Hostility, Anger, Aggressiveness, and Coronary Heart Disease: An Interpersonal Perspective on Personality, Emotion, and Health

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ABSTRACT The related traits of hostility, anger, and aggressiveness have long been suggested as risk factors for coronary heart disease (CHD). Our prior review of this literature (Smith, 1992) found both considerable evidence in support of this hypothesis and important limitations that precluded firm conclusions. In the present review, we discuss recent research on the assessment of these traits, their association with CHD and longevity, and mechanisms possibly underlying the association. In doing so, we illustrate the value of the interpersonal tradition in personality psychology (Sullivan, 1953; Leary, 1957; Carson, 1969; Kiesler, 1996) for not only research on the health consequences of hostility, anger, and aggressiveness, but also for the general study of the effects of emotion, personality and other psychosocial characteristics on physical health.

Hostility, anger and aggressive behavior play a central role in the centuries-old hypothesis that emotions and aspects of personality influence physical health. In the 19th century, physicians suggested that anger and aggressive striving contributed to coronary heart disease (CHD). Psychodynamic theorists in the mid-20th century maintained that excessive anger and inflexible styles of expressing it

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fostered cardiovascular disease. Recent interest in hostility and disease stems from efforts to identify unhealthy elements within the multifaceted, Type A, coronary-prone, behavior pattern (for a review of this history, see Siegman, 1994). Presently, anger, hostility, and aggressiveness collectively represent one of the most widely studied psychosocial risk factors for CHD and premature mortality, and most—but certainly not all—of the available studies support this association (Gallo & Matthews, 2003; Kop, 1999; Krantz & McCeney, 2002; Rozanski, Blumenthal, & Kaplan, 1999; Smith & Ruiz, 2002).

These emotional, cognitive, and behavioral characteristics are often subsumed under the label of hostility, but this term most accurately refers to cognitive factors. Specifically, *hostility* is a "negative attitude toward others, consisting of enmity, denigration, and ill will" (Smith, 1994, p. 26). As components of this characteristic, *cynicism* is the belief that others are motivated primarily by selfish concerns and *mistrust* is the expectation that people are frequent sources of mistreatment. *Hostile attributional style* is the tendency to construe the actions of others as involving aggressive intent. Hence, as a general cognitive characteristic, hostility involves, "a devaluation of the worth and motives of others, an expectation that others are likely sources of wrong-doing, a relational view of being in opposition toward others, and a desire to inflict harm or see others harmed" (Smith, 1994, p. 26). In contrast, anger is "an unpleasant emotion ranging in intensity from irritation or annoyance to fury or rage" (Smith, 1994, p. 25). Related emotions include contempt and resentment. As a personality trait, anger refers to the tendency to experience frequent and pronounced episodes of this emotion. Aggression involves a variety of verbal and physical behavior, "typically defined as attacking, destructive, or hurtful actions" (Smith, 1994, p. 26). As a trait, aggressiveness is the disposition to display such behavior. In a related concept, also studied as a risk factor for cardiovascular disease, modes of anger expression or anger-coping styles refer to individual differences in the tendency to a) outwardly express aggressive behavior when angry or b) withhold (i.e., suppress) such expressions.

It is often difficult to maintain sharp distinctions among hostility, anger, and aggression. For example, anger involves the "relational theme," or cognitive script (Lazarus, 1991), of unfair interference or harm, and both anger and hostility involve the action tendency of inflicting harm through aggression. These personality traits are correlated (Barefoot & Lipkus, 1994), but not so highly as to represent

interchangeable labels for a single construct. Hence, parallel associations with health and similar underlying mechanisms should not be assumed.

In a prior review (Smith, 1992), we described several central topics within the question regarding the health consequences of these traits. First, personality assessments used in this research were not sufficiently validated to permit strong conclusions that hostility (anger or aggressiveness)—rather than other traits inadvertently tapped by these measures—was involved in the associations with health observed in clinical and epidemiological studies. Second, although studies testing this association were generally supportive, there were several failures to replicate the effect. Hence, additional studies were needed, especially prospective designs using well-validated measures and diverse samples. Third, mechanisms underlying this association had been examined only in preliminary ways. We suggested that these limitations combined to preclude definitive conclusions. We also suggested that "virtually all the aspects of research on hostility and health could profit from further consideration of interpersonal processes" (Smith, 1992, p. 148) in that each of these main research issues involved social phenomena.

Since then, considerable progress has occurred, and most of the new evidence supports the basic psychosomatic hypothesis. Yet, some critical reviews have expressed pointed skepticism (e.g., Myrtek, 2001; Petticrew, Gilbody, & Shelton, 1999). In the present article, we revisit the critical topics we identified previously, examining recent progress and remaining challenges. We also discuss the application of the interpersonal perspective in personality psychology (Kiesler, 1996; Pincus & Ansell, 2003). In our view, the conceptual and methodological tools of the interpersonal approach are especially valuable in addressing current issues in research on hostility and health. Therefore, we turn first to the interpersonal approach, and then discuss its relevance to three topics: 1) the measurement of anger, hostility, and aggressiveness; 2) their association with subsequent health; and 3) mechanisms underlying this association.

An Overview of Interpersonal Concepts and Methods

The interpersonal approach (for reviews, see Kiesler, 1996; Pincus & Ansell, 2003) can be summarized through, a) a general assumption about personality, b) a structural model of social behavior, and c) a

process model of personality and social interaction. The basic assumption is evident in Sullivan's (1953, p. 111) definition of personality as, "the relatively enduring pattern of interpersonal situations which characterize a human life." In interpersonal theory, stable characteristics of an individual's social environment (e.g., isolation, persistent conflict) and personality traits such as hostility are assumed to reflect two aspects of one phenomenon (Pincus & Ansell, 2003). This assumption is the reason the interpersonal perspective is well-suited for the integration of research on psychosocial risk factors. Yet, it also suggests a reformulation, as it challenges the implicit parsing of risk factors as characteristics of people *or* the social environment. It instead suggests that risk factors are inherently characteristics of people *and* the social contexts they inhabit (Gallo & Smith, 1999); measuring personality traits, such as hostility, or aspects of the social environment, such as support, is akin to grasping one limb of the larger psychosocial "elephant" that confers risk of disease.

The Interpersonal Circumplex

Since the pioneering work of Leary (1957), the interpersonal circumplex (IPC) has been used to describe social behavior and personality (Wiggins, 1996). Two broad dimensions of friendliness (or warmth) versus hostility (or coldness) and dominance (or control) versus submissiveness (or passivity) define the axes of the IPC (see Figure 1). The IPC can describe specific social stimuli, specific social responses, more stable features of the social environment, and individual differences in social behavior. IPC-based assessments have been developed for interpersonal interactions (Gifford, 1991; Kiesler, 1983; Markey, Funder, & Ozer, 2003), interpersonal traits (Gurtman & Pincus, 2003; Wiggins, 1979), impressions of interaction partners (i.e., "impact messages," Schmidt, Wagner, & Kiesler, 1999; Wagner, Kiesler, & Schmidt, 1995), social support (Trobst, 2000), and interpersonal problems (Alden, Wiggins, & Pincus, 1990). Methods exist for the development of circumplex measures and for their use in validating other assessments (Gurtman & Pincus, 2003; Wiggins & Broughton, 1991).

Several theorists (e.g., Carson, 1969; Wiggins & Trapnell, 1996) have suggested that social interactions involve exchanges of two resources—status (i.e., regard or esteem from others) and love (i.e., acceptance or liking from others)—corresponding to the vertical and

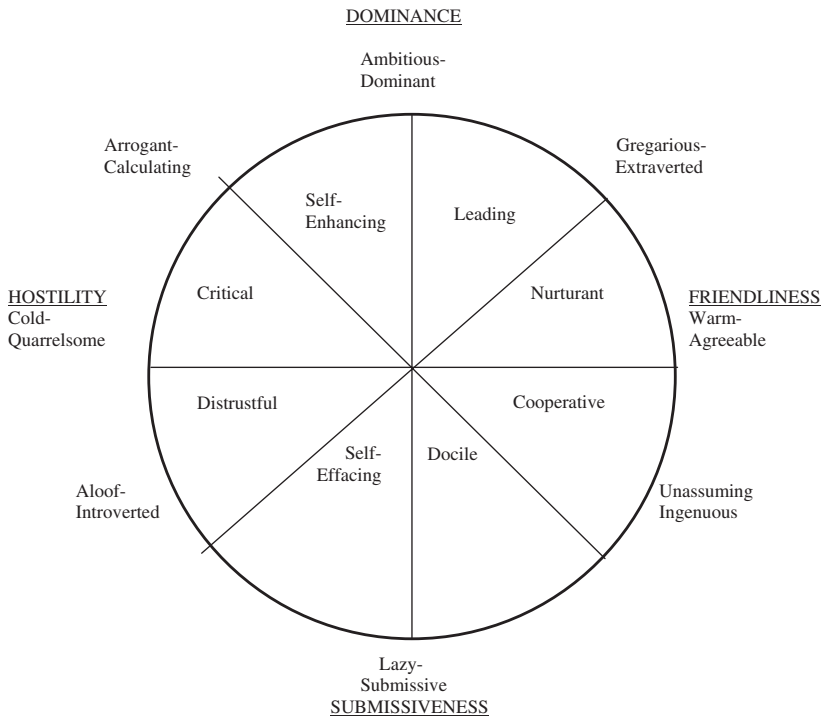

Figure 1
The interpersonal circumplex (from Gallo & Smith, 1999).

horizontal IPC dimensions, respectively. In this view, motives underlying interpersonal behavior concern obtaining or retaining status and acceptance. Wiggins and Trapnell (1996) suggest that the dimensions of the IPC can also be conceptualized as broad motivational complexes; *agency* involves strivings for achievement, status, power, and separateness, whereas *communion* reflects concerns over being part of a larger social group with related strivings for intimacy and the maintenance of relationships. In an account of gender roles and vulnerability to the effects of stress, Helgeson (1994) has suggested that when present in unbalanced or "unmitigated" forms, these motivational dimensions confer risk of emotional and physical dysfunction.

The Transactional Cycle

The basic assumption that personality comprises recurring interpersonal situations has been more formally represented as the *interpersonal*

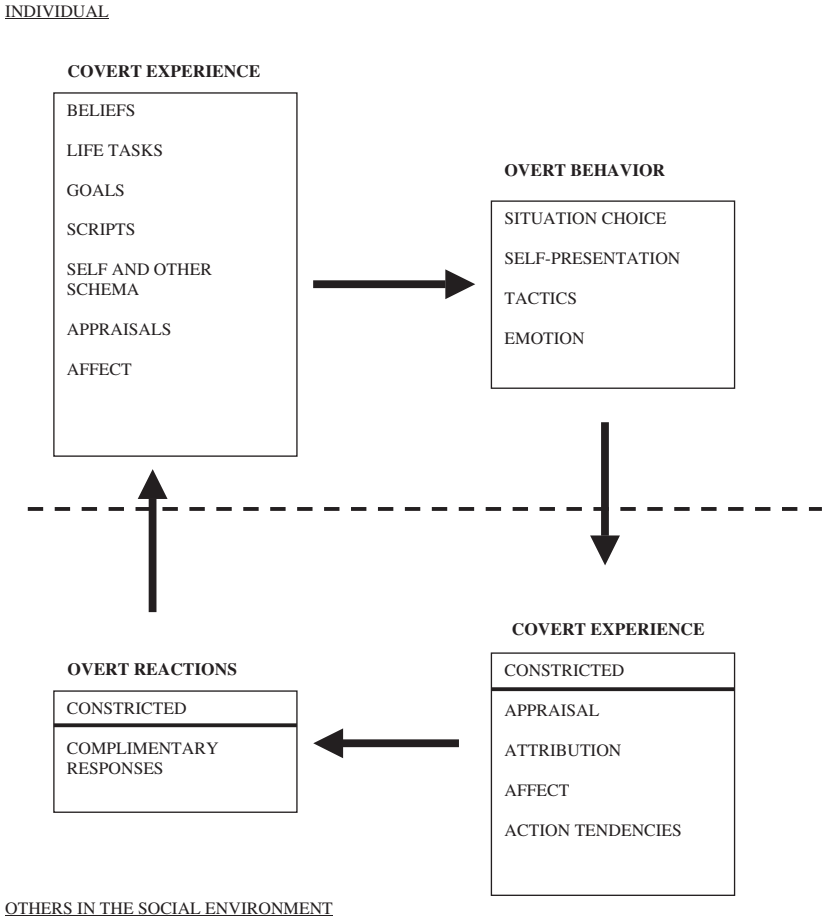

Figure 2
The transactional cycle (from Gallo & Smith, 1999).

or *transactional cycle* (Carson, 1969; Kiesler, 1996; see Figure 2). Through their covert experience and expressive behavior, individuals tend to influence the covert experience and expressive responses of others in their social environments in ways that are consistent with the initial actor's personality. That is, an individual's personality involves a social style that tends to "pull, elicit, invite, or evoke restricted classes of responses from the other" in a "continual, dynamic transactional process" (Pincus & Ansell, 2003, p. 215). Warm and agreeable persons evoke friendliness from others, whereas hostile persons regularly evoke coldness and even conflict. In interpersonal

theory, this mutual influence involves complementarity (Kiesler, 1983), consisting of correspondence on the horizontal IPC axis (i.e., warmth "pulls for" warmth, hostility encourages hostility) and reciprocity on the vertical axis (dominance encourages submissiveness). Methods for quantifying complementarity have produced supportive results (Gurtman, 2001; Markey, Funder, and Ozer, 2003), but strict complementarity frequently does not characterize interactions (Orford, 1986; Kiesler, 1996) and is not the only basis for recurring patterns of social interaction (Kiesler, 1996; Pincus & Ansell, 2003). For example, persistent assertion of dominance may evoke recurring episodes of contested dominance or control from others rather than submissiveness.

The tenet of interpersonal theory involving reciprocal determination between individuals and social situations is quite consistent with contemporary social-cognitive views of personality (e.g., Mischel & Shoda, 1999). In early descriptions of the transactional process (Sullivan, 1953), internal representations of developmentally important people (i.e., "personifications") and the tendency to interpret current interpersonal experiences in part through the "lens" of these knowledge structures (i.e., "parataxic distortions") were central concepts. Elsewhere (Gallo & Smith, 1999; Smith, Gallo, & Ruiz, 2003), we have suggested that a variety of concepts from current social-cognitive approaches to personality (e.g., Mischel & Shoda, 1995; 1998) can be organized within the more detailed transactional framework described by later authors (Carson, 1969; Kiesler, 1996), as depicted in Figure 2. That is, many of the cognitive, affective, and interpersonal processes or "middle units" (McAdams, 1995) in recent models of personality (Cantor, 1990; Mischel & Shoda, 1999)—such as goals and life tasks, and internal representations of self, others, and interaction sequences (i.e., schemas and scripts), tendencies in situation choices, and self-presentation—can be placed in this general schematic.

In interpersonal theory, the processes of personality development and change involve transactions. As in attachment theory (Bowlby, 1973, 1979) and its extension to adult relationships (Cassidy & Shaver, 1999; Hazan & Shaver, 1987, 1994), interpersonal theory emphasizes recurring patterns of parent–child interaction in the initial development of internal representations of the self, important others, relationships, and interaction sequences (i.e., schemas and scripts) (Pincus & Ansell, 2003). Integrations of the attachment and

interpersonal perspectives (Florsheim, Henry, & Benjamin, 1996; Gallo, Smith, & Ruiz, 2003; Pincus, Dickinson, Schut, Castonguay, & Bedics, 1999) suggest that attachment styles (e.g., secure, anxious, avoidant, etc.) can be located in the IPC and related dimensional models of interpersonal behavior and that attachment styles are related to developmental experiences and social functioning in ways that are consistent with interpersonal theory.

Recent interpersonal theory (e.g., Benjamin, 1974, 1994, 2003) emphasizes three developmental processes. In the process of *identification*, individuals, as adults, model behavior that they first observed in their parents during childhood. For example, they might enact toward others the criticism directed toward them by their parents, or they might model conflictual behavior in their own close relationships that they observed between their parents. *Internalization* refers to the development of representations of self, others, and relationships (i.e., schemas and scripts), which, in turn, form the basis of generalized interpersonal expectancies. In this process, individuals come to view themselves and others in a manner that parallels recurring patterns in childhood and to behave in ways that are consistent with these representations. For example, hostile persons might generally come to expect others to display the dismissive, coercive, and blaming actions displayed toward them by parents (Matthews, Woodall, Kenyon, & Jacob, 1996; McGonigle, Smith, Benjamin, & Turner, 1993). They might also continue to display the cold, wary, and defensive stance that complements this expected treatment from others. Finally, in the process of *introjection,* people treat themselves in ways they were treated by key developmental figures. This mechanism is consistent with the observation that hostile persons have vulnerable self-esteem and are critical of both others and themselves (Smith, McGonigle, & Benjamin, 1998; Tangney, Hill-Barlow, Marshal, & Gramzow 1996).

