

Social Environment Effects on Health & Aging:
Integrating Epidemiological and Demographic Approaches and Perspectives

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INTRODUCTION

The past 30 years has witnessed a surge of research interest in the health effects of the social environment. In truth however, this might more accurately be classified as a re=surgence of such interest inasmuch as philosophies of disease susceptibility have included consideration of the social environment as far back as Hippocrates (Dubos, 1959) and up through the seminal work of Emile Durkheim in the late 1800's demonstrating the significant impact of social integration on suicide risk (Durkheim, 1955). With the advent of 'germ theory' in the 19th century, however, interest in the role of the social environment declined as health research focused largely on the identification of specific biological "agents" or risk factors for disease causation (Dubos, 1959). The inability of these more biologically focused models to explain population distributions of disease and mortality has served as a stimulus for a broader search for explanatory factors – resulting in re-consideration of the potential importance of the social environment.

That the social environment should be a potentially influential factor in determining population distributions of health and mortality is rendered all the more plausible by the fact that man has evolved as a social animal, living in groups for some 2 million years (Alcock, 1983). The survival advantages incurred with the formation of groups point to the importance of ties with others in man's evolution. Social bonds were powerful adaptive mechanisms, "facilitating such biologically important activities as the protection and upbringing of offspring, hunting, making shelter and collaborating in defense against predators and hostile others"(Henderson, 1980, pg.64). Such social ties were (and are) sources for many of the material, informational and emotional "goods" needed to function adaptively in the surrounding social and physical/material world, providing things that may either be unobtainable without such social interaction or which could only be independently obtained at much greater psychological or material cost (Baumeister & Leary, 1995). Extensive sociological and anthropological research

documents the central importance of the social environment to attitudes and behaviors. There has been substantial research, for example, on the impact of family setting for child development (Baumrind, 1991), the consequences of the work environment for personality and cognitive performance (Kohn et al, 1990), the relevance of neighborhood sub-cultures for delinquency (*Jessor et al, 1996) and the significance of social networks for political life and community cohesion (Bellah, et al, 1985). Within this context, extending the realm of “outcomes” influenced by the social environment to health outcomes was not such a great leap.

In this chapter, we examine evidence linking three broad aspects of the social environment to health. First, we examine the network of personal social relationships within which most of us live our lives, including ties with our parents, children, other relatives, close friends and other social contacts at work and other social groups to which we may belong. Second, moving to a somewhat broader perspective, we examine evidence linking the individuals’ socio-economic status (SES) to their health and well-being, with particular attention to the ways in which SES relates to a range of other exposures and experiences likely to affect health risks. Finally, we consider the question of community-level social characteristics and their potential impact on the health risks of those exposed to differing socio-cultural/socio-economic environments. In the concluding section of this chapter, we discuss similarities and differences in epidemiological and demographic approaches to questions relating the social environment to health and aging with specific attention to potential points of fruitful collaboration in future research – collaborations that could provide greater insight into the likely population-level effects of efforts to promote health through interventions targeted at individual and/or community-level features of the social environment.

People & Their Social Networks

The latest surge of interest in the social environment as a contributor to health and illness dates largely from the latter half of the 20th century and such seminal articles as John Cassel's "The Contribution of the Social Environment to Host Resistance" (Cassel, 1976). In this article, Cassel reviewed animal and human research illustrating the importance of social integration to health, and the negative health consequences that can be seen to result from a lack of social contacts.

Since that time, two broad aspects of the social environment have most frequently been studied in relation to health outcomes. Structural features of the social environment have largely been studied in terms of the number of social relationships that people have (i.e., their level of social integration); structural analyses have also examined the impact of specific types of relationships (e.g., being married). More qualitative aspects of the social environment have also been studied, largely under the rubric of "social support". In contrast to measures of social network structural characteristics that typically involve counts of existing ties, research on more functional characteristics of the network typically includes measurement of the relational content of social interactions (e.g., amount of emotional and/or instrumental support that is provided).

Mortality: To date, the association between greater social integration and lower mortality risk is perhaps the best documented of the social environment-health relationships, beginning with Durkheim's reknown treatise on suicide (Durkheim, 1955) and continuing with the extensive epidemiologic and demographic research of the past 25 years (Broadhead et al, 1983; Goldman, 1993; House et al, 1988; Berkman, 1995; Rogers, et al., 2000). Figure 1 presents data on 5-year mortality from the New Haven cohort of the Established Populations for the Epidemiological Study of the Elderly, illustrating the general pattern of the findings from various longitudinal, community-based studies. As shown, decreasing levels of social integration (as measured by a summary index reflecting

ties with a spouse, close friends and relatives and participation in church and other types of groups) are associated with increasing mortality (Seeman et al, 1993).

Insert Figure 1

Analyses of other community-based, prospective studies, including the Alameda County (Berkman & Syme, 1979), Tecumseh, Michigan (House, Robbins, Metzner, 1982), Durham, North Carolina (Blazer, 1982) and Evans County, Georgia (Schoenbach et al, 1986) studies, as well as results from a nationally representative sample of Americans (Rogers et al., 2000) have provided further evidence for the protective effects of social integration with respect to longevity. A number of community studies from Sweden (Orth-Gomer & Johnson, 1987; Welin et al, 1985) and Finland (Kaplan et al, 1988) have shown similar results.

Morbidity: Relationships between social integration and more intermediate health outcomes have also been documented, including incidence and prognosis for both physical and mental health outcomes (Broadhead et al, 1983; Seeman, 1996; Cohen & Syme, 1985; Goldmen et al., 1995; Waite and Gallagher, 2000). Available data on relationships between social ties and morbidity most frequently relate to cardiovascular disease, with particular attention to coronary heart disease (CHD) and stroke.

With respect to disease incidence, a longitudinal, community-based study by Orth-Gomer and colleagues in Sweden, examining a cohort of men (aged 50 at the inception of the study), found a significant protective effect of social integration with respect to incidence of CHD (defined as either nonfatal MI or death attributed to CHD in those found to be free from heart disease at the baseline examination) (Orth-Gomer et al, 1993). In a study of members of a health maintenance organization, Vogt and colleagues (Vogt et al, 1992) also found a protective effect of social integration (as indexed by the scope or range of different types of ties): those reporting a wider *range of social ties* experienced significantly lower 15-year incidence of MI (based on medical record review), again

independent of standard CHD risk factors. However, there were no significant associations with incidence of other disease outcomes such as hypertension, stroke and cancer. Other studies, such as the Japanese-American study in Hawaii, have not found significant effects of overall social integration (based on marital status, ties with children and parents, social activities with coworkers, and group memberships) with respect to incidence of heart disease (based on examination and review of medical records) (Reed et al, 1983).

One possible reason for the lack of a consistent association between level of social integration and incidence of CHD is suggested by findings from a study of Israeli men where those reporting higher levels of family problems were found to be at significantly increased risk of developing angina (Medalie & Goldbourt, 1976). As these latter data highlight, and as will be discussed further below, greater social integration, to the extent that it is accompanied by greater interpersonal conflict or other interpersonal problems, may not be uniformly associated with health benefits, including reduced risk of heart disease (or other health outcomes).

With respect to disease severity, several studies of patients undergoing coronary angiography have examined possible associations between social integration and extent of coronary atherosclerosis. In one study of 161 angiography patients, no association was found between level of social integration (based on marital status, ties with close friends/relatives, and group memberships) and the extent of coronary atherosclerosis (CAD) (Seeman & Syme, 1987). However, a significant, inverse association was found between levels of instrumental support provided by others and less CAD. A second study (Blumenthal et al, 1987) also found a significant, inverse association between levels of emotional support and extent of CAD (particularly among Type A patients). Thus, while structural features of social integration (as indexed by social ties with others) do not appear to be associated with disease severity, qualitative features such as levels of social support do exhibit a negative association with disease severity

– more support being associated with less CAD.

