HEALTH PSYCHOLOGY

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Americans currently spend over $400 billion dollars annually on health care—11% of the Gross National Product (Taylor 1987). This figure does not include such health-relevant expenses as health club memberships, special foods, vitamins, stripping asbestos insulation from the home, or installing air bags in the car. In this social and economic context, behavioral medicine and health psychology have become “buzz words for the 1980s” (Pomerleau & Rodin 1986, p. 483). While some continue to deny the importance of psychosocial variables in health and disease (Angell 1985), most investigators in this area believe that behavioral science knowledge and techniques can contribute to the understanding of physical health and illness (Schwartz & Weiss 1977) by complementing biomedical knowledge and applications.
Behavioral medicine has been defined as the integration of behavioral science approaches with biomedical knowledge and techniques (Schwartz & Weiss 1978) and as the application of behavioral therapies to medical disorders (Pomerleau & Brady 1979). Surwit et al (1983) suggest that behavioral medicine applies to all medical disorders, not just those defined as psychological or psychophysiological in nature. Health psychology has been defined as the aggregate of the knowledge base of psychology applied to the understanding of health and illness (Matarazzo 1984). As the disciplines have matured, less attention has been paid to distinguishing one from the other or either from the approach of the medical sciences. Rather, self-defining attention is now paid to practical issues, such as education and training (Taylor 1987), integration with other clinical approaches (Pomerleau & Rodin 1986), and cost-effectiveness (Taylor 1987); and to theoretical issues, such as additive vs interactional models (Cohen & Wills 1985) and the role of applied settings in developing rather than merely testing theory (Rodin 1985).

Another indication of the maturation of the disciplines is reflected in the Annual Review chapters covering this area. In the first (Miller 1983), studies were reviewed that attempted to convince the skeptical medical disciplines that psychosocial and behavioral variables influence onset, maintenance, and treatment of disease. Although the skepticism still remained in some quarters (Angell 1985), the second Annual Review chapter, appearing in the same year as the Angell editorial in the New England Journal of Medicine, presented substantial data indicating not only the role of behavior in health and illness but some mechanisms linking psychosocial variables to these outcomes (Krantz et al 1985). The studies covered in the present review provide further and more detailed evidence for the nature of the physiological mechanisms relating psychosocial and behavioral variables to health and illness, and suggest possible interactions among these variables.

In order to summarize this literature and to highlight further the utility of an approach that emphasizes process models and interactions among variables because of the complex and multiply determined factors that lead to health and illness, we have organized the chapter as follows: First, we describe a group of independent variables shown to correlate most strongly with health and illness outcomes. These include (a) dispositional variables, such as the Type A behavior pattern; (b) cognitive factors, such as representations and appraisals of illness; (c) social environment variables, including social support; and (d) sociocultural variables such as age, gender, ethnicity, and poverty. These variables appear to influence health and disease outcomes either directly or because they influence the second category of variables described below.

In the second section, we review specific behaviors that influence health and illness. These include (a) coping, (b) adherence, (c) substance use and abuse, and (d) exercise and other health behaviors. This group of health-
promoting and health-damaging behaviors is not static. The behaviors fluctuate as a function of motivation, appraisal, social support, and the like. They are also influenced by acute situational variables, such as those covered in the third section. In the third section, we review studies suggesting that the most significant impact of behavioral and psychosocial variables on health and illness may be seen when the system is provoked. The two most widely studied provocative events have been stress in general, whether or not health-relevant, and being sick, in particular.

Finally, in the fourth section we cover the two groups of disease most widely studied in health psychology: cancers and coronary heart disease. We examine studies that consider the direct effects of many behavioral and psychosocial variables, but we emphasize those that posit interactions among them and that focus on explanatory mechanisms.

ANTECEDENT VARIABLES

Dispositional Variables and Health or Illness

Several possible links between personality and disease outcomes have been suggested (Friedman & Booth-Kewley 1987): (a) Certain aspects of personality might result from disease processes; (b) personality might cause disease by motivating unhealthy behaviors; (c) personality could affect disease directly through physiological mechanisms; (d) personality might be related to disease through an underlying biological third variable; and (e) a variety of different causal factors and feedback loops might be at work in the relationship between personality and disease. Conceptual distinctions among these alternatives have led to rich theoretical advances in recent years, as noted below.

In research on personality and disease, subjects cannot be randomly assigned a personality and then observed over time. Therefore, no single study can ever prove a causal link. Some of the strongest evidence for causal connections comes from physiological research that identifies disease mechanisms and shows how they are affected by personality variables through prospective studies. For example, the studies of Levy and coworkers (Levy et al 1985, 1987) prospectively consider the effects of personality and other psychosocial variables on a parameter of immune function, natural killer-cell activity, shown to be important in the progression of breast cancer. Problems still exist in the literature, however, because many personality constructs are still not well operationalized. In some efforts, diseases or health-related behaviors are implicitly construed as an external criterion to help anchor or validate current personality constructs (cf Bowers 1987). When these constructs are then used to predict the same or similar diseases or health-relevant behaviors, the logic becomes circular, hampering efforts to identify causal links.
Many methodologists have argued strongly for a trait taxonomy in the study of health and disease. Without a standard framework for identifying traits, two problems arise (Costa & McCrae 1987b). Scales that measure different underlying constructs may be given the same name (e.g. the Jenkins Activity Survey and the Structured Interview to measure Type A behavior). Scales that measure the same construct may also be given different names (e.g. neuroticism and anxiety), violating the requirement for discriminant validity. A variety of purportedly different traits may all essentially converge on the same underlying personality construct (e.g. hardness, self-efficacy, learned resourcefulness, and internal health locus of control; or repression, denial, defensiveness, and blunting). Both issues continue to plague the field and weaken its conclusions.

**TYPE A** The Type A Behavior Pattern (TABP) has been defined as an action-emotion complex (Rosenman et al, in press) involving (a) behavioral dispositions (e.g. ambitiously, aggressiveness, competitiveness, and impatience), (b) specific behaviors (e.g. muscle tension, rapid and emphatic speech style, and accelerated pace of activities), and (c) emotional responses (e.g. irritation, hostility, and anger). Although many investigators have related the global TABP to chronic heart disease (CHD) end points (see Booth-Kewley & Friedman 1987 for review), in some studies 70–90% of the sample is labeled Type A (Dembroski & MacDougall 1983). Placing such large percentages of individuals in an at-risk category is inconsistent with sound epidemiological practice (Matthews & Haynes 1986).

A first step to resolving this confusion has been to recognize that not all components of the TABP may be coronary prone. A second step, and a more important one to issues of validity of the Type A construct, has been to refine assessment of the empirically different attributes contained in both the conceptual and operational definitions of the TABP (cf Costa et al 1986; Dembroski & Costa 1987). This effort has been problematic because the several different measures of TABP are only modestly correlated (Matthews 1982). In addition to the Structured Interview, based on diagnosis and quantitative assessment of behavior elicited in an interview format, no fewer than ten self-report questionnaires of varying complexity have been used for classifying the TABP (Friedman & Powell 1984). Many of these scales have not been validated as predictors of CHD, and most are distinct from the Structured Interview, which emphasizes behavioral signs indicating the presence of Type A rather than self-report of its consequences. The stability of Type A as measured by the Structured Interview has been high over at least a ten-year period (Carmelli et al in press).

The development of the Structured Interview reflected the belief that Type A is a set of overt behaviors manifested by people in certain situations and not others (Rosenman et al, 1988). Although many investigators have endorsed
this perspective theoretically, they have largely ignored it at the level of assessment and intervention (Thoresen & Ohman 1986), a problem with personality typing generally. Ideally, an interactional analysis would require direct observation of the TAPB over a range of unconstrained natural situations to determine which situational factors were particularly effective in eliciting Type A behavior (cf Magnussen 1983).

An opposing perspective views the TAPB as a fixed personality trait, implying an underlying dimension ranging from Type B personality to Type A. Most current self-report measures share this perspective. A second problem inherent in the dispositional view of Type A is the identification of an appropriate threshold. Classification of Type A becomes tied to some cutoff level (Thoresen & Ohman 1986). This tendency to blur behaviors with traits has pervaded much of the debate about Type A. Nonetheless, as Engel (1986) noted, many investigators of Type A have failed to recognize that traits can be assessed by measuring behaviors.

The most promising avenue for using the TAPB to predict CHD in recent years results from componential scoring of the Structured Interview. CHD among individuals under the age of 50 is predicted, for example, by the Structured Interview component score on “potential for hostility” but not by other components (Dembroksi & Costa 1987). In fact, recent meta-analyses have suggested that anger generally is an important predictor of CHD (Booth-Kewley & Friedman 1987), and a scale to measure the components of anger has been developed (Spielberger et al 1985). Yet, anger is not measured directly by the scale most frequently employed to study hostility and CHD, the Cook-Medley (1954). The two factors in the Cook-Medley scales, cynicism and alienation, appear intercorrelated, and Costa et al (1986) have concluded that a better label for the two scales combined might be “cynical mistrust,” since the total Cook-Medley “hostility” scale correlates .91 with the MMPI cynicism scale. Disappointingly, data from several studies suggest that hostility as measured by the Cook-Medley and behaviorally measured hostility are quite different (Matthews & Haynes 1986).

