Variability and Vulnerability at the Ecological Level: Implications for Understanding the Social Determinants of Health

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Objectives. We examined variability in disease rates to gain understanding of the complex interactions between contextual socioeconomic factors and health.

Methods. We compared mortality rates between New York and California counties in the lowest and highest quartiles of socioeconomic status (SES), assessed rate variability between counties for various outcomes, and examined correlations between outcomes' sensitivity to SES and their variability.

Results. Outcomes with mortality rates that differed most by county SES were among those whose variability across counties was high (e.g., AIDS, homicide, cirrhosis). Lower-SES counties manifested greater variability among outcome measures.

Conclusions. Differences in health outcome variability reflect differences in SES impact on health. Health variability at the ecological level might reflect the impact of stressors on vulnerable populations. (*Am J Public Health.* 2002;92:1768–1772)

Recent research into the role of the social environment as a determinant of individual health has reinvigorated inquiry into the relation between context and health.[1–3] Questions regarding mechanism follow naturally from this work. A key aspect of many contextual variables is that they cannot be measured at the individual level; they are essentially group, or ecological, characteristics. Contextual factors likely interact with the large number of individual characteristics that determine health and illness, such as genetics, behavioral choices, and access to medical care.

Analyses of community factors attempt to elucidate how context affects the health of individuals.[4] Although multilevel analysis allows statistical determination of the relative effect of individual and community factors,[5] the manner in which these measures exert their effects on public health is likely to be more complex than is suggested by generalized multilevel linear models.[6,7] A more accurate understanding of the interplay between individuals and their environments requires construction of models that take into account our knowledge of interactions on various levels, contextual and otherwise, and the fact that system components are interconnected and likely display feedback loops.[8–10]

One approach to understanding complex systems is to examine variability among their components. *Variability* refers to the extent to which a characteristic of a complex system (e.g., heart rate or stock prices) changes over time or space. Variability in a complex system might reflect the effect of external influences ("stressors") through their interaction with the system's homeostatic mechanisms.[11]

Most evaluations of variability in complex physiological systems have been done in the context of individual clinical characteristics. For example, a decrease in heart rate variability has been shown to predict mortality after myocardial infarction.[12] For public health surveillance or for epidemiological analysis, variability in population health or its determinants may be a more informative characteristic than the absolute level of particular components. Similarly, for policy or program evaluation, variability might be a useful measure of the relative effects of different interventions.

We studied the relation between contextual effects and population health outcomes by examining mortality rates associated with several conditions in counties in New York and California. We hypothesized that, first, certain diseases or health outcomes (e.g., traumatic events or communicable diseases) are more sensitive to population socioeconomic factors than are others, reflecting the degree to which those outcomes are avoidable or preventable. Second, the rates of the outcomes that are most sensitive to socioeconomic factors also vary the most among counties, reflecting the wide distribution of responses to the stressors to which populations are exposed.

METHODS

The unit of analysis for this study was the county. We selected New York and California because they are among the most populous states in the United States and their county mortality rates are based on relatively large denominators. We excluded from the analysis any county in either state with a population of less than 15 000 persons.

Data

We used New York State Department of Health and California Department of Health Services data to obtain age-adjusted mortality rates in each county for the various outcomes.[13,14] Table 1 presents the mortality rates for the outcomes studied. We selected outcomes on the basis of data availability, range of clinical conditions, and consistency with previously published studies. Rates for New York were from 1997; rates for California were either from 1997 alone or were an average of rates from 1995 to 1997.

