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Anger/frustration, task persistence, and conduct problems in childhood: a behavioral genetic analysis

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Abstract

Background—Individual differences in conduct problems arise in part from proneness to anger/frustration and poor self-regulation of behavior. However, the genetic and environmental etiology of these connections is not known.

Method—Using a twin design, we examined genetic and environmental covariation underlying the well-documented correlations between anger/frustration, poor attention regulation (i.e., task persistence), and conduct problems in childhood. Participants included 105 pairs of MZ twins and 154 pairs of same-sex DZ twins (4–8 year olds). Independent observers rated child persistence and affect based on behavior during a challenging in-home cognitive and literacy assessment. Teachers and parents provided reports of conduct problems.

Results—Persistence, anger/frustration, and conduct problems included moderate heritable and nonshared environmental variance; conduct problems included moderate shared environmental variance as well. Persistence and anger/frustration had independent genetic covariance with conduct problems and nonshared environmental covariance with each other.

Conclusions—The findings indicate genetically distinct though inter-related influences linking affective and self-regulatory aspects of temperament with behavior problems in childhood.

Keywords

Behavior problems; temperament; genetics

Growth in conduct problems (CP; aggression, delinquency, and oppositional behavior) involves aspects of temperament including negative mood and difficulties in self-regulating behavior. Though evidence of associations between these temperament and behavior problem constructs is well established, the underlying genetic and environmental etiology of the links is not known. In the current twin study, we tested for independent and overlapping genetic and environmental sources of covariation, focusing on specific indicators of negative affect (anger/frustration) and self-regulation (task persistence).

Temperament and behavior problems

The tendency to react in anger and frustration to provocation or challenge, coupled with poor self-regulation of those reactions, are aspects of temperament that put some children at risk for developing conduct problems (CP). There is mounting evidence from behavioral, cognitive and physiological studies of associations between higher levels of anger and frustration, lower levels of self-control of attention and behavior, and higher levels of CP. According to current theories, the correlation between negative affect and self-regulation implicates executive control as a modulator of emotion expression and its impact on behavior problems (Calkins & Fox, 2002; Eisenberg et al., 2005; Rothbart, Ellis, Rueda, & Posner, 2003).

Contemporary models of brain activation and behavior posit separate neural systems in the differentiation of positive and negative affects, arousal, and approach/avoidance. Though separate, these systems are connected with each other, as well as with more primitive regions involved in emotion (e.g., amygdala). They also interact with distinct cortical regions involved in the orientation and regulation of attention as well as cognitive control of many aspects of emotion and behavior (e.g., prefrontal cortex) (Davidson, Jackson, & Kalin, 2000; Gray, 2004; Heilman, 1997; Posner & Rothbart, 2005). In parallel, there also should be evidence of distinct but correlated genetic influences on CP operating through affective and self-regulatory phenotypes, although that research has not been conducted. A first step in testing for such evidence is to conduct multivariate behavioral genetic analyses that consider simultaneously the overlapping and distinctive genetic and non-genetic influences that account for the links between affect and CP, and self-regulation and CP – the goal of the current study.

Considerable research on genetic influences of CP has been conducted, although it remains unknown whether the correlations with anger and self-regulation reflect common or distinctive genetic and environmental etiologies. Individual differences in CP include moderate genetic, shared environmental and nonshared environmental sources of variance (Rhee & Waldman, 2002). Negative affect (and anger in particular) includes moderate to substantial genetic variance and nonshared environmental variance (i.e., non-genetic influences that cause family member differences), with evidence of shared environmental variance (i.e., non-genetic influences that cause family member similarity) being scant (e.g., Goldsmith, Buss, & Lemery, 1997; Oniszczenko et al., 2003; Emde, Robinson, Corley, Nikkari, & Zahn-Waxler, 2001). Genes in the serotonin neuro-transmitter system (e.g., TPH) have been implicated in studies of trait anger and frustration (Manuck et al., 1999; Rujescu et al., 2002). Attention span and persistence are moderately heritable, with recent cross-sectional data pointing to growth in heritable variance and dissipation of shared environmental variance over the transition to middle childhood (Deater-Deckard et al., 2005; Goldsmith et al., 1997). Genes in the dopamine neurotransmitter system have been implicated in pathological and non-pathological attention regulation (Maher, Marazita, Ferrell, & Vanyukov, 2002; Schmidt, Fox, Perez-Edgar, Hu, & Hamer, 2001).

