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HEALTH PSYCHOLOGY: Mapping Biobehavioral Contributions to Health and Illness

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ABSTRACT

Our evolving understanding of how psychosocial and behavioral factors affect health and disease processes has been marked by investigation of specific relationships and mechanisms underlying them. Stress and other emotional responses are components of complex interactions of genetic, physiological, behavioral, and environmental factors that affect the body's ability to remain or become healthy or to resist or overcome disease. Regulated by nervous, endocrine, and immune systems, and exerting powerful influence on other bodily systems and key health-relevant behaviors, stress and emotion appear to have important implications for the initiation or progression of cancer, HIV, cardiovascular disease, and other illnesses. Health-enhancing and health-impairing behaviors, including diet, exercise, tobacco use, and protection from the sun, can compromise or benefit health and are directed by a number of influences as well. Finally, health behaviors related to being ill or trying to avoid disease or its severest consequences are important. Seeking care and adhering to medical regimens and recommendations for disease surveillance allow for earlier identification of health threats and more effective treatment. Evidence that biobehavioral factors are linked to health in integrated, complex ways continues to mount, and knowledge of these influences has implications for medical outcomes and health care practice.

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CONTENTS

INTRODUCTION 138
BEHAVIOR AND HEALTH 139
Stress 140
Coping 141
BIOBEHAVIORAL INFLUENCES IN DISEASE PROCESSES 143
Stress and Disease Processes 143
Stress, Behavior, and Cancer 145
Stress, HIV, and AIDS 146
HEALTH-PROTECTIVE AND HEALTH-IMPAIRING BEHAVIORS 147
Diet, Obesity, and Health 147
Exercise 150
Cigarette Smoking and Tobacco Use 150
Sun Protection and Skin Cancer Prevention 152
HEALTH BEHAVIOR 152
Screening and Surveillance 153
Adherence 154
CONCLUSIONS 155

INTRODUCTION

Thoughts, feelings, and behaviors affect our health and well-being. Recognition of the importance of these influences on health and disease is consistent with evolving conceptions of mind and body and represents a significant change in medicine and the life sciences. This history of medicine is marked by the rise and fall of misconceptions and ineffective therapies and by ill-conceived notions of how the body works were dominant for hundreds of years. The development of modern theories of atherosclerosis, for example, explained heart disease in ways that prompted effective medical interventions just as germ theories and models of immunity revolutionized the treatment and prevention of infectious illnesses. More recent developments include the idea that emotional processes such as stress moderate activity in nearly all systems of the body and can directly influence the pathophysiology of disease. Discovery of these and other relationships between behavior and health has changed the way health and disease are portrayed.

Interest in health and behavior is expanding at the National Institutes of Health, in the media, at work and school, and increasingly in medical schools and medical practice. This interest is also reflected in the rapid development of health psychology and its more multidisciplinary cousin, behavioral medicine. These fields grew rapidly in the 1980s and now constitute major endeavors in most university and medical center settings. The research upon which these activities is based embraces a broad agenda, including studies of prevention, screening, and early detection, etiology of disease, predictors of prognosis to treatment of disease, rehabilitation, and post-illness adjustment and quality of

life. Basic research on the mechanisms and outcomes of bodily changes associated with stress, tobacco, diet, exercise, or other psychosocial variables and clinical studies of intervention and/or treatment outcomes provide compelling evidence of the extent and importance of psychosocial or behavioral variables.

BEHAVIOR AND HEALTH

Although experimental evidence is still inconsistent in some cases, the weight of data from studies of health and behavior strongly suggests that psychological processes and emotional states influence the etiology and progression of disease and contribute to overall host resistance or vulnerability to illness. In general, psychosocial or behavioral factors exert their influence on health or illness in three basic ways (e.g. Krantz et al 1985). First, some of these influences involve direct biological changes that parallel, precede, are induced by, or occur as part of an emotional reaction or behavior pattern. Research has shown, for example, that stress involves increases in blood pressure, heart rate, and sympathetic arousal and is associated with hematological changes that can contribute directly to heart disease, hypertension, or cardiac events (e.g. Manuck & Krantz 1984; Patterson et al 1994, 1995; Schneiderman 1983). Similarly, stress appears to affect the immune system through a complex array of neural and hormonal pathways (Besedovsky & DelRey 1991, Maier & Watkins 1998). Whether these immune system changes are strong or prolonged enough to enhance vulnerability to infection or illness is not clear, but they have been implicated in the etiology and progression of viral infections, wound healing, cancer, and HIV disease (e.g. Andersen et al 1994, Baum & Nesselhof 1988, Cohen & Williamson 1991, Kiecolt-Glaser et al 1985).

A second way in which health and disease are influenced is by behaviors that convey risks or protect against them. *Health-enhancing* behaviors are activities that convey health benefits or otherwise protect people from disease. *Health-impairing* behaviors are activities that have harmful effects on health. Diet and exercise are often cited as protective behaviors, and tobacco use and alcohol abuse as health-impairing behaviors. Diet and exercise can help minimize the conditions underlying cardiovascular disease and cancer. Tobacco use is associated with biological changes in the lungs, heart, and other bodily systems that appear to predispose disease. Similarly, drug use, high-risk sexual activity, and other potentially harmful behaviors are important mediators of disease processes.

