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Self-Esteem, Negative Emotionality, and Depression as a Common Temperamental Core: A Study of Mid-Adolescent Twin Girls

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Abstract

We tested the structure and magnitude of genetic and environmental influences on the overlap among self-esteem, negative emotionality, and major depression symptoms in adolescent girls (N = 706) from the Minnesota Twin Family Study. Genetic and environmental influences on all three operated via a general, heritable factor. Genetic influences explained the majority of overlap among the three constructs, as well as most of the variance in self-esteem and negative emotionality. Genetic influences on depression were more modest, and largely due to genetic factors specific to depression. These findings support the theory that self-esteem, depression, and neuroticism represent aspects of a common temperamental core. The interrelations among the three constructs in mid-adolescence is consistent with their interrelations in adulthood.

Keywords

Self-Esteem; Depression; Neuroticism; Behavior Genetic; Adolescence

It is both a psychological truism and a psychological fact that people who feel better about themselves are also more emotionally stable and less prone to depression (Sedikides, Gregg, & Hart, 2007; Sedikides, Rudich, Gregg, Kumashiro, & Rusbult, 2004; Taylor, Lerner, Sherman, Sage, & McDowell, 2003). Attempts to delineate the structure of the relation among self-esteem, neuroticism, and depression, however, rest on shaky ground. One difficulty with the empirical literature is the wide diversity in models concerning the relations among the three constructs. For example, self-esteem has been conceptualized as both cause (Roberts, Kassel, & Gotlib, 1995) and effect (Kistner, Ziegert, Castro, & Robertson, 2001) of depression, or as anchoring one end of a bipolar continuumopposite depression (Watson, Suls, & Haig, 2002). Neuroticism may predispose people to depression (Krueger, McGue, & Iacono, 2001) or depression may "scar" people, leading to increased levels of neuroticism (Fanous, Neale, Aggen, & Kendler, 2007; Hirschfeld et al., 1983; Kendler, Neale, Kessler, Heath, & Eaves, 1993). Positive self-views are markers of both self-esteem and the absence of neuroticism (Eysenck, 1990), and self-esteem and neuroticism are markers of a broader core construct (Judge, Erez, Bono, & Thoresen, 2002). Thus, given their apparent interchangeability, might

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it be possible that self-esteem, depression, and neuroticism are aspects of a single, core construct?

Conceptually, self-esteem, depression, and neuroticism overlap. Self-esteem is the overall evaluation of the self (Blascovich & Tomaka, 1991; Sedikides & Gregg, 2003), reflecting how much individuals accept and like themselves. Clinical depression is marked by a period of dysregulated mood, thinking, sleep, appetite and behavior. Notably, feeling worthless or helpless is one of the clinical symptoms. Although terminology may differ, a factor corresponding to neuroticism is found across multiple personality theories (Watson, Clark, & Harkness, 1994), including the "Big Five" (McCrae & Costa, 1987) and "Big Three" models (Tellegen, 1985). Generally, neuroticism refers to an enduring trait marked by emotional instability and a propensity to interpret events negatively. Characteristics of someone high on neuroticism include a tendency to experience anxiety, depression, self-criticism, negativisitic appraisal of events, and somatic complaints (Watson et al., 1994). In other words, people who are low in self-esteem, depressed, or neurotic all share a tendency to feel negatively about the self. In addition, depression and neuroticism share more general negative affective tendencies. Despite the conceptual overlap, however, the three constructs are most often viewed as separable and distinct.

Theoretical accounts may well justify complex interrelations among self-esteem, depression, and neuroticism. We highlight just a few such accounts to illustrate purported connections among the three constructs. Terror Management Theory (Pyszczynski, Greenberg, Solomon, Arndt, & Schimel, 2004) posits that self-esteem functions as an anxiety buffer to protect psychological well-being in the face of reminders of our own mortality. Neurotic individuals are thought to be especially susceptible to mortality cues related to the physicality of the body (Goldenberg et al., 2006), which may in turn affect their self-esteem strivings and mood. Cognitive models of depression note that people who attribute negative events to internal (directed toward the self), stable, and global causes are at increased risk for depression (Abramson, Seligman, & Teasdale, 1978). A negative attributional style (one of the characteristics of neurotic individuals) is more problematic in the face of negative life events for individuals with low self-esteem (Metalsky, Joiner, Hardin, & Abramson, 1993).

Thus, perhaps we should not be surprised that empirical studies support multiple models of these interrelations. Nonetheless, a few studies suggest that some caution should be applied to the accumulation of complex models. For example, factor analytic results indicate that self-esteem, depression, and neuroticism form a single factor in a model that includes other depression-related cognitive styles such as rumination or dysfunctional attitudes (Hankin, Lakdawalla, Latchis Carter, Abela, & Adams, 2007). This finding is consistent with research showing that self-esteem and neuroticism load on a common factor (Judge et al., 2002) or that self-esteem, neuroticism, and depression load on the same factor, albeit one that includes other scales such as loneliness, anxiety, and locus of control (Hojat, 1982). One explanation for these findings is that self-esteem, depression, and neuroticism represent aspects of a common temperamental core. That is, a stable and consistent set of thoughts, feelings and behaviors leads simultaneously to lower self-esteem, higher depression, and greater neuroticism (or, alternatively, to higher self-esteem, more positive emotions, and greater emotional stability).

