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Effect of specific aspects of community social environment on the mortality of Individuals diagnosed with serious illness

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Abstract

The purpose of this study was to examine the prospective and contextual effects of urban community environment on mortality among Medicare beneficiaries who were 67 years old or older in 1993, lived in the city of Chicago, and were hospitalized for one of 13 serious diseases. As expected, we found that advantageous socioeconomic context helps lower mortality risk among elderly patients over and above individual demographic and health background. We also found that collective efficacy was a health-enhancing social resource whereas criminal and violent activities in the community appeared to be deleterious. Inconsistent with our hypotheses, community social network density (measured by the size of social network and frequency of social interaction) was not protective but detrimental. Moreover, social support and the civic involvement of residents in the community do not seem to affect mortality. The complex relationship between community social environment and health found in this study may suggest that community-level social interventions based on social capital/social cohesion models are not likely to achieve fruitful results without concomitant effort in the economic and health care realm, at least in terms of influences on the health of older people once they are already ill.

Keywords: Neighborhood/community; Aging; SES; Collective efficacy; Social network density; Crime; Mortality

Introduction

Health and health care are key products of social systems. One reflection of this phenomenon is the contextual impact of residential communities on health outcomes. Evidence from a large number of studies has supported the hypothesis that living in neighborhoods of low socioeconomic status (SES) has negative effects on health-related outcomes including but not limited to self-rated health, low birth weight, depression, disease incidence, physical functioning, number of chronic conditions, psychological distress and life satisfaction, health-behaviors (e.g., substance use), and mortality (Anderson, Sorlie, Backlund, Johnson, & Kaplan, 1997; Aneshensel & Sucoff, 1996; Boardman, Finch, Ellison, Williams, & Jackson, 2001; Chang & Christakis, forthcoming; Diez-Roux et al., 1997; Ennett, Flewelling, Lindrooth, & Norton, 1997; Kawachi, & Berkman, 2003a; LeClere, Rogers, & Kimberley, 1997; O'Campo, Xue, & Wang, 1997; Ramirez-Valles, Zimmerman, & Newcomb, 1998; Robert, 1998; Ross & Mirowsky, 2001; Yen & Kaplan, 1999). The influence of community

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social environment on health also has been examined. Although the literature is relatively sparse, results of a handful of ecological and multi-level studies have shown that the social resources of a residential area are important community-level factors that affect health (Browning & Cagney, 2002; Kawachi, Kennedy, & Glass, 1999; Lochner, Kawachi, Brennan, & Buka, 2003; Veenstra, 2002; Wen, Browning, & Cagney, 2003).

Identifying the collective characteristics of communities that contribute to population health status is clearly important (Kawachi & Berkman, 2000). In this pursuit, it is helpful to recognize that the patterns of the relationship between community and health may differ across different subgroups. For example, older people may be more influenced by the environment of their residential area because they may spend more time at home, rely more on local resources to achieve a better quality of life, and be more emotionally attached to their communities. Although it has long been recognized that "place effects" are more salient for older people (Diez-Roux, 2002; Krause, 1996; Robert & Li, 2001), research using a *prospective* design to investigate this relationship is limited.

This study was designed to examine community effects on mortality of people over age 67 after they were hospitalized for the onset of 1 of 13 serious conditions in 1993. The primary goal of this study is to evaluate any prospective and contextual effects of residential community on the *individual* hazard of death for seriously ill older patients starting from the point of their diagnosis. We examine two broad aspects of community environment: economic structure (e.g., concentrated affluence), and social environment (e.g., collective efficacy). We develop and use data with detailed measures regarding both individual health and community social attributes.

Background

Community effects on mortality in later life

While findings from neighborhood research generally point to a protective effect of living in a higher SES community (Kawachi & Berkman, 2003b; Robert, 1999; Robert & House, 2000; Yen & Syme, 1999), the contextual effect of community environment on mortality among the elderly has not been firmly established. To date, only a handful of studies have used prospective contextual designs to investigate community effects on the mortality of elderly people, and a consistent pattern has not yet emerged.

Waitzman and Smith (1998a) conducted a prospective multilevel study of adults in the 1971–1974 wave of the National Health and Nutritional Examination Survey (NHANES I). After controlling for individual characteristics, they found a significant contextual effect of living in federally designated poverty areas on all-cause mortality in younger adults (25–54) but not in subjects over 54 years old. They then concluded that residence in poverty areas contributed to socioeconomic gradients in mortality among non-elderly adults in the United States. Anderson et al. (1997) linked data from the National Longitudinal Mortality Study to census tract information to assess 11-year mortality risk among black and white men and women associated with median census tract income. After adjusting for individual family income, they found significant area effects on mortality among persons age 25–64 years but not for persons 65 years or greater. Similar findings were reported in an earlier study conducted by Haan, Kaplan, and Camacho (1987) where the effect of residence in a poverty area on 9-year risk of all-cause mortality was highest in the younger group (45–64) but was non-significant for persons age 65 years or older.

One interpretation for the weakened area effect on mortality in old age is that the area measures that have been used in previous studies may not be as relevant to older people. Indeed, alternative community measures may exhibit stronger contextual effects on mortality among the elderly. For example, using the National Health Interview Surveys (NHIS), Waitzman and Smith (1998b) conducted a second contextual analysis of neighborhood effects on mortality focusing on 33 of the largest Metropolitan Statistical Areas (MSAs) in the United States. In this study, they found that concentrations of poverty were significantly associated with elevated risk of mortality for both elderly and non-elderly residents of large urban areas in the United States during the late 1980s and early 1990s. Moreover, they found that urban concentrated affluence had a consistent and robust protective effect against mortality among the elderly (65+) whereas the affluence effect was only sporadic among the younger cohort (30–64). The authors speculated that the disparate findings regarding area effects on the mortality risk of the elderly between their two analyses may be attributable to different sample characteristics and different area measures and geographic units used in the analyses. Recently, Cagney et al. (forthcoming) also documented that concentrated affluence in the neighborhood, rather than concentrated poverty, was significantly associated with self-rated health of Chicago residents age 55 or older.

Another plausible explanation is that the absence of a mortality effect associated with residing in poor areas among the elderly may be due to the so-called "cross-over" effect or "selective mortality." Presumably, the most disadvantaged individuals afflicted with poor life conditions have higher risk of death prior to reaching the elderly stage of the life course, leaving survivors in this group genetically and/or psychological more robust

than their counterparts living in more advantaged social conditions (Glass & Balfour, 2003; Waitzman & Smith, 1998a). As a result, social disparities in health may dwindle in very old age, including those associated with residential areas.