When repeated over time and across contexts, developmentally based transactional cycles contribute to continuities in personality, relationships, and other social experiences in a reciprocal process similar to those described in recent accounts of personality stability and lifecourse coherence (e.g., Caspi & Roberts, 1999). As depicted in Figure 3, a recurring pattern of transactional cycles would be expected to foster a health-relevant trajectory (Smith & Spiro, 2002). For example, early positive attachment experiences would foster a warm, trusting, and agreeable interpersonal style, and through the

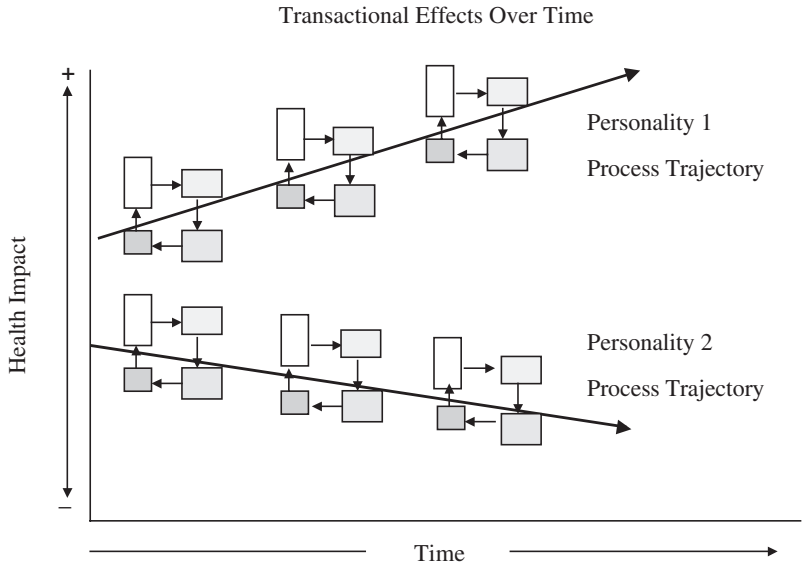

Figure 3
Transactional effects over time (from Smith & Spiro, 2002).

transactional processes described above, this would in turn lead to an accumulation of exposure to health-promoting social contexts (e.g., social support, intimacy, low conflict) over longer periods of time. In contrast, negative attachment experiences would contribute to less adaptive internal representations of self, others, relationships, and interaction sequences, as well as to a cold, mistrusting, and antagonistic interpersonal style. Through recurring transactional cycles, these antagonistic schemas, scripts, and expressive behaviors would promote cumulative exposure to unhealthy social contexts (i.e., isolation, conflict) over time. Through reciprocal processes, these healthy or unhealthy social contexts would, in turn, foster additional adaptive or unhealthy transactional cycles, respectively, regardless of whether they initially result from imposed social circumstances or the individual's impact on the social environment. In these ways, healthy and unhealthy interpersonal trajectories are maintained, similar to Friedman's (2000) concept of *tropisms* in which individuals seem to be consistently drawn to healthy or unhealthy contexts over time. In the interpersonal perspective, therapeutic interventions produce change by disrupting maladaptive transactional patterns (Kiesler, 1996). A wide variety of approaches

could produce such change by targeting various general locations and specific elements of the transactional cycle.

Current Issues in the Assessment of Anger, Hostility, and Aggressiveness

As noted above, assessments of anger, hostility, and aggressiveness used in prior studies of their health consequences have sometimes lacked sufficient information regarding psychometric characteristics, especially construct validity (Smith, 1992; Smith & Gallo, 2001). As a result, the specific individual difference assessed is unclear in some instances. This problem sometimes arises because investigators use preexisting data in which some sort of personality information was collected previously, and the later health of participants is determined in order to test the psychosomatic hypotheses. This approach facilitates the rapid accumulation of longitudinal studies, but at the potential cost of less than ideal assessments of personality traits.

Common Assessments

The two most common types of assessments in this literature are behavioral ratings derived from the Type A structured interview (SI) (Rosenman, 1978) and self-report questionnaires (for a review, see Smith & Gallo, 2001). The most significant recent advance in SI-based behavioral ratings of hostility has been the Interpersonal Hostility Assessment Technique (IHAT) developed by Barefoot and colleagues (Brummett, Maynard, Haney, Siegler, & Barefoot, 2000; Haney et al., 1996). Four types of hostile behavior are rated on the basis of style rather than content of responses: direct challenges to the interviewer, indirect or more subtle challenges, hostile withholding of information or evasion of the question, and irritation. The IHAT ratings can be made reliably, are stable over time, and have been found to have significant, expected associations with the outcome criteria of coronary artery disease (CAD) and CHD incidence (Brummett et al., 2000; Haney et al., 1996; Matthews, Gump, Harris, Haney, & Barefoot, 2004). The construct validity of IHAT ratings, as reflected in significant correlations with other measures of hostility (i.e., convergent validity) and smaller associations with conceptually dissimilar constructs (i.e., divergent or discriminant validity), is less well established. Recently, measures of facial expression of emotion have been used in this context. For example, IHAT rating

are associated with expressions of disgust and anger (Brummett et al., 1998), and similar behavioral ratings of hostility have been associated with fewer facial displays of friendly appeasement (Prkachin & Silverman, 2002).

The most widely used self-report measure has been the Cook and Medley (1954) Hostility (Ho) Scale. Evidence of convergent and discriminant validity, using several methods, supports the interpretation of the Ho scale as a measure of cynicism and mistrust (see Barefoot & Lipkus, 1994, Smith, 1992, and Smith & Gallo, 2001, for reviews). However, the Ho scale consistently correlates with characteristics outside the conceptual definition of hostility (e.g., anxiety and depressive symptoms) and has a poorly defined internal structure (e.g., Contrada & Jussim, 1992; Steinberg & Jorgensen, 1996). Several more homogeneous subsets of Ho scale items have been proposed (e.g., Barefoot, Dodge, Peterson, & Dahlstrom, 1989; Costa, Zonderman, McCrae, & Williams, 1986), but these subscales also require further evaluations of construct validity.

The Buss-Durkee Hostility Inventory (Buss & Durkee, 1957) has been used in this literature, and factor analytic studies suggest that it assesses two correlated dimensions—expressive or antagonistic hostility and experiential or neurotic hostility (Bushman, Cooper, & Lemke, 1991; Costa, McCrae, & Dembroski, 1989; Musante, MacDougall, Dembroski, & Costa, 1989). The former dimension involves verbal and physical aggressiveness and has larger correlations with the Five-Factor Model (FFM) trait of antagonism than with neuroticism. The latter involves subjective experiences such as resentment, suspicion, mistrust, and irritation, and has a larger correlation with neuroticism than antagonism. Buss and Perry (1992) developed a revised measure—the Aggression Questionnaire (AQ)—that includes subscales assessing trait anger, hostility, verbal aggressiveness, and physical aggressiveness. Although refinements in the scale have been suggested (e.g., Harris, 1995), evidence of construct validity has emerged in analyses of self-reports and spouse ratings (Gallo & Smith, 1998; Smith & Gallo, 1999).

Other scales derived from the MMPI-2 have been used in this literature (e.g., Kawachi, Sparrow, Spiro, Vokonas, & Weiss, 1996). There is at least some evidence of construct validity for these scales, but systematic evaluations with an established personality framework are rare. The State-Trait Anger Expression Inventory (Spielberger, et al., 1985) contains subscales for state and trait anger, and

styles of anger expression (i.e., anger-in and anger-out). These scales have excellent psychometric properties, including construct validity. However, in other health studies, scales intended to assess anger, hostility, or aggressive behavior are developed in an ad hoc manner from existing data sets, with little formal evidence of construct validity (e.g., Chang, Ford, Meoni, Wang, & Klag, 2002; Gallacher, Tarnell, Sweetnam, Elwood, & Stanfield, 1999). This is often true for scales intended to assess anger-expression styles, and it is clear that overt expression and suppression are not the only two possible characteristic styles of responding when angry (e.g, Linden et al., 2003).

Using the Interpersonal Perspective in Assessment Research

Understandably, more attention has been paid to the grand hypothesis that these traits predict health outcomes than to the smaller, but essential, embedded hypothesis that the relevant scales do indeed tap the intended trait. Further, despite mounting evidence that these individual differences are correlated with each other and with conceptually quite different risk factors (e.g., low social support, interpersonal conflict), most studies report analyses of a single personality trait. When more than one risk factor is studied, their overlap is often treated as a problem to be overcome through statistical control, rather than as a clue to more basic dimensions or processes conferring risk. We have suggested that the assessment tradition and techniques in the interpersonal approach are useful in addressing these concerns (Gallo & Smith, 1998, 1999).

The IPC provides a conceptual framework with well-established assessments (i.e., a nomological net; Gurtman, 1992) for validating anger, hostility, and aggressiveness scales. In an extension of the IPC as a trait taxonomy, Trapnell and Wiggins (1990) suggested that the friendliness versus hostility and dominance versus submissiveness dimensions can replace the agreeableness versus antagonism and extraversion versus introversion traits in the five-factor model (FFM) of personality, given their close correlation. Within the IPC, agreeableness maps closely onto the friendliness axis, with a small secondary association with submissiveness. Extraversion is closely associated with dominance, with a moderate secondary association with friendliness (Hofstee, deRaad, & Goldberg, 1992; McCrae & Costa, 1989). The IPC is even more useful as a nomological net when combined the other three FFM traits (i.e., neuroticism, conscientiousness, and

openness to experience), as the full FFM has been used to compare, contrast, and integrate health-relevant personality traits (Costa & McCrae, 1987; Marshall et al., 1994; Smith & Williams, 1992).

In an example of this use of the IPC and FFM, we evaluated the construct validity of the AQ scales (Gallo & Smith, 1998) using a version of Interpersonal Adjective Scales (Trapnell & Wiggins, 1990). In this approach, the multiple correlation between the AQ subscales and the two dimensions of the circumplex is equivalent to the "vector length" or strength of association with the dimensions of the IC, providing an index of the "interpersonalness" of the trait (Gurtman, 1992). The relative associations with the two IPC dimensions describe the specific type of social behavior assessed by the scale (e.g., Gurtman, 1992; Wiggins & Broughton, 1991). As depicted in Figure 4, all four AQ subscales were correlated with the cold and unfriendly style of IPC hostility. However, the subscales displayed differing associations with dominance; trait anger and physical aggressiveness were unrelated to this IC dimension, the AQ hostility scale was associated with submissiveness, and verbal aggressiveness was associated with dominance. Consistent with

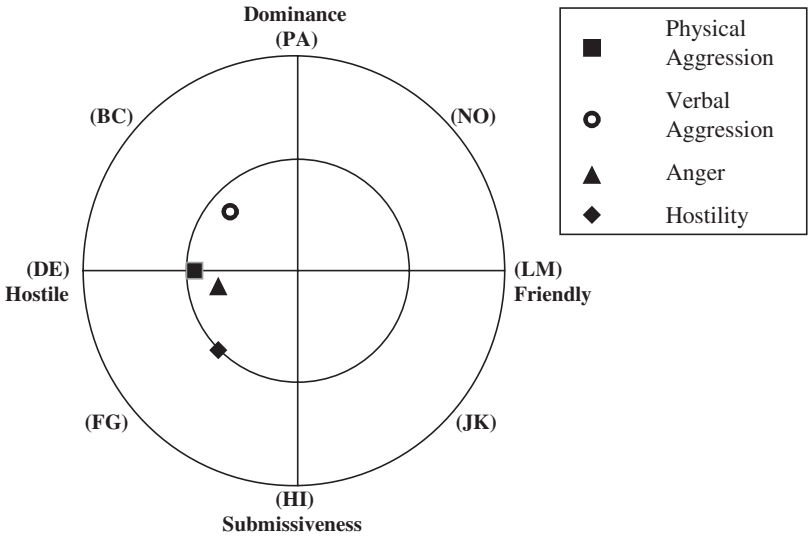

Figure 4
Association of AQ scales with circumplex dimensions (from Gallo & Smith, 1998).

prior research (Costa et al., 1989), trait anger and hostility were more closely related to neuroticism than were verbal and physical aggressiveness.

In a similar analysis of the Spielberger et al. (1985) anger-in and anger-out scales (Ruiz, Smith, & Uchino, 2002), we found that anger-in was associated with hostile submissiveness in the IPC and with neuroticism. In contrast, anger-out was associated with hostile dominance, had a smaller correlation with neuroticism than did anger-in, and was also associated with low conscientiousness. It is important to note that both anger-in and anger-out were associated with the cold and unfriendly style of the IPC hostility dimension. Perhaps this common correlate explains why both anger styles have been found to confer risk of disease.

These IPC analyses are also useful in the evaluation of scales intended to assess other interpersonal traits where associations with hostility would be examined as evidence of divergent, rather than convergent, validity. For example, studies using both behavioral ratings (Houston, Babyak, Chesney, Black, & Ragland, 1997; Houston, Chesney, Black, Cates, & Hecker, 1992; Siegman, Townsend, Civelek, & Blumenthal, 2000) and self-reports of dominance (Whiteman, Deary, Lee, and Fowkes, 1997) have suggested that this trait is associated with increased risk of CHD and premature death. These results are consistent with a well-developed nonhuman primate model of susceptibility to CAD under conditions of chronic stress (Kaplan & Manuck, 1998). Siegman, Kubzansky, and colleagues (2000) recently developed MMPI-2 scales to assess dominance and trait anger, and in a large prospective study they found that these two scales were independently associated with subsequent CHD. We examined the IPC and FFM correlates of these two scales (Corral, Smith, Glazer, & Allen, 2003) and found that, as expected, the trait anger scale was associated most closely with the FFM trait of neuroticism and the hostility axis of the IPC. Also as expected, the MMPI-2 dominance scale was associated with dominance in the IPC, but it also had a significant, albeit slightly smaller, association with the hostility axis. Hence, the dominance scale is more accurately described as a measure of hostile dominance or aggressiveness. The fact that the dominance scale predicted CHD even when trait anger was controlled statistically suggests that dominance itself confers risk, but the overlap with hostility warrants caution in interpreting the results of Siegman, Kubzansky et al. (2000).

Associations of anger, hostility, and aggressiveness scales with dimensions of the IPC clarify the constructs they assess. These patterns also indicate different interpersonal styles that could, in turn, reflect differing mechanisms linking these traits with health. That is, construct validation using the interpersonal framework could also suggest refinements in models of the processes underlying associations between these traits and subsequent health. For example, the hostile dominance associated with verbal aggressiveness and anger-out scales suggests that these traits might influence health through a pattern of "moving against" people (Horney, 1950). In an adversarial and controlling style, persons high in hostile dominance are likely to make frequent effortful attempts to influence others, often eliciting equally unfriendly resistance. Efforts to influence and control others evoke increases in blood pressure and heart rate (Smith, Allred, Morrison, & Carlson, 1989; Smith, Ruiz, & Uchino 2000), and this reactivity could contribute to cardiovascular disease (Manuck, Marsland, Kaplan, & Williams, 1995; Treiber et al., 2003). In contrast, the hostile submissiveness associated with hostility and anger-in scales suggests a pattern of "moving away" in which a less confident and assertive individual may engage in chronic vigilance to detect potential mistreatment he or she cannot otherwise easily manage. This wariness could lead to low levels of social support. Vigilance and low social support have also been associated with heightened cardiovascular reactivity (Kamarck, Peterman, & Raynor, 1998; Lepore, 1998, Smith et al., 2000).

These speculations regarding recurring social styles and interpersonal consequences associated with anger, hostility, and aggressiveness suggest an additional use of the interpersonal approach to assessment, one that addresses the implications of the transactional cycle. As described above, the transactional cycle suggests that characteristics of the person and recurring features of the social environment are two aspects of a single personality process. We would argue further that, in assessing psychosocial risk, personality traits and characteristics of the social environment should be measured more comprehensively, rather than the typical practice of assessing only characteristics of the person or the social environment. These multiple measures could then be considered simultaneously to capture interpersonal patterns. When studies have assessed multiple risk factors, correlated predictors are often forced to compete in multivariate analyses examining their independent predictive effects.

Although the independence of risk factors is clearly important in epidemiological research, this traditional approach can also be seen as forcing artificial independence upon components of a single risk process.