Children and adolescents have been studied to clarify the Type A syndrome. Most psychophysiological studies show that Type A children are more reactive to stress than non-Type A children (Matthews & Woodall, in press). In general, boys are more likely than girls to model the Type A behavior of their parents. The competitive aspect of Type A apparently leads to early and important achievements independent of ability, perhaps because caregivers and teachers respond to Type A behaviors in children by encouraging them to continue to strive to achieve (Matthews et al 1986a). Parents of Type A children appear to do the same (Bracke 1985). From these data it appears that Type A develops as an interaction between constitutional predisposition and parenting styles (Matthews & Woodall, in press; Thoresen & Patillo, in press).
BOLSTERING DISPOSITIONS  Several somewhat overlapping personality variables, all focusing on individuals' abilities to respond to difficulties in optimistic, persistent, and flexible ways, have been studied in relation to positive health outcomes. Viewing them together in this broader theoretical framework may have greater heuristic value in predicting health outcomes. One of these constructs—hardiness (Kobasa 1982)—is a composite of commitment, control, and challenge, each measured empirically by several scales. Studies of the stress-buffering role of hardiness are contradictory (Kobasa et al. 1983; Ganellen & Blaney 1984; Schmied & Lawler 1986). These inconsistent results may be partly attributable to the construct validity and stability of the factor structure in the hardiness measure (Funk & Houston 1987; Hull et al. 1987). Scheier & Carver (1987) have suggested that aspects of hardiness serve as a buffer against stress merely because of the undercurrent of optimism in certain constituents. Alternatively, it may turn out that one or another of the variables presently confounded in the hardiness construct, such as internal/external control, will prove to be a critical mediator (Funk & Houston 1987; Hull et al. 1987).

In arguing for a more generic construct of optimism (measured by the LOT; Scheier & Carver 1985), Scheier & Carver (1987) have suggested that differences in well-being between optimists and pessimists could derive from the way individuals select and use the general strategies for coping available to everyone. For example, optimists appear to display coping patterns that involve continued positive striving and making the best of whatever situations they confront. The second possibility is that optimism/pessimism differences directly affect physiological functioning. For example, Van Treuren & Hull (1986) suggested that optimists show less cardiovascular reactivity to stress.

Optimism and pessimism have also been studied from an attribution/learned-helplessness perspective. Peterson & Seligman (1987) posit that attributional dimensions influence one or more manifestations of helplessness. The emphasis on attributions as a determinant of expectancies is one difference between the theory of optimism/pessimism discussed above and Seligman's view of this construct. In other words, in attribution research, explanatory style is measured rather than expectations per se, and optimistic/pessimistic expectancies are inferred from the explanatory style. Peterson, Seligman, and their colleagues have developed two ways of measuring explanatory style, a self-report questionnaire called the Attributional Style Questionnaire (ASQ; Peterson & Villanova 1988) and a content analysis procedure called the CAVE (Peterson et al. 1983). Some studies report inverse correlations between pessimistic explanatory style (the use of causal explanations focusing on internal, stable, and global factors for negative events) and physical well-being (reviewed in Peterson & Seligman 1987).

Other research has emphasized perceived self-efficacy as a mechanism
linking psychosocial influences to health (Bandura 1986; O'Leary 1985a), and theoretical developments in efficacy have been advanced by application to the health domain (Litt 1988). There is no generic self-efficacy questionnaire. Rather, a set of specific efficacy judgments is developed for each experimental context regarding the necessary behaviors in a specific domain. For this reason, measures of self-efficacy are typically good predictors of behavior (e.g. Bandura & Schunk 1981).

Perceived self-efficacy may affect physiological systems that mediate health functioning. Bandura et al (1985) found that under conditions of phobic threat, self-doubts regarding coping efficacy produced substantial increases in circulating catecholamines. Catecholamine secretion declined as phobics gained mastery over phobic threats through guided mastery treatment. In arthritic patients, perceived coping efficacy was associated with increases in the number of suppressor T cells, which inhibit production of antibodies (O'Leary 1985b). Others have suggested that perceived self-efficacy may be linked to health-promoting and health-impairing behavior (e.g. Kaplan et al 1984).

DIFFICULTIES WITH EMOTIONAL EXPRESSION Alexithymia, an inability to use language to describe emotional experiences (Apfel & Sifneos 1979; Ahrens & Deffner 1986), has been linked to a variety of psychosomatic disorders—e.g. chronic pain (Acklin & Bernat 1987) and respiratory disorders (Sifneos 1973). However, a reliable and valid instrument for assessing this deficit has yet to be developed. Some investigators have found alexithymia related to a variety of disease states (e.g. hypertension; Fava et al 1980); others have reported weaker findings (Heiberg 1980; Kleger & Dirks 1980).

Another aspect of difficulty with emotional expression has been termed the repressive personality style, characterized by avoidance of potentially threatening social encounters or lines of associative thought that might lead to conscious conflict or embarrassing experience (Bonanno & Singer, in press). The repressor style was extensively explored in studies initiated by Byrne et al (1963) and has more recently been revived with greater emphasis on its psychophysiological components by Weinberger, Schwartz, and their collaborators (Weinberger, in press). Weinberger et al (1979) use a combination of the Taylor Manifest Anxiety Scale (Taylor 1953) and the Marlowe-Crowne Social Desirability Scale (Crowne & Marlowe 1964) to measure repression. Repressors on these measures report low anxiety but high defensiveness (i.e. social desirability). The combined method of measuring repression proposed by Weinberger has been criticized on the grounds that the Marlowe-Crowne as originally intended is not a measure of defensiveness but of social desirability [but see the discussion of measurement issues in Weinberger (in press)]. Self-deception or repression is indicated by subjects' refusal to endorse these
items. Temoshok (1985) has proposed a Type C behavioral pattern, reflecting inability to express emotion, particularly negative emotion, in an open fashion, and has related it to health endpoints (Temoshok 1987).

Although the Weinberger et al (1979) study emphasized blood pressure reactivity under stress among repressors, the bulk of the literature in recent years has explored the effects of difficulty in emotional expression on the immune system. Levy and her coworkers have suggested effects on natural killer-cell activity as a function of this constellation of personality variables (Levy et al 1987). This group of investigators and Jensen (1987) have also related the repressive style to breast cancer progression.

It has been suggested that anxiety may underlie all of these difficulties with emotional expression—denial, repression, suppression, and alexithymia (Friedman & Booth-Kewley 1987). Watson & Pennebaker (in press) come to the more general conclusion that a common underlying disposition of somatopsychic distress called negative affectivity accounts for the correlation of physical symptoms and negative emotion.

**Cognitive Activities**

Individuals' thoughts and beliefs influence their responses to situations in which health is salient. In earlier decades, these beliefs were studied in a framework called the Health Beliefs Model and characterized by dimensions concerning perceived susceptibility, severity, benefits, and barriers (see review by Janz & Becker 1984). More recently, these beliefs have been organized according to strategies suggested by social cognition researchers, allowing the opportunity for researchers in the area of health psychology to benefit from and contribute to the rich theoretical developments in the field of cognition.

**MENTAL REPRESENTATIONS OF ILLNESS**  According to Leventhal and colleagues, people evaluate a physiological perturbation or symptom against an implicit or "commonsense" cognitive representation of illness, unique to each individual, that includes expectations about illness and examples of particular diseases. Respondents typically construct a mental representation of their physical problems containing four attributes: (a) identity (label and symptoms), (b) cause, (c) consequences, and (d) timeline (Leventhal et al 1980). Lau & Hartman (1983) have added a fifth dimension, cure. A slightly different set of illness attributes was recently suggested by Turk et al (1985), including seriousness, personal responsibility, controllability, and changeability. This approach has been applied to individuals' understanding of hypertension (Baumann & Leventhal 1985; Meyer et al 1985), cancer (Nerenz 1979), and aging (Prohaska et al 1987).

A different method for understanding mental representations of illness has been suggested by Bishop and colleagues (Bishop 1987; Bishop & Converse
who believe that lay people cognitively organize and recall information about physical symptoms according to disease prototypes. The prototype approach differs somewhat from the dimensional approach in that it posits the existence of concrete instances (e.g. specific diseases) rather than generalized abstractions (e.g. illness schemas). Common disease prototypes exist for frequently occurring diseases, for diseases portrayed in the popular media, and for diseases that present clear and obvious symptoms. These disease prototypes may help individuals to understand bodily changes and symptoms because they provide ready-made interpretations for given internal experiences and help the individual to access other information about his or her condition (Bishop & Converse 1986).

