

Stress

The idea that exercise reduces stress has become a part of folk wisdom, much like views that exercise improves mood, self-esteem, and sleep—topics of later chapters. The benefits of enhanced ability to cope with chronic stress are significant, considering the evidence for the role of mental stress in disease risk and recovery. For example, stress is linked to leading causes of death, such as heart disease, cancer, accidents, and suicide. This chapter discusses whether the scientific evidence supports the idea that exercise alters physiological responses during stress. Because other chapters deal with the effects of physical activity on stress emotions related to anxiety and depressive disorders, this chapter emphasizes whether physical activity or physical fitness blunts physiological responses during types of stress other than exercise.

Background and Definitions

The term *stress* appeared in the English language in the 17th century, borrowed from the French words *destresse* and *estrece*, which meant “hardship” and “oppression.” The origin is the Latin word *strictia*, from a verb meaning “to draw tight.” Since 1660, when the British scientist Robert Hooke reported his law of elasticity (the tension exerted by a stretched string is proportional to the extension), stress has been viewed by engineers as the way that load (i.e., external force) impinges on a physical object. Stress is distinguished from **strain**, which is

the deformation, distortion, or tension in the object that results from stress.

In the life sciences, stress is viewed as an imbalance in physiological systems that activates both physiological and behavioral responses to restore balance. In this way, a stressor is like load, a force that acts on a biological system. Because it is easy to see the analogy between objects resisting or breaking under strain and human tolerance for the burdens of living, it is commonly accepted that stress (imbalance) leads to strain (distortion, tension) in animals as it does in physical objects.

Stress is an imbalance in physiological systems that activates physiological and behavioral responses to restore balance.

Stressors are the forces that act on a biological system to cause stress.

A Brief History of Stress

In the mid-1800s, the French physiologist Claude Bernard (1867) proposed that life depended on maintaining the “*milieu interieur*” (i.e., internal environment) in a constant state during changes in the external environment. It is now accepted that mammalian cells can exist only when certain ranges of temperature and acid-base balance are

maintained and when water, nutrients, and oxygen are available. Systems of cells also depend on such balances. In the 1920s, Harvard physician-scientist Walter Cannon extended Bernard's views through his research on the roles of adrenaline (i.e., epinephrine) and the autonomic nervous system in regulating and maintaining physiological balance during rage and fear. Cannon (1929) introduced the term **homeostasis** to describe this balance, or harmony, of physiological systems. In the early 1930s, Hartman, Brownell, and Lockwood (1932) added to Cannon's ideas by proposing a general tissue hormone theory, which stated that steroids secreted by the cortex of the adrenal gland (e.g., cortisol) are needed by all cells for resisting infection and muscular and nervous fatigue, and for regulating body temperature and body water. Each of these events was credited by the Swiss physician Hans

Selye in forming his theory of a **general adaptation syndrome (GAS)** and the diseases of adaptation, based on activation of the adrenal cortex in response to stress (Selye 1936, 1950).



Homeostasis is the balance or harmony of physiological systems.

To Selye, altered homeostasis was not merely a passing response to changes in the environment. He believed that an animal's physiological systems could learn and maintain adaptive defenses against future exposure to stress. Hence, Selye theorized that many diseases result from maladaptations to the environment that are either insufficient, exces-

Good and Bad Stress

If chronic stress can increase your risk of conditions such as heart disease, high blood pressure, a suppressed immune system, eating disorders, headaches, sleep disorders, and ulcers, you might be wondering if you should attempt to remove *all* stress from your life. The answer is no. A certain amount of stress is needed for optimum health and performance—life without stress would be very boring! Stress researcher Hans Selye clearly distinguished between *distress* (“bad stress”) and *eustress* (“good stress”). Some level of stress (eustress) is desirable for optimal performance and well-being; however, all of us can reach a point where stress can become too much (distress) and it starts to inhibit our mental, emotional, and physiological abilities to function effectively (see figure 4.1).

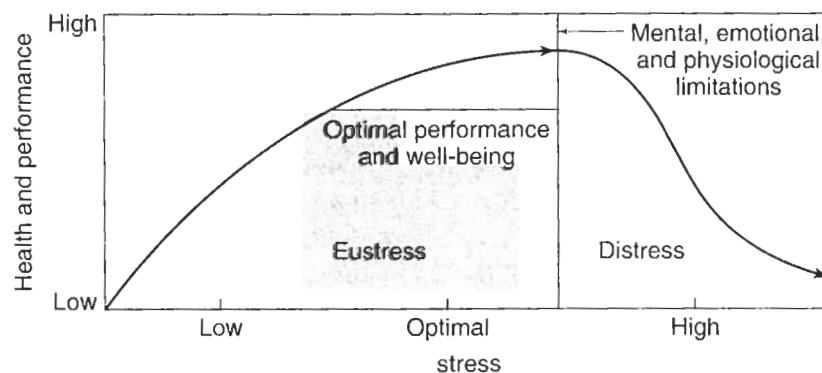


Figure 4.1 A certain amount of stress enhances performance and well-being, but excessive stress can impair mental, emotional, and physiological abilities.

Excessive stress can blunt positive emotions, such as love, joy, and surprise, and exaggerate negative emotions, such as anger, sadness, and fear. Exercise can contribute to your levels of good stress when it is enjoyable and isn't so intense that it causes strain or so frequent that you don't recuperate. Moderate exercise performed regularly can offset negative stress emotions and possibly enhance positive stress emotions.

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sive, or poorly regulated (Selye 1950). He proposed that “conditioning factors,” such as prior exposure and controllability of a stressor, could alter the GAS. Also, Selye believed that stressors, including muscular exercise, might lead to cross-stressor adaptations that would enhance resistance to psychosomatic and neurotic diseases. His research provided a scientific basis for the development of the **cross-stressor adaptation hypothesis** of exercise, which states that exercise training or increased levels of fitness are associated with an attenuation of stress responses in non-exercise situations (Michael 1957; Sothmann et al. 1996).

Recently, Bruce S. McEwen (1998), a neuroscientist at Rockefeller University, has used the term **allostatic load** to describe the long-term effects of physiological response to stress (including activation of the autonomic nervous system; the hypothalamic-pituitary-adrenal axis; and the metabolic, cardiovascular, and immune systems). **Allostasis** is a term derived from Greek and means the ability to achieve stability through change (i.e., adaptation) (see figure 4.2). Like Selye, McEwen believes that the price paid for such adaptation to stress is allostatic load, the strain that results from overactivity or underactivity of these allostatic systems.

