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Changes in Genetic and Environmental Influences on Trait Anxiety from Middle Adolescence
to Early Adulthood

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Abstract

Background. Middle adolescence to early adulthood is an important developmental period for the emergence of anxiety. Genetically-influenced stable traits are thought to underlie internalizing psychopathology throughout development, but no studies have examined changes in genetic and environmental influences on trait anxiety during this period. **Method.** A longitudinal twin study design was used to study same-sex twin pairs (485 monozygotic pairs, 271 dizygotic pairs) at three ages, 14, 18, and 21 years, to examine developmental shifts in genetic and environmental effects on trait anxiety. **Results.** The heritability of trait anxiety increased with age, particularly between ages 14 and 18, no significant new genetic influences emerged after age 14, and the genetic influences were highly correlated across the three ages, supporting developmentally stable genetic risk factors. The environmental effects shared by members of a family decreased in influence across adolescence, while the influence of environmental effects unique to each individual twin remained relatively stable over the course of development and were largely age-specific. **Limitations.** The twin study design does not inform about specific genes and environmental risk factors. **Conclusions.** Genetic influences increased in importance from middle to late adolescence but common genetic factors influenced trait anxiety across the three ages. Shared environmental influences decreased in importance and demonstrated negligible influence by late adolescence/early adulthood. Nonshared environmental effects were almost entirely age-specific. These findings support the importance of developmentally-sensitive interventions that target shared environmental factors prior to middle adolescence and shifting non-shared environmental risks at each age. **Keywords:** anxiety, genetics, environment, adolescence.

Changes Genetic and Environmental Influences on Trait Anxiety from Middle Adolescence to Early Adulthood

Adolescence and early adulthood are periods of major life transitions as well as periods of risk for the development of anxiety (for review see Beesdo et al., 2009). Investigating the etiological influences on anxiety across this period has important implications for understanding the mechanisms underlying the development and stability of anxiety. The purpose of the current study was to investigate developmental changes in genetic and environmental influences on trait anxiety at three ages across the risk period spanning middle adolescence to early adulthood.

Research suggests that anxiety develops through complicated risk processes involving genetic and environmental influences (Franić et al., 2010; Hettema et al., 2001). Experiences shared among individuals of a particular age or developmental stage likely result in general trends for genetic and environmental mechanisms to have greater or lesser influence at various points during development. As individuals progress through adolescence and early adulthood, they spend less time with parents and more time with peers (Larson and Richards, 1991) and typically gain greater independence (Roisman et al., 2004). They are thus freer to shape their environments and social interactions, seeking environments that reinforce their genetically-influenced dispositions, a phenomenon termed active gene-environment correlations (rGEs; Plomin et al., 1977; Scarr and McCartney, 1983). For example, studies have shown that adolescents select friends with behaviorally similar dispositions (Haselager et al., 1998; Kendler et al., 2008c; Newcomb et al., 1999). Age-related increases in the heritability of various behavioral traits (Bergen et al., 2007) may be explained by these developmental rGEs. Genetic and environmental factors specific to a given individual probably also shape risk processes leading to anxiety. Furthermore, some genetic and environmental factors may influence anxiety

across development while other factors may exert influence at certain developmental stages but not others. Despite theoretical indication and some empirical evidence that the magnitude of genetic and environmental influences on anxiety varies with age, longitudinal studies of changes in these influences on anxiety are scarce.

Questions about genetic and environmental influences on anxiety across development can be answered using longitudinal twin study designs. Twin studies are based on comparisons of co-twin similarity in a trait (e.g., anxiety proneness) between monozygotic (MZ) twins, who share 100% of their genetic material, and dizygotic (DZ) twins, who share on average 50% of their segregating genetic material. Biometric twin models are used to statistically quantify the proportions of variance in the trait that can be attributed to genetic, shared environmental (i.e., environmental effects shared by reared-together relatives that are sources of behavioral similarity), and nonshared environmental (i.e., environmental effects that differ for reared-together relatives and are sources of behavioral dissimilarity) factors. Longitudinal biometric twin models, such as the one used in the current study, are methods of estimating and teasing apart genetic and environmental influences that are shared across different ages versus those that are specific to certain ages.

