

# A Twin Study of State and Trait Anxiety in Childhood and Adolescence

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Little research has addressed the relative influence of genetic and environmental factors on subclinical levels of anxiety in children. Of the two twin studies to date, one concluded that measures of adolescents' self-reported trait anxiety were best explained by shared environmental factors (Thapar & McGuffin, 1995), while the second determined that approximately half the variance was attributable to genetic effects (Topolski et al., 1997). The present study, using a sample of 547 twin pairs, reached conclusions similar to those of Topolski et al. Heritability was estimated at 45%. Measures of state anxiety conformed more closely to Thapar and McGuffin's findings, with environmental factors accounting for the variance.

*Keywords:* Anxiety, behavioural genetics, genetics, twins.

*Abbreviations:* AIC: Akaike's Information Criterion; DZ: dizygotic; MTFs: Minnesota Twin Family Study; MZ: monozygotic; STAI: State-Trait Anxiety Inventory; STAIC: State-Trait Anxiety Inventory for Children.

## Introduction

Anxiety, both as a symptom and as a syndrome, is frequently reported by children and adolescents. Community-based studies have found anxiety disorders to be the most prevalent psychiatric disorder for these age ranges (Kashani & Orvaschel, 1988, 1990). In one sample of 14- to 16-year olds, 17.3% reported a sufficient number of symptoms to meet criteria for an anxiety disorder (Kashani & Orvaschel, 1988). Other estimates of anxiety-disorder prevalence include 8.9% of 7- to 11-year-olds in a pediatric primary care setting (Costello et al., 1988), and 10.4% of children, aged 4 to 16, from a Puerto Rican community sample (Bird et al., 1988). Anxiety is thus not uncommon in children and adolescents. Neither is it necessarily innocuous, even at subclinical levels, as one study found children's self-reported anxiety to be predictive of poorer adaptive functioning 4 years later (Ialongo, Edelsohn, Werthamer-Larsson, Crockett, & Kellam, 1994).

It has been established that anxiety disorders aggregate within families (see Marks, 1986, for a review). For example, Weissman, Leckman, Merikangas, Gammon, and Prusoff (1984) found the children of panic disorder probands to be at an increased risk for anxiety disorders. Last, Hersen, Kazdin, Orvaschel, and Perrin (1991) discerned a higher prevalence of anxiety disorders in the first-degree relatives of affected children, as compared to controls. Familial aggregation, however, does not necessarily

imply heritability, as common genes are confounded with common environment. Twin or adoption methodology must be employed to disentangle these effects and quantify the genetic component. Two large studies of adult twins have done this, yielding heritability estimates for a number of different anxiety disorders ranging from .30 to .44 (Kendler, Neale, Kessler, Heath & Eaves, 1992; Kendler et al., 1995).

Symptoms of anxiety have also been shown to have a significant genetic component. One large sample of adult twins produced heritability estimates for state anxiety symptoms similar to the previously mentioned heritability estimates for adult anxiety disorders (Kendler, Heath, Martin, & Eaves, 1986). A family study calculated an upper limit heritability of .43 for anxiety symptoms (Tambs, 1991). Two other population-based samples, one a combination family/twin study, the other a large adult twin study, generated heritability estimates ranging from approximately .20 to .35 (Jardine, Martin, & Henderson, 1984; Tambs & Mow, 1993). Within the 8- to 16-year-old range, Topolski et al. (1997) recently determined that the heritability of trait anxiety ranged from .23 to .45 for males and .42 to .57 for females.

The majority of studies have been in agreement that there exists a genetic predisposition to anxiety at both clinical and subclinical levels. They have also been in accord that the remaining variance is almost entirely attributable to environmental influences unique to each individual (Jardine et al., 1984; Kendler et al., 1986, 1992). Those influences shared by members of a family appear to be of negligible importance in the etiology of anxiety symptoms and disorders.