Attributions regarding health and illness are another type of mental representation. Attributions here are causal inferences people make about events or states of being (Jones et al 1972). Individuals attribute responsibility for the onset and course of illness to themselves or to the environment (Brickman et al 1982). Studies suggest that these attributions affect health outcomes through a variety of mechanisms. For example, Taylor et al (1984b) found that the belief that one had personal control over the progression of cancer was associated with better adjustment, although attributions of responsibility for the cause of the cancer itself were not associated with more positive outcomes. Attributional processes also affect coping by influencing how individuals understand their role in the illness itself (Tennen et al 1986). Attributional processes also play a role in the interpretation of physical symptoms (Michela & Wood 1986).

PERCEPTIONS OF RISK AND VULNERABILITY Perhaps as a result of attributional processes, individuals tend to underestimate their own risk relative to other people for illnesses and other negative life events (Kirsch et al 1966; Weinstein 1984). This unrealistic optimism can have important health consequences. Beliefs concerning increased susceptibility to an illness are generally associated with greater interest in prevention and learning prevention behaviors (Cummings et al 1979; Weinstein 1983), although there are exceptions (Joseph et al 1987).

Several factors affect these perceptions of vulnerability. Individuals who have not experienced major negative life events tend to see themselves as especially invulnerable (Perloff & Fetzer 1986). Conversely, sick individuals view their risk of future illness as similar to (rather than less likely than) that of their peers, but they still see themselves as relatively invulnerable to other kinds of negative events (Jemmott et al 1988; Kulik & Mahler 1987). This “false consensus” bias (Ross et al 1977) extends to initiation of health-relevant behaviors. When compared with their nonsmoking peers, adolescents who were smokers or who intended to smoke greatly overestimated the prevalence of adult or peer smoking and greatly underestimated negative adult
attitudes toward smoking. Adolescents who intended to smoke believed they would be less likely than others to contract a smoking-related illness if they became smokers (Leventhal et al 1987). Finally, risk estimates are influenced by mood. Dysphoric affect tends to make individuals believe that future victimizations are more likely (Johnson & Tversky 1983; Salovey & Birnbaum 1988).

Awareness and acceptance of information also vary with motivational status. A compelling example is provided by Wagener & Taylor (1986), who discovered that failed renal transplant patients recalled the circumstances of their original decision to have the transplant as involving less personal responsibility than did successful transplant patients. These individuals recalled that they had little choice but to make the decision they had made. Perhaps the emerging interest in motivational influences on social cognition will provide the framework for investigating such mechanisms (Showers & Cantor 1985; Sorrentino & Higgins 1986).

**CONTROL.** The cognitive activities discussed thus far may all serve to help individuals feel in control of their lives and futures. Control, or perceived mastery over one's circumstances, can be thought of as a basic human motivation (Rodin et al 1986). The presence or absence of a sense of control has a profound influence on individuals' emotional, cognitive, and physical well-being (Rodin 1986a). Efforts to link loss of control and hopelessness to the development of illness were pioneered by Engel and Schmale (Engel 1968; Schmale 1972). More recent work has emphasized a potentially important role for loss of control in cancer (Visintainer et al 1982) and cardiovascular disease (Frankenhaeuser 1986a; Matthews 1982).

Numerous mechanisms could mediate the effects of control on health (see Rodin 1986b for review): stress reduction (Lazarus & DeLongis 1983; Rodin et al 1982), increased noticing of symptoms (Pennebaker 1982), direct physiological effects on the immune system (Laudenslager et al 1983; Rodin 1986b; Stein et al 1982) and neuroendocrine system (Frankenhaeuser 1986a), and increased health-enhancing actions (Ewart et al 1984; Manning & Wright 1983).

Many of the health-relevant variables discussed in this review interact with control to influence health and disease outcomes. Lower levels of work stress, for example, result from the ability to exert control (Frankenhaeuser 1986b). Type A individuals appear particularly vulnerable to physiological ill-effects of uncontrollable events, and hypertensives appear relatively unable to profit from the beneficial effects of personal control (Frankenhaeuser 1986a).

**Resources from the Social Environment**

Health and illness are influenced by resources from the social environment, most notably spouses and close others, family members, and the workplace.
Each of these aspects of the social environment has some impact on the health of the individual, but, reciprocally, an individual’s health influences the thoughts and behaviors of significant others and, perhaps, alters the work environment as well.

SOCIAL SUPPORT For several years, social scientists have noticed that physical and psychological illnesses are more prevalent in communities in which social ties and networks are disrupted by changes in employment patterns, migration, aging, and death. It was felt that something about the social context of the individual had a profound impact on that individual’s health and well-being (Caplan 1974; Cassel 1976; Cobb 1976). The process by which the social context influences the individual has been termed “social support,” defined as the resources provided by other persons (Cohen & Syme 1985).

It might seem obvious that support from others should improve health and adjustment to illness, but the research literature is mixed. In general, social support provides relief from psychological distress during crises (Holahan & Moos 1986; Sarason & Sarason 1985). Social support may (Berkman & Syme 1979) or may not (Wallston et al 1983) directly inhibit the development of illness. Once an individual is ill, social support plays an important and positive role in promoting adjustment and recovery (DiMatteo & Hays 1981; Wortman 1984).

Theoretical developments have included better specification of the mechanisms by which social support may influence health, either through direct or buffering effects (e.g. Cohen & Syme 1985). By being integrated into a social network, individuals may experience greater positive affect, higher self-esteem, or feel more in control of environmental changes (all direct effects). Each of these cognitive factors might then protect an individual from physical illness through a variety of physiological mechanisms (e.g. immune system functioning; Jemmott & Locke 1984) or by encouraging the individual to make healthy life-style changes. At a more general level, social support via an integrated social network may have direct effects on health by providing the individual with a predictable set of role relationships, a positive social identity, and experiences of mastery and control (Thoits 1983, 1985). In comparison, social support could play a role in buffering the impact of negative events and other stressors by eliminating or reducing the stressor itself, bolstering the ability of the individual to cope with the stressor, or by attenuating the experience of distress after it has already been triggered (Cohen & McKay 1984; Gore 1981; House 1981). Direct effects are more likely to be obtained when support is defined as the degree to which a person is integrated into social networks; buffering effects are typically discovered when support is operationalized as the social resources available to one undergoing stressful events (Cohen & Syme 1985; Kessler & McLeod 1985; Wethington & Kessler 1986).
Social support research, although certainly flourishing, has been hindered by difficulties in measuring support (House et al. 1988). Cohen & Syme (1985) concluded that there are “almost as many measures [of social support] as studies” (p. 14). Bruhn & Philips (1984) reported on 14 strategies for measuring social support, but noted that basic psychometric properties for more than half of these have never been investigated. Among the more promising (and reliable) techniques for measuring social support are assessment via family and work environment scales (Billings & Moos 1982), social relationship scales (Schaefer et al. 1981), and direct self-report (Wilcox 1981).

Kessler et al. (1985) note that one of the reasons why the relationship between social support and health may be complicated is that those in greatest need of support from others may be least able to obtain it. Individuals with severe health problems, because they increase others’ sense of vulnerability, may be seen as threatening, particularly if others are worried about experiencing a similar fate—e.g., when the disease has a poor prognosis (Peters-Golden 1982).

Social support may also play a role in promoting illness or maladaptive behaviors. Adolescent substance abuse, for example, is often encouraged and reinforced by similar behavior among the child’s peer group (e.g., Kandel & Maloff 1984). Even in adults, the perception of a supportive community may delay or prevent consultation with medical professionals (Pilisuk et al. 1987). Moreover, network size can exert a negative influence on adhering to behavioral regimens necessary for the management of chronic diseases like diabetes (Kaplan & Hartwell 1987).

**Marriage and Family** Perhaps the most important source of social support is one’s spouse and family, and the loss of social support resulting from the death of one’s spouse is a significant stressor (Osterweis et al. 1984). Adjustment following illness onset is influenced by successful marital adjustment and general support by the spouse (Morris 1979; Vess et al. 1985a, b). Additionally, a stable and happy marital relationship may have a preventive effect on future illness. Kiecolt-Glaser and her colleagues (1987), for example, have reported that poor marital quality is associated with poorer immune function. Similarly, chronically ill individuals who have the active support and participation of their spouses in treatment may show better adjustment and faster recovery (Taylor et al. 1985).

Although poor marital quality has been associated with negative health outcomes, it has been more difficult to demonstrate that family functioning in general is related to health and illness (Brown et al. 1982). More research attention, instead, has been directed toward the consequences of illness for marital relations and family functioning (reviewed below).
WORK ENVIRONMENTS A variety of aspects of work have an important
effect on health, including (a) physical, chemical, and biological hazards;
(b) the physical demands of a given job; (c) job security; (d) psychosocial
demands; (e) control and decision latitude; and (f) social support.