The behavior genetic perspective offers an important method for assessing the *common temperamental core* hypothesis. Numerous studies document substantial genetic influence on self-esteem, depression, and neuroticism (e.g., Fanous et al., 2007; Neiss, Sedikides, & Stevenson, 2002, 2006; Roberts & Kendler, 1999). Here, we focus on multivariate behavior genetic studies that examine the genetic and environmental architecture underlying these three constructs, although few genetic studies have included all of them in one model. Such studies allow researchers to decompose the covariation among constructs in order to understand their

common and their distinct genetic and environmental etiology. These studies, then, can provide new insight into the origins of the observed relations among these constructs.

Two longitudinal analyses of twins found that neuroticism predicted subsequent onset of depression in twins with no prior history and that occurrence of a major depression episode increased levels of neuroticism in phenotypic models (Fanous et al., 2007; Kendler et al., 1993). However, after modeling shared genetic and environmental factors, the direct causal path from neuroticism to major depression was non-significant in both studies. Rather, the overlap among the two constructs was due largely to shared genetic and environmental factors. In men, genetic influences on neuroticism and depression overlapped entirely (Fanous et al., 2007), whereas women showed specific genetic influences on neuroticism and depression in addition to the substantial overlap in genetic factors between the two (Kendler et al., 1993). Together, these results illustrate that relations found at the observed (phenotypic) level may not replicate after taking into account genetic etiology. In addition, the results support the idea that the relation between neuroticism and depression is explained by shared genetic etiology.

Other behavior genetic studies also demonstrate genetic overlap between self-esteem and negative affectivity (Neiss et al., 2005), or between depression and neuroticism (Hettema, Neale, Myers, Prescott, & Kendler, 2006; Kendler, Gardner, Gatz, & Pedersen, 2007). In one study that modeled explicitly the relation among self-esteem, depression, and neuroticism, a common genetic factor explained most of their covariation (Roberts & Kendler, 1999). By comparing two Cholesky decompositions that varied the order of constructs, Roberts and Kendler concluded that self-esteem had no remaining genetic links with depression, after accounting for the common genetic factor. Neuroticism and depression, however, shared a separate genetic link. As a whole, behavior genetic studies establish substantial genetic overlap among neuroticism, self-esteem, and depression. It is unclear, however, whether this genetic overlap represents common genetic factors that influence all three constructs simultaneously, or whether the constructs are best understood as lower-order factors of one broad latent variable. This is the issue that the present study seeks to clarify. In addition, the present study aimed to extend past research by examining the genetic and environmental structure in a younger population.

In particular, we tested the genetic and environmental structure underlying the covariance among depression, neuroticism, and self-esteem in a sample of adolescent female twins. We compared behavior genetic common pathway and independent pathway models to clarify whether self-esteem, neuroticism (operationalized as negative emotionality), and depression are influenced separately by common genetic and environmental influences or whether the covariation among the three is determined by genetic and environmental influences operating through a single latent phenotypic variable (e.g., broad Neuroticism or Negative Affectivity).

Method

Participants

Participants were female twins recruited to the Minnesota Twin-Family Study (MTFS), a population-based, longitudinal investigation of twin adolescents and their families. Boys did not complete the self-esteem questionnaires and thus are not included in the current study. Detailed information about the full MTFS is available elsewhere (Iacono, Carlson, Taylor, Elkins, & McGue, 1999). The current study included the younger cohort of girls, who ranged in age from 10 to 12 at their initial visit. We investigated those twins who participated in the first follow-up visit, which occurred about 3 years after the initial intake (M_{interval} = 3.05 yrs, *SD* = .28), when participants were about 14 years old (M_{age} = 14.77, *SD* = .57). We excluded 54 girls from the sample of 760 participants due to invalid or missing data for all of our study variables (i.e., depression, self-esteem, negative emotionality). The final sample of 706 girls

included 431 members of monozygotic (MZ) pairs and 275 members of dizygotic (DZ) pairs. We fit behavioral genetic models to raw data, thus retaining in the analyses individuals from incomplete pairs and individuals with partial data.

Zygosity Determination

Zygosity determination relied on information from three separate estimates. Indices of physical similarity included parental report, assessment by study staff (e.g., evaluations based on visage, hair color, or ear shape), and an algorithm based on ponderal and cephalic indices and fingerprint ridge counts. Twins were asked to provide a blood sample for serological analysis when the three estimates did not agree.

Measures

Self-esteem—Participants completed the Piers-Harris Children's Self-Concept scale (Piers, 1984). This is an 80-item self-report inventory which assesses how children and adolescents feel about themselves in several domains. Higher total scores indicate greater self-esteem. Participants responded with a "yes" or "no" to whether each item was self-descriptive. Self-esteem was negatively skewed, so we reflected and log transformed self-esteem scores (Tabachnick & Fidell, 2001). High scores thus, in our study, indicate low self-esteem, a reversed scoring we maintained to limit negative path coefficients in the genetic models.

