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Genetic Relations Between Effortful and Attentional Control and Symptoms of Psychopathology in Middle Childhood

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Abstract

Elucidating the genetic and environmental etiology of effortful control (mother and father report at two time points), attentional control (observer reports) and their associations with internalizing and externalizing symptoms (mother and father report) is the central focus of this paper. With a sample of twins in middle childhood participating in the Wisconsin Twin Project, broad sense heritability for parental report effortful control ranged from 68–79%, with a slightly higher heritability estimate of 83% for observer report attentional control, and no influence of the shared environment on either trait. Further, measures of control were negatively correlated with internalizing and externalizing symptoms longitudinally, concurrently, and across reporters. Importantly, shared additive genetic influence accounted for the covariation between the control variables and symptoms of psychopathology. These results encourage identification of common genes that affect both effortful control and symptoms, and environmental triggers that uniquely influence symptoms of psychopathology.

The regulatory temperament dimension of effortful control has consistently been identified as one pathway that protects children from psychopathology, although we continue to know little of *how* this relationship operates. Rothbart and Bates (2006) propose three explanations for the relationship between positive aspects of temperament and psychopathology: (1) *Protective model*—the person has a predisposition to handling stressful situations well; (2) *Boost from adaptation model*—positive temperament is strengthened by the event of conquering challenge; and (3) *Spectrum or continuity model*—a case in which the positive temperament constitutes positive outcomes. With effortful control and child psychopathology symptoms, a protective model might provide the best explanation, with effortful control moderating the influence of other risk factors (e.g., characteristics of the rearing environment) on symptoms of child psychopathology (Lengua & Long, 2002; Morris et al., 2002; Patterson & Sanson, 1999; Rubin, Burgess, Dwyer & Hastings, 2003). Model Two is difficult to test because it requires measuring conquering a challenge, as well as pre and post measures of positive temperament. Previous studies documenting that measures of effortful control and symptoms do not share item content (e.g., Lemery, Essex & Smider,

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2001) suggest that Model Three does not represent the association between these constructs well.

Although they do not provide immediate answers to the “how” question, twin studies can assist in our conceptualization of the association between effortful control and symptoms of psychopathology by estimating the extent to which genetic and/or environmental influences account for their negative association. For example, if some of the genetic influence on psychopathology were shared with low effortful control, this would suggest that a set of common genes influences both. Such a finding would encourage identification of these common genes as well as isolation of environmental triggers for psychopathology. In addition, results from twin studies can be consistent with some models and not others. The finding that a common genetic factor accounts for the association between low effortful control and symptoms, for example, would discourage the hunt for environmental mediators of the association.

Rothbart and Bates (2006) define temperament as including both reactive and regulatory dimensions. Effortful control is one aspect of the regulatory system; it consists of the abilities to focus attention, to be flexible and adapt attention (i.e., attentional control), and to regulate behaviors or emotions (i.e., inhibitory control). At the same time, low effortful control is often predictive of both externalizing (e.g., conduct disorder, oppositional defiant disorder) and internalizing (e.g., depression, anxiety) types of child psychopathology (Caspi et al., 1995; Eisenberg et al., 2001; 2004; Lemery et al., 2002; Lengua, 2003; Oldehinkel et al., 2004). Theoretically, the inverse relationship between effortful control and externalizing is straightforward, since impulsive and acting out behaviors characterize externalizing behaviors. A relation between effortful control and internalizing is less clear. Perhaps the inability of individuals with internalizing symptoms to regulate negative emotions such as fear and sadness is due to poor attentional control (Shoda, Mischel, & Peake, 1990). Then again, effortful control and internalizing could have a more complicated relation such that the association could be mediated by other factors, such as resiliency (Eisenberg et al., 2004).

While numerous behavioral genetic studies have considered genetic and environmental influences on reactive temperament, many have neglected to consider effortful control or any other variations of regulatory temperament. With a somewhat small sample of young twins, Goldsmith, Buss and Lemery (1997) reported that parent-reported effortful control was 43–58% heritable (with significant additive, but not interactive, genetic influences), with a small shared environmental influence (0–12%) and the rest of the variance attributed to nonshared environmental influences. Similarly, with a sample of young adult Japanese twins self-reporting on their effortful control, Yamagata and colleagues (2005) reported a heritability of 49% (with significant additive, but not interactive, genetic influences), with the rest of the variance attributed to nonshared environmental influences. No teacher report or observed measures of effortful control have been considered in genetically informative designs.

