effects models also are useful in estimating individual growth curves or within-subject change over time. In these examples, we might specify the ecological effects of school or neighborhood as fixed effects and individual household or family influences as random effects. This specification approximates the presumed hierarchy of influences. One may reverse the specification as well, comparing estimates to assess reciprocal effects between the two sets of predictors. Mixed effects models simulate the hierarchy of effects by estimating the differences in error-co-variance matrix structures for each set of effects (Singer & Willett, 2003).[8]

In this analysis, the contagion models are specified using both fixed and random effects. The fixed effects of the neighborhood (outward model) and adjacent community (inward model) structural characteristics and violence rates are interpreted as standard regression coefficients. But the analytic strategy also assumes that neighborhoods and communities have varying mean violence levels and that they exhibit distinct trajectories of violence over time. We account for the variance in average levels of violence by specifying random intercepts in our models. To estimate changes over time, we use a repeated measures design in which year is included as a random effect that approximates a developmental growth curve (Goldstein, 2003). We also estimate an autoregressive co-variance structure to account for the strong correlations of homicide and violence rates through time in census tracts and their surrounding neighborhoods.

We use the Poisson form of the model to estimate counts of both gun and nongun homicides and assaults. Poisson techniques are appropriate to identify factors that predict the number of occurrences of an event within a specific observation period (Gardner, Mulvey, & Shaw, 1995; Land, McCall, & Nagin, 1996). Such "count" models are appropriate, given the relatively low number of homicides in most tracts in most years and the sharp right-hand skew in the distribution of both homicides and assaults. To estimate the adolescent component of the total violence rate, including both homicides and assaults, we included co-variates that estimate the age composition of neighborhoods including the ratio of youths aged 15–24 to persons over age 50 in each tract. Because homicide and assault victimization during this period was disproportionately due to gun violence and also concentrated among African Americans, we estimate separate models for total homicide or assault, gun homicide or violence, and gun victimization for African Americans.

Data

DEPENDENT VARIABLES

The dependent variables include counts of homicides, gun homicides, injury assaults that lead to hospitalization, and gun injury assaults. We also estimate specific counts of these variables for African American victims. These data were obtained from the

Table 36.3: Means and standard deviations for census tracts, New York City, 1990

Variable	N	*Mean*	*Standard Deviation*
% Households with Public Assistance Income	2,157	13.81	13.28
Gini for Total Household Income	2,157	0.37	0.09
% Households Under Poverty Level	2,157	18.10	15.06
% High-School Graduates – Total – 25+	2,174	66.33	16.34
% Employed in Managerial, Professional, or Technical Jobs	2,162	29.69	15.17
Employment Rate	2,164	90.27	7.08
Labor Force Participation Rate	2,175	60.64	11.55
% Non-White	2,175	54.86	36.17
Racial Fragmentation Index	2,175	0.38	0.19
% Female Headed Households with Children <18	2,157	10.09	10.43
Supervision Ratio (25–64 by 5–24)	2,162	2.42	2.08
% Youth Population (5–15)	2,175	14.69	6.74
Residential Mobility – Same House as 1985	2,175	62.93	11.84
Population – 1990	2,216	3,304.41	2,465.07
Foreign Born	2,175	27.32	15.13
Linguistic Isolation	2,175	10.95	10.13
Vacancy Rate	2,160	5.53	5.93
% Occupied Units That Are Rentals	2,157	65.10	25.89
Density – Mean Persons Per Occupied Room	2,157	0.67	0.61

Source: U.S. Census Bureau, 1990 Census of Population and Housing, Summary File STF 3 AFile.

Injury Surveillance System of the New York City Department of Health and Mental Hygiene. The Injury Surveillance System collates information from the New York City Department of Health (DOH) to form a database of injury and fatality locations for cases involving violence victims. All weapon assault injuries, fatal and nonfatal, are generated through this archive. The data come from two public health data sources: Vital Statistics (mortality records) and the New York State hospital admissions database, SPARCS, for hospitalized assault injuries. The latter data were compiled from the hospital patient discharge summaries, with ICD-9 E-Codes (injury codes) for the supplementary classification of external causes of injury. The data are hierarchically organized to avoid duplications for persons who were initially hospitalized but then died.[9] Unfortunately, nonlethal injury data were unavailable until 1990, and so the time series begins at the point when the trends already had begun their decline.

INDEPENDENT VARIABLES

Independent variables operationalize the model of neighborhood risk or susceptibility described earlier. Data reflecting the structural and demographic composition of neighborhoods and communities are predictors in the contagion models. Following Land et al. (1990), we selected 19 tract-level variables from the 1990 Census (STF3A and 3B files) to characterize social areas. Means and variances for the 19 initial variables are presented in Table 36.3. Principal components analysis was used to eliminate autocorrelation among the 14 variables and identify 3 orthogonal and conceptually distinct factors. Table 36.4 shows that neighborhoods and communities are characterized along three dimensions: deprivation, population characteristics, and social control. Because communities are actually composites of census tracts, their factor scores are actually weighted factor score composites. Estimates were weighted by the 1990 tract population.

