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A failure of resilience: Estimating response of New York City's public health ecosystem to sudden disaster

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Abstract

Adapting methodology from resilience theory in ecology, we develop an empirical model of the response of the New York City public health ecosystem to sudden disaster. Contrary to cultural expectation, 'good' and 'bad' neighborhoods—starkly differentiated by public health status reflecting longstanding economic and racial segregation—respond similarly to challenge. This suggests that the difference in health between neighborhoods is primarily predicated on the extent to which they have been, and continue to be, exposed to differing patterns of stressors and affordances, rather than to any difference in underlying socio-economic vulnerability. Paradoxically, then, these urban neighborhoods constitute a single, highly interdependent, health ecosystem, despite substantial socioeconomic and racial segregation.

Keywords: Ecosystem; Epidemiology; HIV; Health; Homicide; Resilience

1. Introduction

Many US cities have substantial spatial segregation along racial/ethnic and socioeconomic lines; New York city is no exception and it is in fact one of the most segregated cities in the US (Massey and Denton, 1993; Charles, 2003). In addition to this spatial segregation, there is substantial concentrated disadvantage in many of the city's 'bad' neighborhoods. Neighborhoods that have higher crime rates, worse physical infrastructure, and so forth, also tend to have higher rates of morbidity and mortality (e.g. Wallace, 1990; McCord and Freeman, 1990). Given both this concentrated disadvantage, and the segregation between 'good' and 'bad' neighborhoods in the City, it is generally assumed that neighborhoods characterized by many disadvantages (low SES, decaying physical infrastructure) are also the most susceptible to worsening health in the event of unanticipated external stressors (Galea et al., 2004).

There is some evidence that neighborhoods characterized by concentrated disadvantage may also have underlying socioeconomic vulnerability that is associated with the poor health indicators that typically characterize these neighborhoods. This reasoning is grounded in well-established and substantiated research that has shown, in many contexts, that socioeconomic conditions are important and fundamental determinants of human

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health. However, this reasoning then suggests implicitly, that neighborhoods that are characterized by concentrated disadvantage ('bad' neighborhoods in typical formulation in the media and in public discourse), are particularly vulnerable to the consequences of intermittent stressors, including for example disasters, economic downturns, or discriminatory policies. The corollary assumption has been that disaster or disastrous policies, even when directly affecting better neighborhoods, will have inherently less impact on 'good' than they will on 'bad' neighborhoods.

An ecosystem approach to the same problem offers a different perspective. Ecosystem theory suggests that within urban areas, neighborhoods are far more interrelated than they are independent. Since development of the railroads in the late 19th century, cities and their underlying subcommunities have become increasingly interconnected, linked by travel patterns into a sequence of ever more tightly interwoven hierarchical structures (e.g. Abler et al., 1971). Therefore, we hypothesized that despite the racial/ethnic segregation that characterizes urban neighborhoods, in the event of unanticipated external stressors, both neighborhoods which are characterized by salutary or by detrimental factors will manifest substantial vulnerability and that the response of advantaged neighborhoods to such stressors is entrained into the same underlying structure—that, ideology aside, 'good' and 'bad' neighborhoods are, in fact, interlinked parts of a consistent whole (e.g. Memmi, 1969; Fanon, 1966).

In order to assess this hypothesis, we model how patterns of illness and violence in 'good' and 'bad' neighborhoods of New York City are likely to respond to sudden changes, including, but not limited to, those initiated by overt disaster or by disastrous economic or public policy.

2. Methods

Public policy in New York City, particularly allocation of essential services such as sanitation, housing inspection, police, and the like, is officially determined at the level of the Community District (CD), the 59 administrative divisions which have come to reflect the political, economic, social, and ethnic composition of the city. Public resource allocation has thus made this geographic structure 'real'.

As in Wallace et al. (2004) and Galea et al. (2005), we focused our initial study on the population rate of HIV/AIDS deaths for 2000, at the CD level, since that is a highly sensitive composite of infection mode, underlying population health status, availability of antiretroviral drug treatments, and availability of health care.

A stepwise regression model on a spectrum of publicly available health variates found the HIV death rate best predicted, at the citywide CD level, by homicide, liver, and drug death rates, also for 2000. As described by Wallace et al. (2004), the four death rates—HIV/AIDS, homicide, liver and drug—constitute a related 'disease guild' in the ecosystem sense. These rates were standardized by dividing them by the citywide medians, and a composite index created by summing the four normalized variates for each CD. The city was then partitioned into two sectors, those with composite index less than or greater than 4. See Fig. 1.