There have been a number of investigations testing for common versus independent genetic liability for behavioral disorders in childhood and adulthood. For instance, Coolidge, Thede, and Young (2000) and Dick, Viken, Kaprio, Pulkkinen, and Rose (2005) reported that the covariance in levels of symptoms for ADHD and other disorders (CD, ODD, and Executive Function deficits) is accounted for by a moderate to substantial genetic correlation involving attention regulation and CP. However, in these studies, the separable influence of angry affect was not examined, and attention and CP were confounded within measures and diagnoses. What is needed are studies that test for genetic and environmental correlations in the links between specific aspects of temperament and child behavior problems based on theoretical models, ideally using different methods and informants for assessing temperament and CP (Lahey & Waldman, 2003).

Anger, persistence, and conduct problems: independent genetic influences?

In the current study, we used multivariate genetic methods to examine the associations between CP and two indicators of negative affectivity and self-regulation – anger/frustration and task persistence. We did so in order to address questions regarding the etiology of CP. Are the genes that account for variation in task persistence also accounting for variation in CP – and are those genes distinct from or the same as those that account for variation in anger/frustration? And how might environmental influences operate with respect to overlap between and independence of these three behaviors? To answer these questions, multivariate genetic analyses are required, because univariate genetic analyses address the sources of variance for only one phenotype at a time. Multivariate analyses test whether there are overlapping and independent genetic and non-genetic influences on correlated behaviors such as task persistence, anger/frustration, and CP – a first step toward identifying specific genetic and environmental pathways linking temperament and behavior problems (Martin & Eaves, 1977; Plomin, DeFries, McClearn, & Rutter, 1997, pp. 301–302).

The multivariate approach provides estimates of genetic correlation (i.e., extent of overlap in genetic variance between two constructs), as well as shared and nonshared environment correlation. There are only a few relevant multivariate genetic studies in which researchers used bivariate models to examine the correlation between one CP variable and one temperament variable at a time – typically using the same method and informant to assess both constructs. In a twin study, Gjone and Stevenson (1997) found evidence of a genetic correlation in the link between emotionality (labile negative affect) and behavior problems, and in separate twin and adoption studies, Schmitz and her colleagues found substantial genetic correlation in the link between emotionality and CP (Schmitz et al., 1999; Schmitz & Saudino, 2004). In a twin study that focused on ADHD symptoms rather than CP, Goldsmith, Lemery, and Essex (2004) found a substantial genetic correlation, as well as a nonshared environment correlation, linking ADHD symptoms with a lab-based measure of effortful control. Taken together, this small literature on bivariate quantitative genetic analyses suggests that the genetic correlations linking CP with persistence, and CP with anger/frustration, are likely to be moderate to substantial in magnitude, nonshared environmental correlations may be present, and shared environment correlations are not likely to be found.

A bivariate genetic analysis approach is an important first step, but it does not address whether the genetic and environmental influences on the associations between anger/frustration, persistence, and CP are overlapping or independent. The literature suggests that anger/frustration and persistence are genetically influenced behaviors that contribute to CP, and that anger and persistence stem from distinct albeit inter-connected biological pathways. If true, independent genetic influences on the link between anger/frustration and CP, and the link between persistence and CP, should be evident. In the current study, we examined the etiology of the correlations between anger/frustration, task persistence, and CP simultaneously, and more specifically, tested whether the estimated genetic and environmental correlations were independent, overlapping, or both. We used different methods and multiple independent informants for assessing temperament (observer ratings) and behavior problems (parent and teacher ratings) to minimize method effects.

Method

Participants

The data were drawn from the first wave of the Western Reserve Reading Project (WRRP), a longitudinal volunteer community twin study that includes 105 monozygotic (MZ) and 154 dizygotic (DZ) same-sex twin pairs (58% female, age $M = 6.09$ yrs, $SD = .69$ yrs, range = 4.33–7.92 yrs; no differences by zygosity). Parental education levels in the sample varied widely

and were similar for women and men: 12–17% high school or less, 23–29% some college or associate's degree, 30–31% bachelor's degree, 4–6% some post-graduate education, and 5% post-graduate degree. The sample included an over-representation of European Americans (92% vs. 86% in Ohio census) and under-representation of African Americans (5% vs. 12% in Ohio census; <http://www.census.gov/census2000/states/oh.html>).

