

Associations of Self-Reports Versus Spouse Ratings of Negative Affectivity, Dominance, and Affiliation With Coronary Artery Disease: Where Should We Look and Who Should We Ask When Studying Personality and Health?

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Objective: Aspects of negative affect and social behavior studied as risk factors for coronary heart disease are usually examined separately and through self-reports. Using structural models of these personality domains, we tested associations of self-reports and spouse ratings of anxiety, depressive symptoms, anger, affiliation and dominance with coronary artery disease (CAD). **Design:** In 154 healthy older couples, the authors tested cross-sectional associations with CAD of three facets of negative affectivity and two dimensions of the Interpersonal Circumplex, (IPC) using scales derived from the NEO-PI-R. **Main Outcome Measures:** CAD was quantified as Agatston scores from CT scans of coronary artery calcification (CAC). **Results:** Self-reports were generally unrelated to CAC, whereas spouse ratings were consistently associated, largely independent of potential confounds. When considered simultaneously, anxiety and anger were related to CAC but depression was not. When considered together, both dominance and (low) affiliation were related to CAC. **Conclusions:** Structural models of negative affectivity and social behavior can facilitate integrative study of psychosocial risk factors. Further, self-report measures of these traits might under-estimate related CHD risk.

Keywords: coronary artery disease, dominance, anger, anxiety, depression

Studies of personality play a central role in the evolving understanding of psychosocial influences on coronary heart disease (CHD) (Matthews, 2005). The most extensively studied traits involve negative affect and social behavior. For affective traits, anxiety, depressive symptoms, and anger confer increased risk (Suls & Bunde, 2005). For social behavior, hostility and aggressiveness also predict CHD (Smith, Glazer, Ruiz, & Gallo, 2004). Although conceptualized as an individual difference in social behavior, measures of hostility and aggressiveness often include anger and therefore overlap with affective traits. Dominance, a second individual difference in social behavior, also confers CHD risk (Houston et al., 1992; Siegman et al., 2000). The present study addressed conceptual and methodological issues in personality

measurement that have complicated this area of research on psychosocial risk for CHD.

Anxiety, depressive symptoms, and anger are usually examined separately as CHD risk factors (Suls & Bunde, 2005). Yet, they are intercorrelated aspects of the broader trait of negative affectivity (NA) or neuroticism (*N*) (Costa & McCrae, 1992; Watson & Clark, 1984), which contrasts negative emotionality with emotional stability. When studied separately, associations with CHD could reflect the specific trait assessed, the broader *N/NA* dimension, or an unmeasured but correlated component of *N/NA* (Suls & Bunde, 2005). The few studies assessing multiple negative affective traits indicate that their associations with CHD are not necessarily independent (Boyle, Michalek, & Suarez, 2006; Kubzansky et al., 2006). However, these studies have not used measures based on related structural models of *N/NA* (e.g., Costa & McCrae, 1992) that would provide an integrative and comprehensive view of negative emotionality as a CHD risk factor.

Elsewhere we have suggested that the interpersonal circumplex (IPC) provides similar integration for individual differences in social behavior studied as CHD risk factors (Smith, Gallo, & Ruiz, 2003; Smith et al., 2004). Depicted in Figure 1, the IPC (Kiesler, 1983) comprises two dimensions—affiliation and dominance. The affiliation dimension contrasts cold-heartedness and quarrelsomeness with warmth and friendliness. The dominance dimension

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encompasses behavior related to exerting dominance and control over others and achieving status, contrasting them with submissiveness and passivity (Pincus & Ansell, 2003). Personality measures associated with hostility in the IPC confer coronary risk (Smith et al., 2004), and dominance may as well. Using ratings from the Type A Structured Interview, Houston and colleagues (1992) found that hostility and a socially dominant interaction style (e.g., emphatic speech, talking “over” others) independently predicted subsequent CHD. Siegman and colleagues (2000) found that self-reports of dominance were related to incident CHD, while controlling self-reported anger. However, the independent roles of individual differences in dominance and hostility would be better demonstrated using measures directly tied to the IPC.

