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JOB STRESS AND BURN OUT

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ABSTRACT

Job stress is the psychophysiological arousal resulting from workplace demands. Optimally, it results in enhanced job performance. When mismanaged, job stress leads to job strain, which includes psychological, medical, and behavioral costs. The growing awareness of the impact of job stress is best illustrated by the sheer increase in the volume of job stress research in recent years. Job stress is a condition familiar to many. In some form or another, it influences all working lives, leaving those who experience job stress emotionally drained and frequently more vulnerable to other illnesses and disease. It is not just individuals who experience the impact of job stress. Organizations, families, communities, and society all bear the costs of job stress to such an extent that job stress is frequently viewed as an inevitable feature of 21st-century living.

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INTRODUCTION

The difficulty when talking about job stress is that it means different things to different people, with the result that the term stress has taken on a variety of been influenced and guided by definitions that focus on stress as a stimulus, a response, or on the interaction between the two. These three approaches to defining stress frequently reflect the discipline or orientation of researchers. Stress as a stimulus is defined in terms of those events or properties of events that place demands on an individual over and above what is normal. Response definitions of stress are concerned with identifying particular responses or patterns of responses that indicate that the individual is confronted with a demanding event. Stimulus and response definitions of stress have provided researchers with an opportunity to identify both a range of events that have demanding properties and responses that indicate that the individual is under stress. However, such definitions have been criticized because each reflects just one aspect of the stress process; defining stress in this way fails to provide any understanding of the nature of the stress process. They have also been criticized because they fail to take into account individual differences and therefore cannot account for the fact

that what may be stressful for one individual may not be stressful for another. Interactional definitions of stress tried to overcome these difficulties by describing stress in terms of the interaction between the stimulus and the response. This definition is one in which a relationship, usually correlational, is hypothesized between the stimulus and the response. Cooper et al. argue that defining stress like this, where the focus is simply on the interaction between the stimulus and the response, fails to provide any understanding of the complexity of the interaction or of the nature of the processes that may be involved. As a consequence of the difficulties associated with stimulus, response, and interactional definitions of stress, more contemporary definitions adopt a definitional approach described by Lazarus in which stress is viewed as relational in nature involving some sort of a transaction between the individual and the environment. Transactional definitions of stress imply that stress is neither solely in the individual nor in the environment but in the transaction between the two. It is the transaction that links the individual to the environment. Furthermore, it is by thinking of stress in transactional terms that draws attention to what the nature of the transaction may be. In this way, defining stress in transactional terms achieves what earlier definitions failed to do: It focuses attention on those processes that link the individual and the environment and that are at the heart of the transaction. Lazarus describes these processes as primary and secondary appraisal. Primary

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appraisal is when the individual evaluates the importance of the event in terms of harm, threat, or challenge to well-being. In short, it is the realization by the individual that something important is at stake. Secondary appraisal is when the individual considers what can be done. It involves an evaluation of those resources that are available to cope with the stressful event. Identifying the processes that are at the heart of a stressful transaction in terms of how an event is appraised and how it can be coped with means that contemporary definitions of stress are more concerned with capturing its transactional qualities than focusing, as was once the tradition, on individual components to that transaction. As a result, stress is best defined in terms of those encounters in which the demands from the encounters are appraised as taxing the individual's resources, thus threatening well-being.

Models of job stress have, quite naturally, closely followed how stress has been defined. Early models of job stress followed an interactional framework. That is, they were interested in the interaction between the cause of job stress (stimulus) and its impact (response). As outlined, the interactional framework suggests a relationship between the perceived presence of demanding work conditions (stressors) and different stress responses (strain). The relationship, usually correlational, postulated that the more demanding the stressor, the greater the probability it would result in strain. As job stress researchers began to explore different relationships between stressors and strains, they soon realized that the stressor-strain relationship may be influenced by other variables, such as age, gender, organizational level, and individual differences. Therefore, a second stage in the development of job stress models began. This stage is described as the moderator stage and was concerned with identifying organizational and individual variables that moderate (influence) the stressor-strain relationship. The interactional and moderator models, although important in providing information on the causes and consequences of job stress and on variables that influenced the relationship between the two, were limited in their ability to explain the stress process. So began in the development of job stress models the transactional stage.

