

Strategies for Measuring Stress in Studies of Psychiatric and Physical Disorders

Sheldon Cohen, Ronald C. Kessler, and Lynn Underwood Gordon

Stress has long been a major focus among researchers interested in environmental and psychosocial influences on health. However, the way in which the term "stress" has been used in this voluminous literature has not been consistent. Indeed, some commentators have gone as far as to argue that the term stress has so many different meanings that it has become a useless concept (Ader, 1980; Elliot & Eisdorfer, 1982). Although we disagree with this premise, we recognize that there is confusion about the meaning and measurement of stress. This volume attempts to clear up some of that confusion by providing conceptual and practical advice for formulating questions about the relation between stress and disease and for selecting appropriate stress measures to test these questions.

What Is Stress?

As noted above, there is disagreement about the meaning of the term "stress." Numerous definitions have been provided, varying in the extent to which they emphasize stressful events, responses, or individual appraisals of situations as the central characteristic of stress (e.g., Appley & Trumbull, 1967; Mason, 1975; McGrath, 1970). We have no illusions that we can resolve the differences among these perspectives in this volume. However, we do see a strong commonality among these approaches that allows them to be integrated in a theoretical model of the role of stress in disease. They all share an interest in a process in which *environmental demands tax or exceed the adaptive capacity of an organism, resulting in psychological and biological changes that may place persons at risk for disease.*

Three broad traditions of assessing the role of stress in disease risk can be distinguished. The environmental tradition focuses on assessment of environmental events or experiences that are normatively (*objectively*) associated with substantial adaptive demands. The psychological tradition focuses on individuals' *subjective* evaluations of their abilities to cope with the demands posed by specific events or

experiences. Finally, the biological tradition focuses on activation of specific physiological systems that have been repeatedly shown to be modulated by both psychologically and physically demanding conditions. The following sections provide a brief orientation to these traditions and attempt to distill the central assumptions of each. We then present an organizational model of the stress process that shows how these traditions relate to one another. Our premise is that each tradition focuses on a different stage of the process through which environmental demands are translated into psychological and biological changes that place people at risk for disease. Finally, we discuss the conceptual and practical issues involved in selecting appropriate categories of stress measurement. Once appropriate categories are selected, subsequent chapters of this volume can be consulted to help select measures within each category.

Throughout the volume we use the term stress exclusively to refer to the general process through which environmental demands result in outcomes deleterious to health. However, we distinguish between the components of the process by referring to environmental experiences as *environmental demands, stressors, or events*; to subjective evaluations of the stressfulness of a situation as *appraisals or perceptions of stress*; and to affective, behavioral, or biological responses to stressors or appraisals as *stress responses*.

The Environmental Stress Perspective

Most evidence on the role of stressors in human disease has derived from interest in stressful life events. Interest in the role of life events in illness began with the work of Adolf Meyer in the 1930s. Meyer advocated that physicians fill out a life chart as part of their medical examination of ill patients (Lief, 1948; Meyer, 1951). Meyer believed that the life events elicited in this way could be shown to have etiologic importance for a variety of physical illnesses. Meyer's ideas were highly influential and led to a substantial body of research which, by the late 1940s, had documented that stressful life events were associated with a variety of physical illnesses (see review by Wolff, Wolf, & Hare, 1950). Although some of this early work was based on inadequate research designs, a number of studies were quite impressive. The work of Wolff and his associates, for example, followed a large sample of telephone operators over many years and documented that illness was much more likely to occur during periods of inordinate demands, frustrations, and losses than at other times (Hinkle & Wolff, 1958).

An important advance in this area of research came in 1957 when Hawkins and his collaborators developed the Schedule of Recent Experiences (SRE) in an effort to systematize Meyer's life chart (Hawkins, Davies, & Holmes, 1957). This instrument was used by a great many researchers over the next decade to document associations between stressful life events and heart disease, skin disease, and many others (reviewed by Holmes & Masuda, 1974). In a subsequent modification of the SRE, the Social Readjustment Rating Scale (SRRS), each event was assigned a standardized weight based on judges' ratings of the degree of difficulty required to adjust to the event (Holmes & Masuda, 1974). These weights were called "life

change units" (LCU). The summing of LCUs associated with reported events allowed for a summary measure of environmental stressors (Holmes & Rahe, 1967). This instrument had an enormous impact on research on the relations between life events and illness, due in large part to the documentation of dramatic associations, such as an effect of stressful life events on sudden cardiac death (Rahe & Lind, 1971). It also had an important conceptual impact on the field in advancing the notion of the life change unit and the conceptual model underlying the creation of this metric, which argued that the effects of stressors operate largely through the creation of excessive adaptive demands. This conception led users of the SRRS to be more concerned with the magnitude of life change than with whether the change was positive (e.g., a promotion) or negative (e.g., a job loss).

Beginning in the 1970s, a new generation of stressful life event researchers began to challenge many of the basic assumptions involved in the construction and scoring of the SRRS. New ideas were advanced about the implications of different means of weighting and summing multiple events into cumulative scales (Shrout, 1981). A subjective element was introduced into some modifications of the SRRS by having individuals estimate the stressfulness of their own experiences as a way of generating measures that are more sensitive indicators of event stressfulness than judges' ratings (e.g., Sarason, Johnson, & Siegel, 1978). More drastic differences are reflected in the development of a life event interview in which investigators rate the importance of events while taking into account the context in which they occur (Brown & Harris, 1978). The investigator-based rating is an attempt to estimate the impact of an event in a specific context for the average person, avoiding individual subjective reactions. A major distinction among competing measures of life events in the current literature is between these contextual measures and more traditional checklist measures (Brown & Harris, 1989; Dohrenwend, Raphael, Schwartz, Stueve, & Skodol, 1993). Separate chapters on these two approaches are included in Section II of this volume.

New concerns were also raised during this period that existing life event scales may not include an adequate and representative sample of the major events that occur in people's lives. Newer checklists were developed to expand the range of experiences evaluated (Dohrenwend, Askenasy, Krasnoff, & Dohrenwend, 1978). Scales were also developed to assess stressful events in specific populations whose experiences might be different from those represented on the more general SRRS. These included scales for children (e.g., Sandler & Ramsay, 1980), adolescents (e.g., Newcomb, Huba, & Bentler, 1981) and the elderly (e.g., Murrell, Norris, & Hutchins, 1984).

The basic assumptions of the SRRS, that the effects of all stressful events are cumulative and that the change per se is the most important dimension of stressors, were also challenged during the same time period (Paykel, 1974). Some newer life-event scales were based on a multidimensional conception of stressors that separately assessed the extent of threat, loss, danger, and other aspects of stressful events (Brown & Harris, 1978).

On the substantive side, there has been a continuation of basic research to document the effects of stressful events on a variety of physical and mental health outcomes using newer stressful life event measures (Chapters 2 & 3, this volume).

There also has been interest in studying the cumulative effects of experiencing two or more stressful life events in the same short interval of time (McGonagle & Kessler, 1990) and the joint effects of experiencing a stressful event in the context of an ongoing chronic stressor in the same life domain (Wheaton, 1990).

In addition, there is now considerable interest in studying *vulnerability factors*—characteristics that make people more or less susceptible to stressor-induced disease. This interest derives from the repeated finding that although environmental stressors are often associated with illness onset, the majority of people confronted with extreme or traumatic stressful events do not become ill (Thoits, 1983). Differential vulnerability to the health-damaging effects of environmental stressors has been documented in a number of investigations (see review by Kessler, Price, & Wortman, 1985). A search for the determinants of this differential vulnerability has become the main focus of researchers interested in the role of life events in illness. In addition, the decade of the 1980s saw a movement away from an earlier tradition of focusing exclusively on the acute health-damaging effects of discrete life events toward an investigation of the long-term health-damaging effects of chronic stressors (Chapter 5, this volume). Work stressors (Neilson, Brown, & Marmot, 1989), marital disharmony (Beach, Sandeen, & O'Leary, 1990), and work-family conflicts (Eckenrode & Gore, 1990) have been the primary areas of investigation in research on chronic stressor effects. In addition, there is a new interest in the cumulative effects of minor daily stressors on both emotional health (Bolger, DeLongis, Kessler, & Schilling, 1989) and physical health (Chapter 4, this volume; Stone, Reed, & Neal, 1987). In all of this work, the researcher attempts to identify characteristics of the environment that promote illness. In the initial stages of this work the focus is on description. As the work evolves, the focus shifts to the processes involved in creating the observed association, the topic of Parts III and IV of this volume.

