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HEALTH PSYCHOLOGY: Why Do Some People Get Sick and Some Stay Well?

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INTRODUCTION

The biomedical community has increasingly recognized the importance of psychological factors in the natural history of disease, prevention of disability and illness, and promotion of recovery. In *Health and Behavior Research* (USDHHS 1991), a report on behavioral research by the National Institutes of Health (NIH), the NIH director indicates, "Our research is teaching us that many common diseases can be prevented, and others can be postponed or well-controlled, simply by making positive life style changes. For these reasons, intensifying such research and encouraging all Americans to make health-enhancing behaviors a part of their daily lives has taken on more and more importance in our efforts to conquer disease" (p. 1). Likewise, *Healthy People 2000* (USDHHS 1991), a report of health promotion and disease prevention objectives for the United States population, notes that "achievement of the agenda depends heavily on changes in human behavior" (p. 8).

Similarly, the psychology community has increasingly embraced questions of essential importance to physical health. Concepts that originated in relation to health problems have become topics of interest themselves. Theories that were developed to account for mental health (e.g. attributional style and depression, or coping with stress and related interventions) are being used to understand and promote good physical health.

As a result, health psychology no longer has clearly demarcated boundaries. Examining the contents of four American Psychological Association (APA) journals that span general psychology and large subdisciplines (*American Psychologist, Psychological Bulletin, Journal of Personality and Social Psychology,* and *Journal of Consulting and Clinical Psychology*), we found that over one third of the articles in 1990–1992 either examined physical health issues or involved concepts from health psychology such as stress or Type A. Specialized journals have also prospered. Division 38 of the APA established *Health Psychology* in 1982, with its almost 1700 members receiving the journal. In 1993, over 8200 APA members received the journal, which is jointly published by Division 38 and the APA. Articles relevant to health psychology also appear in journals in several other fields, including many medical journals.

Although the domain of health psychology is broad and the enormity of its knowledge base is daunting, many of the new developments and concepts in the past five years pertain to three essential questions: First, who becomes sick and why? Second, among the sick, who recovers and why? Third, how can illness be prevented or recovery be promoted? Given the explosion of re-

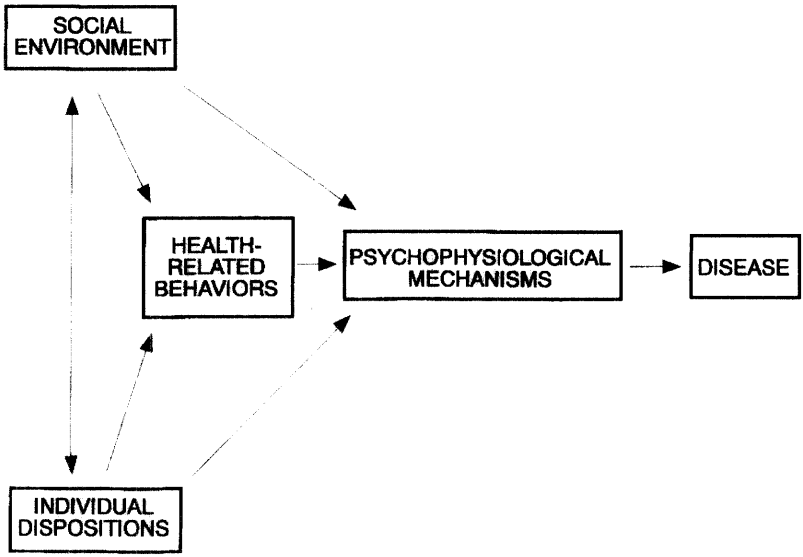

Figure 1 The organization of this review.

search, which precludes a complete review, we had a difficult choice about which question to address. We chose to review the first question—the psychological characteristics of individuals prone to disease and underlying mechanisms accounting for the associations between psychological variables and disease—because it is the basis for the other questions and suggests possibilities for prevention. We applied strict criteria, limiting ourselves whenever possible to longitudinal studies with adequate sample sizes to test predictions about the contribution of psychological variables to the onset of disease. We have excluded for the most part (*a*) cross-sectional studies because of difficulties interpreting cause and effect and (*b*) studies of patients because of biases owing to diagnosis and treatment. More importantly, the latter studies pertain more to the psychological factors in recovery and treatment than to those in the early etiology of disease. We have included some studies of mortality because it is a hard outcome, although we realize that associations between psychosocial factors and mortality could reflect recovery processes as well as etiologic factors.

In this review, we examine two broad domains of variables affecting the onset of disease: factors residing in the social environment and factors residing in the individual (see Figure 1). The double-headed arrow between the two domains signifies the importance of the person-environment interaction. For

example, dispositions may predispose individuals to be susceptible to specific environmental elicitors of clinical episodes or dispositions may affect the likelihood that individuals will be exposed to situations to which they are vulnerable. Variables in the environmental and individual domains may affect disease onset through physiological mechanisms and/or through health behaviors that in turn affect these mechanisms.

SOCIAL ENVIRONMENT AS CONTRIBUTOR TO DISEASE

Stress

Individuals who experience stress may be more susceptible to disease. The evidence is strongest for cardiovascular disease, infectious disease, and pregnancy complications. Prospective evidence for the role of stress in the etiology of cancer and endocrine diseases, such as diabetes, thyroid disorder, and Cushings disease, is not substantial (Cox & Gonder-Frederick 1992, Beardsley & Goldstein 1993). Additionally, evidence is stronger for studies using subjective evaluations of stress than for those using other measures. The latter is consistent with the more recent emphasis on contextual, subjective components of stress, including appraisal processes (Lazarus & Folkman 1984).

CARDIOVASCULAR DISEASE Rosengren et al (1991) followed 2000 men with no prior history of myocardial infarction (MI) for 12 years. Those men who reported substantial stress in the previous 1 to 5 years at the initial interview were more likely to experience coronary artery disease over the next 12 years compared to those who indicated no stress or only sporadic instances. Men reporting more stress also reported more adverse health behaviors such as smoking, alcohol abuse, and lack of physical exercise, but disease risk associated with heightened stress was only slightly reduced when these behaviors were controlled for. Severe stress also predicted subsequent risk of stroke (Harmsen et al 1990).

In contrast, several studies failed to find a link between life events and subsequent mortality. In the Multiple Risk Factors Intervention Trial (MRFIT), over 12,000 men who showed no signs of coronary heart disease (CHD) but who were at high risk were followed for 6 years. Although life events predicted angina, neither death due to CHD nor fatal or non-fatal MI was affected by the experience of life events in general or of life events that were clearly undesirable (Hollis et al 1990). The most severe life event, death of a loved one, was unrelated to subsequent mortality in two studies. Levav et al (1988) found that mortality rates over ten years were not significantly higher among parents in Israel who had lost a son either in war or in an accident than in the general population. In a United States population, women whose part-

ners died during the five years in which they participated in a health study showed no higher mortality than age-matched controls (Avis et al 1991).

