Published in final edited form as:
Twin Res Hum Genet. 2006 February ; 9(1): 24–29. doi:10.1375/183242706776403028.

The Genetic Architecture of Neuroticism in 3301 Dutch Adolescent Twins as a Function of Age and Sex: A Study From the Dutch Twin Register

David C. Rettew[1], **Jacqueline M. Vink**[2], **Gonneke Willemsen**[2], **Alicia Doyle**[1], **James J. Hudziak**[1], and **Dorret I. Boomsma**[2]

[1]Department of Psychiatry, University of Vermont College of Medicine, Burlington, Vermont, United States of America [2]Department of Biological Psychology, Vrije University, Amsterdam, the Netherlands

Abstract

The objective of this study was to estimate the magnitude of genetic and environmental influences to variation in adolescent neuroticism as a function of age and sex. Neuroticism was assessed using the Amsterdamse Biografische Vragenlijst (ABV): a self-report personality instrument similar in content to the Eysenck Personality Questionnaire. Genetic modeling procedures, including age as modifier, were fitted to the total sample of 3301 Dutch adolescent twins aged 12 to 17 years (mean age 15.5). Significant influences of additive genetic factors (.59, 95% confidence intervals [CI] .54–.63) and unshared environmental factors (.41, 95% CI .37–.45) were found. Our data did not support a role of shared environment. Results showed that different genes may influence variation in neuroticism between girls and boys. No interaction was found between the variance components and age. Results generally support prior findings in adults and young children that neuroticism is influenced principally by additive genetic and unique environmental factors. The magnitude of the genetic component appears higher in the present sample of adolescents than in most studies of adults. The present study suggests that, in adolescence, different genes are expressed in boys and girls.

The personality trait of neuroticism refers to the relative tendency to experience negative emotions such as fear, sadness, and anger. Over the years, this trait has been variably referred to as neuroticism (Costa & McCrae, 1985; Eysenck & Eysenck, 1975), negative affectivity (Rothbart et al., 2000), or emotionality (Buss & Plomin, 1984), among others. The existence of this dimension has been well supported, and neuroticism has been studied extensively in part because of its links to psychopathology, particularly anxiety and depression (Middeldorp et al., in press).

The current diagnostic nomenclature presupposes the association between personality dimensions and Axis II but not Axis I disorders (American Psychiatric Association, 1994). Despite this theoretical conceptualization, however, strong associations have been found both with personality disorders (Jang et al., 1996; Svrakic et al., 1993; Warner et al., 2004) and the ‘major’ Axis I disorders (Caspi et al., 1996; Frick, 2004), shedding doubt about this historical distinction. Debate remains, however, as to whether it is best to conceptualize neuroticism as an important risk factor for psychopathology or as different points along a common continuum (Hettema et al., 2004; Nigg & Goldsmith, 1998; Rettew & McKee,

Address for correspondence: Dr David Rettew, Department of Psychiatry, University of Vermont College of Medicine, 89 Beaumont Avenue, Given Building Room B227, Burlington, Vermont 05405, USA., david.rettew@uvm.edu.

2005). Clarifying the relations between personality and psychopathology could be a major step towards the critical task of phenotypic refinement on which all of clinical research depends (Hudziak, 2002).

Research has demonstrated moderate genetic influence of neuroticism in line with most other major personality dimensions (Johnson et al., 2004; Loehlin, 1992; Riemann et al., 1997). However, most of these studies have been carried out in adults. A Finnish sample of approximately 15,000 twins aged 18 to 59 found heritabilities of .17 to .54 for men and .34 to .53 for women across different age groups with evidence of higher heritabilities in women and decreasing heritabilities from late adolescence into early adulthood (Viken et al., 1994). In perhaps the most comprehensive adult study to date involving more than 45,000 individuals in an extended twin design, Lake and coworkers (Lake et al., 2000) found evidence for the influence of both additive (.25 males, .28 females) and nonadditive genetic factors (.10 males and .13 females) in addition to nonshared environment (.65 males, .58 females). Noteworthy from this study was the finding that despite the study's statistical power to test more complex models of transmission, this relatively simple model, similar to studies using less complicated designs, provided the best fit.