Finally, although stressful events have been studied primarily as risk factors for disease, it is becoming increasingly clear that confronting and adapting to stressful events can result in positive outcomes such as personal growth, reprioritization of life goals, increased feelings of self-esteem and self-efficacy, and strengthening of social networks. A greater emphasis on the benefits of stressful events for successful adaptors is likely in the future and would broaden our understanding of the stress process.

The Psychological Stress Perspective

The psychological stress tradition places emphasis on the *organism's perception and evaluation* of the potential harm posed by objective environmental experiences. When their environmental demands are perceived to exceed their abilities to cope, individuals label themselves as stressed and experience a concomitant negative emotional response. Psychological models of stress argue that events influence only those persons who appraise them as stressful—that is, perceive stress. It is important to emphasize that stress appraisals are determined not solely by the stimulus condition or the response variables, but rather by persons' interpretations of their

relationships to their environments. That is, the perception that one is experiencing stress is a product of both the interpretation of the meaning of an event and the evaluation of the adequacy of coping resources.

The most influential model of the appraisal process has been the one proposed by Lazarus (Lazarus & Folkman, 1984). In the original formulation of his model, Lazarus (1966) argued that an appraisal of a stimulus as threatening or benign, termed *primary appraisal*, occurs between stimulus presentation and stress reaction. In his later writings, Lazarus (1977, 1981) argued that a situation will also result in a stress reaction if it is evaluated as a harm/loss, threat, or challenge. Primary appraisal is presumed to depend on two classes of antecedent conditions: the perceived features of the stimulus situation and the psychological structure of the individual. Some stimulus factors affecting primary appraisal include the imminence of harmful confrontation, the magnitude or intensity of the stimulus, the duration of the stimulus, and the potential controllability of the stimulus. Factors within individuals that affect primary appraisal include their beliefs about themselves and the environment, the pattern and strength of their values and commitments, and related personality dispositions. When a stimulus is appraised as requiring a coping response, individuals evaluate their resources in order to determine whether they can cope with the situation—that is, eliminate or at least lessen the effects of a stressful stimulus. This process is termed “secondary appraisal.” Coping responses may involve actions designed to directly alter the threatening conditions (e.g., fight or flight) or thoughts or actions whose goals are to relieve the emotional stress response (i.e., body or psychological disturbances). The latter group of responses, referred to as “emotionally focused” coping, may be somatically oriented—for example, the use of tranquilizers, or intrapsychic responses such as denial of danger (Lazarus, 1975). If one perceives that effective coping responses are available, then the threat is short-circuited and no stress response occurs. If, on the other hand, one is uncertain that she or he is capable of coping with a situation that has been appraised as threatening or otherwise demanding, stress is experienced. It is important to note that this process of evaluating the demands of a situation and evaluating one’s ability to cope not only occurs at the onset of a stressful event but often recurs during the course of the event (cf. Folkman, 1970; Lazarus, 1981). Thus, an event that is initially appraised as threatening may be later reappraised as benign, and coping strategies that are initially found to be lacking may later be found to be adequate. Conversely, events that one initially evaluates as nonthreatening may be later reevaluated as stressful. Although it is recognized that certain events are almost universally appraised as stressful (e.g., the death of a loved one), the impact of even these events can be expected to depend on an individual’s appraisal of the threat entailed and his or her ability to cope with it. For example, the death of a spouse for someone with neither family nor friends may be experienced as more severe than the same event for someone with close ties to family and friends.

As suggested earlier, appraisals of threat elicit negative emotional responses. They also can elicit a range of other outcomes including self-reported annoyance, changes in health practices such as smoking, drinking alcohol, diet, exercise, and sleeping; changes (usually deficits) in performance of complex tasks; and alterations in interpersonal behaviors (Cohen, Evans, Krantz, & Stokols, 1986). Unfortunately,

psychological stress models tend to be vague in their predictions of the particular measures that will be affected in any instance, and of the nature of the relations among these outcome measures.

The Biological Stress Perspective

The biological perspective focuses on the activation of physiological systems that are particularly responsive to physical and psychological demands. Prolonged or repeated activation of these systems is thought to place persons at risk for the development of a range of both physical and psychiatric disorders. Two interrelated systems that are viewed as the primary indicators of a stress response are the sympathetic-adrenal medullary system (SAM) and the hypothalamic-pituitary-adrenocortical axis (HPA). Although detailed descriptions of these two systems and their relations to each other are beyond the scope of this chapter (see Baum, Singer, & Baum, 1981; Levi, 1972; and Chapter 8, this volume), each is discussed in brief in order to provide a basic understanding of their roles in the stress process.

Sympathetic-Adrenal Medullary System

Interest in the impact of SAM activation on bodily reactions to emergency situations may be traced to Walter Cannon's early work on the flight or flight response (Cannon, 1932). Cannon proposed that the SAM system reacts to various emergency states with increased secretion of the hormone epinephrine. There is a large body of evidence indicating increased output of epinephrine and norepinephrine in response to a wide variety of psychosocial stressors (Levi, 1972). Other components of the SAM response elicited by stressors include increased blood pressure, heart rate, sweating, and constriction of peripheral blood vessels. It has been claimed that if SAM activation is excessive, is persistent over a period of time, or is repeated too often, it may result in a sequence of responses that culminate in illness. The responses include functional disturbance in various organs and organ systems (cf. Dunbar, 1954) and ultimately permanent structural changes of pathogenic significance at least in predisposed individuals (e.g., Raab, 1971). Particularly culpable in this regard is the secretion of the hormones epinephrine and norepinephrine by the adrenal medulla and/or sympathetic nerve endings. Excessive discharge of these substances is believed to induce many of the pathogenic states associated with the perception of stress including (1) suppression of cellular immune function (e.g., Rabin, Cohen, Ganguli, Lysle, & Cunnick, 1989); (2) hemodynamic effects, such as increased blood pressure and heart rate (McCubbin, Richardson, Obrist, Kizer, & Langer, 1980); (3) provocation of variations in normal heart rhythms (ventricular arrhythmias) believed to lead to sudden death (Herd, 1978); and (4) production of neurochemical imbalances that contribute to the development of psychiatric disorders (Anisman & Zacharko, 1992).