Research on the health effects of stress has frequently focused on stresses in the work environment, much of it stimulated by Karasek's (1979) model of job strain. The model posits that jobs involving a combination of high demand (e.g. time pressure) and low control (e.g. little latitude over decisions and choice of skills to use) engender mental and physical health problems. Initial studies used individuals' own ratings of their jobs, but such ratings may be influenced by the individual's dispositions, which may themselves influence health outcomes. Karasek et al (1988) developed a system for categorizing occupations, and cross-sectional analyses of two large surveys found that men in high stress occupations had an increased prevalence of MI.

Several studies have established a prospective link between job strain and CHD. Siegrist et al (1990) examined both workers' self-reports of their work environment and scores based on job descriptions for a sample of male, blue-collar workers in Germany. Both types of variables predicted risk of CHD over the next six and a half years. Independent of the association with medical risk factors (e.g. blood lipids and systolic blood pressure), CHD risk increased with high levels of one external indicator of job strain (discrepancy between occupational grades of individuals and their educational levels) and three self-rated measures (low perceived job security, high perceived work pressure, and a vigilant coping strategy). Effects of job strain on risk of CHD were minimal at low medical risk factor levels. However, among individuals at higher medical risk, men in low reward/high effort jobs showed a substantial risk of disease; men in jobs with only one or the other characteristic showed a moderate CHD rate, and those with neither indicator of job strain had little risk. In a subsequent analysis of this data set, Siegrist et al (1992) broadened the set of cardiovascular diseases to include stroke. Biomedical and psychosocial risk factors accounted for almost 85% of the risk of cardiovascular disease. At each level of biomedical risk, the probability of a cardiovascular event increased as job strain indicators increased.

Job strain predicted health problems in two other studies. Among employees of a metal fabrication plant in Finland, job strain, indexed by lack of variety and control and high physical strain, was associated with CHD over a ten-year period. The association remained significant when age, sex and risk factors (e.g. smoking, blood pressure) were controlled (Haan 1988). In a community sample in Sweden, men who had recently retired from high strain jobs (high demand and low scheduling latitude) had a significantly higher relative risk of mortality in the subsequent six years (Falk et al 1992).

Disconfirming evidence of the role of job strain in CHD emerged in a study of Hawaiian men of Japanese ancestry (Reed et al 1989). Over 4000 men, who were in the same occupation at follow-up that they had earlier reported as their

usual occupation, were assigned job strain scores based on occupation ratings. Risk of developing CHD over 18 years was not associated with either the demand or the control dimension of their occupation. Men in high job strain occupations, surprisingly, had the lowest incidence rates of disease, although the rates were not significantly lower.

Research on job strain as a contributor to cardiovascular disease is promising, but several important questions remain. First, it is not clear whether classification of occupations sufficiently captures individual experiences. An advantage of classification ratings is that they can be applied to data sets lacking information on individual perceptions. Also, these ratings are uncontaminated by traits of individuals that may affect perceptions of their jobs, and that may also be linked to disease risk. Further work is needed to clarify relationships among job stress as determined by occupational ratings, organizations' descriptions of specific jobs, and workers self-ratings, and the relationship of these to health outcomes. A second question concerns the generalizability of findings. Most of the studies have used male subjects, and many have been done in Scandinavia and other parts of Europe. It is not clear if the measures capture pathogenic aspects of work settings for women and for different national and ethnic groups.

INFECTIOUS DISEASES Stress has also been linked to increased susceptibility to infectious diseases. In one study, medical students reported more instances of infectious disease during exam periods than in comparison months preceding these periods (Glaser et al 1987). Immune indicators such as a reduction in interferons and increased lymphocyte antibodies to the Epstein-Barr virus, suggesting reduced control of the virus at the cellular immune level, also changed in the exam periods. In a sample of married couples keeping daily diaries, undesirable events increased and positive events decreased three to four days before the onset of symptoms of infectious disease (Stone et al 1987). Positive and negative events reported in the two days immediately preceding symptom onset did not differ from average levels, suggesting that reported events were not responsive to early stages of infection, but rather, that the increase in stress contributed to infection risk.

Viral challenge studies, which follow volunteers who are experimentally exposed to viruses, rule out differential exposure to viruses as a potential alternative explanation for the link between stress and infection. These studies also provide clinical verification of infection. Stone et al (1992) exposed subjects to a rhinovirus and observed them for five days. Subjects who developed a clinical cold following the viral challenge had reported a greater number of both positive and negative life events in the prior year. Neither perceived stress nor mood at time of induction related to risk of subsequent cold,

which is surprising given the prior study's findings that life events at the time of exposure might lower resistance to infection (Stone et al 1987).

Using a similar protocol to Stone et al's (1987), in which healthy volunteers were assessed and then exposed to one of five respiratory viruses, Cohen et al (1991) found a linear relationship between a stress index encompassing negative life events, negative affect, and perceived stress and the probability of developing a clinical cold following viral challenge. The association held across all five viruses and after controlling for health practices (e.g. smoking, diet, sleep). Subsequent analyses (Cohen et al 1993) showed that life events related differently to biological mediators than did perceived stress or affect. Perceived stress and negative affect predicted infection following viral exposure (determined from culture or increases in antibodies), whereas life events predicted who, among those infected, developed a clinical cold. These findings suggest that aspects of stress may play a different role at different stages of disease onset, and indicate the need for better specification of the stress process and its impact on the body.

PREGNANCY The effect of stress on pathological processes can also be seen in research on pregnancy outcomes such as preterm delivery, birth weight, and Apgar scores. The latter are standardized ratings of newborn functioning encompassing heart rate, respiratory effort, reflex response, muscle tone, and color. Pagel et al (1990) found that women who, in their third trimester, reported more life events in the year before pregnancy, had babies with lower birth weights and lower Apgar scores than those born to women experiencing fewer events. No association with life events during pregnancy was found. Williamson et al (1989) found that although life events during the year before pregnancy were unrelated to obstetrical outcome, an increase in life events from the second to third trimester predicted adverse outcomes (e.g. neonatal death, low 5-minute Apgar, low birth weight). Norbeck & Anderson (1989) and McCormick et al (1990) found no association of life events with obstetrical outcome.

