ENCYCLOPEDIA OF PSYCHOLOGY

Alan E. Kazdin
Editor in Chief

VOLUME 2

AMERICAN PSYCHOLOGICAL ASSOCIATION

OXFORD
UNIVERSITY PRESS

New York

2000

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Michael M. Sokal

STRESS. [*This entry comprises three articles. The lead article discusses various definitions of stress and the physiological components and manifestations of stress. The two companion articles provide overviews on the measurement of stress and on its effects on health.*]

Definition and Physiology

The challenge of stress physiology is to elucidate the mechanisms that underlie the relationship between the risk factor of stress and the outcome measure of health. How and under what conditions does stress promote disease?

For far too long stress physiologists have relied on the rather barren concept of homeostasis as the basis for understanding the concept of organisms fighting tenaciously to maintain physiological systems within the range of some ill-defined set point. Within this framework, a stressor is simply any event that threatens homeostasis. This was a useful starting point for the contributions of Canon and others in the 1930s, but the time has long passed since this idea was of value to health scientists (Sterling & Eyers, 1988). Such simplistic notions defy Darwinian biology (Weiner, 1992). Life is full of struggle against the obstacles, challenges, and threats in our environment. Success is most often measured not in the defense of set points, but in our ability to adapt to such conditions, and the ease with which such adaptations occur. Adaptation, not stability, is the essence of life, biological fitness, and health (Selye, 1978).

Adaptation may be considered a set of changes that occur in order to maintain a normal level of function. One of the most important innovations in stress research is the understanding that illness is best understood not in terms of the nature of the stressful events to which an individual is exposed, but in terms of the magnitude of the individual's physiological and psychological responses to the stressor that are necessary to maintain function. The magnitude of these responses determines the likelihood of disease. The same set of responses that are activated in order to survive a stressor and maintain function can produce vulnerability to disease. This idea was advanced by Selye, who referred to many forms of chronic illnesses as "diseases of adaptation."

The brilliance of Selye's science was to recognize the common set of reactions that occurred in response to a wide variety of conditions, including various disease states, and involved fatigue, loss of appetite, and muscle wasting. These reactions formed the general adaptation syndrome, which lay at the heart of the relationship between stress and disease.

Physiological Responses to Stress

For Selye, the first phase of the general adaptation syndrome was the alarm reaction, a set of responses designed to meet the immediate needs of the body and analogous to a fight/flight response (Canon, 1929). Two systems have always been considered primary here. The hypothalamic-pituitary-adrenal (HPA) axis, emphasized by Selye, produced adrenocorticosteroids, mainly cortisol, and mineralocorticoids, including deoxycortisol and aldosterone. Second, the sympathetic nervous system, emphasized by Canon, focused on adrenomeduallary catecholamines, including adrenaline and noradrenaline (or epinephrine and norepinephrine). Together, these hormones furnish the front line of defense for mammals in response to stressors.

As Selye pointed out, there are common pathways that mediate any attempt to adapt to environmental demands and sustain function. A primary concern here is the change in metabolism that occurs in response to a stressor. One reasonably simple way to see how stress can compromise health is to simply appreciate the demands placed on the body during a period of stress. During a stress reaction, many cells are working harder to meet the demands of the situation. If these cells are more active they require more fuel—a greater ability of energy substrates. In the absence of a sufficient level of energy substrates, cells begin to shut down and can even run the risk of ischemic death. A basic feature of the body's response to a stressor is designed to ensure the adequate availability and distribution of energy substrates to specific organs in the body, notably those involved in assuring survival (Sapolsky, 1992). The resultant increase in plasma glucose levels also assists cognitive performance levels during periods of distress.