Most studies of personality and CHD use self-reports scales, yet individuals might not give accurate self-descriptions of undesirable traits (Paulus, 1984). In personality assessment, self-reports and ratings by others converge significantly but moderately (Costa & McCrae, 1992), and findings often differ across these methods (Oltmanns & Turkheimer, 2006). Further, interview-based ratings of personality and ratings provided by significant others are often more closely related to CHD than are self-reports (Kneip et al., 1993; Miller et al., 1996). Hence, self-reports of personality could underestimate associations with CHD. However, when comparing the utility of self-reports and spouse ratings of personality risk factors it is important to consider marital adjustment, as spouse ratings of negative personality traits could reflect a troubled relationship, which also confers risk for CHD (Matthews & Gump, 2002). Studies comparing self-reports of personality risk factors and ratings by others often include clinical samples undergoing invasive diagnostic procedures (e.g., angiography) or CHD patients (Ketterer et al., 2004; Kneip et al., 1993; Siegman, Townsend, Blumenthal, Sorkin, & Civelek, 1998). When patients are compared to healthy controls in cross-sectional designs, associations of disease with emotional or social functioning could reflect reactions to CHD. In clinical samples undergoing invasive procedures, individuals with disease are overrepresented and disease-free individuals are not representative of the general population, producing potentially misleading results regarding risk factors for CAD (Miller, 1994).

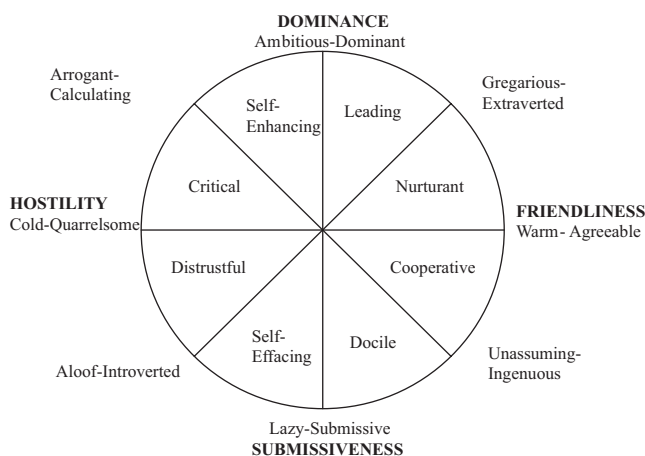


Figure 1. The interpersonal circumplex.

The present study addressed these issues by examining associations of self-reports and spouse ratings of negative affect and social behavior, based on structural models of these personality domains, with asymptomatic CAD as assessed by coronary artery calcification (CAC) in healthy older adults. CAC is closely related to CAD assessed invasively, and is a strong predictor of subsequent CHD (Pletcher, Tice, Pignone, & Brower, 2004). Further, CAC can be used to assess the presence and severity of asymptomatic CAD in representative samples of otherwise healthy persons.

Specifically, we tested associations of affective facets of N/NA (i.e., anxiety, depressive symptoms, and anger) and the IPC dimensions (hostility, dominance) with CAC in healthy older couples. In a previous report from this sample (Smith et al., 2007), the angry hostility facet of *N* and trait antagonism (vs. agreeableness) of the Five Factor model (FFM) of personality were associated with CAC. Analyses reported here extend this prior work in two ways. First, associations of CAC with the two other negative affect facets of *N* are tested. Second, antagonism is closely associated with low affiliation in the IPC, but is also significantly associated with dominance (McCrae & Costa, 1989). Because either IPC dimension could contribute to the association of antagonism with CAC, it was important to examine these two social-behavioral CHD risk factors directly. We predicted that anxiety, depression, and anger would be associated with CAC when considered separately, though it was not clear if these traits would be independently associated when considered together. We predicted that the dominance and hostility dimensions of the IPC would both be related to CAC. Finally, we predicted that spouse-ratings of these traits would be more consistently related to CAC than would self-reports.

Method

Participants

The Utah Health and Aging Study, approved by the University of Utah Institutional Review Board, enrolled 300 middle-aged and older couples during 2001 through 2005. All participants gave informed consent. Couples from the Salt Lake City metropolitan area were recruited through a polling firm, newspaper advertisements, and community programs. Participants met the following criteria: (a) couples had at least one member between 40 and 50 years old (middle-aged group) or between 60 and 70 years old (older group), (b) no more than 10 year age difference, (c) no history of cardiovascular disease, and (d) not taking cardiovascular medications (e.g., beta-blockers). The present report is based on the 154 older couples, because CAC prevalence was too low in middle-aged couples to permit sensitive tests of associations with psychosocial risk factors (Smith et al., 2007). For this sample, women averaged 62.2 years of age (range = 50 – 71) and men 64.7 (52 – 76), and 95.4% were non-Hispanic White. Mean length of marriage was 36.4 years (5 – 53 years), and for 76% of participants the current marriage was their first (17% second). Median household income was \$50,000 to 75,000 per year.

