Psychosocial Models of the Role of Social Support in the Etiology of Physical Disease

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Although there has been a substantial effort to establish the beneficial effects of social support on health and well-being, relatively little work has focused on how social support influences physical health. This article outlines possible mechanisms through which support systems may influence the etiology of physical disease. I begin by reviewing research on the relations between social support and morbidity and between social support and mortality. I distinguish between various conceptualizations of social support used in the existing literature and provide alternative explanations of how each of these conceptualizations of the social environment could influence the etiology of physical disease. In each case, I address the psychological mediators (e.g., health relevant cognitions, affect, and health behaviors) as well as biologic links (e.g., neuroendocrine links to immune and cardiovascular function). I conclude by proposing conceptual and methodological guidelines for future research in this area, highlighting the unique contributions psychologists can make to this inherently interdisciplinary endeavor.

Key words: social support, physical disease, psychosocial models

Social support has been prospectively associated with mortality (Berkman & Syme, 1979; Blazer, 1982; House, Robbins, & Metzner, 1982; Schoenbach, 1982). Requests for reprints should be sent to Sheldon Cohen, Department of Psychology, Carnegie-Mellon University, Pittsburgh, PA 15213.
Kaplan, Fredman, & Kleinbaum, 1986) and has been implicated in the
etiology of both physical illness and psychological distress (see reviews by
Berkman, 1985; Broadhead et al., 1983; S. Cohen & Wills, 1985; Kessler &
McLeod, 1985; Leavy, 1983; Wallston, Alagna, B. M. DeVellis, & R. F.
Devellis, 1983). Although there has been a tremendous effort to establish
the beneficial effects of support on health and well-being, relatively little
work has focused on how social support influences health (cf. Heller,
Swindle, & Dusenbury, 1986). I believe that differentiation between various
conceptions of social support and specification of the processes by which
each conceptualization influences health and well-being are requisite for
further progress in understanding the role of support in the maintenance of
health and prevention of disease (see early call for such distinctions by
Kaplan, Cassel, & Gore, 1977). In service of this goal, I suggest some
distinctions in social support based on existing research, selectively review
studies of the role of social support in the etiology of physical disease, and
propose a series of models linking different conceptualizations of social
support to physical health. My discussion is limited to the etiology of
physical illness (onset and progression but not recovery) and focuses on
disease endpoints (morbidity and mortality) rather than on illness behaviors
such as symptom reporting and use of medical services.

DIFFERENTIATING SOCIAL SUPPORT

There is little agreement among the scientific community in regard to a
precise definition of social support (S. Cohen & Syme, 1985; Shumaker &
Brownell, 1984; Wilcox & Vernberg, 1985). Moreover, existing studies
apply the term to a broad range of conceptualizations of social networks
and the functions they provide. Rather than attempt an all-encompassing
definition, I propose broad categorical classifications of the concepts
commonly included under the social support rubric and define some specific
concepts that I believe may be linked with physical disease.

Several investigators have proposed typologies of social support measures
to help provide organization to the field. For example, House and Kahn
(1985; also Turner, 1983) suggested three categories of support measures:
social networks, social relationships, and social supports. Social networks
refer to measures deriving from formal network theory, including measures
of network size, density, multiplexity, reciprocity, durability, intensity,
frequency, dispersion, and homogeneity. Social relationship measures
assess the existence, quantity, and type of existing relationships. Finally,
social support measures assess resources provided by others with various
measures assessing type (e.g., emotional, informational), source, quantity,
or quality of resource.
Cohen and his colleagues (S. Cohen & Syme, 1985; S. Cohen & Wills, 1985) proposed a distinction between structural and functional support measures. Structural refers to measures describing the existence of and interconnections between social ties (e.g., marital status, number of relationships, or number of relations who know one another). Functional measures assess whether interpersonal relationships serve particular functions (e.g., provide affection, feelings of belonging, or material aid). House and Kahn's (1985) social network and social relationship categories would be classified as structural, whereas their social support category would be classified functional.

As discussed in the following review, only a small sample of possible conceptions of social support have been used with any frequency in studies of morbidity and mortality. The most common measure is a structural index of social ties that is often termed social integration (SI). A prototypic SI index includes marital status, close family and friends, participation in group activities, and church/religious affiliations. Functional measures used in the physical disease literature include network satisfaction and perceived availability of material aid or psychological support.

RESEARCH SUGGESTING A LINK BETWEEN SOCIAL SUPPORT AND THE ETIOLOGY OF PHYSICAL DISEASE

I now present a select review of the studies linking various conceptions of social support to the etiology of disease. (See an earlier review by Berkman, 1985.) My review focuses on social support conceptions that are examined across several studies. As a whole, the data are suggestive of important links among social environment, disease, and mortality but provide only clues as to the processes by which such links occurs.

Mortality Studies

The best documented effects in this literature are of the role of social integration on total mortality. In general, after controlling for traditional risk factors such as blood pressure, cigarette smoking, and serum cholesterol levels, healthy persons with higher SI scores are at lower risk for mortality than their more isolated counterparts (Berkman & Syme, 1979; House et al., 1982; Schoenbach et al., 1986). Although there is evidence that SI decreases risk only or primarily for men (House et al., 1982; Schoenbach et al., 1986), some studies have found social integration effects for both men and women (Berkman & Syme, 1979; Orth-Gomér & Johnson, 1986).
Even studies that have failed to find effects for women in analyses of total mortality reported SI-mortality associations for women in secondary analyses. Hence, House et al. (1982) reported that women with lower SI scores were at higher risk of deaths due to ischemic heart disease, and Schoenbach et al. (1986) found an SI-total mortality effect for elderly women (70 to 80 years old). Associations between SI and mortality in this literature are generally weaker for non-Whites than for Whites (Berkman, 1986).

There is also evidence that SI predicts mortality for persons who are unhealthy at the onset of the study, namely, for male survivors of acute myocardial infarctions or MIs (Ruberman, Weinblatt, Goldberg, & Chaudhary, 1984). These men were followed for 1 to 3 years after their MIs. After controlling for traditional risk, relatively isolated survivors were found to have more total deaths and more sudden cardiac deaths than their less isolated counterparts.

