Racial Disparities in Context: A Multilevel Analysis of Neighborhood Variations in Poverty and Excess Mortality Among Black Populations in Massachusetts

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Objectives. We analyzed neighborhood heterogeneity in associations among mortality, race/ethnicity, and area poverty.

Methods. We performed a multilevel statistical analysis of Massachusetts all-cause mortality data for the period 1989 through 1991 (n = 142 836 deaths), modeled as 79 813 cells (deaths and denominators cross-tabulated by age, gender, and race/ethnicity) at level 1 nested within 5532 block groups at level 2 within 1307 census tracts (CTs) at level 3. We also characterized CTs by percentage of the population living below poverty level.

Results. Neighborhood variation in mortality across CTs and block groups was not accounted for by these areas' age, gender, and racial/ethnic composition. Neighborhood variation in mortality was much greater for the Black population than for the White population, largely because of CT-level variation in poverty rates.

Conclusions. Neighborhood heterogeneity in the relationship between mortality and race/ethnicity in Massachusetts is statistically significant and is closely related to CT-level variation in poverty. (*Am J Public Health.* 2005;95:260–265. doi: 10.2105/AJPH.2003.034132)

Despite documentation of substantial variations in US mortality, whether by state and county or by race/ethnicity, gender, and socioeconomic position,[1–10] few analyses have examined mortality variation at the local geographic level, for example, by neighborhood. Even fewer have attempted to elucidate whether the well-known average racial/ethnic disparities in mortality vary across neighborhoods. We offer a systematic examination of such *contextual heterogeneity*, defined as geographic variation in the individual relationship between race/ethnicity and mortality, conditional upon adjustment of other individual covariates.

Why would an empirical evaluation of contextual heterogeneity by population subgroups (e.g., White and Black) be important? Patterns of all-cause mortality are shaped by a complex constellation of individual as well as contextual factors that may conceivably vary for Whites and Blacks, as suggested, for example, by the fact that leading causes of death differ for different racial/ethnic groups.[11] An investigation of race/ethnicity–specific contextual heterogeneity in mortality at the neighborhood level can give insight into the relative importance to mortality of individual and contextual factors for different racial/ethnic populations. From a methodological standpoint, addressing this question requires that context be included as an intrinsic part of analytical strategies, as achieved by multilevel statistical models.[12,13]

To date, however, multilevel methods in public health research have principally been applied to estimate the "average" effect of a predictor measured at an area level on individual-level health outcomes, including mortality.[14,15] While this approach is important, it can potentially obscure the contextual heterogeneities underlying such average effects. We examined contextual heterogeneity in mortality in relation to race/ethnicity and area poverty by analyzing 1989–1991 Massachusetts mortality data, prepared for the US Public Health Disparities Geocoding Project,[16] in conjunction with 1990 census data. Specifically, we addressed 3 questions: (1) What is the neighborhood variation in mortality rates for different racial/ethnic groups? (2) What is the magnitude of neighborhood variation in the average Black–White disparity? (3) Does neighborhood variation in poverty account for the racial/ethnic contextual variation and racial/ethnic disparities in mortality?

To characterize individuals' neighborhood context, we employed 2 levels of census geography: the census tract (CT), which on average contains 4000 persons, and the census block group (BG), a subdivision of the CT, which on average contains 1500 persons.[17] The 2-fold appeal of CTs is that "when first delineated, [they] are designed to be homogeneous with respect to population characteristics, economic status, and living conditions,"[17(ppG10–G11)] and that, once created, they constitute administrative units used by federal, state, and local governments, including public health departments, to characterize jurisdictions, determine eligibility for diverse programs, and allocate resources.[18–20] Therefore, CTs have real-life implications for their residents.

METHODS

Data

The Massachusetts Department of Public Health provided data for all deaths occurring between January 1, 1989, and December 31, 1991, to Massachusetts residents (n = 156 366). The denominator data were obtained from the 1990 US census for Massachusetts (n = 6 016 425). Mortality records were geocoded to the CT and BG level with an accuracy of about 96%.[21] Death records missing information on age, gender, or race/ethnicity were excluded from the analyses, along with records geocoded to BGs with populations of 0. The final data consisted of 142 836 death records corresponding to 18 049 275 person-years.

