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PERSONALITY AND RISK OF PHYSICAL ILLNESS

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■ **Abstract** Several personality characteristics have been linked in multiple well-designed prospective studies to subsequent physical health outcomes, such as longevity and the development and course of cardiovascular disease. The evidence is strongest for negative affectivity/neuroticism, anger/hostility and related traits, and optimism. Models of mechanisms underlying these associations have emphasized physiological effects of stress, exposure to stressors, and health behavior. Preliminary evidence supports the viability of some mechanisms, but formal mediational tests are lacking. In addition to addressing limitations and inconsistencies in this literature, future research should address developmental aspects of these psychosocial risk factors, contextual moderators of their health effects, and intervention applications in the prevention and management of disease. In these efforts, greater incorporation of concepts and methods in the structural, social-cognitive, and interpersonal perspectives in the field of personality are needed.

CONTENTS

INTRODUCTION

The hypothesis that personality influences the development and course of physical illness has appeared many times and in many forms throughout the history of medicine (McMahon 1976, Smith & Gallo 2001). In recent decades, it played a central role in the emergence and evolution of the interrelated fields of behavioral medicine, health psychology, and psychosomatic medicine (Stone et al. 1979, Surwit et al. 1982, Weiss et al. 1981), and contributed to a resurgence of interest in personality research within psychology (Contrada et al. 1999, Wiebe & Smith 1997). Long-standing skepticism in the medical community (e.g., Angel 1985) has eroded with the accumulation of methodologically sound research. However, inconsistent findings, alternative interpretations, and unresolved questions pose important challenges for future research.

Some topics in personality and health research were so widely reported that they entered popular culture, as in the case of Friedman & Rosenman's (1959) groundbreaking description of the Type A behavior pattern. Other hypotheses and conclusions, such as the role of chronic negative affect in disease (Friedman & Booth-Kewley 1987), were initially met by thoughtful and heuristically valuable critiques (e.g., Matthews 1988, Stone & Costa 1990), and appeared again later with more convincing support (Suls & Bunde 2005). Topics such as the concept of psychological hardiness (Kobasa 1979) were central in the emergence of personality and health research (Suls & Rittenhouse 1987) and can still be seen as important influences on subsequent developments, even though they faded in prominence as the field evolved. In addition, new topics based in mainstream personality theory have emerged as potentially important influences on health, such as the role of conscientiousness (Friedman et al. 1993).

Answers to age-old questions about mind-body associations are inherently interesting and important. However, research on personality and health can also guide the design of potentially useful interventions for the prevention and management of physical illness. In this chapter, we provide a review of the current state of research on personality characteristics as predictors of the development and course of physical illness. The equally important role of personality as an influence on adaptation to physical illness (Contrada et al. 1999, Smith & Ruiz 2004) is beyond our present scope. To provide a context for our review, we first present basic issues in the field. After reviewing the literature linking personality and health, we conclude with a discussion of future research directions and implications for clinical application.

CONCEPTUAL AND METHODOLOGICAL ISSUES

The hypothesis that personality influences health appears straightforward but has proven to be conceptually complex and methodologically challenging. This is true for the conceptualization and measurement of its two major components, as well

as for tests of their association and the explication of mechanisms underlying such associations.

Conceptualizing and Measuring Personality

Current personality theory and research have much to offer in the study of psychosocial risk, beginning with the measurement of personality risk factors. A wide variety of personality constructs and measures have been used as predictors of health, posing challenges for the interpretation and integration of findings. In many instances, scales are developed and used without adequate examination of their psychometric properties, especially construct validity. There is often little beyond the scale content to support the assertion that it measures the intended construct as opposed to another, perhaps better-established, characteristic. In such instances, studies might "reinvent constructs under new labels" (Holroyd & Coyne 1987). Similarly, in any specific topic in personality and health research, it is possible that despite similar labels scales may actually assess quite distinct traits.

The emergence of the Five Factor model (FFM) as a generally accepted taxonomy of broad personality traits (Digman 1990, McCrae & John 1992) and the related availability of well-validated measures can address this problem. The five factors and their facets or components (Costa & McCrae 1992) provide a nomological net (Cronbach & Meehl 1955) to guide efforts to compare, contrast, and integrate personality constructs and scales used in health research. Associations of scales under consideration with the FFM traits and facets can clarify the nature of the construct(s) they assess, as well as identify similarities and differences with scales used in other health studies (Friedman et al. 1995, Marshall et al. 1994, Smith & Williams 1992). Research of this sort could help bring order to what often seems to be an unwieldy, unsystematic, and uncritical proliferation of personality measures and constructs in health research. The general trait perspective also includes well-established methodological principles for the development and evaluation of personality scales (Ozer 1999, West & Finch 1997). More frequent, theory-driven application of these procedures is needed.

The FFM and other trait approaches can help to clarify *which* personality factors predict health, but do less in describing *how* these risk factors are related to cognitive, emotional, and behavioral processes that in turn affect health. Although there are important exceptions (McCrae & Costa 1996), most presentations of the FFM and other trait taxonomies focus on the structure of personality or characteristics that persons *have* as opposed to personality processes and things that people *do* (Cantor 1990). The social-cognitive tradition in current personality theory and research is particularly useful in this regard. Although no consensus exists regarding a taxonomy of social-cognitive personality constructs, major theoretical statements have described an extensive list (e.g., Mischel & Shoda 1998). These include mental representations (i.e., schemas) of self, others, relationships, and social interaction sequences (i.e., scripts); expectancies, goals, motives, and life tasks; appraisals or encoding of people and situations; self-regulation and coping;

and strategies, competencies, and tactics in goal-directed action. This perspective describes personality through the content of such characteristics, as well as the manner in which the characteristics are activated or accessible and the associations among them. These concepts provide a more active and specific process account of individual differences that complements the broader and more static description inherent in most trait approaches. Research examining associations between global traits and social-cognitive characteristics has identified mechanisms through which traits influence affect and behavior (e.g., Graziano et al. 1996). This approach is likely to prove useful in explicating the mechanisms through which personality characteristics influence health, as well as the identification of specific targets for risk reducing interventions.

