Covariation of Psychosocial Characteristics Associated With Cardiovascular Disease: Genetic and Environmental Influences

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Objective: Three psychosocial characteristics associated with cardiovascular disease (CVD)—depression, hostility, and social support—tend to correlate with one another. However, the causes of each characteristic and why they tend to co-occur are not completely understood. Therefore, the current study used a twin design to examine the relative contributions of genetic and environmental influences to the variation and covariation of these three psychosocial characteristics. Methods: The sources of variation and covariation among the Beck Depression Inventory, the Cook-Medley Hostility Scale, and the Interpersonal Support Evaluation List were examined in a young adult community sample of 157 monozygotic and 75 dizygotic twin pairs. Results: Phenotypic confirmatory factor analysis indicated that a single latent factor could account for their moderate intercorrelations. Twin analyses indicated that the Beck Depression Inventory and Interpersonal Support Evaluation List were each influenced by genetic and nonshared environmental factors, whereas the Cook-Medley Hostility Scale was influenced by familial (genetic and/or shared environmental) and nonshared environmental factors. Bivariate associations between these scales were largely determined by common genetic effects and, to a lesser degree, common nonshared environmental effects. Covariation among the three scales could be explained by a single common genetic factor and a common nonshared environmental factor. Environmental factors shared within families did not contribute to covariation among the psychosocial characteristics. Conclusions: The results challenge the conventional approach of examining three psychosocial variables as independent risk factors for cardiovascular disease and argue for the importance of investigating specific causes for their covariation. Key words: twins, genetic, depression, hostility, social support, cardiovascular disease.

INTRODUCTION

Cardiovascular disease (CVD) is the leading cause of mortality in the United States, accounting for 41.4% of all deaths in 1996 (1). Although many biological (eg, hypercholesterolemia, hypertension) and behavioral (eg, smoking, physical inactivity) risk factors for CVD are well established, a growing body of evidence is consistent with the hypothesis that certain psychosocial characteristics, including depression, hostility, and lack of social support, may also predict increased disease risk (2–8). Despite the accumulating literature on the potential consequences of these characteristics, considerably less is known about their causes.

Researchers have tended to investigate putative psychosocial risk factors separately, contributing to what Kaplan (9) has called a “risk-factor-of-the-month-club.” However, it is unclear whether this independent approach is justified. Indeed, it is plausible to hypothesize on a variety of theoretical grounds that depression, hostility, and social support should be intercorrelated. For instance, those who are depressed may view the world in a cynically hostile manner by virtue of their negative affective state. Hostile individuals may drive people away or may not accurately perceive others’ affiliative intentions. Individuals lacking the perception that support is available may be lonely and depressed as a result. An expanding body of research suggests that these putative psychosocial risk factors do covary empirically (10–12); indeed, it has been hypothesized that an underlying characteristic of negative affectivity (13) may also be shared among individuals who are depressed, hostile, and lack support.

A number of twin studies have examined the genetic and environmental determinants of each of these psychosocial characteristics. For clinical diagnoses of depression, Moldin et al. (14) reviewed six early twin-proband studies and found that despite the variety of operational definitions of clinical depression, concordance rates for monozygotic (MZ) twins (ie, identical twins sharing 100% of genes) were significantly higher than rates for dizygotic (DZ) twins (ie, fraternal twins sharing on average 50% of genes) in each study. More
recently, Kendler et al. (15) reported a twin study with 1033 female pairs in the community (mean age = 30.1 years) in which 33% to 45% of the variation in liability to major depression, based on seven diagnostic approaches, was found to be heritable (ie, percentage of variation in liability to depression attributable to genetic variation). Lyons et al. (16), in their large community-based twin study of major and less severe depression among 3372 male pairs (mean age = 44.6 years), also found significant heritability (36%) for liability to major depression, but less severe forms of depression were affected by environmental factors only.

Although there is a body of evidence for the heritability of depression as assessed by diagnostic criteria, less is known about the heritability of depressive symptomatology as measured by continuous self-report scales. Wierzbicki (17) conducted a study with 92 twin pairs (mean age = 37.1 years) and found significant heritabilities for two Minnesota Multiphasic Personality Inventory (MMPI) indices of depression (41–43%) but nonsignificant heritabilities for the Beck Depression Inventory (BDI, Ref. 18; 23%) and the MMPI Depression scale (28%). Fogue-Geile and Rose (19) conducted a 4-year prospective study with 133 twin pairs (mean age at baseline = 20.3 years) and found significant heritability at both time points for the MMPI D30 scale (43% and 61%, respectively). Silberg et al. (20) conducted a community-based study of 771 female twin pairs (mean age = 31.2 years) and found that 13% of the variation in depressive symptoms could be accounted for by a single genetic factor. In sum, both categorical and continuous measures of depression seem to be genetically influenced, but that genetic variation usually accounts for less than half of the total variation.

The behavioral genetics literature on hostility has produced variable results. In two studies of adult male twins with adequate sample sizes (Carmelli et al. (21), 261 twin pairs, mean age ≈ 65 years; Smith et al. (22), 121 twin pairs, mean age = 41.7 years), there was evidence of a significant genetic contribution to a commonly used index of hostility, the Cook-Medley Hostility (Ho) Scale (23), with estimated heritabilities ranging from 28% to 64%, respectively. Similarly, in a large sample of 410 young adult twins pairs (ages 14–34 years, 58% female), Rose (24) reported a significant heritability estimate of 34% across sexes for the Cynicism factor of the MMPI; in a study of 160 male twin pairs (mean age = 48 years), Matthews et al. (25) reported that concordance rates of interview-based potential for hostility were significantly higher among MZ than among DZ twin pairs.