Perhaps the strongest evidence linking social integration and support to morbidity relates to the evidence of beneficial effects with respect to recovery from CVD events such as MI and/or stroke. Beginning with the reports of Ruberman et al (1984) from the B-HAT trial and continuing with reports from Williams et al (1992) and Case et al (1992), more socially isolated individuals have been shown to experience greater mortality post-MI. A study by Berkman et al (1992), also examining survival post-MI, provides evidence suggesting that levels of emotional support may be one important reason why social isolation conveys greater mortality risk in post-MI patients. Emotional support was measured prospective to the actual MI as all subjects were part of the longitudinal EPESE study in New Haven, CT. MI's were identified through hospital monitoring for all cohort members, with chart review to confirm MI diagnoses. In multivariate logistic analyses, controlling for age, severity of MI and other comorbidity, subjects reporting no sources of emotional support experienced nearly three-fold greater 6-month mortality than those reporting one or more sources.

Analyses of data on incidence and recovery from stroke from the New Haven EPESE study have shown a similar pattern of associations between social integration and incidence versus recovery from stroke. As in the previous study of MI's, strokes among cohort members were identified through hospital monitoring for all cohort members. Level of social integration was not a significant predictor of incident stroke in this older cohort, though the direction of the effect was toward increased incidence among the less socially integrated (Colantonio et al, 1992). By contrast, post-stroke recovery was related to pre-stroke level of social integration (Colantonio et al, 1993). As in the case of the Berkman et al (1992) study of post-MI recovery, several studies have also found that levels of emotional support post-stroke are predictive of better recovery (Friedland & McColl, 1987; Glass & Maddox, 1992; McLeroy et al, 1984), suggesting that greater available emotional support may be one reason that

increased social integration predicts better functional status post-stroke.

Research examining relationships between social ties and overall level of physical functioning has largely focused on the positive or protective effects of network structural characteristics (e.g., network size, marital status). Marital status, for example, has been identified as a protective factor in some (Mor et al, 1989; Palmore et al, 1985) but not all (Roos & Havens, 1991) studies of physical functioning. Measures of social network size have more consistently indicated a protective effect with respect to physical disability (e.g., see Boult et al, 1994; Kaplan, Strawbridge et al, 1993). Indirect evidence of the role of social ties in the etiology of physical disability is also suggested by evidence of protective effects of social ties with respect to two important outcomes of disability: institutionalization and mortality (Freedman et al, 1994; Steinbach, 1992; Wolinsky et al, 1992).

Most recently, effects of social ties on cognitive aging has also been a focus of research. Several recent studies have demonstrated significant protective effects of social integration with respect to lower risk for dementia (Bassuk et al, 1999; Fratiglioni et al, 2000). In both studies, older adults reporting greater numbers of social ties and social engagement at baseline were less likely to exhibit incident dementia or cognitive impairment during follow-up (for follow-up periods ranging from 3 to 12 years). Analyses from the MacArthur Study of Successful Aging, focusing on a more selected, high functioning cohort of older adults, and looking at characteristics of the social environment as predictors of cognitive decline (rather than impairment or dementia) suggest that levels of social support (specifically emotional support) may be more strongly related to earlier stages of cognitive decline (Seeman et al, submitted).

Mental Health/Distress: The effects, both positive and negative, of family and friends on mental health have been extensively documented (see George, 1989 for review). Studies of the impact of social ties on risk of depression, for example, have generally found protective effects associated with

greater social integration, particularly as this is reflected by the presence of primary ties with spouse, children and/or supportive significant others (see Broadhead et al, 1983; Biegel et al, 1985; Brown & Harris, 1978; and George, 1990 for reviews). And, marital disruption, either through bereavement or marriage dissolution, has been associated with increased risks for psychological distress (Aseltine & Kessler, 1993; Bloom et al, 1978; Oxman et al, 1992; Williams, Takeuchi & Adair, 1992). "Losing your spouse" -- not through death, but through their cognitive impairment -- has also been shown to result in increased risk for depression (Moritz et al, 1989). The latter data also shed light on potential reasons for this effect-- among other things, increasing levels of cognitive impairment of one's spouse result in dramatic decreases in the availability of emotional support from this important source; this effect is particularly strong for husbands of cognitively impaired wives. The broader family/friend environment (i.e., children, close relatives and friends) can, however, compensate for this loss of "spouse" support, serving as a modifier of the link between the wife's cognitive impairment and the husband's risk of depression. Where the family/friend environment is perceived to provide adequate instrumental, emotional and/or financial support, the husband's risk of depression is not increased.

Negative Health Effects of Social Relationships: While the vast majority of the social epidemiological and social psychological research on health has focused on the hypothesized benefits of social ties, and in particular, on the postulated benefits of the emotional and instrumental support such ties provide (Cohen & Syme, 1985; Sarason & Sarason, 1985), social exchange theorists and others have long argued that social relationships are frequently characterized by costs as well as benefits (Blau, 1964; Duck, 1982; Thibaut & Kelley, 1959, Wellman, 1985). Such costs can take the form of requests/demands for assistance, criticism or other forms of interpersonal conflict (Averill, 1982; Kessler, McLeod & Wethington, 1985). More specifically, while the positive health effects of social ties have been hypothesized to result from their ability to enhance the individual's self-esteem, sense of

belonging and efficacy or mastery through positive, supportive actions (Antonucci & Jackson, 1987; Thoits, 1985), social ties can also be sources of demands, conflict, embarrassment, envy, disappointment, and devaluation as well as serving as models for "risky" or "unhealthful" behaviors (Thoits, 1985; Rook, 1985; Schuster et al, 1990). Furthermore, the fact that supportive and nonsupportive aspects of social relations appear to be only weakly correlated (Schuster et al, 1990; Finch et al, 1989) indicates that these are two relatively independent domains of social experience and that both need to be considered to understand the influence of family and other social ties on health.

The mental health consequences of social relationships characterized by more negative qualities has received considerable attention from health psychologists. Social relationships characterized as intrusive, demanding, critical, sources of conflict or "non-supportive" have been shown to predict increased depression and/or decreased well-being (Schuster et al, 1990; Abbey et al, 1985; Lepore, 1992; Pagel et al, 1987; Pearlin, 1975; Rook, 1984; for review see Burg & Seeman, 1994). Research has also shown that the failure of the family/friend environment to provide anticipated or expected support can result in increased psychological distress (Pagel et al, 1987; Brown & Harris, 1989). Indeed, negative and/or non-supportive social interactions have been reported as one of the major (and more distressing) sources of daily stress (Bolger et al, 1989) and evidence suggests that negative qualities of relationships may have stronger impacts on affect and mental health than positive qualities (Rook, 1984).

Interestingly, while both men and women report more supportive than negative social interactions, women have been found to report relatively more negative interactions with non-discretionary, family/kin ties (Schuster et al, 1990; Lefler et al, 1986). Such negative interactions, especially when they involve a spouse or close friends, have been shown to be more strongly predictive of psychological distress than similar interactions with more distant social relations (Abbey et al, 1985).

Negative interactions with close social relations also appear to have stronger effects on psychological distress than supportive interactions, especially among women, who appear to be more emotionally responsive to such negative interactions (Lefler et al, 1986; Wethington et al, 1987).

Qualitatively negative aspects of social relationships have also been related to physical health outcomes. Poor marital quality appears to be particularly potent in this regard, having been shown to predict increased risk of angina (Medalie & Goldbourt, 1976) and greater reported physical illness (Coyne & DeLongis, 1986); declines in marital satisfaction have also been related to declines in reported health status (Levenson & Gottman, 1985). Poorer marital quality and chronic family stress associated with caring for family members with Alzheimer's disease have also been shown to predict poorer immune function (Kiecolt-Glaser et al, 1987b; Kiecolt-Glaser et al, 1988); and, perceived lack of support from family and others has been related to elevations in serum cholesterol and illness symptoms (Gore, 1978).