Hypothalamic-Pituitary-Adrenocortical Axis (HPA)

The hormonal responses of the HPA axis were emphasized in Hans Selye's (e.g., 1956, 1974) influential description of a nonspecific (general) physiological reaction that occurs in response to excessive stimulation. Selye argued that pathogens, physical stressors (e.g., shock or noise), and psychosocial stressors all elicit the same pattern of physiological response. This response is said to proceed in a characteristic three-stage pattern referred to as the general adaptation syndrome (GAS). During the first stage of the GAS, the *alarm stage*, the organism's physiological changes reflect the initial reactions necessary to meet the demands made by the stressor agent. The anterior pituitary gland secretes adrenocorticotrophic hormone (ACTH), which then activates the adrenal cortex to secrete additional hormones (corticosteroids [primarily cortisol in humans]). The hormone output from the adrenal cortex increases rapidly during this stage. The second stage, *resistance*, involves a full adaptation to the stressor with consequent improvement or disappearance of symptoms. The output of corticosteroids remains high but stable during the resistance stage. Finally, the third stage, *exhaustion*, occurs if the stressor is sufficiently severe and prolonged to deplete somatic defenses. The anterior pituitary and the adrenal cortex lose their capacity to secrete hormones, and the organism can no longer adapt to the stressor. Symptoms reappear, and, if the stress response continues unabated, vulnerable organs (determined by genetic and environmental factors) will break down. This breakdown results in illness and ultimately death. Selye argued that any noxious agent, physical or psychosocial in nature, would mobilize a similar GAS response. In contrast, critiques of Selye's model suggest that each stressor elicits its own distinct physiological reactions (Lazarus, 1977; Mason, 1975). These authors agree that there is a nonspecific physiological response to stressors. They argue, however, that the response is a concomitant of the *emotional reaction* that occurs when situations are appraised as stressful. When conditions are designed to reduce the psychological threat that might be engendered by laboratory procedures, there is no nonspecific reaction to a physical stressor (Mason, 1975). For example, by minimizing competitive concerns and avoiding severe exertion, the danger that young men would be threatened by treadmill exercise was reduced, and the GAS pattern was not found. It is noteworthy that, in Selye's later work (1974, 1980) he acknowledged that there are both specific as well as general (nonspecific) factors in physiological response to a stressor but maintained that the nonspecific response is not always psychologically mediated. He also suggested that the GAS does not occur (or is at least not destructive) in response to all kinds of stressors. For example, he suggested that there may be a pleasant stress of fulfillment and victory and a self-destructive distress of failure, frustration, and hatred. However, there is little empirical evidence to support this position.

Since the late 1970s, interest in the biological bases of psychiatric disorders has stimulated an alternative focus on the HPA. Most of this work has pursued the possible role of HPA dysregulation in depression. Relatively pronounced HPA activation is common in depression, with episodes of cortisol secretion being more frequent and of longer duration among depressed than among other psychiatric

patients and normals (Stokes, 1987). However, it is still unclear whether the hyper-HPA activation is a cause or effect of depressive disorders. HPA regulation may play a role in other psychiatric disorders as well. For example, anxious patients tend to have higher cortisol levels than normal controls (Sachar, 1975).

Other Stress Associated Changes

Although hormones of the SAM and HPA are those most often discussed as the biochemical substances involved in stress responses, alterations in a range of other hormones, neurotransmitters, and brain substances have also been found in response to stress and may play an important role in stress influences on health. These include stressor-associated elevations in growth hormone and prolactin secreted by the pituitary gland, and in the natural opiates beta endorphin and enkephalin released in the brain (see Chapter 8, this volume; also Baum, Grunberg, & Singer, 1982). These substances are also thought to play a role in both immune-mediated (Rabin et al., 1989) and psychiatric diseases (Stokes, 1987).

A Unifying Model of the Stress Process

The perspectives represented by the three traditions discussed earlier can be viewed as emphasizing different points in the process through which objective environmental experiences can influence disease. A model integrating these approaches is presented in Figure 1.1. The sequential relations between the central components of the model (dark arrows) can be described as follows. When confronting environmental demands, people evaluate whether the demands pose a potential threat and whether sufficient adaptive capacities are available to cope with them. If they find the environmental demands taxing or threatening, and at the same time view their coping resources as inadequate, they perceive themselves as under stress. The appraisal of stress is presumed to result in negative emotional states. If extreme, these emotional states may directly contribute to the onset of affective psychiatric disorders. They may also trigger behavioral or physiological responses that put a person under risk for psychiatric and physical illness. This model implies that each sequential component of the stress process is more proximal to and hence more predictive of the illness outcome. For example, a disease-relevant biological stress-response measure should be a better predictor of a disease outcome than measures of stressful life events or perceived stress.

We thought it important that the model also represent the possibility that environmental demands can put persons at risk for disorder even when appraisal does not result in perceptions of stress and negative emotional responses. This is represented by the arrow directly linking environmental demands to physiological or behavioral responses. For example, it has been argued that the process of coping itself (even when it is successful and environmental demands are appraised as benign) may *directly* result in physiological and behavioral changes that place persons at risk for disease (Cohen et al., 1986; Cohen, Tyrrell, & Smith, 1993).

We want to emphasize that this is a *heuristic model* designed to illustrate the

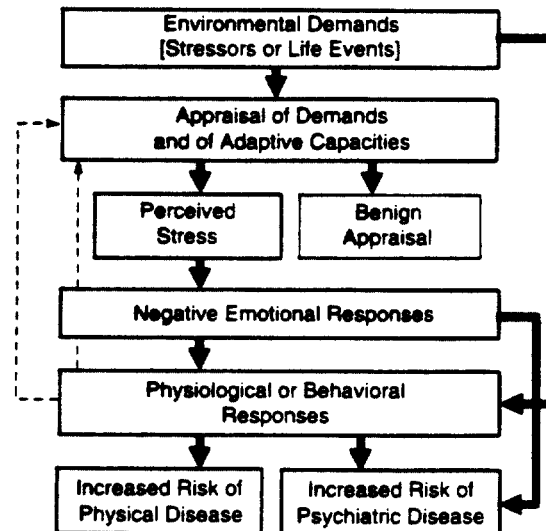


Figure 1.1 A heuristic model of the stress process designed to illustrate the potential integration of the environmental, psychological, and biological approaches to stress measurement.

potential integration of the environmental, psychological, and biological approaches to stress measurement. The model is primarily unidirectional (flowing from environmental demands to disease) and does not include all possible pathways linking these concepts. We identified two of *many possible* feedback loops (dashed lines) to illustrate the potential role of feedback in the model. One of the loops suggests that emotional states may alter appraisals. For example, depressed affect may result in negatively biased views of either the threat posed by stressors or the adequacy of one's own resources. The other loop suggests that physiological arousal may alter appraisals and emotional responses: For example, persons may mistakenly attribute arousal that was elicited by exercise, drugs, or nonrelevant emotional responses to a stressor (Schachter & Singer, 1962). The exclusion of alternative paths is not intended to reflect hypotheses about their existence.

Historically, the environmental, psychological, and biological traditions have each focused on a specific part of the model, thus often ignoring other parts. For example, sociologists and epidemiologists have addressed the question of whether life events increase disease risk but usually ignore the psychological and biological pathways through which this influence might occur. Psychologists have focused on the role of appraisal and emotional response in disease risk, with less emphasis on the environmental causes of these states and the biological pathways responsible for links between psychological states and disease. Finally, biological stress researchers have focused primarily on the links between stressors and hormonal and cardiovascular response, and between these responses and disease risk, without concerning themselves with the psychological pathways through which stressors might influence biological states.

How Could Stress Influence Disease?

We have provided a generic model of the stress process. In this section, we elaborate the implications of the generic model for physical and psychiatric illness. Our hope is that this presentation of how various conceptions of stress can influence illness will help generate appropriate questions and, consequently, the selection of measures and research designs.

Stress and Physical Illness

In general, stressors are thought to influence the pathogenesis of physical disease by causing negative affective states (such as anxiety and depression) which in turn exert direct effects on biological processes or behavioral patterns that influence disease risk (see Cohen et al., 1986; Krantz, Glass, Contrada, & Miller, 1981). The primary biological pathway linking emotions to disease is thought to be hormonal. Hormonal responses associated with stressful experiences include elevations in the catecholamines epinephrine and norepinephrine secreted by the adrenal medulla, in cortisol secreted by the adrenal cortex, in growth hormone and prolactin secreted by the pituitary gland, and in the natural opiates beta endorphin and enkephalin released in the brain (see Baum, Grunberg, & Singer, 1982). A number of these hormones have been implicated in the pathogenesis of cardiovascular disease (Herd, 1986) and diseases involving the immune system including cancer, infectious diseases, and autoimmune diseases (Cohen & Williamson, 1991; Laudenslager, 1988; Rabin et al., 1989). Emotionally induced responses of the cardiovascular system such as increased heart rate and blood pressure have also been implicated in the development of cardiovascular disease and of immune changes that might alter susceptibility to immune-mediated disease (Herbert et al., 1994; Manuck et al., 1992). As discussed earlier, emotional responses are not always required for stressful events to influence disease processes (Cohen, Tyrrell, & Smith, 1993). The effort involved in actively coping with a stressor may also alter many of the same biological processes influenced by the emotional response and hence influence the development of disease independently of the emotional response (Cohen et al., 1986).

