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Nutritional and Botanical Interventions to Assist with the Adaptation to Stress

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Abstract

Prolonged stress, whether a result of mental/emotional upset or due to physical factors such as malnutrition, surgery, chemical exposure, excessive exercise, sleep deprivation, or a host of other environmental causes, results in predictable systemic effects. The systemic effects of stress include increased levels of stress hormones such as cortisol, a decline in certain aspects of immune system function such as natural killer cell cytotoxicity or secretory-IgA levels, and a disruption of gastrointestinal microflora balance. These systemic changes might be a substantial contributor to many of the stress-associated declines in health. Based on human and animal research, it appears a variety of nutritional and botanical substances - such as adaptogenic herbs, specific vitamins including ascorbic acid, vitamins B1 and B6, the coenzyme form of vitamin B5 (pantethine) and B12 (methylcobalamin), the amino acid tyrosine, and other nutrients such as lipoic acid, phosphatidylserine, and plant sterol/sterolin combinations - may allow individuals to sustain an adaptive response and minimize some of the systemic effects of stress. (Altern Med Rev 1999;4(4):249-265.)

Introduction

Stress is a broad, ambiguous, and often poorly understood concept. In its most simplified sense, stress is what one feels when life's demands exceed one's ability to meet those demands. In a much more elaborate sense, stress goes far beyond what one actually feels, causing predictable changes in immune function, hormone levels, enzymes, and gastrointestinal function. In fact, prolonged stress, whether a result of mental/emotional upset or due to physical factors such as malnutrition, surgery, chemical exposure, excessive exercise, sleep deprivation, or a host of other environmental causes, results in predictable systemic effects.

All individuals have different capacities to perform and accommodate when faced with stress. But ultimately we all have a breaking point; add enough total stress and performance suffers. The work of Hans Selye provides the classic model for adaptation to stress (Table 1). He observed that given any source of external biological stress, an organism would respond with a predictable biological pattern in an attempt to restore its internal homeostasis. He termed this the General Adaptation Syndrome or Biological Stress Syndrome, and divided the response into four categories: 1) the "alarm reaction" characterized by an immediate activation of the nervous system and adrenal glands; 2) a "resistance phase" characterized by hypothalamic-pituitary-adrenal (HPA) axis activation; 3) a stage of adrenal hypertrophy, gastrointestinal ulceration, along with thymic and lymphoid atrophy; and 4) an exhaustion phase which may culminate with death. [1]

Selye observed that prolonged stress ultimately forces organisms to accommodate to maintain a relative balance in the face of the continued challenges. But, at some point, all organisms reach a point beyond which compromises are no longer possible and function suffers. If stress persists long enough or with enough intensity, one begins to experience a decline in performance (maladaptive response). Based on human and animal research, a variety of substances, such as adaptogenic herbs, specific vitamins including ascorbic acid, vitamins B1, B5, and B6, the amino acid tyrosine, nutrients such as lipoic acid and phosphatidylserine, and plant sterol/sterolin combinations may allow individuals to sustain an adaptive response to stress.

Physiology of Stress

Within seconds after an acutely stressful event or danger, norepinephrine is released from nerve endings in preparation for a rapid response. Almost instantly, the adrenal glands release epinephrine and norepinephrine into the bloodstream. The combination of the release of norepinephrine and epinephrine results in the familiar "fight or flight" response. Within minutes of a stressful event (and possibly lasting for several hours), a much more elaborate interaction between the nervous and endo-crine systems and other forms of internal communication occurs, resulting in a very complex adaptive response to deal with the stress. At this point adrenal glands release extra amounts of cortisol into the circulation.

Several other endocrine glands are also critical to the stress response. The hypothalamus, located in the brain, is often thought of as the "master" gland; it responds to stress by releasing a hormone called corticotropin-releasing factor (CRF). This hormone signals the pituitary gland to release adrenocorticotropic hormone (ACTH), which stimulates the adrenal glands to release cortisol. With the rise in stress hormones, a complex mechanism of feedback controls is set in motion, eventually signaling the hypothalamus to stop producing its messenger hormone (Figure 1).

A wide range of events, based on their ability to prompt the adrenal glands to release stress hormones, are considered physiologically stressful. These occurrences include calorie restriction, [2-6] surgery, [7] sleep deprivation, [8,9] and excessive exercise. [2,10-12] Even one's mental state can induce an increase of cortisol and catecholamine stress hormones. [13,14]

Stress exerts a disruptive influence on normal circadian release of the adrenal hormone cortisol. A study was conducted on military cadets subjected to a five-day training course of heavy physical exercise and food and sleep deprivation. Not surprisingly, due to the stressful nature of this training, cortisol levels went up and performance deteriorated. The researchers also found, "the circadian rhythm was extinguished." Even after 4-5 days of rest, circadian rhythms had not completely normalized. [2]

As this and other research demonstrates, the physiological and psychological consequences of acute and chronic stress can and do persist well beyond actual cessation of a stressful event. [2,15]

Health Consequences of Chronic Stress

General Effects

From headaches to heart disease and immune deficiencies to digestive problems, stress is a factor in many illnesses. A substantial contributor to stress-induced decline in health appears to be an increased production of stress hormones and subsequent decreased immune function. [16]

