The Coming Crisis of Public Health in the Suburbs

RODRICK WALLACE and DEBORAH WALLACE

New York Psychiatric Institute; Consumers Union

GEOGRAPHIC DIFFUSION, OR THE VARYING FORMS of the spatial spread of contagious phenomena—including, but not limited to, disease—has been a central topic of intellectual study in a variety of disciplines ranging from history to economics, anthropology, and sociology, as well as epidemiology and, more recently, urban studies. Rumors, fads, innovations, epidemics, and urban decay have all been found to spread in space and time in similar patterns, reflecting the social, economic, and geographic structuring of the underlying human communities. These patterns, on decreasing spatial scales, may be generally classified as *hierarchical*, *spatially contagious*, or *network* diffusion, usually in combinations with varying emphasis (Cliff et al. 1981; Abler, Adams, and Gould 1971).

Hierarchical diffusion refers to the spread from larger, more socially dominant central cities to smaller ones along the transportation network. Two geographically distant places may be close together in a "sociogeographic" space if many people travel frequently between them.

Spatial contagion, or expansion diffusion, involves radial spread from an infected geographic epicenter into nearby communities, like a wine stain on a tablecloth.

The Milbank Quarterly, Vol. 71, No. 4, 1993

Network diffusion, often on a relatively smaller scale, spreads along personal, domestic, and community social networks whose structure may sometimes differ from that imposed by simple spatial adjacency or larger organizational hierarchy.

Social, economic, geographic, and other structures, which can change over time and vary with place, determine which modes will predominate in a specific circumstance (Abler, Adams, and Gould 1971).

Here we will explore the implications of these geographic processes for the more general transmission of contagious diseases now intensifying and evolving within physically and socially disintegrating minority urban neighborhoods of the United States. Because one-quarter of the U.S. population resides within central cities, and another half lives in surrounding rings of suburbs, phenomena of spatial spread will increasingly affect the health status of even affluent, suburbanized communities.

We briefly examine the nature of the physical and social disintegration of inner-city neighborhoods and its linkage to new epidemics, and explore implications for the spread of disease to the suburbs.

The "Hollowing Out" of Cities and the Deterioration of Urban Public Health

Many large U.S. cities besides New York now display a characteristic "hollowed out" pattern of one or more rapidly expanding zones of decayed and partly abandoned minority neighborhoods surrounded by overcrowded and threatened "transitional" communities. The hollowing-out process itself is often associated with massive loss of housing and great population transfer. For example, large areas of the South-Central Bronx lost between 50 and 80 percent of their housing and population between 1970 and 1980. Other examples range from the Central Ward of Newark, parts of North Philadelphia, Washington, D.C., and Detroit, to South-Central Los Angeles and the Overtown and Liberty City districts of Miami.

Michael Dear (1976) first described the contagious nature of this hollowing-out process, finding that initial building abandonments "seeded" a neighborhood for subsequent ones, and concluding that such contagion has major implications both for understanding the dynamics of abandonment and for later policy considerations. In particular, once

abandonment has begun it is likely to be very difficult to stop, and may become self-sustaining under the force of contagion.

Odland and coworkers, following Dear's lead (Odland 1983; Odland and Barff 1982; Odland and Balzer 1979), reported a similar phenomenon for Indianapolis.

With our colleagues, we have independently observed the contagious nature of the spatially spreading "South Bronx" process of fire and housing abandonment in several neighborhoods of New York City, particularly noting the critical role of municipal service delivery in either slowing or accelerating community burnout. Our work also identified an interaction between housing overcrowding, poverty, and municipal service levels as determining the "susceptibility" of an area to rapid, contagious urban decay. We described the pattern of forced mass migration from burning areas, which not only disrupts underlying community structures, but also spreads susceptibility to nearby communities (R. Wallace 1978, 1981, 1982, 1989, 1990a,b; R. Wallace and D. Wallace 1977, 1983).

Once a spatially contagious process of urban decay begins in a system where the density of susceptible housing exceeds a threshold, there will be relentless geographic spread of the affected area and massive displacement of population (R. Wallace 1990b; Schuerman and Kobrin 1986; Skogan 1986a,b, 1990; Bursik 1986). See R. Wallace (1981, 1988, 1991a) for a more formal analysis.

McCord and Freeman (1990) estimate that by 1980 some 650,000 people lived in partly devastated minority neighborhoods of New York City, which, like Central Harlem, began to experience raised levels of contagious and chronic disease, substance abuse, and violence, leading to life expectancies for adult males lower than those observed in Bangladesh.

A large body of current research on public physical and mental health, as well as on crime and deviant behavior, provides a basis for understanding McCord and Freeman's observations. This work focuses on the status of social networks and larger social structures as essential both for socialization and control of unacceptable activity and for maintenance of individual and community health. We briefly summarize that literature.

Recent research has found a strong association between rapid mass migrations and behavioral deviance, including crime and violence (South 1987; Crutchfield, Geerken, and Gove 1982; Sampson and Groves 1989). Part of the deviance arises from weakening of normative community ac-

tivity, such as the socialization and supervision of adolescents, which requires strong social organization.

A parallel line of inquiry examines the connection between social structure and health, finding that social relations profoundly affect general health and buffer individuals from stressful events (Cohen and Wills 1985; Rabkin and Struening 1976; Lin and Dean 1984; Lin and Ensel 1984; House, Landis, and Umberson 1988). The poor particularly need cushioning from the effects of lack of resources, but they are especially vulnerable to forces that disperse their social networks, as are minorities and the elderly (House, Landis, and Umberson 1988). Earlier research showed that the destruction of a familiar place and its social networks is itself a stressful event (Fried 1963, 1965; Key 1967; Niebanck 1968).

Forced displacement may be expected to have particularly adverse impacts (Kasl 1977). Cassel (1974) describes psychophysiological mechanisms for this phenomenon, while the association between social disintegration and mental illness was explored in the classic Stirling County studies (A. Leighton 1959; D. Leighton et al. 1963).

Substance abuse and its correlates form a characteristic nexus associated with social and environmental instability. Alcohol abuse is significantly correlated with violence (Collins 1981) and with use of other drugs (Kane 1981). Adolescence and young adulthood, in particular, are times of elevated susceptibility to development of this nexus (Osgood et al. 1988), which features intertwining of drug use and sexual activity (Belcastro and Nicholson 1982; Zabin et al. 1986), matters of obvious importance for the control of HIV infection in urban minority adolescent populations.

