Effect of Community Distress and Subcultural Orientation on Mortality Following Life-Threatening Disease in the Elderly*

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We conducted a multilevel study of the prospective effects of community subcultural orientation on mortality following onset of thirteen life-threatening diseases in later life. We also examined the inter-relationship between the effects of community distress (i.e., poverty, physical disorder, and low collective efficacy) and subcultural orientation on the survival chances of serious ill older patients. Three data sources were combined to construct the working sample: the 1990 Census, the 1994-95 PHDCN-CS, and the COSI data. 51 ZIP code areas in Chicago and 12,672 elderly patients were studied. Community distress is significantly and positively associated with a higher hazard of death. The data also show that a measure of anomie at the community level significantly predicts higher rates of mortality for older patients after the onset of serious diseases over and above personal characteristics and community-level social structural stress (HR=1.26). Moreover, we find that anomie plays an important role of mediating the effect of community stress on health. Gender-specific analyses stratified on diagnosis reveal different patterns in community effect on elderly health for different clinical groups, suggesting the importance of finding specific patterns of community effects for different subgroups. In conclusion, the social, economic, physical, and cultural environment in which people live exerts an effect on whether they live or die, even among the elderly facing life-threatening illness.

Effect of Community Distress and Subcultural Orientation on Mortality Following Life-threatening Disease in the Elderly

Recent interest in how characteristics of residential places contextually affect health of individual residents living in the community has generated an abundant literature that has provided strong evidence for the link between residence and health over and above individual attributes (Blafour and Kaplan 2002; Bond Huie, Hummer, and Rogers 2002; Haan, Kaplan, and Camacho 1987; Kawachi and Berkman 2003b; Latkin and Curry 2003; LeClere, Rogers, and Kimberley 1997; LeClere, Rogers, and Kimberley 1998; Robert 1998; Ross 2000). This work has pointed to a promising research method that simultaneously examines factors located at the different layers of social structure in their influences on health.

The bulk of this work in this area has focused on community socioeconomic status (SES) which is typically measured by the average level of poverty, family income, wealth, and education of local residents. The major conclusion from these studies is that community SES is a significant contextual determinant of individual mental and physical health, although the effect size is relatively smaller than that of individual-level SES (Kawachi and Berkman 2003a; Pickett and Pearl 2001; Robert 1998; Robert 1999). Another important aspect of community life, social organizations or resources, have also been examined, although to a lesser extent. Following the hot debate about whether social capital is relevant to individual health outcomes (Chang and Christakis; Kawachi,

Kennedy, and Glass 1999; Kawachi, Kennedy, Lochner, and Prothrow-Stith 1997; Lomas 1998; Lynch 2000; Muntaner and Lynch 2002), recent empirical investigations that focus on social capital/social cohesion and health are directly testing the hypothesis that social capital at the community or neighborhood level exerts contextual effect on individual health status. Much of this research suggests that social capital, both at the community level and at the state level, has significant and positive effects on health of individuals (Browning and Cagney 2002; Kawachi and Berkman 2000; Kawachi, Kennedy, and Glass 1999; Lochner, Kawachi, Brennan, and Buka 2003; Veenstra 2002; Wen, Browning, and Cagney 2003).

Neighborhood physical environment also has been proposed and examined as a potential mediator on the pathway from community SES to health. For example, previous work has found that broken glass or trash on streets and graffiti on buildings are associated with negative effects on health (Barr, Diez-Roux, Knirsch, and Pablos-Mendez 2001; Browning, Cagney, and Wen 2003). These phenomena of physical disorder, along with other characteristics of the built environment (e.g., land use and street connectivity), may have a direct impact on health-related behaviors of local residents (e.g., physical activity) and in turn affect their health (Ewing and Cervero 2001).

The literature on place effects on health has more or less revolved around these structural, social, and physical dimensions of community environments, whereas other important aspects of community life have been much less studied. As Macintyre and her colleagues (2003) recently argued, research on the link between place and health need not

be constrained by socioeconomic and psychosocial factors as studied by social cohesion/social capital theorists, but could also incorporate other aspects including cultural, religious, political, and historical characteristics of residential community. Indeed, studying these other aspects of community environment may not only reveal important yet previously unknown patterns of social ecological influences on health, but also help identify mechanisms that can explain the effects of community SES and social and physical disorders on individual health.

The current investigation is intended to focus on the prospective impact of community *subcultural orientation* on mortality of a particularly vulnerable population—older people afflicted with life-threatening diseases. We will further explore the linkages among economic forces, social organization, physical environment, and cultural values at the community level and their unique and combined impact on the odds of mortality in late life. Of particular interest is our endpoint that measures the survival time between the incidence or diagnosis of disease and death, which both assesses the impact of contextual factors at a very vulnerable stage of life—being old and seriously ill, and also conditions the effect on a given health state. We will also specifically test whether the tolerance of risky behaviors and prevailing values and beliefs in the community exert contextual and prospective effects on health and whether these subcultural patterns explain the effects of community economic, social, and physical environments on health.

THEORETICAL AND EMPIRICAL BACKGROUND

Poverty, low collective efficacy, and physical disorder

Poverty is the key source of many social problems. The earlier work of Chicago school urban sociologists has suggested that economic problems are often spatially clustered with other social problems involving crime, delinquency, mental disorders, and physical illness (Faris and Dunham 1960; Shaw and McKay 1969). Arguably, a place seriously deficient in economic resources may have a problem of sustaining good physical condition, quality health services, efficient social institutions (e.g., family, church, sport club and other voluntary associations), and adequate local employment opportunities. Thus, individuals who reside in distressed places likely suffer from environmental stress and ill health.

A sophisticated literature has consistently described the deleterious effect of neighborhood poverty on health even after controlling for individual characteristics (Barr, Diez-Roux, Knirsch, and Pablos-Mendez 2001; Haan, Kaplan, and Camacho 1987; Yen and Kaplan 1999). In other words, this work finds that neighborhood poverty *contextually* and sometimes *prospectively* increases one's risks for poor health or death, and this relationship is not completely confounded or mediated by individual SES and other factors.

The protective effect of social resources at the community level has also been documented. For example, a recent study finds multilevel evidence that individuals residing in Chicago neighborhoods with higher levels of collective efficacy[1] report better

overall health (Browning and Cagney 2002). Another Chicago-based study also shows 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 (Lochner, Kawachi, Brennan, and Buka 2003). The social capital/collective efficacy perspective parallels to the institutional theories that emphasize the role that schools, businesses, political organizations, and social services play in the community (Crane 1991).

Not surprisingly, poverty, social, and physical environments at the community level are empirically linked. Ross and Mirowsky (2001) conducted a study examining whether living in a disadvantaged census tract damaged health and whether neighborhood social and physical disorder mediated the association. They constructed an index of objective neighborhood disadvantage that measured physical signs of disorder such as graffiti and abandoned buildings as well as negative social signs such as crime and people drinking or using drugs. They found that residents of disadvantaged neighborhood had worse self-reported health and physical functioning and more chronic conditions than residents of more advantaged neighborhoods. Furthermore, the association was mediated entirely by perceived neighborhood physical and social disorder and the resulting fear.