At present, the few studies decomposing the genetic and environmental underpinnings of the temperament—psychopathology relationship have focused solely on reactive dimensions of temperament (e.g., negative emotionality). Gjone and Stevenson (1997a) reported that a

common genetic factor accounted for the association between negative affect at 7–12 years and either attention problems or aggressive behavior measured two years later. Schmitz et al. (1999) extended this finding to toddlers and preschool age children, showing that the covariance between negative affect as a toddler and externalizing problems at age 4 was accounted for by shared genetic influence. Likewise, a genetic correlation explained the relationship between negative affect and later internalizing symptoms. Goldsmith and Lemery (2000) accounted for the association between temperamental fear and two forms of anxiety, over-anxiousness and separation anxiety, two and a half years later when children were between the ages of five and ten. While a common genetic factor accounted for the relationship between fear and over-anxiousness, common shared environmental variance explained the association between fear and separation anxiety.

In the only adoption study to investigate this association, Schmitz and Saudino (2003) showed that nonshared environmental influences accounted for the relation of mother report of negative emotionality with internalizing and externalizing. However, for teacher report, both overlapping genetic influence and nonshared environmental influence explained the correlation of negative emotionality with internalizing and externalizing. Across raters, the association between teacher report of negative emotionality and mother report of internalizing-externalizing was accounted for by shared genetic influence. Each of the genetically informative studies considering the association between temperament and symptoms was longitudinal, which ensures that any findings of shared genetic influence were not situational.

Based on these previous results, we hypothesize that shared genetic influence will explain the majority of the association between effortful control and internalizing or externalizing symptoms, with the shared environment perhaps playing a small role, because Goldsmith et al. (1997) reported a small shared environmental influence on Effortful Control with young children, and variation in both internalizing and externalizing symptoms is partially due to shared environmental influences (see review by Lemery & Doelger, 2005).

Method

Participants

The sample consisted of 563 twin pairs recruited from birth records for the longitudinal Wisconsin Twin Project (WTP; Lemery-Chalfant, Goldsmith, Schmidt, Arneson & Van Hulle, 2006). Twins were born between the years 1992–1994 and tested when they were approximately eight-years-old ($M = 7.58$; $SD = 1.00$). Because rates of psychopathology are relatively low in population-based samples (around 12%; Roberts, Attkisson, & Rosenblatt, 1998), we screened for inclusion in the sample. Specifically, participants were selected as “at risk” if they scored greater than 1.5 *SD* above the mean on any of the mother report MacArthur Health and Behavior Questionnaire (HBQ) symptom scales, and were selected as control if they scored less than the mean on all symptom scales. Co-twins of selected twins were also a part of the sample and participated in all aspects of the study, i.e., both twins in a pair were included in all analyses and at least one twin of a pair met criteria for being either at risk or control. The sample contained approximately 35% at-risk individuals, and 31%

control. The remaining 34% of the sample were co-twins of the selected at-risk or control individuals; these cotwins did not meet criteria for either category.

The sample was 50% female (*N*=565) and 50% male (*N*=561). There were 214 MZ twin pairs, 198 same-sex DZ twin pairs, and 151 opposite-sex DZ twin pairs. The vast majority of the sample was Caucasian (95%), with 2% African-American, and 3% biracial, which is representative of Wisconsin. Four percent of the families made under $20,000 a year; 32% made $20,000–50,000; 54% made $50,000–100,000; and 10% made over $100,000. For fathers' education (mothers' in parentheses), 33% (22%) graduated from high school or had some high school, 33% (39%) had some college education, 22% (28%) were college graduates, and 13% (11%) held a graduate degree or had some graduate level courses.

A sub-sample had participated in an earlier assessment of temperament (*M* age = 5.5; *SD* = 1.3) and these assessments were included in some analyses to add a longitudinal component. There were 283 twin pairs (48% female) who participated in the earlier assessment, with 112 MZ pairs, 98 same-sex DZ pairs, and 73 opposite-sex DZ pairs.

Measures

Parents provided demographic information and zygosity was determined through agreement between parent and observer ratings. Parents completed the *Zygosity Questionnaire for Young Twins* (Goldsmith, 1991), which measures physical similarities. The agreement of this particular questionnaire with genotyping has been estimated at 96% (Forget-Dubois et al., 2003), rendering it a cost-efficient alternative. Observers also completed questions concerning zygosity after the home visit. For 12 of the twin pairs in this sample, parents and observers did not agree and these pairs were omitted from genetic analyses.

