
Stress Processes and the Costs of Coping

Increased public attention in the late 1960s and early 1970s to the importance of the physical environment in shaping our health and behavior stimulated a renewed interest in these issues on the part of social and health scientists. To a great extent, work during this period was stressor-specific. Independent groups of researchers studied noise, population density, temperature variations, and the like with little cross-stressor integration of theory or data. This work was also conducted in relative isolation from theoretical and empirical work on similar problems. For example, even though Lazarus (1966), Janis (1958), and others had described and empirically demonstrated the role of cognition in mediating the associations between stressor exposure and behavior and/or health, those studying environmental stressors tended to focus on stressor intensity alone with only a handful of researchers considering cognitive factors. Since the mid-1970s research on the impact of environmental stressors on behavior and health has become increasingly integrated into the larger field of psychological stress research. This book documents that integration and discusses its implications for the study of stress and coping processes. We develop a broad theoretical framework that emphasizes the commonalities between psychosocial stress and environmental stress as well as the commonalities between various environmental stressors. We focus on issues that we view as central to understanding the stress and coping process and present original data that exemplify and occasionally provide answers to the questions we raise. In order to place this discussion in context, we include critical reviews of the literatures on the impact of environmental stress on cognition, control, and health.

The data that we report are from the Los Angeles Noise Project—a set of naturalistic studies of the effects of aircraft noise on children. Noise project data are presented in the context of discussions of the issues we view as central to the understanding the generic stress-coping process. Our theoretical perspective places particular emphasis on the deleterious effects of the process of coping, the importance of the context in which a stressor occurs, and the role of one's perceptions of the stressful situation as a predictor of health and behavioral outcomes. In many instances, the scope of our theoretical formulations is considerably broader than that of the data. Our hope is that our perspective will stimulate others to fill in missing evidence.

I

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Plan of the Book

This chapter presents an overview of our major theoretical perspective. We begin by outlining the physiological and psychological stress traditions and then present our views on the "costs of coping" with stressful events. Chapter 2 presents a discussion of methodological approaches employed in the study of environmental and psychosocial stressors. We outline a set of criteria for evaluating correlational research and discuss a number of practical problems that arise in the design and implementation of such work. This discussion is followed by a description and rationale for the designs employed in the noise project studies. Statistical techniques used in this work are also summarized.

Each of the remaining chapters of this book reports data from the studies of the Los Angeles Noise Project in the context of broadly based discussions of the relationship of environmental and psychosocial stressors to the outcome measures being considered. Chapters 3, 4, and 5 focus on the effects of the stress process on personal control, health, and attentional processes respectively. These chapters reflect our concern with the cost of the process of coping and provide evidence relevant to a number of the issues discussed in Chapter 1. The chapters include selective/critical reviews of existing research on the effects of stress on control, health, and attention. The noise project data presented in these chapters examine the question of whether mere exposure to the stressor, for example, attending a noisy as opposed to a quiet school, is associated with the outcome variable under consideration. Evidence on the possibility that children habituate or otherwise adapt to noise with prolonged exposure is also examined in this section of the book.

Chapter 6 argues for the importance of viewing stressors within their broader social and physical environmental contexts. We discuss issues one should consider in doing contextual research and suggest criteria for designing contextual based studies. Analyses of noise project data that reflect the role of contextual factors in determining response to noise are presented.

The final chapter summarizes the theoretical points made in the book and the major findings of the noise project. Implications of this work are drawn for a broad range of environmental psychosocial stressors and for policy decisions regarding environmental stress.

Models of Stress

Some Preliminary Definitions

Although the term *stress* is used widely by psychologists, sociologists, epidemiologists, medical practitioners, and lay persons, there appears to be little agreement either within or across disciplines in regard to a scientifically precise definition. Numerous definitions have been provided that vary in the extent to which they emphasize stimulus, response, or intervening mechanism (e.g., Appley

& Trumbull, 1967; Mason, 1975; McGrath, 1970). They also differ in the processes under consideration with some concerned with cognition, others with motivation, and others with physiology.

Like a number of contemporary writers (e.g., Monat & Lazarus, 1977; Singer, 1980), we avoid a semantic controversy by using the term *stress* to refer to a broad area of study. In particular, it is used to refer to the study of situations in which the demands on individuals tax or exceed their adaptive capabilities. Our own emphasis will be on specifying several mechanisms that may be responsible for deleterious effects on behavior and health that sometimes occur in these situations.

Actually, there are not one but several fields of study that are identified as stress research (cf. Singer, 1980). That is, there are a number of research traditions, each with a different emphasis or focus on the problem. Our own conceptions of the stress process have been influenced by work in what can be termed the physiological and psychological stress traditions. These traditions, although related, provide distinct clues as to the effects of psychosocial and environmental stressors on people. The following pages will provide a short description of the orientations of these two traditions and attempt to distill the central assumptions of each. We will then focus on a number of stress processes we feel are important in understanding response to chronic stress.

Physiological Model of Stress

Many of the pathophysiological concomitants of stress are believed to result from activation of the sympathetic-adrenal medullary system (SAM) and the pituitary-adrenocortical axis (PAC). Although detailed descriptions of these two systems and their relationships to one another are beyond the scope of this chapter (see Baum, Singer, & Baum, 1981; Levi, 1972), each will be 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 Cannon's early work on the fight or flight response (Cannon, 1932). Cannon proposed that the SAM system reacts to various emergency states with increased adrenalin (epinephrine) secretion. Although 25 years passed before this increased secretion was actually demonstrated, there is now a large body of evidence indicating increased output of epinephrine and norepinephrine in response to a wide variety of psychosocial stressors (Levi, 1972).

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, 1976). Particularly culpable in this regard is the secretion of the catecholamines, epi-

nephrine and norepinephrine by the adrenal medulla and/or sympathetic nerve endings. Catecholamine discharge is believed to induce many of the pathogenic states associated with psychological stress, including (a) hemodynamic effects, such as increased blood pressure and heart rate (McCubbin *et al.*, 1980); (b) induction of myocardial lesions (Raab, 1971; Raab, Chaplin, & Bajusz, 1964); (c) increased cardiac demand for oxygen (Raab, 1971); and (d) provocation of ventricular arrhythmias believed to lead to sudden death (Herd, 1978).

Pituitary-Adrenocortical Axis: Selye's Model

The hormonal responses of the PAC axis were emphasized in Selye's (e.g., 1956, 1974) influential description of a nonspecific (general) physiological reaction that occurs in response to aversive 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 ACTH, which then activates the adrenal cortex to secrete additional hormones (cortical steroids). 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 cortical steroids 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 stress continues unabated, vulnerable organs (determined by genetic and environmental factors) will break down. This breakdown results in illness and ultimately death.