Analytical Structure

The data had a hierarchical multilevel structure of 79 813 cells at level 1, consisting of individuals in numerators and denomina-

TABLE 1—Description of Data Used for Multilevel Models Analyzing Neighborhood Heterogeneity in Associations Between Mortality, Race/Ethnicity, and Area Poverty: Massachusetts, 1989–1991

Predictor	No. of Cells (% of Total)	No. of Deaths	Person-Years	Mortality Rate per 100 000 (SD)
Overall	79 813	142 836	18 049 275	967 (2644)
Level 1: cell (n = 79 813)				
Age 0–14 y				
White male	5243 (6.6)	675	1 496 835	61 (293)
Black male	1284 (1.6)	147	116 898	103 (556)
Other male	1849 (2.3)	120	135 519	88 (625)
White female	5234 (6.6)	498	1 416 618	44 (241)
Black female	1274 (1.6)	116	115 398	104 (673)
Other female	1886 (2.4)	96	131 454	61 (457)
Age 15–24 y				
White male	5210 (6.5)	860	1 196 304	99 (401)
Black male	1076 (1.3)	158	80 268	159 (675)
Other male	1486 (1.9)	86	93 798	92 (487)
White female	5230 (6.6)	289	1 198 560	34 (212)
Black female	1110 (1.4)	37	82 476	39 (364)
Other female	1511 (1.9)	23	98 478	24 (265)
Age 25–44 y				
White male	5431 (6.8)	4265	2 681 712	196 (415)
Black male	1718 (2.2)	549	151 275	297 (918)
Other male	2391 (3.0)	373	161 760	214 (822)
White female	5460 (6.8)	1807	2 748 753	76 (253)
Black female	1653 (2.1)	263	155 595	119 (514)
Other female	2525 (3.2)	130	164 478	83 (593)
Age 45–64 y				
White male	5379 (6.7)	12 570	1 500 516	1080 (1450)
Black male	999 (1.3)	597	58 539	825 (1870)
Other male	1312 (1.6)	228	48 906	470 (1570)
White female	5375 (6.7)	8029	1 627 536	598 (878)
Black female	1019 (1.3)	429	73 383	449 (1170)
Other female	1424 (1.8)	143	54 435	282 (1440)
Age ≥ 65 y				
White male	5233 (6.6)	46 725	900 960	6079 (5160)
Black male	492 (0.6)	726	21 948	3133 (4650)
Other male	460 (0.6)	191	14 193	1177 (3140)
White female	5341 (6.7)	61 535	1 467 528	4636 (4320)
Black female	620 (0.8)	965	35 238	2560 (3880)
Other female	588 (0.7)	206	19 914	939 (2470)
Level 2: block group (n = 5532)	...	...	...	...
Level 3: census tract (n = 1307)	...	...	...	...
% of CT population below poverty	base: CT poverty 0%–4.9% (n = 492; 37.6%); contrast: CT poverty 5%–9.9% (n = 386; 29.5%); CT poverty 10%–19.9% (n = 225; 17.2%); CT poverty 20%–100% (n = 204; 15.6%)			

Note. CT = census tract. Data on deaths of Massachusetts residents occurring between January 1, 1989, and December 31, 1991, are from the Massachusetts Department of Public Health (n = 156 366). Denominator data were obtained from the 1990 US census for Massachusetts (n = 6 016 425). Mortality rate was calculated on the basis of the means of the proportion of deaths for each cell type across all block groups and CTs. The data on CT poverty came from the 1990 US Census.

tors cross-tabulated by age, gender, and race/ethnicity, which were nested within 5532 BGs at level 2, nested within 1307 CTs at level 3. Each BG had between 1 and 30 cells, with the numerator specifying the number of persons who died and the denominator providing the total population used to calculate the proportion of deaths in each cell. In the analytical model we considered both BG and CT levels; however, given the greater policy appeal for health monitoring of populations, our substantive interest was at the CT level. Structurally, our models were identical to models with individuals at level 1.[22]

Response and Predictors

The response, mortality, is defined as a proportion: the number of deaths as a proportion of the total population for each cell. The cell predictor variables relate to 5 age categories (0–14 years, 15–24 years, 25–44 years, 45–64 years, and 65 years and older), 2 gender categories (male and female); and 3 racial/ethnic categories (White, Black, and all others, each including non-Hispanics and Hispanics; these 3 groups made up, respectively, 89.9%, 4.9%, and 5.1% of the population enumerated in Massachusetts in the 1990 census). Because of the heterogeneity of the population in "all others," we focused our interpretation mainly on the White–Black comparisons. On the basis of the cross-tabulation of age, gender, and racial/ethnic categories, we obtained 30 unique population groups, or cells, as shown in Table 1.