To illustrate an alternative, we administered the AQ, a multidimensional measure of social support (Cohen, Mermelstein, Kamarck, & Hoberman, 1985) and other measures to two samples (Gallo & Smith, 1999). In replicated cluster analyses, we identified three groups as a function of both AQ and social support subscale scores. As depicted in Figure 5, an affiliative, low-risk group reported low levels of anger, hostility, and aggressive behavior and high levels of social support. A hostile-isolated group reported low levels of social support and had high scores on the AQ subscales, with their highest scores on the hostility subscale. The third group reported high social support similar to the low-risk group, but had elevated AQ subscale scores. Unlike the hostile-isolated group, this latter hostile group had higher scores on AQ aggressiveness subscales than AQ hostility. In IPC analyses, both hostile groups described themselves as much less friendly than did the affiliative group, and the hostile-isolated group differed from the other two by reporting greater submissiveness. Although limited by the use of a single source and method, these analyses illustrate the assessment of psychosocial risk though concurrent measurement of "both halves" (i.e., the person and the social environment) of the transactional cycle. We identified transactional patterns that could be used in studies of health, by emphasizing, rather than ignoring or controlling, covariation between personality and social processes.

Associations of Anger, Hostility, and Aggressiveness With Subsequent Health

At the time of our earlier review (Smith, 1992), most studies of hostility and subsequent health found significant effects, but there were several failures to replicate this finding. To update that review and provide a more accurate summary of the literature, we conducted a meta-analysis and found that behavioral ratings and self-reports of hostility were significantly associated with CHD and premature mortality, despite inconsistencies across studies (Miller, Smith, Turner, Guijarro, & Hallet, 1996). Subsequent reviewers have reached similar conclusions (e.g., Gallo & Matthews, 2003; Kop,

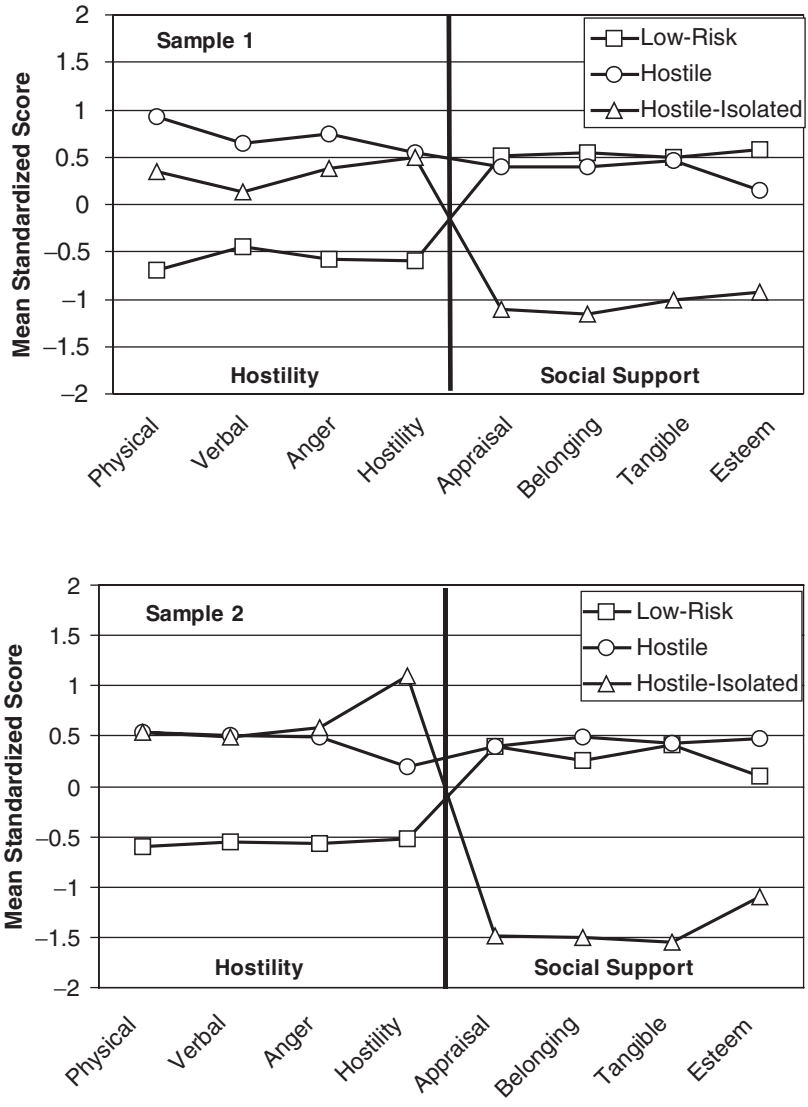

Figure 5
Psychosocial risk clusters (from Gallo & Smith, 1999).

1999; Rozanski et al., 1999). Another recent meta-analysis found that these traits predict the development of hypertension (Rutledge & Hogan, 2002), and a subsequent study using a large sample of black and white young men and women replicated this effect

(Yan et al., 2003). However, our quantitative review has been criticized as possibly reflecting a publication bias against null results or inadequate control of confounds in the studies included in the meta-analysis (Petticrew et al., 1999). A more recent quantitative review concluded that the association was reliable, but so small as to be unimportant (Myrtek, 2001). We now briefly review studies of CHD incidence and mortality appearing after our quantitative review, as well as recent studies of the association of these risk factors with CHD across various stages of its development.

Recent Prospective Studies

Recent case-control studies have reported significant cross-sectional associations between measures of anger or hostility and CHD in both men (e.g., Meesters, Muris, & Backus, 1996) and women (e.g., Lahad, Heckbert, Koepsell, Psaty, & Patrick, 1997). However, cross-sectional studies are an inherently weaker test of this psychosomatic hypothesis than prospective studies. Most—but not all—of the recent prospective studies have reported significant effects. For example, in a sample of 1305 older men in the VA Normative Aging Study (NAS), those reporting high levels of trait anger on a scale derived from the MMPI-2 were three times as likely either to die from CHD or experience a nonfatal myocardial infarction over a 7-year follow-up period than were men reporting low scores (Kawachi et al., 1996), even when demographic, medical, and behavioral risk factors were controlled. In two other analyses of this sample, the Cook and Medley (1954) Ho scale and an MMPI-derived dominance scale similarly predicted CHD (Niaura et al., 2002; Siegman, Kubzansky, et al., 2000). As noted above, our IPC-based validation suggests that this dominance scale assesses hostile dominance (Corral et al., 2003), akin to the construct of aggressiveness. Obviously, these three reports are not independent tests, but as a group they support the association of these traits and CHD.

In an analysis of the Multiple Risk Factor Intervention Trial, Matthews et al. (2004) found that IHAT ratings above the sample median were associated with a 60% increased risk of cardiovascular death over a 16-year follow-up, compared to men with lower hostility scores, even when controlling traditional risk factors. These men were initially free of CHD, but at elevated risk based on the traditional risk factors. In a study of over 2,000 Finish men, scores

on an 8-item measure adapted from the Ho scale predicted cardiovascular death and all-cause mortality over a 9-year follow-up period; among men with no prior CHD, hostility was associated with increased risk of myocardial infarction (Everson et al., 1997). Compared to men in the lowest quartile of hostility scores, those in the highest quartile were at approximately twice the risk for these negative health outcomes. Although adjustment for biomedical and demographic risk factors did not alter these effects, statistical control of behavioral risk factors (e.g., smoking, physical activity, alcohol consumption) largely eliminated them. In subsequent reports from this sample, men reporting high levels of expressed anger were at significantly increased risk of stroke, controlling for several other risk factors (Everson et al., 1999).

In another recent study, high scores on a 3-item, self-report measure of trait anger were associated with cardiovascular disease, CHD, and myocardial infarction in a sample of 1,000 men followed for more than 30 years (Chang et al., 2002). The risk for these outcomes attributable to high levels of anger ranged from a three- to six-fold increase. These effects were significant when controlling for traditional medical, demographic, and behavioral risk factors, and the associations were much stronger for disease outcomes occurring before the age of 55. In a study of nearly 3,000 middle-aged men, anger suppression (i.e., low anger-out and high anger-in) was associated with increased risk of CHD over a 9-year follow-up period, independent of several demographic, behavioral, and medical control variables (Gallacher et al., 1999). However, as with the Chang et al. (2002) study, the measure used to assess these aspects of anger coping style has not undergone extensive validation.

In the prospective studies described above, the samples have consisted largely of white men. Hence, a recent study of nearly 13,000 white and black men and women has been particularly informative. In the Atherosclerosis Risk in Communities Study (ARIC), higher scores on the Spielberger et al. (1985) trait anger scale were associated with a 50%–75% increased risk of CHD (Williams et al., 2000) over a 4.5 year follow-up. This association occurred even when controlling behavioral, biomedical, and demographic risk factors and was larger among initially normotensive individuals. This well-validated trait anger scale contains two correlated factors, and subsequent analyses indicated that angry temperament (i.e., quick, intense, and minimally provoked anger) was more closely related to subsequent CHD than

was a factor reflecting angry reactions to potentially aggravating events (e.g., criticism, unfair treatment, etc.) (Williams, Nieto, Sanford, & Tyroler, 2001). In another report from the ARIC study, high trait anger was associated with increased risk of stroke among participants 60 years of age or younger, even with statistical control of biomedical, demographic and behavioral risk factors (Williams, Nieto, Sanford, Couper, & Tyroler, 2002). Importantly, across all three of these reports, effects were similar across sex and ethnicity.

In contrast to the prospective results from the six samples described above, two studies reported null findings. In a sample of over 20,000 initially healthy male health professionals, scores on the anger-out subscale of the Spielberger scale did not predict the development of CHD over a 2-year follow-up period (Eng, Fitzmaurice, Kubzansky, Rimm, & Kawachi, 2003). Similarly, in a sample of over 9,000 initially healthy French and Irish men, self-reported hostility was not associated with CHD over a 5-year follow-up (Sykes et al., 2002).

Some recent studies indicate that anger and hostility are not associated with morbidity or mortality among patients with established CHD (Kaufman et al., 1999; Welin, Lappas, & Wilhelmsen, 2000). However, in a study of nearly 800 women with CHD, those in the highest quartile of Ho scale scores were twice as likely to suffer a recurrent myocardial infarction than women with low Ho scores, controlling for a wide range of biomedical, behavioral, and demographic variables (Chaput et al., 2002). Similarly, among men in the MRFIT sample who experienced a nonfatal cardiovascular event during the initial study phase, high levels of hostility indicated by IHAT ratings were associated with a five-fold increased risk of cardiovascular death later in the follow-up period (Matthews et al., 2004). Behavioral ratings and self-reports of hostility have been found to predict restenosis of coronary arteries following angioplasty (Goodman, Quigley, Moran, Meilman, & Sherman, 1996; Mendes De Leon, Kop, de Swart, Bar, & Appels, 1996). In a study of 150 patients with preexisting, angiographically documented CAD, self-reported anger-out predicted the progression of CAD at the time of a second angiogram 2 years later, but Ho scale scores did not (Angerer et al., 2000). Hence, although supportive findings have been reported, the role of these traits in the course of established CHD is less clear than it is for cardiovascular events and mortality among initially healthy persons.

Associations with Early and Late Components of CHD

Diagnosable CHD occurs in later adulthood after a decades-long period of silent progression of CAD, beginning with microscopic changes in the artery wall and progressing to larger but still asymptomatic plaques. Only when occlusions become quite extensive or when advanced plaques become unstable do symptoms of CHD emerge. Hence, associations of anger, hostility, and aggressiveness with myocardial infarction and coronary death could reflect effects on the initial or later development of CAD, the emergence of manifestations of CHD (e.g., ischemia and precipitation of acute events) among persons with advanced CAD, or a combination of these effects (Kop, 1999; Krantz & McCeney, 2002; Smith & Ruiz, 2002).

Prior studies of these traits and the severity of CAD among patients undergoing coronary angiography generally revealed significant associations (for reviews, see Rozanski et al., 1999; Miller et al., 1996). Given that most of these studies included disproportionately male samples, it is important to note that in a recent study, behavioral ratings of hostility were associated with angiographically documented CAD in a small sample of women (Low et al., 1998). However, due to the invasive nature of angiography, patients are rarely referred without compelling reason. Therefore, these studies are limited by a) an overrepresentation of participants with significant disease and b) the fact that the disease-free persons in clinically selected samples are not representative of the general population. Thus, angiography studies provide an awkward test of the association between psychological traits and asymptomatic atherosclerosis.

Recently, noninvasive methods of assessing atherosclerosis have provided more informative tests. Given their low-risk nature, these tests can be used more easily with representative samples. For example, in a study of nearly 200 postmenopausal women, brachial ultrasound tests of endothelial dysfunction indicated that a composite measure of anger and aggressiveness was associated with this early indication of atherosclerosis (Harris, Matthews, Sutton-Tyrell, & Kuller, 2003), and similar associations have been reported between hostility and endothelial dysfunction following psychological stressors (Gottdiener et al., 2003). Ultrasound tests can also provide information about the presence and severity of carotid atherosclerosis. Self-reports of hostility have been associated with more extensive and more rapidly progressing carotid artery disease in such

studies (Julkunen, Salonen, Kaplan, Chesney, & Salonen, 1994; Matthews, Owens, Kuller, Sutton-Tyrrell, & Jansen-McWilliams, 1998). This association could account for the prospective association between hostility and ischemic stroke described above, but it also provides indirect information about CAD, given the reliable association between carotid atherosclerosis and CAD.

Recent developments in CT-scan technology have provided similarly noninvasive assessments of CAD itself, through the imaging of calcium deposits (a component of atherosclerotic plaques) in the coronary arteries. In a representative sample of nearly 400 white and black men and women aged 28–40 at the time of CT scans, Ho scale scores above the sample median (obtained at least 5 years previously) were associated with a 2.5 times greater risk of detectable calcium deposits and 9.5 times greater risk of larger deposits. Further, these associations were independent of demographic, lifestyle, and biomedical risk factors (Iribarren et al., 2000). However, a second study using this approach reported no association in a sample of active duty military personnel (O'Malley, Jones, Feuerstein, & Taylor, 2000). Studies of the progression of CAD on repeat angiography (Angerer et al., 2000) and restenosis following angioplasty (Goodman et al., 1996; Mendes De Leon et al., 1996) are also consistent with the view that hostility is associated with more rapid progression of CAD, but these select clinical samples, and the late stage of the disease warrant caution in such a conclusion.

Several research strategies have been used to examine anger, hostility, and related factors in the emergence of manifestations of CHD. For example, among patients with established disease, hostility is associated with more readily evoked myocardial ischemia during psychological and physical stressors, as well as during daily activities (Burg, Jain, Soufer, Kerns, & Zaret, 1993; Helmers et al., 1993). In a study of men with CHD, facial displays of anger but not Ho scale scores were associated with ischemia during the Type A Structured Interview (Rosenberg et al., 2001). Among patients with CAD, experimentally induced anger can evoke myocardial ischemia (Gabbay et al., 1996; Ironson et al., 1992). In two studies in which patients hospitalized for myocardial infarction were interviewed about the period of time immediately preceding their heart attack and a control period of time, episodes of anger were twice as common during the two hours before MI than during the control period (Mittleman et al., 1995; Möller et al., 1999), suggesting that anger

can precipitate acute coronary events. Although the recall methodology warrants caution, this method also found that smoking and vigorous physical activity were more common immediately before MI. These studies of patients with CHD suggest that anger and hostility can contribute to later stages of the disease process. However, as noted above, in prospective studies, anger, hostility, and related traits are more consistently related to the initial occurrence of CHD than to recurrent coronary events (Hemingway & Marmot, 1999; Miller et al., 1996). It may be that studies of recurrent events are subject to a selection bias; angry or hostile persons who survive the initial occurrence of their disease may more be resilient for some reason and therefore at lower risk of recurrence (Williams, 2000).

Conclusions Regarding Association and Implications of the Interpersonal Perspective

Overall, measures of anger, hostility, and aggressiveness are associated with increased risk of CHD and reduced longevity. In multiple reports from six of eight prospective studies of initially healthy samples published since our prior quantitative review (Miller et al., 1996), measures assessing these traits predict CHD incidence and/or mortality. Further, the effect sizes in these studies are as large as those associated with many traditional risk factors, and the associations are found even when a wide variety of potential confounding factors are controlled. However, wide variations in study quality and two conflicting findings suggest a continuing need for additional research, especially studies with large and diverse samples, adequate follow-up periods, well-validated personality measures, and careful statistical controls. Hence, this literature has continued to improve in methodological quality and the consistency of findings, but not to the point that additional sound studies are unnecessary. Prospective studies of these traits and the course of established CHD are also needed, given inconsistencies in this literature and its variable quality. Other recent research suggests that anger, hostility, and aggressiveness may contribute to CHD through both the initial development of CAD and the precipitation of manifestations of CHD among those with more advanced disease.

