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Temperament and Character Associated With Depressive Symptoms in Women: Analysis of Two Genetically Informative Samples

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Abstract

Although previous research has explored associations between personality and depressive symptoms, a limited number of studies have assessed the extent to which genetic and environmental influences explain the association. This study investigated how temperament and character were associated with depressive symptoms in 131 pairs of twin and sibling women in early adulthood, as well as 326 pairs of twin women in middle adulthood. Results indicated that genetic influences accounted for a moderate to substantial percentage of the association between these personality features and depressive symptoms, emphasizing the role of genetic influences. Nonshared environmental influences made important contributions to the association between character and depressive symptoms, particularly in the sample of middle-aged twin women. These findings suggest that unique social experiences and relationships with a partner in adulthood may play an important role in these associations between character and depressive symptoms.

Keywords

Temperament; Character; Depressive symptoms; Twin study; Family study

Depression is a leading mental disorder that involves the interaction between biological and psychosocial factors. One of the main clinical research areas in understanding complex depression is studying personality features in association with depression. Earlier studies have addressed the nature of relationships between personality and depression. For example, Clark, Watson, and Mineka (1994) have conceptualized the relationship between personality

and depression in four different ways. They suggest that personality is a vulnerability factor for developing depression and that personality may be a key factor in the course of depression. They also propose that personality may be different from depression by degree on the same process, considering an extreme manifestation of the personality as disorder. Finally, they explore the notion that personality may be the result of depression. With regard to the vulnerability model of depression, other researchers have speculated that particular personality types may increase the risk for developing depression. For example, Blatt suggests that dependency and self-criticism are specific vulnerability factors that increase the probability of developing depression (Blatt, 2004; Blatt, Quinlan, Chevron, McDonald, & Zuroff, 1982). Regardless of the foci studied by researchers, it is likely that certain personality types underlie depression.

Personality tends to be defined broadly, including individual differences in thoughts, feelings, and behaviors. Personality is thought to represent more than temperament. Although temperament seems to be influenced by both genetic and environmental influences, temperament is seen as basic dispositions of reactivity to stimuli with a more biological basis. Whereas early theorist Allport viewed temperament as individual differences in emotional reactivity, Chess and Thomas, who initiated the influential New York Longitudinal Study, included individual differences in attention and activity level into temperament (Rothbart & Bates, 2006). Beyond dispositional traits such as temperament, personality is conceptualized to include characteristic adaptations such as personal goals and motives, as well as integrative life stories (McAdams & Adler, 2006).

One widely-influential personality model is the factor model on phenotypic structure of personality, which prominently includes the Five Factor Model (FFM) or the Big Five. These traits include extraversion, neuroticism, conscientiousness, agreeableness, and openness to experience. Recent studies have explored the etiologic contributions of the FFM, applying the behavior genetics approach to explain personality models. Behavior genetics research has identified the underlying genetic factors of the phenotypic structure on the FFM (Jang, Livesley, & Vernon, 1996; Jang, et al., 2006; McCrae, Jang, Livesley, Riemann, & Angleitner, 2001). For example, recent longitudinal research from the Minnesota Twin-Family Study has demonstrated that genetic influences on personality are consistent from late adolescence to early adulthood when analyzing personality factors from the Multidimensional Personality Questionnaire (Blonigen, Carlson, Hicks, Krueger, & Iacono, 2008).

Another systematic approach to explaining personality is the psychobiological model proposed by Cloninger and Svrakic (Cloninger & Svrakic, 1997; Cloninger, Svrakic, & Przybeck, 1993; Cloninger, Svrakic, & Svrakic, 1997). That model distinguishes temperament from character dimensions, which are known as individual differences in voluntary goals and values, developed through maturation and learning. The Tridimensional Personality Questionnaire (TPQ; Cloninger, 1987) was originally developed to measure temperament dimensions; later, the Temperament and Character Inventory (TCI; Brandstrom et al., 1998) was developed to assess both temperament and character dimensions. In the TCI, temperament dimensions are measured by harm avoidance, novelty seeking, persistence, and reward dependence; character dimensions consist of self-directiveness, cooperativeness, and self-transcendence. Since the psychobiological model proposed by Cloninger provides a promising theoretical foundation that can be tested by the behavior genetics approach, studies have been conducted to verify genetic and environmental contributions of the factors, mainly with the temperament dimensions (Heiman, Stallings, Hofer, & Hewitt, 2003; Stallings, Hewitt, Cloninger, Heath, & Eaves, 1996). Less research has been undertaken, using the behavior genetics approach, on the association of both the temperament and character dimensions in Cloninger's model with

psychopathology. The current study extends previous studies to identify the genetic and environmental contribution to the associations between dimensions of Cloninger's model and depression.