CTs were characterized by percentage of the population living below the poverty line, with the following cutoff points: 0%–4.9%, 5%–9.9%, 10%–19.9%, and 20%–100%. The rationale for considering area poverty was based on previous research showing the consistency with which area poverty detected socioeconomic gradients across a range of health outcomes, including mortality.[16,23,24] The federal definition of "poverty areas"[25] as areas where more than 20% of persons are living below the poverty line (in 1989, equal to $12 575 for a family of 2 adults and 2 children)[26] was a key consideration in determining the cutoff points. Although data on educational level were available from the death certificates, 1990 census population data stratified simultaneously by age, gender, race/ethnicity, and educational level were available only at the

county—not CT or BG—level, precluding the use of education in specifying the cell structure.

Statistical Analysis

We employed multilevel statistical procedures[27–29] because of their ability to model complex variance structures at multiple levels. The principles underlying multilevel modeling procedures are now well known,[13] and in the context of the present analysis they allow estimation of the relationship between mortality and race/ethnicity, conditional on individual age and gender variations ("fixed parameters") and CT- and BG-level variations ("random parameters"). They also enable an estimation of the extent to which the relationship between mortality and race/ethnicity varies across CTs (random parameters) and the degree to which CT poverty explains this variation (fixed parameters).

The response variable, proportion of deaths in each cell, was modeled with allowances made for the varying denominator in each cell.[27] The fixed and random parameter estimates (along with their standard errors) for the 3-level binomial logit link model were calibrated using predictive/penalized quasi-likelihood procedures with second order Taylor series expansion,[30] as implemented within the MLwiN program.[31] We allowed for extrabinomial variations at level 1, because proportions may exhibit more or less variation than a binomial distribution.[32] We calibrated 3 models.

Model 1. Model 1 was a 3-level model of cells (level 1) within BGs (level 2) within CTs (level 3), with cell characteristics related to age, gender, and race/ethnicity specified in the fixed part of the model and a residual variation estimated at the CT and BG levels in the random part. The fixed-part specification included the main effects for the age, gender, and racial/ethnic categories, along with a second-order interaction between age–gender, age–race/ethnicity, and gender–race/ethnicity. We did not find any empirical support for third-order interaction terms. The estimates from model 1 allowed an evaluation of the racial/ethnic differences in mortality and the magnitude of variation in mortality at the BG and CT levels, conditional on the relationship between mortality and age, gender, and race/ethnicity within each BG and CT.

Model 2. Model 2 was similar to model 1, but it allowed the fixed racial/ethnic differential on mortality to vary across CTs in the random part to obtain differential CT-level variation in mortality for Whites, Blacks, and others. CT-level random parameters from model 2 were used to test the hypothesis pertaining to race/ethnicity–based contextual heterogeneity, that is, whether neighborhood-level variation in mortality was different for different racial/ethnic groups.

Model 3. Model 3 was similar to model 2, but it included a fixed cross-level interaction effect between CT-level poverty and individual race/ethnicity. In this way, we ascertained the relationship between neighborhood poverty, individual race/ethnicity, and mortality, as well as the extent to which CT-level poverty accounted for the CT-level racial/ethnic variation in mortality.

RESULTS

When we controlled for age, the mortality odds ratio (OR) was 25% higher for men than for women (OR=1.25, 95% confidence interval [CI]= 1.11, 1.41) and twice as high for Blacks as for Whites (OR=1.96, 95% CI=1.65, 2.33; Table 2, model 1), in the reference age group of 0-14 years. The odds ratio for the 3 interaction terms (age×gender, age×race/ethnicity, gender×race/ethnicity) represent the unique additional impact of these interactions, not including the main effects. Crucially, and of relevance to this study, the between-CT variance (σ^2_{v0}=0.09; Table 3, model 1) was statistically significant ($P<.001$) even after we took into account the fixed main effect and the 3 two-way interactions, of age, gender, and race/ethnicity on mortality *and* after we took into account the between-BG (within-CT) differences in mortality.

Model 2 (Table 3) allows an assessment of the neighborhood heterogeneity (across CTs) in racial/ethnic disparities in mortality. We found a statistically significant CT-level variation ($P<.001$) in the individual relationship between mortality and race/ethnicity. The between-CT variation in mortality was substantially greater for Blacks (0.524) than for Whites (0.085; Table 4). Accounting for this substantial CT-level heterogeneity in mortality by race/ethnicity reduced the fixed mortality differential for Blacks (aged 0–14 years) from a nearly 2-fold odds ratio (Table 2, model 1) to an odds ratio of 1.30 (95% CI=1.08, 1.56). Understandably, it did not attenuate any of the associated interaction effects.