A third general pathway for behavioral influences on health and illness is through behaviors associated with illness or the possibility that one is ill. Beginning with early detection, these influences include secondary prevention, surveillance, detection and interpretation of symptoms, and the decision to seek care. Adherence with medical advice or prescription can also affect diag-

nosis and treatment and affect health outcomes. Interference with any of these can disrupt the process of interpreting and presenting symptoms, obtaining prompt and effective medical care, and complying with treatment, surveillance, or prevention regimens.

Elaborations of this basic model of health and behavior have been developed to help explain the comorbidity of physical and mental health problems and the etiology and progression of cancer and stress-related diseases (e.g. Andersen et al 1994, Cohen & Rodriguez 1996). Evidence linking key pieces of these models has been reported, and although it is far from complete in many instances, it supports the notion that behavioral variables affect health and illness at these three different levels. This review considers evidence for this psychosocial perspective, focusing on major life-threatening illnesses such as cardiovascular disease, cancer, and HIV disease. Because of its complex role in health and illness, stress will be considered briefly before turning to evidence of direct biological effects on health and illness, of indirect and direct effects of health-impairing behaviors, and of the counter-influence of health-enhancing behaviors. Health behaviors, particularly adherence and early detection behavior, are also considered.

Stress

Stress is a particularly important mediator of health-behavior relationships because it is a common and seemingly inevitable aspect of life and because its broad effects can influence a range of bodily systems and behaviors. Its pervasiveness belies the controversy that surrounds its definition, and its breadth has led to debates over measurement and the utility of stress as a singular construct. Stress generally refers to the nonspecific aspects of dealing with environmental change, demand, and/or threat, though some models characterize it as a stimulus and others describe it solely in terms of responses (Cannon 1929, Selye 1976, Mason 1975). Depiction of stress as nonspecific mobilization was consistent with evolutionary theory, suggesting that stress evolved because nonspecific arousal strengthens and supports efforts to adapt to environmental threats. Some have argued that stress is best viewed as a process, with a signature psychological state and associated physiological changes (Lazarus 1966). Similar to emotions, stress consists of characteristic physiological, affective, cognitive, and behavioral changes that can have costs for well-being whether or not successful adaptation is achieved. Integrated biobehavioral patterns of activation such as stress appear to exert fundamental effects on health.

STRESS AND EMOTION Stress appears to involve more or less simultaneous activation of psychological and biological systems. Recognition of a threat or challenge is accompanied by immediate systemic arousal produced by

the sympathetic nervous system (SNS) and the hypothalamic-pituitary-adrenocortical (HPA) axis. At the same time, stress is associated with changes in mood, alertness, attention, memory, problem-solving, task performance, and well being. This cascade of changes is initiated by detection of a stressor and/or appraisal of the extent to which threat, danger, or challenge are likely (Lazarus 1966). This appraisal also includes evaluation of the resources one can bring to bear on a stressor, and appears to affect subsequent response (Tomaka et al 1997). Once threat or demand is recognized, ensuing responses may all occur simultaneously and changes in arousal and discomfort are rapid. It is not clear whether one or another aspect of the stress response triggers the others or whether these changes emerge more or less as a single integrated pattern. Regardless, stress is typically characterized by the same kind of central and peripheral activation as are emotions such as anger or fear.

EFFECTS OF STRESS Another implication of the foregoing discussion is that the arousal associated with stress may be a primary mechanism by which stress affects health. Nonspecific biological and behavioral changes that accompany appraisal and/or emotional arousal can be thought of as the *stress response*, including heightened cardiovascular and respiratory function, and changes in blood flow, digestion, and skeletal muscle tone. Behavioral changes in attention and alertness are also part of this basic stress response and help focus and support coping activity. Endocrine, immune, and neural changes also occur, and stress responses reflect those that are related to supporting maximal strength and vigilance.

All these changes have negative side-effects or byproducts or can cause harm if stress is prolonged or very intense. Persistent, sustained physiological arousal or frequent, rapid increases in arousal have a range of consequences, including wear and tear on arteries and coronary vessels, formation of thrombi, suppression of host resistance, and other direct biological effects. Changes in alertness, narrowed attention, and irritability could produce consequences such as poorer problem-solving and task performance, disrupted social relationships, and poorer quality of life, which could affect decisions involving health risks and affect health indirectly. The emotional, cognitive, behavioral, and physiological changes that occur as part of the stress response are the mechanisms by which stress conveys consequences for health and well-being.

Coping

Coping constitutes an important aspect of stress, one of the principal routes that behavioral and cognitive responses can take during stress. Ultimately, coping is the main focus of stress responses that support strong, rapid reactions. Stress appears to have two basic functions: to motivate people to ma-

nipulate or accommodate stressors and to support activity directed at reduction or elimination of them. Although stress can be generated by positive as well as negative events, it is generally experienced as discomfort, tension, or negative affect. Harm, loss, anger, threat, and uncertainty are all associated with negative emotions, and the arousal experienced as a function of stress is also considered unpleasant or uncomfortable. This discomfort or tension produced during stress motivates the individual experiencing stress to do something about it. This suggests that the changes associated with stress support coping, the primary product of stress. We have suggested, for example, that stress involves arousal designed to support rapid, strong response to danger. Coping is that response to danger consisting of behavior or other activities that are intended to resolve the stressor or minimize its effects.