Other studies, incorporating subjective reports, show more consistent results. Lobel et al (1992) found that a latent stress factor, incorporating anxiety, perceived stress, and the impact of life events, predicted both timing of delivery and infant birth weight in a low socioeconomic status sample. Medical risk factors contributed independently to birth outcomes and did not reduce the association of stress and outcome. The experience of adverse working conditions also appears to contribute to complications. Vartiainen (1990) studied women in Finland before conception and followed them throughout their pregnancies. Women who reported having a psychologically stressful job had a higher risk of delivering infants with low Apgar scores; measures of life events were not related to outcome. Among women participating in the National Longitudinal Survey of Labor Market Experience, a significant associa-

tion of job strain and risk of delivering a low birth weight, preterm infant became nonsignificant in the sample as a whole after controls were entered for education, degree of physical exertion in the job, and health-related behaviors. However, the association remained significant for the subset of women who reported that they didn't want to remain in the work force. These women may have been particularly affected by adverse job characteristics (Homer et al 1990).

Social Connections

The degree to which an individual is connected to others may also influence health. One's degree of social connection has been reflected in measures of social isolation, number of individuals in the social network, and social integration [i.e. involvement in clubs, churches, and other organizations (House et al 1988)], and in measures of perceived support from others, including the type and degree of support (Cohen 1988).

MORBIDITY AND MORTALITY Many studies of social connections are from Scandinavia and use mortality as the outcome. In a sample of elderly Swedish men, Hanson et al (1989) found that social network and social support indicators related independently to mortality. Men who participated less in formal and informal groups, those who reported less emotional support, and those who were unmarried were at greater risk of dying over a five-year period. In a community sample of middle-aged healthy Swedish men, those who were lower on social integration were significantly more likely to suffer a heart attack during a six-year follow-up; those low on social support were also at increased risk but the difference was of borderline statistical significance (Orth-Gomer et al 1993). Social connections may interact with stress to affect health. Falk et al (1992) found mortality especially high among retired Swedish men who were low in social support and high on job strain.

Reynolds & Kaplan's (1990) study of a United States sample provides mixed evidence for the role of social connections in cancer onset. They examined the incidence of newly diagnosed cancer and mortality over a 17-year period in a sample of 6848 adults in Alameda County, California, as related to several aspects of social connection. For men, none of the measures predicted either cancer onset or mortality. For women, social isolation, specifically having fewer contacts with friends and relatives, predicted onset for total cancers across all sites. Lack of church membership and feeling socially isolated predicted increased rates of hormone-related cancers. Relative risk of mortality for all-site cancer was greater for women who scored high on social isolation, had fewer contacts with friends and relatives, and who felt isolated. Relative risk of mortality from hormone-related cancers was higher in women with fewer contacts and who felt isolated.

Vogt et al (1992) found stronger links between social connections and mortality than with onset for a variety of diseases in over 2600 members of a health maintenance organization. Individuals who had social networks of greater scope and size and who had more contacts within them showed lower mortality over 15 years even after adjusting for sociodemographic variables and health status at baseline. Network measures were unrelated, however, to subsequent incidence of cancer, stroke, or hypertension. Network scope (but not size or frequency of contact) significantly predicted relative risk of CHD. Using the same data set, Hibbard & Pope (1993) examined social support at work and in the marital role. Aspects of marriage were unrelated to morbidity or mortality for men, but women who were married and those who reported greater equality with their marriage partner in decision-making were less likely to die over the 15-year span. Women who reported greater social support at work were less likely to die and also had lower rates of stroke over the 15 years. Work stress, but not support, predicted CHD.

Social support during pregnancy appears to contribute to better obstetrical outcomes. Collins et al (1993) found that three aspects of social support measured during pregnancy predicted birth outcomes. Women who received more support and those who received higher quality support delivered babies with higher Apgar scores, and those with more support also had shorter labors; women who reported larger social networks delivered babies who were of higher birth weight. However, the beneficial effects of social support may differ by ethnicity. Norbeck & Anderson (1989) found that support from partners and from mothers contributed substantially to a reduction in complications and increased gestational age at birth in black women. However, among Hispanic women, outcome was unrelated to social support and among whites, social support from mothers was associated with more complications.

EXPERIMENTAL EVIDENCE Experimental studies that manipulate degree of support and show health advantages provide strong evidence for the role of social support. A recent analog study of social support (Kamarck et al 1990) suggests that social resources may reduce cardiovascular reactivity to stress, a possible risk for CHD (see below). In this study, women brought a friend with them to a laboratory experiment. The women either had the friend in the experimental room with them while they performed several challenging tasks and had their blood pressure and heart rate measured, or they had the friend wait in another room. The former group showed smaller cardiovascular responses to challenge than did the latter.

Kennell et al (1991) replicated in a United States sample earlier findings from Guatemala showing fewer birth complications for women randomly assigned a *doula,* a woman who uses talk and touch to aid a woman in labor (Sosa et al 1980, Klaus et al 1986). Kennell et al found that women assigned a

doula had better outcomes, including shorter labors, fewer caesarean sections, and fewer infants requiring extended hospitalization compared to women receiving standard care or to those assigned a passive observer who only monitored them through labor.

An intervention providing social support during pregnancy to high-risk, poor women in Latin America failed to show positive effects, however (Villar et al 1992). Pregnant women in the first trimester randomly assigned to receive additional visits from a nurse or social worker showed the same rates of complications as those assigned to standard care. The authors argue that the intervention was as intensive as could be expected in a public health effort in a developing country (Villar 1993), but it is not clear how well the support intervention functioned and whether a more intensive or meaningful effort would have shown results. For example, Reite & Boccia (1993) questioned the effectiveness of the social support because it was not provided by someone who had a personal relationship to the woman.

Further clarification is needed about the nature and functioning of social connections and social support. Additionally, it is not clear how support that is experimentally provided mirrors naturally occurring support; resolution of this question will both further our understanding of the function of social support and facilitate the development of more effective interventions.

DISPOSITIONS AS CONTRIBUTORS TO DISEASE

Type A and Hostility

Continuing the trend reported in the last *Annual Review of Psychology* chapter on this topic (Rodin & Salovey 1989), recent prospective studies do not indicate that Type A individuals (i.e. those who are competitive, achievement oriented, easily annoyed, and time urgent) are more likely to be at risk for CHD mortality and MI than their Type B counterparts (Eaker et al 1989, Eaker et al 1992, Matthews 1988, Orth-Gomer & Unden 1990). Moreover, in the MRFIT sample, Type As who reported high levels of life events generally, or loss events specifically, were not at higher risk for CHD mortality or MI than other groups (Hollis et al 1990). Newly recognized is that Type A may be a risk factor for poor health-related quality of life, including chest pain (Eaker et al 1989), general health problems (Shoham-Yakubovich et al 1988), and injuries, especially among Type As that colleagues rated low in amicability (Lee et al 1989).