In contrast, in a sample of 700 Swedish twin pairs (mean age = 58.6 years, 60% female), Pedersen et al. (26) did not find significant genetic effects using a translated subsample of Ho scale items. Nonsignificant heritability for the Ho scale was also reported for a sample of 98 female twin pairs (mean age = 45 years), although three subscales of the Buss-Durkee Hostility Inventory showed evidence of heritability ranging from 70% to 98% (27). Taken as a whole, these findings suggest a small or moderate genetic contribution to individual differences in hostility, perhaps most clearly among middle-aged American men.

Three twin studies have also investigated social support. Kessler et al. (28) studied eight subjective and objective dimensions of social support (29) in a sample of 821 female twin pairs (mean age = 28.9 years) and found significant genetic effects for a majority of the social support dimensions, explaining between 28% and 52% of the variance. Bergeman et al. (30), using a combined twin/adoption study design with 424 Swedish twin pairs (age 50 years and older, 62% female), examined the Interview Schedule for Social Interactions (31) and found that genetic factors accounted for 30% of the variance in subjective support and zero percent of the variance in objective support. Lastly, Kendler (32) conducted a prospective twin study using a 16-question interview of subjective and objective social support in a sample of 851 twin pairs (mean age = 34.6 years). All six indices of support were significantly influenced by genetic factors in this study, with heritability estimates ranging from 43% to 75%. In sum, these findings suggest that subjective, and to a lesser extent objective, measures of social support are genetically influenced.

Although there is a growing literature examining the genetic and environmental determinants of depression, hostility, and social support as individual characteristics, to our knowledge only one published study has investigated the causes of covariation among these factors. Bergeman et al. (33) examined the determinants of the association between depression and social support (r = .20) with the same sample used in the previously described Bergeman et al. study (30). Using structural equation modeling, they found that genetic influences accounted for 65% of the covariation between depression and social support and that nonshared environmental influences (ie, environments uncorrelated between twins, such as accidents) accounted for 35%. This study suggests that environments shared in common by twins, such as familial socialization, do not meaningfully affect the relationship between depression and social support. Clearly there is a scarcity of published investigations assessing genetic and/or environmental mediation among two or more putative psychosocial risk factors for CVD.
The current study used a twin design to examine the relative contributions of genetic and environmental influences to the variation and covariation of depression, hostility, and social support during young adulthood. Whereas a body of research already exists on the determinants of these individual characteristics, this is one of the first studies to examine genetic and environmental influences on their covariation as well. Finding evidence of common genetic or environmental influences on the covariation of these characteristics would raise questions about the distinctiveness of these psychosocial constructs (eg, Ref. 34) and their associations with disease. The following specific questions were addressed in this study: 1) Can the phenotypic covariation among depression, hostility, and social support be explained by a single latent factor? 2) What are the relative contributions of genetic and environmental influences to each putative risk factor individually? 3) What are the relative contributions of genetic and environmental influences to the covariation among pairs of these measures? 4) Can the covariation among all three phenotypic measures be explained by single common latent genetic and/or environmental factors?

METHODS

Participant Ascertainment

The data reported here were collected as part of a larger study, the Pittsburgh Twin Study, designed to investigate genetic and environmental influences on cardiovascular and psychopathological risk factors and neuropsychological functioning (35–37). Young adult (18–30 years old), male and female, same-sex twin pairs were recruited using driver’s license, voter registration, and student registration records as well as some advertisements. Individuals with the same last name, sex, and date of birth on these lists were contacted by mail about their twin status and for permission to be telephoned. Those responding affirmatively were screened over the telephone for the following exclusion criteria: diagnoses of CVD or hypertension, obesity, cancer, kidney disease, Type I diabetes, HIV, and current use of cardiovascular, psychotropic, or glucocorticoid medications or illegal drugs. If both members of the pair passed these criteria, they were each scheduled for two laboratory sessions. In general, co-twins were assessed within 1 week of each other.

Procedure

On the participants’ arrival at the first session, the study was reviewed and informed consent was obtained. At this time blood was drawn for subsequent zygosity diagnosis (MZ or DZ) using DNA fingerprinting. The first afternoon laboratory session involved cardiovascular monitoring, and the second afternoon session included computerized self-report questionnaires and neuropsychological testing. Participants were paid $100 on completion of the study protocol.

Measures

The questionnaires used in the current study were part of a larger battery of computer-administered self-report tests. The BDI (18), Ho scale (23), and Interpersonal Support Evaluation List (ISEL) (38) were used to index the three constructs of depression, hostility, and social support, respectively. These measures have been used extensively in community samples and have satisfactory test-retest and internal consistency reliabilities. Several commonly used personality measures were also administered, including short forms of the Eysenck Neuroticism and Extraversion scales (39); NEO-Agreeableness, NEO-Openness to Experience, and NEO-Conscientiousness (40) scales; Spielberger Trait Anger (41) and Trait Anxiety (42) scales; Life Orientation Test (43); and Marlowe-Crown Social Desirability scale (44).

RESULTS

Sample Description

The final sample consisted of 464 individuals, composed of 157 MZ twin pairs (77 female, 80 male) and 75 same-sex DZ twin pairs (42 female, 33 male). To be included in analyses, all participants, along with their respective co-twins, were required to have complete data for all three scales. Twin pairs with either member missing one or more of the three scales (N = 13) were excluded from analyses. The average age of participants was 20.94 years (SD = 2.8), and the ethnic composition was 93.1% European American (N = 432), 5.2% African American (N = 24), 1.3% Asian American (N = 6), and less than 1% other ethnic groups (N = 2).

