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**Does Attachment Style Influence the Effects of
Social Support on the Stress-Illness Relationship?**

By

Matthew Paul Novak

A THESIS

**Submitted to
Michigan State University
in partial fulfillment of the requirements
for the degree of**

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ABSTRACT

Does Attachment Style Influence the Effects of Social Support on the Stress-Illness Relationship?

By

Matthew Paul Novak

This prospective study used path analysis to examine the effects of attachment style on the stress-illness relationship as it acts through social support. The subjects were 268 undergraduate students who received class credit for their participation. It was found that two factors of attachment, anxiousness about and avoidance of relationships, did have a negative impact both on the size of and satisfaction with a person's social support network. Social network size was a significant predictor of stress at Time 1, thus supporting a direct effects model. Physical symptoms at Time 1 were also found to predict stress. However, the only significant predictor of symptoms at Time 2 (4-6 weeks later) was symptoms at Time 1. The moderating effects of social support on the stress-illness relationship were not supported by the results of this study.

DEDICATION

To Karen,
my wife and my best friend,
without whose love, support, and understanding
this work would not have been possible.

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Stress and Physical Illness

Introduction

The relationship between mind and body has been pondered at least since the time of Hippocrates and his humoral theory. However, scientific theories about mind-body interactions and the testing of those theories are fairly recent developments.

In 1929 Walter Cannon produced some of the first scientific evidence in support of the relationship between mind and body. He labeled this the "Flight-or-Flight" response, and discovered that when psychologically stressed, organisms show physiological responses.

In 1956, work in the area of mind-body connections was furthered by Hans Selye in his pioneering book *The Stress of Life*. Here Selye promoted his theory, the General Adaptation Syndrome, to explain the mind-body connection we so familiarly speak of today. The General Adaptation Syndrome consists of three stages. In the first stage, called Alarm Reaction, the experience of a stressor, a threatening situation, causes the mobilization of the body's resources. Blood pressure rises, epinephrine, norepinephrine, and cortisol are secreted by the endocrine system, heart rate increases, and blood flow to the skeletal musculature is increased. However, this level of arousal cannot be maintained for long without the organism being in crisis. The Stage of Resistance follows. In this second stage, the body attempts to adapt to the stressor. The level of arousal declines somewhat from that of stage one, but continues at a higher than normal rate. However, such a heightened state of arousal impairs the organism from resisting new stressors or attacks. At this stage the organism is susceptible to what Selye termed "diseases of

adaptation". Should the stressor continue beyond this point, the organism will pass into the Stage of Exhaustion, marked by a depletion of the body's energy reserves, extreme susceptibility to disease, and physiological damage. Thus, the effects of prolonged psychological stress could be detrimental to one's physical health.

In 1976, Cassel echoed Selye when he stated that "the microbial diseases most common in our communities today arise from the activities of microorganisms that are ubiquitous in the environment, persist in the body without causing obvious harm under ordinary circumstances, and exert pathological effects only when the infected person is under conditions of physiological stress" (p. 108). Since his address to the American Public Health Association, much research has attempted to answer the question of how psychological stress affects human health.

Selye (1956) defined stress as "the state manifested by a specific syndrome which consists of all the nonspecifically induced changes within a biologic system" (p. 54). While this biological definition suited his work, for the purposes of this paper, stress will be defined as the condition which results when a person perceives a discrepancy between the demands of a situation and the resources at his/her disposal (Sarafino, 1990).

Life events and physical health

Much of the research done to investigate the relationship between stress and physical health has focused on major life events - prominent occurrences in one's life which have been identified by consensus as stressful. This research has found that life events,

primarily negative life events, do have an adverse effect on physical health (Dienstbier, 1989). Such stress does not appear to be illness-specific (Gottlieb, 1985). Life events have been found to be related to such varying diseases as upper respiratory tract infections, allergies, increases incidence of hypertension, heart disease (Jemmott & Locke, 1984), throat infections, tuberculosis, and influenza (Minter & Kimball, 1978). The relationship also does not appear to be dependent upon the type of stressor, as the studies cited examined such disparate stressors as marital problems, death of a loved one, change in housing, and having a child out of wedlock.

Many of the early studies investigating the effects of stress on physical health did not report correlations between stress and health or illness. But when correlations were reported they ranged from $r = .12$ (Rabkin & Struening, 1976) to $r = .43$ (Kobasa, Maddi, & Puchetti, 1982), and were most often in the $r = .20$ s range. Such correlations were inconsistent and often of a low order. Furthermore, the studies which examined this relationship often used retrospective methods of data collection that rely on the memory of the subjects. Such methods of data collection are often subject to error and omission.

In addition, much of the early research in this area used self-report methods to collect data, which may have created biases in the results. Self-report data are problematic because people under stress may become more sensitive to physical symptoms and adopt the sick role without actually being ill (Minter & Kimball, 1978) and because people experiencing negative life changes may selectively

remember more negative events, such as being sick. Recent research has attempted to correct some of these problems.

For instance, to eliminate the subjective reporting of illness, Jacobs and Charles (1980) examined life events in the families of 25 children with cancer and 25 other children brought to a clinic. The families of the children with cancer reported experiencing significantly more life events prior to diagnosis than did the controls. This study improved on the efforts of its predecessors, but it is possible that the life events may have been preceded by the onset of the disease and therefore were the result of the children's illnesses rather than the cause. And again, the retrospective nature of the data collection may have skewed the results in favor of a significant finding.

The most powerful studies have been prospective and have used objective measures to assess health. For instance, Funch and Marshall (1983) obtained stress data from 352 women with breast cancer, and found that stress was a significant predictor of survival 20 years later. But even studies such as these have produced mixed results. This may be due in part to studies which have looked at single major life events.

Cohen and Wills (1985) suggested that "although a single stressful event may not place great demands on the coping abilities of most persons, it is when multiple problems accumulate, persisting and straining the problem-solving capacity of the individual, that the potential for serious disorder occurs" (cf. Wills & Langner, 1980). Another explanation may be seen in the Illness Onset model of disease. Proposed by Dorian and Garfinkel (1987), this model

suggests that acute stressful events do not have a primary effect on one's health, but rather increase one's susceptibility to becoming ill at a certain point in time. This is based on the belief that adaptation is necessary to effectively cope with life change and that a lack of adaptation may lead to alterations in one's immune functioning. In their review of literature, Dorian and Garfinkel (1987) note a study done by Dorian et al. (1985) in which immune *enhancement* followed the onset of a stressful situation (unemployment). It was not until the situation became prolonged (chronic) that a decline in immune function was noted.

Daily Events and Physical Illness

DeLongis, Coyne, Dakof, Folkman, and Lazarus (1982) suggested that major life events may not cause stress in an individual's life directly but may instead be stressful due to the way in which they impact day-to-day living. They have proposed that life events are "distal", that they have lost "specific functional significance" for the individual, and therefore are described in nonpsychological terms (DeLongis et al., 1982). Therefore, everyday disruptions in living caused by major life events, called "hassles", would correlate more highly with measures of health than major life events because hassles are "proximal" events which often consist of "person-environment interactions" that are particularly salient to an individual and his/her well-being. The data supported their hypotheses.

DeLongis et al. (1982) found correlations between the frequency of hassles and health status ranging from -.21 to -.33, with

the intensity of the hassles also correlating significantly with health status ($r = -.29$ to $-.38$). The only significant correlation reported between life events and health status was between prestudy life events and health status at the end of the study ($r = -.26$). This suggests that there is a delayed effect of stress on health. In order to support their hypothesis that major life events affect health through hassles, a multiple regression analysis was conducted. If hassles were entered first, they accounted for 13% of the variance in overall health status, with major life events not adding any significant variance beyond this. And when life events were entered first, they accounted for 7% of the variance, but hassles added an additional 9% of the variance to health status. The analysis of the relationship between hassles, life events, and somatic symptoms produced similar results. Hassles were significantly correlated with symptoms ($r = .27$ to $.35$), but life events were not. And a hierarchical multiple regression demonstrated that hassles accounted for 13% of the variance, with life events not adding significantly to this association. When life events were entered first, they accounted for only 0.4% of the variance, with hassles contributing an additional 14%. While such results seem to lend overwhelming support to the hypothesis in question, the study was retrospective. The authors therefore suggest caution in interpreting their findings because it is possible that one's health status may contribute to the hassles one reports.

