Diffusion in Homicide: Exploring a General Method for Detecting Spatial Diffusion Processes

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This article proposes a new method for examining dynamic changes in the spatial distribution of a phenomenon. Recently introduced exploratory spatial data analysis (ESDA) techniques provide social scientists with a new set of tools for distinguishing between random and nonrandom spatial patterns of events (Anselin, 1998). Existing ESDA measures, however, are static and do not permit comparisons of distributions of events in the same space but across different time periods. One ESDA method—the Moran scatterplot—has special heuristic value because it visually displays local spatial relationships between each spatial unit and its neighbors. We extend this static cross-sectional view of the spatial distribution of events to consider dynamic features of changes over time in spatial dependencies. The method distinguishes between *contagious diffusion* between adjoining units and *hierarchical diffusion* that spreads broadly through commonly shared influences. We apply the method to homicide data, looking for evidence of spatial diffusion of youth-gang homicides across neighborhoods in a city. Contagious diffusion between neighboring census tracts is evident only during the year of peak growth in total homicides, when high local rates of youth-gang homicides are followed by significant increases in neighboring youth-nongang rates. This pattern is consistent with a spread of homicides from gang youth to nongang youth. Otherwise, the increases in both youth-gang and youth-nongang homicides generally occur simultaneously in nonneighboring tracts.

KEY WORDS: homicide; exploratory spatial data analysis; spatial diffusion; contagious diffusion; hierarchical diffusion.

1. INTRODUCTION

The decade following the mid-1980s was a period of sharp changes in homicide rates in the United States, especially in cities. While total rates nationally were relatively stable, cycling between 8 and 10 homicides per 100,000 population, selected population groups—notably young black males in cities—experienced staggering increases in their homicide rates

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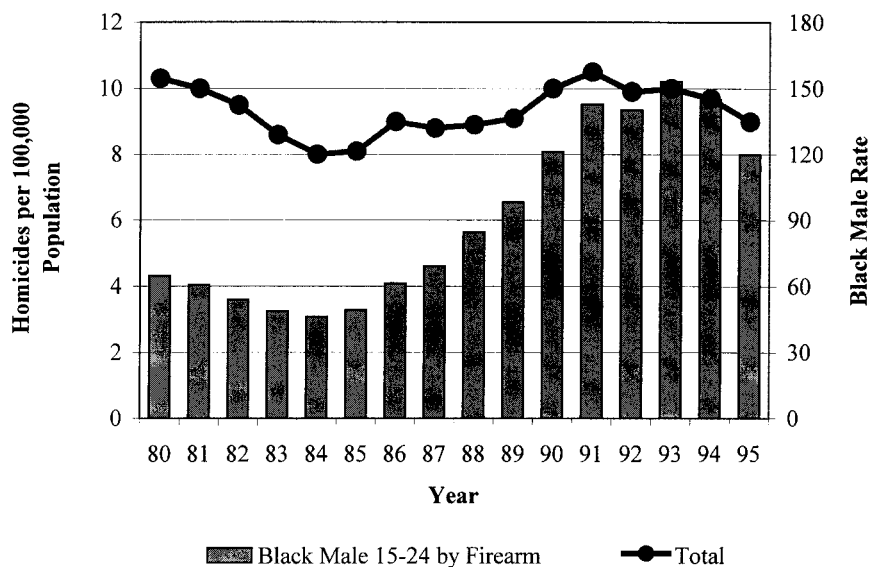
Fig. 1. Annual homicide rate in United States, 1980–1995.

(Fig. 1). Rates for black males ages 15 to 24 more than tripled between 1984 and the most recent peak year in 1993, with similar growth rates evident among victims (Fingerhut, 1993; Fingerhut *et al.*, 1998) and offenders (Blumstein, 1995). Increased reliance on firearms was a distinguishing feature of the rise of homicides among young black males (Blumstein and Cork, 1996; Cork, 1996).

The term "epidemic" is often invoked colloquially to describe this dramatic growth in homicides. However, certain structural features must be satisfied before an observed increase in some social phenomena can be labeled an epidemic in the strict mathematical or biological sense. Simplifying somewhat, an epidemic involves nonmonotonic changes, characterized first by a sudden shift to a period of rapidly accelerating growth, which is followed by a period of slower declines. This distinctive temporal pattern depends fundamentally on the presence of some mechanism for transmitting the "infective" agent among susceptible individuals in the population.[3] There is convincing evidence of nonlinear growth and an increasing concentration of homicides within a subpopulation at both the national and the local levels (nationally in Blumstein and Rosenfeld, 1998; Kellerman, 1996; locally in Block and Block, 1993; Cohen and Tita, 1998; Fagan *et al.*, 1998; Kennedy and Braga, 1998; Klein *et al.*, 1991). The challenge for researchers

[3]For a thorough treatment of the essential structure of an epidemic, see Cliff *et al.* (1981).

lies in identifying the mechanisms of homicide diffusion and understanding the social processes that drive the spread of these events.

This paper addresses both theoretical and methodological issues relating to the diffusion of homicide. Features of the recent homicide epidemic (i.e., it was limited to young black males) suggest that certain social processes—notably, illegal drug markets and gang rivalries—may be important for explaining the pattern and mechanisms of the spread of homicides. We also propose a simple and intuitive method for detecting the presence of spatial diffusion. Epidemics by definition are processes that evolve over time. Using Anselin's (1998) LISA statistics and strategies for their use in exploratory spatial data analysis, we extend the prior cross-sectional method to accommodate the dynamics of changes over time in spatial dependencies.

The paper is organized as follows. We first explore the distinguishing features among various types of diffusion, focusing especially on the mechanisms of diffusion and their application to homicide incidents. The discussion then turns to the features of some homicides that make them candidates for diffusion. The focus is on identifying organizational features of homicides that tie these violent incidents to specific locations. Street-level drug markets and youth gangs are especially implicated. Finally, empirical analyses explore the evidence of spatial diffusion within the same type of homicide and across different types of homicide in a city. While the results are suggestive about the nature of homicide diffusion, the main contribution of the paper is methodological in the form of proposed methods for detecting spatial diffusion processes that can be applied broadly to a wide array of social phenomena.

2. DIFFUSION PROCESSES

Though often used interchangeably (even by geographers), the terms diffusion and contagion refer to different aspects of the process by which a particular phenomenon spreads and recedes. Diffusion refers to the general process of movement, while contagion is one mechanism for achieving that movement. *Contagious diffusion* depends on direct contact and is the classic way of spreading disease. In the medical/disease context, a single person may be responsible for infecting a large number of people over a large geographic area. The contagious spread of violence, particularly homicide, is not likely to involve a single perpetrator who by committing a singular act precipitates many homicidal episodes committed by others. However, when a homicide is instigated by, or furthers, the objectives of a continuing organization, organizational interests may well fuel a series of related homicides. For example, rivalries between gangs or drug markets in the same city may lead to a series of attacks and reprisals among competing enterprises.

Attacks that involve nonparticipants as victims further spread the threat of danger, possibly triggering an increase in violent defensive actions by populations outside the competing organizations. Furthermore, when those organizational interests are tied to specific locations, geographic proximity may be important in that spread.

Two main types of contagious diffusion are possible, *relocation diffusion* and *expansion diffusion*. In *relocation diffusion* the object being diffused leaves the point of origin and spreads outward from that point. A forest fire exemplifies this type of diffusion, as the fire moves along the landscape toward new fuel sources and abandons previously affected areas as the fuel there is exhausted. In the case of crime, *crime displacement* represents one form of relocation diffusion. This form of geographic dispersion might emerge, for example, following effective policing practices that displace homicide incidents from one location to another. Responding to a series of retaliatory strikes between rivals (gangs or drug markets), law enforcement might focus its resources in the area of the most recent homicide. In response, the gang or drug market in the targeted area may move their activities to a nearby location just beyond the concentration of police resources. If repeated over several successive homicide incidents, homicide incidents may appear to be spreading out from an origin.[4]

Like relocation diffusion, *expansion diffusion* also spreads from the center, but the center or place of origin continues to experience high incidence rates of the diffusing phenomenon. A strong attachment to a specific place is inherent to both youth gangs and drug markets (see discussion in Section 3). This increases the likelihood that the center of activity (gang turf or market location) will continue to exhibit high numbers of homicides. Abandoning an area may be only temporary during periods of intensified attention from law enforcement or from rivals. Once that attention from an adversary decreases, gang members and drug dealers will likely move to reclaim a location. Expansion diffusion may also be a factor in a receding epidemic as the benefits of effective treatments are spread to populations in

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[4]A growing body of evidence suggests that crime displacement to nearby areas generally does *not* accompany crime prevention interventions (Eck and Weisburd, 1995, p. 20). Focusing specifically on homicide displacement, recent evaluations of police targeting efforts in two California cities find that gang crimes and homicides linked to street drug markets actually were inhibited rather than simply displaced. Lasley (1998) reports that a Los Angeles Police Department strategy (Operation Cul de Sac) that targeted gang-violence hot spots by erecting steel barriers to impede ingress and egress by gang rivals was successful in eliminating gang crimes without any evident displacement. Another effort by police in Oakland, California targeted aggressive police presence in known drug market locations. This strategy effectively reduced homicides in those locations without displacement elsewhere (Canela-Cacho *et al.*, 1998).

adjoining geographic areas. In the case of crime prevention interventions, recent evaluations have noted a previously unexpected "*diffusion of* [crime prevention] *benefits*" from treated areas to immediately adjacent untreated areas (Clarke and Weisburd, 1994).

The second mechanism for the spread of homicide events is through *hierarchical diffusion*. "This [process] describes transmission through an ordered sequence of classes or places. The process is typified by the diffusion of innovations (such as new styles in women's fashions or new consumer goods, for example, television) from large metropolitan centers to remote villages" (Cliff *et al*., 1981, p. 9). In this instance the spread of a phenomenon does not require direct contact but, rather, occurs through *spontaneous innovation* or *imitation*. Transmission of events is through broad cultural influences that affect the general population or a particular subgroup that may be widely dispersed geographically. We are particularly interested in instances of hierarchical diffusion that occur relatively quickly in time as in the spread of fads or fashions. Within a city, the effects of hierarchical diffusion might be manifested close in time in many physically disjoint neighborhoods.

Spontaneous innovation might accompany the introduction of a new product like crack cocaine that is widely accessible to potential new dealers. Because it is cheap and easy to produce, this product might spawn the simultaneous development of several new and unrelated marketing organizations in different parts of a city. Imitation may also figure prominently in the spread of homicide through mass media popularization of a distinctive lifestyle that is common in one area of the country, e.g., gang-related violence, that is then copied in other cities. There is little evidence that well-established gangs in some parts of the country (e.g., Los Angeles and Chicago) cultivated newly emerging gangs in many smaller U.S. cities (Maxson, 1998). Instead, it appears that violent urban street gangs emerged independently in many cities as local youth adopted a lifestyle they saw portrayed in the media.

As in the case of contagious diffusion, hierarchical diffusion may also be a factor during the receding phase of an epidemic. This is most evident in the case of passing fads that may be widely dispersed initially, but with only weak commitment by adopters, so that they quickly recede again. If highly visible lifestyle changes of new youth gang members are not accompanied by the development of an institutional infrastructure for sustaining the organization, new gangs will not be able to sustain themselves as early members leave. Popularization of newly emerging attitudes that discourage youth involvement in crack markets or gangs likewise may spread broadly throughout a population without requiring direct contact.

3. ORGANIZATIONAL FEATURES OF HOMICIDE AND THEIR IMPACT ON SPATIAL DIFFUSION

Just documenting clustering in the spatial distribution of homicides at some point in time does not inform us about the social and behavioral factors that are driving the actual spread of the phenomenon. Instead, we need to examine the social organization, or organizing principles, of homicide incidents to discover what processes of contagious or hierarchical diffusion facilitate the observed spatial patterns.

The concentration of growth in homicides involving urban youth and firearms suggests that certain organizational and structural features of these homicides—namely, growing youth participation in urban gangs and crack drug markets—may be crucial in the initial spread and subsequent decline of youthful homicides with guns. These youthful enterprises are likely to be especially relevant to diffusion because they involve organizations that are sustained over time through continuing social interactions, both among participants and between participants and outsiders. The particular nature of these social interactions, with their heavy reliance on violence to maintain order, is also important, as is the explicit role that spatial locations play in each.[5] The following discussion explores some of the ways that distinctive features of crack markets and of recently emerging urban youth gangs may facilitate both epidemic growth and spatial diffusion of homicides. The purpose is to provide a plausible account of how various presumed features of these enterprises might facilitate the spread of some types of homicide.