Some people develop a *hypoactivity* or *hyperactivity* of the normal stress response. It appears that *too small* a stress response can be just as harmful as *too much* of a response, because it may result in other



A balanced response to stress is optimal: overresponding or underresponding can be dangerous.

responses that compensate. For example, cortisol stimulates blood glucose for energy, but it also keeps the immune system in check by inhibiting inflammation. If cortisol does not rise during stress, inflammation can result even though there is no infection. On the other hand, too much cortisol can make a person susceptible to infection by overly suppressing the inflammation response; and it can lead to bone loss, muscle atrophy, and elevated insulin levels.

For unknown reasons, the stress response does not subside in some people after a stressful event has ended. For example, public speaking activates the hypothalamic-pituitary-adrenal (HPA) axis and increases blood cortisol in most people, but that response goes away after the person has gained experience. However, about 1 in 10 people will continue to have a cortisol response when they speak in public, regardless of their experience. Likewise, it is not understood why some people lose their ability to mount a stress response after chronic exposure to stressful events. Many researchers are convinced that regular exercise of moderate

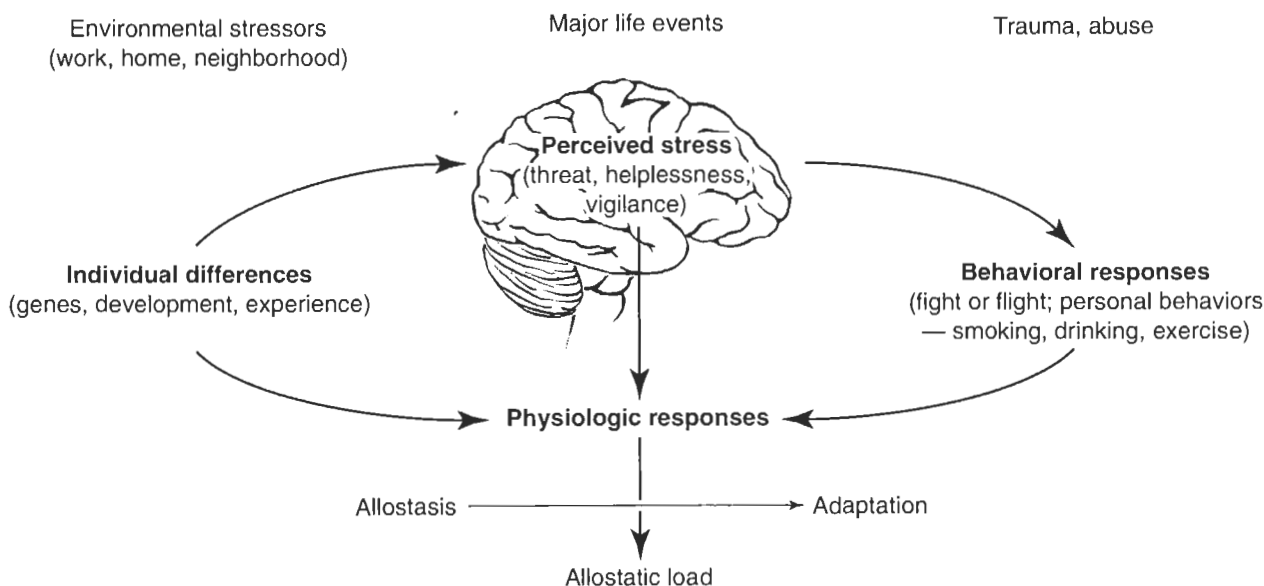


Figure 4.2 Allostasis: the ability to achieve stability through adaptation to stress.

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intensity is one of the best ways to offset the allostatic load of chronic stress. That makes sense because we know, for example, that exercise reduces insulin levels that can be raised by high cortisol, and that exercise training lowers blood pressure and resting heart rate. Before considering evidence for a relationship between exercise and the stress response, it is necessary to define the conditions that elicit stress and the main responses during stress.

Defining Stress

Stress can lead to painful *physical symptoms*, such as muscle tension, headache, and stomach upset; *physiological signs*, such as a racing heart, high blood pressure, sweating, flushing, and dry mouth; and *behaviors* ranging from aggression to hyperactivity to withdrawal. Those signs and symptoms can occur independently or together with stress emotions, which include the physiological and behavior responses that are experienced subjectively by the person (e.g., fear, anxiety, anger, despair). Scientists have identified key physiological responses during stress and their patterns of occurrence.

Early studies of humans during the 1950s and 1960s showed that responses by the adrenal glands are largest when stressful tasks are complex and require rapid decision making, or when people are

responsible for the welfare of others and/or have little control over the outcomes of critical events. During the late 1960s and early 1970s, Marianne Frankenhaeuser of the University of Stockholm demonstrated that in novel, unpredictable, and threatening circumstances, levels of epinephrine and its related hormone norepinephrine were increased during muscular exertion or mental challenge in proportion to people's perception of stress. However, the increases, particularly in epinephrine, were blunted as people became familiar with the challenge (Frankenhaeuser 1971). A later study showed that cortisol responded the same way (Mason et al. 1976). Those findings led to additional definitions of stress according to how people appraise (i.e., define and evaluate) events as threatening and how they cope with the stress (respond mentally or by their behavior) (Lazarus 1993). People can cope with stress *actively* by striving to overcome the source of the stress or by trying to avoid it (**active coping**), or *passively* by taking it without resistance. Events that result in stress can be described in terms of several other dimensions that are related to the nature of the response (see table 4.1).

Stress can occur during a crisis of high impact and for either a short or long duration, or during the smaller, brief but nagging, hassles of daily living. Positive life events or daily uplifts in spirit can

Table 4.1

Dimensions of Events That Can Elicit a Stress Response		
Dimensions of stressors can interact. For example, frequent daily hassles over which we have no control that continue for months may have more cumulative ill effects than one short-lived major life event that we are able to resolve ourselves.		
Quality	Familiarity	Source
Eustress (positive)	Familiar	Mental (i.e., negative thoughts)
Distress (negative)	Novel	Physiological (e.g., virus) Environmental (social, physical)
Quantity	Coping response	Threat level
Duration	Active	No threat
Frequency	Passive	Life threatening
Intensity	Sensory focus	Types (perceived)
Minor hassle	Rejection	Challenge (demands person perceives he or she is confident in overcoming)
Major life event	Intake Intake/rejection	Threat (anticipation of harm) Harm (experienced damage)



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An external stressor provokes psychological and physiological responses.