Preliminary Analyses

Each scale score was checked for outliers, normality, and associations with demographic characteristics. No outliers were apparent. To minimize skewness and kurtosis, the BDI and Ho scores were square root transformed, and the ISEL scores were squared. The skewness and kurtosis values associated with the resulting measures were well within acceptable ranges (skew < 3.0 and kurtosis < 8.0; Ref. 45). Therefore, all subsequent analyses made use of these transformed scores. Means and standard deviations for the raw scale scores were as follows: BDI, mean = 5.46, SD = 7.45; Ho, mean = 21.89, SD = 8.51; and ISEL, mean = 107.57, SD = 15.74. The Ho and ISEL scales were significantly correlated with age (r = −.11 and r = −.14, respectively) and sex (r = .17 and r = −.20, respectively; female = 1, male = 2); the BDI, Ho, and ISEL scales were all significantly associated with ethnicity (r = .19, r = .14, and r = −.16, respectively; European American = 1, other = 2). Given these associations, subsequent analyses for all three scales were based on standardized residuals from regression analyses of the
transformed scores with age and ethnicity entered as covariates.

Phenotypic Confirmatory Factor Analysis

As expected, the BDI and Ho scales were moderately and positively correlated (males: $r = .37$, females: $r = .49$), whereas the ISEL was negatively correlated with both the BDI (males: $r = -.39$, females: $r = -.40$) and Ho (males: $r = -.28$, females: $r = -.28$) measures, with intercorrelations being similar for males and females. Using the Mx structural equation modeling program (46), a phenotypic confirmatory factor analysis (CFA) was performed on these scales using separate variance/covariance matrices for males and females. The CFA model tested included a single common latent factor and independent specific factors for each of the three scales. Initially, different factor loadings were allowed for males and females. This model is “just-identified,” meaning that there are as many estimated parameters (12) as there are observed variances and covariances (12) and thus no degrees of freedom to evaluate model fit. Next, model parameters were equated for males and females, and this model fit quite well: $\chi^2(6) = 3.92, p = .69$ (i.e., a nonsignificant $\chi^2$ value indicates adequate model fit or no significant difference between model-predicted and observed variances and covariances). The percentages of variance (i.e., square of standardized factor loadings) accounted for in each of the measures by both common and specific factors in the model are presented in Figure 1. All of the paths were significant in that none could be dropped from the model without significantly reducing model fit. Therefore, this model suggests that a single common factor can explain much of the observed scale correlations among these three variables and that this structure does not differ across sexes.

To describe further this common factor, correlations between the factor score, estimated using multiple regression (47), and several frequently used personality measures were examined. As might be expected, this common factor score was related in the same direction to several other measures with positive valence, including the Eysenck Neuroticism Short Form ($r = .54, p < .001$), Spielberger Trait Anger ($r = .43, p < .001$), and Spielberger Trait Anxiety ($r = .66, p < .001$); the common factor score was associated in the opposite direction with measures with positive valence, including the Life Orientation Test ($r = -.58, p < .001$), Marlowe-Crown Social Desirability ($r = -.31, p < .001$), NEO-Agreeableness ($r = -.27, p < .001$), NEO-Conscientiousness ($r = -.24, p < .001$), and Eysenck Extraversion Short Form ($r = -.33, p < .001$). Finally, the common factor score was weakly correlated with NEO-Openness to Experience ($r = .10, p > .05$).

Twin Analyses

Twin model-fitting analyses were used to estimate genetic and environmental influences on variation and covariation among the three psychosocial measures. This approach is based on the premise that MZ twins share 100% of genes and DZ twins share on average 50% of genes identical by descent. The twin model-fitting analyses partition the variance and covariances of the self-report measures into that due to latent factors: additive genetic (A), nonadditive or genetic dominance effects (D), common or shared environment (C; e.g., family environment), and nonshared environment (E; e.g., individual experiences, and measurement error). Correlations of these factors between MZ twins are as follows: $A$, 1.0; $D$, 1.0; $C$, 1.0; and $E$, 0.0. Correlations between DZ twins are $A$, 0.5; $D$, 0.25; $C$, 1.0; and $E$, 0.0. The squared path coefficients between these latent factors and the observed measures represent partitions of variance, that is, parameters $a^2$, $d^2$, $c^2$, and $e^2$. The ACE model includes three parameters, and the significance of each is tested by comparing the reduced models (AE, CE, and E) to the full ACE model to determine whether the removal of one or more pa-
rameters significantly increases the $\chi^2$ goodness-of-fit statistic and thus decreases model fit.

Nonadditive genetic, or dominance, effects (D) are tested by comparing the AE to an ADE model. Dominance effects are defined as nonadditive effects and thus cannot be estimated without also estimating additive effects (A). Shared environment (C) and dominance (D) effects cannot be estimated in the same model because they are negatively and perfectly correlated. Therefore, for a variable in which D was not significant, parameter estimates were based on the ACE model. For a model in which D was significant, the parameter estimates were based on the ADE model.

This twin model-fitting approach assumes that assortative mating, epistasis, and gene-environment interactions or correlations are minimal. In addition, it assumes common environments do not differ between MZ and DZ twin pairs in ways relevant to the psychosocial characteristics (for an introduction to behavioral genetics, see Ref. 48; for detailed information on twin statistical methodology, see Ref. 49). The Mx structural equation modeling program (46) was used to fit these etiological models to the twin variance-covariance matrices.

