

Social Supports and Physical Health: Symptoms, Health Behaviors, and Infectious Disease

Sheldon Cohen
Carnegie Mellon University

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Since the mid 1970s, there has been a strong interest among behavioral and medical scientists alike in the possible roles that social networks and social supports play in influencing physical health. This interest received substantial impetus in 1979 from the publication of data from a nine-year prospective study of the residents of Alameda County, California (Berkman & Syme, 1979). The study found that a "social integration" index—including questions about marital status, having friends, associating with neighbors, and belonging to formal and informal groups—was related to mortality. Initially healthy persons who were more socially integrated (i.e., had more friends, family and neighbor interactions, belonged to groups, etc.) lived longer than their less integrated counterparts. Apparently something about the social environment had an important influence on health.

This ground-breaking study raised at least as many questions as it answered. First, it was unclear how the social environment would influence physical health. What psychological, behavioral, and biological mechanisms linked the social environment to physical morbidity and mortality? Second, the quality of measurement of the social environment in the Alameda County Study (as well as later epidemiologic studies reporting similar results, e.g., Blazer, 1982; House, Robbins, & Metzner, 1982; Schoenbach, Kaplan, Fredman, & Klenbach, 1986) was not state-of-the-art, and the conceptual underpinning of the social integration index was virtually nonexistent. What did the index actually measure, and what other conceptualizations of the social environment would influence health?

Because many of these issues are primarily psychological in nature, the time was ripe for psychologists to get involved. Potential contributions included the provisions of: (a) theoretical underpinnings for various conceptualizations of

social networks and supports, (b) hypothesizes about how social supports influence health, and (c) psychometrically valid instruments for the measurement of social networks and social supports.

My own interests in this area were kindled during the late 1970s, and I have been involved in research on the relation between social support and health ever since. This chapter provides an overview of nearly ten years of research that I and my colleagues have done in this area and presents a representative sampling of this work. The work I report is meant to exemplify approaches psychologists can take to this problem but *is not* intended to be a comprehensive coverage of areas where important contributions can be made. I begin by summarizing some of the basic theoretical assumptions made when we began this course of study and by briefly discussing the development of a social support scale. I then summarize a series of studies we conducted on the influences of social supports on psychological and physical symptoms. This work was essential in the process of instrument development and in clarifying a number of theoretical questions. Discussion of the symptom work is followed by summaries of our more recent work on the roles social supports play in determining two "harder" outcomes: health behaviors and biologically verified disease. Included are studies on perceived support as a buffer of the effects of stress on quitting smoking and ongoing work on the role of social supports in susceptibility to infectious disease.

STRESS-BUFFERING VERSUS MAIN EFFECT MODELS

There were few data on the influence of support systems on physical health when the Alameda County study was published. However, John Cassel (1976; also see Cobb, 1976), a social epidemiologist at the University of North Carolina, had proposed that social support positively influenced health and well-being, and he suggested a possible mechanism for this influence—stress-buffering. The idea of the stress-buffering hypothesis is that social support operates by protecting people against the pathogenic effects of stressors. That is, stressors put one at risk for disease, but stressor related risk is reduced or totally ameliorated when those confronted with stressors have strong social support networks. An important corollary of this hypothesis is that outside of facing stressful events, social support is not really important.

Figures 11.1B and 11.1C present two theoretical versions of the data predicted by the stress-buffering model. Figure 11.1B represents an extreme version, suggesting that support totally ameliorates the influence of stress on health outcomes. (Symptoms are used as an example in the figure). Figure 11.1B represents a more moderate version of the hypothesis, suggesting that support attenuates the influence of stress but does not provide total protection. Both versions of the stress-buffering hypothesis are supported by a statistical interaction of stress and social support.

The alternative hypothesis is that stress is not really an issue in social support's

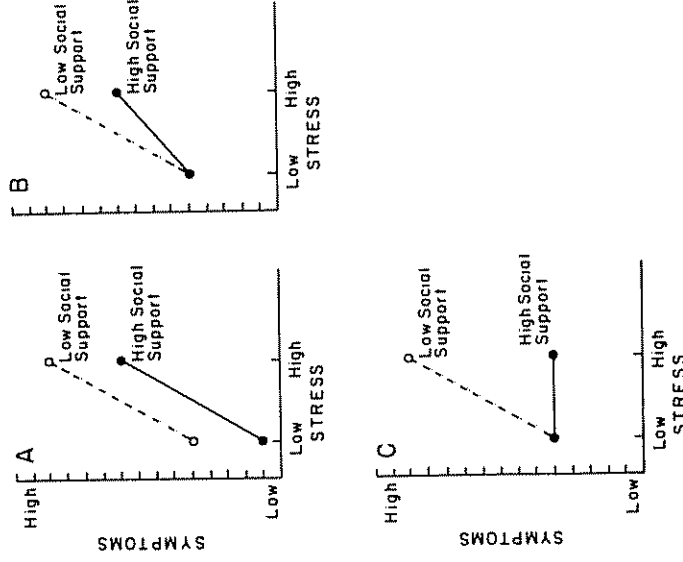


FIG. 11.1. Depiction of main effect of social support on symptomatology and two forms of the stress-buffering interaction. Reprinted from Cohen & Wills (1985).

influence on health. That is, having social support is beneficial to health overall, irrespective of stressor exposure. This is termed the main effect hypothesis, because it predicts a main effect of social support *without* a statistical interaction between stress and social support. Figure 11.1A represents this alternative.