There has not, however, been universal concurrence. In one of the only twin studies of childhood and adolescent

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anxiety symptoms, parents' ratings yielded a heritability estimate of .59; however, this dropped to zero when self-ratings of the adolescents, aged 12 to 17, were employed (Thapar & McGuffin, 1995). Analysis of these twins' self-ratings suggested that any familial transmission was best explained by shared environmental factors only.

Thapar and McGuffin's study is not alone in concluding that a model other than one of genetic and nonshared environmental influences best fits the data. Tambs and Moum's (1993) adult study detected non-trivial shared environmental influences on symptoms of anxiety. In Torgersen's (1983) adult twin study, genetic factors were implicated for panic disorder but not for generalized anxiety disorder. In Andrews, Stewart, Allen, and Henderson's (1990) study of anxiety disorders, contrary to expectations, MZ (monozygotic) twins were not found to be significantly more concordant than DZ (dizygotic) twins; there did appear to be a genetic contribution to anxiety symptoms, but not to specific anxiety disorders. Lastly, in Topolski et al.'s (1997) child and adolescent study, genetic effects were found for overanxious disorder but not for separation anxiety disorder.

The relative contribution of genes and environment in the etiology of anxiety, especially in children, remains uncertain. Although a number of studies have addressed anxiety disorders and symptoms in nonadult populations (Abe & Masui, 1981; Bell-Dolan, Last, & Strauss, 1990; Bird et al., 1988; Ialongo et al., 1994; Kashani & Orvaschel, 1990), few have estimated the strength of genetic contributions to anxiety symptoms. Thapar and McGuffin (1995) and Topolski et al. (1997) have been the only published twin studies to do so, and they have come to conflicting conclusions.

In sum, anxiety is a significant problem among children and adolescents, being neither uncommon nor necessarily harmless. Given the substantial evidence suggesting that genetic factors are important in adult anxiety, it seems reasonable to assume the same for childhood anxiety. However, the only two published genetic analyses are in disagreement. Thapar and McGuffin (1995) concluded that there was no genetic component to self-reported trait anxiety in adolescents, but Topolski et al. (1997) concluded that genetic effects play a modest to moderate role in the etiology of trait anxiety within both the child and adolescent age ranges. With this paper, we hope to add evidence to this debate. A large sample of female twins, ages 11 and 17, completed the State-Trait Anxiety Inventory (STAI) and the State-Trait Anxiety Inventory for Children (STAIC; Spielberger, 1983). Thus, the heritability of subclinical-level anxiety, both in more stable (trait) and fluctuating (state) forms, was investigated.

## Method

### *Participants*

Participants were female twins recruited to the Minnesota Twin Family Study (MTFS), a population-based, longitudinal study of twin adolescents and their families. The twins were born in the state of Minnesota and had been identified from public birth records for 1981–1984 and 1975–1978. At the time of their visit, the younger cohort ranged in age from 10 to 12,

averaging age 11; the older cohort ranged in age from 16 to 18, averaging age 17. They will be referred to as the 11- and 17-year-old groups.

There were 337 twin pairs aged 11 for whom complete anxiety data were available. For 26 pairs, zygosity had not yet been determined, leaving 311 twin pairs (188 MZ pairs, 123 DZ pairs). For the 17-year-olds, complete data were available for 243 pairs, but zygosity was undetermined for 7 pairs, leaving 236 twin pairs (155 MZ, 81 DZ). The excess of MZ pairs does not necessarily reflect a recruitment bias, as there is an overrepresentation of MZ twins in the population from which the sample was drawn (see Hur, McGue, & Iacono, 1995).

### *Zygosity Determination*

Three separate estimates were used to determine zygosity. Parents reported on physical similarity, staff evaluated physical similarity (visage, hair color, ear shape, etc.), and a third assessment was provided by an algorithm that makes use of ponderal and cephalic indices and fingerprint ridge counts. When the three estimates did not agree, a blood sample was requested and a serological analysis done. The twins in our initial sample for whom zygosity was not known were either twins of ambiguous zygosity who refused to have their blood drawn or twins whose blood had been drawn but the serological analysis was not yet completed.