Although MZ and DZ twins did not significantly differ in sex or ethnicity, MZ twins were significantly younger (mean = 20.50 years, SD = 2.63) than DZ twins (mean = 21.80 years, SD = 3.05) ($t(462) = 17.14$, $p < .001$). There were no mean differences between MZ and DZ twins on the BDI, Ho, or ISEL scales ($p$ values $>.10$). As is usually found, MZ twin pairs reported greater degree of current contact (ie, number of months lived with co-twin in past year) than DZ twin pairs (mean = 8.12 vs. 5.74 months) ($t(219) = 6.16$, $p < .001$). However, partial correlations between degree of pair contact and twin resemblance for BDI, Ho, and ISEL scales (absolute pair difference) controlling for zygosity were small and nonsignificant ($r$ values = -.02 to -.12, $p$ values $>.10$), suggesting no violation of the equal environmental covariance assumption.

**Univariate Twin Analyses**

Univariate twin analyses were used to estimate genetic and environmental influences for each of the three measures separately. For each characteristic, different parameters were initially allowed for males and females, and then model parameters were equated across sexes. There were no significant differences between the models permitting different estimates for males and females and the models equating males and females, for any of the three measures.

Table 1 presents twin correlations and partitions of variance ($a^2$, $d^2$, $c^2$, and $e^2$) for each measure for the models equating parameters across sexes. For the BDI and ISEL scales, MZ twin correlations were substantially larger than the correlations between DZ twins, suggesting genetic effects. For the Ho scale, the correlation for DZ twins approached the correlation among MZ twins, raising the possibility of shared environmental contributions. Because the DZ correlations were less than one half of the MZ correlations for BDI and ISEL, genetic dominance is possible.

The overall model fits were satisfactory for the BDI, Ho, and ISEL scales, indicating minimal discrepancy between model-predicted vs. observed variances and covariances. The BDI and ISEL scales both evidenced significant additive genetic effects (A), as indicated by a significant increase in $\chi^2$ between ACE and CE models (ie, reduced model fit without parameter A), whereas the Ho scale did not. Shared environmental factors (C) were not significant for any scale (AE model vs. ACE model fit). In contrast, nonshared environmental factors (including measurement error, E) were significant for all three measures (AC model vs. ACE).

For the Ho scale, either additive genetic (A) or shared environmental effects (C) could be omitted from the ACE model without a significant decrement in model fit, but dropping both A and C together resulted in a significantly poorer fit. This indicates that there exists a familial effect of some nature, but the

<table>
<thead>
<tr>
<th>Variable</th>
<th>Twin Correlations</th>
<th>Parameter Estimates</th>
<th>Model Fit</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$R_{MZ}$</td>
<td>$R_{DZ}$</td>
<td>$a^2$</td>
</tr>
<tr>
<td>BDI</td>
<td>.44*</td>
<td>.13</td>
<td>.43b</td>
</tr>
<tr>
<td>Ho</td>
<td>.49*</td>
<td>.34*</td>
<td>.24*</td>
</tr>
<tr>
<td>ISEL</td>
<td>.61*</td>
<td>-.02</td>
<td>.00b</td>
</tr>
</tbody>
</table>

* Monozygotic pairs = 157; dizygotic pairs = 75.

b $\chi^2$ increased significantly ($p \leq .05$) compared with full model when parameter was dropped, indicating its significant contribution to model.

c For the Ho scale, neither A nor C were significant individually (CE vs. ACE, $\chi^2(1) = 1.22$, $p > .30$ and AE vs. ACE, $\chi^2(1) = 1.05$, $p < .40$, respectively). However, dropping A and C together resulted in significantly poorer fit (E vs. ACE, $\chi^2(2) = 50.42$, $p < .001$). This indicates that there exists a familial effect of some nature, but its nature (A and/or C) cannot be resolved statistically.

d NE indicates that parameters were not estimated. BDI and Ho parameter estimates and $\chi^2$ values are based on the ACE model, whereas the ISEL parameter estimates are based on the ADE model because of its significant A and D effects (CE vs. ACE, $\chi^2(1) = 19.85$, $p < .001$ and AE vs. ACE, $\chi^2(1) = 4.85$, $p < .05$, respectively).

* $p < .0005$ (one-tailed).
type of contribution (genetic and/or shared environmental) cannot be detected statistically. Parameter estimates from the full ACE model suggest approximately equivalent, relatively small effects for both genetic and shared environmental influences on the Ho scale.

Significant genetic dominance effects (D) in addition to additive effects (A) were detected for the ISEL scale, as indicated by a significant decrease in fit for the AE compared with the ADE model, but not for the BDI scale. Interestingly, in parameter estimation with the ADE model, all of the genetic variance was attributed to genetic dominance (D) and none was attributed to additive effects (A) for the ISEL. Like genetic dominance, twin competition effects (50) in which twins negatively influence one another, also may produce lower-than-expected DZ correlations. However, twin competition effects also produce increased total variance for MZ compared with DZ twins. Because no such significant effect was observed, it is unlikely that twin competition effects play a major role for ISEL.

Bivariate Twin Analyses

Table 2 presents the maximum-likelihood estimates

<table>
<thead>
<tr>
<th>Scales</th>
<th>r_{phenotypic}^d</th>
<th>r_a^d</th>
<th>r_e^d</th>
<th>ara^e</th>
<th>ere^e</th>
</tr>
</thead>
<tbody>
<tr>
<td>BDI and Ho</td>
<td>.43</td>
<td>.58**</td>
<td>.31**</td>
<td>61%</td>
<td>39%</td>
</tr>
<tr>
<td>BDI and ISEL</td>
<td>-.40</td>
<td>-.50**</td>
<td>-.31**</td>
<td>62%</td>
<td>38%</td>
</tr>
<tr>
<td>Ho and ISEL</td>
<td>-.29</td>
<td>-.38**</td>
<td>-.17*</td>
<td>72%</td>
<td>28%</td>
</tr>
</tbody>
</table>