The literature on recovery from, or coping with, illness also reveals that family/friend "assistance" in coping with illness and/or disability can result in unintended negative outcomes such as increased reports of dependency and disability (DiMatteo & Hays, 1981; Garrity, 1973). Recent analyses of data from the MacArthur Study of Successful Aging provide similar evidence of the potential for negative effects on physical health (Seeman, Bruce & McAvay, 1996). In these analyses predicting onset of Activities of Daily Living (ADL) disability in a cohort of older men and women, higher baseline levels of instrumental assistance are associated with increased risk of new ADL disability over a two year follow-up among the men, independent of baseline levels of physical functioning.

Pathways for Social Effects on Health:

Though evidence linking social integration to decreased risks for morbidity and mortality

continues to grow rapidly, important questions remain concerning the precise mechanisms or pathways through which the social environment influences health outcomes. Various pathways have been hypothesized as possible avenues through which the family/friend social environment affects health outcomes. These pathways include social environment influences on psychological and behavioral factors that are known to impact on health and longevity as well as more direct social environment influences on physiology (see Figure 2). The following section reviews evidence linking characteristics of the social environment to these various possible pathways and the potential for positive and/or negative social effects on behavior and physiology.

Figure 2

Health behaviors (Pathway a, Figure 2): As both behavioral role models and sources of information and cultural norms, social ties have been shown to influence both the likelihood that particular behaviors will be adopted as well as the likelihood of behavior change. Early in life, family environments are a primary learning environment for children, having the potential to teach healthful or non-healthful behaviors. For example, children of smokers are more likely to smoke (Leventhal & Cleary, 1980), those with obese parents are more likely to become obese themselves (Stunkard, 1979), and those with problem drinkers for parents are more susceptible to alcoholism (Moos et al, 1982). Among adolescents, adoption of detrimental behaviors such as smoking (Cohen et al, 1988), alcohol and other substance abuse has been shown to be importantly linked to peer group norms (Jessor et al, 1991; Jessor & Jessor, 1977).

Among adults, similar peer group effects continue to be seen with respect to the persistence of healthful and non-healthful behaviors. On the positive side, marriage and friendship ties are frequently associated with greater preventive health behavior, including less smoking and drinking (Broman, 1992; Stroup-Benham et al, 1990; Rogers et al., 2000), and more cancer screening (Calnan, 1985; Kang et

al, 1994; Suarez et al, 1994). Social ties more generally have been shown to predict greater preventive health behavior (Seeman et al, 1985), better nutrition (Frongillo et al, 1992; McIntosh et al, 1989; Walker & Beauchene, 1991), increased use of mammography (Kang & Bloom, 1993) and more successful risk reduction efforts such as reducing dietary fat, exercising and smoking cessation (House et al, 1982, Bovbjerg et al, 1995; Duncan & McAuley, 1993; Treiber et al, 1991).

Family/friend environments, however, also carry the potential for encouraging more detrimental health behaviors such as problem drinking (Seeman M, et al, 1988), worse control of diabetes (Kaplan RM, et al, 1985; Kaplan & Hartwell, 1987) and less successful risk reduction efforts (Cohen, 1992; Murray et al, 1995). Spouse pairs, for example, show concordance for smoking, drinking and level of physical exercise (Venters et al, 1984). Heavier drinking has also been shown to be associated with greater social activity (Cahalan et al, 1969; Seeman & Anderson, 1983) with problem drinkers reporting a higher prevalence of fellow drinkers in their social networks (Hobfoll, 1985). And, the presence of other smokers in one's social network has been associated with greater relapse in efforts to quit (Cohen et al, 1988; Mermelstein et al, 1986). Studies among gay men also indicate that engaging in risky behavior such as unprotected anal intercourse is related to perceptions that one's friends and social contacts engage in such behavior (McKusick et al, 1990; Peterson et al, 1992). Family/friend environment can also present more direct health threats in the form, for example, of second-hand smoke exposure from living with a smoker (Emmons et al, 1994; Sandler et al, 1989) or physical violence (McLeer & Anwar, 1989; Mercy & Saltzman, 1989).

Compliance and non-compliance with preventive and/or therapeutic medical regimens have each been linked to the presence of more social ties (Blackwell, 1979; Levy, 1983). Indeed, while significant others such as a spouse or other family members can be particularly important as models of appropriate behavior and normative influences, as well as exerting more direct control over dietary or

medical regimens, they can also serve as models of inappropriate or non-normative behaviors (Becker & Green, 1975) or as sources of discord and dissuasion regarding compliance behavior (Strickland et al, 1981). Such factors may explain why men with larger social networks were found to exhibit poorer control of their diabetes (e.g., significant increases in glycosylated hemoglobin, cholesterol and triglycerides and similar trends for glucose and weight) at 18-month follow-up (Kaplan et al, 1985).

Physiological Mechanisms (Pathway b, Figure 2): The question of possible, direct links between family/friend environment and physiology is one that continues to intrigue researchers, particularly in light of the fact that social ties remain significant predictors of morbidity and mortality in their own right, independent of any associations with health behaviors (Broadhead et al, 1983; House et al, 1988; Seeman & Syme, 1987; Cohen, 1991).

The idea that social relationships influence physiology is congruent with a broader, evolutionary perspective of human physiology and its susceptibility to modulation by external social factors (Alcock, 1983; Washburn, 1961). The basic premise is that, like other social animals, human physiologic homeostasis and ultimate health status are influenced not only by the physical environment but also by the social environment (Bovard, 1985; Henry, 1988; Weiner, 1977). As reviewed in Seeman and McEwen (1996), there is a growing body of evidence for such links in both animal and human populations. In both cases, the data highlight not only the "biological" impacts of such social ties but also the dual nature of these impacts: the presence of more positive, supportive relationships is associated with lower levels of cardiovascular and neuroendocrine activation while more conflictual and non-supportive relationships appear to heighten physiological reactivity.

The effects of the family environment begin at the earliest ages. Secure attachment between infant and mother has been shown to predict lower stress response (as indexed by salivary cortisol) to new or strange situations (Hertsgaard et al, 1995). A broader range of social influences has also been

demonstrated even at these early ages. Gunnar et al (1992), in a study of adrenocortical responses to separation, demonstrated that cortisol responses were attenuated in the presence of a "warm, responsive and interactive" caregiver, much like the effects of a "supportive confederate" or friend in studies of adults discussed below (Lepore, 1992; Gerin et al, 1992; Gerin et al, 1995). Among adults, community-based population studies provide evidence for associations between higher reported levels of support from close friends and family and better physiologic profiles, including lower heart rate and systolic blood pressure (Bland et al, 1991; Dressler et al, 1986; Dressler, 1983; Uden et al, 1991), lower serum cholesterol (Gore, 1978; Thomas et al, 1985; Cobb, 1974; Kushnir & Kristal-Boneh, 1995), and lower urinary catecholamines and cortisol (Kemp & Hatmaker, 1989; Seeman et al, 1994).

Data from smaller experimental studies, where aspects of the social environment are manipulated, provide even stronger evidence that social contact or support can attenuate (or exacerbate) physiologic stress responses as indexed by cardiovascular, autonomic and hypothalamic-pituitary-adrenal (HPA) axis responses. Presence of a friend, for example, has been shown to decrease cardiovascular stress responses to challenging tasks such as mental arithmetic, concept formation and mirror-tracing tasks (Edens et al, 1992; Kamarck et al, 1990). As in the studies of infants discussed above, studies also suggest a broader range of social influences than simply family or "significant others", showing that the presence of "supportive strangers" is also associated with attenuated systolic and diastolic blood pressure responses when asked to complete a challenging task (Gerin et al, 1992; Lepore et al, 1993).