While there have been fewer studies of the genetic and environmental influence on neuroticism in children and adolescents, mainly additive genetic influences have been found in small samples (Graham & Stevenson, 1985). Buss and Plomin reported some evidence of genetic dominance in an early study of 400 pairs of 5-year-old twins (Buss & Plomin, 1984). The extremely low correlations reported between dizygotic (DZ) twins could also have represented a contrast effect (Buss & Plomin, 1984) rather than nonadditive genetic effects. Data in infants and toddlers aged 14 to 36 months from the MacArthur Longitudinal Twin Study (Saudino et al., 2001) showed heritability coefficients of .11–.37 with the remainder of variance primarily due to unshared environmental factors (.47–.89) when using the parent-rated Colorado Childhood Temperament Inventory (Rowe & Plomin, 1977).

Among older children, a study of 198 same-sex twin pairs aged 8 to 16 found the best fitting model for neurotic symptoms, as measured by the Rutter A parent scale (Rutter et al., 1970), included additive genetic (.52) and unshared environmental (.48) factors with no contribution of shared environment (Thapar & McGuffin, 1996). Gillespie and colleagues (2004) studied a group of 540 Australian adolescent twin pairs age 12 to 16 using the Junior Eysenck Personality Questionnaire (Eysenck & Eysenck, 1975). Again, the best fitting models included additive genetic (.28–.53) and unshared environmental components (.51–.70). There was some evidence of shared environmental influence for 16-year-old twins, although the authors reported insufficient power to test that hypothesis. In addition, there were also data supporting higher estimates of additive genetics for males at age 12 in comparison to other ages.

In summary, most studies of the genetic architecture of neuroticism have found additive genetic influences and unshared environmental factors. Little evidence for shared environmental factors has been found. In general, limited data are available on heritability estimates in adolescents. As other personality dimensions and various types of psychopathology reveal different heritability coefficients across sex and age groups (Hudziak et al., 2003; Viken et al., 1994), we decided to investigate potential differences in the magnitude of genetic and environmental factors by age and sex. In the present study, we use self-report data from a sample of 3301 adolescent twins (aged 12 to 18) from the Netherlands Twin Registry (NTR) to study the genetic architecture of neuroticism as a function of age and sex.

Method

Participants

The present study is part of a large ongoing twin-family study of the NTR on health, lifestyle and personality. The details of this study have been presented elsewhere (Boomsma et al., 2000; Boomsma et al., 2002). For this study, we focus on adolescent twins who were assessed through two waves of mailed surveys in 1991 (wave 1) and 1993 (wave 2). For subjects who completed questionnaires at both time points, wave 1 data were used in order to obtain a younger sample.

Out of the 1712 twin pairs aged between 12 and 18 years (3424 individuals), there were 103 individuals without a valid neuroticism score (3%). Zygosity was not known in an additional 20 individuals, leaving 3301 individuals available for the model-fitting analyses which could accommodate twin pairs with partial data. The final sample included 1626 complete twin pairs: 277 monozygotic (MZ) male twin pairs, 382 MZ female twin pairs, 240 DZ male twin pairs, 257 DZ female twin pairs, and 470 DZ opposite-sex twin pairs.

Measures

Neuroticism was assessed using the Amsterdamse Biografische Vragenlijst (ABV) which is a self-report personality instrument similar in content to the Eysenck Personality Questionnaire (Eysenck & Eysenck, 1975), and has demonstrated good reliability and external validity (Wilde, 1970). The neuroticism scale comprises 30 statements such as: Do you think you are a nervous or intrinsically tense person? Are your feelings easily hurt? Are you often moody? Do you take things too personally? Respondents answer the questions on a 3-point scale (*no*, *don't know*, *yes*).

Zygosity

Zygosity was based on DNA typing (33.4% of the same-sex twin pairs) or on questions concerning similarity. Agreement between zygosity based on questionnaire data and zygosity based on DNA is 97% in the total sample.

Statistical Analyses and Model Fitting

Genetic and environmental influences on variation in adolescent neuroticism scores were analyzed using structural equation modeling with the statistical software package Mx (Neale, 1997). The statistical procedures take advantage of the different degree of genetic relatedness between MZ twins, who share all of their genes, and DZ twins, who on average share half of their genes.