Negative emotionality—Participants completed the Personality Booklet – Youth, Abbreviated, a 133-item version of the MPQ (Tellegen, 1982) developed to increase its suitability for younger participants. This version has psychometric properties similar to the full-length adult form of the MPQ in longitudinal research with these same adolescent girls (Johnson, Hicks, McGue, & Iacono, 2007). The MPQ consists of relatively independent primary scales, with three higher order factors: Positive Emotionality (PEM), Negative Emotionality (NEM), and Constraint. NEM is closely associated with neuroticism (Tellegen et al., 1988). Tellegen's model of personality emphasizes the connection between NEM and broad negative affectivity (Patrick, Curtin, & Tellegen, 2002; Watson, Wiese, Vaidya, & Tellegen, 1999), reflected in both dispositions toward negative emotions and to neurobiological-based withdrawal behaviors (Watson et al., 1999). In Tellegen's model, NEM manifests via susceptibility to negative emotional states, such as anxiety, resentment, and anger, and negative interpersonal behaviors such as aggression (Patrick et al., 2002). We summed the alienation, aggression, and stress reactivity sub-scales to create a total Negative Emotionality score (NEM). High scorers experience strong negative emotions, and described themselves as stressed and harassed.

Depression—At the follow-up visit, researchers independently interviewed each twin and their mothers to assess depressive symptoms experienced by the teen during the interval since the initial visit. Symptoms of DSM-III-R major depressive disorder were assessed using a modified version of the Diagnostic Interview for Children and Adolescents-Children or Parent version (DICA-C or DICA-P; Reich & Welner, 1988). We used depression symptom counts based on the "best estimate" procedure, which combined information from both child and mother report (see Iacono et al., 1999). This approach makes the best use of all available information to calculate a composite symptom count. When more than 50% of the items used to formulate the symptom counts were missing, we considered the symptom count invalid and set the total to missing. Given that most participants had no depression symptoms, symptoms counts were positively skewed. We used a Blom transformation to transform and rank normalize the depression symptom count prior to model fitting. A simulation study by van den Oord et al. (2000) reported that rank transformation aids in identifying the correct model in behavior genetic analyses of symptom scores. All three measures were z-scored so that they were expressed in similar units.

Statistical Analyses

Behavior genetic models compare the within-pair resemblance of MZ twins, who share 100% of their genetic material, with that of DZ twins, who share on average 50% of their genetic material. Our models included additive genetic influences (a^2), shared environment (c^2), and non-shared environmental influences (e^2). Greater sibling resemblance among MZ twins as compared to DZ twins indicates genetic influences, and within-pair similarity not resulting from genetic factors is ascribed to shared environmental influences. Non-shared environmental influences create differences among individuals from the same family, and include also measurement error.

Multivariate behavior genetic models decompose the covariance between measures into common genetic and environmental components. The common pathway model posits a hierarchical structure, with common genetic and environmental influences on our measures operating through a higher-order latent phenotypic variable (e.g., broad Neuroticism or Negative Affectivity; See Figure 1). In contrast, the independent pathway model allows for common genetic and environmental influences on self-esteem, NEM, and depression via independent paths (See Figure 2). Both models allow for genetic and environmental influences specific to each phenotype. Comparison of these two models allowed us to test whether self-esteem, NEM, and depression can be seen as indices of one higher-order construct. The hierarchical structure of the common pathway model is more parsimonious than that of the independent pathway model in that it has fewer parameters to explain the covariance between the measures.

We fit structural equation models to raw data using maximum likelihood estimation, which allowed us to use all valid data even if some observations were missing for particular individuals. We conducted the structural equation modeling using Mx software (Neale, Boker, Xie, & Maes, 2003). Fitting models to raw data provides a -2LL (twice the negative log-likelihood) statistic. Subtracting this statistic from the -2LL obtained from a saturated model, one estimating the means, variances, and covariances freely, provides both the chi-square index and Akaike's information criteria (AIC) to assess the degree of fit. We also used the Bayesian Information Criteria (BIC) to compare fit across models. Lower BIC values indicate better fit (Raftery, 1995).

Results

Inspection of the variables prior to transformation revealed that the girls showed relatively positive self-esteem (M = 142, out of a maximum score of 160). Average NEM scores fell somewhat under the midrange of the scale (M = 112, possible range of 54 to 216). The majority of girls (87%) showed no depressive symptoms and the mean number of depression symptoms was 3.63 among the 13% of girls reporting one or more symptoms. The depressive symptom counts corresponded to 4.3% of the girls having had a major depressive disorder episode over the 3-year interval assessed at this visit. Thus the girls are representative of the general population on these measures, as self-esteem scores are usually well above scale means (Baumeister, Tice, & Hutton, 1989; Sedikides et al., 2004). In addition, epidemiological studies find that point estimates of clinical depression rates in children and adolescents range from . 4% to 6.4% (Costello, Mustillo, Erkanli, Keeler, & Angold, 2003; Fleming & Offord, 1990). Less information exists about adolescent norms for neuroticism, but other researchers have reported relatively low values in non-clinical samples of young adolescents (Muris, 2006). Remaining analyses use transformed variables as described above.

Table 1 displays the intraindividual and cross-twin correlations separately for MZ and DZ twins. Correlations outside of the demarcated boxes show the intraindividual relations among self-esteem, NEM, and depression. Twins pairs are separated arbitrarily into twin 1 (indicated

by 1) and twin 2 (indicated by 2). In order to report phenotypic correlations based on the entire sample, we used mixed models to estimate simultaneously the bivariate relations across all individuals (PROC MIXED procedure in SAS 9.1, SAS Institute Inc.). Given that individuals in twin pairs are not sampled independently, we adjusted the standard errors using the Huber/White "sandwich estimator" (Maas & Hox, 2004). Self-esteem was related to both depression ($b = .19$, $p < .001$) and NEM ($b = .65$, $p < .001$), such that people with low self-esteem also had more depressive symptoms and higher NEM scores. In addition, people with more depressive symptoms evidenced higher NEM ($b = .21$, $p < .001$).