Three of the mortality studies assessed satisfaction with social networks. Marital satisfaction (Berkman & Syme, 1979) and satisfaction with social activities (House et al., 1982) were both unrelated to mortality. In a study of older adults, however, perceived adequacy of support was associated with decreased risk of mortality (Blazer, 1982). Unfortunately, the limited number of studies and the lack of conceptual consistency across these studies makes it difficult to draw any firm conclusions about the role of support satisfaction.

**Limits in interpretation of mortality data.** Studies predicting mortality in healthy populations do not provide direct evidence in regard to the role of social support in the etiology of disease. First, mortality can be caused by factors other than disease (e.g., accidents or natural deterioration with age). In the case of support, however, studies breaking mortality down by cause indicate an association between social integration and deaths attributed to disease (e.g., Berkman & Breslow, 1983; House et al., 1982). Second, even if mortality is attributable to disease, mortality studies of initially healthy persons do not clarify the stage of the disease process at which support acts. Hence greater mortality for those with fewer social contacts may be accounted for by increased incidence (onset) of disease, increased severity of disease, faster progression of disease, or reduced recovery from disease. Some specificity is provided by the single study reporting SI prediction of mortality for persons with serious disease (MI survivors; Ruberman et al., 1984). Because this study predicts mortality after disease onset, it suggests that support plays a role in disease progression and/or recovery. These results do not, however, eliminate the possibility of SI influences on disease onset.
Morbidity Studies

Although there are strong suggestions that various conceptions of social support are associated with disease onset, the results of the morbidity studies are less consistent and generally more difficult to interpret. The majority of this evidence examines the association of support to coronary heart disease (CHD), although there are also scattered studies of support and cancer, support and pregnancy complications, and so forth.

CHD. There is evidence from two studies of Japanese-American men that SI measures are associated with prevalence of MI, angina pectoris (AP), and CHD (Joseph, 1980; Reed, McGee, Yano, & Feinleib, 1983).1 Due to the possibility that structures and/or functions of social networks differ across subcultures, caution is urged in generalizing these results to other populations.

More culturally diverse samples were used in two recent studies of the relation between support and the prevalence of coronary artery disease (CAD). T. E. Seeman and Syme (1987) studied men and women undergoing angiography in six San Francisco Bay Area hospitals. They reported that persons with greater instrumental support available from their networks and persons who felt loved had less atherosclerosis than their unsupported counterparts. Atherosclerosis was unrelated to SI, to its structural components, or to emotional support from family or friends. A similar study, focusing on emotional support in a sample of men and women undergoing angiography at Duke University, was reported by Blumenthal et al. (1987). Like T.E. Seeman and Syme (1987), they found no main effect of emotional support on atherosclerosis. However, they did find an interaction between emotional support and the Type A behavior pattern. Greater occlusion of the coronary arteries was related to less emotional support among Type As but to more emotional support among Type Bs. Although T.E. Seeman and Syme (1987) also tested the interaction between emotional support and the Type A behavior pattern, they did not find this effect. These studies suggest that support may play a role in CAD, but they are inconsistent in regard to the nature of this role. Moreover, sample biases in angiography studies suggest cautious interpretation (S. Cohen & Matthews, 1987). Because angiography samples are limited to symptomatic patients, these studies

1Because diagnosis of AP is dependent on symptom reporting, AP incidence may be partly or wholly attributable to processes other than those involved in the development of coronary vascular disease. We include AP studies in this review partly because there is a paucity of evidence with “harder” disease criteria and partly because it is possible that the underlying processes are the same.
attempt to discriminate between those patients with symptoms who have CAD and those patients with symptoms who do not. This is quite different from distinguishing between the diseased and healthy in the general population.

One of the studies of CHD in Japanese-American men, the Honolulu Heart Study, examined SI as a predictor of the onset (incidence) of CHD, as well as the concurrent (prevalence) relations between SI and disease (Reed, McGee, & Yano, 1984; Reed et al., 1983). These incidence data only partly support a relation between SI and CHD. Total CHD rates were higher among more isolated men when an SI scale developed through factor analysis was used. This effect was accounted for primarily through the prediction of nonfatal MIs. However, this scale was not associated with the incidence of AP or of fatal MIs, and a conceptually developed SI scale failed to predict any of these outcomes.

A different conceptualization of support—perception of social support received from a supervisor—was addressed in an analysis of data from working women participating in the Framingham Heart Study (Haynes & Feinleib, 1980). Working women who perceived that their supervisors were nonsupportive had an increased risk of developing CHD over an 8-year period. Interpretation of these data is muddled by the possibility that the perception of supervisor support may be a proxy for job stress rather than an independent assessment of support level.

Three morbidity studies that have examined the possible role of social support as a buffer (moderator) of the increased risk associated with high levels of stress also vary in support measurement and results. Johnson (1986) found that degree of interaction with co-workers buffered the effects of work stress on CHD prevalence for both men and women. Medalie and Goldbourt (1976) found that men who reported that their wives loved and supported them were buffered from the effects of high anxiety on the incidence of AP. However, the Honolulu Heart Study (Reed et al., 1984) found no stress-buffering effect of an SI-like index for men in predicting CHD incidence.

**Other outcomes.** The Honolulu Heart Study data set was also analyzed for evidence of social support relations to cancer (Joffres, Reed, & Nomura, 1985). None of the eight items tested (assessing marital status, number of contacts, and frequency of activities) showed the predicted positive impact of support. In another analysis of these data, four structural support measures—closeness of parents, marital status, number of children, and number of persons in the household—were used to predict incidence of CHD, stroke, cancer, and all diseases (Reed et al., 1984). The inverse relation found between this index and CHD incidence is presented earlier in this article. None of the other outcomes was related to the total SI score.
An SI measure was also used to predict pregnancy complications in a recent prospective study of Navajo women (Boyce et al., 1986). SI was marginally related to complications, with greater complications occurring among the relatively isolated group. Again, the possible importance of culture in support effects suggests caution in generalizing this result.

Two studies have found that psychosocial assets—a single index including personal characteristics, health, and social support—acted as a stress buffer. The first reported increased pregnancy complications among persons with high stress and low assets (Nuckolls, Cassel, & Kaplan, 1972); the second found increased dosage of adrenocorticosteroids (medication used to control asthma) among high-stress, low-assets asthma sufferers (deAraujo, van Arsdel, T. H. Holmes, & Dudley, 1973). Unfortunately, it is unclear whether these effects are attributable to social support or to some other aspect of the assets measure. (See methodological and statistical critique of these studies in S. Cohen & Edwards, in press.)