Negative physiologic effects of social relationships on neuroendocrine and/or cardiovascular activity have also been documented. In the case of marital interaction, spousal interactions characterized by greater hostility and conflict have been shown to elicit greater cardiovascular and

neuroendocrine reactivity (Brown & Smith, 1992; Ewart et al, 1991; Gottman & Levenson, 1992; Kiecolt-Glaser et al, 1993). More generally, experimental studies have shown that individuals exhibit increased cardiovascular and neuroendocrine reactivity when exposed to non-supportive social conditions such as interpersonal challenge or hostility (Lepore et al, 1993; Brown & Smith, 1992; Back & Bogdonoff, 1964; Dembroski et al, 1979; Dembroski et al, 1977; Dimsdale et al, 1988; Krantz et al, 1986). Gerin et al (1992), for example, found a pattern of greater blood pressure response when subjects participated in a discussion where others disagreed with the subject. Interestingly, blood pressure levels were halved if one other person shifted to agreement with the subject.

A number of studies have also indicated that social relationships can have both positive and negative impacts on immune function (see Kiecolt-Glaser & Glaser, 1991 for review). This research has shown that for both men and women, those who were not married exhibited poorer immune function, as indexed by increased Epstein Barr virus antibodies (Kiecolt-Glaser et al, 1993). Among those who are married, the quality of the marital bond has been shown to be important with those reporting less attachment to their relationship exhibiting lower immune function (Kiecolt-Glaser & Glaser, 1991; Kiecolt-Glaser et al, 1987a) and negative behavior during marital conflict has been shown to down-regulate immune function (Kiecolt-Glaser et al, 1993). More generally, levels of social support have been found to relate positively to immune function (Thomas et al, 1985) and to impact on patterns of change over time. A recent study by Theorell et al (1995), for example, found that among HIV-infected men, those reporting lower levels of emotional support from family and/or friends exhibited more rapid decline in CD4 counts over a 5-year follow-up.

Summary: The above data clearly indicate that the network of personal social relationships within which an individual lives his or her life impacts on a wide range of health outcomes, from lifestyle risk factors through morbidity risks to actual mortality risks. These social environment effects appear to

operate through multiple pathways, with evidence for direct effects on physiologic parameters as well as more indirect effects on attitudes and behaviors. Importantly, the health effects of one's social relationships can be positive (i.e., health-promoting) or negative (i.e., health-damaging). It is not surprising that our social relationships should have such widespread effects. As discussed earlier, man has evolved as a social animal and, as such, appears to be "tuned in" to the others in his world. In light of this, it should not be surprising to find that the quality of our interactions with others has profound effects on our psychological, behavioral and physiologic functioning.

Socio-Economic Status & Health

In addition to the personal, social relationships that we have with others, a broader "social" environment that influences our life experiences, and our health and well-being, is that occasioned by our relative social and economic status within the larger society in which we live. Socio-economic status (SES) is traditionally measured in terms of indicators such as education, income and occupation. By these criteria, an extensive and extraordinarily consistent body of evidence has accumulated documenting the negative health outcomes associated with lower SES (for reviews see Adler et al, 1993; Adler et al, 1994; Pincus & Callahan, 1995; Macintyre, 1997; Feinstein, 1993; Rogers et al., 2000; Williams, 1990). Lower SES, for example, has been related to higher prevalence and incidence of most chronic and infectious diseases (Pincus & Callahan; Adler et al 1993; Adler 1994; Hayward et al., 2000; Kaplan & Keil, 1993), and, for exceptions such as breast or prostate cancer, where higher incidence is found among higher SES groups, survival is nonetheless worse among lower SES groups (Dayal et al, 1982; Dayal et al, 1985).

An inverse association between SES and mental distress/disorder has also been extensively documented (Goldstein, 1979; Schwab & Schwab, 1978; Dohrenwend & Dohrenwend, 1969).

Lower SES has been associated with higher incidence and prevalence of both major depression and depressive symptoms (see Adler et al, 1993 for review), as well as with increased prevalence of more general psychological symptomatology (Wheaton, 1980; Dowdall & Meudell, 1982). Indeed, lower SES has been related to higher rates of nearly all major causes of morbidity and mortality both across populations and over time (Macintyre, 1997; Adler et al, 1994; Smith, 1999). Evidence suggests further that SES differentials in morbidity and mortality may be widening rather than narrowing (Wagener & Shatzkin, 1994; Pappas et al; Marmot, 1994; Macintyre, 1997; Feldman et al, 1989; Preston and Elo, 1995; Crimmins & Saito, 2001; Schalick et al., 2000).

This enduring and ubiquitous association between SES and health/longevity tends to elicit interpretations that focus on the obvious detrimental effects of deprivation and difficult circumstances at lower SES (e.g., poverty, living and working conditions). However, the data actually suggest that the association is more linear than threshold, with a gradient of increasingly better health outcomes as one mounts the SES continuum (Adler et al, 1993; Marmot et al, 1984; Marmot et al, 1991), though data from both the US and Britain suggest that the slope may flatten somewhat at higher SES levels (e.g., Kitagawa & Hauser, 1973; Pappas et al, 1993, House et al, 1990; Wilkinson 1986; McDonough et al, 1995). The fact that the relationship takes the form of a linear gradient, rather than showing a threshold effect of "poverty", clearly indicates that a comprehensive explanation for the association of SES with health and mortality will involve more than the obvious factors of inadequate financial resources or poor/dangerous living conditions associated with actual poverty. Any final explanation will have to account not only for the higher morbidity and mortality among the poorest segments of society but also for the relatively higher morbidity and mortality in the middle ranges as compared with the upper ranges of SES.

Another feature of note regarding SES differentials in adult health is the age-related variation in

this association with the greatest SES differentials in health and mortality seen in the 40-65 age range (Ford et al, 1994; House et al, 1990, 1992, 1994). This finding suggests that the effects of SES are, to some extent, the result of a cumulative process beginning in early childhood (Blackwell et al., 2001; Elo and Preston, 1992) which produces differentials in health/mortality only with increasing exposure and increasing biological vulnerability. Thus, differentials are generally smaller prior to middle-age and are again reduced after age 65 where the impact of more general age-related increases in morbidity and mortality is likely to result in higher rates even for those living in more favored SES circumstances. This pattern of data suggests that SES effects on health result largely from a cumulative process, with lower SES resulting in what might be called "premature" aging with attendant increased risks for nearly all types of negative health outcomes. Data on the effects of cumulative economic hardship (i.e., years with income "<200%" of poverty level) also point to increased risks for a range of poor outcomes, including poorer physical, psychological and cognitive functioning (Lynch, Kaplan, Shema, 1997). Indeed, though SES differentials in health and mortality may be greatest in late middle age, these same SES differentials in health risks persist into old age. Indeed, income and education continue to be associated with differential morbidity and mortality among older adults (Seeman, Charpentier, et al, 1994; Albert et al, 1995; Berkman et al., 1989; Crimmins et al., 1996).

Though a variety of possible explanations for these social inequalities in health have been offered, including artifact of measurement and natural or social selection (for discussion see the Black report [Black et al, 1988]), current opinion favors the view that social inequalities are real and little affected by selection (for discussion, see Macintyre, 1997). Rather, SES-related differences in material/structural conditions are seen as contributing importantly to observed differentials both directly (e.g., via pollution, unsanitary conditions) and through indirect effects on cultural/behavioral practices (Macintyre, 1997). Current discussions of SES gradients in health have been cast most frequently in

terms of parallel gradients in: 1). chronic and acute stressors (at both the individual and broader environmental level), 2). coping resources, and 3). health habits and other lifestyle characteristics. In the following sections, we review evidence for the possible role of these factors as intervening pathways through which SES results in a gradient of health and mortality.