The focus of most of the work discussed in this chapter is on establishing and understanding the stress-buffering hypothesis. However, many of the studies pit the main effect and stress-buffering hypotheses against one another. Moreover, near the end of the chapter, I return again to discussing the main effect model. At any rate, FIG. 11.1 provides a template to compare with results of the studies I report, providing an aid in interpreting which of the models is supported by each study.

Support Concepts and Support Mechanisms

Although there are still very few studies comparing the stress-buffering and main effect models in the prediction of physical disease, the literature on the relative efficacy of these models in the prediction of psychological symptomatology has grown enormously over the last ten years. In 1985, Tom Wills and I published

an integrative review article of this work (Cohen & Wills, 1985). The published review included over 40 studies comparing the main-effect and stress-buffering hypotheses.

The Cohen and Wills (1985; also see Kessler & McLeod, 1985) review found support for both main effect and stress-buffering models. However, which model was supported in any specific study depended on the nature of the social support or network measure that was used. Stress-buffering effects were found with measures that assessed *perceived availability* of social support. Main-effects were found with measures that assessed *degree of integration* in a larger social network. This latter measure is the social integration type measure used in the Alameda County mortality study.

Psychologic Mediators of the Relation between Social Support and Health

What are the psychologic and behavior factors that link the social environment to health? In the following section, I selectively review some alternative mechanisms. (A detailed discussion of mechanisms linking support to health can be found in Cohen, 1988). The focus is on the influences of the social environment on the *onset and progression* of disease. Although many of the arguments also apply to disease recovery, a thorough treatment of recovery requires additional considerations such as the influence of support on disease symptoms, rehabilitative behaviors, availability and quantity of caretaking, and so forth. Finally the discussion and models (FIGS. 11.2 and 11.3) are limited to paths moving in one direction, from support to health or illness. Alternative paths are excluded for the sake of brevity. Their exclusion is not, however, intended to reflect any hypotheses about their existence.

Main-effect model. Figure 11.2 represents the main-effect hypothesis—social support directly influences health and well-being irrespective (independent) of stress levels. Because our review suggested that main effects are driven by *social*

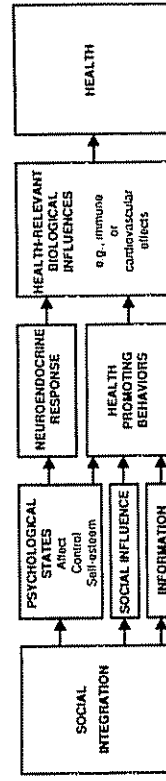


FIG. 11.2. Main-effect model of the psychological and biological pathways linking social integration to the onset and progression of disease. All indicated paths move in one causal direction. The exclusion of alternative paths is not intended to reflect any hypotheses about their existence.

integration, the explanations I present are directed at this conceptualization of social networks. First, social integration may facilitate health-promoting behaviors. Persons with stronger networks may take better care of themselves (e.g., exercise, drink less, and smoke less). Better self-care could occur, because an integrated network makes persons feel better about themselves (e.g., elevated self-esteem and feelings of control), because networks provide accurate information in regard to a healthier life style, or because networks apply social pressures on members to engage in health promoting behaviors. (Of course, networks can also provide inaccurate information or apply social pressures to engage in behaviors detrimental to health, e.g., McKinlay, 1973; Sanders, 1982; Seeman, Seeman, & Sayles, 1985). Second, belonging to an integrated network may cause positive changes in psychological states (affect, control, self-esteem) that influence neuroendocrine response (cf. Bovard, 1959). Hormones released (or suppressed) as a response to these states are presumed to influence disease pathogenesis through their effects on biological systems more proximate to disease outcomes (e.g., the immune and cardiovascular systems) (cf. Ader, 1981; Glass, 1977). Third (not represented in FIG. 11.3), integrated social networks may prevent disease by providing material aid, for example, the provision of food, clothing, and housing could limit exposure to risk factors such as insufficient diet and exposure to cold. Finally social integration may influence health merely because it indirectly reflects exposure to stress (not represented in FIG. 11.3). For example, social networks may provide warnings and information that help persons avoid stressor confrontation. Alternatively weak social networks (isolation) may act as stressors and hence *not belonging* to an integrated network may put persons under disease risk.

Stress-buffering model. Figure 11.3 represents the stress-buffering hypothesis. In this case, I focus on explanations for the role *perceived availability of social support* plays in this process. The theory in developing this model was that people

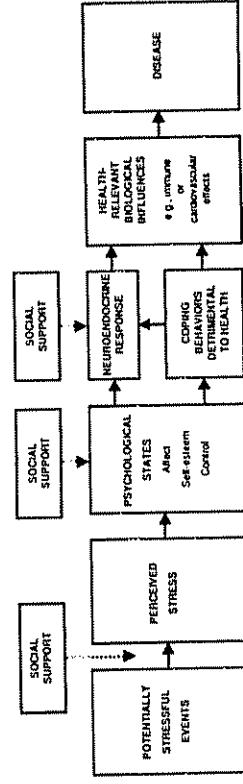


FIG. 11.3. Stress-buffering model of psychological and biological pathways linking perceived availability of support to health and well-being. All indicated paths move in one causal direction. The exclusion of alternative paths is not intended to reflect any hypotheses about their existence.

confront objective stressful events and appraise whether or not they can cope with those events (Lazarus, 1966; Lazarus & Folkman, 1984). If they find the event threatening or otherwise demanding, and at the same time find their coping resources inadequate, they experience stress. We will refer to this state as perceived or appraised stress. The appraisal of stress is presumed to result in negative psychological states that either trigger behaviors detrimental to health, or biological processes (e.g., elevated neuroendocrine response) that put persons under risk for various diseases.

