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Reviews and Commentary

THE EPIDEMIOLOGIC EVIDENCE FOR A RELATIONSHIP BETWEEN SOCIAL SUPPORT AND HEALTH

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The role of the social environment in host resistance had been studied for some time when in the mid 1970s, three major papers were published which reviewed the mounting evidence that "social support" had both a direct positive effect on health status and served as a buffer or modifier of the effects of psychosocial and physical stress on the mental and physical health of the individual (1–3). The study designs reviewed included the whole gamut of scientific inquiry—animal experiments and ecologic, cross-sectional, case-control, cohort and randomized controlled trial studies. The ameliorative and protective effects of social support were reported for numerous disease outcomes ranging from self-reported symptoms and illness behavior through a variety of chronic and infectious diseases, pregnancy outcome, psychiatric morbidity, childhood development, suicide, accidents, recovery from illness, and death from a number of chronic diseases. The measures of social support were as varied as the number of investigators and included the presence of litter mates in animal studies, social disorganization, rapid social change, acculturation, morale, presence of a confidant, presence of a family member, children having been wanted, family competence, and emotionally supportive intervention by clinicians. Likewise, in studies of the buffering effect of social support, the measures of stress ranged from indirect proxy measures and presumed stressful situations to quantitative measures (e.g., the Schedule of Recent Experience (4)).

The attention to this area created by these reviews resulted in an outpouring of publications of social support research since the mid 1970s. The quantity and diversity of this new research necessitates and permits a careful review and re-evaluation of the relationships between

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social support and health suggested earlier.

Cross-sectional studies abound and show a direct association between a variety of social support measures and depression (5–7), anxiety (5, 6), other psychologic or psychiatric symptoms (8–13), physical or somatic symptoms (5, 7, 8, 14), self-reported use of health services (14) and blood pressure (15). There are cross-sectional studies, however, which fail to find these direct effects for both psychologic and physical variables (16). And there is some variation in this association by sex; two studies documented a negative association between social support and number of symptoms for women, but not for men (5, 8). The statistical interaction of social support and stressful life events (or other stressors)—and thus a hypothesized effect modification—has been confirmed by a number of these cross-sectional studies that had also found a direct effect of social support independent of interaction terms (5, 6, 8, 9, 14), and by one study looking only at the buffering hypothesis (17). One study supported the buffering hypothesis but did not find significant direct effects (16). And one study found no interactive effect, although a significant direct effect was present (12).

Several retrospective and case-control studies have also shown a direct effect of social supports in various health/disease states: psychologic adjustment to divorce (18), physical and emotional recovery after an automobile accident (19), and nonpsychotic psychiatric disorders (20, 21). One cohort study which evaluated the presence of social supports retrospectively detected no direct effect, but a significant interactive effect (22). But, despite their contribution to our understanding of the relationship of the social environment to health and disease, these, like the cross-sectional studies, are flawed by their inability to address the direction of causality—especially with respect to the association between low social support and mental illness or psychologic symptoms.

Prospective cohort studies have confirmed the direct beneficial effects of various forms of social support on global mental health (23), incidence of depressive symptoms (24, 25), recovery from a unipolar depressive episode (26), psychologic distress (27, 28), psychologic "strain" (29), physical symptoms (24) and all-causes mortality (30–32). Two of these studies show that the effect of social support is greatest in the presence of social stressors (effect modification): one for serum cholesterol levels (24) and one for psychologic symptoms (28). One study (23), however, did not detect effect modification despite the presence of direct effects. Another (25) detected no effect on physical symptoms, despite the positive association with psychologic status.

Other authors (33, 34) have reviewed numerous clinical and community-based intervention programs which have unfortunately not been properly evaluated with pre- and post-intervention measures of perceived social support and mental or physical health outcomes. Three adequately designed clinical intervention programs do bear mentioning. The first, by Pless and Satterwhite (35) involved a randomized controlled clinical trial of lay family counselors and families of children with chronic diseases. After one year of follow-up, the percentage of children with improved psychologic status was greater in the treatment group vs. the control (60 vs. 41 per cent, respectively). Gottlieb (36) used a randomized control design to evaluate the effect of physician-lead support groups for parents experiencing the stress of having their first child. He was able to document an increase in the amount of support received in the patients' own support networks, outside the groups, but *could not* demonstrate a reduction in subjective ratings of stress or an im-

provement in sense of well-being. The third study, by Sosa et al. (37) used supportive lay companions for women during labor, also in a randomized controlled design. Controls had higher complication rates (cesarean section, meconium staining, etc.), but even when comparing only uncomplicated deliveries, the experimental group had markedly shorter labors than controls (8.8 vs. 19.3 hours), were more often awake after delivery and stroked, smiled at and talked to their babies more than did control mothers.

