JONGIL YUH *Yonsei University*

JENAE M. NEIDERHISER* *Pennsylvania State University*

DAVID REISS** *Yale Child Study Center*

Genetic and Environmental Influences on Dispositional Optimism and Depressive Symptoms in Adolescence

This study explored genetic and environmental contributions to optimism, depressive symptoms, and the association between the two using a genetically informative sample from the Nonshared Environment and Adolescent Development project (NEAD: D. Reiss; J. M. Neiderhiser; E. M. Hetherington; & R. Plomin, 2000)†. At Time 1 of the longitudinal NEAD study, the sample consisted of 720 same-sex twins and sibling pairs from two parent families. The study used parent, adolescent, and observer ratings of depressive symptoms as well as adolescent ratings of optimism. The results revealed that genetic influences explained approximately half of the variability in optimism and depressive symptoms. Nonshared environmental influences also substantially contributed to optimism and depressive symptoms. Bivariate genetic analyses (which partitioned the covariance between optimism and depressive symptoms into genetic and environmental components) indicated that genetic influences accounted for a moderate percentage of the association.

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Research Faculty, Research Institute of Science for Aging, Yonsei University, Seoul, Korea (jonyuh@hotmail.com)

*Professor, Department of Psychology, Pennsylvania State University

**Clinical Professor, Child Study Center, Yale University

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Depression is one of the most prevalent mental health problems during adolescence. Though less common during childhood, depression increases during adolescence. Point prevalence estimates of depression are about 4.3% in adolescents, while lifetime prevalence of major depressive disorder in adolescents ranges from 8.3% to 18.5% (Garber, 2000). Due to the high prevalence rates, there has been increased research attention on the etiology of depression.

Depression is a complex psychiatric problem in which genetic and environmental factors both play roles pertaining to causation and development. For example, a model of vulnerability integrates genetics, biology, and cognitive and social factors in explaining depression (Hammen & Garber, 2001). In an effort to understand the mechanism underlying depression, family, twin, and adoption studies have examined genetic and environmental influences. Although summary figures have yet to be estimated, the findings of these different designs and samples suggest that depression is caused by a combination of genetic and environmental influences. Genetic contributions to adolescent depression are moderate and account for approximately 30-50% of the variance.

Researchers have identified potential risk and vulnerability factors that contribute to depression. Recent models of its pathogenesis posit that certain

individual characteristics are crucial in understanding depression. A number of researchers have paid attention to one individual characteristic in particular, optimism, as a potential protective factor (Nolen-Hoeksema *et al.*, 1992; Robinson *et al.*, 1995). Two different approaches have been used to conceptualize and measure optimism (Reivich & Gillham, 2003). One approach defines optimism as a hopeful disposition or a conviction that future outcomes will be good. The approach uses direct methods of assessing the expectancies that future outcomes will be good or bad (Reivich & Gillham, 2003). The second conception of optimism refers to the belief that the world is the best of all possible worlds (Reivich & Gilliham, 2003). The approach to assessing this particular optimism is more indirect, examining the ways in which people think about causes of events in their lives (Reivich & Gillham, 2003; Scheier *et al.,* 2002).

Although dispositional optimism and explanatory style are conceptually related (Gillham *et al.,* 2002), the current study focuses on dispositional optimism to understand the associations with depressive symptoms. Scheier and Carver (1985) defined dispositional optimism and pessimism as generalized positive and negative outcome expectancies, respectively, and have considered stable characteristics over time. Accumulated evidence from various studies suggests that dispositional optimism is inversely related to the development of depression. Longitudinal studies have indicated that greater optimism is associated with smaller increases in depressive symptoms (Nolen-Hoeksema *et al.*, 1992; Robinson *et al.*, 1995; Brissette *et al.,* 2002). In a twin study, the twin who reported one or more lifetime episodes of depression in at least two assessments over four interviews reported more deviant optimism scores than her sister who was well (Kendler & Gardner, 2001). Current studies of cognitive prevention programs for depression have also indirectly suggested that optimism could serve as an indicator of the onset of depressive symptoms (Seligman *et al.,*1999; Yu & Seligman, 2002).