The link between poverty and low stock in social resources in the community has been illuminated long ago in Shaw and McKay's social disorganization theory (Shaw and McKay 1969). A central premise of this model is that structural barriers such as poverty impede development of the formal and informal ties that promote the ability to solve

common problems (Sampson and Groves 1989). Although later evidence shows that poor urban areas can be tightly integrated with extensive patterns of social interaction (Glass and Balfour 2003; Stack 1974), it is possible that the capacity to achieve common goals among persons living in impoverished neighborhoods (i.e., collective efficacy) is low, given that they may have limited means to support each other.

Community distress signaled by poverty, physical disorder, and low collective efficacy may affect health via multiple mechanisms. It is plausible that as an integral component of our social contexts neighborhood milieu affects individual psychosocial experiences involving stress processes and behavioral patterns that directly affect health. Meanwhile, the social and economic disorganization may nurture a lost sense of community, negative neighborhood identification, and aberrant behaviors (Wilson 1987). The ensuing departure from mainstream patterns amounts to a set of norms, values, orientations, and aspirations that are likely to be negative and health-compromising.

Culture-of-poverty model and the epidemic theory

Here Oscar Lewis's culture-of-poverty arguments on the ghetto underclass may shed light on how community social and structural distress may affect its cultural characteristics which have direct bearings on individual health lifestyles that inevitably affect health (Lewis 1968). Lewis defined the culture of poverty as "both an adaptation and a reaction of the poor to their marginal position in a class stratified, highly individuated, capitalistic society" (Lewis 1968:188). He argued that the culture of poverty "tends to perpetuate itself from generation to generation because of its effect on

the children. By the time slum children are age six or seven, they have usually absorbed the basic values and attitudes of their subculture and are not psychologically geared to take full advantage of changing conditions or increased opportunities which may occur in their life-time" (Ibid). Here it is important to note that Lewis also realized and explicated that culture of poverty is structurally rooted and structural changes in society (e.g., redistributing wealth) can lead to modifications of the basic characteristics of the culture of poverty.

In other words, the culture-of-poverty perspective postulates that the poor who reside in areas plagued by poverty and social problems, by virtue of their exclusion from the mainstream societies and social isolation from positive role models, develop a lifestyle that is by nature different from that of the middle-class societies in which they live and assumes a "life of its own" and passed across generations through cultural transmission (Steinberg 1989; Wilson 1987). This model is essentially consistent with the contagious or epidemic theory suggesting that people's behaviors are influenced by the norms, values, orientations, and aspirations of those around them and spread through peer influence (Crane 1991; Robert 1999). Therefore, living in distressed communities and neighbored by other disadvantaged people enhances one's exposure to health-compromising *attitudes* and *behaviors*. And, at least in part, it is through the spread of risky behaviors and negative attitudes that poverty and social dislocations are transferred to poor health in the community.

Another version of this view is epitomized in the subcultural transmission model. This model suggests that subcultures display distinctive health lifestyles, with specific beliefs, knowledge, and attitudes conditioning risk-taking behavior and health (Fitzpatrick and Lagory 2000). Therefore, in some insulated and deprived places, where long-term poverty, low labor force participation, outside-marriage childbearing, school drop-outs, welfare dependency, and other social problems prevail, deviant role models emerge and encourage health-destructive behaviors such as use of illegal drugs and violence (Wilson 1987; Wilson 1996). Similarly, attitudinal problems may also result from racial and economic segregation, limited opportunities, and little hope for upward mobility. For example, feelings of alienation, powerlessness, fatalism, and cynicism likely result from previous experiences of denial and exploitation that are potentially hazardous to health as they may correlate to recognized pre-disease pathways in individuals such as stress (Thoits 1995) and social isolation (Cacioppo, Hawkley, Crawford, Burleson, Kowalewski, Malarkey, Van Cauter, and Berntson 2002).

In all, a common theme that emanates from these differently labeled theories (i.e., cultural-poverty-perspective, contagion or epidemic model, and subcultural transmission model) is that community economic deprivation (poverty) is complexly intertwined with social disorganizations and physical disorders; and, the combined social-structural distress nurtures deviant cultural characteristics that inevitably expose the inhabitants to health-detrimental psychosocial hazards.

Health lifestyles and ecological "*habitus*"

Among others, health lifestyles are important psychosocial factors that have been theorized and corroborated as important pathways linking the social contexts with health (Williams 1990). Health lifestyles are collective modes of health-related consumptions involving choices in diet, leisure, work patterns, health care use, and other forms of behavior and based on options available to people according to their life chances reflected in variables such as age, gender, race, ethnicity, and SES (Cockerham and Ritchey 1997). It has long been recognized that one's life chances, largely determined by one's position in social hierarchy, shape the choices that people can make and in turn life conduct people undertake (Weber 1978). The relationship between social class and health lifestyles in France has been thoroughly studied by the French sociologist Pierre Bourdieu (1984). According to Bourdieu, *habitus*, or class-based set of durable dispositions to act in particular ways, results from upstream social structure and shapes an individual's cultural tastes and styles that correspond to his or her social position. In the US, given the fact that poverty and affluence have been more and more concentrated spatially in the U.S. large cities over the last three decades (Massey 1996), place of residence can be viewed as an extended marker of one's structural position, at least for urban residents. Indeed, health lifestyles are spatially patterned and inextricably entangled with the spatial distribution of structural resources.

Although research that empirically examines the link between health subculture of residential community and health of individual residents is extremely rare, some evidence

shows that neighborhood collective features are important to individual health behaviors. For example, Yen and Kaplan (1998) found that area characteristics could influence physical activity levels because of lighting, amount of criminal activity, and access to recreational facilities. In addition to Yen and Kaplan's (1999) finding that local poverty prospectively predicted decline in physical activity, a recent study (Craig, Brownson, Cragg, and Dunn 2002) using Canadian data found that a composite score of neighborhood environment based on 18 neighborhood characteristics (e.g., variety of destinations, visual aesthetics, and traffic) was positively associated with walking to work, both with and without adjustment for degree of urbanization. Another study, drawing on the ecological theory, examined the association among participation in regular vigorous exercise, social status, and aspects of prominent life settings including contextual factors at the community level (Grzywacz and Marks 2001). Their analyses indicated that respondents who perceived their neighborhoods as more safe participated in more regular, vigorous exercise than individuals in less safe communities. Other health behaviors have also been examined in their relation to residential environment. Lee and Cubbin (2002) examined whether neighborhood characteristics were associated with cardiovascular health behaviors independently of individual characteristics. The results showed that low SES and high neighborhood social disorganization were independently associated with poorer dietary habits, while high neighborhood Hispanic concentration and urbanicity were associated with healthier dietary habits. There was no association between neighborhood characteristics and physical activity or smoking. On

the other hand, two other studies have found significant effects of neighborhood SES on smoking behaviors. One study found that deprivation of the area of residence remained a significant predictor of smoking status even after the socioeconomic group of the individual had been taken into account (Kleinschmidt, Hills, and Elliott 1995). The other study found that the age- and gender-adjusted prevalence of smoking was higher in deprived urban areas and the SES of residents could only partially explain this effect (Reijneveld 1998). Evidence also shows that neighborhood deprivation, perhaps through exerting environmental stress, increases one's likelihood of drug use even after controlling for individual socio-demographic characteristics (Boardman, Finch, Ellison, Williams, and Jackson 2001).