Genetic and Environmental Influences on Anxiety in Youth Samples

Previous twin research supports genetic, shared environmental, and nonshared environmental influences on anxiety in youth samples (e.g., Thapar and McGuffin, 1995). Estimates of the proportions of variance attributed to genetic and shared environmental sources vary across studies, with estimates of genetic influences on anxiety in children and adolescents ranging from 15% (Eley and Stevenson, 1999) to 59% (Thapar and McGuffin, 1995) and estimates of shared environmental influences ranging from 0% (Thapar and McGuffin, 1995) to

35% (Eley and Stevenson, 1999). One potential explanation for the discrepancy in estimates across studies is the difference in mean ages and large age ranges of the samples in these studies. Samples with wide age ranges may produce estimates that do not accurately represent the size of the effects across the entire age range, for example estimates of genetic influences on anxiety may be larger in studies with older samples. Consistent with hypothesized age-related increases in active gene-environment correlations (resulting from individuals increasingly seeking experiences that fit their underlying genetic traits), cross-sectional research generally supports age-related increases in heritability estimates of anxiety (for review see Bergen et al., 2007).

To our knowledge, only one longitudinal twin study examined anxiety over the period of middle adolescence into early adulthood (age 8 to 20 years). This study of phobias/fears supported a decrease in shared environmental influences and relatively stable heritability estimates across this developmental period. Some of the genetic influences on phobias at later ages were present at earlier ages (developmental stability), but the majority of genetic influences on phobias later in this developmental period emerged throughout adolescence and into early adulthood (genetic innovation) and the influence of early genetic influences decreased with age (genetic attenuation; Kendler et al., 2008a). Additionally, a few twin studies investigated age-related changes in internalizing symptoms, primarily using the Anxious/Depressed scale of the Child Behavior Checklist, Youth Self-Report (CBCL-YSR; Achenbach, 1991) and Adult Behavior Checklist/Self-Report (Achenbach and Rescorla, 2001), in samples of adolescents. Lamb et al. (2010) estimated genetic and environmental influences on the Anxious/Depressed scale of the YSR at ages 12, 14, and 16 years and found significant genetic and nonshared environmental effects at all three ages and shared environmental effects only at age 12. Separate cross-sectional models were analyzed at each age, and thus inferences about changes in genetic

and environmental influences were not made. Kendler et al. (2008b) used longitudinal twin models to examine influences on Anxious/Depressed symptoms (from the CBCL, YSR, and Adult Behavior Checklist/Self-Report) at four ages between ages 8 and 20. Heritability estimates were relatively stable across this period, and they found no significant shared environmental influences. There was evidence of temporal stability of genetic influences as some genetic influences on anxious-depression symptoms at later ages were present at earlier ages (developmental stability), but the influence of early genetic effects declined with age (attenuation) as new genetic effects (innovation) emerged during adolescence and early adulthood. Despite evidence of some common genes influencing depression and anxiety (Eley and Stevenson, 1999; Kendler et al., 1987; Kendler et al., 1992), research indicates changing patterns of genetic covariation in depression and anxiety across childhood and adolescence (Silberg et al., 2001). Further, a meta-analysis concluded that age-related increases in heritability were greater for anxiety than for depression (Bergen et al., 2007). This suggests the importance of studying anxiety separate from depression when examining changes in genetic and environmental influences. A few studies have investigated genetic and environmental influences on depression across adolescence (e.g., O'Connor et al., 1998; Tully et al., 2010), though similar research has not been conducted on measures of general anxiety.

Trait Anxiety

Etiological models of psychopathology (e.g., Iacono et al., 2008; Tully and Iacono, in press) suggest that stable traits are more proximal to risk genes than manifest behaviors or symptoms, and stable traits are increasingly understood to underlie internalizing psychopathology throughout development (De Pauw, 2010; Kendler and Gardner, 2011; Mineka and Zinbarg, 2006; Van Ameringen et al., 1998). For example, trait anxiety proneness has

demonstrated stability from adolescence into adulthood (Usala and Hertzog, 1991) and is predictive of anxiety symptoms across development (Watson and Walker, 1996). This suggests that genetic influences on trait anxiety may have developmental stability (i.e., the same genetic influences contribute to anxiety across development). Thus, longitudinal twin studies employing a general trait measure of anxiety may be particularly insightful for informing about the roles of genetic and environmental contributions to anxiety across adolescence and early adulthood. Few studies have examined genetic and environmental influences on trait anxiety and to our knowledge none have tested longitudinal models. Two studies examined trait anxiety cross-sectionally in youth samples. A previous study from our research group used a twin design to examine trait anxiety in twins aged 11 or 17 (with parameters constrained across ages; Legrand et al., 1999), and Lau et al. (2006) studied a sample of twins aged 8 to 16 years. Both studies found significant genetic and nonshared environmental effects, but not shared environmental effects, although Eley and Stevenson (1999) found significant shared environmental effects on trait anxiety in a subgroup of eight to eleven year olds in their sample. As previously stated, wide age ranges of the samples may be masking important developmental changes in the relative influences of these factors and questions about genetic attenuation and innovation were not addressed in these studies.