Recall that Selye argued the *generality* position that any noxious agent, physical or psychological in nature, could mobilize the GAS. In contrast, recent critiques of Selye's model (Lazarus, 1977; Mason, 1975) argue a more specificitylike position, that is, that particular stressors have their own distinct physiological reactions. 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 to stressful situations that occurs only when situations are appraised as stressful. In short, their position is that the nonspecific response is psychologically mediated (e.g., occurs when there is a cognitive appraisal of threat). When conditions are designed to reduce the psychological threat that might be engendered by laboratory procedures, there is no nonspecific reaction to the 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 more recent work (1974, 1980), he acknowledges that there are both specif-

ic as well as general (nonspecific) factors in one's physiological response to a stressor but maintains that the nonspecific response is not always psychologically mediated. He also suggests that the GAS does not occur (or is at least not destructive) under all kinds of stress. For example, he suggests that there may be a pleasant stress of fulfillment and victory and a self-destructive distress of failure, frustration, and hatred.

Two aspects of Selye's model have exerted a profound influence on the development of behavioral and psychological conceptions of the stress process. The first is his assumption that there is a finite amount of adaptive energy that can be invested in one's transaction with a stressor or stressors. A depletion of this energy results in a deleterious impact on the organism. Although Selye's energy was physiological in nature, a number of other theorists (e.g., Cohen, 1978; DuBos, 1965; Glass & Singer, 1972) have proposed similar limitations of psychic energies. A second influential aspect of Selye's theory is his argument that pathogenic effects occur as a result of the body's attempt to cope with the stressor. This assertion is often termed the "adaptive-cost hypothesis." Specifically, the hypothesis suggests that the process of adaptation itself causes deleterious effects that occur either during or after exposure to a stressor. This notion has been borrowed by those interested in behavioral adaptations and their consequences for both behavior and health (e.g., Cohen, 1980; Glass & Singer, 1972; Holmes & Rahe, 1967). Some specific implications of these assumptions for behavioral and psychological formulations of the stress process will be discussed later.

Psychological Model of Stress

The psychological stress tradition places emphasis on the organism's perception and evaluation of the potential harm posed by a stimulus. The perception of threat arises when the demands imposed upon an individual are perceived to exceed his or her felt ability to cope with those demands. This imbalance gives rise to the experience of stress and to a stress response that may be physiological and/or behavioral in nature. Psychological stress then is not defined solely in terms of the stimulus condition or solely in terms of the response variables but rather in terms of the transaction between the person and the environment. It involves interpretation of the meaning of the event and the interpretation of the adequacy of coping resources. In short, the psychological stress tradition assumes that stress arises totally out of persons' perceptions (whether accurate or inaccurate) of their relationship to their environment.

The most influential model of psychological stress has been the one proposed by Lazarus (1966, 1977). The model is depicted in Table 1. In the original formulation of his model, Lazarus (1966) argued that an appraisal of a stimulus as threatening or benign (primary appraisal) occurs between stimulus presentation and stress reaction. For a situation to be deemed threatening, the stimulus must be evaluated as harmful. In his later writings, Lazarus (1977, 1980) argued that a situation will also result in a stress reaction if it is evaluated as a harm/loss, threat, or challenge.

Table 1. Lazarus's Model of Stressor Appraisal

Appraisal stage	Process	Determinant factors
Primary	Determining whether a situation poses a threat, challenge, or potential harm or loss	Perceived features of the stimulus situation Psychological structure of the individual
Secondary	Evaluating resources in order to determine whether one can cope with the situation	Perceived availability of coping resources from either within the individual or from the environment
Reappraisal	Repetition of primary and secondary appraisal as perception of the stressor or available resources changes over time	Change in the situation or person

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 the 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, the 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 impact of stress (i.e., bodily 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 in nature, for example, 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 does not occur only at the onset of a stressful event but will often recur during the course of the event (cf. Folkman, 1970; Lazarus, 1980). 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, for example, the death of a loved one, the impact of even these events can be expected to depend on the individual's appraisal of the threat entailed and 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.

Unlike the physiological stress tradition in which stressor stimuli presumably lead to a restricted range of physiological responses, the psychological tradition has posited a broad range of outcomes as indicators of stress. Typically, response variables in this work include self-reported annoyance or stress, negative-toned affect, changes (usually deficits) in performance of complex tasks, and alterations in interpersonal behavior as well as the physiological changes discussed earlier. Unfortunately, cognitive stress models tend to be vague in their predictions of the particular measures that will be affected in any instance, and the nature of the relationships among these outcome measures.

Effects of the Coping Process

We noted earlier that one of the influential aspects of Selye's model is his argument that there is a cost of the adaptive process. Specifically, the cost refers to the deleterious effects of an encounter with a stressor that occurs as a consequence of the coping processes that are employed (cf. Cohen, 1980; DuBos, 1968; Glass & Singer, 1972). We will call these *secondary*, or *indirect*, effects of one's exposure to a stressor in that they occur because of the coping process rather than because of the stressor itself.

In the following section, we suggest a number of conditions under which both behavioral and psychological costs of the adaptive process occur. Although, to some degree, these theoretical speculations have been shaped by the physiological and psychological stress traditions discussed previously, they diverge from these traditions in a number of significant ways. First, let us consider our divergence from Selye's model. The adaptive process discussed by Selye is both automatic and nonspecific. By automatic, we mean that the PAC responses are not voluntary strategies chosen by the subject but are rather part and parcel of a biologically programmed reaction to threatening events. In contrast, many of the coping strategies that will be discussed in the following pages are voluntary.

The nonspecific nature of the physiological reactions studied by Selye was discussed earlier. As noted before, the nonspecific aspects of one's physiological response to a stressor may be elicited by an emotional reaction to the stressor rather than by the physical stressor itself. Unlike Selye's model, several of the strategies discussed in this section are specific in nature. That is, they are strategies that are developed to cope with a particular stressful event. It is possible, however, that

these various coping strategies share some common underlying physiological outcome(s).

Finally, we note an important divergence we will make from the psychological stress tradition. Recall that the psychological tradition suggests that people experience stress when they appraise a situation as threatening or otherwise demanding and when they perceive that they lack the ability to adequately cope. We will argue, however, that secondary effects of stressor exposure can occur when a situation is appraised as potentially threatening and one perceives adequate coping responses are available. Specifically, we will propose that successful (as well as unsuccessful) coping can have a deleterious impact on both one's behavior and health.