### *Measures*

Anxiety was assessed using the STAI and the STAIC (Spielberger, 1983). State and trait scales, for both ages, are each 20 items in length. The response format differs slightly for the 11- and 17-year-olds, with the 11-year-olds responding on a 3-point scale and the 17-year-olds on a 4-point scale.

On the state scales, subjects rate how they feel at that very moment, using questions such as "I feel upset" and "I feel nervous." On the trait scale, the intensity of more typical feelings of tension, apprehension, and nervousness is assessed. For instance, it contains questions such as "I worry about making mistakes" and "I have trouble making up my mind." Eleven-year-olds are asked how they feel in general, while 17-year-olds are asked how often, in general, a statement applies to them.

The state questions were designed to tap temporal fluctuations in anxiety that are influenced by situational stressors. The trait questions measure more stable, individual differences in anxiousness or anxiety proneness. The two are believed to be related in that trait anxiety may be expressed through the tendency to regard situations as threatening and to respond with state anxiety. Reynolds (1980) determined that the trait and state scales of the STAIC correlated .35, thus conforming to theory.

### *Procedure*

As part of the MTFS, subjects participate in a morning of psychophysiological assessment. The laboratory procedures are unfamiliar to most and thus potentially anxiety provoking. Each twin filled out a State Anxiety self-report form before the laboratory work and another after completion. As expected, mean state scores decreased from first to second administration. The Trait form was administered midway through the laboratory assessment. This resulted in four scores for each twin: a trait anxiety score, two state scores (before and after), and a difference score representing the change in state scores.

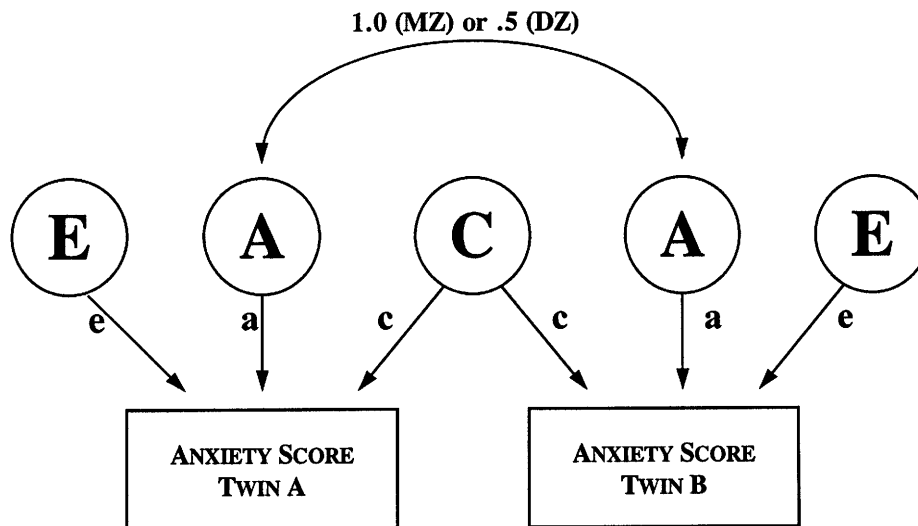


Figure 1. Univariate model of twins' anxiety scores. Etiological factors for anxiety are partitioned into additive genetic factors (A), common environment (C), and nonshared environment plus measurement error (E). Path coefficients are indicated by lower-case letters. The squares of the path coefficients equal the proportion of variance in the anxiety scores accounted for by A, C and E. The correlation for additive genetic factors (A) is 1.0 for MZ twins and .5 for DZ twins. The correlation for shared environmental factors (C) is 1.0 for both MZ and DZ twins.