Since this pioneering work, others have found evidence to support its findings (Cox et al., 1984; Gannon, Banks, Shelton, & Luchetta, 1989; Gannon, & Pardie, 1989; Holahan, Holahan, & Belk, 1984 ; Weinberger, Hiner, & Tierney, 1987) with correlations

between hassles and symptoms ranging from .19 to .64. However, some have found evidence contradicting this relationship between minor everyday events and physical health.

Evans, Pitts, and Smith (1988) had 30 college students complete the Assessment of Daily Experiences and a symptom checklist each day for nine weeks. They found that there was a significant decline in desirable events in the four days preceding an illness. However, the subjects of their study reported no significant change in undesirable events in the same time period. The authors suggest that it is the "absence of uplifts rather than the presence of hassles [which] seems to be the danger signal" (p. 539) prior to an illness episode. Similar results have been obtained by others (Stone, Bruce, & Neale, 1987; Zarski, 1984), but the preponderance of evidence appears to support the notion that chronic stressful events such as daily hassles are correlated with physical symptomology.

Introduction

The modest findings of stress' effects on physical health have led psychologists to search for other factors that may be involved in the process. In 1976, John Cassel stated that "it is evident that any disease process, and in fact any process within the living organism, might be influenced by the reaction of the individual to his social environment or to other people" (p.109). The area of social support seemed to promise to explain some of the inconsistencies in stress-illness research. This section will present research exploring social support's effects on the stress-illness relationship.

For the purposes of this discussion, social support will be defined as "1. Information leading the subject to believe that he [sic] is cared for and loved. 2. Information leading the subject to believe that he is esteemed and valued. 3. Information leading the subject to believe that he belongs to a network of communication and mutual obligation." (Cobb, 1976, p.300)

In their pioneering work Berkman and Syme (1979) looked for a connection between social support and illness. They examined 4725 people (2229 men and 2496 women) who had previously taken part in the 1965 Human Population Laboratory survey in Alameda County, California. The size (a measure of structure) of each subject's social support network at the time of original data collection was negatively related to mortality during the following nine years. After adjusting for age and prior health status, men with low levels of social support were 2.3 times more likely to die than those with high social support. For women the increased risk was 2.8. The

results remained significant at the $p < .001$ level even after simultaneously controlling for smoking, obesity, alcohol consumption, physical activity, and age. Berkman and Syme also suggested that since ill people with poorer social networks did not die in the first few years of this follow-up study, then physical illness could not account for all of the connection between social isolation and mortality.

While the Alameda County Study was ambitious in its sheer size, it had many flaws which have since been addressed by other studies. For instance, while it was prospective in nature, it assumed that the data collected at the beginning of the study remained consistent for the subjects during the nine subsequent years. Also, there was poor control for factors such as physical health at the outset of the study.

House, Robbins, and Metzger (1982) corrected some of these flaws in their study of 1322 men and 1432 women. They collected social integration and medical examination data during a ten year period (1959-1969). Mortality from 1978-1979 was the dependent variable. A wide variety of factors including prior health status, lifestyle, and SES were controlled. Cumulative indices of social relationships and activities were significant predictors of mortality for men. For women, however the significant relationship between cumulative indices of social integration became nonsignificant when other factors were entered as covariates. They found no evidence to support the proposition that satisfaction with relationships and activities has a significant impact on mortality. Prior health status did not appear to be related to social support. That is, those who

were ill did not necessarily have lower scores on social support indices.

In addition to mortality studies, decreases in social support have also been associated with increases in physical symptoms (see Broadhead et al., 1983 for a detailed review). High levels of social support have been related to lower risks for heart disease (Cohen, 1988; Siegrist, Siegrist, & Weber, 1986), decreases in low birth weight, and better recovery from tuberculosis, asthma, heart disease, and surgery (Cobb, 1976). The effects of social support do not appear to be stressor specific, as social support has been noted to be beneficial in alleviating the effects of a wide variety of stressors such as unemployment, bereavement, and retirement (Cobb, 1976). But there are enough negative findings to "make it clear that social support is not a panacea" (Cobb, 1976, p. 310).

Poor social support may lead to anxiety and depression, which may affect health through increasing susceptibility to disease, or indirectly through changes in behavioral patterns that increase one's risk for disease (Cohen & Wills, 1985). It has been suggested that there are different types of social support including esteem support, informational support, social companionship, and instrumental support (Cohen & Wills, 1985). However, the four types are often so highly correlated that the distinction between them becomes unimportant (Caldwell & Reinhart, 1988; Stokes & Wilson, 1984). But, the amount, type, source, and structure of social support are all important dimensions in the defining the effectiveness of one's social support network (Thoits, 1982).

Research in the area of social support has led to the proposal of two models of social support: (a) the buffering model and (b) the main effect model (Cohen & Wills, 1985). The main effects model holds that social support is beneficial regardless of whether one is stressed, while the buffering model suggests that support only works for people while they are under stress. In general "evidence for a buffering model is found when the social support measure assesses interpersonal resources that are responsive to the needs elicited by stressful events. Evidence for a main effect model is found when the support measure assesses a person's degree of integration in a large community social network" (pp.347-348). Put more simply, the buffering model is usually supported when one examines one's satisfaction with his/her support system, and main effects generally found when measuring the structure of one's support network.

Direct Effects of Social Support

One way in which social support eases the effects of stress on one's body is by protecting the individual from experiencing stress in the first place. This model is tested by examining the effects of social support on symptoms without taking into account the level of stress experienced by the subject. It has been suggested that it is the structure of one's support system which best demonstrates the main effects model (Cohen & Wills, 1985). That is, the better the structure (a measure which may include dimensions such as size, density, and integration) of one's network, the fewer symptoms one is likely to experience.

The main effects model of social support was supported in a study by Gore (1978). She compared 100 unemployed men to 74 employed controls over a period of two years to determine the effects of social support in alleviating the effects of the stress on physical health. Men with less support who became unemployed reported more symptoms of illness than men who were more supported or who remained employed. More important in light of the main effects model was that "the well supported report[ed] lower mean levels of symptoms than [did] the quickly reemployed" (p.162). However, the author cautions that since the study did not include adequate baseline levels (the study was started during anticipation of unemployment, a stressor in itself), the results are equivocal.

Monroe (1983) corrected this error in design. He collected life events and physical symptom data from seventy employees of a corporation. While retrospective analysis showed that social support was not significantly related to physical symptoms, prospective analysis showed that support was a significant predictor of subsequent symptoms regardless of stress level.

However "if social support [is] to be considered [an] independent predictor of disorder, [it] should demonstrate an association with subsequent symptoms once initial symptoms have been taken into account" (Monroe, 1983, p.83). Monroe (1983) found evidence to support this. Social support continued to contribute significantly to the variance in physical symptoms even after prior physical status was taken into account. This adds further support to the main effects model.

According to Cohen and Wills (1985) the main effect model may work because "large social networks provide persons with regular positive experiences and a set of stable, socially rewarded roles in the community" (p.311) thus providing a "positive affect, a sense of predictability and stability in one's life situation, and a recognition of self-worth" (p.311), or by decreasing stressful experiences. Such factors may affect the immune or neuroendocrine systems, or may influence one's health-related behaviors.

While there is some evidence to support a main effects model, there is also evidence to suggest that the quality of one's social support network is a better predictor of health than the structure of that network (Broadhead et al., 1983). "When structural measures of social network size are used to indicate the benefits of social relationships, two questionable assumptions are made, namely, that any benefits are directly proportional to the size and range of the network and that having a relationship is equivalent to getting support" (Schaefer, Coyne & Lazarus, 1981, p.383).

