

The Influence of Stress upon Mood, Our Sense of Well-Being, Behavior, and Health

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Abstract: *Stressors have a major influence upon mood, our sense of well-being, behavior, and health. Acute stress responses in young, healthy individuals may be adaptive and typically do not impose a health burden. However, if the threat is unremitting, particularly in older or unhealthy individuals, the long-term effects of stressors can damage health. The relationship between psychosocial stressors and disease is affected by the nature, number, and persistence of the stressors as well as by the individual's biological vulnerability (i.e., genetics, constitutional factors), psychosocial resources, and learned patterns of coping. Psychosocial interventions have proven useful for treating stress-related disorders and may influence the course of chronic diseases.*

Keywords: psychosocial stressors, stress responses, homeostasis, psychosocial interventions, host vulnerability-stressor interactions

1. Introduction

The actual or perceived threat to an organism is referred to as the “stressor” and the response to the stressor is called the “stress response.” Based on the appraisal of perceived threat, humans and other animals invoke coping responses (Lazarus & Folkman 1984). Our central nervous system (CNS) tends to produce integrated coping responses rather than single, isolated response changes (Hilton 1975). Thus, when immediate fight-or-flight appears feasible, mammals tend to show increased autonomic and hormonal activities that maximize the possibilities for muscular exertion (Cannon 1929, Hess 1957). In contrast, during aversive situations in which an active coping response is not available, mammals may engage in a vigilance response that involves sympathetic nervous system (SNS) arousal accompanied by an active inhibition of movement and shunting of blood away from the periphery (Adams et al. 1968). The extent to which various situations elicit different patterns of biologic response is called “situational stereotypy” (Lacey 1967).

Although various situations tend to elicit different patterns of stress responses, there are also individual differences in stress responses to the same situation.

2. Psychological Aspects of Stress

Stressors during Childhood and Adolescence and Their Psychological Sequelae. The most widely studied stressors in children and adolescents are exposure to violence, abuse (sexual, physical, emotional, or neglect), and divorce/marital conflict (see Cicchetti 2005). McMahon et al. (2003) also provide an excellent review of the psychological consequences of such stressors. Children of divorced parents have more reported antisocial behavior, anxiety, and depression than their peers (Short 2002). Adult offspring of divorced parents report more current life stress, family conflict, and lack of friend support compared with those whose parents did not divorce (Short 2002). Exposure to nonresponsive environments has also been described as a stressor leading to learned helplessness (Peterson & Seligman 1984).

Studies have also addressed the psychological consequences of exposure to war and terrorism during childhood (Shaw 2003). A majority of children exposed to war experience significant psychological morbidity, including both post-traumatic stress disorder (PTSD)

Stressors during Adulthood and Their Psychological Sequelae

Life Stress, Anxiety, and Depression

It is well known that first depressive episodes often develop following the occurrence of a major negative life event (Paykel 2001). Furthermore, there is evidence that stressful life events are causal for the onset of depression (see Hammen 2005, Kendler et al. 1999). A study of 13,006 patients in Denmark, with first psychiatric admissions diagnosed with depression, found more recent divorces, unemployment, and suicides by relatives compared with age- and gender-matched controls (Kessing et al. 2003). The diagnosis of a major medical illness often has been considered a severe life stressor and often is accompanied by high rates of depression (Cassem 1995). For example, a meta-analysis found that 24% of cancer patients are diagnosed with major depression (McDaniel et al. 1995).

Disorders related to trauma

Lifetime exposure to traumatic events in the general population is high, with estimates ranging from 40% to 70% (Norris 1992). Of note, an estimated 13% of adult women in the United States have been exposed to sexual assault (Kilpatrick et al. 1992). The Diagnostic and Statistical Manual (DSM-IV-TR; American Psychiatric Association 2000) includes two primary diagnoses related to trauma: Acute Stress Disorder (ASD) and PTSD. Both these disorders have as prominent features a traumatic event involving actual or threatened death or serious injury and symptom clusters including re-experiencing of the traumatic event Variations in Stress Responses

Biological Responses to Stressors

Acute Stress Responses

Following the perception of an acute stressful event, there is a cascade of changes in the nervous, cardiovascular, endocrine, and immune systems. These changes constitute

the stress response and are generally adaptive, at least in the short term (Selye 1956). Two features in particular make the stress response adaptive. First, stress hormones are released to make energy stores available for the body's immediate use. Second,

Chronic Stress Responses

The acute stress response can become maladaptive if it is repeatedly or continuously activated (Selye 1956). For example, chronic SNS stimulation of the cardiovascular system due to stress leads to sustained increases in blood pressure and vascular hypertrophy (Henry et al. 1975). That is, the muscles that constrict the vasculature thicken, producing elevated resting blood pressure and response stereotypy, or a tendency to respond to all types of stressors with a vascular response. new pattern of energy distribution emerges.