These empirical studies demonstrate that individual health lifestyles are indeed affected by community social and structural characteristics, which make it a plausible hypothesis that community subcultural orientation corresponds to social, economic, and physical aspects of community environments and influences individual choices of health promotion or compromising consumption that eventually affect health. Even if we may suspect that healthy or risky behaviors have a larger impact on the onset of disease than on the outcome, it is reasonable to expect that lifestyles as manifest in diet, exercise, smoking, and alcohol use are still important factors that would affect disease progression among ill older adults.

CONCEPTUAL FRAMEWORK

Based on these theoretical considerations and empirical findings, Figure 1 shows a conceptual model that guides our empirical analyses. We focus on poverty as a socioeconomic marker, collective efficacy as an indicator of health-promoting social resources, and physical disorder as a measure of local physical environment. We examine two aspects of subcultural orientation—anomic attitudes and tolerance for risk behaviors—in terms of their impact on health and role in the pathway from community distress to health. Health is objectively indexed by mortality after the onset of serious diseases in old life. Acknowledging additional individual-level pathways linking place with health, this model postulates that poverty, low collective efficacy, and physical disorder are bundled together constituting *community distress* that foster a sense of *anomie* and raise the level of *tolerance for risky behavior* in the community which exacerbates already existing medical conditions among seniors, accelerates health deteriorating process, and increase mortality.

(Figure 1 about here)

METHODS

Data

Three data sources were used to construct the working sample in this study: the 1990 Census, the 1994-95 Project on Human Development in Chicago Neighborhoods-Community Survey, and the Care after the Onset of Serious Illness.

ZIP code level poverty rate was obtained from the 1990 Census and was used as a structural marker of community conditions. The benefit of using a single variable versus a composite index to indicate community economic condition is that it is conceptually clear and methodologically simple.

Measures of community physical environment (physical disorder), social resources (i.e. *collective efficacy*), and cultural characteristics (*tolerance for risk behavior; anomie)* were constructed from the PHDCN-CS (Sampson, Raudenbush, and Earls 1997). The PHDCN-CS is a probability sample of 8782 residents of Chicago focusing on respondent assessments of the communities in which they live. Each record in the PHDCN-CS data was identified by a census tract in Chicago. Using the geographic centroids of census tracts, we linked each census tract with its corresponding ZIP code area.

The core data of COSI are rooted in the 1993 inpatient hospitalization records from the Health Care Financing Administration's Medicare program (Christakis, Iwashyna, and Zhang 2002; Iwashyna, Zhang, and Christakis 2002). The COSI data set consists of a cohort of patients *newly* diagnosed in 1993 with one of thirteen serious illnesses and followed for up to six years. The thirteen diseases were selected because they met several COSI conceptual criteria including accuracy of ascertainment and high prevalence. Prior detailed empirical work provided guidance to capture incident cases of disease based on incident hospitalizations. Detailed descriptions about how COSI data

were constructed have been published elsewhere (Christakis, Iwashyna, and Zhang 2002).

Dependent Variable

Our health outcome was the relative hazard of death for COSI cohort members. The survival time was defined as the time period 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). People who were still alive on June 30, 1999 were right censored.

Independent Variables

At the individual level

Individual demographic and baseline health measures controlled in the analyses include age, gender, race (Non-White versus White), three continuous comorbidity scores based on health care use in the three years prior to the index hospitalization,[2] and a dichotomous indicator of Medicaid recipient 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 14% of them receiving Medicaid in 1993. The majority are white and 30% non-white most of whom are African Americans and Latinos. For the COSI cohort, stroke (27%) and congestive heart failure (24.1%) are the most prevalent conditions among the diseases studied.

(Table 1 about here)

At the community level

Health-related subcultural orientations were captured with two scales, a measure of tolerance for youthful deviance and problem behaviors and a measure of detachment from conventional norms (anomie). *Tolerance for risk behavior* was measured by four items. Respondents were asked their opinions regarding how wrong it was for teenagers around nineteen-year-olds to smoke cigarettes, use marijuana, drink alcohol, and get into fistfights. *Anomie* was tapped by a five-point Likert scale asking respondents' agreement with the following statements: 1) "laws were made to be broken," 2) "it's okay to do anything you want as long as you don't hurt anyone," 3) "to make money, there are no right and wrong ways any more, only easy ways and hard ways," and 4) 'fighting between friends or within families is nobody else's business."

Following the operationalization of Sampson et al. (Sampson, Raudenbush, and Earls 1997), the collective efficacy scale was constructed through combining items of social cohesion and informal social control. 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 reverse coded. Health-related informal social control was tapped through respondents' level of agreement with the following statement: "You can count on adults in this neighborhood

to watch out that children are safe and don't get into trouble." An additional informal control item asked respondents how likely it was that 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 ZIP code areas ($r=0.92$, $p<0.0001$). The higher the value in the collective efficacy scale, the more stock of collective efficacy present in the community.

Physical disorder was represented by a three-item Likert-type scale in the PHDCN-CS. Residents were asked about the extent of problems stemming from litter, broken glass or trash on the sidewalks and streets; of graffiti on buildings and walls; of vacant or deserted houses or storefronts. Responses to the three-point Likert scales were aggregated to the neighborhood level as initial measures. The three scales were highly correlated across neighborhoods. I then combined them into a composite measure, with higher scores indicating higher levels of physical disorder.

Community Poverty was measured by the percentage of households that were living in poverty (a household annual income of less than US$13,359 for a household of four in 1990). As a marker of spatially based position in social hierarchy, community poverty goes hand in hand with physical disorder ($r=0.81$; $p<0.001$) while negatively correlates with collective efficacy ($r=-0.68$; $p<0.001$). By contrast, the two cultural variables are not significantly associated with poverty. Relative to anomie, tolerance for risk behavior is more strongly correlated with poverty although the correlation is not statistically significant at a two-sided test ($r=0.21$; $p=0.15$). Meanwhile, it is significantly

linked to collective efficacy ($r=-0.39$; $p<0.05$) and physical disorder ($p=0.23$; $p<0.10$). Table 2 shows the correlation matrix of the five measures of community environments.

