Person Variables and Health: Personality Predispositions and Acute Psychological States as Shared Determinants for Disease

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This article reviews prospective evidence linking certain classes of person variables to multiple disease end points. Included in the review is a consideration of the effects of hostility and anger, emotional suppression, depression, fatalism, and pessimism on coronary heart disease, cancer, and acquired immunodeficiency syndrome. A model is presented that integrates several of these variables into an overall conceptual scheme. In addition, several variables are identified that appear to moderate the strength of the relationships that are found between person variables and health. The article concludes with some suggested directions for future research.

Key words: Personality, acute psychological states, physical health.

INTRODUCTION

The idea that person variables can influence particular disease outcomes is becoming more commonplace. The idea that the same person variable can affect multiple disease outcomes is considerably more controversial. The primary purpose of present article is to explore this possibility more fully. In it, we review the evidence linking personality predispositions and acute psychological states to multiple disease outcomes.

It may be useful at the outset to define the domain of inquiry and to describe how potentially relevant articles were located and selected for review. Perhaps the easiest way of defining the domain of inquiry is to describe what we mean by the term "person variables," which appears in the title of this article. The term person variables is used to represent a superordinate category of constructs encompassing two different classes of variables. On the one hand, there are personality predispositions, i.e., the kinds of things that people traditionally have in mind when they talk about personality traits, characteristics, and dimensions. Personality predispositions represent cognitive, affective, or behavioral tendencies on the part of a person that are relatively stable across time and context. They refer to enduring characteristics that help to define a person's identity and help to distinguish one person from another. Personality predispositions make up one class of person variables that are relevant to the present review.

In addition to these more stable attributes, people also experience a variety of transient psychological states, e.g., fear, fatigue, and depression. These states may last only a few seconds, or they might persist for months or more. Regardless, they are less stable and less enduring than are personality predispositions, which can remain relatively unchanged for very long periods of time (1). We might note that these acute psychological states often have a more stable predispositional counterpart associated with them. For example, one can be the kind of person who is generally and chronically anxious, or one can be in a momentary state of anxiety (2). The difference is that the personality predisposition remains a relatively constant source of influence, whereas the acute state comes and goes more readily. Acute psychological states make up the second class of person variables of interest.

Person variables of the type just described constitute the psychosocial side of the ledger. What about the medical side? What defines the domain of inquiry with respect to illness outcomes? Clearly, a review encompassing all illnesses is too large to be managed effectively without reducing its scope in some way. To this end, we limited the search to articles dealing with coronary heart disease (CHD), cancer, or acquired immunodeficiency syndrome (AIDS). In addition, studies in these three areas were limited to those that assessed more objective disease markers, i.e., incidence, disease progression, or mortality rate. Admittedly, selection of these illness categories was somewhat arbitrary. In part, they were chosen because they are among the ones that are most vigorously being researched at the present

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time. They were also chosen, in part, because each represents a health problem of significant proportion.

We should also say something at the outset about how we went about locating relevant articles. The search strategy we used proceeded along three fronts simultaneously. One tactic was to identify variables for which connections with specific diseases were known to exist and then examine other bodies of literature to determine whether the same variables had effects in other domains. In addition, we systematically searched the available electronic databases for relevant articles, most notably, *PsychLit* and *Medline*. Finally, we engaged in archival, retrospective searches of the literature, using as starting points reference lists contained in recent articles. That is, when a relevant article was found, the reference list for that article was screened for other earlier relevant work. These earlier articles were then located and searched in turn for other, even earlier articles of interest, and so on back through the literature.

Given the nature of the topic and the breadth of the search strategy, it should not be surprising that an enormous body of potentially relevant literature was initially identified. To give some indication of the size of this literature, as part of the search process, *PsychLit* was scanned for several different topics, covering the period from January 1974 through December of 1993. One of these searches, using "depression" as the key word, yielded 33,990 citations. A second search, using "cancer" as the key word, yielded 3569 citations. A third search, using both depression and cancer together, yielded 427 references.

To reduce further the number of studies for review, two additional selection criteria were used. First, the study had to have a prospective design. Most studies linking person variables to health have been cross-sectional in nature. Cross-sectional studies were eliminated because of the inherent ambiguity that they contain regarding direction of causation. We might note that adoption of this prospective criterion had a tremendous effect on reducing the size of the eligible literature. For example, when "prospective" was added to the *PsychLit* search containing the joint key words depression and cancer, the yield of citations dropped from 427 to nine (and not even all of these nine articles were relevant because some sought to examine the prospective effect of cancer on depression and not the prospective effect of depression on cancer).

The second methodological criterion had to do with the control of relevant medical factors. To be included in the review, the study had to incorporate appropriate medical controls. In practice, this usually meant assessing the person's standing on the relevant factors and then controlling statistically for the effect of those factors in subsequent analyses, or showing that the relevant factors were independent of the particular person variable of interest. In addition, some of the studies selected for review satisfied the control criterion by identifying participants who were similar on the relevant medical factor or factors at baseline, e.g., selecting participants who were uniformly healthy at study intake.

VARIABLES IMPLICATED IN MULTIPLE DISEASE OUTCOMES

Our qualitative review of the literature revealed four clusters of variables that seemed to be implicated in multiple disease outcomes. The first cluster of variables has to do with the experience of anger and hostility. The second cluster of variables has to do with emotional suppression. The third cluster of variables has to do with the experience of depression and depressed affect. The final cluster of variables has to do with the adoption of a pessimistic or fatalistic attitude toward life and health. The evidence linking these four clusters of variables to multiple health outcomes is discussed in turn. We begin with the cluster for which the links to one particular health outcome is the clearest. The cluster has to do with the experience of anger and hostility. The particular link has to do with CHD and its manifestations.