3. Psychosocial Stressors and Health

Cardiovascular Disease

Both epidemiological and controlled studies have demonstrated relationships between psychosocial stressors and disease. The underlying mediators, however, are unclear in most cases, although possible mechanisms have been explored in some experimental studies.

Upper Respiratory Diseases

The hypothesis that stress predicts susceptibility to the common cold received support from observational studies (Graham et al. 1986, Meyer & Haggerty 1962). One problem with such studies is that they do not control for exposure. Stressed people, for instance, might seek more outside contact and thus be exposed to more viruses. Therefore, in a more controlled study, people were exposed to a rhinovirus and then quarantined to control for exposure to other viruses (Cohen et al. 1991). Those individuals with the most stressful life events and highest levels of perceived stress and negative affect had the greatest probability of developing cold symptoms. In a subsequent study of volunteers inoculated with a cold virus, it was found that people enduring chronic, stressful life events

Human Immunodeficiency Virus

The impact of life stressors has also been studied within the context of human immunodeficiency virus (HIV) spectrum disease. Leserman et al. (2000) followed men with HIV for up to 7.5 years and found that faster progression to AIDS was associated with higher cumulative stressful life events, use of denial as a coping mechanism, lower satisfaction with social support, and elevated serum cortisol.

Inflammation, the Immune System, and Physical Health

Despite the stress-mediated immunosuppressive effects reviewed above, stress has also been associated with exacerbations of autoimmune disease (Harbuz et al. 2003) and other conditions in which excessive inflammation is a central feature, such as CHD (Appels et al. 2000). Evidence suggests that a chronically activated, dysregulated acute stress response is responsible for these associations. Recall that the acute stress response includes the activation and migration of cells of the innate immune system. This effect is mediated by proinflammatory cytokines. During periods

of chronic stress, in the otherwise healthy individual, cortisol eventually suppresses proinflammatory cytokine production. Inflammation, Cytokine Production, and Mental Health

In addition to its effects on physical health, prolonged proinflammatory cytokine production may also adversely affect mental health in vulnerable individuals. During times of illness (e.g., the flu), proinflammatory cytokines feed back to the CNS and produce symptoms of fatigue, malaise, diminished appetite, and listlessness, which are symptoms usually associated with depression.

Host Vulnerability-Stressor Interactions and Disease

The changes in biological set points that occur across the life span as a function of chronic stressors are referred to as allostasis, and the biological cost of these adjustments is known as allostatic load (McEwen 1998). McEwen has also suggested that cumulative increases in allostatic load are related to chronic illness. These are intriguing hypotheses that emphasize the role that stressors may play in disease. The challenge, however, is to show the exact interactions that occur among stressors, pathogens, host vulnerability (both constitutional and genetic), and such poor health behaviors as smoking, alcohol abuse, and excessive caloric consumption.

Treatment for Stress-Related Disorders

For PTSD, useful treatments include cognitive-behavioral therapy (CBT), along with exposure and the more controversial Eye Movement Desensitization and Reprocessing (Foa & Meadows 1997, Ironson et al. 2002, Shapiro 1995). Psychopharmacological approaches have also been suggested (Berlant 2001). In addition, writing about trauma has been helpful both for affective recovery and for potential health benefit (Pennebaker 1997). For outpatients with major depression, Beck's CBT (Beck 1976) and interpersonal therapy (Klerman et al. 1984) are as effective as psychopharmacotherapy

Behavioral Interventions in Chronic Disease

Patients dealing with chronic, life-threatening diseases must often confront daily stressors that can threaten to undermine even the most resilient coping strategies and overwhelm the most abundant interpersonal resources. Psychosocial interventions, such as cognitive-behavioral stress management (CBSM), have a positive effect on the quality of life of patients with chronic disease (Schneiderman et al. 2001). Such interventions decrease perceived stress and negative mood (e.g., depression), improve perceived social support, facilitate problem-focused coping, and change cognitive appraisals, as well as decrease SNS arousal and the release of cortisol from the adrenal cortex. Psychosocial interventions also appear to help chronic pain patients reduce their distress and perceived pain as well as increase their physical activity and ability to return to work (Morley et al. 1999).