Measures

Personality and marital adjustment. Participants completed portions of self-report and observer rating (Forms S and R) versions of the NEO-PI-R (Costa & McCrae, 1992). Items in-

cluded Neuroticism facet scales of negative emotionality (i.e., anxiety, angry hostility, depression). These scales have high internal consistency, and convergent and discriminant validity (Costa & McCrae, 1992). Other NEO-PI-R items were used to calculate scores for dominance and affiliation IPC dimensions, based on IPC octants (see Figure 1). We confirmed the circumplex structure of these octant scales (Traupman et al., 2007). Principal components analyses (PCA) revealed two factors corresponding to dominance and affiliation for both forms, and octant scales had the predicted circular ordering of factor loadings (Gurtman & Pincus, 2003). Tests of circular ordering of correlations among octant scales (Tracey, 1997) were highly significant, and tests of correspondence between self-report and spouse-rating octant scales provided strong evidence of their convergent and discriminant validity (cf. Wagner, Kiesler & Schmidt, 1995). Octant scales were combined through circumplex weighting (Gurtman & Pincus, 2003), and resulting dominance and affiliation scores demonstrated convergent and discriminant associations with a second IPC-based measure. Participants also completed the Locke-Wallace Marital Adjustment Test (MAT) (Locke & Wallace, 1959).

Risk factors. A fasting blood draw was taken to measure glucose levels and plasma lipids. Behavioral risk factors (i.e., smoking status, exercise/activity level) were assessed via self-report. Mean values for these risk factors are presented in Table 1.

Coronary artery calcification. Participants underwent two coronary artery scans on a multidetector scanner (Phillips MX8000, Philips Medical Systems, Cleveland, OH). Scans used 2.5-mm thick transverse slices obtained from 2-cm inferior to the carina to the inferior margin of the heart. Four 2.5-mm slices were obtained with each gantry rotation. Each scan was obtained in a single breath-hold using 500 msec exposure and an axial (non-spiral) mode of imaging. ECG-triggering was used to acquire images during diastole corresponding to 50% of the R-R interval.

Table 1
Risk Factors

	Wives	Husbands
Smoking status %		
Never	79.2	62.7
Former	19.5	35.3
Current	1.3	2.0
Alcohol use %		
Rare or never	61.1	56.0
Low	20.8	16.0
Moderate to high	18.1	28.0
Exercise level %		
Sedentary	10.1	5.3
Mild	26.8	29.3
Moderate	36.2	29.3
High	26.8	36.0
BMI	27.4 (4.8)	27.8 (4.0)
Total cholesterol	202.7 (34.5)	182.0 (33.2)
Triglycerides	137.8 (82.5)	128.0 (64.1)
HDL	60.7 (16.5)	47.3 (12.9)
LDL	118.6 (28.3)	111.3 (28.8)
VLDL	26.1 (15.4)	25.1 (14.3)
SBP mmHg	124.7 (18.4)	127.0 (16.4)
DBP mmHg	70.5 (10.2)	77.1 (9.4)
Glucose	90.6 (20.1)	93.3 (18.0)

Note. Standard deviations in parentheses.

Image reconstruction was performed with a 220-mm field of view using a 512×512 matrix with a standard reconstruction filter to give a nominal pixel area of 0.18-mm^2 and voxel volume of 0.46-mm^3 . To correlate CAC scores to those reported on electron-beam scanners using the method of Agatston et al. (1990), multi-detector imaging scores were multiplied by a factor of 0.833 (2.5 mm/3.0 mm) to compensate for the smaller slice thickness. For women and men, respectively, 51.3% and 81% had detectable CAC (i.e., nonzero Agatston scores).

Procedures

Participants were screened in telephone interviews. They completed NEO-PI-R items and other questionnaires independently. They underwent the blood draw, height and weight were determined, and sitting blood pressure readings were taken via random zero mercury sphygmomanometer. Computed tomography (CT) scans were performed as described above.

Statistical Analyses

The primary analyses utilized Proc Mixed (SAS Institute) to test predictors of CAC. Proc Mixed uses a random regression model to derive parameter estimates both within and across individuals (Singer, 1998). All factors were treated as fixed, and Proc Mixed treats unexplained variation within individuals as a random factor. Following Campbell and Kashy (2000), we modeled individuals (i.e., husband, wife) within a dyad as a repeated factor using the compound symmetry covariance structure ("type = cs"). This model allowed us to examine predictors (e.g., anxiety) of CAC scores, while controlling the dependency within married couples. To illustrate, the equation for the main analyses of anxiety on CAC scores (excluding covariates) was as follows:

$$\text{CAC}_{\text{person } i, \text{Dyad } j} = b_{0j} + b_{1j}X_{\text{anxiety } ij} + e_{ij}$$

where b_{0j} represents the average CAC intercept for dyad j as all measures were centered at their grand mean (see Singer, 1998). The coefficient b_1 represents restricted maximum likelihood estimates of the slopes estimating person i in dyad j 's CAC scores from partner anxiety ratings. Output of random regression models were parameter estimates (b) with appropriate within-subjects covariance structure. As recommended (Campbell & Kashy, 2000), we used the Satterthwaite approximation to determine appropriate degrees of freedom. Models examining statistical interactions (e.g., anxiety \times gender) were an extension of the basic model above. As recommended, we transformed CAC scores as $\log(\text{CAC} + 1)$ to reduce positive skew, but did not form groups through CAC cutpoints as this has been shown to reduce statistical power (Reilly, Wolfe, Localio, & Rader, 2004).