Table 36.4: Factor composition, New York City census tracts, 1990

Factor	*Rotated Factor Score*	*Eigenvalue*	*% Variance Explained*
Poverty/Inequality		2.54	84.73
% Households Under Poverty Level	0.96		
% Households with Public Assistance Income	0.93		
Gini for Total Household Income	0.87		
Labor Market/Human Capital		2.60	65.03
% High-School Graduates – Total – 25+	0.91		
% Managerial, Professional, or Technical Jobs	0.84		
Employment Rate	0.74		
Labor Force Participation Rate	0.72		
Segregation		1.31	65.27
Racial Fragmentation Index	0.81		
% Non-White	0.81		
Social Control #1 – Supervision		1.97	65.56
% Youth Population (5–15)	0.92		
% Female Headed Households w/Children <18	0.80		
Supervision Ratio (25–64 by 5–24)	−0.70		
Social Control #2 – Anonymity		1.02	50.78
Population – 1990	0.71		
Residential Mobility – Same House as 1985	0.71		
Immigration		1.52	76.25
Linguistic Isolation	0.87		
Foreign Born	0.87		
Housing Structure		1.24	41.48
% Occupied Units That Are Rentals	0.76		
Density – Persons Per Occupied Room	0.55		
Vacancy Rate	0.60		

CONTAGION EFFECTS

The "contagion effect" of adolescent homicide is not expected to be immediate. Similar to the concept of incubation, it is more reasonable to assume that some period of time must elapse between the occurrence of an adolescent homicide (threshold effect) or change in the adolescent homicide rate (positive co-variation) and the realization of a related occurrence or rate change. For this study, the time elapsed is estimated to be 1 year. For example, with the outward contagion model, the adolescent homicide rate in a neighborhood for 1990 is used to predict the rate in the surrounding community for 1991. Conversely, the adolescent rate for a neighborhood in 1993 under the inward contagion model is estimated using the community adolescent homicide rate from 1992. However, 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.

Results

On a preliminary note, we remind readers that the critical parameter in this class of models is the interaction of the time by contagion measures. The parameter estimate for the interaction indicates whether the rate of change in the parameter is a significant predictor of the rate of change of the dependent variable. The main effects can be interpreted as explanations for

differences in the average rates over the entire duration of the panel. The results in Tables 36.5 and 36.6 show parameter estimates for the factors of interest in the contagion story, including spatial autocorrelation. All models are controlled statistically for the neighborhood susceptibility factors discussed earlier. We use a quadratic form of the time parameter to reflect the nonlinear distribution of the homicide and injury rates over time.

There is evidence of both inward and outward contagion for homicide and injury assault, but the patterns vary by type of violence, means of violence, and race of the victims. For total homicide, including gun and nongun homicides, Table 36.5, Panel A shows results of three outward contagion models. Neither total homicides nor gun homicides are statistically significant. The parameter estimate for the interaction of time by homicide in the surrounding tracts is significant only in the model for gun homicide victimization of African Americans, and the direction is negative. The interpretation for the coefficient is somewhat counterintuitive – it does not imply that there are fewer homicides in the surrounding tracts over time. Instead, as the overall homicide trend declines, a negative coefficient suggests that this factor is opposing that trend in the dependent variable. In this case, then, the negative coefficient for the time by homicide interaction indicates that the rate of homicide victimization of African Americans in the surrounding neighborhoods is increasing, controlling for the rates in the surrounded neighborhood in the previous year. The exponentiated coefficient is 0.989, suggesting that an increase of one homicide in a neighborhood in T_0 predicts an increase of 1.1% in the number of homicides in any of the surrounding areas.

Panel B in Table 36.5 presents consistent evidence of homicide contagion. The estimates for time by contagion are significant and negative in all three models. The exponentiated coefficient for total homicide is 0.992, suggesting that an increase of one homicide in the surrounding tracts in T_0 predicts an increase of 0.8% in the number of homicides in the surrounded tracts in T_1. For the other models in Panel B, the effects also are significant and are larger: an increase of 1.0% for gun homicides for an increment of one homicide in the surrounding tracts, and 2.0 for gun homicide victimizations of African Americans.

The results for contagion of injury assault suggest that there are contagion effects. All the models in both panels of Table 36.6 are significant, but the effects seem at first glance to be small. The exponentiated coefficients suggest that assaults increase by less than 1% for each increment in assaults in the surrounding tracts for outward contagion or in the surrounded tracts for inward contagion. Contagion is evident both for gun violence and nongun violence in these models, suggesting a more ambiguous role of guns. Although there may be a cross-over contagion effect of gun violence to nongun violence in the surrounding areas, it is unlikely conceptually and empirically to be any larger than these observed effects for within-type contagion.

Several features of this analysis merit further note. First, the coefficients for outward homicide contagion parameters are relatively small and significant only for the model for African American gun homicide victimization. This is not surprising, given that homicides are comparatively rare events. But the primary tale in Table 36.5 is one of inward contagion. Consider first the sheer magnitude of the estimates for spatial lag in the outward models, and second the large and significant coefficients for the inward contagion parameters. Taken together, these findings confirm that the inward contagion of homicide is more potent than outward contagion, that a neighborhood is affected more by its adjacent community than it affects that same community.