Coping is the most specific of stress responses. Unlike the readying response described above, coping is thought to be selected by individuals because it is well-suited to the stressor or situation (Lazarus & Folkman 1984). Application of particular kinds of coping is also affected by the resources one brings to the situation and by person variables that influence one's choices or predispositions to act (e.g. Scheier & Carver 1992, Lester et al 1994). The effectiveness of coping aimed at manipulating the stressor or attacking the problem posing the threat can usually be readily assessed. However, coping directed at minimizing, deflecting, or managing distress are more difficult to evaluate and may become more persistent, generalized responses to threat or demand. In this regard, well-learned responses like social withdrawal or helplessness may become predominant coping devices and potentially harmful behaviors such as smoking, drinking, and drug use may be used routinely to reduce distress or self-medicate discomfort.

The arousal that motivates and supports these actions is thought to be one mechanism by which stress affects health, increasing wear and tear on bodily systems and damage to arteries, neural systems, and organ systems, and reducing resistance to pathogenesis. Coping that conveys specific effects on physiological systems (e.g. drug use) can add to the potential for calamity by further suppressing immune system function, taxing the heart and circulatory system, damaging the lungs, or depriving the body of the nutrients it needs during sustained or repeated activation.

If coping is the product of stress response, its potential health consequences are a byproduct of this activity and reflect nonspecific costs of coping. Alternatively, stress-related disease may reflect breakdown in compensatory systems designed to relieve the arousal built up during stress or otherwise protect the body from its harmful effects. Exercise, for example, may help reduce arousal or manage its negative impact. The complex balance that defines good health and the many ways in which stress can disrupt it are among the factors that explain why stress exerts such pervasive effects on health and disease.

BIOBEHAVIORAL INFLUENCES IN DISEASE PROCESSES

At a neural or molecular level, the similarities and interconnectedness of behaviors and biological events are unmistakable, and at higher levels of functioning it appears increasingly certain that psychological or behavioral variables affect and are affected by their biological context. At some levels, behavior is produced by a complex series of neural, hormonal, and effector systems and these systems are regulated by bodily milieu. The biological correlates and consequences of emotional reactions, stress, and many thoughts and behaviors also contribute directly to host defense, health, and illness.

Emotional events are processed in the CNS by several partially redundant structures, including the limbic system and reward centers in the brain. They can be viewed as neural events that have been selected through evolution and that have helped assure our species' survival (LeDoux 1996). Emotional patterns such as stress responses are hardwired in the CNS, but one's experience also defines the content and conditions that give rise to particular responses (LeDoux 1996). In a sense, the emotional system "learns" about the external correlates of internal responses. This "learning" about the external correlates of internal responses may also be characteristic of stress. Factors that affect the magnitude and duration of stress responses are learned but the responses themselves are highly integrated and automated. These response patterns appear to involve nonspecific changes in most bodily systems and more specific reactions associated with the situation and sources of stress affecting the organism.

Stress and Disease Processes

Many biological changes accompanying stress or emotional arousal are extended and intensified by a cascade of endocrine and immunological changes that help to modulate bodily response. These regulatory systems induce circulatory changes, increase heart rate and respiration, and otherwise prepare the organism for action or attention. Research suggests that acute stress is accompanied by increased blood pressure, heart rate, and SNS arousal (e.g. Santagostino et al 1996, Krantz & Manuck 1984, McFetridge & Yarandi 1997). Most systemic arousal associated with stress and emotions such as anger, fear, and sadness reflects catabolic activation in which stored energy is converted into a usable resource and in which growth and repair functions are inhibited (Baum 1990, Mason 1975).

STRESS AND CARDIOVASCULAR RISK Research on the effects of stress and emotional arousal on the cardiovascular system suggests that these states affect pathophysiology as well as trigger disease-related events (e.g. Johnston

1997). For example, anger is associated with responses that promote ischemia or arterial occlusion, heart attacks, and arrhythmias (R Verrier & Mittelman 1996). Anxiety and bereavement are also associated with cardiac events, and stress appears to predispose cardiovascular disease or precipitate ischemic episodes, heart attacks, or other pathological states (Niedhammer et al 1998, Carney et al 1998). Presumably this occurs through a series of interrelated effects of stress or emotional arousal, including direct effects on the heart, on the vasculature, on blood flow and shear stress, and on the constituents of blood such as platelets (Ku 1997; Niebauer & Cooke 1996; Patterson et al 1994, 1995). Hostility has also been linked to sodium consumption and to cardiovascular hyperactivity and risk for high blood pressure (e.g. Miller et al 1998).

Cardiovascular reactivity reflects the magnitude and duration of response to acute stress or challenge and may reflect mediators of cardiovascular risk (Krantz & Manuck 1984, Matthews et al 1990). The magnitude and elasticity of stress responses vary; larger increases in blood pressure or heart rate that follow introduction of a stressor may predict a range of intermediate and more distal outcomes. Blood pressure reactivity appears to be related to future blood pressure and hypertension (e.g. Matthews et al 1993, Pickering & Gerin 1990). Exaggerated blood pressure reactivity is associated with atherosclerosis, although these relationships appear to be modest and most evident among young men (Manuck et al 1989, Jennings et al 1997). Effects of behavior and stress on cholesterol, lipids, neurohormonal regulatory systems, silent ischemia, and oxidative damage have been observed (e.g. E Verrier & Boyle 1996, Howes et al 1997, Pool 1997, Mosca et al 1997). These effects are also related to age, behaviors like smoking, and psychological variables or personality styles such as defensiveness and repressive coping (e.g. Jennings et al 1997, Girdler et al 1997, Helmers & Krantz 1996).