Results

Correlations Between Self-Reports and Spouse Ratings of Personality

Table 2 presents correlations between self-reports and spouse ratings. Convergent associations (i.e., two methods assessing one trait) are presented on the diagonal. In each of 80 possible comparisons, convergent correlations are larger than the absolute

Table 2
Correlations Between Self-Reports and Spouse Ratings of Individual Differences in Negative Affect and Social Behavior

		Wives' self-report				
Husbands' ratings of wives	1.	2.	3.	4.	5.	
1. Dominance	.59**	.08	-.21**	.10	-.17*	
2. Affiliation	-.06	.35**	-.07	-.27**	-.21**	
3. Anxiety	-.13	-.15	.57**	.26**	.39**	
4. Angry hostility	.06	-.23**	.23**	.44**	.39**	
5. Depression	-.25**	-.19*	.45**	.27**	.54**	
		Husbands' self-report				
Wives' rating of husbands	1.	2.	3.	4.	5.	
1. Dominance	.58**	.15	-.29**	-.05	-.31**	
2. Affiliation	-.04	.47**	-.13	-.26**	-.17**	
3. Anxiety	-.06	-.09	.44**	.23**	.35**	
4. Angry Hostility	.14	-.31**	.17*	.48**	.20*	
5. Depression	-.15	-.17*	.39**	.31**	.47**	

Note. $N = 154$. * $p < .05$. ** $p < .001$ (two-tailed). Convergent associations in bold.

value of all other correlations in a given row and column, providing strong evidence of convergent and divergent validity. Dominance is easily distinguished from affiliation, whereas correlations among the negative affects are higher, as expected for aspects of a higher-order construct (i.e., N/NA). The weakest discriminant validity is between anxiety and depression, consistent with prior research and theory (Clark, Watson, & Mineka, 1994).

Associations with Coronary Artery Calcification

Negative affect. Our first set of analyses tested the separate associations between spouse ratings of their partner's negative affect (i.e., anxiety, angry hostility, depression) with that partner's CAC, along with potential interactions with gender while controlling age and gender. Older individuals ($p < .001$) and men ($p < .001$) had higher CAC scores. When considering these covariates, spouse ratings of their partner's anxiety ($b = .53, p < .002$), angry hostility ($b = .53, p < .002$), and depression ($b = .34, p < .05$) were associated with higher partner CAC. No interaction with gender approached significance ($p > .70$).

We next examined alternative explanations or mediational pathways that might account for these effects. When controlling SES (i.e., household income and occupational status), effects for spouse ratings of anxiety ($b = .47, p < .008$) and angry hostility ($b = .49, p < .004$) were not altered. However, the association between depression and CAC became nonsignificant ($b = .26, p < .14$). We next considered biomedical and behavioral risk factors. Preliminary analyses tested univariate associations of risk factors in Table 1 with CAC. We selected those with a $p < .10$ for further consideration. Alcohol consumption ($p < .04$), smoking status ($p < .01$), resting DBP ($p < .07$), triglycerides ($p < .001$), VLDL ($p < .02$), and fasting glucose ($p < .07$) were related to higher CAC, whereas HDL ($p < .07$) was related to lower CAC. Controlling these risk factors did not alter associations of spouse ratings of anxiety ($b = .43, p < .02$) or angry hostility ($b = .40, p < .02$) with CAC, but the effect for depression again became nonsignificant ($b = .23, p < .18$).

We next examined marital quality. Spouse ratings of partner's anxiety ($b = .52, p < .003$) and angry hostility ($b = .52, p < .005$)

continued to predict CAC while controlling MAT scores, but spouse ratings of depression were marginally related ($b = .31, p < .08$). In additional models we considered each personality trait separately, while controlling demographic, health behavior, and biomedical risk factor that had associations with CAC approaching significance (i.e., $p = .10$ or less) when the full set of control variables was considered simultaneously (i.e., age, gender, alcohol use, and triglyceride levels). Anxiety ($b = .44, p < .01$) and angry hostility ($b = .46, p < .01$) were significantly related to CAC, whereas depression was not ($b = .28, p < .10$). In a more conservative model, we included all of the demographic, health behavior, and biomedical control variables considered above, regardless of their independent associations with CAC. Again, anxiety ($b = .37, p < .04$) and angry hostility ($b = .38, p < .03$) were significantly related to CAC in these analyses, whereas depression was not ($b = .16, p < .36$).