3.1. Crack Markets

The timing of the 1985 onset of crack cocaine markets in the United States, the heavy involvement of black youth and juveniles in these markets and the proliferation of guns within these markets have fueled widespread speculation that these markets are implicated in the escalation of youth gun violence, especially homicides. Crack is a relatively low-cost product that can be marketed in small inexpensive quantities. The low cost of the product greatly reduces barriers to entry facing individuals who want to become sellers, and there is no shortage of young and inexperienced dealers on the streets. The addictive quality of crack, compounded by its very short-lived high, produces a market that is driven and sustained by a relentless demand among users. Repeated transactions increase the likelihood of confrontation between buyers (in an agitated state of craving) and dealers (nervous to allude detection by the police and falling prey to robbery). Furthermore, because these transactions are illicit, violence is an important means of

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[5]Cohen *et al*. (1998) provide an earlier treatment of the spread of homicides within cities.

regulating and maintaining control within these markets and protecting participants against robberies and other attacks.

The prevailing wisdom is that participants in crack markets quickly succumbed to the need to carry and use guns as tools of the trade. As hypothesized by Blumstein (1995) and empirically supported by Blumstein and Cork (1996), arming participants in crack markets increases the risk of violence for nonparticipants as well. Faced with increased risks to personal safety, youth outside crack markets increasingly carry guns and use them to settle interpersonal disputes, thereby spreading gun violence more broadly among the youth population. While spreading gun homicides to new segments of the population, crack markets are less likely to serve as a vehicle for the geographical spread of homicides to new locations. As an illicit market, continued transactions often depend on maintaining stable market locations so that customers can easily locate sellers. There are strong incentives for locational persistence.

3.2. Violent Youth Gangs

Identity with a place is a strong motivating factor for gangs. Research has repeatedly demonstrated the importance that gang members attach to the specific areas in which they hang out (e.g., Klein, 1995; Moore, 1991; Tita *et al*., 1998). Klein (1995) believes that the most important role of gang "turf" is as a tangible symbol of gang identity. Gang dress codes and hand signals may serve a similar function. Violence is presumed to be another integral part of daily gang life. Used internally, it might serve as a means for maintaining control over and assuring loyalty by gang members. Used against outsiders, it may serve as a means of inflicting harm on rivals and garnering cooperation, or even submission from the surrounding host population. The likely crucial role of violence for gangs is signaled by the heavy reliance on violence in many gang rituals. This is especially evident in the symbolically important transitions into and out of a gang membership.

Gang locales tend to be centers of persistently high crime rates (Spergel and Curry, 1990). This is especially so for violent crimes. By mapping gang territories in Boston, Kennedy *et al*. (1997) find a strong spatial association between gang location and violent, gun-related crime. Block (1998) finds a similar relationship of gang locations with drug crimes and assaults. Using data from Pittsburgh in Pennsylvania, Tita (1999a) demonstrates that youth gangs are more likely to form in areas that already have high crime rates and that those high crime rates continue unabated after gangs emerge in an area. Rates of some crimes specifically linked to gang activities, like shots fired, actually increase after gangs form. The same areal concentration and increasing rates characterize gang-related homicides. Most gang-related

homicides in Pittsburgh occur within a short distance from gang locations (75% in the same census block group and 90% in the same census tract as gang "set space"—the term used to describe the primary daily activity space of gang members). Persistence of violent gang rivalries, which transcend the participation of particular gang members, is likely to be an important factor in sustaining high levels of violence. Persistence of particular locales for gang activity would further ensure that gang violence remains spatially concentrated.

Other features of violent youth gangs may help to diffuse violence spatially, including homicides. Media portrayals of gang violence as glamorous and sometimes even heroic may increase the status of gangs among urban youth. Widespread cultural disaffection from mainstream aspirations and icons is likely to provide fertile ground for the spread of a "gangster" lifestyle in economically and socially devastated inner-city neighborhoods. Wider acceptance of a gang culture among nonmembers would facilitate the further spread of lethal violence as an increasingly acceptable response to any form of personal insult or challenge. Through these general cultural routes, violence once limited to hard-core gang members can spread into the lives of nongang youth, potentially affecting both those in daily contact with gangs and those in areas more remote from gangs. Gangs may also be directly involved in the spread of violence to nongang areas when they commit violent acts against ousiders who venture into a gang's activity space or when gang members engage in violent forays to outside areas.

A proliferation of guns among gang members adds a particularly lethal dimension to gang activities. In the presence of guns, threats and attacks among gang members and by outsiders are potentially far more dangerous, to both gang members and innocent bystanders. The increasing danger can fuel an expanding local arms race in personal protection weapons, first among rival gang members and then increasingly among nongang members. An increasingly armed youth population, set within the context of cultural changes that increasingly devalue human life, is a particularly lethal combination. Even relatively benign disputes that previously might be resolved in a brawl may become occasions for potentially lethal violence.

3.3. Self-Limiting Features of Crack Markets and Violent Youth Gangs

In addition to features that facilitate the growth and spread of homicides, crack markets and youth gangs also involve self-limiting features that may stem the tide of an epidemic. Drug dealers realize that too much violence is bad for business, because it attracts increased police attention and encourages customers to find other less violent outlets. Technological changes also affected the operation of crack markets. Notably, new communications technology in the form of beepers, pagers, and cellular phones,

and new ways of marketing these communications services through large one-time cash purchases, substantially reduced the importance of stable market locations. Rather than hawking their wares in known—and possibly increasingly dangerous—street locations, dealers could be anywhere waiting for a customer's call. The actual location of the transaction became more flexible, and could be chosen to maximize dealer and customer safety.

The types of places where crack markets and youth gangs locate may also help to limit the spread of these types of violence. Continuing patterns of racial residential segregation, compounded by further segregation within minority communities along social and economic status dimensions, effectively isolate disadvantaged urban minority communities spatially and socially from contact with others. This physical and social isolation also may serve to limit the diffusion of homicides across space, and more importantly across population subgroups. To the extent that virtually all aspects of the daily social life of a population subgroup are limited to other members of the same group, there are few occasions for spreading violent encounters to other groups. This may help to explain why minority youth, especially young black males were so susceptible during the most recent homicide epidemic. Crack and violent youth gangs first emerged and flourished in poor minority communities. Sharp differences in social and economic structure—notably in the capacity for exercising informal social control—between afflicted communities and their neighbors likely served effectively to repel the encroachment of these violent enterprises into neighboring areas.[6]

4. DATA

The analysis uses microdata on 287 homicides in the city of Pittsburgh during the period 1991 to 1995.[7] The data were culled from individual case files of all homicides in the city (excluding a handful of cases involving police officers). Attributes of both offenders and victims and a variety of circumstances of the incident were collected, including weapon type and gang and drug involvement in the incident. Locations were coded for the homicide incident and participants' residences.

The two dimensions that are potentially of greatest relevance to spatial diffusion are homicides associated with crack drug markets and/or with youth gangs. Fortunately, these two features arrived in the city at different

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[6]Tita *et al.* (1998) empirically examine differences in the economic and social structure of neighborhoods with and without violent youth gangs and find significant effects for factors often associated with social control. Also see Curry and Spergel (1988).

[7]The authors thank the Pittsburgh Bureau of Police, especially detectives in the Investigations Bureau, for providing access to the data and assistance during the collection phase.

times. Crack cocaine arrived in Pittsburgh during the latter half of 1989. It was accompanied by substantial increases in drug arrests (up more than 100% from 1988 to 1989 and an additional 61% from 1989 to 1990), especially arrests of younger offenders through age 20 (up 4.7-fold from 1988 to 1989 and then almost 2-fold from 1989 to 1990). Local gang members reported in interviews that recent gang "sets" in Pittsburgh began emerging during the latter half of 1991 and continued forming through 1992 (Tita, 1999b; Tita et al., 1998). This start-up period was confirmed in parallel interviews with youth service workers and police officers working in the affected neighborhoods, as well as by sharp increases in the volume of citizen initiated 911 calls reporting shots fired (a common gang activity that was up 78% between 1991 and 1992).

Any indication of drug involvement in a case file was sufficient to classify a homicide as drug-related. These homicides overwhelmingly involved drug trafficking activities (63%) and rarely involved drug use by participants (under 1%). A homicide was classified as gang-involved if the homicide involved some gang motivation (e.g., intergang disputes, initiation activities, or spontaneous drive-by killings) or any participant was a gang member. The special subclass of "member-only" homicides involved at least one gang member, but no gang motivation.

Throughout most of the study period, gang-motivated homicides were about two-thirds and member-only homicides one-third of all gang-involved homicides in the city. The one exception was the end of the study period in 1995 when member-only homicides—usually involving domestic disputes or robberies—increased to become 68% of all gang-involved homicides. This transformation in the nature of gang-involved homicides may be symptomatic of a decline in active gangs in the city. Intergang conflicts were the bulk of all gang-involved homicides (68%). There was very little intersection between gang-involved and drug-involved homicides, with only 21% of gang-involved homicides also being drug related.

On one hand, Pittsburgh is well suited for an analysis of the dynamics of a homicide epidemic because it experienced substantial changes in both the volume and character of homicide during the study period. During the decade of the 1980s the total homicide rate was relatively stable, at about 10 per 100,000 population (Fig. 2). During the early 1990s there is an unusual departure from these characteristically low rates, and the homicide rate nearly doubled in a single year increase during 1993. The rate then declined slowly through 1997, but remained above the previous low rates of the 1980s. On the other hand, however, the total number of homicides in any year is relatively small, peaking at 82 homicides in 1993. The numbers involved become smaller still as total homicides are disaggregated by type of homicide and by geographic areas within the city. We will return to the problem of small numbers in the discussion of results, but note here that
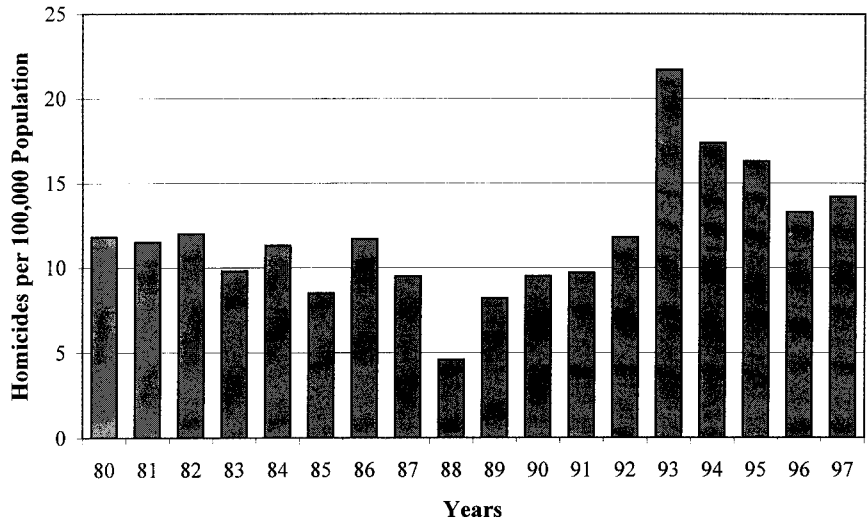
Fig. 2. Annual homicide rate in Pittsburgh, 1980–1997.

the problem is obviated somewhat by concentration of homicides in selected types and in relatively few geographic areas of the city.