As noted above, the interpersonal approach has several implications for future research on the association of anger, hostility, and aggressiveness with CHD. First, IPC analyses suggest that variations

among these traits in their association with the second major dimension of social behavior—dominance—may be an important consideration. Hence, the simultaneous measurement of these traits and dominance—perhaps with IPC based scales—may be useful in future epidemiological research. This is particularly true given that dominance influences CAD and CHD in both animal (Kaplan & Manuck, 1998) and human studies (Houston et al., 1992; Houston et al., 1997; Siegman, Kubzansky, et al., 2000; Whiteman, Deary, Lee, & Fowkes, 1997).

Prospective studies of anger-expression styles suggest that these traits may predict later health, but results are inconsistent. Further, it is somewhat contradictory that both styles can confer risk. Several studies have used poorly validated measures of these traits, and hence, use of refined measures of these styles (perhaps validated using the IPC/FFM approach described above) is important. Further, anger-in and anger-out are both associated with trait anger and the hostility dimension of the IPC. Simultaneous assessment of anger, hostility, dominance, and anger-expression styles would help to determine if their associations with health involve what the anger expression styles have in common (i.e., anger and hostility) and/or what is unique about them (i.e., hostile dominance vs. hostile submissiveness).

Also, as discussed above, consistent with the transactional cycle, these traits and aspects of the social environment could be sampled simultaneously and used together to quantify risk factors. The approach outlined above (Gallo & Smith, 1999) could be used to identify subgroups on the basis of patterns of scores on measures of the social environment and anger, hostility, and aggressiveness. Some initial support for this general view is suggested by studies in which the combination of high anger or hostility and low social support was associated with atherosclerosis or CHD (Angerer et al., 2000; Knox, et al., 2000; Knox, et al., 1998).

Mechanisms Linking Anger, Hostility, and Aggressiveness With CHD

In our prior review (Smith, 1992), we discussed research on several different mechanisms potentially underlying the statistical associations of hostility with subsequent health. This literature has grown considerably, and the interpersonal perspective may facilitate further progress in evaluating most if not all of these models.

The Psychophysiological Reactivity Model

As initially described by R. B. Williams, Jr. and colleagues (1985), this model suggests that hostility confers risk through the mechanism of exaggerated cardiovascular (e.g., increases in blood pressure and heart rate) and neuroendocrine (e.g., epinephrine, norepinephrine, cortisol, etc.) responses to potential stressors. A growing body of research supports the hypothesis that these responses can contribute to the initiation and progression of CAD and to the manifestations of CHD (Kop, 1999; Rozanski et al., 1999; Smith & Ruiz, 2002; Treiber et al., 2003). Prior reviews suggested that the nature of the stressor was a key consideration in the association of hostility and related traits with these stress responses. Specifically, initial evidence suggested that hostile persons responded to relevant interpersonal stressors with heightened reactivity relative to nonhostile individuals but not to nonsocial stressors often used to study reactivity, such as mental arithmetic tasks (Houston, 1994; Smith, 1992). Subsequent research has produced a variety of findings consistent with this view (Smith & Gallo, 2001).

For example, Suarez, Kuhn, Schanberg, Williams, and Zimmermann (1998) randomly assigned men with high and low Ho scores to undergo a solvable anagram task while experiencing either harassing or nonharassing comments from the experimenter. Compared to low-hostile participants, hostile participants displayed larger increases in blood pressure, heart rate, and neuroendocrine levels (i.e., norepinephrine, cortisol, and testosterone) during and shortly after the task, but only in the harassment condition. Similar studies have also reported results consistent with this basic prediction of a statistical interaction between hostility and relevant social stress (e.g., Miller et al., 1998; Smith, Cranford, & Green, 2001). However, when the friendly versus hostile style of the experimenter is manipulated within the context of an already engaging social stressor (e.g., evaluative public speaking), some studies have found main effects for both trait hostility and the situational manipulation of hostility (e.g., Gallo, Smith, & Kircher, 2000). Other relevant social stressors that evoke heightened reactivity among hostile participants include the recall and discussion of past anger-inducing events (Frederickson et al., 2000), current events discussions or debates (e.g., Davis, Matthews, & McGrath, 2000), watching anger-inducing films (Fang & Myers, 2001), and self-disclosure (Christensen & Smith, 1993, but see

Kurylo & Gallant, 2000, for a negative study). Importantly, not all studies have supported the model, and some negative results may be attributable to the fact that hostile participants respond to less compelling stressors with decreased task engagement (Piferi & Lawler, 2000). Recent theory and research suggests that impaired physiological recovery from social stressors among hostile persons might be at least as important as heightened reactivity (Brosschot & Thayer, 1998; Llabre, Spitzer, Siegel, Saab, & Schneiderman, 2004). Consistent with prior reviews (Houston, 1994), the association of individual differences in anger-coping style with reactivity has not been straightforward in recent studies (e.g., Sudchay & Larkin, 2001). This might reflect a complex pattern in which the fit between situational requirements for expression and dispositional expression styles determines reactivity (Engebretsen, Matthews, & Scheier, 1989).

These traits are also associated with heightened reactivity during interactions with actual social network members (Miller et al., 1999; Smith, Uno, Uchino, & Ruiz, 2000). Further, these effects occur outside the laboratory, as trait hostility has been associated with ambulatory blood pressure (e.g., Benotsch, Christensen, McKelvey, 1997; Guyll & Contrada, 1998; Polk, Kamarck, & Shiffman, 2002; Raikkonen, Matthews, Flory & Owens, 1999).

Hostility and physiological reactivity during marital interaction. Research in our laboratory has examined the association between hostility and physiological responses during marital interaction. Stressful marital interactions are common and evoke pronounced cardiovascular and neuroendocrine reactivity (Kiecolt-Glaser & Newton, 2001). Further, marital discord and disruption are associated with increased risk of cardiovascular disease and related mortality (e.g., Matthews & Gump, 2002; Orth-Gomer, et al., 2000). Hence, the social psychophysiology of marital interaction is a potentially important context for examining reactivity as a mechanism potentially linking hostility and health.

In a recent study of 60 young married couples (Smith & Gallo, 1999), we manipulated an agency stressor (i.e., high vs. low evaluative threat) and a communion stressor (i.e., agreement vs. disagreement) for couples participating in a current events discussion task. Among husbands, high AQ total scores (and hostility subscale scores) were associated with larger systolic blood pressure responses

in the high, but not the low, evaluative threat condition. The disagreement manipulation did not interact with AQ scores to influence husbands' reactivity; wives' AQ scores were not related to their reactivity. After the task, husbands and wives rated each other's behavior during the discussion on the IAS measure of IPC hostility and dominance. Interestingly, in the high evaluative threat condition (i.e., agency stressor) wives rated husbands with high AQ scores as expressing more dominance during the interaction than did wives of husbands with low AQ scores, but not in the low threat condition.

We interpreted this pattern as suggesting that hostile husbands responded to the agency stressor with increased assertion of social dominance, and that consistent with prior research (Smith et al., 1989; 2000), such efforts evoked heightened reactivity. This interpretation is consistent with the results of a previous study, in which we similarly found that husbands' Ho scale scores were associated with heightened cardiovascular reactivity when they had an incentive to assert social influence over their wives (Smith & Brown, 1991). Interestingly, in the current study we also found that in the disagreement condition (but not the agreement condition), the wives of husbands with high total AQ scores displayed greater heart rate reactivity than did wives of men with lower AQ scores. Wives' AQ scores were unrelated to their husbands' cardiovascular responses. Hence, again consistent with prior research (Smith & Brown, 1991), women interacting with angry, hostile, and aggressive husbands displayed cardiovascular reactions hypothesized to place them at risk for CHD.

In a second recent study, we examined a stressor intended to resemble more closely the stressful interactions commonly experienced in close relationships. Further, we examined the association of both the AQ anger and hostility subscales with cardiovascular responses. A recent prospective study in our laboratory indicated that wives' trait anger was more closely related to increases in their own and their husbands' marital distress than was wives' trait hostility (Glazer, Smith, Nealey-Moore, & Hawkins, 2002a), and we hypothesized that wives' trait anger might prove to be a better predictor of reactivity as well. We asked 80 couples to discuss either their typical daily schedule as a low-stress task or characteristics they disliked about each other as a high-stress task (Smith, Nealey-Moore, Uchino, & Hawkins, 2003). Consistent with the psychophysiological reactivity model (Houston, 1994; Smith, 1992), high-hostile husbands in the

high-stress condition displayed larger increases in blood pressure, heart rate, and cardiac output (i.e., the volume of blood leaving the heart each minute), as well as larger decreases in cardiac pre-ejection period (an indication of heightened sympathetic nervous system stimulation of the heart) than did low-hostile husbands in this condition and both groups of husbands in the low-stress discussion. As in our prior study, wives of hostile husbands also displayed greater cardiovascular reactivity during the high-stress discussion. Husbands' trait anger was unrelated to their own or their wives' reactivity.

A different pattern emerged for wives. Consistent with the effects of wives' trait anger on marital adjustment (Glazer et al., 2002a), high-trait-anger wives in the high-stress discussion displayed larger increases in blood pressure and heart rate than did the other three groups of wives. The husbands of high-trait-anger wives in the high-stress condition displayed larger increases in cardiac output and larger decreases in total peripheral resistance than did the other three groups of husbands, a physiological pattern that reflects effortful task engagement. High-trait-anger wives in the high-stress discussion reported much larger increases in state anger than did the other three groups and rated—and were rated by—their husbands on IPC scales as less warm. In addition, the high-trait-anger wives in this condition were rated by their husbands as much more dominant than any other group. We interpreted these results and our prior studies on reactivity during marital interaction to suggest that marital stressors can evoke heightened reactivity consistent with the psychophysiological reactivity model, but that the specific traits most closely related to reactivity in this context may differ for men and women. Further, interactions evoking both dominance and unfriendliness influence the psychophysiological correlates of trait anger and hostility in the marital context. Finally, these traits are also related to the spouses' physiological responses, perhaps indicating that either partner's anger and hostility can make marital interactions generally stressful.

Recent extensions of the psychophysiological reactivity model. These traits have been linked to resting levels and stress—induced increases in plasma lipids (Finney, Stoney, & Engebretson, 2002; Siegman et al., 2002; Suarez, Bates, & Harralson, 1998; Vogele, 1998). Elevations in total and low-density cholesterol and triglycerides could certainly contribute to the effects of hostility on CAD and CHD. Further, these effects may be part of a broader association between

hostility and the metabolic syndrome, a combination of an unhealthy lipid profile, high levels of insulin, insulin resistance, visceral body fat, and elevated blood pressure (Niaura et al., 2000; Raikkonen, Matthews, Kuller, Reiber, & Bunker, 1999; Raikkonen, Matthews, & Kuller, 2002; Surwit et al., 2002). Although one recent study indicated that these characteristics do not account for the prospective association between Ho Scale scores and subsequent CHD (Niaura et al., 2002), these factors certainly could contribute to the health effects of hostility. Other possible mechanisms identified in recent research include activation of blood platelets (Markovitz, 1998) and elevated plasma homocysteine (Stoney & Engebretsen, 2000).

Another set of potential psychophysiological mechanisms is suggested by recent models of CAD. Rather than passive deposition of lipids in the arterial wall, newer models describe the initiation and progression of CAD as an inflammatory process (Becker, de Boer, & van der Wal, 2001; Libby, 2003; Ross, 1999). Hence, anger, hostility, and aggressiveness could be linked to CAD and CHD through mechanisms involving inflammatory processes and other components of the immune system (Kop, 2003). In novel observations consistent with this framework, Suarez, Lewis, and Kuhn (2002) recently reported that scores on the AQ total, hostility, and aggressiveness scales were positively associated with levels of tumor necrosis factor alpha, a pro-inflammatory cytokine implicated in CAD and CHD. Similar results have been reported for a second pro-inflammatory cytokine, interleukin-6 (Suarez, 2003a; Suarez, 2003b), though these effects may be moderated by levels of other psychosocial risk factors such as depressive symptoms (Miller, Freedland, Carney, Stetler, & Banks, 2003). Hence, future research on mechanisms linking these traits with health should examine psychosocial influences on the immune system (Kiecolt-Glaser, McGuire, Robles, & Glaser, 2002).

The Psychosocial Vulnerability Model

A second possible mechanism linking hostility and health focuses on the experience of social support and various sources of stress. Hostile persons report greater levels of interpersonal conflict and stress and lower levels of social support, and these psychosocial vulnerabilities could mediate the association between hostility and health (Smith,

1992). Since our prior review, several studies have supported at least portions of this model. For example, as in prior studies (e.g., Smith, Pope, Sanders, Allred, & O'Keeffe, 1988), hostile persons report low levels of social support in general (Hart, 1999; O'Neil & Emery, 2002), from friends and spouses (Glazer et al., 2002a; Smith, Uno, et al., 2000), and co-workers (McCann, Russo, & Benjamin, 1997). Further, prior observations that hostility is associated with greater interpersonal conflict and stress in general and in the context of close relationships (see Smith, 1992) have been replicated in recent studies (Gallo & Smith, 1999; Glazer et al., 2002a). These traits are also associated with increasing difficulties in close relationships over time (Miller et al., 1995; Newton & Kiecolt-Glaser, 1995; Siegler et al., 2003). For example, in a study of approximately 100 young married couples conducted in our laboratory, higher levels of wives' hostility, and especially their trait anger, were associated with decreases in their own and their husbands' reports of marital satisfaction over a 14-month follow-up (Glazer et al., 2002a).

These associations with greater social stress and reduced support can have physiological consequences, as in the reactivity model described above. In laboratory studies, for example, unlike their more agreeable counterparts. hostile persons do not respond to social support with decreased cardiovascular responses to experimental stressors (Lepore, 1995; Smith, Nealey-Moore, et al., 2000). Hence, hostility is associated with less social support and less psychophysiological benefit from it when it is available. As described above, hostility is also associated with increased reactivity to marital stressors (Smith & Gallo, 1999; Smith et al., 2003). Similarly, a recent study found that high levels of anger-out were associated with increased salivary cortisol levels among persons with high job stress (Steptoe, Cropley, Griffith, & Kirschbaum, 2000).

Recent research on the health consequences of social support has suggested that providing support to others might be more important than receiving support. The beneficial health effects of receiving support were eliminated when giving support was statistically controlled (Brown, Nesse, Vinokur, & Smith, 2003). Yet providing support reduced risk of death, even when its association with receiving support was controlled. If this result is replicated, the psychosocial vulnerability model should be expanded accordingly. In our research to date, spouses of hostile persons report getting less support from their partners than do spouses of nonhostile individuals (Smith et al.,

1988; Glazer et al., 2002a), perhaps suggesting that low levels of providing support is another psychosocial pathway linking hostility and health.

The Transactional Model

As noted above, hostile persons both experience more interpersonal difficulties and respond to them with greater physiological reactivity. They also experience less social support and display less favorable physiological responses to it when available. Hence, hostility is characterized by both greater *exposure* and *reactivity* to unhealthy psychosocial contexts (Bolger & Schilling, 1991; Bolger & Zuckerman, 1995). In the transactional model, the greater exposure occurs because hostile persons create more conflict and undermine sources of social support through a variety of cognitive and behavioral process, as in the cycle depicted in Figure 2. Given their negative views of self, others, relationships, and interaction sequences (i.e., schemata and scripts), hostile persons are likely to behave toward others in ways that increase conflict and decrease support (Smith, 1995). As depicted in Figure 6, hostility is therefore associated not only with heightened reactivity to a given stressor (e.g., the first two stressors depicted in Figure 6) but also with more frequent, pronounced, and prolonged exposure to stressors.

Although this model has provided a useful integrating framework for research on the psychophysiological and psychosocial correlates of hostility, it is difficult to test directly. One promising avenue involves ambulatory methods, in which the personality trait, level of physiological response, and exposure to relevant stress are all measured. In a preliminary test of this type, self-reported levels of interpersonal stress accounted for some of the association between hostility and ambulatory blood pressure (Benotsch et al., 1997). In another ambulatory study, hostility was associated with both more exposure to aversive social interactions and greater blood pressure responses to such interpersonal stressors (Brondolo et al., 2003).