Though not as dramatic, the same pattern is evident in Table 36.6. Here, there is evidence of both outward and inward contagion of nonlethal assaults. The coefficients for the outward contagion of assault are not large, but they are consistently significant. In relative terms, however, the coefficients

Table 36.5: Poisson regression results for models of inward and outward contagion of homicide, New York City census tracts, 1985–2000 (coefficient, *t* statistic, statistical significance of predictor, exponentiated coefficient)

A. Outward Contagion	*Total Homicides*				*Gun Homicides*				*African American Gun Homicides*			
Predictor	B	Exp (B)	t	p(t)	B	Exp (B)	t	p(t)	B	Exp (B)	t	p(t)
Intercept	−0.069	0.942	−3.35	b	−0.4919	0.611	−26.00	c	−2.169	0.114	−63.32	c
Time	0.112	1.127	26.56	c	0.149	1.160	28.94	c	0.155	1.167	20.35	c
Homicide Contagion	0.003	1.003	1.01	ns	0.008	1.008	2.22	a	0.040	1.041	4.34	c
% African American Population	0.010	1.010	22.54	c	0.012	1.012	24.20	c	0.030	1.030	35.14	c
Homicide Spatial Lag	0.888	2.429	119.73	c	1.169	3.219	117.99	c	1.425	4.157	100.77	c
Time *Homicide Contagion	−0.0009	0.999	−0.84	ns	−0.002	0.990	−1.30	ns	−0.011	0.989	−2.73	c
Time *Homicide Spatial Lag	−0.072	0.931	−20.88	c	−0.098	0.906	−19.88	c	−0.085	0.918	−9.11	c

B. Inward Contagion	*Total Homicides*				*Gun Homicides*				*African American Gun Homicides*			
Predictor	B	Exp (B)	t	p(t)	B	Exp (B)	t	p(t)	B	Exp (B)	t	p(t)
Intercept	−1.743	0.175	−70.27	c	−2.204	0.110	−77.92	c	−2.176	0.113	−58.44	c
Time	0.156	1.168	14.80	c	0.1888	1.207	15.56	c	0.173	1.189	11.28	c
Homicide Contagion	0.050	1.052	26.61	c	0.066	1.069	25.88	c	0.078	1.081	10.18	c
% African American Population	0.012	1.102	23.52	c	0.014	1.014	23.97	c	−0.016	0.984	−13.66	c
Time *Homicide Contagion	−0.008	0.992	7.47	c	−0.010	0.990	−8.02	c	−0.020	0.980	−5.30	c

p(t): a < 0.05; b < 0.01; c < 0.001; ns = not significant.

1. All models were estimated using mixed effects Poisson regression models with autoregressive co-variance structures.
2. Outward contagion models include adjustment for spatial autocorrelation of homicides in the surrounding census tracts.
3. All model results were adjusted for effects of tract social and economic factors: percent of persons below poverty, percent in labor market, ratio of adults to juveniles, percent population living in tract 5 years or less, percent population not born in U.S., percent of households in public housing, and total population.
4. All models were estimated with time as quadratic (nonlinear) predictor.

Table 36.6: Poisson regression results for models of inward and outward contagion of assault, New York City census tracts, 1990–2000 (coefficient, *t* statistic, statistical significance of predictor, exponentiated coefficient)

A. Outward Contagion	*Total Assaults*				*Gun Assaults*				*African American Gun Assaults*			
Predictor	B	Exp (B)	t	p(t)	B	Exp (B)	t	p(t)	B	Exp (B)	t	p(t)
Intercept	2.226	9.263	120.58	c	0.086	2.356	37.04	c	−0.338	0.713	−9.98	c
Time	−0.004	0.996	−42.87	c	−0.008	0.992	−46.81	c	−0.008	0.992	−31.33	c
Assault Contagion	0.0002	1.002	3.45	c	0.008	1.008	2.33	c	0.004	1.004	0.81	ns
% African American Population	0.008	1.008	17.31	c	0.012	1.012	21.41	c	0.026	1.025	30.70	c
Assault Spatial Lag	0.100	1.105	67.64	c	0.283	1.328	33.88	c	0.303	1.354	24.70	c
Time *Assault Contagion	−0.0001	0.999	−2.45	c	−0.0002	0.998	−3.57	c	−0.0002	0.998	−2.19	a
Time *Assault Spatial Lag	0.001	1.001	44.94	c	0.004	1.004	31.17	c	0.005	1.005	26.18	c
B. Inward Contagion	*Total Assaults*				*Gun Assaults*				*African American Gun Assaults*			
Predictor	B	Exp (B)	t	p(t)	B	Exp (B)	t	p(t)	B	Exp (B)	t	p(t)
Intercept	0.506	1.659	22.13	c	−1.113	0.328	−36.43	c	−2.64	0.071	−58.43	c
Time	−0.007	0.993	−11.99	c	−0.021	0.979	−18.27	c	−0.023	0.978	−12.89	c
Assault Contagion	0.005	1.005	15.66	c	0.020	1.020	13.91	c	0.021	1.021	10.87	c
% African American Population	0.011	1.01	21.37	c	0.016	1.016	24.47	c	0.031	1.032	34.65	c
Time *Assault Contagion	−0.0001	0.999	−3.59	c	−0.001	0.999	−9.06	c	−0.001	0.999	−7.99	c

p(t): a < 0.05; b < 0.01; c < 0.001.