STRESS AND IMMUNE STATUS Research on psychosocial mediation of the immune system suggests it is responsive to changing moods and behaviors. Research in human and animal models is consistent in suggesting that immune system activity is regulated by the CNS (Moynihan & Ader 1996). Further, evidence indicates that stress affects activity in the immune system. Research has been complicated by the fact that responses among different components of the immune system are variable (e.g. Zakowski 1995). Some cells, like natural killer cells, appear to respond differently depending on the chronicity of the stressor (Delahanty et al 1996). Some immune cells also appear to respond to stress differently at different times of day and to follow clear circadian rhythms (Wang et al 1998). Direct effects of stress include acute activation and chronic suppression of natural killer cells, increased latent viral activity, decreased lymphocyte proliferation, and cytokine production (Andersen et al 1998, Pariante et al 1997, Schedlowski et al 1995, Kiecolt-Glaser et al 1994).

These influences are thought to be caused by neural and hormonal regulation, principally through arousal of the SNS, the HPA axis, and opioid peptide systems (e.g. Webster et al 1997). Nerve growth factor and cytokine influences are also possible and suggest a broad interface between the nervous and immune systems (e.g. Jenkins & Baum 1995, Aloe et al 1997). The importance of the SNS in observed stress-immune relationships is suggested by studies reporting correlations between SNS indices and immune system change or by studies of the effects of adrenergic blockade (Bachen et al 1995, Manuck et al 1991, Zakowski et al 1992a,b). Exercise and exertion also affect the immune system, often in ways that resemble the impact of stress or emotional arousal (e.g. Perna et al 1997, Nieman 1997).

The immune system communicates directly with the CNS and appears to have a regulatory function in much the same way that the endocrine system supplements and extends neural activation (Maier & Watkins 1998). In addition, some mental health problems are associated with abnormal immune system function (Weisse 1992, Herbert & Cohen 1993). Some of these effects are mediated by cytokines that are activated by neural stimulation (Muller & Ackenheil 1998). Pro-inflammatory cytokines in turn activate the HPA axis and induce fever and illness symptoms, but these effects may also be influenced by glucocorticoids and other stress-related agents (Goujon et al 1997). Interleukin-6 (IL-6) appears to activate the HPA axis as well and may be a component of CNS integration of the stress response (Path et al 1997).

We do not yet know whether observed patterns of change in the immune system reflect alterations that have implications for vulnerability to disease (e.g. Cohen & Rabin 1998). However, there is considerable evidence of changes within the immune system associated with social support, negative affect, stress, and other behavioral or psychological factors, and evidence that these factors affect infectious disease, progression of cancer and HIV disease, and other health outcomes (e.g. Leserman et al 1997, Antoni 1997, Andersen et al 1994, Cohen et al 1991).

Stress, Behavior, and Cancer

Cancer is a generic name for a number of genetic diseases in which normal cells mutate and begin to grow uncontrollably. Cancer typically requires considerable time to develop or to be detected, and there are currently few good markers of disease or disease progression for most forms of cancer. With the lack of good biomarkers for disease processes, surveillance efforts seek to quantify people's risk for cancer and maximize the likelihood of detecting it early in more readily treatable stages.

These limits on our ability to detect very early stage disease or to follow its slow early development make it difficult to study the role of emotions and behavior in the etiology of cancer. Most relevant studies are limited by one or an-

other methodological problem. However, it appears likely that emotional states like stress affect primary cellular functions, such as DNA repair, that could contribute to cancer (e.g. Kiecolt-Glaser et al 1985). It also appears that emotional reactions and adjustment to having cancer or to its treatment can affect progression of disease, survival, quality of life, and other important outcomes of cancer (e.g. Andersen et al 1994, Ramirez et al 1989, Levy et al 1991, Helgeson & Cohen 1996). Some of the strongest evidence of stress or psychosocial mediation of cancer-related outcomes is drawn from intervention programs that provide supportive and/or psychoeducational interventions for cancer patients (e.g. Spiegel et al 1989, Fawzy & Fawzy 1994, V Helgeson et al 1998). Several reviews have concluded that stress management, coping skills training, support, and other aspects of these interventions affect progression of disease, but mechanisms governing these effects are not known (Meyer & Mark 1995, Baum et al 1995, Posluszny et al 1998).

Stress, HIV, and AIDS

HIV disease and AIDS are unusually important as examples of behavior-influenced outcomes. They reflect both direct effects on bodily functioning and on behaviors that may affect disease risk and prognosis. They also affect caregivers in powerful ways that can harm their health as well (e.g. Catalan et al 1996, Folkman et al 1997, Folkman 1997, Park & Folkman 1997). The transmission of the virus that causes HIV disease is typically accomplished through behaviors (sexual activity, IV drug use), and because HIV attacks the immune system directly, behaviors or states that affect the immune system should affect HIV. Mental health problems appear to be associated with an increased risk for HIV infection (Hoff et al 1997, O'Leary & Jemmott 1996), presumably because of distress-related increases in risky behavior. High-risk behaviors such as unprotected sexual activity or IV drug use can be minimized, but stress can trigger impulsive behavior, reduce countervailing constraints against high-risk behaviors, or temporarily inhibit or interfere with intentions to avoid them (e.g. Fishbein et al 1998, O'Keefe et al 1990). These impulsive behaviors may occur because of stress-related drug and alcohol use (e.g. Testa & Collins 1997, Robins et al 1997). The implications of increased drug or alcohol abuse include reduced motivation, a false sense of invulnerability, and lack of attention to details or sanctions leading to increased risk and infection (Dingle & Oei 1997, Chandra et al 1996). As a result, people who are intoxicated by drugs or alcohol may not use good judgement, may be more likely to end up in compromising situations, or may simply not care about risks to their health at that moment.

