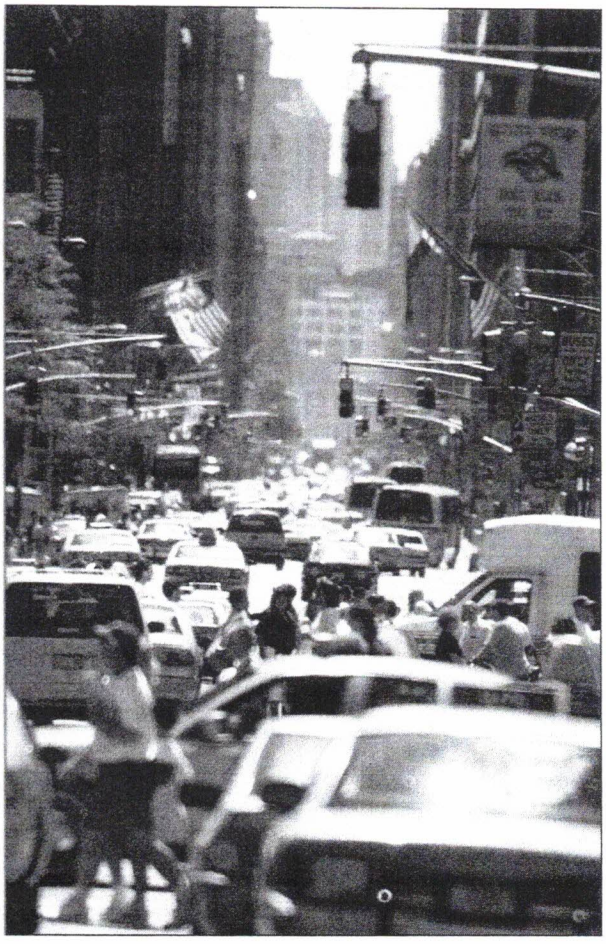
Taylor Reading

Class 7

Stress 1

CHAPTER 6

Stress



CHAPTER OUTLINE

The night before her biology final, Lisa confidently set her alarm and went to sleep. A power outage occurred during the night, and her alarm, along with most of the others in the dorm, failed to go off. At 8:45, Lisa was abruptly awakened by a friend banging on the door to tell her that her final started in 15 minutes. Lisa threw on some clothes, grabbed a muffin and a cup of coffee from the machines in her dorm, and raced to the exam room. Ten minutes late, she spent the first half hour frantically searching through the multiple-choice questions, trying to find ones she knew the answers to, as her heart continued to race.

WHAT IS STRESS?

Most of us have more firsthand experience with stress than we care to remember. Stress is being stopped by a police officer after accidentally running a red light. It is waiting to take a test when we are not sure that we have prepared well enough or studied the right material. It is missing a bus on a rainy day full of important appointments.

Psychologists have been studying stress and its impact on psychological and physical health for several decades. **Stress** is a negative emotional experience accompanied by predictable biochemical, physiological, cognitive, and behavioral changes that are directed either toward altering the stressful event or accommodating its effects (see Baum, 1990).

What Is a Stressor?

Initially, researchers focused on stressful events themselves, called **stressors.** Such events include noise, crowding, a bad relationship, a round of job interviews, and the commute to work. The study of stressors has helped define some conditions that are more likely to produce stress than others, but a focus on stressful events cannot fully explain the experience of stress. An experience may be stressful to some people but not to others. If the "noise" is the latest rock music playing on your radio, then it will probably not be stressful to you, although it may be to your neighbor. Whereas one person might find the loss of a job highly stressful, another might see it as an opportunity to try a new field, as a challenge rather than a threat. How a potential stressor is perceived determines whether it will be experienced as stressful.

Person-Environment Fit

Stress is the consequence of a person's appraisal processes: the assessment of whether personal resources are sufficient to meet the demands of the environment. Stress, then, is determined by **person-environment fit** (Lazarus & Folkman, 1984b; Lazarus & Launier, 1978).

When a person's resources are more than adequate to deal with a difficult situation, he or she may feel little stress and experience a sense of challenge instead. When the person perceives that his or her resources will probably be sufficient to deal with the event but only at the cost of great effort, he or she may feel a moderate amount of stress. When the person perceives that his or her resources will probably not suffice to meet an environmental stressor, he or she may experience a great deal of stress.

Stress, then, results from the process of appraising events (as harmful, threatening, or challenging), of assessing potential responses, and of responding to those events. To see how stress researchers have arrived at our current understanding of stress, it is useful to consider some of the early contributions to the field.