Rothbart's Children's Behavior Questionnaire (CBQ)—The CBQ is a parent-report measure of temperament for children from three to eight years of age (Rothbart et al., 2001). The WTP used 10-item versions of 12 of the 15 scales; Attentional Focusing (alphas in the .70s across mother and father reports at both time points in the current study) and Inhibitory Control (alphas in the .80s) were used. Parents rated the child's behavior over the past six months on a 7-point Likert scale ranging from “extremely untrue of your child” to “extremely true of your child”. Both mothers and fathers completed the CBQ. The Effortful Control composite was the mean of the Attentional Focusing and Inhibitory Control scales. These two scales were significantly correlated .53 at time one (T1), and .73 at time two (T2), $p < .01$. Mother and father reports of Effortful Control were correlated (T1 average $r = .59$, $p < .01$; T2 average r = .57, p <.01), and we computed mother-father mean composites.

Bayley Rating Scale (BRS)—revised items—Items based on the BRS (Bayley, 1993) but revised to be age-appropriate provided an observer report measure of attentional control reported by the two trained experimenters who conducted the home visit. These experimenters based their ratings on the child's behavior throughout the visit, including transitions from one task to the next. The measure consisted of 28 items rated on five-point scales. An example item is, “Consistently attends well; rarely, if ever, is off task”. A mean composite called Attentional Control was created from the Adaptation to Change in Test Materials, Attention to Tasks, and Persistence in Completing Tasks items. These items had

relatively high intercorrelations: adaptation and attention to tasks (average $r = .48$), adaptation and persistence (average $r = .39$), and attention to tasks and persistence (average $r = .54$). Attentional Control was significantly correlated across rater, [$r(196) = .48$ for twin 1 subsample, $r(194) = .53$ twin 2 subsample], and a mean composite was formed.

MacArthur Health and Behavior Questionnaire (HBQ)—The HBQ is a caregiver report questionnaire designed to assess the physical and mental health of 4 to 8 year old children (Armstrong et al., 2003; Boyce et al., 2002; Essex et al., 2002). Parents are asked to respond to questions by rating their child's behavior on three point scales, from 0 for not true for the child to 2 corresponding to very true for the child. Test-retest reliability over a one-week period was .80 for mother report (Ablow et al., 1999). Caregivers evaluate externalizing symptoms on a mean composite of four scales: Oppositional Defiant (9 items, alpha = .87 mother report, .83 father report), Conduct Problems (12 items, alpha = .83 mother report, .82 father report), Overt Hostility (4 items, alpha = .74 mother report, .69 father report) and Relational Aggression (6 items, alpha = .84 mother report, .76 father report). Internalizing symptoms are rated on three scales and a mean composite was formed: Overanxious (12 items, alpha = .82 mother report, .77 father report), Depression (7 items, alpha = .74 mother report, .63 father report), and Separation Anxiety (10 items, alpha = .84 mother report, .83 father report). Mother and father reports of internalizing (average $r = .51$, $p < .01$) and externalizing (average $r = .53$, $p < .01$) were correlated, and thus averaged for additional analyses.

Procedure

Mothers completed the HBQ, demographic information, and the zygosity questionnaire via a phone interview. Around three to four months later (M=3.72, SD=4.77), mothers participated in another phone interview during which they completed Rothbart's CBQ. At this time, the father also completed the HBQ and CBQ over the telephone. In addition, families participated in a four-hour home visit, which involved a number of assessments. Within a week of the home visit, the two trained experimenters who administered tasks to the twins during the home visit independently completed measures of each child's behavior (including BRS attentional control) after reviewing the entire four-hour videotape of the visit.

Approximately two and a half years earlier, mothers and fathers of a sub-sample completed questionnaire packets that included the CBQ.

Twin Methodology

Two independent samples were formed for use with statistical tests that assume independent observations. The Twin 1 subsample was created by randomly selecting one twin from each pair. The Twin 2 subsample was the co-twins of the Twin 1 subsample. All correlations and t-tests that could potentially be affected by the statistical dependence created by using two twins from each pair were tested separately with the Twin 1 and Twin 2 subsamples. Average correlations (using Fisher's Z transformation) across the two samples are reported.