The criminology literature describes a vicious circle of community physical destruction leading to social disintegration, causing violent and deviant behaviors, and resulting in yet more physical destruction and social disintegration (Skogan 1986a,b, 1990). Indeed, physical destruction in parts of Los Angeles presaged increased criminal behavior, which triggered further physical deterioration (Schuerman and Kobrin 1986). For a detailed analysis of this feedback mechanism and its spatial expression as an expanding zone of physical devastation and behavioral pathology, see R. Wallace (1991a).

L. Hinkle summarizes much of this process, concluding that it is the social environment, and not the physical environment per se, that is the primary determinant of the health and well-being of people who live in cities (Hinkle and Loring 1977). Thus, disruption of that social environment may be expected to precipitate deterioration in public health.

This general framework provides a perspective for understanding many of the spatial and temporal patterns of the recent deterioration of public health and public order within New York City and elsewhere, which was caused by the rapid hollowing out of minority urban neighborhoods (R. Wallace 1990b, 1993a,b,c,d; Wallace and Fullilove 1991; Wallace, Fullilove, and Wallace 1992; R. Wallace and D. Wallace 1990; D. Wallace and R. Wallace 1993; Struening, Wallace, and Moore 1990; D. Wallace 1990).

These results have implications as well for future disease outbreaks. Recent application of principles from evolutionary biology to disease ecology—"Darwinian epidemiology"—strongly suggests that raised rates of the transmission of contagious disease resulting from the coupled physical and social disintegration of inner-city neighborhoods can cause the development of more rapidly reproducing—that is, more virulent—strains of many disease agents (Ewald 1991, 1993; Cowley 1993; Yoon 1993). This is because what Ewald characterizes as "cultural vectors"—behaviors driven by social, economic, or other collective factors—can result in greater rates of transmission of virulent strains between infected and susceptible persons than the incapacitation caused by the disease alone would ordinarily permit.

In sum, these literatures and empirical results strongly imply that public health and public order in congested urban areas have much the same foundation in the stability and integrity of personal, domestic, and community social networks and institutions embedded in the physical structures of a community. Programs aimed at "controlling disease"—including AIDS, multiple-drug-resistant tuberculosis (MDRTB) and drug-resistant gonorrhea (PPNG)—or at "controlling crime" in disintegrating neighborhoods will have a severely limited effect unless they are in partnership with programs to stabilize and revitalize the closely interlinked community physical and social structures.

The Diffusion of Contagious Disease from Inner City to Suburbs

Addressing the central point of this analysis, we can ask, Why worry about the U.S. suburbs? As poverty, social disintegration, and their consequences for public health and public order were supposedly left behind in the central cities by the outward suburban migrations, won't infectious diseases, new and old, be left behind as well?

There are compelling reasons to believe otherwise.

First, population displacement, at least in New York City, has not just been the movement of minority population within the confines of the city itself, but has involved as well the increasing suburbanization of blacks. Analysis of census data for 1970, 1980, and 1990 shows that, whereas the total population of nearby Westchester, Nassau, and Suffolk counties near New York has remained near 3.5 million, the percent classified as "nonwhite" by the census rose from 6.4 in 1970 to 10.2 by 1980 and to 13.9 by 1990. For Nassau the rate of change is highest, as the percentage of the nonwhite population more than doubled between 1970 and 1990.

In a recently published book, Massey and Denton (1992) show clearly that for blacks in the North this suburbanization brought not integration, but a slightly less extreme form of segregation. They found that within most northern areas, including New York City, black isolation in suburbs is remarkably high given the relatively small proportions of blacks they contain. Even where blacks constitute a relatively high fraction of the suburban population, their uneven distribution results in a high degree of isolation. Massey and Denton found that, although suburbanization of blacks had begun in most metropolitan areas by 1980, their entry into suburbs did not bring integration. Rather, suburban blacks have experienced considerable segregation, isolation, and continued economic deprivation, which tended to be high in suburbs where blacks were represented in large numbers.

They write:

> Black suburbanization often does not eliminate black-white disparities in residential quality. Indeed, suburbs that accept black residents tend to be older areas of relatively low socioeconomic status and high population density. . . . In many ways, black suburbs replicate the problems of the inner city. (1992, 69)

Thus, black suburbanization has produced a spatially dispersed pattern recognizably similar to the congregated/segregated urban neighborhoods from which many suburban blacks migrated. We suspect suburban minority enclaves may also begin to experience a similar decay and deterioration of public health and order because suburban governments are not structured to provide levels of municipal services required to manage both the high housing overcrowding and population densities of poor

populations and the sociopathic consequences of mass migration and social disruption.

If another wave of burnout were to consume newly overcrowded minority neighborhoods within New York City, nearby suburban "enclaves" would likely become even more crowded, and perhaps would begin to suffer patterns of contagious decay similar to those of crowded minority neighborhoods in central cities. In addition, they would attract the problems of public health and public order associated with forced displacement from the city itself.

Thus, black suburbanization has not been random, but rather has involved intensification of a citylike patchwork of relatively poor, crowded minority communities surrounded by more affluent, exclusively zoned, and largely white suburban townships. Parts of Yonkers and Mt. Vernon in Westchester County and areas of Hempstead Village, Freeport, and Amityville on Long Island are examples. Indeed, streets in the black part of Mount Vernon are being renamed for black national heroes such as King and DuBois.

In sum, the standard racially and economically congregated/segregated pattern of neighborhoods in the central city has been replicated in the extended urban/suburban complexes that have sprung up around U.S. central cities since the end of World War II in tandem with the widespread automobile ownership that is closely coupled with patterns of highway construction.

Second, a large and growing body of research in medical geography suggests that, from the viewpoint of disease ecology, suburban isolation is at best fragile, even without continued forced displacement of population from inner-city neighborhoods. This is particularly true for infection by agents like HIV or primary MDRTB, for which there are no early self-limiting symptoms, no immunization, and no cure.

Figure 1A, from Gould (1993), shows the early pattern of spatial diffusion of AIDS from Manhattan into nearby counties between 1982 and 1984. The vertical axis is the county's rate of AIDS cases per 100,000 population and the horizontal axis indicates the centroid distance from Fifth Avenue and 42nd Street in Manhattan. The classic distance-decay pattern of diffusive spread is clear, as is the temporal intensification of that pattern between the two years. Figure 1B shows similar information for Washington, D.C., in the years 1986 and 1990.

Whereas figure 1A appears to represent the early stages of infection

There are compelling reasons to believe otherwise.