Social Support as a Moderator

The buffering effect of social support occurs after a stressful event has been experienced and is evident when there is an interaction between levels of stress and levels of social support. That is, people with different levels of support may report the same number of symptoms when stress levels are low, but when stress levels are high, those people with more social support report fewer symptoms than those with low levels of support. The buffering effect is most frequently noted when an experimenter is measuring

the functioning of the social support (e.g. satisfaction with social support) rather than size or structure. Often such measures involve satisfaction with a small subset of one's social network such as a confidant, spouse, or family (Cohen & Wills, 1985).

A study conducted in Finland found that high school students who had reported both a high level of life events and poor relationships with their families and friends reported more symptoms than students with good relationships or low levels of stress (Aro, Hanninen, & Olavi, 1989). However, this relationship was found to be significant only for boys.

Similar effects were found in a study of residents of Three Mile Island (TMI) after the nuclear accident that happened there. TMI residents with moderate or high levels of social support had symptom levels comparable to control subjects with similar support (Fleming, Baum, Gisriel, & Gatchel, 1982). Buffering effects have also been implicated in research investigating a variety of health conditions including herpes simplex virus (VanderPlate, Aral, & Magder, 1988), and coronary heart disease (reviewed by Cohen, 1988).

However, these studies were done retrospectively and therefore the results are questionable. But such effects have also been noted in prospective research. For example, Monroe (1983) found that subjects with initially high levels of symptoms and social support tended to experience fewer symptoms during follow-up. High levels of life events and low levels of social support together were most predictive of high levels of physical symptoms, while high

support among those who experienced large numbers of life events demonstrated the buffering effect.

Similar results were also found by Cohen and Hoberman (1983). Using prospective measures of support they found support for the buffering model. Interestingly, they found that when they used retrospective measures, they did not find similar support. The authors suggested that retrospective measures may not be good measures of social support because they may tap a person's prior need for support as well as its availability.

There is some evidence to suggest that social support may reduce psychological stress, but does not do particularly well in reducing physiological arousal to stress (Fleming, Baum, Gisriel, & Gatchel, 1982). But support for the buffering model has also been found in research which used objective measures of symptoms (e.g., Fontana, Kerns, Rosenberg, & Colonesse, 1989; Norbeck & Tilden, 1983; Sarason, Sarason, Potter, & Antoni, 1985).

The buffering effects of social support may occur by (a) preventing a "stress appraisal response" (i.e., may allow a person to believe that s/he has sufficient resources to handle the stressor due to social support's added resources) or (b) by reducing or eliminating a stress reaction after its onset (through providing a solution to the stressor, reducing the importance of the event, or by encouraging healthful behaviors) (Cohen & Wills, 1985).

Possible Extraneous Variables

It should be noted that there may be some other variables which affect the stress-illness relationship. A study conducted by

McFarlane, Norman, Streiner, and Roy (1983) found that the number of neutral or undesirable events experienced were associated with physical health when measured concurrently. But they proposed that "while stressful events may be related to consistent patterns of health, they may not predict *change* in health. Alternatively, individuals suffering from poor health may, as a result, experience an increase in stressful events" (p. 165). After controlling for prior health, they found that stressful events which occurred in the previous six months were not related to current health, although there was still a significant relationship between current life events and current illness. Prior illness was significantly correlated with current life events. They concluded that "the more immediate effect of stressful events on health has been considerably over-estimated" (p. 169), and offer three possible explanations for their finding that stress is related to subsequent health problems: (a) Stressful events may lead to later health problems, (b) symptoms may lead to higher levels of stress rather than the other way around (but they note that this does not explain the baseline level of stressful events reported by the subjects of this experiment), and (c) some third factor, such as personality, may cause both stressful events and poorer health.

Other possible extraneous variables are found under the heading of lifestyle. People have good and bad habits which affect health and may be unrelated either to attachment style or social support. The most well known of these is diet.

A balanced diet consisting of meats, cereals, vegetables, fruits, and dairy products is important to maintaining good health (Katch & McArdle, 1993). Poor diet is associated with many health problems

including cardiovascular disease (Havel, 1989), hypertension (Kaplan, 1989), diabetes (Wolever & Jenkins, 1989), and cancer (Sugimura, Wahabayashi, Nagao, & Ohgaki, 1989). Exercise also seems to promote good health, apparently through improving immune system functioning (Keast & Morton, 1992; Verde, 1992).

In addition to behaviors which may improve a person's health, there are behaviors which may lead to an increased incidence of illness. Alcohol abuse is correlated with various health problems including nervous system disorders (Bigby, 1987), liver disease (Moulton & Cyr, 1987), gastrointestinal problems (Cyr & Moulton, 1987), and cardiovascular disease (Ende, 1987). Likewise, people who abuse drugs tend to have a higher incidence of illness than those who do not use drugs (Andersen, 1981).

Why Social Support May Not Always Be Of Help

While there has been much research supporting social support's beneficial effects on health, many criticisms have been raised surrounding this research. It has been proposed that people with strong networks may be more prevalent in studies which examine such disorders such as coronary artery disease because their social networks will encourage them to seek treatment (Cohen & Matthews, 1987), thus suggesting that people who most need social support receive it.

One study found that while those with higher levels of social support had less trouble with their ability to carry out ordinary daily activities and maintain relationships than the others, when physical status and health were entered first, social support did not

contribute significantly to the variance of this level of functioning (Lee, Graydon, & Ross, 1991). Similar results have been found in other studies (Cox et al., 1984; Gannon & Pardie, 1989; Schaefer, Coyne, & Lazarus, 1981).

One possible explanation for negative findings in the area of social support was reported by Cohen and Hoberman (1983). Although demonstrating social support's buffering effect, they found a decrease in symptoms for those with low stress and low levels of social support. This is not typical of evidence supporting the buffering model. They hypothesized that "the increased responsibilities ... of the interpersonal relationships ... may themselves contribute a small increment to one's stress level and consequently in symptomatology" (p.116).

Schaefer, Coyne, and Lazarus (1981) agree with Cohen and Hoberman. They state that the assumption that network size and amount of social support are positively associated "ignores the demands, constraints, and conflicts also associated with social relationships", which "comprise a significant share of the stresses people experience in their daily lives" (pp. 383-4). They further propose that these "demands and constraints of network membership may dilute or vitiate the beneficial effects, and so weaken any relationship with health outcomes" (p.385). Such social interaction would also be harmful if such interaction were improper and would thus promote illness rather than health (Orth-Gomér & Undén, 1987).

One study (McFarlane, Norman, Streiner, & Roy, 1983) found an inverse relationship between stressful events and social supports.

The authors suggested that the size of one's social network may be related to the perception of it being less helpful. Another possibility is that increases in stressful events lead to the perception that one's social network is less helpful.

But there is some evidence to refute such ideas. Fontana, Kerns, Rosenberg, and Colonese (1989) tested the hypothesis that illness, distress, and stress mobilize one's social support system to action against the hypothesis that illness decreases support. The data revealed no significant interactions. Therefore, there was no support for buffering or moderating effects. They did, however, find that intimacy ameliorated psychological distress and physical symptoms, thus supporting the main effects model.

It may be that the positive health effects of social support are countered by other factors. One's social network may decrease in size because the demands of illness restrict the amount of time and energy some people have to spend with the members of their networks (Bloom, 1990). Or disease may decrease social support because the sufferer overuses the system while coping with his/her illness (Cohen & Matthews, 1987).

Finally, some people may be better able to use their social support systems when faced with a stressful situation. Research in the area of attachment theory seems to point in this direction.

Attachment and Symptoms

In 1958 John Bowlby proposed a theory explaining interpersonal behavior. This theory stated that very young children develop behaviors which maintain proximity to their primary caregiver. Bowlby called these behaviors attachment behaviors. Over time, the child develops internal representations of the relationship with his/her caregiver. And eventually they become a pattern of expectations for all interpersonal interactions which color the individual's interactions with others. The tone of the patterns of expectations is based on the success or failure of these behaviors and the responsiveness of the caregiver.