- Monozygotic pairs = 157; dizygotic pairs = 75.
- Parameter estimates for the BDI-Ho, BDI-ISEL, and Ho-ISEL models are based on bivariate AE Cholesky models without C or D parameters, none of which were significant. For BDI and Ho: significant A (CE vs. ACE, Δχ^2(3) = 7.84, p < .05); nonsignificant C (CE vs. ACE, Δχ^2(3) = 2.28, p < .70); nonsignificant D (AE vs. ADE, Δχ^2(3) = .81, p < .80). For BDI and ISEL: significant A (CE vs. ACE, Δχ^2(3) = 24.72, p < .001); nonsignificant C (CE vs. ACE, Δχ^2(3) = 0.0, p < .99); nonsignificant D (AE vs. ADE, Δχ^2(3) = 5.62, p < .20). For Ho and ISEL: significant A (CE vs. ACE, Δχ^2(3) = 19.35, p < .001); nonsignificant C (CE vs. ACE, Δχ^2(3) = 1.85, p < .70); nonsignificant D (AE vs. ADE, Δχ^2(3) = 5.24, p < .20). The AE bivariate models all provided adequate fit to the data (p values > .40).
- Maximum likelihood estimates of phenotypic correlations, which may differ somewhat from singletons’ Pearson correlations.
- r_a represents additive genetic correlation; r_e represents nonsquared environmental correlation. Significance represents a significant decrease in χ^2 model fit when the relevant covariance parameter is dropped from the AE Cholesky model.
- Standardized ara and ere are the percentages of phenotypic covariance due to additive genetic and nonshared environmental effects, respectively.
- * p < .05, ** p < .001.

of phenotypic correlations, genetic and environmental correlations, and percentage of phenotypic covariance due to additive genetic and nonshared environmental factors. These bivariate twin analyses were performed to determine the effects of common genetic, shared environmental, and nonshared environmental factors on phenotypic covariation between pairs of measures. Given that univariate analyses revealed no significant differences between males and females, all bivariate and multivariate (see below) analyses are based on the combined sample of males and females with sex, age, and ethnic group as covariates.

Despite the significant univariate genetic dominance effect for the ISEL scale (in addition to additive genetic effects) and the nonsignificant trend for shared environmental effects on the Ho scale, neither C nor D contributed significantly to the covariation of the psychosocial characteristics. As detailed in Table 2 (footnote b), there were no significant reductions in model fit between the AE and ACE or AE and ADE bivariate models for any pair of measures. Such findings should not be unexpected because it is not necessary that effects that contribute to measures univariately must contribute to covariation with other measures. In contrast, A effects were significant for each pair of measures, as indicated by significant reductions in fit for CE vs. ACE bivariate models. Thus, only additive genetic and nonshared environmental factors are included in Table 2.

First, Table 2 presents maximum-likelihood phenotypic correlations calculated from the twin models, which, similar to the Pearson correlations among singletons, suggested a moderate degree of covariation among the BDI, Ho, and ISEL scales. Next, genetic and environmental correlations (r_a, r_e) indicate the degree to which two variables are affected by the same additive genetic or nonshared environmental factors, respectively (48). In contrast, the percentage of the phenotypic covariation attributable to common additive genetic or nonshared environmental factors is calculated by taking the product of the individual genetic or environmental influences and the correlation between these influences and dividing it by the phenotypic covariation. For instance, ara is computed by multiplying the square root of the additive effects of two measures by the correlation between these genetic effects ([a_1 * r_a * a_2]/cov_{phenotypic}). These correlations and the percentages of phenotypic covariation differ in that the former do not include information about the degree of genetic (a^2) or environmental (e^2) influences. For instance, r_a could be large even if heritabilities are low (ie, small genetic effects on two variables that highly overlap), or r_a could be small even though heritabili-
ties are high (ie, large but totally distinct genetic influences for the two variables) (51).

As seen in Table 2, the additive genetic correlations between each pair of measures were significant between \( r_g \) values ranged from .58 to -.38), indicating that common genetic influences were approximately equal to genetic influences specific to each variable. The nonshared environmental correlations were all significant but smaller \( r_e \) values ranged from .31 to -.17), indicating that environmental influences tended to be more specific to these measures than common between them. The observed phenotypic correlations were largely attributable to additive genetic influences \( (ar) \) ranged from 72% to 61%) and less so to common nonshared environmental influences \( (er) \) ranged from 39% to 28%).

Multivariate Twin Analyses

Similar to the phenotypic CFA described above, multivariate twin analyses were used to determine whether covariation among the three putative psychosocial risk factors could be explained more parsimoniously by one or a few latent factors. However, these twin analyses extend the phenotypic CFA by assessing the degree to which the covariation among the measures can be explained by genetic and/or environmental factors that are common across measures in addition to genetic and/or environmental influences that are measure specific.

The model tested was the “biometric common factor model.” The full model posits that all covariation among the three measures is explained by single factors (A, D, C, E), each of which is common across the three psychosocial measures. Variability unique to each measure is accounted for by specific influences (A, D, C, E) (see Ref. 49). A full ACE common factor model was consistent with the observed data, indicating that single common additive genetic and environmental effects along with specifics can account for variation and covariation among the BDI, Ho, and ISEL scales (Table 3). A biometric common factor model including ADE common and specific factors was also tested because of genetic dominance effects initially detected with the ISEL. Similar to the ACE model, this ADE full common factor model was also consistent with the observed data.