The data set, including ICD-9 (1990–1998) and ICD-10 codes (1999–2000), is described in great detail by Galea et al. (2005). The data subset used in this paper is available on request from the senior author (rdwall@ix.netcom.com).

As Table 1 shows, the two sectors differ markedly on the means of many socioeconomic and public health variables, in particular income, poverty, and 'racial' composition.

We next expanded the citywide CD stepwise regression model of Wallace et al. (2004) for HIV/AIDS death rate to include a number of economic and other census and municipal service variates as well as the health variates, all for the year 2000, finding the log of the HIV death rate best predicted by a model which included the logs, respectively, of structural (building) fires per unit area, homicide death rate and liver death rate. Citywide, this model accounted for 85% (adjusted) of the variance in the log of the HIV death rate. See Table 2.

The log of the number of fires per unit area was itself best predicted, under stepwise regression ($R^2 = 74\%$), by a model including the log of the unemployment rate, the log of the fraction of the population in the lowest two income divisions (out of 10 possible), and the median income. This represents a very powerful economic structuring which, in its positive relation to median income, mirrors the finding of Wallace et al. (2004) that gentrifying neighborhoods, having both high median income and high percent of those in poverty, were subject to higher rates of health problems. See Table 3.

Building on an alternative formulation of Ives stationary time series formulation (Ives, 1995; more

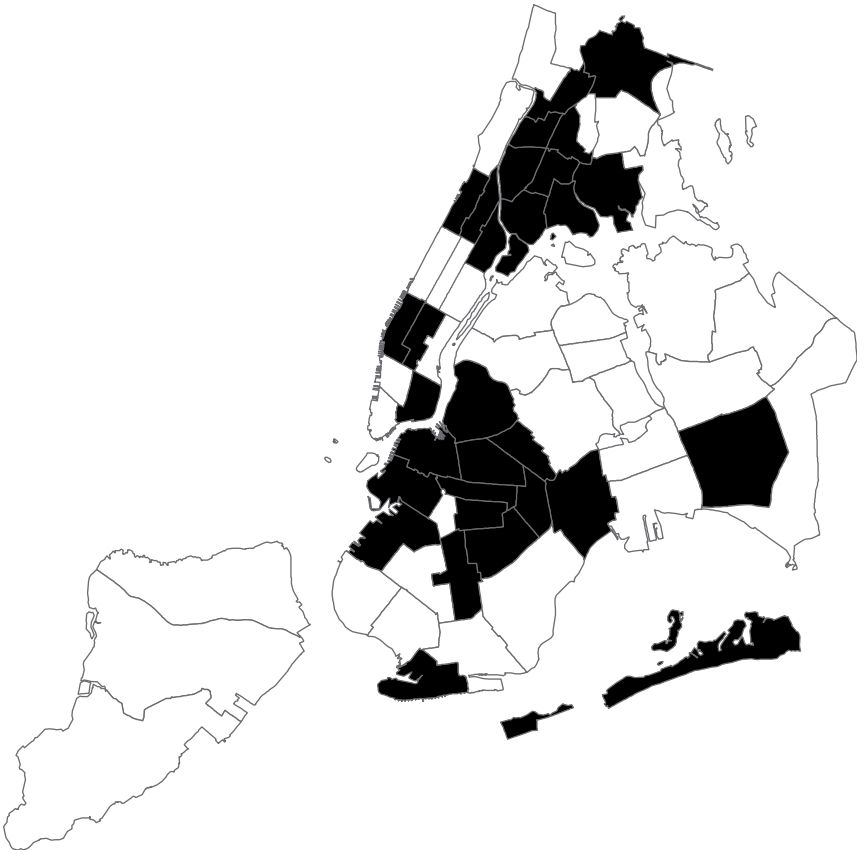

Fig. 1. Map of the 59 New York City Community Districts. Filled areas have composite index of HIV/AIDS, homicide, drug, and liver death rates for 2000 greater than citywide median.

Table 1
2000 Census demographics of more and less healthy sectors of Fig. 1

Percent white	50.5	17.1
Precent black	11.2	38.9
Precent below poverty	14.0	30.0
Median income ($)	47638	29482

details are found in Wallace and Wallace, 2000) we conducted a round-robin of regression models relating the logs of HIV death rate, fires per unit area, homicide death rate and liver disease death rate for the 'good' and 'bad' sections of the city shown in Fig. 1. That is, for each of the two sectors, we made four regression models, with each variate serving, in turn, as independent, and dependent. The result is a statistical model which, as a matrix equation, has, for each sector, the form

$$X = \mathbf{B}X + b + \varepsilon. \quad (1)$$

Table 2
Citywide multiple regression analysis of 2000 HIV/AIDS death rate

Parameter	Estimate	S.E.	*T*	*P*
Dependent variable: Log(HIV/AIDS)				
Constant	−1.686	0.302	−5.578	0.000
Log(fires/landarea)	0.523	0.067	7.785	0.000
Log(homicide)	0.478	0.080	5.973	0.0000
Log(liver)	0.608	0.110	5.551	0.0000
R-SQ.(ADJ).	85.9%			