Suggestion of a stress-buffering effect more clearly attributable to support is provided by a pregnancy complication study by Norbeck and Tilden (1983). Support measures in this study included perceived availability of emotional and tangible support. Women's perceptions of tangible support acted as a buffer of the stress occurring during pregnancy in the prediction of two of three categories of complications but not for total complications. However, neither the four interactions between life stress and emotional support nor any of the eight interactions between life stress during the prior year and support were consistent with the hypothesis that support operates as a stress buffer.

Summary

In sum, there are reasonable evidence for a tie between social integration and mortality and a suggestion of such an effect in the morbidity (specifically CHD) studies. Although less consistent in both conceptualization and results, the morbidity studies also suggest that perceived availability of support may operate as a stress buffer. A major weakness in the morbidity work is the lack of evidence of support effects in studies that prospectively predict the onset (incidence) of disease. (For example, only the Honolulu Heart Study provided incidence data for a link between social integration and CHD in an initially healthy sample.) As a result, this work is similar to the mortality work in that it is unclear whether support influences onset, progression, or recovery from disease. Moreover, prevalence studies are also open to an interpretation of reverse causation—illness resulting in smaller and/or less accessible social networks.

The morbidity literature is also weak from another perspective. The vast majority of existing studies focus on CHD. Because heart disease is the
major cause of death in the United States, CHD (and CAD in particular) is an important disease to study. The widely accepted model of CAD pathogenesis attributes MIs to atherosclerosis, a relatively unique (among diseases) pathogenic process. Development of atherosclerosis, however, is presumed to be facilitated by psychologically mediated neuroendocrine response, particularly the release of the catecholamines epinephrine and norepinephrine, and by standard behavioral risks such as smoking and poor diet (Glass, 1977). Because neuroendocrine and behavioral mediators are thought to be involved in the development of a wide range of diseases (e.g., infectious diseases, cancer, and stroke), evidence implicating social support in CHD suggests the likelihood that support may play a role in other diseases as well.

MODELS LINKING SOCIAL SUPPORT TO THE ONSET OF ILLNESS

The models to be presented address the hypothetical roles of social support in onset, severity, and progression of disease. Although much of what is discussed could also be applied to recovery, there is enough difference between these stages and recovery to require additional assumptions. For example, adequate models of recovery require a focus on support influences on disease symptoms, rehabilitative behaviors, availability and quality of caretaking, and so forth. The interested reader is referred to reviews and discussions of recovery in DiMatteo and Hays (1981), Wallston et al. (1983), and Wortman and Conway (1985).

I discuss three levels of analysis for modeling the possible influence of social support on the etiology of disease: generic models, stress-centered models, and psychosocial process models. This categorization of levels is in some ways arbitrary but provides a tool for illustrating the incremental development that has occurred in the conceptualization of the relation between social support and disease. The generic and stress-centered models represent early and relatively simple approaches; the psychosocial process models build on these early models by specifying the complexity of psychosocial processes in greater detail. Specificity of support conception occurs only in the context of the psychosocial process models. The models I present all focus on either a lack of support (e.g., isolation as a cause of illness) or on support as a promoter of health. These models are recursive in nature (i.e., they move in one direction, from support to health, without addressing alternative directions and feedback loops). Exclusion of these alternative paths is not intended to reflect any hypotheses about their existence. Finally, models within each category are not considered mutually exclusive. That is, it is possible (and even likely) that support influences health through more than one mechanism.
At the most elementary level, it can be posited that social support is linked to illness either through its influence on behavioral patterns that increase or decrease risk for disease (Model 1) or through effects on biological responses that influence disease (Model 2). These models are depicted in Table 1.

Examples of health behaviors that may be influenced by social support include diet, exercise, smoking, and alcohol intake. Depending on social norms and on the nature of information provided by one's social network, support may increase or decrease these behaviors. Poor health habits could put persons at risk for practically any disease. For example, smoking is a major risk factor for stroke, heart disease, and lung cancer. Biological processes presumed to be influenced directly or indirectly by social support include neuroendocrine response, immune response (either directly or as a reaction to neuroendocrine reactivity), and hemodynamic responses. Increased support is presumed to result in suppression of neuroendocrine and hemodynamic response and in increased immune competence. Diseases associated with immune functioning include infectious diseases, allergies, autoimmune diseases, and cancer (see Ader, 1981; Ader & N. Cohen, 1984; Jemmott & Locke, 1984). Neuroendocrine response may modulate immune response (e.g., Laudenslager, in press), but elevated neuroendocrine response may also directly damage artery walls and facilitate other pathogenic processes presumed to be involved in CAD (e.g., Glass, 1977; Krantz & Manuck, 1984). Finally, hemodynamic responses (e.g., sheer turbulence in blood flow) are also associated with CAD pathogenesis (e.g., Manuck, Kaplan, & Matthews, 1986).

Models 1 and 2 converge when the effects of behavior on disease are mediated by biological response (Model 3). Behaviors may be linked to the same biological processes proposed in Model 2. For example, active forms of coping result in increased neuroendocrine response that may be directly or indirectly (through the immune system) linked to disease (e.g., Manuck, Harvey, Lechleiter, & Neal, 1978; Obrist, 1981; Solomon, D. S. Holmes, & McCaul, 1980). Alternatively, behaviors may influence other biological processes. For example, poor diet may be associated with elevated serum cholesterol levels (which may promote CAD), and excessive alcohol intake may result in direct damage to the liver.

<table>
<thead>
<tr>
<th>TABLE 1</th>
<th>Generic Models Linking Social Support to Physical Disease</th>
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<tbody>
<tr>
<td>Model 1:</td>
<td>support — behavior — disease</td>
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<tr>
<td>Model 2:</td>
<td>support — biological response — disease</td>
</tr>
<tr>
<td>Model 3:</td>
<td>support — behavior — biological response — disease</td>
</tr>
</tbody>
</table>
STRESS-CENTERED MODELS

The next level of analysis addresses whether social support is only important for persons under stress or whether it is potentially beneficial irrespective of stress level. The stress-buffering model proposes that support is related to well-being only (or primarily) for persons under stress (see early discussion by Cassel, 1976). This is termed the stress-buffering model because it posits that support "buffers" (protects) persons from the potentially pathogenic influence of stressful events. The alternative model proposes that social resources have a beneficial effect irrespective of whether persons are under stress. Because the evidence for this model derives from the demonstration of a statistical main effect of support with no Stress × Support interaction, this is termed the main-effect model. Implied (but not specified) in both stress-buffering and main-effect models is that support is linked to disease endpoints through behavioral and/or biological processes discussed earlier.