First, high levels of stress hormones, including the catecholamines, adrenaline, and noradrenaline, as well as the glucocorticoids, principally cortisol in humans, are produced from the adrenal gland. These hormones

orchestrate a move to catabolism, increasing lipolysis and mobilizing glucose reserves. Glucose, fatty acids, and amino acids are then liberated from their macromolecular storage forms of glycogen, fat, and protein. Glycogen is converted into glucose and released into the bloodstream. The increase in lipolysis serves to break down fats and thus elevate circulating levels of fatty acids and glycerol (Brindley & Rolland, 1989), both of which can be used effectively as energy substrates by cells. Certain protein stores are broken down into amino acids, which then serve to fuel the stress hormone–induced increase in hepatic gluconeogenesis. These actions serve to defend blood glucose levels during stress. In addition, both the catecholamines and the glucocorticoids serve to antagonize the effects of insulin, decreasing the tissue response to it. This is no time to be storing sugars away in the form of glycogen. Rather, glycogen stores are broken down into glucose and poured into the bloodstream. Collectively, these responses serve to increase the availability of energy substrates. This is essential for survival.

Second, not only does the production of energy substrates increase, but so does their distribution. During stress, sympathetic activity increases arterial blood pressure through vasoconstriction as well as through heart rate. These actions increase blood flow. Interestingly, these actions are highly selective. Blood flow to active muscles, the liver, and the brain are increased. In contrast, circulation to the skin and mesentery is decreased. Digestion is thus compromised—this is no time for an all-dressed pizza.

Third, we also respond to stress as if performing a small cost-benefit analysis of our investment of resources. During a period of serious threat, survival is at risk. Growth hormone production is shut off and anabolism is suppressed. The elevated levels of glucocorticoids also serve to dampen tissue sensitivity to growth hormone. These events ensure that during a crisis growth-related processes are not draining energy reserves from vital organs. This is no time to be investing precious energy substrates into bone density and muscle mass. These processes can wait until the threat has subsided. Likewise, since processes such as ovulation are not essential to survival at this time, we turn off our reproductive function. Leutinizing hormone production from the anterior pituitary is suppressed during periods of stress as part of a general suppression of anabolism.

Fourth, there are cognitive responses to stressors that include systems that mediate attentional processes as well as learning and memory. During a stress reaction, we become hypervigilant; the level of attention directed to the surrounding environment is increased at the expense of our ability to concentrate on a focused set of tasks that are not essential for survival. As a function of these changes in attentional processes, as well as the effects of glucocorticoids on brain structures such as the hippocampus, episodic memory is less functional during periods of stress. At the same time, glucocorticoids act on areas of the brain such as the amygdala to enhance emotional learning and memory (Lupien & McEwen, 1997). These changes in psychological function obviously greatly depend on changes in central nervous system activity and are associated with altered emotional states (Davis, 1992; Le Doux, 1995). Feelings of apprehension and fear predominate during a stressful experience. Thus, stress has effects on cognition and emotions that are consistent with the demands of the situation.

Corticotropin-Releasing Hormone

Perhaps one of the most significant advances in stress research is the identification of a neural hormone, corticotropin-releasing hormone (CRH), which appears to initiate and coordinate behavioral, emotional, autotonic, and endocrine responses to stressors. The CRH is a 41-amino-acid peptide, and while it is widely distributed throughout the brain, two CRH pathways appear to be principally involved in the expression of autotonic, behavioral/emotional, and endocrine response to stress (Sawchenko & Swanson, 1981). The first is a CRH pathway from the paraventricular region of the hypothalamus (PVNh) to the hypophysial-portal system of the anterior pituitary, a system that serves as a principal network for the neural regulation of the HPA axis. In response to stressors, CRH, and often arginine vasopressin (AVP), are released from PVNh neurons into the portal blood supply of the anterior pituitary, where they provoke the synthesis and release of adrenocorticotropin hormone (ACTH; Plotsky, 1991). In turn, pituitary ACTH causes the release of glucocorticoids from the adrenal gland. This hypothalamic-releasing factor system thus serves as the means by which a neural signal associated with the stressor is transduced into an endocrine response.