Successful Coping

First, consider situations in which a person is successfully coping, that is, the direct (primary) effects of the stressor are neutralized by an apparently adjustive coping strategy. There are (at least) three types of secondary effects that can occur for those involved in an apparently successful adaptation to their environment. These effects can be labeled (a) cumulative fatigue effects; (b) overgeneralization of a coping strategy; and (c) coping side effects.

Cumulative Fatigue Effects. Many previous theoretical discussions of the adaptive cost hypothesis have proposed what we term a *cumulative fatigue effect*. This concept assumes that a person has a limited amount of energy (whether biological or psychological in nature) and that prolonged coping demands deplete this energy supply. Recall, for example, that Selye (1975) asserted that after prolonged exposure to a stressor, one's adaptive biological reserves are drained, resistance breaks down, and exhaustion sets in. Symptoms appear, and, if stress continues unabated, vulnerable organs will break down, and ultimately death will ensue. Glass and Singer (1972) similarly suggest that the process of adaptation requires cognitive effort. This work includes searching for appropriate coping responses and/or attempting to define the stimulus. Moreover, they assumed that the work required to adapt to unpredictable and uncontrollable stressors was substantially greater than that required to adapt to predictable and controllable stimulation. Glass and Singer further argued that increased adaptive effort depletes one's available energies and thus results in deficits on subsequent task performance.

Cohen (1978) similarly argued that there were psychic costs of prolonged exposure to unpredictable and uncontrollable stressors, but he described these losses in terms of one's attentional capacities or information-processing abilities. Specifically, he suggested that exposure to unpredictable, uncontrollable stressors substantially increases demands on one's attentional capacity. This increased demand might occur because individuals are required to continually monitor threatening stimuli to evaluate their adaptive significance and decide on appropriate coping responses (cf. Lazarus, 1966). Increased demand may also occur because of effort required in inhibiting response to a distracting stimulus. Cohen further asserts that

an individual's attentional capacity is not fixed but shrinks when there are prolonged demands. This shrinkage, or "cognitive fatigue," presumably increases with both the attentional load (information rate) of an activity and the duration of an activity. Thus, prolonged exposure to an environmental stressor and/or to a high information rate task should result in an insufficient reserve of attention to perform demanding tasks.

Others (Basowitz, Persky, Korchin, & Grinker, 1955; Dubos, 1965; Wohlwill, 1966) make similar claims concerning the depletion of psychological reserves over the course of one's exposure to a stressor. These various models have been used to explain poststress deficits in task performance (e.g., Cohen, 1980; Glass & Singer, 1972), poststress insensitivity toward others including increased aggression and decreased helping (e.g., Cohen, 1978; Cohen & Spacapan, 1978), and the increased deficits that occur over time on demanding tasks performed under stress (Cohen, 1978; Wilkinson, 1969).

The reduced capacity to cope with environmental demands for those *already coping* with life stressors may also be interpreted as a result of cumulative fatigue. An example is provided by a study of effects of smog exposure on a large sample of Los Angeles residence (Evans *et al.*, 1982). Evans and his colleagues found that higher smog levels resulted in increased risk of mental health problems *only* for those who had recently experienced a stressful life event.

Overgeneralization. Another secondary effect of successful coping is the overgeneralization of a coping strategy. This effect occurs when a strategy employed to deal with a stressor persists even in situations where a person is not confronted with the offending stressor. Overgeneralization results in deleterious effects if the coping responses are inappropriate in other situations in which they are employed. The persistent use of a particular strategy may be due to an overlearning of the coping response.

Overgeneralization has been shown to occur both in laboratory and field settings. For example, Epstein and Karlin (1975) reported that the competitiveness and withdrawal displayed by men during crowding and the cooperativeness and cohesiveness displayed by women during crowding persisted into the poststress session. Baum and Valins (1977) similarly reported that students from dormitories whose architectural designs fostered high levels of forced interaction made more active attempts to avoid the possibility of contact with a stranger outside of the dormitory than students from dormitories with lower levels of interaction. Thus, an avoidance response that presumably developed as an attempt to cope with dormitory life persisted outside of the dormitory setting.

Evidence on the use of denial in coping with heart attacks (myocardial infarctions) similarly suggests that the persistence of a coping strategy can be detrimental after the stressor is terminated (cf. Krantz & Schulz, 1980). Several studies have found that deniers show less anxiety when first admitted to the coronary care unit (CCU) than those not employing a denial strategy (e.g., Froese, Hackett, & Cassem, 1974; Gentry, Foster, & Haney, 1972). These patients deny that they have had a heart attack, often protest detention in the CCU, and insist on returning to normal

activities. Although useful during the initial phases of the attack, use of denial has been related to rehabilitation problems due to long-term resistance to following medical instructions (e.g., Croog, Shapiro, & Levine, 1971; Garrity, 1975). In sum, use of denial may make for better coping with the early stress of illness in the CCU. However, in the long term, patients may endanger their chances of recovery by ignoring medical recommendations that are important for satisfactory rehabilitation.

An example of overgeneralization of a strategy employed to cope with noise was proposed by Deutsch (1964). She suggested that children reared in a noisy environment eventually become inattentive to acoustic cues; that is, they learn to "tune out" sound. In tuning out his or her noisy environment, a child is not likely to distinguish between speech-relevant and speech-irrelevant sounds. Thus, he or she will lack experience with appropriate speech cues and will generally show an inability to recognize relevant sounds and their referents. The inability to discriminate sound could account, in part, for subsequent problems in learning to read. A child who cannot readily discriminate basic speech sounds faces a difficult task in learning to associate these sounds with their appropriate signs. A number of studies have found auditory discrimination and reading deficits on the part of children living and/or attending school in noisy environments (e.g., Bronzaft & McCarthy, 1975; Cohen, Glass, & Singer, 1973; Cohen, Evans, Krantz, Stokols, & Kelly, 1980). However, it is still unclear whether these deficits are attributable to a tuning-out strategy or to the noise masking parent speech (cf. Cohen & Weinstein, 1982).

Although the data just described are limited to situations in which the coping strategy is one that develops as a response to a particular stressor (e.g., withdrawal as a response to crowding), it is possible that nonspecific strategies that are used to cope with a wide range of stressors persist outside of stressor exposure (cf. Milgram, 1970). For example, the strategy of focusing one's attention on the essential aspects of a task during stressor exposure (e.g., Broadbent, 1971; Hockey, 1970; Wachtel, 1968) may persist even after exposure is terminated. Deficits on complex tasks administered after exposure to unpredictable and uncontrollable stressors including noise (e.g., Glass & Singer, 1972; Sherrod, Hage, Halpern, & Moore, 1977), electric shock (e.g., Glass *et al.*, 1973), and crowding (e.g., Evans, 1979; Sherrod, 1974) are explicable within this context (cf. Cohen, 1980).