Hypotheses

In summary, previous cross-sectional research supports age-related changes in genetic and environmental influences on anxiety and developmentally dynamic changes, both genetic innovation and genetic attenuation, in the influence of genes on internalizing and phobia symptoms, but little is known about the dynamic changes in genetic and environmental influences on general anxiety. The current study used a longitudinal twin model to examine

genetic and environmental influences on trait anxiety at three specific ages across the important developmental period of adolescence to early adulthood (14, 18, and 21 years) when dynamic changes in the influence of genes on internalizing symptoms are thought to occur. Four hypotheses were tested. First, the heritability of anxiety was hypothesized to increase with age due to developmental processes such as increased independence and niche-fitting during adolescence and early adulthood. Second, the increase in heritability was expected to reflect both relative, that is compared to environmental influences, increases in the influence of *developmentally stable* genetic influences as well as the emergence of new genetic influences, though this *genetic innovation* (and corresponding *genetic attenuation* of early genetic influences on later trait anxiety) was expected to be relatively small given the phenotype of interest is a trait measure of anxiety. Third, age-related decreases in shared environmental influences on anxiety were expected as adolescents transition to spending less time with family. Fourth, nonshared environmental influences on trait anxiety were hypothesized at each age but the effects were expected to be largely *age-specific*, reflecting sources of environmental risks that change across development, and were not expected to contribute to covariance in trait anxiety across ages.

Method

Sample

Participants were drawn from the Minnesota Twin Family Study (MTFS), a large, population-based, longitudinal study. Same-sex twins and their families were recruited from birth records of twins born in Minnesota between 1971 and 1985. The participation rate was 83% for families who met inclusion criteria (i.e., twins had no major cognitive and physical handicaps, lived within a day's drive from the laboratory, and had not been adopted by non-relatives). The participating families are representative of the Minnesota population at the time

the twins were born. The sample is 98% Caucasian and mean Hollingshead occupational statuses for fathers and mothers were 3.9 and 3.7, respectively. Written, informed consent was obtained from all participants. Additional recruitment information and sample characteristics have been detailed elsewhere (Iacono et al., 1999; Iacono et al., 2006).

Twins ($N=756$ twin pairs; 50.3% female; 64% monozygotic) first participated when they were 11 years old ($M=11.72$, $SD=0.43$). They completed three follow-up assessments, approximately every three years. The measure of trait anxiety was added to the study protocol after most male participants completed the first assessment, and thus this project will focus on the three follow-up assessments. Trait anxiety scores are available for 345 twin pairs (66% MZ; 11.3% female; mean age 14.18, $SD=.51$) at the first follow-up, 495 twin pairs (66% MZ; 50.5% female; mean age 18.16, $SD=.65$) at the second follow-up, and 555 twin pairs (64% MZ; 54.1% female; mean age 21.46, $SD=.77$) at the third follow-up. Due to budgetary constraints, the trait anxiety measure was administered to a subset of participants at the first follow-up, and the sample size, especially for female twins, was smallest at this assessment. There was little evidence that those who completed the measure at one age differed in trait anxiety from those who did not. The mean trait anxiety scores at age 14 differed by only .07 SDs for participants who completed the age 18 follow-up versus those who did not and by .14 SDs for participants who completed the age 21 follow-up versus those who did not. Mean trait anxiety scores at age 21 differed by .02 SDs for individuals who did and did not complete the age 14 assessment, and all individuals who completed the age 18 assessment also completed the age 21 assessment.

Measures

Participants completed the Spielberger's State-Trait Anxiety Inventory (STAI; Spielberger, 1983) at ages 18 and 21 and the Spielberger's State-Trait Anxiety Inventory for

Children (STAIC) at age 14. The trait anxiety scale of the STAI and STAI-C were used in this study to provide a measure of individual differences in proneness to anxiety. Both scales contain 20 items that require the respondent to rate how often they generally experience tension, nervousness, and apprehension on a 4-point scale (1=*almost never*, 2=*sometimes*, 3=*often*, 4=*almost always*) in the STAI and a 3-point scale (1=*hardly ever*, 2=*sometimes*, 3=*often*) in the STAIC. Examples of items include: "I worry too much over something that really doesn't matter" on the STAI and "I worry too much" on the STAIC. Internal consistency reliabilities in this sample were .85, .89, and .90 for ages 14, 18, and 21 respectively.