Another subset of hypothalamic CRH neurons project to midbrain sites regulating autonomic activity, resulting in changes in sympathetic nervous system activity (Brown, 1989). The second CRH pathway arises from the central nucleus of the amygdala and projects to the noradrenergic cell body regions of the locus coeruleus, the n. tractus solitarius, and the parabrachial n. as well as the serotonergic cell bodies in the raphe nuclei. Activation of these CRH projections results in an increase in the firing of targets and an increased release of serotonin and noradrenaline (Dunn & Berridge, 1990). These monoaminergic responses mediate behavioral/emotional responses to stress (Koob et al., 1994). The CRH antagonists can attentuate or even block the behavioral/emotional responses to stressors (Le Doux, 1995; Davis, 1992; Koob et al., 1994). These CRH systems therefore play an essential role in medi-

ating the effects of stressors on emotional and behavioral states, as well as regulating autonomic activity.

Chronic Stress

Chronic, intermittent, or chronic continuous stress generally has two important effects on stress hormone levels. First, it increases basal plasma levels of glucocorticoids and cathecholamines. In response to chronic stressors, there are increases in the resting levels of heart rate and blood pressure, as well as in the circulating levels of triglycerides, cholesterol, and fatty acids. The second effect is an increase in reactivity to new forms of stress. This effect is referred to as facilitation by neuroendocrinologists studying the HPA axis, and sensitization by neurochemists studying either peripheral or central catecholamine responses to novel forms of stress. In addition, chronic stress, at least in studies of rodents, can serve to prolong the stress hormone response, such that the hormonal response persists even following the termination of the stressor. Together, these effects increase the cumulative exposure to stress hormones.

While these changes have been seen as the hallmarks of chronic stress, it appears that the long-term effects of a serious form of acute stress may have been underestimated. Severe forms of acute stress produce enduring changes in both basal HPA activity (Tannenbaum et al., 1997) as well as hypothalamic-releasing factor systems.

Chronic stress increases CRH and/or AVP gene expression in both the PVNh and CRH expression in the central nerve of the amygdala (Schulkin, McEwen, & Gold, 1994). These effects form the basis for the chronic stress–induced increase in endocrine, autotonic, and emotional responses to acute stress. Increased CRH activity has been associated with depression and anxiety disorders (Stout & Nemeroff, 1994), and may provide a neural basis for stress-induced affective disorders. Interestingly, antidepressant drugs have been found to serve as useful prophylactic substances, dampening the effects of chronic stress. These compounds have been found to decrease CRH gene expression in both the PVNh and the central n. of the amygdala (Brady et al., 1991). These findings may underlie the anxiolytic effects of certain antidepressant medications.

Resistance, Adaptation, and Illness

Allostasis. Allostasis is the establishment of a new level of activity in the brain and the rest of the body in response to the demands imposed on an individual over an extended period of time (i.e., chronic stress). Stability of function is maintained through allostasis. This is the essence of adaptation. For example, under conditions of stress we experience an increase in blood pressure and higher levels of fat metabolites, triglycerides, and fatty acids in circulation. When the conditions become chronic, the body begins to establish a higher baseline level of blood pressure and fat metabolism, and seems to defend this new state with appropriate changes in the circulatory system and increased fat consumption. The net result of these changes is the allostatic load—the burden an individual carries around as a result of adapting to adversity. In this case the burden is represented by increased blood pressure and circulating fat levels, but the reader can certainly add to this list. Irritability (perhaps associated with higher vigilance), listlessness, fatigue, and changes in appetite and digestion come readily to mind (see Sapolsky, 1992; Weiner, 1992, for reviews).

The critical assumption here is that the greater the allostatic load, the greater the risk of illness (McEwen, 1998). Seeman, Singer, and Charpentier (1995) recently tested this hypothesis using a cumulative index of allostatic load based on a variety of measures, including systolic blood pressure, urinary cortisol and cathecholamine levels, hip-to-waist ratio, glycosylated hemoglobin, measures of serum-high density and lipoprotein, and cholesterol. Based on a 3-year follow-up, it was found that individuals with higher allostatic loads were more likely to have cardiovascular disease and declines in physical and cognitive functioning. In our longitudinal studies with an otherwise healthy population of elderly clients, we found that basal cortisol levels over several years were negatively correlated with hippocampal volume and cognitive performance (Lupien et al., 1998).