Anger and Hostility

CHD. Interest in hostility as a risk factor for CHD grew out of earlier work on the Type A behavior pattern (3). This interest can be traced back to pioneering work by Matthews et al. (4), who reanalyzed data from the Western Collaborative Group Study to identify the variables (derived from the Type A structured interview) that were most predictive of CHD. The variables providing the best discrimination between cases and controls included ratings of potential for hostility, anger directed outward, frequent experience of anger, and irritation when waiting in lines (5).

There is now a host of prospective evidence linking anger and hostility to various aspects of CHD (6). For example, Barefoot et al. (7) examined the relationship between scores on the Cook and Medley (8) Hostility Scale (Ho) and 25-year CHD incidence among a group of 255 men who, in the 1950s, had

taken the Minnesota Multiphasic Personality Inventory (MMPI) while in medical school. They found that those with Ho scores above the median had nearly five times the incidence of clinical CHD events as those below the median. Moreover, the relationship between Ho scores and CHD incidence was statistically independent of hypertensive status and appeared to be unconfounded with age and other traditional CHD risk factors such as family history. In a follow-up study with lawyers, Barefoot et al. (9) found that Ho scores were associated with reduced survival over a 28-year period. Conceptually similar findings have been reported repeatedly by others (4, 5, 7, 10–12).

Studies that examine CHD incidence and clinical CHD events (7, 11) often combine CHD death with other outcomes (e.g., myocardial infarction or angina pectoris) into an overall index, which makes it difficult to evaluate the independent effects of hostility on CHD morbidity and mortality rates. In this regard, we could find only two studies that successfully linked hostility to the CHD mortality rate when the CHD mortality rate was assessed by itself, uncombined with other factors. In reanalyses of the Western Electric Study, Almada et al. (13) found that scores on the MMPI Cynicism Factor, the items of which overlap substantially with Ho items, were an independent predictor of the 25-year cardiac mortality rate among a group of 1871 middle-aged men. This association was independent of age, smoking status, alcohol use, blood pressure, and serum cholesterol. Similarly, Koskenvuo et al. (14) found that hostility (assessed by a three-item scale that reflected argumentiveness, irritation, and proneness to anger) was associated with higher CHD mortality rates in a large sample of Finnish men.

In addition to its association with CHD incidence, events, and mortality rate, hostility has also been associated with factors that predict CHD risk. For example, Siegler et al. (15) examined the ability of Ho scores to predict a host of coronary risk factors over a 21- to 23-year period. Although the effect sizes were not large, in a sample of 4710 men and women, Ho scores measured in college predicted greater caffeine consumption, a larger body mass index, greater likelihood of being a current smoker, a larger lipid ratio, and more hours of exercise at follow-up (11).

Taken together these findings provide rather compelling evidence linking hostility to various aspects of CHD. Although a number of negative studies have been reported (16–19), the number of positive studies outnumber those with no effects. Several years ago, Matthews (3) concluded her meta-analytic review of prospective studies by saying that hostility was a significant risk factor for CHD. Since then, the balance of positive to negative studies has remained fairly constant. As a result, it should not be surprising that Adler and Matthews (20) recently came to the same conclusion regarding the importance of hostility, anger, and anger expression as etiological factors in CHD.

Other Health Outcomes. When one examines health outcomes other than CHD, links to hostility become more tenuous. Perhaps the best evidence implicating hostility to other disease end points comes from studies that assess its relationship to all-cause mortality. A number of studies examined the association between hostility and all-cause mortality rate and reported positive associations (9, 13, 14, 21). There is a problem interpreting most of these studies, however, in that the CHD mortality rate is not controlled when the effects of hostility on the all-cause mortality rate is evaluated. Thus, it often remains unclear whether hostility predicts the all-cause mortality rate, independent of its association with CHD death.

We could find only one study that examined the all-cause mortality rate and controlled for CHD deaths. Shekelle et al. (11) found a significant positive association between Ho scores and 20-year crude mortality rates from all causes, excluding cardiovascular-renal diseases and malignant neoplasms. This association remained significant even after the data were adjusted for major CHD risk factors. Thus, there is some rigorous evidence that hostility might have an impact on diseases other than those that are CHD related.

Interestingly, the Shekelle et al. (11) study provides some suggestive evidence that hostility may also be related to cancer deaths. In their study, Ho scores were also significantly related in a positive direction with 20-year crude mortality rates caused by malignant neoplasms. However, when those data were adjusted for major CHD risk factors (including smoking), the significance of the association became marginal. Thus, until the effect is replicated, the link between hostility and cancer death should be considered somewhat tenuous.

With the exception of these few studies on all-cause and cancer mortality rates, we could find no other studies that linked hostility to health outcomes other than those relating to CHD. It remains an open question whether associations between hostility and other health outcomes will continue to emerge in the future.

Emotional Suppression

The preceding section focused on the experience of emotion, specifically anger and hostility, and its role in the development and progression of disease. The simple experience of anger or hostility, however, may not be the only aspect of emotional experience that has disease-related consequences. The degree to which emotion, particularly negative emotion, is expressed may also play an important role as a cause of disease (22–25). Of particular interest in this regard is the role of suppressed expression of emotion. Indeed, a central feature of what has been coined the Type C or cancer-prone personality (26) is an exaggerated suppression of the expression of negative emotion such as anger. Research addressing the consequences of suppressed emotion has begun to suggest that it might be associated with the development of disease.