To ascertain a more parsimonious account of the covariation of BDI, Ho, and ISEL scales, reduced sub-models of the ACE common factor model were tested. When all common and specific shared environmental influences were omitted, there was no significant loss of model fit in comparison to the full ACE model, indicating that shared environmental factors do not significantly influence overall the variation or covariation among the three psychosocial measures. Similarly, when comparing the full AE common factor model against a model including all common and specific genetic dominance influences (the full ADE model), there was no significant decrement in model fit. Therefore, genetic dominance effects also do not significantly influence overall the variation or covariation among the three psychosocial measures. In contrast, exclusion of either the common genetic factor or the common nonshared environmental factor resulted in significant loss of fit as compared with the full ACE model. These results suggest that additive genetic and nonshared environmental factors significantly influence covariation among the three measures.

<table>
<thead>
<tr>
<th>Model for BDI, Ho, and ISEL</th>
<th>Goodness of Model Fit</th>
<th>( \chi^2 )</th>
<th>df</th>
<th>( p )</th>
<th>AICb</th>
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<tbody>
<tr>
<td>2. Biometric common factors ADE model</td>
<td>19.94</td>
<td>24</td>
<td>.700</td>
<td>-28.06</td>
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<tr>
<td>3. Biometric common factors AE model (see Figure 2)</td>
<td>25.42</td>
<td>30</td>
<td>.705</td>
<td>-34.58</td>
<td></td>
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<tr>
<td>4. Biometric common factors ACE model without common A factor</td>
<td>38.53</td>
<td>27</td>
<td>.070</td>
<td>-15.47</td>
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<tr>
<td>5. Biometric common factors ACE model without common E factor</td>
<td>56.01</td>
<td>27</td>
<td>.001</td>
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</tbody>
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Questions Model Comparisons Tests of Parameter Significance

<table>
<thead>
<tr>
<th>Question</th>
<th>Model</th>
<th>( \Delta \chi^2 )</th>
<th>df</th>
<th>( p )</th>
</tr>
</thead>
<tbody>
<tr>
<td>A. Any common or specific shared environmental effects?</td>
<td>No</td>
<td>3 vs. 1</td>
<td>3.95</td>
<td>6</td>
</tr>
<tr>
<td>B. Any common or specific dominance genetic effects?</td>
<td>No</td>
<td>3 vs. 2</td>
<td>5.48</td>
<td>6</td>
</tr>
<tr>
<td>C. Any common additive genetic effects?</td>
<td>Yes</td>
<td>4 vs. 1</td>
<td>17.06</td>
<td>3</td>
</tr>
<tr>
<td>D. Any common nonshared environmental effects?</td>
<td>Yes</td>
<td>5 vs. 1</td>
<td>34.54</td>
<td>3</td>
</tr>
</tbody>
</table>

*Tests of individual measures’ specific genetic and environmental paths from the AE model are not shown.

b Akaike’s Information Criteria (lower values indicate more parsimonious models).
All subsequent comparisons were nested within and tested against the AE common factor model. Specific genetic and nonshared environmental factors also significantly influenced the Ho and ISEL measures when tested individually in comparison with the AE model. Specific genetic and nonshared environmental factors were not significant for the BDI. Therefore, the most parsimonious model underlying the covariation of the BDI, Ho, and ISEL scales was composed of common genetic and nonshared environmental factors as well as genetic and nonshared environmental influences specific to the Ho and ISEL scales (Figure 2).

The relative influences of common vs. specific factors in the AE common factor model were derived by summing the squared path coefficients (ie, percentages of variances; Figure 2). For the Ho and ISEL scales, the relative influence of measure specific factors (27% + 42% = 69% and 40% + 35% = 75%, respectively) exceeded that of common factors (22% + 9% = 31% and 18% + 7% = 25%, respectively). In contrast, common factors (64%) accounted for a greater proportion of variability in the BDI scale than did specific factors (36%). Similar comparisons indicate that the majority of variance common across variables was due to genetic factors (BDI = 52% [eg, 33% + (33% + 31%) = 52%], Ho = 71%, and ISEL = 72%) with a smaller contribution of nonshared environment (BDI = 48%, Ho = 29%, and ISEL = 28%).

DISCUSSION

The goal of this study was to examine the influence of genetic and environmental factors on three putative psychosocial risk factors for CVD—depression, hostility, and social support—in young adults and to explore the extent to which these factors may be common across two or more of these psychosocial characteristics. The results showed the following:

1. The BDI, Ho, and ISEL scales were moderately intercorrelated, and a single latent phenotypic factor could account for this observed covariation.
2. The BDI was influenced by additive genetic effects, and the ISEL scale was primarily affected by genetic dominance; both scales were also influenced by nonshared environment but not shared environmental factors. The Ho scale showed a familial effect that could not be resolved between additive genetic and/or shared environmental influences.
3. The moderate phenotypic correlations between each pair of these measures were primarily attributable to common genetic factors and, to a smaller degree, common nonshared environment.
4. At the multivariate level, the moderate covariation of the BDI, Ho, and ISEL scales could be explained by single common genetic and common nonshared environmental factors.

Phenotypic Analyses

Few published reports have included factor analyses of these putative psychosocial risk factors for CVD (cf, Ref. 52), and to the best of our knowledge none have performed CFA on the constructs of depression, hostility, and social support. Although the variance shared among these characteristics is relatively modest, the present CFA confirmed that the intercorrelations among the BDI, Ho, and ISEL scales could be explained by common latent factors and factors specific to each of the measures. No significant shared environmental or genetic dominance factors were significant. Significance indicates a significant increase in model $\chi^2$ value if the parameter is omitted compared with the full AE model. $^*p < .005$, $^{**}p < .001$.

![Figure 2](image-url)
accounted for by a single factor. The ability to fit a single factor suggests important commonality among the measures that may improve the prediction of disease risk (ie, factor scores may possess increased reliability due to aggregation and reduction of error variance). However, it is also possible that variances specific to each of the psychosocial characteristics are the best predictors of disease risk. These and other data recommend that future studies of disease risk use a multivariate strategy to address this important issue.