Type A is a multidimensional concept, and efforts to disentangle coronary-prone and noncoronary-prone components have, with few exceptions, pointed to the importance of hostility, anger, and anger expression in the etiology of CHD. Clinical ratings of potential for hostility based on interview responses

were significant predictors of CHD morbidity and mortality in the MRFIT and Western Collaborative Group Study (WCGS) data (Dembroski et al 1989, Hecker et al 1988, Houston et al 1992). The Cook-Medley scores of hostile or cynical attitudes predicted CHD or total mortality in three of six prospective studies that Smith (1992) reviewed.

The aforementioned exceptions include a cluster analysis of WCGS participants who differ in patterns of Type A characteristics: hostile men and controlling, dominant men with little hostility were more prone to CHD (Houston et al 1992). In a sample of blue collar men, those who scored highly on combined dimensions of need for approval, competitiveness, impatience and irritability, and inability to stop working had a higher risk of coronary disease, while being hard driving and perfectionistic did not relate to CHD (Siegrist et al 1990).

Problems of measurement have contributed to the difficulty of estimating the true effect of hostility (or Type A). Measures of hostility and anger are heterogeneous and some overlap with dispositions that may be noncoronary-prone. For example, clinical ratings of potential for hostility have little or no association with Cook-Medley scores (Matthews et al 1992). Three of six factors from a rational analysis of the Cook-Medley scale predicted CHD: cynical attitudes, hostile affect, and aggressive behavior (Barefoot et al 1989). Reanalysis of the Western Electric Study suggested that cynical attitudes, a component of hostile attitudes as measured by the Cook-Medley scores, predicted all cause and CHD mortality, independent of neuroticism. Neuroticism, independent of cynicism, was unrelated to outcomes, except for alcohol-related deaths (Almada et al 1991). Health psychology would benefit from further development of valid measures of the major domains of hostility for use in large scale, prospective studies.

Depression, Distress, and Exhaustion

The concepts of depressive symptoms, distress, and vital exhaustion are all conceptualized as negative mood states, which are somewhat reliable across time, although affected by environmental stress. Below, we review these concepts in relation to risk for cancer and for cardiovascular disease or total mortality.

CANCER RISK Interest in the association of depression and cancer stems from a proposed connection between emotional distress and compromised immune function (see below) and an early prospective study in which Minnesota Multiphasic Personality Inventory (MMPI) depression scores predicted a two-fold increase in death from cancer (Shekelle et al 1981). Hahn & Petitti (1988) examined the incidence of breast cancer in almost 9000 women over 15–20 years. MMPI scale scores for depression and repression/sensitization were

similar in women with and without incident breast cancer; the lying score was higher in breast cancer patients. The National Health and Nutrition Examination Survey I cohort was administered the Center for Epidemiologic Studies Depression scale and the depression subscale from the General Well-being Schedule and were followed for 10 years. Neither measure was associated with cancer morbidity or mortality (Zonderman et al 1989). In the Alameda County Study of almost 7000 cancer-free persons, those who scored one standard deviation or above on an 18-item scale of depressive symptoms had similar rates of cancer morbidity and mortality to those with lower scores. However, depressed individuals were at higher risk for early mortality from all causes and from non-cancer related illnesses, which presumably are largely constituted by cardiovascular diseases (Kaplan & Reynolds 1988). It may be biologically naive to expect depression to play a major role in determining all cancers because pathogenic factors differ for different types of cancers.

CARDIOVASCULAR DISEASE RISK Evidence for the role of depression and psychological distress in the etiology of cardiovascular diseases, including hypertension, elevated blood pressure, MI, and cardiac death, is more substantial than it is for cancers.

Anxiety scores predicted change in women's blood pressure across three years controlling for age, parental history of hypertension, diet, obesity, and other predictors of blood pressure status (Markovitz et al 1991). Among middle-aged men in the Framingham Heart Study, anxiety scores predicted 20-year incidence of hypertension, independent of age, obesity, glucose intolerance, smoking, hematocrit, and alcohol intake (Markovitz et al 1993). Among women in that study, those who reported high levels of tension, low educational attainment, and no vacations were at heightened risk for MI or cardiac death (Eaker et al 1992).

In the Evans County study, high scores on the Health Opinion Survey, a measure of psychosomatic symptoms, predicted greater mortality over 12 years controlling for gender, race, and age, as well as preexisting disease and blood pressure (Somervell et al 1989). In a prospective longitudinal study of the elderly, a significant association of high levels of depressive symptoms and low frequency attendance at religious services with incidence of stroke over six years became nonsignificant when statistical controls for age, sex, housing, hypertension, diabetes, physical function, and smoking were entered (Colantonio et al 1992).

Appel and colleagues' concept of vital exhaustion is defined as a mental state characterized by unusual fatigue, a feeling of being dejected or defeated, and increased irritability. Excess fatigue and general malaise may be immediately precursory to a clinical event (i.e. within a few days or weeks). Recent data suggest that vital exhaustion might also be a long-term predictor.

In the Rotterdam Civil Servants Study of men, feelings of vital exhaustion predicted four-year incidence of angina and non-fatal MI, but not cardiac death. The effect for each individual year was not significant by the fourth year, however (Appels & Mulder 1989). In this sample and in the Kaunas-Rotterdam Intervention study, men who reported being burned out or exhausted at the end of the day were at higher risk for subsequent MI (Appels & Schoeten 1991, Appels & Otten 1992).

Interpretation of findings regarding vital exhaustion or distress more generally has been a matter of some debate. Some believe that exhaustion may simply reflect a poorly functioning heart muscle. Others suggest that feelings of mild depression, anxiety, and fatigue, when superimposed on an already compromised heart, may lower the threshold for triggering clinical events. Future studies on mechanisms using new ambulatory technologies (see below) will help disentangle these hypotheses.

Neuroticism and Negative Affectivity

In the last five years controversy emerged about the extent that neuroticism or negative affectivity contaminates relationships between correlated dispositions (e.g. hostile attitudes) and health outcomes. Self-reported health outcomes or diseases diagnosed largely by symptom reports are especially vulnerable to possible confounding. Neuroticism is defined as a broad dimension of individual differences characterized as the tendency to experience negative emotions, including anger, anxiety, and depression. Costa & McCrae (1987) reviewed data suggesting that although neuroticism scores are moderately correlated with chest pain and angina, they are unrelated to MI or cardiac death. Similarly, Watson & Pennebaker (1989) have demonstrated that the disposition of negative affectivity (i.e. persistent differences in the general negative affect level) leads to self-ratings of poor health, but is unrelated to biological markers such as elevated blood pressure or serum lipids in relatively healthy samples.