Coping Side Effects. The third form of the secondary effect occurs when coping behaviors, which were successful in ameliorating the possible effects of the stressor, are detrimental to the individual in other ways. We have labeled this phenomenon *coping side effects*. We will discuss two ways in which the act of coping itself may be detrimental: (a) the coping process directly produces pathogenic effects and (b) coping behaviors interfere with health maintenance.

The first approach suggests that direct coping may be effective in initially removing a threat, but at the same time, it may itself produce pathogenic physiological reaction (Obrist, 1981). For example, Obrist reported that situations in which active coping or behavioral adjustment are required result in cardiovascular responses, for example, elevated blood pressure and increased water retention by the

kidneys, above the level that is efficient for the body's metabolic needs. Active coping situations are those in which the organism can exert some control over a noxious stimulus by some aspect of his or her behavior, for example, attempting to escape or avoid shock by pressing a key. By contrast, these physiological responses do not seem to be elicited in similar intensity or kind in stressful situations where the individual copes passively or intrapsychically and does not take direct action to attempt to control the situation. In related studies, active coping with a stressor has also been found to lead to increased specific discharge of norepinephrine (e.g., Elmadjian, 1963; Elmadjian, Hope, & Larson, 1958). Moreover, norepinephrine levels in blood and urine remain elevated in subjects engaged in active efforts to escape or avoid stressors (Contrada *et al.*, 1982; Frankenhaeuser, 1971; Frankenhaeuser & Rissler, 1970; Weiss, Stone, & Harrell, 1970).

Incidentally, this notion of active coping seems applicable to descriptions of those psychosocial conditions (e.g., rapid cultural change, migration, socioeconomic mobility) shown by epidemiologic research to be associated with blood pressure elevations in some human populations (Gutmann & Benson, 1971; Krantz *et al.*, 1981). As Gutmann and Benson have pointed out, this research literature similarly suggests that situations that require active coping, that is, continuous behavioral adjustment, likely play a causal role in hypertension. Although there is no convincing evidence at this point, it has also been proposed that the effects of stressful life events on the etiology of disease are attributable to the effort of coping with life change (cf. Holmes & Rahe, 1967).

Recent work (e.g., Manuck, Harvey, Lechleiter, & Neal, 1978; Solomon, Holmes, & McCaul, 1980) suggests that the detrimental effects of active coping occur under conditions of high- but not low-effort coping. In particular, when control is easy to exercise, subjects report a level of anxiety and display physiological arousal patterns that are similar to subjects who are not threatened by an aversive event. However, when control is difficult to exercise, subjects report a level of anxiety and display physiological response patterns that are like those of subjects threatened with an aversive event who are unable to exercise control over it.

There is recent evidence that there are predictable *individual differences* in physiological (sympathetic) reactivity for those involved in high-effort coping. Specifically, these data come from experiments comparing Type A's—those who respond to challenge by showing competitive drive, impatience, hostility, and vigorous voice characteristics—with those not showing this behavioral style (Type B's). First, a number of studies have found that A's respond with higher systolic blood pressure than B's when performing a challenging task (e.g., Dembroski *et al.*, 1978; Glass *et al.*, 1980). If we assume that challenge leads to high-effort coping, the greater elevation of blood pressure on the part of A's may be explicable in effort terms. Second, two recent studies compared A's and B's in an experimental situation where subjects exerted effort to control an aversive stimulus. Pittner, Houston, and Spiridigliozze (1983) found that under high-effort control conditions, Type A subjects had greater elevations in blood pressure and pulse rate than did Type B subjects. In low-effort control conditions these differences were absent or

weaker. Contrada *et al.* (1984) similarly reported evidence of greater plasma nor-epinephrine secretion among A's than among B's when effortful control was required. Hence, Type A's may show greater physiological reactivity when effortfully coping, and hence coping A's may be under greater risk for illness than coping B's.

A further refinement of the conditions under which active coping results in pathogenic physiological responses is suggested by Weiss's (e.g., 1977) discussion of the conditions under which stress produces ulcers. Like the theorist discussed previously, Weiss suggested that effortful coping, in his terms a high rate of coping responses, is related to increased pathology. He also suggests, however, that pathogenic effects will occur only when one's coping responses do not continually produce feedback that one is manipulating his or her environment. The theory's first proposition is that the more coping responses one emits, the greater is the ulcerogenic stress. Note that Weiss does not say that increased responses cause increased ulceration, only that they rise together. Although coping may cause ulceration, a strong alternative would view both increased response rate and increased ulcerations as a consequence of a high level of anxiety or fear. The theory's second proposition is that if the response immediately produces appropriate feedback—brings about stimuli that are not associated with the stressful situation—ulcerogenic stress will not occur. Weiss's emphasis is on immediate changes in one's environment following each coping attempt. Hence, those coping attempts that are successful in avoiding an aversive situation are considered stressful unless there is continuous feedback signaling the effectiveness of each coping response.

Evidence supporting Weiss's theory is provided by a set of experiments in which he manipulated whether or not coping (lever-pressing) rats received relevant feedback. Ulceration was low for those rats who received feedback, higher for those who coped and received no feedback, and highest for those who coped but received inappropriate feedback (short, low-intensity shocks). Studies in which number of coping responses and rate of feedback are independently manipulated by the experimenter would determine whether the effort involved in coping is the cause of the pathogenic response (as mentioned before there is evidence that this is true), and whether feedback does cancel out this effect.

Cassel's (1975) analysis of social stress and its implications for the etiology of disease resulted in a similar emphasis on coping and feedback. In Cassel's words: "Stressful social situations might be those in which the actor is not receiving adequate evidence [feedback] that his actions are leading to anticipated consequences" (p. 543). He suggests that these stressful situations are particularly likely to occur when individuals are unfamiliar with cues and expectations of the society in which they live. Thus, like Guttman and Benson (1971), Cassel would expect that migrants, those undergoing rapid social change or social disorganization, would be at risk for disease. He argues, however, that it is not only the effortful adaption that results in increased pathology but rather the adaptive efforts in the absence of information about their effectiveness.