It has been well demonstrated that unemployment is associated with high
levels of stress and significant disease outcomes (Cobb 1974; Kasl & Cobb
1970). More recently, it has been discovered that unemployed individuals
differ from employed workers on physiological indicators of stress (such as
circulating catecholamine levels) and also perform more poorly on complex
tasks, often exhibiting behaviors associated with learned helplessness (Baum
et al 1986).

Work, too, can be stressful. Occupational stress is usually associated with
employment situations in which multiple competing demands are placed on
the individual and in which the individual can exert little authority or control
(Singer et al 1986). The perception of these job characteristics seems more
closely linked to health behavior and morbidity than are the actual characteristics
of the job (House et al 1986). For example, perceived job pressures
resulted in a tendency to work longer hours and increase smoking and
drinking behaviors.

The workplace can also benefit health. Coworkers can be important sources
of social support. They can provide health information and encourage healthy
life-style changes. Increasingly, organizations have implemented health-
promotion activities for their employees. Because such programs tend to
boost company morale (Felix et al 1985), participating individuals may
receive both the direct benefit of the program (e.g. weight loss, smoking
cessation) and also more positive social interactions with their coworkers. For
example, worksite weight-loss programs are as effective as self-help and
commercial groups (Brownell et al 1985) and often result in positive changes
in morale, better employer/employee relations, and a cost-effective change in
participants' life-styles (Brownell et al 1984).

Demographic Variables

GENDER The effects of gender on health have only recently been explored.
Study is urgently needed, however, given epidemiologic demonstrations that
women are on the average significantly less vulnerable than men to most
modern life-threatening diseases (Bush & Barrett-Connor 1985). Women
have until recently been less likely to smoke than men (Waldron 1986), show
fewer stress-induced lipid responses (Stoney in press), and have lower num-
bers and/or density of peripheral vascular adrenergic receptors than men
(Freedman et al in press), suggesting several possible reasons for their
substantially lower incidence of CHD. Differences in reproductive hormones
may also be linked to gender differences in disease epidemiology (Bush &
Barrett-Connor 1985), either directly or as stimuli for other physiological processes.

Because of the high employment rates of women at the present time, it is possible to compare men and women in the same occupation at the same worksite, thus controlling for effects of occupational role on health. Detre et al (1987) studied federal government employees holding high service grades (14 or above). The occupational experience of men and women in similarly responsible and demanding positions appears to reduce the mortality advantage of women, suggesting that variables unrelated to sex-specific hormones may strongly contribute to the overall mortality differences between men and women in national databases. There are also differences in the stress hormone reactivity of males and females. At rest, men and women do not differ in levels of the stress hormones epinephrine, norepinephrine, and cortisol, but when a challenge is applied, males release more epinephrine than females (Frankenhaeuser 1986a). Men also appear to have greater blood pressure responses to many types of stressors than women (Matthews & Stoney 1988).

In Western countries, males tend to engage in more risky behaviors than females, particularly those involving physical daring or illegal activities, thus influencing health (Waldron 1986). One important generalization is that gender differences in specific types of health-related behavior are strongly influenced by the compatibility of the behavior with general sex role expectations (Waldron, in press).

In Western countries, women visit physicians more than men (Verbrugge 1985; Waldron 1983). Although many psychological reasons have been posited for this difference, it is important to note that women’s more complex and demanding reproductive functions are the major reason for their higher rates of physician visits, at least among young and middle-aged adults (Verbrugge 1985; Waldron 1983). Women may also be more likely to visit physicians because they have more self-reported symptoms and poorer self-rated health. Survey data in the United States suggest that men may put up with more pain than women (Mechanic 1964; Verbrugge 1982). Also women rate the importance of health somewhat higher than do men (Verbrugge 1982). Gender differences in attitudes toward health care are suggested by the findings that women more often than men have a personal physician or a regular source of health care (Cleary et al 1982; NCHS 1985). In the United States, at least, women appear to have greater faith than men in the value of preventive medical care (Cleary et al 1982; Waldron 1983). There is no evidence for the hypothesis that gender differences in employment and other role obligations are the major cause of gender differences in physician visits (Waldron, in press).

Finally, women and men are treated differently by the health-care system. For example, physicians respond less to female patients’ requests for informa-
tion (Todd 1984). However, most evidence suggests little gender difference in physician’s recommendations for medical care for a given condition (Verbrugge 1985). In some non-Western societies, females obtain less adequate medical care than males, reflecting sex discrimination and other general sex role differences in these societies.

AGE Physical health of the aged population reflects both normal biological changes and increased incidence of many major diseases. For example, about 86% of individuals over age 65 living in the community have at least one chronic disease, and 50% have two or more (Jarvik & Perl 1981). Increased vulnerability is additionally reflected in the fact that the impact of acute illness is often more severe in older adults and recovery more protracted (Atchley 1977). Many of the chronic conditions experienced by the elderly result in part from the life-styles and health practices of their youth (Siegler & Costa 1985).

In understanding the effects of age as a variable on health, it is extremely important to note that all data suggest greater interindividual variability with aging (see Rodin 1986b). Second, changes that appear to be related to biological aging may actually result from psychosocial factors associated with being old, such as widowhood. Third, the outcome of the aging process is not always decline. Some diseases (e.g. autoimmune disease) are less likely to occur in old age. Moreover, the impact of many life events, such as the death of a spouse, is often smaller in old age, since these events occur “on time” in old age. Recent theoretical emphasis has shifted to characterization of “successful” vs “usual” aging (Rowe & Kahn 1987).

It has been suggested that the relation between an individual’s health and the effects of psychosocial variables might grow stronger in old age (Kasl & Berkman 1981). Rodin (1986b) focused on one psychosocial variable, control, in elaborating further this position, emphasizing that experiences related to control increase markedly in older age and have different social meanings. Further, the association between control and an indicator of health status may be notably altered or conditioned by age, or age may influence the relation between the sense of control and health maintenance behaviors and the seeking of medical care.

There has been substantial attention to changes in social support that occur with aging, which may influence health and well-being. Many of these have focused on bereavement and relocation (see Rowe & Kahn 1987 for review). One outcome of increasing concern is the use of health services. Widowed persons go to the doctor and hospital more often than married persons (Verbrugge 1979). Not being in the labor force any longer has also been considered a cause of the increased use of ambulatory physician services by older adults. However, a recent analysis of data on almost 20,000 individuals
aged 55 and over taken from the 1978 Health Interview Survey showed that 
widowhood and retirement per se are not the issue in utilization of health 
services among elderly adults. Rather, living alone is the crucial factor 
(Homan et al 1986), suggesting a theoretically richer set of variables 
operative. Other suggestions of spurious interpretations of earlier data include 
the notion that older adults show better rates than younger people of 
commonly accepted favorable health practices (e.g., abstinence from alcohol use 
and cigarette smoking; NCHS 1980), leading to suggestions that they are 
more adherent to health-promoting regimens (Besdine 1981). However, it is 
reasonable to suggest that people who have engaged in a set of distinctly 
unfavorable health practices are more likely to have died prior to age 65 
(Breslow & Endstrom 1980).

A variety of behaviors relevant to health may be influenced by the fact that 
older adults are more likely to attribute symptoms to normal and irreversible 
aging, to underreport symptoms, and to anticipate a health decrement as an 
equated part of the aging process (Besdine 1981; Brody & Kleban 1981; 
Rodin 1986b). There are conditions under which this expectation may 
pro-mote certain forms of health behavior and others where it may inhibit them. 
Moreover, Leventhal (1984) found few age differences in cognitive repre-
sentations of various illnesses, but significant age differences in associated 
emotions. Anger generated by the cancer label decreases with age, as do fear 
and shame associated with alcoholism and arthritis. These age differences in 
affective responses to illness could have significant implications for responses 
to treatment and family relations.

The age of the patient can influence clinical decisions that affect treatment 
(Riley 1987). Older people are more likely than younger people with identical 
symptoms to be given a poor prognosis for recovery and more likely to be 
given palliative rather than intervention treatments (Ciliberto et al 1981). 
Older people are also less likely to challenge their physicians and more likely 
to adopt a passive role in health care (Haug 1981). Thus, in viewing all of 
these data, one must be attentive to the interactive effects of patients’ and 
physicians’ expectations and behaviors.

ETHNICITY AND SOCIAL CLASS Until recently, little of the literature in 
health psychology focused on racial or ethnic minorities. As noted by 
Anderson & Jackson (1987), this lack of attention is problematic for at least two 
reasons. First, sociocultural and socioeconomic differences in behavior and 
reactions to illness, both among and within racial and ethnic groups, have 
implications for physical health and illness (Harwood 1981; Hamburg 1982). 
Second, members of racial and ethnic groups within this society have higher 
incidence and prevalence rates for many physical illnesses (NCHS 1984; 
Jackson 1981).
Comparisons of cancer death rates provide evidence of the extra burden of illness experienced by Blacks (NCHS 1984). Blacks (and Hispanics) exhibit a higher incidence of AIDS, and the transmission pattern of this disease in these groups differs from that in Whites [non-Whites are more likely to transmit AIDS through intravenous drug use or sexual contact with drug users (Bakerman et al 1986; Peterson & Bakeman, in press)].