In the case of the stress-buffering model, support presumably operates by short-circuiting or preventing behavioral and biological responses to stress that are inimical to health. The possible stress-buffering mechanisms of social support are depicted in Table 2. As indicated by the table, support may play a role at two different points in the causal chain linking stress to illness (cf. S. Cohen & McKay, 1984; Gore, 1981; House, 1981; also see discussion of coping and appraisal process in Lazarus & Folkman, 1984). First, support may intervene between the potentially stressful event (or expectation of that event) and a stress reaction by attenuating or preventing a stress appraisal response. That is, the perception that others can and will provide necessary resources may redefine the potential for harm posed by a situation and/or bolster one's perceived ability to cope with imposed demands and hence may prevent a particular situation from being appraised as highly stressful. Second, adequate support may intervene between the experience of stress and the onset of the pathological outcome by reducing or eliminating the affective reaction, by directly dampening physiologic processes, or by altering maladaptive behavior responses. Support may alleviate the impact of stress appraisal by providing a solution to the

| TABLE 2 |
| Stress-Centered Models |

<table>
<thead>
<tr>
<th>Stress-Buffering Model</th>
<th>Support 2</th>
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<tbody>
<tr>
<td>Support 1</td>
<td>1</td>
</tr>
<tr>
<td>Stress</td>
<td>1</td>
</tr>
<tr>
<td>Neuroendocrine Response</td>
<td>1</td>
</tr>
<tr>
<td>Negative Health Behaviors</td>
<td>1</td>
</tr>
<tr>
<td>Disease</td>
<td>1</td>
</tr>
</tbody>
</table>

Support 1 refers to benign appraisal of stressful events due to support
Support 2 refers to dampening of endocrine response due to reappraisal, adjustive counterresponse, inhibition of maladjustive response
problem, by reducing the perceived importance of the problem, by providing a distraction from the problem, by tranquilizing the neuroendocrine system so that people are less reactive to perceived stress, or by facilitating healthful behaviors such as exercise, personal hygiene, proper nutrition, and rest (cf. S. Cohen & Wills, 1985; House, 1981).

PSYCHOSOCIAL PROCESS MODELS

A final category of models includes those that provide elaborate descriptions of the nature of psychosocial mediation of the social support–health relation. These models specify the psychological and biological processes implied in the generic models and separately address main and stress-buffering effects.

The models proposed in this section rely on hypothesized links between specific conceptions of support and specific psychosocial and biological processes. It is theoretically possible to derive different models for each of the multiple conceptions of social support proposed and/or measured in the literature. However, I felt it would be more useful and less cumbersome to model only those relations that are substantially supported by existing empirical work. Such a basis is provided by combining the conclusions of this review of studies of the role of support in physical disease with earlier reviews of studies of the role of support in psychological distress (S. Cohen & Wills, 1985; Kessler & McLeod, 1985). These reviews suggest that social integration is the primary cause of main effects of social support and that perceived availability of support is the primary cause of stress-buffering effects. As a result, the models discussed here focus on how social integration and perceived availability of support are linked to disease outcomes. This choice should not be interpreted as my conclusion that these are the only relevant conceptualizations of support in this context. I am merely using existing empirical work to help determine a focus that merits detailed analysis at this time.

Main-Effect Models

Recall that the main-effect model predicts that the influence of support on health is independent of current stress level and that main effects of support have been primarily found when support is defined as social integration. Hence, our main-effect models focus on plausible links between social integration and health. Social integration has been conceptualized byThoits (1983) as having multiple identities (i.e., ties to network members with different roles). Table 3 summarizes several psychological pathways
TABLE 3
Main-Effect Models of Psychological Influence

<table>
<thead>
<tr>
<th>Information-based models (assumes advice is health promoting)</th>
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</thead>
<tbody>
<tr>
<td>Social integration  →  (advice)  →  ability to obtain preventive medical care</td>
</tr>
<tr>
<td>Social integration  →  (advice)  →  health-promoting behaviors</td>
</tr>
<tr>
<td>Social integration  →  (advice)  →  information to avoid stressors and other health risks</td>
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<tr>
<th>Identity and self-esteem models</th>
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<tbody>
<tr>
<td>Social integration (role identity)  →  sense of identity + evaluative basis for sense of mastery</td>
</tr>
<tr>
<td>→  meaning to one's life  →  less despair and anxiety  →  health-promoting behaviors</td>
</tr>
<tr>
<td>Social integration  →  self-esteem and perceived control  →  greater motivation to care for oneself  →  health-promoting behaviors</td>
</tr>
</tbody>
</table>
| Social integration  →  increased positive affect, sense of well-being, control over environment  
  →  suppressed neuroendocrine reactivity and enhanced immune function or  →  health-promoting behaviors |
| Isolation  →  increased negative affect, sense of alienation, lack of perceived control  
  →  elevated neuroendocrine response and immune suppression or  →  negative health behaviors |
| Lacking ties  →  stress  →  negative affective state  →  . . . |

<table>
<thead>
<tr>
<th>Social influence models</th>
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</table>
| Social integration  →  social controls and peer pressures, constraints to behave as others do  
  →  health-enhancing behaviors (assumes pressures toward health-promotive behaviors) |

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<tr>
<th>Tangible-resource models</th>
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</table>
| Social networks  →  aid and tangible and economic services  
  →  network taking better care of members  →  limited exposure to risk factors |

through which social integration may influence the susceptibility to and recovery from disease.

**Information-based models.** Having a wide range of network ties presumably provides multiple sources of information and hence an increased probability of having access to an appropriate information source. Information could influence health-relevant behaviors or could help one to avoid stressful or other high-risk situations. For example, network members could provide information regarding access to medical services or information regarding the benefits of behaviors that positively influence health and well-being. Appropriate information could also aid in avoiding stressful life events or in avoiding exposure to infectious or carcinogenic agents (Berkman, 1985; S. Cohen & Syme, 1985). It is noteworthy, however, that integration in a social network could also operate to the detriment of health: discouraging use of medical services, providing inadequate alternative care, or influencing people to adopt behaviors inimical to health (e.g., McKinlay, 1973; Sanders, 1982; M. Seeman, T. E. Seeman, & Sayles, 1985).