We also tested the independent associations of spouse ratings of these negative affects with CAC. When simultaneously considering anxiety, angry hostility, and depression while controlling age and gender, the associations for anxiety ($b = .52, p < .04$) and angry hostility ($b = .43, p < .03$) remained significant, but depression did not ($p > .29$).

Finally, we tested separate associations of self-reported negative affects and CAC, controlling age and gender. Self-reports of anxiety ($b = .09, p > .65$), angry hostility ($b = .009, p > .90$), and depression ($b = -.004, p > .90$) were not related to CAC.¹

Interpersonal behavior CAC. We next tested associations of spouse ratings of dominance and affiliation with CAC, again controlling age and gender and modeling the interactions of dominance and affiliation with gender. Spouse ratings of dominance were related to higher CAC ($b = .11, p < .004$), whereas spouse ratings of affiliation were related to lower CAC ($b = -.09, p < .02$). When tested simultaneously, spouse ratings of dominance and affiliation were independent predictors of CAC (dominance: $b = .11, p <$

¹ Spouse reports of overall neuroticism were related to CAC, controlling age and gender, $p < .05$. However, self-reports and spouse ratings of the nonaffective NEO-PI-R facets of N (i.e., self-consciousness, perceived vulnerability, and impulsiveness) were not.

.002, affiliation: $b = -.10, p < .01$). No interactions involving gender were significant (p 's $> .33$).

Controlling income and occupational status did not alter the association of spouse ratings of dominance ($b = .13, p < .001$), or affiliation ($b = -.08, p < .03$) with CAC. Controlling behavioral and biomedical risk factors (i.e., alcohol consumption, smoking status, resting DBP, triglycerides, VLDL, HDL, fasting glucose levels) also did not alter the effects of spouse ratings of dominance ($b = .10, p < .008$), but the association between spouse ratings of affiliation and CAC was reduced to marginal significance ($b = -.07, p = .06$). Controlling MAT scores did not influence the significant associations of spouse ratings of dominance ($b = .11, p = .005$) or affiliation ($b = -.09, p < .04$) with CAC. In an additional model controlling all demographic, health behavior, and biomedical risk factors for which the independent association with CAC approached significance (i.e., age, gender, alcohol use, and triglyceride levels), dominance ($b = .11, p < .01$) and affiliation ($b = -.08, p < .02$) were significantly associated with CAC. In the more conservative model including all of the demographic, health behavior, and biomedical control variables considered above regardless of their independent associations with CAC, dominance was significantly associated with CAC ($b = .12, p < .001$) but the association for affiliation became marginal ($b = -.06, p < .08$).

We also examined the association of self-reports of affiliation and dominance with CAC, again controlling age and gender. The affiliation axis of the IPC was unrelated to CAC ($b = .04, p < .26$). However, self-reported dominance was significantly associated with CAC ($b = .10, p < .01$). This association remained significant in analyses controlling SES ($b = .13, p < .002$), risk factors ($b = .08, p < .03$), and MAT scores ($b = .10, p < .02$).

Self versus partner ratings and CAC scores. We next tested independent effects of parallel self-reports and partner ratings of each personality trait, by entering pairs of self-reports and spouse ratings simultaneously and controlling age and gender. For negative affects, self-reports of anxiety ($b = -.25, p > .26$), angry hostility ($b = -.33, p > .14$), and depression ($b = -.25, p > .21$) were not associated with CAC when controlling spouse ratings. However, spouse ratings of anxiety ($b = .64, p < .002$), angry hostility ($b = .65, p < .001$), and depression ($b = .46, p < .02$) predicted higher CAC independent of self-ratings. Similarly, spouse ratings of dominance ($b = .09, p < .05$) predicted higher CAC, and spouse ratings of affiliation ($b = -.13, p < .001$) predicted lower CAC independent of self-ratings. When controlling spouse ratings, self-reports of dominance were not related to CAC ($b = .04, p > .40$). However, self-reports of affiliation were significantly and *positively* related to CAC scores ($b = .10, p < .02$) when controlling spouse ratings.

Controlling income and occupational status did not influence these independent associations for spouse ratings of anxiety ($b = .60, p < .003$), angry hostility ($b = .63, p < .001$), depression ($b = .38, p = .05$), dominance ($b = .09, p < .05$), and affiliation ($b = -.12, p < .003$), and self-ratings of affiliation ($b = .10, p < .03$). When controlling traditional risk factors, spouse ratings of anxiety ($b = .50, p < .02$), angry hostility ($b = .52, p < .005$), dominance ($b = .09, p = .05$), and affiliation ($b = -.11, p < .005$), and self-ratings of affiliation ($b = .11, p < .02$) still predicted CAC, but the association for spouse ratings of depression was no longer significant ($b = .32, p = .09$). Finally, control of MAT scores did not affect these significant associations for spouse ratings of anxiety ($b = .63, p < .002$), angry

hostility ($b = .64, p < .001$), depression ($b = .43, p < .03$), dominance ($b = .09, p = .05$), affiliation ($b = -.12, p < .003$), and self-ratings of affiliation ($b = .10, p < .02$) on CAC scores.