The study was conducted with approval from Institutional Review Boards at the co-authors' institutions. The families in WRRP live in the Cleveland and Cincinnati metropolitan areas, as well as outlying areas throughout the state of Ohio. Recruiting was conducted through school nominations, Ohio State Birth Records, and media advertisements. Schools were asked to send a packet of information to parents in their school system with twins who had entered at least kindergarten but had not finished first grade. Participating families enrolled by completing a demographics questionnaire and returning it by mail with a signed consent form.

Procedures

Twins and parents completed a battery of behavioral and cognitive assessments (see Petrill et al., in press, for more information) during a three-hour home visit. Also, the mother was videotaped while interacting with each twin separately for 16 minutes while they completed two moderately frustrating games together: drawing pictures (standardized across families) using an Etch-A-Sketch toy, and moving a marble through a tilting maze box. Both toys were operated using two dials. The mother and child were assigned their own dials and asked not to touch each other's dials. Testers completed a brief questionnaire at the end of the home visit, and parents returned questionnaires to the testers that had been completed before the home visit. Different research assistants who had not conducted the home visits coded the videotaped interactions. For each twin pair, different testers rated each child and different observers coded each child's behaviors on videotape, thereby minimizing rater effects on estimates of twin similarity.

At the beginning of each home visit, the research assistants conducted informed consent and assent procedures in which the consent form was read, discussed and signed by the parent, and during which assent was gained from the twins. Families were given a \$100 honorarium. Once a home visit was conducted, each twin's teacher also was invited to complete a questionnaire. Those teachers who completed and returned a questionnaire received a \$10 gift certificate at an office supplies store.

Measures

Persistence—Task persistence is a component of a larger system of self-regulated attention and behavior that comprises executive function and effortful control (Anderson, 2002). Our measure of task persistence captured effortful, sustained attention and shifting of attention, corresponding to widely used measures of attention focusing, shifting, and inhibitory control that are substantially inter-correlated with each other while being distinct from reactive control (i.e., impulsivity, inhibition to novelty; see Derryberry & Rothbart, 1997; Eisenberg & Morris, 2002; Kochanska & Knaack, 2003).

The child's task persistence was assessed using observer ratings by independent testers and research assistants. At the end of the home visit, testers completed items from Bayley's (1969) Behavior Record, which are rated on 5-point Likert-type scales. We used the two items (reverse-scored for analyses) relevant to attention span and persistence (1 = lacks attention/persistence; 3 = half the time; 5 = constantly attends/persists). In addition, different research assistants completed observer ratings of each twin from the videotaped parent-child interactions after each home visit, using the Parent-Child Interaction System or PARCHISY (see Deater-Deckard et al., 2005) which involves completing a short questionnaire of items

rated on 7-point Likert-type scales. Coders achieved Cronbach's $\alpha > .75$ during training and maintained this level of reliability throughout data collection. For the current study, we used the item regarding on-task behavior (1 = none evident to 7 = constant). Thus, for each twin we gathered independent observers' ratings that also were independent from ratings of the co-twin. Principal components factor analysis supported combining the three items into a composite score (61% of variance explained, with loadings ranging from .55 to .88). The items were standardized, averaged, and standardized again to yield z -scores.

Anger/frustration—Like persistence, anger and frustration during completion of the in-home assessment battery and interaction tasks were rated by the testers (using the Behavior Record) and other research assistants (using the PARCHISY). We used two reverse-scored items from the testers' observer ratings pertaining to anger and frustration (1 = none/never displayed; 3 = three or more brief displays/occasionally frustrated; 5 = three or more intense displays/consistently becomes frustrated). We also used one item from the research assistants' observer ratings of negative affect (1 = none to 7 = constant). Principal components factor analysis again supported combining the three items (54% variance, loadings from .58 to .84). As with persistence, the items were standardized, averaged, and standardized again to derive z -scores.

Conduct problems—Teachers rated child behavior using the Teacher Report Form or TRF, and mothers and fathers completed the similar Child Behavior Checklist (Achenbach, 1991). These include items rated on a 3-point Likert-type scale (0 = not true, 1 = some-what or sometimes true, 2 = very true or often true). In this study, we analyzed the Externalizing Problems syndrome score that includes Aggression and Delinquency sub-scales. Again, principal components factor analysis supported combining the three respondents' scores into a reliable composite (55% variance, loadings from .50 to .86). As with persistence and anger/frustration, and consistent with other researchers (e.g., Valiente et al., 2003), the TRF and CBCL scores were standardized, averaged, and standardized again to yield z -scores. There were missing data for CP, especially for fathers' reports. Rather than choosing a criterion data source and discarding data (e.g., including only those with mothers' reports), we aggregated in a way that allowed any child with at least one valid mother-, father-, or teacher-rated CP score to receive a composite score. Twenty-one percent of the children had CP data from one source (usually the mother or teacher), 43% had CP data from two respondents (usually mother and teacher), and 36% had CP data from mother, father, and teacher.