We obtained the following measures of county socioeconomic status (SES) from the US Census Bureau: unemployment rate (1997), percentage living in poverty (1995), percentage of children aged less than 18 years living in poverty (1995), median household income (1995), and high school graduation rate (1990).[15] Percentage living in poverty is defined as the percentage of households under the federal poverty threshold, adjusted for family size and composi-

TABLE 1—Mortality Rates from Various Causes in New York and California, 1997.[a]

Outcome	New York			California		
	Mean	Range	Range / Mean	Mean	Range	Range / Mean
All-cause mortality (all ages)	829	274	0.3	453.8	228.5	0.5
All-cause mortality (≥ 75 y)	21 655	9070	0.4	19 284.2	8714	0.5
All-cause mortality (10–24 y)[b]	120	310	2.6	75.0	155.1	2.1
Infant mortality	6.7	12.5	1.9	7.0	12.3	1.8
AIDS	4.9	53.7	10.9	3.4	24.6	7.3
Pneumonia[c]	34.4	65.1	1.9	15.8	22.3	1.4
COPD	42.7	58.3	1.4	23.5	29.6	1.3
Cardiovascular disease	286	234	0.8	89.5	75.0	0.8
Stroke	47.9	64.0	1.6	26.4	22.6	0.9
All neoplastic disease	204	107	0.5	118.8	56.1	0.5
Lung cancer	n/a	n/a	n/a	34.9	32.3	0.9
Female breast cancer	n/a	n/a	n/a	19.1	19.1	1.0
Cirrhosis	8.0	15.6	2.0	10.1	24.7	2.4
Accidents[d]	29.2	33.8	1.2	18.5	29.5	1.6
Homicide	2.6	16.7	6.3	6.7	17.7	2.6
Suicide	n/a	n/a	n/a	12.1	18.3	1.5

Note. COPD = chronic obstructive pulmonary disease; n/a = not available.
[a]New York data from 1997. California data from 1997 or aggregated from 1995–1997. New York rates are age-adjusted to New York State census population 1990. California rates are standardized to US standard population 1940. (As such, these rates are not directly comparable.) All rates are per 100 000 population.
[b]California—ages 15–24 years.
[c]California—includes influenza.
[d]California—motor vehicle accidents only.

tion.[16] These socioeconomic factors were chosen on the basis of data availability and consistency with previously published studies.[11,17]

Analysis

We analyzed counties in California and New York separately. We stratified counties into quartiles by each of the SES measures, calculated the average rate of each health outcome for the bottom and top quartiles, and obtained the rate ratio for a given health outcome by comparing counties in the lowest and highest socioeconomic quartiles.

We also calculated the variability of each health outcome across all counties in each state. Following Levins and Lopez, we used the range of values divided by the mean value as the measure of variability; this measure provides a useful estimate for qualitative analyses.[11] The larger the range divided by the mean value, the higher the variability of a particular health outcome across counties. No statistical inferences were based on this measure of variability. We calculated Pearson correlation coefficients between rate ratios and variability measures and examined variability in outcomes across counties, stratified by SES.

We also calculated smoothed county-specific rates, in which the observed rate in a county was "stabilized" by replacing it with the weighted average of the county rate and all adjacent county rates; weights were proportionate to population size.[18] We repeated all of the analyses described here on the smoothed rate estimates. Finally, we compared outcome rankings and correlations derived using the range-divided-by-mean measure with rankings obtained using 2 other variability measures: interquartile range divided by mean, and the coefficient of variation (SD / mean × 100%).

RESULTS

We included 61 (98%) of 62 counties in New York and 53 (91%) of 58 counties in California in the analysis. For each outcome, Table 1 shows the mean, range, and range divided by mean across counties in both states.

In New York, the largest variability in outcomes was in AIDS mortality (range / mean = 10.9), followed by homicide (6.3), all-cause mortality among persons aged 10–24 years (2.6), and mortality from cirrhosis (2.0). The smallest variability was observed in all-cause mortality across all ages (0.3) and among persons aged more than 75 years (0.4), as well as in mortality from neoplastic disease (0.5) and mortality from cardiovascular disease (0.8).

In California, variability was highest for AIDS (range / mean = 7.3), followed by homicide (2.6), mortality from cirrhosis (2.4), and mortality among persons aged 15–24 years (2.1). The lowest variability was in rates for all-cause mortality across all ages and for persons aged more than 75 years as well as mortality from neoplastic disease (0.5 for each) and mortality from cardiovascular (0.8) or cerebrovascular (0.9) disease. The ordering of diseases by their intercounty variability was similar between the 2 states.

TABLE 2—Relative Mortality Rates Comparing Counties in the Lowest to Highest Quartiles of Economic Indicators, New York and California, 1997.