Emotional arousal, stress, and behaviors such as smoking or drug use may affect resistance to the disease or disease progression. Evidence of this possibility is scant and is largely limited to early outcomes related to testing (Evans

et al 1997, Antoni et al 1994). However, stress and related states or behaviors are associated with changes in immune system activity that could affect the body's ability to resist or combat the virus. A study of 104 HIV+ individuals enrolled in the Oslo HIV Cohort Study suggested that negative affect was related to somatic symptoms associated with progression, but there was no evidence of mediation of these effects by the immune system (Vassend et al 1997). Distress associated with concealment of sexual identity was positively related to cancer morbidity and incidence of pneumonia and other infectious illnesses (Cole et al 1996). Stress management interventions that enhance or buffer immune system activity have proven useful in treatment of HIV+ individuals (Littrell 1996, Goodkin et al 1997, Pomeroy et al 1997, Schneiderman et al 1992). Aerobic exercise interventions that also reduced distress have had similar effects (LaPerriere et al 1994).

HEALTH-PROTECTIVE AND HEALTH-IMPAIRING BEHAVIORS

Direct effects of stress and emotions are supplemented or modified by behaviors that affect health and disease processes. Behaviors such as diet or exercise can affect health independently but may mitigate, enhance, or modify the effects of stress. Conversely, stress may suppress these behaviors or their benefits. Diet, exercise, sleep, and relaxation are considered to be protective factors that can exert direct effects on physiological sources of risk and indirect effects by reducing the effects of stress and enhancing mood. In addition, stress can affect the likelihood of maintaining these behaviors. A healthy diet appears to directly reduce risk of disease, but maintenance of good nutrition is based on complex motivation and reward systems, and stress can affect the likelihood that one will maintain healthy dietary behavior. Poor diet, conversely, appears to contribute to pathophysiology of disease. In addition, smoking, alcohol consumption, drug use, and other health-impairing behaviors also have direct effects on disease processes and indirect effects on bodily systems and mood or behavior. Together these behaviors reflect insufficiently studied elements in the etiology of disease and keys to health promotion efforts that seek to minimize risk. Maximum sustained reduction of modifiable risk is an important implication of this reasoning and research.

Diet, Obesity, and Health

What people eat and how much they weigh are inherently behavioral processes. The fact that some people are able to maintain a normal or healthy weight while others become obese is almost certainly a result of behavioral and psychological factors working in concert with genetic and metabolic characteristics. Increasingly, one's diet and weight have been implicated in a

number of health problems or adult onset diseases, and weight management and nutritional risk management programs have become a standard part of wellness or health promotion campaigns (e.g. Wing 1995a, Weiss et al 1991). Use of vitamin supplements, increasing consumption of fruits, vegetables, and fiber, and moderation of consumption of animal fat are widely believed to predispose better health. Healthier diets and proper nutritional balance may also facilitate weight management and help to prevent obesity. However, dietary change often requires substantial lifestyle change, and weight control has proven difficult for many people to maintain (Wing 1995b).

OBESITY AND CARDIOVASCULAR RISK Perhaps the clearest links between diet and health or disease outcomes are for cardiovascular disease, where fat and cholesterol intake as well as salt consumption, obesity, and weight gain have been implicated as major contributors to coronary artery disease, hypertension, and stroke. Many of the dietary characteristics that affect weight gain and obesity are also risk factors for cardiovascular disease, and some programs seek to intervene to address both issues simultaneously. For example, high-fat meals are less satiating than are high-carbohydrate meals (Golay & Bobbioni 1997), and high-fat diets may be associated with greater overall food consumption and weight gain than are high-carbohydrate diets. In addition, people who are or have been obese do not appear to adapt appropriately to dietary fat and respond to it with increased fat storage (Golay & Bobbioni 1997). Consumption of fat affects eating behaviors as well as metabolism of food, weight gain, and cardiovascular risk in several different ways.

Interventions to prevent obesity and help people to lose weight are successful in achieving short-term benefits, but show more complex and poorer outcomes over longer periods (Wing 1995b). Some are very successful as long as the intervention is maintained and participants comply (Metz et al 1997). Interventions targeting specific ethnic or socioeconomic groups and recognizing cross-group differences in diet and disease risk have also had some success in changing dietary behaviors (e.g. Barry & Wassenaar 1996, Fitzgibbon et al 1996, Stolley & Fitzgibbon 1997).

DIET AND CANCER The literature on the impact of diet and overall weight on the etiology and progression of cancer is more speculative and difficult to evaluate than is research on diet and heart or vascular disease. Nonetheless, research suggests that careful dietary management may help to prevent or control cancers. The literature on the impact of vitamins, foods, and natural products on cancer is growing rapidly.

Data provide some support for the possibility that what people eat affects their risk of developing cancer, but findings are mixed. For example, high-fat/low-fiber diets are associated with mammography results indicating high risk of breast cancer, while lower-fat/high-fiber diets are associated with much

lower risk mammography profiles (Nordevang et al 1993). However, research has also failed to find associations between fat or fiber intake and cancer-relevant outcomes (Negri et al 1996). Estrogen levels are associated with fat consumption, and dietary fat has been associated with recurrence of breast cancer among women with estrogen-receptor–positive tumors (Longcope et al 1987, Holm et al 1993). These findings are provocative, but more definitive evidence of these relationships must await the results of several ongoing trials.