### Analysis

Twin study methodology was employed. This method uses the difference between MZ and DZ twins, in proportion of shared genes, to estimate genetic and environmental contributions to observed behaviors or characteristics (i.e. phenotypes). MZ twins are genetically identical whereas DZ twins share, on average, 50% of their segregating genes. Assuming both types of twins share, to equal extents, those environmental factors that are etiologically relevant (see Kendler, 1993, for a defense of this assumption), the MZ correlation is expected to be greater than the DZ correlation for any genetically influenced trait. Thus, the twin design allows an estimate of the degree of genetic influence—and, alternatively, of environmental influences—from the differences between the MZ and DZ twin correlations. (See Plomin, DeFries, McClearn, & Rutter, 1997, for a more detailed explanation of twin study methodology.)

### Model Fitting

Univariate model-fitting analyses were done using the Mx structural equation modeling program developed by Neale (1995). Mx fits models to covariance and correlation matrices, employing maximum-likelihood model fitting. This method maximizes the fit between the model and the data, and thus determines those parameter estimates providing the smallest discrepancies with the data. Model fitting offers the advantage of not only testing the fit of a particular model, but allowing a comparison in fit of alternative models.

The chi-square statistic is used as a goodness-of-fit index, representing the degree of fit between model expectations and observed data. Statistically significant chi-square values are associated with a relatively poor fit. Improvements in the model's fit, from adding or omitting parameters, can be assessed by noting the change in chi-square, as the difference between the chi-square of an initial model and a nested model is itself distributed (under the null hypothesis) as chi-square.

When the parameters of one model are not a subset of the parameters of the other (i.e., not tested), Akaike's Information Criterion (AIC; Akaike, 1987) may be used to compare them. The AIC statistic is the model's chi-square minus twice its

degrees of freedom. In using the AIC, one is considering goodness of fit while penalizing overparameterization. As the aim of model fitting is to explain the data as parsimoniously as possible, the model with the lowest AIC value is generally considered to be the best.

### Results

Table 1 shows the means and standard deviations of the MZ and DZ twins' scores on the four measures. MZ and DZ score means were compared using two-tailed *t*-tests. In only one case was the difference between the means statistically significant: For the 11-year-olds' trait scores, it was significant at the  $p < .01$  level. At this age, MZ and DZ mean trait scores differed by 1.40 points on a scale with a 40-point range; thus, although statistically significant, the mean difference was not substantial. Levene's test for equality of variance revealed no statistically significant differences between MZ and DZ score variances. Considering both MZ and DZ scores together, data were approximately normally distributed within the two age groups, although generally somewhat positively skewed (range of skewness:  $-0.1$  to  $1.0$ ).

Prior to model-fitting analyses, intraclass twin correlations were calculated using the double-entry method (see Table 2). A double-entered intraclass correlation removes variance differences resulting from the arbitrary ordering of the twins within each pair. This provided a preliminary indication of the extent to which genetic and environmental factors influenced anxiety scores. Because different scales were used with the 11- and 17-year-old children, correlation rather than covariance matrices were used for the univariate model-fitting analyses.

The univariate model-fitting analysis began with the full ACE model. A between-groups analysis was performed in which the three parameters were alternately freed and fixed. In the free or unconstrained condition,

Table 1.  
*Means and Standard Deviations for State and Trait Anxiety Scores*

	11-year-old twins				17-year-old twins			
	MZ ( <i>N</i> = 188 pairs)		DZ ( <i>N</i> = 123 pairs)		MZ ( <i>N</i> = 155 pairs)		DZ ( <i>N</i> = 81 pairs)	
	Mean	<i>SD</i>	Mean	<i>SD</i>	Mean	<i>SD</i>	Mean	<i>SD</i>
Trait anxiety	31.86	5.98	33.26	6.24	37.76	8.02	38.18	7.70
State anxiety (before)	29.60	4.72	29.95	4.33	34.11	7.60	35.56	8.08
State anxiety (after)	26.18	4.28	26.40	4.44	29.39	7.75	30.78	7.23
Difference score	3.42	5.11	3.55	5.34	4.72	7.07	4.78	7.12

In only one case was the difference between the MZ and DZ mean scores statistically significant. For the 11-year-old trait scores, the difference in MZ and DZ mean scores was significant at  $p < .01$ . Levene's test for homogeneity of variance revealed no significant differences between MZ and DZ score variances.