Cancer. In a quasiprospective study of 160 women hospitalized for breast biopsy, Greer and Morris (27) found that an abnormal release of emotion, assessed the day before biopsy and corroborated through interviews with husbands or close relatives, was significantly associated with subsequent diagnoses of breast cancer. Specifically, the 69 women who were diagnosed with breast cancer demonstrated significantly higher levels of suppression of anger and of other feelings than did those who were diagnosed with benign breast disease. This effect, however, reached statistical significance only among women younger than 50 years of age. The same research group reported similar results with a second group of women with breast tumors (28). Conceptually similar results have also been provided by others (29–31).

Because the assessment of emotional suppression in these studies clearly occurred after the cancer had been established, it is a bit more difficult to establish causal relations, even though the patients were naive with respect to their diagnoses. For example, it is possible that the predisposition to emotional suppression and susceptibility to cancer both develop as a result of some other unmeasured third factor (perhaps biologic or genetic in origin). It is also possible that the patients with cancer were responding to some unspecified set of occult or prodromal symptoms and that it was this set of symptoms that caused them to suppress emotion in some way. In spite of these drawbacks, the consistency of the findings is noteworthy.

We should also mention in this context a set of two large-scale prospective studies done in Europe (32–34). One study involved 1353 participants and took place in Crevenka, a village in what was Yugoslavia. The second study involved 1026 participants and took place in Heidelberg, Germany. In both cases, a variety of psychosocial and medical variables were assessed in participants at baseline, and then their health status was tracked over a 10-year period. Results from these studies revealed that emotional suppression was one of the major psychosocial predictors of cancer incidence (35). We should also note, however, that the findings reported by Grossarth-Maticek, Eysenck and colleagues (32–34) are somewhat difficult to evaluate because the researchers tended to provide few details about questionnaires, statistical analyses, and other aspects of the protocol. Still, the findings that have emerged are consistent with the results reported by others on breast cancer (27–29, 31).[1]

CHD. The evidence linking suppressed emotion to disease has not been limited to cancer. Reporting data from the Framingham Study, Haynes et al. (36) found that suppressed hostility was a significant predictor of CHD incidence among white-collar men and working women. In this study, 1674 coronary disease-free men and women completed measures that assessed their manner of expressing or coping with anger. These included measures of anger in (keeping it to oneself), anger out (taking it out on others), and of discussing, anger with a friend. Follow-up 8 years later revealed that men younger than age 65 in whom CHD developed scored significantly lower on the anger-out scale than did disease-free men. Similarly, working women younger than age 65 in whom CHD developed scored significantly lower on the discussing-anger scale than did disease-free women. When the data were adjusted for standard CHD risk factors (i.e., blood pressure, serum cholesterol, and smoking), the association among women between not discussing anger and CHD remained significant, whereas the association among men between not expressing anger and CHD did not. Thus, anger suppression appears to be a CHD risk factor for women, but not for men.

Depression

Several studies have begun to document links between depression and physical health. Some of

[1] Consistent with the findings described in the previous section, these studies also provide evidence further linking hostility and anger to CHD-related illness and death.

these studies have gathered information that is relevant to clinical depression. Other studies are limited to a consideration of depressive mood and depressive symptoms because of the nature of the measures that were used or the analyses that were done. Inasmuch as these latter studies report significant effects, the available data suggest that depressed mood is sufficient in certain circumstances to induce poor health, even in the absence of clinical depression.

CHD. Data that establish associations between depression and health are clearest for various kinds of cardiac-related events. Ladwig et al. (37), for example, gathered psychological data from more than 500 male survivors of myocardial infarction (MI) during the 3rd week after the onset of the MI. The assessment battery included a composite measure of depression that consisted of several discrete markers, including self-reported emotional isolation, vulnerability, and manifest depression. At the time of the 6-month follow-up, there were 17 patients who had arrhythmic events and 12 who had suffered cardiac deaths. Both categories of events were significantly predicted by post-MI depression using univariate analyses. When the data were adjusted for clinical predictors of death, the association between depression and cardiac death remained marginally significant ($p < .07$). The association between depression and arrhythmias became nonsignificant.

Stronger findings relating depression to post-MI mortality rates have been reported by Fraser-Smith et al. (38), using a more standardized measure of depression. Fraser-Smith et al. interviewed more than 200 men and women, hospitalized for MI, during the week after the onset of the MI. Depression was assessed using a modified version of the National Institute of Mental Health Diagnostic Interview Schedule (39), the purpose of which is to allow trained lay interviewers to gather the information needed to provide reliable DSM-III-R psychiatric diagnoses. In addition, a host of clinical variables were assessed, including history of previous MI, left ventricular ejection fraction, Killip class (a clinical estimate of left ventricular function), frequency of preventricular contractions, thrombolytic treatment, and prescription of beta-adrenergic blockers. At 6 months, 12 deaths had occurred, all cardiac related. Depression was a significant predictor of the mortality rate. Patients who met criteria for clinical depression during the week after their MI were more than five times more likely to have died at the time of the 6-month follow-up. Moreover, the effect of depression remained significant in this case even after the data were adjusted for all significant clinical predictors of death, as determined in univariate analyses. Thus, depression seems to be an independent risk factor for death after MI (see Powell et al. (40), however, for a report of negative findings using a six-item unstandardized measure of clinical depression).

Depression has also been linked to the onset of new cardiac events. Wassertheil-Smoller et al. (41) examined the impact of depression on subsequent cardiovascular events in the Systolic Hypertension in the Elderly Program (SHEP), a multicenter placebo-controlled clinical trial of drug therapy in persons older than 60 years of age with isolated systolic hypertension. More than 4000 SHEP participants were followed an average of 4.5 years to assess the effects of low-dose drug therapy on the primary end point of stroke. Depression was measured at baseline using the Center for Epidemiologic Studies Depression Scale (42). Baseline depression was not related to subsequent cardiovascular events (i.e., MI, coronary artery bypass graft surgery (CABG), stroke, or endarterectomy), but change in depression was. Persons who experienced events had a significant increase in depression up to 6 months before the event compared with those with no events. This effect was significant, however, only for those participants in the placebo group. The effect for participants in the active group was in the same direction, but weaker.