We have reviewed a large number of reported associations, but we should not hastily conclude that a causal relationship exists between low social support and unfavorable health outcomes based on the sheer weight of numbers. Rather, we should take the advice of Austin Bradford Hill (38) and closely examine the characteristics of this association before making interpretations of causality. Hill proposed eight criteria to be considered when inferring causality: temporality, strength, consistency, biologic gradient, biologic plausibility, coherence, experimental/intervention and specificity of outcome. We discuss current social support research with respect to these and other criteria and summarize this discussion in table 1.

Temporality. In traditional prospective cohort studies of specific chronic diseases, diseased persons are excluded from the original sample and the nondiseased cohort is followed and evaluated for disease incidence over time. However, in studies evaluating the incidence of changes in global measures such as mental health or general physical health status, dichotomization of subjects into diseased vs. nondiseased groups would be arbitrary. But since the mental health or physical status of a person at one point in time can be presumed to affect his or her mental health and physical status (including mortality) into the future, it is necessary to take baseline status into account. This *can* be accomplished by stratified analysis or by multivariable methods which include baseline status as an independent variable. It is only after such analyses that the causal direction between low social support and health outcome can be inferred; unfortunately, the bulk of the studies we have reviewed to this point have not met this criterion.

There are five recent exceptions. Williams et al. (23) demonstrated a significant relationship between baseline social support and mental health at one year of follow-up in a multiple regression model controlling for baseline mental health as an independent variable (standardized regression coefficient of 0.12). The magnitude of the association was somewhat decreased from the model lacking baseline status as an independent variable (standardized coefficient of 0.19), but it was still significant ($p < 0.01$). Holahan and Moos (27) showed similar minor reductions in measure of effect of work-related support on men and family-related support on women. However, a striking effect occurred for work-related support and women; in this case, controlling for baseline psychologic distress decreased the presumed (uncontrolled) measure of effect from a significant value ($r = -0.225, p = 0.02$) to an insignificant level ($r = 0.005$, nonsignificant). Turner (28), using similar statistical methods, found a significant effect of social support on the psychologic well-being of new mothers after controlling for baseline status. Berkman and Syme (30) used stratification analysis to control carefully for age, baseline health status, health practices, and utilization of preventive health services in their study of low social support and mortality and found the relationship independent of these baseline risk factors. And, most recently, Blazer (32) demonstrated a strong association between perceived social support and 30-month mor-

TABLE 1

Characteristics of the association between social support and health

Temporality:
Poor social support precedes adverse psychologic outcomes and mortality.
Strength:
Social support explains from 1.0–7.0% of the variance in psychologic outcomes. The relative risk of mortality given poor social support is in the range of 1.5–3.5.
Consistency:
There is a similar direction and magnitude of effect across all major study designs and across a wide variety of age, sex, race, ethnic, and health status groups. But the effect is greater for women than for men in most studies.
Biologic gradient:
There is an apparent increase in numbers of physical and psychologic symptoms and mortality with incremental increases in numbers or frequency of social contacts. The relationship is less clear for perceived qualitative measures of social support.
Biologic plausibility:
Experimental evidence (animal and human) suggests neuroendocrine mechanisms, possibly mediated by β-endorphin, which might explain both the proposed direct and stress-modifying effects of social support.
Coherence:
Social support theory is supported by studies in ethology, and existing psychosocial theory and biologic evidence can be used to explain the effect of social support at six or more points in the proposed causal chain between exposure and disease.
Experimental/intervention:
Social support intervention has improved psychologic outcome of chronically ill children and pregnancy outcome of women in labor. Otherwise there is a dearth of adequately evaluated intervention.
Specificity of outcome:
The wide number of physical and psychologic outcomes associated with variations in social support are consistent with cognitive and neuroendocrine mechanisms of effect.
Measurement of exposure:
A wide range of definitions of social support have been used. Factor analytic studies suggest constructual differences between measures of quality vs. quantity. Inappropriate summary indexes and contamination of indexes by nonsupport variables plague the literature.
Determinants of social support:
A large number of environmental and individual characteristics interact to produce a person's social support system at any one point in time. Health outcomes affect each of these determinants. (*Social support is not merely an environmental exposure.*)
Dynamics of social support:
The nature of all these determinants changes with sequential role changes and other life events as an individual proceeds through the life cycle. (*Social support is not independent of life events*).

tality in an elderly group of community residents, controlling for baseline physical health, activities of daily living, depressive symptoms, age and six other potential confounders.