Results from model 3 (Table 3) supported the hypothesis that CT poverty accounts for the racial/ethnic-specific heterogeneity in mortality at the CT level. While between-CT variances in mortality declined only slightly for Whites (from 0.08 in model 2 to 0.07 in model 3), CT poverty accounted for 63% of the CT-level mortality variation for Blacks (from 0.52 in model 2 to 0.19 in model 3; Table 4).

The odds ratio for mortality increased with neighborhood poverty, and the relationship was substantially stronger for Blacks than for Whites (Table 2, model 3). When Whites living in the 3 higher CT-poverty strata were compared with the reference group (Whites living in CTs with lower than 5% poverty), their odds ratios were 1.05 (CTs with 5%–9.9% poverty), 1.23 (CTs with 10%–19.9% poverty), and 1.42 (CTs with 20% or higher poverty). For Blacks, however, compared with the same reference group (Whites living in CTs with lower than 5% poverty), the odds ratios for the unique interactions were, respectively, 1.67 (CTs with 5%–9.9% poverty), 2.34 (CTs with 10%–19.9% poverty), and 3.00 (CTs with 20% or higher poverty).

DISCUSSION

We found, first, that between-CT variation in mortality was some 6 times greater for Blacks than for Whites. Second, neighborhood poverty contributed substantially to the observed area variations in Black excess mortality. Indeed, if we consider the estimated variation between CTs as a "true" estimate of race/ethnicity–specific contextual heterogeneity, then the mortality odds ratio for Blacks compared with Whites can range from 0.31 to 5.36 (with the "average" Black–White disparity 1.30). While the existing literature is conceptually (and to a large extent, empirically) rich in descriptions of average racial/ethnic differences in mortality and possible explanations for such differences,[11,33,34] there has been little documentation—let alone investigation—of why the racial/ethnic disparities are much greater in some neighborhoods than others.

TABLE 2—Odds Ratios and 95% Confidence Intervals for Fixed Parameters From Models 1, 2, and 3 Analyzing Neighborhood Heterogeneity in Associations Between Mortality, Race/Ethnicity, and Area Poverty: Massachusetts, 1989–1991

	OR (95% CI)		
	Model 1	Model 2	Model 3
Individual-level predictors			
Main effect			
Age, y			
0–14	1.00	1.00	1.00
15–24	0.64 (0.54, 0.75)	0.64 (0.54, 0.75)	0.64 (0.54, 0.75)
25–44	1.82 (1.63, 2.03)	1.81 (1.62, 2.02)	1.81 (1.62, 2.02)
45–64	13.92 (12.59, 15.38)	13.89 (12.56, 15.36)	13.87 (12.53, 15.36)
≥65	119.46 (108.36, 131.70)	119.22 (108.09, 131.51)	118.99 (107.77, 131.36)
Gender			
Female	1.00	1.00	1.00
Male	1.25 (1.11, 1.41)	1.25 (1.11, 1.41)	1.25 (1.11, 1.41)
Race/ethnicity			
White	1.00	1.00	1.00
Black	1.96 (1.65, 2.33)	1.30 (1.08, 1.56)	0.72 (0.53, 0.98)
Other	1.47 (1.21, 1.78)	0.98 (0.80, 1.21)	0.54 (0.39, 0.74)
Interaction effect			
Male, 15–24 y	2.56 (2.12, 3.09)	2.57 (2.12, 3.10)	2.57 (2.12, 3.11)
Black	0.95 (0.75, 1.22)	0.97 (0.75, 1.24)	0.97 (0.75, 1.24)
Other	0.63 (0.47, 0.84)	0.66 (0.48, 0.90)	0.64 (0.47, 0.87)
Male, 25–44 y	1.93 (1.69, 2.21)	1.94 (1.70, 2.22)	1.95 (1.70, 2.23)
Black	0.90 (0.74, 1.08)	0.92 (0.76, 1.11)	0.93 (0.77, 1.12)
Other	0.73 (0.59, 0.90)	0.78 (0.63, 0.97)	0.81 (0.65, 1.00)
Male,45–64 y	1.38 (1.22, 1.56)	1.38 (1.22, 1.57)	1.38 (1.22, 1.57)
Black	0.42 (0.35, 0.51)	0.42 (0.35, 0.50)	0.43 (0.36, 0.51)
Other	0.28 (0.22, 0.34)	0.30 (0.24, 0.37)	0.32 (0.25, 0.40)
Male, ≥65 y	1.04 (0.93, 1.18)	1.05 (0.93, 1.18)	1.05 (0.93, 1.18)
Black	0.22 (0.18, 0.26)	0.21 (0.18, 0.26)	0.22 (0.18, 0.26)
Other	0.12 (0.10, 0.15)	0.13 (0.11, 0.17)	0.14 (0.11, 0.17)
Male, Black	1.01 (0.93, 1.09)	1.02 (0.94, 1.11)	1.02 (0.94, 1.11)
Male, Other	1.08 (0.95, 1.23)	1.10 (0.96, 1.25)	1.10 (0.96, 1.25)
CT-level predictor			
Main effect			
5%–9.9% poverty	. . .	. . .	1.05 (1.00, 1.10)
10%–19.9% poverty	. . .	. . .	1.23 (1.16, 1.30)
20%–100% poverty	. . .	. . .	1.42 (1.33, 1.51)
Cross-level interaction effect (individual race and CT poverty)			
White, 0%–4.9% poverty	. . .	. . .	1.00
Black, 5%–9.9% poverty	. . .	. . .	1.67 (1.24, 2.26)
Black, 10%–19.9% poverty	. . .	. . .	2.34 (1.76, 3.12)
Black, 20%–100% poverty	. . .	. . .	3.00 (2.28, 3.94)
Other, 5%–9.9% poverty	. . .	. . .	1.32 (0.95, 1.84)
Other,10%–19.9% poverty	. . .	. . .	2.26 (1.67, 3.07)
Other, 20%–100% poverty	. . .	. . .	3.37 (2.56, 4.45)