The Health Behavior Model

Some evidence suggests that hostile persons have a less healthy lifestyle (Siegler, 1994) and that these health behaviors (e.g., smoking, activity level, etc.) could account for the association with subsequent health. For example, one recent study found that hostility in young

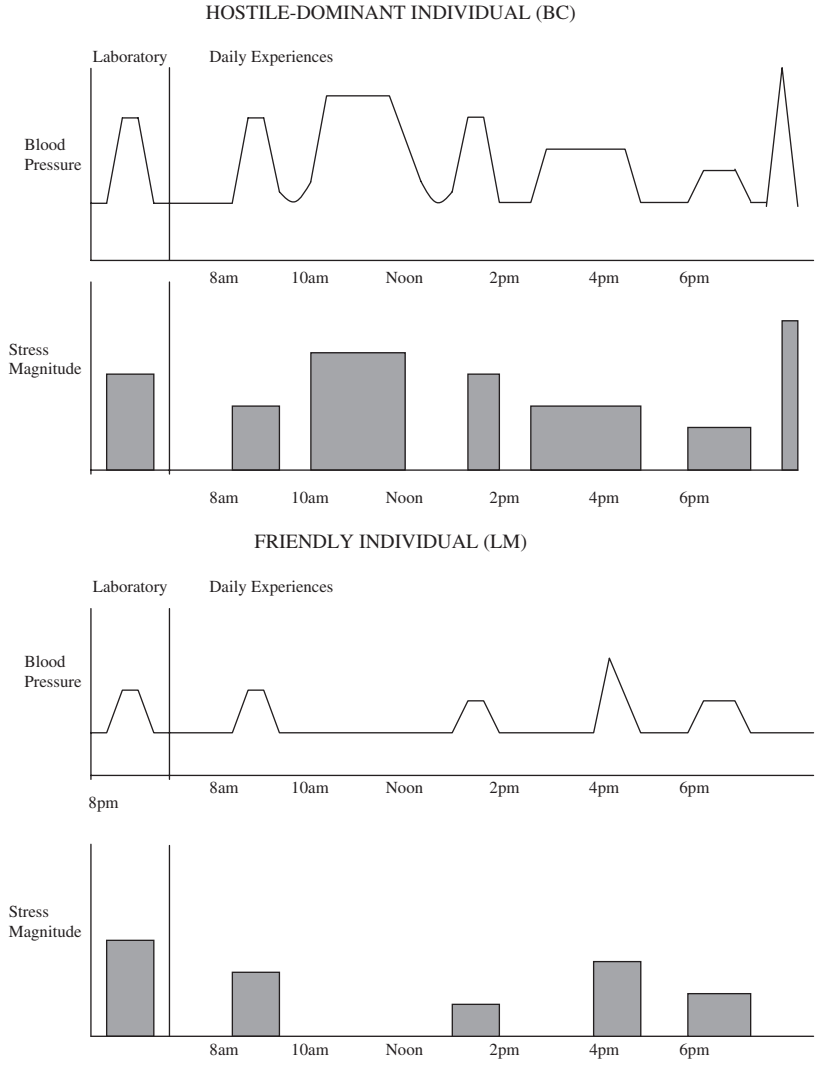

Figure 6
Transactional approach to personality, stress exposure, and reactivity (from Smith, Gallo, & Ruiz, 2003).

adults was associated with smoking and excessive alcohol use 20 years later (Siegler et al., 2003). In most prospective studies that control such risk factors, trait anger, hostility, and aggressiveness still confer risk of CHD. However, at least one large epidemiological study found that the significant association between self-reported

hostility and subsequent CHD was accounted for by these health behaviors (Everson et al., 1997).

Although this model has typically been tested by evaluating the role of traditional behavioral risk factors for the initial development of CAD and CHD (e.g., smoking, diet, etc.), many other health behaviors and other stages of the disease process could be involved. For example, hostility is also associated with decreased likelihood of smoking cessation among patients with CAD (Brummett et al., 2002) and with less adherence to medical regimens in other life-threatening chronic diseases (Christensen, Wiebe, & Lawton, 1997). Poor sleep is associated with increased risk of CHD and other causes of premature morality (e.g., Dew et al., 2003), and hostile persons are particularly susceptible to sleep disruption after interpersonal conflicts (Brissette & Cohen, 2002). Hence, future research should address a broad range of health behaviors across the course of CHD.

The Constitutional Vulnerability Model

As a general view of mechanisms linking personality and health, the constitutional vulnerability model suggests that basic, possibly genetically determined, biological, individual differences are responsible both for the personality phenotype and the increased risk of disease (Smith & Gallo, 2001). Further, the personality trait is not necessarily causally related to the later occurrence of disease at all, as both may be determined by the third variable of constitutional vulnerability. Individual differences in central serotonergic functioning have been discussed as a basic biologic trait underlying disease (Williams, 1994), and recent progress in molecular genetics has created new opportunities to test this hypothesis. For example, polymorphisms involved in serotonergic systems have been related to individual differences in hostility and aggressiveness (e.g., Manuck et al., 1999; Manuck, Flory, Muldoon, & Ferrell, 2002) and to cardiovascular responses to psychological stress (R. B. Williams et al., 2001; 2003). Currently, it is possible to test the hypothesis that these genotypes explain the statistical associations of hostility and related traits with subsequent disease, though no such studies have been reported.

Although recent studies of serotonergic genes are consistent with this model, current theory in behavioral genetics maintains that genetically influenced personality traits are shaped by social processes (Rutter & Silberg, 2002). For example, expression of such genes in

determining behavioral and psychophysiological phenotypes is moderated by exposure to aspects of the social environment (i.e., gene × environment interactions). Further, genetic factors are often correlated with these same social environmental vulnerability and resilience factors because the parents' genes have influenced the environment they provide for the developing child (i.e., passive gene–environment correlation). Finally, these genetic factors in the offspring can influence exposure to these same aspects of the social environment (i.e., active or evocative gene–environment correlation). For example, in animal models, the development of genetically based individual differences in behavioral and neuroendocrine stress reactivity can be moderated by environmentally influenced maternal behavior (Meaney, 2001). In humans, genotypes that place children at risk for aggressiveness are associated with exposure to the type of negative parenting (i.e., negative control) in adoptive families that also increases risk of aggressiveness (O'Connor, Deater-Deckard, Fulker, Rutter, & Plomin, 1998).

Hence, even if additional research supports the role of genetically determined individual differences in the development of hostility and in its association with CHD, transactional processes are likely to be important. Genetically vulnerable children tend to be born into higher- risk social environments, characterized by cold and controlling parenting and interparental conflict. Further, by virtue of their difficult emotional style or temperament, children at genetic risk for developing hostility and highly reactive, unhealthy physiologies may also evoke and maintain such parental behavior. Through the processes of identification, internalization, and introjection described above, these experiences would, in turn, promote the development of cognitive, emotional, and behavioral vulnerabilities, as well as the further expression of the genetic potential for a reactive constitution Through the transactional processes described above, these vulnerabilities would lead to increased exposure to social conflict and reduced support over time, thereby maintaining unhealthy personality processes and fostering recurring exposure to social environments that are especially damaging to individuals with reactive constitutions.

Conclusions Regarding Mechanisms and Implications of the Interpersonal Approach

Since our prior review (Smith, 1992), considerable progress has occurred in the study of these models. Results generally support the

hypothesis that hostile persons display heightened physiological responses to social stressors, though complete mediational tests of this model examining personality traits, psychophysiological responses, and health outcomes have not been reported to date. These traits are also consistently associated with increased levels of conflict and other social stressors and with lower levels of social support. However, no mediational analyses have examined directly the role of these psychosocial vulnerabilities in the association between hostility and subsequent health. The transactional model provides a useful framework for integrating research on psychophysiological reactivity and psychosocial vulnerability, but to date, it has been subjected only to preliminary tests. Although most of the evidence suggests that the health behavior model does not provide a complete account of the health effects of hostility, at least one well-controlled study was supportive (Everson et al., 1997). Further, other possible health behavior mediators (e.g., adherence to medical regimens, sleep) have yet to be investigated thoroughly. Finally, recent developments in the molecular genetics of personality, social behavior, and stress physiology have created unprecedented opportunities to pursue the constitutional vulnerability model. It is important to note that these models are not mutually exclusive or even necessarily conceptually distinct. It may be that hostility confers risk of CHD and premature mortality through more than one of the mechanisms outlined in these models.

Future research on each of these models may benefit from integration of the interpersonal perspective. Regarding psychophysiological mechanisms, we have discussed elsewhere the IPC as a conceptual framework for organizing research on the social psychophysiology of reactivity (Gallo et al., 2000; Smith, Gallo, et al. 2003). Transitory social stimuli and social behavior, as well as more stable social situations and individual differences in social behavior, can be located in this conceptual space. Hence, a variety of potential influences on reactivity can be integrated in this framework. As described above, the IPC can also clarify the specific concepts assessed by individual-difference measures, and what are often complex experimental manipulations of the social situation or social behavior can be verified fairly precisely with well-validated IPC measures. As in our marital studies (Smith & Gallo, 1999;Smith, Nealey-Moore, et al., 2003), such assessments can help elucidate the interpersonal determinants of physiological responses associated with anger, hostility,

and aggressiveness. For example, harassment routinely evokes heightened reactivity from hostile persons, but in the IPC framework this social stimulus (i.e., criticism) is a blend of hostility and dominance. It is not clear whether the threatened loss of status inherent in being "put down," the threatened loss of liking inherent in being "put off," or the combination of these interpersonal concerns produces this physiological response.

Given its conceptual accommodation of a wide variety of social processes and characteristics and the availability of well-validated measures, the IPC provides a valuable integrative approach for studying the psychosocial vulnerability model. For example, we have used circumplex-based measures to demonstrate that hostile persons both give and receive less social support (Corral et al., 2003; Smith, Uno, et al., 2000). The interpersonal perspective is obviously well suited to the study of the transactional model, and IPC measures could be used in daily experience sampling designs (cf., D'Antono, Ditto, Moskowitz, & Rios, 2001) to capture the social situations hypothesized to influence stress responses through the combined effects of greater exposure and reactivity. In examining the health behavior model, the interpersonal perspective could identify social processes that contribute to unhealthy lifestyles and unwise health decisions. Finally, the developmental and transactional elements of the interpersonal perspective could be useful in research on the social processes through which genetic vulnerabilities influence personality and disease.

General Conclusions, Additional Issues, and Future Directions

Over the past decade, there has been considerable progress in addressing the limitations identified in our prior review (Smith, 1992). Measures of these traits used in studies at that time have since been subjected to more thorough evaluation, and better measures have been developed, though additional work is still needed on this essential issue. Large, well-controlled prospective studies have replicated the association between these traits and CHD, though not all have produced positive result, and some have used insufficiently validated personality measures. Recent research suggests that anger, hostility, and aggressiveness may contribute to CHD by both promoting the development of CAD and the emergence of manifestations of CHD later in the course of disease. Importantly, recent

studies of the association between these traits and CHD have included more diverse samples, but here, too, more work is required. Finally, several plausible mechanisms linking these traits and CHD have been supported in additional research, though more definitive tests are still lacking.

Previously, we suggested that research in the area had reached a crossroads (Smith, 1992). A large body of supportive evidence encouraged continued research. However, limitations and inconsistencies in this literature could either be seen as the outlines of an agenda for future research or sufficient reason to curtail such efforts without resolution of the basic questions. Pessimistic views have appeared, but research over the past decade demonstrates how vigorously the field has continued to pursue the topic. Although many issues are far from resolved, the progress is compelling. In the present review, we have discussed the usefulness of the concepts and methods of the interpersonal approach in personality psychology in addressing the key remaining problems. We turn now to additional implications of this perspective.

Development of Risk

A comprehensive understanding of anger, hostility, aggressiveness, and health must include a developmental perspective. Early signs of CAD can be seen in childhood and adolescence. Further, indications of angry and socially difficult temperament in young children are associated with anger, hostility, and aggressiveness in adulthood (Caspi, 2000; Caspi, Harrington, Milne, Amell, & Moffitt, 2003), as well as exposure to the life experiences described in the psychosocial vulnerability and transactional models. The heritability of these traits is often found to be modest (e.g., Raynor, Pogue-Geile, Kamarck, McCaffery, and Manuck, 2002). As discussed above, even if stronger evidence of heritability emerges and specific genetic factors are identified, interpersonal processes are likely to be important in the development of anger, hostility, and aggressiveness (e.g., Luecken, 2000; Matthews, et al., 1996; Raikkonen, Katainen, Keskivaara, & Keltikangas-Jarvinen, 2000). In our brief review of the interpersonal perspective, we illustrated how it provides a valuable framework for exploring these aspects of social and emotional development (Gallo et al., 2003; McGonigle et al., 1993; Smith et al., 1998).

Prevention and Clinical Intervention

If CAD, these personality traits and related interpersonal process, begins in childhood, one important potential application of this research area involves early prevention. The well-developed literature on prevention of aggressive behavior and poor peer relations in childhood and adolescence (e.g., Blechman, 1996) could be adapted to an additional goal—improving physical health rather than emotional and social functioning alone. The interpersonal approach illustrates the interconnections among these outcomes and could guide the adaptation of existing preventive interventions to this new purpose. For persons with CHD, psychological interventions can reduce morbidity and mortality (Linden, Stossel & Maurice, 1996; Dusseldorp, van Elderen, Maes, Meulman, & Kraaij, 1999; Smith & Ruiz, 2002). However, anger, hostility, and aggressiveness have been a specific focus in only a few controlled trials (e.g., Gidron, Davison, & Bata, 1999). A variety of cognitive and behavioral interventions can reduce anger and aggressiveness (DiGiuseppe & Tafrate, 2003). The interpersonal perspective could provide an integrative framework for these diverse approaches, and specific interpersonal techniques could augment existing interventions.

Understanding Other Risk Factors

The interpersonal approach could also be useful in examining other psychosocial risk factors for CHD. For example, depressive symptoms and disorders confer increased risk of incident CHD and poor prognosis among CHD patients (Krantz & McCeny, 2002). Models of depression suggest that it is both a cause and a consequence of problematic social processes (Joiner & Coyne, 1999), and the interpersonal perspective we have described could easily provide an integrative and heuristic framework for examining its effect on health. Optimism exerts a protective effect on CHD (Smith & Ruiz, 2002). Although typically considered to be an intra-individual trait, optimism is associated with friendly dominance in the IPC, and consistent with the transactional cycle, it is also associated with greater social support and less exposure to interpersonal difficulties over time (Glazer et al., 2002b; Smith & Ruiz, 2000).

Whether indexed as a characteristic of individuals or the places they live, low socioeconomic status (SES) confers risk of CHD and

premature mortality (Gallo & Matthews, 2003). Low SES environments contribute to the development of many characteristics identified as psychosocial risk factors for serious disease, including hostility and aggressiveness (Leventhal & Brooks-Gunn, 2000). Further, there is some evidence that negative emotional and interpersonal characteristics like depression and hostility account for at least some of the effects of low SES on health (Gallo & Matthews, 2003; Williams, 2003). The interpersonal perspective might help to identify the reciprocal processes connecting people and their social contexts that mediate the effects of broad risk factors like SES on the health of individuals.

Hence, the interpersonal approach described here may be relevant to the study of many psychosocial influences on health and disease. Such applications of interpersonal theory begin with the notion that any given psychosocial risk or protective factor may reflect a "relatively enduring pattern of interpersonal situations which characterize a human life" (Sullivan, 1953, p. 111). They continue with the hypothesis that the effect of risk factors on health is most accurately conceptualized as involving an active reciprocal process rather than a static characteristic of the person or the social environment. From this starting point, the concepts and methods of the interpersonal approach can be applied to age-old questions about emotion, personality, and disease, and perhaps even improvement of physical health.

REFERENCES

Alden, L. E., Wiggins, J. S., & Pincus, A. L. (1990). Construction of circumplex scales for the Inventory of Interpersonal Problems. *Journal of Personality Assessment*, **55**, 521–536.

Angerer, P., Siebert, U., Kothny, W., Muhlbauer, D., Mudra, H., & von Schacky, C. (2000). Impact of social support, cynical hostility and anger expression on progression of coronary atherosclerosis. *Journal of the American College of Cardiology*, **36**, 1781–1788.

Barefoot, J. C., Dodge, K. A., Peterson, B. L., & Dalhstrom, G. W. (1989). The Cook-Medley Hostility scale: Item content and ability to predict survival. *Psychosomatic Medicine*, **51**, 46–57.

Barefoot, J. C., & Lipkus, I. M. (1994). The assessment of anger and hostility. In A. W. Siegman & T. W. Smith (Eds). *Anger, hostility, and the heart*. (pp. 43–66). Hillsdale, NJ: Lawrence Erlbaum Associates, Inc.