First, population displacement, at least in New York City, has not just been the movement of minority population within the confines of the city itself, but has involved as well the increasing suburbanization of blacks. Analysis of census data for 1970, 1980, and 1990 shows that, whereas the total population of nearby Westchester, Nassau, and Suffolk counties near New York has remained near 3.5 million, the percent classified as "nonwhite" by the census rose from 6.4 in 1970 to 10.2 by 1980 and to 13.9 by 1990. For Nassau the rate of change is highest, as the percentage of the nonwhite population more than doubled between 1970 and 1990.

In a recently published book, Massey and Denton (1992) show clearly that for blacks in the North this suburbanization brought not integration, but a slightly less extreme form of segregation. They found that within most northern areas, including New York City, black isolation in suburbs is remarkably high given the relatively small proportions of blacks they contain. Even where blacks constitute a relatively high fraction of the suburban population, their uneven distribution results in a high degree of isolation. Massey and Denton found that, although suburbanization of blacks had begun in most metropolitan areas by 1980, their entry into suburbs did not bring integration. Rather, suburban blacks have experienced considerable segregation, isolation, and continued economic deprivation, which tended to be high in suburbs where blacks were represented in large numbers.

They write:

> Black suburbanization often does not eliminate black-white disparities in residential quality. Indeed, suburbs that accept black residents tend to be older areas of relatively low socioeconomic status and high population density. . . . In many ways, black suburbs replicate the problems of the inner city. (1992, 69)

Thus, black suburbanization has produced a spatially dispersed pattern recognizably similar to the congregated/segregated urban neighborhoods from which many suburban blacks migrated. We suspect suburban minority enclaves may also begin to experience a similar decay and deterioration of public health and order because suburban governments are not structured to provide levels of municipal services required to manage both the high housing overcrowding and population densities of poor

populations and the sociopathic consequences of mass migration and social disruption.

If another wave of burnout were to consume newly overcrowded minority neighborhoods within New York City, nearby suburban "enclaves" would likely become even more crowded, and perhaps would begin to suffer patterns of contagious decay similar to those of crowded minority neighborhoods in central cities. In addition, they would attract the problems of public health and public order associated with forced displacement from the city itself.

Thus, black suburbanization has not been random, but rather has involved intensification of a citylike patchwork of relatively poor, crowded minority communities surrounded by more affluent, exclusively zoned, and largely white suburban townships. Parts of Yonkers and Mt. Vernon in Westchester County and areas of Hempstead Village, Freeport, and Amityville on Long Island are examples. Indeed, streets in the black part of Mount Vernon are being renamed for black national heroes such as King and DuBois.

In sum, the standard racially and economically congregated/segregated pattern of neighborhoods in the central city has been replicated in the extended urban/suburban complexes that have sprung up around U.S. central cities since the end of World War II in tandem with the widespread automobile ownership that is closely coupled with patterns of highway construction.

Second, a large and growing body of research in medical geography suggests that, from the viewpoint of disease ecology, suburban isolation is at best fragile, even without continued forced displacement of population from inner-city neighborhoods. This is particularly true for infection by agents like HIV or primary MDRTB, for which there are no early self-limiting symptoms, no immunization, and no cure.

Figure 1A, from Gould (1993), shows the early pattern of spatial diffusion of AIDS from Manhattan into nearby counties between 1982 and 1984. The vertical axis is the county's rate of AIDS cases per 100,000 population and the horizontal axis indicates the centroid distance from Fifth Avenue and 42nd Street in Manhattan. The classic distance-decay pattern of diffusive spread is clear, as is the temporal intensification of that pattern between the two years. Figure 1B shows similar information for Washington, D.C., in the years 1986 and 1990.

Whereas figure 1A appears to represent the early stages of infection

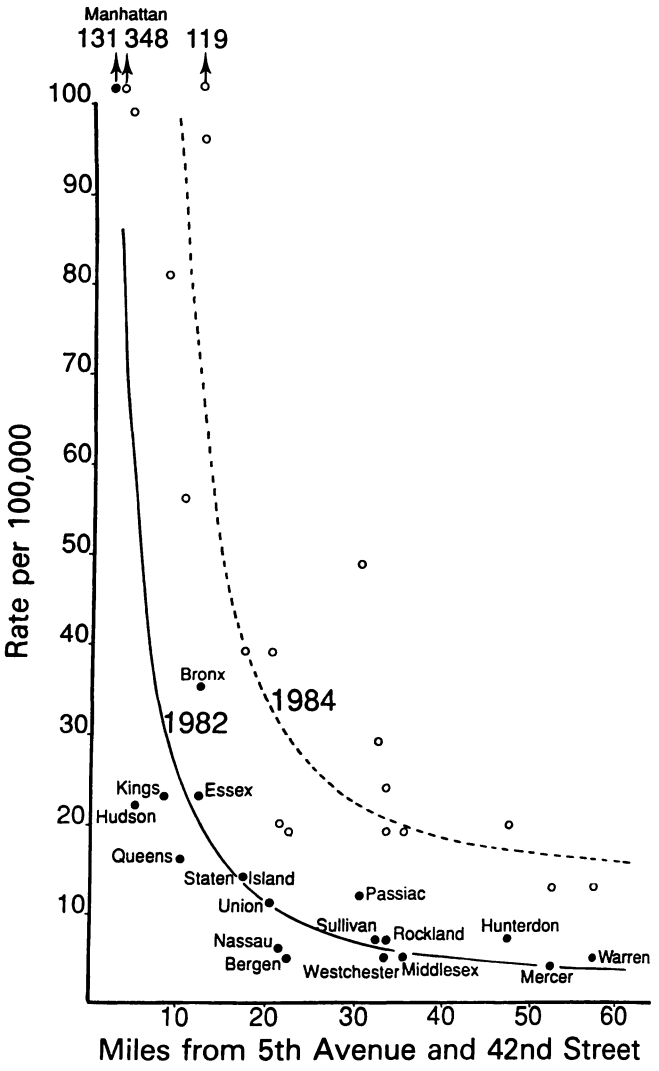

FIG. 1.A. Rate of AIDS cases per 100,000 population as a function of county centroid distance from Fifth Avenue and 42nd Street in Manhattan. ●, 1982; ○, 1984. (*Source:* Gould 1993)

for male homosexuals, figure 1B seems to show the later convolution of both intravenous drug users and male homosexuals.