Perceived support can enter into this model through its influence on the stress appraisal process; that is, a supportive network may help by providing information regarding how threatening an event actually is, suggesting effective coping responses and/or providing aid in coping (e.g., Cohen & McKay, 1984; Thoits, 1985). The perception of support may merely reflect these processes. However, because the appraisal process is cognitive in nature (i.e., it all goes on in your head), the mere belief that such resources are available may be enough to short-circuit stressor appraisal. This argument suggests that perceived support works not because it provides a reasonable approximation of available resources, but because the *belief* that support is available is what is critical in appraising whether events are stressful (e.g., Wethington & Kessler, 1986).

Perceived support may also operate after a situation is appraised as stressful, between stress appraisal and the illness outcome (cf. House, 1981). This could occur through reappraisal of a situation initially judged as stressful, through the facilitation of successful coping with stress-induced affect (Thoits, 1985), through the discouragement of unhealthy behavioral coping styles, or by directly short-circuiting a stress-triggered biological process (e.g., damping neuroendocrine response).

There is another theoretical prediction regarding stress-buffering effects that is important for the work I am going to discuss. We predicted that there must be a *match* between the needs elicited by a stressor and the available support resources in order to get a stress-buffering effect (Cohen & McKay, 1984; Cohen & Wills, 1985). For example, if you lose your job, having friends who will gladly give (or loan) you money may be appropriate support. On the other hand, if a spouse, parent, or friend dies, having friends who will give you money is not very helpful. So there is a need for a match between the kinds of resources that are available and the kinds of needs that are elicited by the stressful events that one confronts.

What is proposed earlier is a specific matching hypothesis (i.e., there needs to be a match between the specific needs elicited by a stressor and available social resources). Tom Wills and I also suggested a more global matching hypothesis; there are some globally useful resources that are helpful in confronting a wide range of stressors (Cohen & Wills, 1985). One of these resources is appraisal support—having people you can talk to about problems, who can help define problems, provide ways of coping with problems, and ways of coping with stress-

triggered affect. The other one is self-esteem support—having people who make you feel better about yourself. We felt that appraisal would serve as a global resource because the appraisal of most stressors is subject to information provided by others. (For exceptions, see discussion of social comparison as a model for social support in Cohen & McKay, 1984). We felt that self-esteem would operate as a global resource, because many stressors are in fact threats to self-esteem (cf. Wills, 1985).

Measuring Perceived Social Support

Our early work focused on the stress-buffering hypothesis. Although the literature suggested that studies using perceived support measures (and appropriate methodologies) often found stress-buffering effects, there were almost as many measures as there were studies. Moreover, the measures available at that time were impure in terms of what concept they represented and lacked in terms of traditional psychometric validation.

For the reasons I just outlined and because I wanted a measurement instrument that assessed different types of social resources, the first step was to design a measure of perceived availability of social support. I wanted a scale score that reflected the total perceived availability of social support as well as subscale scores reflecting different types (or functions) of social resources. In developing the Interpersonal Support Evaluation List (ISEL), we focused on four functions that I had proposed as important in stress-buffering: appraisal support—having people to talk to about problems; self-esteem support—having people who make you feel better about yourself; belonging support—having others to do things with; and tangible support—having people who would provide material aid (Cohen & McKay, 1984). Separate forms of the ISEL were designed for both college students (Cohen & Hoberman, 1983) and for the general population (Cohen, Mermelstein, Kamarck, & Hoberman, 1985). Psychometrics on these scales have been reported in over 10 studies and are summarized in Cohen et al. (1985).

Subscale independence. There has been some controversy recently as to whether the ISEL subscales actually differentiate between different support functions (House & Kahn, 1985; Sarason, Shearn, Pierce, & Sarason, 1987). In response, let me refer you to the recent work of Jeff Brookings (Brookings & Bolton, 1988) that reports a confirmatory factor analysis of the student scale. Brookings found that the models that held up best were the ones that combined all of the subscales (entire ISEL score) and the one that proposed the four separate subscales. In our own work with more standard factor analysis, we have consistently (across samples) found independent factors for appraisal and tangible support. We often find that self-esteem support is also relatively independent, but it occasionally (especially in adult samples) clusters with appraisal. In most

cases, however, belonging is so highly correlated with appraisal that we are unable to separate them empirically. As will be apparent from the studies we discuss later, even with some overlap between scales, discriminating between support functions has proved to be empirically useful.

THE SYMPTOM STUDIES

The first study using the ISEL examined the possibility that perceived support buffered the effects of negative life events on symptomatology. The subjects were 70 undergraduates at the University of Oregon (Cohen & Hoberman, 1983). Figure 11.4 presents the data when the entire ISEL is considered. As apparent from the figure, when depressive symptoms were the outcome, we found the stress-buffering interaction that was predicted (similar to FIG. 11.1B).¹ We find a similar effect with physical symptoms, although there is an unpredicted crossover with those under low stress having somewhat higher symptomatology related with higher levels of support. Figure 11.5 presents similar data from a study of 112 Oregon undergraduates conducted one year later. In this case, we found a stress-buffering effect with both depressive and physical symptoms without a cross-over in the physical symptoms data.

Also of importance are the subscale data. Subscale data from the Cohen and Hoberman study indicate stress-buffering effects for appraisal, belonging, and self-esteem support. Neither a main nor buffering effect was found in the case of tangible support. Unfortunately subscale data from the second Oregon student study are not available.