THEORETICAL CONTRIBUTIONS TO THE STUDY OF STRESS

Fight or Flight

One of the earliest contributions to stress research was Walter Cannon's (1932) description of the **fight-or-flight response.** Cannon proposed that, when an organism perceives a threat, the body is rapidly aroused and motivated via the sympathetic nervous system and the endocrine system. This concerted physiological response mobilizes the organism to attack the threat or to flee; hence, it is called the fight-or-flight response (Kemeny, 2003).

At one time, fight or flight literally referred to fighting or fleeing in response to stressful events such as attack by a predator. Now, more commonly, *fight* refers to aggressive responses to stress, whereas *flight* may be seen in social withdrawal or withdrawal through substance use such as alcohol or drugs. On the one hand, the fight-or-flight response is adaptive because it enables the organism to respond quickly to threat. On the other hand, it can be harmful because stress disrupts emotional and physiological functioning, and when stress continues unabated, it lays the groundwork for health problems.

Selye's General Adaptation Syndrome

Another important early contribution to the research into stress is Hans Selye's (1956, 1976) work on the **general adaptation syndrome.** Although Selye initially intended to explore the effects of sex hormones on

physiological functioning, he became interested in the stressful impact his interventions seemed to have. Accordingly, he exposed rats to a variety of stressors—such as extreme cold and fatigue—and observed their physiological responses. To his surprise, all stressors, regardless of type, produced essentially the same pattern of physiological changes. They all led to an enlarged adrenal cortex, shrinking of the thymus and lymph glands, and ulceration of the stomach and duodenum.

From these observations, Selye (1956) developed his concept of the general adaptation syndrome. He argued that, when an organism confronts a stressor, it mobilizes itself for action. The response itself is nonspecific with respect to the stressor; that is, regardless of the cause of the threat, the individual will respond with the same physiological pattern of reactions. Over time, with repeated or prolonged exposure to stress, there will be wear and tear on the system.

The general adaptation syndrome consists of three phases. In the first phase, *alarm,* the organism becomes mobilized to meet the threat. In the second phase, *resistance,* the organism makes efforts to cope with the threat, as through confrontation. The third phase, *exhaustion,* occurs if the organism fails to overcome the threat and depletes its physiological resources in the process of trying. These phases are pictured in Figure 6.1.

Selye's model continues to have an impact on stress research. One reason is that it offers a general theory of reactions to a wide variety of stressors over time. As such, it provides a way of thinking about the interplay of physiological and environmental factors. Second, it posits a physiological mechanism for the stress-illness relationship. Specifically, Selye believed that repeated or prolonged exhaustion of resources, the third phase of the syndrome, is responsible for the physiological damage that lays the groundwork for disease. In fact, prolonged or repeated stress has been implicated in a broad array of disorders, such as cardiovascular disease, arthritis, hypertension, and immune-related deficiencies, as we will see in Chapters 13 and 14.

Criticisms of the General Adaptation Syndrome

Selye's model has also been criticized on several grounds. First, it assigns a very limited role to psychological factors, and researchers now believe that the psychological appraisal of events is important in the determination of stress (Lazarus & Folkman, 1984b). A second criticism concerns the assumption that responses to stress are uniform (Hobfoll, 1989). Not all stressors produce the same endocrinological responses (Kemeny, 2003). How people respond to stress is substantially influenced by their personalities, perceptions, and biological constitutions. A third criticism concerns the fact that Selye assessed stress as an outcome, such that stress is evident only when the general adaptation syndrome has run its course. In fact, people experience many of the debilitating effects of stress while a stressful event is going on and even in anticipation of its occurrence. Despite these limitations and reservations, Selye's model remains a cornerstone in the field.

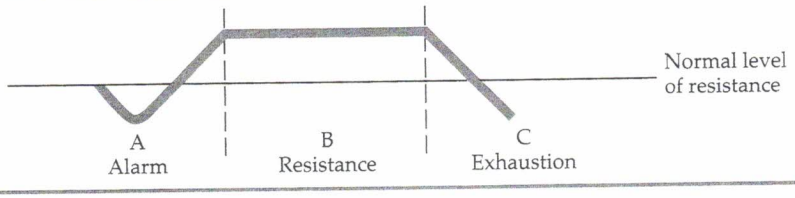
Tend-and-Befriend

Animals, whether nonhuman or human, do not merely fight, flee, and grow exhausted in response to stress. They also affiliate with each other, whether it is the herding behavior of deer in response to stress, the huddling one sees among female rats, or the coordinated responses to a stressor that a community shows when it is under the threat of flood, tornado, or other natural disaster.