	New York						California					
Outcome	Employment	Poverty	Under-18 poverty	Income	% high school grad	Row mean	Employment	Poverty	Under-18 poverty	Income	% high school grad	Row mean
All-cause mortality (all ages)	1.05	1.07	1.05	1.05	1.03	1.05	1.16	1.19	1.22	1.21	1.13	1.18
All-cause mortality (≥ 75 y)	0.97	0.97	0.96	1.01	0.95	0.97	0.95	0.94	0.94	0.91	0.92	0.93
All-cause mortality (10–24 y)	1.00	1.41	1.22	1.21	1.47	1.26	1.32	1.16	1.27	1.32	1.46	1.31
Infant mortality	1.04	1.14	1.04	0.96	1.17	1.07	1.24	1.35	1.41	1.29	1.17	1.29
AIDS	3.47	3.13	4.00	1.64	2.59	2.96	0.40	0.68	0.88	0.57	0.68	0.64
Pneumonia	0.92	1.11	1.09	1.10	1.01	1.04	0.93	1.07	1.09	0.88	0.99	0.99
COPD	1.01	1.22	1.10	1.16	1.12	1.12	1.35	1.32	1.34	1.47	1.19	1.33
Cardiovascular disease	1.12	1.06	1.07	1.05	1.04	1.07	1.18	1.28	1.33	1.18	1.23	1.24
Stroke	0.85	1.16	1.13	1.02	0.96	1.02	1.11	1.11	1.17	1.09	1.15	1.12
All neoplastic disease	1.01	0.97	0.97	1.00	0.94	0.98	1.07	1.02	1.05	1.12	1.01	1.05
Lung cancer	n/a	n/a	n/a	n/a	n/a	n/a	1.15	1.11	1.12	1.23	0.99	1.12
Female breast cancer	n/a	n/a	n/a	n/a	n/a	n/a	1.00	0.86	0.85	0.94	0.88	0.90
Cirrhosis	1.42	1.40	1.37	1.20	1.00	1.28	1.13	1.44	1.38	1.43	0.98	1.27
Accidents	0.91	1.25	1.13	1.17	1.23	1.14	2.56	1.71	1.75	2.38	1.76	2.03
Homicide	1.40	1.94	2.23	1.13	1.20	1.58	1.40	2.02	2.61	1.39	1.86	1.85
Suicide	n/a	n/a	n/a	n/a	n/a	n/a	0.93	1.00	0.98	1.06	0.73	0.94
Column mean	1.23	1.36	1.39	1.13	1.20		1.18	1.20	1.27	1.22	1.13	

Note. COPD = chronic obstructive pulmonary disease; n/a = not available.

Rate ratios comparing mean disease-specific mortality rates between counties in the lower and upper quartiles of various socioeconomic markers are shown in Table 2. Rate ratios greater than 1.0 imply that counties with lower SES have higher disease-specific mortality than do those with higher SES.

The range of all rate ratios in New York was 0.85–4.00. The mean ratios across economic marker categories ranged from 0.98 for neoplastic disease to 2.96 for AIDS. Mean homicide and cirrhosis mortality ratios were 1.58 and 1.28, respectively. For all economic markers, all-cause mortality, all-cause mortality for persons aged more than 75 years, and mortality from neoplasms had ratios less than 1.10. Mortality from all causes in persons aged 10–24 years had a mean rate ratio of 1.26. The mean ratios for all outcomes across economic measures ranged from 1.13 (median household income) to 1.39 (percentage of persons aged <18 years in poverty).

In California, ratios ranged from 0.40 to 2.61. The mean ratio across economic indicators for neoplastic disease rates was 1.05 (0.90 for female breast cancer and 1.12 for lung cancer). The highest mean ratios were for mortality rates from motor vehicle accidents (2.03) and for homicide rates (1.85). The mean ratio for cirrhosis was 1.27. All-cause mortality for persons aged more than 75 years, suicide, pneumonia and influenza mortality, and female breast cancer mortality each had rate ratios of less than 1.10 for all economic markers. In addition, ratios for AIDS mortality ranged from 0.40 to 0.88. Mortality from all causes in persons aged 10–24 years had a mean rate ratio of 1.31, whereas for persons aged more than 75 years the mean ratio was 0.93. The mean ratios for all outcomes across economic measures ranged from 1.13 (high school graduation rate) to 1.27 (percentage of persons aged <18 years in poverty).