Third, the observed weaker effect of neighborhood context on mortality in old age may be due to high prevalence of mortality among the elderly, which would make it more difficult to detect risk factors that are common in younger populations (Glass & Balfour, 2003; Kaplan, Haan, & Wallace, 1999).

If the "cross-over-effect" and the "high-mortality-rate" phenomena are truly at work contributing to the observed deterioration of neighborhood impact on mortality in the late stage of the life course, we should then expect a non-significant or an even weaker area effect on mortality in a more vulnerable group—i.e., an elderly patient population with serious diseases—especially if we control for their baseline health status. It is arguable that the mortality selection bias is less of a problem among seriously ill elderly patients who have a higher frequency of mortality than a general elderly population. Hence, evidence regarding an elevated mortality risk associated with disadvantaged areas in this group of elderly would suggest that area effects on mortality in later life may have been underestimated in previous work. Focusing on an elderly patient population in the neighborhood effects research should, in a unique way, shed light on the debate regarding whether area effects on mortality extend to later life.

Nevertheless, there has not been much evidence on whether area of residence exerts a contextual effect on the mortality of elderly patients after the initial onset of disease. Most neighborhood research on aging has studied elderly people in general (Glass & Balfour, 2003). But even if a contextual mortality effect were to be found in this type of research, it is not possible to know whether this is due to differences in disease incidence or case fatality rates (Horne et al., 2004). Several studies have tested area effects on mortality following a specific type of condition such as cardiovascular diseases and cancer and have documented a significant link between residence in low-SES areas and increased risk of dying from theses diseases (Horne et al., 2004; Kapral, Wang, Mamdani, & Tu, 2002; O'Malley, Le, Glaser, Shema, & West, 2003). However, no evidence is available on whether neighborhood environment (e.g., SES and/or other social resources) contextually and prospectively affects mortality of seriously ill and elderly patients suffering from a broad variety of medical conditions. Studying area effects on mortality in this group is valuable in that findings from such research should hold considerable promise expanding previous evidence on contextual effects into a secondary-prevention population with known life-threatening diseases.

Theoretical perspectives

The hypothesis that community SES matters to health has intuitive appeal because communities rich in economic and educational resources are generally more equipped to provide for residents with better physical (e.g., high-quality housing), social (e.g., solidarity and safety), service (e.g., medical and other social services), and cultural environments (e.g., prevailing positive attitudes) (Macintyre & Ellaway, 2003; Macintyre, Maciver, & Sooman, 1993; Robert, 1999).

Several of these plausible pathways linking community SES and health have been confirmed in empirical studies (Browning & Cagney, 2002; Ross & Mirowsky, 2001; Wen et al., 2003). For example, in a multi-level study, Wen et al. (2003) found that an index of community social resources (e.g., collective efficacy and reciprocity) and an index of physical environment largely explained the observed link between self-rated health and concentrated affluence at the neighborhood level after adjusting for social, demographic, and behavioral factors at the individual level. The effect of health-promoting social resources also has been examined in their own right. For instance, Browning and Cagney (2002) showed that collective efficacy had a significant and positive effect on self-rated health even after controlling for individual demographic and health background as well as relevant neighborhood-level variables. Lochner, Kawachi, and Brennan et al. (2003) also reported that neighborhood social capital—as measured by reciprocity, trust, and civic participation—was associated with lower neighborhood death rates, after adjustment for neighborhood material deprivation.

On the other extreme, spatially distributed negative social indicators such as crime and violence also have been examined in neighborhood research. For example, as early as the 1940s, Shaw and McKay (1969) elaborated the concept of social disorganization and demonstrated that social problems such as crime and violence, poverty, prevalence of tuberculosis, and prevalence of mental disorders were geographically clustered. One major thesis in the social disorganization theory is that geographic variation in criminal events can be attributed to the strength or absence of local social cohesion and social control. Shaw and McKay (1969) argued that more socially organized neighborhoods or communities were better able to control youth deviant behaviors and maintain local social order. More recently, collective efficacy theorists explicitly stated and empirically substantiated that some communities had lower ability to realize the common values of residents and maintain effective social controls—consequently, they had more reports of neighborhood perceived violence and personal victimization (Sampson, Raudenbush, & Earls, 1997).

The previous literature suggests that enduring social inequalities in health may in part originate from the unequally distributed social resources and hazards of urban communities. It is arguable that the effects of local social resources such as social control and social cohesion, as well as social hazards such as crime and violence, should be examined at the neighborhood or community level, rather than a larger geographic aggregation, because local communities can capture the immediacy of social context that may be lost in cross-metropolitan analyses (Guest, Almgren, & Hussey, 1998). This is particularly true for older people, who have been afflicted with serious diseases and consequently are less mobile and more reliant on local services and supports compared with their healthy and younger neighbors. After all, part of the reason our residence is important to our health is that, as "spatial animals" (Fitzpatrick & Lagory, 2000), we are psychologically, emotionally, and physically affected by the milieu of our everyday living space. The objective and perceived conditions of our residential environment are important markers of our quality of life and may structure our opportunities. Places rich in social resources are usually safe, clean, convenient, and have sufficient health-enhancing services. Conversely, a disorganized social fabric coupled with poverty is characteristic of disadvantaged neighborhoods; residents of these distressed neighborhoods are more often exposed to social and environmental health hazards such as noise, violence, crime, poor physical conditions, and poor opportunities for social networking. Indeed, these patterns have been routinely observed by early and recent Chicago school scholars whose work has consistently shown that social disorganization, typically measured by low collective efficacy (lack of social cohesion and informal social control), family disruption, and other structural disadvantages, were strongly associated with higher crime and violence rates (Sampson & Groves, 1989; Sampson et al., 1997; Shaw & McKay, 1969). It is conceivable that neighborhood social hazards such as crime and violence not only directly harm victims but also have indirect detrimental effects on the physical and psychological health of all residents.

Other mechanisms underlying the place-health relationship involve access to health and social services, physical environment, subcultural orientation, political processes, and neighborhood reputation (Fitzpatrick & Lagory, 2000; Kawachi et al., 1999; Macintyre, Ellaway, & Cummins, 2002). To keep our research focused, however, we chose to emphasize two important aspects of community environment—economic context and social environment—in this prospective and contextual study of how place affects mortality among seriously ill elderly adults. We aim to decompose the effects of multiple aspects of the social environment on mortality and investigate the role of the social environment in the routinely observed relationship between residential economic context and health.