Zygoty of the twins was determined by three measures: (1) parental report on a standard measure of zygoty, (2) subjective evaluations of twin's physical similarity (e.g., hair color and face and ear shape) by an MTFS researcher, and (3) anthropometric measures of ponderal index, cephalic index, and finger print ridge count. When these measures were discrepant, zygoty was determined through serological analysis. Validation of this zygoty method was supported through analysis of a subsample ($N=50$), which confirmed agreement among the three measures with serological analysis.

Data Analyses

Biometric model-fitting. A 3-factor Cholesky decomposition model (Figure 1) was used to estimate additive genetic effects (A), shared environmental effects (C, environmental effects that are common to reared-together relatives and are sources of behavioral similarity), and nonshared environmental effects (E, environmental effects that differ for reared-together relatives and are a source of behavioral dissimilarity) on trait anxiety at the three ages. Genetic and environmental contributions to variance in age 14 trait anxiety were obtained by squaring the respective age 14 path-coefficients (a_{11} , c_{11} , e_{11}). Variance in trait anxiety at age 18 was divided into

components attributable to genetic and environmental influences present at age 14 (a_{21} , c_{21} , e_{21}) and residual (new) components that were independent of the genetic and environmental variance at age 14 (a_{22} , c_{22} , e_{22}). Variance in the phenotypes at age 21 was divided into components attributable to genetic and environmental influences present at age 14 (a_{31} , c_{31} , e_{31}), genetic and environmental effects present at age 18 but not 14 (a_{32} , c_{32} , e_{32}), and residual (new) components that were independent of the genetic and environmental influences present at age 14 or 18 (a_{33} , c_{33} , e_{33}). If all the genetic liability in trait anxiety across the three ages is accounted for by genetic influences present at age 15 (A_1), the genetic influences are *developmentally stable*. Diminishing influence of A_1 on trait anxiety with age is evidence of *genetic attenuation*. The emergence of significant new genetic influences at age 18 or 21, that is A_2 or A_3 accounting for significant variance in trait anxiety at age 18 and 21, would be evidence of *genetic innovation*. To test for age-related increases in genetic influences and decreases in shared environmental influences, a series of Cholesky models in which the A and C components were constrained to be equal across ages were calculated and fit statistics for these models were compared. In addition, to determine if parameters could be constrained across sexes, the fit of a model in which separate parameters were estimated for male and female participants was compared to the same model with estimates constrained across sexes.

Data were standardized within age groups to account for the change in STAI measures. Data were then fit to the models using the maximum likelihood option in the Mx statistical software system (Neale et al., 1999). Parameters were estimated to minimize two times the natural logarithm of the multivariate normal likelihood ($-2\ln L$). Differences in the minimized value of $-2\ln L$ between the baseline model (parameters free to vary across ages) and more restrictive models (A, C, and/or E parameters constrained across ages 14, 18, and/or 21) yield a

likelihood χ^2 test that is used to test the significance of the model with constraints. A nonsignificant χ^2 difference test indicates that the more restrictive model (with age constraints) provides an appropriate fit to the data, and in general this more parsimonious model is preferred. The Akaike Information Criteria ($AIC = \chi^2 - 2\Delta df$) is an alternative to the χ^2 goodness-of-fit test statistic that is less influenced by large sample sizes and thus less prone to rejecting a more restrictive model when deviations between the baseline and restricted model are relatively small.

Results

Descriptive Statistics, Phenotypic Correlations, and Twin Correlations

Means, standard deviations, and phenotypic correlations for the anxiety measure across the three ages are provided in Table 1. All phenotypic correlations are significant and generally moderate in magnitude, with the smallest correlations between anxiety at age 14 and age 21.

Twin intraclass correlations by zygosity and age are presented in Table 2 to provide information about genetic and environmental influences on trait anxiety at each age. The correlations for trait anxiety at age 14 were positive, significant, and moderate in magnitude for both MZ and DZ twins with the correlation only slightly larger for MZ twins than DZ twins. These findings indicate that genes contribute little to variance in anxiety at age 14 but shared environmental effects do contribute to the variance. At age 18, the MZ twin correlation was significant, positive, and moderate in size, and the DZ correlation was not significantly different from zero. These correlations support genetic influences but no shared environmental influences on trait anxiety at this age. The magnitude of the MZ twin correlation was significant and moderate in magnitude at age 21. The DZ twin correlation was also significant, but it was small in magnitude and considerably smaller than the MZ twin correlation. These correlations at age 21 suggest continued genetic influences on trait anxiety. Altogether, these correlations indicate

the presence of age-related increases in heritability and decreases in shared environmental influences. Separate twin correlations for males and females were also calculated, and there were no gender differences in the correlations for the MZ twins or the DZ twins at any of the ages, suggesting similar genetic and environmental influences for males and females