1. All models were estimated using mixed effects Poisson regression models with autoregressive co-variance structures.
2. Outward contagion models include adjustment for spatial autocorrelation of homicides in the surrounding census tracts.
3. All model results were adjusted for effects of tract social and economic factors: percent of persons below poverty, percent in labor market, ratio of adults to juveniles, percent population living in tract 5 years or less, percent population not born in U.S., percent of households in public housing, and total population.
4. All models were estimated with time as quadratic (nonlinear) predictor.

for the inward contagion assault model again indicate that neighborhoods exert less of an influence over their surrounding communities and instead are more susceptible to events in those communities.

The presence of gun contagion of both gun homicides and gun assaults underscores the importance of guns in the dynamics of social contagion at the population level. In an earlier article, Fagan and Davies (2004) showed that the neighborhoods with the highest homicide, injury, and gun violence rates are New York City's poorest neighborhoods. Those finding were confirmed in these models in the significant contribution of the co-variates that express the susceptibility of the poorest neighborhoods to contagion (data available from authors). Accordingly, the corollary finding of socioeconomic risk as a contributor to the spread of violence captures both the significance of susceptibility and the importance of structural equivalence in shaping the trajectory of diffusion. That is, the adoption at the population level of gun violence as a means of social control and exchange was facilitated by the social concentration of poverty and of the close social synapses intrinsic to poor neighborhoods. Accordingly, the social and spatial clustering of homicide suggests that it is concentrated within overlapping social networks in small areas (Fagan & Wilkinson, 1998b).

Social contagion theory suggests that individuals are likely to mutually influence the behaviors of others with whom they are in frequent and redundant contact (Bovasso, 1996, p. 1421; Burt, 1987). The social interactions underlying assaultive violence suggest its spread by social contact (Loftin, 1986), and, as we show below and in other articles, by specific forms of social interaction (Wilkinson, 2003). We explore these themes next.

Violent Events, Social Networks, and Social Contagion

We turn next to an analysis of the individual- and group-level processes of social contagion. We identify dynamic social processes that fuel the social contagion of youth violence. At the heart of this process are the interactions of individuals within and across social networks. Violence plays a central role in the maintenance of organizational boundaries, norms, and cohesion. Two elements in the contagion of youth violence are conflict between networks of youths and the role of violence in resolving conflicts. The violent events in which these processes unfold and change over time represent opportunities to build or maintain status within networks; in some events, violence is an imperative with costs when it is not invoked (Wilkinson, 2003). Third parties are especially important in the spread of violence between networks; third parties can animate or intensify violence once a conflict begins, or they can help mediate and suppress it. They also convey the outcomes of violent events to others in the larger social worlds that surround these social networks, helping sustain norms and provide context for the next conflicts that may arise. The strategic role of guns in these processes intensifies the dynamics that fuel the epidemic. In some instances, the presence of guns in events links together persons and events across time and sustains the processes of social contagion.

This section unfolds in three parts. First, we discuss the mechanisms through which violence may spread between social groups or networks and show how these mechanisms are best understood through the analysis of specific events. Next, the chapter focuses on the importance of third parties in violent events. The third section presents three scenarios that illustrate the intersection of these themes that produce violent events and often set the stage for future ones.

Violent Events

For any attitude, expectation, behavior, or virus to be "spread" from person to person or group to group, interpersonal contact is typically required. Exposure can be direct or indirect. Direct exposure to gun use and violent behaviors among similarly

situated networks of young men would likely increase the risk of transmission. Observations of young men's decision-making processes in violence use and avoidance provide a window into the processes that shape interpersonal transmission. Studying violence from an event perspective combines the study of offenders, victims, and social context to yield a more complete picture of its etiology (Meier, Kennedy, & Sacco, 2001). The event perspective considers the co-production by victim(s), offender(s), and others of a violence experience. It emphasizes event precursors; the event as it unfolds; and the aftermath, including reporting, harm, and redress. The event perspective integrates concepts from symbolic and situational interactionism, routine activity, and rational choice theories. The social geometry of violent conflict provides clues to understanding what distinguishes one conflict situation from another or more precisely what distinguishes a nonviolent conflict from a violent conflict (Phillips & Cooney, 2005).

Several investigators have found evidence that the interplay between the primary actors determines, in part, the outcome. For example, Felson and Steadman (1983) found that violent incidents usually began with identity attacks, were followed by attempts and failures to influence the opponent, then included verbal threats, and finally, ended in physical attack. In a study of ex-offenders, ex-mental patients, and a sample drawn from the general population, Felson (1982, 1984) found a similar pattern. Hughes and Short (2005) confirmed Felson's earlier findings with a sample of gang-involved youth.

Similarly, Oliver (1994) used detailed narratives of violent confrontations between Black males in bars and bar settings. Oliver employed both participant observation and interview methods over a 5-year period (1983–1987) to "systematically examine the social functions of the black bar and how black males interacted with each other and with females in this setting." The sample consisted of 41 Black men 28 to 45 years old who frequented the research locations. Oliver examined violent behaviors to identify the "rules of engagement" and situational causes of violence in the bar setting. He observed a five-stage sequence of events similar to Felson and Steadman's previous classification. His work added insights about violent events from a sample of African American men, especially with regard to understanding event closure and the aftermath of violent events. In all of these studies, victim actions, including retaliation, denial of claims, and aggressiveness, were found to be important factors.