Carney et al. (43) provided conceptually similar data on a group of patients with diabetes mellitus. Thirty-eight depressed and 32 nondepressed diabetic patients who were free of clinically apparent CHD at baseline were followed for 10 years. At the time of the 10-year follow-up, a significantly greater number of the depressed patients were found to have CHD. The only other significant predictors of CHD status at the time of the 10-year follow-up were age and hypertension status at baseline. Both of these variables were unrelated to baseline levels of depression. Taken together, these results suggest that depression may be an independent risk factor for CHD among patients with diabetes (44).

Human Immunodeficiency Virus (HIV) Disease Progression. Data are also beginning to emerge that suggest that depression may hasten HIV disease progression (as indexed by changes in CD4 lymphocyte counts). Burack et al. (45) studied 277 HIV-positive homosexual and bisexual men over a 5-year period. Depression was assessed in two ways, using the overall score on the Center for Epidemiologic Studies Depression Scale (CES-D) and a subscale of the CES-D that measured affective depression in which somatic items were removed. The unadjusted mean rate of CD4 change was 38% greater for sub-

jects classified as overall depressed (i.e., a CES-D score of 16 or higher) and 34% greater for subjects classified as affectively depressed (i.e., a score that was 1 SD above the mean). Both of these effects remained significant in multivariate analyses that controlled for antiretroviral medication, symptoms, and other baseline predictors of disease progression (e.g., age, absolute CD4 count, and hematocrit levels). Thus, overall depression and affective depression predicted a more rapid decline in CD4 lymphocyte counts, and this association seemed not to be attributable to baseline differences in health status.

In addition to the study by Burack et al. (45), there have been two other attempts to examine the effects of depression on HIV disease progression (46, 47), neither of which reported positive findings. In this regard, it is interesting to note that participants in both of the studies with no effects seemed to be in poorer health at baseline. This suggests the possibility that initial health status may moderate the effects of depression on HIV disease progression. We return to this issue more generally later in this article when we discuss moderators of relationships between person variables and health.

Pessimism and Fatalism

Pessimism refers to the tendency to expect negative outcomes in the future. Although fatalism formally refers to the belief that outcomes (both good and bad) are predetermined, in the context of existing research on illness and disease, it typically refers to the belief that the worst of all possible consequences will come to pass. As such, both pessimism and fatalism share a common core that involves negative expectations regarding future outcomes. Researchers have studied the effects of pessimism and fatalism in one of two ways. Either the variables are conceptualized as stable, enduring personality dispositions (48), or they are conceptualized as situation-specific attitudes toward particular contexts or particular health outcomes (49). Studies examining both types of variables simultaneously typically find a moderate correlation between the two (50), although sometimes the correlation is considerably lower (49). Other data suggest that the links between dispositional measures of fatalism and pessimism and their situational counterparts may become weaker over time as the person gains experience with a particular illness (51). Regardless of whether pessimism and fatalism are approached from a dispositional or a situational perspective, the emerging evidence is beginning to suggest that they have important implications for health.

AIDS. As part of the Multicenter AIDS Cohort Study, Reed et al. (49) examined the effect of what they called realistic acceptance on AIDS-related death. As used by Reed et al., realistic acceptance refers to a particular style of coping with AIDS, which is largely characterized by a sense of disease-specific pessimism/fatalism and resignation. In a sample of 74 male patients diagnosed with AIDS, endorsement of a fatalistic orientation toward their illness was inversely associated with survival time over the 50-month term of the study. The median survival time for participants low on realistic acceptance (i.e., fatalism) was 9 months greater than for participants high on realistic acceptance (i.e., fatalism). This effect was independent of self-reported health status, other styles of coping, established medical risk factors (e.g., age, CD4/CD8 ratio, and use of AZT, and health-related behavioral risk factors (e.g., smoking or use of marijuana or cocaine).

Cancer. Schulz et al. (52) followed a group of 238 patients with recurrent cancer for a period of 8 months. All patients were receiving palliative radiation treatment as a result of their cancer. At the time of the 8-month follow-up, a total of 70 patients had died. A pessimistic orientation, as assessed at baseline by the pessimism items of the Life Orientation Test (LOT) (48), was a significant predictor of death. However, this effect was true only among younger patients with recurrent cancer (age 30–59 years). The association between pessimism and death among the younger patients remained significant even after site of cancer and level of symptoms at baseline were controlled.

CHD. Scheier and Carver (50) studied the effects of dispositional pessimism on recovery from CABG surgery. Pessimism was assessed presurgically using the total score on the LOT, which combines both optimism and pessimism items into an overall index. They found evidence that pessimism was significantly associated with perioperative MI. Specifically, pessimists were significantly more likely than optimists to have developed new Q-waves on their electrocardiograms as a result of the surgery. Pessimist were also significantly more likely than optimists to have shown a clinically significant release of the enzyme aspartate aminotransferase. Both of these measures are widely taken as markers for MI. The data thus suggest that pessimists were significantly more likely than optimists to have had an infarct during surgery. The effect of pessimism on perioperative MI was also shown to be statistically independent of the extent of the patient's surgery

(i.e., the number of grafts performed), the severity of the patient's CHD (i.e., number of coronary arteries occluded 50% or more), and a composite index of coronary risk factors (composed of current smoking status, hypertensive status, and serum cholesterol level).