Transactional models, like transactional definitions of job stress, were concerned with the sequence of events and the processes that linked the stressor to the strain. McGrath had for some time urged researchers to approach the investigation of job stress by developing models of job stress that mirrored the sequencing of events from stressor to strain. To capture this sequence, many models of job stress adopted the concept of fit. The idea of fit was coupled with the idea of equilibrium or balance. In general, where there was a misfit or imbalance between the person and the environment, such as in the case of a person failing to cope with the demands of a job, a state of disequilibrium would exist and this state would be associated with strain. Implicit in the concept of misfit is the notion of an individual's ability to manage or deal with a demanding job event. As job stress researchers began to explore the nature of this misfit, models of job stress began to outline more explicitly what misfit may involve. Job stress models began to build into their frameworks different transactional qualities, such as how stressors are appraised, how individuals cope, and organizational and individual resources available to individuals to manage the demanding encounter. What

distinguishes the transactional model from the earlier interactional and moderator models is its focus on process. It requires researchers to identify the processes that link the individual to the environment and to consider the sequencing of those processes and their role over time. However, although job stress researchers have long accepted at the theoretical level the importance of identifying process elements in job stress models, empirical work is predominately still being carried out using a interactional-moderator perspective. Nevertheless, job stress research has contributed much to our understanding of its causes and consequences, not at times without controversy and intense debate.

BURNOUT

Cooper et al. describe burnout as "a special form of strain." These authors point to the concept of burnout as reflecting a state of psychological strain initially associated with those working in the human service professions. Since the early studies on burnout, researchers have extended their investigations of the phenomenon to working life in general, considering in detail the consequences of burnout for individuals and their organizations. In the early 1980s, Maslach provided a description of burnout involving three major elements: emotional exhaustion described in terms of not having the emotional energy to sufficiently manage the encounter; depersonalization, in which individuals simply become seen as objects and are treated in a detached way; and a lack of personal accomplishment, in which the tendency is to devalue performance in negative ways. Since this three-dimensional view of burnout was first proposed by Maslach, much discussion has centered on whether emotional exhaustion is the essential feature of burnout, with the roles played by the other two dimensions being disputed. Cordes et al. describe burnout as a developmental process. These authors go on to describe burnout as a gradual eroding process and note that by emphasizing the process of burnout, researchers and organizations are provided with a mechanism for understanding what to look for and the types of interventions that may be necessary.

Their work supports a process in which the onset of burnout is marked by emotional exhaustion. Depersonalization follows, as Ashforth and Lee suggest, because it is a "means (albeit futile) of staunching the flow of emotional energy, of coping with growing exhaustion." The issue of whether, as depersonalization occurs, the individual begins to sense a loss of accomplishment and hence a degrading of achievements is less clear. Cordes et al. note that one possible reason for this is that a lack of personal accomplishment may also be explained in terms of a range of constraining organizational factors and hence depersonalization may develop somewhat independently of the other two dimensions. Correlates of burnout are many and varied, and researchers have explored these at a number of levels, including the individual level (e.g., gender, age, commitment, and individual differences), the job level (e.g., work role demands, client relationships, and autonomy), and the organizational level (e.g., organizational culture, management style, and communications). In general, it is clear that a range of individual, job, and organizational level factors influence the experience of burnout. However, it is also clear that more work is still needed to understand where in the process these different factors have their most significant

effect, how far their effect can be generalized, and what this means in terms of the development of intervention strategies.

HEALTH EFFECTS

Life stressors inevitably produce some emotional strain and physical tension. Stress researchers have posed the question of whether life stressors are also associated with more significant illnesses. In fact, life stressors are linked to a number of psychological and physical illnesses. Psychological and physical illnesses are often linked reciprocally, with each category of illness exacerbating the other. Moreover, psychological and physical illnesses often function as life stressors themselves, initiating a new cycle in the stress process.

Psychological Illness

Life stressors are associated with psychological stress reactions that involve depression and anxiety. For example, life stressors are linked both to the onset of depressive disorders and to relapse among individuals recovering from depressive disorders. Interpersonal problems and losses are especially likely to be associated with depressive reactions. Life stressors can also precipitate both onset and relapse of anxiety disorders such as generalized anxiety disorder, panic disorder, agoraphobia, and obsessive-compulsive disorder, and they can play a role in the development and progression of alcohol and drug abuse. Moreover, life stressors can trigger schizophrenic episodes among individuals who are vulnerable to this disorder. Trauma exposure produces a recognized pattern of PTSD symptoms, including re-experiencing the trauma psychologically through flashbacks and nightmares, emotional numbing, and experiencing heightened arousal and vigilance. Exclusive of traumatic events, chronic stressors are more strongly linked to psychological distress than are acute events. Although chronic stressors generally are less severe than acute life events, their effects often last longer and are more pervasive.