It is noteworthy that the factor scores describing the common variance for these measures correlated in the same direction as those traits that are socially undesirable (eg, neuroticism, anger, and anxiety) and inversely with those traits that are socially desirable (eg, optimism, agreeableness, conscientiousness, and extraversion), albeit with magnitude of associations generally small to moderate (r values = .24–.66). The moderate association between the factor scores and neuroticism (r = .54) is of particular interest given the controversy surrounding the extent to which neuroticism or negative affectivity is thought to confound the relationships between self-reported individual dispositions and health outcomes (13, 53). Given that each of the three psychosocial constructs contributes to CVD outcomes and their underlying factor shares variance in common with neuroticism, the assumption that neuroticism is not an important health determinant may be questioned. This debate is yet to be resolved, and it is hoped that future research on disease will shed more light on the extent to which variance common to the latent factor and neuroticism (25% in this sample) predicts disease risk.

Univariate Analyses

The present analyses indicated that 43% of the variance in the BDI was attributable to genetic influences, which is higher than the nonsignificant heritability estimate of 23% reported by Wierzbicki (17), the only published twin study of the BDI to our knowledge. A possible explanation for the discrepancy of findings is sampling variation because Wierzbicki’s (17) sample was relatively smaller (92 twin pairs), older (mean age = 37.3 years), and predominantly female (78%). In contrast, our results for the BDI are similar to those of several studies that have examined other questionnaire measures of depression (17, 19, 20). Moreover, the present results indicate that shared environmental influences did not influence the variance of BDI scores, which is consistent with results of studies examining the BDI and other questionnaire measures of depression (19, 20).

Unlike the significant heritability estimates reported by Smith et al. (22) and Carmelli et al. (21), the present study’s univariate analyses did not detect significant genetic influences on the Ho scores, although significant familiality was apparent. However, comparison of the actual heritability estimates across studies reveals a generally consistent pattern (present study, h² = 24%; Carmelli et al. (21), h² = 28%; Pederson et al. (26), h² = 21%; Cates et al. (27), h² = 34%; and Rose (24), h² = 34%), with the exception of the estimate (h² = 64%) reported by Smith et al. (22), which was substantially higher. This discrepant finding by Smith et al. may be attributable to the uniqueness of their sample, which was the only one composed entirely of middle-aged men (mean age = 41.7 years). Taken together, these results indicate that genetic influences may make a small contribution to the variability of the Ho scale, but these may be statistically significant only some of the time.

The effects of shared environmental influences on the Ho scores could not be statistically resolved in the present study. Although the other twin studies examining the full Ho scale did not use twin-model fitting analyses (21, 22, 27), we estimated shared environmental influences using twin correlations presented in the articles (c² = 2rDZ − rMZ) and found them to be minimal (Carmelli et al. (21), c² = 2%; Cates et al. (27), c² = 1%; Smith et al. (22), c² = 17%). Rose (24) reported that the effect of shared environmental influences on the Cynicism factor of the MMPI was small (17%) and nonsignificant, whereas Pedersen et al. (26) found a small but significant shared environmental contribution (20%) to a reduced version of the Ho scale. In sum, the few available results indicate that environmental influences shared by family members may contribute to variation in hostility during adulthood, albeit minimally at most.

Although the ISEL has not been used in other behavioral genetics studies of social support, these results are generally comparable to those of previous studies of social support using other self-report measures (28, 30, 32). That is, the present results indicate that 59% of the variance of ISEL scores was attributable to genetic factors, whereas the other studies reported that genetic factors explained 28% to 75% of the variance in other self-report and interview measures. Although Kessler et al. (28) reported significant genetic dominance effects for a single question concerning the availability of a confidant, seven other indices of social support were not influenced by genetic dominance effects in that study. Bergeman et al. (30) did not find evidence for genetic dominance effects on social support, and Kendler (32) did not present data for this effect. Therefore, the present study is the first to report evidence of significant genetic influences on social support.
geneic dominance effects in addition to additive effects using a full-scale measure of social support. The present results also indicate that shared environmental influences did not influence the variance of ISEL scores, which is consistent with findings of other studies of social support.

With the possible exception of the Ho scale, there was little support for the contribution of environmental factors shared within twin pairs to the psychosocial characteristics examined here. This finding is especially interesting given the developmental stage of the sample. That is, twin pairs in young adulthood have lived together more recently than older twin pairs and therefore should be expected to have in common more recent experiences (54). Given the lack of observed shared environmental effects at this young age, it would be difficult to expect that shared family experiences germane to these psychosocial characteristics would have any additional impact as these individuals aged into middle or late adulthood.

Bivariate Analyses

Phenotypic correlations between the characteristics were moderate ($r$ values $= .28-.43$). Bivariate twin analyses showed that the correlations between genetic factors were moderate ($r_g$ values $= .38-.58$), whereas the correlations between nonshared environmental factors were smaller ($r_e$ values $= .17-.31$). These analyses also indicated that the moderate phenotypic correlations were due largely to genetic influences ($ara = 61-72\%$) and, to a lesser degree, nonshared environmental influences ($ere = 28-39\%$). These findings are somewhat discrepant from those of the only other study with comparable bivariate analyses (33). The additive genetic and nonshared environmental correlations between the BDI and ISEL scales in the present study were .50 and .31, respectively, whereas the additive genetic and nonshared environmental correlations among other measures of depression and social support reported by Bergeman et al. (33) were 0.13 and 0.07, respectively. These differences may be due in part to differences between the samples, because the Bergeman et al. (33) sample was composed of Swedish older adults. Also, the self-report measures varied; the Bergeman et al. (33) study used nine translated items of the Interview Schedule of Social Interaction (31) to index social support and a scale based on factor analyses of 15 mental health items from the OARS' Short Psychiatric Evaluation Schedule (55) to index depression.