X is a four-dimensional column vector representing the logs of HIV death rate, fires per unit area, homicide death rate, and liver disease death rate, $\mathbf{B}$ is a 4×4 matrix of regression coefficients *having zeros on the diagonal*, b is the column vector of regression constants, and ε is a column vector of normally distributed 'errors' unaccounted for by regression.

Table 3
Citywide multiple regression analysis of 2000 structural fires/area

Parameter	Estimate	S.E.	T	P
Dependent variable: Log(fires/landarea)				
Constant	12.92	0.685	18.87	0.0000
Log(unemp/totalpop)	1.457	0.277	5.258	0.0000
Log(inc1 + inc2)/totalpop)	2.176	0.298	7.311	0.0000
Medinc/10000	0.4614	0.0715	6.448	0.0000
R-SQ.(ADJ)	74.0%			

Again, two such models were made, one each for the two sectors of Fig. 1.

Estimating the effect of 'perturbations' is equivalent to solving this equation in terms of ε. Taking **I** to be the four-dimensional unit matrix—ones on the diagonal, zeros elsewhere— the argument proceeds as follows:

$$(\mathbf{I}-\mathbf{B})X = b + \varepsilon,$$

$$X = (\mathbf{I}-\mathbf{B})^{-1}b + (\mathbf{I}-\mathbf{B})^{-1}\varepsilon$$

so that

$$\delta X \equiv X - (\mathbf{I}-\mathbf{B})^{-1}b = (\mathbf{I}-\mathbf{B})^{-1}\varepsilon$$

or

$$\delta X = (\mathbf{I}-\mathbf{B})^{-1}\varepsilon \equiv \mathbf{C}\varepsilon. \quad (2)$$

Since **B** is constructed by regression, it is fairly easy to show (e.g. Wallace et al., 1998, 2000) that **C** has eigenvalues of the form $1/(1-\lambda)$ where λ is an eigenvalue of **B**, with at most one $\lambda \to 1$. The eigenvectors of **B** (which are the same as those of **C**) are not, however, generally orthogonal.

Assuming simple regularities in the time occurrence of the 'perturbations' ε, in particular fixed mean and standard deviation $\sigma(\varepsilon)$, we obtain, for the 'excited eigenmodes' of **C**, the relation

$$\sigma(\delta X) = \frac{\sigma(\varepsilon)}{|1-\lambda|}. \quad (3)$$

Systems for which $\lambda \to 1$ would not be characterized as 'resilient' under this analysis, since their response to perturbation would be much greater than the perturbation itself: $\lim_{\lambda\to 1} 1/(1-\lambda) = \infty$.

Note that, using the **C** matrices, it is possible to predict the effects of particular forms of perturbation—i.e. of the vector ε—on the two systems. For example, unit changes in log(fires/area) would be correspond to the column vector [0, 1, 0, 0]. Multiplying this vector by the **C** matrices gives the effect of this change on the system, and selects out the second column of each, predicting the response in terms of changes in the four variates, including the effects of feedback on fire itself.

3. Results

The round-robin of regressions relating the logs of HIV death rate, fires per unit area, homicide rate, and liver disease death rate for the year 2000 over the two sectors of Fig. 1 gives the results of Table 4, expressed in terms of the **B** matrices. Table 5 shows the two **C** matrices, and their normalized principal eigenmodes—corresponding to the largest amplification factors—as well as the amplification factors themselves, along with the inner product of those eigenmodes.

The maximum amplification factors, $1/(1-\lambda)$, are virtually identical: 5.04 for the less healthy and 5.81 for the more healthy sectors. Thus, neither is a 'resilient' ecosystem, whose requirement is that $1/(1-\lambda) \approx 1$. The normalized principal eigenmodes corresponding to these amplification factors have an inner product of 0.94, which means that they are very highly parallel—virtually identical.

Like the principal eigenmodes, the **C**-matrix method described at the end of the methods section for estimating the effects of perturbation finds the second columns of both sector's matrices to be very similar, a likewise counterintuitive and striking result which implies that increase in fires per unit area has similar impact in both regions.