Behavior Genetic Analyses

The cross-twin correlations, both within and between traits, are demarcated by the solid boxes in Table 1. The cross-twin correlations within traits are highlighted by the dotted lines. The MZ twins showed greater resemblance in self-esteem, and slightly greater resemblance in NEM, but not in depression. This pattern of results suggests genetic influence on self-esteem, perhaps on NEM, and shared environmental influences on depression.

We tested common pathway and independent pathway models to clarify the genetic and environmental structure relating self-esteem, NEM, and depression. Table 2 provides fit estimates for both models, based on comparison of each to a saturated model. The common pathway model provided a better fit to the data, as evidenced by lower AIC values and greater parsimony. Comparison of the BIC values provides further support for the common pathway model. Lower BIC values indicate better fit, with the observed difference here of about 9 indicating strong evidence in favor of the common pathway model (Raftery, 1995). This model suggests that the covariance among self-esteem, NEM, and depression can be explained by genetic and environmental influences on a latent, broad, and heritable personality factor. Figure 3 provides the path estimates from the full common pathway model. We tested also several sub-models to check for the significance of the genetic and shared environmental factors. Dropping the common genetic path to the latent phenotype worsened significantly the model, but dropping the common shared environmental path did not do so. In fact, it was possible to drop all shared environmental paths and still result in a model with a negative AIC value, suggesting a more favorable fit overall.

Figure 4 illustrates the proportion of total variance ascribed to common and specific influences in the final reduced model. We present the estimates for the latent temperament factor, which we have termed Core Negative Affect (NA), as well as the specific observed phenotypes. Core NA shows evidence of strong genetic influences (69%; 95% Confidence Interval [CI] 59 – 78%), suggesting that genetic factors explain a large amount of the variance in this higher order personality construct. The remaining variance can be attributed to non-shared environmental influences, which are environmental influences unique to each person or measurement error (30%; CI 22 – 41%).

The substantial total genetic influences on self-esteem and NEM stemmed primarily from genetic influences via Core NA. The specific genetic effects on both self-esteem (14%; CI 0 – 26%) and NEM (8%; CI 0 – 22%) were modest and not statistically significant. Depressive symptoms had modest overlap with self-esteem and NEM; of the overlap, however, the majority was due to genetic influences. The smaller overlap arose because of a relatively low loading for depressive symptoms on the latent phenotype. Genetic influences on depression stem more from specific genetic influences (24%; CI 12 – 35%). Total genetic influences (sum of common and specific parameter estimates) were 59%, 57%, and 29% for self-esteem, NEM, and depressive symptoms, respectively. Specific non-shared environmental influences explained the majority of variance in depression (69%; CI 57 – 82%). This suggests that the unique environmental events in these girls' lives account for their depressive symptoms.

Discussion

In the bulk of the relevant literature, self-esteem, depression, and neuroticism have been linked causally and correlationally (Roberts et al., 1995). In another research tradition, higher-order factors have been postulated. These factors encompass self-esteem and depression (Watson et al., 2002), self-esteem and neuroticism (Judge et al., 2002), or self-esteem, depression, and neuroticism (Hankin et al., 2007). Our research falls in the latter tradition. In particular, our research complements that of Hankin et al. (2007) and Hojat (1982). Whereas these researchers implemented factor analytic techniques to address the issue of a common temperamental core, we used behavior genetics methodology.

Our results are consistent with the common temperamental core hypothesis. Self-esteem, neuroticism, and depression emerged as facets of a general, heritable factor. Although our behavior genetic models did not test the underlying phenotypic structure of the three constructs, our finding indeed extends past factor analytic work on the relations among them (Hankin et al., 2007; Hojat, 1982). In particular, our behavior genetic models tested specifically whether the shared variance among self-esteem, neuroticism, and depression was best explained by independent genetic and environmental effects or genetic and environmental influences via a common pathway. The results support the presence of a phenotypic latent variable, which is consistent with the hypothesis of a common latent temperamental core.

Our finding that the genetic and environmental links among self-esteem, neuroticism, and depression stem from a common latent core construct supports another behavior genetic study that found strong genetic overlap among these three (Roberts & Kendler, 1999). Our work extends this to a sample of adolescent girls, and our comparison of two competing models provides a stronger test of the underlying genetic and environmental structure. Specifically, a broad, heritable factor explains the overlap among self-esteem, neuroticism, and depression. Furthermore, the overlap among all three stems primarily from common genetic etiology. We speculate that the latent phenotype modeled in our research is broad Negative Affectivity or Neuroticism (Core NA). A large body of work points to the importance of personality in explaining the onset and course of psychiatric disorders (Brown, 2007; Clark, Watson, & Mineka, 1994) and comorbidity of disorders (Khan, Jacobson, Gardner, Prescott, & Kendler, 2005; Krueger et al., 2002). We suggest that a similar recognition of the links among self-esteem, personality, and depression in non-clinical samples might help researchers winnow down the existing proliferation of models and bring greater understanding of the underlying relation among these constructs.