In both states, the variability (range/mean) of health outcomes across counties was strongly correlated with the mean ratio of rates between counties in the lowest and highest quartiles of economic status (measured by percentage of children <18 years living in poverty). In New York, the Pearson correlation coefficient was 0.97; in California, it was 0.70 (0.01 when AIDS was included in the calculation).

Figure 1 shows the relation between the homicide rates in New York counties and their socioeconomic status. Homicide rates for each county were plotted against tertiles of poverty (percentage of persons aged <18 years living in poverty). Counties of lower economic status displayed greater variability in their rates of homicide than do counties with high economic status. The general trend of the relation was linear, with a positive slope; however, among counties with the lowest economic status, there were both low and high rates.

Smoothed rates showed less variability than did observed rates (range/mean for all outcomes varied from 0.2 to 6.4, compared with 0.3 to 11.0 in the original analysis); nonetheless, the trends and correlations present in the original analysis were preserved. In both New York and California, AIDS rates and homicide rates continued to display the most variability

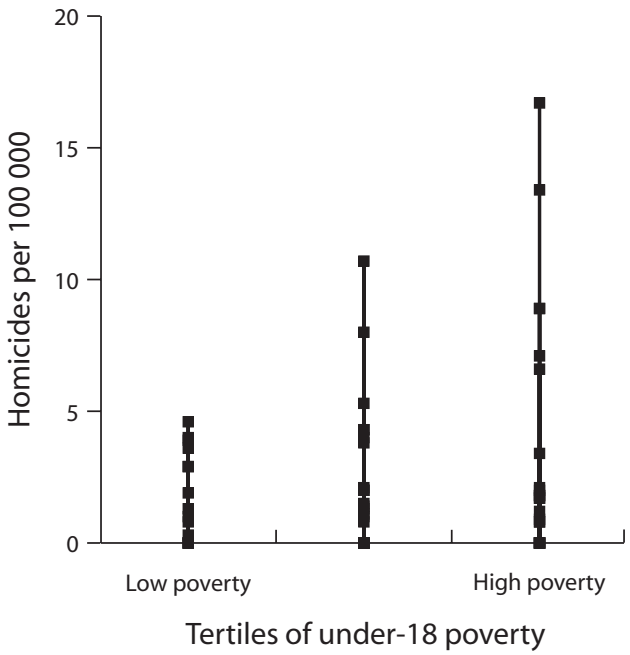

FIGURE 1—Intercounty variability in homicide rates stratified by tertiles of child (under 18 y) poverty, New York State, 1997.

(New York AIDS range/mean=6.4, homicide range/mean=3.4; California AIDS range/mean=2.0, homicide range/mean=1.4), and mortality among persons aged more than 75 years and neoplastic disease mortality continued to display the least (range/mean= 0.2–0.3). Similarly, as expected, the magnitudes of variability for each outcome were attenuated when the coeffecient of variation or the interquartile range divided by mean were used (ranges 12%–104% and 0.1–0.9, respectively, in New York, and 7%–200% and 0.2–0.7 in California); the ordering of outcomes by variability, however, remained largely unchanged in comparison with the ordering obtained with the range-divided-by-mean statistic (e.g., AIDS and homicide rates remained most variable and all-cause mortality and mortality from neoplasms remained least variable), as did the strong association between variability and SES sensitivity.

DISCUSSION

This analysis investigated the effects of counties' SES on their mortality rates and the variability of those rates across counties. We hypothesized that the outcomes most sensitive to SES would also exhibit the most variability. Outcomes in both New York and California that displayed high sensitivities to SES were AIDS, homicide, cirrhosis, and accidents. Outcomes in both states were most sensitive to the percentage of children living in poverty as a single indicator of SES.