The Dynamics of Depression, Fatalism, and Pessimism: A Theoretical Integration

Depression, fatalism, and pessimism obviously share a variety of common features. For example, in the context of the research we reviewed, each is negative in tone, and each implies a certain bleakness about future possibilities. Given the conceptual overlap, it would be beneficial to have some means of integrating them into an overall conceptual scheme. In this regard, Scheier and Carver (53) proposed a model of motivation that encompasses all of these various elements.

A graphic representation of the model, adapted to the present context, can be found in Figure 1. As can be seen, the model takes as its starting point the idea that an individual's behavior is essentially goal directed. Goals, and the activities to which the goals give rise, are seen as providing the structure that defines people's lives, imbuing those lives with meaning, both in the long and short term. The goals and activities in question are not just limited to performance or achievement contexts. Most people have as an important life goal the maintenance of good health. Similarly, activities such as developing and maintaining good relationships can be seen as goal driven, as can relaxing by digging in the garden or playing tennis. Indeed, even routine activities of living such as going to the mall to shop for clothes or going to the service station to have the oil changed on the car can be seen as intrinsically goal directed.

As long as no obstacles or problems are encountered during the course of one's goal-directed efforts, life proceeds smoothly and comfortably. However, people do sometimes have difficulties attaining their goals. People cannot always do or achieve what they want. According to the model, people's responses to the adversity of goal disruption depends to a great extent on the nature of their expectations with respect to obtaining the goals toward which they are striving. People who expect to overcome the problems confronting them stay engaged in life's activities. In contrast, people who expect to be unsuccessful in the attainment of their goals disengage from their goal-directed efforts. As outlined in Figure 1, such disengagement is thought to manifest itself in

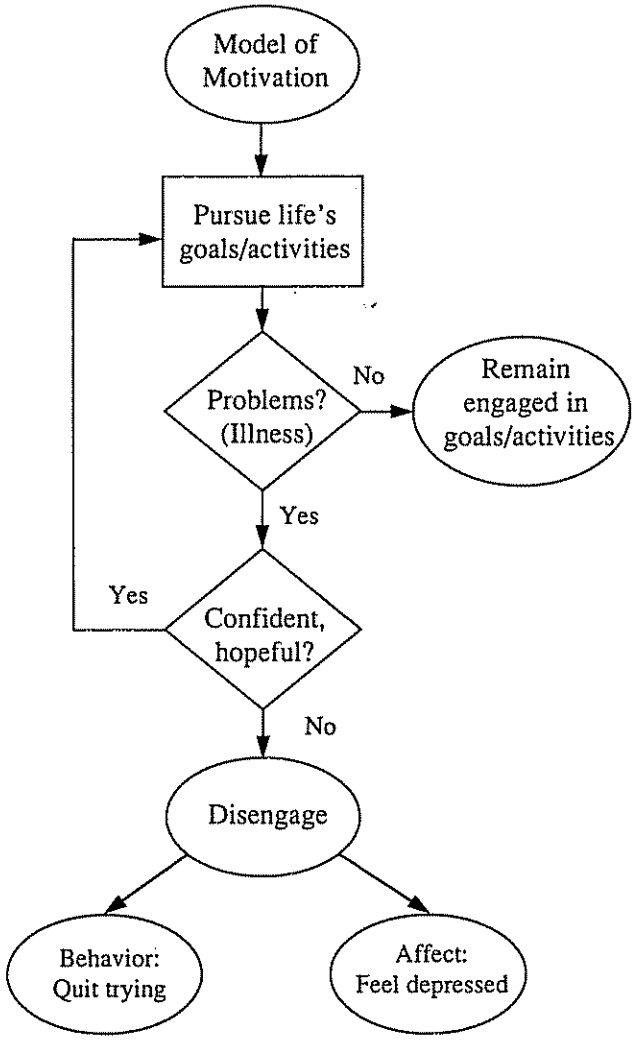

Fig. 1. Illness as a disrupter of life goals and activities.

two different ways. Behaviorally, people quit trying. They stop doing the things they were doing in the service of goal attainment. Affectively, people feel depressed. They become dysphoric over the realization that important goals will go unrealized.

The relevance of the model to the present context is straightforward. From this perspective, illness represents one general and significant class of events that can interfere with the pursuit of life's activities and goals, both those that are health related and those that are not. At one extreme, are the minor inconveniences of a muscle pull, sore throat, or cold, which can slow one down for a couple of days. At the other extreme, are the major disruptions caused by a life-threatening illness. Each can interfere, to a lesser or greater extent, with the general set of plans and activities that give a person's life its form and

meaning. Each can also interfere, to a lesser or greater extent, with the specific desire to feel well.

As we have seen, fatalism and pessimism are two variables that seem to have a profound effect on the manner in which a person responds to illness. In terms of the model, fatalism and pessimism have the effects that they do because of the negative expectancies to which they are attached. Fatalists and pessimists do not expect to get better or recapture old goals. Indeed, their fatalism and pessimism may also prevent them from targeting new goals scaled back to match their present circumstances. As such, they are more likely to withdraw prematurely from the activities of life and to give up on the hope of recovering health. The extracted cost is more rapid disease progression or death.

Depression from the present perspective is something of an epiphenomenon, reflecting the affective component of the disengagement process—a process that is set in motion by the negative expectations concerning eventual goal attainment. Although real, the depression derives from the realization that sought-after goals are no longer attainable and, as such, is secondary to the person's negative expectations. Consistent with this view that expectations are ascendant, there are data to suggest that pessimism is a prospective predictor of changes in depressive symptoms over time (54, 55).