As with interventions to reduce risk of cardiovascular disease, efforts to manage cancer risk through diet have achieved significant changes in diet. Randomized trials seeking to decrease the percentage of caloric intake from fat have shown that people can modify their diet and achieve recommended or target levels of fat intake or weight loss (e.g. Heber et al 1992, Meyskens et al 1985, Schapira et al 1991). Similar efforts have been linked to increased consumption of healthy foods and fiber (Domel et al 1993, Atwood et al 1992). Attitudes, knowledge, or beliefs about associations between diet and disease can also be manipulated to help alter patterns of food consumption. People who believe that diet and cancer are linked, who know what recommendations to follow, and who believe that others support modification of diet to reduce cancer risk are more likely to make healthy changes in diet than people who do not (Patterson et al 1996). Among people considering their risk of cancer, it appears that many adults are willing and able to modify diet when there is clear evidence for it.

STRESS AND DIET Stress is thought to affect diet and weight at several different levels. Negative mood may lead people to eat more and may result in their seeking "comfort foods" or foods that make them feel better. Most of these foods are relatively high in fat and salt or sugar, meaning that stress may increase consumption of less healthy fatty, salty, or sweet foods. These effects can be traced at any of a number of levels, as in stress-related enhancement of metabolic rate, stress-related increases in physical activity or in time pressures and busy schedules that can increase consumption of fast or convenience foods. Enhanced metabolic demand during stress may increase consumption of food without necessarily affecting weight.

The literature on stress and eating behavior, weight gain, and obesity is complicated and focused most intently on acute stress-eating relationships (e.g. Greeno & Wing 1994). The relationship between stress and eating is complicated by personality or behavioral factors that qualify stress-related changes in food consumption, and thoughtful analysis of chronic stress and weight or diet changes have not been evaluated. Systematic examination of these and other possibilities will clarify the relationship between stress and diet and should help to design programs that more effectively manage weight and risk of disease.

Exercise

Exercise appears to be important as a means of managing weight, managing stress, and modifying the impact of stress- or other-induced disequilibrium. Regular exercise alters endocrine activity, circulatory function, muscle tone, and a number of other aspects of physical functioning. As a result, some risks for disease may be altered. Its influence on cardiovascular risk is well established and is not discussed here. Exercise is also very important in weight control and prevention of obesity. Some of the mechanisms linking exercise to these health outcomes have been identified, but many are not well characterized.

EXERCISE AND CANCER Results of several large population studies of cancer risk indicate that exercise decreases the relative risk of developing cancer (e.g. Francis 1996, Shepard 1993, Thune et al 1997). Sedentary activities appear to increase the risk for colon cancer (Shephard 1993), and evidence suggests that breast cancer and some reproductive cancers in women are negatively correlated with exercise history, although these findings are mixed (Bernstein et al 1994, Friedenreich & Rohan 1995, Kramer & Wells 1996, Mink et al 1996, Paffenbarger et al 1987, Thune et al 1997, Vena et al 1987). Adult weight gain also appears to contribute to risk for breast cancer (e.g. Huang et al 1997). Systematic evidence of links between exercise and cancer will await determination of the extent to which exercise suppresses risk factors, reduces stress, and/or increases the elasticity and adaptability of the organism.

STRESS AND EXERCISE Exercise has also received attention as a coping strategy or means of dealing with stress (e.g. Rostad & Long 1996, Perna et al 1997). Evidence of psychological benefits of exercise would suggest another layer of influence on health; in addition to fitness benefits, exercise may be related to mood and to perceived stress. Some studies support these possibilities, and many suggest that regular exercise has psychological and emotional benefits (Leith & Taylor 1990, Plante & Rodin 1990). For example, regular exercise appears to attenuate physiological reactivity to stressors in the laboratory (Anshel 1996, Holmes & Roth 1988). Mood effects and perceived control may also be associated with exercise and may help buffer stress. Ironically, this form of coping may be one of the first casualties of stress; although regular exercise may convey benefits, research suggests that stress reduces physical activity (Steptoe et al 1996).

Cigarette Smoking and Tobacco Use

Tobacco use is a primary cause of premature mortality and a modifiable risk for many debilitating or fatal diseases. Smoking tobacco, using snuff, and ingesting other smokeless tobacco have become pervasive around the world.

The primary active ingredient in tobacco is nicotine, which has stimulant properties that appear to increase SNS arousal and alertness and to reduce appetite. Once tobacco use is established as a habit, it is highly resistant to change. Among tobacco users, the relief from withdrawal, appetite suppression, arousal, and sensation of well being associated with tobacco make it a very desirable behavior (Kassel 1997, Parrott et al 1996).

Smoking and other forms of tobacco use are major contributors to heart disease, hypertension, stroke, cancer, and several serious diseases of the lungs and airways. These effects are generated by a combination of constituents and smoke associated with smoking tobacco as well as by tobacco itself. Tobacco use also affects endocrine and immune system activity (e.g. Canals et al 1997, Mol et al 1993). Direct effects of tobacco use underlie its broad negative impact on health (e.g. Lykkesfeldt et al 1997). Passive exposure to tobacco smoke is also a problem, and research suggests that it has many effects consistent with exposure to nicotine (Hausberg et al 1997).