A focus on violent events demonstrates that most violence is a process of social interactions with identifiable rules and contingencies (Campbell, 1986; Felson, 1982; Felson & Steadman, 1983; Luckenbill, 1977; Luckenbill & Doyle, 1989; Oliver, 1994; Polk, 1994; Sommers & Baskin, 1993; Wilkinson, 2003). Contrary to common wisdom, violent acts can be understood as rational or purposive behavior. Most experts agree that rationality is "bounded"; that is, individuals rarely have all of the information necessary to make a truly "rational" decision. The likelihood of violence reflects the progression of decisions across a series of identifiable stages. Much of this research concludes that there are contingencies in each stage, shaped by external influences and social interactions of the actors. Yet the data have generally not been available to answer more useful questions, such as what the contingencies are, how actors take them into account, and how they vary as an event progresses through these stages. Although prior studies provide generalized classifications of violent event stages, a finer-tuned assessment of the actions and reactions of actors in violent encounters than is possible from detailed event narratives will shed new light on the micro-decisions and contextual influences across a range of types of violent encounters. Previous studies have generally ignored information about precipitating actions, as well as the aftermath of violent events. The analysis of event stages must also take into account the heterogeneity of violent events by examining a wide range of violent acts.

Studies of Third Parties in Violent Events

Third parties witness or somehow become involved in an estimated two thirds of acts of interpersonal violence in the United States (Planty, 2002). The percentage is even greater (approximately 73%) for violence among young people. Despite the common nature of third-party presence, researchers know little about the specific contributions that third parties make in promoting or preventing the escalation of interpersonal conflict to violence. Previous research concludes that bystanders and third parties contribute significantly to the outcome of violent encounters (see Black, 1993; Cooney, 1998; Decker, 1995; Felson, 1982; Felson, Ribner, & Siegel, 1984; Oliver, 1994; Phillips & Cooney, 2005; Wilkinson, 2003). For example, Felson (1982, 1993) found that, when a dispute occurred between parties of the same sex, the presence of third parties increased the likelihood that a verbal disagreement would turn into a physical fight. Third parties may be viewed both as part of the sociocultural context and as participants in the co-production of violent events. Largely due to the work of Donald Black and his followers, theory in the area of third parties has evolved while empirical studies of third-party roles in violence remain rare and unfocused within criminology.

Black's (1993) theoretical work on the social structure of conflict includes a typology of third parties with specification across two domains: the *nature* and *degree* of the intervention. He identified 12 third-party roles, "including five support roles (informer, adviser, advocate, ally, and surrogate) and five settlement roles (friendly peacemaker, mediator, arbitrator, judge, and repressive peacemaker)." Two other roles that do not fit within either category are the "negotiator" whose partisanship cross-cuts both sides and the "healer." The types are rank ordered in terms of degree of intervention, with supporting roles organized by the extent of partisanship and settlement roles by the authoritative status of the third parties. Settlement roles come into play when, according to Black (1993, p. 108), "third parties intervene without taking sides."

As Black explains, the role of third parties often depends on personal allegiance (or lack of it) to the main actors. He argues that audience members allied with either the protagonist or the antagonist may contribute to the escalation or de-escalation of a dispute through verbal statements, body language, cheering, nonverbal social pressure, or physical acts of violence. *Partisanship* and *solidarity* are key features of Black's thesis. Cooney (1998) elaborated Black's theory to include variables on group membership status and articulated hypotheses for four configurations of third-party social locations in determining their influence over the principals in a conflict situation. They specified the predictive power of third parties with close and distant ties to individuals, close and distant ties to groups, cross-cutting ties, and no ties. Using interviews with 100 incarcerated offenders of assault or homicide, Phillips and Cooney (2005) found moderate support for these hypotheses in the first empirical test to date.

Wilkinson (2007) examined the role of network peers and third parties as potential agents of social control in 237 violent events reported by 159 urban youth. The study classified third parties by their relational ties to the focal respondent and his opponent(s). The study showed that third parties who were closely tied to the primary participants were mostly likely to *join in* the violence, rather than doing anything to stop it (55% of the respondent's associates actively used violence, whereas 42% of the opponent's associates did). Bystanders who were neutral parties toward either side rarely became involved in the violence itself, although they actively engaged in some type of social control action in about 20% of events. Bystander actions typically involved yelling to try to stop the violence (8%), actions to break up the conflict (10%), and coming to the aid of participants nonviolently (3%). From the respondents' perspective, bystanders very rarely called the police. Third-party presence seems to coincide with police becoming

involved in both serious and nonserious events. Events that occurred at night and without neutral bystanders present were less likely to come to the attention to police (at least from the respondents' experience).

Three Scenarios

Moving beyond these descriptive findings, in the remainder of this section we examine *how* third-party involvement relates to social contagion in 782 violent events. An event-level analysis is the best way to examine how violence "flows" as a process from individual to individual, as well as from group to group. By focusing specifically on the dynamics of gun violent events reported by 418 New York City male youth aged 16 to 24, we identify dynamic social processes that fuel the social contagion of youth violence. Peer network involvement in violent acts takes several forms. First, and most common, is co-participation or co-offending. The decision to co-participate happens at any stage as violent conflicts unfold. These processes are evident in analyses of the action by actor sequences of violent events. Peer network members become actively involved in conflicts that lead to violence through several avenues: (1) Their involvement in the violent event is strategic and anticipated from the outset; (2) they come to the aid of an associate who is losing in the battle; (3) they are threatened/offended/disrespected at some point during the course of observing a dispute unfold; (4) they use violence either in the moment or after the fact to obtain justice or right some wrong that was perpetrated against a group member; or (5) they are influenced by gossip about the performance and reputation of violence participants and take up conflicts to restore the reputation of group members. Peer network members who are present during disputes that escalate into violence play different roles depending on the relationship among the combatants, weapon type, and injury outcomes. Three scenarios of violence are presented that illustrate the nuances of how violence is diffused across peer networks.