Responses to stress can be active (resistance or avoidance) or passive (taking it without resistance).

also be stressful, but in a good way, because they reduce boredom and offset negative emotions. Though it is true that some people simply are exposed to more events that cause stress or strain (e.g., family conflicts, money problems, loss of a loved one, too many hard exams), it is also true that personality and coping skills can lessen one's vulnerability to stress. About 50% of a person's temperament—whether it is usually calm or is nervous or fiery—is explained by heredity and early childhood learning. Nonetheless, people can improve their ability to deal with stress by learning skills to reduce exposure to stressful events or by changing their outlook on life. People who view change as a challenge or an opportunity for success, who feel in control, and who have a strong commitment to a purpose in their life (e.g., career, other people, spirituality) seem to deal better with misfortune than people whose outlook interprets change as a threat, who feel out of control, and who lack a guiding purpose in their lives.

Stress Management: Controlling Emotion by Maintaining Rational Thinking

- ▼ Avoid fatalistic thinking (*No matter what I do, I will never be any stronger than I am now*).
- ▼ Avoid all-or-none thinking (*I must get an A in this class, or it will be a waste of my time; I can't have any chocolate or I will blow my whole diet*).
- ▼ Avoid catastrophic thinking, that is, making mountains out of molehills (*I couldn't keep up on the runs this week—I must have a metabolic disease that keeps me from adapting to exercise training*).

Candidates for Chronic Stress

People may be at risk for chronic stress if they

- ▼ feel overwhelmed by responsibilities,
- ▼ think they have too much to do in too little time, or
- ▼ feel uncertain about important consequences that seem out of personal control.

Does Exercise Reduce Stress?

Most researchers in exercise psychology who have studied stress have looked at whether exercise affects perceived stress. And many studies have confirmed that people generally report reduced or fewer symptoms of stress when they have been physically active. It appears that aerobic types of exercise lasting up to about 30 min generally are associated with the largest reductions in perceived stress. Aerobic exercise programs lasting at least a few months seem best for reducing reports of chronic stress. Though exercise usually won't eliminate the source of stress, exercise could help reduce stress temporarily by providing a short-term distraction from a problem. An exercise program might increase feelings of control or commitment (e.g., success in doing something important for yourself), which could buffer the impact of stressful events.

Relying on people's self-ratings of stress has problems, however. Using self-reports of reduced stress makes it difficult to disentangle the contribution of

exercise to stress reduction from a placebo effect. As in research on anxiety and depression, many participants enter the testing environment with expectations that exercise will decrease tension and improve mood. In addition, a self-rating of perceived stress does not adequately determine whether becoming physically active or physically fit indeed reduces behavioral or physiological responses during stressful events.



Acute exercise decreases behavioral and physiological manifestations of the stress response.

Studies that used objective measures of stress seem to confirm the self-reports, though. A few studies have shown that a single exercise session can reduce tension in muscles of the face, arms, and legs, as measured by **electromyography (EMG)** after exercise (deVries and Adams 1972; Smith et al. 2001). Other studies have shown that a single session of exercise can increase electrical brain waves (measured from the scalp by electroencephalography; see chapter 3) in the α frequency band (i.e., 8-12 cycles per second) by a half standard deviation when measured during and after exercise (Crabbe and Dishman 2001). Alpha waves are usually believed to reflect a mental state of relaxed wakefulness. However, exercise also increases smaller, faster β waves (i.e., 13-30 cycles per second) that are increased during brain activation, so it is not yet possible to view the brain wave studies as physiological evidence that exercise reduces stress.

Additionally, these studies did not show that people also *perceived* less tension or stress when the muscle or brain measurements were made. That research is discussed in more detail in chapter 5 on affect, mood, and emotion and in chapter 6 on anxiety. We should note too that most studies of physiological measures related to stress have been in experiments conducted in laboratory conditions and examined whether heart rate and blood pressure during mildly stressful tasks were lower among young and middle-aged adults who were physically active or fit versus people who were more sedentary and less fit, or examined whether those responses were lessened by a

single session of exercise (Jackson and Dishman 2002). Some of the limitations of research on exercise and stress are discussed in later sections of this chapter.

Physiological Mechanisms of the Stress Response

Understanding the possible mechanisms for a reduced stress response from exercise requires a basic familiarity with the key physiological responses during stress, how they are controlled by the nervous and **endocrine** systems, and how they differ among types of stressors. (See table 4.2 for types of stressors and associated responses.) Key components of the stress response involve neural and endocrine responses that are regulated by the brain and the autonomic nervous system. They include regions of the brain modulated by the neurotransmitters norepinephrine and serotonin, the sympathetic (including the adrenal medulla) and parasympathetic arms of the autonomic nervous system, and the HPA cortical axis.

Brain Norepinephrine and Serotonin

Norepinephrine and serotonin cells in the brain influence attention and vigilance, pituitary hormone release, and cardiovascular function during stress. They also influence pain, fatigue, and sleep. Neural discharge of the locus coeruleus and the raphe nuclei is increased during arousal, decreased during sleep, and absent during rapid eye movement sleep (when motor activity is inhibited). During stress, cells from the locus coeruleus release norepinephrine, and cells from the raphe nuclei release serotonin into the brain's frontal lobe and into the limbic system, including the hippocampus, amygdala, and hypothalamus (see figure 3.11, p. 52). Norepinephrine regulates other brain cells involved in vigilance against threat, helping to initiate behavioral, cardiovascular, and endocrine responses during stress. Serotonin helps the body return to rest after energy-expending behaviors (e.g., feeling satisfied and full after eating and feeling fatigued after exercise). In these ways, the locus coeruleus and the raphe nuclei operate in the brain similarly to the way the sympathetic and parasympathetic branches of the peripheral autonomic

Table 4.2

Characteristic Features and Physiological Responses to Common Tasks Used in Human Studies of Stress and the Autonomic Nervous System

Task	Coping (active vs. passive)	Sensory focus (intake vs. rejection)	Response pattern	ANS pattern
Mental arithmetic	Active	Rejection	↑HR, ↑SBP, ↑DBP, ↔ SV, ↑CO, ↔ TPR	Strong vagal withdrawal, β-adrenergic
Psychomotor reaction time	Active	Intake/rejection	↑HR, ↑SBP, ↑DBP, ↑SV, ↑CO, ↓TPR	Moderate vagal withdrawal, β-adrenergic
Stroop word-color conflict	Active	Intake/rejection	↑HR, ↑BP, ↑DBP, ↔ SV, ↑CO, ↔ TPR	Moderate vagal withdrawal, β-adrenergic
Forehead cold	Passive	?	↓HR, ↑SBP, ↑DBP, ↓SV, ↔ CO, ↑TPR	Vagal activity, α-adrenergic
Cold pressor	Passive	?	↑HR, ↑SBP, ↑DBP, ↓SV, ↔ CO, ↑TPR	Vagal withdrawal α-adrenergic

ANS = autonomic nervous system; CO = cardiac output; DBP = diastolic blood pressure; HR = heart rate; SBP = systolic blood pressure; SV = stroke volume; TPR = total peripheral resistance; ↑ = increase; ↓ = decrease; ↔ = little change.