Another major tenet of this view is that the consistency in personality may be better captured at the level of patterns of behavioral response to variation in specific types of situations rather than at the level of broad traits and aggregated behavior (Mischel 2004). For example, a pattern of hostile responses to perceptions of mistreatment by persons in authority and warmth toward lower-status persons reflects a potentially very different personality characteristic than does a pattern of warmth toward persons in authority and hostility toward subordinates, even if the aggregated mean level of hostility versus warmth is equivalent. Hence, "if–then" patterns of situation-specific behavior or "behavioral signatures" (Mischel 2004) provide an intriguing alternative to trait conceptualizations in predicting health outcomes.

Most research on psychosocial risk factors for disease characterizes these variables as either aspects of people (e.g., personality traits) or the social-environmental circumstances they inhabit (e.g., social isolation, conflict). Yet personality risk factors are consistently associated with social-environmental risk factors. For example, social support (versus social integration) and job stress are two of the most well established social-environmental risk factors. Personality characteristics both predict and are predicted by experiences in personal relationships and at work (Roberts et al. 2003, Robins et al. 2002). Further, some social-environmental risk factors such as social support display stability over time and across situations, strong correlations with personality characteristics, and even evidence of heritability. That is, these variables sometimes appear more like personality traits than independent external social circumstances to which individuals are simply exposed. Conventional trait approaches describe personality and social circumstances or situations as independent domains that interact only statistically to influence behavior, emotion, and other responses (Endler & Magnusson 1976). However, personality traits seem to influence exposure to health-relevant social circumstances rather than simply moderate reactions to this purportedly separate class of influences on health. Hence, the distinction between person characteristics and social environmental factors is somewhat artificial and potentially impedes the development of a more integrative view of risk.

The interpersonal approach to personality (Kiesler 1996, Pincus & Ansell 2003) provides additional resources in this regard. This approach assumes an inherent

association between personality and social circumstances, evident in Sullivan's (1953, p. 111) definition of personality as "the relatively enduring pattern of interpersonal situations which characterize a human life." Consistent with social-cognitive models suggesting that personality and social situations are reciprocally related (Bandura 1978, Mischel & Shoda 1998), the interpersonal approach describes the ways in which people shape and are shaped by the social contexts they encounter. In interpersonal theory, this concept is articulated as the transactional cycle (Carson 1969, Kiesler 1996). Intraindividual factors such as those described in the social-cognitive perspective (e.g., expectancies, goals, appraisals) guide overt social behavior. Once expressed, the actor's behavior tends to restrict the experience of interaction partners in such a way as to evoke interpersonal responses that are consistent with the actor's original expectancies, affect, or internal representations. In this way, trusting or optimistic individuals behave in a warm manner toward others, tending to evoke positive responses that confirm and maintain their positive outlook. The resulting stability of the reciprocal interaction patterns contributes to the apparent stability of both personality and aspects of the social environment (Caspi et al. 1989, Smith & Spiro 2002, Wagner et al. 1995).

The interpersonal approach describes social behavior as varying along two basic dimensions (i.e., dominance versus submissiveness and friendliness versus hostility), forming a structural model of interpersonal behavior—the interpersonal circumplex (Kiesler 1983, Wiggins 1979). The circumplex can be used equally well to describe aspects of the social environment such as social support (Trobst 2000) or personality traits (Wiggins & Broughton 1991). In this manner, it provides common concepts and methods for integrating personality and social–environmental risk factors (Gallo & Smith 1999) and for examining psychophysiological mechanisms underlying their association with health (Smith et al. 2003).

The circumplex can be used to compare and contrast personality characteristics through the use of several quantitative approaches (Gurtman & Pincus 2003), much like the construct validation process (described above) utilizing the FFM. One particularly useful version of the FFM replaces introversion versus extraversion and agreeableness versus antagonism with the dimensions of the interpersonal circumplex (Trapnell & Wiggins 1990), and has been used to clarify the nature of constructs studied in personality and health research (e.g., Gallo & Smith 1998). The dimensions of the circumplex have also been conceptualized as broad social motives. Agency refers to striving for separateness, achievement, and power, whereas communion refers to striving for connection and concern for others (Wiggins & Trapnell 1996). This motivational framework has been discussed as relevant to risk for physical disease (Helgeson 2003).

Quantifying Health Outcomes and Testing Associations

Many different health endpoints have been studied in personality and health research. Some are straightforward, as in the case of longevity in initially healthy samples or survival among persons with pre-existing disease. Increasingly in this

literature, the incidence and course of specific diseases assessed through accepted diagnostic procedures (e.g., myocardial infarction verified by ECG changes and cardiac enzyme elevations) serve as health outcomes. In contrast, earlier studies often utilized convenient but ambiguous health endpoints, such as self-reported symptoms and self-rated health status. These variables are most accurately seen as reflecting illness behavior—things that people often do when ill—rather than the underlying disease itself. Such measures are certainly associated with actual health and disease. For example, self-ratings of general health or physical function predict longevity in prospective studies, even when initial diagnoses of disease and traditional medical risk factors are statistically controlled (Idler & Benyamini 1997, Myint et al. 2005). However, self-reports of illness or health status and other measures of illness behavior also likely contain systematic variance that is unrelated to actual health (Costa & McCrae 1987, Watson & Pennebaker 1989), such as the tendency to deny or minimize illness (e.g., stoicism) or the tendency to exaggerate descriptions of health problems (i.e., unfounded or excessive somatic complaints). As a result, associations between these measures and personality traits could reflect an association with actual disease, illness behavior independent of disease, or a combination of these effects. This is not to say that personality and health research should always be limited to endpoints such as mortality and verified disease. Symptoms and well-being are important in comprehensive models of health and quality of life (Ryff & Singer 1998), and health care utilization is an increasingly important outcome, given economic considerations. However, when research questions concern actual disease, measures even partially reflecting illness behavior have obvious limitations.