Note. OR = odds ratio, CI = confidence interval, CT = census tract. ORs for interaction terms are the unique (additional) effect of a particular variable and do not include the main effect.

Our findings suggest a need to conceptualize contextual effects (e.g., neighborhood poverty) in more complex ways. Typically, the examination of contextual effects has proceeded with an assumption of "main contextual effects" (e.g., that neighborhood poverty affects individual mortality in a similar manner for all racial/ethnic groups). However, it is entirely reasonable (and perhaps more realistic) to anticipate that contextual differences as well as contextual effects inherently interact with individual characteristics. Our finding that neighborhood-level poverty *directly* contributes to the greater geographic heterogeneity in mortality rates for Blacks suggests that the consequences of neighborhood deprivation may be particularly exacerbated for Blacks, compared with Whites. That is, there is a joint and synergistic shaping of population patterns of mortality by individual (e.g., race/ethnicity) and contextual (e.g., CT poverty) factors.

The following caveats should be considered in interpreting the empirical findings of our study. First, there is a distinct possibility of a potential misspecification of the individual-level factors that are associated with mortality. Our specification of individual variables was constrained by the degree of cross-tabulation (i.e., by age, race/ethnicity, and gender) that was possible on the numerator and denominator populations simultaneously at the BG level. This constraint precluded estimation of the impact of other important (unmeasured) socioeconomic markers on clustering in mortality.

Had we constructed our cells by county, it would have been possible to include educational data at the individual level (thus increasing the "social resolution" at the individual level), but this would have come at the cost of ignoring the critical level of BGs and CTs (thus decreasing the "spatial resolution"). While it is difficult to evaluate the sensitivity of our findings, given the lack of data, it is likely that including additional socioeconomic markers might have attenuated both the magnitude of racial/ethnic disparities in mortality and the effect of CT poverty. It is *unlikely,* however, that such data would have accounted for the contextual heterogeneity in racial/ethnic disparities in mortality. For this to have happened, the omitted individual socioeconomic markers not only would have to

TABLE 3—Random Parameters at the Census Tract (CT) and Block Group (BG) Levels From Models 1, 2, and 3 Analyzing Neighborhood Heterogeneity in Associations Between Mortality, Race/Ethnicity, and Area Poverty: Massachusetts, 1989–1991