Some very elementary analysis and a reformulation of figure 1A shows matters in more detail. We basically propose that a sudden, explosive

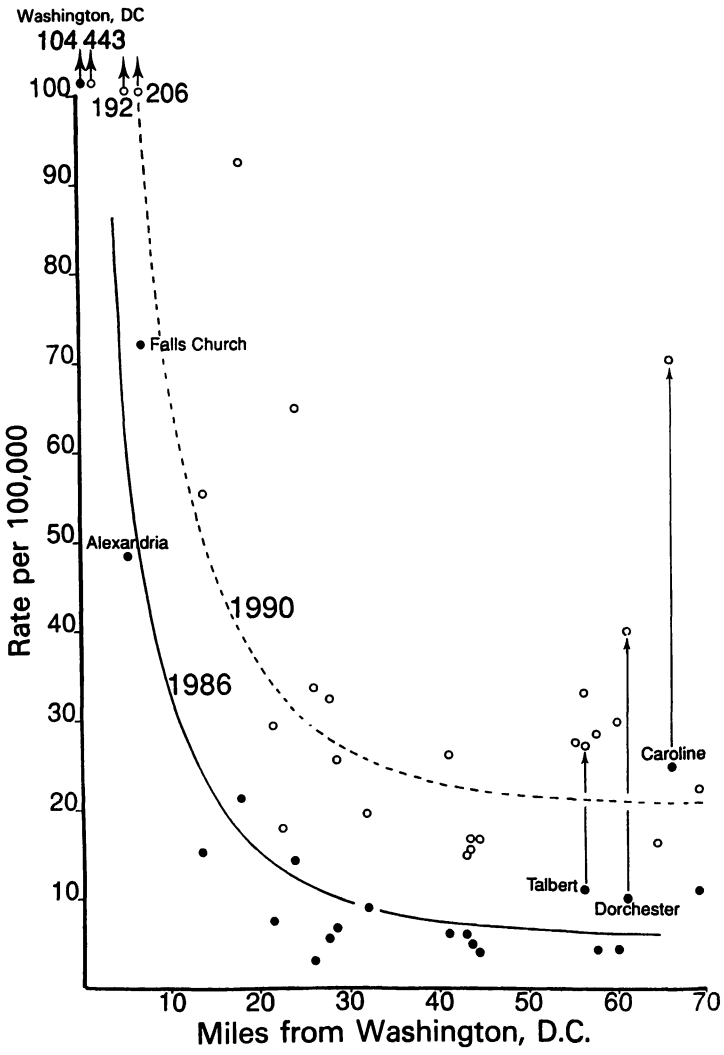

FIG. 1.B. Same as (A) for county distance from Washington, D.C. ●, 1986; ○, 1990. (*Source:* Gould 1993)

spread of disease within a central-city epicenter will diffuse along transport routes into nearby suburbs in proportion to the magnitude of that initial outbreak, as modulated, in some sense, by the "commuting field" between city and suburbs. We look at the very early stage of the infection process, before large numbers of susceptible individuals within a suburb are infected.

Let S represent the suburb and E a nearby inner-city epicenter. $I_0(E)$ is taken as the number of individuals infected within the epicenter during a very sudden, "explosively" rapid disease outbreak. Let $N(S)$ be the number of susceptible individuals within the suburb before a significant proportion of the population becomes infected. We look at the initial rise in number of suburban infecteds, $\Delta I(S)$, in a very short time period, Δt, following the explosive epicenter disease outbreak. The simplest, and indeed the standard, model of disease transmission (Bailey 1975) under these circumstances is just

$$\Delta I(S)/\Delta t \approx \beta I_0(E) N(S) f(E,S) \tag{1}$$

where $\Delta I(S)/\Delta t$ is the rate of increase per unit time of the number of infective individuals within the suburb, β is the rate of infectivity of contacts between individuals in the epicenter and the suburb, and $f(E,S)$ is the *commuting field*, that is, the probability of contact between individuals living in the epicenter E and the suburb S.

A slight algebraic rearrangement of this expression gives us something we can actually test. Multiplying both sides of equation (1) by the time interval Δt and dividing by the number of susceptible suburban individuals $N(S)$ gives

$$\Delta I(S)/N(S) \approx I_0(E) f(E,S) \beta \Delta t \propto I_0(E) f(E,S) \propto f(E,S). \tag{2}$$

This relation states that, for a suburb in the early stages of an infection spreading primarily from contact with a central-city epicenter—before establishment of independent local disease epicenters within the suburbs themselves—the per capita infection rate will be directly proportional to the infection burden of the central city epicenter, as modulated by the commuting field between epicenter and suburb.

Figure 2 explores this proposition for cumulative reported AIDS cases in the eight New York State suburban counties around Manhattan, the most heavily infected regional epicenter. On the vertical axis are reported cumulative AIDS case rates per 100,000 population through 1987 and through 1991, for Queens, Richmond, Westchester, Nassau, Rockland, Putnam, Suffolk, and Orange Counties (New York State Department of Health 1988, 1991). On the horizontal axis is the percent of the county workforce employed in Manhattan, as determined by the 1990 census. This is taken as an environmental index of the probability of

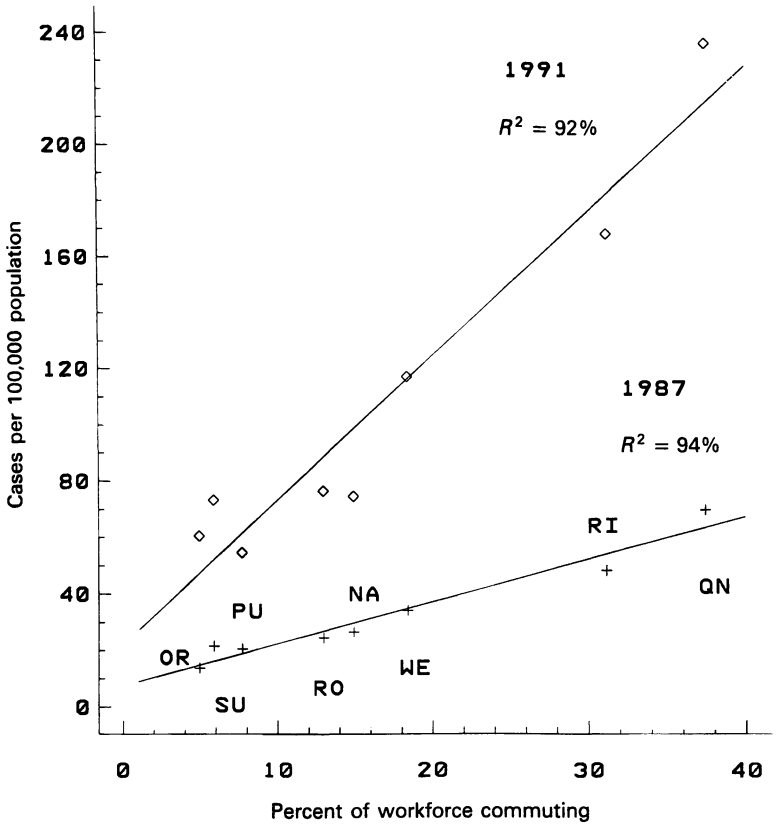

FIG. 2. Cumulative AIDS case rates per 100,000 population for downstate suburban New York counties as a function of the percent of their workforce commuting into Manhattan for 1987 and 1991. The counties include Queens, Richmond, Westchester, Nassau, Rockland, Putnam, Suffolk, and Orange. (*Source:* New York State Department of Health 1988, 1991)