Chronic Stress and CHD. The concept of allostasis leads to an understanding of the relationship between stress and disease. Disease emerges not directly as a function of the stressor but rather as a function of responses to the stressor. The constant pressures of a hated job does not directly promote heart disease. What leads to illness is the increased blood pressure and heart rate along with the elevated levels of fat metabolites produced in response to stress. Likewise, the suppression of immune function, the antagonism of insulin action, and the inhibition of growth hormone release are all natural features of response to stress, and all are conditions in which stress can promote illness. The question then becomes one of whether stress will promote illness.

In attempting to address the complex question of individual differences I have always found it useful to return to basics. Lazarus defined stressful conditions as those in which the demands of a situation exceed the resources of an individual (Lazarus & Folkman, 1984). This effort follows in the tradition of Mason (1975), in emphasizing both the stressful stimulus and the individual. This is a simple yet important definition, for it recognizes not only the stimulus event and its impact on the individual, but also the fact that individuals differ in their resources. Moreover, and this is essential,

the definition crosses disciplinary boundaries. The stimulus event can refer to verbal abuse, a horrible recurrent image, or the presence of an antigen. Likewise, the resources can refer to the ability of certain neurotransmitter systems to maintain normal function, the capacity of an individual to call upon a social network of friends and family, or the capacity of the immune system to attack and destroy the viral assailant.

There are features of a situation that can produce greater stress responses very reliably. Obviously, the intensity or severity of a stressor is a major factor, but so too are the features that determine the nature of cognitive responses or appraisals. When there is a high degree of uncertainty surrounding events, when outcomes are unpredictable—these are situations generally seen as being far more effective in producing physiological responses. Perhaps the critical feature is that of controllability: Conditions in which we feel we have no effective means of coping with events produce the greatest set of stress responses. This is, of course, entirely consistent with the definition of Lazarus, who suggested that a situation is seen as stressful when one appraises it as being beyond control.

There are at least two obvious sources of individual difference here. The first lies in the appraisal of the meaning of the stressor; the second lies in the resources available to the individual. The two are intimately related. A stressor is evaluated by an individual in terms of available resources. We respond not to events, per se, but rather to our perception of events. There is clearly room for tremendous individual variation in the appraisal of stressors; the same events can have very different meaning and significance to different people, and thus elicit very different reactions. Such variations are partly associated with stable individual differences in activity in the left and right prefrontal cortex (Davidson, 1992), which are, in turn, associated with individual difference in stress hormone release (Wittling, 1995). It is tempting to think that these findings might provide a neurobiological basis for the effect of optimism on responses to stress, and a relationship between optimism and allostatic load.

The research of Hellhammer and colleagues provides a wonderful insight into the basis for individual differences in hormonal responses to stress. Following years of study, Hellhammer found that the single most influential factor in determining individual differences in cortisol responses to acute stress was self-esteem (Seeman, Singer, & Charpentier, 1995). Individuals high in self-esteem showed far more modest hormonal responses to an acute social stressor than did persons low in self-esteem: The assessment of one's resources, reflected in the measure of self-esteem, determined the magnitude of the response to stress. This idea seems to parallel the older ideas of mastery (White, 1960) and would seem to be an essential feature in determining the psychological basis for individual differences in stress reactivity. The greater our estimation of our resources, the smaller the stress response. Interestingly, early developmental histories that undermine self-esteem also increase the sensitivity of neural CRH pathways to stress.

There is obviously a long path to travel before we can clearly understand these issues, but more recent studies provide the basis for some fascinating hypotheses concerning the mechanisms through which social support, self-esteem, and optimism might influence health (Taylor, Repetti, & Seeman, 1997). Do these factors produce good outcomes because, over the lifetime, they serve to dampen the impact of stressors and reduce allostatic load? In addition to self-esteem, it appears that a case could be made for the effect of social support on cardiovascular, endocrine, and immune responses to provocation (Uchino, Kiecolt-Glaser, & Cacioppo, 1996), allostatic load, and health outcomes (Cohen, 1988).