Effects of Related Variables. The Scheier and Carver (53) model of motivation provides a theoretical framework for understanding the effects of depression, fatalism, and pessimism on health. In addition, the model provides a heuristic by which the effects of other variables might also be understood. Consider first the effects of active efforts to cope behaviorally with illness on disease progression and death. In terms of the model, active coping efforts represent the antithesis of disengagement and giving up. As such, efforts to remain engaged with the attempt to overcome one's illness should be beneficial.

In a study evaluating the effectiveness of a 6-week structured psychiatric group intervention, Fawzy et al. (56) found that lower baseline levels of active behavioral coping were predictive of higher rates of recurrence and death among 68 patients with malignant melanoma. Active behavioral coping, as assessed by the Dealing with Illness Coping Inventory (57), reflects a style of coping with illness characterized by behavioral engagement. Specifically, patients try to alter some aspect of their disease course by activities such as exercising, relaxation techniques, and frequent consultations with physicians. Interestingly, the effects of active behavioral coping on outcomes was independent of treatment group, with those demonstrating higher levels of active behavioral coping in both the treatment and control groups having the best health outcomes.

These findings sound similar conceptually to findings reported by Greer et al. (58) who examined recurrence and death among a group of patients with breast cancer. In an initial prospective study of 69 women with nonmetastatic breast cancer, Greer et al. found that those women who reacted with fighting spirit or denial, as assessed 3 months postoperatively, were significantly more likely to experience recurrence-free survival at the 5-year follow-up than those women who reacted with stoic acceptance (a kind of fatalism) or feelings of hopelessness and helplessness. A similar pattern of results emerged at the time of the 10-year follow-up (59). Moreover, at the 15-year follow-up, multivariate analysis revealed that those women who reacted with fighting spirit and denial were significantly more likely to be alive and recurrence-free than women who reacted with stoic acceptance or hopelessness and helplessness (60). Psychological responses at the 15-year follow-up were not associated with clinical stage, histological grade, mammographic appearance, or measures of tumor load.

Next, consider the work of Appels and Mulder (61) regarding a construct termed vital exhaustion, which overlaps considerably with many measures of depressive symptoms such as dysphoria, tiredness, hopelessness, and sleep disturbances. In a study that prospectively assessed 3877 men, these researchers found an association between vital exhaustion and angina and nonfatal acute MI (62–64). This effect was independent of standard coronary risk factors. From the current perspective, vital exhaustion might be viewed as a manifestation of the giving-up response. As such, signs of disengagement would once again be associated with poorer health outcomes.

Finally, it may also be possible to integrate aspects of the literature involving hostility, anger, and CHD (reviewed previously) into the model. This possibility rests on the observation that measures of hostility and anger often do not covary (6, 65). For example, clinical ratings of the potential for hostility have little or no association with Ho scores (66). Unlike the potential for hostility, Ho scores seem to tap a negative social attitude, feelings that others are generally nonsupportive and untrustworthy. Negative social expectations of this kind could easily feed into a sense of pessimism more generally (67). If so, they would represent yet another set of results that relate negative expectations to poor health outcomes.

TABLE 1. Studies Showing Positive Effects on Disease Categories by Person Variables

Person Variables	Outcomes			
	CHD-Related	Cancer-Related	AIDS-Related	All-Cause Deaths
Hostility/anger	4, 5, 7, 9–15, 21	11		7, 9, 11, 13, 14, 21
Suppression of emotion	36	26–35		
Disengagement cluster				
Depression	37, 38, 41, 43, 44		45	
Pessimism/fatalism	50	52	49, 51	
Fighting spirit/active coping		56, 58–60		
Vital exhaustion	61–64			

Summary of Current State of Knowledge

A summary of the studies linking particular person variables to multiple health outcomes can be found in Table 1. Presentation of the findings of our review in this fashion makes it easy to see where the clearest connections lie and where future work is needed. As can be seen, persuasive evidence exists associating hostility and anger to a variety of CHD-related health outcomes. Indeed, the evidence is stronger for this particular connection than for any other connection that was examined. Evidence relating hostility and anger to other health outcomes is considerably more meager, however, and more research is needed to determine how broad the effects of these variables might be.

The same is generally true for research that involves emotional suppression. There is some evidence linking emotional suppression to breast cancer incidence, but the research is either quasiprospective in design or is difficult to evaluate because of the brief descriptions that have been provided in published accounts of the studies. There is some evidence that suppressed emotionality might be associated with the development of CHD. Unfortunately, only one study shows this effect, and the effect was observed only in women. No study that we know of has linked suppressed hostility to any aspect of AIDS. Future research on emotional suppression might benefit from a consideration of research on alexithymia (which involves the absence of emotional expressivity). Although research on alexithymia has not been applied to the domain of physical health, the research areas are similar enough to each other conceptually that one might profitably be used to inform the other.

The evidence involving depression, fatalism, and pessimism is more impressive. As a group, these variables clearly showed the widest effects of any of the variables surveyed. Merging these variables with fighting spirit, active coping, and vital exhaustion into a "disengagement cluster" (as Table 1 does) yields even a wider set of effects. In absolute terms, although the number of available studies is still rather small, the initial set of findings for the variables in this cluster would seem to be suggestive enough to warrant further work along similar lines in the future.[2]

POTENTIAL MODERATORS OF PERSON VARIABLE-HEALTH RELATIONSHIPS

Our specific charge in conducting the present review was to locate studies that demonstrate positive associations between particular types of person variables and multiple health outcomes. As such, the review is somewhat biased toward studies showing significant relationships. Negative findings have been mentioned as they were encountered, but finding negative studies was not the intent. Moreover, negative studies are easy to miss because negative findings are rarely publicized. Variables that do not show significant associations rarely work themselves into the titles, abstracts, or key word lists of articles. This also made it easier to miss or overlook negative findings.