Understanding the etiology of optimism is thought to be one of the most interesting and valuable research areas (Gillham *et al.,* 2002; Zuckerman, 2002); however, only a few studies have examined genetic and environmental influences on optimism. By examining correlations between 115 pairs of identical twins and 27 pairs of fraternal twins across a wide age range, one study revealed heritability in attributional styles for both the composite and negative events scales and shared environmental effects in attributional styles for positive events scale (Schulman *et al.,* 1993). However, zero correlation between fraternal twins in the study seemed to indicate an entire genetic mechanism or unreliable data because of small number of fraternal twins (Zuckerman, 2002). In another study with a mean age of 60.7 years, which compared older adult twins raised together and twins raised apart, heritability accounted for 23% of the total variance in optimism and 27% of the total variance in pessimism. Furthermore, genetic factors accounted for more than one-third of the phenotypic associations among optimism, pessimism, and depression (Plomin *et al.,*1992). In an effort to extend this line of research, the present study investigated the etiology of optimism using the data collected during adolescence.

Over the past several decades, researchers have investigated genetic and environmental influences in order to address individual differences in behavior and adjustment. A behavioral genetics methodology allows researchers to statistically estimate genetic and environmental influences for the study of their relative importance for adjustment outcomes (DiLalla, 2004). Current directions in the field have included investigating the nature of relations between variables, and estimating the proportions of genetic and environmental influences of single variables. More genetically informative research is needed to determine whether optimism is linked to depression and to assess the extent to which genetic and environmental influences contribute to that association. In order to capture the nature of the relationship, the current study investigated the influence of genetic and environmental factors on the relationship between optimism and depressive symptoms.

The current study, using a genetically informative sample of adolescents, aims to estimate the relative contributions of genetic and environmental factors

to optimism and depressive symptoms. This study addresses two specific questions: (1) What is the relative importance of genetic and environmental influences on optimism and depressive symptoms? And (2) Do the same genetic and environmental influences explain the associations between optimism and depressive symptoms in adolescents? Genetic influences are a possible source of the association between optimism and depressive symptoms. Genetic factors that influence an individual's optimism may directly manifest as genetic vulnerability to depressive symptoms or may be indirectly associated with depressive symptoms interacting with other variables. Another potential source of the association could be shared environmental influences or common environments that influence optimism and the development of depressive symptoms. Alternatively, nonshared environmental experiences that are unique to each individual may be related to the development of optimism and depressive symptoms. Unique experiences, such as experiences with one's peers, may have an influence on the association for adolescents. Efforts to clarify the underlying genetic and environmental influences would expand our knowledge of the pathways leading to depression and provide information on the etiology of optimism and depression as well.

METHODS

Subjects

Participants in the NEAD project included 720 same-sex sibling pairs of adolescents between the ages of 10 and 18 and their parents (M=13.5; SD=2.0). The NEAD project is a longitudinal study of adolescents and parents from adolescence into young adulthood. Reiss and colleagues provided a detailed sample description elsewhere (e.g., Neiderhiser *et al.,* 2007; Reiss *et al.,* 2000). The NEAD project consisted of a geographically representative sample of two-parent families with a pair of same-sex adolescent siblings. The nationwide sample consisted of 93 monozygotic (MZ) and 90 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 sample represented two family types: Families that had experienced no divorce and stepfamilies in which the parents were married for at least 5 years. Pairs of adolescents were no more than four years apart and the adolescents in the case of dual custody in stepfamilies were required to have lived together at least 50% of the time. Most of the twin pairs in non-divorced families and sibling pairs in stepfamilies were obtained through a national market panel of 675,000 households. Full sibling pairs in non-divorced families were nationally recruited through a random digit dialing of 10,000 telephone numbers. The families were primarily middle class. The average number of years of education was 13.6 for mothers and 14.0 for fathers. For twin pairs, zygosity was determined by reports from interviews, parents, and the adolescents of physical similarity using a modified version of a zygosity questionnaire for adolescents who reported an accuracy of over 90% as compared with the DNA assessment of zygosity (Nichols & Bilbro, 1966).

Measurement

Optimism Optimism was assessed by the Life Orientation Test (LOT; Scheier & Carver, 1985) that consisted of 12 items using a 5-point Likert-type scale, with 4 positively worded, 4 negatively worded, and 4 non-scored items. The authors of the instrument hypothesized that optimism and pessimism represents a single personality dimension with bipolar ends, rather than two distinct dimensions. A single summary score was used in the present study. The internal consistency estimates for the Life Orientation Test was .74.