Results

The descriptive statistics for the study variables are presented in Table 1. Although linear composite z -scores were used for subsequent analyses, information regarding the individual raw scores comprising the composites is shown for descriptive purposes. Indicators for all three of the study variables were skewed but highly variable – a typical pattern in community samples. The distributions of parent- and teacher-rated CP were much like those found in community-sample studies of non-twin children of the same ages (e.g., Lemery et al., 2002; Valiente et al., 2003). Also consistent with other findings (e.g. Valiente et al., 2003), transforming the variables to normalize the distributions did not influence the results.

Because the sample contained children nested within twin families, multilevel modeling procedures were employed (Snijders & Bosker, 1999) to account for the nonindependence of children living in the same family. In particular, a series of bivariate multilevel regression analyses were conducted using z -scored independent and dependent variables. Thus, the fixed effect estimates were analogous to Pearson correlations between the variables of study, but controlling for the fact that individual twins are nested within families. There were no significant associations between zygosity, gender, and the study variables. CP and anger/

frustration were not correlated with child age, but we found that older children showed higher levels of task persistence, *standardized fixed effect* (240) = .25, $p < .01$. Turning to inter-correlations between study variables (Table 2), all three variables were associated as expected; lower task persistence, higher anger/frustration, and higher CP covaried. Note that in the behavioral genetic analyses that follow, we estimated quantitative genetic parameters using the original z -score composites as well as standardized residuals controlling for age and gender. Controlling for age and gender had no effect on the results, so we report results for original z -scores only.

We also estimated twin intra-class correlations for each of the three constructs (Table 2). For all three variables, MZ twin similarity was higher than DZ twin similarity, suggesting the presence of genetic variance. To test whether there were common or independent genetic, shared environmental (i.e., leading to family member similarity), and nonshared environmental (i.e., leading to family member differences) parameters, a trivariate Cholesky decomposition was used to partition the variances of, and covariances between, persistence, anger/frustration, and CP (see Figure 1, which shows the model for one twin for ease of presentation). In this model, latent variables are estimated that represent independent and overlapping additive genetic effects (A), additive shared environment effects (C), and additive nonshared environment effects including error (E), as well as residual genetic (a), shared environmental (c), and nonshared environmental (e) variance. The pathways between latent variables representing genetic variance or covariance across twins are set at 1 for MZ twins and .5 for DZ twins. The pathways for shared environmental variance or covariance across twins are set at 1 for MZ and DZ twins, whereas the pathways for nonshared environmental variance or covariance across twins are set at 0 for MZ and DZ twins.

The path estimates derived from the Cholesky decomposition are used to compute the univariate variance estimates and genetic, shared environmental, and nonshared environmental correlation estimates, which are shown in Table 3 and which we focus on in interpreting the data. The univariate variance estimates for each variable are shown in the left half of Table 3. All three constructs had significant heritable variance (.2 range). Shared environmental variance estimates for persistence and anger/frustration were evident but not significant due to the relative lack of power afforded by the sample size, though shared environmental variance was significant for CP. Nonshared environmental variance estimates were moderate and significant for all three constructs.

Turning to estimates of covariance in the right half of Table 3, significant and substantial genetic correlations were found for covariance between persistence and CP, and between anger and CP, suggesting the presence of independent genetic influences on these links between phenotypes. None of the shared environmental correlations was significant (though the shared environmental correlation between persistence and anger was 1.0), ruling out shared environmental influences on the covariation between study variables. Finally, there was a moderate significant nonshared environmental correlation for persistence–anger/frustration, suggesting the presence of overlapping nonshared environmental influences on these two covarying components of temperament.

Discussion

There is an emerging consensus that both genetic and environmental factors contribute to the development of CP (Goldsmith et al., 2004; Moffitt, 2003). There also is growing evidence from studies of community and clinical samples that higher levels of anger and frustration, along with poorer self-regulation of attention and behavior, covary with higher levels of CP (Caspi, Moffitt, Newman, & Silva, 1996; Shiner, Masten, & Roberts, 2003). However, there has been little research addressing whether and how genetic and environmental influences

operate to explain the observed links between CP, negative affect, and self-regulation. Therefore, we conducted a behavioral genetic analysis to test for overlapping and independent genetic and environmental influences on the connections between CP, anger/frustration, and task persistence.