Moreover, an event is more likely to have an adverse psychological outcome when it threatens or disrupts a domain in which a person has central commitments. Psychological reactions themselves can exacerbate the stress process in two important ways. First, psychologically distressed individuals are more likely to perceive benign situations as threatening, and these perceptions of threat can trigger additional stress reactions. Second, persons who are psychologically distressed often create social conditions in their lives, such as conflictual family or work relationships, that are likely to produce new life stressors.

Physical Illness

Stressor exposure also initiates a characteristic biological response that is associated with the onset or exacerbation of a wide spectrum of physical illnesses. The biological stress response involves interconnections among the nervous, endocrine, and immune systems. The two most heavily studied stress-related biological mechanisms have been sympathetic arousal and activation of the hypothalamic-pituitary-adrenocortical (HPA) axis. Both mechanisms involve initial central nervous system input from the hypothalamus at the

base of the brain, and both mechanisms operate through the adrenal glands located above the kidneys. Short-term activation of these biological mechanisms is adaptive, mobilizing energy and enhancing alertness to respond to adaptive demands. However, chronic stimulation of these mechanisms can lead to (a) hyperarousal and subsequent wear and tear on body systems and (b) suppression of key components of the immune system. Sympathetic arousal is mediated by the sympathetic nervous system, which during stressful experiences releases norepinephrine at multiple sites throughout the body and stimulates the adrenal medulla to release epinephrine (adrenaline). These catecholamines mobilize the body to deal with immediate adaptive demands. However, chronic sympathetic arousal is associated with tension-related complaints such as headache. In fact, stress is the triggering factor reported most often by migraine and tension-type headache patients. Sustained or frequent intense sympathetic arousal is also linked to wear and tear on arteries and coronary vessels and to shear stress associated with sharply increasing catecholamines.

Chronic sympathetic arousal also increases blood clotting through coronary vasoconstriction, increased circulating lipids, and increased platelet aggregation. Hemodynamic and biochemical changes, in turn, may lead to health-related problems such as atherosclerosis hypertension, and coronary artery disease. For example, occupational stressors have been linked to elevated blood pressure in studies of activity at work. Moreover, stressful work conditions, particularly the combination of high work demands and low job control, have been associated with coronary heart disease in population studies of workers in Europe and the United States. Activation of the HPA axis is mediated by the pituitary gland, which during stressful experiences stimulates the adrenal cortex to release glucocorticoids (primarily cortisol in humans). Chronic expression of glucocorticoids and excessively high levels of glucocorticoids have been associated with suppression of key components of the immune system in human *in vitro* studies (i.e., examining tissue or blood samples outside the body) and in experimental studies using animal models. For example, glucocorticoids are linked to reduced T cell proliferation and lower natural killer cell cytotoxicity. In addition, glucocorticoids can impede the maturation of developing lymphocytes and can destroy mature lymphocytes.

These changes inhibit the ability of the cellular arm of the immune system to attack target pathogens directly. Glucocorticoids are also linked to decreased production of antibodies and certain components of complement. These changes inhibit the ability of the humoral arm of the immune system to target pathogens for destruction. It is unclear, however, whether the levels of immune system downregulation observed in the context of stressful experiences are sufficient to increase vulnerability to disease.

In studies examining clinical illness, there has been evidence of a link between life stressors and the onset and progression of infectious diseases such as the common cold and influenza. Life stressors are also related to latent viral activity in the herpes viruses, resulting in complaints such as mononucleosis and cold sores. In addition, life stressors may be associated with the onset and exacerbation of some autoimmune diseases, such as rheumatoid arthritis, and with poor diabetic control and progression of diabetes. Experimental research with

animals has demonstrated that stress can increase the development of artificially induced tumors; however, there is no clear evidence that stress can accelerate the growth of common human tumors. Historically, stress research has emphasized the link between sympathetic arousal and cardiovascular response as well as that between the HPA axis and immune system response. However, there is increasing evidence that sympathetic arousal also affects the immune system and that the HPA axis also affects the cardiovascular system. In addition to these direct biological pathways between stressors and illness, life stressors can relate to physical illness indirectly through health risk behaviors. Life stressors are associated with common health risk behaviors such as increased cigarette smoking, alcohol abuse, poor dietary and exercise habits, disturbed sleep, and reduced adherence to medical regimens. In turn, each of these health risk behaviors plays a role in increasing the risk of physical illness