Mary Ainsworth and her colleagues (1978) furthered Bowlby's work and found three broad patterns of infant attachment to caregivers. The first pattern has been termed secure attachment. Those in this category appear to experience his/her primary caregiver as accessible and responsive, and are characterized by being easily consoled when distressed, compliant and cooperative, outgoing, enthusiastic, positive, and persistent. Children displaying the second pattern, the anxiously or ambivalently attached, tend to have a less consistently positive experience of their caregivers, and are characterized as anxious, clingy, easily frustrated, dependent, passive-aggressive, and are not as easily soothed as secure children. Finally, the avoidantly attached often experience his/her primary caregiver as rejecting, and tend to be angry, aloof, and stand-offish.

Research over the past decade has supported these models. Infants labeled secure children have shown a greater ability to respond flexibly, persistently, and resourcefully in a preschool

setting (Arend, Gove, & Sroufe, 1979; Sroufe, 1983) and it appears that avoidant children cut off anger or distress-related emotions (Kobak, 1986; Lutkenhaus, Grossmann, & Grossmann, 1985). Thus, experiences which lead to internal representations of others who are unavailable lead to dysfunctions of attachment behavior (Sheldon & West, 1990).

It is believed that these patterns "will be persistent and resistant to change" (Mrazek, Casey, & Anderson, 1987, p. 516). There is research supporting this assertion. A longitudinal study done in Australia demonstrated a correlation between early attachment experiences with parents and later marital quality (Kotler & Omodei, 1988).

Simpson, Rholes, and Nelligan (1992) stated that:

The utility of Bowlby's attachment theory for adult relationships, however, does not require that attachment styles observed in adulthood date back to infancy. As long as the patterns of attachment identified in children are phenotypically similar to those that characterize adults, and as long as the consequences of these styles for behavior and emotions are similar across different developmental levels, attachment theory remains a viable model for understanding adult relationships (p. 443).

Research suggests that securely attached adults tend to report little distress and high levels of social support (Kobak & Sceery, 1988). They feel valued and worthy of others' concern, support, and affection, and develop mental models of significant others who are accessible, reliable, trustworthy, and well-intentioned (Simpson,

Rholes, & Nelligan, 1992). Securely attached adults tend to express trusting attitudes towards others (Feeney & Noller, 1990) and to be involved in romantic relationships marked by higher levels of satisfaction (Simpson, 1990).

Avoidant individuals picture themselves as aloof, and emotionally distant, feel uncomfortable being around others, and find it difficult to completely trust and depend on others (Simpson, Rholes, & Nelligan, 1992). They report more distant relationships (i.e., more loneliness) and lower levels of social support than the other two groups (Kobak & Sceery, 1988).

Ambivalently attached persons report higher levels of distress than the securely attached (Kobak & Sceery, 1988), express dependence (Feeney & Noller, 1990), and feel that they are misunderstood and underappreciated (Simpson, Rholes, & Nelligan, 1992). While they view their social network as more supportive than the avoidantly attached (Kobak & Sceery, 1988), they believe that others are reluctant to get as close as they wish (Simpson, Rholes, & Nelligan, 1992).

Bowlby (1969) has argued that an individual's attachment system should be most strongly activated under conditions of distress. There have been several studies supporting this assertion. For example, Simpson, Rholes, and Nelligan (1992) found that securely attached women were more likely than avoidant women to seek out support from their romantic partners when made to feel anxious. However, no significant effects were found for the anxious attachment style. Therefore,

secure attachment would be organized by rules that allow acknowledgment of distress and turning to others for support, avoidant attachment by rules that restrict acknowledgment of distress and the associated attachment attempts to seek comfort and support, and ambivalent attachment by rules that direct attention toward distress and attachment figures in a hypervigilant manner that inhibits the development of autonomy and self-confidence (Kobak & Sceery, 1988, p. 142).

Simply stated, because securely attached people are relatively comfortable with social relationships, they tend to have large social networks and tend to be more satisfied with their networks.

Alternately, those people who are ambivalently or avoidantly attached are less comfortable with social relationships, they tend to have small networks and report low levels of satisfaction.

There is also evidence to indicate that attachment style is correlated with physical illness. Mrazek, Casey, and Anderson (1987) found that asthmatic children were more likely to be insecurely attached than children without asthma. It is possible, therefore, that attachment style moderates the effects of stress on physical health through its effects on how one develops and uses his/her social support system.

This study was designed to examine the effects of social support on the relationship between stress and physical illness and how attachment style might influence these effects. The following hypotheses were tested:

Hypothesis 1: The amount of stress one experiences causes the amount of physical illness experienced, after accounting for lifestyle and prior illness.

Hypothesis 2: Given the same amount of stress, those persons with relatively higher levels of social support experience less physical illness than those with relatively lower levels of social support. The effects of social support will be evident in two ways: One, people with a higher number of people in their social support networks will show less stress (i.e. direct effects model of social support) and, two, people who are more satisfied with their social support networks will have a smaller number of physical symptoms even though their stress levels may be similar to those who are unsatisfied with their networks (i.e. moderating model of social support).

Hypothesis 3: A person's level of secure attachment is positively correlated with the size of his/her social network as well as with the level of satisfaction with the support received from said network. Alternately, one's level of anxious and avoidant attachment (as defined by Hazan and Shaver, 1987) is negatively correlated with a person's social network size and the satisfaction received from that network.

Hypothesis 4: This hypothesis proposed the path model depicted in Figure 1.

Based on the literature reviewed in this paper, there is a correlation between stress and health, social support can have a direct effect on illness, or it can moderate the stress-illness relationship, and attachment style appears to be related to health

and social support. Therefore, the above model purports that, after accounting for prior illness and lifestyle factors, attachment will moderate the relationship between stress and physical illness by acting through social support.

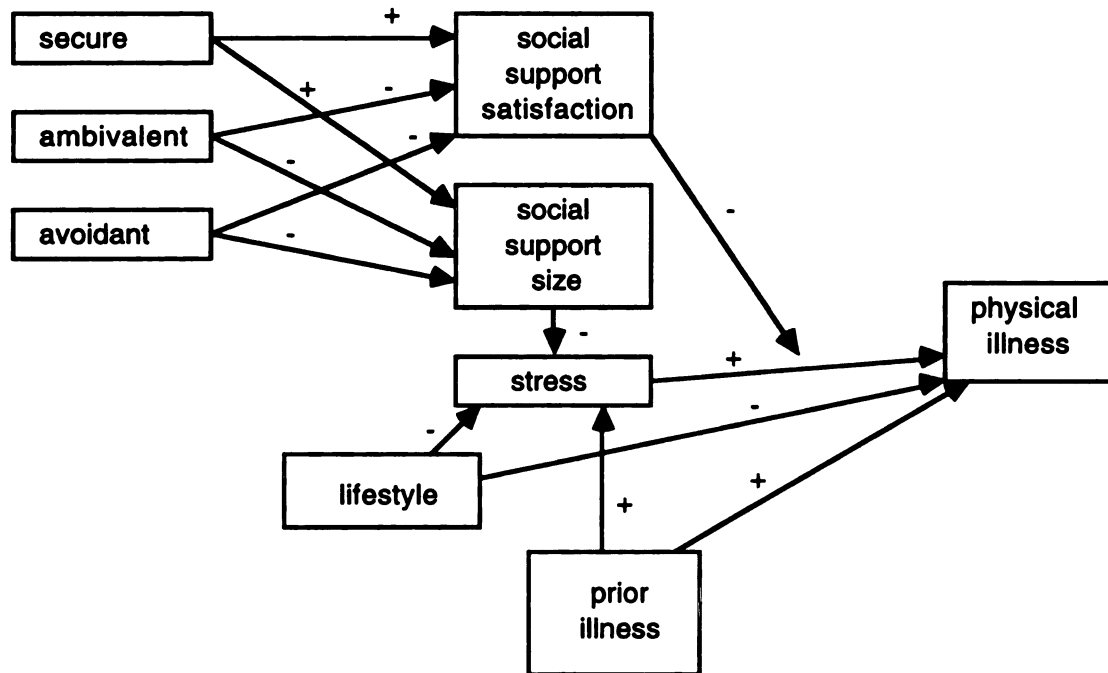


Figure 1. A diagram of the path model which was proposed for this study. (The plus or minus sign next to each proposed path indicates the expected direction of that relationship.)