Biometric Model Fitting

A model with separate estimates for males and females was calculated first. The fit of the full Cholesky model (i.e., age-unconstrained) with estimates constrained to be equal across sexes was not significantly worse than the fit of the same model with separate parameters estimated for male and female participants [$\Delta X^2(18)=20.42, p=.31$]. Therefore, in subsequent models, sex was accounted for by using separate matrices for males and females, but parameters were constrained to be equal across sexes.

Consistent with the twin correlations, findings from the Cholesky models supported age-related increases in genetic influences and decreases in shared environmental influences on trait anxiety. The standardized variance estimates (Figure 1) indicate that a significant proportion of variance in trait anxiety could be attributed to genetic effects at each age. The proportion of variance attributed to genetic influences increased considerably between age 14 (15%) and age 18 (39%) and only slightly between ages 18 and 21 (45%). A significant proportion of variance in trait anxiety was accounted for by shared environmental influences at age 14 (26%), and shared environmental influences decreased dramatically to nonsignificance from 14 to 18 (1%) and remained negligible at age 21 (1%). The proportions of variance attributable to nonshared environmental influences were significant, relatively large, and similar in size across the three ages.

Figure 1 displays the standardized variance estimates for the fully age-unconstrained (baseline) model. These standardized estimates indicate that despite changes in the relative magnitude of genetic effects across ages, there was considerable overlap in genetic influences across ages. All of the genetic influences on trait anxiety present at age 18 were present at age 14 (a^2_{21}), that is no variance in trait anxiety at age 18 could be attributed to "new" influences. Similarly, all of the genetic influences present at age 21 were present at earlier ages (98% at age 14, a^2_{31} ; 2% at age 18, a^2_{32}). Given the negligible variance attributable to shared environmental influences at ages 18 and 21, estimates of common shared environmental influences across ages are not meaningful. Nonshared environmental influences were almost entirely age-specific, with small but significant variance at age 18 present at age 14 (7%, e^2_{21}) and small but significant variance at age 21 attributed to influences present at age 14 (7%, e^2_{31}) and age 18 (15%, e^2_{32}). As expected, given the common genetic influences across the three ages, genetic correlations were very large: $r_{A1A2}=1.00$ (.52, 1.00), $r_{A1A3}=.98$ (.35, 1.00), $r_{A2A3}=.97$ (.85, 1.00). Reflecting the largely age-specific nonshared environmental effect, the nonshared environmental correlations were smaller but all were significant: $r_{E1E2}=.27$ (.14, .39), $r_{E1E3}=.26$ (.14, .38), $r_{E2E3}=.44$ (.35, .53). Since the shared environment covariance paths were essentially zero, it is not meaningful to present the shared environmental correlations.

Table 3 displays fit indices for model comparisons. Model 2, which constrained A to be equal at ages 14 and 18 (but allowed A to be free at age 21), provided a significantly worse fit than the fully age unconstrained baseline model (model 1). This indicates that A cannot be constrained across these two ages without significantly worsening the fit of the model, i.e., the estimates of genetic influence are different at ages 14 and 18. Model 3, which constrained A to be equal at ages 18 and 21, did not provide significantly worse fit than the baseline model as

indicated by the nonsignificant χ^2 test, suggesting that the small differences in the magnitude of genetic influences on TA at ages 18 and 21 are not meaningful. Model 4 constrained C to be equal at age 14 and 18, and produced a significant χ^2 test, indicating that the shared environmental influences could not be constrained across ages 14 and 18. Model 5, which constrained C to be equal at ages 18 and 21, provided a fit that was not significantly different from the baseline model, meaning the shared environmental influences could be constrained at ages 18 and 21. Model 6 constrained E across all three ages, and this model did not significantly reduce the fit of the model. Given these findings, model 7, which constrained A and C to be equal at 18 and 21 and E to be equal at 14, 18, and 21, was fit to the data, and this model provided a fit that was not significantly worse than the baseline model and had the lowest AIC value. Thus, model 7 is the best-fitting model.

In summary, genetic contributions to the variance in anxiety increased with age while the proportion of variance attributed to shared environmental contributions decreased with age. Considerable genetic influences were common across all ages, providing evidence for *developmental stability* of genetic influences, and nonshared influences were almost entirely age-specific.