Still, negative findings were encountered. In reviewing the literature, it became apparent that the negative findings did not seem to be randomly distributed. Rather, negative findings were more likely to occur in certain types of studies than others using certain types of samples than others. In the next few paragraphs, we share some of the insights we obtained with respect to the nature of the variables that seemed to moderate the strength of the relationships between person variables and health. This discus-

[2] We might also note that there is an emerging prospective literature linking anxiety to changes in blood pressure (68) and incidence of hypertension (69). Although this is interesting, these studies fall outside the purview of the present article. As a result, they are not reviewed here.

sion is offered in part as an attempt to begin to understand why the negative effects might have occurred. It is also offered to sensitize future researchers to the potential importance of the variables involved.

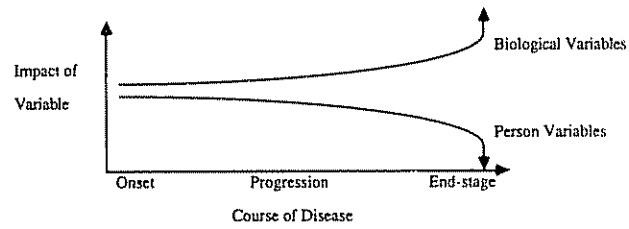

Fig 2 Changing impact of person variables and biological factors across the disease course

Age

Age is one variable that may moderate the strength of the associations between person variables and health. A number of studies were encountered, across a range of variables and outcomes, in which the effects of person variables were stronger for younger persons than for older persons. For example, Greer and Morris (27) report a positive association between emotional suppression and the development of breast tumors, but the effect was significant only for women younger than age 50. Similarly, Haynes et al. (36) found that the tendency to suppress talking about anger was positively related to the development of CHD, but only for women younger than 65. Finally, Schulz et al. (52) report that pessimism predicts the mortality rate among a group of patients with recurrent cancer, but only for patients between the age of 30 and 59, nor are these the only studies that report stronger associations for younger persons (10, 26). In brief, we encountered no study in which relationships between person variables and health were stronger for older than for younger persons. When differences were found, the favor was always in the direction of the younger age group. Thus, age seems to be systematically related to the strength of the effects that are observed.

It is less clear why these age differences have occurred. One possibility is simply that biological factors become more important in determining disease outcomes as people grow older, overshadowing the impact of psychosocial variables. Alternatively, it is possible that the wrong variables have been assessed. Perhaps a different set of psychosocial variables become important as the person ages. Finally, the problem may lie in the manner in which psychosocial factors are typically analyzed. Researchers often limit their analyses to main effects when assessing the effects of person variables on health. Perhaps such models are inappropriate or inadequate for elderly populations. Prediction might be enhanced if more complex models were tested that encompassed interactions between predictors. Indeed, the entire field might be enhanced if more complex predictive models were routinely tested.

Place in Disease Course

In a well-publicized and often-cited study, Cassileth et al. (70) examined relationships among several different psychosocial predictors, including several person variables, and both survival time and time to relapse among two groups of patients with cancer. No significant associations emerged. In this regard, it is interesting to note that the groups Casselith et al. were studying were composed of patients who were fairly ill. One group of patients consisted of persons with advanced malignant disease, who had an expected survival time of less than 1 year. The second group of patients consisted of a heterogeneous mix of persons with either a cutaneous malignant melanoma, which placed them in an intermediate- or high-risk group, or with Stage II breast cancer.

As suggested in Figure 2, the potency and importance of person variables (and other psychosocial factors) may vary, depending on where the patient is in the disease course. At disease onset, both person variables and biological factors may influence disease outcomes.[3] Over time, however, the influence of person variables may wane, whereas the influence of biological factors may increase. At the end stage of an illness, biological factors may completely determine, or almost completely determine, the course of the disease. If so, the effects of person variables might be expected to be more pronounced among samples that are healthier at study intake. This may

[3] We do not intend that Figure 2 should be overinterpreted. For simplicity, we have set the importance of biological and person variables to be at an equivalently moderate level at disease onset. One can imagine circumstances in which the potency of biological factors would be greater than person variables even in the early stages of an illness. Alternatively, one can imagine the effects of person variables being initially stronger. The point of the figure is simply to convey the idea that, as the disease experience unfolds, the effects of person variables become weaker at the same time that the effects of biological variables become stronger.

help to explain why it was that Cassileth et al. (70) did not find any significant associations.

One of the clearest examples of the importance of initial disease status comes from the previously cited research examining the effects of depression on HIV disease progression. Recall that Burack et al. (45) found a positive association between depression and illness progression, as indexed by changes in the level of CD4 count, whereas Lyketsos et al. (46) and Perry et al. (47) found no association between these variables. In this regard, it is noteworthy that participants in the Burack et al. study had the highest baseline level CD4 count of any group of participants. Indeed, the baseline level CD4 count for participants in the Burack et al. study was slightly more than 20% higher than the baseline level CD4 count for participants in the Lyketsos et al. study and nearly 50% higher than the baseline level CD4 count for participants in the Perry et al. study. Although these differences may be due to differences in laboratory protocol, the differences are also consistent with the idea that participants in the Burack et al. study were healthier at baseline.

To explore whether differences in baseline status might have contributed to the differences among these studies, Burack et al. (personal communication, 1994) went back to their dataset and performed the following secondary analysis. In brief, they divided their sample at the median baseline CD4 count. They then separately evaluated the relationship between depression and disease progression among participants in the two resulting groups. With other risk factors controlled, overall depression was significantly associated with decline in CD4 count for the group whose baseline CD4 count was above the median but not for the group whose baseline CD4 count was below the median. Stated differently, the association between depression and disease progression emerged only for participants who were healthier at study intake. Thus, it appears important to take initial health status into account when evaluating the effects of person variables on health.