Becker, A. E., de Boer, O. J., & van der Wal, A. C. (2001). The role of inflammation and infection in coronary artery disease. *Annual Review of Medicine*, **52**, 289–297.

Benjamin, L. S. (1974). Structural Analysis of Social behavior (SASB). *Psychological Review*, **81**, 1129–1133.

Benjamin, L. S. (1994). SASB: A bridge between personality theory and clinical psychology. *Psychological Inquiry*, **5**, 273–316.

Benjamin, L. S. (2003). *Interpersonal reconstructive therapy (IRT): Promoting change in nonresponders*. New York: Guilford Press.

Benotsch, E. G., Christensen, A. J., & McKelvey, L. (1997). Hostility, social support and ambulatory cardiovascular activity. *Journal of Behavioral Medicine*, **20**, 163–176.

Blechman, E. A. (1996). Coping, competence, and aggression prevention: Part 2. Universal school-based prevention. *Applied and Preventive Psychology*, **5**, 19–36.

Bowlby, J. (1973). *Separation: Anxiety and anger. Vol 2. Of attachment and loss*. London: Hogarth Press.

Bowlby, J. (1979). *The making and breaking of affectional bonds*. London: Tavistock.

Bolger, N., & Schilling, E. A. (1991). Personality and the problems of everyday life: The role of neuroticism in exposure and reactivity to daily stressors. *Journal of Personality*, **59**, 355–386.

Bolger, N., & Zuckerman, A. (1995). A framework for studying personality in the stress process. *Journal of Personality and Social Psychology*, **69**, 890–902.

Brissette, I., & Cohen, S. (2002). The contribution of individual differences in hostility to the associations between daily interpersonal conflict, affect, and sleep. *Personality and Social Psychology Bulletin*, **28**, 1265–1274.

Brondolo, E., Rieppi, R., Erickson, S. A., Bagiella, E., Shapiro, P. A., & McKinley, P., et al. (2003). Hostility, interpersonal interactions, and ambulatory blood pressure. *Psychosomatic Medicine*, **65**, 1003–1011.

Brosschot, J. F., & Thayer, J. F. (1998). Anger inhibition, cardiovascular recovery, and vagal function: A model of the link between hostility and cardiovascular disease. *Annals of Behavioral Medicine*, **20**, 326–332.

Brown, S. L., Nesse, R. M., Vinokur, A. D., & Smith, D. M. (2003). Providing social support may be more beneficial than receiving it: Results from a prospective study of mortality. *Psychological Science*, **14** (4), 320–327.

Brummett, B. H., Babyak, M. A., Mark, D. C., Williams, R. B., Siegler, I. C., & Clapp-Channing, N., et al. (2002). Predictors of smoking cessation in patients with a diagnosis of coronary artery disease. *Journal of Cardiopulmonary Rehabilitation*, **22** 3, 143–147.

Brummett, B. H., Maynard, K. E., Babyak, M. A., Haney, T. L., Siegler, I. C., Helms, M. J., & Barefoot, J. C. (1998). Measures of hostility as predictors of facial affect during social interaction: Evidence for construct validity. *Annals of Behavioral Medicine*, **20**, 168–173.

Brummett, B. H., Maynard, K. E., Haney, T. L., Siegler, I. C., & Barefoot, J. C. (2000). Reliability of interview-assessed hostility ratings across mode of assessment and time. *Journal of Personality Assessment*, **75** (2), 225–236.

Burg, M. M., Jain, D., Soufer, R., Kerns, R. D., & Zaret, B. L. (1993). Role of behavioral and psychological factors in mental stress-induced silent left ventricular dysfunction incoronary artery disease. *Journal of the American College of Cardiology*, **22**, 440–448.

Buss, A. H., & Durkee, A. (1957). An inventory for assessing different kinds of hostility. *Journal of Consulting Psychology*, **21**, 343–349.

Buss, A. H., & Perry, M. (1992). The aggression questionnaire. *Journal of Personality and Social Psychology*, **63**, 452–459.

Bushman, B. J., Cooper, H. M., & Lemke, H. M. (1991). Meta-analysis of factor analyses: An illustration using the Buss-Durkee Hostility Inventory. *Personality and Social Psychology Bulletin*, **17**, 344–349.

Cantor, N. (1990). From thought to behavior: "Having" and "doing" in the study of personality and cognition. *American Psychologist*, **45**, 735–750.

Carson, R. C. (1969). *Interaction concepts of personality*. Oxford: Aldine.

Caspi, A. (2000). The child is father of the man: Personality continuities from childhood to adulthood. *Journal of Personality and Social Psychology*, **78**, 158–172.

Caspi, A., Harrington, H., Milne, B., Amell, J. W., & Moffitt, T. E. (2003). Children's behavioral styles at age 3 are linked to their adult personality traits at age 26. *Journal of Personality*, **71**, 495–513.

Caspi, A., & Roberts, B. W. (1999). Personality continuity and change across the life course. In L. A. Pervin & O. P. John (Eds.), *Handbook of personality: theory and research* (2nd ed.). (pp. 300–326). New York: Guilford.

Cassidy, J., & Shaver, P. R. (1999). *Handbook of attachment: Theory, research, and clinical applications*. New York: Guilford Press.

Chang, P. P., Ford, D. E., Meoni, L. A., Wang, N. Y., & Klag, M. J. (2002). Anger in young men and subsequent premature cardiovascular disease: the precursors study. *Archives of Internal Medicine*, **162**, 901–906.

Chaput, L. A., Adams, S. H., Simon, J. A., Blumenthal, R. S., Vittinghoff, E., & Lin, F., et al. (2002). Hostility predicts recurrent events among postmenopausal women with coronary heart disease. *American Journal of Epidemiology*, **156**, 1092–1099.

Christensen, A. J., & Smith, T. W. (1993). Cynical hostility and cardiovascular reactivity during self-disclosure. *Psychosomatic Medicine*, **55**, 193–202.

Christensen, A. J., Wiebe, J. S., & Lawton, W. J. (1997). Cynical hostility, powerful others Control expectancies, and patient adherence in hemodialysis. *Psychosomatic Medicine*, **59**, 307–312.

Cohen, S., Mermelstein, R. J., Kamarck, T., & Hoberman, H. M. (1985). Measuring thefunctional components of social support. In I. G. Sarason & B. Sarason (Eds.), *Social support: Theory, research, and applications* (pp. 73–94). The Hague: Martines Niijohff.

Cook, W. W., & Medley, D. M. (1954). Proposed hostility and pharisaic virtue scales for the MMPI. *Journal of Applied Psychology*, **38**, 414–418.

Contrada, R. J., & Jussim, L. (1992). What does the Cook-Medley hostility scale measure? In search of an adequate measurement model. *Journal of Applied Social Psychology*, **22**, 615–627.

Corral, I., Smith, T. W., Glazer, K. M., & Allen, N. (2003, March). *Construct validation of MMPI-2 anger and dominance scales: Interpersonal and five-factor analyses*. Poster session presented at the annual meeting of the Society of Behavior Medicine, Salt Lake City.

Costa, P. T. Jr., & McCrae, R. R. (1987). Neuroticism, somatic complaints and disease: Is the Bark worse than the bite? *Journal of Personality*, **55**, 299–316.

Costa, P. T. Jr., McCrae, R. R., & Dembroski, T. M. (1989). Agreeableness versus antagonism: Explication of a potential risk factor for CHD. In A. Siegman & T. M. Dembroski (Eds.) *In search of coronary-prone behavior: Beyond Type A* (pp. 41–63). Hillsdale, NJ: Lawrence Erlbaum Associates.

Costa, P. T., Zonderman, A. B., McCrae, R. R., & Williams, R. B. (1986). Cynicism and paranoid alienation in the Cook and Medley HO Scale. *Psychosomatic Medicine*, **48**, 283–285.

D'Antonio, B., Ditto, B., Moskowitz, D. S., & Rioz, N. (2001). Interpersonal behavior and resting blood pressure in college women: a daily monitoring study. *Journal of Psychosomatic Research*, **50**, 309–318.

Davis, M. C., Matthews, K. A., & McGrath, C. E. (2000). Hostile attitudes predict elevated vascular resistance during interpersonal stress in men and women. *Psychosomatic Medicine*, **62** (1), 17–25.

Dembroski, T. M., MacDougall, J. M., Costa, P. T. Jr., & Grandits, G. A. (1989). Components of hostility as predictors of sudden death and myocardial infarction in the Multiple Risk Factor Intervention Trial.. *Psychosomatic Medicine*, **51**, 514–522.

Dew, M. A., Hoch, C. C., Buysse, D. J., Monk, T. H., Begley, A. E., & Houck, P. R., et al. (2003). Healthy older adults' sleep predicts all-cause mortality at 4 to 19 years of follow-up. *Psychosomatic Medicine*, **65**, 63–73.

DiGuiseppe, R., & Tafrate, R. C. (2003). Anger treatment for adults: A meta-analytic review. *Clinical Psychology, Science and Practice*, **10**, 70–84.

Dusseldorp, E., van Elderen, T., Maes, S., Meulman, J., & Kraaij, V. (1999). A meta-analysis of Psychoeducational programs for coronary heart disease patients. *Health Psychology*, **18**, 506–519.

Eng, P. M., Fitzmaurice, G., Kubzansky, L. D., Rimm, E. B., & Kawachi, I. (2003). Anger expression and risk of stroke and coronary heart disease among male health professionals. *Psychosomatic Medicine*, **65**, 100–110.

Engebretson, T. O., Matthews, K. A., & Scheier, M. F. (1989). Relations between anger expression and cardiovascular reactivity: Reconciling inconsistent findings through a matching hypothesis. *Journal of Personality & Social Psychology*, **57**, 513–521.

Everson, S. A., Kaplan, G. A., Goldberg, D. E., Lakka, T. A., Sivenius, J., & Salonen, J. T. (1999). Anger expression and incident stroke: prospective evidence from the Kuopio ischemic heart disease study. *Stroke*, **30**, 523–528.

Everson, S. A., Kauhanen, J., Kaplan, G., Goldberg, D., Julkunen, J., & Tuomilehto, J., et al. (1997). Hostility and increased risk of mortality and myocardial infarction: The mediating role of behavioral risk factors. *American Journal of Epidemiology*, **146**, 142–152.

Fang, C. Y., & Myers, H. F. (2001). The effects of racial stressors and hostility on cardiovascular reactivity in African American and Caucasian men. *Health Psychology*, **20**, 64–70.

Finney, M. L., Stoney, C. M., & Engebretson, T. O. (2002). Hostility and anger expression in African American and European American men is associated with cardiovascular and lipid reactivity. *Psychophysiology*, **39**, 340–349.

Florsheim, P., Henry, W. P., & Benjamin, L. S. (1996). Integrating individual and interpersonal approaches to diagnosis: The structural analysis of social behavior

and attachment theory. In N. Kaslow & W. Florence. (Ed). *Handbook of relational diagnosis and dysfunctional family patterns* (pp. 81–101). Oxford: John Wiley & Sons.

Fredrickson, B. L., Maynard, K. E., Helms, M. J., Haney, T. L., Siegler, I. C., & Barefoot, J. C. (2000). Hostility predicts magnitude and duration of blood pressure response to anger. *Journal of Behavioral Medicine*, **23**, 229–243.

Friedman, H. S. (2000). Long-term relations of personality and health: Dynamisms, mechanisms, tropisms. *Journal of Personality*, **68**, 1089–1107.

Gabbay, F. H., Krantz, D. S., Kop, W., Hedges, S., Klein, J., & Gottdiener, J., et al. (1996). Triggers of myocardial ischemia during daily life in patients with coronary artery disease: Physical and mental activities, anger, and smoking. *Journal of the American College of Cardiology*, **27**, 585–592.

Gallacher, J., Tarnell, J., Sweetnam, , Elwood, P., & Stanfeld, S. (1999). Anger and incident heart disease in the Caerphilly Study. *Psychosomatic Medicine*, **61**, 446–454.

Gallo, L. C., & Matthews, K. A. (2003). Understanding the association between socioeconomic status and physical health: Do negative emotions play a role? *Psychological Bulletin*, **129**, 10–51.

Gallo, L. C., & Smith, T. W. (1998). Construct validation of health-relevant personality traits: Interpersonal circumplex and five-factor model analyses of the Aggression Questionnaire. *International Journal of Behavioral Medicine*, **5**, 129–147.

Gallo, L. C., & Smith, T. W. (1999). Patterns of hostility and social support: Conceptualizing psychosocial risk factors as a characteristic of the person and the environment. *Journal of Research in Personality*, **33**, 281–310.

Gallo, L. C., Smith, T. W., & Kircher, J. C. (2000). Cardiovascular and electrodermal responses to support and provocation: Interpersonal methods in the study of psychophysiological reactivity. *Psychophysiology*, **37**, 289–301.

Gallo, L. C., Smith, T. W., & Ruiz, J. M. (2003). An interpersonal analysis of adult attachment style: Circumplex descriptions, recalled developmental experiences, self-representations, and interpersonal functioning in adulthood. *Journal of Personality*, **71**, 141–181.

Gidron, Y., Davidson, K., & Bata, I. (1999). The short-term effects of a hostility-reduction intervention on male coronary heart disease patients. *Health Psychology*, **18**, 416–420.

Gifford, R. (1991). Mapping nonverbal behavior on the interpersonal circle. *Journal of Personality and Social Psychology*, **61**, 279–288.

Glazer, K., Smith, T. W., Nealey, J., & Hawkins, M. (2002a). *Hostility and marital adjustment*. Paper presented at the annual meeting of the Society of Behavioral Medicine.

Glazer, K., Smith, T. W., Nealey, J., & Hawkins, M. (2002b). *Dispositional optimism and relationship quality in married couples*. Paper presented at the International Conference on Personal Relationships, Halifax, Nova Scotia.

Goodman, M., Quigley, J., Moran, G., Meilman, H., & Sherman, M. (1996). Hostility predicts resentosis after percutaneous transluminal coronary angioplasty. *Mayo Clinic Proceedings*, **71**, 729–734.

Gottdiener, J. S., Kop, W. J., Hauser, E., McCeney, M. K., Herrington, D., & Krantz, D. S. (2003). Effects of mental stress on flow-mediated brachial arterial

dilation and influence of behavioral factors and hypercholesterolemia in subjects without cardiovascular disease. *American Journal of Cardiology*, **15**, 687–691.

Gurtman, M. B. (1992). Construct validity of interpersonal personality measures: The interpersonal circumplex as a nomological net. *Journal Personality and Social Psychology*, **63**, 105–118.

Gurtman, M. B. (2001). Interpersonal complimentarity: Integrating interpersonal measurement with interpersonal models. *Journal of Counseling Psychology*, **48**, 97–110.

Gurtman, M. B., & Pincus, A. L. (2003). The circumplex model: Methods and research applications. In J. A. Schinka & W. , F. Velicer (Eds). *Handbook of psychology: Research methods in psychology* , Vol. 2. (pp. 407–428). New York: John Wiley & Sons, Inc.

Guyll, M., & Contrada, R. (1998). Trait hostility and ambulatory cardiovascular activity: Responses to social interaction. *Health Psychology*, **17**, 30–39.

Haney, T. L., Maynard, K. E., Houseworth, S. J., Scherwitz, L. W., Williams, R. B., & Barefoot, J. C. (1996). Interpersonal Hostility Assessment Technique: Description and validation against the criterion of coronary artery disease. *Journal of Personality Assessment*, **66**, 386–401.

Hart, K. E. (1999). Cynical hostility and deficiencies in functional support: The moderating role of gender in psychosocial vulnerability to disease. *Personality and Individual Differences*, **27**, 69–83.

Harris, J. A. (1995). Confirmatory factor analysis of the aggression questionnaire. *Behaviour Research and Therapy*, **33**, 991–993.

Harris, K. F., Matthews, K. A., Sutton-Tyrrell, K., & Kuller, L. H. (2003). Associations between psychological traits and endothelial function in post-menopausal women. *Psychosomatic Medicine*, **65**, 402–409.

Hazan, C., & Shaver, P. (1987). Romantic love conceptualized as an attachment process. *Journal of Personality and Social Psychology*, **52**, 511–524.

Hazan, C., & Shaver, P. R. (1994). Attachment as an organizational framework for research on close relationships. *Psychological Inquiry*, **5**, 1–22.

Helgeson, V. S. (1994). Relation of agency and communion to well-being: Evidence and potential explanations. *Psychological Bulletin*, **116**, 412–428.

Helmers, K. F., Krantz, D. S., Howell, R., Klein, J., Bairey, N., & Rozanski, A. (1993). Hostility and myocardial ischemia in coronary artery disease patients: Evaluation by gender and ischemic index. *Psychosomatic Medicine*, **50**, 29–36.