Discussion

The purpose of this study was to investigate changes in proportions of genetic and environmental contributions to trait anxiety from middle adolescence into early adulthood as well as to examine the extent to which genetic influences are developmentally stable, genetic innovations emerge, and the effect of early genetic influences attenuate throughout this period. This study builds on previous research supporting shifts in the relative contributions of genetic and environmental influences as well as dynamic changes in these influences on phobias and

anxious-depressive symptoms from childhood to early adulthood (Eley and Stevenson, 1999; Kendler et al., 2008a; Kendler et al., 2008b; Thapar and McGuffin, 1995).

As expected, we found shifts in the relative influence of genes and shared environment on trait anxiety during adolescence. We found an increase in heritability of trait anxiety from middle to late adolescence, which is consistent with past cross-sectional research supporting increases in the heritability of anxiety across adolescence (e.g., Bergen et al., 2007), but stability of heritability from late adolescence to early adulthood, which provides new insight given the dearth of longitudinal studies examining anxiety from adolescence into early adulthood. Active gene-environment correlations may contribute to this rise in heritability as adolescents typically expand their social networks (La Greca and Prinstein, 1999), spend less time with families and more time with peers (Larson and Richards, 1991), and form friendships with peers who are behaviorally similar to themselves (Haselager et al., 1998; Newcomb et al., 1999). This increased freedom to influence their environment in ways consistent with their genetic predispositions is consistent with research suggesting anxious individuals seek environments that reinforce their anxiety. For example, anxiety-prone adolescents have been found to associate with peers who are similarly anxious and withdrawn (Güroğlu et al., 2007; Van Zalk et al., 2011), and over time the friends influence each other to increase their anxiety (Oh et al., 2008; Van Zalk et al., 2011).

Although heritability increased from middle to late adolescence, no significant “new” genetic influences emerged over the course of adolescence and early adulthood. In other words, *developmentally stable* sources of genetic risk that became relatively more important compared to environmental risks were responsible for the increased heritability, and genetic *innovation* did not contribute to increased heritability. Consistent with etiological models positing genetic influences on traits that underlie symptoms of psychological disorders (Iacono et al., 2008; Tully

and Iacono, in press), our findings indicate that stable genetic influences on trait anxiety contribute to anxiety across adolescence and into adulthood, and previous research by Kendler and colleagues provides evidence of genetic innovation on recent symptoms of anxious-depression (Kendler et al., 2008b) and phobias/fears (Kendler et al., 2008a). Thus, our findings support the usefulness of trait measures of anxiety in molecular genetic studies aimed at identifying genes involved in risk for anxiety disorders (e.g., Fakra et al., 2009; Zhou et al., 2008).

Consistent with our hypothesis, the contribution of shared environmental influences to trait anxiety decreased from middle to late adolescence, providing negligible contributions to trait anxiety in late adolescence and early adulthood. This finding is also consistent with developmental transitions as adolescents spend less time in their family environment and more time engaged in activities with peers (Larson and Richards, 1991). The previous cross-sectional research on shared environmental influences on trait anxiety found no support for shared environmental effects (Lau et al., 2006; Legrand et al., 1999), but these studies used samples that had wide age ranges. The use of a longitudinal design and more discrete age ranges allowed us to detect the developmental period when the shared environmental influences lose their influence.

Finally, nonshared environmental influences were almost entirely age-specific. Different sources of nonshared environmental risk across development are consistent with the presence of developmental transitions that provoke anxiety uniquely at middle adolescence, late adolescence and early adulthood. This finding indicates a need for research on the nature of environmental risks specific to each stage to inform developmentally-sensitive components of interventions, such as degree of family involvement and interpersonal emphasis in cognitive-behavioral therapy protocols (Kendall et al., 2010; Weisz and Kazdin, 2010), for psychological problems influenced

by trait anxiety. Furthermore, our finding that shared environmental influences become negligible during the transition from middle to late adolescence highlights the need for interventions targeting shared environmental factors to occur prior to this period.