Table 2  
*Intraclass Twin Correlations for State and Trait Anxiety Scores*

	11-year-old twins		17-year-old twins	
	MZ ( <i>N</i> = 188 pairs)	DZ ( <i>N</i> = 123 pairs)	MZ ( <i>N</i> = 155 pairs)	DZ ( <i>N</i> = 81 pairs)
	Trait anxiety	.475	.176	.394
State anxiety (before)	.464	.296	.341	.446
State anxiety (after)	.119	.121	.280	.255
Difference score	.165	.141	.161	.143

In only one case was the difference between the MZ and DZ twin correlations statistically significant. For the 11-year-old trait scores, the difference in MZ and DZ twin correlations was significant at  $p < .01$ .

Table 3  
*Model-fitting Results for Parameters Fully Constrained across Age Groups*

Scale	Model	Parameter estimates (and 95% CIs)			Model fit					
		$a^2$	$c^2$	$e^2$	$\chi^2$	<i>df</i>	<i>p</i>	AIC	$\Delta\chi^2$	$\Delta df$
Trait anxiety	ACE	.40 (0.11, 0.51)	.04 (0.00, 0.29)	.56 (0.49, 0.64)	2.36	3	.50	-3.65	—	—
	AE	.45	—	.55	2.44	4	.66	-5.56	0.08	1
	CE	—	.37	.63	9.90	4	.04	1.90	7.54**	1
	E	—	—	1.00	88.62	5	.00	78.62	86.26**	2
State anxiety (before)	ACE	.11 (0.00, 0.40)	.30 (0.05, 0.45)	.59 (0.51, 0.67)	3.71	3	.29	-2.29	—	—
	AE	.44	—	.56	8.99	4	.06	0.99	5.28*	1
	CE	—	.39	.61	4.27	4	.37	-3.73	0.56	1
	E	—	—	1.00	94.05	5	.00	84.05	90.34**	2
State anxiety (after)	ACE	.04 (0.10, 0.29)	.16 (0.00, 0.27)	.80 (0.71, 0.89)	3.37	3	.34	-2.63	—	—
	AE	.22	—	.78	4.56	4	.34	-3.44	1.19	1
	CE	—	.19	.81	3.43	4	.49	-4.57	0.06	1
	E	—	—	1.00	23.84	5	.00	13.84	20.47**	2
Difference score	ACE	.06 (0.00, 0.27)	.11 (0.00, 0.24)	.83 (0.73, 0.92)	0.01	3	1.00	-5.99	—	—
	AE	.19	—	.81	0.62	4	.96	-7.38	0.61	1
	CE	—	.16	.84	0.12	4	1.00	-7.88	0.11	1
	E	—	—	1.00	14.63	5	.01	4.63	14.62**	2

$a^2$  = additive genetic variance;  $c^2$  = shared environmental variance;  $e^2$  = nonshared environmental variance plus measurement error.

The significance of the difference in a reduced versus the full ACE model is assessed using the change in chi-square ( $\Delta\chi^2$ ); statistically significant values for this statistic are \*  $p < .05$  and \*\*  $p < .01$ .

the parameters were free to vary across the 11- and 17-year-old groups; in the constrained condition, the parameters' values were fixed across the two groups. For all four variables, the fully constrained model fit best, suggesting the proportion of variance attributable to each of the parameters did not change with age.

Fixing the three parameters across ages, and thus treating all MZ twins as one group and all DZ twins as another, analyses were conducted to determine whether any parameters could be dropped without compromising model fit. These results are summarized in Table 3. Also given in Table 3 are the 95% confidence interval estimates for the proportion of variance attributable to genetic ( $a^2$ ), shared environmental ( $c^2$ ), and nonshared environmental factors ( $e^2$ ). The confidence intervals were estimated using the procedure described by Neale and Miller (1997).