A model designed to test interactions of a latent genetic variable with a measured continuous moderator variable (Purcell, 2002) was used for the total sample (average age 15.5 years, *SD* = 1.4). In this model, the phenotypic variance (in neuroticism score) is not only partitioned into the usual genetic (*a*), common environmental (*c*), and nonshared environmental (*e*) components, but also incorporates the interaction between these components and a measured moderator variable, in this case, age. The expected trait variance is $\mathrm{var}(T_i) = (a + \beta_x M)^2 (c + \beta_y M)^2 + (e + \beta_z M)^2$ where M is the age of the twin. The expected MZ covariance is: $\mathrm{cov}_{mz}(T_1,T_2) = (a + \beta_x M)^2 + (c + \beta_y M)^2$; and the expected DZ covariance is: $\mathrm{cov}_{dz}(T_1,T_2) = .5(a + \beta_x M)^2 + (c + \beta_y M)^2$. Furthermore, this model incorporates the noninteractive main effect of age (β_m) on the neuroticism score. The expected trait mean is $\mu + \beta_m M$.

Quantitative sex differences were tested by allowing the magnitude of the genetic and environmental effects to be different in males and females. Qualitative sex differences, addressing the question of whether the same genes are expressed in boys and girls, were

explored by allowing the correlation between the genetic factors in opposite-sex twin pairs to be less than .5. As the raw neuroticism scores approximated a normal distribution, no transformation was applied.

Results

Correlational Analyses

Correlations between MZ and DZ twins are presented in Table 1. Correlations for all types of twins, regardless of zygosity, were statistically significant for the entire sample. For same-sex twin pairs, DZ correlations are nearly half the MZ correlations, suggesting the influence of additive genetic effects. The opposite-sex DZ twin correlations are somewhat less than those for same-sex DZ twin, which indicates that different genes may influence neuroticism levels for girls and boys. Because of the possibility that opposite-sex DZ twins might share less than half of the relevant genes for neuroticism, we tested whether or not we could fix the degree of genetic relatedness of opposite-sex twins to .5 in the genetic models (Eaves et al., 1998).

Preliminary Model Testing

A number of factors were tested using Mx before proceeding to the specific models expressed in the hypotheses. We found evidence for a significant effect for sex, age, and zygosity on means. Specifically, higher neuroticism scores were associated with female sex, higher age, and being a DZ as opposed to a MZ twin. The test for homogeneity of the variance across sex showed significant sex differences.

The test for homogeneity of the variance across zygosity (MZ vs. DZ) showed no significant difference. However, the variances between males and females were significantly different. Variances were larger for females than for males. We found no evidence that the covariance between twins differed between girls and boys.

Model Fitting

Results of the models that included age as modifier showed that the interactions between the variance components *a*, *c* and *e* with the moderator variable age were not significant (model, 2, 4 and 6). Compared to an ACE model, the model which dropped C (model 3) did not worsen the fit. The main effect of age on the mean was significant for females only (model 7, 8). Next, we tested a model that constrained the variance components for A and E across sexes (model 9) and a model with the genetic correlation in the DZ opposite-sex group fixed on .5 (model 10) but for both models, a significant deterioration of the fit was observed. For model 7, the parameter estimates included an additive genetic component of 16.19 for males ($16.19^2/457.00 = .57$) and 17.91 for females ($17.91^2/528.99 = .61$) and an unshared environment component of 13.96 for males ($13.69^2/457.00 = .43$) and 14.43 for females ($14.63^2/528.99 = .39$). In a final step (not shown in the table), the standardized estimates for A and E were constrained to be equal across sexes which did not worsen the fit of the model ($\chi^2 = .537$, 1 *df*, $p = .464$).

Thus, the best fitting model was an AE model with an effect of age on the mean for females. The estimates of the genetic variance and environmental variances differed between males and females (i.e., the variances were larger for females than for males) but the standardized estimates were the same in both sexes (i.e., .59, 95% confidence intervals [CI] .54–.63 for additive genetic influences, and .41, 95% CI .37–.45 for unique environmental factors). The main effect of age on the mean for females was 2.08 such that the expected neuroticism scores for females increased by 2.08 for each increased year from 12 to 18. The genetic

correlation in the opposite-sex twins was estimated at .34, suggesting that there may be some unique genes that influence neuroticism in boys and girls.