Researchers have found that people dramatically increase their use of the medical system during times of job insecurity. Visits to doctors increased 150 percent, episodes of illness increased 70 percent, and visits to hospital outpatient departments increased 160 percent. [17]

Other evidence clearly demonstrates workers reporting the highest level of perceived stress due to job dissatisfaction (with working conditions or supervisory style being the most common stress reported), family problems, and personal conflict are the most likely to experience somatic symptoms. [18]

Cardiovascular Health

Stress and emotions associated with stress are important risk factors for cardiovascular problems. The Mayo Clinic reported psychological stress is the strongest risk factor predictive of future cardiac events, including myocardial infarction and cardiac death, among individuals with existing coronary artery disease. In this study, the economic cost of high and low stress was compared in terms of the mean rehospitalization costs: \$9,504 versus \$2,146. [19]

When researchers interviewed survivors of heart attacks, they found the intensity and timing of a stressful emotion like anger dramatically increased their risk. [20] The Normative Aging Study also provided compelling evidence that emotions associated with a higher stress level are significant risk factors for coronary heart disease (CHD) and myocardial infarction (MI):

Anger: Compared with men reporting the lowest levels of anger, relative risk among men reporting the highest levels of anger is 3.15 ((95% confidence interval) [CI]: 0.94-10.5) for total CHD (nonfatal MI plus fatal CHD). A dose-response relation was found between level of anger and overall CHD risk. [21]

Anxiety: Compared with men reporting no symptoms of anxiety, men reporting two or more anxiety symptoms had elevated risks of fatal CHD (age-adjusted odds ratio [OR] = 3.20, 95% CI: 1.27-8.09), and sudden death (age-adjusted OR = 5.73, 95% CI: 1.26-26.1). [22]

Worry: Compared with men reporting the lowest levels of worry, men reporting the highest levels had multivariate adjusted relative risks of 2.41 (95% CI: 1.40-4.13) for nonfatal MI and 1.48 (95% CI: 0.99-2.20) for total CHD (nonfatal MI and fatal CHD). A dose-response relation was found between level of worry and both nonfatal MI and total CHD. [23]

Immune Performance

Research clearly indicates a bout of acute stress in virtually any form will cause, at the very least, a temporary decrease in functioning of the immune system, while chronic stress will result in continued decline in immune system function (Table 2).

Natural Killer Cell Cytotoxicity: Overwhelming evidence has demonstrated virtually any type of stress has a detrimental effect on the ability to maintain optimal levels of natural killer (NK) cell cytotoxic activity. [24-27] In fact, a severe life stress may be associated with up to a 50-percent reduction of NK cell activity. [28] Since NK cell activity plays a vital role in immune system surveillance against viral-infected and cancer cells, one can ill afford any sustained decrease in this aspect of immune performance.

A study of breast cancer patients found test scores assessing an individual's overall stress level due to the diagnosis of breast cancer were strongly correlated to NK cell activity. In these women, a high degree of stress predicted a lowered ability of NK cells to destroy cancer cells. A high degree of stress also significantly predicted a poorer response to interventions aimed at improving NK cell activity. [29]

Chronic stress preceding an acutely stressful event significantly impacts NK cell activity. A study examined two groups, one consisting of individuals experiencing chronic stress, and a second group who were relatively stress-free. A single stressful event experienced by both groups resulted in the people who suffered chronic stress in a much greater sense of subjective distress, higher peak levels of epinephrine, a more pronounced immediate reduction in NK cell activity, and a protracted decline of NK cell activity. Individuals without chronic stress readily rebounded from the acute stress with no long-term impact on NK cell activity. This study clearly demonstrates chronic stress measurably reduces the ability of the immune system to respond to an acute psychological challenge. [30]

Secretory IgA: The ability to produce secretory IgA (sIgA) also appears to be influenced by stress. [31-33]

sIgA, as the first line of defense, is probably the single most important aspect of humoral immunity in the mucus secretions of the digestive system, mouth, lungs, urinary tract, and other body cavities. Any decline in levels of sIgA decreases one's resistance to microbial pathogens. [34]

Higher levels of the catecholamine stress hormone epinephrine are significantly associated with lower sIgA concentrations. [35] Daily problems, lack of a sense of humor, [36] and negative emotions can decrease sIgA levels. [14] To demonstrate the profound effect of emotions associated with stress on sIgA levels, a single five-minute experience of anger can produce a significant decrease in sIgA levels that can still be measured up to five hours after the emotional experience. [14]

Intestinal Microflora

Stress has a significant influence on the balance of intestinal microflora. [37] In fact, Moore et al found, "the composition of the flora was not significantly affected by drastic changes in diet, but statistically significant shifts in the proportions of some species were noted in individuals under conditions of anger or fear stress." [38]

To examine the impact of high stress on intestinal microflora, Lizko et al investigated the preparation for and participation in space flight. During the preparation phase they found a distinct decrease in the numbers of Bifidobacterium and Lactobacilli, and a corresponding increase in the numbers of E. coli and of Enterobacteria. These imbalances worsened until launch, illuminating the effect of nervous-emotional stress on altering the balance of beneficial and pathogenic organisms. After the flight the number of potentially pathogenic Enterobacteria and Clostridia were also substantially increased, while the number of Lactobacilli were decreased, suggesting the physiological strain of space flight also disrupted microflora balance. [39]