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nervous system function to modulate the heart, vessels, and adrenal glands during stress.



During stress, norepinephrine helps to initiate behavioral, cardiovascular, and endocrine responses. Serotonin helps the body return to rest after energy-expendng behaviors.

Autonomic Nervous System

Features of the autonomic nervous system (ANS) most relevant for understanding cross-stressor adaptations to regular exercise include (1) innervation of the heart, blood vessels, and the adrenal gland by sympathetic nerves and the vagus nerve and (2) hypothalamic-pituitary hormone responses (see chapter 3, figure 3.13). Though there is great specificity in the ANS in response to different types of stressors, common neuroanatomy in the brain also permits coactivation of ANS responses during intense stress.

Sympathetic and Vagal Effects: Emotional Stress Versus Exercise Stress

Usually when someone experiences stress, activation of the sympathetic nervous system (SNS) increases. Sympathetic nerves from the portions of the spinal cord in the areas of the trunk stimulate organs such as the heart, the adrenal glands, and arteries. Under physical or emotional stress, sympathetic nerves stimulate the heart to beat faster and more forcefully, the adrenal glands to secrete epinephrine and norepinephrine, and the arteries that supply the heart and skeletal muscles to dilate so that blood flow is increased (see figure 4.3). During exercise, these actions help supply the extra blood needed to carry oxygen to the muscles used in locomotion. While the heart is beating, systolic blood pressure rises to help drive blood to muscle. While the heart rests between beats, diastolic pressure remains low, so there is little resistance to the flow of blood to the skeletal muscles (see figure 4.3).

During emotional stress, the same responses occur, though usually to a lesser degree, because the nervous, cardiovascular, and endocrine systems are preparing for a threatening situation, the so-called flight-or-fight response to danger. A major

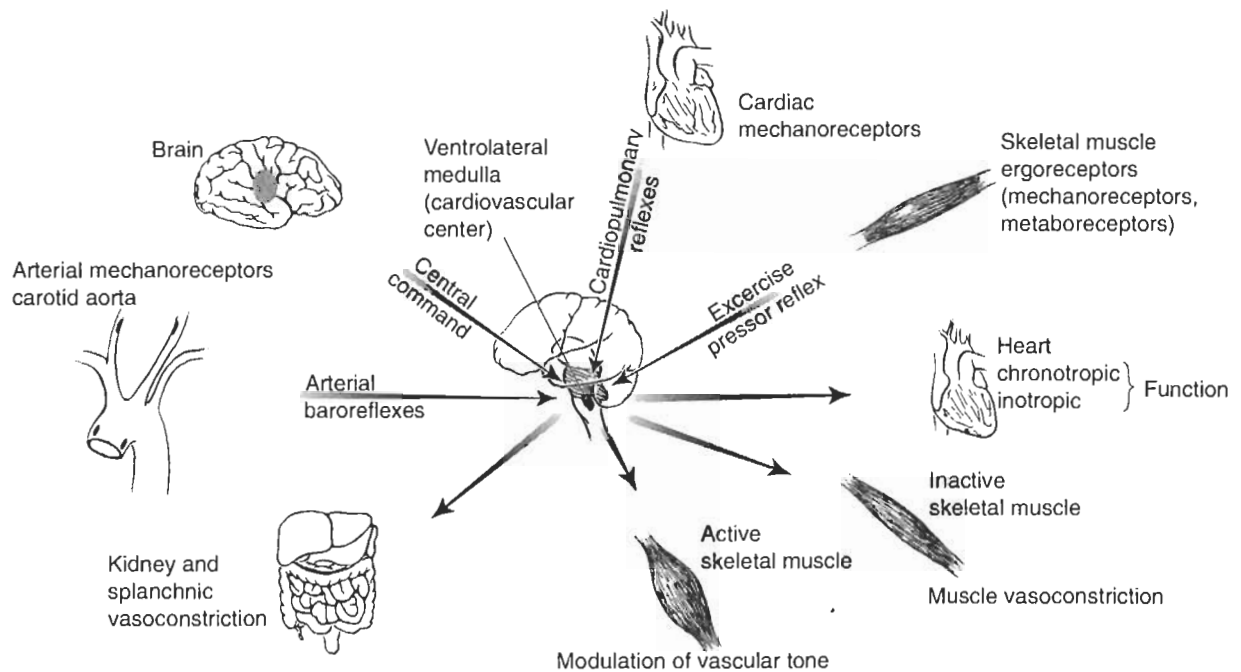


Figure 4.3 Depiction of the control of cardiovascular responses during exercise by central command from the brain and reflexive processing of sensory signals.

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difference between the two situations, exercise and emotional stress, is that the responses during exercise are necessary for the large increase in body metabolism (i.e., the need for extra energy). Emotional stress usually occurs at near-resting metabolism while people are anticipating a response such as fighting or fleeing from the danger. It is this elevated sympathetic response to perceived, not necessarily real, threat that is common among people with depression or anxiety disorders. Chronic elevations in several of the stress hormones when people feel threatened—but do not physically respond by fighting or fleeing—can make tissues in the brain, heart, and vessels vulnerable to injury or death. The physiological effects of chronically elevated stress hormones can contribute to diseases such as coronary heart disease and suppression of the body's immune system (see figure 4.4).

The main functions of the ANS during exercise are to regulate the increases in heart rate (HR) and blood pressure (BP) needed to increase cardiac output in order to support the increased metabolism of skeletal muscle cells. Secondary functions include the regulation of breathing and temperature. The cardiovascular pressor response, which regulates systolic BP during exercise, is understood to depend on a central command of autonomic efferent



In emotional stress, the ANS prepares the body for the flight-or-fight response to actual and perceived threat; metabolism is at or near rest with anticipation of threat. In exercise stress, the ANS supports increased metabolism of skeletal muscles and regulates breathing and body temperature; metabolism is elevated to perform physical work.

neural activity in the region of the temporal sensory/motor cortex. The centrally controlled pressor response is integrated at the ventrolateral medulla of the spinal cord with a pressor reflex arising from mechanoreceptors (e.g., sensitive to muscle tension) and metaboreceptors (e.g., sensitive to hydrogen ions) in exercising muscle (Mitchell and Raven 1994). Cardiopulmonary and arterial baroreflexes modulate the exercise pressor response, apparently by an upward and parallel resetting of the operating (i.e., set) point of the arterial baroreflexes resulting from central command (Rowell 1993). This means that BP is still regulated