The association between personality characteristics and physical health can be tested in a variety of designs. A common approach compares persons with and without a given disease on the personality construct of interest (i.e., case-control designs). In this cross-sectional design, it is difficult to determine if associations reflect a potential cause of disease or a psychological reaction to it (Cohen & Rodriguez 1995), as when patients with clinically apparent coronary disease score higher on measures of anxiety, depression, or other negative affects than do matched controls. Recent developments in medical imaging have created opportunities for more informative cross-sectional designs. Ultrasound and computed tomography scan technologies, for example, provide noninvasive assessments of asymptomatic or preclinical indications of disease. Associations between personality traits and these "silent" disease states are less likely to reflect psychological reactions to disease than are effects involving clinically apparent disease. These assessments have the further advantage of testing associations between personality and earlier stages of disease. In studies of mortality or the incidence of diagnosed disease, it is unclear at what point in a potentially decades-long etiology personality may be playing a contributing role.

The advantages of newer cross-sectional designs notwithstanding, prospective designs are much more informative. However, the correlational nature of prospective designs poses other threats to internal validity. Unmeasured third variables are

always a concern, especially in light of the fact that the etiology of most serious illness is multifactorial. Even when potential third variables are carefully articulated, the measures may not exhaust variance in the confounding factor. The possible undercorrection of confounds is a common source of alternative interpretations even for prospective associations between carefully assessed personality characteristics and unambiguous health outcomes (Phillips & Davey Smith 1991).

Other cautions are important in the statistical analysis of correlated risk factors. Traditional epidemiological methods emphasize the identification of independent risk. However, some confounds may actually reflect mediating mechanisms (Baron & Kenny 1986). Personality traits might influence subsequent disease through the mechanisms of health behavior (e.g., smoking) or biological consequences of psychological stress (e.g., elevated blood pressure or cholesterol). Statistical control of these previously established risk factors might lead to the conclusion that associations between personality and health outcomes are unimportant, when the analysis can also be interpreted as suggesting a mediational explanation. In other cases, correlated personality traits might compete in analyses, forcing their independence when their overlap exists for good reason, as in the case when they reflect distinct but related facets of a multicomponent construct (Suls & Bunde 2005). A narrow view of the statistical independence criteria could lead to the erroneous conclusion that neither characteristic is an important risk factor. Similarly, if a measure of a social-environmental risk factor (e.g., social isolation) and a related personality risk factor (e.g., negative affectivity) are forced to be independent, a causally important pattern of covariation between personality and social environment might be erroneously interpreted as conferring no independent risk. Statistical control procedures are essential in nonexperimental research. However, they should be used in a theory-driven manner rather than in a simplistic pursuit of independent risk. The articulation and examination of possible confounds are invaluable in psychosocial epidemiology, but this process must be balanced by considering the implications of creating counterfactual (Meehl 1970) independence among naturally bundled risk factors.

Mechanisms Linking Personality and Disease

Several general models of mechanisms underlying associations between personality and subsequent health have been described (Cohen 1979, Suls & Sanders 1989, Wiebe & Smith 1997). Health behavior models suggest that personality influences health-relevant daily habits (e.g., smoking, diet, exercise) and other health practices (e.g., medical screening). A wide variety of health behaviors are associated with the major domains of personality (Booth-Kewley & Vickers 1994), and these factors could mediate the association between personality and subsequent disease. The interactional stress moderation model specifies physiological rather than behavioral mechanisms. This view suggests that personality influences the appraisal of potentially stressful life circumstances as well as coping responses. Appraisal and coping influence physiological responses to stress (i.e., neuroendocrine responses,

immune functioning and inflammation, cardiovascular responses) that in turn contribute to the development of disease (Schneiderman et al. 2005).

The transactional stress moderation model also identifies personality effects on appraisal, coping, and physiological reactivity as an important mechanism, but posits an additional pathway in the stress process. As in the interpersonal approach, personality is seen as influencing the individual's exposure to potential stressors (e.g., interpersonal conflict) and the degree of stress-reducing interpersonal resources (e.g., social support). Through their decisions to enter some situations and not others, the reactions they unintentionally evoke from other people, and their intentional alteration of social situations, people influence the interpersonal contexts they encounter (Buss 1987). In this way, personality can influence the frequency, magnitude, and duration of exposure to stressors in daily life, as well as the availability of stress-reducing social resources. This exposure mechanism would augment the contribution of personality to reactivity to everyday stressors (Bolger & Schilling 1991, Bolger & Zuckerman 1995).

Constitutional predisposition models describe a noncausal association between personality and health. In this view, an underlying genetic or other constitutional factor produces both a physiologic vulnerability to disease and the behavioral, emotional, and cognitive phenotype of personality. However, the personality characteristic and the disease are otherwise causally unrelated coeffects of this underlying factor. In the illness behavior model, personality influences perception of and attention to normal physiological sensations, the labeling of such sensations as symptoms of illness, the reporting of symptoms, and the utilization of health care—but not actual disease (cf. Williams 2004).

Each of these models could account for prospective associations between personality and health outcomes. Inclusion of measures of health outcomes that reflect disease rather than illness behavior provides a test of the illness behavior model. Thorough assessment and statistical control of health behavior can test the health behavior model, with the cautions regarding statistical control and undercorrection of correlated risks (i.e., residual confounding) described above. Both of these methodological features have been sufficiently common in the literature to support the conclusion that neither the illness behavior model nor the health behavior model provides a complete account of the prospective association between personality and health.

Tests of the other three models are possible, but far less common. For example, molecular genetic studies have identified—at least in preliminary findings—genes associated with several personality traits studied as health risk factors. Measurement of these genotypes in prospective studies of personality and health could provide a test of this general view. However, these advances in molecular genetics also provide opportunities to test other potentially important models of risk (e.g., genetic diathesis by stress interactions, gene-environment correlations) that are the focus of research in other psychological fields (see, e.g., Moffitt et al. 2005, Rutter & Silberg 2002) but not yet addressed in the study of personality and health. Similarly, assessment of physiological stress responses and stress exposures could

provide an opportunity to test the interactional and transactional stress moderation models. However, few studies have been designed in such a way as to permit the evaluation of these mechanisms.

PERSONALITY CHARACTERISTICS LINKED TO HEALTH

Rather than attempt to comprehensively review all traits studied as risk factors, we focus here on research testing prospective associations with objective health outcomes. Much of this research has focused on longevity or mortality as a health outcome, or coronary heart disease (CHD) when specific diseases are studied. As a result, these outcomes are common in the studies reviewed below. In addition to conclusions regarding associations with subsequent health, we also briefly describe measurement issues and research evaluating potential mechanisms.