Our review focuses on studies of health outcomes that are less susceptible to reporting biases (e.g. occurrence of MI, stroke, or cardiac death). However, concern about how dispositions may relate to perceptions of health versus pathophysiological processes or objective clinical events is an important one. Experimentally-induced negative mood increases reports of physical symptoms (Salovey & Birnbaum 1989). Neurotic persons clearly report more stressful events and uncomfortable physical symptoms (Affleck et al 1992, Aldwin et al 1989) and magnify the effects of a given stressful event (Bolger 1990). On the other hand, data reviewed above demonstrate that specific negative emotions relate to specific health outcomes. Furthermore, perceived health predicts mortality, independent of biological risk factors (Kaplan & Comacho 1983), suggesting that self-report provides useful information not captured by biological markers. Finally, self-reported ill health and health-related quality

of life are important outcomes themselves because of their influence on utilization of services and the burden they impose on individuals and families (Weiner 1991). Such measures are used to evaluate the effectiveness of many clinical treatments (Kaplan 1988). Whether neuroticism should be viewed only as a nuisance variable, as a disposition that exacerbates the effects of environmental stress, or as a risk factor itself remains unresolved. In the meantime, it is premature to write off associations between psychosocial factors and self-reported health status because of possible contamination from neuroticism or negative affectivity.

Optimism, Explanatory Style, and Self-Esteem

Recent models of disease are emphasizing positive factors, especially those having to do with positive cognitions. Several overlapping concepts—optimistic expectations, optimistic explanatory style, self-efficacy, and high self-esteem—are important determinants of mental health (Scheier & Carver 1992, Taylor & Brown 1988); these may also contribute to physical health outcomes.

Measures of dispositional optimism (i.e. general positive expectations) predicted reports of few physical symptoms in relatively healthy populations (Smith et al 1989), active coping responses during stress (Aspinwall & Taylor 1992), as well as faster physical recovery and high quality of life after bypass surgery (Scheier et al 1989). A non-optimistic explanatory style—making internal, stable, and global attributions for negative events—predicted subsequent physician-diagnosed poor health in mid-life, controlling for initial health status, in a sample of male Harvard undergraduates (Peterson et al 1988). This style was also associated with lowered immunocompetence in elderly individuals (Kamen-Siegel et al 1991).

In contrast, Friedman et al (1993) found that bright children rated highly by parents and teachers on two items, good sense of humor and optimism/cheerfulness, died younger in adulthood. In addition, being conscientious was related to longevity, whereas high self-esteem was unrelated (as a main effect). High self-esteem, however, may interact with stress to affect physical symptoms of illness. On days following a stressful day, individuals with low self-esteem and poor social networks reported more health problems, such as flu, sore throat, and backaches (DeLongis et al 1988). After experiencing a positive life event, students with low self-esteem reported lower physical well being and used the student health center more often than those with high self-esteem (Brown & McGill 1989).

The concepts of optimism, explanatory style, and self-esteem appear to have promise as predictors of physical health, especially in individuals experiencing stressful events or distress. Self-efficacy, a more situation-specific variable, may affect health more through its impact on health-related behaviors (Bandura 1989).

HEALTH-RELATED BEHAVIORS

Health-related behaviors constitute a pathway by which environmental and dispositional variables affect physiological mechanisms and disease risk. In this section, we review evidence for linkage between these variables and health-related behaviors. We also discuss alternative ways of analyzing the role of behavior in disease risk and new issues in health behavior research.

Links with Stress and Support

Individuals under stress may find it more difficult to engage in health-promoting behaviors because of the emotional and behavioral demands of such behaviors. In addition, some individuals may offset stress-induced emotional distress by engaging in behaviors that have health-damaging consequences. Perkins & Grobe (1992) demonstrated that smokers subjected to a stressful task had an increased desire to smoke compared to those experiencing a non-stressful task. In a prospective study of individuals quitting smoking (Cohen & Lichenstein 1990), individuals who had decreasing levels of perceived stress over six months were more likely to remain abstinent over that period.

Social support's impact on health-related behaviors can occur in several ways. Insofar as individuals use health-damaging behaviors to cope with stress, and social support reduces the adverse effects of stress, individuals with more support in the context of stressful experiences may be less likely to engage in health-damaging behaviors. For example, Jennison (1992) reanalyzed several national surveys to examine the influence of recent loss on alcohol use among older adults. More losses (eg. death of spouse) were associated with greater excess use of alcohol. Social support buffered the effects of loss—the relationship of loss to drinking was reduced for those with more support.

Social connections can also provide specific support for engaging in health-promoting behaviors. Individuals who report receiving more support for given health-related behaviors (e.g. exercising, avoiding alcohol or tobacco while pregnant) are more likely to engage in those behaviors (Aaronson 1989, Treiber et al 1991). Zimmerman & Conner (1989) obtained reports of anticipated support among participants in a 7-week worksite program aimed at changing 4 cardiovascular risk behaviors. Anticipated support did not predict change. However, those who reported at follow-up having received more support from others for changing their behavior showed greater behavior change. The best prospective data come from a study of relapse following treatment for alcohol use, smoking, or opiate use. Individuals successfully completing treatment were followed once a week until relapse (for up to 12 weeks). At the first follow-up, indicators of general and of abstinence-specific social support were

obtained. Both types related to subsequent risk of relapse. Individuals who had a partner and those who reported greater social participation were less likely to relapse. There was no effect of general functional support but individuals who had more support specifically for abstinence were less likely to relapse (Havassy et al 1991).

Links with Dispositions

Dispositions, too, can affect health-related behaviors. For example, recent data suggest that hostile individuals experience more adverse changes in coronary risk factors over time, thereby contributing to their higher risk (Scherwitz et al 1992). Prospective studies beginning with adolescents and college students and following them for three to twenty years have shown that hostility and Type A behavior predict later smoking and alcohol use, as well as higher lipid levels and body mass index (Siegler et al 1992, Raikkonen & Keltikangas-Jarvenen 1991, Keltikangas-Jarvenen & Raikkonen 1989). Similarly, Anda et al (1990) found in cross-sectional analyses that individuals who were depressed were more likely to smoke. In a 9-year follow-up of these smokers, individuals who were initially depressed were 40% less likely to quit than were smokers who were not depressed.