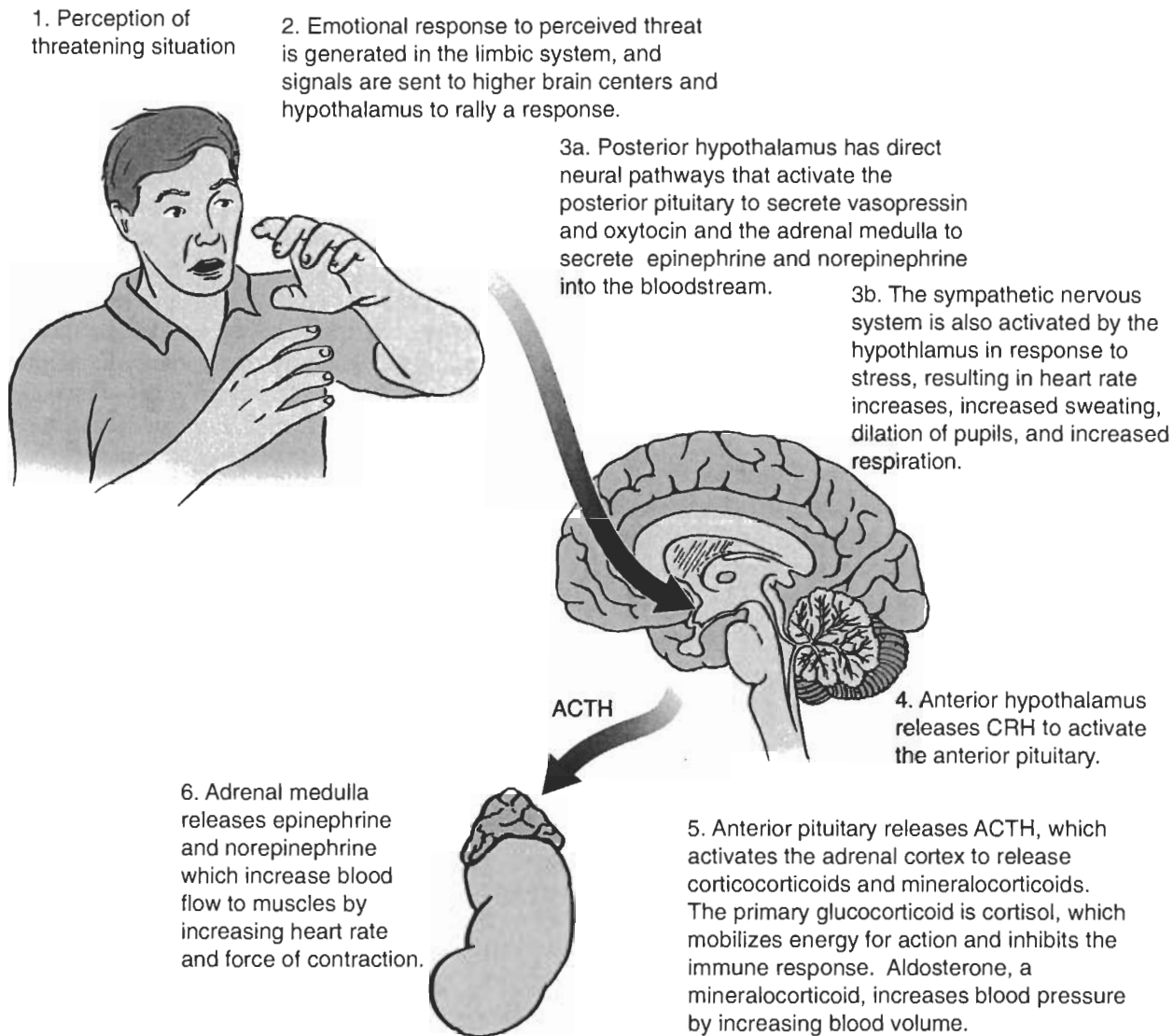


Figure 4.4 Several different systems are involved in supporting the cardiovascular response to stress.

by changes in HR but at much higher levels than at rest. Increased HR during exercise results from an initial withdrawal of cardiac-vagal inhibition of the heart. Vagal withdrawal and subsequent increase in heart rate are followed by increased activation of the heart by sympathetic nerve activity and by hormonal stimulation from catecholamines secreted by the adrenal medulla during intense exercise.

During exercise, the increase in blood levels of norepinephrine comes mainly from sympathetic nerves to the heart, with some of the increase also coming from the exercising skeletal muscles. Some may come from the brain. Exercise training does not usually change levels of norepinephrine in the blood or sympathetic nerve activity to muscles measured while people rest. However, after exercise

training, levels of norepinephrine in the blood are lower at a given **absolute intensity** (standard intensity) of exercise (e.g., running a mile in 6 min) and unchanged when that intensity of exercise is expressed as a percentage of **maximal aerobic capacity** (e.g., running a mile at 80% of top speed), but higher than normal at maximal exercise. This means that exercise training seems to increase the capacity of the sympathetic nerves to respond to maximal exercise, but does not change their responses to exercise of the same relative strain as before training. Also, there is no evidence that exercise training leads to a reduced sympathetic response to mental stress, when sympathetic response is measured by epinephrine or norepinephrine in the blood or by activity of sympathetic nerves to muscle.

Exercise adaptations for plasma norepinephrine are no change at rest, lower levels at a given absolute intensity, no change at the same relative intensity, and increased levels at maximal exercise.

Studies have shown that fitter people, especially women, have lower HR and BP during active mental stress (e.g., mental arithmetic and public speaking) compared to less fit individuals (Spalding et al. 2000); but that is mainly explained by their lower resting HR and BP, common adaptations to regular exercise. In other words, they have lower levels during stress because they have lower levels to start with, not because they have a smaller reaction to the stressors (Buckworth, Dishman, and Cureton 1994; Graham et al. 1996; Jackson and Dishman 2002).

Physically fit people have lower HR and BP during active mental stress because they typically have lower HR and BP at rest than unfit people have.

Lower HR among fit people could result from lower intrinsic rate of the heart (i.e., the rate of the heart's internal pacemaker) or lower activity by sympathetic nerves to the heart, but studies mainly show that it results from increased cardiac-vagal tone (see table 4.3 for measurement of ANS activity). The vagus nerve is part of the parasympathetic branch of the ANS. Its neurotransmitter is acetylcholine. Recall that the sympathetic nervous system stimulates energy expenditure and that the parasympathetic nervous system helps store and conserve energy (see chapter 3). The two systems work together to maintain a balance of the body's energy resources both at rest and during stress. For example, the vagus nerve slows the heart's frequency and force of beating and relaxes or dilates arteries that supply blood to skeletal muscle. So, a person who has increased vagal tone after exercise training can better offset the effects of the sympathetic nerves on heart and blood vessels and thus have lower HR and BP at rest and during stress. Increased cardiac-vagal tone also decreases the risk for irregular heartbeats and sudden death in people who have heart disease.