Hypotheses

Based on previous theoretical and empirical developments, we hypothesize that, over and above individuals' demographic attributes and baseline health status, (1) higher community SES is protective against mortality among older patients; (2) the social resources and social hazards of residential areas are strong contextual forces that affect the likelihood of mortality after the onset of serious disease in later life; (3) crime and violence, as markers of social hazards, help explain the effects of community health-promoting social resources; and (4) the social environment can, at least partially, explain the effects of community SES on mortality.

Data and methods

Data sources

Three data sources were used in this study: the 1990 Decennial Census, the 1994–95 Project on Human Development in Chicago Neighborhoods—Community Survey (PHDCN-CS), and the Care after the Onset of Serious Illness (COSI) data set (1993–1999). ZIP code boundaries were used to define residential communities and to link the three data sources into one merged file. Although ZIP code boundaries do not perfectly circumscribe neighborhoods, they do represent local residential areas and are frequently used in studies of neighborhoods (Finch, Kolody, & Vega, 1999; Lipton & Gruenewald, 2002; Merkin, Stevenson, & Powe, 2002; Zwanziger, Mukamel, & Indridason, 2002). We studied 12,672 patients from the COSI data residing in 51 ZIP code areas in Chicago.[1] Geo-coding to smaller levels of aggregation (e.g., census tracts) was not possible because of sample size constraints and data limitations.

Decennial census

Measures of *contextual SES* were derived from the 1990 Census, including contextual ZIP code level affluence, poverty, and education.

PHDCN-CS

Measures of *contextual social environment* were constructed from the PHDCN-CS (Sampson et al., 1997). The PHDCN-CS is a probability sample of 8782 residents of Chicago focusing on respondents' own assessments of the communities in which they live. Each

[1] In 1990, there were on average approximately 50,000 persons per ZIP code area in Chicago.

record in the PHDCN-CS data set was linked to a census block group in Chicago. Using the geographic centroids of census block groups, we linked each record in the PHDCN-CS with a corresponding ZIP code. Thereafter, individual data from the PHDCN-CS were aggregated to the ZIP code level. On average, there were 293 PHDCN respondents per ZIP code.

COSI

The core data of COSI are rooted in the 1993 inpatient hospitalization records from the Health Care Financing Administration's Medicare program.[2] The COSI data set consists of a cohort of patients *newly* diagnosed in 1993 with one of thirteen serious illnesses and were followed for up to 6 years. These conditions include acute myocardial infarction (MI), congestive heart failure (CHF), central nervous system (CNS) cancer, colorectal cancer, hip fracture, head/neck cancer, liver/biliary cancer, leukemia, lung cancer, lymphoma, pancreatic cancer, stroke, and urinary cancer. The thirteen diseases were selected because they met several COSI conceptual criteria including accuracy of ascertainment of incident cases and high prevalence. Prior detailed empirical work provided guidance for us to capture incident cases. For example, prior research has demonstrated that for lung, colon, and esophageal cancers, 3 years of look-back in the Medicare claims was adequate to eliminate prevalent cases. That is, if an individual had not been hospitalized in the prior 3 years before the putative index hospitalization for onset of his/her serious disease, it was very likely that they had never previously been hospitalized for the disease. Detailed descriptions about how COSI data were constructed have been published elsewhere (Christakis, Iwashyna, & Zhang, 2002).

In summary, the final data used in this study consist of ecological measures of economic and social context at the ZIP code level along with individual level data (i.e., age, race, sex, poverty status, diagnostic category, comorbidity,[3] and survival time after disease inception). Given that Medicare covers nearly 96% of the elderly population in the US (Hatten, 1980), this research is essentially a complete population study of patients who were 67 years old or over,[4] who resided in the City of Chicago, and who were hospitalized for the onset of one of 13 serious diseases in 1993.

Dependent variable

Our health outcome was the relative hazard of death for COSI cohort members. The survival time was defined as the time period (number of days) from the date of the index hospitalization for the onset of his/her disease to death or to the end of the study (i.e., June 30, 1999). Survival times of people who were still alive on June 30, 1999 were right censored.

Independent variables

Individual controls

Individual demographic and baseline health measures controlled in the analyses include diagnosis, age, gender, race (white versus non-white), three continuous comorbidity scores for 3 years prior to the index hospitalization, and a dichotomous indicator of Medicaid receipt at any point in 1993 as a proxy measure of individual income status (poverty). Table 1 illustrates characteristics of COSI patients in Chicago. The average age of this cohort is about 79 with 13% in poverty. For the COSI cohort, stroke, MI, CHF, hip fracture and lung cancer are the most common conditions.

[2]Medicare is a federally sponsored health insurance program administered by the Centers for Medicare and Medicaid Services (CMS) whose beneficiaries include more than 96% of *all* US citizens aged 65 and older, whether they use health care or not (Hatten, 1980). CMS maintains billing records of outpatient, inpatient, and other claims for all beneficiaries not enrolled in risk contract health maintenance organizations (HMOs).

[3]Comorbidity is measured by the Charlson method (Charlson, Pompei, Ales, & MacKenzie, 1987), which is extremely popular and has been used extensively in claims data research (Christakis et al., 2002; Christakis, Iwashyna, & Zhang, 2002). Having examined the effects of alternative data sources and lookback periods on the performance of Charlson scores in the prediction of mortality following hospitalization, Zhang, Iwashyna, and Christakis (1999) have found that, compared

(*footnote continued*)
with a 1-year lookback involving solely inpatient claims, statistically and empirically significant improvements in the prediction of mortality were obtained by incorporating alternative sources of data (particularly 2 years of inpatient data and 1 year of outpatient and auxiliary claims), but only if they were entered into the regression simultaneously. Although they parameterized the Charlson score as indicator variables, they also tested a linear, continuous specification of the Charlson score and found by and large the same patterns. In our sample, 3 years of in-patient Charlson scores were available, all of which were parameterized as linear, continuous measures and were entered into our regression model distinctly as controls for pre-hospitalization health status.

[4]Models that involve comorbidity as a covariate, such as ours, require us to impose a criterion of $\geqslant 67$ years of age because patients who were less than 67 at the time of 'diagnosis' could not have had Medicare claims filed for an antecedent 2-year period (since, with certain exceptions, such as dialysis patients, people do not become eligible for Medicare until they are 65). This (2-year) duration of retrospective ascertainment of health problems has been shown by us and others to be adequate for the detection of prevalent chronic conditions (McBean, Warren, & Babish, 1994; Zhang, Iwashyna, & Christakis, 1999).