Although there was evidence of significant genetic dominance effects along with additive effects for the ISEL scale in the univariate analyses, no significant dominance effects were detected in the bivariate analyses. This may be because dominance effects did not contribute at all to covariation between the ISEL and other measures or perhaps because the relatively small overall dominance effects were split between ISEL specific and common effects and thus were not statistically detectable. In contrast, common additive genetic effects were significant for each bivariate analysis. Given the presence in the univariate analyses of significant additive genetic effects for two of the three measures (BDI and ISEL) and a nonsignificant trend for the third (Ho), this should not be unexpected.

Implications

The present findings have implications for a range of hypotheses—psychological, biological, and etiological—predicting covariation of psychosocial factors associated with CVD risk. As mentioned previously, several plausible psychological explanations are available to explain covariation of these psychosocial characteristics. Watson and Pennebaker (13) have suggested that negative affectivity is a psychological characteristic underlying various measures of distress. Similarly, cognitive theorists like Beck et al. (56) and Ellis (57) have hypothesized that depressed individuals have maladaptive thinking patterns, which are exemplified by cynically hostile beliefs (eg, others can never be trusted or the world is threatening). It has also been posited that poor social support may increase susceptibility to depression and also be a consequence of depression (58). Smith and Anderson (59) have likewise described an interactive process in which hostile individuals may elicit aversive reactions from potential sources of social support, which in turn confirm their cynical beliefs. A recent hypothesis that also includes biological predictions postulates a “hostility syndrome” in which health-damaging psychosocial (ie, hostility, depression, social isolation, job strain), behavioral (ie, physical inactivity, high-calorie or
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high-fat diet, smoking, alcohol consumption), and physiological (ie, sympathetic nervous system and HPA axis hyper-reactivity) characteristics correlate as a result of reduced serotonergic function in the central nervous system (60–63). At an etiological level, the covariation of negative psychosocial characteristics has often been attributed to environmental experiences, such as ineffective parental rearing practices (64), negative peer influence (65), and racial discrimination (66). Socioeconomic status (SES) has also been of interest here because it has been shown that low SES is associated with many negative emotions, risk for later health problems (67), and even diminished central serotonergic responsivity (68). Indeed, the hostility syndrome model has been elaborated recently to suggest that reduced central serotonergic activity is caused in part by environmental experiences associated with low SES, particularly in childhood (63).

Evidence from the present study indicating that phenotypic covariation of depression, hostility, and social support measures may be explained by a single latent factor is consistent with all of the above psychological and biopsychosocial hypotheses that predict that these psychosocial traits should correlate. At an etiological level, however, the absence of any detectable effects of shared environmental factors on variation (with the possible exception of the Ho scale) or covariation of the three measures in this study tends to argue against a major causal role for experiences that are highly correlated among family members, such as parental rearing and SES in childhood. That is, because childhood SES, for example, is highly correlated within both MZ and DZ twin pairs, any causal effect of such experiences should lead to increased resemblance for both MZ and DZ twins and thus would contribute to estimates of shared environmental influence. However, in the absence of a completely representative sample, it is possible that the effects of SES extremes are underestimated in the current study. In contrast, the current data emphasize the importance of genetic variation in the covariation among putative risk factors. Perhaps this genetic variation affects serotonergic function, as implied by the hostility syndrome model, although nonserotonergic factors could also be important. Thus, the current findings suggest that future research should emphasize investigations of the extent to which common genetic and nonshared environmental influences underlie covariation among these putative psychosocial risk factors for CVD.

Limitations

One potential limitation of the present study is its reliance on self-report questionnaires, which is the case in almost all behavioral genetics research on personality (cf. Ref. 69). In addition to the typical limitations of questionnaires, a particular concern in the bivariate and multivariate analyses is that the measures share common method variance. This may contribute to their phenotypic covariation, which in turn, potentially may contribute to both genetic and non-shared environmental correlations. Although most effects of common method variance should contribute to nonshared environmental correlations, genetically influenced factors, such as “acquiescence,” may also be important. Only further research using multiple methods (eg, experimenter observation, peer ratings) will be able to address this issue.

A second limitation of the study is the difficulty of drawing inferences about the direction of causation among the three psychosocial factors (eg, Ref. 59). It is very difficult to determine from cross-sectional data to what extent genetic factors contribute to multiple phenotypes (ie, pleiotropy) or contribute to only one phenotype, which then causes the others (eg, Ref. 70). For example, it is possible that genetic influences could contribute to the variability of depression, which in turn causes individuals to perceive a lack of social support. Alternatively, genetic variation could contribute to some common mechanism that influences both depression and social support. With a cross-sectional design, both possibilities are consistent with the present finding that a common genetic factor contributes to the covariation between depression and social support. A prospective twin design could aid in distinguishing such hypotheses.

Lastly, the use of factor analyses in the present study cannot provide information about psychosocial measures that were not included. The selection of the three constructs was not an attempt to discover a new, fundamental dimension of personality. Rather, they were selected because of their predictive associations with CVD in addition to their intuitive and empirically documented associations with each other. Future research could investigate the extent to which other psychosocial characteristics putatively relevant to CVD, such as anger, anxiety, job strain, and optimism, may share common genetic and environmental influences. Also, it should be determined whether variance common across such health-related characteristics is redundant with more broad-band measures of personality, such as the “Big Five” personality factors. Another promising approach for future research is to explore common genetic and environmental influences on the covariation between psychosocial and physiological risk factors for CVD.

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