Recent studies have shown the phenotypic relationship between dimensions of Cloninger's model and depression. Harm avoidance among temperament dimensions has been shown to be associated not only with depressive symptoms among the general population (Grucza, Przybeck, Spitznagel, & Cloninger, 2003), but also with a mood disorder diagnosis among psychiatric outpatients in clinical settings (Young et al. 1995). In addition to harm avoidance, self-directiveness from character dimensions has been found to be inversely associated with severe depressive symptoms in adults (Cloninger, Bayon, & Svrakic, 1998). Moreover, studies across different countries have consistently reported that specific dimensions of temperament and character are closely related to depression or depressive symptoms (Hirano et al., 2002; Marijnissen, Tuinier, Sijben, & Verhoeven, 2002; Peirson & Heuchert, 2001; Richer, Polak, & Eisemann, 2003; Tanaka, Kijima, & Kitamura, 1997).

These phenotypic associations of temperament and character dimensions with depressive symptoms have led to questions about the extent to which genetic and environmental influences explain the association. Current directions in behavior genetics studies have included investigating the nature of the relations between variables and developmental changes in addition to identifying the proportions of genetic and environmental influences of one variable. Although genetically informative studies have more recently been carried out to address genetic and environmental contributions to the association, the literature has been limited to assessing the relative genetic and environmental contributions in the association between personality dimensions of Cloninger's model and depression. One relevant behavior genetics study demonstrated that siblings who had never reported being depressed, but who had first-degree relatives with depression, reported higher scores for harm avoidance and reward dependence than never-depressed siblings who had healthy control probands (Farmer et al., 2003). The never-depressed siblings who had depressed relatives also reported lower scores for novelty seeking and self-directiveness than never-depressed siblings who had healthy control probands (Farmer et al., 2003). This finding suggests that these temperament and character traits may represent a potential genetic vulnerability to depression. The other exceptional study is a Japanese twin study using only the temperament dimensions of Cloninger's model; It suggests that the same additive genetic effects account for depression and dimensions of temperament (Ono et al., 2002). More genetically informative research is needed to determine which temperament and character dimensions are linked to depression and to assess the extent to which genetic and environmental influences contribute to the association. Those studies will help to capture clearly the nature of the relationship as well as to provide the information on the etiology of personality and depression. The current study, focusing on women, extends previous studies by relating both temperament and character dimensions to depressive symptoms in two genetically-informative samples.

Women may be particularly vulnerable to depression and depressive symptoms. Previous studies have shown that the rate of depression in women is two times higher than in men (Weissman & Olfson, 1995). Despite that high prevalence, studies on depression have been underinvestigated in women. The present study does not address the causal relationships between personality and depressive symptoms, but does examine how personality features and depressive symptoms are associated in women. Genetic influences are a possible candidate for explaining such associations. One possible scenario is that genetic liability to depression involves the manifestation of certain features of personality. In fact, recent molecular genetic studies and genomic mapping have explored candidate genes for personality traits in possible links with psychopathology. Additional processes, perhaps

certain stressful life events, may initiate a transformation of risk in manifesting symptomatology. It is also possible that personality is linked to depression through shared environmental influences, meaning that common environments that influence personality may influence the development of depression. Alternatively, nonshared environmental experiences that are unique to each individual may also favor the development of certain aspects of personality and depressive symptoms.

Using two independent genetically-informative samples, the current study aims to estimate the relative contributions of genetic and environmental factors in the association of temperament and character with depressive symptoms in women. Behavior genetics studies indicate that stability of personality is due to largely genetic influences from late adolescence to early adulthood (Blonigen et al., 2008) and even in late adulthood (Johnson, McGue, & Krueger, 2005). Based on previous work in this area, we hypothesize that genetic factors would account for much of the association of temperament and character with depressive symptoms. The hypothesis will be tested using two comparable, genetically-informative samples with identical measures, which results in firm evidence. Furthermore, the importance of sibling-specific environments increases across the life span (Loehlin, 1992a; McCartney, Harris, & Bernieri, 1990), and specific social interactions, such as marital relationships, also appear to contribute to depression or mental health in middle adulthood (Spotts et al., 2004; Spotts et al., 2005; Zlotnick, Kohn, Keitner, & Grotta, 2000). Congruent with the literature, we expect that nonshared environmental influences would make a substantial contribution to this association for the middle adulthood twin sample.

Method

Participants and Procedures

Data used in this study were from the Nonshared Environment in Adolescent Development (NEAD) Project (Reiss et al., 1994; Neiderhiser, Reiss, & Hetherington, 2007) and the Twin Mothers (TM) Project (Reiss, Pedersen, et al., 2001). Each sample is separately described. Since the same measures were used in both the NEAD and TM projects for the personality and depressive symptoms, the measures are described here only once.