contact, based on daily travel patterns, between those suburban counties and Manhattan. It can be used to construct a probability-space "socio-geographic metric" convoluting both distance and ease of transport (R. Wallace 1993b).

Some 94 percent of variance of cumulative AIDS case rates through 1987 is accounted for by the percent of the county workforce commuting to the central-city epicenter—the commuting field; 92 percent is accounted for through 1991.

The empirical relation suggested by equation (2) is strikingly good for AIDS.

For a suburb surrounded by urban and suburban disease epicenters we would expect the rate of infection per unit population to be determined by a sum of terms like the right-hand side of equation (2) over those epicenters.

Although some local government analysts and academic researchers have produced maps of AIDS covering individual cities or states, detailed regional maps and serious analytic studies of the spatial and temporal spread of the disease within the United States are curiously rare. The earliest reasonably complete analysis seems to be that of Gardner et al. (1989), which examined HIV seropositivity rates in military recruits. They found that, by 1987, mixed hierarchical and expansion diffusion was readily apparent in Florida, Texas, and Northern California. For the East Coast, expansion diffusion was particularly apparent near New York City and within northern New Jersey.

Even now no federal agency, including the Centers for Disease Control and Prevention, either routinely produces detailed maps of the geographic spread of AIDS or other diseases in the United States, or conducts the sophisticated geographic analysis needed to understand the underlying national or even regional spread dynamics. Given the unambiguous implications of figure 2, this failure is difficult to understand.

Peter Gould's analysis of AIDS spread on the West Coast (Gould 1991) and, more recently, his series of national maps published by *Time* (Gould 1992) are among the first detailed maps widely published. They clearly show initial hierarchical diffusion from larger to smaller cities, followed by spatial contagion into the suburbs of those cities. Similarly, Cassetti and Fan (1991) show for Ohio the avalanche through the urban hierarchy, followed by expanding spatial contagion around the central cities.

The recently published *London International Atlas of AIDS*, by Smallman-Raynor, Cliff, and Haggett (1992), provides what is perhaps the most complete summary of the spatial structure of AIDS in the United States and elsewhere, and shows the hierarchical diffusion/spatial contagion pattern of spread to be ubiquitous.

These analyses represent matters at what was still a fairly early stage of AIDS in the United States, when concentrated populations of male homosexuals and intravenous drug users (IVDUs) in the largest central cities were approaching epidemiologic saturation, but before the disease entered a more widespread phase. As the disease affects different populations, and inevitably "changes"—after gay male and IVDU popula-

tions become saturated—to one increasingly transmitted by heterosexual contact, an intensified form of the standard pattern of geographic diffusion may well manifest itself: from large central city to middle-sized city, and from central locus to suburb.

In any event, given the sociogeographic system that produced these characteristic diffusion patterns for the early stages of the AIDS pandemic in the United States, the analysis leading to equation (2) suggests that the rate of spread of an intensified HIV outbreak within large central cities to middle-sized cities, where in total about 25 percent of the population of the United States resides, and to the suburbs of both, where another 50 percent lives, will be strongly determined by the rise in prevalence of the disease within the largest central urban epicenters.

Figure 2 suggests this as likely to be true, even if the more affluent communities, including suburbs, attempt to establish local control programs, because contact with more heavily infected regions, including internal deteriorating suburban minority "enclaves" that become local epicenters, will inevitably result in constant cascades of cases. Indeed, marked changes in sexual behavior within the New York City and San Francisco gay male epicenters occurred only under truly draconian conditions: rampant, unchecked disease spread and HIV seroprevalence levels approaching 50 percent. There are now reports of increasing behavioral lapses in younger gay male populations. Evidence with regard to changing behavior patterns of at-risk heterosexuals is anything but encouraging: Catania et al. (1992) recently found that 31 percent of heterosexuals nationally, and as many as 41 percent in cities with high AIDS prevalence, admit to at least one HIV risk factor. Condom use was miniscule, involving only 17 percent of those with multiple sexual partners and 12.6 percent of those with risky sexual partners.

Similar scenarios, depending in detail on the particular disease and its ecology, can be created for drug-resistant tuberculosis and gonorrhea, and even for the spectrum of treatable diseases that thrive in extreme poverty and social disintegration. For example, a whole literature now exists on the role of geographically focused, socially marginalized "core groups" in maintaining high endemic concentrations of a spectrum of sexually transmitted infection (STI), including drug-resistant gonorrhea (Arya, Reese, and Turner 1984; Potterat et al. 1985; Rice et al. 1991; Rothenberg 1983, 1991; Rothenberg and Potterat 1988; Zenilman et al. 1988).

Gonorrhea remains the most widely reported STI in spite of rapid de-

tection and the relatively simple medical cure of infected individuals. Brandt, who analyzed in *No Magic Bullet* the failure to control gonorrhea and syphilis in the United States, writes:

> Sexual contact is one of a number of ways in which microorganisms are transmitted from human to human. New or altered infectious agents are passed this way; no single medical treatment has proven effective for these infectious organisms. . . . Venereal disease, and indeed all infectious diseases, constitute complex bio-ecological problems in which host, parasite, and a number of social and environmental forces interact. No *single* medical or social intervention can thus adequately address the problem. (1987, 203)

Expanding this view somewhat, we believe that the nexus of sexual, substance-abuse, and related economic and behavioral factors, such as homelessness and sex-for-drugs, which has developed or has been intensified as a result of the public-policy-driven hollowing out of minority communities within the largest U.S. cities over the past two decades, provides fertile ground for the establishment of high prevalences of virulent pathogens. These diseases will, according to long-studied patterns, diffuse from larger to smaller central cities, and from central cities into their surrounding suburbs, at rates dependent on those prevalences.