Subjects

The subjects for this experiment 304 undergraduates enrolled in introductory psychology classes at Michigan State University. In exchange for their voluntary participation, they were given class credit and were debriefed as to the nature of the experiment after they completed the questionnaires. Twenty-one did not return for the second testing session. In addition, fifteen surveys were incomplete, and, due to the nature of the analysis, these data were not included in the analysis. The final number of valid protocols was 268; 105 males and 163 females.

Procedure

The subjects were tested at two times. The second testing occurred four to six weeks after the first time. At Time One they were asked to complete a lifestyle assessment questionnaire, the Hassles scale, the Social Support Questionnaire, the Relationship Style Questionnaire, and the Cohen-Hoberman Inventory of Physical Symptoms. At Time Two they were asked to complete again the lifestyle assessment questionnaire, and the Cohen-Hoberman Inventory of Physical Symptoms.

Materials

The lifestyle assessment questionnaire is a 34-item self-report measure derived from the Olin Health Center Lifestyle Assessment questionnaire. It was designed to assess various lifestyle factors such as eating, exercise, smoking, and self-care habits, as well as

alcohol and drug use patterns and sexual health. The subject responds to questions on a five-point Likert scale. This measure was used to control for lifestyle factors which may have influence the stress-illness relationship. A lifestyle rating was obtained by adding the responses for each question with a higher score indicating a more healthful lifestyle. For this sample alpha reliability was .82 and test-retest reliability was .87.

The Hassles Scale (Kanner, Coyne, Schaefer, & Lazarus, 1981) is a 117-item self-report measure which assesses the "frequency and severity of a person's transactions with the environment that are considered by the person to be stressful events" (Lazarus & Folkman, 1989, p.1). In this experiment it measured perceived stress by summing up the severity for all items endorsed. The authors of the Hassles Scale reported test-retest reliability for severity was .48, which is an acceptable level for a state measure. An alpha reliability analysis was done using the sample chosen for this study and was found to be .96. The scale also appears to have face and content validity (Lazarus & Folkman, 1989).

The Social Support Questionnaire (Sarason, Levine, Basham, & Sarason, 1983) is a 27-item self-report measure which asks the respondent for the number of people to which s/he can turn in a variety of situations. Each situation also includes a 6-point Likert scale which asks about the subjects satisfaction with his/her support system. Overall number and satisfaction scores were computed by summing the number and satisfaction portions of each item, respectively. Test-retest reliability with an undergraduate

population was .90 for number and .83 for satisfaction (Sarason, Levine, Basham, & Sarason, 1983).

The Relationship Style Questionnaire (Bartholomew, 1990) is a self-report measure which is based on a three previously designed measures: The three-item measure by Hazan and Shaver (1987) divided into its constituent statements, the four-style Relationship Questionnaire (Bartholomew & Horowitz, 1991), again, divided into its constituent statements, and Collins and Read's (1990) Adult Attachment Scale. This questionnaire asks the subject to rate on a 5-point Likert scale 30 statements as to how "like me" each is. Originally it was intended that each subject would receive a score for each of Hazan and Shaver's attachment categories by adding their scores on statements corresponding to each category. However, it was demonstrated that this method was undesirable. Although the alpha reliability for the Ambivalent and Avoidant categories was adequate (.71 and .72, respectively), reliability for the Secure scale was only .60. Since others (Simpson, 1990; Simpson, Rholes, & Nelligan, 1992) have reported that a factor analysis of similar scales resulted in two factors, a forced two-factor exploratory factor analysis was done. The factor analysis resulted in two factors which closely resembled what have been labeled by others as Anxiousness and Avoidance (Simpson, 1990; Simpson, Rholes, & Nelligan, 1992). After modifying the factors to more closely fit these labels, alpha reliability was .85 for Anxiousness and .81 for Avoidance. The items contained in each factor are presented in Table 1. Hypothesis 3 was, therefore, restated to take into account this change in factors: A person's level of anxiousness about and avoidance of close

relationships is correlated with the size of his/her social network as well as with the level of satisfaction with the support received from said network. That is, the higher one's levels of anxiousness and avoidance, the smaller the person's network and the less satisfied they will be with said network (see Figure 2).

Finally, the Cohen-Hoberman Inventory of Physical Symptoms (Cohen & Hoberman, 1983) is a 33-item self-report measure which asks the subject how much each item bothered or distressed the subject. Items are rated on a 5-point Likert scale ranging from "not at all" to "extremely". Reliability for this measure was .90 which is similar to what Cohen and Hoberman found ($\alpha = .88$). Test-retest reliability was .66 for the sample used in this study. A physical symptom score was derived by adding the responses from all items. Therefore, a higher score indicated more symptoms reported.

Table 1

Two factor solution with factor loadings. (An * indicates that this item was moved from the other factor. A § indicates that the item was deleted from the factor to increase reliability. An "r" indicates an item which is reverse scored.)

RSQ Item	Factor 1	Factor 2
Factor 1: Anxiousness		
184. I often worry that romantic partners won't want to stay with me.	.77	.13
186. I worry about being abandoned.	.72	.04
174. I often worry that romantic partners don't really love me.	.66	.25
172. I worry about being alone.	.66	-.01
179. I worry that others don't value me as much as I value them.	.65	.16
181. My desire to merge completely sometimes scares people away.	.60	-.17
188. I find that others are reluctant to get as close as I would like.	.57	-.05
191. I worry about having others not accept me.	.54	.04
180. People are never there when you need them.	.52	.35
168. I worry that I will be hurt if I allow myself to become too close to others.	.52	.38
§167. I want to merge completely with another person.	.49	-.40
*170. I am not sure that I can always depend on other to be there when I need them.	.41	.43
*r190. I know that others will be there when I need them.	.18	.56

Table 2 (continued)

Two factor solution with factor loadings. (An * indicates that this item was moved from the other factor. A § indicates that the item was deleted from the factor to increase reliability. An "r" indicates an item which is reverse scored.)

RSQ Item	Factor 1	Factor 2
Factor 2: Avoidance		
r193. I find it relatively easy to get close to . others	.01	.65
r173. I am comfortable depending on other people.	.15	.62
164. I find it difficult to depend on other people.	.30	.61
183. I am nervous when anyone gets too close to me.	.38	.58
189. I prefer not to depend on others.	.11	.56
r166. I find it easy to get emotionally close to . others	-.15	.54
176. I worry about others getting too close to me.	.29	.53
175. I find it difficult to trust other completely.	.45	.52
r177. I want emotionally close relationships.	.40	-.52
187. I am somewhat uncomfortable being close to others.	.33	.47
192. Romantic partners often want me to be closer than I feel comfortable being.	.09	.47
r171. I want to be completely emotionally intimate with others.	.42	.46
165. It is very important to me to feel independent.	-.05	.42
169. I am comfortable without close emotional relationships.	-.21	.33
r178. I am comfortable having other people depend on me.	.01	.33
182. It is very important to me to feel self- sufficient.	-.01	.26
185. I prefer not to have other people depend on me.	.26	.26

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Results

The means and standard deviations for each measure are listed in Table 2 and the correlations between variables are listed in Table 3.

Hypothesis 1

The mean scores for Symptoms at Time 1 (SX1) and Symptoms at Time 2 (SX2) were 56.28 (s.d. =17.15) and 59.14 (s.d. = 22.33), respectively. The mean for Stress (STR) was 188.41 with a standard deviation of 40.53. The mean score for Lifestyle at Time 1 (LFS) was 175.32 with a standard deviation of 14.98.

Table 2

Means and standard deviations for all variables.