Discussion

This study tested the relative contribution of genetic and environmental influences to neuroticism in a large sample of adolescent Dutch twins. Results were generally consistent with previous reports in both adults and pediatric samples in that the variance was explained by a combination of additive genetic and unshared environmental factors.

Our estimates of additive genetics appear to be somewhat higher than some previous reports. These differences may be related to assessment approach. The present study used 30 items to measure neuroticism in contrast to many previous reports which have used as few as five. In addition, this is one of the few studies to examine self-reports in a nonadult sample. While the use of self-report measures may be subject to bias, it is less likely to be distorted by contrast effects which are possible when one parent rates more than one offspring.

However, there may indeed be a stronger genetic influence on levels of neuroticism in adolescents in comparison to adults. One stereotype of adolescence is that it is a period of ‘moodiness’ thought to be due in part to hormonal changes. While this stereotype has been challenged, less controversial is the claim that adolescence represents a period of heightened risk for the onset particularly of many mood and anxiety disorders (Bernstein et al., 1996; Birmaher et al., 1996).

Interesting findings with regard to sex differences emerged. The best fitting model included additive genetic and unique environmental influences. The variances were different for boys and girls but the magnitude of the genetic and environmental influences could be constrained to be equal. In addition, we found the genetic correlation in the opposite-sex twins to be less than .5, which suggests that it is possible that different genes underlie neuroticism in young adolescent boys and girls. Evidence from other studies is lacking.

Also interesting but perhaps more difficult to explain is the effect of zygosity on mean neuroticism scores. DZ twins had higher neuroticism scores than MZ twins. This difference was tested and not found in a previous study, using a slightly younger sample (Thapar & McGuffin, 1996) while a study in adult women found slight indications for the opposite direction (Heath et al., 1992). Replication, especially in another adolescent sample, is needed to confirm this finding. Nevertheless, it is possible that the increased similarity of MZ twins compared to DZ twins results in a strong bond that protects them somewhat from higher levels of neuroticism.

Limitations

While this study provides important information from a large sample, there are limitations that influence the generalizability of the conclusions. First, this study relied on self-report data which, while free of contrast effects, may still contain bias. Additionally, this sample comes from a relatively homogenous population with regard to ethnic background and results, therefore, may not generalize to other groups.

Clinical Implications

The pathways that accentuate or diminish temperamental traits in general and neuroticism in particular become of clinical interest in light of the links between personality and later psychopathology. Researchers have increasingly promoted the hypothesis that continuously distributed traits such as personality traits may underlie categorical diagnoses and that studying the etiology of psychiatric disorders may be more fruitfully explored by studying

these endophenotypes (Rutter et al., 1997). Neuroticism has been linked to a number of psychiatric disorders including not only anxiety and depressive disorders (Compas et al., 2004), but also some disruptive behavior disorders (White, 1999), eating disorders (Westen & Harnden-Fischer, 2001), and substance abuse (Wills & Dishion, 2004). Bivariate genetic analyses (Hettema et al., 2004) and receiver operating characteristic analyses (Rettew et al., 2005) have revealed substantial, although not complete, overlap between neuroticism and corresponding psychiatric conditions such as generalized anxiety disorder. It is likely that through genetic environmental (GE) correlations, many environmental factors are more likely to occur based on genetic predispositions (Plomin et al., 1999). For example, due to parents' genetic influences (passive GE correlation) or evoked through the child's anxious tendencies (evocative GE correlation), it is possible that these children may experience less mastery opportunities to anxiety producing situations and may even be taught explicitly or implicitly more avoidant or fear-based responses (Lenuga & Long, 2002). Future investigations will be able to delineate the complex pathways through which various personality predispositions propel children towards or away from the development of significant behavioral disturbance. This understanding, in turn, can be used to provide insights into possible intervention strategies that could moderate the developmental outcomes of genetically based predispositions (Kaufman et al., 2004).

In summary, this study represents one of the largest adolescent twin studies to date to investigate the genetic and environmental contributions to the personality trait of neuroticism. Results support previous work which has found influence of both additive genetic and unshared environmental factors. No interaction was found between age and the magnitude of genetic and environmental influences; however, results suggested that different genes may be involved in neuroticism between girls and boys. Future studies are in progress to investigate other major personality dimensions such as extraversion as well as studies to test whether or not the same genes are involved in both a personality trait such as neuroticism and corresponding psychiatric conditions.