	Estimate (SE)		
	Model 1	Model 2	Model 3
	Between-CT variation		
Constant/constant (σ^2_{v0})	0.095 (0.005)	0.085 (0.005)	0.066 (0.004)
Constant/Black (σ^2_{v0v1})	. . . (. . .)	0.050 (0.015)	–0.017[a] (0.012)
Black/Black (σ^2_{v1})	. . . (. . .)	0.337 (0.053)	0.158 (0.034)
Constant/other (σ^2_{v0v2})	. . . (. . .)	0.054 (0.016)	–0.034 (0.013)
Black/other (σ^2_{v1v2})	. . . (. . .)	0.136 (0.047)	0.028[a] (0.030)
Other/other (σ^2_{v2})	. . . (. . .)	0.325 (0.064)	0.120 (0.040)
	Between-BG variation		
Cell-level dispersion	1.399 (0.007)	1.405 (0.007)	1.442 (0.007)
Constant/constant (σ^2_{u0})	0.111 (0.004)	0.111 (0.004)	0.1083 (0.004)

Note. The parameter σ^2_{v0} represents the variance for the base category (Whites), whereas σ^2_{v1} and σ^2_{v2} represent the differential variance for Blacks and others, respectively. The parameters σ_{v0v1}, σ_{v0v2}, and σ_{v1v2} present the covariance associated with the random variables, v_{0k}, v_{1k}, and v_{2k}, associated with the race/ethnicity categories. The random parameters were tested with "Wald-like" tests,[27,31] and *P* values were based on a χ^2 distribution. Tests were conducted for both individual random parameters and the entire random part at the CT level.

[a]These terms were *not* significant at $P \leq .001$; all other terms were.

TABLE 4—Variation (on Logit Scale) in Mortality Between Census Tracts (CTs) by Race/Ethnicity, Before and After Taking Into Account CT-Level Poverty: Massachusetts, 1989–1991

	White	Black	Other
Before taking account of poverty	0.08	0.52	0.52
After taking account of poverty	0.07	0.19	0.12

Note. Estimates for Whites relate to the CT-level random parameter estimate associated with σ^2_{v0} in Models 2 and 3 (see Table 3). Estimates for Blacks were not estimated directly but were based on the sum of the random parameter variance related to Whites (σ^2_{v0}), the differential variance associated with Blacks (σ^2_{v1}), and 2 times the covariance between these 2 variances (σ_{v0v1}) in Models 2 and 3. Estimates for "others" were not estimated directly but were based on the sum of the random parameter variance related to Whites (σ^2_{v0}), the differential variance associated with others (σ^2_{v2}), and 2 times the covariance between these 2 variances (σ_{v0v2}) in Models 2 and 3.

be to perfectly collinear with individual race/ethnicity but would also need to have exactly the same spatial clustering as race/ethnicity.

It is, however, worth noting that neighborhood effects continue to be thought of as an extension of individual effects on health, as is reflected in the overbearing concern for "controlling" for individual confounders. Such an approach, arguably, obscures the need to reconceptualize the very notion of "individual effects" that results from incorporating neighborhoods in our conceptual model to elucidate health disparities. The differential response to neighborhood environments by individual Blacks and Whites, demonstrated in our analysis, is a simple illustration of this point.

A second limitation is our focus on fixed, discrete, and hierarchical census-defined contexts to define neighborhoods. While these are important, there may be other nonspatial contexts (e.g., households, nongeographic communities) and nonhierarchical contexts (e.g., workplaces and subjectively defined neighborhoods) that are also important to understanding the patterning of mortality. Thus, in addition to the issue of missing covariates, the issue of missing "levels" remains, especially in multilevel models.

Third, our characterization of neighborhoods was based on a single dimension—poverty. Future researchers might consider systematically developing a "typology" of neighborhoods to take into account the complex interaction of multiple characteristics.

A fourth concern is that it is problematic to demonstrate a "neighborhood effect" on mortality on the basis of cross-sectional observational data, since the magnitude of both neighborhood heterogeneity and neighborhood poverty is for individuals at the time of their death; therefore, the issue of potential mobility (social as well as spatial) over the life course is ignored.

Fifth, the partitioning of variation by different levels (e.g., individual, BG, and CT) in logistic models is not straightforward.[35] Specifically, the magnitude of variance at the BG and CT levels cannot be evaluated for its size in relation to the individual-level variance, since the latter is a known function in logistic models. Thus, the magnitude of CT-level contextual heterogeneity in mortality by race/ethnicity reported here is simply a *conditional* (not absolute or relative) estimate, that is, conditional on the fixed part and the random part of the model at the BG level.

Finally, our findings can be generalized only to populations (of neighborhoods and individuals) that are similar in their characteristics to those in Massachusetts. The replicability of our results in other states or geographic settings is an empirical question warranting further research.