All-cause mortality and certain neoplastic disease mortality rates did not differ greatly between poorer and wealthier counties. The underlying mechanisms for these health outcomes might account for the findings. Diseases with an incidence or course that is influenced by behavioral or environmental factors would be expected to exhibit sensitivity to SES, whereas diseases with genetic or other nonmodifiable causes would not. Our analysis is consistent with earlier findings and builds on previous small-area analyses in Kansas, Saskatchewan, and Cuba.[11]

A general process underlying these observations has been articulated by Link and Phelan, who postulated that access to protections and avoidance of harms underlie health outcomes that are sensitive to SES.[19] For example, in California, lung cancer mortality (largely a consequence of smoking) had a higher mean ratio than did female breast cancer mortality. In New York, cirrhosis mortality (largely a consequence of alcohol misuse) had a higher rate ratio than did neoplasms. A comparison of age-specific mortality further supports this hypothesis. In New York and California, county rates between economic strata exhibit low ratios for mortality among older persons and high ratios for youth mortality. Youth mortality has more potentially modifiable behavioral and social causes at its root than does mortality among older persons.

The economic sensitivity of AIDS was reversed in New York and California. In New York State, counties in the lowest economic quartiles had an average of 2.96 times the AIDS rates of counties in the highest quartiles, and AIDS is particularly prevalent in poor communities of New York City. By contrast, in California, the populations with the lowest SES are both urban and rural, and AIDS incidence is more widely distributed in populations of varying SES.

Our principal measures of interest were variabilities in outcomes rather than absolute rates. Variability in mortality rates across counties was highest for the outcomes with larger SES rate ratios. AIDS mortality and homicide were the outcomes with the largest variability in the 2 states. Rates for all-cause mortality across ages and for mortality in older persons, as well as rates for neoplastic disease mortality, exhibited small variability and were generally not sensitive to socioeconomic conditions.

Although we hypothesized that variability reflects system-specific conditions (i.e., the balance among vulnerability, stressors, and protectors), variability may also be the product of random events, especially when the outcome of interest is rare or the population within which it occurs is small. Moreover, there may be confounding of the SES–sensitivity/variability relationship if rare events are also more sensitive to SES. To address these possibilities, we repeated all analyses using smoothed county-specific rates, which reduce intercounty population variability, as well as robust measures of variability,

which have low sensitivity to outliers. The observed rate variability was attenuated under these circumstances, but trends in health outcome variability and associations with county-level SES were preserved.

In their examination of ecological factors contributing to adverse health effects, Levins and Lopez suggested that the relation between economic deprivation and variability in health status might be mediated by the vulnerability of populations.[11] They cited an observation by a geneticist, I. I. Schmalhausen, that "a system at the boundary of its tolerance along any dimension of its existence is more vulnerable to small differences in circumstance along any dimension."[20(p.276)] Populations enduring social or economic deprivation will be more vulnerable to potential stressors than will populations of higher status. Thus, acute outbreaks of infectious diseases, environmental risks, or transient gaps in public health services will likely affect an economically deprived population to a greater degree than they would a less marginalized population.

We note, however, that these external stressors are not uniformly distributed across all disadvantaged communities, and therein might lie the source of the observed variability. Although vulnerability might result from chronic economic deprivation, the range of adverse health outcomes will depend on the degree to which each community experiences stressors and the distribution within communities of counteracting protective factors.

Variability in biological systems is increasingly seen as a marker for stresses to systems in homeostasis. Applying this insight to communities and health, we postulate that economic deprivation produces vulnerability to stressors whose nonuniform distribution across populations manifests as variability in health outcomes. One possible implication of this model is that interventions to improve public health might exert the greatest effect not by targeting particular stressors, but rather by focusing on improving general social and economic well-being, thus reducing populations' overall vulnerability. ■

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Contributors

A. Karpati and S. Galea collected the data, performed the analysis, and wrote the article. T. Awerbuch contributed to the study design, oversaw the analysis, and contributed to the editing of the article. R. Levins contributed to the study formulation and design, oversaw the analysis, and contributed to the editing of the article.

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