NEAD Project—The sample used in this report consisted of 131 same-sex female siblings and twin pairs. The average age of the twin/sibling women in the analysis was 25, with ages ranging from 20 to 35 years. This subsample was part of the third assessment of the NEAD Project, a nationwide sample of two-parent families residing in two family types: families that had experienced no divorce and stepfamilies in which the parents were married at least five years. At the time of the first assessment, this nationwide sample consisted of 93 monozygotic (MZ) twin pairs; 99 dizygotic (DZ) twin pairs; and 95 full sibling (FI) pairs in non-divorced families; and 181 full sibling (FS), 110 half sibling (HS), and 130 unrelated sibling (US) pairs in stepfamilies. The six types of siblings showed few significant differences for age or number of children in the family. Most of the twin pairs in non-divorced families and sibling pairs in stepfamilies were recruited through a national market survey of 675,000 households. Full sibling pairs in non-divorced families were recruited through random-digit dialing of 10,000 telephone numbers throughout the United States. The sample at the time of the first assessment consisted of adolescent sibling pairs and their parents. The adolescents ranged in age from 10 to 18 years and their siblings ranged in age from 9 to 18 years, with the age differences between siblings within families no greater than four years. The participating siblings had to reside at home at least half-time. The sample represented generally middle class families; 94 percent of the mothers were Caucasian. Additional details about the sampling strategy and sample characteristics can be found elsewhere (e.g., Neiderhiser et al., 2007; Reiss, Neiderhiser, Hetherington, & Plomin, 2000).

The NEAD sample was assessed longitudinally during middle adolescence (first time), three years later during late adolescence (second time), and during early adulthood (third time). The third time data collection included all family members who participated in the first time and the eligibility resulted in a 7- to 13-year time span, depending on the participation on the second time data collection. Despite the long time span from 7 to 13 years between the last contact and the third assessment, data on the third assessment was collected from more than 50% of families who participated in the first assessment. Demographic characteristics such as the parents' age or education for the families who participated in the third assessment were not different from the families who did not participate in the third assessment. The present study is based on the third assessment of the NEAD sample and includes data from all six sibling types. Among families who participated in the third assessment, only the female subsample was analyzed for the present study in order to be comparable to the sample from the TM Project (described below). The female subsample included 28 MZ twin pairs; 21 DZ twin pairs; 19 full siblings pairs in non-divorced families; and 28 full sibling, 16 half sibling, and 19 unrelated sibling pairs in stepfamilies. Although multiple measures on adjustment, including depressive symptoms, were collected at all three time periods for the NEAD Project, only data from the third period were considered for the present study since they included the same personality measure of interest employed in both NEAD and TM projects.

TM Project—The TM sample comprised 326 pairs of adult female twins. The twin pairs consisted of 150 MZ twin pairs and 176 DZ twin pairs. The sample included twin women between 32 and 54 years of age (mean age = 44 years). Although the TM sample included pairs of women drawn from the Swedish Twin Registry, their male partners, and one adolescent child for each pair, the current research included data on only twin mothers. Details regarding the Swedish Twin Registry are provided in Lichtenstein et al. (2002), and details about the TM sample and measurement are available in Reiss, Cederblad, et al. (2001). Because the TM study emphasized genetic and social influences on maternal adjustment, the twin pairs of the sample reflected a random representative sample of two-household, two-generation families. Of the 652 couples in the sample, 613 couples were married and 39 couples were cohabiting; cohabitation is a common alternative to marriage in Sweden and no differences between these two groups were found. All married and cohabiting couples were required to have lived together for at least five years prior to the time of data collection.

Zygosity assessment—For twin pairs, zygosity was determined utilizing self-reports and interviewer ratings in the NEAD Project and with self reports in the TM Project, based on a modified version of a zygosity questionnaire. Questionnaire methods of assigning zygosity have been found to be more than 95 percent accurate when compared to DNA (Nichols & Bilbro, 1966; Spitz, Moutier, Reed, Busnel, & Marchaland, 1996). In the NEAD sample, zygosity was unclear for 12 twin pairs and these twin pairs were excluded from all analyses. DNA was also collected from the twins who participated in the TM Project; zygosity was verified via DNA samples for all but four twin pairs who refused to provide a DNA sample or provided an unusable sample. For these four twin pairs, zygosity was assigned according to their questionnaire results. In the TM Project, no significant differences existed between the MZ and DZ twins with regards to characteristics such as age, occupation level, education, or age of partner.

Measures

The measures of interest in the present study, used in both the NEAD and TM projects, include the Temperament and Character Inventory (TCI; Brandstrom et al., 1998) and the Center for Epidemiological Studies-Depression Scale (CES-D; Radloff, 1977).