Our conclusion echoes arguments put forth by others (Judge et al., 2002; Watson et al., 2002), in that we propose a common temperamental core underlies self-esteem, negative emotionality, and depression. Our work extends previous findings by identifying a specific mechanism for their covariation in shared genetic etiology. We note that genetic or environmental effects may explain shared covariance; for example, specific negative events provide plausible explanations for the co-occurrence of low self-esteem, emotional instability, and depressive symptoms. Stated otherwise, although we expected genetic influences to explain variability in each construct, it is not the case that shared genetic influences must thus necessarily explain their covariation. This is shown by the association between reading disability and antisocial behavior, where although genetic factors play a significant role in both, their co-occurrence is accounted for by shared environmental influences (Trzesniewski, Moffitt, Caspi, Taylor, & Maughan, 2006).

Given the shared genetic influences underlying these personality traits, we would expect to find specific genotypes linked to all three. Recognition of genetic links between personality and clinical disorders has informed recent molecular genetic work. For example, research by

Hettema, An, Neale and their colleagues (2006) show a link between *GAD1* and a latent phenotype based on common genetic factors underlying neuroticism, major depression, and anxiety disorders. *GAD1* contributes to the production of glutamic acid decarboxylase (GAD), which synthesizes gamma-aminobutyric acid (GABA), a neurotransmitter implicated in mood disorders (Hasler et al., 2007). An important next step would be to investigate whether *GAD1* is associated with self-esteem as well.

Limitations

In our study, depression showed modest correlations with NEM and self-esteem. Several factors may account for this. One methodological issue is that the depression variable includes child and mother reports, which may result in lower correlations between it and measures based solely on child report. In addition, the interview assessed clinical symptoms of depression during the 3 year interval from the previous visit. Self-ratings of self-esteem and personality might be more strongly related to current symptomatology as compared to symptoms over a longer time span. One result of the modest interrelations was the modest loading of depression on the latent temperamental factor. We note, however, that we might have thus expected the independent pathway model to be the better fitting model, which was not the case.

Given the young age of the girls, our depression variable does not account for lifetime prevalence of depression. Thus, the genetic and environmental estimates from the current study reflect only estimates of the magnitude of genetic and environmental influences during this stage of the participants' lives. However, adolescence is an important period in the development of self-esteem for girls (Wigfield, Eccles, Iver, Reuman, & Midgley, 1991), a time when most have experienced puberty and when the prevalence of depression is markedly increased (Nolen-Hoeksema & Girgus, 1994). Interestingly, our findings indicate that by this early point in life, the nature of the relation among self-esteem, negative emotionality, and depression is very much in line with what other investigators have observed for adult samples. Our findings suggest that, despite developmental changes occurring in all three areas, a heritable core temperament is a stable feature of the relation among self-esteem, negative emotionality, and depression. Future longitudinal research may investigate whether the shared genetic influences contribute to the understanding of developmental changes in all three constructs as well as of between-person stability. Such work would enhance our knowledge of the underlying developmental processes. Moreover, it would be important to incorporate multiple time-points and multiple raters (e.g., parental report of personality as well as depressive symptoms) for the elucidation of the structure of core NA. The inclusion of other well-being measures in such a study would allow the question to be addressed as to whether the broad measure of core NA better predicts long term outcomes than the three individual constructs.

Conclusion

Our results showed that self-esteem, neuroticism, and depression form a general, heritable factor which we label Core Negative Affect (Core NA). The majority of variance in both self-esteem and neuroticism stemmed from common genetic and non-shared environmental influences operating via this general factor, although depression showed evidence of substantial separate influences. Our results suggest that researchers should consider the overlapping genetic and environmental etiology that may influence simultaneously self-esteem, neuroticism, and depression.