Figure 3 displays the year-to-year changes in the total number of homicides. There is a modest increase in total homicides following the arrival of
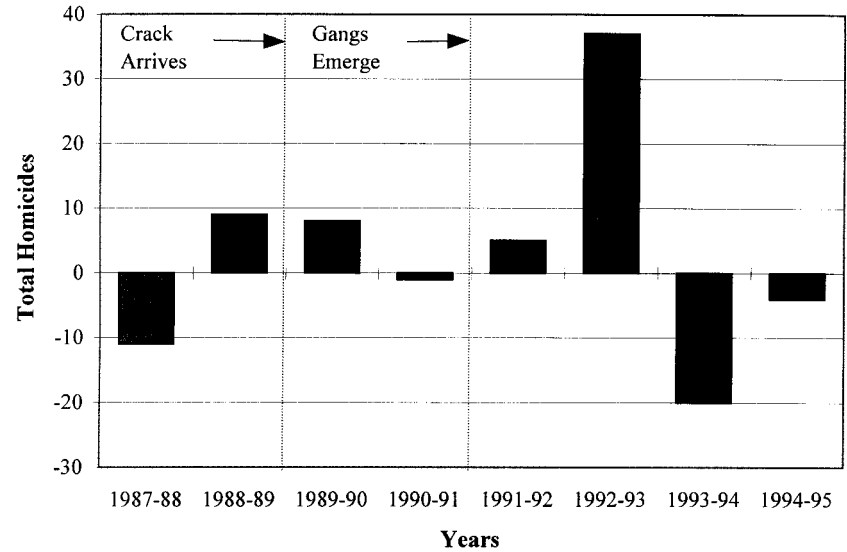

Fig. 3. Yearly change in Pittsburgh homicides, 1987–1995.

crack markets in the city (+8, or up 24% between 1989 and 1990). This increase is dwarfed by the near-100% increase of 37 additional homicides in 1993, 2 years after the emergence of youth gangs. The sharp increase in 1993 does not reflect a general increase that is distributed uniformly across all types of participants or circumstances. Rather the change is concentrated in only one segment of the population (with homicides involving youthful participants under age 25 more than tripling between 1991 and 1993) and in selected types of incidents (homicides by gun grew almost fourfold from 1990 to 1993). The data covering the period of the early 1990s coincides with the arrival of crack markets in Pittsburgh at the end of 1989 and the emergence of violent youth gangs beginning in late 1991. It will be evident in the next section that these two factors had quite different effects on homicides in the city.

We are interested mainly in spatial features of the location of homicides within a city, especially the role of contagious diffusion processes that involve the spread of homicide spatially to neighboring areas. The analysis in this paper focuses narrowly on neighbors defined by physical or geographical proximity between census tracts.[8] Tracts are small enough to distinguish meaningfully between areas with and without gangs or crack markets (Tita, 1999b) and to observe diffusion across space. As the units of analysis increase in size and include more diverse areas within the same areal unit, it becomes more difficult to observe the spread of homicides *between units*, as opposed to an undetectable redistribution among locations within the same spatial unit. Changes over time are measured annually. Each homicide incident can be located in time and space by year and census tract of occurrence, respectively.[9]

4.1. Emphasis on Diffusion Associated with Youth-Gang Homicides

Drug-related homicides do not appear to be a factor in the changes in homicides in Pittsburgh (Fig. 4a). Despite the anticipated structural changes that might accompany the arrival of crack markets in Pittsburgh late in 1989, the number of drug-related homicides changed hardly at all over the entire period 1987 to 1995. This may be a reflection of the fluid and open

[8]A broader view might include "neighbors" defined more generally in terms of shared social attributes that form a basis for the transmission of attitudes and behaviors. Such extensions are beyond the scope of the present paper but may benefit from the conceptual and methodological developments that we present here in the more limited context of diffusion through physical proximity.

[9]The analysis relies on the location of the incident rather than participants' residences. This more reliably reflects the influence of the proximate activities that may give rise to the homicide incidents.
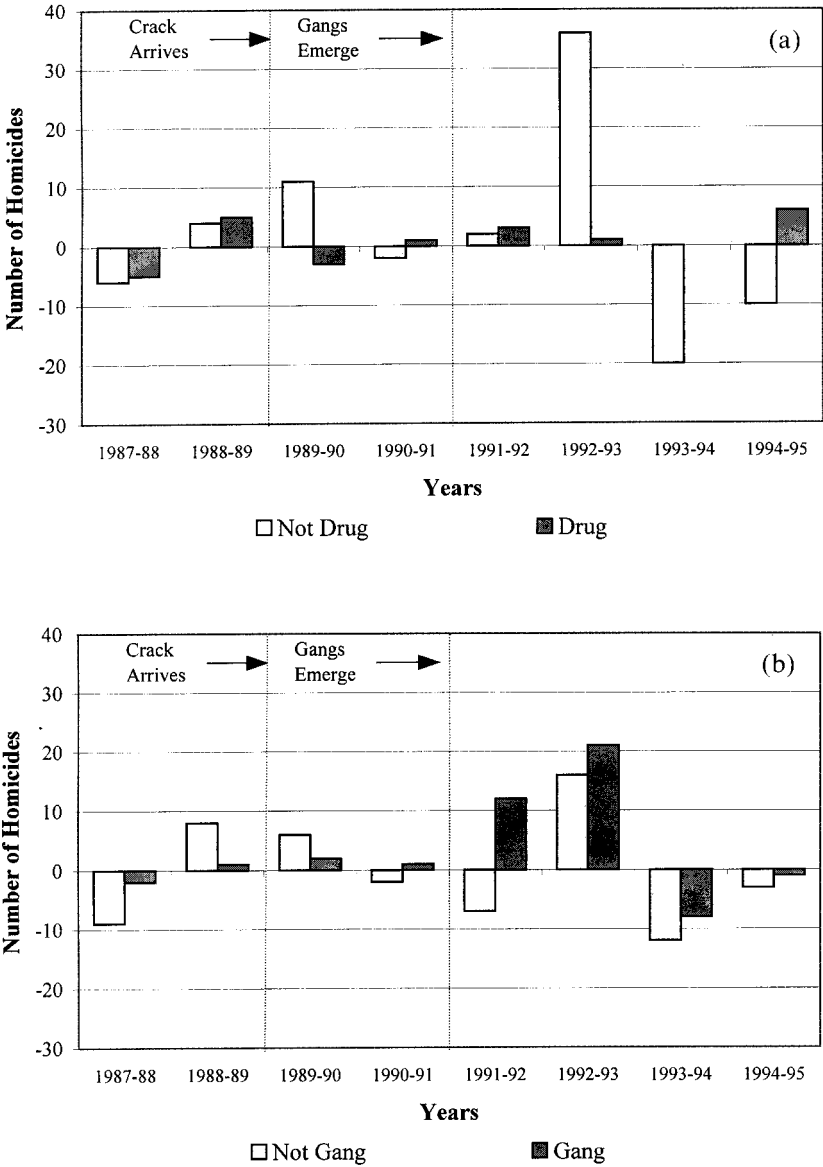
Fig. 4. Role of drugs and gangs in yearly changes in Pittsburgh homicide. (a) Drug involved (using or dealing) or not. (b) Gang involved (members or motives) or not.

character of illicit drug markets in Pittsburgh, where the same dealers could be observed dealing in more than one venue throughout the city. Crack's arrival in Pittsburgh also occurred several years after crack markets began to flourish in large cities on the East and West coasts. This was closer in time to the arrival of beeper and cell phone technologies that freed many drug dealers from vending their product in specific market locations. This reduced face-to-face conflicts between competing dealers and, possibly, reduced the occasions that might result in drug-related homicides.

The emergence of youth gangs beginning in late 1991 was a more compelling factor in the citywide changes in homicides (Fig. 4b). The increase of 12 additional gang-involved homicides during 1992—a 30% increase from 40 total homicides in 1991—was previously not evident in total homicides because of an offsetting decline in nongang homicides that year. The larger increase of 21 additional gang-involved homicides in 1993 comprised almost 60% of the total growth that year. The homicidal violence that started in gang-involved incidents apparently spread quickly from gang youth to nongang youth, with nongang homicides accounting for 38% of the growth during the peak year of homicides in 1993.[10] Hence, the analysis that follows will focus on gang-involved homicides looking for evidence that these homicides not only escalated in numbers, but also diffused spatially among neighboring census tracts.

Since youth gangs were virtually nonexistent in the city before 1992, we restrict the analysis to the years 1991 to 1995. This allows for the initial appearance of youth-gang homicides between 1991 and 1992 and then their spread to neighboring areas. As we examine the spatial dimensions of changes in homicide in Pittsburgh, we focus on three distinct types of homicide during the period 1991 to 1995. *Youth-gang* homicides ($n = 112$) involve youthful (ages 12 to 24) participants as offenders or victims, and gang members or gang motives. *Youth-nongang* homicides ($n = 104$) also involve youthful participants, but there is no gang involvement in the incident. Finally, *nonyouth* homicides ($n = 67$) include all the remaining incidents with no youthful participants.[11]

Spatial diffusion of *youth-gang* homicides might follow two distinct paths. Because of the importance of gang turf, both symbolically and practically for gang members and their activities, we might expect the greatest spatial dependencies among neighboring tracts to be evident in *youth-gang* homicides. This pattern would be consistent with *contagious* diffusion through *expansion* from original-source innovator gangs out to imitator

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[10]Only 1 of 37 additional homicides between 1992 and 1993 involved no youthful participants.
[11]There is little overlap between gang-related and drug-related homicides in the city. Homicides with both elements are 6% of all homicides. This subset of homicides represents 20.8% of gang-related homicides and 26.9% of drug-related homicides.

gangs that form in reaction to gangs in neighboring tracts and to the threat posed by gang violence in nearby locations. Alternatively, the emergence of youth gangs in Pittsburgh might have been influenced by broad cultural influences leading to *hierarchical* diffusion, with distinct youth gangs emerging simultaneously in many different locations in the city.

Youth-nongang homicides existed before youth gangs emerged in the city. We expect, however, that the emergence of youth gangs will have an effect on the spatial distribution of homicides by nongang youth. Growth in *youth-nongang* homicides is expected initially to follow *youth-gang* homicides in the same neighborhoods, as local nongang youth react to rising gang violence by arming themselves as a means of providing personal protection. To the extent that nongang youth are not as rigidly tied to specific locations, they are expected to spread *youth-nongang* homicides more widely to areas outside gang turf. *Nonyouth* homicides will include many of the more traditional forms of homicide emerging from interpersonal conflicts between intimates, friends, and acquaintances. These homicides are expected to exhibit little spatial clustering during any time period, and little or no spatial diffusion to neighboring areas over time.

Aside from geographic proximity, the rate of diffusion of homicides will also depend on the size of the population at risk in neighboring tracts. In the extreme, an area with little or no population is unlikely to be the location of homicides—except perhaps premeditated execution-style killings. Likewise,the larger the population at risk in an area, the greater the likelihood that activities that involve face-to-face contacts will diffuse into that area. To isolate the effects of spatial proximity, the analysis relies on population-based homicide rates to control for differences in the size of the population at risk across census tracts.

The appropriate population at risk varies among the three types of homicide. *Youth-gang* homicides are scaled relative to the size of the black youth population in a tract. Just 1 of 112 youth-gang homicides in the 1991–1995 period involved only white youths as participants. All the remaining youth-gang homicides involved black youths among participants, including all of the offenders and 96% of the victims. This contrasts with *youth-nongang* homicides, where 19% of 104 homicides involved no black youths among participants. Thus, youth-nongang homicides are scaled relative to the total youth population in a tract. Finally, *nonyouth* homicides involve both black and white participants, with white offenders in 24% of 67 incidents. Nonyouth homicide counts are scaled relative to the size of the total adult population (ages 25 to 61).[12]

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[12]Population counts disaggregated by age, race, and census tract are available only from the decennial census. Rates in each year (1991 to 1995) use the relevant population counts from the 1990 census.
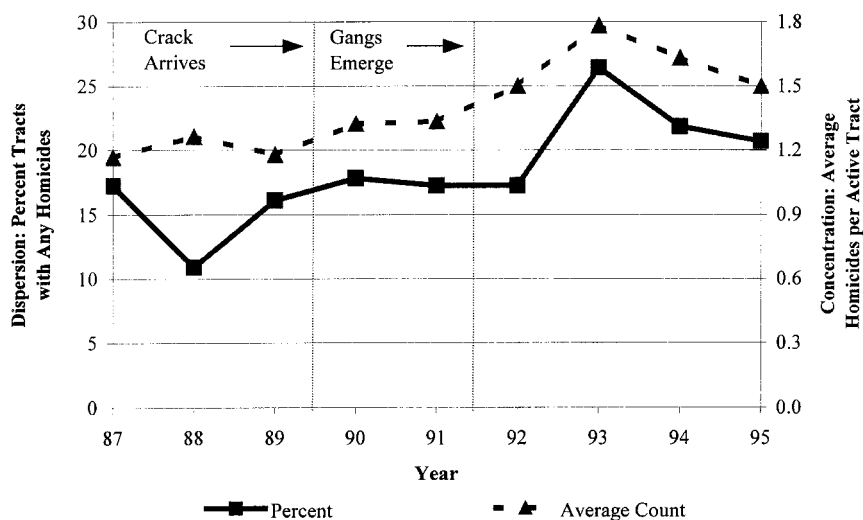
Fig. 5. Concentration and dispersion of homicide in Pittsburgh.