SCENARIO 1: STREET CORNER STORE AND RESIDENTIAL NEIGHBORHOOD

In this scenario, let's call the respondent (Aron) and the opponent (Bruce). Aron argues with Bruce at a neighborhood corner store over cutting in line. They step outside and begin to fight. None of the witnesses in the setting were closely tied to either party, but they did know both youths by face and reputation. After about 5 minutes of fighting with fists, Bruce pulls out a razor on Aron and uses it to slice him across his arm that Aron had extended to protect his face. Someone in the audience yells at the youth to stop before the police are called. Both youth flee. Aron goes back to his block and recounts the story to his associates. He rallies their support for a counterattack by highlighting the ways that his opponent was trying to destroy his attractiveness by scarring his face and how he disrespected him. After a few days pass and the group was fueled by visions of revenge, Aron and four of his associates armed themselves with handguns and went to Bruce's block. Aron's group finds Bruce, two guys, and one girl sitting on the front steps of a neighborhood building. Without verbal warning, Aron pulls his 9 mm from his waist and starts shooting in the direction of Bruce's group. Bruce was caught off guard as he was not expecting conflict that day. One of Bruce's companions pulls out his gun and returns fire. After a few brief minutes the shooting stops. Two people are shot – one relatively minor wound to the leg on Aron's side and one serious injury to Bruce's friend. According to the respondent, the beef or conflict remains ongoing. Aron is anticipating a retaliatory attack to avenge the injury to Bruce's friend and because he escalated the beef to a "life and death" issue.

SCENARIO 2: A CLUB AT 2 A.M.

Here, Rich, Mike, and four of his associates go to a club to party and socialize with females. In the club, Mike sees a girl named Becky with whom he has a causal relationship. She came to the club with six of her girlfriends to dance and have fun. Rich sees Becky dancing and joins her. Rich rubs

his body up against Becky. Mike observes the violation directly. Mike informs Rich of his wrongdoing and asks for an account. Rich denies wrongdoing, states claim to the girl, and places blame on Mike. Mike pulls the girl away from Rich and returns to his group. Mike's boys comment on the violation. Mike watches Rich, thinks about the violation, and is angry. Mike's boys report back information about Rich and his boys. Mike returns to his prior activity. Mike's girl goes back to dancing.

The girl's friends compliment her on being desired by two guys. Rich talks with his buddies about the girl and Mike's capabilities. Both parties wait to see what the other will do. After some time passes, Rich begins dancing provocatively with Mike's "girl" again. Mike is not watching, but hears about it from his man. Rich's friends watch to see if Rich gets the girl. Mike walks up to Rich and punches him in the face. Rich hits Mike back. Both sets of friends watch initially. Rich lands some good punches. Mike's first friend jumps on Rich. Club security breaks up the fight, issuing warnings. The youths go back to drinking and partying.

Mike discusses ways of punishing Rich. Both sides watch the other. The status of who "gets" the girl remains open. Both sides plan to attack at the end of the night. Mike believed that Rich must have called some of his friends for additional reinforcements and to make sure that when Rich got outside he would have a gun available. Mike and his boys essentially make the same type of preparations. As soon as Mike moved toward exiting the club, Rich's group was preparing for a gun battle. Mike recalls that his side had three guns that they retrieved from nearby stashes, whereas it seemed like the other side had five or more guns. With more than 20 shots fired, injuries were sustained on both sides. The police came to the scene, but no one was arrested.

The injured were transported to the hospital, and one youth from Rich's side died at the hospital as a result of this gun event. Mike heard rumors that Rich had been seen with Becky following the shoot-out, which fueled his anger and, in his mind, justified his need for revenge on Rich. Mike's boy suffered serious damage to his knee, which angered Mike as well. Rumors of revenge for the death of Rich's associate were spreading around. Both sides were on guard and looking for strategic advantage for the next violent event. Mike anticipated that more violence would follow from this event.

SCENARIO 3: A DRUG STASH HOUSE NEAR A STREET DRUG SPOT

Pete and his two associates planned a robbery of a drug stash house manned by a Dominican crew. Pete got information about the best day and time to rob the house, what types of weapons would be used to defend the stash house, and so on. However, his information was incomplete. When he and his associates made their armed robbery attempt, they were confronted by additional armed drug dealers who were protecting the drug stash. Pete's group exchanged fire with the drug dealers as they fled the building following a failed robbery attempt. Pete's friend Franky almost got shot. Franky was recognized by the Dominican drug crew. They came to Pete's neighborhood to find and shoot him as revenge for the attempted robbery incident. Pete describes the situation to our interviewer:

> (Pete): They recognized him and shit. So it was some Dominican kids. They went to our block and we seen like this blue Lincoln Towne or Escort or some shit. We seen this coming around and coming around. And we was like, 'yo Franky, I was like you know them niggas right there man?' He was like, 'nah, I was like the niggas got something with us, either they scheming at us or the niggas ready to hit somebody else.' Niggas is sitting up in there either waiting for somebody or waiting for us to make a move. He gave a peek and as soon as he looked, Franky told me, 'yo Pete that's one of them nigga from that stash house. My hands got real sweaty, we didn't have our ghats on us, My hands got real sweaty. And it's like I could front it off and I call you

from across the way, hey 'yo what's up come here.' You know what I'm saying by that time, Franky tells me yo Pete just run your way and I ran inside the building. And I said alright I'm going to run to this Alicia's house right there where the gate is at. You know what I'm saying the car all of the sudden just right down the block see but we didn't want to run as soon as that shit come. You know what I'm saying as soon as that car come down slowly, it didn't do nothing. The Dominican kid didn't do nothing, he just looked at us. And went right around the block. We was like that ain't them, that ain't them. Give it like two to three minutes they came walking, walking, it was like a good little 50 yards. And I said, 'yo, Franky that's them right there.' He was like, 'where I don't see the car.' I said, 'nah they on foot right there.' And Franky said, 'oh shit, that's them.' When me and Franky look at them they looked at us and I see them real quick running to pull out they ghat. And . . . .they just started busting. Blah, blah, blah and I caught my reaction I ran where I said I was going to run. Franky ran in there but niggas was just kept blah, blah, blah and they went after Franky more than me. They kept just shooting blah, blah, blah and they ran in the building. That was like oh shit, I jumped over the gate and I was oh shit. I didn't have my ghat or nothing. I was damn Franky, Franky.

Interviewer: What happened?

(Pete): Really they didn't caught Franky, You know what I'm saying they didn't get him. But the next day they caught, they shot him up.

(Interviewer): So they came back the next day?

(Pete): Early in the morning cause Franky was out there pitching, early in the morning. They came back the next day and they were shooting at him and he didn't feel it while he was running but it cause the bullet went in and out. I was like you know, everybody was yo what happen why they niggas shot up Franky and shit. We didn't want to tell nobody that we went to stash house to hit. It was like, 'nah we had beef with these kids and they came around and shit like.'

(Interviewer): Why didn't you want to tell?

(Pete): Because if he tell niggas that we hitting other peoples stash houses they going to probably. God forbid they hit up the stash house in our block they the first thing they going to say is 'yo I think it was Pete and Franky and Zee 'cause niggas like hitting stash houses. Everybody in my block think shystyness, so the first nigga they will probably will is to us and they will probably try to smoke us and it ain't us. You know what I'm saying.

The event process can be dissected into specific stages: anticipatory stage, opening moves, countermoves and brewing period, persistence stage, intensification stage, early violence stage, stewing period, assessment stage, the casting/recasting stage, and the retaliatory stage. The examples above demonstrate that network peers play important roles at almost every stage of a conflict that escalates into violence. The communication of normative expectations, violence scripts, and violence strategies filters through direct observation, word of mouth via rumors, and telling of "war stories."

Social Contagion and Social Norms

The dynamics of social contagion can be accommodated within concepts of social influence and social norms. The social influence concept of behavior borrows from both economics and sociology (Harcourt, 1998; Lessig, 1995). Its economic component suggests that people will act to maximize their utility, whereas its sociological dimension suggests that conduct is shaped through direct and vicarious social interactions. A simple version of this nexus suggests that the choice of conduct is influenced by

observation and practice of the most effective options. Choices are contextualized as well, reflecting both the range of available options and the specific contingencies in which they are applied (Fagan, 1999).

In the contagious dynamics of violence, the social meaning of violence is constructed through the interrelationship of its action and its context. The social meaning in this case involves actions (violence) that have both returns (identity, status, avoidance of attack) and expectations that, within tightly packed networks, are unquestioned or normative. Conduct impregnated with social meaning has influence on the behaviors of others in immediate proximity. The social meaning of violence influences the adaptation of behavioral norms, expected responses (scripts), and even beliefs (memes) about systems of behavior. Social norms are the product of repeated events that demonstrate the meaning and utility of specific forms of conduct. Social influence thus has a dynamic and reciprocal effect on social norms (Harcourt, 1998; Lessig, 1995). In poor neighborhoods, social interactions are dominated by street codes, or local systems of justice, that reward displays of physical domination and offer social approval for antisocial behavior.

The endogeneity of social contagion to networks and neighborhoods illustrates the differences in the two types of epidemics. The origins of a contagious epidemic that travels through a population become distal influences on the pathway and dynamics of transmission through populations over time. The setting or context of contagion reflects the susceptibility of populations to the transmission of a socially meaningful behavior, as well as its exposure to the behavior that has acquired meaning (Fullilove et al., 1998). This can be true both for fashion and art (Gladwell, 1997; Servin, 1999) and for problematic social behaviors, such as drug use (Rowe & Rodgers, 1991), teenage sexual activity (Rodgers & Rowe, 1993), teenage pregnancy (Crane, 1991), child maltreatment (Coultin, Korbin, Su, & Chow, 1995), and violence (Anderson, 1999; Cork, 1999; Fagan, 1999; Loftin, 1986).