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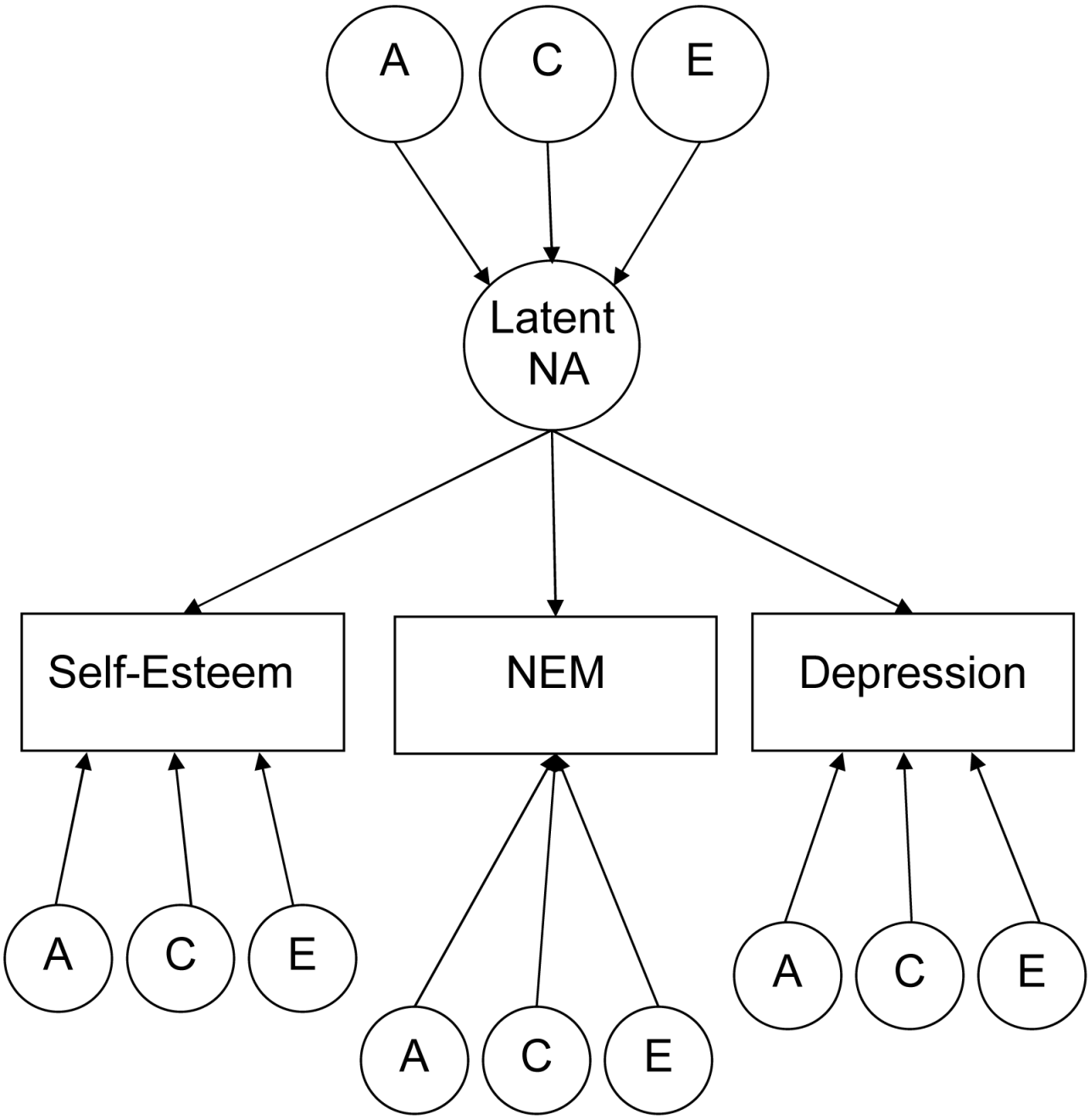