Table 1
Characteristics of COSI patients in Chicago

Variables	Mean	St. dev.
Demographic		
Age	78.598	7.204
Male	0.403	0.491
Medicaid recipient	0.130	0.339
Race (white)	0.676	0.468
Baseline Health Status (in 1993)[a]		
Charlson score for year 1	1.356	1.198
Charlson score for year 2	1.238	0.977
Charlson score for year 3	1.182	0.873
Baseline Diagnosis (in 1993)		
Acute myocardial infarction	0.159	0.365
Congested heart failure	0.241	0.428
CNS	0.004	0.063
Colorectal cancer	0.069	0.252
Hip fracture	0.134	0.339
Head/neck cancer	0.010	0.098
Liver/biliary cancer	0.008	0.089
Leukemia	0.013	0.115
Lung cancer	0.070	0.255
Lymphoma	0.021	0.144
Pancreatic cancer	0.014	0.116
Stroke	0.229	0.421
Urinary cancer	0.029	0.166

$N = 12{,}672$.

[a]Baseline health status was measured by the Charlson scores for the first, the second and the third year of lookback.

Community characteristics

Measures of community SES were obtained or constructed from the 1990 US Census Summary Tape File STF 3B (data at the ZIP code level). These measures include the percentage of residents with household annual incomes $50,000 or over *(concentrated affluence*), the percentage of households in a neighborhood that were below the Federal poverty threshold in 1990—$13,359 for a household of four (*concentrated poverty*), and the percentage of college graduates (*aggregate education*). Because the three factors represent important dimensions of SES and are highly correlated, we integrated them into a summary measure labeled *contextual SES,* weighted by factor loadings that ranged from 0.71 to 0.94.[5] The neighborhood SES scale has a high level of internal consistency (Cronbach's $\alpha = 0.83$). Higher scores indicate higher SES.

Ecological measures of community social environment were constructed from the PHDCN-CS. Following the operationalization of previous works (Browning & Cagney, 2002; Sampson et al., 1997), the *collective efficacy* scale was constructed through combining items of social cohesion and informal social control. The social cohesion items from the PHDCN-CS assessed the respondent's level of agreement (on a five-point scale) with the following statements: (1) "People around here are willing to help their neighbors," (2) "This is a close-knit neighborhood," (3) "People in this neighborhood can be trusted," (4) "People in this neighborhood generally don't get along with each other," and (5) "People in this neighborhood do not share the same values." The last two items were reversely coded. Informal social control was tapped through respondents' level of agreement with the following two statements: (1) "You can count on adults in this neighborhood to watch out that children are safe and don't get in trouble" and (2) "People in their neighborhood would intervene if a fight broke out in front of their house." Social cohesion and informal social control were closely correlated across the ZIP code areas ($r = 0.92$; $p < 0.0001$). The seven items were combined to form a single scale of health-related collective efficacy. The reliability of the collective efficacy scale was 0.80.[6]

The *social network density* scale encompassed a number of items measuring the size of neighborhood-based kinship/friendship networks as well as the frequency of parties and visits among neighbors. The four relevant items were based on questions asking PHDCN-CS respondents (1) "How often do you and people in this neighborhood have parties or other get-togethers where other people in the neighborhood are invited?" (2) "How often do you and other people in this neighborhood visit in each other's homes or on the street?" (3) "How many relatives or in-laws do you have in the neighborhood?" and (4) "How many friends do you have in the neighborhood?" Unlike the social support and sociability scale examined in previous work (Browning & Cagney, 2003) which combined objective and functional measures of neighborhood social networking, this scale specifically taps the size of social networks and the extent of social integration at the community level. The purpose here is to detect different effects on health between the objective structure of social networks and the quality of social interaction in community life. The reliability of the social network density scale was 0.54.

The *social support* scale contained four items corresponding to the following questions: (1) "How often do you and other people in the neighborhood ask each other advice about personal things such as child-rearing or job openings?" (2) "How often do you and people in your neighborhood do favors for each other?" (3) "When a neighbor is not at home, how often do you and other neighbors watch over their property?" (4) "If I

[5]These factor loadings were produced from orthogonal principal factor analyses (Harman, 1976).

[6]For detailed discussion of neighborhood reliability, see Raudenbush and Sampson (1999).

were sick I could count on my neighbors to shop for groceries for me?" These items attempt to tap the instrumental and informational dimensions of actual or perceived social support within the community. The reliability of the social support scale was 0.76.

Measures *of local organizations* and *voluntary associations* are intended to capture institutional neighborhood processes (Morenoff, Sampson, & Raudenbush, 2001). *Local organizations* is an index of the number of survey-reported organizations and programs in the neighborhood—the presence of a community newspaper, block group or tenant association, crime prevention program, alcohol/drug treatment program, mental health center, or family health service. The reliability of local organizations was 0.82. *Voluntary associations* taps the civic involvement of residents in (1) local religious organizations; (2) neighborhood watch programs; (3) block group, tenant associations, or community councils; (4) business or civic groups; (5) ethnic or nationality clubs; and (6) neighborhood ward groups or local political organizations. The reliability of voluntary associations was 0.70.

Social hazards of residential areas were tapped by two variables. First, the *personal victimization* scale measures the frequency of victimization that the residents personally experienced in the last 6 months. The reliability of this scale was 0.56. Second, the *perceived violence* scale was created to reflect the perceived prevalence of armed fights, violent arguments between neighbors, gang fights, sexual assault or rape, and robbery or mugging. The reliability of the perceived violence scale was 0.91.

In order to parsimoniously examine the effect of community social environment on mortality, we created a global measure of community social environment. Based on a series of orthogonal principal factor analyses, collective efficacy, social support, and perceived violence appeared to be clustered around a single concept. Their factor loadings in absolute value ranged from 0.63 to 0.92. A factor score was then generated and labeled as *contextual social index*. The contextual social index has reasonably good internal consistency (Cronbach's $\alpha = 0.81$) and is significantly and positively correlated with the contextual SES scale ($r = 0.52$; $p = 0.0001$). Higher scores indicate more social resources in the community.