(Table 2 about here)

To better understand the conceptual relationship among these community factors, we performed factor analyses to identify the latent structure underlying the five variables at the community level examined in this study. These analyses show that physical disorder, poverty, and collective efficacy are tightly clustered around a dominant factor, whereas tolerance for risk behavior and anomie appear to be distinct. We then constructed a single factor score as a composite measure of general community social, economic, and physical environment.[3] We labeled this composite measure as "community distress," which is positively correlated with high poverty, low collective efficacy, and high level of physical disorder in the community.

Analytical strategy

Following the '*ecometric*' method assessing ecological settings illustrated in the work of Raudenbush and Sampson (1999), we used HLM-adjusted empirical Bayes' residuals as measures of community *collective efficacy, tolerance of risky behavior*, and *anomie*. The major advantage of this approach to measuring community characteristics based on survey data is that ecological measures thus constructed are simultaneously adjusted for item specific factors (e.g., severity), response bias, different sample size

within each ecological unit, and random errors specific to each community have all been taken into account.

After data construction, we fit a series of Cox proportional hazards models to test the effects of community characteristics on individual hazard of death among the elderly patients in the COSI data set. Huber-White robust method of calculating the variance-covariance matrix is used to account for the possible correlation in survival experiences among patients living within the same ZIP code area (Lin and Wei 1989). The proportionality assumption of Cox models was tested to examine the sensitivity of findings from non-stratified Cox models to the violation of the proportionality assumption. No meaningful violation of the proportionality assumption was detected.

RESULTS

Table 3 shows the results of six Cox proportional hazards models that examine the effects of community distress, anomie, and tolerance for risk behaviors on mortality and the mediating role of subcultural orientation in the link between community distress and the hazards of death among elderly patients. First, we find that community physical, social, and economic distress prospectively and contextually increases the mortality risk of individuals after diagnosis (model 3.1). Living in a community with one unit higher level of distress is associated with 3.9% higher risk of mortality in this elderly patient population controlling for individual poverty status, age, gender, race, baseline comorbidity, and diagnosis.

Second, a measure of *anomie* at the community level significantly predicts higher rate of mortality for older patients with serious diseases over and above personal characteristics (model 3.2). Living in a community with one unit higher level of anomie is associated with 26% higher risk of mortality in this elderly patient population regardless of individual risk factors. Tolerance for risk behavior also exerts a positive effect on mortality yet the magnitude of the effect is not statistically significant (model 3.3).

Third, the data also show that community distress can be partially explained by the contextual impact of anomie. After adding anomie to model 3.1, the effect of community distress decreases about 18% but remains significant at the 5% level (model 3.4). Similarly, adding community distress to model 3.2 reduces anomie effect nearly 20% which is now only marginally significant at the 10% level (model 3.4).

Fourth, both model 3.5 and model 3.6 show that tolerance of risky behaviors is not an important mechanism that explains the contextual effect of community distress on mortality. The effect of community distress remains virtually unchanged with tolerance of risky behaviors added to the model.

In summary, the key message conveyed by Table 3 is that the overall level of community disadvantage exercises a strong positive effect on the odds of dying from medical conditions among elderly people; and community subcultural orientation manifest in anomic attitudes holds considerable promise as a mediator of this contextual and prospective effect. It is also noteworthy that the effects of individual risk factors are

consistent across all the models. Age, male gender, poverty, and co-existing morbidity are all significant risk factors of mortality. The coefficients of diagnosis reveal that relative to acute myocardial infarction (MI; the reference diagnosis), stroke has comparable effect on the hazard of death, congestive heart failure has stronger mortality force, most cancers have yet stronger effect on mortality, and hip fracture appears to be the least fatal condition among the thirteen diseases examined.

The findings on these individual-level risk factors suggest that since the nature and deteriorating effect of these conditions vary so dramatically, it might be informative to examine the effect of community distress and anomie stratified by diagnosis and gender as well. The large sample size of this study makes it feasible to do fine-tuned gender-specific analyses for several clinical subgroups that differ in terms of their mortality forces.

Table 4 through table 7 present models that do this. Table 4 examines the effects of community distress and anomie for the MI/stroke patients. The first three models are for male MI or stroke patients. It appears that community distress is a salient contextual predictor of mortality for this particular group of elderly persons (model 4.1 and model 4.3), whereas the effect of anomie is positive but not statistically significant at the 5% level (model 4.2) although it reduces 9% of the distress effect (model 4.1 and model 4.3). The last three models are for elderly women with MI or stroke conditions; and, for these women, community environments are completely irrelevant for the likelihood of their surviving MI or stroke (model 4.4 through model 4.6).

Table 5 presents theses models for congestive heart failure (CHF). For both men and women, community distress significantly predicts the hazard of death (model 5.1 and model 5.4). For women, anomie is also an important contextual factor (HR=1.78; model 5.5), substantially mediating the path from community distress to mortality with the distress effect decreasing from 0.07 to 0.052 (a 26% reduction from model 5.4 to model 5.6).

Table 6 shows the results for cancers of nine different sites. A pattern is fairly steady for men and women, that is, community environments do not appear significant in predicting survival from cancer in old life, rather, the individual-level risk factors such as age, minority race (non-white), poverty, and comorbidity in the preceding year before the index hospitalization exhibit a strong predictive power for the hazard of dying from cancer (model 6.1 through model 6.6).

Lastly, Table 7 tests our hypothesis on hip fracture sufferers. We find that for seniors afflicted with hip fracture, community distress does not contextually affect their chances of survival (model 7.1; model 7.4 and 7.6). For men, anomie is a strong predictor for mortality (model 7.2); yet it is not the case for women (model 7.5). There is also some evidence to show that the anomie effect can be explained by community distress in a small degree (the effect size of anomie decreases about 10% from model 7.4 to model 7.6).

(Table 3, 4, 5, 6, 7 about here)

Table 8 summarizes the findings from the stratified analyses. In general, it seems that the effect size of anomie is larger than that of community distress. Although sometimes the anomie effect is statistically non-significant, it has a large hazard ratio that should not go unnoticed. For example, the model for female cancer patients finds a positive effect of anomie with a hazard ratio of 1.31, although the effect fails to reach the conventional 5% level. The finding is suggestive and supports further efforts addressing community subculture and health link for cancer patients. Another pattern that emerges from this table is that community effects tend to be stronger for CHF than for other diagnoses. Except for the anomie effect on males, where community factors are found to affect patients with MI/stroke, hip fracture, or cancer, it always affects the CHF patients; where community factors do not affect the non-CHF patients, it still affects the CHF patients. As shown in Table 3, CHF is less fatal than cancers but more life-threatening than MI, stroke, or hip fracture. We speculate that the effect of community environments on mortality following serious illnesses peaks when one's condition is not so bad (e.g., lung cancer) nor so mild (e.g., hip fracture) in terms of the force of mortality. It is also conceivable that CHF, which is exquisitely responsive to daily variation in compliance with medications and dietary restrictions (Iwashyna 2001), is the most environment-sensitive condition among the diseases we studied. In fact, we know little about how place effects change by cause-specific mortality or by different health outcomes. Our basic knowledge would be strengthened by systematically examining subgroup variations in the link between residential community and health. Lastly, our data show no

consistent gender pattern in community effects. Table 8 conveys a heterogeneous picture of community effects by gender. For some conditions like MI, Stroke, or hip fracture, men appear to be more affected. Yet for other diseases such as CHF, at least in terms of the anomie effect, women are more sensitive to the contextual environment. These variations may reflect a real phenomenon, that is, men and women are differentially affected by the community environments for different health markers.