Figure 1.
Path Diagram Illustrating the Common Pathway Model

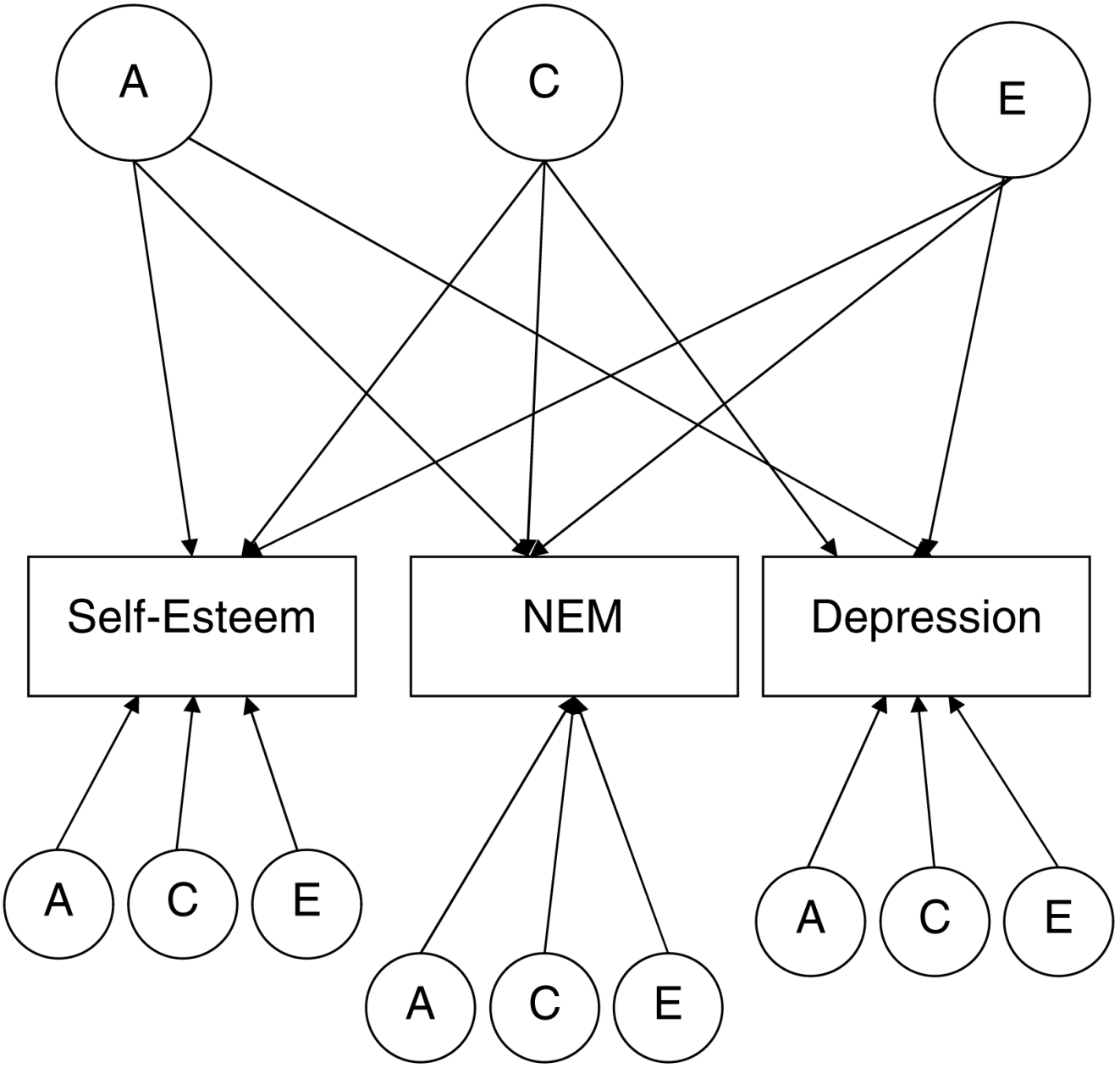

Figure 2.
Path Diagram Illustrating the Independent Pathway Model

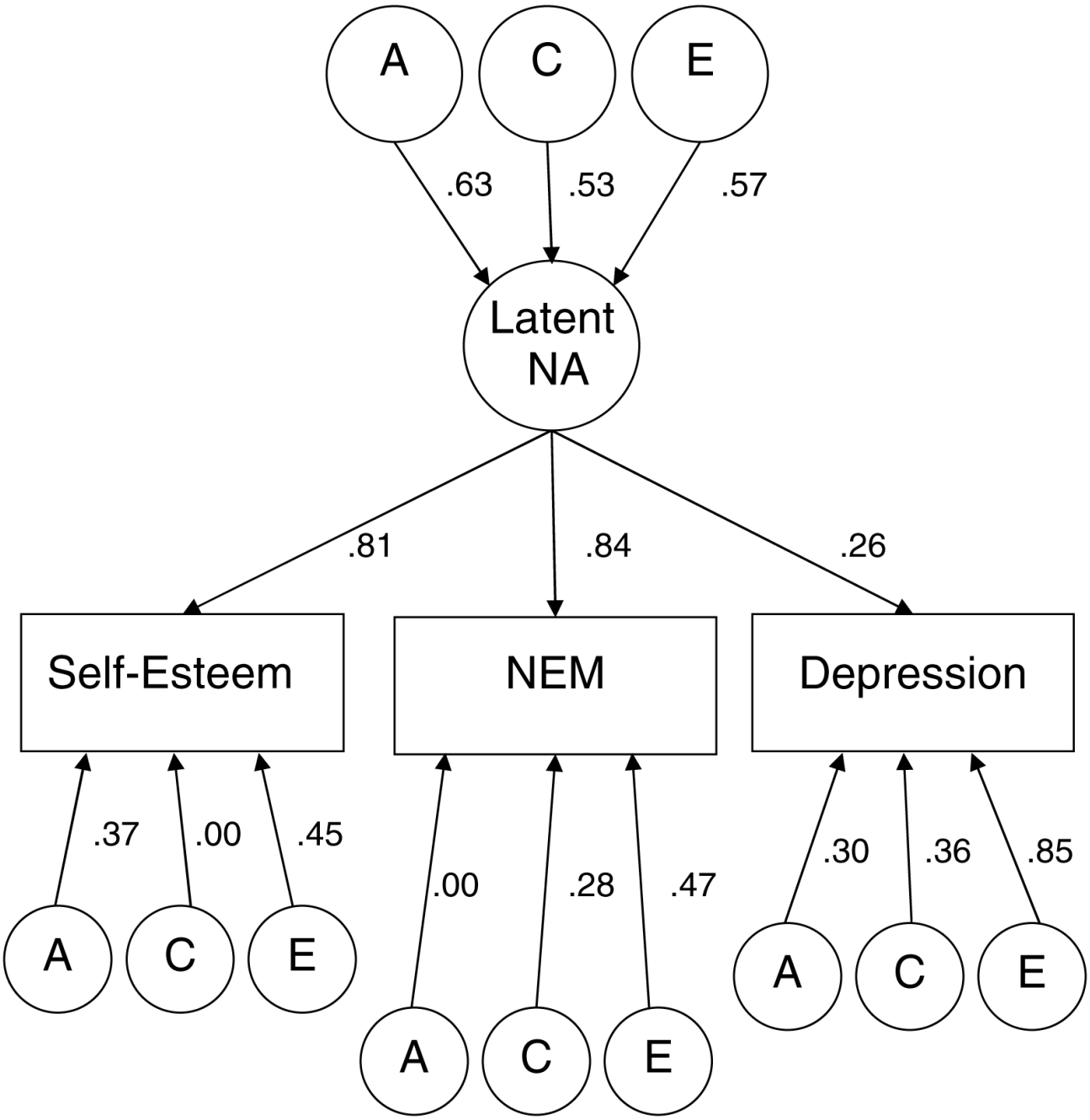

Figure 3.
Standardized Path Estimates from Full Common Pathway Model

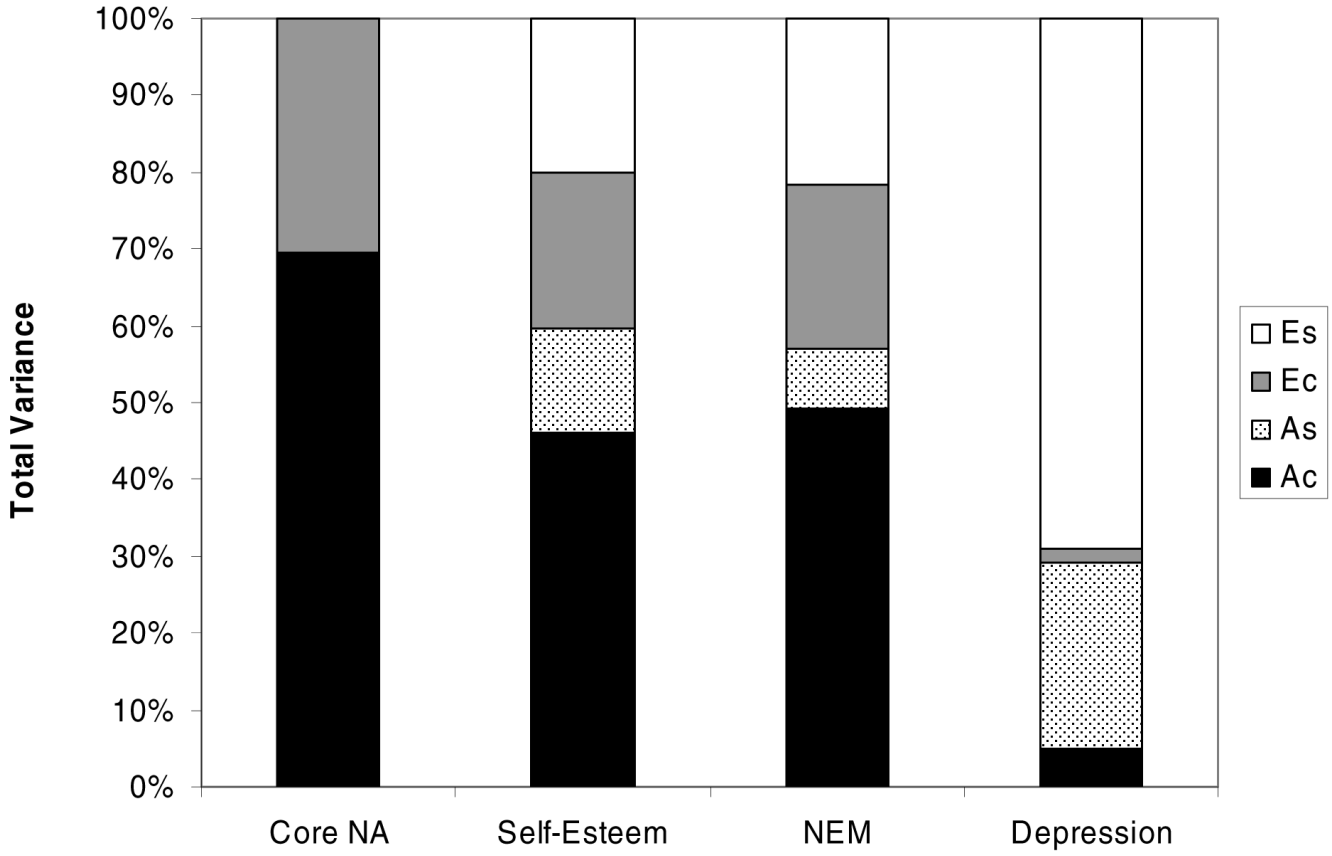

Figure 4. Genetic and Environmental Estimates from Final Model

Note. Bars represent total variance in each measure or latent construct. Segments display proportion of the total variance accounted for by genetic and environmental parameters. Ac = Common Genetic Influences; As = Specific Genetic Influences; Ec = Common Non-Shared Environmental Influences; Es = Specific Non-Shared Environmental Influences.

Table 1

Descriptive Information and Twin Resemblance for Self-Esteem, NEM, and Depression

Variable	Self-Esteem_1	NEM_1	Depression_1	Self-Esteem_2	NEM_2	Depression_2
DZ Twins						
Self-Esteem_1	-					
NEM_1	.74***	-				