Analytic Issues

Researchers frequently treat health-related behaviors and risk indicators (e.g. cholesterol level, which may reflect eating habits) as confounders rather than as mediators of relationships between dispositions or environmental variables and health. For example, studies of hostility and CHD test the association controlling for other CHD risk factors such as blood pressure, cigarette smoking, and physical activity. This approach may underestimate the true risk associated with hostility on CHD because, as noted above, hostility influences these risk factors. The same argument would hold for the impact of other variables such as stress and depression.

It may be most useful to use combinations of variables to predict health outcomes, which may not be predicted by a single variable. For example, Linkins & Comstock (1990) found that the combination of depression and smoking strongly predicted cancer incidence in a cohort of over 2000 individuals who were cancer-free at least 2–4 years into a follow-up period of 12 years. A similar interaction was observed by Grossarth-Maticek et al (1988), who found that individuals who felt distant from loved ones and who smoked were at higher risk for cancer. Rather than simply controlling for health-related behaviors and observing if the residual relationship of psychosocial variables with health remains significant, it will be more revealing to test for main effects and interactions of both types of variables.

Other Influences on Health-Related Behaviors

Studies linking stress, social support, and dispositions to health-related behaviors constitute a narrow cut through the vast literature on behavior and health. In the past five years, there has been increasing awareness of the critical role that behavior plays in risk of disease, generating a great deal of research on determinants and consequences of health-related behaviors.

Major shifts have occurred in some health-risk behaviors. Key examples are reductions in smoking prevalence (Lichtenstein & Glasgow 1992), which are credited along with other behavioral changes for a substantial drop in heart disease (USDHHS 1989), and reduction in unsafe sexual behaviors in high-risk gay male populations (Ekstrand & Coates 1990, Catania et al 1990). The processes by which these changes occurred are not fully known, but each involved shifts in social and community norms governing behavior. As a result, more attention is being paid to public health and social approaches to behavior change (Chesney 1993, Lichtenstein & Glasgow 1992). At the same time, research on individual determinants of health-related behaviors has flourished. The Health Belief Model (Rosenstock et al 1988), Theory of Reasoned Action and Theory of Planned Behavior (Ajzen & Fishbein 1980, Ajzen 1985), Self-Efficacy Theory (Bandura 1986), Protection Motivation Theory (Rogers 1983), Self-Regulation Theory (Leventhal et al 1984), and Transtheoretical Stages of Change Model (Prochaska & DiClemente 1984, Prochaska et al 1992) all have been used to elucidate the cognitive and affective factors associated with specific health-related behaviors including cigarette smoking, exercise, diet, breast self-examination, care-seeking, and sun exposure. Increasingly, researchers are combining and elaborating on the models to improve prediction of behavior (e.g. Seydel et al 1990, Boyd & Wandersman 1991, Aspinwall et al 1991, Weinstein 1993). There have been innovations in both individual and community approaches to behavior change. Research evaluating these interventions addresses our third question, regarding ways to prevent disease and promote recovery, but are unfortunately beyond the scope of this review.

Diverse studies on risk behavior point to the importance of placing the health implications of risk behaviors in the context of other costs and benefits of engaging in that behavior. Some models of health-related behavior include only elements that relate to the health risks and benefits of the target behavior. This may be appropriate for simple behaviors, such as obtaining a vaccination, but are not helpful for understanding ongoing lifestyle behaviors, such as eating habits, exercise, and sexual practices. When studies have assessed broader aspects of behavior, better prediction has been achieved. So, for example, studies of condom use have revealed that use is more affected by considerations of the physical and interpersonal aspects of use, including the impact on

the pleasure and spontaneity of sex, than by health concerns (Kegeles et al 1989, Hays et al 1990).

Findings that health concerns are generally not the predominant influence on health-risking behaviors has been used as evidence for the irrationality of human behavior. Adolescents have been characterized as being particularly irrational in their behavior patterns. However, empirical tests have shown that adolescents are no less rational than adults. Applications of rational models to adolescent decision-making show that adolescents are consistent in their reasoning and behavior after the salient set of beliefs is assessed (Adler et al 1990). Quadrel et al (1993) demonstrated that adolescents are no more biased in their estimates of vulnerability to adverse health outcomes than are their parents. These and the above findings suggest that interventions aimed at modifying perceived consequences of health-damaging behaviors will be more effective for adults and adolescents if they consider a full range of salient consequences.

New Health Risks

In recent years, the adverse consequences of some behaviors have received greater attention. With the spread of AIDS, there has been increasing concern about unsafe sexual practices. We know a lot about the behavioral epidemiology of the disease and many of the models of health-related behavior have been used to understand sexual risk behaviors. Currently, the only prevention for AIDS requires behavioral change. There has been increasing attention paid to new high-risk populations, including adolescents and women (Ickovics & Rodin 1992), and efforts at prevention span individual to community-level interventions (Coates 1990).

A second area of increasing interest is unintentional injury. Injuries are closely tied to behavior and are the primary cause of death for individuals under age 44 in the United States. Although relatively little empirical work has been done, some studies have examined contributors to injuries in children (Boyce & Sobolewski 1989, Horwitz et al 1988). This will be an increasingly important arena for research and intervention (Spielberger & Frank 1992).

Newly recognized as a potential risk are some health promotion efforts that may have unintended health-damaging consequences. A prominent example is dieting for weight reduction. It has generally been assumed that dieting is beneficial because short-term weight loss is accompanied by improvement in cardiovascular risk factors. However, weight loss is usually not maintained, thereby leading to repeated cycles of loss and regain of weight. Further, many dieters are unsuccessful even in the short run and feel a sense of loss of control and lowered sense of self-worth (Brownell 1991). Of special concern are epidemiological findings suggest that body-weight variability leads to heightened risk for CHD and all cause mortality (Lee & Paffenbarger 1992, Lissner

et al 1991). The robustness of these associations and their underlying mechanisms must await further research, but the possibility that unsuccessful dieting is hazardous clearly deserves careful consideration.

Another example of unintended negative consequences of health promotion activities comes from interventions to reduce blood cholesterol levels. Although low or lowered cholesterol levels are associated with reduced risk for CHD mortality and morbidity (Muldoon et al 1990, Rossouw et al 1990), they are also associated with increased risk of death from non-illness related causes, including accident, suicide, and homicide. Low or lowered cholesterol levels are associated with increased aggression and risk-taking behavior, which are thought to be related to altered central serotonergic function (Muldoon & Manuck, 1992), and may explain in part the association between non-illness related death and lowered cholesterol.

PHYSIOLOGICAL MECHANISMS

There has been an explosion of research on physiological mechanisms that may connect psychological variables to disease processes. Below, we review progress in the most active areas, cardiovascular reactivity to stress, and psychoneuroimmunology.