Turning first to the phenotypic associations between variables, higher levels of anger/frustration covaried with lower levels of task persistence. This is consistent with the idea that regulation of attention serves to control affect expression. The $-.57$ correlation that we found was larger than usual, perhaps due to inflation arising from shared method effects (see Kochanska & Knaack, 2003, $r = -.31$; Olson et al., 2005, $r = -.34$; Rothbart et al., 2003, r s in $-.2$ range; Rydell et al., 2003, r s in $-.4$ range). We found a correlation of $.16$ between anger/frustration and CP, which also is consistent with the literature, though on the low end of the range of effect sizes found (see Jenkins & Oatley, 2000, *betas* in $.3$ range; Lawson & Ruff, 2004, *betas* in $.3$ range; Lemery et al., 2002, average r s in $.3$ to $.4$ range; S. Olson, personal communication, June 20, 2005, regarding Olson et al. 2005, average r s in $.2$ range; Rydell et al., 2003, average $r = .33$; Schmitz et al., 1999, average r s in $.2$ range). This may be because our method of assessing anger was based on observations of low-frequency events of short endurance. We also found the expected association between lower task persistence and higher CP. The effect size in the current study ($r = -.19$) was similar to effects reported in previous studies that have used a variety of methods, although again this effect was smaller than found by others (Copeland et al., 2004, r s in $-.3$ range; Kochanska & Knaack, 2003, $r = -.37$; Lemery et al., 2002, average r s in $-.2$ range; Olson et al., 2005, average $r = -.29$; Rydell et al., 2003, average $r = -.20$; Valiente et al., 2003, average $r = -.24$).

Though informative, phenotypic analyses do not address whether and how genetic and environmental sources of variance operate to account for associations between temperament and behavior problems. Current theories posit distinct but inter-related influences on CP from limbic affective/motivational systems and cortical self-regulatory systems (Davidson et al., 2000; Gray, 2004; Heilman, 1997), which in turn may reflect distinct but correlated genetic influences on CP. To test this, we conducted multivariate genetic analyses to examine the sources of variance in, and covariance between, anger/frustration, task persistence, and CP. For anger/frustration and persistence, heritability approached $.3$ and nonshared environment was $.5$ – results that are consistent with the literature (e.g., Emde et al., 2001; Goldsmith et al., 1997). For CP, we found moderate genetic, shared environmental, and nonshared environmental variance estimates that also were consistent with previous studies (see Rhee and Waldman's (2002) meta-analysis of externalizing problems; $h^2 = .32$; $c^2 = .16$; $e^2 = .43$).

We then turned to estimate genetic, shared environmental, and nonshared environmental correlations to see whether there was evidence of independent or overlapping genetic and non-genetic influences across the anger-CP and persistence-CP phenotypic links. We found independent genetic correlations between anger/frustration and CP ($.80$), and task persistence and CP ($.86$). There also was a moderate though non-significant genetic correlation between persistence and anger/frustration ($.43$). This pattern of results is consistent with the idea that the biological pathways involved in affect and self-regulation are distinctive but inter-related in their operation and connections to other phenotypes such as CP. Anger/frustration and task persistence reflect independent genetic links with CP, operating through affective/motivational and self-regulatory mechanisms respectively.

Rater and method effects can bias multivariate genetic and environmental correlation estimates, a limitation in much of the previous phenotypic and behavioral genetic research. To reduce these effects on parameter estimation, we used parents' and teachers' reports of CP (behaviors that are difficult to observe in brief visits to the home or school), and multiple observers' ratings of anger/frustration and persistence (behaviors that can be observed during brief visits in which

children face challenging tasks). Observers' ratings of attention span and regulation converge with teachers' and parents' ratings (e.g., Goldsmith et al., 2004; Valiente et al., 2003), and observers' ratings of anger/frustration yield correlations with CP that are similar to those based on parents' reports of child mood (Jenkins & Oatley, 2000; Kochanska & Knaack, 2003; Valiente et al., 2003). For these aspects of temperament, observers' global ratings capture all of the information on individual variation that is assessed in more time-consuming event-based codes (see Lawson & Ruff, 2004), though our method is limited by its focus on brief, constrained observations. Another caveat is that we had low power for detecting shared environment effects due to the sample size. The small sample also prevented us from making better use of the cross-sectional design to consider possible age differences, an important consideration given the age range (4–8 years) of children in the study.