SUMMARY AND DISCUSSION

In this prospective and multi-level study, we examined four dimensions of community environments, including physical disorder, social resources, economic deprivation, and subcultural orientation in terms of their contextual effects on mortality risk following serious conditions in an elderly population in Chicago. We found that a dominant latent factor underlies physical disorder, collective efficacy, and poverty. An encompassing composite index was then constructed to measure the level of community distress and subsequently used to test our conceptual model (see Figure 1). The theoretical framework received broad empirical support.

The conclusion that we can draw from the research is that community distress and the amount of anomie present in the community (a subcultural aspect) exert powerful contextual forces affecting the mortality risk among elderly patients over and above individual risk factors. By contrast, we found no effect of the level of tolerance for risk behaviors on mortality of elderly people stricken with debilitating illnesses. When we set

off to test the mortality effect of subcultural orientation, we explicitly focused on deviant attitudes in general and views on risk behaviors in particular as two important dimensions of subculture. The anomie scale was intended to tap the first dimension and the tolerance for risk behavior scale was orientated to capturing the latter. Recall the tolerance for risk behavior scale was based on respondents' opinions regarding how wrong it was for *teenagers around nineteen-year-olds* to engage in risk behaviors. It is possible that this measure is primarily based on views about teenager risk behavior and therefore is less relevant for older and diseased adults. Or, perhaps more likely, norms of healthy or risky behavior *at the community level* indeed have a more preventive function and are less crucial for elderly people who have already been afflicted with serious diseases, even if at the individual level lifestyles may still be relevant for health at this stage of life. On the other hand, the level of anomie, which may reflect a certain degree of cynicism, fatalism, and "present-time-consumption" that spread in the community, appears to have a strong impact on the hazard of death contextually as well as prospectively in this elderly population with serious illnesses.

Following the findings of significant effects of community distress and subculture, we further examined whether the effect of community distress is mediated through subcultural orientation as measured by the level of anomie and the tolerance for risk behaviors in the community. Our data show that the two types of community forces, social-structural distress and subculture, are inter-related yet clearly distinct from each other. Although their effects on mortality can each be understood by the other in some

degree, they have unique or independent impacts on mortality which are perhaps channeled through alternative pathways. This finding confirms the hypothesis put forward in the conceptual model (Figure 1), that is, community social-structural distress affects mortality in part via subcultural orientation but additional mechanisms exist probably through other community ecological processes and/or individual pathways that are not examined in this study.

The conclusion that community subcultural orientation plays a significant role in the link between residential community distress and health may be provocative. This evidence essentially lends support to the culture-of-poverty perspective that stresses the unhealthy subcultures in marginalized social groups and poor urban communities in explaining the so-called "ghetto" problems as well as individual disadvantage. Critics of the culture-of-poverty thesis argue that it places blame on the victim no matter whether at the individual level or community level, and hence, clouds the social causes of poverty; believers of this view may therefore erroneously favor social policies that aim to indoctrinate the poor with mainstream values over those more expensive and painful policies that promote structural changes in resource allocation (Steinberg 1989). Clearly, it is not thoughtful to assume that cultural patterns just spontaneously evolve without being inextricably tied up with the social structure. As Oscar Lewis noted in his original conception of the culture-of-poverty perspective, "by creating basic structural changes in society, by redistributing wealth, by organizing the poor and giving them a sense of belonging, of power and of leadership, revolutions frequently succeed in abolishing some

of the basic characteristics of the culture of poverty even when they do not succeed in curing poverty itself" (Lewis 1966:9).

On the other hand, it is also important to recognize the fact that although poor places tend to have health-compromising subcultures and social problems, not all poor places have become "ghettos" (Wilson 1987); and some communities, despite suffering from economic deficiency, may yet manage well in maintaining the mainstream values or social functioning, and in doing so, alleviate the deleterious effect of structural disadvantage. These communities are probably uncommon but their experiences would be particularly informative to policy makers and researchers who are concerned with health disparities across social groups. Further quantitative and qualitative research is needed to elucidate the processes through which some impoverished communities protect their residents against health hazards better than their peers of similar poverty level. Data from in-depth ethnographic field work may provide some answers to questions like "What are the circumstances, apart from or in conjunction with affluence, that impel a community to mobilize an efficient apparatus to instill positive cultural values into the residents and successfully maintain social order?" Such research would potentially narrow the knowledge gap in the relationship between community subcultural orientation and the residents' physical and mental health. Some cultural orientations are not exclusively rooted in the unequal distribution of resources. Future work could extend the research to investigating other cultural aspects in the community such as health values

and attitudes toward health services that are most often embedded in the community's ethnic and minority background.

In sum, the general findings from this research are strongly supportive of the perspective that place effects extend to late life. Theory suggests that neighborhood effect should be stronger for elderly people because they tend to be less mobile, spend more time in the neighborhood, and be more vulnerable and/or attached to their neighborhoods than younger people (Diez-Roux 2002; Glass and Balfour 2003). However, empirical evidence is at most mixed about this posited age pattern in the neighborhood-health link, with more studies in fact finding non-significant or less significant neighborhood effect (SES) at the late stage of the life course (Anderson, Sorlie, Backlund, Johnson, and Kaplan 1997; Haan, Kaplan, and Camacho 1987; Waitzman and Smith 1998). It has been argued that inadequate measures of neighborhood environment may be one reason for the seemingly reduced neighborhood effects in older persons (Glass and Balfour 2003). For example, Robert and House (1996) found that neighborhood disparities in mortality can be seen until at least age 85 when measures of wealth rather than just income were used to indicate neighborhood SES. Using more sophisticated measures of the community environments, our study reinforces the idea that the social, economic, physical, and cultural environments of residential community matter to the course of disease progression to death in late life.

Several strengths of this study merit comments. Using different data sources to measure community environments (i.e., 1990 Census and the PHDCN-CS) and capture

individual risk factors and health event (the COSI) minimizes method-induced associations between outcomes and predictors. Our outcome measure, the hazard of death, was objective. The study was prospective and population-based. The sheer size of our sample allows an in-depth study focusing on different subgroups by diagnosis and gender. In addition, we tested the prospective and contextual effect of an under-researched aspect of community life—*subcultural orientation*—on mortality following serious illnesses in old life, and further revealed the mediating role of cultural characteristics (i.e., anomie) in the link between community physical, social, and economic disadvantage and the hazard of death among elderly urban patients.