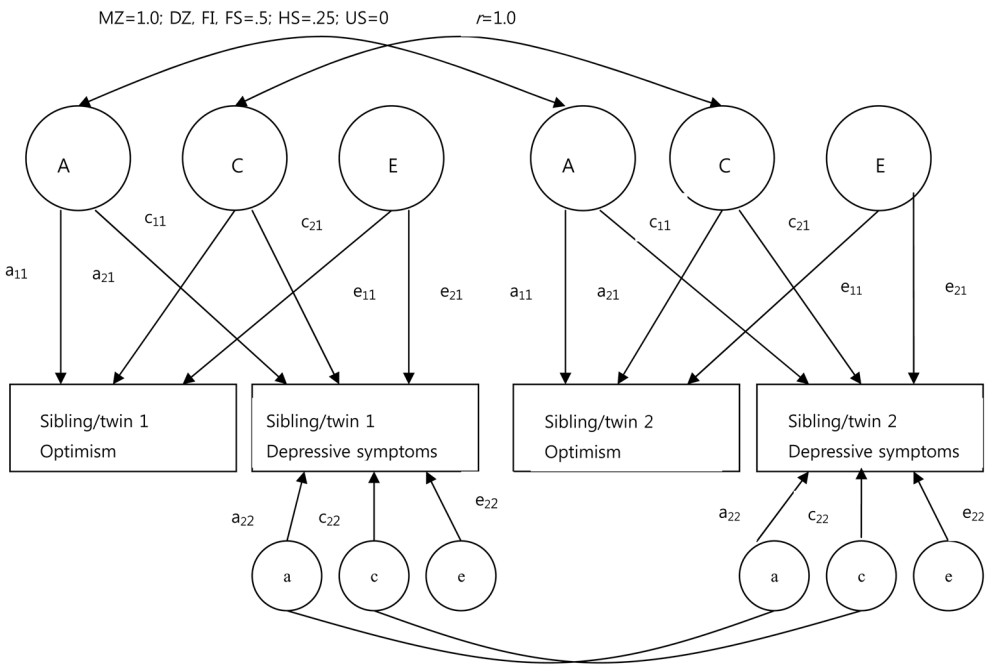
Depressive symptoms Depressive symptoms were assessed by multiple measures and multiple raters, including mother, father, and adolescent reports of total scores for the Child Depression Inventory (CDI; Kovacs, 1985), the depressive behavior subscale from the Behavior Problems Index (BPI; Zill, 1985), and the depression subscale from the Behavior Events Inventory (BEI; Hetherington & Clingempeel, 1992) as well as observer ratings of depressed affect during dyadic interactions between

the adolescent and each of the family members (Hetherington & Clingempeel, 1992). All measures were standardized before constructing the depression composite. The process was used to minimize rater bias by compositing across raters and across different measures. The internal consistency reliability of the depression composite was .85.

Analyses

The effects of age, gender, and their interaction were removed from all scores by regression and age differences within sibling pairs were corrected for non-twin sibling pairs. Because the depression composite was skewed, the data were log transformed for the adolescent NEAD sample (Loehlin *et al.,* 2003). As an initial step, intraclass correlations were computed using double-entered data. An intraclass correlation is the correlation of twin/sibling 1 with twin/sibling 2, computed separately by twin/sibling type. The range of genetic relatedness varies from 0% among unrelated siblings, 25% half siblings in stepfamilies, 50% among full siblings in non-divorced families and stepfamilies and dizygotic twins, and 100% among monozygotic twins. By examining the pattern of intraclass correlations, genetic, shared environmental, and nonshared environmental influences can be anticipated. As a next step, model-fitting allows the twin and sibling covariances to be considered simultaneously. Univariate genetic analyses estimate genetic, shared environmental, and nonshared environmental influences in each measure.

The focus of this study is on the association between optimism and depressive symptoms. Figure 1 depicts the bivariate model used to partition the covariance between two variables. Using Figure 1 as a guide, the covariance between optimism and depressive symptoms is broken into three components, additive genetic (A), shared environmental (C), and nonshared environmental influences (E). Genetic influences and environmental influences on optimism were broken down into components



Note. A, C, and E represent genetic, shared environmental, and nonshared environmental influences respectively. a, c, and e represent genetic, shared environmental, and nonshared environmental influences specific to depressive symptoms.
MZ = monozygotic twins; DZ = dizygotic twins; FI = full siblings in non-divorced families; FS=full siblings in stepfamilies; HS=half siblings in stepfamilies; UN = unrelated siblings in stepfamilies.

Figure 1. *Bivariate Model for Optimism and Depressive Symptoms*

accounted by the genetic and environmental influences shared with depressive symptoms and the influences unique to optimism, represented by the paths a_{11}, a_{21}, c_{11}, c_{21}, e_{11}, and e_{21}. Specific genetic, shared, and nonshared influences on depressive symptoms independent of optimism are represented by a_{22}, c_{22}, and e_{22}.