The next study is not from our lab, but because it used the ISEL in testing the stress-buffering hypothesis, it provides relevant information for our discussion. These data are from a sample of 92 University of Delaware students collected by Larry Cohen (L. Cohen, McGowan, Fooskas, & Rose, 1984). The study was longitudinal—stress and symptoms were measured at the onset of the study and stress, social support, and symptoms measured two months later. Cohen analyzed the data cross-sectionally at time 2 and prospectively, predicting time 2 symptomatology from time 1 stress and time 2 support while controlling for time 1 symptoms. Both the cross-sectional and prospective analyses indicated support for a stress-buffering effect of the overall ISEL. Figure 11.6 shows the effect for the cross-section at

¹No statistical analyses are reported in this chapter. Most of the individual studies have been published elsewhere with detailed statistical descriptions of the analyses reported in the original publications. When an effect of stress-buffering is indicated, a significant ($p < .05$) stress $\times$ support interaction was found. The interaction is tested after the main effects of stress and main effects of social support have already been entered into the equation (see Cohen & Wills, 1985; Reis, 1984). In order to avoid Type I error, subscale analyses of the ISEL have been done only when the overall stress $\times$ ISEL effect is significant. They are considered exploratory. Data reported in the cold study are all based on correlations with $p < .05$ or better.

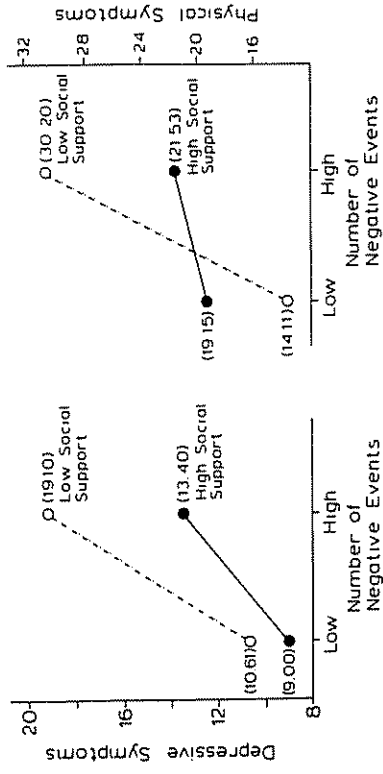


FIG. 11.4. Depiction of the interaction between number of negative life events and perceived availability of social support in the prediction of physical and depressive symptoms from a sample of 70 University of Oregon undergraduates. Reprinted from Cohen and Hoberman (1983) with permission of V. H. Winston and Sons, Inc.

time 2. Subscale contributions varied somewhat across analyses. Time 2 analyses predicting scores on the Beck Depression Inventory (BDI) indicated appraisal, self-esteem, and belonging were operative, while the prospective analysis of BDI scores indicated that only the self-esteem scale was operative.

Figure 11.7 presents data from a longitudinal study of 64 people participating in a clinic-based smoking cessation program (Cohen et al., 1985; Mermelstein,

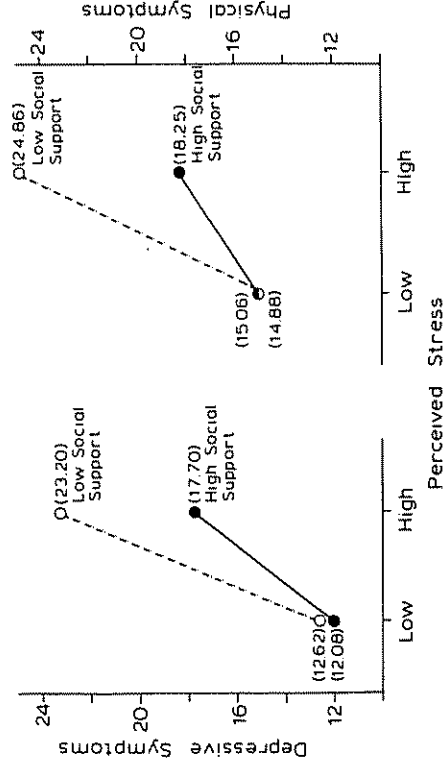


FIG. 11.5. Depiction of the interaction between perceived stress and perceived availability of social support in the prediction of physical and depressive symptoms from a sample of 112 University of Oregon undergraduates.

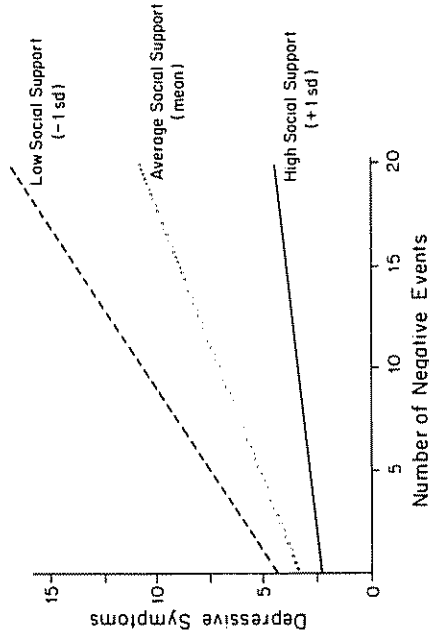


FIG. 11.6. Depiction of the interaction between number of negative life events and social support from a sample of 92 University of Delaware undergraduates. Adopted from FIG. 2 in Cohen et al. (1984).