To address this issue, S. E. Taylor and colleagues (Taylor, Klein, et al., 2000) developed a theory of responses to stress termed **tend-and-befriend.** The theory maintains that, in addition to fight or flight, humans

FIGURE 6.1 | The Three Phases of Selye's (1974) General Adaptation Syndrome

Plase A is the alarm response, in which the body first reacts to a stressor. At this time, resistance is diminished. Phase B, the stage of resistance, occurs with continued exposure to a stressor. The bodily signs associated with an alarm reaction disappear, and resistance rises above normal. Phase C is the stage of exhaustion that results from long-term exposure to the same stressor. At this point, resistance may again fall to below normal. (*Source: Stress Without Distress* by Hans Selye, M.D. Copyright © 1974 by Hans Selye, M.D. Reprinted by permission of HarperCollins Publishers, Inc.)



respond to stress with social affiliation and nurturant behavior toward offspring. These responses may be especially true of women.

During the time that responses to stress evolved, men and women faced somewhat different adaptive challenges. Whereas men were responsible for hunting and protection, women were responsible for foraging and child care. These activities were largely segregated, with the result that women's responses to stress would have evolved so as to protect not only the self but offspring as well. These responses do not characterize only humans. The offspring of most species are immature and would be unable to survive were it not for the attention of adults. In most species, that attention is provided by the mother.

Like the fight-or-flight mechanism, tend-and-befriend may depend on underlying biological mechanisms—in particular, the hormone oxytocin. Oxytocin is a stress hormone, rapidly released in response to at least some stressful events, and its effects are especially influenced by estrogen, suggesting a role in the responses of women to stress (Taylor, Gonzaga et al., 2006). The potential contribution of oxytocin to stress responses is to act as an impetus for affiliation. In both animals and humans, oxytocin increases affiliative behaviors of all kinds, especially mothering (Taylor, 2002). In addition, animals and humans with high levels of oxytocin are calmer and more relaxed, which may contribute to social and nurturant behavior (McCarthy, 1995). Opioids may also contribute to affiliative responses to stress in females (Taylor, Klein, et al., 2000).

In support of the theory, there is evidence that women are consistently more likely than men to respond to stress by turning to others (Luckow, Reifman, & McIntosh, 1998; Tamres, Janicki, & Helgeson, 2002). Mothers' responses to offspring during times of stress also appear to be different from those of fathers in ways encompassed by the tend-and-befriend theory. Nonetheless, men, too, show social responses to stress, and at present, less is known about men's social responses to stress than women's.

In addition to offering a biobehavioral approach to differences in male and female responses to stress, the tend-and-befriend theory brings social behavior into stress processes. We are affiliative creatures who respond to stress collectively, as well as individually, and these responses are characteristic of men as well as women (Taylor, Klein, et al., 2000).

Psychological Appraisal and the Experience of Stress

In humans, psychological appraisals are an important determinant of whether an event is responded to as stressful.

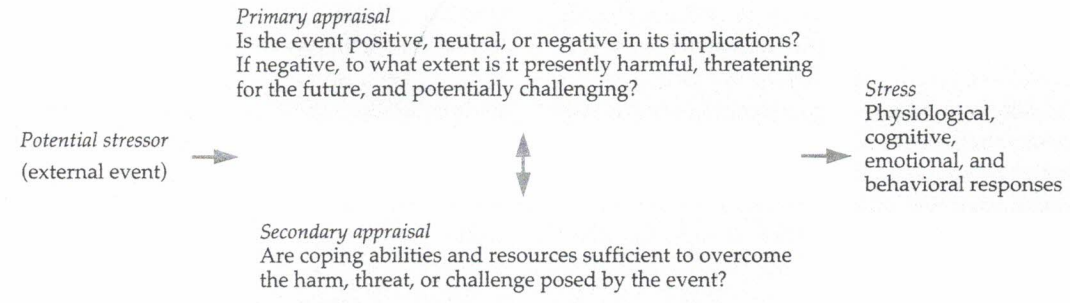
Primary Appraisal Processes

R. S. Lazarus, a chief proponent of the psychological view of stress (Lazarus, 1968; Lazarus & Folkman, 1984b), maintained that, when individuals confront a new or changing environment, they engage in a process of **primary appraisal** to determine the meaning of the event (Figure 6.2).

Events may be perceived as positive, neutral, or negative in their consequences. Negative or potentially negative events are further appraised for their possible harm, threat, or challenge. *Harm* is the assessment of the damage that has already been done by an event. Thus, for example, a man who has just been fired from his job may perceive present harm in terms of his own loss of self-esteem and his embarrassment as his coworkers silently watch him pack up his desk.