Blacks are roughly twice as likely to develop hypertension as Whites (Saunders & Williams 1975). There is beginning to be evidence with regard to cardiovascular disease that the differences between Blacks and Whites are attributable to biological mechanisms. Most of the research attention has focused on the sympathetic nervous system (see Anderson & Jackson 1987 for a review). There are also striking racial differences in sodium homeostasis (Luft et al 1985). Racial differences in styles of coping with stress have also been implicated in hypertension (James et al 1984).

Racial and ethnic variables are confounded with socioeconomic factors that may influence health and disease. Low socioeconomic status usually results in a less stable physical environment, a less stable and supportive social environment, altered perceptions of oneself and one's group, and altered capacities to adapt psychologically and behaviorally. Inadequate resources, low-status jobs, social stigma, and inadequate education interact with differential physiology, nutrition, environmental risks, and coping styles to create a circle of disadvantage (Kessler et al 1985; Jenkins 1982). These active ingredients of social disadvantage might relate to health risks in a variety of ways including, for example, a noxious environment; lack of knowledge, funds, or availability of early care; economic or cultural barriers to screening or diagnostic services or treatment opportunities; and inadequate follow-through (Jenkins 1982). Differential health behavior is also evident. For example, several surveys indicate that individuals most likely to exercise tend to be younger, more highly educated, and more affluent (Government of Canada 1982).

MEDIATING VARIABLES

Coping Processes

In studying how individuals adapt to stressful life circumstances, coping resources are sometimes distinguished from coping responses. Coping resources are personality, attitudinal, and cognitive factors that provide the psychological context for coping responses (Moos & Billings 1982). Coping responses occur as the result of appraisal processes that may be influenced by coping resources.

A number of investigators have been concerned with the classification of coping responses. Moos & Billings (1982) suggest three primary coping
domains can be identified in the literature on coping: (a) appraisal-focused coping, attempts to define the meaning of a situation; (b) problem-focused coping, trying to modify or eliminate the source of the stress; and (c) emotion-focused coping, managing emotions aroused by stressors and trying to maintain affective equilibrium. The distinction between problem-focused and emotion-focused coping has also been suggested by others (e.g. Lazarus & Folkman 1984; Lazarus & Launier 1978). Recently, coping responses have been classified as avoidant and nonavoidant (or attention) types (Suls & Fletcher 1985). Stone et al (in press) argue that current schemes for classifying coping responses are oversimplifications of the way people actually cope with stress. They suggest grouping coping strategies in terms of several general themes: seeking social support, seeking information, religiosity, situation redefinition, behavioral and cognitive avoidance, tension reduction, and problem solving. These themes are similar to those derived empirically by Folkman et al (1986).

Do individuals tend to use the same coping responses across situations or do they select a specific coping response for each stressor? Although more of the variance in coping responses seems attributable to situational differences than to individual differences in coping styles or coping resources (McFarlane et al 1983; Pearlin & Schooler 1978), there is clearly a person × situation interaction (House et al 1988).

Are certain coping responses consistently more efficacious than others? Suls & Fletcher (1985) conducted a meta-analysis of 43 studies of coping efficacy in various domains. Overall, coping strategies involving avoidance were just as effective as those involving active attention, and both types of coping yielded better adjustment than no-coping control conditions. But when the time course of the stressor was considered, avoidant coping responses seemed to be more effective in the short term, unless attentional coping strategies focused on sensory rather than emotional interpretational sets. In terms of longer-term outcome, attention was associated with better adjustment.

Effective coping may play an important role in health promotion, disease prevention, and more rapid recovery from illness (for review see Kessler & Wortman, in press). How individuals cope with stress is an important mediator of the stress-illness relationship (Cohen 1984). Cohen (1984) reviewed several mechanisms by which successful coping can affect the etiology and course of a disease. Coping can influence hormone levels, cause direct tissue changes, or affect the immune system (Jacobs et al 1985; Kielcolt-Glaser et al 1987). Interpersonal coping styles may influence the type of care received (e.g. demanding, task-oriented patients may have their complaints acted upon more quickly). In fact, cancer patients who cope by complaining and expressing high levels of negative affect survive longest (Jensen 1987). Conversely, positive coping, including strong feelings of a “will to live” and
high morale, may also have positive physiological consequences (Scheier & Carver 1987; Peterson & Seligman 1987). Effective coping has been linked to quicker recovery from illness, and active participant coping strategies may be especially effective in this regard (Cohen & Lazarus 1979).

Efficacious coping reduces stressor effects, but it also has costs (Cohen et al 1986). Coping processes require effort, and prolonged coping depletes the individual’s supply of energy. Successful coping also can result in the overgeneralized use of an effective strategy in inappropriate situations. For example, the Type A behavior pattern might be thought of as an overgeneralization to nearly all life domains of a coping strategy that was effective for coping with competing demands in one domain. Finally, coping may produce pathogenic effects (Contrada et al 1982). Moreover, coping behaviors themselves can be detrimental to health (e.g. smoking and drinking alcohol; Pomerleau & Pomerleau 1984).

Substance Use and Abuse

Cigarette smoking, excessive use of alcohol, and obesity represent major health risks and therefore continue to be the focus of considerable research attention. For example, the 1982 Surgeon General’s Report noted that 30% of all cancer deaths were attributable to tobacco use. Smoking is also a major factor in death from cardiovascular disease (Abbott et al 1986; Dawber 1980). Alcoholism, via its effects on the liver, nutrition, and risk for head and neck cancer, represents one of the three leading causes of death in modern societies (Fourth special report on alcohol and health, 1981). Other health risks include the strong relationship between alcohol abuse during pregnancy and poor fetal outcome (Sokol et al 1980).

Obesity has also been associated with mortality and morbidity. Similarly, there is a small increase in excess mortality with low body weight. The correlates of obesity contributing to excess mortality include non-insulin dependent (Type 2) diabetes, digestive disease, hypertension, cardiovascular diseases, and cancers (Bray 1984).

Over the last several years, models of substance use and abuse have moved away from traditional theories of addiction that focus on pharmacological properties of drugs and their physiological effects (e.g. Lettieri et al 1980; Jellinek 1960). In these theories, the primary emphasis is on uncontrollable endogenous processes as the basis for addiction. The paradigm shift toward a more cognitive-behaviorally based model is most evident in the work of Marlatt and his colleagues on the determinants and prevention of relapse (Marlatt et al 1988 see also Shumaker & Grunberg 1986). These models focus on the cognitions and behaviors that lead to the initiation and maintenance of substance use and abuse.

The field has once again begun to generate biological perspectives, howev-
Swan et al, 1988b) are now the focus of considerable research, based on new analyses of cohorts of twins and other behavioral-genetics approaches (e.g. Stunkard et al 1986 for obesity; Cadoret et al 1987 and Cloninger 1987 for alcoholism). Recent data for obesity emphasize the possible inheritance of biological parameters such as low metabolic rate (Ravussin et al 1988; Roberts et al 1988). It appears that variability in sensitivity to alcohol among individuals, and in different organ systems within individuals, may also be genetically determined (Straus 1986).

Over the last few years there has also been increasing emphasis on the study of common processes across substance abuse domains. The idea of commonalities was first discussed by Hunt et al (1971), who found nearly identical patterns of relapse in heroin addicts, alcoholics, and cigarette smokers. Adding an analysis of dieters and exercisers, the picture appears the same today (Marlatt & Gordon 1985). Many differences also exist both among the disorders and among persons afflicted with the same disorder. For example, genetic contributions to both alcoholism and obesity suggest separate pathways for their development. There are also key differences in the pharmacology of nicotine and alcohol (Best et al in press; Pomerleau & Pomerleau 1984), and food abuse fits even less neatly with concepts of dependency, withdrawal, and tolerance. Nonetheless, there has been great enthusiasm in the field recently for the study of commonalities.

A National Academy of Sciences report (Levison et al 1984) emphasized common processes at the sociocultural, psychological, and biological levels. There is a strong association between young age and a variety of risk-taking behaviors, including the use of substances such as cigarettes and drugs. Fleming et al (in press) have suggested that cigarettes may be the entry-level drug. Individuals who have tried cigarettes are significantly more likely to use other substances two years later (e.g. coffee, beer, marijuana). This study extended the previous efforts of Kandel (see Kandel & Maloff 1984 for review) demonstrating the tendency for use of illicit drugs to occur in a relatively fixed temporal sequence.