Hemingway, H., & Marmot, M. (1999). Psychosocial factors in the aetiology and prognosis of coronary heart disease: Systematic review of prospective cohort studies. *British Medical Journal*, **318**, 1460–1467.

Hofstee, W. K., deRaad, B., & Goldberg, L. R. (1992). Integration of the Big Five and circumplex approaches to trait structure. *Journal of Personality and Social Psychology*, **63** (1), 146–163.

Horney, K. (1950). *Neurosis and human growth: The struggle toward self-realization*. Oxford: W. W. Norton.

Houston, B. K. (1994). Anger, hostility, and psychophysiological reactivity. In A. W. Siegman & T. W. Smith (Eds.), *Anger, hostility, and the heart* (pp. 97–115). Hillsdale, NJ: Lawrence Erlbaum.

Houston, B. K., Babyak, M. A., Chesney, M. A., Black, G., & Ragland, D. R. (1997). Social dominance and 22-year all-cause mortality in men. *Psychosomatic Medicine*, **59**, 5–12.

Houston, B. K., Chesney, M. A., Black, G. W., Cates, D. S., & Hecker, M. L. (1992). Behavioral clusters and coronary heart disease risk. *Psychosomatic Medicine*, **54**, 447–461.

Iribarren, C., Sidney, S., Bild, D. E., Liu, K., Markovitz, J. H., & Roseman, J. M. et al, (2000). Association of hostility with coronary artery calcification in young adults: The CARDIA study. *Journal of the American Medical Association*, **283**, 2546–2551.

Ironson, G., Taylor, C. B., Boltwood, M., Bartzokis, T., Dennis, C., & Chesney, M, et al. (1992). Effects of anger on left ventricular ejection fraction in coronary disease. *American Journal of Cardiology*, **70**, 281–285.

Joiner, T., & Coyne, J. C. (1999). *The interactional nature of depression: Advances in interpersonal approaches*. Washington, DC: American Psychological Association.

Julkunen, J., Salonen, R., Kaplan, G. A., Chesney, M. A., & Salonen, J. T. (1994). Hostility and the progression of carotid atherosclerosis. *Psychosomatic Medicine*, **56**, 519–525.

Kamarck, T. W., Peterman, A. H., & Raynor, D. A. (1998). The effects of the social environment on stress-related cardiovascular activation: Current findings, prospects, and implications. *Annals of Behavioral Medicine*, **20**, 247–225.

Kaplan, J. R., & Manuck, S. B. (1998). Monkeys, aggression, and the pathobiology of atherosclerosis. *Aggressive Behavior*, **24**, 323–334.

Kaufmann, M. W., Fitzgibbons, J. P., Sussman, E. J., Reed, J. F. III, Einfalt, J. M., & Rodgers, J. K., et al. (1999). Relation between myocardial infarction, depression, hostility, and death. *American Heart Journal*, **138**, 549–554.

Kawachi, I., Sparrow, D., Spiro, A., Vokonas, P., & Weiss, S. T. (1996). A prospective study of anger and coronary heart disease: The Normative Aging Study. *Circulation*, **94**, 2090–2095.

Kiecolt-Glaser, J., McGuire, L., Robles, T. F., & Glaser, R. (2002). Psychoneuroimmunology: Psychological influences on immune function and health. *Journal of Consulting and Clinical Psychology*, **70**, 537–547.

Kiecolt-Glaser, J., & Newton, T. (2001). Marriage and health: His and hers. *Psychological Bulletin*, **127**, 472–503.

Kiesler, D. J. (1983). The 1982 Interpersonal Circle: A taxonomy for complementarity in human transactions. *Psychological Review*, **90**, 185–214.

Kiesler, D. J. (1996). *Contemporary interpersonal theory and research: Personality, psychopathology, and psychotherapy*. New York: John Wiley & Sons.

Knox, S. S., Adelman, A., Ellison, R. C., Arnett, D. K., Siegmund, K., & Weidner, G. (2000). Hostility, social support, and carotid artery atherosclerosis in the National Heart, Lung, and Blood Institute Family Heart Study. *American Journal of Cardiology*, **86**, 1086–1089.

Knox, S. S., Siegmund, K. D., Weidner, G., Ellison, R. C., Adelman, A., & Paton, C. (1998). Hostility, social support, and coronary heart disease in the National Heart, Lung, and Blood Institute Family Heart Study. *American Journal of Cardiology*, **82**, 1192–1196.

Kop, W. J. (1999). Chronic and acute psychological risk factors for clinical manifestations of coronary artery disease. *Psychosomatic Medicine*, **61**, 476–487.

Kop, W. J. (2003). The integration of cardiovascular behavioral medicine and psychoneuroimmunology: new developments based on converging research fields. *Brain Behavior and Immunology*, **17**, 233–237.

Krantz, D. S., & McCeney, M. K. (2002). : Effects of psychological and social factors on organic disease: A critical assessment of research on coronary heart disease. *Annual Review of Psychology*, **53**, 341–369.

Kurylo, M., & Gallant, S. (2000). Hostility and cardiovascular reactivity in women during self-disclosure. *International Journal of Behavioral Medicine*, **7**, 271–285.

Lahad, A., Heckbert, S. R., Koepsell, T. D., Psaty, B. M., & Patrick, D. L. (1997). Hostility, aggression and the risk of nonfatal myocardial infarction in postmenopausal women. *Journal of Psychosomatic Research*, **43**, 183–195.

Lazarus, R. S. (1991). *Emotion and adaptation*. London: Oxford University Press.

Leary, T. (1957). *Interpersonal diagnosis of personality*. New York: Ronald.

Lepore, S. J. (1995). Cynicism, social support, and cardiovascular reactivity. *Health Psychology*, **14**, 210–216.

Lepore, S. J. (1998). Problems and prospects for the social support-reactivity hypothesis. *Annals of Behavioral Medicine*, **20**, 257–269.

Leventhal, T., & Brooks-Gunn, J. (2000). The neighborhoods they live in: The effects of neighborhood residence on child and adolescent outcomes. *Psychological Bulletin*, **126**, 309–337.

Libby, P. (2003). Vascular biology of atherosclerosis: overview and state of the art. *American Journal of Cardiology*, **91** (3A), 3A–6A.

Linden, W., Hogan, B. E., Rutledge, T., Chawla, A., Lenz, J. W., & Leung, D. (2003). There is more to anger coping than ''in'' or ''out''. *Emotion*, **3**, 12–29.

Linden, W., Stossel, C., & Maurice, J. (1996). Psychosocial interventions for patients with coronary artery disease: A meta-analysis. *Archives of Internal Medicine*, **156**, 745–752.

Llabre, M. M., Spitzer, S., Siegel, S., Saab, P. G., & Schneiderman, N. (2004). Applying latent growth curve modeling to the investigation of individual differences in cardiovascular recovery from stress. *Psychosomatic Medicine*, **66**, 29–41.

Low, K. G., Gleisher, C., Colman, R., Dionne, A., Casey, G., & Legendre, S. (1998). Psychosocial variables, age, and angiographically-determined coronary artery disease in women. *Annals of Behavioral Medicine*, **20**, 221–226.

Luecken, L. J. (2000). Attachment and loss experiences during childhood are associated with adult hostility, depression, and social support. *Journal of Psychosomatic Research*, **49**, 85–91.

Markey, P. M., Funder, D. C., & Ozer, D. J. (2003). Complementarity of interpersonal behaviors in dyadic interactions. *Personality and Social Psychology Bulletin*, **29**, 1082–1090.

Manuck, S. B., Flory, J. D., Ferrell, R. E., Dent, K. M., Mann, J. J., & Muldoon, M. F. (1999). Aggression and anger-related traits associated with a polymorphism of the Prytophan Hydroxylase Gene. *Biological Psychiatry*, **45**, 603–614.

Manuck, S. B., Flory, J. D., Muldoon, M. F., & Ferrell, R. E. (2002). Central nervous system serotonergic responsivity and aggressive disposition in men. *Physiology and Behavior*, **77**, 705–709.

Manuck, S. B., Marsland, A. L., Kaplan, J. R., & Williams, J. K. (1995). The pathogenicity of behavior and its neuroendocrine mediation: An example from coronary artery disease. *Psychosomatic Medicine*, **57**, 275–283.

Markovitz, J. H. (1998). Hostility is associated with increased platelet activation in coronary heart disease. *Psychosomatic Medicine*, **60** (5), 586–591.

Marshall, G. N., Wortman, C. B., Vickers, R. R., Kusulas, J. W., & Hurvig, L. K. (1994). The five-factor model of personality as a framework for personality-health research. *Journal of Personality and Social Psychology*, **67**, 278–286.

Matthews, K. A., & Gump, B. B. (2002). Chronic work stress and marital dissolution increase risk of posttrial mortality in men from the Multiple Risk Factor Intervention Trial. *Archives of Internal Medicine*, **162**, 309–315.

Matthews, K. A., Gump, B. B., Harris, K. F., Haney, T. L., & Barefoot, J. C. (2004). Hostile behaviors predict cardiovascular mortality among men enrolled in the Multiple Risk Factor Intervention Trial. *Circulation*, **109**, 66–70.

Matthews, K. A., Owens, J. F., Kuller, L. H., Sutton-Tyrrell, K., & Jansen-McWilliams, L. (1998). Are hostility and anxiety associated with carotid atherosclerosis in healthy postmenopausal women? *Psychosomatic Medicine*, **60**, 633–638.

Matthews, K. A., Woodall, K. L., Kenyon, K., & Jacob, T. (1996). Negative family environment as a predictor of boys' future status on measure of hostile attitudes, interview behavior, and anger expression. *Health Psychology*, **15**, 30–37.

McAdams, D. P. (1995). What do we know when we know a person? *Journal of Personality*, **63**, 365–396.

McCann, B. S., Russo, J., & Benjamin, G. A. (1997). Hostility, social support, and perceptions of work. *Journal of Occupational Health Psychology*, **2**, 175–185.

McCrae, R. T., & Costa, P. T. (1989). The structure of interpersonal traits: Wiggins's circumplex and the five-factor model. *Journal of Personality and Social Psychology*., **56**, 586–595.

McGonigle, M. M., Smith, T. W., Benjamin, L. S., & Turner, C. W. (1993). Hostility and non-shared family environment. *Journal of Research and Personality*, **27**, 23–34.

Meaney, M. J. (2001). Maternal care, gene expression, and the transmission of individual differences in stress reactivity across generations. *Annual Review of Neuroscience*, **24**, 1161–1192.

Meesters, C. M., Muris, P., & Backus, I. P. (1996). Dimensions of hostility and myocardial infarction in adult males. *Journal of Psychosomatic Research*, **40**, 21–28.

Mendes De Leon, C. F., Kop, W. J., de Swart, H. B., Bar, F. W., & Appels, A. P. (1996). Psychosocial characteristics and recurrent events after percutaneous transluminal coronary angioplasty. *American Journal of Cardiology*, **77**, 252–255.

Miller, G., Dopp, J. M., Myers, H. F., Stevens, S. Y., & Fahey, J. L. (1999). Psychosocial predictors of natural killer cell mobilization during marital conflict. *Health Psychology*, **18**, 262–271.

Miller, G. E., Freedland, K. E., Carney, R. M., Stetler, C. A., & Banks, W. A. (2003). Cynical hostility, depressive symptoms, and the expression of inflammatory risk markers for coronary heart disease. *Journal of Behavioral Medicine*, **26**, 501–515.

Miller, S. B., Dolgoy, L., Friese, M., Sita, A., Lavoie, K., & Campbell, T. (1998). Hostility, sodium consumption, and cardiovascular response to interpersonal stress. *Psychosomatic Medicine*, **60**, 71–77.

Miller, T. Q., Marksides, K. S., Chiriboga, D. A., & Ray, L. A. (1995). A test of the psychosocial vulnerability and health behavior models of hostility: Results from an 11-year follow-up study of Mexican Americans. *Psychosomatic Medicine*, **57**, 572–581.

Miller, T. Q., Smith, T. W., Turner, C. W., Guijarro, M. L., & Hallet, A. J. (1996). A meta-analytic review of research on hostility and physical health. *Psychological Bulletin*, **119**, 322–348.

Mischel, W., & Shoda, Y. (1995). A cognitive-affective system theory of personality: Reconceptualizing situations, dispositions, dynamics, and invariance in personality structure. *Psychological Review*, **102**, 246–268.

Mischel, W., & Shoda, Y. (1998). Reconciling processing dynamics and personality dispositions. *Annual Review of Psychology*, **49**, 229–258.

Mischel, W., & Shoda, Y. (1999). Integrating dispositions and processing dynamics within a unified theory of personality: The cognitive-affective personality system. In L. A. Pervin & O. P. John (Eds.) *Handbook of personality: Theory and research* 2nd ed., (pp. 197–218). New York: Guilford Press.

Mittleman, M. A., Maclure, M., Sherwood, J. B., Mulry, R. P., Tofler, G. H., & Jacobs, S. C., Et al for the Determinants of Myocardial Infarction Onset Study Investigators. (1995). Triggering of acute myocardial infarction onset by episodes of anger. *Circulation*, **92**, 1720–1725.

Möller, J., Hallqvist, J., Diderichsen, F., Theorell, T., Reuterwall, C., & Ahlbom, A. (1999). Do episodes of anger trigger myocardial infarction? A case-crossover analysis in the Stockholm Heart Epidemiology Program (SHEEP). *Psychosomatic Medicine*, **61**, 842–849.

Musante, L., MacDougall, J. M., Dembroski, T. M., & Costa, P. T. (1989). Potential hostility and dimensions of anger. *Health Psychology*, **8**, 343–354.

Myrtek, M. (2001). Meta-analyses of prospective studies on coronary heart disease, type A personality, and hostility. *International Journal of Cardiology*, **79**, 245–251.

Newton, T. L., & Kiecolt-Glaser, J. K. (1995). Hostility and erosion of marital quality during early marriage. *Journal of Behavioral Medicine*, **18**, 601–619.

Niaura, R., Banks, S. M., Ward, K. D., Stoney, K. M., Spiro, A. 3rd Aldwin, C. M., Et al. (2000). Hostility and the metabolic syndrome in older males: The normative aging study. *Psychosomatic Medicine*, **62**, 7–16.

Niaura, R., Todaro, J. F., Stroud, L., Spiro, A. III, Ward, K. D., & Weiss, S. (2002). Hostility, the metabolic syndrome, and incident of coronary heart disease. *Health Psychology*, **21**, 588–593.

O'Connor, T. G., Deater-Deckard, K., Fulker, D., Rutter, M., & Plomin, R. (1998). Genotype-environment correlations in late childhood and early adolescence: antisocial behavior problems and coercive parenting. *Developmental Psychology*, **34**, 970–981.

O'Malley, P. G., Jones, D. L., Feuerstein, I. M., & Taylor, A. J. (2000). Lack of correlation between psychological factors and subclinical coronary artery disease. *New England Journal of Medicine*, **343**, 1298–1304.

O'Neil, J. N., & Emery, C. F. (2002). Psychosocial vulnerability, hostility, and family history of coronary heart disease among male and female college students. *International Journal of Behavioral Medicine*, **9**, 17–36.

Orford, J. (1986). The rules of interpersonal complementarity: Does hostility beget hostility and dominance, submission? *Psychological Review*, **93**, 365–377.

Orth-Gomer, K., Wamala, S. P., Horsten, M., Schenck-Gustafsson, K., Schneiderman, N., & Mittleman, M. A. (2000). Marital stress worsens prognosis in women with coronary heart disease: The Stockholm Female Coronary Risk Study. *Journal of the American Medical Association*, **284**, 3008–3014.

Petticrew, M., Gilbody, S., & Sheldon, T. A. (1999). Relation between hostility and coronary heart disease. Evidence does not support link. *BMJ*, **319**, 917–918.

Piferi, R. L., & Lawler, K. A. (2000). Hostility and the cardiovascular reactivity of women during interpersonal confrontation. *Women and Health*, **30**, 111–129.

Pincus, A. L., & Ansell, E. B. (2003). Interpersonal theory of personality. In T. Millon & M. J. Lerner, *Handbook of psychology Personality and social psychology* Vol. 5 (pp. 209–229). New York: John Wiley & Sons, Inc.

Pincus, A. L., Dickinson, K. A., Schut, A. J., Castonguay, L. G., & Bedics, J. (1999). Integrating interpersonal assessment and adult attachment using SASB. *European Journal of Psychological Assessment*, **15**, 206–220.

Polk, D. E., Kamarck, T. W., & Shiffman, S. (2002). Hostility explains some of the discrepancy between daytime ambulatory and clinic blood pressures. *Health Psychology*, **21**, 202–206.