The Evolution of the Coronary-Prone Behavior Pattern

Following Friedman & Rosenman's (1959) description of the Type A behavior pattern (TABP) as a coronary risk factor, 20 years of research generally supported this hypothesis (Cooper et al. 1981). However, notable failures to replicate appeared soon thereafter (e.g., Ragland & Brand 1988; Shekelle et al. 1985a,b). A quantitative review indicated that the overall association between the TABP and CHD was significant and that much of the inconsistency could be attributed to methodological factors (Miller et al. 1991). The effects of the TABP were more apparent in studies of the incidence of CHD among initially healthy individuals than in studies of high-risk populations, such as persons with pre-existing CHD. Furthermore, these associations were stronger in studies using interview-based behavioral ratings of the TABP than in those using self-reports. The most extensively studied model of the mechanism underlying this association is the interactional stress-moderation model. Compared with their more relaxed Type B counterparts, Type A individuals display larger cardiovascular and neuroendocrine responses to a variety of stressors (Houston 1989). Transactional mechanisms also may contribute to this association, in that Type A individuals select more challenging tasks and often evoke competitive and antagonistic behavior from others (Smith & Anderson 1986).

The inconsistent association between the TABP and CHD led investigators to examine individual facets of this multicomponent construct on the assumption that some specific characteristics may be more relevant to health than are others. These studies identified behavioral ratings of hostility as the best predictor of CHD among the various Type A traits (Dembroski et al. 1989, Hecker et al. 1988, Matthews et al. 1977). Prospective studies using self-report measures of hostility supported this conclusion (Barefoot et al. 1983, Shekelle et al. 1983). Despite the appearance of some negative findings, a quantitative review of studies published before 1995 supported the conclusion that hostility was associated with increased risk of CHD and all-cause mortality (Miller et al. 1996).

Although some subsequent prospective studies of initially healthy individuals have failed to replicate this effect (Eng et al. 2003, Surtees et al. 2005, Sykes et al. 2002), the majority of such studies support the prior conclusion (Chang et al. 2002, Everson et al. 1997b, Gallacher et al. 1999, Kawachi et al. 1996, Matthews et al. 2004a, Williams et al. 2000). Studies of various stages of CHD suggest that hostility might play a role across the development and course of the condition. Individual differences in hostility and anger are associated with early indications of atherosclerosis such as endothelial dysfunction (Gottdiener et al. 2003, Harris et al. 2003) and with measures of more advanced but still asymptomatic atherosclerosis in otherwise healthy individuals (Iribarren et al. 2000, Matthews et al. 1998, Raikkonen et al. 2004), although some studies have not found this association (O'Malley et al. 2000).

Some recent studies suggest that anger and hostility are not associated with the course of established CHD (Kaufman et al. 1999, Welin et al. 2000). However, others (Smith et al. 2004a) indicate that self-reports or behavioral ratings of hostility are associated with increased risk of progression of atherosclerosis, recurrent coronary events, and death among individuals at high risk for disease as well as patients with pre-existing CHD (Angerer et al. 2000, Boyle et al. 2004, Chaput et al. 2002, Matthews et al. 2004a, Olson et al. 2005). Hostility also predicts restenosis of coronary arteries following angioplasty (Goodman et al. 1996, Mendes De Leon et al. 1996). Measures of trait anger and hostility have also been associated with the occurrence of myocardial ischemia among persons with CHD (Burg et al. 1993, Helmers et al. 1993, Rosenberg et al. 2001). Hence, prospective associations of these personality traits with the incidence of CHD and mortality could reflect effects during early, middle, and late stages of disease development, although the effects are generally stronger and more consistent for the initial development of disease as opposed to its course (Miller et al. 1996). Anger and hostility have also been found to predict the development of hypertension and stroke (e.g., Everson et al. 1999, Rutledge & Hogan 2002, Williams et al. 2002, Yan et al. 2003).

A wide variety of scales and rating systems are used in these studies (Barefoot & Lipkus 1994, Smith et al. 2004a). Interview-based behavioral ratings demonstrate generally consistent associations with subsequent health, but evidence of construct validity in the form of associations with other measures of anger, hostility, or aggressive behavior is limited. Of the many self-report instruments used in this research, the Cook & Medley (1954) hostility (Ho) scale is the most widely used, in large part because it is from the Minnesota Multiphasic Inventory (MMPI) item pool. The availability of several large samples in which the MMPI was administered previously facilitated the accumulation of longitudinal tests of hypotheses about hostility by conducting follow-up health assessments. Supportive findings from these studies encouraged the continued use of the Ho scale. Although the Ho scale demonstrates expected associations with other measures of this trait, it also has a poorly defined internal structure and substantial overlap with personality traits beyond the conceptual definition of this domain (Smith et al. 2004a). Measurement research has clarified the structure of this general personality domain

(e.g., Martin et al. 2000), and applications of the FFM and interpersonal circumplex have provided ties to well-established personality frameworks (Costa et al. 1989, Gallo & Smith 1998).

Research on mechanisms linking hostility and health has focused primarily on interactional stress moderation models. In this view, hostile individuals are seen as responding to potential stressors with larger and more prolonged heart rate, blood pressure, and neuroendocrine (e.g., cortisol, catecholamines) changes, relative to nonhostile persons. These responses are hypothesized to contribute to cardiovascular and other diseases (Williams et al. 1985). Many studies have demonstrated that hostile people display such responses in the laboratory (e.g., Smith & Gallo 1999, Suarez et al. 1998) and during daily life (Benotsch et al. 1997, Brondolo et al. 2003, Guyll & Contrada 1998, Pope & Smith 1991). In recent studies, hostility is positively associated with inflammatory markers (Suarez 2003, Suarez et al. 2002, Miller et al. 2003), suggesting another psychophysiological mechanism linking hostility with CHD (Libby 2003) and other negative health outcomes (Kiecolt-Glaser et al. 2002). Hostility is consistently associated with increased exposure to interpersonal stressors and reduced levels of social support (Smith et al. 2004a). This psychosocial vulnerability could contribute to the health consequences of hostility and could reflect transactional processes through which hostile persons engender a social environment high in conflict and low in support.