Further commonalities exist in the social influences that underlie the etiology of substance use, such as peer group influence and situational and social contexts. The seminal work of Jessor & Jessor (see Jessor 1984, for review) focuses on adolescence as a relatively high-risk stage of life. Many health risk behaviors in adolescence tend to covary. According to Jessor & Jessor (1982), the term that best captures the content of the dimension underlying psychosocial proneness is conventionality/unconventionality. From their work, it appears that with a permissive environment, unconventionality promotes proneness to at-risk behavior that carries through to adulthood.

Studies of common processes have also focused on the role of learning theory in understanding substance use and abuse. Donegan et al (1984) have identified six properties common to the development of many habitual health-
damaging behaviors: (a) the ability of the substance to act as a reinforcer; (b) acquired tolerance—reduced effectiveness of the same dose or exposure over time; (c) physical dependence and withdrawal; (d) affective contrast—often euphoria followed by dysphoria; (e) the capacity of the substance or activity to act as an effective Pavlovian unconditioned stimulus; and (f) the capacity of states like arousal, stress, or pain to influence use.

Lang (1984) has reviewed the evidence for the suggestion that a common personality pattern leads some individuals into the heavy use of recreational drugs. Lang concludes that no unique personality trait or profile is a necessary or sufficient condition for substance abuse, but some personality factors (such as impulsiveness and difficulties in delaying gratification) appear to be predisposing factors that may act in concert with situational and other variables.

Biological commonalities have emphasized the relation of naturally occurring substances and sites in the central nervous system to biological mediators of continued substance use. This work has focused on the body’s natural opiate system (e.g. Simon 1984; Bloom 1984). Substances may also alter the bioavailability of several behaviorally active neuroregulators, thus helping to explain the remarkable persistence of substance use behaviors. For example, nicotine alters the bioavailability of acetylcholine, norepinephrine, and dopamine, leading to the suggestion that nicotine is used by smokers to produce temporary improvements in performance and affect (Pomerleau 1986).

The second domain of recent interest in common processes has focused on the relationship between stress and coping and the use of substances. For example, it is a widely held belief that alcohol reduces stress (Sher 1988). Several psychosocial, physiological, and pharmacological variables can influence alcohol’s effects on stress, including the amount of alcohol consumed, the person’s prior experience with alcohol, individual experiences based on physiological responsiveness to ethanol, learned expectations about alcohol and its effects, the social setting in which drinking occurs, and the specific way stress is measured. Similar proposals have been made for the effects of stress on excessive food intake (Cattanach & Rodin 1988) and on smoking (Pomerleau 1986; Pomerleau & Pomerleau 1984).

Questions about the effects of stress on increasing substance use lead to considerations of the use of these substances as coping strategies (Marlatt & Gordon 1985; Perri et al 1984; Supnick & Colletti 1984). Abrams et al (1987) showed that quitters cope better than nonquitters with relapses involving intrapersonal (negative mood) smoking-specific situations; thus differences in coping skills exist between individuals who are successful and those who are unsuccessful in controlling substance abuse. Substance use may directly affect coping processes by reducing stress. Alternatively, coping processes may involve increases in social skills for dealing with social influences on substance use (Biglan et al 1985). Another possibility is that coping processes
are related to third variables, such as self-esteem, and influence substance abuse in this way.

Health-Promoting Behaviors

Individuals may engage in consistent clusters of positive health practices. The belief in a pattern of associations has led to the hypothesis that positive health behavior is part of an overall style of living reflecting the ability to anticipate problems, mobilize to meet them, and cope actively (Mechanic & Cleary 1980). However, in a review of the health behavior literature, Kirscht (1983) concluded that there is little evidence for a health-protective life-style. Studies have examined correlations between the ways people organize their thinking with respect to health and how they ultimately behave to protect their health, with modest findings (e.g. Harris & Guten 1979; Salovey et al 1987). These studies have focused on belief structures and behavioral intentions (Ajzen & Timko 1986), values (Kristiansen 1985, 1986), and current practice (Salovey et al 1987).

In addition to these intrapersonal motivators of health-protective behaviors, interpersonal variables are also important. This view is best illustrated by the impressive data from community based interventions, which combine state of the art communication and educational strategies with community organization and social support to encourage health-protective behaviors across an entire community. The two earliest were the North Karelia project (see Puska 1984 for a review) and the Stanford Three Community Project (see Farquhar 1984 for review). Studies in Rhode Island, Minnesota, and Belgium are still collecting data (see Matarazzo et al 1984).

The role of social support in promoting health behaviors is complex. While some data indicate a relationship between perceived level of support (especially from significant others) and long-term maintenance of positive health behaviors (e.g. Mermelstein et al 1986), several experimental attempts to enhance social support (e.g. by actively involving partners and instructing them in useful supportive skills) have proved unsuccessful (Lichtenstein et al 1986; Colletti & Brownell 1982; Cohen et al 1987).

An entire volume devoted to behavioral health (Matarazzo et al 1984) considered exercise, healthful diet, dental health, and safety. The determinants of these health-promoting practices remain unclear. Nowhere is this truer than about exercise. Only 15% of the general population is highly active, and as much as 70% of the entire population can be characterized as inactive. Fewer than 30% of those volunteering to engage in exercise programs continue to be active over a mean of 3.5 years (see Oldridge 1984 for review). Even less encouraging is the rate of recruitment of subjects into exercise programs. In employee-based exercise programs, approximately 30% or fewer of the potential population actually volunteer (Cox et al 1981). However, as many as 25% of these volunteers report themselves to be regularly active at the time of
volunteering (Shephard et al. 1981). Participation in employee-based exercise programs has been shown to influence other health-relevant behaviors, resulting in fewer sick days and lower health care costs (Der-Karabetian & Gebhart 1986).

Some have argued that the major problems with poor exercise adherence rates are similar to those for all formal health-behavior programs and stem from poor recruitment, marketing, and motivational strategies (Morgan 1977). Sensenig & Cialdini (1984) suggest that a few basic social psychological principles account for most specific compliance tactics observed in commercial settings, and these could be applied to the health behavior domain to increase adherence. These principles include (a) commitment induced via the pressure for consistency; (b) the use of social evidence to convince target persons to behave in desired ways (social validation); (c) high source credibility; and (d) reciprocation (e.g. rewarding adherence by providing something in return). It is worth noting however, that quite different factors may influence a person’s decision to attempt an exercise regimen, determine ongoing participation in the exercise program, and affect the decision to continue to exercise once a formal program is completed (Fontana et al. 1986).

Several behaviors believed to be health promoting also have potential health damaging consequences. For example, smoking cessation leads to weight gain (Hall et al. 1986; Rodin 1987). This negative consequence is the second most widely cited reason in the Surgeon General’s Report that people give for not trying to stop smoking. Rodin & Wack (1984) have reviewed the major pathways by which weight gain may occur, including metabolic changes caused by smoking cessation and increased caloric intake. Rodin (1987) recently demonstrated that ex-smokers are especially responsive to sweet taste, which is consistent with observations in experimental studies with animals (Grunberg et al. 1985).

Another example of a paradoxical effect of health-promoting behavior is the recent demonstration that dieting itself may decrease metabolic rate, increase the uptake of fat into storage, and make people generally more food efficient (i.e. able to exist on fewer calories; Brownell et al. in press). This phenomenon appears to become increasingly severe with repeated cycles of gaining and losing weight (Brownell et al. 1986). After repeated cycles of gaining and losing weight, people and animals may weigh the same as when they started but be significantly fatter (i.e. their fat/lean body mass ratio has increased). In animals, at least, the effect appears stronger for females than for males (Brownell et al., in press).

Adherence

Issues of compliance have long been a focus of attention for medical personnel and health psychologists. One important development has been a shift from use of the term compliance, connoting patient obedience, to the term
adherence, implying voluntary effort by the individual (Kristeller & Rodin 1984; Turk et al 1986). This theoretical shift has led to different types of research questions as well, since adherence implies choice and mutuality in the planning and implementation of treatment. Levels of nonadherence vary considerably, depending on the treatment or health recommendation. The highest adherence is for treatments with direct medical procedures (e.g. an injection), high levels of monitoring, and acute onset. For example, Taylor et al. (1984a) reported a rate of 94% for cancer patients’ adherence to chemotherapy. In contrast, the lowest adherence rates occur with patients who have chronic disorders with no immediate discomfort or evident risk, when lifestyle changes are required, and when prevention instead of symptom reduction or cure is the desired outcome. Treatment nonadherence is not limited to medication. Problems of adherence have been noted for performance of numerous health behaviors such as wearing seat belts, reducing weight, and increasing exercise (cf Brownell et al 1986). In general, level of adherence is difficult to ascertain because adherence may be conceptualized and operationally defined in various ways (Meichenbaum & Turk 1987).

Meichenbaum & Turk (1987) have systematically reviewed research on the determinants of adherence. These include numerous patient variables (e.g. characteristics of the individual, competing ethnic folk concepts of disease and treatment, environments that support nonadherent behavior, and lack of resources). It has also been suggested that nonadherence may represent a person’s attempt to gain some control over an illness or treatment. In other circumstances, the decision not to adhere may be based on misunderstanding or inadequate information (Deaton 1985; Janis 1984). In addition, patient beliefs affect adherence (e.g. Buckalew & Sallis 1986).