Acknowledgments

This work was supported by the Netherlands Organization for Scientific Research (NOW 985-10-002 and NOW 900-562-137) and by grants K08 MH069562, MH58799 and MH52813 from the National Institute of Mental Health, Rockville, MD.

References

American Psychiatric Association. Diagnostic and statistical manual of mental disorders. 4. Washington, DC: Author; 1994.

Bernstein GA, Borchardt CM, Perwien AR. Anxiety disorders in children and adolescents: A review of the past 10 years. Journal of the American Academy of Child and Adolescent Psychiatry. 1996; 35:1110–1119. [PubMed: 8824054]

Birmaher B, Ryan ND, Williamson DE, Brent DA, Kaufman J, Dahl RE, Perel J, Nelson B. Childhood and adolescent depression: A review of the past 10 years: Part i. Journal of the American Academy of Child and Adolescent Psychiatry. 1996; 35:1427–1439. [PubMed: 8936909]

Boomsma DI, Beem AL, van den Berg M, Dolan CV, Koopmans JR, Vink JM, de Geus EJ, Slagboom PE. Netherlands twin family study of anxious depression (NETSAD). Twin Research. 2000; 3:323–334. [PubMed: 11463154]

Boomsma DI, Vink JM, van Beijsterveldt TC, de Geus EJ, Beem AL, Mulder EJ, Derks EM, Riese H, Willemsen GA, Bartels M, van den Berg M, Kupper NH, Polderman TJ, Posthuma D, Rietveld MJ, Stubbe JH, Knol LI, Stroet T, van Baal GC. Netherlands twin register: A focus on longitudinal research. Twin Research. 2002; 5:401–406. [PubMed: 12537867]

Buss, AH.; Plomin, R. Temperament: Early developing personality traits. Hillsdale, NJ: Erlbaum; 1984.

Caspi A, Moffitt TE, Newman DL, Silva PA. Behavioral observations at age 3 predict adult psychiatric disorders: Longitudinal evidence from a birth cohort. Archives of General Psychiatry. 1996; 53:1033–1039. [PubMed: 8911226]

Compas BE, Connor-Smith J, Jaser S. Temperament, stress reactivity, and coping: Implications for depression in children and adolescents. Journal of Clinical Child and Adolescent Psychology. 2004; 33:21–31. [PubMed: 15028538]

Costa, PT.; McCrae, RR. The neo personality inventory manual. Odessa, FL: Psychological Assessment Resources; 1985.

Eaves LJ, Heath AC, Neale MC, Hewitt JK, Martin NG. Sex differences and non-additivity in the effects of genes on personality. Twin Research. 1998; 1:131–137. [PubMed: 10051337]

Eysenck, HJ.; Eysenck, SBG. Manual for the Eysenck Personality Inventory (Adult and Junior). San Diego, CA: Digits; 1975.

Frick PJ. Integrating research on temperament and childhood psychopathology: Its pitfalls and promise. Journal of Clinical Child and Adolescent Psychology. 2004; 33:2–7. [PubMed: 15028536]

Gillespie NA, Evans DE, Wright MM, Martin NG. Genetic simplex modeling of Eysenck's dimensions of personality in a sample of young Australian twins. Twin Research. 2004; 7:637–648. [PubMed: 15607015]

Graham P, Stevenson J. A twin study of genetic influences on behavioral deviance. Journal of the American Academy of Child and Adolescent Psychiatry. 1985; 24:33–41.

Heath AC, Neale MC, Kessler RC, Eaves LJ, Kendler KS. Evidence for genetic influences on personality from self-report and informant ratings. Journal of Personality and Social Psychology. 1992; 63:85–96. [PubMed: 1494987]

Hettema JM, Prescott CA, Kendler KS. Genetic and environmental sources of covariation between generalized anxiety disorder and neuroticism. American Journal of Psychiatry. 2004; 161:1581–1587. [PubMed: 15337647]

Hudziak, JJ. Importance of phenotype definition in genetic studies of child psychopathology. In: Helzer, JE.; Hudziak, JJ., editors. Defining psychopathology in the 21st century: DSM-V and beyond. Washington, DC: American Psychiatric Publishing; 2002. p. 211-230.