Temperament and character—The 125-item version of the TCI was used to assess dimensions of temperament and character in the adulthood assessment of the NEAD and TM samples. The TCI has two subscales assessing temperament and character; these subscales are theoretically conceptualized as independent. The temperament subscales assess novelty seeking, harm avoidance, persistence, and reward dependence dimensions. For example, the harm avoidance dimension is designed to assess a heritable bias in behavioral inhibition. The character subscales assess self-directiveness, cooperativeness, and self-transcendence dimensions. For example, the self-directiveness dimension is designed to assess the extent to which an individual perceives himself or herself as responsible, purposeful, resourceful, and self-acceptable. The self-transcendence dimension is known to be related to spirituality and assumes that low self-transcendence represents materialistic behavior without absolute ideals. The TCI has shown high internal consistency and robust construct validity in patients with major depression (Sato et al., 2001).

Internal consistency estimates for the TCI subscales for the NEAD sample were .79 for novelty seeking, .86 for harm avoidance, .61 for reward dependence, .62 for persistence, .83 for self-directiveness, .72 for cooperativeness, and .72 for self-transcendence. Internal consistency reliability estimates of the TCI subscales for the TM sample were .68 for novelty seeking, .83 for harm avoidance, .47 for reward dependence, .56 for persistence, .83 for self-directiveness, .62 for cooperativeness, and .83 for self-transcendence.

Depressive symptoms—The CES-D was used to measure depressive symptoms over a one-week recall period for the NEAD sample assessed during early adulthood and for the TM sample. The measure consists of 20 items scored on a 4-point scale ranging from rarely or none of the time to most or all of the time. The CES-D has shown adequate reliability and validity with community and psychiatric populations (Boyd, Weissman, Thompson, & Myers, 1982; Weissman, Sholomskas, Pottenger, Prusoff, & Locke, 1977). A cross-national comparison for the CES-D supported the use of the measure to assess self-reported depressive symptoms (Gatz, Johansson, Pedersen, Berg, & Reynolds, 1993). In the current study, the internal consistency of the CES-D was .93 for the NEAD sample and .90 for the TM sample.

Statistical Analyses

Prior to all analyses for both NEAD and TM samples, the effect of age was regressed out of the scores (McGue & Bouchard, 1984). Nontwin sibling scores for the NEAD sample were also corrected for age differences within sibling pairs. Because the depressive symptoms scores were skewed to the left, scores on the CES-D were transformed using a square root transformation in both samples.

Genetic model-fitting analysis—The current study focused on how temperament and character are associated with depressive symptoms. Figure 1 depicts the bivariate genetic model used to partition the covariance between two variables. This model is designed to analyze the covariance between temperament and depressive symptoms as well as the covariance between character and depressive symptoms. Double-headed arrows connecting the latent genetic and environmental factors are set to be equal to the model expectations. Specifically, the correlation between the latent genetic factors varies according to the genetic relatedness of the twin/sibling pair. The range of genetic relatedness varies from 0 percent among unrelated siblings, to 25 percent among half siblings in stepfamilies, to 50 percent among full siblings in nondivorced families, stepfamilies, and dizygotic twins, and is 100 percent among monozygotic twins (MZ=1, DZ, FI, FS=.50, HS=.25, US=0). The correlation between the latent shared environmental factors is set to 1.0 for all twin/sibling types, and the correlation between the nonshared environmental latent factors is set to 0. Using Figure

1 as a guide, the covariance between personality features and depressive symptoms is broken into three components: additive genetic (A), shared environmental (C), and nonshared environmental (E) factors. Additive genetic factors assume that multiple genes independently influence personality. Shared environmental influences include all nongenetic factors that make family members similar to one another. It is worth noting that shared environmental influences for both of these adult samples include effects of shared rearing experiences and influences of current contact. Finally, nonshared environmental influences and measurement error are all nongenetic factors that cause family members to differ. Genetic and environmental influences on personality measurements were broken down into components accounted by the genetic and environmental effects on depressive symptoms, denoted as a_{21} , c_{21} , and e_{21} while the paths a_{11} , c_{11} , and e_{11} indicated the genetic and environmental influences on personality only. Specific genetic, shared, and nonshared environmental influences on depressive symptoms independent of personality are represented by the paths a_{22} , c_{22} , and e_{22} .

The overall fit of each model was tested using χ^2 and Akaike's Information Criterion (AIC; Akaike, 1987). The best-fitting model can be evaluated by nonsignificant χ^2 values and an AIC that is low or negative, considering that as AIC decreases the fit of the model increases. Previous research has found that a χ^2 test is likely to reject a model that fits the data well but imperfectly, is very sensitive to sample size, and improves when more parameters are added to the model (Mulaik et al, 1989; Neale & Cardon, 1992; Tanaka, 1993). AIC, which is equal to the χ^2 value minus two times the degree of freedom, considers both the goodness of fit as well as parsimony, thereby providing a more comprehensive fit index when used in conjunction with χ^2 (Williams & Holahan, 1994). Further discussion of fit indices is available elsewhere (Bollen & Long, 1993; Loehlin, 1992b; Neale & Cardon, 1992). The computer program Mx was used to estimate the models (Neale, Boker, Xie, & Maes, 2002).