As we have shown in the argument leading to equation (2), even at the earliest stages of epidemic development, disease rates within a suburb will be proportional to the number of infective individuals within the inner-city epicenter, as modulated by the commuting field between city and suburbs. Continued export of disease from those central-city epicenters is likely to result in establishment of self-sustaining local disease epicenters.

Discussion and Conclusions

The period of rapid suburbanization after World War II has produced a series of extended "urban–suburban complexes" in which some three-quarters of the population of the United States now resides. The standard central city pattern of disjoint, congregated and segregated, rich and poor neighborhoods has now become intensified and spread over much larger areas, while political fragmentation has made comprehensive public health management of this enlarged but coupled ecosystem

difficult (Johnston 1982). Since 1968 the hollowing-out process affecting central cities and its resulting social disintegration created vast incubators for contagious diseases. Some of these, like tuberculosis, had been declining since the successes of the Great Urban Reform Movement of the late ninteenth and early twentieth centuries.

Massey and Denton (1992) show in some detail that minority suburban populations in the north are "hypersegregated," as well as "replicat[ing] the problems of the inner city." The work cited above by Rothenberg (1983, 1991), Rothenberg and Potterat (1988), and Potterat et al. (1985) suggests that *hypersegregation in and of itself* is a primary risk factor for the establishment of high-prevalence "core groups" for STIs.

For example, Potterat et al. (1985) found that gonorrhea in Colorado Springs, a predominantly white, middle-class city, was primarily a disease of poor, minority military personnel, who congregated in six of the city's 96 nightspots. Within this tightly self-interactive and sociogeographically concentrated "sexual cloud" the risk of gonorrhea infection was some 300 times greater than within the general population. In addition, some 45 percent of case-contact pairs in this relatively stable subpopulation had known each other for two months or more before sexual contact, and nearly a fifth for two years or more before contact.

Sexual interaction leading to the high prevalence of disease was not simply a matter of the rapid temporal cycling of sexual contact, but the consequence of a hypersegregated pattern of social interaction. The high prevalence of infection within this population serves as a reservoir for the constant reseeding of the larger city, much as does the high prevalence of tuberculosis within other disadvantaged subpopulations (Youmans 1979).

One of the authors (R. Wallace 1991b,c; R. Wallace and Fullilove 1991) has provided a theoretical context for understanding the role of hypersegregation in the rapid establishment of a newly introduced STI by examining the spread of behaviorally transmitted disease on tightly interwoven, strongly geographically concentrated social networks—what they characterize as "low dimensional sociogeographic networks." Wallace (1991c) found that the consonance of hypersegregation and population mobility associated with social disintegration could spread disease very rapidly indeed, quickly resulting in the establishment of new epicenters.

In the context of the standard, indeed canonical, patterns of geographic and social diffusion of disease from intensifying epicenters, the challenges to public health in the suburbs will increase and become less

amenable to even the most draconian of purely "medical" interventions within the suburbs themselves. Even "regional coordination" of "service provision" among and within the suburbs will fail to stem the rising incidence of disease, which will spread from physically, socially, and economically deteriorating central cities and from distressed local minority enclaves.

The political fragmentation of the extended urbanized regions has obscured the fact that, from the viewpoint of the health status of populations within those regions, they remain coupled human ecosystems recognizably similar to the earlier, more centrally focused systems upon which they were built. This similarity includes the spatially dispersed patterns of congregation and segregation of population, which were always found within the earlier, more concentrated, urban system.

When New York City pursues a policy of "planned shrinkage," which has the impact of not putting out fires and leaving garbage uncollected in crowded minority neighborhoods (Duryea 1978; Roberts 1991; Wallace and Wallace 1990), when Detroit and Newark do not have the money to put out their fires and collect their garbage, when economic and social marginalization of inner-city populations and suburban minority "enclaves" exacerbates poverty, when policies of political fragmentation prevent effective regional governance, then affluent and politically powerful suburbs, only a few dozen miles from disintegrating city centers, must expect to be drawn into the epidemics that disintegration engenders.

Developing immediate, temporary measures to stabilize disease epicenters, or even designing a full-scale reform program, will require deep understanding of the extended urban ecosystems themselves, to create mutually reinforcing and highly cost-effective "synergistic" programs whose impacts amplify each other.

Our own work on New York City suggests that proper use of administrative data sets to understand the ecology of the larger urban–suburban system may indeed permit identification of mutually reinforcing, and possibly counterintuitive, regional strategies, which may greatly amplify their impact.

For example, simply stopping the contagious hollowing-out process in the central city by providing adequate levels of relatively inexpensive "hard" municipal services like fire extinguishment, sanitation, and building code enforcement would, in the current context, be a considerable advance. The failure to deliver such fairly inexpensive hard services,

we have shown, creates a growing avalanche of demand for very expensive "soft" services, such as health care, remedial education, welfare, shelters for the homeless, criminal justice, and the like.

Greatly improved public health was brought to a vast population of the United States at the turn of the last century through an integrated series of programs, initiatives, and policies that ameliorated both living and working conditions in urban areas. Maintaining, and indeed reclaiming, the gains of that era at the turn of this century will require the physical and social rehabilitation and stabilization of minority communities within both central cities and their suburbs, a regional problem requiring regional address.

References

Abler, R., J. Adams, and P. Gould. 1971. *Spatial Organization: The Geographer's View of the World.* Englewood Cliffs, N.J.: Prentice-Hall.

Arya, O., E. Rees, and C. Turner. 1984. Epidemiology of Penicillinase Producing Neisseria Gonorrhoeae in Liverpool from 1977 to 1982. *Journal of Infectious Diseases* 8:70–8.

Bailey, N.T.J. 1975. *The Mathematical Theory of Infectious Diseases.* New York: Hafner Press.

Belcastro, P., and T. Nicholson. 1982. Chemical Foreplay among Black and White Students. *Journal of Drug Education* 12:373–83.

Brandt, A. 1987. *No Magic Bullet; A Social History of Venereal Disease in the United States since 1880.* New York: Oxford University Press.

Bursik, R. 1986. Ecological Stability and the Dynamics of Delinquency. In *Communities and Crime*, eds. A. Reiss and M. Tonry, 35–66. Chicago: University of Chicago Press.

Cassel, J. 1974. Psychosocial Processes and "Stress": Theoretical Formulation. *International Journal of Health Services* 4:471–80.