The high metabolism during exercise produces feedback to the brain and central nervous system from peripheral nerves and hormones in the blood; this feedback is used to regulate physiological homeostasis. It is easy to understand why regular exercise and increased fitness would lead to lower HR,

Table 4.3

Measurement of Autonomic Nervous System Activity: Heart Rate Variability

It is not feasible to directly measure the firing rates of the sympathetic and vagus nerves that innervate the heart. Thus, their relative activity is commonly estimated by heart rate variability (HRV).

Experiments in nonhuman animals have shown that electrical stimulation of the cardiac sympathetic nerves results in fluctuations in heartbeat that are large but slow (LF), whereas stimulation of the vagus nerve results in rapid changes in heart rate (HF).

Term	Definition
Heartbeat	The period of the heart; the time between the R waves in successive QRS complexes
Hertz (Hz)	Frequency, or the number of cycles per second
Low frequency (LF)	.05 to .15 Hz
High frequency (HF)	.15 to .50 Hz
<i>Estimates of autonomic balance during short-term fluctuations in heart rate:</i>	
Cardiac-vagal component:	<i>HF is normalized to total power:</i> $[HF / (HF + LF) \times 100]$
Sympatho-vagal component:	<i>LF is expressed relative to HF:</i> (LF / HF)

BP, and stress hormones during exercise and to a quicker recovery, because that adaptation would preserve homeostasis and reduce allostatic load. However, it is more difficult to understand why such adaptations to exercise would transfer to other stressors that do not have a high energy cost and do not involve exertion using skeletal muscles. It is not apparent that cardiovascular adaptations to exercise should be expected to generalize to other stressors that do not impose similar psychomotor demands.

In contrast to exercise, most non-exercise stressors elicit little or no sensory afferent activity to regulate cardiovascular responses. Thus, much of any cross-stressor adaptation after exercise must involve central command (i.e., motor nerve discharge to the heart, vessels, or adrenal medulla) or altered responses by organs to central command (e.g., decreased number or sensitivity of receptor cells that bind with epinephrine or norepinephrine). Though such propositions are plausible, the studies done to this point do not support the idea that regular exercise or cardiorespiratory fitness results in a blunted physiological response to stress other than exercise.



Much of any cross-stressor adaptation after exercise must involve central command or altered responses by organs to central command.

On the other hand, studies in rats have shown that voluntary running in activity wheels leads to a blunted stress response during foot-shock stress (as measured by less release of norepinephrine in the brain cortex) (Soares et al. 1999) and a protection against suppression of the innate immune system (Dishman et al. 1995), which appears to be modulated by the sympathetic nervous system during stress (Dishman, Hong, et al. 2000).

Hypothalamic-Pituitary-Adrenal Cortex System: Emotional Stress Versus Exercise Stress

The ANS is activated during stress, but the brain also stimulates energy production by activating endocrine organs, including the pituitary gland and the outer part (cortex) of the adrenal gland (see fig-

ure 4.5). During stress and exercise, the anterior pituitary gland releases adrenocorticotropin (ACTH), which stimulates the adrenal cortex to secrete cortisol. The amount of ACTH available for release is regulated by a gene that is activated by corticotropin-releasing hormone (CRH). This gene is located mainly in the arcuate nucleus of the hypothalamus, which expresses a macromolecule, **proopiomelanocorticotropin (POMC)**. ACTH and **beta-endorphin** are cleaved from POMC and are secreted together from the pituitary during stress. Although nerve cells containing CRH are located throughout the brain, most of the CRH that increases ACTH levels comes from the paraventricular nucleus (PVN) of the hypothalamus and is released into the portal blood supply to ACTH-secreting cells in the pituitary. During stress, release of norepinephrine and dopamine in the PVN activates CRH to increase ACTH. Secretion of CRH is inhibited by the hippocampus.

Moderate exercise training results in a diminished HPA response during the same absolute exercise intensity compared to that before training. However, heavy exercise training can be associated with abnormal HPA responses under resting conditions (see chapter 5, p. 113). Generally, fit people have an increased capacity to respond to severe stress. Whether exercise training or aerobic fitness influences HPA responses to milder mental stresses such as daily hassles is not yet known. One study (Sinyor et al. 1983) indicated that trained men had higher levels of cortisol at rest, under mental stress, and during recovery when compared to sedentary men; but the rates of response and recovery were the same for the trained and the untrained men. In other studies, men differing in fitness levels had similar levels of cortisol or ACTH in plasma after mental stress regardless of whether it was novel (Sothmann et al. 1988) or familiar (Blaney et al. 1990). Animal studies have shown no effects of chronic activity-wheel running on plasma levels of ACTH and cortisol after repeated foot-shock in female and male rats (Dishman et al. 1995, 1997).

Exercise studies usually have not measured or controlled reproductive hormones known to influence physiological responses to non-exercise stressors, despite evidence of an interaction between the HPA cortical and the HPA gonadal systems in highly trained women. Treadmill exercise training of female rats treated with estrogen was accompanied by an attenuated ACTH response to familiar treadmill running but a hyperresponsiveness of