Characteristics of the disease also affect adherence. For example, the more complex the demands of the treatment, the poorer the rates of adherence (e.g. Glasgow et al 1986). The intrusiveness and duration of the regimen are also important variables in this category (e.g. Meichenbaum & Turk 1987). Issues such as inconvenience of the behavior or treatment, inadequate supervision, and side effects also strongly influence the likelihood of adherence. Finally, relationship variables, particularly in the health-care setting, correlate strongly with rates of adherence (e.g. poor communication or rapport; Janis 1984).

MOTIVATORS

Stress

Stress calls into play dispositional variables, appraisal processes, coping mechanisms, reliance on social support, use of substances, and the like (Elliott & Eisdorfer 1982). In many cases, stress may damage health through a combination of these processes. For example, Type A behavior leads to
significantly greater cardiac reactivity under certain kinds of stress that are most meaningful to the Type A individual (Matthews et al 1986b). To take another example, the combination of stress and cigarette smoking may have a greater than additive effect on blood pressure and heart rate responses (Dembroski et al 1985; Pomerleau & Pomerleau, in press). Similarly, consumption of caffeine under conditions of stress affects blood pressure, plasma cortisol level, and serum cholesterol, especially among borderline hypertensives (Pincomb & Lovallo 1987; Goldstein & Shapiro in press).

Controversy has surrounded whether it is useful to define stress in terms of stimuli external to the organism (Selye 1974) or as an interaction among an individual’s appraisal processes, reactions, and an external event (Lazarus & Folkman 1984b; see Krantz et al 1985 for a review of this controversy). Large-scale efforts to resolve the issue (Elliott & Eisdorfer 1982) have had little impact (Engel 1986). However, we now know that the objective nature of the stressor is less significant for health outcomes than the person’s perceptions of the stress. Reactions to the same stressor (e.g. the death of a spouse; Osterweis et al 1984) vary with the meaning of this stressor for the bereaved.

Another shift in the study of stress is toward the investigation of daily hassles rather than catastrophic life events. Hassles are experiences and conditions of daily living appraised as salient and harmful or threatening to well-being (Lazarus 1984). Lazarus suggested that daily hassles show a stronger relationship to health-relevant variables such as psychological distress and physical symptom reporting than do stressful life events. Major life events may affect health by creating new hassles or increasing the intensity of existing ones (Osterweis et al 1984). However, in such studies subjective stress is often measured in terms of health-relevant annoyances, confounding the operationalization of stress and the health-relevant results of stress (Lazarus et al 1985).

Stress effects on pathophysiological processes have been widely studied (reviewed in Miller 1983). Earlier work focused on corticosteroids and sympathetic nervous system variables; more recent research has examined immune system responses as well (reviewed in Justice 1985; Krantz et al 1985). The controllability of the stressor determines whether or not stress has physiological effects (e.g. Hanson et al 1976; Laudenslager et al 1983). Even variables such as tumor growth and proliferation may be influenced by whether or not a stressor is controllable (Visintainer et al 1982). Use of the triadic design (Seligman 1975) in these newer studies allows separation of the controllability of the stressor from the intensity of the noxious stimulation. Many other physiological systems (including such hormones as testosterone and prolactin, and peptides such as the endorphins and more central immune parameters) also merit further investigation in this context.
Being Sick: The Impact of Illness

The initial illness episode involves the self-evaluation of health status as a reaction to the experience of unusual symptoms (Feist & Brannon 1988). Nerenz & Leventhal (1983) described a theory of how individuals adapt to acute illness episodes, which includes attributional search, potential dysphoria, and preoccupation with bodily experience. Illness-appraisal behaviors include information seeking, social comparison, and even denial. These behaviors contribute to “appraisal delay,” the time it takes an individual to determine that new symptoms and sensations indicate illness. Other forms of delay, later in the process, include (a) the time between the recognition that one is ill and the decision to get medical help (illness delay), and (b) the time that elapses between the decision to seek treatment and one’s entrance into the medical-care system (utilization delay).

These illness behaviors (determining health status, deciding to seek treatment, and actually obtaining treatment) are related to a variety of variables: gender (Edlin & Golanty 1988; Rosenstock & Kirscht 1979); age (Prohaska et al 1987); socioeconomic status, ethnicity, cultural attitudes toward illness and pain (Mechanic 1978); and the salience of the symptom itself (DiMatteo & DiNicola 1982; Mechanic 1979). The individual’s reactions to medical treatment are not well predicted by severity of illness or actual diagnosis, but rather by the individual’s stance with regard to the medical system (e.g., positive vs negative affect; perceived physician support) (Ben-Sira 1980; Felton et al 1984).

Once people have an illness, the variables reviewed thus far affect subsequent adjustment. For example, in a study of spinal-cord-injured persons, the psychological well-being of patients who reported high levels of social support and satisfaction with their social contacts was as high as that of healthy individuals (Schulz & Decker 1985). The literature on successful adjustment to cancer is burgeoning. The cancer patient must learn to cope with problems ranging from the side effects of radiation therapy (Andersen 1985; Andersen & Karlsson 1986; to sexual dysfunction (Andersen 1985; Andersen et al 1986). Successful adjustment, at least among breast cancer patients, is often better predicted by prognosis and the invasiveness of treatment than by degree of disability. The impact of prognosis on adjustment seems to be mediated by beliefs about one’s current control over the cancer (Taylor et al 1984b), while the effect of surgery seems to be mediated by the patient’s sense of disfigurement and by changes in the sexual and affectional patterns in marriage (Taylor et al 1985). Cancer patients often compare themselves to others worse off in order to bolster how they feel about their own progress (Molleman et al 1986; Wood et al 1985).

A growing literature reveals that daily caregiving to a chronically ill person usually depresses and dissatisfies the caregiver (Schulz et al 1987). Much
attention has been focused on caregivers of patients with dementing diseases (Maletta & Hepburn 1986; Scott et al 1986). Moreover, family interactions are profoundly affected by the diagnosis of chronic illness in children (Hauser & Jacobson 1986; Walker et al 1987). Family members tend to become overinvolved in the chronically ill person's problem, producing negative consequences for family functioning and the illness itself.

Chronic illness challenges the family. Although the response to this challenge can be dissolution or distress, it is often an increase in cohesiveness (Boss 1988; Leventhal et al 1985). Illness has less impact on well-organized families in which individual goals and ambitions are less important than family goals, a setting that promotes effective problem-solving and coping (Lewis et al 1976; Leventhal et al 1985).

DISEASE END POINTS

Most of the earlier studies in this area (see Krantz et al 1985) were characterized by a "one variable, one outcome" approach in which a single antecedent variable was correlated with the presence or absence of a disease. Investigators then sought to understand the pathophysiological mechanism by which that psychosocial variable influenced the disease process. This approach has been followed most strongly in the area of personality variable effects on disease. For example, Booth-Kewley & Friedman (1987) reviewed the evidence for associations between personality variables (e.g. components of Type A, anxiety, depression, anger, and extraversion) and CHD in a meta-analysis of 83 studies.

Another approach has been to examine interactions among sets of variables. This work is based on several kinds of hypotheses. The most straightforward view holds that since disease is multiply determined, multifactorial approaches are appropriate to the prediction of disease outcome. More interesting models, however, conceptualize interaction effects as the most important predictors. So, for example, recent studies consider the stress-buffering effects of social support or the role of dispositional variables in influencing health practices under certain conditions (e.g. high stress) or in individuals of a certain gender or age.

Cancer

Central nervous, endocrine, and immune systems form a complex regulatory homeostatic network (e.g. Blalock 1984; Roszman et al 1985; Irwin et al 1987; Besedovsky et al 1986). Therefore, for tumors that are also at least partially under hormonal and/or immunological control, known biological pathways make it appropriate to ask whether psychosocial variables can influence tumor outcomes. Levy (1985) has noted that only certain tumors are
relevant when considering behavior as a potential modifier of biological responses. The study of indolent tumors with a variable time course for recurrence and subsequent death is most appropriate (e.g. breast cancer and melanoma). More virulent malignancies (e.g. pancreatic or primary lung cancers, with their rapid and aggressive course) leave little time for the host's behavior to play much of a role in progression and outcome.

Levy (1985) noted that behaviors can have both direct and indirect effects on the initiation of cancer and on the progression of the disease. The most important direct behavioral cause of disease initiation is tobacco use. Doll & Peto (1981) estimated that in the United States in 1981, tobacco caused 130,000–140,000 deaths, accounting for 33% of all cancer deaths during the 1980s. Alcohol consumption and occupational exposure to carcinogens also produce direct effects.