**Identity and self-esteem models.** There are several theoretical perspectives suggesting that social support increases feelings of self-esteem, of
self-identity, and of control over one's environment—feelings that result in better health. Social integration is presumed to provide a source of generalized positive affect, a sense of predictability and stability in one's life, and a recognition of self-worth due to demonstrated ability to meet normative role expectations (Cassel, 1976; Hammer, 1981; Thoits, 1983; Wills, 1985). These positive psychological states are presumed to be facilitative because they lessen psychological despair (Thoits, 1985), result in greater motivation to care for oneself (e.g., S. Cohen & Syme, 1985), or result in suppressed neuroendocrine response and enhanced immune function (Bovard, 1959; Cassel, 1976).

A popular model in this category assumes that it is isolation that causes disease rather than social integration that enhances health. This approach assumes that isolation increases negative affect and sense of alienation and decreases sense of control. Alternatively, one can merely view isolation as a stressor. In any case, as noted earlier, these negative psychological states could induce increases in neuroendocrine response, suppress immune function, and interfere with performance of health behaviors.

**Social influence models.** A socially integrated person is subject to social controls and peer pressures that influence normative health behaviors. To the extent that these pressures promote healthful behaviors (e.g., exercise, better diet, not smoking, moderating alcohol intake), social integration would promote better health. To the extent that normative behaviors within a social network promote behaviors that are deleterious to health, social integration would result in poorer health and well-being.

**Tangible-resource models.** A network may operate to prevent disease by providing aid and tangible and economic services that result in better health and better health care for network members. For example, network members could provide food, clothing, and housing that operate to prevent disease and limit exposure to risk factors. Networks may also provide (nonprofessional) health care that prevents minor illness from developing into more serious disease.

**Stress-Buffering Models**

Recall that the stress-buffering model posits that support "buffers" (protects) persons from the potentially pathogenic influence of stressful events. As suggested earlier, the following models focus on the perceived availability of social support because this conception has been found to result in stress-buffering effects. S. Cohen and McKay (1984) and S. Cohen and Wills (1985) have also proposed that stress-buffering occurs only when there is a match between the needs elicited by the stressful event and the functions of
TABLE 4
Stress-Buffering Models of Psychological Influence

Information-based models
Stress — network provision of stress-buffering information (or perception of the availability of that information) resulting in benign appraisal of stressful events or enhanced ability to cope with stressful events or their affective consequences
— prevention of stress-induced biological and behavioral responses imimical to health
— health maintenance

Identity and self-esteem models
Stress — network-assisted or -enhanced coping
— increased control and/or self-esteem
— suppression of neuroendocrine response, enhanced immune function, and/or health-enhancing behaviors
— health maintenance

Stress — network-assisted or -enhanced coping (or perceived availability of network assistance)
— increased control and/or self-esteem
— suppression of neuroendocrine response, enhanced immune function, and/or health-enhancing behaviors
— health maintenance

Social influence models
Stress — network pressures (or expected pressures) to adopt particular modes of coping with stressors resulting in benign appraisal of stressful events or (when coping strategies are appropriate) enhanced ability to cope with stressful events and the emotional effects of stress appraisal
— prevention of stress-induced biological and behavioral responses imimical to health
— health maintenance

Tangible-resource models
Stress — network provision of aid and tangible and economic services resulting in benign appraisal of stressful events or enhanced ability to cope with stressful events
— prevention of stress-induced biological and behavioral responses imimical to health
— health maintenance

support that are perceived to be available. For example, having someone who would loan you money may be useful in the face of a temporary job loss but useless in the face of the death of a friend. A matching of support with need is implied, although not specified, in many of the models to be discussed. S. Cohen and Wills also argued that certain types of support may be useful in coping with all or many stressors. Specifically, having people to talk to about problems (appraisal support) and having people who make you feel better about yourself (self-esteem support) may be generally useful because these are coping requirements elicited by most stressors. The stress-buffering models are depicted in Table 4.
Information-based models. Stress may elicit network provision of information about the nature of the potential stressful events or about ways of coping with those events (see discussion of social comparison under stress by S. Cohen & McKay, 1984; Wills, 1983). To the extent that this information reduces the evaluation of potential threat or harm in the context of existing coping resources, the event would be appraised as less threatening and/or harmful and hence the risk of illness decreased. It is also likely that in many cases the perception of available support operates without any actual support being provided. That is, knowing (or at least believing) that others will provide needed information if it becomes necessary can similarly result in a potentially stressful event or events being appraised as benign (e.g., Wethington & Kessler, 1986). In both cases, a reduction in stress appraisal would be presumed to reduce negative affect, negative health behaviors, and concomitant physiological reactivity.

Emotional responses to stressful events may also elicit network provision of information. In this case, the information would be health enhancing if it helped to control undesirable feelings (Thoits, 1985). The reduction of negative affect produced by a stress appraisal is presumed to influence health outcomes in the same ways as already discussed—that is, by reducing negative health behaviors and physiological reactivity.

Identity and self-esteem models. These models are minor twists on the information models already described. They suggest that others' willingness to help and/or the enhanced ability to cope that results from receiving help increase feelings of personal control and self-esteem. As noted earlier, such feelings may influence health through increased motivation to perform health behaviors or through suppression of neuroendocrine responses and enhanced immune function. Again, the mere perception that help is available may similarly trigger these processes.

Social influence models. Social controls and peer pressures could influence persons to cope with stressors in particular normative manners. Such influence processes would promote health to the extent that the normative coping behaviors were effective in reducing perceptions of stress, nonadjustive behavioral adaptations, and negative affective responses. Inappropriate norms, however, could lead to less effective coping and hence greater risk of stress-elicited disease. To the extent that social coping norms were internalized, or that persons expected others to pressure them to cope in a particular manner, the mere perception of availability could influence stress–disease process in the same manner as actually receiving support.

Tangible-resource models. Network contribution of aid or of tangible or economic services could reduce the probability of potentially
stressful events being appraised as threatening or harmful and hence could reduce the behavioral and affective concomitants of such an appraisal. Again, the mere perception of the availability of aid may operate without actual receipt of help. Tangible resources could also help resolve specific (tangible-related) problems after a stress appraisal is made.