Cohen, Lichtenstein, Kamarck, & Baer, 1986). It is a six-week prospective study, with perceived stress, social support, and physical symptoms measured at time 1, and physical symptoms measured again at time 2. The analysis predicted symptoms at time 2 from time 1 stress and support, controlling for time 1 symptoms (cf. Monroe, 1983). As apparent from the left side of the figure, there is a stress-buffering effect for the overall ISEL scale. If you examine the role of the separate subscales, the effect is almost entirely attributable to the appraisal scale. The effect of appraisal is depicted on the right side of the figure.

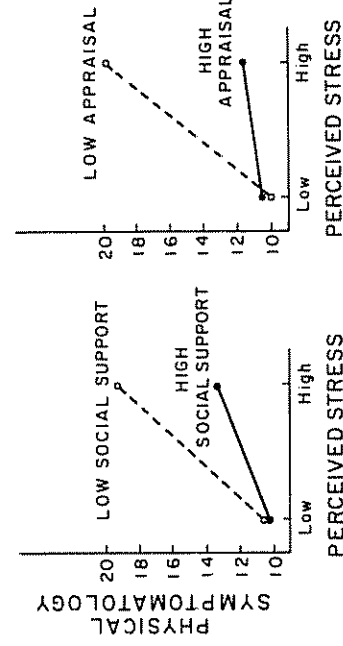


FIG. 11.7. Depiction of the interaction between perceived stress and perceived availability of social support in the prediction of physical symptomatology from a sample of 64 persons enrolled in a clinic smoking cessation program. The figure on the left is based on the scores from the entire ISEL. The figure on the right is based solely on the ISEL Appraisal subscale.

Threshold or continuous effect? Figure 11.8 is reversed in orientation to attract your attention, because it is slightly different in content. These are data from a study of 609 incoming freshman at Carnegie Mellon University [CMU] (Cohen, Sherrod, & Clark, 1986). One of the questions we addressed in this study was whether stress-buffering effects occurred at a particular threshold of perceived support, or whether the apparent protective influence of perceived support increased in a continuous linear fashion. In short, does some minimum amount of social support protect persons, with additional social support not making any difference? The first thing to notice about the figure is that we again found a stress-buffering effect of the ISEL. Second, as social support increases, the difference in symptomatology between low and high stress decreases. In other words, the more social support available to persons under high stress, the better off they are (i.e., the closer they are to being like the low-stress group). Hence, we found a linear effect, not a threshold effect. Finally the stress-buffering effect of the ISEL in this study was attributable to the appraisal, self-esteem, and belonging subscales. All three demonstrated stress-buffering effects, but there was no buffering effect of tangible support.

Are stress-buffering effects an artifact of personality? My concern with this question arose out of an issue raised by Ken Heller (1979; Heller & Swindle, 1983). Specifically Heller suggested that stress-buffering effects of social support may be due to differences in social competence. In short, the argument was that socially competent people are both more able to attract social support and more able to cope with stressful events.

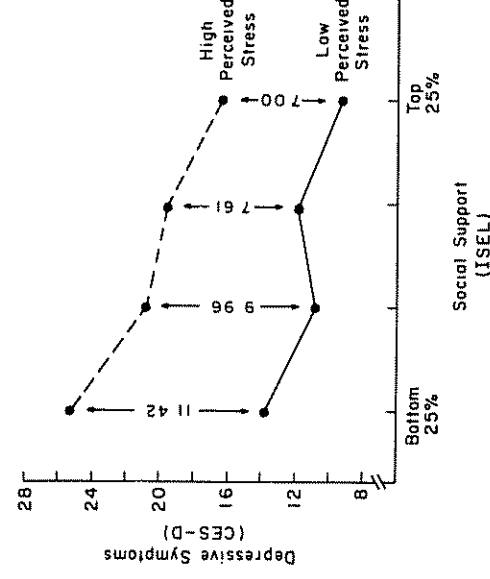


FIG. 11.8. Depiction of the interaction between perceived stress and social support from a sample of 609 freshman undergraduates at Carnegie Mellon University.

We examined the issue of whether social skills could account for the stress-buffering effects in the study of CMU freshman discussed earlier. As you recall from FIG. 11.8, perceived support operates as a stress buffer in this study. In an additional analysis, we investigated the possible role of three social skills: social confidence, self-disclosure, and social anxiety. In a regression analysis, we forced the three main effects of these scales in first, then life events and social support, then the interactions between stressful life events and the social skills scales, and finally the interaction between life events and social support. In short, we tested for the possibility that the stress-buffering effect of social support occurred above and beyond (independent of) any similar effects of the social skills measures. The results indicated that the support effect held up, even after controlling for the possible influence of these three social skills (Cohen, Sherrod, & Clark, 1986).

These are not, of course, the only personality measures that may account for the stress-buffering effects. However, to make a strong argument that perceived support is acting as a proxy for any personality variable, one would need to show that the variable under consideration operates as a stress buffer. In order to determine which factors may provide an alternative explanation for support effects, we critically reviewed the published studies on personality factors as stress buffers (Cohen & Edwards, 1989). Studies of the following personality factors were included in our review: anomie, arousal, and sensation seeking; coping flexibility; coping styles; hardiness; locus of control; private self-consciousness; self-esteem; social interests; social skills; and Type A behavior. We concluded that locus of control is the only personality factor whose stress-buffering capabilities receive any substantial support. When defined as a generalized concept applying only to control of things outside oneself, the data are fairly consistent with the operation of internal control as a stress buffer. I do not want to deny that any of these other factors act as stress buffers but to suggest that there is no convincing evidence at this time that this is true.