5. SPATIAL FEATURES OF HOMICIDE

We know from citywide data that the levels of homicide changed dramatically during the study period. There are also indications that these changes in citywide totals involved both increasing concentration of homicides in already affected locations and the dispersion of homicides to new locations. Figure 5 partitions overall changes in the level of homicides between citywide indicators of *concentration* and *dispersion*.

Concentration (dashed line in Fig. 5) is the average number of homicides experienced in those tracts that have any during a year. From 1989 to 1993, the annual number of homicides in affected tracts increased by 50% from just under 1.2 homicides per tract to just under 1.8. This average then declined, as did the total number of homicides after the 1993 peak. *Dispersion* (solid line in Fig. 5) is measured by the percentage of tracts that have any homicides. In 1988 the unusually low total number of homicides[13] was due entirely to a reduction in active tracts. Those tracts that remained active that year continued to average about 1.2 homicides each.

The percentage of active tracts remained fairly stable between 1989 and 1992, at about 17%, while the *concentration* increased 27%, from 1.18 to

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[13]There were only 24 homicides citywide in 1988, compared to an average of 37 annually in the surrounding years from 1987 to 1991.

1.50 homicides per active tract. The sharp increase in total homicides from 1992 to 1993 was due primarily to dispersion of homicides to an increasing number of tracts that year, as active tracts rose 1.53-fold, from 17 to 26% between 1992 and 1993. The rise in *concentration* during the same year was only 1.19-fold, from 1.500 to 1.782 homicides per tract. Following the peak year in 1993, both *dispersion* and *concentration* declined, with dispersion decreasing slightly faster than concentration.

The rise and fall of homicides in Pittsburgh were associated primarily with changes in the geographic dispersion of homicides. But how were the affected tracts distributed spatially in the city? Was the growth restricted primarily within clusters of neighboring tracts or distributed more broadly across the city? To answere these questions, we look first to available measures of spatial association.

Global measures of spatial dependence look for spatial associations in the distribution of some phenomenon. Positive associations exist when neighboring locations share similar levels of a variable, for example, clusters of high (or low) values in geographically proximate locations. The association is negative when neighboring locations are dissimilar, with high-level locations adjacent to low-level neighbors.[14] One of the most commonly used measures of global spatial association is Moran's *I*, a statistic that measures the extent of similarity or dissimilarity in a variable across neighboring spatial units. For the combined years from 1992 to 1995, the global Moran's $I = 0.149$ ($p \leq 0.05$ in two-tailed test) for annual homicide rates in each tract. While low in magnitude, this citywide measure is statistically significant.[15]

In the context of city homicides, the global Moran's *I* statistic is most useful for detecting a single common *citywide* pattern of spatial dependency. As a global indicator, it measures the presence of a homogeneous pattern of spatial association across the entire study area. Global measures are poorly suited when there is heterogeneity in spatial dependencies, for example, a case with varying levels of association across different regions of the study area. Such nonstationarity in the association across space might be manifested in the extreme by local "hot spots," or even positive associations in some regions and negative associations in others. Local indicators of spatial association (*LISA*) statistics are more appropriate for detecting such spatial instabilities (Anselin, 1993, 1995a).

[14]Like temporal dependencies in time series data, spatial dependencies in cross-sectional data introduce a number of concerns about spatial autocorrelation in multivariate statistical analyses. These concerns are not a focus in this paper, but interested readers may consult any of several texts on spatial statistical analysis for discussion of this topic (Anselin, 1988; Haining, 1990; Cressie, 1993).

[15]Significance is gauged relative to a near-zero expected value obtained from $-1/(n-1) = -0.006$. The Moran's *I* statistics are lower in value, with no clear patterns of significance for individual years and for distinct types of homicide related to youth and gangs.
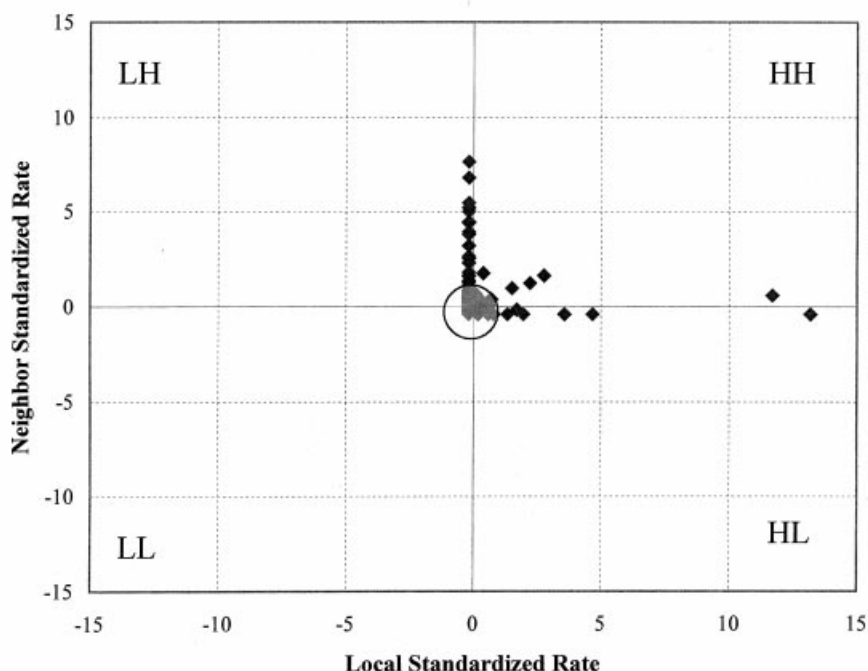
Fig. 6. Scatterplot of local and neighbor standardized rates for youth-gang homicides in 1993.

5.1. Local Indicators of Spatial Association

LISA statistics are especially valuable in exploratory spatial data analyses looking for patterns across space in spatial dependencies. The values of a variable for local and neighbor pairs provide the most basic representation of local spatial associations. Each pair (L, N) consists of the standardized level of a variable in the local (L) spatial unit and the standardized value of the same variable in neighboring (N) spatial units—both standardized relative to their respective mean and standard deviation across the spatial units.[16] Each element of the pair is either low (L) or high (H) relative to the distributions of the respective local and neighbor values across all observations. The standardized values locate each local-neighbor pair within a two-dimensional euclidian space (Fig. 6). Pairs in which both the local and

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[16]The "neighbor" value, also known as the spatially lagged value for each local spatial unit, is obtained from a spatially weighted average value across all "neighboring" units. Neighbors are typically defined by their physical proximity to the local unit. For example, first-order neighbors are adjacent to the local unit and usually share a common border. Alternatively, the spatial weights might reflect physical or "social" distance between the local unit and its "neighbors." Throughout this paper, neighbor values are the simple average value of a variable in all physically adjacent units (obtained using the row-standardized version of a simple 0/1 contiguity matrix).

the neighbor values are above their respective means fall in the upper-right HH quadrant. When both elements of the pair are below their means, the pair falls in the lower-left LL quadrant. When the relative values of local and neighbor units differ, the pair falls in either the HL or the LH quadrant.
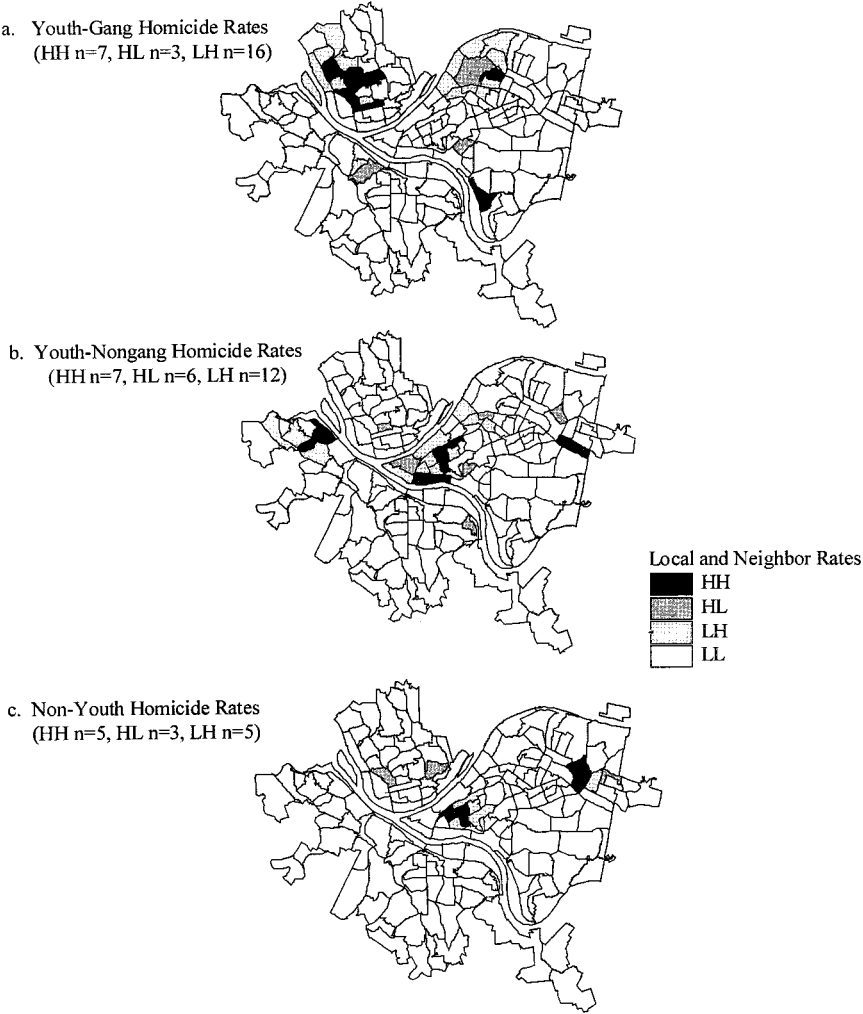
By standardizing the local and neighbor homicide rates relative to their respective means and standard deviations, a simple scatterplot of local-neighbor pairs like that in Fig. 6 provides a basis for judging the relative strength of spatial relationships. While the appropriate distributional assumption for the spatial units is unknown, we invoke a simple "two-sigma" rule to approximate the tails of the distribution. In a variation from the approach suggested by Anselin (1995b), "significant" tracts fall outside the circle defined by an origin at the local and neighbor means and radius that is 2 standard units in length.[17] Extreme outliers in the HH quadrant may be symptomatic of "hot spots," while those in the LL quandrant may be relatively "immune" to the phenomenon under study.

Figure 6 plots local-neighbor pairs for youth-gang homicide rates in 1993, the peak year of homicides. There is a total of 10 tracts in the HH and HL quadrants that experienced high local youth-gang homicide rates in 1993 relative to the overall distribution of local and neighbor pairs. Another 16 tracts in the LH quadrant have local values near the mean in 1993 but are surrounded by relatively high-rate neighbors. None of the local-neighbor pairs in the LL quadrant ever exceed the two-sigma distance filter. Figure 7 displays the 1993 outlier tracts on a map. Two distinct clusters of outlier *youth-gang* homicide rates are evident in Fig. 7a, one large cluster of five adjoining high-rate tracts in the northwestern part of the city and another smaller cluster of two high-rate tracts toward the northeast of city. During the same year there are three distinct *youth-nongang* clusters (Fig. 7b) and two *nonyouth* homicide clusters (Fig. 7c). These high rates of nongang and nonyouth homicides occur in the same areas in the center and northeast of the city. The areas affected by high *youth-nongang* and *non-youth* clusters during 1993 do not overlap the *youth-gang* clusters in the same year.[18]

Table I reports the prevalence of different types of local-neighbor pairs and of outlier tracts over the 5 years of data. The top panel in the table

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[17]Anselin (1995b) defines outliers as local-neighbor pairs that fall more than two standard units away from the origin on *either* dimension (i.e., outside the square whose corners are two standard units away from the origin for *both* the local and the neighbor axes).