Despite these challenges, quantifying and studying the distribution of mortality across different local geographic units disaggregated by key sociodemographic and economic markers may provide important input into social and health policy as well as sharpen etiological analysis of variations in mortality. For example, our results suggest that while the routine monitoring of how population groups are doing needs to be area-specific, the monitoring of how areas are doing (a prerequisite for developing area-based public health interventions) needs to be population group specific. Such multilevel thinking has yet to permeate the research geared toward public health practice. Such considerations, we believe, may lead to a fairer, and clearly more sensitive and realistic, means of target-setting and evaluation for population groups and areas. If we rely only on across-the-board, average targets for areas, it is difficult to distinguish improvements consisting of changes only in groups that are already "better-off" in those areas. The analytical approach developed here offers a framework for understanding such public health issues.

In conclusion, our analysis points to the importance of generating a quantitative descrip-

tion of the varying impact of neighborhood on health, in relation to diverse population subgroups, rather than focusing solely on average differences in health disparities across these groups. Extending this approach to investigating health patterns over time could provide opportunities to quantify the impact of various social policies on health inequalities between population groups and areas. Indeed, the complex geographic and social variation in important health outcomes such as mortality is an intrinsically important attribute of society that needs to be routinely described and understood. ■

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Contributors

S. V. Subramanian originated the study, analyzed and interpreted the data, and wrote the article. J. T. Chen contributed to the data preparation, interpretation of the results, and editing of the article. D. H. Rehkopf assisted with data analysis and presentation. P. D. Waterman obtained and arranged geocoding of the mortality data. N. Krieger contributed to the interpretation of the results and editing of the article.

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Human Participant Protection

Use of the data employed in this study was approved by all relevant institutional review boards/human subjects committees at the Harvard School of Public Health and the Massachusetts Department of Public Health.

References

1. Barnett E, Casper ML, Halverson JA, et al. *Men and Heart Disease: An Atlas of Racial and Ethnic Disparities in Mortality.* Morgantown: Office for Social Environment and Health Research, West Virginia University; 2001.

2. Casper ML, Barnett E, Halverson JA, et al. *Women and Heart Disease: An Atlas of Racial and Ethnic Disparities in Mortality.* Morgantown: Office for Social Environment and Health Research, West Virginia University; 2000.

3. Casper ML, Barnett E, Williams GI Jr, Halverson JA, Braham VE, Greenlund KJ. *Atlas of Stroke Mortality: Racial, Ethnic and Geographic Disparities in the United States.* Atlanta, Ga: Centers for Disease Control and Prevention; 2003.

4. Devesa SS, Grauman DJ, Blot WJ, Pennello GA, Hoover RN, Fraumeni JF Jr. *Atlas of Cancer Mortality in the United States, 1950–94.* Bethesda, Md: National Cancer Institute; 1999.

5. Geronimus AT, Bound J, Waidmann TA, Hillemeier MM, Burns PB. Excess mortality among Blacks and Whites in the United States. *N Engl J Med.* 1996; 335:1552–1558.

6. Murray CJL, Michaud CM, McKenna MT, Marks JS. *US Patterns of Mortality by County and Race: 1965-1994.* Cambridge, Mass: Harvard Center for Population and Development Studies; 1998. Also available at: http://www.hsph.harvard.edu/organizations/bdu/images/usbodi. Accessed September 10, 2004.

7. *US Decennial Life Tables for 1989 to 1991.* Hyattsville, Md: National Center for Health Statistics; 1997. Also available at: http://www.cdc.gov/nchs/products/pubs/pubd/lftbls/decenn/1991-89.htm. Accessed September 10, 2004.

8. Pickle LW, Mungiole M, Jones GK, White AA. *Atlas of United States Mortality.* Hyattsville, Md: National Center for Health Statistics; 1996.

9. Singh KG, Yu SM. Trends and differentials in adolescent and young adult mortality in the United States, 1950 through 1993. *Am J Public Health.* 1996;86: 560–564.

10. *Health, United States, 2002 With Chartbook on Trends in the Health of Americans.* Hyattsville, Md: National Center for Health Statistics; 2002.

11. Kington RS, Nickens HW. Racial and ethnic differences in health: recent trends, current patterns, future directions. In: Smelser NJ, Wilson WJ, Mitchell F, eds. *America Becoming: Racial Trends and Their Consequences.* Washington, DC: National Academy Press; 2001.