Cassetti, E., and C. Fan. 1991. The Spatial Spread of the AIDS Epidemic in Ohio: Empirical Analyses Using the Expansion Method. *Environment and Planning A* 23:1589–96.

Catania, J., T. Coates, R. Stall, et al. 1992. Prevalence of AIDS-related Risk Factors and Condom Use in the United States. *Science* 258: 1101–6.

Cliff, A., P. Haggett, J. Ord, and G. Versey. 1981. *Spatial Diffusion: An Historical Geography of Epidemics in an Island Community.* London: Cambridge University Press.

Cohen, S., and T. Wills. 1985. Stress, Social Support and the Buffering Hypothesis. *Psychological Bulletin* 98:310–21.

Collins, J. 1981. Alcohol Use and Criminal Behavior: An Empirical, Theoretical and Methodological Overview. In *Drinking and Crime*, ed. J. Collins. New York: Guilford Press.

Cowley, G. 1993. The Future of AIDS: New Research Suggests HIV Is Not a New Virus but an Old One. *Newsweek* (March 22):47–51.

Crutchfield, R., M. Geerken, and W. Gove. 1982. Crime Rate and Social Integration: The Impacts of Metropolitan Mobility. *Criminology* 20:467–78.

Dear, M. 1976. Abandoned Housing. In *Urban Policy Making and Metropolitan Development*, ed. J. Adams. Cambridge: Ballanger.

Duryea, P. 1978. Press release dated Friday, January 27. Albany: Office of the New York State Assembly Republican Leader.

Ewald, P. 1991. Transmission Modes and the Evolution of Virulence with Special Reference to Cholera, Influenza and AIDS. *Human Nature* 2:1–30.

———. 1993. The Evolution of Virulence. *Scientific American* 268(4):86–93.

Fried, M. 1963. Grieving for a Lost Home. In *The Urban Condition*, ed. L. Duhl, 151–71. New York: Simon and Schuster.

———. 1965. Transitional Functions of Working Class Communities: Implications for Forced Relocation. In *Mobility and Mental Health*, ed. M. Kanor, 65–78. Springfield, Ill.: Charles C Thomas.

Gardner, L., J. Brundage, D. Burke, J. McNeil, R. Visintine, and R. Miller. 1989. Spatial Diffusion of the Human Immunodeficiency Virus Infection Epidemic in the United States, 1985–87. *Annals of the American Association of Geography* 79:25–34.

Gould, P. 1991. Modeling the Geographic Diffusion of the AIDS Epidemic. *Science* 251:1022.

———. 1992. Inexorable March. *Time* 20 (August 31):20.

———. 1993. *The Slow Plague*. London: Blackwell.

Hinkle, L., and W. Loring. 1977. *The Effect of the Man-Made Environment on Health and Behavior*. DHEW pub. no. (CDC) 77-8318. Atlanta, Ga.: Centers for Disease Control.

House, J., K. Landis, and D. Umberson. 1988. Social Relationships and Health. *Science* 241:540–5.

Johnston, R. 1982. Voice as a Strategy in Locational Conflict: The Fourteenth Amendment and Residential Separation in the United States. In *Conflict, Politics and the Urban Scene*, eds. K. Cox and R. Johnston, 1–22. New York: St. Martin's Press.

Kane, G. 1981. *Inner City Alcoholism: An Ecological Analysis and Cross Cultural Study*. New York: Human Sciences Press.

Kasl, S. 1977. The Effects of the Residential Environment on Health and Behavior. In *The Effect of the Man-Made Environment on Health and Behavior*, eds. L. Hinkle and W. Loring, 65–128. Atlanta, Ga.: Centers for Disease Control.

Key, W. 1967. *When People Are Forced to Move*. Topeka, Kans.: Menninger Foundation.

Leighton, A. 1959. *My Name Is Legion: Foundations for a Theory of Man in Relation to Culture*. New York: Basic Books.

Leighton, D., J. Harding, D. Macklin, A. Macmillan, and A. Leighton. 1963. *The Character of the Danger*. New York: Basic Books.

Lin, N., and A. Dean. 1984. Social Support and Depression: A Panel Study. *Social Psychiatry* 19:83–94.

Lin, N., and W. Ensel. 1984. Depression-Mobility and Its Social Etiology: The Role of Life Events and Social Support. *Journal of Health and Social Behavior* 25:176–88.

Massey, D., and N. Denton. 1992. *American Apartheid: Segregation and the Making of the Underclass*. Cambridge: Harvard University Press.

McCord, C., and H. Freeman. 1990. Excess Mortality in Harlem. *New England Journal of Medicine* 322:173–9.

New York State Department of Health. 1988. *AIDS in New York State*. Albany.

———. 1991. *AIDS in New York State*. Albany.

Niebanck, P. 1968. *Relocation in Urban Planning: From Obstacle to Opportunity*. Philadelphia: University of Pennsylvania Press.

Odland, J. 1983. Conditions for Stability and Instability in Spatial Diffusion. *Modeling and Simulation* 14:627–32.

Odland, J., and B. Balzer. 1979. Localized Externalities, Contagious Processes and the Deterioration of Urban Housing: An Empirical Analysis. *Socio-Economic Planning Science* 13:87–93.

Odland, J., and R. Barff. 1982. A Statistical Model for the Development of Spatial Pattern: Application to the Spread of Housing Deterioration. *Geographical Analysis* 14:326–34.

Osgood, D., L. Johnston, P. O'Malley, and J. Bachman. 1988. The Generality of Deviance in Late Adolescence and Early Adulthood. *American Sociological Review* 53:81–7.

Potterat, J., R. Rothenberg, D. Woodhouse, J. Muth, and J. Fogle. 1985. Gonorrhea as a Social Disease. *Sexually Transmitted Diseases* 12:25–32.

Rabkin, J., and E. Struening. 1976. Life Events, Stress and Illness. *Science* 194:1013–20.

Rice, R., P. Roberts, H. Handsfield, and K. Holmes. 1991. Sociodemographic Distribution of Gonorrhea Incidence: Implications for Pre-

vention and Behavioral Research. *American Journal of Public Health* 81:1252–7.

Roberts, S. 1991. A Critical Evaluation of the City Life Cycle Idea. *Urban Geography* 12:431–43.

Rothenberg, R. 1983. The Geography of Gonorrhea: Empirical Demonstration of Core Group Transmission. *American Journal of Epidemiology* 117:688–93.

———. 1991. Those other STD's. *American Journal of Public Health* 81:1250–1.