Threat is the assessment of possible future damage that may be brought about by the event. Thus, the man who has just lost his job may anticipate the problems that loss of income will create for him and his family in the future. Primary appraisals of events as threats have important effects on physiological responses to stress.

FIGURE 6.2 | The Experience of Stress



For example, blood pressure is higher when threat is higher or when threat is high and challenge is low (Maier, Waldstein, & Synowski, 2003).

Finally, events may be appraised in terms of their *challenge,* the potential to overcome and even profit from the event. For example, the man who has lost his job may perceive that a certain amount of harm and threat exists, but he may also see his unemployment as an opportunity to try something new. Challenge appraisals are associated with more confident expectations of the ability to cope with the stressful event, more favorable emotional reactions to the event, and lower blood pressure (Maier et al., 2003; Skinner & Brewer, 2002).

The importance of primary appraisal in the experience of stress is illustrated in a classic study of stress by J. Speisman and colleagues (Speisman, Lazarus, Mordkoff, & Davidson, 1964). College students viewed a gruesome film depicting unpleasant tribal initiation rites that included genital mutilation. Before viewing the film, they were exposed to one of four experimental conditions. One group listened to an anthropological account about the meaning of the rites. Another group heard a lecture that deemphasized the pain the initiates were experiencing and emphasized their excitement over the events. A third group heard a description that emphasized the pain and trauma that the initiates were undergoing. A fourth group was given no introductory information, and the film they viewed had no sound track. Measures of autonomic arousal (skin conductance, heart rate) and self-reports suggested that the first two groups experienced considerably less stress than did the group whose attention was focused on the trauma and pain. Thus, stress not only was intrinsic to the gruesome film itself but also depended on the viewer's appraisal of it.

Secondary Appraisal Processes At the same time that primary appraisals of stressful circumstances are occurring, secondary appraisal is initiated. **Secondary appraisal** is the assessment of one's coping abilities and resources: whether they will be sufficient to meet the harm, threat, and challenge of the event. Ultimately, the subjective experience of stress is a balance between primary and secondary appraisal. When harm and threat are high and coping ability is low, substantial stress is felt. When coping ability is high, stress may be minimal.

Potential responses to stress are many and include physiological, cognitive, emotional, and behavioral consequences. Some of these responses are involuntary reactions to stress, whereas others are voluntarily initiated in a conscious effort to cope.

Cognitive responses to stress include beliefs about the harm or threat an event poses and beliefs about its causes or controllability. They also include involuntary responses such as distractability and inability to concentrate, disruptions on cognitive tasks (Cohen, 1980; Shaham, Singer, & Schaeffer, 1992), and intrusive, repetitive, or morbid thoughts. Cognitive responses are also involved in the initiation of coping activities, as we will see in Chapter 7.

Potential emotional reactions to stressful events range widely; they include fear, anxiety, excitement, embarrassment, anger, depression, and even stoicism or denial. Emotional responses can be quite insistent, prompting rumination over a stressful event, which, in turn, may keep biological stress responses elevated (Glynn, Christenfeld, & Gerin, 2002).

Potential behavioral responses are virtually limitless, depending on the nature of the stressful event. Confrontative action against the stressor ("fight") and withdrawal from the threatening event ("flight") constitute two general categories of behavioral responses. We will examine others in the course of our discussion.

The Physiology of Stress

Stress is important, both because it causes psychological distress and because it leads to changes in the body that may have short- and long-term consequences for health. Two interrelated systems are heavily involved in the stress response. They are the sympathetic-adrenomedullary (SAM) system and the hypothalamic-pituitary-adrenocortical (HPA) axis. These components of the stress response are illustrated in Figure 6.3.

Sympathetic Activation When events are encountered that are perceived as harmful or threatening, they are labeled as such by the cerebral cortex, which, in turn, sets off a chain of reactions mediated by these appraisals. Information from the cortex is transmitted to the hypothalamus, which initiates one of the earliest responses to stress—namely, sympathetic nervous system arousal, or the fight-or-flight response first described by Walter Cannon. Sympathetic arousal stimulates the medulla of the adrenal glands, which, in turn, secrete the catecholamines—epinephrine (EP) and norepinephrine (NE). These effects result in the cranked-up feeling we usually experience in response to stress. Sympathetic arousal leads to increased blood pressure, increased heart rate, increased sweating, and constriction of peripheral blood vessels, among other changes. As