Limitations

A few limitations of this study should be noted. First, although biometric modeling partitions variance into additive genetic, shared environmental, and nonshared environmental effects, it does not provide information about specific genes, environmental risks, or targets for interventions. Future research should investigate the interplay of specific genes at the molecular level and particular environmental risks to inform the nature of changing etiological factors across this developmental period. Second, the estimates of influence on anxiety variance at age 14 may be influenced by the sample at that age being primarily male and the STAI form changing after age 14. However, the effect of these anomalies on the estimates appears to be minimal. The MZ and DZ twin correlations did not differ significantly for males and females, the parameters in the Cholesky model could be constrained across sexes without reducing the fit of the model, and a gender effect would have resulted in higher shared environmental influences at ages 18 and 21 (ages with greater gender diversity), though the opposite effect was found. Moreover, previous research indicates negligible sex differences in estimates of genetic and environmental influences on various measures of anxiety and anxious-depression symptoms (e.g., Franić et al., 2010; Kendler et al., 2008b; Topolski et al., 1997). Similarly, the very high genetic correlations across all three ages suggest a minimal impact of the change in STAI forms. Third, estimates of nonshared environmental influence include an estimation of measurement error, and age-specific sources of measurement error would partially contribute to the demonstrated age-specificity of sources of nonshared environmental influence. Fourth, though

reflective of local demographics, the primarily Caucasian composition of the sample limits generalizability to other ethnic/racial groups. Fifth, while the participants were necessarily twins, the findings may not apply to singletons. However, past research supports the applicability of findings from twin samples to other samples (e.g., Kendler et al., 1995).

Conclusion

This study extends knowledge of genetic and environmental influences on trait anxiety over the course of middle adolescence to early adulthood. Genetic and shared environmental factors evidenced changes in their relative contributions to variance in trait anxiety across adolescence with increasing genetic influences and decreasing shared environmental influences. Common genetic influences contributed to trait anxiety across ages 14, 18, and 21, supporting a genetically-influenced mechanism underlying the developmental stability of trait anxiety. Sources of nonshared environmental influences were unique to each age, indicating developmental shifts in relevant environmental risks. Future research should seek to identify the specific genes that consistently influence trait anxiety across development, describe the non-shared environmental risk factors that are important at each age, and investigate gene-environment interplay as it influences trait anxiety across this developmental period.

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Table 1. Means, Standard Deviations, and Phenotypic Correlations for Trait Anxiety at Ages 14, 18, and 21

	14 years	18 years	21 years
Means	31.56	34.68	33.83
(SD)	(5.79)	(7.71)	(7.62)
Phenotypic Correlations			
Age 18	.42***		
Age 21	.33***	.64***	

Notes. *** $p < .001$.

Table 2. *Twin Intraclass Correlations for Trait Anxiety by Age*

	Age 14			Age 18			Age 21		
	<i>N</i>	<i>r</i>	95% CI	<i>N</i>	<i>r</i>	95% CI	<i>N</i>	<i>r</i>	95% CI
MZ	226	.40	(.29, .51)	319	.43	(.35, .52)	356	.46	(.38, .54)
DZ	119	.36	(.19, .50)	176	-.01	(-.15, .14)	199	.16	(.03, .30)