The same logic can potentially be applied to understanding why psychotherapy might serve to decrease the risk of illnesses such as heart disease or diabetes, or assist in the management of these disorders. Several forms of psychotherapy, such as cognitive-behavioral or interpersonal therapies, can be viewed as increasing the resources of the individual (or the perception of existing resources) in the face of existing demand. Therapies that focus on coping skills and decision-making as a means of enhancing controllability can decrease stress hormone response (e.g., Dienstbier, 1989). Increased resources and a greater sense of control should thus serve to dampen stress hormone responses, decrease allostatic load, and improve health outcomes.

We must accept that stress research involves a degree of ambiguity. We are not unique here. The concept of heart disease is equally vague and nonspecific. Heart disease takes many forms and frequently involves kidney dysfunction, metabolic disorders such as diabetes, as well as the myriad effects associated with circulatory failure. Yet we continue to pursue the study of heart disease despite the fact that we know its form will differ for each individual—so too is it with stress research. The advantage of the allostatic model is that it provides a clear set of hypotheses concerning the mechanisms through which stress can influence health, as well as a basis for collaboration between individuals whose focus is psychosocial or psychological mediators. Only through such multidisciplinary efforts will we fully understand how stress promotes illness.

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tory, Revised Research Edition. Odessa, FL: Psychological Assessment Resources.

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Impact on Health

Stress has broad effects on mood, behavior, and health. It involves changes in response of nearly every system in the body, affects how we feel, and influences our behavior. By causing these whole-body changes, stress contributes directly to disease processes and affects health by increasing high-risk behavior and reducing quality of life. Evidence for these broad effects is increasing despite the difficulty in doing research on stress and illness. Recent data suggest that stress can affect health in many different ways and that it can be managed so that its effects are reduced.

Stress has been hard to define, but most investigators agree that it is caused by events or thoughts that convey threat, harm, or loss. These stressors are appraised or interpreted, and to the extent that they are perceived as dangerous, stress is experienced. Stress is a general term referring to a state or response pattern characterized by changes in most bodily systems, mood, and behavior. Biological and psychological arousal alters mood and behavior and elicits coping responses directed at reducing or eliminating the source of stress or its effects. This arousal is characteristic of stress and heightens the motivation to cope with the stressor and increases attempts to eliminate or reduce its impact.

Stress responses appear to have been naturally selected. Walter Cannon, a noted American physiologist (writing in 1929) believed that stress evolved to serve as a general energizer and regulator of emergency reactions and response to life threat. Often referred to as the fight/flight response, stress has helped people survive by increasing their strength, alertness, and stamina. Stress increases the body's metabolism and blood flow to skeletal muscles and the brain, and enhances peoples' ability to react. Biological aspects of stress are initiated by the nervous and endocrine systems, principally by sympathetic nervous system (SNS) arousal. Release of epinephrine, norepinephrine, and cortisol (among other hormones and neurotransmitters) is characteristic, and these actions signal bodily systems to break down and release stored energy and to increase the speed and efficiency of its distribution, resulting in an alert, stronger, and more robust organism. Digestive processes are slowed, and the availability of glucose is increased in circulating blood to fuel the major muscle groups. Heart rate, blood pressure, and respiration increase to improve circulation and the delivery of nutrients to muscles and brain. These readying responses work well when stressors are acute and/or amenable to coping characterized by physical responses. However, when stress is unusually intense or prolonged, or when activity is not appropriate, wear and tear on the organism results and health may be compromised.

This wear and tear was described by Hans Selye, an endocrinologist who is considered the most prolific stress researcher of this century. He observed three changes in the body that occurred as a consequence of any stressor. These changes included an increase in the size of the adrenal glands, shrinkage of the thymus gland and lymphoid (immune) tissue, and increased incidence of ulcers in the digestive tract. In his 1956 book *The Stress of Life*, Selye theorized that organisms are born with a fixed capacity to resist stressors, but that repeated exposure to danger and activation of stress responses can deplete these reserves and cause tissue and organ damage. Although this theory has been criticized and psychological components have been added, research suggests that repeated or prolonged exposure to stress can harm our health.