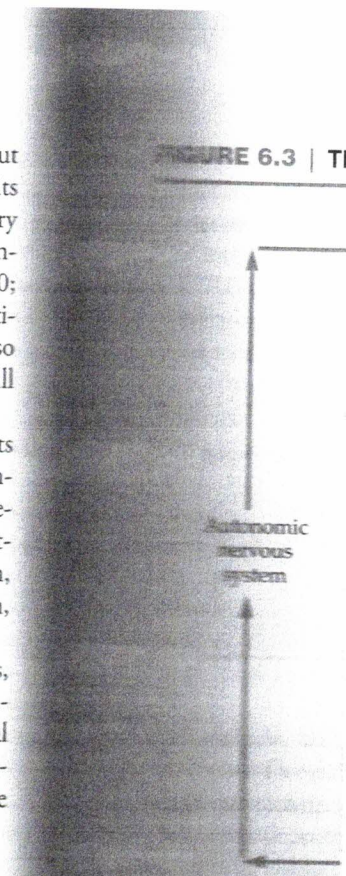
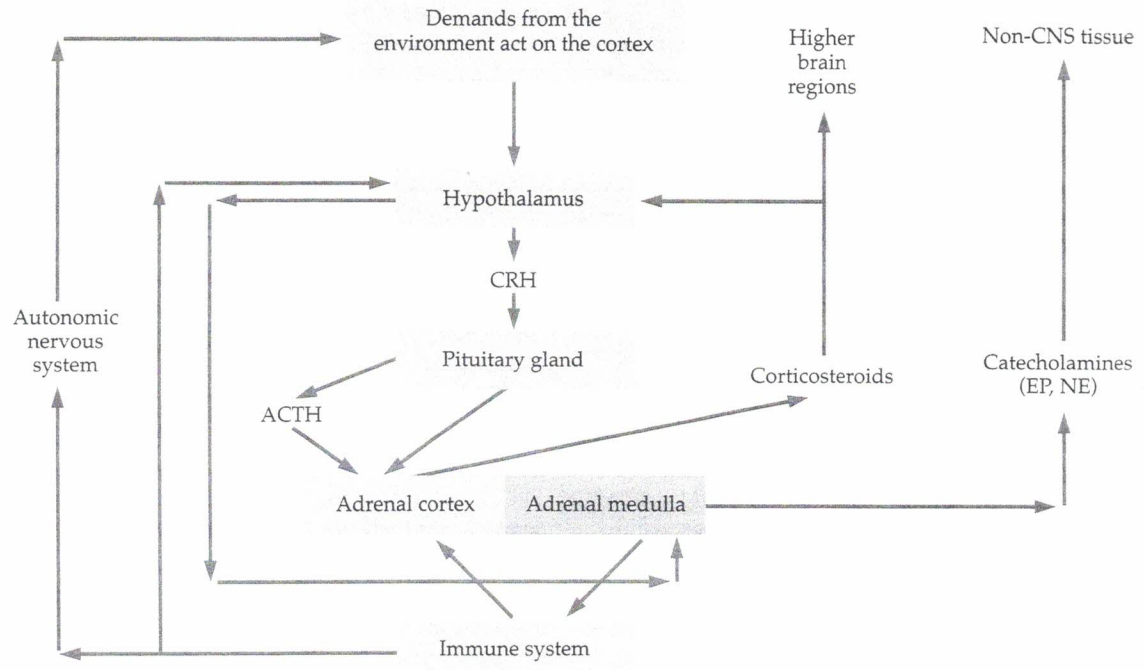


FIGURE 6.3 | The Body's Stress Systems



can be seen in Figure 6.3, the catecholamines have effects on a variety of tissues and modulate the immune system as well.

Stress can affect heart rate variability, including variability during sleep. Parasympathetic modulation is an important restorative aspect of sleep, and thus, changes in heart rate variability may both represent a pathway to disturbed sleep and help to explain the relation of stress to illness and increased risk for mortality (Hall, Vasko, et al., 2004).