<u>Variable</u>	<u>Mean</u>	<u>S. Dev.</u>
Anxiousness	31.97	9.65
Avoidance	46.80	10.32
SS Satisfaction	140.01	18.56
SS Number	104.59	39.57
Lifestyle T1	175.24	14.95
Stress	188.41	40.53
Symptoms T1	56.28	17.15
Symptoms T2	59.14	22.33

Multiple regression on SPSS was used to test Hypothesis 1. Symptoms at Time 2 was the dependent variable with Stress, Lifestyle at Time 1, and Symptoms at Time 1 entered as predictor variables. SX1 was significantly positively related to SX2 ($\beta = .62$, $p < .05$). However, while both LFS and STR were significantly correlated with SX2 when either was taken in isolation ($r = -.25$, $p < .05$ and $r = .46$, $p < .05$; respectively), neither was significantly related to SX2 after

SX1 was accounted for ($\beta = -.09$ and $\beta = .04$, respectively). Therefore, the best predictor of Symptoms at Time 2 was Symptoms at Time 1. Thus, hypothesis 1 was not supported by these data.

Table 3

Correlation matrix for all variables (N=268). ($r > .12$, $p < .05$; $r > .16$, $p < .01$) (ANX=Anxiousness, AVO=Avoidance, SSN=Social Support Number, SSS=Social Support Satisfaction, LFS=Lifestyle at Time 1, SX1=Symptoms at Time 1, STR=Stress, SX2=Symptoms at Time 2)

	ANX	AVO	SSN	SSS	LFS	SX1	STR	SX2
ANX	1.00							
AVO	.37	1.00						
SSN	-.24	-.28	1.00					
SSS	-.35	-.28	.39	1.00				
LFS	-.31	-.12	-.01	.08	1.00			
SX1	.38	.17	.01	-.07	-.24	1.00		
STR	.45	.24	-.11	-.09	-.30	.63	1.00	
SX2	.29	.09	.01	-.02	-.25	.66	.46	1.00

Hypothesis 2

To test whether Social Support Number (SSN) had a direct and Social Support Satisfaction (SSS) had a moderating influence on the STR-SX2 relationship, SSN and SSS, along with SX1 and LFS, were entered into a path analysis using PATH (Hunter & Hamilton, 1992) (see Figure 2). The mean Social Support Number was 104.59 with a standard deviation of 39.57, and the mean Satisfaction with Social Support was 140.01 with a standard deviation of 18.56.

The direct effect of SSN on STR was not supported by the path analysis ($\beta = -.11$, $p > .05$), although the path was close to being significant. The moderator effect of SSS on the STR-SX2

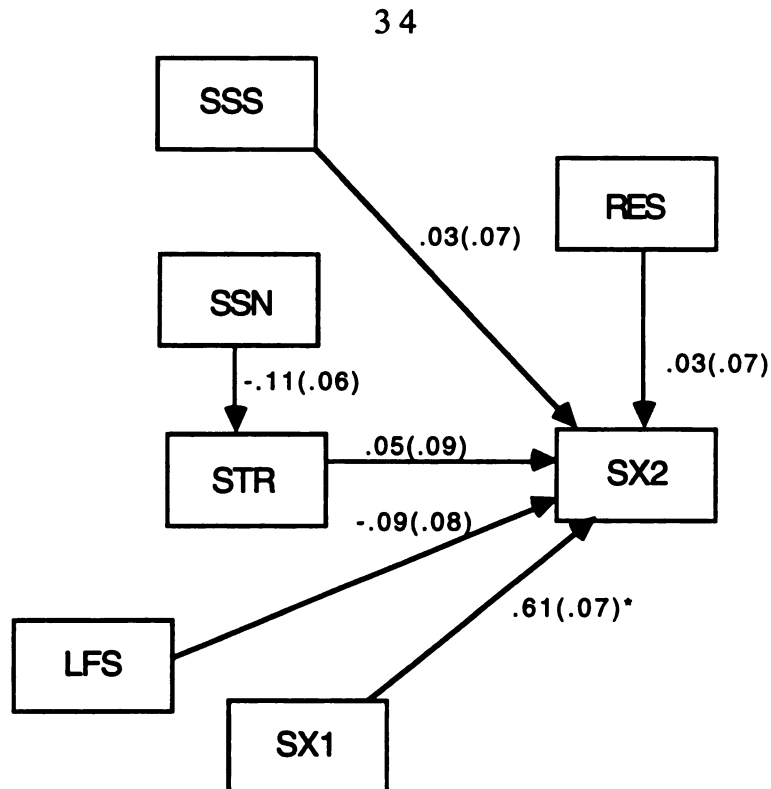


Figure 2. Model to test Hypothesis 2 with path coefficients. (Beta weights are given with standard errors in parentheses. * indicates $p < .05$. SSN= Social Support Number, SSS=Social Support Satisfaction, RES=residual term of STR*SSS, LFS=Lifestyle at Time 1, SX1=Symptoms at Time 1, STR=Stress, SX2=Symptoms at Time 2.)

relationship was tested by first identifying a path between SSS and SX2. Next, the product of the deviation scores of STR and SSS ($\Sigma(\text{STR} - \bar{\text{STR}})(\text{SSS} - \bar{\text{SSS}})$) was calculated using SPSS. Then, again using SPSS, STR and SSS were regressed onto the product mentioned above, and the residual was obtained. This procedure was undertaken to maintain the integrity of this new variable as a representation of the cross between STR and SSS while ensuring that it would not be correlated with either STR or SSS. Indeed, this was accomplished as the correlation between the resulting variable, RES, and the product of the deviation scores was .97 while there was no correlation between RES and STR or SSS ($r = .00$ in both cases). Finally, a path was set between RES and SX2. If SSS had a moderating effect, RES should have shown a significant effect on SX2 after the path between SSS and SX2 was taken into account (Baron & Kenney, 1986). Neither the path between SSS and SX2 nor that between RES and SX2 was significant ($\beta = .03$, $p > .05$ in both cases). Therefore, there was no support for the moderating effect of social support. However, it should be noted that the mean for Social Support Satisfaction was 140 out of a possible 162, which may indicate a ceiling effect. This may have attenuated the correlation between RES and SX2. Therefore, the only significant predictor of SX2 remained SX1 ($\beta = .61$, $p < .05$). SX1 also was significantly related to STR ($\beta = .63$, $p < .05$).

The overall Chi-square for the model in Figure 2 was 54.74 ($df = 5$), which gave it a tail probability of .000. This suggests that the model was a very poor fit to the data. Hypothesis 2 was not supported.

Hypothesis 3

For Hypothesis 3, Anxiousness (ANX) and Avoidance (AVO) were each regressed onto both Social Support Number and Social Support Satisfaction using SPSS. The mean for ANX and AVO were 31.97 (s.d.= 9.65) and 46.80 (s.d.= 10.32), respectively. Hypothesis 3 was supported by the data. SSN was significantly predicted by both ANX and AVO ($\beta = -.16$, $p < .05$ and $\beta = -.23$, $p < .05$, respectively), thus indicating that securely attached persons (people low on ANX and AVO) tended to have more people in their social networks. In predicting a subject's SSS, again, both ANX and AVO were significant ($\beta = -.28$, $p < .05$ and $\beta = -.17$, $p < .05$, respectively), suggesting that people who are securely attached are more satisfied with their social networks.

Hypothesis 4

In order to test hypothesis 4, the full path model was run using the Least Squares method in PATH. For each endogenous variable a formula was designed based on the variables in the model which were purported to be related to that variable. For example, Social Support Satisfaction was set to be equal to the effects of each of the two attachment factors. Reliability scores for each of the variables was used to derive standard errors for each of the path coefficients. The beta weights, standard errors, and significance levels are shown in Figure 3.