Behavioral changes occurring as adaptations or coping responses to stressors may also influence disease risk. For example, persons exposed to stressors or viewing themselves as under stress tend to engage in poor *health practices*. They may smoke more, drink more alcohol, eat poorly, exercise less, and sleep less (e.g., Cohen & Williamson, 1988; Conway, Vickers, Ward, & Rahe, 1981). Smoking, drinking alcohol, and poor diets have been established as risk factors for a range of different physical illnesses. Both stressors and negative affect have also been associated with failure to *comply* with medical regimens. Such failure could result in more severe and longer-lasting illness, either because undesirable behaviors aggravate existing problems or because failure to perform desirable behaviors (e.g., following medication regimens) results in disease progression. Other stressor-elicited behaviors—for example, unsafe sexual practices or poor hygienic practices—could also increase exposure to infectious agents.

Stress and Illness Behaviors

Stress may also influence behaviors that appear to be manifestations of a disease state. Of particular interest is the role of stress in recognizing and acting on symptoms. The recognition and reporting of symptoms, and seeking of medical care often indicate underlying pathology. However, stress and other psychological factors can independently influence these behaviors (Cohen & Williamson, 1991). Because appraised stress often triggers physiological arousal, people under stress may be more (possibly overly) attentive to their internal physical states (Pennebaker, 1982). Stress may also facilitate the labeling of sensations as symptoms because people are reminded (in cognitive parlance, a schema is triggered) of previous times when stress was associated with symptoms or simply because they believe that stress triggers symptoms. Alternatively, stressors or stress appraisals may result in physical sensations whose causes are mistakenly attributed to disease symptoms rather than the stressor (e.g., Mechanic, 1972; Schachter & Singer, 1962). Labeling symptom constellations as disease may similarly be activated by stress-disease schemas. For example, it is widely believed that stressors cause the recurrence of oral herpes. Under stress, a minor oral lesion that would be ignored under nonstressful conditions may be defined as disease recurrence. Reports of symptoms and illness are also ways to avoid stressful situations (Mechanic, 1977). The prototypical example is the child who reports symptoms to avoid attending school on an especially stressful day (playing ill). Finally, stressors or stress appraisals may influence the decision to seek medical care when persons label themselves as ill. The perception of stress could interfere with deciding whether it is necessary to seek care, increasing care-seeking for minor symptoms or decreasing care-seeking for serious ones. Persons under stress may also seek medical care unnecessarily because medical providers are viewed as persons to whom one can confide problems. Stressors could also *decrease* care-seeking because the time demands of many stressors make such visits inconvenient (Schulz, Visintainer, & Williamson, 1990).

Stress and Psychiatric Illness

As noted above, most models of the relations between stress and physical illness postulate an intervening link through negative affective states such as anxiety and depression. But how is it that stressors cause these affective states, particularly those that are so extreme and enduring that they are considered disorders in their own right? There are two broad perspectives on this question. One holds that stressful experiences are sometimes so extreme that they naturally lead to enduring fear or sadness. This is the notion, for example, underlying the diagnosis of post-traumatic stress disorder, a disorder defined by the American Psychiatric Association as one in which there is enduring distress related to recurrent recollections of a traumatic event that is so far "outside the range of usual human experience . . . that (it) would be markedly distressing to almost anyone" who experienced it (American Psychiatric Association, 1987:250). The second perspective holds that stressful

events lead to psychiatric disorder only in the presence of some preexisting personal vulnerability. A number of different vulnerability factors have been postulated for different disorders. Some of these are thought to be environmental in origin (U.S. Congress Office of Technology Assessment, 1992). For example, some researchers believe that exposure to abnormal parental attachment behaviors early in life can lead children to develop disturbed interpersonal styles which persist throughout their lives and create vulnerability to depression (Kessler & Magee, 1993). This vulnerability is thought to be triggered by stressful experiences associated with interpersonal loss, such as death of a loved one, divorce, or forced residential relocation. Other vulnerabilities are thought to be biological in origin. For example, there is evidence consistent with the possibility that HPA abnormalities are involved in vulnerability to depression (Anisman & Zacharko, 1992; Checkley, 1992) and that these HPA abnormalities may be under genetic control (Checkley, 1992).

Temporal Characteristics of Stressors, Appraisals, and Stress Responses

One of the most challenging issues in the measurement of stress is the characterization of the temporal course of stressors, appraisals, and stress responses. Most of the discussion of the role of stress duration focuses on the chronicity of stress. Although the terms "acute" and "chronic" stress are used liberally in the literature, there have been few attempts (for an exception, see Brown & Harris, 1989) to set even arbitrary standard cutoffs delimiting acute from chronic durations. Our own view is that such cutoffs should be attempted only in the context of considering the implications for a specific outcome. For example, 3 months of persistent stress might prove important in risk for depression, and prove inconsequential for a disease that develops over years such as coronary artery disease. Assuming we were correct about the implications of these durations, it might be appropriate to define 3 months of persistent stress as "chronic" in the study of depression, but it would be uninformative to call 3 months of stress chronic in the study of heart disease (see Cohen & Matthews, 1987; Cohen, Kaplan, & Manuck, 1994).

A view of temporal characteristics of stress consistent with the process model discussed earlier is proposed by Baum and his colleagues (Baum, Cohen, & Hall, 1993). They categorize stress duration through the use of a $2 \times 2 \times 2$ matrix that crosses duration of event exposure (i.e., event present for short or long duration), duration of perceived threat (i.e., appraised threat or demand present for short or long duration), and duration of stress responding (i.e., behavioral, emotional, or physiological stress responses present for short or long duration). This procedure suggests a more sensitive approach to understanding the role of stress duration. For example, consider the difference between a persistent stressful event that is no longer appraised as stressful or responded to with a stress response, and a stressful event that has terminated but continues to be appraised as stressful and responded to with a stress response (e.g., traumatic experiences).

Other work highlights some of the complexities of this issue by emphasizing the importance of temporal characteristics other than duration such as continuousness or

repetitiveness. The following classification of stressful events (from Elliott & Eisdorfer, 1982) provides an indication of the complexity of this issue: (1) *acute time-limited events* (e.g., awaiting surgery); (2) *stressful event sequences*—when one event initiates a series of different events that occur over an extended period of time (e.g., bereavement or being fired from a job); (3) *chronic intermittent stressful events*—events that occur periodically (once a week, once a month, or once a year; e.g., sexual difficulties or conflicts with neighbors); and (4) *chronic stress conditions*—situations that may or may not be initiated by a discrete event (e.g., being disabled, chronic job stress).

The temporal course of events, stress appraisals, and stress responses have multiple implications for understanding the risk for disease. For example, one can ask how long an exposure, appraisal, or response is required to alter a disease process. Does persisting exposure result in a greater impact or in habituation? Are stress responses maintained over long periods, or do response mechanisms fatigue or trigger feedback mechanisms? What roles do repetitiveness and interval between repetitions play in these processes? Also of interest is the role that chronic stressors play in moderating the effect of more acute stressors. Unfortunately, there are few domains in which we know very much about the temporal role of stress. It is hoped that future longitudinal research addressing specific questions about the temporal course of stress will begin to provide us with a better understanding about how these issues should be treated.