PROTECTIVE AND DAMAGING EFFECTS OF STRESS ON BRAIN

Stress mediators have both positive and negative effects on the brain, just as they do on other systems of the body. The stress mediators enhance formation of the so-called “flashbulb memories” of events associated with strong emotions, including fear but also positive emotions. These involve the amygdala, and the pathway for encoding these memories involves the interaction between neurotransmitters in the amygdala and in related brain areas such as the hippocampus along with circulating stress hormones of the adrenal cortex and adrenal medulla. Indeed, encoding of these memories is strengthened by glucocorticoids acting in the amygdala and hippocampus, among other brain regions, and epinephrine acting in the sensory vagus outside of the blood–brain barrier, with information transmitted into the brain via the nucleus of the solitary tract. These findings may have relevance to posttraumatic stress disorder and also to symptoms of depression, in which an overactive amygdala appears to be involved. At the same time as the brain encodes information and controls the behavioral responses, it is also changed structurally and chemically by those experiences.

Studies of learning and memory have revealed levels of plasticity involving structural changes in brain cells and changes in gene expression. On the one hand, this can be seen by the remodeling of neuron structure brought about by training. On the other hand, transcription factors involved in regulating expression of groups of genes in brain cells appear to be essential for the formation of long-term memories in species ranging from fruit flies to mice. Although short-term response of the brain to novel and potentially threatening situations may be adaptive and result in new learning and acquired behavioral strategies for coping, as may be the case for certain types of fear-related memories, repeated stress can cause cognitive impairment via at least four different mechanisms:

Impairing neuronal excitability: Adrenal steroids biphasically modulate long-term potentiation (LTP), with low levels enhancing it and high levels impairing LTP in regions of the hippocampus that use NMDA receptors; other measures of excitability are also affected by adrenal steroids.

Causing atrophy of nerve cells in the Ammon’s horn region of the hippocampus: Adrenal steroids facilitate a remodeling of apical dendrites of pyramidal neurons in the CA3 region of the hippocampus that is caused by excitatory amino acids; such remodeling is reversible as long as stress is terminated after a number of weeks.

Inhibiting neurogenesis in the dentate gyrus region of the hippocampus: The adult hippocampus continues to produce nerve cells in adult life, and this process is inhibited by certain stressors and by activation of NMDA receptors as well as by elevated glucocorticoids.

Causing permanent loss of nerve cells in hippocampus: Prolonged psychosocial stress causes damage and apparent neuron loss in the hippocampus. These processes may occur somewhat independently of each other and contribute in various degrees to different pathophysiological situations involving traumatic stress, depression, or aging.

Effects of Stress and Stress Hormones on Cognitive Function

Having reviewed the potential mechanisms by which stress and stress hormones can biphasically modulate learning and memory processes, we now consider the information available on stress and glucocorticoid effects on cognitive function in human subjects. The cognitive effects of elevated concentrations of glucocorticoids in human populations have been studied in disorders affecting corticosteroid levels and by using exogenous administration of the synthetic compound to healthy subjects. Mental disturbances mimicking mild dementia (such as decrements in simple and complex attentional tasks, verbal and visual memory, encoding, storage, and retrieval) have been described in depressed patients with hypercortisolism and in those with steroid psychosis following corticosteroid treatment. Similar cognitive deficits are also reported in patients suffering from Cushing’s disease. During human aging, a significant proportion of elderly individuals present an endogenous increase of glucocorticoid levels, and this increase has been related to impaired memory performance. Moreover, many investigators have reported inverse relationships between mean 24-hr cortisol levels and severity of cognitive decline in Alzheimer patients.