Using structural equation modeling, full univariate twin models decompose the variance in a variable into three components: additive genetic influence (A; the sum of the average effects of individual genes across the genotype), shared environmental influence (C; aspects of the environment that make twins similar to one another), and non-shared environmental influence (E; aspects of the environment that make twins dissimilar to one another as well as measurement error). An alternative full model decomposes the variance into A, D (nonadditive, or dominant genetic influence, or the interactive influence of multiple alleles of the same gene), and E. These components were allowed to vary in magnitude across sex in a model testing for quantitative sex differences, but qualitative sex differences were not tested (because the opposite sex twin correlations were not less than the same sex twin correlations in this study).

C is completely shared between co-twins growing up in the same home, and E is completely independent across individuals. The latent A and D influences are correlated between individuals: for MZ twins, both the A and D genetic correlations equal 1 because they share 100% of their genes. For DZ twins, the A genetic correlation equals .5 because they share on average 50% of their genes, and the D genetic correlation is .25 because they inherit the same alleles (genes) at a locus from both parents 25% of the time. Thus, if genetic effects are additive in nature (A), MZ twins should be approximately twice as similar as DZ twins. If shared environment is significant, however, MZ twins are less than twice as similar as DZ twins because these influences act on both types of twins in similar ways. In contrast, if dominance is present, then MZ intraclass correlations would be more than half DZ intraclass correlations. Because C and D capitalize on the same variance (deviation from MZ similarity being 2 times DZ similarity), they cannot be tested in the same model.

Reduced models of the form AE, CE, or E can be compared to the full ACE or ADE model. D is not estimated independently from A because interactive genetic influences do not occur without additive influences for complex traits, and E can never be dropped because it also contains measurement error. A nonsignificant chi-square difference test and a negative Akaike's Information Criterion (AIC; Akaike, 1987) indicate more parsimonious fit of a reduced model over the full model. The statistical program Mx (Neale, Boker, Xie & Maes, 2003) was used to fit the models.

Bivariate twin models are a simple extension of the univariate model to two measured variables (e.g., effortful-attentional control and symptoms), and the purpose is to uncover the etiology of the covariances between the variables. The variances and covariance between two variables are parsed into latent constructs representing A, D and E (or A, C, and E although C was found to be nonsignificant in this study and was not estimated in the bivariate models). Figure 1 depicts a bivariate model, the Cholesky decomposition, illustrating the traits for both siblings and the correlations between sibling genetic and shared environmental latent factors. The three latent variables on the left represent the ACE influences common to both traits. The latent variables on the right reflect influences unique to variable 2. The paths from the common latent factors to variable 1 indicate genetic and environmental influences on the first variable. The common A, D, and E paths to variable 2 indicate the extent to which genetic and environmental influences on variable 1 also influence variable 2 (i.e., decomposing the covariance). The paths from the ADE unique

factors (on the right) to variable 2 represent genetic and environmental influences independent of the common genetic and environmental influences. Paths were systematically dropped to fit nested (reduced) models. The best model was indicated by the highest probability chi-square difference test and the lowest value for the AIC.

Results

Means, standard deviations, and ranges were first examined (see Table 1). Gender and age differences in parent report of Effortful Control were explored with repeated-measure ANOVAs. Parent-reported Effortful Control was significantly higher at T2 than at T1 for sample 1 only, $F(1, 136) = 9.70$, $p < .01$, $F(1, 136) = 1.71$, $p = .19$. Girls were significantly higher in parent-reported Effortful Control across both time points, $F(1, 136) = 7515.86$, $F(1, 136) = 5126.85$, $p<.01$. At time two (T2), girls were also higher in observer report Attentional Control, $t(375) = 2.51$, $t(375) = 2.32$, $p < .05$. Boys had significantly higher rates of parent report Externalizing, $t(561) = -3.36$, $t(562) = -4.45$, $p < .01$. Equality of variances across twin pairs for each variable was also examined, as one would expect equal variances for MZ and DZ twins. Unequal variances suggest possible sibling contrast effects, when the rater contrasts the twins while completing the measures (Saudino et al., 1995). There were no consistent differences in variances by sex or zygosity group across the two twin samples for any of the measures, so contrast effects were not considered further.