In sum, there is considerable evidence that perceived support operates as a stress buffer but little that plausible proxy variables do. There are no published studies directly addressing the possibility that locus of control, the one variable that appears to operate as a stress buffer, underlies the perceived support effect. However, studies demonstrating that persons with *internal* control and high levels of social support are less impacted by stress than persons with *external* control and high levels of social support indicate that control operates as a moderator of stress by support interaction, not as a proxy for support (Lefcourt, Martin, & Saleh, 1984; Sandler & Lakey, 1982). Hence, up until this point, proxy explanations of perceived support effects seem unfounded.

Are perceptions of available social support valid? An issue related to the discussion of personality proxies is whether measures of perceived social support reflect the actual availability of support or merely a grossly distorted representation of the availability of social resources. It is conceivable that persons have

biased cognitive representations of their social environments. Moreover, these biases could be driven by stable individual differences in personality. Several investigators have addressed the issue of whether perceived availability of social support really is a reflection of people's social support systems. Carolyn Cutrona (1986), Amiram Vinokur and his colleagues (Vinokur, Schul & Caplan, 1987), and Harry Reis (personal communication) have all done independent work where they measured the perceived availability of social support in people and subsequently had them track their interpersonal interactions over some period of time. All three studies found that perceived availability of social support corresponded with people's reports of the support available to them in their environment. Particularly interesting in this context is Harry Reis' work. Using the ISEL, Reis found that people who are high on the appraisal scale (who said that they had people they could talk to about problems, who could help them cope with problems) subsequently reported having more interactions in which they talked to friends about problems than people who were lower on that scale.

Summary. Our work on perceived availability of support as a buffer of cumulative stress effects on symptomatology clearly and consistently indicates stress-buffering effects of the overall ISEL scale. These effects were found in both cross-sectional and prospective studies, and typically suggest a partial (see FIG. 11.1b) ameliorative effect. The effect also appears to be linear in nature, that is, it increases with increasing perceptions of support. In most studies, appraisal and/or self-esteem support were at least partly responsible for stress-buffering effects. However, belonging was often similarly operative. Only tangible support was consistently ineffective. We used cumulative stress measures as opposed to looking at specific stressors (e.g., exams, bereavement, or unemployment), therefore, the reviewed work does not provide a good test of the hypothesis that support is effective only when it matches the needs elicited by stressful events. The general effectiveness of appraisal and self-esteem support are, however, consistent with our argument that these two types of support operate as global resource.

The studies examining possible alternative explanations for the perceived social support effect suggest little evidence for such alternatives. Stress-buffering effects of perceived support survive analyses where social-skill effects are partialled out, and perceived support seems to reflect the actual availability of social resources.

PERCEIVED SOCIAL SUPPORT AND SMOKING CESSATION AND MAINTENANCE

As mentioned earlier, a primary path through which social support could influence health is by influencing health relevant behaviors. Smoking is the behavior most strongly linked with physical health outcomes, thus, studying the role of support in smoking behavior seemed like the appropriate place to start. Hence, Ed

Lichtenstein and I began a program of research on the role of social supports in quitting smoking and maintaining abstinence. We began by considering how social networks could influence people's attempts to quit smoking. We looked at a number of different possibilities (see Cohen, Lichtenstein, Mermelstein, Kingsolver, Baer, & Kamarck, 1988, for a detailed discussion). However, for the purpose of this chapter, I would like to stick with the stress-buffering issue. Our thinking in this area was that stress makes it harder to quit smoking and maintain abstinence, and that support should help protect quitters from the deleterious effects of stress on the quitting process (see Shiffman et al., 1986; Wills & Shiffman, 1985). Moreover, although environmental and social stressors were important considerations in this context, we felt that it was even more important to consider the stress created by the act of quitting. If perceived social support operated as a stress buffer, persons with higher scores on the ISEL would be more likely to succeed at this stressful task. We assume all quitters are under relatively high levels of stress and, hence, are concerned here with a main effect of perceived availability of support rather than an interaction between stress and support.

Our analysis got a little more complicated in that we broke up the sequence of quitting into different stages: the decision to quit, quitting itself, early maintenance (defined as the first three months after quitting), and late maintenance (three to twelve months after quitting) (see discussion of stages by Prochaska & DiClemente, 1983; and by Brownell, Marlatt, Lichtenstein, & Wilson, 1986; our theoretical discussion of support and stages in Cohen et al., 1988). We hypothesized that stress would be particularly important for cessation and early maintenance but relatively unimportant for late maintenance. This hypothesis partly derived from existing evidence that we and others had collected suggesting that stress was important during the early stages of quitting (Cohen, 1986; Cohen, Kamarck, & Mermelstein 1983; Shiffman, 1982) and partly from our gut feelings that the early stages were the most likely points for stress to interrupt the quitting process. The studies I will discuss do not address the decision to quit stage but only the last three stages. We predicted that perceived availability of social support would predict who quits smoking and success at early but not late maintenance.

We have, in the last eight years, conducted four one-year prospective studies. In these studies, people are given the perceived social support measure (the ISEL) upon entering the study—before starting to quit smoking. The first three studies each involved between 50 and 70 persons enrolled in formal smoking cessation programs run at a university clinic (Baer, 1987; Cohen et al., 1988; Mermelstein et al., 1986). The fourth study included 465 people in the Pittsburgh community who were quitting smoking by themselves, 70 of whom were administered the ISEL. As noted previously, in all four studies, we assessed perceived support before participants began the quitting process and then followed them for one year after their expected quit date.