Independent Associations of Negative Affect and Social Behavior with CAC

We also considered simultaneous associations with CAC of spouse ratings of the two affective traits that were independently related to CAC (i.e., anxiety, angry hostility), dominance and affiliation, controlling age and gender. In this analysis, anxiety ($b = .53, p < .01$) and dominance ($b = .13, p < .001$) were independently related to CAC, whereas angry hostility ($b = .07, p = .77$) and affiliation ($b = -.05, p = .27$) were not.

Discussion

As predicted, individual differences in negative affect and social behavior were concurrently associated with asymptomatic CAD, as measured by CAC in otherwise healthy older adults. When considered individually, anxiety, angry hostility, depression, dominance, and low affiliation were each associated with greater CAC. Except for depression, these effects were generally independent of age, gender, SES, and traditional biomedical and behavioral risk factors. However, these associations were largely nonsignificant when personality was assessed via self-reports and instead emerged when spouse ratings were used, an effect that could not be attributed to marital satisfaction. Further, these risk factors were correlated, and when the three negative affects were considered together anxiety and anger were independently related to CAC but depression was not. Dominance and (low) affiliation were independently related to CAC when considered together, but anger and affiliation were not significantly related to CAC when negative affects and dimensions of social behavior were considered together. Hence, some effects of conceptually distinct personality risk factors with CHD were overlapping.

The Role of Assessment Methods in Studies of Personality and CHD Risk

The stronger effects for spouse ratings compared to self-reports are consistent with previous studies (Ketterer et al., 2004; Kneip et al., 1993; Miller et al., 1996; Siegman et al., 1998). It is important to note, however, that the present study is—to our knowledge—the first to compare the utility of parallel self-reports and spouse ratings of a comprehensive set of individual differences in negative affect and social behavior as CAD risk factors in an outwardly healthy sample and using well-validated scales.

The better predictive utility of spouse ratings could indicate that individuals are unable or unwilling to provide highly accurate self-reports of undesirable traits. Among the self-report scales examined here, only dominance was significantly related to CAC, perhaps because it is less socially undesirable. In previous studies the discrepancy between self-reports and spouse ratings of negative affect has been associated with CHD morbidity; individuals who report less negative affect than expected on the basis of spouse ratings are at increased risk (Ketterer et al., 1998, 2004), perhaps reflecting adverse effects of denial. In the present results, higher self-reported affiliation was associated with *greater* CAC

when controlling spouse ratings. Perhaps, variance in self-reported affiliation that is independent of spouse ratings reflects unhealthy denial of hostility. However, this pattern did not emerge for measures of negative affect, providing no evidence that denial of negative emotion confers risk. Failure to appreciate the extent of one's quarrelsomeness could be an independent CAD risk factor beyond more objective indications of this aspect of social behavior. Also, self-reports capture the experience of these aspects emotional and social functioning, whereas spouse reports also capture whether or not an individual regulates the expression of potentially problematic tendencies. The greater predictive utility of spouse reports could be because of the fact that they assess this combination of risky personality traits and self-regulatory capacity.

Regardless of the meaning of discrepancies between self-reports and spouse ratings, the present results provide evidence of incremental validity (Hunsley & Meyer, 2003) of spouse ratings of personality risk factors. Ease of administration encourages use of self-reports in studies of CHD risk, and a large literature attests to their predictive utility (Smith & MacKenzie, 2006). Yet, this method might provide an underestimate of the role of psychosocial risk factors. Null findings for associations between self-reports of psychosocial risk factors and CAC (e.g., Diez Roux et al., 2006) have been interpreted as evidence that these risk factors do not contribute to early stages of CHD (Whooley, 2006). Such conclusions based solely on self-reports could lead to an inaccurate view of the role of psychosocial risk factors in the decades-long development of CHD.²