The research would be strengthened by a true longitudinal design, however. Without time-varying information on individual residence (only ZIP code at the origin of the study was available for the COSI cohort) we can not examine how residential mobility affects our findings. Presumably, some people moved between their initial diagnosis and their death or censorship. Failure in taking this issue into account may result in bias due to exposure misspecification. The direction of this bias is not clear, largely depending on the causes of residential move. Future investigation equipped with data longitudinal both at the individual level and community level should be able to explore and clarify this issue. Another data limitation concerns the lack of individual-level controls. For example, we did not control for education which is clearly related to one's residence as well as health; and our measure for individual income was crude and only based on a dichotomous indicator of Medicaid recipient. Nevertheless, we

controlled for diagnosis in 1993 at the index hospitalization and comorbidity status for the three preceding years. It is arguable that these health outcomes are largely reflective of one's social-demographic background and can be viewed as complementary to other individual-level risk factors. Finally, we want to mention that this study is Chicago-based and designed for a vulnerable subgroup of the population—people who are old and ill. It would be interesting to see if the main findings are also replicable for other populations in other urban or rural settings. Our knowledge of social determinants of health would be further enhanced by finding specific patterns of community effects for different subgroups on a variety of health outcomes.

In conclusion, the research finds significant evidence of the prospective and contextual effect of the social, economic, physical, and cultural environment in which people live on whether they live or die, even among the elderly facing life-threatening illness. It appears that reducing poverty, regaining community collective efficacy, improving physical environment, and redirecting subcultural orientation via, say, providing role models and adequate institutional support may be effective to regenerate disadvantaged neighborhoods and benefit local residents even in the dire straights faced by the seriously ill elderly. In other words, this evidence suggests that policy makers should act on the places where people live, in tandem with improving the life chances and encouraging healthy behaviors of individuals.

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[1] Sampson and his colleagues (1997) has recently developed "collective efficacy" as a related social ecological concept in their study of neighborhood effects on crime. Essentially collective efficacy is a subsumed notion of social capital, which has been defined as "social cohesion among neighbors combined with their willingness to intervene on behalf of the common good" (Sampson, Raudenbush, and Earls 1997). This notion emphasizes two important dimensions of social capital—social solidarity and informal social control. In physical setting, it emphasizes the *willingness* and *ability* of neighborhoods to act collectively in order to realize the common goals of residents. According to collective efficacy theory, high collective capacity is achieved not necessarily through the existence of dense local civic networks but mainly through the agreed collectivism manifest in a community's cohesive culture and the informally controlled normative behaviors of its residents (Morenoff, Sampson, and Raudenbush 2001; Sampson, Raudenbush, and Earls 1997).

[2] Comorbidity is measured by the Charlson method (Charlson, Pompei, Ales, and MacKenzie 1987), which is extremely popular and has been used extensively in claims data research (Christakis, Iwashyna, and Zhang 2002; Iwashyna, Zhang, and Christakis 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 et al. (1999) have found that, compared 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.

[3] Factor loadings for poverty, collective efficacy, and physical disorder are 0.83, -0.83, and 0.93 respectively. The community distress scale has satisfactory internal reliability with coefficient alpha of 0.76.

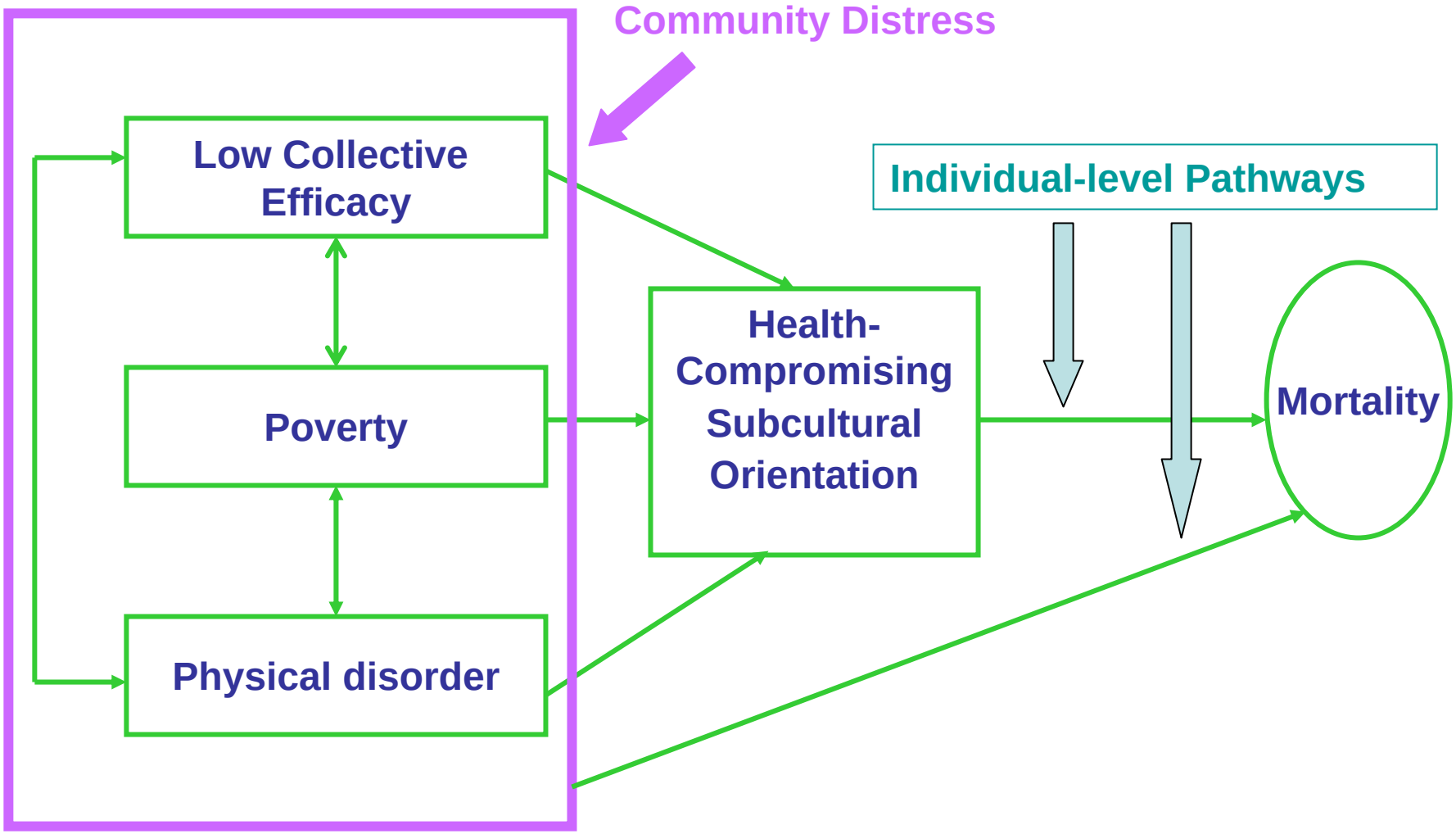

Figure 1 Conceptual Model

Table 1. Characteristics of COSI patients in Chicago

Variables	Mean/Percent	St dev.
Demographic		
Age	78.598	7.204
Male	0.403	0.491
Poverty (Medicaid recipient)	0.130	0.339
Race (White)	0.676	0.468
*Baseline Health Status (in 1993)**		
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
Congestive 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