The overall fit of each model was tested by χ^2 and Akaike's Information Criterion (AIC; Akaike, 1987). The best-fitting model can be evaluated by nonsignificant χ^2 and an AIC that is low. Previous research has found that χ^2 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 considers both the goodness of fit and parsimony, thereby providing a useful fit index to be used in addition to χ^2 (Williams & Holahan, 1994). Further discussion of fit indices is available elsewhere (Loehlin, 1992; Neale & Cardon, 1992). The models estimated in this study were examined using the statistical package Mx (Neale *et al.*, 2002).

We assume that shared and nonshared environmental effects are the same across twin/sibling types, and that the genotype-environment interaction is negligible. We also assume there is no selective placement of the stepsiblings as well as no assortative mating. A more detailed discussion of these assumptions for the NEAD sample can be found in Pike, McGuire, Hetherington, Reiss, and Plomin (1996). General discussions on assumptions of quantitative genetic model fitting can be found in Loehlin (1992) and Plomin, Defries, and McClearn (1990).

RESULTS

Genetic and Environmental Contributions to Optimism and Depressive Symptoms

Univariate maximum likelihood model-fitting analyses were conducted for optimism and depressive symptoms using the standard ACE model. Results from the full and best-fitting models for optimism and depression are summarized in Table 1. The AE model was fitted for optimism and the full model was fitted for depressive symptoms. For optimism, genetic effects accounted for 47%, with the remaining 53% accounted for by nonshared environmental effects. For the depressive symptoms, genetic effects accounted for 60% of the variance, with the remaining 40% accounted for by shared and nonshared environmental effects.

Genetic and Environmental Contributions to the Association between Optimism and Depressive Symptoms

Optimism was significantly and negatively associated with depressive symptoms, showing a phenotypic correlation of -.38. Genetic and environmental influences on the association between measures are limited by the size of the phenotypic correlation; the genetic and environmental influences on the covariance between two measures can only be present if the two measures covary at least modestly (Plomin *et al.*, 1990).

Standardized parameter estimates and 95% confidence intervals for bivariate genetic model-fitting of optimism and depressive symptoms are presented in Table 2. The bivariate model is decomposed into effects due to genes, shared environmental experiences that create twin and

Table 1. *Univariate Best Model-fitting Results for Optimism and Depressive Symptoms*

Measures	Parameter Estimates (95% CI)				
	A	C	E	χ^2 (*df*)	AIC
Optimism					
Full model	.47 (.26-.58)	.00(.00-.11)	.53 (.42-.67)	11.562 *(15)*	-18.438
Best-fitting model	.47 (.35-.58)	--	.53 (.42-.66)	11.562 *(16)*	-20.438
Depressive symptoms					
Full model	.60 (.41-.76)	.12(.00-23)	.27 (.29-.39)	42.807 *(15)*	12.807

Note. AIC represents Akaike's Information Criterion. A, C, and E represent additive genetic, shared environmental, and nonshared environmental effects respectively. Parameters are standardized. Parenthetic values give the 95% confidence intervals.

Table 2. *Bivariate Model-fitting between Optimism and Depressive Symptoms*

	Common to optimism, and depressive symptoms			Unique to depressive symptoms			Fit indices
	A	C	E	A	C	E	
Optimism	.70 (.54-.77)	.05 (.00-.32)	.72 (.64-.80)				$\chi^2(df)$ = 83.805 (51) AIC = -18.195
Depressive symptoms	.41 (.22-.56)	.36 (.00-.48)	.10 (.00-.21)	.65 (.46-.76)	.00 (.00-.46)	.52 (.43-.62)	

Note. AIC represents Akaike's Information Criterion. A, C, and E represent additive genetic, shared environmental, and nonshared environmental effects respectively. Parameters are standardized. Parenthetic values give the 95% confidence intervals.

sibling similarity in both measures, and nonshared environmental experiences that make only one sibling vulnerable (O'Connor *et al.,* 1998). As can be seen by the model-fit indices, the full model provided an adequate fit to the data with a negative AIC value. The full model includes all parameters, while the constrained models have set all of the shared environmental paths to be zero. The results from the full model are interpreted because the differences in chi-square between full model and constrained model are not significant and that the parameter estimates between optimism and depressive symptoms are relatively sizable.

The first noteworthy finding was that genetic and nonshared environmental influences explained adolescents' optimism and depressive symptoms. In optimism, approximately 50% of variances were attributed to genetic and nonshared environmental factors, respectively; whereas in depressive symptoms, approximately 60% of the variances were attributed to genetic factors in depressive symptoms, and 40% were explained by shared and nonshared environmental factors. Second, genetic influences on the covariance between optimism and depressive symptoms were substantial. Specifically, the bivariate analyses revealed that genetic factors were an important explanation for the association between optimism and depressive symptoms. The sum of multiplying the path coefficients for genetic, shared environmental and nonshared environmental factors common to optimism and depressive symptoms produces the phenotypic correlation between the two measures. For example, (.70 X .41) + (.05 X .36) + (.72 X .10) in the full model, was within rounding error of the phenotypic correlation of .38. Finally, there were genetic and nonshared environmental influences on depressive symptoms that were not correlated with optimism.