Because of the difficulty in getting people to stop using tobacco once they have started, considerable attention has been paid to prevention, particularly with children and adolescents (e.g. Chassin et al 1997). Risk factors for smoking vary by culture, but in the United States, adolescent cigarette smoking has been associated with perceived availability of cigarettes, cost, social models, social pressures, and mental health (Robinson et al 1997, Milberger et al 1997, Wang & Chou 1996, Unger et al 1997). The combination of social pressures and immediate reinforcers may induce people to try smoking when they do not intend to smoke (Engels et al 1997). Prevention efforts are most effective when continued for long periods (Eckhardt et al 1997), but a recent meta-analysis suggests that the average effect of social programs is small (Rooney & Murray 1996). Stress is one cause of tobacco use. Smoking and tobacco use appear to reduce stress or ameliorate its aversive effects (e.g. Viinamaki et al 1997, Todd et al 1996, Naquin & Gilbert 1996, Ogden & Mitandabari 1997). Smoking also alters acute stress responses. Smokers exhibit greater increases in peripheral resistence than nonsmokers during challenge (Girdler et al 1997). The synergistic effects of stress and smoking may heighten the negative effects of smoking and increase the incidence of tobacco-related illnesses (Maser 1997). Stress increases the amount and frequency of tobacco use (e.g. Steptoe et al 1996, Acierno et al 1996, Beckham et al 1996).

Stress also appears to be a major cause of relapse after cessation and often leads to resumption of smoking (Shiffman et al 1996, Matheny & Weatherman 1998). Newer approaches to cessation and relapse prevention hold some promise for success, appropriate motivation, and pharmacological adjuncts, while addressing individual level of addiction and recognition of barriers and supports (Lichtenstein & Glasgow 1997). Consideration of openness to change may also increase the efficacy of interventions by matching people to all ap-

propriate intervention and fashioning changes in pre-contemplation behavior (e.g. Parrott et al 1996).

Sun Protection and Skin Cancer Prevention

Another behavior associated with serious health consequences is sun exposure. The majority of skin cancers are caused by exposure to ultraviolet (UV) radiation in sunlight. Cumulative lifetime exposure to sun is associated with basal-cell and squamous-cell cancers. More serious melanomas are more likely to be associated with intermittent but intense exposure (infrequent or periodic sunburn) (Albino et al 1997, Balch et al 1997). Use of sunscreens that block UV radiation or more prudent exposure (e.g. sitting in the shade, covering more of the body with clothing) should reduce or eliminate most skin cancers. Estimates range up to 78% of lifetime basal-cell and squamous-cell skin cancers that could be prevented by regular use of sunscreens rated at SPF 15 or greater during childhood and adolescence (Stern et al 1986).

Efforts have been made to increase knowledge about the risks of sun exposure and skin cancer, but the results have not been encouraging. Surveys suggest that fewer than half of sunbathers use sunscreen, and that of those who do, fewer than half use sunscreens that convey adequate protection (Bak et al 1992, Banks et al 1992). Educational interventions have increased awareness and knowledge of skin cancers, but there is little evidence of associated behavior change (Katz & Jernigan 1991, Mermelstein & Riesenberg 1992, Hughes et al 1993). Beliefs persist that tanning makes people look healthy and more attractive, that exposure to the sun is good for health, and that the risks of skin cancer are outweighed by these more immediate factors (e.g. Baum & Cohen 1998, Grob et al 1993, Maducdoc et al 1992, Miller et al 1990, Keesling & Friedman 1987). Some intensive intervention programs have produced evidence of short-term behavior change (Girgis et al 1993, Lombard et al 1991). However, long-term behavior change is usually not evaluated and there is little evidence that these programs achieve measurable reductions in skin cancer morbidity (Baum & Cohen 1998).

HEALTH BEHAVIOR

A third general mechanism linking behavior and health is the behavior that characterizes how people behave when they are ill, suspect they are ill, or learn they are at risk for serious illness. Early detection of disease is a critical element of health care because identification and treatment of early disease is usually more effective. Even more basic are prevention behaviors that help people manage modifiable risk for disease. Prevention behaviors include proper diet and exercise, cessation of smoking, and elimination of other

health-impairing behaviors. Genetic risk testing programs for an increasing number of diseases reflect important public health advances and suggest immediate implications for health psychology (C Lerman 1998). Increasingly precise risk estimates of disease are attractive from a number of perspectives but also may have far-reaching psychological, social, and economic consequences that need to be studied (Baum et al 1997). Screening and surveillance have received a great deal of attention as means of early detection, and success in achieving good screening rates varies considerably. In general, there are a number of barriers to such behavior, including stress, social or support-related factors, and the perceived risk and emotional reaction to this perception (Rimer et al 1998).