Studies in both animals and humans have shown that the glucocorticoid-induced memory impairment is related to an atrophy of the hippocampus. Hippocampal atrophy associated with chronic exposure to high levels of glucocorticoids is reported in Cushing patients, elderly individuals, depressed patients, and individuals suffering from posttraumatic stress disorders. This is a significant finding and implicates the hippocampus since the declarative memory impairments that are induced by chronic exposure to high levels of glucocorticoids are those attributed to the hippocampus in memory function. It is known that the hippocampus plays a significant role in declarative memory function, whereas it has little function in non declarative memory function. Declarative memory refers to the conscious and voluntary recollection of information that was previously learned, whereas non declarative memory function refers to the facilitation in performance observed after exposure to a given information, without necessary consciousness of recall of this

information. Many studies have shown that hippocampal damage in animals and humans leads to declarative memory impairments, whereas non declarative memory is unimpaired. This is the pattern of memory dysfunction reported to occur in all cases of chronic exposure to high levels of glucocorticoids. However, studies of endogenous disorders generally fail to discriminate the cognitive deficits related to HPA hyperactivity from those due to the underlying illness. Thus, most of the cognitive deficits associated with corticosteroids are derived from those observed during acute exogenous administration of synthetic glucocorticoids to healthy subjects. In general, studies measuring the acute impact of glucocorticoids on cognitive function report that this steroid impairs selective attention (i.e., the ability to discriminate relevant from irrelevant information), which thus impairs encoding of incoming information. This finding is in accordance with electrophysiological results showing that acute administration of cortisol to human subjects reduces the average evoked potential response to relevant but not to irrelevant stimuli. These findings are also consistent with studies showing that glucocorticoids can impair neuronal electrophysiology and hippocampal long-term potentiation. Recent studies have reported that an acute increase of glucocorticoids also impairs working memory function.

Working memory is the cognitive mechanism that allows us to keep a limited amount of information active for a limited period of time. Working memory impairments have been found in several experiments using a variety of delay task procedures. In these tasks, a temporal gap is introduced between a stimulus and a response, which creates the need to maintain the stimulus in a temporary memory storage. Interestingly, data obtained in monkeys show that cells in the lateral prefrontal cortex become particularly active during delayed response tasks, suggesting that these cells are actively involved in holding on to the information during the delay. This result is in accordance with studies reporting a high density of corticosteroid receptors in the cerebral cortex of both rat and human. Receptor binding studies in rats have shown the presence of adrenal steroids in the cortex, particularly in the medial prefrontal regions. Further studies in rats and humans have shown that the prefrontal cortex is a significant target for the negative feedback actions of circulating glucocorticoids, which suggests that this area could play a significant role in the acute effects of corticosteroids on cognitive function. Thus, the hippocampus is not likely to be the only brain area affected in this way since atrophy of the amygdala and prefrontal cortex has also been reported in depressive illness. Reversibility and/or preventability of such atrophy is a major topic for future research, as is the implication of such treatment for cognitive function. A recent study showed that treatment of Cushing's patients induces a 10% reversibility in the hippocampal atrophy that was induced by chronic exposure to high levels of glucocorticoids

Stress and Mental Illness

Stress is generally acknowledged to play a paramount role in the pathogenesis of many psychiatric disorders. However, not everyone exposed to a given stressor generally considered likely to precipitate a psychiatric syndrome becomes ill. For example, pancreatic cancer has long been believed to convey a substantial risk for developing a major depressive episode. In

fact, depression commonly predates the onset of physical symptoms of the cancer. Nevertheless, one-half of patients with pancreatic cancer do not become depressed. Similarly, PTSD remains a significant burden to many Vietnam combat veterans nearly 30 years after the conclusion of that war. However, most Vietnam combat veterans (85%) do not suffer from PTSD. Why do some individuals succumb to a particular stressor and become ill while others do not? Stressful life events can obviously serve as acute precipitants to psychiatric and medical illness. However, some individuals tolerate stress of great magnitude and long duration without becoming ill. Others exhibit a constitutional vulnerability to the effects of stress (i.e., a lower threshold of tolerance for stress that predisposes them to stress-induced illness). This inherent vulnerability to the adverse effects of stress is known as a diathesis and provides the basis for the diathesis/stress disease model. The diathesis/stress model has in recent years been applied to a broad range of psychiatric and medical disorders, including major depression, schizophrenia, chronic fatigue syndrome, PTSD and other anxiety disorders, sexual disorders, and pain disorders such as fibromyalgia and arthritis.