Chronic (and Acute) Stress (Pathway c, Figure 2): Differential exposure to both chronic and acute stressors constitutes one of the foremost factors postulated to contribute to observed health differentials by SES. Increased risks for poor health outcomes associated with lower SES are hypothesized to result from the relatively greater exposure to environmental stress in lower SES environments, including a physical environment characterized by crime, crowding, poor physical amenities, and greater exposure to physical hazards such as industrial and hazardous wastes (Williams & Collins, 1995); a financial/occupational environment characterized by inadequate and/or unpredictable resources, nonexistent or little job security or personal autonomy (Williams, 1990; Kohn & Schooler, 1983); and a socio-cultural environment characterized by discrimination and impoverished social and psychological resources (e.g., fewer socio/political resources, individual perceptions of powerlessness, alienation, lack of self-esteem; Mirowsky & Ross, 1986; Pincus & Callahan, 1995; Kessler et al, 1995; Williams, 1990; Adler et al, 1994; House et al, 1992; Williams, 1999). Recent evidence from a community-based survey (Turner et al, 1995) provides further support for the view that SES differentials in mental health are at least partially explained by differential exposure to stressful life events. Using occupational prestige level as their index of SES, Turner et al (1995) found the expected associations between lower occupational prestige levels and increased depressive symptomatology as well as between lower occupational prestige and greater exposure to stressful life events. However, in multivariate analyses, the differential stress exposure by occupational prestige levels was found to account for 38% of the initial bivariate association between occupational prestige and depressive

symptoms.

Evidence for such SES-related differentials in exposure to stressors, particularly chronic stressors, is abundant, with lower SES individuals consistently reporting more chronic and acute stressors (Dohrenwend & Dohrenwend, 1970; Dohrenwend 1973; Kessler & Cleary, 1980; Kessler 1979; House et al, 1992; Turner et al, 1995). Analyses by *McLeod* & Kessler (1990), using data from five epidemiologic surveys, indicate that this increased exposure to stressors cuts across life domains (i.e., it is not confined to events related to financial issues but includes more events related to health and one's social network as well). Kessler and colleagues (Kessler & Cleary, 1980; *McLeod* & Kessler, 1990) have also demonstrated the synergistic effects of such stress exposure in the context of lower SES, showing that, in addition to their more frequent exposure to stressful events, lower SES individuals exhibit greater negative impact of such events on psychological functioning. These findings parallel those from other studies examining SES differences in mental health outcomes for individuals exposed to similar levels/intensities of stressful experience which also show greater vulnerability among lower SES groups (for review see Thoits, 1995).

Further contributing to the chronic stress of lower SES status are neighborhood characteristics such as police-documented higher crime rates (Macintyre et al, 1993) as well as greater perceived threat of crime and more local problems (Sooman & Macintyre, 1995), more refused services (e.g., taxi, credit, ambulance; Sooman & Macintyre, 1995), and poorer transportation services and recreational facilities (Macintyre et al, 1993). Lower SES neighborhoods have also been associated with greater exposure to physical hazards such as air and water pollutants, hazardous wastes, pesticides and industrial chemicals (Calnan & Johnson, 1985).

Clearly, lower SES status (especially if, as is generally the case, it is accompanied by residence in a lower SES neighborhood) is more likely to result in increased exposure to chronic and acute

stressors stemming from physical, economic, and social characteristics of the environment. The potential mediating pathways from such exposure to increased disease susceptibility are suggested by findings that chronic stress as indexed by residence in higher population-density areas is associated not only with increased reports of "stress" but with increased biochemical indices of stress such as urinary excretion of norepinephrine and epinephrine (Fleming et al, 1987). The immediate and long-term health consequences of the greater "stress" exposure of lower SES individuals is illustrated by two sets of findings. The first relates to immediate consequences in terms of pregnancy outcomes where greater prenatal stress has been associated with lower birthweight (Lobel et al, 1992). The second set of findings relates such differential prenatal exposures to longer-term health consequences. Barker has proposed that adult differentials in health and mortality are influenced by biological programming occurring *in utero* or early infancy (Barker, 1994), suggesting, for example, that lower SES may exert an impact prenatally not only on ultimate birthweight (a known correlate of infant health outcomes) but on future adult health outcomes as well.

Psychological Resources (Pathway d, Figure 2): The association of lower SES with reduced coping resources has also been suggested as a contributor to the observed health differentials. Indeed, reduced coping resources may serve to heighten the impact of the chronic and acute stressors discussed above (e.g., contributing to the pattern of greater impact of such stressors in lower SES groups; Kessler, 1979; Kessler et al, 1995). Lower SES, for example, has been related to a number of characteristics which have been found to serve as risk factors for health or as moderators of the relationship between stressful experiences and health outcomes including lower optimism (Pincus & Callahan, 1995), lower self-esteem and feelings of self-worth (Pearlin & Schooler, 1978; Rosenberg & Pearlin, 1978; Matthews et al, 1989; Turner & Roszell, 1994), lower self-efficacy beliefs (House et al, 1992), and a weaker sense of control (Marmot, Davey-Smith, Stansfeld et al, 1991; Gurin & Gurin,

1976; Mirowsky & Ross, 1986) and greater fatalism (Wheaton, 1980). The latter two characteristics in particular are thought to play important roles in facilitating and promoting coping efforts (Wheaton, 1980; Syme, 1989; Mirowsky & Ross, 1986). Higher SES, in the form of more education and greater occupational autonomy, as also been shown to foster development of more positive self-perceptions and greater cognitive flexibility (characteristics which influence coping resources and responses) (Kohn & Schooler; 1983). Pearlin & Schooler (1978) also document education-related differentials in the coping strategies used to deal with life events, with the less educated using relatively less effective strategies.

Exacerbating such individual psychological vulnerabilities, lower SES individuals also tend to have fewer social resources to call on for support and assistance in coping with events (e.g., fewer social ties and less support, and less contact with more formal social institutions such as religious or other groups; Berkman & Breslow, 1983; Murrell & Norris, 1991; Matthews et al, 1989; House et al, 1994; Fischer 1982; Turner & Marino, 1994; Comstock & Partridge, 1972) (Pathway e, Figure 2). The connections and interactions between various SES-related pathways to disease are further suggested by evidence indicating that chronic and acute stressors such as financial strain and fear of crime (which are more commonly found in lower SES environments) tend to foster a distrust of others which can promote greater relative social isolation (Krause, 1992). The potential importance of both social and psychological resources as mediators of the greater stress exposure in lower SES environments is suggested by a study of SES (indexed by occupational status) and psychological vulnerability to stressful life events. In this study, Turner and Noh (1983) found evidence for a buffering effect of both social support and a sense of control on the relationship between lower occupational status and vulnerability to life events -- among women with high social support and stronger feelings of personal control, the relationship between lower status and vulnerability to life events disappeared.

Notwithstanding such buffering effects of social and psychological factors, evidence on the "costs of coping" (Cohen, 1980) would suggest that the greater exposure to chronic and acute stressors of lower SES individuals may nonetheless exact a cumulatively greater toll, even for those with good coping skills.