Table 2 displays the correlation matrix of these ZIP code level variables, all of which are standardized. The higher the scores of these variables, the greater amount of either social resources or problems present in the community. As expected, community SES is positively associated with collective efficacy and is negatively correlated with crime and violence measures. Contrary to our a priori expectation, the higher the level of social network density, the lower the level of community SES, and the more criminal and violent problems present. Social network density also appears to be negatively correlated the prevalence of local organizations.

Analytical strategy

Ecometric approach to assessing ecological context

Instead of simply computing neighborhood mean scores from individual responses to the survey questions

Table 2
Correlation matrix of ZIP code area characteristics

	1	2	3	4	5	6	7	8	9
1. Contextual SES	1.000								
2. Collective efficacy	0.470**	1.000							
3. Social support	0.123	0.642***	1.000						
4. Social network density	−0.551***	−0.180	0.045	1.000					
5. Local organizations	0.154	0.098	−0.145	−0.391**	1.000				
6. Voluntary associations	0.206	0.388**	0.515***	0.077	−0.074	1.000			
7. Perceived violence	−0.721***	−0.772***	−0.339**	0.336**	0.031	−0.199	1.000		
8. Personal victimization	−0.215	0.111	0.269^	0.412**	−0.444***	0.332**	−0.009	1.000	
9. Contextual social index	0.517***	0.992***	0.672***	−0.199	0.053	0.395**	−0.828***	0.117	1.000

ZIP code $N = 51$.
^$p \leqslant 0.10$; *$p \leqslant 0.05$; **$p \leqslant 0.01$; ***$p \leqslant 0.001$.
a. Contextual SES is a factor score based on % of household with annual income $50,000 or over (concentrated affluence), % of household in poverty (concentrated poverty), and % of college graduates (aggregate education).
b. Collective efficacy is a summary measure of social cohesion and informal social control.
c. Higher scores of collective efficacy, social network density, social support, local organizations, voluntary associations, perceived violence, and personal victimization indicate higher stocks in these social resources or hazards.
d. Contextual social index is a factor score based on collective efficacy, social support, and perceived violence. Higher scores indicate more social resources in the community.

to assess community social environment, we replicated the operationalization of Sampson et al. (1997) using a three-level linear item response model (Rasch model). As an illustrative example, we describe the method used to construct the measure of collective efficacy at the ZIP code level.

At level one (within individuals)—the seven items comprising the collective efficacy scale were modeled as follows:

$$Y_{ijk} = \pi_{jk} + \alpha_i + e_{ijk},$$

where Y_{ijk} is the response to item i of person j in ZIP code k, π_{jk} is the intercept and is interpreted as the respondent's latent perception of collective efficacy in neighborhood k, α_i represents the effects of item-specific factors such as item difficulty, and e_{ijk} is the item-specific measurement error.

At level two (between individuals)—respondent-specific latent perceptions of collective efficacy were adjusted for the individual-level characteristics of the PHDCN-CS respondents as follows:

$$\pi_{jk} = \eta_k + \sum_{q=1}^{7} \beta_q x_{qjk} + r_{jk} \qquad r_{jk} \sim N(0, \sigma^2),$$

where η_k is the intercept and represents the mean perception of collective efficacy in ZIP code k, X_{qjk} is the value of person-level predictor q for individual j in neighborhood k, B_q is the effect of q on individual j's expected score, and r_{jk} is an independently, normally distributed error term with variance σ^2 (within-area variance). Controlling for response bias, these models adjust for seven individual variables including age, gender, race, education, family income, marital status, and years of residence in the ZIP code area. The "individuals" here are those who participated in the PHDCN-CS.

At level 3, each ZIP code area's mean collective efficacy varies randomly around the grand mean:

$$\eta_k = \gamma + u_k \qquad u_k \sim N(0, \tau^2),$$

where γ is the grand mean collective efficacy across the ZIP code areas, u_k is a normally distributed random effect associated with ZIP code area k, and τ^2 is the between-area variance.

According to this setup, the object of measurement is η_k—the average perception of collective efficacy in ZIP code k. To account for measurement error and missing data, we used Empirical Bayes (EB) residuals to measure community collective efficacy (Bryk & Raudenbush, 1992; Whittemore, 1989). The entire three-level model was estimated simultaneously via maximum likelihood.

This method has been coined as ecometric approach to assessing ecological context from survey data (Raudenbush & Sampson, 1999).

All the scales of community social environment including *collective efficacy, social support, social network density, local organizations, voluntary associations, perceived violence*, and *personal victimization* were constructed using the ecometric method. These EB estimators of ZIP code area social environment were then used as independent covariates in the subsequent multivariate models of the mortality hazards using the individual-level COSI data.

Statistical models

After data construction, a series of Cox proportional hazards models were fit to test the independent effects of area characteristics on the individual hazard of death among the members of the COSI cohort (Collett, 1996). Instead of using random effects or hierarchical modeling techniques, we conducted survival analyses fitting Cox proportional hazards models with robust standard errors to analyze our survival data (which contains a substantial amount of censoring).[7] Of 12,672 patients, 3,211 patients (25.34%) were censored at the end of the study.[8] The Breslow method was adopted to handle tied values.[9] The proportionality assumption of Cox models was tested.[10] The test was based on the generalization by

[7]Because our data are clustered by ZIP code, the Huber-White robust method of calculating the variance-covariance matrix was used to obtain standard errors for the model coefficients that account for correlation among individuals in the same ZIP code area (Lin & Wei, 1989).

[8]Death dates were obtained from the highly accurate Vital Status file of the Health Care Administration as of July 6, 1999 (Christakis et al., 2002; Christakis, Iwashyna, & Zhang, 2002). This file is updated regularly from the Social Security Administration. This file has been shown to be highly accurate (Kestenbaum, 1992).

[9]When there are tied failure times, we must decide how to handle the calculation of the risk pools for these tied observations. Assume that there are two subjects that died on the same day. In the calculation involving the second person, the first person is not in the risk pool since failure (or death) has already occurred. We employ the Breslow method for handling tied values which uses the largest risk pool for each of the tied failure events (Breslow, 1974).

[10]For all the models presented here, we tested the proportionality assumption. First, we found that poverty (receiving Medicaid) and race (non-white/white) and some indicators of diagnosis were the most significant violators. However, while the chi-square tests were significant, the slopes were rather small (for example, in the model including contextual SES, ρ for poverty was 0.03 and ρ for race was 0.02). The existence of slight violations such as these is actually not surprising given the size of the data set. Next, we refit these Cox models stratified on poverty and race. We found that none of the community effects was sensitive to this correction. The estimated coefficients and significance levels were quite similar with or without stratification. We further examined the proportionality assumption by fitting models that include interaction terms between community measures and time-at-risk. Again, our findings on the community effects were not subject to the proportionality assumption. Therefore, we decided to present the results without correcting the proportionality violation of the individual-level variables.