Matching Measures, Designs, and Research Questions

The choice of appropriate stress measures depends on the *disease* (or stage of disease) under study, on the *specific question* posed by the investigator about the relation between stress and disease, and on other *methodological and practical issues*. Although we discuss each of these criteria separately, all three criteria need to be optimized if the most appropriate measure is to be selected.

Matching the Temporal Courses of the Stress Measure and Disease

Choosing an appropriate stress measure requires investigators to be informed about the disease outcome they are studying. Of particular importance is an understanding of the *temporal course* of the disease or disease stage under investigation (Cohen, Kaplan, & Manuck, 1994; Cohen & Matthews, 1987). Take, for example, the possible role of stressors in coronary artery disease (CAD). Atherosclerosis (occlusion of the coronary arteries) does not occur suddenly, over a few weeks or months, or even over several years. Instead it develops over decades. What kind of stressor measure would be most appropriate in studying the potential impact of stressors on CAD? Answering this question requires the investigator first to specify plausible pathways through which stressors might influence the development of the disease. Logically, there are two primary ways in which stressors could play a part in a

disease with this course of development. First, a traumatic stressful event or events might trigger behavioral or biological processes that contribute to the *onset* of a disease process. After this process is set into action, the disease could then develop over many years. In this case, the trigger could occur at any time in the life course. Testing this assumption would require a technique that allows for reporting and timing of traumatic events over several decades, such as appropriately adapted event interviews or checklists. Second, long-term exposure to a chronic stressful experience might facilitate the development of disease during stressor exposure. Exposure to a chronic stressor might result in permanent or at least long-term psychological, biological, and behavioral responses that alter the progression of the disease. This hypothesis assumes exposure to chronic stressor(s) and hence requires measurement of chronic stressor experiences. Alternative types of measures include event interviews focusing on chronic experiences and scales designed to assess specific chronic stressors such as marital discord, unemployment, and disability. Obviously, measures assessing acute stressful events such as daily events, or acute stress perceptions or stress responses such as perceived stress, and negative affect measures would be inappropriate in testing either of these hypotheses. An alternative example is the role of stressors in triggering heart attacks (myocardial infarctions). The assumption in this case is that sudden and acute stressful experiences trigger coronary events (Cohen, Kaplan, & Manuck, 1994). Here, acute measures of life events, perceived stress, and negative affect would be appropriate, whereas chronic strain measures covering months or years would be inappropriate.

Other models are possible, and these are intended only as examples. Our point is that investigators need to understand enough about the course of a disease outcome to be able to generate hypotheses about the *duration and timing* of stressor, appraisal, or stress-response exposure required to influence disease onset or progression. With this information, they can choose a stress measure with a temporal span that matches that of the disease process. Different hypotheses may imply very different types of measures as well as different research designs.

Posing a Specific Question About the Relations Between Stress and Disease

The most important issue in designing a research study is to state clearly the question an investigator wants to answer. The more specific the question, the more guidance it provides for choice of instruments and study design. Global questions—for example, What role do stress-related physiological responses play in overall health?—are important. However, a specific question—for example, Does increased heart rate responsivity mediate the relationship between environmental stressors and the development of cardiovascular disease?—is required for the development of a research protocol and selection of appropriate instruments.

The model in Figure 1.1 suggests a variety of questions one might ask about the relation between stress and disease. In this section, we give examples of some of these questions and of the types of measures one would use for each.

When studying the relation between *environmental demands* and health, the

focus is on characterizing the environment or environmental/social changes that influence the onset or progression of disease. As discussed earlier, the majority of research in this area has focused on stressful life events. Event characteristics can include the type or domain of events, the magnitude of events, the temporal characteristics of events, or the nature of relations between combinations of events. Part II of this volume addresses different approaches to assessing environmental demands including checklist life event measurement (Chapter 2), interview measurement of stressful events (Chapter 3), daily and within-day event measurement (Chapter 4), and chronic stressor measurement (Chapter 5). Life event measures, whether checklist or interview, are generally used to assess the *cumulative* impact of events over a prolonged period (ranging from 3 months to a life-time, although these measures most often span a period of 6 months to 2 years). Sophisticated versions of these measures can also provide additional information about the context in which the events occurred to aid in determining the potential impact of the events. For example, in the case of a natural disaster, some people will lose all of their possessions and perhaps even some of their loved ones, whereas others will suffer only minor property loss or be fortunate enough to avoid any loss whatsoever. One would expect this variability to influence the stress perceptions and responses. The sensitivity of event measurement can be improved to the extent that a measurement technique allows this kind of information to be obtained.

Chronic stressor measures generally assess the impact of *individual* stressors that last for prolonged (but often unspecified) periods—for example, enduring economic, work, or marital problems. Although such measures can focus solely on environmental events, they also include assessments of long-lasting appraisals or affective responses to specific environmental characteristics. Finally, daily and within-day event measures are generally used to assess the cumulative impact of the events that occur during a single day. Most of these events (or hassles) are so small that they would not be considered contributors to perceived stress and stress-responses when the other, more chronic approaches to event measurement are employed. Daily event measures have many of the same characteristics of major event measures. For example, they can be used to examine the importance of specified domains of events and to elicit information about the meaning and impact of individual events. They are generally employed in studies of the role of very acute (simultaneous or 1- or 2-day lag) changes in health. However, if administered over long periods of time, they can also be used to assess persistent difficulties and nagging day-to-day problems that continue for long periods of time.

As discussed earlier, appraisals refer to the psychological interpretation of events as demanding, threatening, or challenging as opposed to benign (Lazarus & Folkman, 1984). The study of stress appraisal focuses on what are often termed *vulnerability factors*—the nature of persons and their contexts that make them more or less vulnerable to stress-induced disease. For example, variables such as social support and feelings of control that are thought to contribute to adaptive capacities and hence to protect persons from the pathogenic effects of stressful events are thought to act by short-circuiting the appraisal of stress. Alternatively, variables like type A behavior pattern are thought to increase vulnerability to stressful event-induced disease by accentuating stressor appraisal (Cohen & Edwards, 1989). One

can also address whether the type of (or reason for) appraisal plays a role in disease risk. For example, events can be viewed as stressful because of the excessive demands they represent, potential bodily or psychological harm, or because of a high level of challenge (Lazarus & Folkman, 1984). Like stressor measures, measures of appraisal may also assess cumulative perceptions of stress combining responses to multiple events or may assess responses to specific individual events. Measurement of stressor appraisal is discussed in Chapter 6.

Environmental demands that are appraised as stressful are generally thought to influence disease risk through negative emotional responses. Although this association is commonly accepted, there are many interesting unanswered questions about the role of emotion in the stress process. For example, do different stressors produce different emotions? Do the same stressors produce different emotions in different people? Are certain types of emotional response associated with certain types of biological responses or diseases? Do positive and negative emotional responses have the same or opposite effects on biological responses and disease risk? There are several alternative approaches to categorizing emotions that are represented by different measurement techniques. For example, emotions can be categorized on the basis of valence (positive or negative), level of associated arousal, or emotional states such as anxiety, depression, and anger. Choice of the appropriate scale should be based on how close a specific breakdown approximates the question about emotional response that the investigator is concerned with. For example, a study of the role of stress in depressive disorder may focus on a measure of depressed mood, whereas a study of emotion as a pathway linking events to immunity or physical disease may require an assessment of the whole range of emotions. A discussion of the different approaches to the measurement of emotional response to stress is presented in Chapter 7 of this volume.