Health Behaviors (Pathway f, Figure 2): Lower SES is also associated with a higher prevalence of unhealthful lifestyle characteristics, including less exercise, more smoking and alcohol consumption, and poorer diets (Winkleby et al, 1990; Marmot et al, 1991; Macintyre 1994; House et al 1992, 1995; Rogers, 1992; for reviews see also Williams & Collins, 1995; Adler et al, 1994; Kaplan & Keil, 1993; Preston and Taubman, 1994) Indeed, national survey data indicate that lower SES is associated with a relative disadvantage on every risk factor considered (House et al, 1995; Rogers et al., 2000) -- a finding that is corroborated by other community-based surveys from Alameda County in California and Kuopio, Finland (Lynch et al, 1997). The important influence of broader environmental characteristics on these patterns of behaviors is suggested by findings from Macintyre and colleagues documenting that lower SES neighborhoods in Glasgow are characterized not only by fewer recreational facilities and poorer transportation service (e.g. to facilitate access to other areas) (Macintyre et al, 1993; Macintyre, 2000) and by less availability and higher cost for foods recommended for health-promotion (Sooman & Taggart, 1992; Sooman, Macintyre & Anderson, 1993). Parallel data have also been reported for the US (Troutt, 1993). These data may also help to explain why lower SES groups have been less likely to adopt widely promulgated recommendations for exercise and healthy diets (Macintyre, 2000; Adler et al, 1993).

Not surprisingly, in light of the behavioral data outlined above, an SES gradient in other known biological risk factors for major chronic diseases such as CHD has also been documented (e.g., higher cholesterol, more obesity and hypertension; Macintyre, 1994; Matthews et al, 1989; Kaplan & Keil,

1993; Winkleby et al, 1990). Importantly, while much of the early research on CHD and its risk factors focused on these associations among men, more recent research documents a similar SES gradient for CHD risk factors among women (Winkleby, et al, 1990; Matthews et al, 1989).

Medical Care: Differential access to quality health care is another factor generally thought to contribute to observed SES differences in health and mortality. Indeed, data from England and the US indicate that despite universal health insurance in the former and federally-funded programs such as Medicaid and Medicare in the latter, lower SES individuals continue to have access to less extensive and lower quality medical resources (Williams, 1990; Macintyre et al, 1993; Wyke et al, 1992). However, recent reviews have suggest that there are several reasons to believe that differential access to medical care is not the primary, or perhaps even a major contributor, to observed SES differentials in health and mortality (Williams, 1990; Adler, et al, 1993). For example, health and mortality data from countries such as Britain, where universal health insurance has been available for some time, nonetheless continue to the "traditional" SES gradient in health and mortality and, indeed, show evidence for a widening of the social class differences (Wilkinson, 1986). In addition, the inverse relationship between SES and mortality is seen not only for causes of death "amenable to medical care" but for causes "not amenable to medical care" as well (Marmot et al, 1984; Kaplan & Keil, 1993; Woolhandler et al, 1985) suggesting again that differentials in medical care, though a possible contributory factor, are unlikely to play a definitive role in the SES gradient in health. SES differentials in access to and quality of medical care remain, however, one of what are clearly a multiplicity of sources of SES-related "health disadvantage" (Macintyre et al, 1993).

Summary: To date, the documented association between lower SES and negative health outcomes has generated considerable research and policy interest, but our explanations for and understanding of this relationship remain incomplete. While research has shown that the association can

be "accounted for" in part by differences in health behaviors, psychological and social factors, even the most complex multivariable analyses leave a significant, unexplained variance in outcomes across the SES gradient (Lynch et al, 1996; House et al, 1994; Marmot, 1994). Clearly, much remains to be elucidated regarding the actual mechanisms through which something as complex and multifaceted as SES "gets under the skin". This will necessarily include consideration of external characteristics of the environments (both physical and sociocultural) where people live and work, and individual characteristics as well as possible interactions between these in producing the observed SES gradients in health and mortality.

Of particular significance in this matrix of factors may be those reflecting the individual's social/psychological "environment". Evidence clearly points to the importance of such factors in considering the greater "resilience" of some adults and children in the face of hardship -- hardship which is itself usually largely grounded in their lower SES. Those exhibiting better developmental and health outcomes despite the hazards of lower SES are quite routinely found to be characterized by better social and psychological resources (e.g., social support and sense of control [Turner & Noh, 1983], as well as supportive/positive adult role models [Werner, 1990]) which appear to buffer against the "expected" negative outcomes usually associated with their lower SES environments.

Whatever the relative importance of differing factors to the SES-health gradient, the ultimate picture of the SES-health relationship will be one in which SES exerts its effects on health via multiple potential pathways with the specific individual-level pathways depending on specifics of both the environment (e.g., physical, economic, sociocultural factors) and the individual (e.g., genetic, social, psychological and economic factors). Two known features of the SES-health relationship already point to such a multiplicity of potential pathways. First, there is the remarkable consistency of the observed association, irrespective of the various indicators of SES (e.g., income, education or occupation as well

as more multidimensional indices; Liberatos, et al, 1988). Second, there is the remarkable stability of SES-related differentials in mortality which have remained largely constant in the face of shifting patterns of disease and risk factors, including most notably the shift from predominantly infectious to chronic diseases and their very different risk factors. Such consistencies, despite shifting measurement of SES and shifting disease patterns, suggest the availability of multiple pathways through which SES-related variables influence health and well-being -- it is these pathways that will need to be fleshed out in any comprehensive explanatory model.

It is important to note that the foregoing discussion of SES and health has focused almost exclusively on how SES, broadly conceived, affects the individual -- i.e., how does one's experience of social and/or economic status within the larger society impact on one's health and well-being. There are two further features of the SES environment, however, that deserve special note. The first relates to the multi-dimensionality of the SES environment; the second relates to the broader societal context in which these different SES environments are embedded.

Here, we have focused on SES as broadly defined rather than considering a more detailed evaluation of the relative contributions of various dimensions of SES (e.g., income, education, occupation) to health outcomes. While each of these has been shown to demonstrate the general, inverse SES gradient, these dimensions as well as other aspects of the SES environment likely play importantly differentiated roles in the genesis of different health outcomes -- an area of potential importance for research on the multiple pathways through which SES affects on health.

In addition to this multidimensionality, it is important to note the broader contextual features of what might best be characterized as a gradient of SES environments. These environments are not generated, nor do they persist, in isolation from each other or from the surrounding socioeconomic order. Rather, there are broader socio-economic-political forces that importantly shape and maintain

these socio-economic environments (i.e., what have been termed the "upstream determinants" of such environments; Kaplan, 1995; Lynch, Davey Smith, Kaplan, House, 2000). These forces are likely to factor importantly in any successful effort to understand and ameliorate the progressively worse health outcomes found as one moves down the SES gradient. It is to these "upstream", more global environments that we turn our attention now, examining the growing body of research focusing on relationships between broader community-level social and economic characteristics in relation to the health outcomes of those living in such environments.

Community-Level Socio-Economic Effects on Health

The broader socio-economic environment within which people live out their individual lives has become a focus of increasing research interest over the past decade. Here, we present evidence from two major streams this research -- one focusing on the socio-economic/resource characteristics of these broader communities and the other on more specifically social features of these environments.

Community SES characteristics: Analyses of US census-tract level data have shown consistent effects of area-level SES characteristics on the health experience of inhabitants: those living in lower SES neighborhoods experience higher mortality (Anderson et al., 1996, 1997; Geronimus et al., 1999; Cubbin et al, 2000). Significantly, this area-level effect appears to compound that of individual-level SES. In analyses which include both area-level and individual-level data on SES where area-level SES (i.e., census tract or neighborhood) has been shown to have a significant effect on health and mortality, independent of the effects of individual-level SES (Blaxter, 1990; Jones & Duncan, 1995; Haan et al, 1987). As indicated earlier in the discussion of chronic stress exposure among lower SES groups, some of the community-level environmental factors likely to be contributing to these observed area-level effects include neighborhood characteristics such as police-documented higher crime rates

(Macintyre et al, 1993) as well as greater perceived threat of crime and more local problems (Sooman & Macintyre, 1995), more refused services (e.g., taxi, credit, ambulance; Sooman & Macintyre, 1995), and poorer transportation services and recreational facilities as well as higher costs for “healthy” foods (Macintyre et al, 1993; Troutt, 1993; Mooney, 1990) – the latter perhaps contributing to observed differences in diet (Diez-Roux et al, 1999) and other health behaviors (Duncan et al, 1996; VonKorff et al, 1992; Diez-Roux et al, 2000). Lower SES neighborhoods have also been associated with greater exposure to physical hazards such as air and water pollutants, hazardous wastes, pesticides and industrial chemicals (Calnan & Johnson, 1985). More recently, community differences is what has been referred to as “social capital” have become a focus of considerable research interest.