Hudziak JJ, van Beijsterveldt CE, Bartels M, Rietveld MJ, Rettew DC, Derks EM, Boomsma DI. Individual differences in aggression: Genetic analyses by age, gender, and informant in 3-, 7-, and 10-year-old Dutch twins. Behavior Genetics. 2003; 33:575–589. [PubMed: 14574134]

Jang KL, Livesley WJ, Vernon PA, Jackson DN. Heritability of personality disorder traits: A twin study. Acta Psychiatrica Scandinavica. 1996; 94:438–444. [PubMed: 9020996]

Johnson AM, Vernon PA, Harris JA, Jang KL. A behavior genetic investigation of the relationship between leadership and personality. Twin Research. 2004; 7:27–32. [PubMed: 15053851]

Kaufman J, Yang BZ, Douglas-Palumberi H, Houshyar S, Lipschitz D, Krystal JH, Gelernter J. Social supports and serotonin transporter gene moderate depression in maltreated children. Proceedings of the National Academy of Sciences USA. 2004; 101:17316–17321.

Lake RI, Eaves LJ, Maes HH, Heath AC, Martin NG. Further evidence against the environmental transmission of individual differences in neuroticism from a collaborative study of 45,850 twins and relatives on two continents. Behavior Genetics. 2000; 30:223–233. [PubMed: 11105396]

Lenuga LJ, Long AC. The role of emotionality and self-regulation in the appraisal-coping process: Tests of direct and moderating effects. Journal of Applied Developmental Psychology. 2002; 23:471–493.

Loehlin, JC. Genes and environment in personality development. Newbury Park, CA: Sage; 1992.

Middeldorp, CMC.; Danielle, C.; van den Berg, M.; Beem, AL.; van Dyck, R.; Boomsma, DI. The association of personality with anxious and depressive psychopathology. In: Canli, T., editor. The biological basis of personality and individual differences. in press

Neale, MC. Mx: statistical modeling. Richmond, VA: Department of Psychiatry, Medical College of Virginia; 1997.

Nigg JT, Goldsmith HH. Developmental psychopathology, personality, and temperament: Reflections on recent behavioral genetics research. Human Biology. 1998; 70:387–412. [PubMed: 9549245]

Plomin, R.; Caspi, A.; Pervin, LA.; John, OP. Behavioral genetics and personality. In: Pervin, LA.; John, OP., editors. Handbook of personality: Theory and research. New York: Guilford Press; 1999. p. 251-276.

Purcell S. Variance components models for gene–environment interaction in twin analysis. Twin Research. 2002; 5:554–571. [PubMed: 12573187]

Rettew DC, McKee L. Temperament and its role in developmental psychopathology. Harvard Review of Psychiatry. 2005; 13:14–27. [PubMed: 15804931]

Riemann R, Angleitner A, Strelau J. Genetic and environmental influences on personality: A study of twin reared together using the self- and peer report neoffi scales. Journal of Personality. 1997; 65:449–476.

Rothbart MK, Ahadi SA, Evans DE. Temperament and personality: Origins and outcomes. Journal of Personality and Social Psychology. 2000; 78:122–135. [PubMed: 10653510]

Rowe DC, Plomin R. Temperament in early childhood. Journal of Personality Assessment. 1977; 41:150–156. [PubMed: 856967]

Rutter M, Dunn J, Plomin R, Simonoff E, Pickles A, Maughan B, Ormel J, Meyer J, Eaves L. Integrating nature and nurture: Implications of person–environment correlations and interactions for developmental psychopathology. Development and Psychopathology. 1997; 9:335–364. [PubMed: 9201448]

Rutter, M.; Tizard, J.; Whitmore, K. Education, health and behaviour. London: Longman; 1970.

Saudino, KJ.; Cherny, SS.; Emde, RN.; Hewitt, JK. Parental ratings of temperament in twins. In: Emde, RN.; Hewitt, JK., editors. Infancy to early childhood: Genetic and environmental influences on developmental change. Oxford, UK: Oxford University Press; 2001. p. 73-88.