The results of these studies are summarized in Table 11.1. As apparent from the table, in one clinic study, the total ISEL score predicted *cessation*, and the appraisal and self-esteem subscales were responsible for the effect. However, the consistent finding is clearly for *early maintenance*. Across three of the four studies, we found that those who quit (for at least 24 hours) were more likely to still be abstinent at three months, if they had high scores on the ISEL.² In the first two of those studies, the effects were due primarily to the appraisal scale. In the self-quitting study, none of the subscales were independently related to outcome. The one piece of datum that is inconsistent with our *stage* prediction occurs in the third study. Unexpectedly the ISEL also predicted long-term maintenance. That is, persons with higher ISEL scores who were abstinent at three months were more likely to be abstinent at twelve months than their counterparts with lower scores.

In short, the data roughly fit our model. Overall, there is strong evidence for the positive influence of high levels of perceived social support and suggestive evidence for the argument that stress and, therefore, the perceived availability of support are most important during quitting and early maintenance.

THE COMMON COLD STUDY

Although there has been a great deal of research on the influence of social supports on symptomatology, there are few data linking any conceptualization of support to the onset of a biologically verified disease. This section provides a description of our ongoing work on the effects of social integration and social support on susceptibility to infectious disease. The foci of this work include establishing relations between support and biologically verified disease, and identifying behavioral, psychological and biological mediators of support-disease links.

This study is being conducted in collaboration with David Tyrrell and Andy Smith of the Common Cold Unit (CCU), which is part of the Medical Research Council in Britain. The CCU runs clinical trials to assess the effects of various cold viruses. Over the 30 or so years that the Common Cold Unit has been running trials, they have found that there is tremendous variability in who among those exposed to viruses develop colds. Up until now, attempts to explain this variability have primarily been immunologic in nature. (For exceptions, see Broadbent, Broadbent, Phillips, & Wallace, 1984; Totman, Kiff, Reed, & Craig, 1980).

²Given a directional hypothesis and the replicative purpose of studies 2 through 4, the use of a two-tailed test in evaluating the relation between support and three-month continuous abstinence is quite conservative. One-tail probabilities for studies 2 and 3 would be .03 and .05 respectively. It is also possible to calculate the joint probability corrected for chance differences of finding these three-month results across the four studies. The nonsignificant probability for study 2 is not readily available, therefore, I will make the conservative assumption that it is 1. Hence, the joint probability corrected for chance is $1 \times .05 \times .06 \times .10 / .5^4 = .005$.

TABLE 11.1
Stages at Which Pretreatment ISEL (and ISEL Subscales) Predicted
Smoking Cessation and Maintenance in Four Prospective Studies¹

	Stage of Cessation ²		
	Cessation (End of Treatment)	Early Maint. (3 Mo. Post)	Late Maint. (12 Mo. Post)
Clinic Study I	ISEL Total (Appraisal & Self-Esteem)	ISEL Total (Appraisal)	
Clinic Study II			
Clinic Study III		ISEL Total* (Appraisal)	ISEL Total (Appraisal)
Self-quit Study IV		ISEL Total**	

¹There were no significant effects in cells where the scale and subscale are not indicated. In all cases where effects occurred, persons with higher social support scores were more likely to quit or remain abstinent.

²The cessation state represents the predictions of quitting smoking for at least 24 hours. The early maintenance stage represents prediction of smoking status at 3 months for 24-hour quitters. The late maintenance stage represents prediction of smoking status at 12 months for persons who were abstinent at 3 months.

p < .05, if no asterisk

*p < .06

**p < .10

Overall, however, the previral challenge antibody levels standardly used by the CCU have not been extremely successful in predicting outcome. It is clear that persons with specific neutralizing antibody to a virus (those who have been previously exposed to the virus) are at least partially protected from reinfection. However, beyond that, there has not been much variance accounted for.

In my collaboration with the CCU, we are examining whether we can predict response to viral challenge from a psychological profile taken prior to challenge. In particular, the emphasis is on assessing the possible roles of social integration and perceived social support in susceptibility to infection and in specifying the psychologic, behavioral, and immunological pathways that link support to susceptibility. When completed, the study will include over 1,000 volunteers, allowing adequate power for testing hypotheses that require multiple controls and an opportunity for split-sample replication.

Main effect and stress-buffering models. We are testing both the main effect and buffering models discussed earlier (FIGS. 11.2 & 11.3). In the case of main effects, we are specifically testing the proposal that social integration has a main effect on susceptibility to infection, with more integrated persons showing less

infection and less symptomatology. Psychological mediators of a social integration-infection link that we are considering include increased personal control, self-esteem, and positive affect, and decreased negative affect. Health behavior mediation under consideration includes decreased smoking and drinking, increased exercise, improved diet, and quality of sleep. Finally immune mediation is being assessed by a number of measures of both humoral and cellular immunity.

In the case of stress-buffering effects, we are testing the proposal that stress increases susceptibility to infection and that high levels of perceived support protect (or buffer) against this increased risk. We hope to determine the point at which perceived support short circuits the stress-disease process by assessing objective stressful events, stress perceptions, and negative affective responses. (Recall that we proposed earlier that support may intervene between the objective event and perception of that event and/or after an event has been appraised as stressful—see FIG. 11.3.) We are also interested in identifying psychological, behavioral, and biological pathways that link stress to infection. Hypothesized psychological pathways we are testing include lowered personal control, self-esteem, and positive affect, and elevated negative affect. The five measures of health behavior discussed earlier will be used to assess whether stress influences on susceptibility are mediated by health behaviors. Here the assumption is that stress may result in coping behaviors deleterious to health (e.g., increases in smoking and drinking, decreases in exercise and sleep, and a poorer diet), and as a consequence place persons under greater risk for infection. If perceived support operates as a stress buffer in this case, no relation between stress and health behaviors would be expected for those with high levels of support.