Hostility is associated with a variety of negative health behaviors (Siegler et al. 2003), and at least one study supports the hypothesis that health behavior mediates the association between hostility and subsequent health (Everson et al. 1997b). However, in most prospective studies, statistical control of these factors does not eliminate the effects of hostility (Miller et al. 1996). Individual differences in hostility demonstrate moderate heritability (e.g., Smith et al. 1991), and specific genotypes have been identified in preliminary studies (Jang et al. 2001, Manuck et al. 1999). Such findings are consistent with constitutional predisposition models positing genetically based central mechanisms accounting for the statistical association between hostility and health (Kaplan et al. 1994, Williams 1994).

There is some evidence that hostility is not the only unhealthy aspect of the TABP. Behavioral ratings of hostility and a socially dominant style—consisting of vigorous speech and the tendency to talk over interaction partners—are independently related to incident CHD and premature death (Houston et al. 1992, 1997). Self-reports of dominance are also associated with increased risk of CHD (Siegman et al. 2000, Whiteman et al. 1997). These results converge with findings from a nonhuman primate model of social behavior and atherosclerosis (Kaplan & Manuck 1998). Socially dominant male macaques develop atherosclerosis more readily than do subordinate males in response to chronic social stress. This vulnerability was eliminated through the administration of beta-adrenergic blockade, a finding that suggests sympathetic activation associated with chronic challenges to social status contributes to this association between individual differences in social behavior and subsequent cardiovascular disease. This is consistent with the interactional stress moderation model of personality and health. In humans, the

act of asserting dominance and attempting to influence others evokes heightened cardiovascular responses (Smith et al. 1989a, 2000).

Chronic Negative Affect

Individual differences in negative affect such as anxiety and sadness have figured prominently in research on personality and health, although the topic has at times been controversial. Mostly commonly labeled neuroticism or negative affectivity, this personality characteristic is generally defined as "the tendency to experience distress, and the cognitive and behavioral styles that follow from this tendency" (McCrae & John 1992, p. 195). An influential quantitative review concluded that this trait conferred risk of serious illness and premature mortality (Friedman & Booth-Kewley 1987), but the review was criticized for the inclusion of studies assessing illness behavior rather than actual disease and others possibly demonstrating consequences of disease rather than contributing causes (Matthews 1988, Stone & Costa 1990). Neuroticism and negative affectivity are associated with excessive somatic complaints (Costa & McCrea 1987, Watson & Pennebaker 1989), and serious physical illness certainly can cause emotional distress. However, a large body of research now supports the prior conclusion that this personality trait predicts serious health problems (Smith & Gallo 2001, Suls & Bunde 2005).

There are important issues in the conceptualization and measurement of this risk factor. This global trait includes several more specific characteristics, including anxiety, depressive symptoms, worry, anger and irritability, self-consciousness, and low self-esteem (Costa & McCrae 1987, Watson & Clark 1984). Scales with labels that imply the measurement of specific dimensions or constructs (e.g., trait anxiety, depressive symptoms, self-esteem) are often psychometrically indistinguishable from measures of the broader domain and measures of other specific elements within it (Watson & Clark 1984). Further, many measures with labels quite different from this personality domain are actually quite closely related. Hence, research in this area is often complicated by measurement problems (described above) in which scale labels imply more specificity than can be demonstrated in construct validation research. In addition, this domain reflects variation in negative affect and related responses within the range of normal functioning, as opposed to clinically diagnosable emotional disorder. Important differences exist between individual differences in chronic negative emotion and emotional disorder (Cohen & Rodriguez 1995, Coyne 1994, Watson et al. 1994). However, individuals with clinically diagnosable anxiety or depressive disorders score high on measures of negative affectivity or neuroticism (Clark et al. 1994), and high levels of this trait are associated with increased risk of subsequent anxiety and mood disorders (Hirschfeld et al. 1989, Zonderman et al. 1993). Hence, unless clinical disorders are assessed, studies of the associations between this personality trait and later disease could involve the effects of undiagnosed mood or anxiety disorders. Similarly, prospective associations between anxiety and mood disorders with health outcomes could involve the effect of this personality trait.

Several studies have demonstrated that measures of anxiety and depression are prospectively associated with increases in blood pressure and the development of hypertension (Davidson et al. 2000, Jones et al. 1997, Markovitz et al. 1991, Rutledge & Hogan 2002, Spiro et al. 1995), though others have not supported this association (Yan et al. 2003). Among persons with hypertension, negative affectivity has been associated with increased risk of stroke and death from cardiovascular disease (Simonsick et al. 1995). In studies of initially healthy individuals, various measures of anxiety, depressive symptoms, general emotional distress, and other specific constructs in this domain (e.g., self-esteem, worry) have been associated with subsequent CHD (Albert et al. 2005, Anda et al. 1993, Barefoot & Schroll 1996, Eaker et al. 1992, Ford et al. 1998, Kawachi et al. 1994, Kubzansky et al. 1997, Pennix et al. 2001, Rowan et al. 2005, Todaro et al. 2003), atherosclerosis (Haas et al. 2005), stroke (May et al. 2002), diabetes (Golden et al. 2004), and earlier all-cause mortality (Gump et al. 2005, Herman et al. 1998, Martin et al. 1995, Somervell et al. 1989, Stamatakis et al. 2004). Among persons with existing CHD, measures of emotional distress predict recurrent coronary events and reduced survival (Ahern et al. 1990; Barefoot et al. 1996; Blumenthal et al. 2003; Denollet et al. 1995; Follick et al. 1998; Frasure-Smith et al. 1995a,b; Lesperance et al. 2002; Moser & Dracup 1996; Strik et al. 2003). In initial survivors of stroke, depressive symptoms have been found to predict reduced longevity (House et al. 2001). Similarly, neuroticism is associated with reduced survival among patients with end-stage renal disease (Christensen et al. 2002).

It is important to note that several well-controlled prospective studies have failed to find associations between measures of negative affect and health outcomes (Kaplan & Reynolds 1988, Lane et al. 2001, Shekelle et al. 1991, Zonderman et al. 1989). Further, several of the supportive studies cited above assessed multiple health outcomes, and effects on some specific health outcomes were not demonstrated consistently across studies. Nonetheless, results generally support the conclusion that this broad individual difference is associated with increased risk of objectively assessed, serious health problems.