Strength. The strength of the significant associations of low social support with poor health status reported in the numerous cross-sectional and retrospective studies cited above ranges from r = 0.14 to r = 0.55 for psychiatric symptoms, and, for those using tabular data, prevalence ratios range from 1.44 for depressive symptoms (39) to 2.54 for self-perceived health and symptom status (14). Unfortunately, we can predict such associations in advance if we presume that mental or physical illness has a direct effect on access to social supports or perception of socially supportive behavior by others. Moving on to prospective cohort studies we find multiple partial correlation coefficients ranging from 0.17 to 0.33 for psychologic variables, and relative risks of 2.6 for mortality (31) and 2.72 for complications of pregnancy in a high-stress group of mothers (40). But, as was demonstrated in the previous section, the

apparent strength of these associations will be reduced—even in prospective studies controlling for other risk factors—when baseline health status is added to the model as an independent variable. In those studies which meet this latter criterion the multiple partial correlation coefficients range in magnitude from 0.11 to 0.27, explaining from 1–7 per cent of the variance in mental health outcome. Similarly, Berkman and Syme (30) demonstrated age-adjusted relative risks of mortality for persons with low social network indexes which ranged from 1.5 in persons with no health problems to 3.5 in disabled individuals. And Blazer (32), as discussed above, described an estimated relative risk of 3.40 (95 per cent confidence limits 1.88–6.16) for 30-month mortality of elderly individuals with poor perceived social support, adjusting for 10 important potential confounders recorded at the baseline of entry into his study.

Consistency. By this, Hill (38) suggested that causal inferences could be strengthened by the knowledge that the findings have been observed repeatedly "by different persons, in different places, circumstances and times." The studies reviewed have found the association of social support and mental or physical health in a number of groups of people in different situations: community surveys (5, 11, 22, 23, 27, 30), elderly community residents (7, 14, 16, 32, 39), middle-aged and elderly men (15, 31, 41), women at all stages of the life cycle (6, 13, 28, 37, 42), young men (29), parents (10), employed men (17, 43), unemployed men (24), college students (9), Chinese-Americans (12), Swedish men (31), psychiatric patients (20, 21, 26) and general practice patients (8). The majority of findings have been of a similar magnitude (and all have the same direction of effect) using measures of social support as varied as the investigators (see "Specificity of outcome" below) and under a variety of stressful conditions and health outcomes. Inconsistencies are found, as we have discussed above, in whether the effect of social support is direct or indirect (effect modification) or both (see "Biologic plausibility" below), and in the effect by sex. However, where sex differences have been examined, the results *are* consistent; the effect of social support on health outcome is always greater for females (5, 8, 27, 30).

Biologic gradient. A simple cause-effect relationship is more tenable if we find that the measure of effect increases with increasing exposure levels. If intermediate values of exposure had higher measures of effect than the highest values of exposure, for example, a more complex relationship may exist.

A number of studies have demonstrated a biologic gradient. Ware and Donald (11) noted a gradual and steady increase in positive well-being with increasing numbers of close friends and relatives in a community survey. Stephens et al. (7) showed a similar result for five different morale scales and three social activity scales; morale and activity scores increased consistently across five levels of an informal social support index. In a prospective cohort study, Medalie et al. (41) showed consistently increasing age-adjusted angina pectoris incidence rates with increasing severity scores for five levels of family problems and four levels of co-worker problems and "superior" problems. These problem scores included both low social support and conflict in interpersonal relationships. Tibblin (44), again in a prospective cohort study, showed consistent increases in mortality rates with decreases in scores on his social network scale. Berkman and Syme (30) showed incremental increases in mortality rates with decreasing numbers of social connections for men and women across all age categories.

Blazer (32) was unable to confirm a consistent gradient of increasing mortality rates with progressive decreases in perceived social support or frequency of

social interaction. He did note, however, that there was a "consistent but nonsignificant pattern of increased mortality" with gradational decreases in "roles and attachments," a summary measure of marital status and the number of living siblings and children. Although not significant in his sample of 331 elderly persons, this suggests a dose-response relationship between the number of possible familial sources of support and mortality.

Two studies were not able to demonstrate such a gradient. When controlling for the presence or absence of a confidant, Miller and Ingham (8) found a relationship between the number of acquaintances a person had and a number of psychologic and physical symptom scores. But, while persons with "few acquaintances" had the highest symptom scores and persons with "some acquaintances" had the lowest scores, persons with "many acquaintances" had intermediate scores—an inconsistency in the proposed gradient. This may reflect the differences between quantity and quality of social support (see "Specificity of outcome" below) and the suggestion that the number of social contacts does not necessarily reflect the number of socially supportive relationships (22), that social relationships are both sources of stress and support (45) and that an increase in the number of social acquaintances may produce environmental demands for reciprocal support which exceed the person's abilities to meet them (46).

Brown et al. (42) tried to demonstrate a change in risk of developing psychiatric disorder across four levels of support: *a* = women with a spouse or boyfriend as confidant, *b* = women with a confidant other than spouse or boyfriend and seen at least once per week, *c* = women with a confidant seen less than weekly, and *d* = women with no confidant. They were able to detect a significant effect only for the highest level of support (group *a*). That is, a gradient was not demonstrable. While this may reflect a threshold effect of confidant support on the development of psychiatric disorder, it does not address the relative importance of various degrees of other types of support.