[18]The extent of overlap in significant local-neighbor pairs is generally small across different types of homicides. Combining annual results from 1991 to 1995, significant local-neighbor rates for *youth-gang* homicides occur in only 11.6% of tracts with significant local-neighbor rates for *youth-nongang* homicides,and 1.7% of tracts with significant *nonyouth* rates in the same year. There is slightly more overlap between *youth-nongang* and *nonyouth* homicide rates, with significant local-neighbor pairs for *youth-nongang* homicide rates occurring in 23.7% of tracts with significant local-neighbor rates for *nonyouth* homicides in the same year.

a. Youth-Gang Homicide Rates
    (HH n=7, HL n=3, LH n=16)

b. Youth-Nongang Homicide Rates
    (HH n=7, HL n=6, LH n=12)

Local and Neighbor Rates
- HH
- HL
- LH
- LL

c. Non-Youth Homicide Rates
    (HH n=5, HL n=3, LH n=5)

Fig. 7. "Significant" local and neighbor homicide rates by tract in Pittsburgh during 1993. "Significant" local-neighbor pairs of homicide rates are outliers whose rates are more than 2 standard units away from the origin formed by the means of local and neighbor rates over all tracts and years.

reports total tract-years falling in each quadrant, and the middle panel identifies the number of outlier tract-years that are designated as "significant." Finally, the bottom panel in the table reports the relative share of outlier tracts in each quadrant. HH and HL tracts each occur in 5% or less

Table I. Annual Local-Neighbor Pairs of Homicide Rates for Tracts in Pittsburgh, 1991–1995 Combined

	Homicide rate in local-neighbor pairs[a]			
	HH	HL	LH	LL
Count of all tracts (percentage of total)				
Youth-gang	36 (4.1%)	47 (5.4%)	157 (18.0%)	630 (72.4%)
Youth-nongang	31 (3.6%)	50 (5.7%)	238 (27.4%)	551 (63.3%)
Nonyouth	19 (2.2%)	37 (4.2%)	209 (24.0%)	605 (69.5%)
Count of "significant"tracts (percentage of total)[b]				
Youth-gang	17 (2.0%)	7 (0.8%)	32 (3.7%)	0 (0%)
Youth-nongang	23 (2.6%)	17 (2.0%)	29 (3.3%)	0 (0%)
Nonyouth	14 (1.6%)	16 (1.8%)	29 (3.3%)	0 (0%)
Percentage "significant" in all-tract total				
Youth-gang	47.2	14.9	20.4	0
Youth-nongang	74.2	34.0	12.2	0
Nonyouth	73.7	43.2	13.9	0

[a] Each *local-neighbor* pair consists of the annual homicide rate locally in a tract and the weighted-average homicide rate in neighboring tracts (i.e., the spatially lagged homicide rate for each tract). Both rates are standardized relative to their respective distributions across all tracts and all years (174 tracts × 5 years = 870 observations). HH tracts have high homicide rates locally and border on neighboring tracts that also have high average homicide rates. HL tracts have high local homicide rates and are surrounded by neighboring tracts with low average homicide rates. In LH tracts, the local homicide rate is low, while the average rate in neighboring tracts is high. Both the local rate and the average rate in neighboring tracts are low in LL tracts.

[b] A tract is counted as "significant" if the euclidian distance from the pair defined by the local and neighbor standardized homicide rates is more than 2 standard units away from the origin (i.e., away from the local and neighbor means across all tracts and years). "Significant" tracts are outliers with the highest or lowest rates found in the distribution of all tracts and years.

of all tracts during the 5 years. Outliers are common among the HH tracts, especially for *youth-nongang* and *nonyouth* homicides. Thus, HH tracts are extreme in two ways in these data. Not only do they fall within clusters of adjoining high-rate tracts, the rates in these tracts are also more likely to be outliers relative to the distribution of rates across all tracts and years.

The above illustrates the use of exploratory spatial data analysis to identify localized clusters of adjoining high-rate tracts. Such analyses, however, provide static views of the spatial distribution of homicides at just one point in time. They do not address the dynamics of changes in that distribution over time. To partially address this limitation, Exhibits 1 to 3 visually display animated sequences of outlier clusters in successive years for each homicide type.[19] While there are indications of some potential diffusion

[19] The animations are available on the web at http://hdm.heinz.cmu.edu/jackie.

Table II. Dynamics of Change in Spatial Distribution of Homicide Rates Over Successive Observations

Direction of change	Type of diffusion	Mechanism of change	Year-to-year change in *local-neighbor* pairs	
			Local is diffusion outcome	Local is diffusion source
From low to high levels	Contagious	Expansion among Neighbors	LH to HH	HL to HH
		Relocation among neighbors	LH to HL	HL to LH
	Hierarchical	Isolated increase	LL to HL	LL to LH
		Global increase	LL to HH	LL to HH
From high to low levels	Contagious	Expansion among neighbors	HL to LL	LH to LL
		Relocation among neighbors	HL to LH	LH to HL
	Hierarchical	Isolated decrease	HH to LH	HH to HL
		Global decrease	HH to LL	HH to LL
No change	None	Stationary		

among neighboring census tracts, the visual displays alone do not help in discerning what types of processes might underlie the evident movement among locations. Is it just random relocations, or is some more systematic process of diffusion at work? These questions arise naturally from viewing the animations, but they are difficult to answer by relying solely on visual displays.

6. DYNAMICS OF CHANGE IN SPATIAL RELATIONSHIPS

Local-neighbor pairs are a simple but powerful tool for identifying spatial clusters. We introduce here a simple method for extending the analysis of these pairs to address dynamic features of changes over time in the spatial patterns. The method focuses on changes in the levels of local-neighbor pairs and looks for evidence of diffusion that involves the spread of high (or low) rates to increasing numbers of spatial units. This spread may be spatially dependent among adjoining spatial units or more general throughout nonadjoining units. We apply the method to look for evidence of the diffusion of homicides among census tracts within one city. The method has the potential for much wider application as a means of characterizing changes over time in spatial units.

6.1. Method for Calibrating the Extent of Diffusion

Table II identifies the full array of possible combinations of local-neighbor pairs over successive time periods. The various combinations of pairs in successive time periods are each compatible with a different type of

diffusion. The types of change are distinguished in terms of the direction and mechanism of change and whether the local spatial unit is the outcome or source of diffusion in local-neighbor pairs.

The direction of diffusion may be monotonic or nonmonotonic over time. An epidemic is a particular form of nonmonotonic diffusion, with an "infectious" period of rapid spread of high levels, followed by a period of slower proliferation of low levels as the epidemic recedes. We would expect to see changes in both directions in annual tract-level homicide rates.

Four mechanisms lead to changes from low to high levels in spatial units. *Expansion* and *relocation* are forms of *contagious diffusion* in which the status among neighboring spatial units affects the future status of adjoining units, either by increasing the level locally or in the neighbors located in the same local-neighbor pair. Spatial units can also change from low to high levels through *hierarchical diffusion* in the form of *isolated* or *global increases*. Both types of hierarchical diffusion reflect increases that do not depend on contact with nearby high-level spatial units. Instead, individual or adjoining spatial units "heave" upward without any neighbor effects, possibly in response to broad cultural influences.

Four parallel mechanisms lead to changes from high to low levels in spatial units. *Expansion* and *relocation* operate through contagious effects to spread declining levels among adjoining spatial units, while *isolated* and *global decreases* reflect decreases experienced broadly by nonadjoining spatial units. Finally, the spatial pattern of local-neighbour pairs may remain stationary over successive observations with no evident changes in levels from prevailing high or low levels among spatial units.

Large changes can be distinguished from small changes using the euclidian distance, D, between successive local-neighbor pairs, (L_t, N_t) and (L_{t+1}, N_{t+1}):

$$D = \sqrt{(L_{t+1} - L_t)^2 + (N_{t+1} - N_t)^2}$$

where L_t is the standardized value at time t of a local spatial unit, and N_t is the standardized value at time t of the spatially lagged weighted average value in neighboring spatial units. Without making any strong assumptions about the appropriate distributional form for this distance measure, we invoke a simple 2σ rule as an approximation to identify unusually large changes in either an increasing or decreasing direction. Changes over time that involve a move of at least two standard units in the value of a local-neighbor LISA pair are designated "significant."

A more detailed description of the method for assessing the nature of changes in local-neighbor pairs is given in Appendix A.[20] The basic strategy

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[20]Appendices A and B are available from the authors or from the web at http:// hdm.heinz.cmu.edu/jackie.

is to compare the relative prevalence of transitions among the different local-neighbor LISA pairs in successive time periods. The question is whether transitions that are compatible with diffusion, especially diffusion of homicides among neighboring tracts, are more likely than expected based on the prevalence of other transitions to the same local-neighbor outcome. The spread of high youth-gang homicide rates among neighboring tracts is of special interest. An excess of significant transitions from LH youth-gang rates at time t to HL or HH rates at time $t + 1$ is compatible with contagious diffusion of high homicide rates. In this case, the comparison transition rate is based on the prevalence of other nonstationary, significant transitions to HL and HH outcomes at time $t + 1$. A simple t test of the difference between proportions evaluates whether the diffusion transition occurs more often than might be expected based on the prevalence of other transitions to the same outcome.

6.2. Dynamics of Change in the Same Homicide Type

We first examine the dynamics of spatial changes within individual types of homicide, looking for evidence consistent with the various types of diffusion processes. For example, How does youth-gang homicide spread among census tracts? and Is the spread among neighboring tracts more likely than expected? This *same-type* diffusion involves the influence of local-neighbor rates at time t on subsequent rates in the same type of homicide at time $t + 1$.

Table III reports the resulting tests of diffusion transitions for all three types of homicides. In each case we test whether the diffusion transition rate is significantly larger than the rate of other nonstationary transitions to the same local-neighbor pair of homicide rates. In other words, are transitions that involve diffusion to higher or lower homicide rates more likely than other types of transitions to the same local-neighbor pairs of homicide rates? Table III reports the results for all years combined. Combining all tracts ($n = 174$) and pairs of adjacent years ($n = 4$ for 1991–1992, 1992–1993, 1993–1994, 1994–1995), there is a total of 696 transitions for each homicide type.[21]

Table IIIA looks for evidence of diffusion from neighbor rates to local tracts serving as the *outcomes* of diffusion. There is no evidence of upward

[21]The local and neighbor rates of homicides in Pittsburgh are standardized relative to the distribution of rates in all tracts and all years combined (i.e., 174 tracts by 5 years = 870 total observations). We include all years together so that changes in overall levels from year to year are also detectable as unusually large values in standardized rates. If each year were standardized separately, then extreme values in low-rate years would be treated as similar to extreme values in high-rate years.

contagious diffusion where increases in local tracts follow high neighboring rates (row 2 in Table IIIA). Instead, the significant increases in youth-gang and youth-nongang rates that occur during the study period are compatible with hierarchical diffusion that results in modestly significant upward heaves in noncontiguous tracts (row 1 in Table IIIA). In results compatible with both contagious and hierarchical diffusion, there are significant declines in local rates for youth-nongang and nonyouth homicides (rows 3 and 4 in Table IIIA).[22] Similar declines are not evident in local youth-gang homicide rates.[23]

Table IIIA explores whether local homicide rates are outcomes of diffusion. Some diffusion is evident, especially in *decreasing* youth-nongang and nonyouth homicide rates. Table IIIB examines diffusion from the other direction to assess whether homicide rates in local tracts served as *sources* of diffusion to other tracts. Again, there is no evidence of contagious upward diffusion: high local rates are not associated with subsequent higher rates in neighboring tracts (row 6 in Table IIIB). Nevertheless, local youth-gang homicide rates do emerge as significant sources. They are accompanied by moves to higher rates in other locations through hierarchical diffusion (row 5 in Table IIIB) and by moves to lower rates in neighbors through contagious diffusion (row 7 in Table IIIB). The latter contagion to lower youth-gang rates is evident only during the later years in the 1993–1994 and 1994–1995 transitions. Youth-nongang homicides display evidence of significant diffusion in both an increasing and a decreasing direction, and serving as both outcomes (Table IIIA) and sources (Table IIIB) of diffusion over spatial units. As expected, nonyouth rates are never significant sources of diffusion (last column in Table IIIB).