Community “Social Capital”: The term “social capital” refers to the cooperative network of social relationships between citizens (or members of any defined community or group) that facilitates collective action for mutual benefit and problem resolution (Coleman, 1988). As such, social capital is a community or group level characteristic rather than an individual-level characteristic. Communities characterized by high social capital include such features as high levels of interpersonal trust and norms of reciprocity and mutual aid (Coleman, 1988; Putnam et al, 1993). Such communities have been found to benefit from more effective government (Putnam et al, 1993), lower crime and delinquency (Sampson & Groves, 1989; Kennedy, Kawachi...Gupta, 1998; Sampson et al, 1997) and better health outcomes including lower mortality (Kawachi et al, 1997) and better self-rated health status (Kawachi et al, 1999). Kawachi et al (1997) used state level data from the US to demonstrate a strong negative relationship between mortality rates and levels of social capital (measured in terms of levels of trust, perceived reciprocity, and membership in various types of groups or associations) – those areas with the highest reported levels of social capital showing lower mortality rates; Kawachi et al (1999) have also shown a similar pattern of association with self-rated health status. Some postulated mechanisms

for these observed health effects of social capital include such things as community promotion of health behaviors (through diffusion of information, stronger norms for healthy behaviors, and exerting social control over deviant health-related behavior) and greater community success in acquiring and maintaining health-related services and amenities (Kawachi et al, 1999; Veenstra, 2000).

Community Inequalities: In addition to this interest in varying community-level social characteristics, the potential health consequences of *relative* socio-economic inequalities across and within various “communities” have become a focus of considerable research and debate. Income inequality (i.e., the relative distribution of wealth within a community [variously defined in terms of metropolitan areas (Lynch et al, 1998), states (Kaplan et al, 1996; Kennedy et al, 1996) or countries (Wilkinson, 1992; van Doorslaer et al, 1997)]) has been found to correlate with differential health outcomes, particularly mortality -- communities characterized by greater inequality also showing higher mortality rates (Hayward et al., 1997; Lynch et al, 1998; Lynch et al, 2000; Kaplan et al, 1996; Kennedy et al, 1996; Wilkinson, 1992; van Doorslaer et al, 1997). Greater income inequality has also been related to poorer self-rated health (Kennedy, Kawachi, Glass, Prothrow-Stith, 1998) as well as higher rates of disability, higher percentages of low birth weight babies, and greater prevalence of smoking and sedentary life-styles (Kaplan et al, 1996; Diez-Roux et al, 2000). The potential importance of such findings is further underscored by the fact that the US leads the industrialized countries in terms of the extent of such income inequality and the trends in the US are toward greater inequality (Atkinson, Rainwater, Smeeding, 1995).

The growing evidence linking income inequality to various health outcomes has led to a considerable debate regarding the reasons or pathways for these health effects of income equality. The two most vigorously debated positions can be characterized (grossly) as reflecting on one side a greater emphasis on differences in material conditions while the other puts greater emphasis on differences in the

nature of social relationships (see Kawachi & Kennedy, 1999 and Lynch et al, 2000 for general overviews). As proposed by Kaplan, Lynch and colleagues, income inequality has its broadest and most invidious impact on health through its association with poorer material conditions and greater disinvestment in human and social capital (e.g., lower spending on education, unemployment, transportation and other public services ; see Kaplan, Pamuk et al, 1996; Lynch, Davey-Smith, Kaplan House, 2000; Lynch, Due et al, 2000 for discussions). Others such as Wilkinson, and Kawachi and colleagues, have postulated that the primary pathways through which income inequality impact health are through its association with poorer psychosocial conditions (see Kawachi & Kennedy, 1999; Wilkinson, 1999). Here, the emphasis is on the nature of social relations (and the psychological consequences of such relations) in communities characterized by greater income inequality. In such communities, particularly among those at the bottom of the income distribution, there is postulated to be an erosion of the social fabric (reducing social capital) and more negative social comparison processes resulting from the greater power and status differentials associated with the income differentials (Wilkinson, 1999; Kawachi & Kennedy, 1999). Support for this position comes from evidence that more inegalitarian societies and communities are characterized by lower levels of interpersonal trust and civic participation, greater hostility and sense of competition (Kawachi & Kennedy, 1997, Wilkinson, 1999, 2000; Fernandez & Harris, 1992). As William Julius Wilson points out in *The Truly Disadvantaged* (1987), the worst of these socially isolating effects are frequently seen among the “underclass” (i.e., those in inner-city communities characterized by extremes of poverty) where there is a “lack of contact or of sustained interaction with the individuals or institutions that represent mainstream society (Wilson, 1987, p.60). The lower social capital in communities with greater income inequality has been hypothesized to contribute to the increased mortality rates in such communities (Kawachi, Kennedy, Lochner, Prothrow-Stith, 1997).

Summary of Community-level evidence: Research on community/area-level characteristics as risk factors for health outcomes of residents provide intriguing evidence for such “environmental” effects on health risks. One important caveat however, is that to date there has not been a great deal of research that tests for the impact of such community-level effects net of individual-level characteristics. Thus, it is not yet completely clear to what degree community-level SES or social capital impact on health net of the individual’s own SES or level of social integration. Available evidence does suggest that there are area-level effects net of individual SES (Haan et al, 1987, Daly et al, 1998; Kennedy et al, 1998; Hayward et al., 1997; Cubbin et al, 2000) though further research is warranted (see Fiscella & Franks, 1997 for negative findings and Diez-Roux, 1998 for discussion of issues). Also, the relative importance of material versus social conditions remains a topic of hot debate.

Evidence that area/community-level characteristics have significant independent effects on health has important public health implications. As the history of epidemiology and public health intervention clearly documents, some of the most efficacious interventions were those targeted not at changing individuals, but rather at changing "unhealthy" environmental conditions that generated health risks for those living in them (Goldberger, 1920; Snow, 1936; Wallack & Winkleby, 1987). Indeed, this has led many to argue that interventions targeting individuals (e.g, to get them to stop smoking, exercise more, eat a more healthful diet, or cope more effectively) hold much less promise of altering health risks at a population level because they do not address the broader environmental forces generating the risks (e.g., availability of cigarettes and fatty foods, lack of easily accessible recreational/exercise facilities, and presence of chronic stressors). A policy targeting these broader, environmental sources of "health risk" would be akin to earlier, highly successful public health efforts to reduce water-borne diseases through wholesale water treatment (Wallack & Winkleby, 1987). It is only through understanding both the pathways through which the unhealthy effects of lower SES environments get under the skin (the

"micro" processes of individual effects) as well as understanding the broader, "upstream" forces that help to generate and maintain these environments (the "macro" processes) that we will arrive at a more complete understanding of the sources of socio-economic differentials in health and well-being and, with this knowledge, hope to achieve a modicum of success in reducing them.

CONCLUSIONS

This paper provides an overview of epidemiological and demographic research linking social characteristics of both individuals and communities to differences in both morbidity and mortality risks. Large and consistent bodies of literature from each discipline provide clear evidence for the generally health promoting effects of social ties. In both fields, the bulk of the evidence relates to mortality though both fields have begun to examine other health outcomes including aspects of physical and cognitive functioning (e.g., see Crimmins & Saito, 2001; Manton & Stallard, 1997; Seeman, Lusingolo, Albert & Berkman, submitted; Seeman, Berkman et al, 1995) as well as disease outcomes (Hayward et al., 2000; Kington and Smith, 1997; see also Seeman, 1996 for review of earlier work). Despite these general similarities, there are some interesting differences in the epidemiological and demographic approaches to the question of social environment effects on health.