Cardiovascular Reactivity to Stress

Large and frequent increases in cardiovascular responses to stress have been proposed as a key mechanism relating Type A, hostility, and low social support with risk for hypertension and CHD. Considerable progress has been made in the conceptualization and measurement of cardiovascular responses to stress as an individual difference variable. Longitudinal studies ranging from two weeks to four years have demonstrated that the magnitude of blood pressure and heart rate responses to cognitive tasks are reliable over time (Manuck et al 1989b), even among children (Matthews et al 1990). Reliability is further increased if responses to laboratory tasks are statistically averaged (Kamarck et al 1992, Llabre et al 1988).

Recent improvements in noninvasive measures of cardiovascular function using impedance cardiography now allow reliable measurement of hemodynamic factors underlying observed stress-induced changes in blood pressure and heart rate [i.e. total peripheral resistance and cardiac output (Saab et al 1992)]. Based on these measures, individuals appear to fall into several types: (*a*) cardiac output responders, (*b*) total peripheral resistance responders, and (*c*) undifferentiated (Allen et al 1991, Kasprowicz et al 1990). It is not yet known if these types are reliable across time or if they are differentially useful for predicting disease.

The external validity of laboratory task–induced measures of cardiovascular responses has been of considerable interest. Many studies have examined the association between laboratory measures of responses during cognitive challenges and ambulatory measures of blood pressure. Although some studies report associations, especially if the ambulatory measures were taken during the workday (Fredrikson et al 1991), many have not (Pickering & Gerin 1990), leading some to question the validity of laboratory measures of stress responses. Part of the lack of correspondence between ambulatory and laboratory measures is the result of greater variability in posture, time of day, and activity level during ambulatory rather than laboratory measurement (Gellman et al 1990). Perhaps just as important is the greater variability of stress during ambulatory measures, which are typically averaged without regard to the occurrence of stress; laboratory measures are, by definition, averaged only during periods of stress. A recent analysis showed that individuals who exhibit large elevations in blood pressure during laboratory tasks also exhibit elevated ambulatory blood pressure levels, but only for those measures taken during times when participants simultaneously reported being under stress (Matthews et al 1992).

Additionally, most laboratory tasks are asocial [e.g. performing serial subtraction or reaction time tests (Smith et al 1989)]. Life stress is often interpersonal, and cardiovascular responses to laboratory stress and to ambulatory measures will not correlate if individuals differ in how they respond to asocial and social stress. Indeed, Ewart & Kolodner (1993) found that adolescents exhibiting large cardiovascular responses to a stressful interview had elevated ambulatory blood pressure throughout the schoolday; responses to asocial tasks were less predictive of ambulatory levels. An important direction for future research is to classify individuals not only by their responses to asocial stressors, but also to social stressors (Lassner et al 1993).

Increased attention has also been paid to influences of ethnicity, gender, and age on cardiovascular responses to stress. Accumulating data suggest that African-Americans, males, and older individuals exhibit larger blood pressure and total peripheral resistance responses to stress, whereas Caucasian-Americans, females, and younger individuals exhibit larger heart rate and cardiac output responses (e.g. Anderson 1989, Girdler et al 1990, Tischenkel et al 1989). These data are of particular interest because African-Americans, males, and older individuals are at greater risk for cardiovascular diseases.

Despite the above advances, longitudinal data linking cardiovascular responses to psychological stress and risk for cardiovascular disease remain sparse. Menkes et al (1989) followed a group of male medical students for 18–20 years for incidence of hypertension. Students who had exhibited an increase of 20 mm Hg or more in systolic blood pressure while immersing their hands in cold water were more likely to become hypertensive during the

follow-up period. This relationship was independent of baseline blood pressure, parental history of hypertension, age, and smoking status. Similar analyses for incidence of MI, sudden death, stroke, and other cardiovascular diseases showed no effect (Coresh et al 1992). In another study, third grade school children performed a video game while their blood pressure and heart rate were monitored. Those children who exhibited the largest stress-induced increases in blood pressure had the greatest increase in resting blood pressure 4 years later, independent of age, body mass index, and resting pressure and heart rate at study entry (Murphy et al 1992). Another study of children aged 7–18 at study entry and their parents showed that cardiovascular responses to serial subtraction, mirror image tracing, and isometric exercise predicted increases in resting pressure 7 years later, adjusted for age, body mass index, resting pressure at study entry, and length of follow-up, in men, women, and boys (Matthews et al 1993). Light et al (1992) reported that among a small subgroup of an initial sample of undergraduate men who were able to be followed, systolic blood pressure and heart rate responses to a reaction time task were related to subsequent blood pressure status 10–15 years later. Clearly, longitudinal studies of reactivity and disease are a pressing need in this area.

Advances in our understanding of psychosocial factors and coronary disease also have emerged from animal model research. Cynomolgus monkeys, when fed a North American diet, develop atherosclerosis that resembles that of humans. Social stress induced by periodic reorganization of housing accelerates atherosclerosis and endothelial dysfunction (paradoxical vasoconstriction) in the coronary arteries of male monkeys (Kaplan et al 1991, Williams et al 1991). Cross-sectional data show that dominant males under social stress and subordinate females (not ovariectomized), regardless of social stress, have the greatest atherosclerosis (Kaplan et al 1991). Perhaps most relevant to our review, male and female monkeys who exhibit the largest heart rate changes in response to threat of capture have the greatest atherosclerosis upon autopsy (Manuck et al 1989a).

Psychological stress may not only lead to exaggerated cardiovascular responses in susceptible individuals, but also to enhanced platelet aggregation, coronary vasoconstriction, plaque rupture, myocardial ischemia, and arrhythmias, all part of the pathogenic process leading to MI or sudden death (see Kamarck & Jennings 1991, Markovitz & Matthews 1991 for reviews). A particularly important series of studies has demonstrated that stress leads to transient myocardial ischemia that is often symptomless or silent in coronary patients (e.g. Rozanski et al 1988) and that those patients who exhibit the largest blood pressure response to mental stress are more likely to experience myocardial ischemia (Krantz et al 1991). The extent that dispositional and environmental factors influence pathophysiological processes other than exag-

gerated cardiovascular reactivity is largely unknown and is an important area for research.

Immune Competence

Lowered immune competence has been proposed as a key mechanism linking psychological variables with susceptibility to infectious diseases as well as with progression of cancer, AIDS, and other diseases. Ader & Cohen (1993) reviewed animal research on conditioning of the immune system. In this section, we briefly examine human research on psychological influences on the immune system.