Similar questions can be asked about physiological responses to stress. Do different stressors have similar influences on responses of the cardiovascular, endocrine, and immune systems? Do the same stressors produce different physiological responses in different people? Do different kinds of appraisal produce differential physiological responses? Endocrine and cardiovascular measures have traditionally been used as biological indicators of stress. As discussed earlier, a variety of hormones have been found to be altered by stressors, appraisals, and negative affective responses, and these hormones have been implicated in the pathogenesis of a wide range of mental and physical disease outcomes. However, there are tremendous differences in the characteristic response of different hormones and in their potential importance for specific disease outcomes. One important response characteristic that is essential in choosing a hormonal measure is the temporal course of the response. Some hormones respond quickly or return to baseline quickly, whereas others have a delayed response or slow recovery. Chapter 8 discusses issues in choosing hormone measures. Cardiovascular measures also differ in their importance as potential precursors of different cardiovascular disorders and in their sensitivity and course of response. These measures are discussed in Chapter 9. Immune measures have not traditionally been viewed as indicators of stress. In fact, it is only recently that stressors, appraisals, and negative affect have been closely tied to immune response (see reviews by Herbert & Cohen, 1993a; 1993b). We include a

chapter on immune response here because of the increased interest in the role of stress associated immune changes in physical and psychiatric disorders (Rabin et al., 1989). Individual immune measures differ substantially in both their associations with other categories of stress measures, and their implications for disease susceptibility. Chapter 10 discusses these differences and suggests appropriate criteria for choosing immune measures.

Up to now, our discussion has focused primarily on the questions that can be asked within each of the traditional perspectives. However, our hope is that the model we have presented will encourage broader views of the stress process. In particular, it suggests the possibility of studies integrating multiple perspectives. At the simplest level, one could test the basic assumptions of the model. For example, do stressful events that result in perceptions of stress increase disease risk through changes in biological responses and health practices? Studies combining perspectives have the potential to answer many of the most important questions about the role of stress in disease.

Methodological and Practical Considerations

We feel that it is inappropriate to discuss measurement as if it were the most important issue in designing studies of the relation between stress and illness. We consider appropriate and unbiased sampling, study designs that maximize the ability to make causal inferences, and appropriate statistical analyses equally important criteria for any study (e.g., Cohen et al., 1986; Kessler, 1987). Our choice, in this volume, to focus on measurement is based on our perception that the literature has failed to deal adequately with issues involved in the appropriate selection of stress measures and that compendia of available instruments are lacking. Our focus on measurement, however, does not imply that we feel that measurement is more important than any of these other considerations. Good measurement does not compensate for weak or poor study design.

Because of limitations of space and our desire to maintain a coherent focus on measurement, we cannot adequately address the issues involved in designing studies of stress and disease. However, there are design issues that influence the choice of a measure and hence need to be raised in this context. The central concern is the importance of the interplay of the stress measure and the outcome. We introduced this problem earlier in the discussion of the need to match the temporal courses of the stress measure and the disease. However, there are other issues as well. A common problem is the use of stress and outcome measures that assess closely related if not identical concepts. This overlap (or confounding) is often a problem in studies of psychiatric symptoms since measures of symptoms and stress appraisal often include similar if not identical items (Dohrenwend & Shrout, 1985). Similarly, stressful life event scales often include serious illness and hospitalization as events. These (and other) items overlap with outcome measures assessing serious physical or psychiatric illness (Rabkin & Struening, 1976). The possibility of this type of bias is maximized in cross-sectional designs, and minimized in prospective designs where a stress measurement is used to predict changes in the outcome from the point

of measurement to a follow-up. It can also be minimized by selecting stress measures that are not confounded with an outcome or by carefully removing items from a scale that are potentially confounded with an outcome. This last procedure must be done with great care since dropping items may alter the psychometric characteristics of the scale or bias the domain that the scale assesses. For example, removing hospitalization decreases the overlap of the life event and disease measures, but also results in the omission of a potentially important and impactful stressful event.

Another problem is the potential for shared response biases that occur when the same type of measurement method is used to assess both stress and outcome. Take, for example, paper-and-pencil self-reports. These types of measures are influenced by a range of idiosyncratic response biases such as responding positively and avoiding extreme responses. If this method is used in assessing both stress and outcome, the relation between these measures will be artificially inflated because it will reflect the identical bias tendencies picked up by both measures. This problem can be avoided by using different methods—for example, a paper-and-pencil stress measure and biological or behavioral outcome.

Realistically, practical limitations and limited resources often prevent the use of measures of stress that one would choose based solely on the conceptual and methodological issues discussed earlier. Primary among these considerations is the amount of *time required from subjects*. Studies employing multiple measures, those conducted over the phone, and those with samples to whom there is limited access or among whom there is limited interest, often require stress measures that take a minimal amount of time. As a consequence, interview measures and detailed self-report measures may not be appropriate in these cases. *Cost of administration* is another important consideration. For example, although interview measures of stressful events provide more sensitive measurement of events and their contexts, they require hours of effort by trained personnel, including the interview process itself as well as labor-intensive coding of materials. Choices based on the cost and time of instrument administration require the researcher to balance these constraints with the need for measurement accuracy.

Moderators of the Relation Between Stressors and Disease Risk

Correlations between life events and illness have rarely risen above .30, suggesting that life events may account for less than 10 percent of the variance in illness. A number of investigators have proposed that relations between stress and illness vary with preexisting vulnerability factors (see reviews by Cohen & Edwards, 1989; Cohen & Wills, 1985; Kessler & McLeod, 1985). That is, differences in social support systems, skills, attitudes, beliefs, and personality characteristics render some persons relatively immune to stress-induced illness and others relatively susceptible. Although this book does not directly address the issues of moderation, interest in moderation does have implications for the choice of stress measurement. As we noted earlier, moderators are often thought to act through their effects of

stress appraisal. For example, a person may not view potentially threatening events as stressful if they believe that their social network will aid them in coping. However, moderators could act at any stage in the stress process. For example, social networks may enforce norms about both emotional and behavioral responses to events that are appraised as stressful. By specifying the point in the stress process where moderation is thought to occur, one can choose the type of stress measure most likely to demonstrate moderation. Again, the choice of the appropriate assessment tool depends on the questions under consideration.

Purpose and Organization of the Book

The purpose of the proposed volume is to serve as a resource for state-of-the-art assessment of stress in studies of physical and psychiatric illness in humans. Our major goal in producing the volume is to aid researchers in making decisions about the appropriate measures to use in specific studies. In this chapter we provided a broad conceptual overview of stress and its relation to psychiatric and physical disorder. We have addressed the conceptualization of stress and how environmental, psychological, and biological approaches can answer different questions about the stress process. The overall theme of this chapter and of the volume is that the measures one selects should reflect the kinds of questions being asked and research designs employed. In Parts II through IV of the volume, we address three broadly defined types of stress measurement: environmental demands, psychological stress, and biological stress. We do not attempt to cover all the measures that might broadly be construed as falling in these categories. Instead we choose those that we believe will be of interest to the broadest possible audience. For example, we do not discuss measures of physical environment such as noise and air pollution, behavior changes such as deficits in task performance, or maladaptive coping responses such as smoking and drinking, or alternative biological stress responses such as skin conductance, eye movement and pupillary diameter, or muscle tension. The exclusion of these measures does not imply that they are not as important as or more important than those we discuss. It merely reflects our decision to address a few of the most central categories of measures in depth rather than try to cover all possible measures superficially.

Each chapter provides a conceptual underpinning of the approach it addresses, discusses the important measures within the approach, the kinds of studies suited to each, and the various costs and benefits of using each alternative measure. The purpose of these chapters is to aid researchers in making decisions about the appropriate measures to use in specific studies. Questions addressed in each chapter include: What kinds of questions about the relation between stress and illness can be answered using this kind of measure? Under what conditions would an investigator use one version of a measure versus another? What are the kinds of study designs in which this particular measure works well? What are the logistical issues that must be considered in using this measure? What is known about the psychometrics of these kinds of measures (and, when appropriate, specific measures)? Are these measures population sensitive, and are appropriate measures available for different

populations? What are the key references for finding the measures, the psychometrics, and instructions for use? Our intent is to provide valuable information for audiences with a wide range of expertise; to aid persons without extensive experience, but at the same time to provide sufficient information for experts to select state-of-the-art measurement instruments.