This model theoretically has practical application to other psychiatric syndromes, including somatoform disorders, eating disorders, attention-deficit hyperactivity disorder, and impulse control disorders. In addition, neurological disorders including epilepsy and migraine headaches, rheumatological diseases such as systemic lupus erythematosus and other illnesses such as irritable bowel syndrome and diabetes mellitus may be appropriate to consider from the framework of a diathesis/stress model. What is the origin of the diathesis? The relative contributions of genetic inheritance and environmental exposure to the susceptibility to illness have been deliberated in often contentious nature versus nurture debates. Such arguments are often couched in overly absolute terms, but the diathesis/stress model permits a more balanced consideration. Diathesis/stress models recognize that both inherited and acquired factors may contribute to the vulnerability to stress. A full accounting of the genetic contribution to the predisposition to stress-related illness is beyond the scope of this article. Nevertheless, human twin studies have clearly revealed a significant genetic contribution to the vulnerability to many psychiatric syndromes, including depression, bipolar disorder, and schizophrenia.

To date, it has not been possible to identify with certainty the chromosomal localization much less the precise gene(s) that forms the basis of the genetic contribution to the diathesis for psychiatric disorders. From years of largely unsuccessful research by psychiatric geneticists, it is clear that the genetic contribution to the vulnerability for psychiatric illnesses is unlikely to arise from simple single gene Mendelian transmission. It is more likely that the heritable vulnerability to psychiatric illness arises either from complex polygenic patterns of inheritance or from even more complex epigenetic modification of genotypic risk. For example, in recent years, the existence of resistance genes that interact with susceptibility genes has been postulated. Epidemiological research also indicates that the environment makes a substantial contribution to the vulnerability to psychiatric illnesses. Environmental contributions to the diathesis may emerge from any of a variety of biopsychosocial stressors. Consequently, stress plays a dual role in the pathogenesis of

psychiatric illness. When stress is coincident with the onset of an illness, it serves as an acute precipitant to the disorder. In contrast, when stress predates the onset of a disorder, it may well shape the predisposition to future illness. Although the major environmental contributions to the diathesis occur during the formative childhood years, the diathesis remains mutable throughout adult life. Stresses during adulthood continue to modify the predisposition to illness. In fact, disease is a stressor that can increase the risk for future episodes of illness. Theoretically, the stress-induced predisposition to illness should be both psychometrically and biologically measurable. It is this impression that underlies a burgeoning line of research investigating the persistent neurobiological sequelae of early life adverse experiences.

EARLY WARNING SIGNS OF STRAIN

From the prevention perspective, it is important to recognize the earliest warning signs of strain in order to intervene early in the process. Worker strain can result following prolonged exposure to stressors when paired with poor response patterns. Pains of unknown origin, fatigue, inability to concentrate, and irritability are early warning signs for individuals. In addition, symptoms of depression, increased anger and hostility, increased accidents, or signs of substance abuse can be clues that interventions are warranted. These early warning signs may be accompanied by increased aggressive behavior, disrespect, and withdrawal from relationships. For organizations, early warning signs that normal work stressors are leading to strain include general patterns such as slight changes in productivity with decreased quantity and/or quality of work, increased absenteeism, decreased commitment to the organization, poor interpersonal work relationships, increased tardiness, and more conflict among workers. Other early signals of distress include general dissatisfaction, low motivation, and low morale. Increased accident rates and machine breakdowns may also constitute early warnings. There may be a general loss of vitality within the organization and an atmosphere of distrust and animosity. As with any preventive strategy, early detection of strain can help to reduce the long-term negative consequences. Key to early detection and sound prevention is vigilance on the part of the management team.

PREVENTIVE STRESS MANAGEMENT

Prevention is the best public health strategy for any disease epidemic. Because job stress is a health epidemic, prevention holds the best hope for addressing this epidemic. The theory of preventive stress management translates the public health notions of prevention into an organizational context and overlays them on a stress process model. Stress is one of several chronic organizational health problems—with others being workplace violence, sexual harassment, and suicide—for which prevention is appropriate. Preventive stress management is an organizational philosophy and set of principles that employs specific methods for promoting individual and organizational health while preventing individual and organizational distress. This philosophy is based on the following five guiding principles that motivate the practice of preventive stress management and provide a framework for healthy organizations and healthy leaders:

Principle 1: Individual and organizational health are interdependent.

Principle 2: Leaders have a responsibility for individual and organizational health.

Principle 3: Individual and organizational distress are not inevitable.

Principle 4: Each individual and organization reacts uniquely to stress.

Principle 5: Organizations are ever-changing, dynamic entities.