Negative Affect and CAD

The associations of anxiety, depression, and angry hostility with CAC are consistent with prior research on CHD risk (Suls & Bunde, 2005); these traits may contribute to CHD at least in part through the development of CAD. However, only anxiety and anger were independent affective correlates of CAC, suggesting that it is important to consider overlapping effects of correlated aspects of this trait domain (Suls & Bunde, 2005). Some scales implying measurement of distinct negative affects (e.g., anxiety vs. depressive symptoms) are psychometrically indistinguishable (Watson & Clark, 1984), and as expected in the present sample these traits are closely interrelated. Hence, tests of associations with CHD of an individual negative affect considered separately could provide potentially misleading evidence of specificity or fail to identify broader dimensions of risk. Similarly, when nonaffective aspects of N/NA (e.g., self-esteem, helplessness) are studied as CHD risk factors, significant associations could reflect unmeasured affective aspects of N/NA (see Footnote 1). The issue of general versus specific emotional risk also has implications for risk-reducing interventions. Future interventions might usefully incorporate approaches that target general emotional dysfunction (e.g., Moses & Barlow, 2006), rather than specific emotional symptoms or disorders. The present findings suggest that candidate interventions should have demonstrated effectiveness in the reduction of both anxiety and anger.

The weak association of depression scores with CAC in this sample may indicate that a fluctuating characteristic like depressive symptoms influences CHD onset and course at later stages of the disease, rather than through the initiation and progression of CAD.³ In contrast, anger or anxiety may be sufficiently stable as to influence the decades-long process of initiation and progression

of CAD (Kop, 1999). Lifetime history of depression is related to atherosclerosis, perhaps reflecting effects of more chronic negative affect (Agatista et al., 2005). However, although the NEO-PI-R depression scale includes clinically relevant symptoms (e.g., negative mood, guilt, hopelessness), it is a measure of normal variation in depressed affect rather than depressive disorder.

Social Behavior and CHD Risk

The independent associations of hostile and dominant social behavior with CAC extend prior evidence regarding these risk factors and support suggestions that both IPC axes confer risk for CHD (Smith et al., 2003; 2004). To our knowledge, this is the first evidence using a well-validated IPC-based measure that these aspects of social behavior are independently related to CAD or CHD. Previous analyses of this sample indicated that the FFM trait of agreeableness was inversely associated with CAC. Agreeableness is closely correlated with high affiliation in the IPC, but also with low dominance (McCrae & Costa, 1989).⁴ Either of these IPC correlates could account for the association of agreeableness with CAD. Further, the other FFM social behavior trait, extraversion, combines high dominance and affiliation. Because dominance and affiliation have opposite associations with CHD, the blend of social behavior captured by extraversion could mask associations of a second aspect of interpersonal behavior with CHD.⁵ Hence, conceptualization and measurement of individual differences in social behavior as CHD risk factors may be better guided by the IPC framework than the FFM taxonomy.

Beyond the empirical issue of predictive utility, IPC and FFM conceptualizations of individual differences in social behavior also differ in their correspondence with potentially health-relevant biobehavioral systems. Depue (2006) has argued that the IPC version of this two-dimensional space corre-

² Another possible explanation for the greater predictive utility of spouse ratings is that they are influenced by that spouse's own personality, and that spouse personality is related to partner's CAC (i.e., cross-spouse or "partner" effects of personality on CAC). However, no measures (i.e., self-report or spouse rating) of one partner's personality trait were related to the other partner's level of CAC—across any of the five traits studied.

³ Sample means on self-reports and spouse ratings of depression were at or somewhat above reported norms for adults (Costa & McCrae, 1992). Hence, it is unlikely that low sample levels of depressive symptoms explain the null result.

⁴ Spouse ratings of agreeableness were closely associated with the IPC affiliation axis for both men, $r = .87$, and women, $r = .85$. Hence, our prior report of an association of agreeableness with CAC in this sample overlaps with effects of IPC affiliation reported here. However, as in prior research (e.g., McCrae & Costa, 1989), spouse ratings of agreeableness were also associated with low dominance for men, $r = -.49$, and women, $r = -.38$. Hence, the effect for agreeableness versus antagonism confounds these two IPC dimensions, found here to be independently related to CAD. Given that the two-dimensional space defined by the IPC can be seen as a rotated equivalent of that defined by the FFM traits (i.e., agreeableness and extraversion), it is important to note that neither self-reports or spouse ratings of extraversion were associated with CAC.

⁵ In the present sample, spouse ratings of extraversion were associated with high dominance for men, $r = .85$, and women $r = .87$, and with high affiliation for men, $r = .36$, and for women, $r = .52$. As described in Footnote 4, neither self-reports or spouse ratings of extraversion were significantly related to CAC.

sponds more closely to neurobiological underpinnings of social behavior than does the FFM version; dominance reflects the activation and/or responsivity of a dopaminergic incentive or approach motivational system, whereas the affiliation axis reflects variation in an opioid reward system. The FFM trait of extraversion combines these two systems. Hence, the IPC could provide a more specific account of biological underpinnings of psychosocial risk for CHD.