The results have implications for how it is that scientists and clinicians conceptualize the connection between temperament and behavioral problems in childhood. Correlational studies implicate proneness to anger/frustration and poor self-regulation of behavior in the development of CP. Our results suggest that the underlying genetic mechanisms involved in affective and self-regulatory aspects of temperament may be distinct, while still being inter-related. Given the heterogeneity of symptoms within diagnosed groups of children (e.g., conduct disorder, ADHD), it may be worthwhile to identify sub-groups using measures of anger/frustration and persistence that are more homogeneous and that reflect different patterns of genetic risk, which in turn may lead to improvements in diagnosis and treatment. Although we view community studies like ours as informative in regards to the etiology of clinical disorders, it is important to bear in mind that the children in the current study showed behaviors in the 'normal', not 'clinical', range of symptoms of CP.

With regard to intervention, statistically significant genetic variance and genetic correlation do not imply intractable individual differences. The connections between motivational factors, self-regulation, and CP very likely are modifiable. There is evidence from correlational and quasi-experimental studies of systematic environmental influences on attention and persistence in early childhood (Calkins & Fox, 2002; Deater-Deckard et al., 2005), and experiments with humans and non-human primates show that these behaviors and underlying neural systems may be altered through manipulation of environmental inputs (Posner & Rothbart, 2005). It remains to be seen how specific environmental influences on negative affect and self-regulatory capacity interact with specific genetic effects (i.e., dopamine and serotonin genes) in accounting for individual differences in behavior problems.

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Abbreviations

CP	Conduct problems
MZ	monozygotic
DZ	dizygotic

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Figure 1.

A multivariate Cholesky decomposition was used to partition the variances of, and covariances between, task persistence (TP), anger/frustration (A/F), and conduct problems (CP), into genetic and non-genetic components. For ease of presentation, the figure shows one half of the model for one twin's scores. Latent variables represent overlapping additive genetic effects (A), additive shared environment effects (C), and additive nonshared environment effects including error (E), as well as residual genetic (a), shared environmental (c), and nonshared environmental (e) variance, are identified in the model. The pathways between latent variables representing genetic variance or covariance across twins are set at 1 for MZ twins and .5 for DZ twins. The pathways for shared environmental variance or covariance across twins are set at 1 for MZ and DZ twins, whereas the pathways for nonshared environmental variance or covariance across twins are set at 0 for MZ and DZ twins.

Table 1
Descriptive statistics

	U	SD	Range
Task persistence			
Tester rating: Attention to tasks	4.32	.81	1–5
Tester rating: Persistence with tasks	4.23	.88	1–5
Research asst rating: Attention/Persistence	6.46	.75	3–7
Anger/Frustration			
Tester rating: Negative affect (anger, irritability)	1.59	.87	1–5
Tester rating: Frustration	1.45	.72	1–5
Research asst rating: Negative affect (anger, irritability)	1.27	.52	1–5
Conduct problems			
Teacher rating: TRF	3.33	6.28	0–42
Mother rating: CBCL	7.46	5.86	0–30
Father rating: CBCL	7.09	5.79	0–31

Table 2
Standardized fixed effect estimates from multi-level modeling and twin intra-class correlations for MZ/DZ twins

	Intra-class <i>r</i>		
	Standardized fixed effects		DZ
	MZ		
Task persistence	.51	1.00	.30
Anger/Frustration	.50	1.00	.33
Conduct problems	.72	.16	.48

Note: All significant, two-tailed $p < .05$.

Table 3

Genetic, shared environmental, and nonshared environmental variance estimates (with 95% confidence intervals) and correlations

	Heritability		Genetic correlations	
Task persistence	.28	(.06–.56)	—	
Anger/Frustration	.25	(.02–.58)	.43	—
Conduct problems	.29	(.09–.53)	.86*	.80*
		Shared environment		Shared environment correlations
Task persistence	.22	(.00–.40)	—	
Anger/Frustration	.23	(.00–.46)	1.00	—
Conduct problems	.38	(.15–.57)	.00	.00
		Nonshared environment		Nonshared environment correlations
Task persistence	.50	(.39–.62)	—	
Anger/Frustration	.51	(.40–.65)	.49*	—
Conduct problems	.32	(.23–.38)	.00	.00