Researchers should also be aware of the possibility that entirely different sets of person variables might become important in predicting illness, depending on where the patient is in the disease course. As the pathophysiology of the disease changes, so too might the nature of the person variables that have an impact on that disease also change. It would not be surprising to learn, for example, that the person variables that are involved in the development of a chronic disease such as CHD during adolescence and early adulthood are different from the person variables that contribute to fatal events in persons for whom the disease is already firmly established (71).

It is also conceivable that the effects of a single person variable will reverse from one point in the disease course to the next or as a function of whether the effects of the variable are assessed over the short or the long term. Perhaps the best illustration of this latter point comes from research that examined the effects of denial on medical outcomes after an acute coronary event. Recent research that involves acute coronary events shows that denial often produces more beneficial medical outcomes in the short run (72–74) but contributes to more negative medical outcomes in the long run (73). As a field, we need to do a more systematic job of tracking the impact of different person variables across different times in the disease course. Extant data suggests that person variables may interact in subtle and complex ways with the course of the person's disease.

Importance of Acute-Chronic Distinction

The last point that needs to be made with respect to moderating variables concerns the importance of the acute-chronic distinction. We began this article by drawing a distinction between personality predispositions and acute psychological states. Personality predispositions are stable and enduring. Acute psychological states are more transient and fleeting. In our view, it may not be reasonable to expect acute states to affect long-term outcomes. Unless an acute state recurs frequently over time, the putative characteristic simply may not be present or operative long enough to affect outcomes that take a long time to transpire. In contrast, predictors such as personality predispositions tend to be present for both the short and long run. It follows that they should affect all types of outcomes, i.e., those that occur quickly and those that take longer to develop.

The available data certainly support this general point of view. Take depression, for example, which would seem to represent an acute psychological state. It may last for months or even longer, but it rarely lasts forever. The kinds of disease outcomes to which depression has been linked are all ones that can occur quickly over time, e.g., progression of disease among HIV-positive men (45), onset of new CHD-related clinical events (41), and the 6-month mortality rate after acute MI (38). Inasmuch as a depressive episode might last most (if not all) of the time frame encompassed by these studies, it should not be surprising that depression can affect outcomes of this type.

The data are far less promising for outcomes that take longer to occur. Persky et al. (75), for example, provided initial prospective evidence that seemed to link depression to 10-year cancer incidence and 20-year cancer mortality rate. However, attempts to replicate these findings have not been generally successful (76–79). Thus, the most reasonable conclusion at present is that depression is not related to longer term outcomes such as cancer incidence and long-term cancer mortality rates.

Findings from other areas serve to reinforce this point. For example, vital exhaustion, a state-like variable, has been linked to the onset of angina and acute MI (61, 63), but not to CHD incidence. It does not take long to have a heart attack. It takes longer to have CHD develop. On the other hand, hostility, more of a chronic, trait-like variable, has been shown to predict CHD clinical events and incidence (5, 7) and long-term CHD mortality rate (13, 14). Similarly, emotional suppression, another trait-like variable, has been linked to CHD incidence (36) and to the development of breast cancer (27, 28). In brief, the effects of acute psychological states seem largely limited to short-term outcomes or outcomes that occur quickly, i.e., they seem to act as precipitators of acute clinical events. The effects of personality predispositions seem to be more general, affecting both short- and long-term health outcomes.

FUTURE DIRECTIONS

We would like to close this article by suggesting ways in which research in this domain might be improved. First, we believe that future research could benefit by greater use of theory. Prior research has proceeded in a largely haphazard and atheoretical fashion. Often variables are included in protocols simply because the relevant measures are readily available. This gives rise to a disjointed literature that is hard to integrate and understand. Future research needs to be more focused and more systematic. To accomplish this task, more comprehensive biobehavioral models are needed that integrate psychological and biological characteristics and mechanisms. In developing these models, it will be important to give equal weight to each side of the biobehavioral ledger.

Second, the field could benefit from a greater number of "prospective" prospective studies. Too often data are used that were collected for reasons other than to explore the impact of person variables on health. Researchers have made do with what was available, developing proxy measures for variables of interest, and looking backward through existing prospective data sets rather than designing future protocols around the best measures that exist. In defense of this strategy, it seems pointless not to utilize significant datasets that already exist, and it takes a long time to run a 5-, 10-, or 20-year prospective study. In using proxy measures, gleaned from existing studies, however, we should be aware that our measures will often just miss the mark and not quite capture the essence of the main variables of interest. To test hypotheses about person variables and health rigorously and fairly, it will be necessary to design future prospective studies that incorporate the relevant measures into baseline assessment procedures.

Along with the call for new prospective studies comes the need for more reliable and valid measurement techniques. If long-term studies are conducted, it is critical that the measurement base for these studies be as strong as possible. Controversies surrounding the meaning of measures need to be resolved and new measures need to be created as needed to measure central variables as soundly as possible. The need for conceptually focused measures underscores further the need for greater conceptual and theoretical clarity in the models that are developed. Without a clear understanding of the nature of the variables under study, effective measurement instruments cannot be constructed.

Finally, it will be important for researchers to realize that the effects of certain person variables may not be disease specific. The mind set of the field is currently centered around disease foci. It is reflected in the way in which researchers align themselves with their fields of interest, and it is reflected in the way in which government funding agencies are structured. The prevailing mind set often leads to the belief that, to be interesting, a particular variable has to be related to one and only one type of illness or disease. It may be time to relax the uniqueness standard just a bit and to begin to explore more thoroughly the possibility that certain person variables may be linked to multiple outcomes across diseases. If not, we may never come to understand that certain categories of person variables are uniquely related to many different disease end points.

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