Figures 8 and 9 display maps of the tracts experiencing significant annual changes in homicide rates some time during the observation period 1991 to 1995. Effects consistent with contagious diffusion are darkly shaded and those with hierarchical diffusion are lightly shaded. Moves to high rates are in Fig. 8 and those to low rates are in Fig. 9. Animations of the changes for successive years are in the accompanying electronic Exhibits 4 to 6 for increasing rates and Exhibits 7 to 9 for decreasing rates.[24] Several general observations emerge from these visual displays and are confirmed in statistical analyses.

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[22]The modestly significant contagious diffusion to lower local youth-nongang rates occurs only in the earliest years (1991 to 1992) when youth-gang homicides began to emerge in the city.

[23]Table IIIB, however, reveals evidence that is consistent with contagion of low youth-gang rates from local tracts to their neighbors. This occurs during the later years in the 1993–1994 and 1994–1995 transitions.

[24]Exhibits are available on the web at http://hdm.heinz.cmu.edu/jackie.

Table III. Patterns of Yearly Changes in Local and Neighbor Tract Homicide Rates in Pittsburgh from 1991 to 1995: "Same-Type" Effects[a]

Diffusion type	Proportion of Tracts with a "significant" change in homicide rate in successive years[b]					
	Youth-gang		Youth-nongang		Nonyouth	
	Diffusion[c]	Other[d]	Diffusion	Other	Diffusion	Other
A. Local tract is outcome of diffusion: Effect of neighbor rate at t on local rate at t+1						
Hierarchical: isolated or global increases (LL to HL, LL to HH)	0.138 (145) *[e]	0.076 (118)	0.143 (140) *	0.071 (198)	0.075 (107)	0.089 (168)
Contagious: expansion or relocation increases (LH to HH, LH to HL)	0.069 (73)	0.126 (190)	0.080 (138)	0.115 (200)	0.107 (122)	0.065 (153)
Contagious: expansion or relocation decreases (HL to LL, HL to LH)	0.130 (23)	0.238 (240)	0.394 (33) *	0.216 (305)	0.552 (29) ***	0.146 (246)
Hierarchical: isolated or global decreases (HH to LH, HH to LL)	0.318 (22)	0.220 (241)	0.741 (27) ***	0.190 (311)	0.647 (17) ***	0.159 (258)
B. Local tract is source of diffusion: Effect of local rate at t on neighbor rate at t+1						
Hierarchical: isolated or global increases (LL to LH, LL to HH)	0.324 (145) ***	0.068 (118)	0.250 (140) ***	0.101 (198)	0.084 (107)	0.125 (168)
Contagious: expansion or relocation increases (HL to LH, HL to HH)	0.043 (23)	0.225 (240)	0.212 (33)	0.157 (305)	0.207 (29)	0.098 (246)

Contagious: expansion or relocation decreases							
(L<u>H</u> to H<u>L</u>, <u>L</u>H to <u>L</u>L)	0.274 (73)	***	0.074 (190)	0.210 (138)	0.145 (200)	0.188 (122)	0.144 (153)
Hierarchical: isolated or global decreases							
(H<u>H</u> to H<u>L</u>, <u>H</u>H to <u>L</u>L)	0.273 (22)		0.116 (241)	0.481 (27) ***	0.145 (311) ***	0.294 (17)	0.155 (258)

[a] The *same-type diffusion* refers to the influence of homicide rates of type i in period t on the same homicide type in period $t + 1$. Panel A focuses on local tracts as *outcomes* of diffusion as reflected in the influence of boldface neighbor rates on rates in the underlined local tracts. Panel B focuses on local tracts as the *source* of diffusion as reflected in the influence of boldface local rates on rates in underlined neighbor tracts.

[b] The transition rates exclude completely all stationary transitions that result in *no change* in local-neighbor pairs. The proportion stationary in each type of local-neighbor pair is in Appendix Fig. B1 available from authors or from the web at http://hdm.heinz.cmu.edu/jackie. The number of tracts eligible for each type of transition is noted in parentheses. A change in rates for a tract is "significant" if the euclidian distance between the local-neighbor pair of rates at time $t + 1$ is more than 2 standard units away from the local-neighbor pair of rates a time t.

[c] Transitions involving diffusion are described in the row headings. For example, in the analysis of outcome effects, contagious diffusion that increases local rates for homicide type i involves transitions from L_iH_i local-neighbor pairs in year t to either H_iL_i or H_iH_i pairs in year $t + 1$. Alternatively, in the analysis of source effects, contagious diffusion that increases neighbor rates for homicide type i involves transitions from H_iL_i local-neighbors pairs in year t to either L_iH_i or H_iH_i pairs in year $t + 1$.

[d] The comparison group of "other" transitions includes all other nonstationary transitions to the same destination local-neighbor pair. For example, in the case of outcome effects in which neighbor rates increase local homicide rates of type i, "other" transitions include all nonstationary changes from L_iL_i, H_iL_i, and H_iH_i at time t to H_iL_i or H_iH_i at time $t + 1$. The individual counts that are the basis for the reported transition rates are in Appendix Fig. B1 available from authors or from the web at http://hdm.heinz.cmu.edu/jackie.

[e] Results of a one-tail test that diffusion transition rates are larger than other transition rates. Significance levels in the one-tail t test are *$p < 0.05$, **$p < 0.01$, and ***$p < 0.001$.
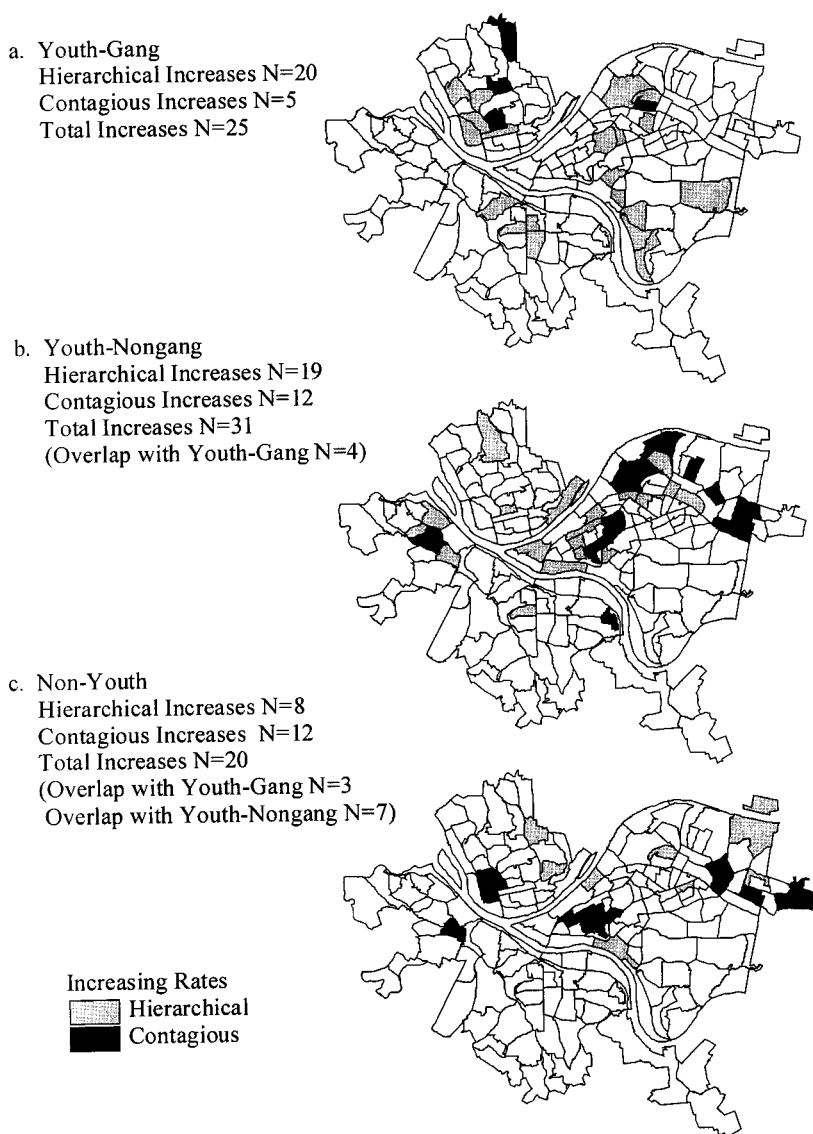
a. Youth-Gang
   Hierarchical Increases N=20
   Contagious Increases N=5
   Total Increases N=25

b. Youth-Nongang
   Hierarchical Increases N=19
   Contagious Increases N=12
   Total Increases N=31
   (Overlap with Youth-Gang N=4)

c. Non-Youth
   Hierarchical Increases N=8
   Contagious Increases N=12
   Total Increases N=20
   (Overlap with Youth-Gang N=3
    Overlap with Youth-Nongang N=7)

Increasing Rates
   Hierarchical
   Contagious

Fig. 8. Significant yearly increases in homicide rates, Pittsburgh 1991–1995.

a. Youth-Gang
   Hierarchical Decreases N=7
   Contagious Decreases N=3
   Total Decreases N=10

b. Youth-Nongang
   Hierarchical Decreases N=18
   Contagious Decreases N=12
   Total Decreases N=28
   (Overlap with Youth-Gang N=0)

c. Non-Youth
   Hierarchical Decreases N=10
   Contagious Decreases N=14
   Total Decreases N=23
   (Overlap with Youth-Gang N=1
    Overlap with Youth-Nongang N=6)

Decreasing Rates
   Hierarchical
   Contagious

Fig. 9. Significant yearly decreases in homicide rates, Pittsburgh 1991–1995.

Table IV. Cooccuring Homicide Types: Tracts with "Significant" Change in More than One Homicide Type[a]

	Number of tracts		
Homicide types	Ever significant change in either homicide type	Significant change in both homicide types	Percentage both
A. Significantly increasing rates			
Youth-gang/Youth-nongang	52	4	7.7
Youth-gang/Nonyouth	42	3	7.1
Youth-nongang/Nonyouth	44	7	15.9
B. Significantly decreasing rates			
Youth-gang/Youth-nongang	40	0	0.0
Youth-gang/Nonyouth	33	1	3.0
Youth-nongang/Nonyouth	48	6	12.5

[a]A change in rates for a tract is "significant" if the euclidean distance between the local-neighbor pair of standardized rates at time $t + 1$ is more than 2 standard units away from the local-neighbor pair of rates at time t.

First, there is very little overlap in tracts that experience significant changes in homicide rates. This is true on more than one dimension. Tracts that experience contagious diffusion in either direction almost never experience hierarchical diffusion in the same direction.[25] There are also very few tracts that experience significant changes in more than one homicide type (Table IV). Youth-gang homicide rates overlap least with the other types of homicide. Fewer than 8% of eligible tracts experience significant increases in youth-gang homicide rates and in youth-nongang or nonyouth homicides, and no more than 3% of eligible tracts have decreasing rates in these same pairs of homicide types. There is more overlap between youth-nongang and nonyouth homicide rates—15.9% of eligible tracts experience substantial increasing rates in both types and 12.5% experience substantial decreasing rates in those types.

Second, it is very common to observe significantly increasing rates followed by significantly decreasing rates for the same homicide type in the same tract (Table V). This pattern—compatible with the sharp changes up and then down that are expected during an epidemic—occurs in more than half of the eligible tracts for each type of homicide in Table V.

The final observation is that tracts experiencing substantial annual changes in homicide rates are widely dispersed throughout the city and not compactly clustered in just a few areas. This is not surprising in view of the

[25]The only exceptions are two tracts with significant decreases consistent with both contagious and hierarchical diffusion in youth-nongang homicide rates and one tract with significant contagious and hierarchical decreases in nonyouth rates.