Chronic diseases do not arise suddenly; instead, they develop gradually through a progression of disease stages, a “natural life history.” This is true for chronic individual disorders, such as heart disease, as well as for chronic organizational problems, such as workplace violence. The natural history of most diseases is one of evolution from a stage of susceptibility to a stage of early disease and finally to a stage of advanced or disabling disease. At the stage of susceptibility, the individual is healthy but is exposed to certain risk factors or disease precursors. For example, individuals who choose a sedentary life or who choose to smoke cigarettes are at the stage of susceptibility for coronary artery disease as well as several other diseases. When these and other risk factors lead to the development of arteriosclerosis or hardening of the arteries to the heart, the individual is at the stage of early disease or preclinical disease. In other words, the person’s body has responded to the disease precursors, but there are few, if any, symptoms. As the disease advances, it becomes symptomatic or clinical disease. Angina pectoris (heart pains) and heart attacks are advanced manifestations of coronary artery disease.

Preventive stress management is rooted in the public health notions of prevention, which were first used in preventive medicine. The term public health encompasses a broad array of health protection activities inspired by the practice of viewing illnesses within a social context. The dominant diagnostic model in public health involves the interaction between a host (the individual), an agent (health-damaging organism or substance), and the environment. One of the fundamental concepts of preventive medicine is that there is an opportunity for preventive intervention at each stage in the life history of a disease, as noted previously. These interventions are aimed at slowing, stopping, or reversing the progression of disease. There are three stages of prevention strategies: primary prevention, secondary prevention, and tertiary prevention. Primary prevention aims to modify and manage the job stressors and other demands in the work environment. Secondary prevention aims to modify and manage the individual’s response to these job stressors and other demands. Tertiary prevention aims to help and provide aid to those who are experiencing behavioral, psychological, or medical strain symptoms. From a public health perspective, primary prevention is always the preferred point of intervention. For job stress and workplace health, this implies that job redesign efforts and other interventions that alter, modify, or eliminate stressful work conditions are the preferred category of preventive stress management interventions. Primary prevention is the protection of health directed at the stage of susceptibility and aims to eliminate or reduce the impact of risk factors; it is intervention before the onset of problems or disorders. Primary prevention may be either organizational or individual. Organizational strategies include the following:

- Job redesign
- Participative management
- Flexible work schedules
- Career development
- Design of physical settings
- Individual strategies include the following:
 - Learned optimism
 - Planning and time management
 - Social supports and secure attachments
 - Maintaining a work-home balance
 - Changing type A behavior pattern

It is important to recognize that some primary prevention techniques directly alter stressors. Job redesign, for example, may incorporate more control into a job, or more decision latitude, thereby reducing the stress from lack of control. On the other hand, some primary prevention methods alter the individual's perception of the stressor. Social support, for example, can help individuals reframe stressors as challenges because they believe they possess the resources to manage a stressor. In both cases, primary prevention is the preferred first defense against stressors. Secondary prevention aims at the early detection of problems or disorders and prompt, early interventions to correct departures from health. Secondary prevention may also be either organizational or individual. Organizational strategies aim at improving relationships in the work environment. Organizational strategies include the following:

- Goal setting
- Role analysis
- Team building
- Social supports and secure attachments
- Diversity training and programs
- Individual strategies include the following:
 - Relaxation training
 - Prayer, spirituality, and faith
 - Emotional outlets
 - Physical fitness and exercise
 - Nutrition

Secondary prevention efforts focus on the development of strengths for both individuals and organizations. Team-building efforts, for example, can lead members to understand each other better, manage conflict more effectively, and be more productive. Individual strategies, such as relaxation training, meditation, and exercise, make the body and mind more resilient and more capable of dealing with stress. Tertiary prevention is prevention of more serious problems, disorders, or death. It is directed at expediting or improving treatment for symptomatic or advanced disease and aims to alleviate discomfort and restore effective functioning. Tertiary prevention is primarily focused at the individual level rather than the organizational level, as is done in treating mental health referrals. Tertiary prevention also includes psychological counseling and medical or surgical treatment. Individual strategies include the following:

- Individual psychotherapy
- Behavior therapy
- Medical care and treatment
- Traumatic event debriefings
- Physical therapy

Tertiary prevention is thus focused on procuring care from qualified professionals who can help individuals heal. The goal of the preventive stress management approach is to utilize sufficient primary and secondary prevention such that individuals seldom, if ever, need to turn to tertiary prevention for relief.

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