These results raise the obvious question of whether one or more aspects of this broad personality domain are more important in future health or if the larger multifaceted domain predicts health. Well-established models of the structure of this domain suggest that these more specific characteristics are closely correlated. To the extent that these dimensions are difficult to distinguish, limitations in the discriminant validity of measures of closely correlated characteristics will complicate detection of their specific effects (Suls & Bunde 2005). Most of the studies cited above assess only one of the specific facets or include only a broad measure. Regrettably, in the instances where study protocols include measures of multiple components, published reports are often based on a single scale or one specific dimension at a time. Although some studies addressing this issue appear to identify one or another facet as most important (e.g., Strik et al. 2003), it should be noted that the construct validity of the scales they use is rarely sufficiently well established to support strong conclusions regarding specificity. Further, given the expected

high levels of association among aspects of a higher-order construct, estimates of independent statistical associations may be unstable and sample-specific.

Several mechanisms could contribute to the apparent health consequences of negative affectivity/neuroticism. This trait is associated with negative health behavior (Booth-Kewley & Vickers 1994). Consistent with stress moderation models, chronic anxiety and depressive symptoms are associated with altered autonomic regulation of the cardiovascular system (Berntson et al. 1998, Carney et al. 1995, Watkins et al. 1998), immune suppression (Kiecolt-Glaser et al. 2002), and increased inflammation (Suarez 2004, Suarez et al. 2003). Several forms of chronic negative affect are associated with increased exposure to daily stressors (Bolger & Schilling 1991, Bolger & Zuckerman 1995) and future life difficulties (Daley & Hammen 2002, Daley et al. 1997, Davila et al. 1997, Joiner & Coyne 1999, Neyer & Asendorpf 2001, Potthoff et al. 1995), perhaps indicating the operation of transactional mechanisms.

Optimism-Pessimism

The tendency to hold optimistic expectations about the future, as opposed to pessimism or even hopelessness, has been found to be associated with important health outcomes, although this literature is less extensive than the topics reviewed above. There are three conceptual models of this trait domain in personality and health research. Most prominent is the generalized expectancy model of Scheier & Carver (1985). Here optimism is defined as the tendency to "expect good experiences in the future" (Carver & Scheier 2001, p. 31), and a brief self-report scale (i.e., Life Orientation Test) is used to measure the construct. The explanatory style approach of Seligman and colleagues defines optimism as the tendency to attribute life difficulties to "temporary, specific, and external (as opposed to permanent, pervasive, and internal) causes" (Gillham et al. 2001, p. 54), and both self-report scales and expert coding of written or spoken material are used as measures. Finally, several self-report scales assess pessimism, based on cognitive models of depression (e.g., Everson et al. 1996).

Measurement issues pose challenges in this domain. These various measures are often only modestly correlated (Norem & Chang 2001), raising concerns about the extent to which they assess the same construct. Furthermore, the measures are often found to have substantial overlap with neuroticism and other personality traits used to study health (Marshall et al. 1992, Scheier et al. 1994, Smith et al. 1989b). As a result, associations of optimism/pessimism with subsequent health might reflect the effects of other traits. Finally, structural analyses indicate that optimism and pessimism are most accurately seen as distinct albeit inversely related dimensions, rather than opposite poles of a single personality trait (Chang 1998, Kubzansky et al. 2004). In studies of health, this raises the question as to whether it is the presence of optimism or the absence of pessimism that alters risk of disease. An analogous issue is illustrated by a recent study on positive and negative affect as predictors of mortality in CHD patients. Although both affective dimensions

predicted survival when considered separately, only negative affectivity was significant when they were considered simultaneously (Brummet et al. 2005).

In prospective studies of objective health, optimism as assessed with the Life Orientation Test is associated with reduced incidence of medical complications following coronary artery bypass surgery (Scheier et al. 1989, 1999) and angioplasty (Helgeson & Fitz 1999), as well as less progression of atherosclerosis (Matthews et al. 2004b). Pessimism has been associated with decreased survival among women with breast cancer, though optimism was not related to survival (Schultz et al. 1996). In prospective studies with long follow-up intervals, content ratings of optimistic explanatory style have been associated with better physician-rated health (Peterson et al. 1988) and longevity (Peterson et al. 1998). Other measures of optimism have been associated with longevity (Maruta et al. 2000), reduced incidence of CHD (Kubzansky et al. 2001), and longer survival following stroke (Lewis et al. 2001). Hopelessness has been associated with the development of hypertension (Everson et al. 2000), increased incidence of death from cardiovascular disease and cancer (Everson et al. 1996), incidence of myocardial infarction (Anda et al. 1993), greater progression of atherosclerosis (Everson et al. 1997a), and reduced longevity (Stern et al. 2001). However, some studies find no association between optimism and subsequent health (Cassileth et al. 1985), and the role of the association of these traits with neuroticism/negative affectivity in the observed effects is generally unknown.

Several mechanisms could contribute to the effects of this social-cognitive individual difference. Greater optimism and/or lower pessimism has been associated with better immune functioning (Segerstrom et al. 1998) and lower ambulatory blood pressure (Raikkonen et al. 1999), consistent with the stress moderation model. Optimism is also associated with more effective participation in health care (Lin & Peterson 1990, Strack et al. 1987), which suggests a possible role for health behavior mechanisms. Optimism is also associated with greater levels of social support (Brissette et al. 2002, Carver et al. 2003), suggesting the possible operation of transactional mechanisms.