Recent applications of social influence models to crime control emphasize the seminal role of the exogenous influence of "disorder," in which minor crimes signal to would-be criminals that crimes in that area will be tolerated and not reported (Kelling & Cole, 1996; Wilson & Kelling, 1982). At first glance, "Broken Windows" suggests that there is a spread of norms supporting crime that overwhelm norms of orderliness. The spread comes from the continuing signals from disorder. Withdrawal of the signs of disorder will change social norms by allowing the social influence of orderliness to flourish. Apart from the problematic nature of this dichotomous categorization of persons (Harcourt, 1998), Broken Windows medicalizes the conditions of disorder and criminality. It assumes that exposure to the disorder is a constant and recurring process that signals to the motivated offender that crime can succeed. Removing the signs of disorder will change social norms by allowing the social influence of orderliness to flourish. But this theory is limited by focusing only on the introduction of the original cues or sources of crime and relying on the causal effects of these exogenous factors. This is analogous to the food poisoning model of epidemics. Moreover, a literal reading of Broken Windows theory would invite problematic legal policy responses, such as "social quarantine," which have limited efficacy and raise moral quandaries (Markovits, 2005).

The dynamics of social contagion instead suggest an endogenous process, in which the spread of social norms occurs through the everyday interactions of individuals within networks that are structurally equivalent and closely packed. Here, the ill grows and spreads from the inside, often long after the origins have subsided. This is analogous to influenza contagion or to the spread of cultural or political thought (Cavalli-Sproza & Feldman, 1981).

The concept of contagion neutralizes the categorizations of disorder and order that theoretically inform the new path of deterrence. A literal translation of contagion would emphasize guns as a recurring source of violence and as an agent in the

transmission of violence norms. Because the recent epidemic cycle of violence was in reality a gun homicide epidemic, the case for gun-oriented policing strategies (Fagan, 2002; Fagan, et al., 1998) is much stronger than practices based on the more diffuse and unsupported theory of disorder control and order-maintenance strategies. Although disorder opposes orderliness, cleanliness, and sobriety (Harcourt, 2001), violence appears to travel on vectors quite unrelated to that particular set of social norms.

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Notes

1. Cook and Ludwig (2004) developed estimates of the effects of gun availability, controlling for any effects due to reverse causation in which the demand for guns among teenagers may affect prevalence.
2. One important development is a breakdown in the age grading of behaviors, in which the traditional segmentation of younger adolescents from older ones, and behavioral transitions from one developmental stage to the next, are short-circuited by the strategic presence of weapons. Mixed-age interactions play an important role in this process. Older adolescents and young adults provide modeling influences as well as more direct effects. We found that they exert downward pressure on others their own age and younger through identity challenges that, in part, shape the social identities for both parties. At younger ages, boys are pushing upward for status by challenging boys a few years older.
3. With the exception of the decade influenced by both the passage of the Volstead Act and the social and economic instability of the Great Depression, homicide rates in New York City varied little. In its 1996 report, the Office of Vital Statistics and Epidemiology reports homicide rates prior to 1985 in 5-year intervals. Homicide rates rose from an average of 4.9 in 1916–1920 to 7.6 in 1931–1935, and declined to 4.5 by 1936–1940 (NYC Department of Health and Mental Hygiene, 1997).
4. The 1990 spike for male nongun homicides most likely reflects the 90 arson homicide deaths in the Happyland Social Club fire. We could not adjust the age-, race-, or gender-specific rates for these homicide deaths since data were not available on their characteristics. The nongun total for 1990 has been adjusted by deleting 89 of the 90 killings from the Happyland Social Club fire, in effect counting that episode as one homicide.
5. Exceptions include domestic homicides, and homicides that follow rape (see Dugan, Nagin, and Rosenfeld, 1999).

6. Other indicators, such as drug use among arrestees recorded in the Drug Use Forecasting System (DUF), also show little relationship with trends in gun homicide rates. Fagan et al. (1998) show that the incidence of drug-positive arrestees remained unchanged throughout the period, and was unrelated to both firearm and nonfirearm homicide trends.
7. The male divorce rate also is a consistent predictor of violence and homicide rates, and effects are greater for juveniles than for adults. For example, Messner and Sampson (1991) showed that Black family disruption was substantially related to rates of murder and robbery involving Blacks. These findings are consistent with the consistent findings in the delinquency literature on the effects of broken homes on social control and guardianship. The effects of male divorce can be interpreted either as a consequence and correlate of the rise of female-headed households, or as an indicator of weak social control of children who then are raised primarily by women. Whatever its meaning, the male divorce rate has positive, clear cut effects on robbery, assault, rape and homicide (Sampson and Lauritsen, 1994).
8. A second advantage of the mixed models approach is that it allows for greater flexibility in specifying the covariance structure of the data. Specifically, mixed models allow for the analysis of data where the requisite assumptions of Ordinary Least Squares regression concerning error term independence are violated. This is particularly important in research involving aggregate units. Because the neighborhoods and communities are comprised of geographically contiguous census tracts, autocorrelation is inherent in the data structure and it would be inappropriate to assume a simple covariance structure for these analyses. All of the models are instead analyzed with an autoregressive covariance structure.
9. Additional records are available on deaths from other means (accidents, disease classifications, self-inflicted violence) to estimate overall mortality rates by area and by cause. Accordingly, the database has the capacity for spatial, temporal, and demographic disaggregation and analysis of several dimensions of mortality.

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