The nature of stress makes investigation of some of these relationships very difficult. For example, many clinicians and researchers suspect that stress has a role in the development of heart disease, cancer, AIDS, and other illnesses. However, these diseases often develop over decades of life. Correlating or even identifying stressors or critical disease points over such a long period is difficult. Animal models provide an important complement to decades-long naturalistic study of humans under stress. Evidence of stress as a factor in the etiology of chronic diseases has been reported, although it is considerably more controversial than findings suggesting that stress plays a role in progression, recurrence of, or recovery from disease.

There are three main pathways through which excessive stress affects health. First, it causes changes in biological systems in the body, including the endocrine, cardiovascular, and immune systems. These direct effects include tissue damage caused by the repeated release of stress hormones that irritate the lining of arteries and contribute to atherosclerosis. In vulnerable individuals, acute stress can cause platelets to aggregate and form clots that can induce heart attack or stroke. Prolonged stress responding may compromise the immune system, allowing infections and some forms of cancers to develop. For example, Sheldon Cohen, a noted health psychologist, has found that stress affects whether people catch colds or experience viral infections.

Stress also affects behaviors that in turn have effects on physiological systems. Stress can increase cigarette consumption, alcohol and/or drug abuse, and diet. These behaviors may help decrease the tension or discomfort associated with stress, but they have

negative physiological consequences and may interact with the direct effects of stress and affect subsequent coping. Third, stress affects health behaviors; to the extent that it affects whether people follow medical advice or utilize screening, it can also affect the recovery and recurrence of disease. Complex interactions among these pathways appear to contribute to the development and/or progression of hypertension, cancer, depression, asthma, cardiovascular disease, diabetes, and AIDS.

Reducing the Effects of Stress

Given this impact of stress on the body, what aspects of the stress process can be modified to reduce its health consequences? Perhaps the most effective way to reduce stress is to eliminate its source, either by moving away from it, eliminating it, or reducing its effects on us. If people experience stress because of their commute to work, they can change their route, time of commute, or other aspects of the stressor to reduce stress. However, most stressors are not time-limited and are generally beyond our control to eliminate them. An important aspect of coping is accommodation or adapting to a stressful situation and "learning how to live with it." Most stress management interventions focus on modifying people's responses to stressors, innoculating them against stress, and/or learning new coping skills that help people accommodate. One such approach capitalizes on the importance of appraisal and coping in the stress process. In a very influential book (*Psychological Stress and the Coping Process*, 1966) Lazarus argued that psychological appraisal is critical in determining the extent to which stress is experienced. Consequently, stress may be reduced if the threatening event is appraised as less harmful or threatening. Many of the stress-reduction techniques that have been used are targeted at these appraisals. Other interventions are based on learning and conditioning principles, or on increasing people's self-efficacy and perceived control.

People are inclined to cope with stress because it is an unpleasant state. This coping can be detrimental to health if it involves increases in health-impairing behaviors such as smoking or if it reduces that likelihood of health-protective behaviors (such as exercise or safer sex). When coping increases harmful conditions, stress-related illnesses are more likely. Because this can cause absenteeism and lost productivity, organized stress management programs have become increasingly popular in workplace settings. By helping people cope more effectively and/or maintain the physical conditioning that reduces its affects on the body, these programs are intended to reduce stress and its health consequences.

Stress inoculation training involves three phases of treatment: education, rehearsal, and the application of coping skills. The education phase of stress involves identifying sources of distress that stem from negative self-talk. These cognitive distortions may include overgeneralization, personalization, magnifying failures, and minimizing successes. Therapeutic intervention involves showing people the problems with these thoughts and substituting positive statements. Once the stressor is identified and the particular problems accurately interpreted, individuals may prepare to cope with future stressful events by practicing coping skills in increasingly stressful situations.

Extreme stress or fears related to objects or situations associated with harm can be alleviated using a technique called systematic desensitization. This approach requires the client to substitute relaxation for anxiety in response to a particular object or situation. Deep-muscle relaxation is paired with anxiety-provoking images or statements. The therapist and client construct a hierarchy of items ranging from games evoking low levels of anxiety to those causing very uncomfortable levels of anxiety. The individual learns to relax while being led through increasingly difficult imagined situations. Eventually, the client can relax while imagining his or her most stressful situation and ultimately can learn to relax while confronting stressful situations in real life.