For trait anxiety, the full or ACE model fit well [ $\chi(3) = 2.36$ , n.s.]. However, the improvement in fit over the AE model was modest [ $\chi^2(1)$  difference = 0.08, n.s.]. When additional parameters do not significantly improve fit, the rule of parsimony gives preference to the simplest model; here, it was the AE model. The AIC statistic confirmed this. The CE model was easily rejected against the full model, as the difference in  $\chi^2$  between the full and reduced model was highly significant.

In contrast, the CE model provided the optimal fit for the three state-based variables. For the initial state anxiety score, the C parameter could not be dropped without significantly compromising model fit. The A parameter, on the other hand, could be dropped [ $\chi^2(1)$  difference = 0.56, n.s.]. Again, the low AIC value confirmed that the model best balancing parsimony and goodness of fit was the CE model. For the final two variables, either C or A, but not both, could be dropped from the full model without a significant deterioration in fit. However, in each case, the AIC values supported a preference of the CE over the AE model.

In summary, the trait anxiety scores were best fit by an AE model, with heritability estimated at 45%. The three state-based variables were best fit by a CE model, without the inclusion of a genetic parameter. That trait but not state scores were heritable attests to the two types of scales measuring distinct aspects of anxiety, as they were designed to do.

## Discussion

Results, based on analyses of 343 pairs of MZ twins and 204 pairs of same-sex DZ twins, suggest trait anxiety symptoms are moderately heritable, with additive genes accounting for 45% of the variance. The remaining variance is attributable to nonshared environmental influences plus measurement error. The variance of the three state-based variables, on the other hand, could be accounted for without the inclusion of a genetic parameter.

Trait anxiety's moderate heritability is consistent with the literature on personality traits, including neuroticism. Most studies of self-report personality scales have yielded heritability estimates around .50 (see Goldsmith, 1983, and Loehlin, 1992, for reviews). The remaining variance for personality traits has typically been of the nonshared variety, again similar to the present findings.

That Thapar and McGuffin (1995) did not conclude similarly was probably a consequence of their relatively small twin sample (19 MZ and 19 DZ twin pairs), for the questionnaire they used, the Revised Children's Manifest Anxiety Scale (RCMAS), is designed to measure trait anxiety. Its scores correlate highly ( $r = .85$ ) with the trait scale of the STAIC (Roberts, Vargo, & Ferguson, 1989). Yet, if the present study had only examined the scores of 17-year-olds, its conclusions may have resembled Thapar and McGuffin's. Within this older age range, the difference between MZ and DZ trait-score correlations was not statistically significant. Nevertheless, as the difference in magnitude between MZ and DZ correlations did not significantly vary by age, the 11- and 17-year-olds' data could appropriately be combined. When so examined, trait anxiety does appear to be moderately heritable.

Given the relationship between state and trait anxiety, it is somewhat surprising that the state scores were best modeled without a genetic parameter and with a shared environmental parameter. Perhaps this is a consequence of the unique situation in which the twins completed the questionnaires. Conceivably, sharing the uncommon and potentially anxiety-provoking state of filling out the questionnaires in a laboratory was more influential than any genetic similarity.

In conclusion, this study of children and adolescents fits with the majority of adult behavior genetic literature, and fits with one of only two nonadult behavior genetic studies of anxiety, in observing a substantial heritable component to enduring anxiety. This finding is especially apparent within the 11-year-old age range. Measures of more transient anxiety were accounted for by environmental factors only; however, this may have resulted from the unusual circumstances under which the questionnaires were completed. Previous to this study, there had been a paucity of genetic analyses of anxiety in the nonadult age range. Anxiety is a significant problem and greater understanding of its etiological components may lead to greater understanding of those factors necessary in alleviating it.

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