Table V. Sequences of Transitions in Tracts with "Significant" Changes in Homicide Rates[a]

Type of homicide	Eligible tracts	Percentage			
		Increase followed by decrease	Decrease followed by increase	Decrease followed by decrease	No subsequent change
Youth-gang	17	58.8%	0.0%	0.0%	41.2%
		($n = 10$)	($n = 0$)	($n = 0$)	($n = 7$)
Youth-nongang	31	58.1%	12.9%	3.2%	25.8%
		($n = 18$)	($n = 4$)	($n = 1$)	($n = 8$)
Nonyouth	23	56.5%	17.4%	4.3%	21.7%
		($n = 13$)	($n = 4$)	($n = 1$)	($n = 5$)

[a]To be included in the table, tracts must have at least one "significant" year-to-year change in homicide rates (i.e., a change of more than 2 standard units between local-neighbor rates at t and at $t + 1$). Tracts that do not experience any significant rate change before the final 1994–1995 transition are not eligible for follow-up changes and do not appear.

important role of hierarchical diffusion. The dominant pattern of diffusion is one in which increases in homicide rates emerge simultaneously in disjoint tracts throughout the city—a result that is compatible with youth both in and out of gangs increasingly relying on guns for protection and settling disputes. Tracts exhibiting contagious diffusion are also widely dispersed in disjoint clusters throughout the city. This highlights the fact that contagious diffusion does not require tight clustering of the affected spatial units. Instead, contagious diffusion may emerge in small clusters of tracts that border areas experiencing hierarchical diffusion. If hierarchical diffusion is widely dispersed, then contagion may also be dispersed geographically. Thus, while a few tight clusters are highly suggestive of an underlying contagious diffusion process, contagious diffusion need not result in tightly clustered outcomes.

6.3. Dynamics of Change Across Homicide Types

There is little evidence of cross-type effects within the same tract and year for the various types of homicide. Youth-gang homicides, in particular, do not occur at significant rates in the same tracts and years as youth-nongang or nonyouth homicides (see footnote 18). Nevertheless, other forms of contagious cross-type effects are possible in which homicides of one type in a tract increase the likelihood of future homicides of another type in the same or in neighboring tracts. In this section we explore a number of questions relating to cross-type diffusion. Do newly emerging youth-gang homicides occur in tracts that were previously the sites of high-rate youth-nongang homicides, or in tracts bordering the high-rate tracts? Is the emergence of youth-gang homicides followed subsequently by increasing

Table VI. Patterns of Yearly Changes in Local and Neighbor Tract Homicide Rates in Pittsburgh from 1991 to 1995: "Cross-Type" Effects[a]

Diffusion type	Proportion of Tracts with a "significant" change in homicide rate in successive years[b]			
	Youth-gang effects on youth-nongang rates		Youth-nongang effects on youth-gang rates	
	Diffusion[c]	Other[d]	Diffusion	Other
	A. Local tract is outcome of diffusion			
	Effect of neighbor $YG(t)$ on local $NG(t+1)$		Effect of neighbor $NG(t)$ on local $YG(t+1)$	
Hierarchical: isolated or global increases (<u>LL</u> to H<u>L</u>, <u>LL</u> to H<u>H</u>)	0.161 **[e] (149)	0.052 (136)	0.120 * (142)	0.050 (182)
Contagious: expansion or relocation increases (L<u>H</u> to H<u>H</u>, L<u>H</u> to H<u>L</u>)	0.068 (74)	0.123 (211)	0.044 (138)	0.108 (186)
Contagious: expansion or relocation decreases (H<u>L</u> to <u>LL</u>, H<u>L</u> to L<u>H</u>)	0.500 *** (46)	0.247 (239)	0.333 (21)	0.191 (303)
Hierarchical: isolated or global decreases (H<u>H</u> to L<u>H</u>, H<u>H</u> to <u>LL</u>)	0.563 * (16)	0.271 (269)	0.375 * (23)	0.186 (301)
	B. Local tract is source of diffusion			
	Effect of local $YG(t)$ on neighbor $NG(t+1)$		Effect of local $NG(t)$ on neighbor $YG(t+1)$	
Hierarchical: isolated or global increases (L<u>L</u> to LH, L<u>L</u> to H<u>H</u>)	0.225 *** (142)	0.049 (182)	0.289 *** (149)	0.103 (136)
Contagious: expansion or relocation increases (H<u>L</u> to LH, H<u>L</u> to H<u>H</u>)	0.238 (21)	0.118 (303)	0.174 (46)	0.205 (239)
Contagious: expansion or relocation decreases (L<u>H</u> to HL, L<u>H</u> to L<u>L</u>)	0.203 * (138)	0.118 (186)	0.284 * (74)	0.166 (211)
Hierarchical: isolated or global decreases (H<u>H</u> to HL, H<u>H</u> to L<u>L</u>)	0.375 ** (23)	0.136 (301)	0.313 (16)	0.190 (269)

[a]The *cross-type diffusion* refers to the influence of homicide rates of type i in period t on rates of homicide type j in year $t+1$. Two kinds of cross-type diffusion are examined: youth-gang effects on youth-nongang homicide rates and youth-nongang effects on youth-gang homicide rates. Panel A focuses on local tracts as the *outcomes* of diffusion as reflected in the influence of boldface neighbor rates on underlined rates in local tracts. Panel B focuses on local tracts as the *source* of diffusion as reflected in the influence of boldface local rates on underlined rates in neighbor tracts.
[b]The transition rates exclude completely all stationary transitions that result in *no change* in local-neighbor pairs. The proportion stationary in each type of local-neighbor pair is in Appendix Fig. B2 available from authors or from web at http://hdm.heinz.cmu.edu/jackie. The number of tracts eligible for each type of transition is noted in parentheses. A change in rates for a tract is "significant" if the euclidian distance between the local-neighbor pair of rates at time $t+1$ is more than 2 standard units away from the local-neighbor pair of rates at time t.

youth-nongang rates in the same tracts? Do high rates in youth-gang homicides affect subsequent youth-nongang rates in neighboring tracts?

We first investigated how often there are cross-type effects within the same tract—with "significant" youth-gang rates in a tract followed in the next year by significant youth-nongang rates in the same tract, or vice versa.[26] This never occurs during the 5 years of observations. High local youth-nongang rates ($n = 34$ tracts over 4 years) are never followed by high local youth-gang rates in the same tract, and high local youth-gang rates ($n = 16$ tracts over 4 years) are never followed by high youth-nongang rates in the same tract. The same is also true of low rates—significant low rates that cross types never follow significant low rates in the same tract.

Finally, we looked for evidence of cross-type diffusion among neighboring tracts. Table VIA focuses on the changes in local levels of homicide associated with experiences in neighboring tracts during the previous year. The local tract is viewed as an *outcome* of diffusion from cross-type influences from neighbors. Table VIB focuses on the local tract as a *source* of diffusion to its neighbors. Columns 1 and 2 in the table assess the presence of youth-gang effects on subsequent youth-nongang rates, and columns 3 and 4 assess the reverse effect of youth-nongang rates on subsequent youth-gang rates. In both cases, the test assesses whether the diffusion rate to higher or lower homicide rates occurs more often than expected from the general prevalence of changes to the outcome rates.

[26] Continuing the criterion of earlier analyses, the local homicide rate in a tract is "significant" if the standardized local-neighbor pair of rates is more than 2 standard units away from the origin (i.e., away from the point formed by the means of the respective local and neighbor rate distributions). This criterion is somewhat weaker than the requirement that the local rate alone must be more than two standard units away from its mean.

Whether focusing on the source of diffusion or its outcomes, diffusion effects from *youth-gang* rates to neighboring *youth-nongang* rates (columns 1 and 2) are much more common than the reverse effects. Significant hierarchical diffusion from youth-gang to youth-nongang rates is evident in both increasing (rows 1 and 5) and decreasing (rows 4 and 8) directions.

Contagious diffusion across these homicide types is of particular interest. There is no evidence of sustained significant contagion to increasing youth-nongang rates (rows 2 and 6). Within the individual years (not in table), however, there is evidence of modestly significant contagion ($p \leq 0.05$, one-tailed test) from tracts with high local youth-gang rates to increased youth-nongang rates in neighboring tracts during the year of peak growth in homicide (from 1992 to 1993). Some contagion of decreasing rates is also evident. This is generally confined to the earliest transition from 1991 to 1992. Thus, within clusters of local and neighboring tracts, there is some evidence that contagious diffusion across types does occur more often than expected from the general prevalence of the same outcome, but this contagion is generally limited to selected years during the observation period.

Figure 10 displays the locations of significant cross-type diffusion for all years combined.[27] As with same-type diffusion discussed earlier, changing rates that are compatible with cross-type diffusion are generally not clustered together but, rather, are dispersed broadly across tracts in the city.

There is some overlap between tracts that experience cross-type diffusion and those displaying same-type diffusion. Most of this overlap is in hierarchical types of diffusion where general increases or decreases in homicide rates occur broadly and without regard to neighboring levels of either the same type or cross-types of homicide. Just under one-third of tracts that exhibit significant cross-type diffusion from youth-gang to youth-nongang rates also experience significant same-type diffusion. In these tracts it is difficult to distinguish the relative influence of same-type and cross-type diffusion.

6.4. Factors Associated with Diffusion of Homicide—Gangs and Racial Residential Patterns

We do not undertake a formal statistical analysis of what factors might be associated with diffusion of homicides. Two factors, however, do emerge as potentially important in accounting for the spatial distribution of homicides in Pittsburgh. The first factor is the locations where different gang sets hang out. These areas of daily gang activity, which we call "set space," are very localized, within just a few blocks within a neighborhood. Figure 11

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[27]Animations of the cross-type effects in successive years are in the accompanying electronic Exhibits 10 and 11 for increasing rates and Exhibits 12 and 13 for decreasing rates, available on the web at http://hdm.heinz.cmu.edu/jackie
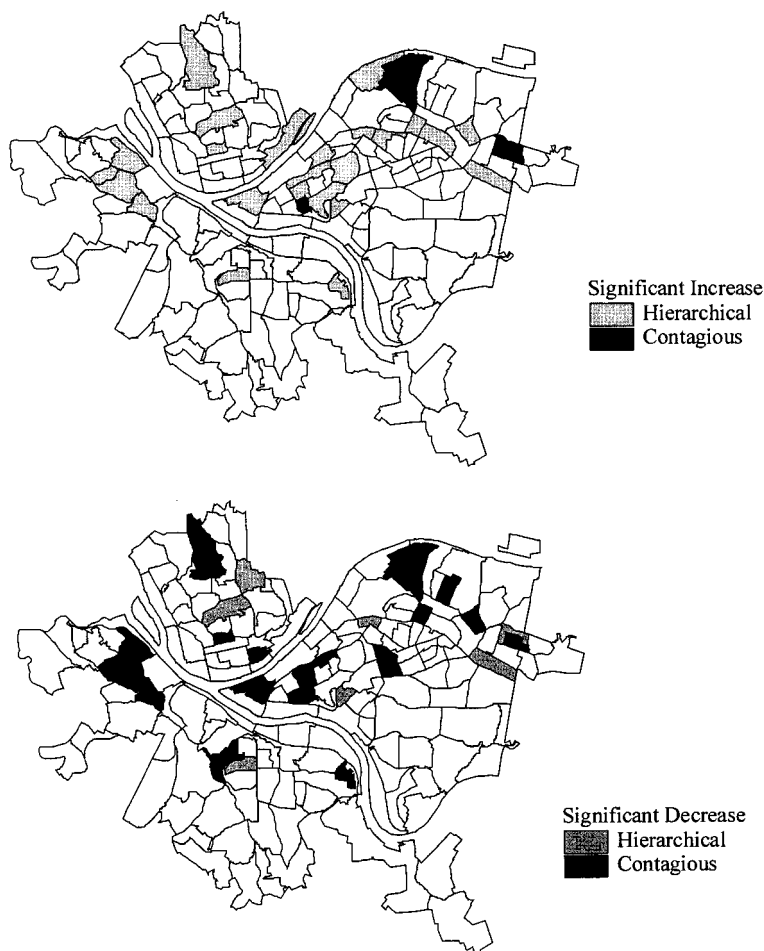
Significant Increase
Hierarchical
Contagious

Significant Decrease
Hierarchical
Contagious

Fig. 10. Cross-type effects of neighbor youth-gang rates on local youth-nongang rates.

displays the tract locations of set space for violent youth gangs in Pittsburgh.[28]

It is not surprising that tracts with set space overlap tracts that experience substantial diffusion of youth-gang homicides in Figs. 8 to 10. Set space tracts also figure prominently in youth-nongang and nonyouth diffusion. Table VII reports the distribution of tracts by the presence of homicide diffusion and set space. When diffusion does occur, it is more likely to

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[28]Tita *et al*. (1998) provide a more comprehensive analysis of set space locations, including a multivariate analysis of the place specific features associated with these locations.
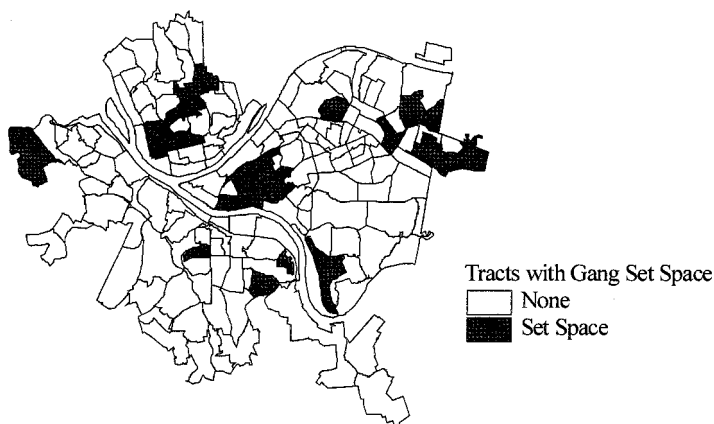
Fig. 11. Tracts with youth-gang set space in Pittsburgh.

be found in tracts with set space. This association is statistically significant for all types of diffusion with one important exception—diffusion to decreasing rates of youth-gang homicides is not more likely in set space tracts. In part this reflects the generally low prevalence of substantial declines in youth-gang rates (found in only 10 tracts for same-type diffusion and 12 tracts for cross-type diffusion). Such declines in youth-gang rates are apparently even less likely in tracts with set space.