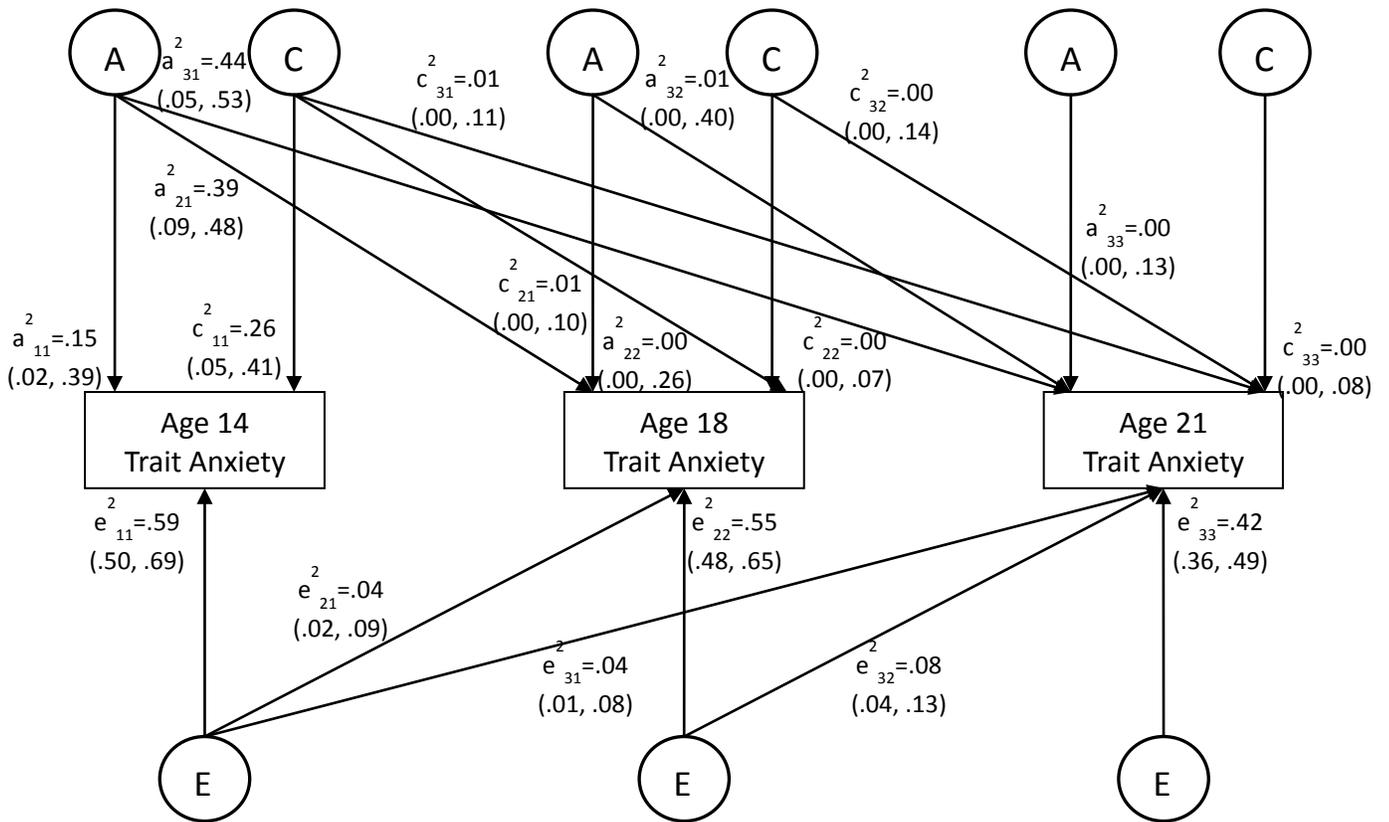
Notes. MZ=monozygotic. DZ=dizygotic. All correlations are significant at $p < .001$.

Table 3. *Test Statistics for Cholesky Decomposition Models for Trait Anxiety*

<i>Model</i>	<i>-2lnL</i>	<i>df</i>	Δ -2lnL (<i>df</i>)	<i>p</i>	<i>AIC</i>
1. Fully age unconstrained (baseline)	7303.29	2814			1675.29
2. Constrain A at ages 14 and 18 years	7307.14	2815	3.85 (1)	.04	1677.14
3. Constrain A at ages 18 and 21 years	7303.88	2815	0.59 (1)	.44	1673.88
4. Constrain C at ages 14 and 18 years	7308.80	2815	5.51 (1)	.02	1678.80
5. Constrain C at ages 18 and 21	7303.29	2815	1.00 (1)	.32	1673.29
6. Constrain E at all ages	7304.65	2816	1.36 (2)	.51	1670.65
7. Constrain:	7304.83	2819	1.54 (4)	.82	1666.825
A: ages 18 and 21 years					
C: ages 18 and 21 years					
E: at all ages					

Notes. Abbreviations: A=additive genetic effects. C=shared environmental effects. E=nonshared environmental effects. -2lnL= -2 times the log likelihood. AIC=Akaike Information Criteria. Δ -2lnL=differences in 2lnL values between the sex-constrained but age unconstrained model (model 1). All χ^2 tests compare models to model 1 (fully age unconstrained model). The best fitting model is bolded.

Figure 1. *Changes in Genetic and Environmental Influences on Trait Anxiety at Ages 14, 18, and 21*



Standardized Variance Estimates with 95% Confidence Intervals

Mean Age	a ²	c ²	e ²
Age 14	.15 (.02, .39)	.26 (.05, .41)	.59 (.50, .69)
Age 18	.39 (.26, .48)	.01 (.00, .10)	.60 (.52, .69)
Age 21	.45 (.29, .53)	.01 (.00, .14)	.54 (.47, .62)

Notes. a², c², e² = proportion of variance accounted for by genetic, shared environmental, and nonshared environmental influences, respectively.

Figure 1. This figure depicts the standardized path diagram for the full Cholesky decomposition model, including 95% confidence intervals for all parameters. Path coefficient estimates have been squared and represent the proportion of variance in trait anxiety accounted for by A_i (additive genetic), C_i (shared environmental), and E_i (nonshared environmental) effects. The table presents the overall proportions of variance in trait anxiety attributed to genetic, shared environmental, and nonshared environmental influences at each age.