Diet and sexual activities indirectly affect the initiation of cancer. Dietary fat may increase cancer risk, and dietary fiber may lower it (Doll & Peto 1981; Boyd 1985; Bright-See & Levy 1985). It has also been suggested (Ames 1983) that even natural foodstuffs expose people to a large variety of naturally derived mutagens and carcinogens.

Nowhere has there been greater attention to the role of behavior in the initiation of disease than in the discussion of the causes and transmission of Acquired Immune Deficiency Syndrome (AIDS). Behaviors involving intravenous drug abuse and unsafe sex practices account for most cases of AIDS (Kelly & St. Lawrence 1988). The incidence of cervical cancer correlates with the character of sexual intercourse (e.g. age of onset, number of sexual partners, etc; American Cancer Society 1985). Moreover, interactions with other psychosocial variables such as race and socioeconomic status are notable. For example, the incidence of cancer of the cervix is highest in low-income groups (Hulka 1982). This social class distinction, of course, may be a proxy for more specific risks generated by particular activities within these groups (Hulka 1982). It is also possible that higher levels of stress accompanied by poverty in this group enhance the risk potential. For example, susceptibility to infectious disease such as herpes simplex virus II has been linked to environmental stress and poor coping ability (Jemmott & Locke 1984). There is strong evidence that cervical cancer is a viral disease with herpes simplex playing a vital role (Graham et al 1982).

Moving next to behavioral and psychosocial variables that may have a direct effect on the progression of disease, social support has been studied in cancer patient samples, and investigators have now begun to suggest a biological advantage of this support (Levy 1982). Second, in an earlier review of this area, Cox & MacKay (1982) concluded that the inability to express emotion was likely the most significant factor related to cancer progression (see also Temoshok 1985, 1987). Jensen (1987), for example, reported that
breast cancer patients who reported a repressive, defensive coping style had worse outcomes. A third theme is related to the variable termed helplessness (or hopelessness, a related, but not identical concept; Greer et al 1985; Jensen 1987), although negative findings have also been reported (Cassilith et al 1985, 1987; Jamison et al 1987).

In general, if any or all of these elements—inadequate social support, lack of negative emotional expression, and helplessness or pessimism—play a role in biological vulnerability to cancer or in cancer progression, multiple causal pathways are possible. As Levy (1985) suggests, social support might facilitate stress reduction if homeostatic, regulatory neuroendocrine, and immune function are relatively buffered from severe chronic stress effects. Social factors could also act directly on emotional expressiveness (e.g. Lehman et al 1986).

Finally, there are potential indirect effects of behavior on cancer progression. For example, screening and detection behaviors, and factors relevant to noncompliance and delay, certainly influence cancer progression (Timko & Janoff-Bulman 1985).

**CHD**

Numerous personality and environmental variables appear involved in the pathogenesis and course of CHD. We attempted to divide this literature into the same four-cell framework suggested by Levy (1985) for cancer and found that while studies could be categorized as specifying the direct versus indirect effects of psychosocial variables, they could not be divided so clearly into those related to the cause versus progression of CHD. It is possible that this distinction has not been made because the pathophysiology of the various forms of CHD depend on the same mechanisms for incidence as they do for progression. In addition, risk factors differ for first CHD episodes vs later events (Matthews, in press). It is also possible that this distinction has not been made as clearly because certain forms of heart disease are risk factors for other forms of heart disease. Alternatively, it may simply be that investigators of behavioral factors relevant to CHD have not distinguished as clearly between etiological factors and those involved in the worsening of the disease.

Behavior can increase the role of physical risk factors. For example, salt intake directly affects blood pressure levels, and diet helps to regulate serum cholesterol levels. There are also, however, indirect effects involving these risk factors. For example, cigarette smoking is a behavioral risk factor itself undoubtedly brought about by psychosocial risk factors (Leventhal & Cleary 1980).

Most work on the role of psychosocial variables in CHD has focused on their indirect effects. Specific psychological factors influence neuroendocrine
and autonomic nervous system function. In particular, new situations and
tasks that exceed perceptions of self-confidence or elicit intense sustained
efforts to cope increase neuroendocrine response (Herd 1986; Rosenman, in
press). The most notable examples, of course, come from studies of the
TABP. Extremely challenging situations with a component of time urgency
elicit sympathetic adrenal medullary responses. Additionally, increased levels
of circulating catecholamines, which are elevated in individuals with certain
elements of the TABP (Glass et al 1980), may affect coronary atherogenesis
through influences on platelet aggregation and on the mobilization of serum
lipids (Schneiderman 1983; Haft 1974).

While it has been suggested the aspects of the TABP are related to
increased psychophysiological responses (Houston et al, in press; Ward et al
1986), Suls & Sanders (in press) found that studies comparing systolic and
diastolic blood pressure in Type A and B individuals obtained various results.
Furthermore, Manuck & Krantz (1986) note little evidence that a physiologic
hyper-reactivity to behavioral stimuli contributes appreciably to the etiology
of CHD or essential hypertension. They argue that it would be premature
to consider reactivity as a proven risk factor. More likely, reactivity operates
in interaction with stress, personality attributes, and consummatory behaviors
in its effects on CHD (Dembroski 1986). For example, smoking, stress, and
reactivity may interact in their effects on heart disease. The cardiovascular
response is larger when people smoke during stress than when they smoke
while relaxed (Epstein & Jennings 1986).

It has also been suggested (Smith & Rhodewalt 1986) that Type As,
through their choices, appraisals, and self-evaluations, actively operate on
their environments in ways that influence the frequency, duration, and inten-
sity of stressors, and, as a result, episodes of cardiovascular reactivity.
Blumenthal et al (1987) show that the presence of social support reduces the
risk of significant coronary artery disease among Type A individuals. Since
heart rate may be attenuated by social closeness (Manuck et al 1983), Type As
who avoid social contact may be deprived of psychological benefits.

Recent meta-analyses (Booth-Kewley & Friedman 1987; Matthews, in
press) have focused on the more general relationship between components of
the TABP and various types of heart disease. Booth-Kewley & Friedman
posit a coronary prone personality that encompasses time urgency, hostility,
and competitiveness but also includes aggression and depression. Matthews’s
more conservative meta-analytic approach finds that Type A/CHD associa-
tions are only significant in population-based studies and in studies using the
structured interview assessment of Type A.

Despite data suggesting that alteration of the TABP significantly reduces
incidence and recurrence of myocardial infarction (MI) (Friedman et al 1986;
Powell et al 1984) and possibly serum cholesterol as well (Gill et al 1985), a
number of recent studies have failed to find a significant relationship between measures of Type A behavior and new (Shekelle et al. 1985) or recurrent (Case et al. 1985; Shekelle et al. 1985) cases of MI. One reason for the inconsistent pattern is that some components of the TABP may be more pathogenic than others (Dembroski 1986). A second is the potential importance of environmental influences (Blumenthal et al. 1987). Finally, the determinants of survival following MI are quite different from the determinants of an initial MI (see Matthews, in press for review). Indeed, while aspects of Type A behavior may increase the risk of heart disease, Type A’s are apparently less at risk for mortality after a heart attack (Ragland & Brand 1988).

Finally, many of the behavioral variables that influence disease outcome in individuals with CHD (e.g. changing exercise and diet, smoking cessation) are moderated by psychosocial variables. Changes in perceived efficacy regarding the likelihood of engaging in a new life-style, and social support for making these changes, strongly influence both physical risk factors (e.g. cholesterol) and recurrent MIs (Bandura 1988; Ewart et al. 1986; Taylor et al. 1985; O’Leary 1985a).

CONCLUSIONS

Despite the demonstration that altering psychosocial and behavioral variables may lessen disease (e.g. Friedman et al. 1986), the absence of disease does not necessarily equal good health. In both psychology and medicine, the major focus of research and theory has been on the abnormal; states of normality have been defined as the absence of pathology. This has led to narrow definitions and assumptions about the desirability of encouraging health promoting behaviors that may not in fact be healthy when one takes the whole person into account. For example, elite athletes are often used as examples of outstandingly healthy individuals. However, many such athletes attain excellence in one area by compromising other areas of their bodies or their lives. A notable example is the high incidence of anorexia nervosa and bulimia in athletes for whom low weight is mandated (Striegel-Moore et al. 1986). Similarly, longevity has been used as the dependent variable for many studies of health, in part because it is definitive and in part because it was assumed to be a valid outcome. Yet there is much debate over the value that should be placed on the quantity of life compared to the quality. Indeed many variables linked in epidemiological research to longevity—e.g. narcissism and mild paranoia—might not indicate good health by other definitions (Lieberman & Tobin 1983).

Studies both of individuals’ subjective perceptions of their own health and of the personal, environmental, and social determinants of these perceptions...
suggest that people's sense of their own health is not only a reflection of their psychological and physical well-being but also a predictor of subsequent physical health. This relationship holds even when diagnosed illness and major risk factors for illness are statistically controlled (Kaplan & Camacho 1983). How these perceptions and other cognitive processes relate to health and illness remains to be understood.

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