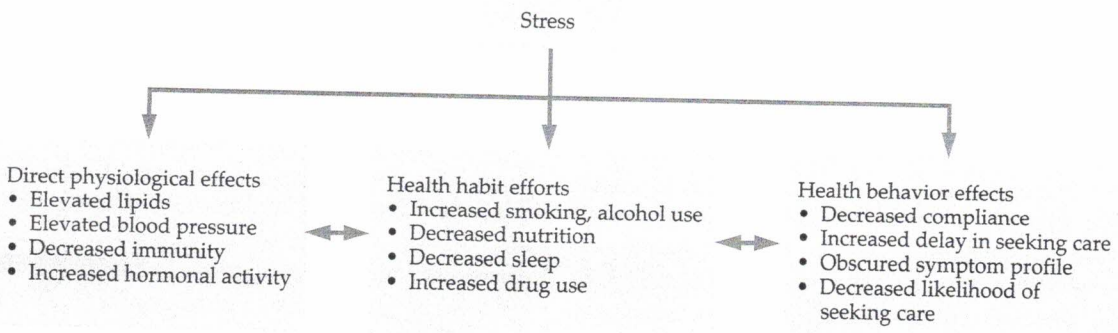
HPA Activation In addition to the activation of the sympathetic nervous system, the HPA axis is activated in response to stress. Hans Selye provided the basis for understanding the effects of stress on the HPA in his general adaptation syndrome, the nonspecific physiological reaction that occurs in response to stress and involves the three phases of alarm, resistance, and exhaustion.

The hypothalamus releases corticotrophin-releasing factor (CRF), which stimulates the pituitary gland to secrete adrenocorticotropic hormone (ACTH), which, in turn, stimulates the adrenal cortex to release glucocorticoids. Of these, cortisol is especially significant. It acts to conserve stores of carbohydrates and helps reduce inflammation in the case of an injury. It also helps the body return to its steady state following stress. In addition, HPA activation produces elevations in growth hormone and prolactin, secreted by the pituitary gland.

Repeated activation of the HPA axis in response to chronic or recurring stress can ultimately compromise its functioning. When the HPA axis becomes dysregulated in response to stress, several things may happen. Daily cortisol patterns may be altered. That is, normally cortisol levels are high upon waking in the morning, but decrease during the day (although peaking following lunch) until they flatten out at low levels in the afternoon. People under chronic stress, however, can show elevated cortisol levels long into the afternoon or evening (Powell et al., 2002), a general flattening of the diurnal rhythms, an exaggerated cortisol response to a challenge, a protracted cortisol response following a stressor, or, alternatively, no response at all (McEwen 1998). Any of these patterns is suggestive of compromised ability of the HPA axis to respond to and recover from stress (McEwen, 1998; Pruessner, Hellhammer, Pruessner, & Lupien, 2003). When researchers study physiological and neuroendocrine stress responses, they look for signs like these (Figure 6.4).

FIGURE 6.4 | Routes by Which Stress May Produce Disease

The text describes how direct physiological effects may result from sympathetic nervous system and/or HPA activation. In addition, as this figure shows, stress may affect health via behaviors—first, by influencing health habits directly and second, by interfering with treatment and the use of services. (*Source:* Baum, 1994)



Effects of Long-Term Stress

We have just examined some of the major physiological changes that occur in response to the perception of stress. What do these changes mean? Although the short-term mobilization that occurs in response to stress originally prepared humans to fight or flee, rarely do stressful events require these kinds of adjustments. Consequently, in response to stress, we often experience the effects of sudden elevations of circulating stress hormones that, in certain respects, do not serve the purpose for which they were originally intended.

Over the long term, excessive discharge of epinephrine and norepinephrine can lead to suppression of cellular immune function; produce hemodynamic changes such as increased blood pressure and heart rate; provoke variations in normal heart rhythms, such as ventricular arrhythmias, which may be a precursor to sudden death; and produce neurochemical imbalances that may contribute to the development of psychiatric disorders. The catecholamines may also have effects on lipid levels and free fatty acids, all of which may be important in the development of atherosclerosis.

Corticosteroids have immunosuppressive effects, which can compromise the functioning of the immune system. Prolonged cortisol secretion has also been related to the destruction of neurons in the hippocampus. This destruction can lead to problems with verbal functioning, memory, and concentration (Starkman, Giordani, Brenent, Schork, & Schteingart, 2001) and may be one of the mechanisms leading to senility. Pronounced HPA activation is common in depression, with episodes of cortisol secretion being more frequent and of longer duration among depressed than nondepressed people, although it is not entirely clear whether HPA activation is a cause or an effect of these disorders. Another long-term consequence of the endocrine abnormalities that result from chronic HPA activation is the storage of fat in central visceral areas, rather than to the hips. This accumulation leads to a high waist-to-hip ratio, which is used by some researchers as a marker for chronic stress (Bjorntorp, 1996).

Which of these responses to stress have implications for disease? Several researchers (Dientsbier, 1989; Frankenhaeuser, 1991) have suggested that the health consequences of HPA axis activation are more significant than those of sympathetic activation. Sympathetic arousal in response to stress may not be a pathway for disease; HPA activation may be required as well. Some researchers have suggested that this reasoning explains why exercise, which produces sympathetic arousal but not necessarily HPA activation, is protective for health rather than health compromising.