Biologic plausibility. The biologic plausibility and mechanisms of effect of stress on physical health have been well established (47–53). These include the "fight or flight" response described by Cannon—a sudden discharge of the sympathetic nervous system resulting in elevated blood pressure, increased cardiac output, elevated serum catecholamines and elevated serum free fatty acids (49)—and "the general adaptation syndrome" of Selye (47)—which includes a delayed and prolonged adrenalcortical-regulated protein catabolic effect. Evidence is accumulating to explain a mechanism for the effects of social support.

Bovard (50, 51) has reviewed the literature supporting the existence of a brain system located in the anterior and lateral hypothalamus which antagonizes the neuroendocrine response to environmental stress by direct inhibition of the posterior hypothalamic zone (responsible for sympathetic activity and adrenocorticotrophic hormone release) and by its own neuroendocrine effects—parasympathetic activity resulting in lowered blood pressure and growth hormone release, which has its own protein anabolic effect. Autostimulation of these areas with implanted electrodes is reinforcing for experimental animals, and electrical stimulation of analogous regions of the unanesthetized human brain have produced feelings of pleasure, euphoria, relaxation, joy and satisfaction. Psychotic episodes in schizophrenic patients have been terminated by such stimulation, and it has been used to control the intractable pain of terminal cancer and rheumatoid arthritis. Bovard hypothesizes that any physical or social stimulus which is reinforcing—such as sexual stimulation, affection, and social approval—would act through this system to mediate the response to stressful stimuli.

Benson and others (54–56) have described an apparent counterpart to Bo-

vard's "positive brain system activity" which Benson calls the "relaxation response." This is analogous to the relationship between the "negative brain system activity" of the posterior hypothalamus and Cannon's "fight or flight" response. The trance-like state of the "relaxation response" can be self-induced by autohypnosis and allows individual control over this portion of the autonomic nervous system. Benson and colleagues (54–56) have shown its effectiveness in reversing the hypertensive effects of stress.

Other recent work has discovered possible biochemical intermediates between sensory perception and the neuroendocrine responses to stressful and socially supportive stimuli. These are the neuropeptides, especially the opioid β-endorphin (57–59). Although the exact relationship between β-endorphin and the positive hypothalamic activity reviewed by Bovard (50, 51) is not known, its effects are quite similar: analgesia sufficient to treat severe pain, growth hormone release, and euphoria. Another effect of β-endorphin is reminiscent of the "relaxation response"; that is, a state of narcolepsy. Indeed, Benson (54) recommends that the "relaxation response" be induced while seated upright to prevent falling asleep and losing its full benefit.

Plasma β-endorphin levels have been shown to increase with extreme exercise and to reach chronic high levels in trained athletes (60–63). It has been suggested that this may be the mechanism whereby running produces a state of euphoria and addiction (opioid effects) and has a positive effect on certain emotional disorders (60)—effects similar to those attributed to "positive brain system activity." But β-endorphins can only be implicated in these positive effects of running when it can be shown that brain tissue levels of β-endorphin are increased by exercise (63, 64).

There has apparently been no research to investigate the possible role of social support in affecting brain or plasma β-endorphin concentrations. But Bovard (50, 51) described a brain system which is antagonistic to the "fight or flight" response, which corresponds anatomically to sites for which repetitive electrical autostimulation in experimental settings is reinforcing and to sites which reflect increased activity in the presence of reinforcing stimuli (such as food, sex, and water), and which produces major effects also associated with β-endorphin (analgesia, growth hormone release, euphoria, and addictive behavior) (50, 51, 58, 59). Hence, it is likely that β-endorphin is a biochemical mediator between reinforcing external stimuli and this response. Reinforcing stimuli such as affection, social approval, and other forms of social support may also produce their euphoria, direct positive health effects and stress-buffering effects through β-endorphin and/or other neuropeptides.

Coherence. Hill (38) insisted that the cause - and - effect interpretation should not seriously conflict with the existing knowledge of biology and the natural history of disease. Indeed, social support theory is congruous with existing knowledge and the mechanism of its beneficial effect can be discussed coherently at all levels of biologic organization.

Alcock (65) has reviewed how Darwinian forces of evolution have resulted in the innate human needs for social contact including copulation and pair bonding, parental care, and the sense of belonging to a group (group bonding). These needs are so important to species survival that, at a level much more primitive than symbiotic or altruistic cooperation, their absence produces distress and dysfunction (3, 66). But low social support may not be a stressor in the sense of a stimulus which produces a "fight or flight" response, but more likely it may be that "positive brain system activity" (50, 51) produced by social support is a necessary condition for the growth, development, and homeostasis of the organism by way of its contribution

to parasympathetic activity and growth hormone secretion. However, the threat of loss of essential levels of social support may be such a stressor.