Depression_1	.24**	.27**	-			
Self-Esteem_2	.37***	.39***	.19*	-		
NEM_2	.33***	.47***	.32***	.76***	-	
Depression_2	.07	.16	.27**	.30**	.27**	-
MZ Twins						
Self-Esteem_1	-					
NEM_1	.61***	-				
Depression_1	.14*	.16*	-			
Self-Esteem_2	.57***	.39***	−.04	-		
NEM_2	.45***	.52***	−.06	.64***	-	
Depression_2	.12	.12	.19*	.13	.17*	-

Note. Twin1 and Twin2 are differentiated by the endings _1 and _2, respectively.

* $p < .05$

** $p < .01$

*** $p < .001$.

Table 2

Model Fitting Results Comparing Common Pathway and Independent Pathway Model

Model	−2LL	Df	BIC	Change From Full Model		AIC
				ΔX2	*p*	
Independent	5068.30	1976	−3264.71	-	-	1116.30
Common	5072.61	1980	−3274.30	-	-	1112.61
No Ac	5077.41	1981	−3274.83	4.80	<.05	2.80
No Cc	5074.95	1981	−3276.30	2.33	.13	.33
No Cc or Cs	5077.33	1984	−3283.68	4.72	.32	−3.28

Note. Ac = Common Genetic paths; Cc = Common Shared Environment Paths; Cs = Specific Shared Environment Paths.