There is also an unexpected result across the different types of homicides. Diffusion of *nonyouth* homicides is more concentrated in set-space tracts (52 to 60% of diffusion tracts also contain set space) than diffusion of youth-gang or youth-nongang homicides (30 to 40% of diffusion tracts also contain set space). The concentration of diffusion in set space tracts is not mirrored in the distribution of homicide incidents, where only youth-gang incidents are much more likely to be concentrated in set space tracts. So, contrasting among the three types of homicide, *diffusion* in nonyouth rates is more likely to be concentrated in set space tracts, while nonyouth homicide *incidents* are not more concentrated in set space tracts. This hints at a possible distinction among nonyouth homicides. Specifically, diffusion may be relevant to only some nonyouth homicides, possibly through links to gang activities and to youth-gang homicides that affect nonyouth homicides in set space tracts.

Race is another compelling distinguishing factor in the diffusion of homicide in recent years. We know that black male youth are disproportionately overrepresented among both offenders and victims in homicide incidents, and increasingly so during the recent period of dramatic growth in

Table VII. Distribution of Tracts with Significant Diffusion by Presence of Gang Set Space or Not, 1991–1995 Combined[a]

Type of diffusion	Homicide type	Total tracts ever diffuse	Diffusion in tracts		Percentage diffusion in set space tracts	Set space tracts without diffusion	Fisher exact probability test (2-tail)
			Without set space	With set space			
Increasing rates of same type	Youth-gang	25	15	10	40.0	22	0.009
	Youth-nongang	31	20	11	35.5	21	0.011
	Nonyouth	20	8	12	60.0	20	<0.001
Decreasing rates of same type	Youth-gang	10	7	3	30.0	29	0.394
	Youth-nongang	28	17	11	39.3	21	0.006
	Nonyouth	23	11	12	52.2	20	<0.001
Cross-type increasing	Youth-gang	23	15	8	34.8	24	0.042
	Youth-nongang	29	19	10	34.5	22	0.032
Cross-type decreasing	Youth-gang	12	5	7	58.3	25	0.002
	Youth-nongang	29	18	11	37.9	21	0.007
Set space in all tracts			142	32	18.4		

[a]Counts are of tracts that ever experience annual diffusion anytime in the period 1991 to 1995. The total number of tracts in the city is 174. The Fisher exact probability test assesses independence between presence of gang set space and diffusion. This test replaces the more customary chi-square test, which is unreliable when the expected number of observations falls below five in any cell in the frequency table.
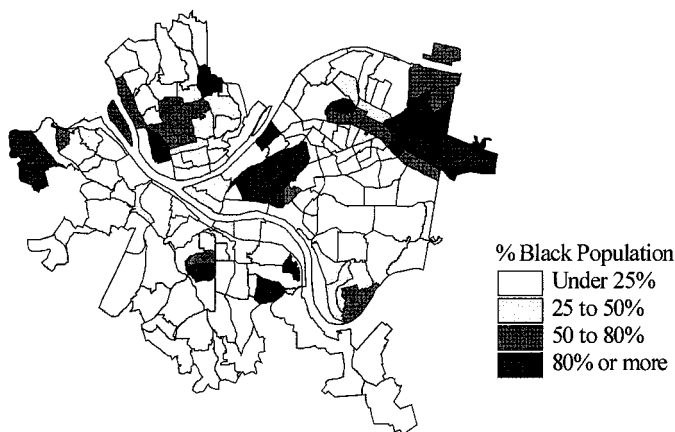
% Black Population
□ Under 25%
▨ 25 to 50%
▦ 50 to 80%
■ 80% or more

Fig. 12. Percentage black population by tract in Pittsburgh.

city homicide rates. This pattern common to so many cities in the United States also occurred in Pittsburgh. The distinctive character of racial residential patterns in the city is also likely to be an important factor in the observed diffusion patterns of homicides.

Figure 12 displays the racial distribution of Pittsburgh's population. Unlike many other cities, Pittsburgh's black population is dispersed over several distinct predominantly black areas. Focusing only on tracts with at least 50% black population in 1990 (citywide the population was 26% black in 1990), three large black areas, each consisting of eight or more census tracts, are evident in the northeastern, central, and northwestern regions of the cities. There are also four isolated tracts containing large public housing developments in the southern part of the city. These multiple points of population concentration are likely to be factors in the pattern of multiple point diffusion observed in the city. A simple visual inspection of Figs. 8 to 10 and 12 confirms the strong overlap between areas of racial residential concentration and the spatial distribution of homicide diffusion.

Table VIII reports the joint distributions of tracts experiencing substantial homicide diffusion and tracts with at least 25% black residents (designated "black" tracts in the table).[29] With one notable exception, when diffusion occurs, it is more likely to occur in "black" tracts. While just over one-third of all tracts have populations that are at least 25% black, 56 to 85% of tracts with diffusion are "black". As in the case of set space, the one exception that is not overrepresented in black tracts is diffusion to

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[29]The associations between diffusion and "black" tracts are weaker when the threshold size used to define "black" tracts increases.

Table VIII. Distribution of Tracts with Significant Diffusion by Percentage Black Population Residing in Census Tract, 1991–1995 Combined[a]

Type of diffusion	Homicide type	Total tracts ever diffuse	Diffusion		Percentage diffusion in black tracts	Black tracts without diffusion	Fisher exact probability test (2-tail)
			Nonblack tracts	Black tracts			
Increasing rates of same type	Youth-gang	25	11	14	56.0	50	0.043
	Youth-nongang	31	12	19	61.3	45	0.003
	Nonyouth	20	3	17	85.0	47	<0.001
Decreasing rates of same type	Youth-gang	10	6	4	40.0	60	1.000
	Youth-nongang	28	10	18	64.3	46	0.002
	Nonyouth	23	4	19	82.6	45	<0.001
Cross-type increasing	Youth-gang	23	10	13	56.5	51	0.061
	Youth-nongang	29	10	19	65.5	45	0.001
Cross-type decreasing	Youth-gang	12	4	8	66.7	56	0.033
	Youth-nongang	29	11	18	62.1	46	0.003
Racial distribution, all tracts			110	64	36.8		

[a]Counts are of tracts that ever experience annual diffusion anytime in the period 1991 to 1995. The total number of tracts in the city is 174. Tracts with at least 25% black residents in 1990 are treated as "black" tracts for this analysis. The Fisher exact probability test assesses independence between black population and diffusion. This test replaces the more customary chi-square test, which is unreliable when the expected number of observations falls below five in any cell in the frequency table.

decreasing youth-gang rates. Also, diffusion of *nonyouth* homicides is more concentrated in black tracts than diffusion of youth-gang or youth-nongang homicides. As before, this may signal a distinction within nonyouth homicides, with those in black tracts subject to greater diffusion, possibly with greater cross-type links to youth-gang and youth-nongang homicides, than prevails in other nonyouth homicides.

The bivariate cross-sectional relationships of homicide with the location of youth gangs and with the racial concentration of the population across different neighborhoods are strong. The link to race-based residential patterns raises important concerns about the causal nature of the processes that underlie the observed spatial diffusion in the various types of homicide. Is diffusion linked in causal ways to features of the homicide incidents, or is the apparent spatial diffusion merely a manifestation of spatial features in residential patterns? The answer has implications for the likely effectiveness of intervention strategies that seek to reduce diffusion by targeting specific features of homicide incidents.

7. CONCLUSIONS

In this paper we propose a new method for examining the dynamics of changes in the spatial distribution of a phenomenon. Recently introduced exploratory spatial data analysis (ESDA) techniques provide social scientists with a new set of tools for distinguishing between random and non-random spatial patterns of events (Anselin, 1998). Existing ESDA measures, however, are static and do not permit comparisons of distributions of events in the same space but across different time periods. One ESDA method—the Moran scatterplot—has special heuristic value because it provides visual display of local spatial relationships between each spatial unit and its neighbors. We extend this static cross-sectional view of the spatial distribution of events to consider dynamic features of changes over time in spatial dependencies. The method distinguishes between *contagious diffusion* between adjoining units and *hierarchical diffusion*, which spreads broadly through commonly shared influences.

Prompted by the apparently epidemic increases in city homicide levels in the 10 years since 1985, we use the proposed method to analyze the dynamics of changing spatial patterns in the distribution of homicide incidents across neighborhoods in one city. We look especially for indications that the sharp increases in citywide homicide totals are accompanied by any systematic spatial diffusion (or spread) of homicide across different neighborhoods in a city. Because of the historically important role of interpersonal conflicts between intimates and acquaintances, homicide is traditionally not viewed as subject to spatial diffusion. Newly emerging crack cocaine markets and violent youth gangs, accompanied by the spread of

handguns among increasingly more youthful populations, substantially changed the landscape of homicide incidents. These enterprises share a number of organizational and structural features that are conducive to spatial diffusion of violence.

A preliminary analysis of citywide levels of drug-related and gang-related homicides indicated that newly emerging crack markets apparently were not a direct factor in the sharp growth in homicides during the early 1990s in Pittsburgh. Gang-related homicides, however, were strongly implicated as a factor in the rise of homicides. Our method for analyzing the spatial dynamics of changes over time provides support for the spatial diffusion of homicides across different census tracts in a city. Spatial diffusion of homicide risk to new locations is evident in the data.

Contagious diffusion of increasing homicide rates across neighboring tracts is evident only during the year of peak growth in total homicides, when high local rates of youth-gang homicides are followed by significant increases in neighboring youth-nongang rates. This pattern is consistent with a spread of homicides from gang youth to nongang youth. Otherwise, the increases in both youth-gang and youth-nongang homicides generally occur simultaneously in nonneighboring tracts. Some contagion is also evident early in the observation period when decreased rates of youth-nongang rates ripple among neighboring tracts in the same year that youth-gang homicides just begin to grow. Youth-gang homicides display similar contagious declines in later years. Such declines are symptomatic of the kind of subsiding rates that are expected during a homicide epidemic.

Based on anecdotal evidence emerging from a number of different cities, there is good reason to expect variation across cities in the relative roles of drug markets and youth gangs in the recent rise of youth homicides. The analysis in Pittsburgh highlights the value of explicitly partitioning among distinct types of homicide when considering diffusion processes. We hope that the ideas presented here are provocative enough to encourage others to adopt a similar framework and methodological approach to examine the city specific homicide patterns. Further extensions might include a broader view of contagion that goes beyond a narrow conception of spatial proximity to include alternative forms of ''proximity'' that depend on various social dimensions that are the basis for connections among population groups. In the case of gangs, for example, contagion from particular patterns of rivalries among gangs might underlie the spread of gang-related homicides among apparently disjoint spatial units.

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