Table 3
Cox Proportional Hazards Model (assessing the contextual effects of different dimensions of community SES)

Independent variables	Models				
	1	2	3	4	5
Individual level variables					
Age	1.050***	1.050***	1.050***	1.050***	1.050***
Male	1.310***	1.311***	1.311***	1.309***	1.310***
Poverty (medicaid recipient)	1.104**	1.093**	1.092**	1.103**	1.092*
Race (non-white/white)	1.047*	1.015	1.016	1.033	1.013
Charlson score (lookup yr 1)	1.106***	1.106***	1.106***	1.106***	1.106***
Charlson score (lookup yr 2)	1.061***	1.061***	1.061***	1.061***	1.061***
Charlson score (lookup yr 3)	1.028*	1.028*	1.028*	1.028*	1.028*
Zip code level variables					
Concentrated poverty		1.029*			
Concentrated affluence			0.965**		
Aggregate education				0.974**	
Contextual SES					0.958**

* $p \leqslant 0.05$; ** $p \leqslant 0.01$; *** $p \leqslant 0.001$ (two-tailed tests).
a. Zip code level $N = 51$; Individual level $N = 12{,}672$.
b. The range of education, poverty, and affluence is between 0 and 1.
c. Contextual SES is a scale that contains concentrated poverty, concentrated affluence, and aggregate education.
d. The estimates presented in this table are hazard ratios
e. To save space, the effects of individual diagnosis are not presented.

Grambsch and Therneau (1994). Both stratified Cox models and models that include the interaction terms between community measures and time-at-risk were fit to examine the sensitivity of findings from non-stratified Cox models to violations of the proportionality assumption.

Results

Analytical results are presented in Tables 3–5. All statistical models have adjusted for age, gender, poverty status, race, diagnosis, and baseline comorbidity.

Table 3 reports the results of five Cox proportional hazards models, assessing the effects of several dimensions of contextual SES on relative hazard of death, independent of individual risk factors (the first hypothesis). Models 3.2 through 3.4 examine the three aspects of community SES respectively. Concentrated affluence and aggregate education have a significant and protective effect against mortality in seriously ill older people, whereas concentrated poverty is a strong positive predictor of mortality. A natural step following these separate models would be to fit a model examining affluence, poverty, and education simultaneously to assess their relative contributions to mortality. However, in this analysis, all the effects were rendered non-significant, potentially due to multi-collinearity; correlation coefficients in absolute value among these SES variables range from 0.42 to 0.79 with all p-value smaller than 0.0001. Next, we examined a composite measure of contextual SES and present the results in model 3.5. Overall, community SES appears to be a significant and positive predictor for the survival of older people with serious diseases. This model estimates that one standard deviation (SD) higher in contextual SES is associated with a 4.4% lower likelihood of death.

Table 4 presents the results of 10 Cox proportional hazards models, examining the effects of different dimensions of community social environment (the second hypothesis). Based on previous evidence and theoretical development, we expect collective efficacy, social support, social network density, the prevalence of local organizations, and the involvement of residents in voluntary associations to be health-enhancing social resources. Controlling for individual background, these dimensions of social environment are first examined separately (Models 4.1–4.7). Consistent with our expectations, collective efficacy is protective against mortality while personal victimization and perceived violence are significantly deleterious. Contrary to our expectation, local social network density is not conducive, but detrimental, to the health of older patients. Also inconsistent with our hypotheses, social support, voluntary associations, and local organizations do not appear to be predictive of the hazard of death.

Table 4
Cox proportional hazards model (assessing the contextual effects of different dimensions of social environment)

Independent variables	Models									
	1	2	3	4	5	6	7	8	9	10
Individual level variables										
Age	1.050***	1.050***	1.050***	1.050***	1.050***	1.050***	1.050***	1.050***	1.050***	1.050***
Male	1.312***	1.311***	1.310***	1.310***	1.310***	1.312***	1.309***	1.311***	1.311***	1.312***
Poverty (medicaid recipient)	1.093**	1.096**	1.102**	1.105**	1.104**	1.091**	1.105**	1.091**	1.092**	1.092**
Race (Non-white/white)	1.035	1.047	1.036	1.047	1.048	1.012	1.046	1.020	1.010	1.034*
Charlson score (lookup yr 1)	1.106***	1.106***	1.106***	1.106***	1.106***	1.106***	1.106***	1.106***	1.106***	1.106***
Charlson score (lookup yr 2)	1.061***	1.061***	1.061***	1.061***	1.061***	1.061***	1.061***	1.061***	1.061***	1.061***
Charlson score (lookup yr 3)	1.028*	1.028*	1.028*	1.028*	1.028*	1.028*	1.028*	1.028*	1.028*	1.028*
Zip code level variables										
Collective efficacy	0.973*							0.983		
Social support		0.984								
Social network density			1.029**						1.014	
Local organization				0.990						
Voluntary association					0.999					
Perceived violence						1.035**		1.019	1.031**	
Personal victimization							1.030***	1.031***	1.022^	
Contextual social index										0.974*

^$p \leqslant 0.10$; * $p \leqslant 0.05$; ** $p \leqslant 0.01$; ***$p \leqslant 0.001$ (two-tailed tests).
a. ZIP code level $N = 51$; individual level $N = 12{,}672$.
b. Contextual social index is a composite scale that contains collective efficacy, social support, and perceived violence (data from the PHDCN-CS).
c. The estimates presented in this table are hazard ratios.
d. To save space, the effects of individual diagnosis are not presented.

Models 4.8 and 4.9 test our third hypothesis—crime and violence, as markers of social hazards, help explain the effects of community health-promoting social resources. We found in model 4.1 that collective efficacy is a significant social resource promoting health in later life. Model 4.8 shows that this beneficial effect is rendered non-significant after measures of perceived violence and personal victimization are added to the model. The coefficient of collective efficacy decreases 37% from model 4.1 to model 4.8,[11] suggesting that reducing violence and crime might be a mechanism through which collective efficacy protects health. As to the unexpectedly detrimental effect of social network density, the data suggest that crime and violence may also play an important role in this association. From model 4.3 to model 4.9, the effect of social network density is reduced by 52%.[12] Finally, to assess the overall effect of community social environment on mortality, model 4.10 estimates the coefficient for contextual social index (see the section under *Independent Variables*). The results show that older patients living in communities with a favorable social environment, compared with their counterparts living in socially disorganized communities, fare better in terms of survival after the onset of serious disease. A one SD increase in the global social index is associated with a 3% lower hazard of death.