A second side effect of successful coping occurs when the coping behaviors one engages in are themselves detrimental to the maintenance of one's health and

well-being. For example, increased coping efforts may result in increased allocation of time to a particular problem. Thus, one might spend long hours at work attempting to complete a demanding project. Although furthering short-term goals, this kind of coping is detrimental to one's health to the extent that it interferes with proper nutrition, exercise, or the practice of hygienic behaviors. Cigarette smoking, coffee drinking, drug consumption, and overeating, which often increase in times of stress, are salient behaviors in this category (e.g., Conway, Vickers, Ward, & Rahe, 1981; Rahe & Arthur, 1978). Smoking, while often resulting in self-reported decreases in anxiety or fear (actually sympathetic increases, Schachter, Kozlowski, & Silverman, 1977), has been implicated as a risk factor for three leading causes of death in the United States (heart disease, cancer, stroke). Similarly, alcohol consumption, although providing a temporary release from stress, is also implicated as a risk factor for physical diseases and social pathology.

Summary of the Effects of Successful Coping. In sum, we have outlined three kinds of effects of successful coping. First, the coping process may drain one's cognitive or physiological energies. Second, a strategy employed to deal with a stressor may persist even in situations where a person is not confronted with the offending stressor. Finally, coping behaviors that are successful in ameliorating the possible effects of a stressor can be detrimental to individuals in other ways.

When Coping Fails

There are also secondary effects that occur when one's attempts to cope with an aversive event fail. These effects occur either as a cost of actively coping with the stressor or because of one's perception of uncontrollability and failure *per se*. They are separate from any primary effects that may occur as a result of being unable to cope with the demands of a specific stressor.

Costs of Active Coping. The cumulative fatigue effects and coping side effects discussed before in the context of the costs of successful coping are also potential effects of failure to cope. These effects would occur to the extent that there is a prolonged active and effortful attempt to cope. Clearly, cognitive fatigue, pathogenic effects of active coping, and the interference of coping processes with health maintenance should occur when one engages in prolonged effortful coping irrespective of whether the coping behavior is successful or not. In cases where the ineffectiveness of coping attempts becomes immediately apparent and active coping is terminated, these mechanisms would not come into play.

A form of overgeneralization may also be operative in situations where coping fails. Instead of overgeneralizing successful coping strategies, it is also possible to overgeneralize the expectation that coping is ineffective. This perspective has been developed in learned helplessness theory and is discussed in that context next.

Learned Helplessness Theory. Specific effects of a continual inability to control important events are suggested by Seligman's (1975) learned helplessness theory. Seligman has proposed that when persons (or animals) learn that their reinforcements are independent of their responses (i.e., that they lack control over their

outcomes), this learning undermines their motivation to initiate further instrumental responses, interferes with learning that other outcomes are controllable, and causes a depression of mood. Extreme effects of helplessness presumably include fear, clinical depression, disease, and even death. A later modification of learned helplessness theory suggests that the impact of helplessness on these outcomes increases when the individual lacks a requisite controlling response that is available to others (personal helplessness), and when persons attribute their helplessness to long-lived and recurrent (stable) causes and to causes that are important for a wide range of outcomes (global) (Abramson, Seligman, & Teasdale, 1978).

In general, laboratory studies of learned helplessness in humans provide strong evidence that depriving someone of control over important (in the context of the experiment) outcomes ultimately (often after an initial period of attempting to reassert control, e.g., Roth & Kubal, 1975; Wortman & Brehm, 1975) results in deficits on subsequently administered tasks (e.g., Cohen, Rothbart, & Phillips, 1976; Hiroto & Seligman, 1975; Krantz, Glass, & Snyder, 1974). These deficits presumably manifest negative transfer that inhibits one from learning that their outcomes are under his or her control as well as a decrease in motivation. Similar studies have indicated that those lacking control over noxious stimulation exhibit increases in reported levels of depression, anxiety, and hostility (Gatchel, Paulus, & Maples, 1975; Miller & Seligman, 1975). This depressed mood, however, apparently occurs only when subjects perceive that they have failed the task (Griffith, 1977). Although we will not pursue alternative explanations here, there is evidence suggesting the possibility that the poor performance in the helplessness group may occur because of the distracting effects of anxiety (Coyne, Metalsky, & Lavelle, 1980; Lavelle, Metalsky, & Coyne, 1979), or that poor performance is the result of a low-effort strategy used to avoid making a self-attribution of low ability (Frankel & Snyder, 1978; Snyder, Smoller, Strenta, & Frankel, 1981).

Helplessness has been manipulated in the laboratory by both the administration of insoluble tasks and by exposing subjects to inescapable noxious stimuli. These two paradigms are presumed to have the same effect because in both cases the probability of various response outcomes (correct or incorrect, noxious stimulation or no noxious stimulation) is independent of one's responses. Both paradigms use a triadic design including groups in which a person's performance on a test phase task is assessed after a pretreatment task in which either (a) reinforcement is contingent on a subject's behavior; (b) reinforcement is not contingent on a subject's behavior; and (c) there is no pretreatment.

An experiment by Hiroto (1974) serves as an example of the helplessness paradigm. Subjects in an escape group received loud noise that they learned to turn off by button pushing. The subjects in the inescapable group received the same noise, but the noise was independent of their responding. A third group received no noise. After the pretreatment session, subjects were again exposed to noise but could escape the loud bursts by mastering a hand shuttle-box task. In order to escape noise in this situation, an individual had only to move his or her hand from one side to the other. Both the no-noise group and the escape group learned readily to shuttle

with their hands. However, those in the inescapable group generally failed to escape and avoid, with many sitting passively and taking the aversive noise.

Deficits on tasks administered after an experience of uncontrollability are not typically limited to the original situation in which responding was ineffective but rather may generalize to a wide variety of new situations. For example, failure to control noxious stimuli can lead to later difficulties in solving mental tasks, and failures on cognitive tasks can interfere with instrumental responding to noxious stimuli (Hiroto & Seligman, 1975).

Most research on the effects of helplessness on physiology and behavior has emphasized deficits in task performance and indications of "giving up" that occur subsequent to a helplessness treatment. In real life, this apparent reluctance to cope with new demands could be detrimental to both health maintenance and to the achievement of challenging goals. In terms of health maintenance, one could react to the onset of illness and even one's own symptoms as if they were uncontrollable. This may well result in failures to perform routine health care behaviors, for example, brushing one's teeth, seeking medical care, or complying with treatment and rehabilitation regimes (e.g., Krantz & Schulz, 1980; Taylor, 1979). Challenging situations on the job in school or at home may similarly be met by a lack of active coping. Performance on difficult tasks would thus be expected to suffer, possibly reinforcing one's previous inappropriate expectancy that his or her outcomes are not contingent on his or her behavior.