There were only minor variations between the path coefficients in the full path model and the beta weights reported earlier in the

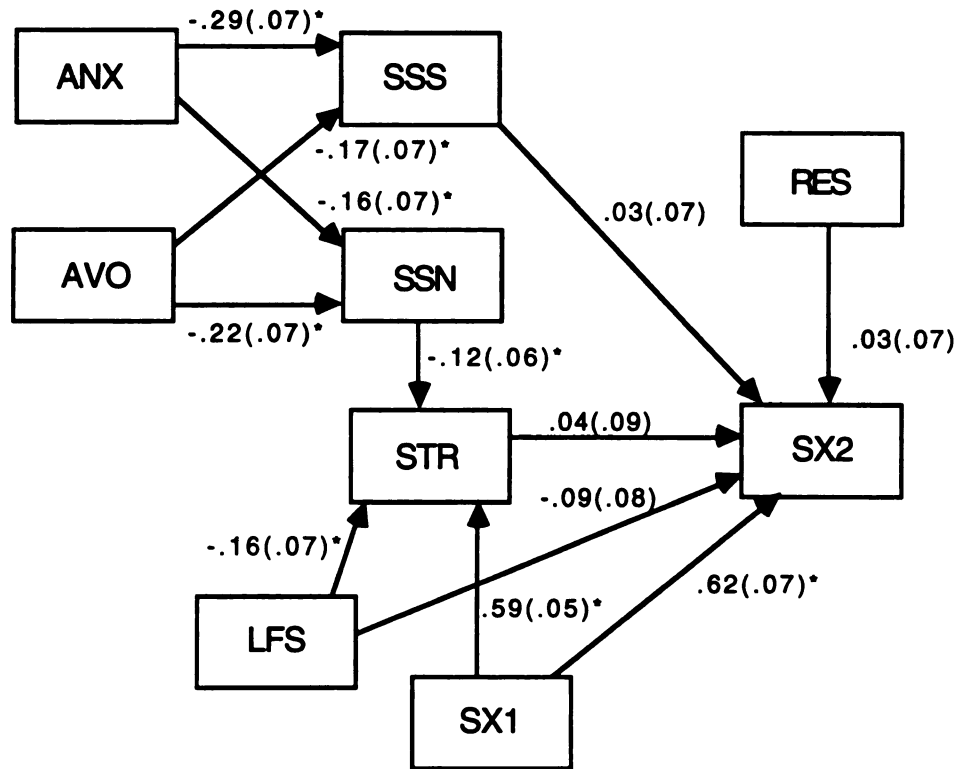


Figure 3. Path model to test Hypothesis 4 with path coefficients. (Beta weights are given with standard errors in parentheses. * indicates $p < .05$. ANX=Anxiousness, AVO=Avoidance, SSN=Social Support Number, SSS=Social Support Satisfaction, RES=residual term of $STR \times SSS$, LFS=Lifestyle at Time 1, SX1=Symptoms at Time 1, STR=Stress, SX2=Symptoms at Time 2.)

analysis of the data for each of the first three hypotheses. SSN was predicted by ANX ($\beta = -.16$, $p < .05$) and AVO ($\beta = -.22$, $p < .05$), as was SSS ($\beta = -.29$, $p < .05$, and $\beta = -.17$, $p < .05$, respectively). SSN and SX1 were significant predictors of STR ($\beta = -.12$, and $\beta = .59$, respectively, all $p < .05$). Interestingly, LFS became a significant predictor of STR when analyzed in this way ($\beta = -.16$). Finally, SX2 was only significantly predicted by SX1. All other paths were non-significant. The Chi-square for this model was 18.69 (d.f. = 14, $p = .177$), therefore this model fit the data somewhat well.

Subsequent analysis using SAS' CALIS produced slightly different results. While the path coefficients were similar, the standard errors were slightly smaller. For the path depicted in Figure 3 (without the residual term), this changed the significance of the path between LFS and SX2. Additionally, although the goodness of fit index was .95, which suggests that the model fit the data reasonably well, the overall Chi-square was significantly different from that found by PATH. The Chi-square was 51.83 (d.f.=11, $p < .01$) suggesting that the model was a poor fit. The root mean square error was .12.

It was thought that the difference between the results found with PATH and CALIS was due to an error in the way PATH calculates Chi-square. Unfortunately, timing (i.e., deadlines) prevented a complete rerun of the data on CALIS. Therefore, except where noted, all of the remaining data analysis was done using PATH. Because of the problems noted, the conclusions reached in the rest of this manuscript are to be considered equivocal. (NOTE: If

anyone would like the analysis done on SAS, please contact the author.)

Since the originally run path model (Figure 3) was not as representative of the data as was thought possible, an attempt was made to find a path model which better fit the data. To this end, all non-significant paths were deleted. This was done in a step-wise fashion, one path removed at a time, to test for significant changes in the model. However, since none of the removed paths individually caused significant differences between the path models, only the path model with all of the deleted paths will be presented.

Figure 4 shows the path model after all non-significant paths were eliminated. Again, ANX and AVO predicted SSS ($\beta = -.29$ and $\beta = -.17$, respectively), and SSN ($\beta = -.16$ and $\beta = -.22$, respectively). STR was ultimately only significantly predicted by SSN, LFS, and SX1 ($\beta = -.12$, $\beta = -.12$, and $\beta = .59$, respectively). And SX1 was the only predictor of SX2 ($\beta = .66$). The overall Chi-square of this model was 20.49 (d.f.=18, $p = .306$). Therefore the revised model fits the data significantly better than the originally tested model.

Although other models were tested (usually created by changing the direction of a causal arrow) these usually resulted in only minor changes in both the path coefficients and the significance of the Chi-square statistic. However, one interesting finding was that the path between SX1 and STR was of the same magnitude regardless of the direction in which causation was presumed to be.

PATH identified potentially significant paths which were not tested in this work. One such path was between Social Support

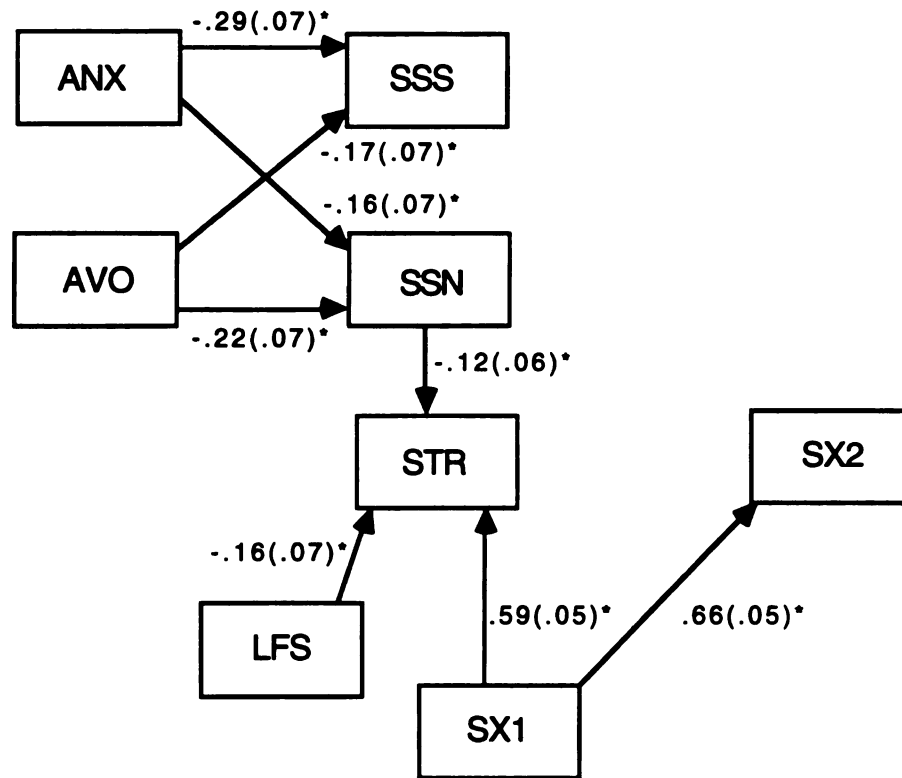


Figure 4. Modified path model with path coefficients. (Beta weights are given with standard errors in parentheses. * indicates $p < .05$. ANX= Anxiousness, AVO=Avoidance, SSN=Social Support Number, SSS=Social Support Satisfaction, LFS=Lifestyle at Time 1, SX1=Symptoms at Time 1, STR=Stress, SX2=Symptoms at Time 2.)