Table 5 presents results from models that examine the effects of community social environment while controlling for community SES, attempting to assess the mediating effect of community social environment in the link between contextual SES and individual mortality (the fourth hypothesis). The protective effect of community SES (model 3.5) can be explained considerably by collective efficacy (model 5.1), social network density (model 5.3), perceived violence (model 5.6), and personal victimization (model 5.7); the reduction in the effect size of contextual SES due to these four variables varies from 12% to 33% respectively. But the contextual SES effect cannot be explained by social support (model 5.2), the number of local organization (model 5.4), or participation in voluntary associations (model 5.5). The last model in this table (model 5.8) presents the

[11] In model 4.1, the coefficient of collective efficacy was $\ln(0.973) = -0.027$. In model 4.8, the coefficient of collective efficacy was $\ln(0.983) = -0.017$. The reduction in the effect size of collective efficacy from model 4.1 to model 4.8 was 37%.

[12] In model 4.3, the coefficient of social network density was $\ln(1.029) = 0.029$. In model 4.9, the coefficient of network density was $\ln(1.014) = 0.014$. The reduction in the effect size of social network density from model 4.3 to model 4.9 was 52%.

Table 5
Cox proportional hazards model (simultaneously assessing the contextual effects of community SES and different dimensions of social environment)

Independent variables	Models							
	1	2	3	4	5	6	7	8
Individual level variables								
Age	1.050***	1.050***	1.050***	1.050***	1.050***	1.050***	1.050***	1.050***
Male	1.311***	1.311***	1.311***	1.311***	1.310***	1.311***	1.309***	1.311***
Poverty (Medicaid recipient)	1.089*	1.091*	1.093**	1.093*	1.093*	1.089*	1.094**	1.089**
Race (Non-white/white)	1.014	1.015	1.014	1.013	1.011	1.007	1.017	1.019
Charlson score (lookup yr 1)	1.106***	1.106***	1.106***	1.106***	1.106***	1.106***	1.106***	1.106***
Charlson score (lookup yr 2)	1.061***	1.061***	1.061***	1.061***	1.061***	1.061***	1.061***	1.061***
Charlson score (lookup yr 3)	1.028*	1.028*	1.028*	1.028*	1.028*	1.028*	1.028*	1.028*
Zip code level variables								
Contextual SES	0.965*	0.960**	0.967^	0.959**	0.957**	0.971	0.963*	0.986
Collective efficacy	0.989							0.981
Social support		0.996						1.000
Social network density			1.017					1.013
Local organizations				0.994				1.009
Voluntary associations					1.005			0.997
Perceived violence						1.016		1.004
Personal victimization							1.024**	1.029*

^ $p \leqslant 0.10$; * $p \leqslant 0.05$; ** $p \leqslant 0.01$; *** $p \leqslant 0.001$ (two-tailed tests).
a. Zip code level $N = 51$; individual level $N = 12{,}672$.
b. Contextual SES is a scale that contains concentrated poverty, concentrated affluence, and aggregate education.
c. The estimates presented in this table are hazard ratios.
d. To save space, the effects of individual diagnosis are not presented.

multivariate results of community effects on mortality. Among different aspects of community environment, it appears that personal victimization, as a direct marker of community social hazards, exerts the most deleterious effects on surviving after the onset of serious disease in later life. This model provides supportive evidence for our fourth hypothesis—the social environment can, at least partially, explain the effects of community SES on mortality. The effect of community SES was reduced 67% and rendered non-significant after the social factors were added to the model (see SES coefficient change from model 3.5 to model 5.8).

Motivated by the empirical result about social network density, we further explored this finding to see whether it is contingent on neighborhood socioeconomic factors. It is possible that frequent neighborly networking is beneficial in good neighborhoods, whereas in poor neighborhoods social interaction may help spread risky behaviors (e.g., crime-related activities) and in turn harm the health of local residents. We tested the interaction effect between social network density and community SES and performed analyses stratified by community affluence, education, and contextual SES (a composite score) respectively. However, we did not find any statistically significant interaction effect between these SES variables and social network density, nor did we find any consistent pattern in the stratified analyses (data not shown).

To help distinguish possible hospital-based effects on mortality from community-based effects, we repeated all the above analyses in subsets of the data restricted to include only those who survived 7 days or 30 days post-hospitalization, and our results were not much different. This may suggest that community effects found in the study are not simply picking up hospital-related factors since long-term survival (e.g., more than 30 days) is probably less sensitive to hospital characteristics.

Discussion

Understanding how community characteristics might affect the health of specific population, and understanding at which point in the course of illness such effects might appear, could help policy makers better design and evaluate community-based disease prevention and health care interventions. This study

contributes to the literature by providing new evidence that the residential environment may shape the survival experiences of seriously ill older patients after the onset of their illness. As such, it sheds light on how the local social and economic environment can affect health in the late stage of the life course.

We find strong evidence supporting our first hypothesis that an advantageous socioeconomic context helps lower mortality risk among the elderly. Community economic and educational resources appear to be strong contextual predictors of post-hospitalization mortality among the elderly, confirming that area effects on mortality do extend to later life and are not negligible even in tertiary prevention.

As for the community social environment, we find that collective efficacy is protective against mortality, net of individual characteristics. The data suggest that living in a cohesive community with effective social control enhances older peoples' survival chances after the onset of serious diseases. We also find that the level of crime and violence are considerable community forces, or markers of community social hazards, that elevate risk of death in older adults following serious illnesses. Moreover, there is evidence to show that the level of crime and violence, to a large extent, can explain the effects of other community social factors such as collective efficacy and social network density. Taking different dimensions of community social environment together, a global measure of the contextual social environment shows a strong effect in mortality among older people. These empirical findings generally support our second hypothesis (i.e., both health-enhancing social resources and health-detrimental social hazards are important) and third hypothesis (i.e., crime and violence help explain the effects of positive social resources).