Screening and Surveillance

Research has addressed screening and surveillance for many diseases and disease risks and has had some success in reducing cholesterol and blood pressure in people at risk for heart disease or hypertension (Heath et al 1995, Hoffmeister et al 1996). One of the most extensively studied diseases for which screening is important is breast cancer. Women are encouraged to have regular mammography and to perform monthly breast self-examination (American Cancer Society 1997), but many do not follow this recommendation. Socioeconomic factors, lack of physician support, perceptions of risk, and emotional reactions to the knowledge of being at risk appear to affect appropriate surveillance (e.g. Calle et al 1993, Fox et al 1991, Costanza et al 1992, Aiken et al 1994). Health beliefs are also associated with surveillance (e.g. Aiken et al 1994), but the focus of research has shifted from health beliefs to the emotional reaction that perceptions of risk may bring, including distress and worry about disease. Several studies suggest that women at higher risk for breast cancer experience heightened distress (e.g. Lerman et al 1994, Valdimarsdottir et al 1995, Gilbar 1997, Kash et al 1992). Women also report elevated anxiety about developing breast cancer and experience intrusive thoughts about their worries (e.g. Lerman et al 1993). Intrusive thoughts about breast cancer were also reported by women undergoing genetic counseling for breast cancer (Lloyd et al 1996). Some studies suggest that distress increases surveillance (e.g. McCaul et al 1996). Studies also suggest the opposite, that distress decreases surveillance activities (e.g. Kash et al 1992). Risk and distress have also been associated with overadherence to surveillance activities (e.g. Epstein et al 1997), but in some studies, distress is not related to surveillance at all (e.g. Sutton et al 1995).

A different kind of surveillance is testing for the human immunodeficiency virus (HIV). Testing for HIV indicates the presence of antibodies to the virus that can ultimately result in AIDS. The decision to undergo HIV screening is not universal, and many patients receive their first test near end-stage disease

(Wortley et al 1995). In one study, almost half of the HIV patients indicated that they waited to be tested for a year or more after first suspecting that they were HIV-positive (Wenger et al 1994). Again, emotional responses may be implicated. Studies have linked AIDS-related anxiety to not getting screened (e.g. Wilson et al 1996), undergoing screening (Stehlow & Kampmann 1993), and failure to return for test results (Bell et al 1997). Other factors also affect decisions regarding HIV testing (Myers et al 1993), but stress and emotional responses may be major factors in this decision.

There have been several interventions to increase knowledge about HIV risk reduction and adoption of less risky behaviors. Some have been encouraging, achieving population-level reductions in rates of risky behavior (Kelly et al 1997). However, results are not always consistent; one study with women at risk has shown some success initially and at three-month follow-up (e.g. Carey et al 1997). Another intervention in men at risk failed to show maintenance of these changes (Kalichman et al 1997). Culturally targeted interventions were also successful (Kalichman et al 1993). Past behavior was often a better predictor of future behavior than was the counseling and negative test result (Ickovics et al 1994).

Adherence

Adherence refers to the extent to which people comply with medical advice or prescription for treatment or surveillance (Haynes et al 1979). Nonadherence is a major problem when treating patients or seeking to prevent or detect disease states early, in part because care providers rarely know whether patients are being compliant.

Nonadherence also complicates treatment by affecting outcomes and may lead to errors in subsequent diagnoses (Becker & Maiman 1980). Successful adherence involves remembering to do something, doing it properly, and being motivated to continue. Unintentional noncompliance reflects adherence problems that result from patients' inadequate understanding of the condition, treatment, or prevention regimen prescribed (e.g. Hussey & Gilliland 1989). Forgetting or misunderstanding instructions or explanations are major sources of adherence problems. Other kinds of noncompliance are more intentional and often indicate attempts to find an alternative treatment (Cameron 1996). Satisfaction, health beliefs, naive theories of illness, preferences for health care, and other factors influence adherence as well (Claydon & Efron 1994, Brownell & Cohen 1995, Morris & Schulz 1993). The costs and consequences of misdiagnosis or failure to detect readily treatable disease associated with noncompliance underscore the importance of this aspect of health behavior.

Research has sought to identify personality or demographic predictors of adherence. Identification of likely noncompliers, for example, would facilitate selective intervention and increase adherence. However, this effort has not

produced robust predictors of compliance or of an "adherent style." The quality of provider-patient interaction and behavior of health care providers in these interactions is related to adherence (Cameron 1996). High-quality interactions, sensitivity to patient concerns, and other aspects of care provision predict better adherence (DiMatteo & DiNicola 1981). Supervision is related to better adherence as well, and increasing supervision of health behaviors or outcomes or reminders to comply are effective means of increasing competence (Haynes et al 1976, Schapira et al 1992). Social support and health beliefs also affect willingness to comply and may influence the extent and accuracy of the understanding of a recommendation, health threat, or health communication (Cameron 1996). Stress interferes with adherence by increasing memory problems, decreasing satisfaction, or altering receptiveness and capacity to adjust to treatment demands (e.g. Brickman et al 1996).

CONCLUSIONS

Research increasingly suggests a strong link between how people think, feel, and behave and how well they withstand illness and poor health. Stress provides one model for understanding and predicting the impact of more specific emotional arousal and distress. The unique interactions of nonspecific stress responses and more specific emotional changes associated with anger, sadness, uncertainty, or other psychological states are not known. However, the behavioral implications of mood are well recognized and behaviors tied to these states, including self-medication with food, alcohol, or drugs, are important aspects of health. Interest has gradually shifted from confirmation of links between psychosocial or behavioral factors and health outcomes to investigation of mechanisms by which health benefits or harm are conveyed. Whether the negative influences of emotional arousal, health-impairing behaviors, and ineffective or disrupted illness behaviors can be managed or minimized is a major question that will be addressed next, and the continued integration of this knowledge into health care practice and prevention/treatment of disease should contribute to better medical outcomes. Stress management, enhanced coping, and reduction of modifiable risk for disease associated with harmful behaviors have already been targeted. A broad approach—one that considers these factors in the context of genetic variables, environmental constraints, and health-protective resources and behaviors— seems most likely to succeed.

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