12. O'Campo P. Invited commentary: advancing theory and methods for multilevel models of residential neighborhoods and health. *Am J Epidemiol.* 2003; 157(1):9–13.

13. Subramanian SV, Jones K, Duncan C. Multilevel methods for public health research. In: Kawachi I, Berkman LF, eds. *Neighborhoods and Health.* New York, NY: Oxford University Press; 2003.

14. Yen IH, Kaplan GA. Neighborhood social environment and risk of death: multilevel evidence from the Alameda County Study. *Am J Epidemiol.* 1999;149: 898–907.

15. Cubbin C, LeClere F, Smith G. Socioeconomic status and injury mortality: individual and neighbourhood determinants. *J Epidemiol Community Health.* 2000;54: 517–524.

16. Krieger N, Chen J, Waterman PD, Soobader MJ, Subramanian SV, Carson R. Geocoding and monitoring US socioeconomic inequalities in mortality and cancer incidence: does choice of area-based measure and geographic level matter? The Public Health Disparities Geocoding Project. *Am J Epidemiol.* 2002;156:471–482.

17. US Census Bureau. Geographical Areas Reference Manual. Available at: http://www.census.gov/geo/www/garm.html. Accessed February 5, 2004.

18. *Race and the Elimination of Health Disparities in the City of Boston: Promoting the Health of the Underserved.* Boston, Mass: Boston Public Health Commission; 2002.

19. Health Resources and Services Administration. Health Professional Shortage Areas. 2002. Available at: http://bhpr.hrsa.gov/shortage. Accessed September 17, 2004.

20. Monmonier M. *Cartographies of Danger: Mapping Hazards in America.* Chicago, Ill: University of Chicago Press; 1997.

21. Krieger N, Waterman P, Lemieux K, Zierler S, Hogan JW. On the wrong side of the tracts? Evaluating the accuracy of geocoding in public health research. *Am J Public Health.* 2001;91:1114–1116.

22. Subramanian SV, Duncan C, Jones K. Multilevel perspectives on modeling census data. *Environ Plan A.* 2001;33:399–417.

23. Krieger N, Chen J, Waterman PD, Soobader MJ, Subramanian SV, Carson R. Choosing area-based socioeconomic measures to monitor social inequalities in low birthweight and childhood lead poisoning: the Public Health Disparities Geocoding Project. *J Epidemiol Community Health.* 2003;57(3):186–199.

24. Krieger N, Waterman PD, Chen J, Soobader MJ, Subramanian SV. Monitoring socioeconomic inequalities in sexually transmitted infections, tuberculosis, and violence: geocoding and choice of area-based socioeconomic measures—The Public Health Disparities Geocoding Project (US). *Public Health Rep.* 2003; 118(3):240–260.

25. US Census Bureau. Poverty areas. Available at: http://www.census.gov/population/socdemo/statbriefs/povarea.html. Accessed February 5, 2004.

26. Census of Population and Housing, 1990: Summary Tape File 3 on CD-ROM Technical Documentation. Washington, DC: US Census Bureau; 1991.

27. Goldstein H. *Multilevel Statistical Models.* 3rd ed. London, UK: Arnold Publishers; 2003.

28. Longford N. *Random Coefficient Models.* Oxford, UK: Clarendon Press; 1993.

29. Raudenbush S, Bryk A. *Hierarchical Linear Models: Applications and Data Analysis Methods.* Thousand Oaks, Calif: Sage Publications; 2002.

30. Goldstein H, Rasbash J. Improved approximations for multilevel models with binary responses. *J R Stat Soc Ser A Stat Soc.* 1996;159:505-513.

31. Rasbash J, Browne W, Goldstein H, et al. *A User's Guide to MLwiN, Version 2.1.* London, UK: Multilevel Models Project, Institute of Education, University of London; 2000.

32. Collet D. *Modelling Binary Data.* London, UK: Chapman & Hall; 1991.

33. Krieger N, Rowley DL, Herman AA, Avery B, Phillips MT. Racism, sexism, and social class: implications for studies of health, disease, and well-being. *Am J Prev Med.* 1993;9(suppl):82–122.

34. Williams DR, Collins C. US socioeconomic and racial differences in health: patterns and explanations. *Annu Rev Sociol.* 1995;21:349–386.

35. Goldstein H, Browne WJ, Rasbash J. Partitioning variation in multilevel models. *Understanding Statistics.* 2002;1:223–232.