Model assumptions—The assumptions of models are implicit in Figure 1 and are somewhat different for the NEAD and TM samples. The assumptions of both models indicate that shared environmental effects are the same across twin and sibling types and that genotype-environment interaction and correlation are negligible. For the NEAD sample, it is also assumed that neither selective placement of the stepsiblings nor assortative mating occurs. Earlier investigation of the equal-environment assumption has supported the validity of the assumption in twin studies of psychiatric disorders (Hettema, Neale, & Kendler, 1995). Although genotype-environment interaction may be present, most research has been unsuccessful in detecting this in non-clinical samples (Plomin, Defries, McClearn, & Rutter, 1997). Assortative mating refers to nonrandom mating, indicating sizable correlations between spouses for the same characteristic. Appropriate measures to test the assortative mating effects were not included for the parents of the twin women. In general, if there are any assortative mating effects, they will serve to decrease heritability estimates and increase shared environmental influences. A general discussion of the assumptions of quantitative genetic model fitting can be found elsewhere (Loehlin, 1992b; Plomin, Defries, & McClearn, 1990).

Results

Descriptive Statistics

The means, standard deviations, and ranges for the study variables are presented in Table 1 for both the NEAD and TM samples, while Table 2 shows the correlations between personality features and depressive symptoms for both samples. The harm avoidance and self-directiveness subscales in the TCI were further analyzed as the phenotypic correlations

with depressive symptoms were high enough to warrant bivariate behavioral genetic analyses.

Genetic Model-Fitting Analyses

Maximum likelihood model-fitting analyses allow estimates of the contribution of genetic and environmental influences by partitioning variances. The full ACE model estimated the variance components with genetic (A), shared environmental (C), and nonshared environmental (E) factors. In the full model, notable genetic and nonshared environmental influences are evident in harm avoidance, self-directiveness, and depressive symptoms. Shared environmental influences were not found in the full ACE model, revealing that C was estimated as zero. Nested models were tested by systematically dropping genetic and shared environmental components from the full model (harm avoidance of the NEAD: $\chi^2=16.294$, $df=15$, $AIC=-13.706$; harm avoidance of the TM: $\chi^2=12.205$, $df=3$, $AIC=6.205$; self-directiveness of the NEAD: $\chi^2=26.135$, $df=15$, $AIC=-3.865$; self-directiveness of TM: $\chi^2=12.347$, $df=3$, $AIC=6.347$; depressive symptoms of the NEAD: $\chi^2=18.013$, $df=15$, $AIC=-11.987$; depressive symptoms of TM: $\chi^2=1.196$, $df=3$, $AIC=-4.804$). The CE model in which the genetic parameter was dropped did not fit the data for personality variables for both samples and depressive symptoms for the NEAD sample (harm avoidance of the NEAD: $\chi^2=22.984$, $df=16$, $AIC=-9.016$; harm avoidance of the TM: $\chi^2=25.889$, $df=4$, $AIC=17.889$; self-directiveness of the NEAD: $\chi^2=35.733$, $df=16$, $AIC=3.733$; self-directiveness of TM: $\chi^2=15.335$, $df=4$, $AIC=7.335$; depressive symptoms of the NEAD: $\chi^2=23.625$, $df=16$, $AIC=-8.375$). The CE model on depressive symptoms for the TM sample did not show a significantly worse fit, which was not surprising given that the confident interval for the genetic effects included zero (.00-.40).

The E model with only nonshared environmental variance showed the worst fit in the data from both samples, based on chi-square and AIC values. In all measures of interest in this study, the AE model in which the shared environmental parameter (c parameter) was dropped was the most parsimonious and best-fitting model; the AE model showed no significant worsening of the model fit and AIC relative to the full model and demonstrated a lower AIC value. Table 3 presents only the results of the univariate best model-fitting analyses for harm avoidance, self-directiveness, and depressive symptoms.

In the bivariate genetic model-fitting analyses, the AE models provided a better fit for the association between harm avoidance and depressive symptoms in both the NEAD sample ($\chi^2=64.477$; $df=54$; $AIC=-43.523$) and the TM sample ($\chi^2=19.090$; $df=14$; $AIC=-8.910$) than in the full models. The AE models also resulted in a better fit for the association between self-directiveness and depressive symptoms for both samples (NEAD: $\chi^2=79.447$, $df=54$, $AIC=-28.553$; TM: $\chi^2=30.989$, $df=14$, $AIC=2.989$).