Rothenberg, R., and J. Potterat. 1988. Temporal and Social Aspects of Gonorrhea Transmission: The Force of Infectivity. *Sexually Transmitted Diseases* 15:88–95.

Sampson, R., and W. Groves. 1989. Community Structure and Crime: Testing Social Disorganization Theory. *American Journal of Sociology* 94:774–802.

Schuerman, L., and S. Kobrin. 1986. Community Careers in Crime. In *Communities and Crime*, eds. A. Reiss and M. Tonry, 67–100. Chicago: University of Chicago Press.

Skogan, W. 1986a. Fear of Crime and Neighborhood Change. In *Communities and Crime*, eds. A. Reiss and M. Tonry, 203–30. Chicago: University of Chicago Press.

———. 1986b. Disorder, Crime and Community Decline. In *Communities and Crime Reduction*, eds. J. Hope and E. Shaw, 121–42. London: Her Majesty's Stationery Office.

———. 1990. *Disorder and Decline: Crime and the Spiral of Decay in American Neighborhoods*. New York: Free Press.

Smallman-Raynor, M., and A. Cliff. 1991. Civil War and the Spread of AIDS in Central Africa. *Epidemiology and Infection* 107:69–79.

Smallman-Raynor, M., A. Cliff, and P. Haggett. 1992. *London International Atlas of AIDS*. London: Blackwell.

South, S. 1987. Metropolitan Migration and Social Problems. *Social Science Quarterly* 68:3–18.

Struening, E., R. Wallace, and R. Moore. Housing Conditions and the Quality of Children at Birth. *Bulletin of the New York Academy of Medicine* 66:463–9.

Wallace, D. 1990. Roots of Increased Health Care Inequality in New York. *Social Science and Medicine* 31:1219–28.

Wallace, D., and R. Wallace. 1993. Community Destruction and the Resurgence of Tuberculosis in New York City. (In press)

Wallace, R. 1978. Contagion and Incubation in New York City Structural Fires, 1964–1978. *Human Ecology* 6:423–33.

———. 1981. Fire Service Productivity and the New York City Fire Crisis: 1968–1979. *Human Ecology* 9:433–63.

———. 1982. The New York City Fire Epidemic as a Toxic Phenomenon. *International Archives of Occupational and Environmental Health* 50:33–50.

———. 1988. A Synergism of Plagues: "Planned Shrinkage," Contagious Housing Destruction and AIDS in the Bronx. *Environmental Research* 47:1–33.

———. 1989. "Homelessness," Contagious Destruction of Housing, and Municipal Service Cuts in New York City: 1. Demographics of a Housing Deficit. *Environment and Planning A* 21:1585–93.

———. 1990a. "Homelessness," Contagious Destruction of Housing, and Municipal Service Cuts in New York City: 2. Dynamics of a Housing Famine. *Environment and Planning A* 22:5–15.

———. 1990b. Urban Desertification, Public Health and Public Order: "Planned Shrinkage," Violent Death, Substance Abuse and AIDS in the Bronx. *Social Science and Medicine* 31:801–13.

———. 1991a. Expanding Coupled Shock Fronts of Urban Decay and Criminal Behavior: How U.S. Cities are Becoming "Hollowed Out." *Journal of Quantitative Criminology* 7:333–47.

———. 1991b. Traveling Waves of HIV Infection on a Low Dimensional "Socio-Geographic" Network. *Social Science and Medicine* 32:847–53.

———. 1991c. Social Disintegration and the Spread of AIDS: Thresholds for Propagation along Sociogeographic Networks. *Social Science and Medicine* 33:1155–64.

———. 1993a. Recurrent Collapse of Fire Service in New York City: The Failure of a Paramilitary System as a Phase Change. *Environment and Planning A* 25:233–343.

———. 1993b. A Fractal Model of HIV Transmission on Complex Sociogeographic Networks: Towards Analysis of Large Data Sets. *Environment and Planning A* 25:137–45.

———. 1993c. Social Disintegration and the Spread of AIDS, Part 2: Meltdown of Sociogeographic Structure in Urban Minority Communities. *Social Science and Medicine* 37:887–96.

———. 1993d. Inner City Disease and the Public Health of the Suburbs: The Sociogeographic Dispersion of Point-Source Infection. *Environment and Planning A*. (In press)

Wallace, R., and M. Fullilove. 1991. AIDS Deaths in the Bronx 1983–1988: Spatiotemporal Analysis from a Sociogeographic Perspective. *Environment and Planning A* 23:1701–20.

Wallace, R., M. Fullilove, and D. Wallace. 1992. Family Systems and De-Urbanization: Implications for Substance Abuse. In *Substance Abuse, A Comprehensive Textbook*, eds. J. Lowinson, P. Ruiz, and R. Millman, 944–55. Baltimore: Williams and Wilkins.

Wallace, R., and D. Wallace. 1977. *Studies on the Collapse of Fire Ser-*

vice in New York City, 1972–1976: The Impact of Pseudoscience in Public Policy. Washington: University Press of America.

———. 1983. Urban Fire as an Unstabilized Parasite: The 1976–1978 Outbreak in Bushwick, Brooklyn. *Environment and Planning A* 15:207–31.

———. 1990. Origins of Public Health Collapse in New York City: The Dynamics of Planned Shrinkage, Contagious Urban Decay and Social Disintegration. *Bulletin of the New York Academy of Medicine* 66:391–443.

———. 1991. Contagious Urban Decay and the Collapse of Public Health. *Health/PAC Bulletin* 13:8–16.

Yoon, C. 1993. What Might Cause Parasites to Become More Virulent. *Science* 259:1402.

Youmans, G. 1979. *Tuberculosis*. Philadelphia: Saunders.

Zabin, L., J. Hardy, E. Smith, and M. Hirsch. 1986. Substance Use and its Relation to Sexual Activity among Inner-City Adolescents. *Journal of Adolescent Health Care* 7:320–30.

Zenilman, J., M. Bonner, K. Sharp, J. Rabb, and E. Alexander. 1988. Penicillinase-producing Neisseria Gonorrhoeae in Dade County, Florida: Evidence of Core Group Transmission and the Impact of Illicit Antibiotics. *Sexually Transmitted Diseases* 18:45–56.

Acknowledgments: The authors thank Drs. P. Ewald, M. Fullilove, R. Fullilove, and P. Gould for fruitful discussions, and we are grateful to four anonymous reviewers for extensive and detailed comments useful in revision.

Address correspondence to: Rodrick Wallace, PISCS Inc., 549 W. 123 St., New York, NY, 10027.