References

- Ader, R. (1980). Psychosomatic and psychoimmunological research. Presidential Address. *Psychosomatic Medicine*, 42, 307-321.
- American Psychiatric Association. (1987). *Diagnostic and statistical manual of mental disorders* (rev. 3rd ed.). Washington, DC: Author.
- Anisman, H., & Zacharko, R. M. (1992). Depression as a consequence of inadequate neurochemical adaptation in response to stressors. *British Journal of Psychiatry*, 160, 36-43.
- Appley, M. H., & Trumbull, R. (Eds.). (1967). *Psychological stress: Issues in research*. New York: Appleton-Century-Crofts.
- Baum, A., Cohen, L., & Hall, M. (1993). Control and intrusive memories as possible determinants of chronic stress. *Psychosomatic Medicine*, 55, 274-286.
- Baum, A., Grunberg, N. E., & Singer, J. E. (1982). The use of psychological and neuroendocrinological measurements in the study of stress. *Health Psychology*, 1, 217-236.
- Baum, A., Singer, J. E., & Baum, C. S. (1981). Stress and the environment. *Journal of Social Issues*, 37, 4-35.
- Beach, S.R.H., Sandeen, E. E., & O'Leary, K. D. (Eds.). (1990). *Depression in marriage*. New York: Guilford Press.
- Bolger, N., DeLongis, A., Kessler, R. C., & Schilling, E. (1989). The effects of daily stress on negative mood. *Journal of Personality and Social Psychology*, 57, 808-818.
- Brown, G. W., & Harris, T. O. (1978). *Social origins of depression: A study of psychiatric disorders in women*. London: Tavistock.
- Brown, G. W., & Harris, T. O. (1989). *Life events and illness*. New York: Guilford Press.
- Cannon, W. B. (1932). *The wisdom of the body*. New York: W. W. Norton.
- Checkley, S. (1992). Neuroendocrine mechanisms and the precipitation of depression by life events. *British Journal of Psychiatry*, 160, 7-17.
- Cohen, S., & Edwards, J. R. (1989). Personality characteristics as moderators of the relationship between stress and disorder. In R. W. J. Neufeld (Ed.), *Advances in the investigation of psychological stress* (pp. 235-283). New York: Wiley.
- Cohen, S., Evans, G. W., Krantz, D. S., & Stokols, D. (1986). *Behavior, health and environmental stress*. New York: Plenum Press.
- Cohen, S., Kaplan, J. R., & Manuck, S. B. (1994). Social support and coronary heart disease: Underlying psychological and biologic mechanisms. In S. A. Shumaker & S. M. Czajkowski (Eds.), *Social support and cardiovascular disease* (pp. 195-221). New York: Plenum Press.
- Cohen, S., & Matthews, K. A. (1987). Social support, type A behavior and coronary artery disease. *Psychosomatic Medicine*, 49, 325-330.
- Cohen, S., Tyrrell, D.A.J., & Smith, A. P. (1993). Negative life events, perceived stress, negative affect, and susceptibility to the common cold. *Journal of Personality and Social Psychology*, 64, 131-140.
- Cohen, S., & Williamson, G. M. (1988). Perceived stress in a probability sample of the

- United States. In S. Spacapan & S. Oscamp (Eds.), *The social psychology of health* (pp. 31–67). Newbury Park, Cal.: Sage Publications.
- Cohen, S., & Williamson, G. M. (1991). Stress and infectious disease in humans. *Psychological Bulletin*, *109*, 5–24.
- Cohen, S., & Wills, T. A. (1985). Stress, social support and the buffering hypothesis. *Psychological Bulletin*, *98*, 310–357.
- Conway, T. L., Vickers, R. R., Ward, H. W., & Rahe, R. H. (1981). Occupational stress and variation in cigarette, coffee, and alcohol consumption. *Journal of Health & Social Behavior*, *22*, 155–165.
- Dohrenwend, B. S., Askenasy, A. R., Krasnoff, L., & Dohrenwend, B. P. (1978). Exemplification of a method for scaling life events: The PERI Life Events Scale. *Journal of Health and Social Behavior*, *19*, 205–229.
- Dohrenwend, B. P., Raphael, K. G., Schwartz, S., Stueve, A., Skodol, A. (1993). The structural event probe and narrative rating method (SEPRATE) for measuring stressful life events. In L. Goldberger & S. Bresnitz (Eds.), *Handbook of stress: Theoretical and clinical aspects* (2nd ed., pp. 174–199). New York: The Free Press.
- Dohrenwend, B. P., & Shrout, P. (1985). "Hassles" in the conceptualization and measurement of life stress variables. *American Psychologist*, *40*, 780–785.
- Dunbar, H. F. (1954). *Emotions and bodily changes; a survey of literature on psychosomatic interrelationships, 1910–1953* (4th ed.). New York: Columbia University Press.
- Eckenrode, J., & Gore, S. (1990). *Stress between work and family*. New York: Plenum Press.
- Elliott, G. R., & Eisdorfer, C. (Eds.). (1982). *Stress and human health: Analysis and implications of research; a study by the Institute of Medicine, National Academy of Sciences*. New York: Springer.
- Folkins, C. H. (1970). Temporal factors and the cognitive mediators of stress reaction. *Journal of Personality & Social Psychology*, *14*, 173–184.
- Hawkins, N. G., Davies, R., & Holmes, T. H. (1957). Evidence of psychosocial factors in the development of pulmonary tuberculosis. *American Review of Tuberculosis and Pulmonary Diseases*, *75*, 768–780.
- Herbert, T. B., & Cohen, S. (1993a). Depression and immunity: A meta-analytic review. *Psychological Bulletin*, *113*, 472–486.
- Herbert, T. B., & Cohen, S. (1993b). Stress and immunity in humans: A meta-analytic review. *Psychosomatic Medicine*, *55*, 364–379.
- Herbert, T. B., Cohen, S., Marsland, A. L., Bachen, E. A., Rabin, B. S., Muldoon, M. F., & Manuck, S. B. (1994). Cardiovascular reactivity and the course of immune response to an acute psychological stressor. *Psychosomatic Medicine*.
- Herd, J. A. (1986). Neuroendocrine mechanisms in coronary heart disease. In K. A. Matthews et al. (Eds.), *Handbook of stress, reactivity and cardiovascular disease* (pp. 49–70). New York: Wiley.
- Herd, J. A. (1978). Physiological correlates of coronary-prone behavior. In T. M. Dembroski, S. M. Weiss, J. L. Shields, S. G. Haynes, & M. Feinleib (Eds.), *Coronary-prone behavior* (pp. 129–136). New York: Springer-Verlag.
- Hinkle, L. E., Jr., & Wolff, H. G. (1958). Ecologic investigations of the relationship between illness, life experiences and the social environment. *Annals of Internal Medicine*, *49*, 1373–1388.
- Holmes, T. H., & Masuda, M. (1974). Life changes and illness susceptibility. In B. S. Dohrenwend & B. P. Dohrenwend (Eds.), *Stressful life events: Their nature and effects* (pp. 45–72). New York: Wiley.
- Holmes, T. H., & Rahe, R. H. (1967). The social readjustment rating scale. *Journal of Psychosomatic Research*, *11*, 213–218.