Physiological Concomitants of Helplessness. The psychological state of helplessness may play a more direct role in the etiology of disease states. For example, it has been suggested that helplessness inhibits the operation of the immune system. This hypothesis is supported by experiments in which tumors have been found to grow more quickly and to be rejected less often in mice given inescapable shock than in mice given escapable shock or no shock (Sklar & Anisman, 1979; Visintainer, Volpicelli, & Seligman, 1982). Helplessness has also been proposed as a possible cause of sudden coronary death (cf. Engel, 1968; Green, Goldstein, & Moss, 1972) and of increased risk of heart disease for those displaying Type A behavior patterns, that is, competitive drive, hostility, and impatience (Glass, 1977). However, the physiological bases for these assertions have not been clearly spelled out, and further research is needed in order to determine mechanisms translating psychological states of helplessness into physiological effects that culminate in disease.

There is, however, research on animal response to hopeless situations that may provide a clue to a physiological mechanism linking helplessness to sudden death. Richter (1957) found that wild rats whose whiskers were cut off apparently gave up any attempt to survive an emersion in a water bath. The dewhiskered rats swam for less than 5 minutes before dying, whereas rats whose whiskers were not removed swam for 6 to 8 hours before drowning. Richter reasoned that the wild rats' whiskers were an important source of contact with their environment and that their removal totally deprived the rats of the perception that they could control their outcomes. This assertion was lent credence by Richter's demonstration that rats

who were given a "hopelessness pretraining," in which they received several experiences of being immersed in water, subsequently did not give up and die but swam for 6 to 8 hours. Richter found that death in the hopeless rats was preceded by a decrease in arousal, including a slowing of heart rate and lowering of body temperature. He interpreted this as evidence that death was due to *underarousal*, possibly brought on by an overstimulation of the parasympathetic system, rather than to overarousal due to the *overactivation* of the pituitary-adrenal cortical axis (as outlined by Selye) and/or the sympathetic-adrenal-medullary system. Others have similarly found that swimming deaths in rats are associated with changes in cardiovascular function indicative of overstimulation of the parasympathetic system (e.g., Binik, Theriault, & Shustack, 1977). However, the interpretation that these changes are induced by helplessness has been challenged and a number of viable alternative explanations suggested (cf. Binik, Deikel, Theriault, Shustack, & Balthazard, 1979; Hughes & Lynch, 1978). Studies directly manipulating helplessness in rats are mixed in their support for a helplessness-linked reduction of arousal. Although Weiss and his colleagues (Weiss, Glazer, & Pohorecky, 1977) found that giving up in the face of an uncontrollable stressor was associated with substantial depletions in norepinephrine, Binik *et al.* (1979) failed to find any increase in swimming deaths or decrease in heart rate during swimming trials for rats pretreated with uncontrollable shock.

Human studies in which helplessness was manipulated in the laboratory have found a similar pattern of decreased sympathetic arousal (after an initial period of increased response) among people deprived of control over their outcomes. These studies indicate that subjects who initially expect that they can control an aversive agent but who learn over the course of the experiment that they are helpless show lower levels of arousal after the initial trials than subjects provided with an effective coping response. For example, as discussed earlier, Obrist *et al.* (1978) found that subjects with a hard coping task showed higher arousal than those with an easy task. They also report that after the first few trials, subjects working on an impossible coping task, designed to foster "giving up," had levels of systolic blood pressure, heart rate, and carotid dP/dt (how quickly a heart beat is transmitted as a pulse rate through the body) equivalent to the easy-task group. Apparently, after learning that their coping attempts were unsuccessful, both their coping efforts and associated sympathetic activation decreased. Further support for lowered physiological arousal in helpless subjects is reported by Gatchel and Proctor (1976). After the first few trials, subjects treated with a standard helplessness pretreatment—a series of inescapable tones—demonstrated lower tonic skin conductance and smaller phasic skin-conductance responses than a group pretreated with escapable tones. The helplessness group did, however, show more spontaneous electrodermal activity, the measure most closely correlated with self-reported arousal. Similar reports of relatively low levels of phasic skin-conductance response on the part of subjects who initially expected that they could control an aversive stimulus but were unable to do so (helplessness condition) as opposed to those who were able to escape have been reported by Gatchel, McKinney, and Koebernick (1977) and Krantz, Glass, and

Snyder (1974). The levels of arousal reported in the studies discussed here were not below those expected in normally functioning people. Thus, this work cannot be viewed as evidence for a pathogenic response but only as supportive of the hypothesis that arousal decreases after prolonged exposure to a helplessness-inducing task or situation.

In sum, there is accumulating evidence that the experience of helplessness is associated with relatively low levels of arousal, which is possibly attributable to reaction of the parasympathetic system. Moreover, extreme instances of such a response may be associated with sudden death as well as susceptibility to disease. This work is, however, merely suggestive, and further research on physiological response to helplessness is required before any definitive conclusions are possible. Overall, it is clear that one's perceived failure to cope with a stressor may result in behavior and physiological changes inimical to the well-being of the organism. Hence we apparently suffer when coping fails as well as when it succeeds.

Helplessness Resulting in Alternative Coping Modes. Learned helplessness theory views helplessness as a state resulting from the inability to *directly* cope with a stressful event. This approach does not address the possibility that persons unable to directly cope may still be able to handle the affective consequences of that event (e.g., Lazarus & Folkman, 1984). It seems likely that the perception of helplessness, although inhibiting further problem-focused coping, may alternatively elicit emotional-focused coping. For instance, Evans, Jacobs, and Frager (1982) found that long-term residents of air polluted neighborhoods did not directly confront the problem but instead tended to adopt emotionally focused coping strategies such as rationalization or denial. In comparison to recently exposed persons, they were more likely to underestimate the extent of the smog problem as well as its impact on their own health. These same residents also felt that community smog abatement programs like car pooling would not do any good. Moreover, long-term residents were less likely to follow health advisories during smog episodes and did not restrict outdoor activities. Hence, many of their actions were consistent with the helplessness model and at the same time suggest that they did not become totally passive but rather adopted alternative emotionally focused modes of coping. An interesting question that is as yet uninvestigated is whether health outcomes differ for persons who just fail at direct coping and those who fail at both problem-focused (direct) and emotionally focused coping.