Systematic desensitization involves progressive muscle relaxation. This intervention is based on the observation that relaxation and stress responses are incompatible, that is, relaxation and stress cannot exist simultaneously, and deep relaxation can actually inhibit anxiety. This method is called progressive muscle relaxation because it proceeds through all of the major groups one at a time until a completely relaxed state is reached. Muscles in the feet are tensed for 6 to 8 seconds, then relaxed. Other muscle groups are tensed and then relaxed, working up to the face. The client learns to identify states of tension and relaxation, and with practice can benefit by unwinding after stressors and by being prepared for future stressful encounters. Progressive muscle relaxation is effective in lowering blood pressure, reducing the side effects of chemotherapy, and has been used to treat ulcers, insomnia, asthma, and headaches. Systematic desensitization is particularly useful for treating phobias, including anticipatory stress associated with medical procedures.

Biofeedback is also useful in reducing stress. Most biofeedback involves learning to control visceral responses such as heart rate or brain activity. It encourages a sense of efficacy and control as well as a direct means of stress reduction. For example, skin temperature, neuromuscular activity, blood pressure, heart rate, and brain-wave activity can be targeted and, with feedback, people learn to control or alter these bodily func-

tions and learn to control physiological responses and reduce stress-related activity. Biofeedback is often combined with other stress reduction techniques for the treatment of stress-linked illnesses such as hypertension or headache.

A very different approach to stress management focuses on increasing physical fitness and overall well-being so that episodes of stress have shorter or less intense impact. Although data are not conclusive, they suggest that diet and exercise may reduce the impact of stress and its effects on health. The plasticity of bodily systems, their ability to refract and respond again, and the habituation of response are all important in this regard. General good health may increase coping ability by augmenting the ability to unwind after stressful encounters or by minimizing physiological reactivity. Vigorous exercise may lead to subsequent deep relaxation and better sleep, both of which are beneficial. Exercise appears to be related to well-being, reduction of anxiety, and depression, may help disperse the arousal associated with stress, and can substitute for negative coping behaviors such as overeating, smoking, and using alcohol to unwind.

The stress response is adaptive in that it motivates and enables an individual to either fight or flee a noxious agent. However, repeated or prolonged activation of the stress response can be detrimental to health. Stress can affect health through several pathways. These include the prolonged presence of stress hormones and the wear and tear of disruptions of homeostasis, the influence of stress on lifestyle variables such as smoking and alcohol consumption, and the influence of stress on seeking health care and adhering to medical advice. Stress, among other modifiable and nonmodifiable factors, contributes to a wide variety of disorders and diseases. Stress reduction techniques enable people to reinterpret events, assert themselves, and relax in times of tension. Ultimately, stress reduction should contribute to better health and a better quality of life.

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Tonya Y. Schooler, Angela Dougall, and Andrew Baum

STROKE. *See* Alzheimer's Disease.

STRONG, EDWARD KELLOGG, JR. (1884–1963), American psychologist. Strong was born in Syracuse, New York, the son of Edward Kellogg Strong and Mary Elizabeth Graves. He entered the University of California at Berkeley, graduating with a bachelor's degree in biology in 1906 and a master's in psychology in 1909. In 1911, Strong received his doctoral degree in psychology at Columbia University where he worked as an assistant at Barnard College and as a fellow at Columbia University. His dissertation, "The Relative Merit of Advertisements: A Psychological and Statistical Study," was an early reflection of his interest in applied issues from an empirical perspective.

After graduation, Strong worked as a research fellow in advertising before accepting a position as professor of psychology and education at George Peabody College for Teachers in Nashville, Tennessee, in 1914. He enlisted in the armed forces in 1917 and was assigned to the Department of the Adjutant General of the United States Army. Strong was a member of the Committee on the Classification of Personnel where he became actively involved in issues of occupational and personnel classification. He aided in the development of a personnel selection system, played a central role in the preparation of the *Index of Occupations*, and was a leader in the education and training of personnel officers. After the war, Strong went to the Carnegie Institute of Technology as professor and head of the department of vo-