Stress may also impair the immune system's capacity to respond to hormonal signals that terminate inflammation. A study that demonstrates this point compared 50 healthy adults, half of whom were parents of children with cancer and half of whom were parents of healthy children. Childhood cancer is known to be one of the most stressful events that parents encounter. The parents of the cancer patients reported more stress and had flatter daily slopes of cortisol secretion than was true for the parents of the healthy children. Moreover, the ability to suppress production of a proinflammatory cytokine called IL-6 was diminished among parents of the cancer patients. Because chronic inflammation is implicated in a broad array of diseases including coronary

artery disease (see Chapter 2), these findings suggest that the impaired ability to terminate inflammation may be another pathway by which stress affects illness outcomes (Miller, Cohen, & Ritchey, 2002).

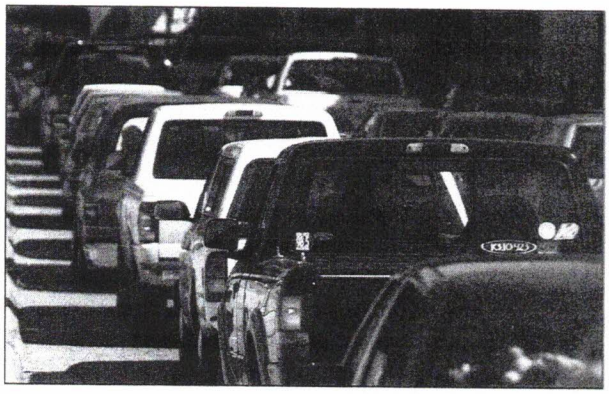
Researchers are increasingly focusing on poor sleep quality as both an indicator of chronic stress and a consequence of chronic stress. It has long been suspected that chronic insomnia can result from stressful events. Evidence suggests that the combination of emotional arousal and neuroendocrine activation due to chronic stress may indeed underlie chronic insomnia (Shaver, Johnston, Lentz, & Landis, 2002). Because sleep represents a vital restorative activity, this mechanism, too, may represent an important pathway to disease (Edwards, Hucklebridge, Clow, & Evans, 2003). The coming years will help clarify the important psychobiological pathways from stress to disease.

Individual Differences in Stress Reactivity

People vary in their reactivity to stress. **Reactivity** is the degree of change that occurs in autonomic, neuroendocrine, and/or immune responses as a result of stress. Reactivity is, in part, a genetically based predisposition to respond physiologically to environmental threats or challenges (Jacobs et al., 2006) that may be implicated in both short- and long-term health complications due to stress. Some people show very small reactions to stressful circumstances, whereas others show large responses. These differences may be genetic in origin or develop prenatally or in early life.

Reactivity to stress can affect vulnerability to illness. For example, in one study, a group of children ranging in age from 3 to 5 years old were tested for their cardiovascular reactivity (change in heart rate and blood pressure) or their immune response to a vaccine following a stressful task. Parents were then asked to report on the number of family stressors during a 12-week period, and illness rates were charted during this period. The results indicated that stress was associated with increased rates of illness only among the children who had previously shown strong immune or cardiovascular reactions. The less reactive children did not experience any change in illness rates under stressful circumstances (Boyce, Alkon, Tschann, Chesney, & Alpert, 1995).

Do changes like this actually lead to illness? S. Cohen and colleagues (2002) found that people who reacted to laboratory stressors with high cortisol responses and who also had a high level of negative life events were especially vulnerable to upper respiratory infections. People who reacted to laboratory stressors with low immune responses were especially vulnerable to upper respiratory infection only if they were also under high stress. High immune reactors, in contrast, did not show differences in upper respiratory illness as a function of the stress they experienced, perhaps because their immune systems were quick to respond to the threat that a potential infection posed.



Stressful events such as being stuck in traffic produce agitation and physiological arousal.

Studies like these suggest that psychobiological reactivity to stress is an important factor that influences the effects that stress has on the body and the likelihood that it will contribute to distress or disease. As will be seen in Chapter 13, differences in reactivity are believed to contribute especially to the development of hypertension and coronary artery disease.

Physiological Recovery Processes Recovery processes following stress are also important in the physiology of the stress response (Rutledge, Linden, & Paul, 2000). In particular, the inability to recover quickly from a stressful event may be a marker for the cumulative damage that stress has caused. Researchers have paid special attention to the cortisol response—particularly, prolonged cortisol responses that occur under conditions of high stress.