Morbidity, Mortality, and Markers of Disease Progression

Psychosocial intervention trials conducted upon patients following acute myocardial infarction (MI) have reported both positive and null results. Two meta-analyses have

reported a reduction in both mortality and morbidity of approximately 20% to 40% (Dusseldorp et al. 1999, Linden et al. 1996). Most of these studies were carried out in men.

4. Conclusion

Stress is a central concept for understanding both life and evolution. All creatures face threats to homeostasis, which must be met with adaptive responses. Our future as individuals and as a species depends on our ability to adapt to potent stressors. At a societal level, we face a lack of institutional resources (e.g., inadequate health insurance), pestilence (e.g., HIV/AIDS), war, and international terrorism that has reached our shores. At an individual level, we live with the insecurities of our daily existence including job stress, marital stress, and unsafe schools and neighborhoods. These are not an entirely new condition as, in the last century alone, the world suffered from instances of mass starvation, genocide, revolutions, civil wars, major infectious disease epidemics, two world wars, and a pernicious cold war that threatened the world order. Although we have chosen not to focus on these global threats in this paper, they do provide the backdrop for our consideration of the relationship between stress and health.

References

- [1] Adams DB, Bacelli G, Mancina G, Zanchetti A. Cardiovascular changes during naturally elicited fighting behavior in the cat. *Am. J. Physiol.* 1968;216:1226–1235. [PubMed]
- [2] Adams MR, Kaplan JR, Koritnik DR. Psychosocial influences on ovarian, endocrine and ovulatory function in *Macaca fascicularis*. *Physiol. Behav.* 1985;35:935–940. [PubMed]
- [3] Affleck G, Urrows S, Tennen H, Higgins P, Pav D, Aloisi R. A dual pathway model of daily stressor effects on rheumatoid arthritis. *Ann. Behav. Med.* 1997;19:161–170. [PubMed]
- [4] American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders IV-TR*. 4th ed. Washington, DC: Am. Psychiatr. Assoc.; 2000.
- [5] Angst J, Vollrath M. The natural history of anxiety disorders. *Acta Psychiatr. Scand.* 1991;84:446–452. [PubMed]
- [6] Antoni MH, Baggett L, Ironson G, LaPerriere A, Klimas N, et al. Cognitive behavioral stress management intervention buffers distress responses and elevates immunologic markers following notification of HIV-1 seropositivity. *J. Consult. Clin. Psychol.* 1991;59:906–915. [PubMed]
- [7] Antoni MH, Cruess DG, Cruess S, Lutgendorf S, Kumar M, et al. Cognitive behavioral stress management intervention effects on anxiety, 24-hour urinary catecholamine output, and T-cytotoxic/suppressor cells over time among symptomatic HIV-infected gay men. *J. Consult. Clin. Psychol.* 2000a;68:31–45. [PubMed]
- [8] Antoni MH, Cruess S, Cruess DG, Kumar M, Lutgendorf S, et al. Cognitive-behavioral stress management reduces distress and 24-hour urinary free cortisol output among symptomatic HIV-infected gay men. *Ann. Behav. Med.* 2000b;22:29–37. [PubMed]
- [9] Appels A, Bar FW, Bar J, Bruggeman C, de Bates M. Inflammation, depressive symptomatology, and coronary artery disease. *Psychosom. Med.* 2000;62:601–605. [PubMed]
- [10] Ballenger JC, Davidson JRT, Lecrubier Y, Nutt DJ, Borkovec TD, et al. Consensus statement on generalized anxiety disorder from the international consensus group on depression and anxiety. *J. Clin. Psychiatry.* 2001;62:53–58. [PubMed]
- [11] Başoğlu M, Parker M, Parker Ö, Özmen E, Marks I, et al. Psychological effects of torture: a comparison of tortured with non-tortured political activists in Turkey. *Am. J. Psychiatry.* 1994;151:76–81. [PubMed]
- [12] Baum A. Stress, intrusive imagery, and chronic distress. *Health Psychol.* 1990;9:653–675. [PubMed]
- [13] Beck AT. *Cognitive Therapy and the Emotional Disorders*. New York: Int. Univ. Press; 1976.
- [14] Berlant JL. Topiramate in posttraumatic stress disorder: preliminary clinical observations. *J. Clin. Psychiatry.* 2001;62:60–63. [PubMed]
- [15] Bernard C. *An Introduction to the Study of Experimental Medicine*. Transl. HC Greene. New York: Collier; 1865/1961.
- [16] Bleich A, Gelkopf M, Solomon Z. Exposure to terrorism, stress-related mental health symptoms, and coping behaviors among a nationally representative sample in Israel. *JAMA.* 2003;290:612–620. [PubMed]
- [17] Borkovec TD, Ruscio AM. Psychotherapy for generalized anxiety disorder. *J. Clin. Psychiatry.* 2001;61:37–42. [PubMed]
- [18] Breslau N, Davis GC, Andreski P, Peterson E. Sex differences in depression: a role for preexisting anxiety. *Psychiatr. Res.* 1995;58:1–12. [PubMed]
- [19] Brindley D, Rollan Y. Possible connections between stress, diabetes, obesity, hypertension, and altered lipoprotein metabolism that may result in atherosclerosis. *Clin. Sci.* 1989;77:453–461. [PubMed]
- [20] Brown GW, Bifulco A, Harris T, Bridge L. Life stress, chronic subclinical symptoms and vulnerability to clinical depression. *J. Affect. Disord.* 1986;11:1–19. [PubMed]
- [21] Brownley KA, Hurwitz BE, Schneiderman N. Cardiovascular psychophysiology. In: Cacioppo JT, Tassinari LG, Berntson GG, editors. *Handbook of Psychophysiology*. 2nd ed. New York: Cambridge Univ.; 2000. pp. 224–264.
- [22] Cannon WB. *Bodily Changes in Pain, Hunger, Fear and Rage*. 2nd ed. New York: Appleton; 1929.
- [23] Carney RM, Rich MW, Tevelde A, Saini J, Clark K, Jaffe AS. Major depressive disorder in coronary artery disease. *Am. J. Cardiol.* 1987;60:1273–1275. [PubMed]
- [24] Cassem EH. Depressive disorders in the medically ill: an overview. *Psychosomatics.* 1995;36:S2–S10. [PubMed]
- [25] Cicchetti D. Child maltreatment. *Annu. Rev. Clin. Psychol.* 2005;1:409–438. [PubMed]
- [26] Classen C, Sephton SE, Diamond S, Spiegel D. Studies of life-extending psychosocial interventions. In: Holland J, editor. *Textbook of Psycho-Oncology*. New York: Oxford Univ. Press; 1998. pp. 730–742.
- [27] *Clinical Practice Guidelines*. No. 5. Depression in Primary Care. Vol. 2: Treatment of Major