A further understanding of the role of social support in health and disease can be gained by a discussion of the possible mechanisms of social support effects beyond the simple concept of a basic biologic need for social interaction and in the context of the person-environment fit theory (46). We first must consider that our responses to the environment (be they physiologic, affective, cognitive or motoric) are a function of both the person and the environment (67). The goodness of fit between person and environment depends upon a match between the *demands* of the environment and the person's *abilities* to meet them on the one hand, and the *needs* of the individual and the *resources* from the environment available to satisfy these needs on the other. Although each of these components has subjective and objective values, it is our subjective person-environment fit which is "the most immediate antecedent" of our response to the environment (67). The cumulative effects of our responses over time may promote health and disease—both mental and physical.

Caplan (67) has described how social support might protect us from the effects of a poor person-environment fit at six levels of functioning. 1) At the most elementary social level, direct aid can alter objective dimensions by increasing the environment's resources or altering its demands. Rather than modifying the effect of external stressors, this prevents them from occurring. Also, in the presence of potentially stressful stimuli, direct assistance (previous and/or concurrent) can modify the effect of the environment by teaching social network building skills (68), providing models of effective coping skills, and providing access to coping strategies (69). 2) At the level of perception, input from socially supportive relationships may allow a person to "form a more veridical view of the objective nature of self and environment" (67). 3) Given an accurate perception of external reality, one important socially supportive behavior may be the provision of information about the availability of other environmental resources, potential changes in demands, and the probable utility of planned coping strategies (69). This type of information may reduce the subjective evaluation of misfit. 4) Once a condition of subjective misfit has occurred, the inhibitory effect of socially supportive stimuli on the brain centers responsible for the physiology of "fight or flight" may modify the effect of environmental stressors by preventing a deleterious response. 5) Alternatively, the direct beneficial neuroendocrine effects of social support may buffer the immediate effect of a "fight or flight" response in progress, or 6) cumulatively prevent or reverse its adverse health consequences (49).

The majority of these plausible mechanisms are examples of effect modification which corroborate the findings of most studies designed to detect interactive effects (5, 6, 8, 9, 14, 16, 17, 22, 28, 40, 42, 43). It might be suggested, then, that the two studies which were unable to detect modification in the presence of direct effects (12, 23) were a result of statistical chance (i.e., Type II or β error). An alternative explanation is that in their study populations the stressful life events measures used were not sensitive or specific enough to reflect meaningful differences in the effect of social support between high and low stress groups. These results might have been different if an instrument one step closer to the actual experience of daily life were used (70).

Experiment or intervention. There has been little good research to evaluate the health promoting effects of social support intervention (33, 34). Three studies, already described, have used adequate randomized controlled designs with pre- and post-intervention measurements of status

(35–37); however the results were equivocal in one (36), and the forms of social support and outcome measures of the others are too different to make meaningful comparisons. More intervention studies are certain to occur, since intervention is the ultimate public health goal of all the previous studies; however, it is important that future intervention be adequately evaluated, as illustrated in a recent study of self-help intervention for widows by Vachon et al. (90).

Specificity of outcome. It is clear that the proposed effect of social support is *not* specific to any one disease state or organ system, but ranges from the mental to the physical. This does not detract from the argument for causality because the proposed mechanism of effect is plausible, consistent with existing theory, and is dependent on a complex interaction of mind and body mediated through neuroendocrine responses that have a wide range of consequences (49). Here is a requirement of Hill's (38) that cannot be met, because in fact some exposures have multiple disease or health promoting effects and are by their nature nonspecific. Another simple example is cigarette smoking with its effects as diverse as lung cancer and coronary heart disease.

Measurement of exposure. One problem with social support research is a lack of specificity in definitions of exposure variables. Various definitions of social support have been put forth (2, 3, 71, 72), but the definition of Kahn and Antonucci (71) is most comprehensive. They feel that social support refers to interpersonal transactions that include one or more of the following: *affect* (expressions of liking, admiration, respect, love), *affirmation* (expressions of agreement or acknowledgement of the appropriateness or rightness of some act or statement of another person), and *aid* (transactions in which direct aid or assistance is given—including things, money, information, advice, time and entitlement). The degree to which this definition has been operationalized varies from study to study.

Measures of social support generally fall into two categories—those dealing with the quality or content of interpersonal relationships and those dealing with quantity and other social network concepts such as size, frequency, density, etc. (73). In those studies which have compared both types of measures it is apparent that the quality of social support is a stronger predictor of health outcome than quantity measures (frequency of contact, number of friends) (14, 32), and quantity of social support is often not significantly related to well-being (5, 11, 13, 19).

Most measures of quality of social support have included questions pertaining to a confidant relationship and three reports found a beneficial effect looking at the confidant relationship only (8, 39, 42). The dimensions of affect and affirmation were present in most indexes of support quality and it can be suggested that these are merely functions of a confidant relationship. Another variable, which may be a component of affect and affirmation, is reciprocity or "reciprocal affective support." This, too, has been shown to positively affect psychologic well-being (13).