In one intriguing study (Perna & McDowell, 1995), elite athletes were divided into groups that were experiencing a high versus a low amount of stress in their lives, and their cortisol response was measured following vigorous training. Those athletes under more stress went a longer time before their cortisol levels returned to normal. Because elevated cortisol affects the immune system, the researchers suggested that stress may widen the window of susceptibility for illness and injury among competitive athletes by virtue of its impact on cortisol recovery.

As the research on recovery processes implies, the long-term effects of stress on the body are of great

BOX 6.1

Can Stress Affect Pregnancy?

Common wisdom has long held that pregnant women should be treated especially well and avoid major stressors in their lives. Research now supports that wisdom by showing that stress can actually endanger human pregnancy.

Stress affects the immune and endocrine systems in ways that directly affect the growing fetus. These changes are potentially dangerous because they can lead to preterm birth and low birth weight, among other adverse outcomes (Coussons-Read, Okun, Schmitt, & Giese, 2005). African-American women and acculturated Mexican American women appear to be especially vulnerable (Dominguez, Dunkel-Schetter, Mancuso, Rini, & Hobel, 2005).

Although a number of theories have been proposed to explain these relations, one that appears to have broad support is the idea that the mother's elevated cortisol levels in response to stress act as a signal to the fetus that it is time to be born (Diego et al., 2006; Hobel, Dunkel-Schetter, & Roesch, 1998; Mancuso, Dunkel-Schetter, Rini, Roesch, & Hobel, 2004).

Are there any factors that can protect against adverse birth outcomes due to stress? Social support, especially from a partner, appears to have a protective effect against potential adverse birth outcomes (Feldman, Dunkel-Schetter, Sandman, & Wadhwa, 2000). Psychosocial resources such as mastery, self-esteem, and optimism may also help guard against adverse birth outcomes such as low birth weight (Rini, Dunkel-Schetter, Wadhwa, & Sandman, 1999). The anxiety that can accompany stress and the prenatal period exacerbates the risk of elevated cortisol levels and adverse birth outcomes, and so interventions to reduce anxiety may be helpful as well (Mancuso et al., 2004).

But the old adage about taking it easy during pregnancy and the more dire warnings about adverse birth outcomes in disadvantaged groups make it clear that pregnancy is an important time to avoid stress as much as possible and to make strong use of psychosocial resources.

importance in understanding the mechanisms by which physiological changes in response to stress may promote illness.

Allostatic Load

As Selye noted, the initial response of the body to stressful circumstances may be arousal, but over time, this response may give way to exhaustion, leading to cumulative damage to the organism. Building on these ideas, researchers have developed the concept of **allostatic load** (McEwen & Stellar, 1993). This concept refers to the fact that physiological systems within the body fluctuate to meet demands from stress, a state called allostasis. Over time, allostatic load builds up, which is defined as the physiological costs of chronic exposure to fluctuating or heightened neural or neuroendocrine response that results from repeated or chronic stress.

This buildup of allostatic load—that is, the long-term costs of chronic or repeated stress—can be assessed by a number of indicators (Seeman, Singer, Horwitz, & McEwen, 1997). These include decreases in cell-mediated immunity, the inability to shut off cortisol in response to stress, lowered heart rate variability, elevated epinephrine levels, a high waist-to-hip ratio (reflecting abdominal fat), hippocampal volume (which is believed to decrease with repeated stimulation of the HPA), problems with memory (an indirect measure of hippocampal functioning), high plasma fibrinogen, and elevated blood pressure. Many of these changes occur normally with age, so to the extent that they occur early, allostatic load may be thought of as accelerated aging of the organism in response to stress. Over time, this kind of wear and tear can lead to illness and increased risk of death (Karlamangla, Singer, & Seeman, 2006). The damage due to chronic or repeated stress is only made worse if people also cope with stress via a higher-fat diet, less frequent exercise, and smoking, all of which stress can encourage (Ng & Jeffery, 2003).

The physiology of stress and, in particular, the recent research on the cumulative adverse effects of stress are important because they suggest the pathways by which stress exerts adverse effects on the body, ultimately contributing to the likelihood of disease. The relationship of stress, both short and long term, to both acute disorders, such as infection, and chronic diseases, such as heart disease, is now so well established that stress is implicated in most diseases, either in their etiology, their course, or both. We explore these processes more fully when we address different diseases such as heart disease and hypertension in Chapter 13 and cancer and arthritis in Chapter 14. Stress can even affect the course of pregnancy, as Box 6.1 shows.