Biologic mechanisms linking affiliation (McCabe et al., 2002) and social dominance (Kaplan & Manuck, 1998) with atherosclerosis have been identified in animal models. In humans, individual differences in dominance and experimentally manipulated expressions of dominant social behavior are associated with increased cardiovascular reactivity (CVR) (Smith et al., 2003), which in turn is related to increased cardiovascular risk (e.g., Matthews et al., 2006). Similarly, individual differences in affiliation and experimental manipulations of affiliative aspects of social context alter CVR and neuroendocrine responses in expected ways (Smith et al., 2003; Uchino, 2006). Hence, both IPC dimensions are related to CHD risk and to plausible underlying mechanisms.

The IPC has an additional use in research on CHD risk; it can describe both individual differences in social behavior (i.e., personality traits) and social-environmental risk factors, such as low social support (Trobst, 2000) or relationship conflict, thereby providing an integrative psychosocial framework. Personality risk factors for CHD are often correlated with social-environmental risk factors (e.g., Gallo & Smith, 1999). Hence, just as studying personality traits in isolation could impede development of a more comprehensive and integrated understanding of psychosocial influences on CHD, so could separation of personality and social-environmental risk factors. For example, hostile personality traits are consistently associated with social environments characterized by high conflict and reduced social support (Smith et al., 2004), perhaps reflecting an ongoing reciprocal process in which antagonistic social behavior both fosters and is maintained by quarrelsome and unsupportive interpersonal relationships. Similarly, social dominance might be associated both with self-selection into social contexts requiring further agonistic striving and evocation of competitiveness in others, thereby maintaining this interaction style. These ongoing interpersonal transactions may provide a better representation of psychosocial risk for CHD than static personality and social-environmental risk factors considered separately (Smith et al., 2004). By providing a common conceptual and methodological framework, the IPC can promote such an integrative study of psychosocial risk factors (Gallo, Smith, & Cox, 2006; Smith et al., 2003, 2004). Further, the tradition surrounding the IPC (Pincus & Ansell, 2003) includes mechanistic accounts of how personality traits and aspects of the social environment reciprocally influence each other (Wagner et al., 1995), and these transactional models could guide the refinement of approaches to risk reduction.

Limitations, Conclusions, and Future Directions

The present sample was largely White and middle class, and all participants were married. Generalizations to other demographic groups must be made cautiously. The cross-sectional

design precludes causal inferences. However, the use of an outwardly healthy sample free from symptomatic CHD reduces the likelihood that associations between personality and CAD reflect emotional and behavioral responses to disease. The noninvasive scans of CAC permitted examination of psychosocial correlates of CAD without worrisome selection artifacts inherent in clinical samples referred for invasive tests (e.g., angiography), but CAC may not capture some features of CAD (e.g., plaque instability) through which personality could influence CHD. Finally, the PROC Mixed HLM analysis used here does not permit calculation of effect sizes.⁶

These limitations notwithstanding, our results illustrate the value of structural models of negative affect and social behavior in clarifying associations of personality with CAD. A great variety of personality traits have been studied separately as CHD risk factors, typically with little regard for their potentially overlapping effects (Smith & MacKenzie, 2006). Structural models of personality can provide a valuable integrative guide, as researchers and clinicians consider, “*Where should we look?*” for general and specific dimensions of risk. The present findings revealed both overlapping and independent associations between CAC and aspects of negative affect that are typically studied separately. The assessment of both dimensions of the IPC demonstrated that two dimensions of social behavior—low affiliation (i.e., hostility) and high dominance—are independently associated with CAD, extending the field’s traditional emphasis on hostility. Constructs and measures from well-established models of personality such as the IPC and FFM can be used directly as predictors of health outcomes and to compare and contrast other personality measures, facilitating an integrated literature in which central dimensions of risk and resilience can be identified by locating risk factors in a common conceptual and empirical space or nomological net (Cronbach & Meehl, 1955).

However, only rarely do researchers or clinicians pause to consider, “*Who should we ask?*” when assessing personality risk factors. Importantly, the present results support prior findings that effects for self-reports of psychosocial risk factors may be weaker than those obtained with other methods (Keterer et al., 2004; Kneip et al., 1993; Siegman et al., 1998). Given that the vast majority of studies of CHD risk have used self-reports of personality, the prior evidence could represent an underestimate of the consistency and magnitude of associations between personality and health.

⁶ *t* test values from the PROC Mixed analyses as well as correlation coefficients (point-biserial and Pearson) pooled across spouses can provide a rough indication of at least relative effect sizes. Using these admittedly imprecise metrics, age and gender were more closely related to CAC than were any of the personality traits, whereas the personality traits were equally or somewhat more closely related to CAC than were the significant biomedical and health behavior risk factors.

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