Evidence for Separate Links in the Psychosocial Models

There is only a handful of studies that provide evidence for the individual links (separate paths) in the proposed psychosocial models. Unfortunately, in many cases, the conceptualizations of support do not match up well with those proposed in this article, and the results are not always directly relevant. However, a short review is presented to provide a summary of what we know about the potential links.

Social support and health behaviors. In their prospective analysis of the Alameda County data, Berkman and Syme (1979) found that health practices were positively associated with SI. Their health practice measure equally weighted smoking, drinking alcohol, eating breakfast, eating between meals, sleeping 7 or 8 hr a night, participation in regular physical activity, and weight (adjusted for height). These health practices explained only a small amount of the association between SI and mortality (Berkman & Breslow, 1983), and the SI-mortality association remained after they were partialled out. This small mediating effect is tempered by the fact that the health practice measure used in these analyses was assessed only at the beginning of the study and thus may not accurately reflect health practice changes that occurred over the course of the study.

In a prospective study of adults 65 years old and older, Blazer (1982) also found evidence for a relation between support and health practices. All three support measures—an SI-type measure, a frequency-of-interaction measure, and a perceived-availability-of-support measure—were associated with relatively beneficial changes in self-care over the course of the 30-month study. The self-care instrument assessed various instrumental and physical (or body care) tasks that permit individuals to live independently.

The possibility that social networks and social supports influence health behaviors is also suggested by recent research on quitting smoking. Successful quitting has been linked to positively supportive behaviors from one’s spouse (Abrams et al., 1985; Lichtenstein, Glasgow, & Abrams, 1986; Mermelstein, S. Cohen, Lichtenstein, Kamarck, & Baer, 1986), and the ability to maintain smoking abstinence over 30 to 60 days has been linked to perceived availability of social support, especially to perceived availability of persons to talk to about one’s problems (Mermelstein et al., 1986).
Negative influences of social networks on smoking have also been found when one's network is made up primarily of other smokers. Initiating, quitting, and staying off cigarettes have been tied to the proportion of smokers in a person's social network (see review in S. Cohen et al., in press). The more smokers, the greater the chance of smoking.

A final study examined the influence of perceived availability of support in times of illness for performance of preventive health behaviors. In a community sample, M. Seeman et al. (1985) found that the perceived availability of instrumental support is unrelated to preventive health behavior, whereas the perceived ability to consult with friends and relatives in times of illness is negatively related to health practices! This work is interesting but difficult to interpret in this context. It is possible that support in times of illness is somewhat independent of support processes that operate during more representative healthy periods. For example, family members may provide support in time of illness but not provide support for coping with the more usual life strains and hassles. The latter form of support is what is proposed earlier—see Stress-Buffering Models—as a predictor of health practices.

Social support and affect. Relatively higher levels of social support have been clearly linked with less negative affect (see reviews by S. Cohen & Wills, 1985; Kessler & McLeod, 1985). The strongest ties have been shown in both prospective and cross-sectional studies of depressed affect and social integration (e.g., Schaefer, Coyne, & Lazarus, 1981; Surtees, 1980; Williams, Ware, & Donald, 1981), confidant relationships (e.g., Dean & Ensel, 1982; Henderson, 1981), and perceptions of informational and emotional support (e.g., S. Cohen & Hoberman, 1983; Schaefer et al., 1981). Perceived availability of support has also been shown to protect persons from the psychological distress usually associated with high levels of stressful life events (e.g., S. Cohen & Hoberman, 1983; Henderson, Byrne, & Duncan-Jones, 1981; Wilcox, 1981).

There is less evidence in regard to the association between social support and positive affect. Positive morale has been found to be positively associated with tangible, emotional, and informational support (Schaefer et al., 1981), number of friends and relatives who could be counted on (Eckenrode, Kruger, & Cerkovnik, 1986), number of friends and relatives who had provided help with problems in the previous year (Eckenrode et al., 1986), and neighborhood cohesion (P. Cohen et al., 1982). Schaefer et al. (1981), however, did not find a relation between SI and positive morale.

Social support, self-esteem, and personal control. In a study of the stress-buffer model, increased self-esteem and personal control were found among stressed persons with a confidant (Pearlin, Menaghan,
Lieberman, & Mullan, 1981). This work suggests the possibility that control and self-esteem mediate stress-buffering effects. A study of the main-effect model found evidence for an association between control and health but not for control as a mediator of the relation between support and health (M. Seeman et al., 1985). Health locus of control and generalized feelings of control were both positively associated with perceived overall health status and preventive care. This study found, however, only minimal correlations between instrumental support in response to illness and general and health-specific control and between availability of someone to talk to about an illness and these control measures. Because this work assessed support in times of illness and did not address the possibility that support operated as a stress buffer—both measures assess perceived availability—it does not provide a direct test of any of the proposed models.

Social support and neuroendocrine response. Support influences on neuroendocrine response have been suggested as possible mediators of relations between support and CAD and between support and immune functioning. At this point, the only study addressing a support–neuroendocrine link provides rather mixed results. Fleming, Baum, Gisriel, and Gatchel (1982) found that perceived availability of emotional support was related to decreased norepinephrine both among persons living near the Three Mile Island nuclear plant and among those living in a control area. Emotional support, however, was not related to epinephrine levels, and the authors failed to find the stress-buffering interaction (effectiveness of support only for those living near Three Mile Island) that they (and I) expected.

Social support and immune function. Suggestive evidence of a link between support and immune function is provided by two recent studies of the relations between loneliness and immune modulation (Glaser, Kiecolt-Glaser, Speicher, & Holliday, 1985; Kiecolt-Glaser et al., 1984). If we assume that loneliness is roughly equivalent to the social isolation pole of SI, then these data can be viewed as suggestive for our purpose (see Rook, 1985, for limitations of this assumption). Specifically, both medical students and psychiatric patients with high scores on a loneliness scale had higher levels of latent viral activity (as indicated by elevated antibody titers to herpes viruses) than those who described themselves as less lonely. An elevation of latent viral activity is considered an indication of a relatively suppressed cellular immune response.

WHERE DO WE GO FROM HERE?

A Role for Psychologists?