First, demography has tended to focus on specific types of social ties, particularly marital status (Lillard & Panis, 1996; Lillard & Waite, 1995; Rogers, 1995; Smith and Zick, 1994; Pienta et al., 2000). By contrast, over the past 30 years, epidemiologists have focused more and more on broader indices of social integration including ties with friends and other relatives as well as group memberships (see Berkman, Glass and Seeman, 2000 and Brissette, Cohen and Seeman, 2000 for discussion of measures).

Second, the differing disciplinary traditions of demography and epidemiology have lead to

differing approaches to the modeling of social effects on health. Epidemiology, with its historical interest in the identification of factors that explain the population distribution of a given health outcome, has approached the question of social effects through multivariate modeling that asks whether such social factors provide additional explanatory information, independent of other known risk factors. As a result, epidemiological analyses of social effects on health have included a broad array of other demographic variables (e.g., age, gender, ethnicity, education, income) as well as health behavior variables (smoking, diet, exercise) and, depending on the health outcome in question, other measures of health status such as the presence of other diseases known to influence the outcome in question (e.g., hypertension, diabetes and other chronic conditions for analyses of mortality). By contrast, demographic analyses of social effects on health have largely focused on models that incorporate other demographic factors but are less likely to include the same range of health status and health behavior variables seen in epidemiological models.

Third, while epidemiological analyses focus on estimating the relative risk for a given outcome associated with differences in social environment exposures (e.g., relative risk of mortality associated with social isolation), demographers have been interested in differential rates of mortality and other health problems in populations and specific population sub-groups (Elo & Preston, 1996; Deaton & Paxson, 1998). More recently demographers have emphasized social environmental effects on life expectancy (Manton et al., 1997) and healthy life expectancy (Crimmins and Saito, 2001).

Consideration of these disciplinary differences in research approaches to understanding the role of the social environment in health outcomes suggests interesting possibilities with respect to potentially valuable collaborative efforts. One avenue, exemplified in our own developing collaborative program of research, would be to integrate the more comprehensive risk factor modeling characteristic of epidemiological analyses with demographic models of population level burdens of disease and/or

mortality. Such integrative efforts, for example, might take risk estimates associated with social factors from comprehensive epidemiological models and apply these estimates in demographic models to provide estimates of population-level burdens of disease, disability or mortality under different social conditions. Similar methodologies could be used to provide more accurate estimates of the potential impact of interventions to change social characteristics (or other factors) (i.e., using risk estimates from epidemiological models as input to demographic population models). Models of this type have been developed to examine the effects of risk factors for heart disease on population levels and trends of cardiovascular disease (Bonneux et al, 1994). Such models also are the basis of the WHO Burden of Disease approach (Murray and Lopez, 1996).

The shared interests of epidemiologists and demographers in understanding population-level distributions of health provides a common base from which to launch potential collaborative efforts that take advantage of the strengths of each discipline – the more comprehensive risk factor approach of epidemiologists to modeling health risks and the more developed modeling approach to estimation of population characteristics of demographers (Manton and Stallard, 1988; Berendregt and Bonneux, 1998). Questions regarding links between social environment conditions and health may be a particularly fruitful area of future collaboration, drawing on demographers and epidemiologists shared interest in understanding how different social conditions promote variation in distributions of better versus worse health outcomes within a population.

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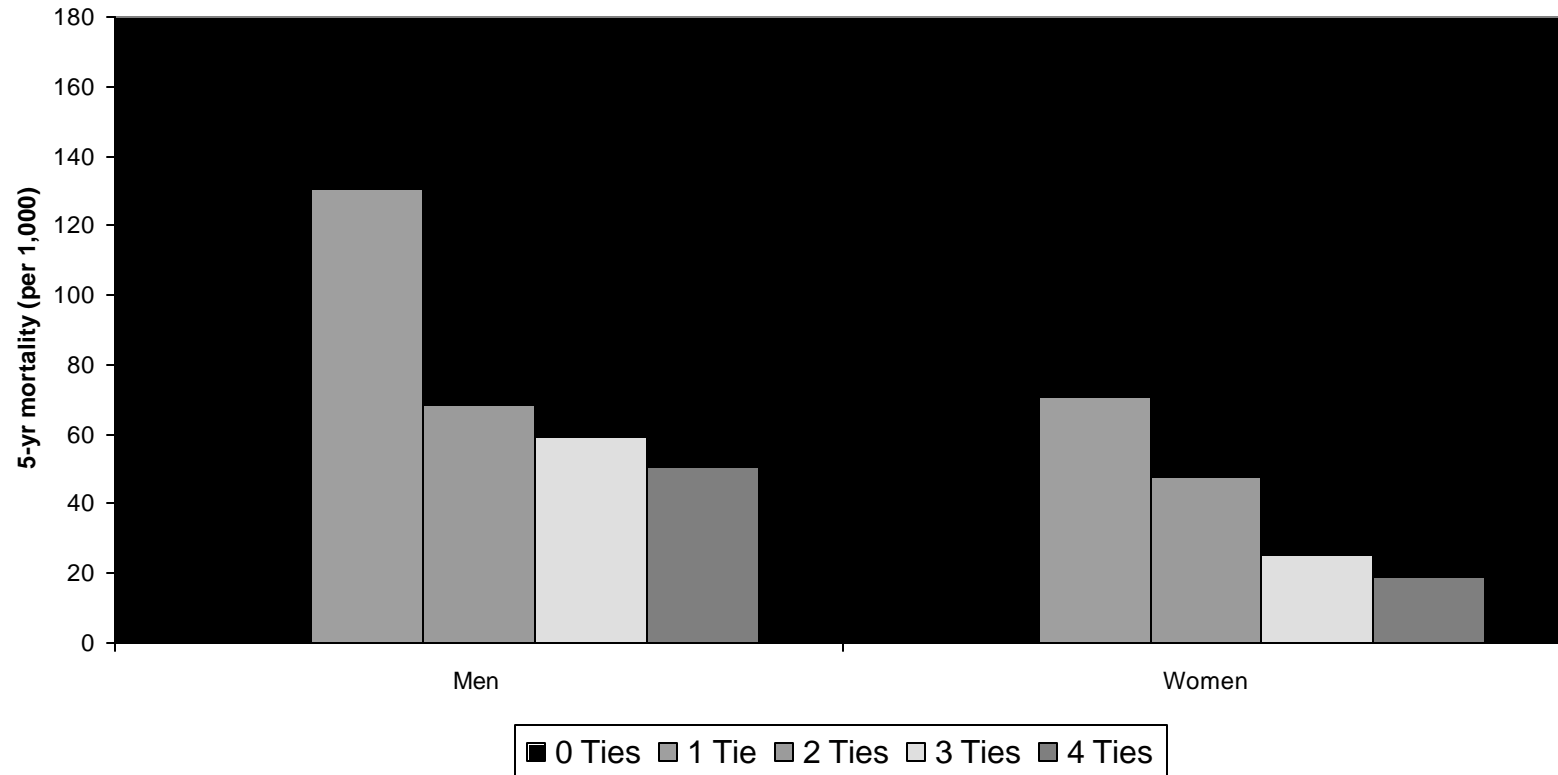
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Figure 1: Sex-specific 5-year Mortality (per 1,000) by Level of Social Integration: New Haven EPESE cohort (adapted from Seeman et al, 1993)



324.

Figure 2: Biopsychosocial Model of Health and Aging – Relationships presented are considered to operate at the level of the individual within a life-span, cumulative effects framework. Relationships are hypothesized to be influenced by broader environmental effects of the socio-cultural and physical environments within which the individual lives during their life-span.