- Kessler, R. C. (1987). The interplay of research design strategies and data analysis procedures in evaluation of the effects of stress on health. In S. V. Kasl & C. L. Cooper (Eds.), *Stress and health: Issues in research methodology* (pp. 113–140). New York: Wiley.
- Kessler, R. C., & Magee, W. (1993). Childhood adversity and adult depression. *Psychological Medicine*, 23, 679–690.
- Kessler, R. C., & McLeod, J. D. (1985). Social support and mental health in community samples. In S. Cohen & S. L. Syme (Eds.), *Social support and health* (pp. 219–240). New York: Academic Press.
- Kessler, R. C., Price, R., & Wortman, C. (1985). Social factors in psychopathology: Stress, social support, and coping processes. *Annual Review of Psychology*, 36, 531–572.
- Krantz, D. S., Glass, D. C., Contrada, R., & Miller, N. E. (1981). *Behavior and health*. (National Science Foundation's second five-year outlook on science and technology). Washington, DC: U.S. Government Printing Office.
- Laudenslager, M. L. (1988). The psychobiology of loss: Lessons from humans and nonhuman primates. *Journal of Social Issues*, 44, 19–36.
- Lazarus, R. S. (1966). *Psychological stress and the coping process*. New York: McGraw-Hill.
- Lazarus, R. S. (1975). A cognitively oriented psychologist looks at biofeedback. *American Psychologist*, 30, 553–561.
- Lazarus, R. S. (1977). Cognitive and coping processes in emotion. In A. Monat & R. S. Lazarus (Eds.), *Stress and coping* (pp. 145–158). New York: Columbia University Press.
- Lazarus, R. S. (1981). The stress and coping paradigm. In C. Eisdorfer, D. Cohen, A. Kleinman, & P. Maxim (Eds.), *Models for clinical psychopathology* (pp. 177–214). New York: Spectrum.
- Lazarus, R. S., & Folkman, S. (1984). *Stress, appraisal, and coping*. New York: Springer.
- Levi, L. (Ed.). (1972). *Stress and distress in response to psychosocial stimuli*. New York: Pergamon Press.
- Lief, A. (Ed.). (1948). *The commonsense psychiatry of Dr. Adolf Meyer*. New York: McGraw-Hill.
- Manuck, S., Olsson, G., Hjemdahl, P., & Rehnqvist, N. (1992). Does cardiovascular reactivity to mental stress have prognostic value in postinfarction patients? A pilot study. *Psychosomatic Medicine*, 54, 102–108.
- Mason, J. W. (1975). A historical view of the stress field, Part 2. *Journal of Human Stress*, 1, 22–36.
- McCubbin, J. A., Richardson, J., Obrist, P. A., Kizer, J. S., & Langer, A. W. (1980). *Catecholaminergic and hemodynamic responses to behavioral stress in young adult males*. Paper presented at the Twentieth Annual Meeting of the Society for Psychophysiological Research.
- McGonagle, K. A., & Kessler, R. C. (1990). Chronic stress, acute stress, and depressive symptoms. *American Journal of Community Psychology*, 18, 681–705.
- McGrath, J. E. (Ed.). (1970). *Social and psychological factors in stress*. New York: Holt, Rinehart and Winston.
- Mechanic, D. (1972). Social psychologic factors affecting the presentation of bodily complaints. *New England Journal of Medicine*, 286, 1132–1139.
- Mechanic, D. (1977). Some modes of adaptation: Defense. In A. Monat & R. S. Lazarus (Eds.), *Stress and coping* (pp. 244–257). New York: Columbia University Press.
- Meyer, A. (1951). The life chart and the obligation of specifying positive data in psychopathological diagnosis. In E. E. Winters (Ed.), *The collected papers of Adolf Meyer*,

- Vol. 3: *Medical teaching* (pp. 52–56). Baltimore: The Johns Hopkins University Press.
- Murrell, S., Norris, F., & Hutchins, G. (1984). Distribution and desirability of life events in older adults: Population and policy implications. *Journal of Community Psychology*, *12*, 301–311.
- Neilson, E., Brown, G. W., & Marmot, M. (1989). Myocardial infarction. In G. W. Brown & T. O. Harris (Eds.), *Life events and illness* (pp. 313–342). New York: Guilford Press.
- Newcomb, M., Huba, G., & Bentler, P. (1981). A multidimensional assessment of stressful life events among adolescents: Derivations and correlates. *Journal of Health and Social Behavior*, *22*, 400–414.
- Paykel, E. S. (1974). Life stress and psychiatric disorder: Applications of the clinical approach. In B. S. Dohrenwend & B. P. Dohrenwend (Eds.), *Stressful life events: Their nature and effects* (pp. 135–149). New York: John Wiley.
- Pennebaker, J. W. (1982). *The psychology of physical symptoms*. New York: Springer-Verlag.
- Raab, W. (1971). Cardiotoxic biochemical effects of emotional–environmental stressors—fundamentals of psychocardiology. In L. Levi (Ed.), *Society, stress, and disease* (Vol. 1, pp. 331–337). London: Oxford University Press.
- Rabin, B. S., Cohen, S., Ganguli, R., Lysle, D. T., & Cunnick, J. E. (1989). Bidirectional interaction between the central nervous system and the immune system. *CRC Critical Reviews in Immunology*, *9*, 279–312.
- Rabkin, J. G., & Struening, E. L. (1976). Life events, stress and illness. *Science*, *194*, 1013–1020.
- Rahe, R. H., & Lind, E. (1971). Psychosocial factors and sudden cardiac death: A pilot study. *Journal of Psychosomatic Research*, *15*, 19–24.
- Sachar, E. J. (1975). Twenty-four hour cortisol secretion patterns in depressed and manic patients. *Progress in Brain Research*, *42*, 81.
- Sandler, I., & Ramsay, T. (1980). Dimensional analysis of children's stressful events. *American Journal of Community Psychology*, *8*, 285–302.
- Sarason, I. G., Johnson, J. H., & Siegel, J. M. (1978). Assessing the impact of life changes: Development of the life experiences survey. *Journal of Consulting and Clinical Psychology*, *46*, 932–946.
- Schachter, S., & Singer, J. E. (1962). Cognitive, social and physiological determinants of emotional state. *Psychological Review*, *69*, 379–399.
- Schulz, R., Visintainer, P., & Williamson, G. M. (1990). Psychiatric and physical morbidity effects of caregiving. *Journal of Gerontology: Psychological Sciences*, *45*, P181–P191.
- Selye, H. (1956). *The stress of life*. New York: McGraw-Hill.
- Selye, H. (1974). *Stress without distress*. Philadelphia: J. B. Lippincott.
- Selye, H. (1980). Stress, aging, and retirement. *Journal of Mind and Behavior*, *1*, 93–110.
- Shrout, P. E. (1981). Scaling of stressful life events. In B. S. Dohrenwend & B. P. Dohrenwend (Eds.), *Stressful life events and their contexts* (pp. 29–47). New Brunswick, NJ: Neale Watson Academic Pub.
- Stokes, P. E. (1987). The neuroendocrine measurement of depression. In A. J. Marsella, R.M.A. Hirschfeld, & M. M. Katz (Eds.), *The measurement of depression* (pp. 153–195). New York: Guilford Press.
- Stone, A. A., Reed, B. R., & Neale, J. M. (1987). Changes in daily event frequency precede episodes of physical symptoms. *Journal of Human Stress*, *13*, 70–74.
- Thoits, P. A. (1983). Dimensions of life events that influence psychological distress: An evaluation and synthesis of the literature. In H. B. Kaplan (Ed.), *Psychological stress: Trends in theory and research* (pp. 33–103). New York: Academic Press.

- U.S. Congress, Office of Technology Assessment (September 1992). *The biology of mental disorders (OTA-BA-538)*. Washington, DC: U.S. Government Printing Office.
- Wheaton, B. (1990). Life transitions, role histories, and mental health. *American Sociological Review*, 55, 209-223.
- Wolff, H. G., Wolf, S. G., Jr., & Hare, C. C. (Eds.). (1950). *Life stress and bodily disease*. Baltimore: Williams & Wilkins.