Passivity in the Face of Stressful Events

Our discussions of successful and unsuccessful coping were concerned with situations where a person actively attempts to cope with a stressful event. We can also ask what happens when a situation is structured such that one responds passively to a stressor, that is, does not engage in any direct coping behavior. The issue of passive exposure to a stressor is both conceptually and empirically controversial and, as is indicated later, the present state of the literature does not lend itself to clear conclusions.

Let us first consider research on the aftereffects of stress. This work focuses on the important difference between instances of passive exposure in which there is an expectancy that, if active coping was engaged in, it would be successful and instances in which there is no reason to expect that coping efforts would be fruitful. Specifically, Glass and Singer (1972) and others (see review by Cohen, 1980) have found that persons passively exposed to a stressor who are not led to expect that it is possible to escape or avoid it (no perceived control group) perform more poorly on tasks administered after stressor termination than a group not exposed to the stressor. However, when a passive exposure group is led to expect that they can terminate the stressor if they desire (perceived control group), even though they do not actually implement that control, the deleterious effects of stressor exposure are partly or wholly ameliorated. These data suggest that passive exposure is deleterious, at least in terms of performance on subsequent task, only if one's passivity is forced on him or her by the structure of the situation, that is, there is no choice of whether or not one copes.

Seligman (1975) has interpreted these data as supportive of learned helplessness. Before discussing Seligman's analysis, it is important to emphasize the differences between the "aftereffects" and "learned helplessness" paradigms. In the helplessness studies, subjects in the control and no-control groups both expect (because of the instructions) that it is possible to control the situation. Subjects with control actively and successfully cope. Those without control cope initially but eventually learn that they are not effectively manipulating their environment. In the aftereffect studies, no one actually copes. The perceived control group is told that there is an effective coping strategy available to them, but they do not actually use it. The no-perceived-control group is never given the expectation that coping is possible. According to Seligman (also Glass & Singer, 1972), passive exposure without perceived control presumably results in the deterioration of poststress task performance because of the motivational and cognitive deficits associated with helplessness (see Cohen, 1980, for alternative explanations of aftereffects).

It is paradoxical that the helplessness literature itself provides data that are apparently inconsistent with the helplessness interpretation of the aftereffects phenomenon. Several learned helplessness studies, in fact, demonstrate that helplessness is not produced in subjects who are passively exposed to noxious stimuli. Moreover, these studies employ a passive exposure condition that is strikingly similar to Glass and Singer's no-perceived-control group. In work reported by Gatchel *et al.* (1977), Gatchel and Proctor (1976), and Hiroto and Seligman (1975), a condition is included in which subjects are passively exposed to a noxious stimulus without being told that it is possible to escape or avoid it. In all cases, subjects in this condition do not show the poststimulation deficits in performance found in the inescapable stressor (helplessness) condition but in fact perform like subjects in the escapable condition.

Because of the difference between the aftereffects and helplessness paradigms, it is difficult to assess whether failure to find poststimulation deficits in the passive exposure condition are inconsistent with results from aftereffect studies. For exam-

ple, subjects may merely perform better under perceived control than under the escape condition in helplessness studies. Unfortunately, there are no experiments at this point that include all of the relevant conditions for comparison of the after-effects and helplessness paradigms. (These would include two groups from the helplessness paradigm—those who actively cope and succeed, those who actively cope and fail, and two groups from the aftereffects paradigm—those who do not cope but expect coping would be successful and those who do not cope and do not expect that coping would be successful.) Hence, it is still uncertain whether a passive response to a stressor is adjustive or not.

When Not Coping Is Adjustive. There are some important implications of the research and theory discussed previously that deserve further attention. Earlier, we pointed out that effortful coping without feedback is associated with increased pathogenesis in rats and increased sympathetic response (possibly a precursor of pathogenesis) in humans. One corollary of this finding is that in the face of an objectively uncontrollable stressor, it is better to remain passive than it is to actively cope. Because there would be little if any feedback indicating that one is successfully manipulating his or her environment in an uncontrollable situation, the active copers would presumably suffer, whereas the passive copers would not. On the other hand, accepting a passive strategy may itself be a sign of helplessness or may eventually result in feelings of helplessness. As noted earlier, such feelings are themselves linked to disease outcomes and behavioral deficits.

The preceding analysis suggests that one cannot win in the face of an uncontrollable stressor. That is, if you actively cope, you increase your risk of disease because of an overarousal of the sympathetic system. If you fail to cope, you become (or already are) helpless and are at higher risk for the behavioral and health consequences of such a state. The possibility does exist, however, that in some cases passivity in the face of an uncontrollable stressor is adjustive (not increasing one's risk for disease or behavioral disorder). These are situations in which one chooses to remain passive as opposed to having his or her passivity forced on them because they have no effective active strategies. One may choose a passive strategy because not responding is perceived to be the most effective (or at least temporarily useful) way of dealing with the situation. In other words, when not responding is perceived by the subject as a coping strategy or chosen as a temporary state that the subject perceives can be replaced at any time with an effective coping strategy (e.g., perceived control conditions in the aftereffects paradigm), it is an effective means of coping with an uncontrollable stressor. When not responding is perceived by a person as a state of not coping, a state that if one had a choice would be avoided (e.g., no-escape condition in the helplessness paradigm), it has the detrimental effects associated with helplessness. Another way of viewing this distinction is whether or not passivity is a preferred state. When it is, it is an effective means of coping and when it is not, it results in helplessness-related effects.

Choosing not to directly cope with a stressor does not necessarily imply total passivity. One strategy that may be employed by those choosing not to directly cope is to accept the event and cope instead with its emotional impact (e.g., Lazarus &

Folkman, 1984). For example, Baum and his colleagues (Baum, Fleming, & Singer, 1983; Collins, Baum, & Singer, 1983) studied persons living near the Three Mile Island nuclear plant. These persons were viewed as confronting an uncontrollable stressor because a sharp decline in property values made moving (the direct response with the most likelihood of being effective) impossible for most. Baum found that over the long run, those using emotionally focused coping strategies reported less distress and fewer psychological symptoms than those using problem-focused strategies. Hence, it is possible that the effectiveness of passivity (a lack of direct coping attempts) in the face of an uncontrollable stressor may be partly or wholly due to refocusing efforts on an aspect of the experience that is controllable. It is to be hoped that future research on the relationship between passivity in the face of uncontrollable stressors will test the predictive validity of the distinction between preferred and nonpreferred states and examine the role of emotionally focused coping for those preferring not to directly cope with the stressful event.