Sophisticated studies of the relations between social supports and physical health are by nature interdisciplinary. To address models proposing social
influences on infection, atherosclerosis, and other pathogenic processes, it is necessary to collaborate with medical scientists with expertise in the specific disease under consideration and in state-of-the-art techniques for assessing pathogenic process and disease. However, the role of psychologists in this work is as important as that of medical scientists. Psychologists are uniquely qualified to provide insight into the mechanisms through which social environments influence cognition, affect, behavior, and physiologic response (cf. Taylor, 1978). In short, psychology has the theory, data, and perspective necessary to propose plausible models that suggest when and why social networks and/or perceptions of support influence health. Psychologists also have the technical skills necessary to develop psychometrically sound measures of social support and of the psychological states and behaviors proposed as important mediators of support-disease links.

Research Priorities

The thrust of this article is an attempt to influence the form of the questions researchers ask when designing studies of the relations between social support and health. The general message is to enter this work with clear hypotheses regarding both the psychosocial and pathogenic processes by which specific conceptualizations of support influence specific outcomes. Choices of social support concepts and of disease outcomes should be driven by theory or theories specifying the psychological and biological pathways by which such outcomes could occur.

This orientation calls for a rather different approach to research design and analysis than has been traditionally applied in this area. First, much of the existing work involves post hoc analyses of data sets that were not designed to test hypotheses regarding social support. In most cases, the choice of support measures has been dictated by the questions about the social environment that happened to be available in an existing data set. Instead, social support measures used in future studies should be chosen because they represent appropriate concepts for predicting the etiology of the diseases under consideration. Second, existing work often measures traditional risk factors for the disease under consideration (e.g., smoking, diet, serum cholesterol) and controls for these factors in the data analyses. In other words, the question being asked up to now is whether social support influences disease outcomes independent of traditional risk. Several models proposed in this article, however, suggest that support may have its influence on morbidity and mortality through its influence on behavioral risk factors. Hence in these cases, these factors should not be treated as controls but rather as mediators (Berkman, 1985). It may be that previous studies controlling for risk underestimated the influence of support on health by subtracting effects on risk that were actually attributable to
support. Finally, existing work seldom looks at the role of psychological states, behavior, and concomitant physiological states in mediating the influence of support on health. Instead, hypothesized psychological (e.g., affect, self-esteem, personal control), behavioral (e.g., smoking, diet, health service utilization), and biological (e.g., neuroendocrine and immune function) mediators should be assessed whenever possible.

What social support concepts do we study? Existing evidence suggests that social integration and perceived availability of support are predictive of disease and mortality. Hence modeling (as in this article) and testing the role of these concepts in disease development and progression are relatively good strategies at this time. However, these models reflect only two of many possible conceptualizations of the social environment, and it would be a mistake to limit our theory and research to these concepts merely because they have been used successfully in the past. Other social support concepts that may play a role in disease etiology include the various structural components derived from social network theory (e.g., density, reciprocity, dispersion, and the qualitative aspects of perceived or received functional support; House & Kahn, 1985).

What diseases do we study? Most models linking psychosocial factors to disease, including the majority of those proposed in this article, assume that the primary link between the psychosocial and biological systems is provided either by health practices or by affect-mediated neuroendocrine response. Because health practices and neuroendocrine response may have implications for a wide range of diseases, insufficient social support (and stress) are often thought to be potential precursors of a variety of disorders (cf. Cassel, 1976; Selye, 1956). For example, negative affective responses could influence cancer and infectious diseases through neuroendocrine-elicited immunosuppression and CHD through neuroendocrine-elicited facilitation of coronary artery occlusion. Similarly, cigarette smoking provides a good example of a health practice that has been implicated in the multiple diseases including CHD, stroke, lung cancer, and upper respiratory infection.

It is clear, however, that some diseases are more likely to be influenced by social support than others and that each disease (or possibly, each category of disease) should be considered separately. Two questions should be addressed in deciding whether a particular disease is susceptible to support influence. First, is it plausible that behavioral and biological processes presumed to be influenced by social support are important precursors of the disease? For example, although modulations of neuroendocrine response and/or changes in health practices are assumed to play a role in the pathogenesis of some diseases (e.g., CAD), their influence on others (e.g.,
Hodgkin's disease) is less clear. Second, is the conception of support under study temporally stable enough to provide an exposure that is long enough to influence the pathogenesis of the disease under consideration (S. Cohen & Matthews, 1987)? The answer to this question depends on the relation between the temporal stability of a particular conception of support and the developmental course of the disease under study. Plausible models assume either (a) that the support conception under examination is relatively stable over the period of disease development or (b) that a short-term exposure to a particular level of social support is sufficient to influence the disease process.

An example of the importance of matching the stability of the support measure and temporal characteristics of disease pathogenesis is provided by the relation between social integration and CAD. Social integration is a temporally stable conceptualization of social support, and CAD has a very long and slow course of development (clinical disease generally requiring many years). Hence there is a reasonable match here between stability of the support measure and the temporal characteristics of disease pathogenesis. That is, exposure to relatively lower levels of social integration lasts over the period of disease development. Conceptualizations of social support with much shorter temporal stabilities such as support satisfaction or perceived availability (see S. Cohen & Matthews, 1987) would not be plausible predictors of CAD pathogenesis, although they may be plausible predictors of disease with short-term developments—diseases such as colds and influenza.

It is possible, however, to propose plausible models of slowly developing diseases that focus on support measures with shorter stabilities. For example, we have been discussing support processes that may influence the development of atherosclerosis. As noted earlier, this disease develops over many years. However, clinically significant progressions in CAD can occur over relatively short periods. Consider, for example, modeling MI incidence. Assume that persons with undetected CAD are more likely to manifest this disease in an MI if they experience stress (e.g., Glass, 1977). A severe stressor might trigger the onset of the detectable disease. In this case, perceived available support, a support concept with less temporal stability, may be important if it is stable over the course of stressor exposure and operates to buffer persons from stress at the trigger point. Hence perceived availability of support might protect such a person from stress-triggered disease progression.

Finally, there are plausible support–disease models in which short-term exposures to a particular support level triggers the onset of the development of a disease with a long (slow) developmental course. Take, for example, the possibility that sudden and severe breaches in social support—such as those produced by divorce or bereavement—may be associated with immu-
nologic changes that set a given disease in motion. Hence, a short term without support may hit with sufficient impact to produce a dramatic but short-lived compromise in immune functioning—triggering the onset of a disease like cancer, which may then be self-perpetuating.