Recent meta-analytic (Herbert & Cohen 1993b) and enumerative (Kiecolt-Glaser et al 1992) reviews clearly show that psychological stress lowers immune competence. More specifically, stress can lead to decreased proliferative response of lymphocytes to mitogens such as phytohemagglutin (PHA) and concanavalin A (Con A), as well as to natural killer (NK) cell activity; to fewer circulating B cells, T cells, helper T cells, suppressor/cytotoxic T cells, and large granular lymphocytes; and to increased herpesvirus antibody titers (thought to reflect poor immune competence).

Personal resources or attributes may also modulate the immune system. Increased in vitro immune competence has been found among persons reporting supportive networks (Baron et al 1990) and feelings of belonging (Kennedy et al 1990), and among nonhuman primates with high rates of affiliation during stress (Cohen et al 1992). A prospective study found that caregivers who reported less social support and greater distress at an initial interview showed significantly greater decrements in immune function over the next 13 months (Kiecolt-Glaser et al 1991). A meta-analytic review documented that depression is associated with large decreases in proliferative response of lymphocytes to PHA, Con A, and pokeweed mitogen (PWM), and natural killer cell cytotoxic activity and decreased numbers of NK cells, T cells, B cells, helper T cells, and suppressor/cytotoxic T cells (Herbert & Cohen 1993a). These associations appear stronger among older and hospitalized populations. Finally, recent data suggest that those who exhibit heightened sympathetic responses to stress, indexed by elevations in blood pressure, heart rate, and catecholamines, are most likely to show immuno-suppression during stress (Manuck et al 1991, Zakowski et al 1992).

Given these associations, psychological interventions to alter immune function have been tried. Both a cognitive-behavioral stress management program and exercise program attenuated immunologic changes following notification of HIV-1 seropositivity (Antoni et al 1991, LaPerriere et al 1990). Writing about traumatic personal experiences led to better lymphocyte response to PHA, compared to a control of writing about topics with no emotional element (Pennebaker et al 1988).

Although associations between psychological parameters and functional and enumerative measures of immune measures are now documented, the precise health consequences of changes in immune responses are not known, particularly in determining infection and emergence of initial clinical symptoms. Decreased NK cell activity is a predictor of disease recurrence in patients with early-stage breast cancer (Levy et al 1991); delayed hypersensitivity to skin testing is associated with mortality, unadjusted for age in the elderly (Wayne et al 1990); and poor PHA response is associated with mortality in one sample of the elderly (Murasko et al 1988), but not in another (Wayne et al 1990). At this point, a high priority should be longitudinal studies on psychologically-induced alterations in immune parameters in relation to health outcomes.

SUMMARY AND CONCLUDING COMMENTS

The past five years have witnessed substantial progress in addressing the question, who becomes sick and why? Links of antecedent environmental and individual variables with future disease have become clearer. Firm evidence has emerged that social support, dispositional hostility, and work strain are linked to health outcomes, whereas accumulating evidence suggests that Type A is not linked with CHD, nor depression with all-site cancers. In addition, appraisal of stress appears to play a more important role in health outcomes than does simple exposure to life events.

Progress has occurred despite methodological challenges in studying the etiology of disease. A relatively small number of initially healthy individuals fall ill over even five to ten years (except for upper respiratory disease and flu). Suitable indicators of subclinical disease are often not available for research purposes. For example, coronary angiograms and thallium scans used to measure atherosclerosis require costly or invasive procedures, making their use hard to justify in people with no apparent disease. The influence of psychosocial factors may be more easily detected in studies of recurrence or of course of disease because of greater incidence rates and because frank disease is more easily measured than subclinical disease. Studies have, for example, documented higher rates of recurrence of herpes and breast cancer (Forsén 1991, Levy et al 1991, Kemeny et al 1989, cf Dean & Surtes 1989), of abnormal cardiac responses in heart patients (Follick et al 1988, Zotti et al 1991, Rozanski et al 1988), and of poor glucose control in diabetics (Gonder-Frederick et al 1990) under increased stress.

Health-related psychological concepts are increasingly being refined. In an effort to understand better the health-damaging aspects of psychological factors, distinctions have been made between subjective stress and life events, functions of social support and social network or integration, hostile attitudes

and neuroticism, and dispositional optimism and attributional style. We are lagging, however, in developing measurement tools that adequately capture the progress made in refining our concepts. We often infer relationships based on secondary analyses of existing items, rather than using a priori scales with demonstrated external and discriminative validity. Furthermore, work establishing the external validity of self-report measures of stress, neuroticism, social support, hostility, etc is less than desirable, leaving questions about the conceptual underpinnings of our measures. Validated tools are essential for establishing precise linkages between behavior and disease and for adequate characterization of individual risk in studies of prevention and intervention.

In contrast, there has been substantial research on assessment of mechanisms. We now have reliable and internally valid assessments of stress-induced cardiovascular reactivity. Although there are many demonstrations that psychological factors impact on cardiovascular and immune function, it is not yet clear, however, that psychologically-induced alterations in cardiac and immune function are predictive of disease status. This is an important agenda for future research.

Future research on mechanisms as well as on measurement of psychological states will benefit from recent technological advances. Of special relevance are ambulatory monitoring techniques for measurement of physiological states (e.g. myocardial ischemia, blood pressure, glucose sampling) and psychological states (e.g. mood) that record data for immediate relay via telemetry to data storage and for later readout. These techniques will permit health psychologists to address different questions; to assess, in a more refined manner, variability in physiological and psychological states for testing of theory; and to avoid biases owing to retrospective recall and poor sampling techniques.

Research on mechanisms has focused mostly on cardiovascular reactivity and psychoneuroimmunology. New directions in research on mechanisms are emerging, including the effects of stress on body fat distribution (Rebuffé-Scrive et al 1992), which is a newly identified risk factor for cardiovascular disease; development of a suitable animal model for noninsulin-dependent diabetes mellitus that has permitted analysis of genetic and environmental determinants of hyperglycemia (Surwit 1992); and the role of the sympathetic nervous system in accounting for the covariation of insulin sensitivity, blood pressure, and body fat distribution (Donahue et al 1990). The interactions among systems influenced by environmental stress will be an important arena for further investigation.

Finally, health psychology is now seeing the development of more sophisticated models that consider genetic predispositions, environmental challenge, and individual differences in behavior in understanding disease risk. We anticipate that the enormous progress made in studying the relationships between psychological variables and health outcomes in the last few years will be

dwarfed by the progress in the next five years as research will benefit from the advances in technology, research on assessment of psychosocial variables and mechanisms, and awareness of the interaction between mechanisms and environmental variables, dispositions, and health-related behaviors.

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