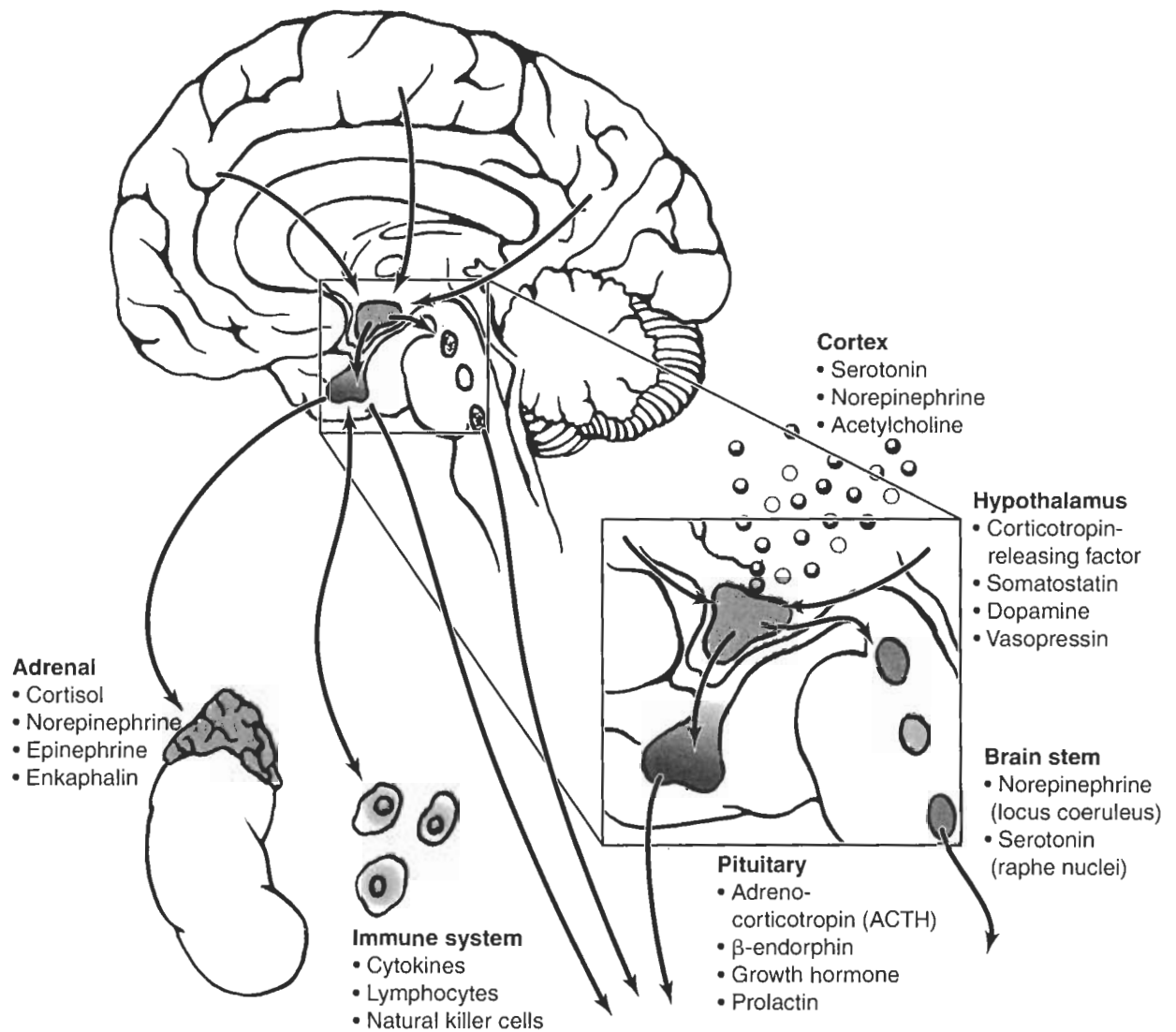


Figure 4.5 Responses of the hypothalamic-pituitary-adrenal cortex system and the sympatho-adrenal medullary system during stress.

Adapted from R.H. Black, 1995, "Psychoneuroimmunology: Brain and immunity," *Scientific American* 2 (6): 17.

ACTH to novel immobilization or foot shock (White-Welkley et al. 1995, 1996). Whether this hyperresponsiveness of ACTH is a healthful adaptation and whether it is due to increased CRH or other factors that release ACTH is not known. The latter seems likely since treadmill exercise training in male rats is accompanied by reduced ACTH after immobilization stress with no change in brain CRH (White-Welkley et al. 1996). Those findings might indicate that the energy and neuromuscular demands of treadmill running lead to an increased potential for HPA responses to novel stressors.

Contemporary Views: Exercise Research

The first review of the cumulative evidence from 25 studies of fitness and physiological stress responses concluded, nearly 15 years ago, that aerobic fitness reduced stress responses by about a half standard deviation, regardless of the type of stressors used or the physiological responses measured (Crews and Landers 1987). Since then, there has not been scientific consensus with that early con-

clusion (see Dishman and Jackson 2000). This is the case largely because research in exercise psychology has done a poor job in building on what is known about the physiology of stress; especially problematic are the absence of a clear characteriza-

tion of the features of the stressor used and the lack of any consideration of the regulatory mechanisms that govern physiological stress responses. Specifically, the lack of consensus is explainable primarily by the five factors described in figure 4.6.

Factors limiting past research on exercise and stress reactivity

1. Measurement of fitness and exercise

Early studies did a poor job defining or measuring fitness or exercise. Thus it was hard to determine whether people differed enough to permit a true test of the influence of fitness or exercise habits on stress responses. Also, the use of submaximal heart rate (HR) to estimate peak oxygen uptake ($\dot{V}O_{2\text{peak}}$) confounded the use of HR as both an independent variable (i.e., level of fitness) and dependent variable in several studies. A test-anxious person could show exaggerated HR responses to the exercise test and to the other stressors and be misclassified as unfit because of the emotional elevation in HR during exercise.

2. Measurement of physiological variables

The manner of reporting the methods used to measure physiological variables and to compute their change in response to stressors made it difficult to determine whether the procedures in many studies met international standards for psychophysiological research. The accuracy of the measures was questionable in some studies; the influence of pretest baselines on the stress variables that were measured during stress and recovery was not accounted for in several studies, likely giving a false measure of responsiveness.

3. Research design

About two-thirds of the studies used a cross section of time to compare stress responses among groups classified according to levels of fitness or exercise (rather than comparing responses after a change in fitness or exercise) and did not match the groups on other factors known to influence stress responses, such as temperament, behavior patterns, or reproductive hormone status.

4. Consideration of integrated physiological responses

Investigators gave inadequate consideration to physiological mechanisms that explain variations in integrated responses of variables, such as HR, blood pressure, or circulating stress hormones like norepinephrine, epinephrine, and cortisol. For example, HR responses to a stressor might be similar between people of different fitness levels, but the reasons might be different. A fit person might have less withdrawal of parasympathetic nerve inhibition of the heart despite a similar or greater sympathetic nerve stimulation of the heart. Though the integrated HR response might not differ from that of an unfit person, the different pattern of cardiac sympatho-vagal balance would be important, as it is known to have health consequences.

5. Consideration of characteristics of stressor tasks

Researchers generally failed to compare standardized stressor tasks of equal novelty or difficulty and did not choose tasks according to common or unique features that induce specific or general stress responses (e.g., different sympatho-vagal and sympatho-adrenal medullary responses). Exercise adaptations might extend to certain types or intensities of stressors but not to others.

Figure 4.6 These five factors help to explain why consensus has not been reached in exercise psychology research.