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

Table 2 Correlation Matrix of Community-level Characteristics

	Poverty		Collective efficacy		Physical disorder		Anomie	Risk behavior
Poverty	**1.000**							
Collective efficacy	**-0.676**	***	**1.000**					
Physical disorder	**0.814**	***	**-0.813**	***	**1.000**			
Anomie	**-0.045**		**-0.088**		**0.135**		**1.000**	
Tolerance of risk behavior	**0.206**		**-0.386**	**	**0.234**	*	**0.025**	**1.000**

*** p < 0.10 ** p<0.05 *** p < 0.001**

Table 3: Coefficients of Cox Proportional Hazards Models

	(1)	(2)	(3)	(4)	(5)	(6)
	Community distress	Anomie	Risk behavior	Distress & Anomie	Distress & Behavior	Distress & Subculture
Age	0.049***	0.049***	0.049***	0.049***	0.049***	0.049***
	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)
Male	0.272***	0.269***	0.271***	0.270***	0.272***	0.270***
	(0.022)	(0.023)	(0.022)	(0.023)	(0.022)	(0.023)
Poverty (Medicaid)	0.085**	0.096***	0.098***	0.085**	0.085**	0.085**
	(0.035)	(0.034)	(0.035)	(0.034)	(0.035)	(0.034)
Race (non-white)	0.016	0.061*	0.042	0.034	0.016	0.034
	(0.033)	(0.031)	(0.033)	(0.031)	(0.032)	(0.031)
Comorbidity 1^	0.100***	0.101***	0.101***	0.100***	0.100***	0.100***
	(0.009)	(0.009)	(0.009)	(0.009)	(0.009)	(0.009)
Comorbidity 2	0.059***	0.059***	0.059***	0.059***	0.059***	0.059***
	(0.011)	(0.011)	(0.011)	(0.011)	(0.011)	(0.011)
Comorbidity 3	0.028**	0.028**	0.028**	0.028**	0.028**	0.028**
	(0.012)	(0.011)	(0.011)	(0.011)	(0.012)	(0.011)
CHF	0.105***	0.106***	0.108***	0.104***	0.105***	0.104***
	(0.039)	(0.039)	(0.039)	(0.039)	(0.039)	(0.039)
CNS	1.068***	1.069***	1.074***	1.064***	1.068***	1.063***
	(0.187)	(0.188)	(0.186)	(0.189)	(0.188)	(0.190)
Colorectal cancer	0.094*	0.094*	0.093*	0.095*	0.094*	0.095*
	(0.055)	(0.055)	(0.055)	(0.055)	(0.055)	(0.055)
Hip fracture	-0.207***	-0.205***	-0.207***	-0.206***	-0.207***	-0.206***
	(0.048)	(0.048)	(0.048)	(0.048)	(0.048)	(0.048)
Head/neck cancer	0.377***	0.383***	0.379***	0.379***	0.377***	0.379***
	(0.137)	(0.136)	(0.135)	(0.137)	(0.137)	(0.137)
Liver/biliary cancer	1.322***	1.323***	1.326***	1.322***	1.323***	1.322***
	(0.146)	(0.145)	(0.147)	(0.145)	(0.147)	(0.146)
Leukemia	0.661***	0.671***	0.663***	0.669***	0.661***	0.669***
	(0.110)	(0.109)	(0.110)	(0.110)	(0.111)	(0.111)
Lung cancer	1.161***	1.164***	1.162***	1.163***	1.161***	1.163***
	(0.050)	(0.051)	(0.050)	(0.051)	(0.050)	(0.051)
Lymphoma	0.492***	0.500***	0.497***	0.496***	0.492***	0.496***
	(0.079)	(0.080)	(0.080)	(0.079)	(0.079)	(0.080)
Pancreatic cancer	1.361***	1.361***	1.357***	1.364***	1.361***	1.364***
	(0.079)	(0.080)	(0.082)	(0.079)	(0.080)	(0.079)
Stroke	0.007	0.009	0.008	0.008	0.007	0.008
	(0.039)	(0.039)	(0.039)	(0.039)	(0.039)	(0.039)
Urinary cancer	0.017	0.020	0.018	0.019	0.017	0.019
	(0.059)	(0.059)	(0.059)	(0.059)	(0.059)	(0.059)
distress	0.039***			0.032**	0.038**	0.032**
	(0.015)			(0.014)	(0.016)	(0.015)
Anomie		0.228**		0.183*		0.183*
		(0.106)		(0.098)		(0.098)
Tolerance of risk			0.049		0.007	0.001
behaviors			(0.061)		(0.065)	(0.063)
Observations	12672	12672	12672	12672	12672	12672

Robust standard errors in parentheses

* significant at 10%; ** significant at 5%; *** significant at 1%

^ Charlson comorbidity score in lookup year 1 (see footnote 2)

Table 4: Coefficients of Cox Proportional Hazards Models for MI/Stroke

	(1)	(2)	(3)	(4)	(5)	(6)
	Community distress; Male	Anomie; Male	Distress & Anomie; Male	Community distress; Female	Anomie; Female	Distress & Anomie; Female
Age	0.059***	0.059***	0.059***	0.053***	0.053***	0.053***
	(0.004)	(0.004)	(0.004)	(0.004)	(0.004)	(0.004)
Poverty	0.131	0.147	0.123	0.052	0.058	0.052
	(0.106)	(0.104)	(0.107)	(0.070)	(0.070)	(0.070)
Race	-0.005	0.085	0.014	0.040	0.060	0.042
	(0.052)	(0.060)	(0.062)	(0.056)	(0.055)	(0.059)
Comorbidity 1^	0.069***	0.071***	0.070***	0.117***	0.117***	0.117***
	(0.025)	(0.024)	(0.024)	(0.017)	(0.017)	(0.017)
Comorbidity 2	0.043	0.043	0.043	0.096***	0.096***	0.096***
	(0.028)	(0.028)	(0.028)	(0.021)	(0.021)	(0.021)
Comorbidity 3	0.089***	0.088***	0.089***	0.056***	0.055***	0.055***
	(0.031)	(0.030)	(0.031)	(0.021)	(0.021)	(0.021)
Distress	0.091***		0.083***	0.021		0.020
	(0.032)		(0.030)	(0.031)		(0.032)
Anomie		0.328	0.206		0.049	0.021
		(0.208)	(0.172)		(0.162)	(0.166)
Observations	2024	2024	2024	2907	2907	2907