Only a few indexes have included measures of instrumental aid (6, 7, 9, 13), and, unfortunately, most of these have been combination indexes. In one study which looked at instrumental support separately, it was not a significant determinant of psychologic well-being, nor was reciprocal instrumental support (13).

One problem with a number of indexes or scales of social support was their combination of unrelated variables. For example, Ware and Donald (11), Broadhead (14), and Blazer (32) have shown by factor analysis that the quality of social support and frequency of social interaction are minimally intercorrelated and that it may be inappropriate to combine them into summary measures. But, some investiga-

tors have intentionally combined these constructs (12, 25), and others, whose measures are ostensibly indexes of quantity, have contaminated their data with a variety of possibly independent constructs—perceived quality or closeness of interaction (6, 7, 16, 30), voluntarily initiated social activity (11, 30, 31), hobbies (31), and even marital status (independent of marital satisfaction) (30). Similarly, the TAPPS score (an index score representing the adaptive potential for pregnancy) used by Nuckolls and colleagues (40) has been referred to by most reviewers as a measure of social support, but in addition a large number of psychologic status items are summed into the score.

Besides specificity of the content areas of social support, there is a need for more research into the relative benefits of specific sources of support and the role situational contexts play in the effect of potentially supportive relationships. For example, Holahan and Moos (27) found social support in the work environment was more beneficial for men while social support from the family was more beneficial for women. Morrow et al. (10) found that, for parents of children with cancer, the sources of social support which were most beneficial varied with whether the child was under treatment, in remission, or had died. Wells et al. (43) found that blue-collar workers' spouses and supervisors were more effective sources of support than friends or relatives. Coworkers were the least effective.

Determinants of social support. A description of the varying distribution of social support by demographic characteristics points out that while much of social support may be environmentally determined (e.g., by social class, size of community), much of it may be determined by characteristics of the person (e.g., age, race, sex, self-initiated social activity). Three good studies of the distributions of social support have been reported in the literature. 1) McFarlane et al. (74) studied family practice patients in Canada age 21–60 and measured size and sources of confidant-type help for six topic areas. Stephens et al. (7) studied informal support quality in a community survey of elderly persons in Texas. Ingersoll and Depner (75) similarly studied social networks of persons over age 55 by a social network analysis system which categorizes helping individuals into three circles of network membership (inner, middle, outer) based on their closeness and importance to the subject.

In general, the mean network size is 9 or 10 (74, 75). The inner circle is the largest component (three to four people) and the outer circle the smallest (two) (75). McFarlane et al. (74) break this down into average numbers for individuals by type: close friends (2.24 people), friends (2.21), work-related relationships (1.43), professionals (1.14), spouse (0.82), other family (0.65), neighbors (0.17) and others (0.02). The breadth of content for six topic areas of possible discussion is widest for one's spouse (5.13), close family (2.81), and friends (2.49); it decreases further with more distant relationships: work-related individuals (1.42), professionals (1.15), other family (0.88), and neighbors (0.19).

There is important variability by sex. Women's networks are slightly larger than men's at all ages greater than 55 with the difference being one more individual on an average in the "inner circle" of closest relationships. Women's networks have a higher proportion of family and friends, while men's are more work-related (74). Women also discuss more content areas with their networks and feel more helped by the people with whom they discuss their concerns. Stephens et al. (7) found that there is no change in average amount of informal support (a measure of quality and availability) by sex.

Marital status is also an important factor. McFarlane et al. (74) found that married individuals, of course, have more con-

tact with a spouse. They also have more work-related individuals in their networks. Single adults have a larger number of friends, and the widowed and divorced have more professional contacts. The widowed and divorced appear to feel more often that their network is not being adequately helpful or supportive. Stephens et al. (7) similarly found a gradient of informal supports with married individuals receiving the most, followed by the never married, the widowed, and then the divorced.

Age (or its consequences) causes a decrease in both network size (75) and amount of informal support (7) for persons over age 55. For women the decrease seems to be steady, but most of the decrease occurs in the "middle and outer circles" with the "inner circle" stable over time. For men, the "inner circle" decreases gradually with age, but the most variability occurs in the "middle circle." Here, the bulk of the decrease occurs at about the time of retirement, and is partially recovered by age 75 or above. Nevertheless, the older an individual is, the less apt he/she is to express a desire for a larger network of support, but the more likely he/she is to report health as a restriction on access to contact with the network. This latter phenomenon is more prominent among females (75).