The first noteworthy finding was that the genetic influences on the covariance between these personality features and depressive symptoms were substantial. Figure 2 depicts estimates of genetic and environmental effects from bivariate model-fitting results. The genetic influences explained a substantial portion of the covariance between harm avoidance and depressive symptoms. As Table 2 shows, the phenotypic correlations between personality features and depressive symptoms can be accounted for by genetic and nonshared environmental influences. The magnitude of the cross correlations can be confirmed by multiplying the path coefficients for each genetic and nonshared environmental influences and then adding genetic and nonshared environmental correlations behind these cross correlations. We calculated the percent of the correlation between harm avoidance and depressive symptoms that can be attributed to genetic influences by multiplying the path coefficients. Specifically, genetic effects were the sole explanation for the phenotypic correlation between harm avoidance and depressive symptoms for the NEAD sample.

Genetic effects explained about 50 percent of the phenotypic correlation for the middle adulthood TM sample ($.64 \times .33/.42$; the phenotypic correlation of .42 between harm avoidance and depressive symptoms for the TM sample). Meanwhile, for self-directiveness and depressive symptoms, the proportions of phenotypic variance attributable to genetic effects were close to 70 percent for the NEAD sample ($.75 \times .54/.59$; the phenotypic correlation of .59 between self-directiveness and depressive symptoms for the NEAD sample), and to 36 percent for the TM sample ($.59 \times .32/.52$; the phenotypic correlation of .52 between self-directiveness and depressive symptoms for the TM sample).

A second noteworthy finding was that nonshared environmental influences contributed to the association between self-directiveness and depressive symptoms, and were quite prominent, particularly in the middle-aged sample. Nonshared environmental effects accounted for 50 percent of the correlation between harm avoidance and depressive symptoms for the middle adulthood TM sample ($.77 \times .27/.42$; the phenotypic correlation of .42 between harm avoidance and depressive symptoms for the TM sample) and none of the correlation for the NEAD sample of young adults. For self-directiveness and depressive symptoms, the proportions of correlation attributable to nonshared environmental effects were 64 percent in the middle adulthood TM sample ($.81 \times .41/.52$; the phenotypic correlation of .52 between self-directiveness and depressive symptoms for the TM sample) versus 29 percent in the NEAD sample ($.66 \times .26/.59$; the phenotypic correlation of .59 between self-directiveness and depressive symptoms for the NEAD sample). For the NEAD sample, only nonshared environmental influences were unique to depressive symptoms once the variance shared with harm avoidance was accounted for.

The results showed genetic and environmental influences on the association between these personality features and depressive symptoms. Figure 3 presents estimates of the genetic and environmental contributions to the phenotypic correlations between these personality features and depressive symptoms.

Discussion

The goal of this study was to examine the genetic and environmental influences on the association of temperament and character with depressive symptoms in women. To this end, two complementary, genetically-informative samples were analyzed. Only the harm avoidance and self-directiveness dimensions of the TCI were highly correlated enough with depressive symptoms to warrant further analysis. Previous empirical research has indicated that harm avoidance is related with depressive symptoms (Grucza et al., 2003) whereas self-directiveness is inversely related with depressive symptoms in adults (Cloninger et al., 1998). According to theoretical background from Cloninger's psychobiological model, personality dimensions that predispose individuals to depression are linked to psychopathology. Particularly, high harm avoidance has been related to pessimistic, fearful, and shy behaviors and low self-directiveness has been related with behavior of poor impulse control and personality disorder (Cloninger et al., 1997). Consistent with previous literature, only harm avoidance and self-directiveness from the TCI were sufficiently highly correlated with depressive symptoms to warrant bivariate behavioral genetic analyses in this study.

Findings of genetic contributions that explain covariance of the dimensions of temperament and character with depressive symptoms from the two samples are consistent with the results of previous behavioral genetic studies (Farmer et al., 2003; Ono et al., 2002). For example, harm avoidance was considered as an index of genetically-influenced vulnerability to depression in behavior genetics studies (Farmer et al., 2003; Yuh et al., 2008). The findings of the present study from two genetically-sensitive designs support that there is evidence of genetic overlap between temperament/character and depression. Furthermore, genetic

influences on the covariance of harm avoidance and depressive symptoms appeared to be greater for the NEAD sample than the TM sample. Previous research on the onset of depression provides indirect evidence for these relative genetic influences. For example, recent research demonstrated that early onset depression at or before 25 years of age is more related to familial contribution and early personality traits, represented by behavioral inhibition or early shyness, compared to a matched control group with late onset depression (Parker, Roy, Hadzi-Pavlovic, Mitchell, & Wilhelm, 2003).