Robust standard errors in parentheses

*** significant at 10%; ** significant at 5%; *** significant at 1%**

^ Charlson comorbidity score in lookup year 1 (see footnote 2)

Table 5: Coefficients of Cox Proportional Hazards Models for CHF

	(1)	(2)	(3)	(4)	(5)	(6)
	Community distress; Male	Anomie; Male	Distress & Anomie; Male	Community distress; Female	Anomie; Female	Distress & Anomie; Female
Age	0.046***	0.045***	0.046***	0.049***	0.049***	0.049***
	(0.005)	(0.005)	(0.005)	(0.003)	(0.003)	(0.003)
Poverty	-0.001	0.028	0.004	0.028	0.052	0.035
	(0.124)	(0.125)	(0.122)	(0.081)	(0.081)	(0.081)
Race	-0.063	-0.035	-0.085	-0.051	0.037	-0.008
	(0.066)	(0.070)	(0.072)	(0.096)	(0.083)	(0.089)
Comorbidity 1^	0.117***	0.116***	0.117***	0.023	0.020	0.019
	(0.035)	(0.035)	(0.035)	(0.029)	(0.029)	(0.030)
Comorbidity 2	-0.002	-0.003	-0.001	0.076**	0.077**	0.078**
	(0.043)	(0.043)	(0.044)	(0.037)	(0.037)	(0.037)
Comorbidity 3	-0.001	0.001	-0.004	-0.060*	-0.062*	-0.062*
	(0.024)	(0.025)	(0.025)	(0.036)	(0.036)	(0.036)
Distress	0.058*		0.069*	0.070*		0.052
	(0.034)		(0.037)	(0.037)		(0.033)
Anomie		-0.131	-0.255		0.579***	0.509**
		(0.251)	(0.266)		(0.221)	(0.203)
Observations	1166	1166	1166	1895	1895	1895

Robust standard errors in parentheses

*** significant at 10%; ** significant at 5%; *** significant at 1%**

^ Charlson comorbidity score in lookup year 1 (see footnote 2)

Table 6: Coefficients of Cox Proportional Hazards Models for Cancer (9 sites combined)

	(1)	(2)	(3)	(4)	(5)	(6)
	Community distress; Male	Anomie; Male	Distress & Anomie; Male	Community distress; Female	Anomie; Female	Distress & Anomie; Female
Age	0.022***	0.022***	0.022***	0.032***	0.032***	0.032***
	(0.005)	(0.005)	(0.005)	(0.005)	(0.005)	(0.005)
Poverty	0.218**	0.230**	0.221**	0.006	-0.006	0.001
	(0.096)	(0.094)	(0.096)	(0.074)	(0.074)	(0.075)
Race	0.190***	0.217***	0.202***	0.189***	0.205***	0.217***
	(0.060)	(0.055)	(0.061)	(0.070)	(0.065)	(0.069)
Comorbidity 1^	0.112***	0.113***	0.113***	0.166***	0.167***	0.167***
	(0.026)	(0.027)	(0.026)	(0.020)	(0.020)	(0.020)
Comorbidity 2	-0.008	-0.008	-0.008	0.058*	0.059**	0.058**
	(0.030)	(0.030)	(0.030)	(0.030)	(0.029)	(0.029)
Comorbidity 3	-0.006	-0.005	-0.006	0.026	0.026	0.025
	(0.032)	(0.032)	(0.032)	(0.038)	(0.038)	(0.038)
Distress	0.022		0.016	-0.007		-0.015
	(0.031)		(0.031)	(0.036)		(0.034)
Anomie		0.158	0.135		0.269	0.284
		(0.221)	(0.220)		(0.231)	(0.227)
Observations	1511	1511	1511	1489	1489	1489

Robust standard errors in parentheses

*** significant at 10%; ** significant at 5%; *** significant at 1%**

^ Charlson comorbidity score in lookup year 1 (see footnote 2)

Table 7: Coefficients of Cox Proportional Hazards Models for Hip Fracture

	(1)	(2)	(3)	(4)	(5)	(6)
	Community distress; Male	Anomie; Male	Distress & Anomie; Male	Community distress; Female	Anomie; Female	Distress & Anomie; Female
Age	0.047***	0.049***	0.049***	0.064***	0.064***	0.064***
	(0.007)	(0.007)	(0.007)	(0.005)	(0.005)	(0.005)
Poverty	0.078	0.113	0.087	0.180	0.186*	0.179
	(0.173)	(0.172)	(0.173)	(0.110)	(0.109)	(0.110)
Race	-0.175	-0.030	-0.092	-0.031	0.004	-0.014
	(0.173)	(0.152)	(0.160)	(0.078)	(0.072)	(0.081)
Comorbidity 1^	0.093*	0.094*	0.093*	0.145***	0.144***	0.145***
	(0.049)	(0.050)	(0.049)	(0.033)	(0.034)	(0.033)
Comorbidity 2	0.034	0.026	0.026	0.058	0.058	0.059
	(0.064)	(0.067)	(0.067)	(0.040)	(0.040)	(0.040)
Comorbidity 3	0.028	0.043	0.037	0.085**	0.086**	0.086**
	(0.084)	(0.081)	(0.082)	(0.038)	(0.038)	(0.038)
Distress	0.090		0.061	0.027		0.021
	(0.066)		(0.065)	(0.041)		(0.044)
Anomie		0.934***	0.844***		0.230	0.200
		(0.340)	(0.320)		(0.273)	(0.287)
Observations	407	407	407	1273	1273	1273

Robust standard errors in parentheses

*** significant at 10%; ** significant at 5%; *** significant at 1%**

^ Charlson comorbidity score in lookup year 1 (see footnote 2)

Table 8: Summary of Table 4 through Table 7

	Male	Male	Female	Female
	Community distress	Anomie	Community distress	anomie
MI/Stroke	Y (HR=1.10) n=2024	N (HR=1.39) n=2024	N (HR=1.02) n=2907	N (HR=1.05) n=2907
CHF	Y (HR=1.06) n=1166	N (HR=0.88) n=1166	Y (HR=1.07) n=1895	Y (HR=1.78) n=1895
Cancer	N (HR=1.02) n=1511	N (HR=1.17) n=1511	N (HR=0.99) n=1489	N (HR=1.31) n=1489
Hip fracture	N (HR=1.09) n=407	Y (HR=2.55) n=407	N (HR=1.03) n=1273	N (HR=1.26) n=1273

Y denotes that there is a statistically significant contextual effect on mortality.
N denotes that there is no statistically significant contextual effect on mortality.
Hazard ratios (HR) are presented in the parenthesis.
Sample sizes (n) are presented for each subgroup analysis.