That is, according this statistical model, the two sections of Fig. 1, which differ remarkably on virtually all health and demographic variates, respond almost identically to increases in fires per unit area, in terms of increases in HIV death rate,

Table 4
B-matrices of round-robin regressions for the two sectors of Fig. 1

Healthier sector				
HIV/AIDS	0	0.472027	0.506685	0.466231
Fires/area	1.04797	0	−0.38925	−0.79205
Homicide	0.850406	−0.206527	0	−.196006
Liver	0.405754	−0.310479	−0.133091	0
Less healthy sector				
HIV/AIDS	0	0.495232	0.095836	0.411038
Fires/area	1.07736	0	0.112734	−0.385774
Homicide	0.257777	0.139385	0	−0.145897
Liver	0.660918	−0.285132	−0.0872159	0

Table 5
C-matrices and principal eigendecomposition corresponding to Table 4

Healthier sector				
C =	3.636872	1.332679	1.271997	0.390755
	2.565171	2.345192	0.487634	−0.757126
	2.494991	0.704069	1.960665	0.221282
	0.347183	−0.281099	0.103771	1.364172
Normalized principal eigenvector				
[0.650073, 0.549519, 0.52406, 0.028279]				
Eigenvalue/amplification factor				
5.80				
Less healthy sector				
	2.966476	1.336984	0.378473	0.648343
	2.908562	2.469403	0.542854	0.163697
	1.018003	0.671189	1.174257	−0.011810
	1.042487	0.120993	−0.007059	1.382856
[0.586009, 0.735408, 0.281606, 0.190964]				
5.04				
Eigenvector inner product: 0.94				

fires per unit area itself, and homicide rate. The liver disease death rate seems independent of these three factors in both sectors, since the coefficients—the last entries of the second columns of the two C-matrices—are relatively small for both.

4. Discussion

Our primary finding is that, in response to unanticipated external stressors—sudden perturbations—the variability in health outcomes exhibited by 'good' neighborhoods of New York City is comparable to the variability exhibited by 'bad' neighborhoods. The city's neighborhoods are, according to our results, highly interconnected, and their response to stressors is dependent, not simply on their own resources, but also on the resources available to the other neighborhoods with which they are closely interlinked. In sum, New York City, in terms of its public health ecosystem, is a single functional unit despite substantial socio-economic and racial/ethnic segregation (Massey and Denton, 1993).

We classified New York City's neighborhoods into two sectors, representing high and low values of a composite index of median-normalized HIV, homicide, liver, and drug death rates (Wallace et al., 2004). Using a statistical model adapted from ecosystem theory to study the predicted effect of sudden changes—perturbations—we found, unexpectedly, that each sector had virtually identical modeled responses to perturbation. At the level of the Community District, by which many essential services are allocated, New York City is a single, highly interdependent ecosystem, whose linkages appear to transcend the city's racial and socio-economic segregation. The more healthy neighborhoods of Fig. 1 are, in their response to sudden perturbation, no more stable than the less healthy neighborhoods, and both are unstable.

Although this finding was unexpected, it is not incompatible with the previously demonstrated importance of specific factors that may influence population heath across an urban area. For example, the strong role of fires per unit area in the model, and the past experience of contagious urban decay modulated by decreased fire service levels as driving a literal fire epidemic in the city (Wallace and Wallace, 1998, 1990; Wallace, 1990), suggests that levels of fire-related municipal services are particularly important for public health and order in New York City as a whole. It is these services, interacting with the synergism between poverty and housing overcrowding (Wallace, 1990), which determine fire occurrence per unit area. The process we suggest in this paper is not a static one. Dear (1976) and Odland et al. (1979, 1982, 1983) first explored the general role of contagious process in urban decay, where abandoned buildings seed for further abandonment in a self-dynamic feedback loop. Wallace (1978, 1981) then showed that, within New York City, contagious urban decay itself was dependent on, and convoluted with, fire occurrence.

Our results add to a growing body of evidence that, in contrast to powerful cultural assumption, concentration of 'bad things' in poor urban and suburban minority neighborhoods does not, in fact, represent their containment, but rather serves as an engine of systemwide instability (e.g. Charles, 2003; Wallace and Wallace, 1997; Memmi, 1969; Fanon, 1966). In addition, resources unavailable to less healthy neighborhoods—for example closed fire companies or other lowered housing-related municipal services—are, in fact, also unavailable to more healthy neighborhoods in the event of need: essential municipal services, in particular emergency services, must, in exact analogy to a flood control system, be designed to meet the challenges of the '100 year storm', not just the time averaged flow of a river, and holes in the dykes protecting 'bad'

neighborhoods mean there will be a good deal of water in the 'good' ones as well.

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