Prkrachin, K. M., & Silverman, B. E. (2002). Hostility and facial expression in young men and women: Is social regulation more important than negative affect? *Health Psychology*, **21**, 33–39.

Raikkoenn, K., Katainin, S., Keshivaara, P., & Keltikangas-Jarvinen, L. (2000). Temperament, mothering, and hostile attitudes: A 12-year longitudinal study. *Personality and Social Psychology Bulletin*, **26**, 3–12.

Raikkonen, K., Matthews, K. A., Flory, J. D., & Owens, J. F. (1999). Effects of hostility on Ambulatory blood pressure and mood during daily living in healthy adults. *Health Psychology*, **18**, 44–53.

Raikkonen, K., Matthews, K. A., & Kuller, L. J. (2002). The relationship between psychological risk attributes and the metabolic syndrome in healthy women: antecedent or consequence? *Metabolism*, **51**, 1573–1577.

Raikkonen, K., Matthews, K. A., Kuller, L. J., Reiber, C., & Bunker, C. H. (1999). Anger, hostility, and visceral adipose tissue in healthy postmenopausal women. *Metabolism*, **48**, 1146–1151.

Raynor, D. A., Pogue-Geile, M. F., Kamarck, T. W., McCaffery, J. M., & Manuck, S. B. (2002). Covariation of psychosocial characteristics associated with cardiovascular disease: Genetic and environmental influences. *Psychosomatic Medicine*, **64**, 191–203.

Rosenberg, E. L., Ekman, P., Jian, W., Babyak, M., Coleman, R. E., & Hanson, M., et al. (2001). Linkages between facial expressions of anger and

transient myocardial ischemia in men with coronary artery disease. *Emotion*, **1**, 107–115.

Rosenman, R. H. (1978). The interview method of assessment of the coronary-prone behaviors in the Western Collaborative Group Study. In T. M. Dembroski, S. M. Weiss, J. L. Shields, & M. Feinleib (Eds.) *Coronary prone behavior* (pp. 55–69). New York: Springer-Verlag.

Ross, R. (1999). Atherosclerosis: An inflammatory disease. *New England Journal of Medicine*, **340**, 115–126.

Rozanski, A., Blumenthal, J. A., & Kaplan, J. (1999). Impact of psychological factors on the pathogenesis of cardiovascular disease and implications for therapy. *Circulation*, **99**, 2192–2217.

Ruiz, J. M., Smith, T. W., & Uchino, B. N. (2002, April). *Anger Expression in social contexts*. Paper Presented at the annual meeting of the Society of Behavioral Medicine, Washington, DC.

Rutledge, T., & Hogan, B. E. (2002). A quantitative review of prospective evidence linking psychological factors with hypertension development. *Psychosomatic Medicine*, **64**, 758–766.

Rutter, M., & Silberg, J. (2002). Gene-environment interplay in relation to emotional and behavioral disturbance. *Annual Review of Psychology*, **53**, 463–490.

Schmidt, J. A., Wagner, C. C., & Kiesler, D. J. (1999). Psychometric and circumplex properties of the octant scale Impact Message Inventory (IMI-C): A structural evaluation. *Journal of Counseling Psychology*, **46**, 325–334.

Siegler, I. C. (1994). Hostility and risk: Demographic and lifestyle variables. In A. W. Siegman & T. W. Smith (Eds.), *Anger, hostility, and the heart* (pp. 199–214). Hillsdale, NJ: Lawrence Erlbaum.

Siegler, I. C., Costa, P. T., Brummett, B. H., Helms, M. J., Barefoot, J. C., & Williams, R. B., et al. (2003). Patterns of change in hostility from college to midlife in the UNC Alumni Heart Study predict high-risk status. *Psychosomatic Medicine*, **65**, 738–745.

Siegman, A. W. (1994). From Type A to hostility to anger: Reflections on the history of coronary –prone behavior. In A. W. Siegman & T. W. Smith (Eds.), *Anger, hostility, and the heart* (pp. 1–21). Hillsdale, NJ: Lawrence Erlbaum.

Siegman, A. W., Kubzansky, L. D., Kawachi, I., Boyle, S., Vokonas, P. S., & Sparrow, D. (2000). A prospective study of dominance and coronary heart disease in the normative aging study. *American Journal of Cardiology*, **86**, 145–149.

Siegman, A. W., Malkin, A. R., Boyle, S., Vaitkus, M., Varko, W., & Franco, E. (2002). Anger, and plasma lipid, lipoprotein, and glucose levels in healthy women: the mediating role of physical fitness. *Journal of Behavioral Medicine*, **25**, 1–16.

Siegman, A. W., Townsend, S. T., Civelek, A. C., & Blumenthal, R. S. (2000). Antagonistic behavior, dominance, hostility, and coronary heart disease. *Psychosomatic Medicine*, **62**, 248–257.

Smith, B. D., Cranford, D., & Green, L. (2001). Hostility and caffeine: Cardiovascular effects during stress and recovery. *Personality and Individual Differences*, **30**, 1125–1137.

Smith, T. W. (1992). Hostility and health: Current status of a psychosomatic hypothesis. *Health Psychology*, **11**, 139–150.

Smith, T. W. (1994). Concepts and methods in the study of anger, hostility, and health. In A. W. Siegman & T. W. Smith (Eds.), *Anger, hostility and the heart* (pp. 23–42). Hillsdale, NJ: Lawrence Erlbaum.

Smith, T. W. (1995). Assessment and modification of coronary-prone behavior: A transactional view of the person in social context. In A. J. Goreczny (ed.), *Handbook of health and rehabilitation psychology* (pp. 197–217). New York: Plenum Press.

Smith, T. W., Allred, K. D., Morrison, C. A., & Carlson, S. D. (1989). Cardiovascular reactivity and interpersonal influence: Active coping in a social context. *Journal of Personality and Social Psychology*, **56**, 209–218.

Smith, T. W., & Brown, P. W. (1991). Cynical hostility, attempts to exert social control, and cardiovascular reactivity in married couples. *Journal of Behavioral Medicine*, **14**, 581–592.

Smith, T. W., & Gallo, L. C. (1999). Hostility and cardiovascular reactivity during marital interaction. *Psychosomatic Medicine*, **61**, 436–445.

Smith, T. W., & Gallo, L. C. (2001). Personality traits as risk factors for physical illness. In A. Baum, T. Revenson, & J. Singer (Eds.), *Handbook of health psychology* (pp. 139–172). Hillsdale, NJ: Lawrence Erlbaum.

Smith, T. W., Gallo, L. C., & Ruiz, J. M. (2003). Toward a social psychophysiology of cardiovascular reactivity: Interpersonal concepts and methods in the study of stress and coronary disease. In J. Suls & K. Wallston (Eds.), *Social psychological foundations of health and illness* (pp. 335–366). Oxford, UK: Blackwell Publishers.

Smith, T. W., McGonigle, M. A., & Benjamin, L. S. (1998). Sibling interactions, self-regulation, and cynical hostility in adult male twins. *Journal of Behavioral Medicine*, **21**, 337–349.

Smith, T. W., Nealey-Moore, J., Uchino, B. N., & Hawkins, M. (2003). Hostility, anger, and cardiovascular reactivity during marital interaction. Paper presented at the American Psychosomatic Society.

Smith, T. W., Pope, M. K., Sanders, J. D., Allred, K. D., & O'Keefe, J. L. (1988). Cynical hostility at home and work: Psychosocial vulnerability across domains. *Journal of Research in Personality*, **22**, 525–548.

Smith, T. W., & Ruiz, J. M. (2000). *On the bright side of love and work: Optimism in social context*. Paper presented at the meeting of the Society of Personality and Social Psychology, San Antonio.

Smith, T. W., & Ruiz, J. M. (2002). Psychosocial influences on the development and course of coronary heart disease: Current status and implications for research and practice. *Journal of Consulting and Clinical Psychology*, **7**, 548–568.

Smith, T. W., Ruiz, J. M., & Uchino, B. N. (2000). Vigilance, active coping, and cardiovascular reactivity during social interaction in young men. *Health Psychology*, **19**, 382–392.

Smith, T. W., & Spiro, A. (2002). Personality, health, and aging: Prolegomenon for the next generation. *Journal of Research in Personality*, **36**, 363–394.

Smith, T. W., & Williams, P. G. (1992). Personality and health: Advantages and limitations of the five factor model. *Journal of Personality*, **60**, 395–423.

Smith, T. W., Uno, D., Uchino, B. N., & Ruiz, J. M. (2000). *Hostility, support from friends, and cardiovascular reactivity in young women*. Society for Psychophysiological Research.

Spielberger, C. D., Johnson, E. H., Russell, S. F., Crane, R. J., Jacobs, G. A., & Worden, T. J. (1985). The experience and expression of anger: Construction and validation of an anger expression scale. In M. A. Chesney & R. H. Rosenman (Eds.), *Anger and hostility in cardiovascular and behavioral disorders* (pp. 5–30). Washington, DC: Hemisphere.

Steptoe, A., Cropley, M., Griffith, J., & Kirschbaum, C. (2000). Job strain and anger expression predict early morning elevations in salivary cortisol. *Psychosomatic Medicine*, **62**, 286–292.

Steinberg, L., & Jorgensen, R. S. (1996). Assessing the MMPI-based Cook-Medley Hostility Scale: The implications of dimensionality. *Journal of Personality and Social Psychology*, **70**, 1281–1287.

Stoney, C. M., & Engebretson, T. O. (2000). Plasma homocysteine concentrations are positively associated with hostility and anger. *Life Sciences*, **66**, 2267–2275.

Suarez, E. C. (2003a). Plasma interleukin-6 is associated with psychological coronary risk factor: moderation by use of multivitamin supplements. *Brain Behavior and Immunology*, **17**, 296–303.

Suarez, E. C. (2003b). Joint effect of hostility and severity of depressive symptoms on plasma interleukin-6 concentration. *Psychosomatic Medicine*, **65**, 523–527.

Suarez, E. C., Bates, M. B., & Harralson, T. L. (1998). The relation of hostility to lipids and lipoproteins in women evidence for the role of antagonistic hostility. *Annals of Behavioral Medicine*, **20**, 59–63.

Suarez, E. C., Kuhn, C. M., Schanberg, S. M., Williams, R. B., & Zimmermann, E. A. (1998). Neuroendocrine, cardiovascular, and emotional responses of hostile men: The role of interpersonal challenge. *Psychosomatic Medicine*, **60**, 78–88.

Suarez, E. C., Lewis, J. G., & Kuhn, C. (2002). The relation of aggression, hostility, and anger to lipopolysaccharide-stimulated tumor necrosis factor (TNF)-α by blood monocytes from normal men. *Brain, Behavior, and Immunity*, **16**, 675–684.

Sudchay, S., & Larkin, K. T. (2001). Biobehavioral responses to interpersonal conflict during anger expression among anger-in and anger-out men. *Annals of Behavioral Medicine*, **23**, 282–290.

Sullivan, H. S. (1953). *The interpersonal theory of psychiatry*. New York: Norton.

Surwit, R. S., Williams, R. B., Siegler, I. C., Lane, J. D., Helms, M., & Applegate, K. L. (2002). Hostility, race, and glucose metabolism in nondiabetic individuals. *Diabetes Care*, **25**, 835–839.

Sykes, D. H., Arveiler, D., Salters, C. P., Ferrieres, J., McCrum, E., Amouyel, P., et al. (2002). Psychosocial risk factors for heart disease in France and Northern Ireland: the Prospective Epidemiological Study of Myocardial Infarction (PRIME). *International Journal Epidemiology*, **31** (6), 1227–1234.

Tangney, J. P., Wagner, P. E., Hill-Barlow, P., Marshall, D. E., & Gramzow, R. (1996). Relation of shame and guilt to constructive versus destructive respons-

es to anger across the life span. *Journal of personality and Social Psychology*, **70**, 797–809.

Trapnell, P. D., & Wiggins, J. S. (1990). Extension of the Interpersonal Adjective Scales to include the big five dimensions of personality. *Journal of Personality and Social Psychology*, **59**, 781–790.

Treiber, F. A., Kamarck, T., Schneiderman, N., Sheffield, D., Kapuku, G., & Taylor, T. (2003). Cardiovascular reactivity and development of preclinical and clinical disease States. *Psychosomatic Medicine*, **65**, 46–62.

Trobst, K. (2000). An interpersonal conceptualization and quantification of social support transactions. *Personality and Social Psychology Bulletin*, **26**, 971–986.

Vogele, C. (1998). Serum lipid concentrations, hostility and cardiovascular reactions to mental stress. *International Journal of Psychophysiology*, **28**, 167–179.

Wagner, C. C., Kiesler, D. J., & Schmidt, J. A. (1995). Assessing the interpersonal transaction cycle: Convergence of action and reaction interpersonal circumplex measures. *Journal of Personality and Social Psychology*, **69**, 938–949.

Welin, C., Lappas, G., & Wilhelmsen, L. (2000). Independent importance of psychosocial factors for prognosis after myocardial infarction. *Journal of Internal Medicine*, **247**, 629–639.

Whiteman, M. C., Deary, I. J., Lee, A. J., & Fowkes, F. G. (1997). Submissiveness and protection from coronary heart disease in the general population: Edinburgh Artery Study. *Lancet*, **350** (9077), 541–545.

Wiggins, J. S. (1979). A psychological taxonomy of trait-descriptive terms: The interpersonal domain. *Journal of Personality and Social Psychology*, **37**, 395–412.

Wiggins, J. S. (1996). An informal history of the interpersonal circumplex tradition. *Journal of Personality Assessment*, **66**, 217–233.

Wiggins, J. S., & Broughton, R. (1991). A geometric taxonomy of personality scales. *European Journal of Personality*, **5**, 343–365.

Wiggins, J. S., & Trapnell, P. D. (1996). A dyadic-interactional perspective on the five-factor model. In J. S. Wiggins (Ed.), *The five-factor model of personality* (pp. 88–162). New York: Guilford Press.

Williams, J. E., Nieto, F. J., Sanford, C P., Couper, D. J., & Tyroler, H. A. (2002). The association between trait anger and incident stroke risk: the Atherosclerosis Risk in Communities (ARIC) Study. *Stroke*, **33**, 13–20.

Williams, J. E., Nieto, F. J., Sanford, C P., & Tyroler, H. A. (2001). Effects of an angry temperament on coronary heart disease risk: The Atherosclerosis Risk in Communities Study. *American Journal of Epidemiology*, **154**, 230–235.

Williams, J. E., Paton, C. C., Siegler, I. C., Eigenbrodt, M. L., Nieto, F. J., & Tyroler, H. A. (2000). Anger proneness predicts coronary heart disease risk: Prospective analysis from the Atherosclerosis Risk in Communities (ARIC) study. *Circulation*, **101**, 2034–2039.

Williams, R. B., Jr. (1994). Basic biological mechanisms. In A. W. Seigman & T.W Smith (Eds.), *Anger, hostility, and the heart* (pp. 117–125). Hillsdale, NJ: Erlbaum.

Williams, R. B., Jr. (2000). Psychological factors, health, and disease: The impact of aging and the life cycle. In S. B. Manuck, R. Jennings, B. S. Rabin, &

A. Baum (Eds.), *Behavior, health, and aging* (pp. 135–151). Mahwah, NJ: Lawrence Erlbaum.

Williams, R. B. (2003). Socioeconomic status, hostility, and health behaviors—Does it matter which comes first? *American Journal of Epidemiology*, **158**, 743–746.

Williams, R. B., Jr., Barefoot, J. C., & Shekelle, R. (1985). The health consequences of hostility. In M. A. Chesney & R. H. Rosenman (Eds.), *Anger and hostility in cardiovascular and behavioral disorders* (pp. 173–185). New York: Hemisphere.

Williams, R. B., Marchuk, D. A., Gadde, K. M., Barefoot, J. C., Grichnik, K., & Helms, M. J., et al. (2001). Seratonin function and cardiovascular responses to stress. *Psychosomatic Medicine*, **63**, 300–305.

Williams, R. B., Marchuk, D. A., Gadde, K. M., Barefoot, J. C., Griebnik, K., & Helms, M. J. (2003). Serotonin-related gene polymorphisms and central nervous system Serotonin function. *Neuropsychopharmacology*, **28**, 533–541.

Yan, L. L., Liu, K., Matthews, K. A., Daviglus, M. L., Ferguson, T. F., & Kiefe, C. I. (2003). Psychosocial factors and risk of hypertension: The coronary artery risk development in young adults (CARDIA) study. *JAMA*, **290**, 2138–2148.