Whereas only genetic factors seem to account for almost all the covariance between harm avoidance and depressive symptoms among women in young adulthood from the NEAD sample, both genetic and nonshared environmental influences explained the association among middle-aged women from the TM sample. The role of nonshared environmental influences for the TM sample may reflect changing social settings in mid-life. Unique social systems experienced by middle-aged women, such as a job, child, or husband, may favor the development of harm avoidance and depressive symptoms. However, this speculation needs to consider the possibility that any differences found between the two samples could have resulted from differences in sample characteristics. Data from the two samples were collected from the United States and Sweden. The differences in the two samples may include differences inherent in the procedures utilized in the two samples from the different countries. Although a comprehensive review of Swedish cultural characteristics did not support any different mechanisms for the origin of depression in Sweden in comparison with other nations (Daun, 1996), we can not rule out the possibility that differences found may be associated with different samples.

Analyses indicate that nonshared environmental influences tend to be substantial in the association between character and depressive symptoms in both samples. Individual differences in specific environmental experiences contribute to the development of certain characters such as self-directiveness and depressive symptoms. In the psychobiological model of personality development proposed by Cloninger and colleagues (Cloninger & Svrakic, 1997; Cloninger et al., 1993; Cloninger et al., 1997), character reflects individual differences in goals and values. It begins with parental attachments, is followed by self-object differentiation, and matures as a continuum throughout life with the interactions among temperament, family environment, and life experiences. Because of the core features of character in the model, dimensions of character such as self-directiveness are influenced substantially more by nonshared environmental influences than dimensions of temperament. The findings of nonshared environmental influences in the TM sample are also compatible with previous studies in that greater nonshared environmental influences, rather than shared environmental influences, tend to exist in association with increasing age. A study that compared genetic and environmental contributions to personality change at different ages using various cross-sectional and longitudinal studies has demonstrated that nonshared environmental influences appeared to increase as individuals grew older, whereas shared environmental influences appeared to become negligible (Loehlin, 1992a). A recent longitudinal study on personality stability in late adulthood also has demonstrated approximately equal genetic and nonshared environmental influences on stability (Johnson et al., 2005). In addition, recent twin studies using the TM sample have consistently shown that nonshared environmental influences play a substantial role in middle-aged women's depressive symptoms and mental health, suggesting husbands are a potential source of nonshared environmental influence (Spotts et al, 2004; Spotts et al, 2005). The experiences unique to individuals may favor certain personality characteristics and the development of depressive symptoms. More study on this matter is needed to identify the specific nonshared environmental influences.

Although the results of the present study demonstrate that genetic and nonshared environmental influences account for the overlap between personality features and depressive symptoms, it is important to note that genetic and nonshared environmental influences exist that are unique to depressive symptoms independent of temperament and character. For example, substantial unique genetic influences and unique nonshared environmental influences were found for the depressive symptoms among the middle-aged TM sample. Although the focus of the present study was the overlapping genetic and nonshared environmental influences between personality features and depressive symptoms, the findings from this study also suggest that future research should explore influences on depressive symptoms that are accounted for by different individual characteristics.

Several limitations of the study should be considered carefully. First, the sample consisted only of females. Although genetic factors have not been found to differ by gender for depression (Lyons et al., 1998), it is not clear whether these results can be generalized to males. Second, as discussed earlier, samples from the United States and Sweden were compared. Moreover, the differences in results may also include differences in the two samples given that the procedures were not completely the same. Previous research that investigated similar patterns in the means, distribution, and associations within and between TCI scales and subscales in the American and Swedish samples suggests that no meaningful differences exist in the constructs by country (Brandstrom et al, 1998). Reiss and colleagues have argued that cultural differences in findings are best explored when guided by clear hypotheses that posit how well-specified cultural differences might influence findings (Reiss, Cederblad, et al., 2001). A comprehensive review of Swedish cultural characteristics did not yield any obvious hypotheses suggesting different mechanisms for the origin of depression in Sweden as opposed to other nations (Daun, 1996). One recent research approach to clarify underlying genetic and environmental influences compares two genetically-sensitive designs (Iervolino et al., 2002; Neiderhiser, Reiss, Lichtenstein, Spotts, & Ganiban, 2007). With the efforts to delineate the underlying processes using two genetically-informative samples, longitudinal studies over considerable periods of time using a large sample are needed to more fully assess developmental change. Third, given that participants were representative of the general population, the present findings may not be generalizable to depressive disorders in clinical settings. Further study is needed to investigate how clinical depression may be associated with personality characteristics. Fourth, although variables of interest in the present study were in adequate range of reliability from .83 to .86 for the TCI and were used for further analyses, some of the reliability estimates for other subscales of the TCI measure were in the low range. Concerning that reliability in the measure of the independent variable, future studies need to consider using other personality measures with high reliability. Finally, personality features and depressive symptoms were measured through self-reporting instruments. Although previous research has demonstrated clear agreement between self-reported data and the informant rating for personality measures (Heath, Neale, Kessler, Eaves, & Kendler, 1992), assessments by multiple raters would increase the confidence in the results.