Other Traits

The FFM trait of conscientiousness has been found to predict longevity among initially healthy persons (Friedman et al. 1993) and survival among patients with end-stage renal disease (Christensen et al. 2002). The mechanism underlying this effect has not been examined extensively, though apparently it is not explained by health behavior (Friedman et al. 1995). Curiosity, perhaps related to the FFM trait of openness to experience, has been found to be associated with longevity (Swan & Carmelli 1996), independent of medical risk factors and health behavior. The concept of sense of coherence (Antonovsky 1987) involves the extent to which individuals find their lives to be comprehensible, manageable, and meaningful. A self-report measure of this trait has been found to predict all-cause mortality in a large, population-based prospective study (Surtees et al. 2003), independent of

medical risk factors, health behaviors, and individual differences in hostility and neuroticism. The concept of a Type D (i.e., distressed) personality has been identified as a predictor of prognosis in CHD patients (Denollet 2005, Denollet et al. 1996). The self-report measure of this construct includes two factors: negative affectivity and social inhibition. These dimensions are closely related to the FFM traits of neuroticism and introversion, respectively (Denollet 2005), placing the Type D construct in the hostile-submissive quadrant of the interpersonal circumplex (Wiggins & Broughton 1991). Given strong associations with well-established personality variables, the Type D construct provides an example of prior critiques of personality and health research as having a tendency to "reinvent constructs under new labels" (Holroyd & Coyne 1987). Further, this model implies that the combination of negative affectivity and social inhibition provides unique prognostic information, yet the incremental effect of the statistical interaction of these traits is not tested. Also, the description of personality risk factors as types rather than dimensions raises a complex issue regarding the existence of discrete classes or groups of individuals within apparently continuous distributions of personality test scores (Trull & Durett 2005).

The tendency to deny or minimize negative emotions (i.e., repressive coping, denial, expressive suppression) has a long history in the study of personality and health. Various measures of this construct have been associated with increased risk of cancer (Dattore et al. 1980), cancer progression (Jensen 1987), and hypertension (Perini et al. 1991). In a related observation, the tendency to conceal their homosexual identity has been found to predict the development of cancer in HIV seronegative gay men (Cole et al. 1996a) and more rapid progression of HIV among seropositive men (Cole et al. 1996b). Mechanisms underlying the health consequences of repressive coping, expressive suppression, or denial could involve stress moderation mechanisms, as these processes have been associated with several aspects of autonomic activity (cf. John & Gross 2004, Smith & Gallo 2001). Transactional mechanisms may also be involved, as repressive coping or expressive suppression is associated with more stressful social interactions and other interpersonal difficulties (Butler et al. 2003, Gross & John 2003, John & Gross 2004).

CONCLUSIONS AND FUTURE ISSUES

This review provides ample evidence that some personality characteristics are prospectively associated with objective health outcomes, including longevity among initially healthy persons, survival among those with serious illness, and the onset of specific diseases (e.g., CHD). Plausible mechanisms underlying these effects have been identified and supported in preliminary research. Most findings are in need of replication, clarification, and extension. A major issue concerns the unique versus overlapping nature of the personality constructs and measures used to predict health. This is obviously an issue within the broad domains, as evident in the difficult psychometric distinctions among anger, hostility, and aggressiveness

or components of negative affectivity and neuroticism. It is also an issue across these domains, as trait anger can also be seen as a facet of the neuroticism trait domain, as can pessimism and hopelessness. Hence, greater use of current structural models of personality and related assessment methods could help to distill a clearer view of a smaller number of broad risk factors or identify more important specific elements within them. This integration could then help to focus future efforts in the difficult task of testing hypothesized mechanisms.

The study of personality and health has been generally separate from research on social-environmental risk factors. The implicit separation of risk factors into characteristics of personality and social circumstances has the potential to impede the emergence of a more integrative view of psychosocial influences on physical health. The health consequences of personality traits may involve their effects on the individual's social environment, and exposure to social environmental risk factors of sufficient duration to influence serious illness may reflect the operation of personality traits. The interpersonal perspective (Pincus & Ansell 2003) can provide useful concepts and methods in this effort. Low socioeconomic status (SES) confers risk of serious health problems, perhaps in part through its effect on psychosocial risk factors reviewed here (Gallo & Matthews 2003). Recent evidence that low SES is also associated with potentially stressful social experiences as assessed through the interpersonal circumplex (Gallo et al. 2006) further illustrates the integrative potential of this perspective.

The Importance of Context

The studies of personality and health outcomes described above generally adhere to a "main effects" model. Future research should pursue the possibility that health consequences of personality characteristics vary across aspects of the context in which they occur. Age and developmental processes represent a potentially important contextual factor. To date, the life-span development perspective has been underemphasized in personality and health research (Smith & Spiro 2002). Although dimensions of personality are fairly stable across adulthood, there are normative changes in levels of some characteristics (Caspi et al. 2005). Further, associations among personality traits and processes can change over time, as can the individual's profile of personality traits. Any one of these various types of change may be related to health risk. For example, high levels of hostility during early adulthood that decrease in middle age may be associated with lower risk than levels that increase during the same period, even if the level of hostility in these two scenarios is the same when averaged over time. That is, the temporal pattern of personality change may provide independent predictive information.

It is also likely that the association between personality and a given health outcome may change across the lifespan. A trait that predicts poor health outcomes in middle-aged adults might not predict longevity when assessed in later adulthood. The latter population may underrepresent individuals susceptible to the effects of the trait due to prior morbidity and mortality and may overrepresent those who are

more resilient (Williams 2000). Consistent with this view, Boyle et al. (2004) found that hostility was associated with mortality in younger but not older CHD patients. Age-related differences in the association between a personality characteristic and health could also reflect the fact that the trait influences one stage in the natural history of the disease but not others. Even when associations are similar across age and stage of disease, determinants of these psychosocial risk factors might vary with age (Nolen-Hoeksema & Ahrens 2002), as could the mechanisms underlying their association with health.

A developmental view encourages consideration of the origins of risk factors and related mechanisms as well as processes underlying their stability and change. Adverse events during childhood (e.g., physical or emotional abuse, neglect) are associated with increased risk for CHD, and this association is mediated to a greater extent by psychosocial risk factors (e.g., anger, depression) than by traditional CHD risk factors (Dong et al. 2004). Individual differences in physiological stress responses may be shaped by early experiences (Luecken & Lemery 2004), and reciprocal relationships between emerging personality characteristics and aspects of the social environment characterize the development, continuity, and change of personality over the lifespan (Caspi et al. 2005). A more complete science of personality and health should incorporate these and perhaps other developmental considerations.