Depression. Rockville, MD: US Dept. Health Hum. Serv., Agency Health Care Policy Res.; 1993. AHCPR Publ. 93-0551.

- [28] Cohen S, Frank E, Doyle WJ, Skoner DP, Rabin BS, Gwaltney JM., Jr Types of stressors that increase susceptibility to the common cold in healthy adults. *Health Psychol.* 1998;17:214–223.[PubMed]
- [29] Cohen S, Tyrrell DA, Smith AP. Psychological stress and susceptibility to the common cold. *N. Engl. J. Med.* 1991;325:606–612. [PubMed]
- [30] Colby JP, Linsky AS, Straus MA. Social stress and state-to-state differences in smoking-related mortality in the United States. *Soc. Sci. Med.* 1994;38:373–381. [PubMed]
- [31] Conway TL, Vickers RR, Ward HW, Rahe RH. Occupational stress and variation in cigarette, coffee and alcohol consumption. *J. Health Soc. Behav.* 1981;22:156–165. [PubMed]
- [32] Danner M, Kasl SV, Abramson JL, Vaccarion V. Association between depression and elevated C-reactive protein. *Psychosom. Med.* 2003;65:347–356. [PubMed]
- [33] Dantzer R. Cytokine-induced sickness behavior: Where do we stand? *Brain Behav. Immun.* 2001;15:7–24. [PubMed]
- [34] David D, Mellman TA, Mendoza LM, Kulick-Bell R, Ironson G, Schneiderman N. Psychiatric morbidity following Hurricane Andrew. *Int. Soc. Trauma. Stress Stud.* 1996;9:607–612. [PubMed]
- [35] Dhabar FS, McEwen BS. Acute stress enhances while chronic stress suppresses cell-mediated immunity in vivo: a potential role for leukocyte trafficking. *Brain Behav. Immun.* 1997;11:286–306.[PubMed]