Dispositional Factors Mediating Coping Costs

Some individuals are more likely than others to cope in ways that accrue costs. It is beyond the scope of this chapter to outline the relationship between personality factors, individual coping styles, and the coping costs discussed here, but the mention of a few salient possibilities should clarify our point. We mentioned earlier that Type A's may respond to challenge with greater effort and the concomitant physiological reactivity. Hence, A's may be more susceptible to arousal induced by effortful coping. Coping flexibility may be similarly important in preventing other coping costs. The importance of flexibility has been raised by Pearlin and Schooler (1978) who found that successful copers are those who are able to adjust their behavioral and emotional repertoires to fit the changing demands of their diverse life domains. Perhaps individuals who can bring a variety of coping strategies to bear on a particular stressor may be less prone to an overgeneralization of inflexible coping behaviors than those persons who have a more limited repertoire. Lazarus's distinction between problem-focused and emotional-focused coping strategies may also be important in determining coping costs. For example, individuals who characteristically combine problem-focused and emotional-focused strategies in dealing with various stressors may be less prone to fatigue effects than people who rely on one or the other coping style exclusively. Persons who tend to use emotional-focused forms of coping may also avoid the potentially pathogenic effects of active problem-focused-coping described before.

Cost of Coping: Summary

As outlined here, it is clear that the process of active coping, whether successful or unsuccessful, can have a severe impact on one's health and behavior. Table 2 provides an outline of the secondary effects of coping discussed previously.

In some cases this impact is initially mediated by physiological processes, whereas in other cases it is mediated by behavioral ones. Deleterious effects may

Table 2. Costs of Coping with a Stressful Event

	Mechanism	Effects
Successful coping		
Cumulative fatigue effect	Adaptive processes drain one's cognitive (psychic) energies	Poststress deficits in task performance, poststress insensitivity toward others, and increased deficits over time on demanding tasks performed under stress
Coping side effect	When coping behaviors, which were successful in ameliorating the possible effects of the stressor, are detrimental to individuals in other ways	Physiological effects that are above the level efficient for the body's metabolic needs. Possible interference with health maintenance behaviors.
Overgeneralization of a coping strategy	A strategy employed to cope with a stressor persists even in situations where a person is not confronted with the offending stressor	Deleterious effects on task performance, interpersonal behavior, and health maintenance behavior occur to the extent that the coping responses are inappropriate in other situations in which they are employed
Unsuccessful coping		
Cumulative fatigue effect	Adaptive processes drain one's cognitive (psychic) energies	Poststress deficits in task performance, poststress insensitivity toward others, and increased deficits over time on demanding tasks performed under stress
Coping side effect	When coping behaviors, which were employed in an attempt to ameliorate the possible effects of the stressor, are detrimental to individuals in other ways	Physiological effects that are above the level efficient for the body's metabolic needs. Possible interference with health maintenance behaviors.
Helplessness	When an expectation that one cannot effectively cope with a stressor persists even in situations where coping is possible	Undermines motivation to initiate further instrumental responses, interferes with learning that other outcomes are controllable, causes depression of mood, possibly results in a level of underarousal
Passivity in the face of stressful events	One does not engage in any coping behavior	When not responding is a chosen (preferred) state, there are no secondary effects. When not responding is not the preferred state, effects associated with helplessness are expected

occur when (a) one engages in effortful coping; (b) one persists in using a coping response in situations where the strategy is not adaptive; (c) coping responses have deleterious side effects; and (d) one perceives that his or her efforts to cope are fruitless. Data and theory on passivity in the face of a stressor allow only tentative conclusions at this time.

On Studying Children under Stress

Our discussion up to this point has not distinguished between the stress-appraisal processes or coping strategies that are used by adults as opposed to those used by children. This issue is critical to our presentation because the data that we report have been gathered exclusively from third- and fourth-grade schoolchildren, whereas our theoretical discussion and research reviews are primarily based on work with adults. It is possible that children in this age group (8 to 9 years old) employ appraisal styles or coping strategies that are peculiar to their stage of development. Although an assessment of these processes, evaluation of their efficacy, and description of the roles they play in child development would be a worthy pursuit, it is one more suited to those concerned with developmental processes *per se* than to those interested in generic forms of coping with stress (see Garnezy & Rutter, 1983). Thus, our own emphasis is on determining whether various processes demonstrated in the adult literature are similarly operative in children.

A central question about the effects of stress on children is whether children are more or less vulnerable than adults. There are a number of reasons to expect that children are more susceptible to pathogenic and behavioral effects of the stress process. Young children (a) may have less cognitive capacity and thus lower thresholds for information overload (Cohen, 1978; Evans, 1978b); (b) may be unable to adequately anticipate stressors and consequently plan coping strategies; (c) may lack well-developed coping repertoires and thus have less flexibility in meeting various adaptive challenges and threats from their surroundings; and (d) have organs that are not fully developed that may be more vulnerable to the physiological effects of stress and/or the coping process than those of physically mature adults.

In addition, the lack of *control* that children have over their physical and social environments may be especially important in determining susceptibility. Unlike adults, children usually cannot regulate their initial choice to expose themselves to stressors nor can they regulate subsequent environmental conditions. For example, children are seldom able to make choices about moving away from a noisy neighborhood, adding sound insulation to their house, or otherwise attempting to attenuate noise exposure. They are also seldom able to directly complain about the noise source. As a result, they must frequently rely on adults to regulate their exposure to stressful conditions. Unfortunately, adult caregivers may also suffer from exposure to the same aversive conditions and be unable or unwilling to attend to the child's needs (Cohen, Glass, & Phillips, 1979; Evans, 1978b; Saegert, 1981).

Stress Processes and the Costs of Coping

In sum, there are many reasons to think that children may be more susceptible to environmental and psychosocial stressors than adults. Hence children may be viewed as a high risk population who can provide an idea of the potential effects of chronic stressor exposure.

Making the Most of this Volume

As we noted earlier, this volume was designed to serve multiple purposes. Because we felt that some persons would be interested in specific topics but not in others, each chapter is written to stand on its own. (The exception is that the details of the design of the Los Angeles Noise Project studies are included only in Chapter 2). However, we have also attempted to produce a volume that holds together as a whole, representing an integration of our theoretical views, previously existing literature, and the data provided by the Los Angeles Noise Project studies. We encourage even those who are primarily interested in one or two of the three categories of outcomes we discuss (control, health, and attention) to read the theoretical discussions in Chapters 1 and 6 and the methodological and practical insights of Chapter 2 because we think that these chapters provide new and interesting ways of viewing these literatures. Our hope is that this organization allows efficient and optimal use of the volume for both those who are broadly interested in the topics we discuss and those who are interested in one or more specific areas.