Gender represents a similarly important aspect of context. Personality and health research includes a well-developed model of gender, personality, and vulnerability. Helgeson (1994) suggests that traditional sex roles render women differentially susceptible to stressors involving communion (e.g., maintenance of connectedness, caregiving, relationship quality) especially when they display high levels of communion traits and the relative absence of agentic characteristics (i.e., unmitigated communion). Conversely, men characterized by high levels of agency striving are susceptible to stressors in this domain (e.g., achievement, status, work) especially when they lack communal traits (i.e., unmitigated agency). Unmitigated agency and unmitigated communion can confer health risks for either men or women, though sex differences in these characteristics make them differentially common in men and women. A growing body of research supports this view of personality, gender, and vulnerability (Helgeson 2003).

Other examples of the importance of gender include sex differences in the type or timing of major health threats. Associations between personality and specific health outcomes may be different for men and women due to sex differences in the prevalence of various diseases or the age at which they occur. For example, associations between hostility and CHD within a given age group might be weaker for women than for men because women tend to develop the condition at a later age. Across the lifespan, there are sex differences in many of the personality traits identified as risk factors, and in childhood temperament precursors to these traits (Williams & Gunn 2006). There may be similar sex differences in the nature or determinants of these risk factors (Nolen-Hoeksema et al. 1999), the magnitude of their association with health, or in underlying mechanisms.

The major sources of morbidity and mortality vary across ethnic and cultural groups. However, research in health psychology and behavioral medicine has only recently begun to consider these issues extensively (Whitfield et al. 2002, Yali & Revenson 2004). The role of ethnicity and culture as moderators of associations between personality factors and health outcomes has been tested infrequently. Furthermore, few culturally relevant personality factors have been identified and examined within current conceptualizations of personality and health. Some of these issues arise from the development of personality theory primarily within European American culture (Triandis 1997). Many current conceptualizations of personality theory are most readily applicable to Western society. For example, much of Western personality theory assumes that social behavior is dependent on stable traits, which can be examined and understood as separate from social experiences and roles within society (Cross & Markus 1999). These assumptions place emphasis on understanding the individual as the primary determinant for behavior and action. However, this perspective is not equally applicable across all cultures. For example, many Asian cultures emphasize social obligations and social roles in relation to others as underlying motivation for individual action. These cultural differences may influence the relative importance of personality factors as opposed to roles, obligations, and other social factors as determinants of health. These cultural considerations might also influence how personality characteristics interact with social circumstances to influence health.

Research with African Americans provides some examples of how ethnicity can be examined as a context for personality and health research. African Americans display higher prevalence of hypertension, a primary risk factor for heart disease and stroke. In addition, African Americans continue to experience discrimination in higher education, housing rentals and sales, automotive sales, and hiring practices (Clark et al. 1999). These experiences may influence the development of personality risk factors or moderate their association with health. Similar considerations have led to the identification of specific personality traits within the African American community that are associated with the development of cardiovascular disease. For example, African Americans tend to score higher than European Americans on measures of hostility and anger (Steffen et al. 2003). These traits and individual differences in anger expression versus inhibition may be involved in African Americans' greater susceptibility to cardiovascular disease. Another culture-specific personality factor that has been identified as a potential influence on the health of African Americans is "John Henryism," in which a "strong behavioral predisposition to cope actively with psychosocial environmental stressors interacts with low socioeconomic status to influence the health of African Americans" (James 1994). This active coping style may contribute to elevated blood pressure for African Americans who diligently attempt to succeed despite limited resources and cultural barriers that impede attainment of goals (Dressler et al. 1998).

The major sources of morbidity and mortality also vary as a function of SES. Several of the personality traits that are reliably associated with health outcomes

are also related to SES, a finding that leads to the hypothesis that personality characteristics and related psychosocial risk factors may mediate some of the effects of SES on health (Gallo & Matthews 2003, Gallo et al. 2005). SES might also influence which personality traits are important for subsequent health, the magnitude of these associations, or the underlying mechanisms. Consideration of gender, age, culture and ethnicity, and SES has the potential to produce a more detailed account of associations between personality and health. Such an endeavor poses many challenges, from the equivalence of personality measures across these contexts to the need for context-specific revision of the conceptual models guiding personality and health research.

Clinical Applications

Drawing implications for interventions from the results of epidemiological research on personality and other psychosocial risk factors is a not a straightforward task (Macleod & Davey Smith 2003). Nonetheless, an important goal of this research is the development of risk-reducing interventions for the prevention and management of physical illness. The potential benefit of such applications was illustrated by the Recurrent Coronary Prevention Project (Friedman et al. 1984, Powell & Thoresen 1988), in which group therapy based on a social-cognitive model of the Type A pattern reduced both Type A behavior and the rate of recurrent cardiac events among CHD patients. Related interventions for reducing stress (e.g., Blumenthal et al. 2005) and hostility (e.g., Gidron et al. 1999) continue to prove useful in the management of CHD (Dusseldorp et al. 1999). In contrast, in the multicenter ENRICHD (Enhancing Recovery in Coronary Heart Disease Patients) trial, cognitive therapy for depression produced improvements in the severity of depressive symptoms among CHD patients but had no effect on cardiac events (Berkman et al. 2003). It is possible that further progress in the study of personality and health will facilitate refinements of such interventions in the future. Further specification of which traits confer risk and subgroups where these effects are strongest, as well as explication of the personality processes and mediating mechanisms involved in these associations, are all important aspects of such progress. To achieve it, the trait, social-cognitive, and interpersonal approaches to personality can be brought to bear.

The modification of existing personality risk factors in adulthood in order to prevent or manage serious illness (i.e., secondary and tertiary prevention) is perhaps the most obvious application of this research. However, personality and health research could also inform primary prevention efforts. Personality risk factors for medical illness in adulthood (e.g., chronic negative affect, antagonistic social behavior) converge with the focus of many primary prevention programs in childhood and adolescence intended to promote emotional health, prosocial functioning, and academic success. However, there is another possible benefit of such efforts—the prevention of prevalent and serious medical illnesses of mid and later adulthood (Smith et al. 2004b). Given the continuity of personality characteristics across the

life course (Caspi et al. 2005) and the decades-long etiology of many major diseases, the prevention of risk is an important implication of the study of personality and health.

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