Despite these limitations, the results of two genetically-informative samples delineate the extent to which genetic and environmental influences account for the associations of temperament and character with depressive symptoms in women. The finding that genetic influences explained the association between harm avoidance and depressive symptoms as well as self-directiveness and depressive symptoms suggests that future research should focus on investigating candidate genes as well as transforming process into symptomatology. A substantial nonshared environmental contribution to the association is also important. Unique experiences could play important roles in the manifestation of certain aspects of personality and depressive symptoms. Identifying the unique environments that could contribute to the shaping of personality and depressive symptoms would be helpful to

the development of interventions. For example, the role of heritable personality features in developing depressive symptoms may be influenced by a unique environment such as marriage or specific social interactions for middle-aged women. Thus, these efforts to clarify underlying genetic and environmental influences through genetically-informative samples can help us better understand the nature of depressive symptoms in women and facilitate the development of interventions for women at risk for depression by considering unique social experiences as a new focus of prevention.

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Figure 1.

Bivariate Cholesky model for personality and depressive symptoms.

Note. A, C, E represent genetic, shared environmental, and nonshared environmental influences respectively. a, c, e represent genetic, shared environmental, and nonshared environmental influences specific to depressive symptoms.

Figure 2.

Bivariate model fitting from the best fitting model between personality features and depressive symptoms.

Note. First value given is for the NEAD wave 3; second value is for the TM sample.

A=genetic influences; E=nonshared environmental influences; a=genetic influences specific to depressive symptoms; g=nonshared environmental influences specific to depressive symptoms. Parenthetical values give the 95% intervals.

Figure 3.

The contribution of common variables A and E to the phenotypic correlation between personality features and depressive symptoms by sample.

Note. Bars represent strength of correlation. A=genetic influences; E=nonshared environmental influences.

Table 1

Means, Standard Deviations, and Ranges for Study Variables.

Variables	NEAD sample			TM sample		
	M	SD	Range	M	SD	Range
Temperament						
Novelty Seeking	9.29	4.36	1–20	9.45	3.35	2–19
Harm avoidance	9.87	4.98	0–20	8.31	4.35	0–20
Reward Dependence	11.34	2.65	1–15	10.09	2.34	1–15
Persistence	3.04	1.51	0–5	2.35	1.41	0–5
Character						
Self-directiveness	18.56	4.73	5–24	20.49	3.91	4–25
Cooperativeness	22.06	2.78	11–25	21.68	2.41	11–25
Self-transcendence	6.01	3.19	0–14	4.44	3.39	0–15
Depressive Symptoms [*]	3.70	1.44	0–7.28	2.92	1.30	0–6.86

Note.

^{*} These are transformed scores for skewness.

Table 2

Phenotypic Correlations between Personality and Depressive Symptoms

	Depressive Symptoms	
	NEAD sample	TM sample
Temperament		
Novelty Seeking	.29 [*]	.03
Harm avoidance	.46[*]	.42[*]
Reward Dependence	-.19 [*]	-.02
Persistence	-.02	.05
Character		
Self-directiveness	-.59[*]	-.52[*]
Cooperativeness	-.29 [*]	-.12 [*]
Self-transcendence	.11	.08 [*]

Note. Bold represents the focus of further analyses.

^{*}
 $p < .05$

Table 3
Univariate Best Model-fitting Results for Harm Avoidance, Self-directiveness, and Depressive Symptoms

Measures	Parameter Estimates (95% CI)			Fit of Model		
	A	C	E	χ^2	df	AIC
NEAD sample						
Harm Avoidance	.53 (.24–.73)	--	.47 (.27–.77)	16.294	16	-15.706
Self-directiveness	.58 (.30–.76)	--	.42 (.24–.70)	26.135	16	-5.865
Depressive Symptoms	.57 (.32–.75)	--	.43 (.25–.68)	18.013	16	-13.987
TM sample						
Harm Avoidance	.41 (.25–.53)	--	.59 (.47–.73)	12.205	4	4.205
Self-directiveness	.35 (.21–.47)	--	.65 (.53–.79)	12.347	4	4.347
Depressive Symptoms	.27 (.13–.40)	--	.73 (.73–.87)	1.196	4	-6.804

Note. The estimates and fit index are the results for the full model. AIC represents Akaike's Information Criterion. A, C, and E represent genetic, shared environmental, and nonshared environmental effects respectively.