Our last hypothesis is about the mediating effect of community social factors on the link between community SES and health. We found considerable evidence supporting this hypothesis. The protective effect of community SES was substantially reduced after the survey-reported neighborhood social predictors were simultaneously examined. The data further suggest that among all the community variables including SES and other social factors examined in the study neighborhood safety appears to be the most powerful force shaping survival experiences of the elderly with serious medical conditions. The level of crime and violence in a neighborhood serves as a social sign of environmental stress, which has a strong symbolic meaning for the reputation of the community and influences the overall life chances and life quality of local residents. Moreover, practically, it is possible that local crime interferes with the elderly leaving their homes in order to get medical care; in the case of many diseases, the lack of regular medical care (e.g., carefully scheduled cancer chemotherapy) can have a detrimental impact on survival.

One intriguing finding is that community social network density (measured by the size of the social network and frequency of social interaction) is not beneficial, but detrimental. Our data suggest that areas with higher levels of social network density have more crime and violence and tend to have lower SES (see Table 2). This empirical finding resonates with Wilson's observation on neighborhood processes: "It appears that what many impoverished and dangerous neighborhoods have in common is a relatively high degree of social integration (high levels of local neighboring while being relatively isolated from contacts in broader mainstream society) and low levels of informal social control (feelings that they have little control over their immediate environment, including the environment's negative influences on their children)" (Wilson, 1996, pp. 63–64). It is also possible that better-off individuals and communities are more likely to have dense social networks and strong social support *outside* the community and hence less within-community activities. In fact, a high level of social integration in low SES communities has been found in other studies (Morenoff et al., 2001), suggesting that a complex relationship between community SES and network density may exist (Glass & Balfour, 2003).

In the social support literature, social network density and social integration are sometimes regarded as structural properties of social support (Lin, Ye, & Ensel, 1999). This work suggests that the nature and exchanges of social interaction are more important than the objective structure of social networks (e.g., size and frequency of contacts) for population health, and a crude measure of social network density fails to capture the complicated nature of local social interaction. Here, it is important to note that our study does not directly address *individual-level* social resources but emphasizes a *contextual* effect of *community-level* network density on the *individual* relative hazards of deaths following a serious condition among older adults. It would be particularly informative if future work explores how community-level network density and individual-level integration interact with each other to affect health.

Several study limitations need to be discussed. First, this study is focused on Chicago, a large American city with a great degree of racial and economic segregation. While it is possible that the study findings are applicable to other urban settings within or outside the US, the national and international relevance of the findings needs to be examined in future research conducted elsewhere. Second, the PHDCN-CS data were collected 2 years later than 1993. As a result, the linked data lacked temporal sequence from social measures to health outcomes. Arguably, however, the features of social

environment we measured were unlikely to have changed significantly over this short period of time. It is a reasonable assumption that the 1995 data measure 1993 social environment relatively well. Third, we used ZIP code boundaries to define residential communities; this may not be the most appropriate unit for analyzing the impact of the social ecological environment on health. Fourth, due to known limitations of Medicare data in reporting socio-demographic background, our models did not control for a substantial array of individual characteristics, thus causing the possibility of a lack of controls for confounding factors at the individual-level. For example, race is dichotomized into white versus non-white group and individual income was measured by a dummy indicator of Medicaid receipt as a proxy for poverty status. While our research has consistently shown that this crude measure of individual poverty status has a strong effect on mortality and can not be "explained away" by any variable in all the models, it is reasonable to suspect that some effects of community SES are picking up the effects of individual SES. On the other hand, as a means-tested program, Medicaid is meant to help those with limited resources. While it does not capture the entire low-income population, it is a reasonably good proxy for poverty. Moreover, we controlled for baseline illness in our empirical analyses. To the extent that baseline health status is a product of comprehensive and cumulative life-course exposure to all sorts of environmental factors including SES, it is not probable that our findings on the prospective mortality effects of community SES and social environment simply reflect relationships held at the individual level. Other potential confounding factors may be related to family structure and relationships. Future work is warranted to explore how the survival of seriously ill older people is affected by the support and care provided by their spouses and adult children (Christakis & Iwashyna, 2003). Our data also lack individual healthcare information which may confound our results. While we did control for access to care by focusing on Medicare beneficiaries, this may not be sufficient insofar as some individuals may concomitantly have other types of insurance and disparities exist in quality care that can not be fully captured by access variables. These issues should be addressed in future research where healthcare data are available. Lastly, as with most research conducted in this area, this study does not address the issue of residential mobility and cannot advance our knowledge about the relative importance of residential selection versus social causation in explaining community effects on health. While all the respondents were older and sick, some may have moved between their initial diagnosis and either death or censorship. The issue of residential mobility has not been well addressed in the literature of neighborhood effects on health and has been noted as a major limitation in the field (Kawachi & Berkman, 2003b; Macintyre & Ellaway, 2003). Indeed, this gap in the literature warrants future investigation equipped with data longitudinal both at the individual level and community level to explore and clarify this issue.

That being said, several strengths of this study are noteworthy. Using multiple independent data sources is one methodological advantage of this work. To minimize method-induced associations between outcomes and predictors, we used Census data, social surveys, and administrative clinical information to empirically investigate our hypotheses. Our outcome measure, the hazard of death, was objective. The study was prospective in design and population-based. In addition, it tested several under-researched hypotheses about the link between residence and health in a population not adequately attended in the field. With few exceptions (Browning & Cagney, 2002; Cagney, Browning, & Wen, Forthcoming), research on the roles that specific dimensions of community SES and social environment play in the link between residence and health especially among the elderly has been scant. Examining the prospective and contextual impact of multifaceted community social ecology on mortality among seriously ill and elderly patients, this study narrowed the gap in our knowledge about the effects of urban community environment on health for older people.

Taken together, the results of this study point to the significant contextual effects of community SES and social environment on mortality for older residents in the City of Chicago after they were diagnosed with one of 13 diseases—all of which are important or major causes of death in the United States. The finding that local network size and social integration are not health-beneficial resources but health-detrimental forces in Chicago communities may signal that interventions, if only based on stimulating local social networking while ignoring the quality of social interaction in disadvantaged communities, are not likely to succeed in enhancing the health status of older people with serious health conditions. For the purpose of reducing health disparities and from a perspective of community development, it may be more efficient to steer governmental endeavors into revitalizing urban disadvantaged neighborhoods (e.g., creating more local jobs and investing in local public schools), strengthening health care services, and wielding stronger formal controls (e.g., policing) to improve neighborhood safety. In other words, the complex relationship between community social environment and health found in this study may suggest that community-level social interventions based on social capital/social cohesion models are not likely to achieve fruitful results without concomitant effort in the economic and health care realm, at least in terms of influences on the health of older people once they are already ill.

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