Svrakic DM, Whitehead C, Przybeck TR, Cloninger CR. Differential diagnosis of personality disorders by the seven-factor model of temperament and character. Archives of General Psychiatry. 1993; 50:991–999. [PubMed: 8250685]

Thapar A, McGuffin P. A twin study of antisocial and neurotic symptoms in childhood. Psychological Medicine. 1996; 26:1111–1118. [PubMed: 8931157]

Viken RJ, Rose RJ, Kaprio J, Kosenvuo M. A developmental genetic analysis of adult personality: Extraversion and neuroticism from 18 to 59 years of age. Journal of Personality and Social Psychology. 1994; 66:722–730. [PubMed: 8189349]

Warner MB, Morey LC, Finch JF, Gunderson JG, Skodol AE, Sanislow CA, Shea MT, McGlashan TH, Grilo CM. The longitudinal relationship of personality traits and disorders. Journal of Abnormal Psychology. 2004; 113:217–227. [PubMed: 15122942]

Westen D, Harnden-Fischer J. Personality profiles in eating disorders: Rethinking the distinction between axis i and axis ii. American Journal of Psychiatry. 2001; 158:547–562. [PubMed: 11282688]

Wilde, GJS. Neurotische labiliteit gemeten volgens de vragenlijstmethode [The questionnaire method as a means of measuring neurotic instability]. Amsterdam, the Netherlands: Van Rossen; 1970.

White JD. Personality, temperament and ADHD: A review of the literature. Personality and Individual Differences. 1999; 27:589–598.

Wills TA, Dishion TJ. Temperament and adolescent substance use: A transactional analysis of emerging self-control. Journal of Clinical Child and Adolescent Psychology. 2004; 33:69–81. [PubMed: 15028542]

Table 1

Correlations (95% Confidence Intervals) in Neuroticism Scores Between Twins by Zygosity

Zygosity	*n*	Correlation Age = 15.47 yrs
MZM	277	.58 (.49–.65)
DZM	240	.32 (20–.43)
MZF	382	.60 (.54–.66)
DZF	257	.36 (.24–.46)
DOS_MF	237	.16 (.03–.28)
DOS_FM	233	.21 (.09–.33)

Note: MZM = monozygotic twins, males; DZM = dizygotic twins, males; MZF = monozygotic twins, females; DZF = dizygotic twins, females; DOS_MF = dizygotic opposite-sex twins, male born first; DOS_FM = dizygotic opposite-sex twins, female born first.

Table 2

Model-Fitting Results for Neuroticism Scores Using a Modifier Model in the Total Sample

Model	Versus model:	$\Delta\chi^2$	*df*	*p* (α = .05)
1. ACE ♂ and ♀, $\beta_{x,}$ $\beta_{y\ and}$ β_z for ♂ and ♀, β_m for ♂ and ♀. R_g free.			3235	
2. ACE ♂ and ♀, $\beta_{x\ and}$ β_z for ♂ and ♀, β_m for ♂ and ♀. R_g free				
–Drop β_y for ♂ and ♀	1	0.726	3237	*ns*
3. AE ♂ and ♀, $\beta_{x\ and}$ β_z for ♂ and ♀, β_m for ♂ and ♀. R_g free.				
–Drop c for ♂ and ♀	2	1.289	3239	*ns*
4. AE ♂ and ♀, β_z for ♂ and ♀, β_m for ♂ and ♀. R_g free.				
–Drop β_x for ♂ and ♀	3	3.534	3241	*ns*
5. E ♂ and ♀, β_z for ♂ and ♀, β_m for ♂ and ♀. R_g free.				
–Drop a for ♂ and ♀	4	255.969	3243	.000
6. AE ♂ and ♀, β_m for ♂ and ♀. R_g free.				
–Drop β_z for ♂ and ♀	4	2.854	3243	*ns*
7. AE ♂ and ♀, β_m for ♀. R_g free.				
–Drop β_m for ♂	**6**	**.413**	**3244**	*ns*
8. AE ♂ and ♀. R_g free.				
–Drop β_m for ♀	7	22.480	3245	.000
9. AE ♂ = ♀, β_m for ♀. R_g free.				
–no sex dif AE	7	7.663	3246	.022
10. AE ♂ and ♀, β_m for ♀.				
R_g .5. – R_g fixed	**7**	18.130	3245	.000

Note: A = additive genetic factors, C = common or shared environment, E = nonshared environmental effects. β_X = modifier age on A, β_Y = modifier age on C, β_Z = modifier age on E. β_M = regression of age on mean. R_g = genetic correlation in dizygotic opposite-sex group. *ns* = not significant. Bold print indicates best fitting model.