A number of other personal characteristics affect availability of informal social supports. The study by Stephens et al. (7) reports that, as to race whites have greater access than blacks, who are more well off than Mexican Americans (in Texas). Persons living in communities smaller than 2500 in population have more informal supports than city-dwellers. There are definite gradients of increasing support with increases in social prestige and numbers of neighbors of a subject's own age. The amount of support decreases with numbers of living children (perhaps a reflection of a relationship between socioeconomic status and access to birth control). For the elderly, social support is lowest when three or more individuals live in a household, intermediate for persons who live alone, and optimal for households of two (perhaps these are intact married couples).

Two personal characteristics bear special mention. The availability of informal social support increases with both the number of organizations or clubs an elderly person belongs to and with his or her frequency of religious service attendance (7). These variables, discussed below, reflect an individual's own active social network building skills/abilities/options and they emphasize the individual's role in controlling and facilitating environmental access to social supports.

Dynamics of social support. Although we might think of social support as the static environmental exposure status of an individual, it, like any exposure, may fluctuate and has its own determinants of variability. That is to say, social support is a dynamic phenomenon. It has determinants which are internal to the individual (e.g., individual temperament or patterns of perceiving and interacting with the environment) and those which are externally mediated (e.g., social role definitions). Both types of determinants are active at all stages of the life cycle.

The internal determinants of social support are, in fact, coping strategies. Coping may occur at a behavioral, cognitive, or physiologic level (76). Although cognitive coping skills may affect perceptions of social support, it is the behavior of an individual in the form of social coping skills which affect the availability of social supports. A person who is either self-reliant or resigned to helplessness and does not seek the advice of others when help is needed may have less social support on an average than a person who copes actively by seeking advice, information, or simply someone to talk to about a problem (5, 77–80). Likewise, a person's

tendency to seek affiliation or social contact with others even in the absence of a problem will partially determine the social supports available (3, 69, 81). Thus, although measures of environmental social support should preferably exclude individual psychologic characteristics, a person's temperament with regard to social affiliation should be considered if an understanding of the process is to be reached.

Those psychologic characteristics which affect social support availability are under continuous change during development and have far reaching effects into adulthood. Even in early infancy we find evidence in discussions of "temperament" for the role of the individual in altering or controlling the social environment—especially with respect to the maternal-infant dyad (82). Affiliative or support-seeking skills learned in infancy are expanded upon during childhood and adolescence "through long sequences of experiences with considerable transfer of learning from one stressful episode to another" (69). The parental social network during childhood (68) and the peer social environment during adolescence (83) are very important in the development of precursors to adult social coping skills. The defenses used by the eventual adult in coping, and their social consequences, are, as we have discussed, primary determinants of social availability (84). Thus a person's "ability" (in person-environment fit terms) changes with age and experience, and these abilities may have direct effects on the "demands" and "resources" of the environment.

In addition to the cumulative effect of experience on one's skills or abilities to mobilize social supports, a person's roles, needs and circumstances also change with age and experience. The form and amount of social support appropriate depends upon these changes, and the nature of one's role after each life transition or life event determines not only the demands made by the environment, but the opportunities or resources for development of a social support system (71). Hence, the distribution of social supports varies, as we have discussed, with demographic and other personal characteristics related to role and is not independent of life events.

We see that the dynamics of social support involves a set of complex relationships between variables. Temperament and other individual psychologic characteristics interact with the social environment during all stages of development to produce behavioral coping styles (or patterns). These in turn interact with the current social environment to allow mobilization of social support or recruitment of a social network. A longitudinal or life-span perspective is essential to an understanding of these dynamics because the availability of social support at any one time is dependent on both the current state of affairs (with respect to needs, abilities, demands, and resources) and cumulative experience.

Clinical implications. What is the clinical significance of social support? The clinician may ask, "How will my knowledge of my patient's social support alter my treatment plan?" Knowledge of the dynamics of social support and its mechanism of association with mental and physical health or disease results in two categories of response—prevention and intervention. Prevention refers to the suggestion by Kaplan et al. (3) of the need for early childhood education and adult training in affiliation and coping skills to teach a person to garner the necessary social resources to maintain his or her health and help withstand the onslaught of stressful life events which are the inevitable consequences of living. In the realm of intervention, others have suggested that physicians and other health practitioners should be trained to identify high risk families and individuals, assess the nature and deficiencies of their social networks and through "anticipatory care"

(85) or "anticipatory guidance" (86) assist and teach them to manipulate their environments to acquire and maintain the social supports necessary for healthy survival (1, 3, 72, 86, 87). Intervention might also include direct mobilization of a patient's informal support system (family, friends, neighbors, community volunteers) by the clinician (88). The quality of support might also be improved by engaging patients in mutual support groups with other patients with similar problems or situations (36).

Conclusions and recommendations. As has been suggested before (3), social support is much more than a simple environmental exposure. It can be studied as an effect modifier or buffer against the stress of life events (1, 2), but also as a direct determinant of health or illness (an independent variable) and as dependent variable with its own causes and determinants. A more complete understanding of social support will result from a research model which considers social support from all three perspectives. We conclude this review by discussing the problems and research needs in each of these areas (summarized in table 2).