In sum, the question of whether a particular disease is susceptible to support influence depends on (a) whether the conceptualization of support under consideration affects processes that influence disease pathogenesis, (b) the temporal stability of the support concept, and (c) the nature and time course of the pathogenesis of the disease. As noted earlier, existing work on morbidity is almost totally limited to CAD. It is clear, however, that stable conceptions of support like social integration are plausibly related to the onset and progression of diseases with both long-term and short-term pathogeneses and that less stable support conceptions like perceived availability are plausibly related both to diseases with short-term developments and to diseases for which stressors may act as triggers of progression or onset. Future work on diseases with plausible links to both stable and unstable conceptualizations of support is a high priority at this time.

High-prevalence diseases such as infectious diseases (ranging from common upper respiratory infections to AIDS) and cancer provide opportunities in this area that have the potential for both theoretical and practical contributions.

**What mediators do we study?** The models proposed in this article are neither mutually exclusive nor independent. For example, it is likely that the positive influence of social integration on health is mediated both through social influences improving health practices and through psychological states, such as control and self-esteem, triggering biological processes. Hence the simultaneous measurement of behavioral and psychological links seems optimal. There are no human studies tying self-esteem and personal control to biologically verified disease, and hence these often-discussed mediators provide an untapped area for exploration. Finally, because the assumption that support triggers neuroendocrine response is critical to several possible mediating paths, further investigation of this link is essential.

**Other variables to consider.** Access to, and the meaning of, social support may vary across other social and psychological dimensions. Sources and forms of support may differ among cultures or social classes and between the sexes (Berkman, 1986; S. Cohen & Syme, 1985; Schoenbach et al., 1986). For example, information provided by a network may be less accurate and hence less helpful to persons with lower educational levels. Support may also be useful to persons with some social skills and personality factors but not with others. One possible moderator is personal
control. That is, persons with control may be more able to make use of their networks than those who feel they cannot control their outcomes (Lefcourt, Martin, & Saleh, 1984; Sandler & Lakey, 1982; M. Seeman et al., 1985). Future empirical work and theoretical modeling should be sensitive to these differences. Initially, it will be useful to stratify samples by subgroups and to examine the relative efficacy of different types of support for different populations. As the data base increases, it may be necessary to develop unique models for unique populations.

The need for biological verification of disease. Although the influence of social support on sensitivity to symptoms, reporting of symptoms, and utilization of health services are interesting issues, it is imperative to understand that these outcomes do not necessarily involve the same mechanisms that influence disease processes. Certainly, the development of models of symptom reporting and health utilization is a worthwhile pursuit and one especially appropriate for psychologists (cf. Leventhal, Meyer, & Nerenz, 1980; Pennebaker, 1982). If our goal is to understand disease, however, it is necessary to focus on "hard," biologically verifiable (as opposed to self-reported symptoms) disease endpoints.

Studies of biologically documented atherosclerosis, MI, and cancer meet the "hard" outcome criterion, although these diseases involve long-term development and hence are expensive and difficult to study prospectively. One strategy for pursuing such questions would be to "piggyback" sophisticated social support measures and measures of psychological and physiological mediators onto ongoing studies designed to examine other (often biological) issues in disease risk. Alternatively, those interested in infectious diseases (e.g., upper respiratory infections) have a more accessible opportunity to develop and test models of the social etiology of disease as measured by "hard" outcomes. This approach allows for a prospective design and for monitoring of changes in antibodies, virus shedding, and other disease-relevant biological changes over the course of diseases with very short developmental periods (days).

Development of psychometrically valid measures. Psychologists' methodological skills are critically needed to improve the quality of work on support and disease. The primary area for contribution is measurement. Psychometric examination of empirically derived scales used in earlier studies would help clarify the reliability of the early measures and aid in appropriate interpretation of study results. Moreover, it is imperative that future studies employ psychometrically reliable and valid measures of support. Several scales are available at this time (see, e.g., review by House & Kahn, 1985; B. R. Sarason, Shearin, Pierce, & I. G. Sarason, 1987), and a priori scale construction is a worthy pursuit for those who are unhappy
with available instruments or for those who need instruments to assess different support concepts.

The need for prospective data on risk onset and disease onset. Although there is a consistent data base in regard to the relation between SI and mortality, there is little work on support and morbidity, and existing studies tend to suffer from inadequate conception and measurement. Moreover, even though we know that SI influences disease-related mortality, we know little about when in the course of the disease support operates. As a consequence, future prospective studies of the incidence of morbidity with adequate controls for disease state at study onset are imperative. Such designs help eliminate causal alternatives and aid in differentiating between the influence of social support on onset, course, and recovery from disease.

Possibly more important is the total lack of evidence in regard to the role of social support in the onset of biologic and behavioral disease risk. Prospective studies of the relation of support to smoking, alcohol abuse, diet change, physical inactivity, and biologic risk such as (in the case of CHD) hypertension, elevated serum cholesterol, and triglycerides would provide some clear evidence for the plausibility of support-disease models presumed to be mediated by health practices and biologic risk.

Summary

The early years of research on the social support concept have resulted in an almost uniform enthusiasm for its importance in the development of disease and maintenance of health (S. Cohen & Syme, 1985). It is only now, after the concept has generated some credibility, that its complexities have become apparent. Shotgun approaches and reanalysis of data sets that were not designed to test links between social support and health will no longer add significantly to our understanding of the construct. Studies need to be designed to test hypotheses about the psychological and biological processes that link social environments and their psychologic representations to health. Psychologists are uniquely qualified to contribute to this effort. They can provide theory and data on the influence of social environments on the behaviors and psychological states that are presumed to influence health, and they have the skills necessary to develop the psychometrically sound measures needed to test proposed links between support and physical well-being.

This article outlines a series of models linking two conceptions of support—social integration and perceived availability of support—to disease onset and progression. Other representations of these specific processes are possible. Moreover, I have addressed only two of the multiple conceptions of social networks and social supports. It is likely that other
characteristics of social networks (e.g., density, range, and multiplicity) and of social supports (e.g., actual support received, adequacy, and source) play an important role in disease etiology. I hope that this work will stimulate others to propose alternative models and that future work will focus on specifying the psychological and biological processes that link the social environment to disease onset, severity, progression, and recovery.

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