Number and Social Support Satisfaction. Another was between Anxiousness and Stress.

The finding that SSS did not demonstrate a buffering effect was surprising given previous research. Therefore, an attempt was made to explore whether SSS had a direct effect. SSS and SSN were changed in the path model of Figure 2 and a new residual term based on a product of SSN and STR. However, none of the new paths were significant and the model fit the data more poorly than the first model tested.

When the reduced model in Figure 4 (with the LFS-SX2 path added) was tested, SAS again provided different results than PATH. The goodness of fit index remained at .95, again suggesting that the model fit the data reasonably well. However, the Chi-square was 53.13 (d.f.=13), which also was significant ($p < .01$). The root mean square error for this model was .11. As noted earlier, this calls into question the validity of the data analysis and the resulting conclusions based on that analysis.

Discussion

The results suggest that one's level of physical symptoms is related to the level of stress one experiences concurrently but not to the physical symptoms one experiences four to six weeks later once prior symptoms are accounted for. Since causal factors must precede the factor they are purported to cause, it is unlikely that the stress experienced at Time 1 caused the symptoms reported at Time 1. However, since illness presents another demand on one's resources (e.g. time, energy), it may be that being ill causes stress, and, indeed, this is supported by other studies. As mentioned earlier, McFarlane, Norman, Streiner, and Roy (1983) suggested that a person's stress and health levels may be consistent across time, much like personality traits. The consistency across models of the beta weights between stress and illness when measured concurrently supports this line of reasoning. This suggests that people with a certain level of symptoms tend to maintain that level over time, and this consistency of symptoms may increase one's stress level. The design did not allow the determination of which might come first. A longitudinal study following people from a very young age may shed more light on this process.

It may be that the self-report measures of symptoms in this study were somewhat inaccurate because people under stress may tend to be more sensitive to unpleasant sensations of their bodies. More objective measures may be effective in correcting this potential source of error.

The lack of a significant relationship between stress and symptoms also may have been an artifact of the time lag between

Time 1 and Time 2 in this study (4-5 weeks). It has been suggested that stress affects a person's physical health through its effects on the immune system (Kiecolt-Glaser et al., 1986). Since the incubation period of many common illnesses is something less than one week (Cohen, Tyrrell, & Smith, 1991; Evans, Pitts, & Smith, 1988) and since most people recover from these illnesses within a period of two to three weeks, it may be that the time lag in this study was too long to demonstrate a connection between stress on later illness. Further studies of this sort should use a one to two week delay between testing sessions.

People with a healthier lifestyle, including good diet, regular exercise, and safe sexual habits, tended to suffer from less stress than those with a less healthy lifestyle, although this relationship was rather small. It may be that it takes time to exercise and to eat a more healthy diet. Therefore, it may be that busy students who are involved in a healthy lifestyle feel added time pressure as well as reaping the benefits of such good habits. Research devoted to identifying the pros and cons of healthy habits might be helpful in revealing such counterbalancing forces.

It was interesting to note that one's lifestyle has little to do with the amount of symptoms s/he experiences four to six weeks later, especially in light of the medical community's evidence that healthy habits tends to reduce health problems. Although the subjects in this study were consistent about responding to the lifestyle measure (as can be seen in the test-retest reliability), it may be that there was an unmeasured social desirability bias. Since it is well-known that people should exercise, eat well, sleep well, etc.,

perhaps the subjects reported that they complied with these guidelines to "look good" in the eyes of the experimenter. Or perhaps people reported healthier lifestyles because of a desire to follow that lifestyle or because of poor recall. Future research might be more accurate using more objective measures of lifestyle factors, such as diaries.

The main effects model for social support was partially supported by this study. Although modest, there was a statistically significant correlation between the number of people in one's social support system and the amount of stress one experiences. But, as reported earlier, that stress was not correlated with subsequent illness. The size of the relationship between social support and stress might be increased if more complete measures of the structure of social support networks were included. These might include measures of density and integration. There is evidence that the more integrated one's core network is, the less resistant it is to loss of one of its members (Hammer, 1983). Therefore, if a person has a group of friends and family who all know each other, it is more difficult to deal with the loss of one of that network's members because everyone in the group is impacted. Also, if one is experiencing stress due to one or two group members, people with social networks which are highly integrated might have difficulty obtaining assistance in dealing with their concerns since all of the people in the network are very familiar with each other.

The buffering portion of the social support hypothesis was not supported. This may have been due to the possible ceiling effects reported earlier in this manuscript. It may also have been due to the

nature of the buffering effect. In light of the data on the physiological effects of denial (Shedler, Mayman, & Manis, 1993), social support may merely increase the likelihood that a person experiencing stress will deny the existence or severity of a stressor. This may increase physical expressions of stress. Such social support may also interfere with the emotional expression which has been demonstrated useful in reducing stress (Pennebaker, Kiecolt-Glaser, & Glaser, 1988). Furthermore, this study did not address the stressful effects that a social network can sometimes produce (e.g., Cohen & Hoberman, 1983; Shaefer, Coyne, & Lazarus, 1981), and, therefore, it is difficult to determine if a high level of satisfaction with one's social network is also accompanied by an increase in responsibilities to the members of the network, and thus an increase in stress.

This study also supported the idea that people who are anxious about relationships tend to be less satisfied with their social networks than those who are not. Avoidant people also tend to be more dissatisfied with their networks than others, perhaps because the people in their networks expect closer connections than they are comfortable with. As a result, it was no surprise to find that people who tend to avoid relationships and maintain their self-sufficiency tend to have smaller networks than more outgoing people. The results also supported the hypothesis that people who are relatively more anxious about relationships have smaller networks than those who are relatively less anxious. Both of these findings suggest that people who are more securely attached tend to have more resources at their disposal, which tends to reduce stress. If these patterns are

consistent throughout the lifespan, there is a strong argument for the effects of early development of socialization patterns on health later in life. However, the results of this study do not allow one to make conclusions about the source of such patterns. If the sources of these patterns could be identified, it might be possible to intervene to change such patterns.

It should also be noted that this study did not address how effectively people of varying levels of anxiousness and avoidance use their social support networks. If those high on anxiousness or avoidance tended to make poor use of their networks, it might have decreased the direct effects of social support on illness and it might explain the lack of support for the buffering hypothesis.

A couple of notes should be made here. First, at the time this manuscript was completed, norms for the measures used were not available. Therefore, it is unknown if the sample used in this study are representative of the greater population. Second, although the measures used in this study had adequate reliability, they necessarily limit the amount of information which can be collected. If an interview format was used, it would have produced information more specific to the individual which might have been more informative from a clinical standpoint. Third, the stresses experienced by this population are unique. College students face challenges not commonly encountered by other adults, such as initial separation from parents, initial encounters with independence, and classroom demands. Because of these unique stressors, the results of this study may be difficult to generalize to other populations.

Finally, a few notes about the potentially significant paths which were not tested in this study. One of these was the connection between social support satisfaction and social support number. It is possible that people who are more satisfied with their social support networks would be more likely to retain the people in their networks and then to augment this steadier base. This would result in people high on satisfaction with their networks having larger networks than those less satisfied. Or it may be that people with larger networks are more satisfied with them, perhaps due to the wider range options this presents them with. If the former is true, it may be that satisfaction with social support affects stress levels indirectly through the structure of the network. Further study in this area is warranted to confirm or deny the existence of such a relationship and to ferret out the nature of it.

The other connection suggested by PATH was between a person's level anxiousness about relationships and the amount of stress s/he experiences. It seems likely that people who are anxious about their interpersonal relationships would experience more stress. Such a connection would also raise the possibility that it is not the number of persons in an individual's social support network that alleviates stress, but rather that one's concern about not having enough support or the most appropriate kinds of social support which causes stress. Further research is needed to test these hypotheses.

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