Social support as an effect modifier has, since the writing of Cassel (1) and Cobb (2), referred to the "buffering hypothesis." That is, social support was conceptualized as an exposure which interacted with the stress of life events to reduce their deleterious consequences. However, previous studies of this buffering relationship are difficult to interpret because they use inventories of life events which are precursors of change in social support, the proposed effect-modifier. Until stress and social support measures are "disaggregated" this relationship will not be adequately evaluated (89). There is a need for the development of stress measures which are not conceptually confounded with social support.

Other forms of effect modification or statistical interaction are often neglected. Three are evident in the literature reviewed here, and deserve closer study. 1) Social support has an effect which is apparently of greater benefit to women than men (5, 8, 27, 30). Is this a measurement error? Do men respond differently to questionnaires asking social support information? Or is this a substantive difference? Are the social supports effective for men different than those for women? 2) Social support may be more beneficial to

TABLE 2

Social support research needs

Social support as an effect modifier:
Need to investigate the effect of social support as a buffer against stress, using measures of stress which are independent from social support.
Need to investigate other statistical interaction with social support including sex, previous health status, education (and other measures of socioeconomic status).
Social support as an independent variable:
Need to address the issue of a possible third factor causing both declines in social support and health.
Need to corroborate social support theory by studies of experiments of nature as well as clinical intervention.
Need to explore various definitions of social support and the relative benefits of various types and sources of social support.
Social support as a dependent variable:
Need prospective study of personal and social characteristics which may be determinants of social support.
Need intervention studies which demonstrate effective increases in social support.
Social support and biologic plausibility:
Need to combine experimental and epidemiologic evidence on biologic mechanisms.
Need to investigate specific mediating biologic mechanisms.

those already in poor health (30). Are there certain groups of patients with certain types of illnesses or conditions who would be expected to benefit more from social support intervention strategies than others? 3) Education may also interact with social support so that the least educated receive the greatest benefit (14). Why then is the relative benefit of social support reduced for the well educated? Could education perhaps be an indicator of verbal and other skills necessary to receive vicarious social interaction from literature, theatre, and other arts, which nullify the effects of low social support? Or does education grant one the earning power to buy the necessary assistance and help, including counseling? Other potential effect modifiers need to be considered as well.

Our review of the characteristics of the association between social supports and health has concentrated on conceptualizing social support as an independent variable, and, given this perspective, a strong argument for a causal relationship can be made (38). However, specific weaknesses in this argument remain, especially with respect to a demonstration of the necessary antecedent-consequent relationship, adequate experimental or interventional corroboration, and clear definitions and quantification of exposure.

Although key studies of social support and mortality (30–32) demonstrate that poor social support precedes mortality even when controlling for baseline health status, the argument for the proposed cause-effect relationship is weakened by considering that social support and health may be independent consequences of a third factor (such as socioeconomic status) with chronic effects which may limit supportive resources and access to health care, thus resulting in poorer health.

Two types of "experimental" studies may help elucidate the nature of this relationship. 1) An "experiment of nature" may be studied in which a cohort of healthy, well supported individuals is observed for losses in social support and subsequent declines in health status. If losses of social support precede declines in health and are statistically independent of suspected third factors (e.g., socioeconomic status), this third factor hypothesis can be dismissed. However, if social support is not independent of a third factor, this would not be sufficient to establish a causal relationship between the third factor and social support. And, therefore, this would not disprove the hypothesis of a causal relationship between social support and health. 2) A more clearcut line of investigation might be one of experimental intervention. The argument for causality would be greatly strengthened by randomized controlled trials in which clinical intervention with poorly supported individuals could be demonstrated to produce a rise in perceived social support in the treatment group followed by a lower rate of morbidity and mortality compared with the control group. This type of study would be most efficient if groups of individuals at high risk of undesirable outcomes were used: e.g., victims of acute myocardial infarction, postoperative patients, pregnant women (37, 40), automobile accident victims (19), or widows (90).

The results of social support research vary with the definitions of social support which are used. A great need, then, is an evaluation of the multiple dimensions of social support to determine which have significant effects on health outcome, and then which have the strongest significant effects. Only after determining the relative effectiveness of the various kinds and sources of support will accurate evaluations of a patient's needs be possible, thus making social support a more useful clinical concept.

Finally, social support's role as a dependent variable has been neglected. Variables such as community characteristics, socioeconomic status, social roles, social coping skills and other personal

and social characteristics should be investigated prospectively as possible factors in the building and maintenance of socially supportive networks. It is this kind of information which would permit the design of prevention and intervention strategies of plausible efficacy. Once designed, adequate evaluations of these strategies are necessary to demonstrate true rises in perceived social support as well as changes in health and outcome.

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