DISCUSSION AND CONCLUSIONS

This study investigated genetic and environmental contributions to optimism, depression, and the association between the two, using a genetically informative sample. With regard to the first research question, the findings suggest that genetic factors are important in explaining individual differences of optimism and depression. Limited studies have assessed the genetic and environmental factors of optimism; however, one exceptional study on older adult twins demonstrated that genetic factors play an important role in optimism (Plomin *et al.,* 1992). Consistent with the previous study, this study showed that genetic factors explained approximately half of the variability in adolescent optimism.

Although the relative contributions of genetic and environmental factors vary depending on age, respondent, and the severity of the depressive symptoms (Garber & Flynn, 2001), there has been a wealth of research supporting the view that genetic factors play an important role in depression. Longitudinal studies on familiar patterns of depression have also indicated that children of parents with an affective disorder or depression have a higher rate of depression than children of parents without such conditions, ranging from 26% (Beardslee *et al.,* 1993) to 50% (Weissman *et al.,* 1997) and suggest a possible genetic risk for depression. Consistent with previous literature, these findings highlight the importance of genetic factors in influencing depression.

Although genetic factors play an important role in optimism, the nonshared environment contributes substantially to individual differences in optimism and depression. Previous twin and family studies reported substantial genetic and nonshared environmental influences on depressive symptoms (Gatz *et al.,* 1992; Kendler *et al.,* 1994; McGue & Christensen, 1997). Consistent with previous studies on depression, univariate model-fitting analyses revealed the significant nonshared influences for depression. Unique social experiences in adolescents, such as school or peer experiences, may favor the development of optimism and depression. Further studies are needed to identify the specific nonshared environmental influences.

With regard to the second research question, multivariate analysis indicated that the liabilities to both optimism and depressive symptoms are linked by common gene influences. In a previous study that combined twin and adoption design, genetic factors were found to account for more than one-third of the phenotypic associations among optimism, pessimism, and depression in older adults (Plomin *et al.,* 1992). Consistent with the previous study, the findings of the current study support the claim that genetic factors play an important role in the association between adolescents' optimism and depressive symptoms, and suggest that optimism is a potential genetic vulnerability to depression. Finally, it is important to note that there were genetic and nonshared environmental influences on depressive symptoms that were not correlated with optimism. Future studies are needed to explore genetic and environmental influences on depressive symptoms in the association with various individual risk factors.

This study had several limitations. First, the findings in this study (based on depressive symptoms in community samples) may be incapable of being generalized as clinical depression. Second, this study is cross-sectional. Although studies have indicated that optimism influences depressive symptoms (Nolen-Hoeksema *et al.,* 1992; Robinson *et al.,* 1995; Brissette *et al.,* 2002), longitudinal behavior genetics data would allow for the examination of how optimism is associated with depression over time. The longitudinal data would also enable us to examine genetic and environmental influences on the stability and change of the association. Third, there may be a possibility that optimism may be a cognitive tendency or a consequence of depressive symptoms. Although previous studies have suggested that optimism is a relatively stable trait and a strong predictor of major depression (Brissette *et al,* 2002; Scheier & Carver, 1985), one needs to be careful in ruling out mood state biases or implying causation.

Despite these limitations, this study support and extend the previous literature on dispositional optimism and depressive symptoms in adolescents. In line with previous review (Sullivan *et al.,* 2000), this study has showed that genetic and nonshared environmental origins play a role in explaining individual differences of depressive symptoms. In addition, the current study has expanded previous research on optimism by estimating genetic and nonshared environmental influences of optimism in adolescents. The findings will help increase the understanding of individual differences in optimism among adolescents. The finding that genetic factors influence optimism and depressive symptoms suggests that future studies need to focus on investigating candidate genes as well as transforming process into symptomatology. Identifying the specific nonshared environmental influences that contribute to the shaping of optimism and depressive symptoms would also be helpful for the development of interventions. Further, genetic influences underlying optimism seemed to mediate (at least partially) genetic influences on depressive symptoms. The results of this behavior genetic analysis help us to clarify the underlying nature of the relationship between optimism and depressive symptoms as well as provide information on their developmental origins in adolescence.

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