The type of stressor used in research on exercise and stress reactivity is a particularly critical issue (see figure 4.6, factor 5). Unless a stressor is strong enough to engage a general flight-or-fight response (e.g., life or death threat), responses during stress differ widely according to the type of stressor (Allen and Crowell 1989; Dishman, Jackson, and Nakamura 2002) (see table 4.2). Active stressors motivate the person to try to control the challenge (e.g., mental arithmetic, quizzes, reaction-time tasks). Physiological responses include increases in HR, cardiac output, systolic BP, and withdrawal of vagal tone. Passive stressors offer little or no opportunity for the person to control an aversive situation and commonly result in increased HR and increased peripheral resistance to blood flow and diastolic BP. Other responses can also occur such that increased vagal tone results in decreased HR and decreased BP (a so-called playing-dead response). One passive test that elicits a cardiac-vagal response in many people is application of cold to the forehead. Forehead cold can increase BP because of increased resistance to blood flow even though HR is reduced. This response is similar to the mammalian diving reflex, which involves a vagally mediated bradycardia and α -adrenergic vasoconstriction of the skin and viscera.

Generally, BP responses are greater during hand immersion in cold water (cold pressor) than during mental arithmetic, which in turn elicits greater responses than does a psychomotor reaction-time task. In contrast, cardiac output is greater during a reaction-time task or mental arithmetic than during a cold-pressor test. Heart rate increases the most during mental arithmetic compared to the cold-pressor test or reaction-time task. Increased HR during mental arithmetic is explainable by vagal withdrawal, whereas during a reaction-time task it is more influenced by sympathetic innervation of the heart. Blood flow also differs according to type of stressor. Increased cardiac output during a reaction-time task is mainly explained by increased stroke volume, whereas during mental arithmetic it is explained by increased HR. Cardiac output during the cold-pressor test is unchanged because the increased HR is offset by decreased stroke volume (Dishman, Jackson, and Nakamura 2002).

Limitations of past research also make it too early to conclude that regular exercise has no effect on responses during stress that are modulated by the sympathetic nervous system. The reason for the absence of effects in past studies might be the narrow range in the increase of plasma catecholamines

evoked by the stressors that researchers have used. Most of the stressors used have been mild, eliciting small increases in a range of about 300 to 500 pg/ml for norepinephrine and 40 to 80 pg/ml for epinephrine. Those levels are below thresholds for norepinephrine (1500-2000 pg/ml) and epinephrine (75-125 pg/ml) that reliably elicit increases in HR and systolic BP (Clutter et al. 1980; Silverberg, Clements, and Fiatarone 1978). A five- to tenfold elevation in norepinephrine and a twofold increase in epinephrine are generally believed necessary for cardiovascular effects, yet the stressors used have seldom resulted in a doubling of catecholamines above basal levels. In contrast, moderate-to-heavy exercise results in a six- to tenfold increase in norepinephrine and a tripling of epinephrine (Clutter et al. 1980).

The types of stressors used in studies of exercise and stress have been milder than many events that are stressful in real life. The experimental stressors usually increase HR by altered sympatho-vagal balance of the ANS's innervation of the heart rather than by hormonal response. For example, during mental arithmetic, heart transplant patients—who have had the autonomic nerves to the heart severed—have an increased BP (Sehested et al. 1995) but not the increase in HR (Sehested et al. 1995; Shapiro et al. 1994) that is observed in people with innervated hearts. Thus, tasks like mental arithmetic do not elicit a stress response by the adrenal gland that is of sufficient magnitude to increase HR.



Evidence for modification in the response to mental stress after exercise training is equivocal, but there might be beneficial effects from enhanced vagal tone.

Nonetheless, it remains plausible that increased cardiac-vagal tone after regular exercise might generalize to blunt responses to mild stressors that elicit increases in HR and BP mainly by vagal withdrawal. In contrast, responses by the sympathetic nervous system seem to be unique to different stress organs during mild stressors. Hence, whether altered regulation of the sympathetic nervous system after regular exercise might lead to a generalizable response seems less clear and could depend on the intensity of exercise and other stressors.

Summary

Adults without stress disorders typically say that they feel less stressed after a single exercise session and after a regular exercise program. However, studies have not yet shown convincingly that those findings were uninfluenced by people's expectations of benefits. There has been no research to determine whether exercise reduces stress among people diagnosed with stress disorders. Studies in which physiological responses were measured have shown that cardiorespiratory fitness is associated with a slight blunting of HR and systolic BP reactions during active mental stress, but not during passive stress, such as that caused by placing the hand in ice water. More often, fit people have a lower overall level of HR and, many times, of BP during stress because they have lower resting levels, not because they have a smaller reaction to the stress than do unfit people.

Research has not shown that fitness affects catecholamine responses during stress, but the stressors used have been mild and have not led to responses large enough to adequately test whether fitness really alters the sympathetic response by the adrenal gland. There are not enough studies of other hormone responses, such as cortisol, to permit us to conclude whether exercise and fitness alter other endocrine responses during stress.

There has been very little use of the traditions and methods of biological psychology and neuroscience in the study of exercise, fitness, and stress. Though many people say that exercise helps them cope with stress, we cannot rely solely on social cognitive models of stress that use people's self-rated perceptions to determine whether, or in what circumstances, cardiorespiratory fitness or regular exercise leads to blunted or augmented physiological responses during stress and enhances

recovery from stress. Modern students of exercise psychology should learn the basics of neuroanatomy, neurophysiology, and psychopharmacology as well as the techniques of neuroscience to help them conduct sound research on exercise and stress or to collaborate effectively with physiologists or biological psychologists. Nonetheless, the subjective experience of stress remains a cornerstone of inquiry into the nature of stress emotions such as anxiety and depression. Discussions of those topics follow.

Suggested Readings

- Dishman, R.K. 1997. Brain monoamines, exercise, and behavioral stress: Animal models. *Medicine and Science in Sports and Exercise* 29 (Jan): 63–74.
- Dishman, R.K., and E.M. Jackson. 2000. Exercise, fitness, and stress. *International Journal of Sport Psychology* 31: 175–203.
- Sapolsky, R.M. 1994. *Why zebras don't get ulcers—A guide to stress, stress-related diseases, and coping*. New York: Freeman.
- Sothmann, M.S., J. Buckworth, R.P. Claytor, R.H. Cox, J.E. White-Welkley, and R.K. Dishman. 1996. Exercise training and the cross-stressor adaptation hypothesis. *Exercise and Sport Sciences Reviews* 24: 267–287.

Web Sites

- www.nimh.nih.gov/publicat/index.cfm
- www.surgeongeneral.gov/library/mentalhealth/index.html
- www.nhlbi.nih.gov/health/index.htm
- www.clas.ufl.edu/users/gthursby/stress/