Trial description. Healthy volunteers are recruited to participate in a nine-day, live-in isolation trial at the CCU. Before arriving at the unit, demographics and medical histories are obtained through the mail. Prior to viral exposure, each volunteer is given a thorough physical examination, has blood drawn, and nasal secretion samples are taken. Psychological measures and health behaviors are also assessed during this three-day, prechallenge period. Late on the third day or early on the fourth, the volunteers are exposed (in nasal drops) to a cold virus or to a placebo. Over the remainder of the trial, their biologic and symptomatologic responses are carefully tracked. Nasal secretion samples are collected daily to detect viral shedding (cellular reproduction of the virus). Handkerchiefs (tissues) used by volunteers are placed in plastic bags and sealed—they are later counted and weighed to assess the amount of nasal secretion, and daily symptom interviews are conducted by a physician blind to psychologic and immune status and to their experimental condition. On the ninth day of the trial, ratings of volunteer satisfaction with their flatmates, physician clinical ratings, and volunteer clinical ratings of their cold are all obtained. Three weeks after the beginning of the trial, a final blood sample is collected (by volunteer's own physician or local hospital) to look at serum antibody levels that take that long to change.

At the end of the trial, volunteers are classified into three outcome categories: clinical cold, subclinical cold, and not infected. To be assigned to the clinical cold classification, a volunteer must show both biologic (a four-fold increase in the production of specific antibodies to the virus and/or virus shedding—cellular reproduction of the virus) and symptomatologic responses (e.g., headache, runny nose, fever, handkerchief use, etc.). Those in the subclinical cold category demonstrate biologic infection but *do not* have cold symptoms. Finally those in the not-infected category show neither biologic nor symptomatologic indications of being exposed to the virus. On the average, roughly one-third of the volunteers fall in each category.

Some preliminary results. The evidence discussed in the following section is preliminary, based on approximately 300 volunteers. These data are cross-sectional in nature. That is, they involve correlations between the psychosocial factors, health behaviors, and immune function at entering the trial. We need very large numbers to provide a fair test of the stress-buffering hypothesis (cf. Reis, 1984), therefore, I will limit the discussion to the main effect (social integration) model.

As discussed earlier, social integration may influence disease susceptibility through a number of psychological states and/or through health behaviors. In this study, we conceptualized social integration as the number of important roles persons hold (Thoits, 1983). A scale—the Social Network Index (SNI)—was developed that assessed both the number of roles (e.g., spouse, parent, child, employee, student) and the number of relationships across these roles. The associations reported here are found for both measures but are generally stronger for the number of important relationships measure. First, let us ask, if social integration is associated with psychological states. The preliminary data indicate positive correlations between social integration and positive affect, self-esteem, and personal control. There is *no* association of social integration and negative affect. Moreover, social integration is also associated with all five health behavior measures. The greater the level of social integration, the less smoking and drinking, the better diet and sleep, and the more exercise. Finally social integration is correlated with a number of the cellular immune measures in the study. However, whether these relations are indicative of relatively higher levels of immunocompetence in regard to the virus challenge must await analyses of infection susceptibility. In sum, preliminary data suggest the plausibility of many of the pathways between social integration and disease discussed earlier (see FIG. 11.2). The lack of a relation between social integration and negative affect suggests, however, that stress triggered by isolation may not be the underlying cause of SI effects on health. However, this sample is less likely to include the truly isolated individuals who are present in the general population samples and, hence, may not provide an adequate test of the isolation hypothesis.

CONCLUSIONS

The epidemiological data on the role of social integration in morbidity and mortality have clearly established that the social environment plays an important role in health and well-being (see review in Cohen, 1988). Our own work suggests that pathways through which such effects occur depend on the particular conceptualization of the social environment one chooses to study. Social integration appears to promote health irrespective of stress levels, whereas perceived availability of social support operates as a stress buffer.

The particular focus of this chapter has been stress-buffering effects. We have discovered that when a perceived availability of social support measure is used, these effects reliably occur in the prediction of psychological and physical symptoms.³ Moreover, perceived support is also related to success in quitting smoking and maintaining abstinence, especially in the first three months after quitting. In both the symptom and smoking behavior data, the effects are primarily attributable to appraisal and self-esteem support—two types of support that are presumed to be effective in the face of a wide range of stressors.

Work on possible artifactual explanations for these effects suggest that they are robust in the face of controls for such artifacts. They are not explicable in terms of social skills or other obvious personality variables. Moreover, perceived support is not a grossly biased estimate of available support but rather provides at least a rough representation of the support available in the social environment.

The role of social integration in producing main effects on health has been discussed only in the context of the study of susceptibility to cold viruses. Preliminary data from this study provide evidence for a number of pathways through which social integration might influence disease susceptibility. Social integration is positively associated with a number of beneficial psychological states and with engaging in health-promoting behaviors. Moreover, social integration is also associated with cellular immune modulation that might provide a more direct (than health behaviors) biologic explanation for the link between social integration and disease susceptibility.

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³This assumes appropriate sample, adequate sample size, an influence of stress on the health outcome, and reliable instruments (Cohen & Wills, 1985).

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