We examined the twin (intraclass) correlations separately by zygosity group and gender to determine the general pattern of genetic and environmental influences for the individual variables (see Table 1). For parent-reported Effortful Control at both ages, we see high MZ twin similarity that is more than twice DZ twin similarity, implicating a possible influence of D (see Method for description of A, C, D and E sources of variance). For observed Attentional Control and the parent report Internalizing and Externalizing composites, we see a pattern of MZ twin similarity twice that of DZ twins, implying A but no C. Interestingly, female DZ twins appear more similar than male DZ twins, which may indicate gender differences in heritability that can be tested with model fitting.

Based on the intraclass correlations, we decided to begin with the ACE full model (which allows for shared environmental influences), as well as the ADE full model (which distinguishes between additive and dominant genetic influences). We allowed the magnitude of the estimates to differ by gender, comparing this model to a constrained model that equated estimates for girls and boys. Estimates could be constrained to be equal for girls and boys for T1 parent-reported Effortful Control, observed Attentional Control, parent-reported Internalizing, and parent-reported Externalizing. However, separate estimates for girls and boys were needed for T2 parent-reported Effortful Control (ACE: $\chi^2_\Delta(3) = 16.16$, $p = 0.001$, AIC = 10.16; ADE: $\chi^2_\Delta(3) = 12.04$, $p = 0.007$, AIC = 6.04), with the constrained model fitting significantly worse than the model that allowed estimates to vary by gender.

Beginning with the best fitting full model, we systematically dropped parameters to test the significance of each influence (A, C, and D; E is always included because it includes measurement error) on trait variation. Table 2 summarizes the fit statistics for each model, as well as the estimates of genetic, shared environmental, and nonshared environmental

influences for the full models and the (bolded) best fitting reduced model. For T1 and T2 parent-reported Effortful Control, the best fitting model was the ADE, with 68–79% of the variance due to broad sense heritability (additive and dominant genetic influences combined), 0% to the shared environment, and 21–32% to the nonshared environment. For observed Attentional Control, the AE reduced model was the best fitting model, with 83% of the variance due to genetic influences (17% nonshared environment). The AE reduced model was also the best fitting model for the Internalizing and Externalizing composites, with remarkably similar estimates of genetic influence (69% and 68%, respectively; 31% and 32% nonshared environment, respectively). Thus, considered individually, genetic influences accounted for the majority of the variance in all traits, with no significant effects of the shared environment.

The phenotypic correlations among the variables were examined next to determine the extent to which parent-reported Effortful and observed Attentional Control were related to the Internalizing and Externalizing composites. These correlations are given in Table 3 separately for girls (above the diagonal) and boys (below the diagonal). All correlations were in the expected direction, with high stability (T1 and T2 parent-reported Effortful Control). Observed Attentional Control was moderately positively correlated with parent-reported Effortful Control. Parent report of Effortful Control at both time points was significantly negatively correlated with the Internalizing and Externalizing composites (with the exception of T2 parent-reported Effortful Control and Internalizing for girls). Observed Attentional Control was negatively correlated with the Internalizing and Externalizing composites for girls, but not boys.

There were a total of six bivariate genetic models that were estimated to decompose the covariance between the traits into genetic and environmental components, one representing each pairing of Effortful/Attentional Control with Internalizing or Externalizing.

First, we considered the longitudinal association between T1 parent-reported Effortful Control and T2 Internalizing, Figure 2 illustrates estimates derived from the full ADE-ADE model [−2LL (1111) = 809.42], as well as the best fitting reduced model, the AE-AE [−2LL (1115) = 809.94; $\chi^2_\Delta(4) = 0.52$, p = 0.97, AIC = −7.49]. D was not a significant influence on either variable, and the covariation between the traits was due to a shared additive genetic influence. Very similar results were found for the model that considered the longitudinal association between T1 parent-reported Effortful Control and T2 Externalizing; see bottom of Figure 2. The full ADE-ADE model yielded a fit of −2LL (1111) = 957.70, with the final reduced model dropping the influence of D on both traits, and indicating that about half of the genetic influence on Externalizing was shared with earlier parent-reported Effortful Control [−2LL (1115) = 967.23; $\chi^2_\Delta(4) = 9.53$, p = 0.05, AIC = 1.53].

Next, we fit a model to represent the association between concurrent T2 parent-reported Effortful Control and Internalizing, the fit of the full ADE-ADE model was −2LL (1897) = 1216.34, with different estimates needed for girls and boys (the constrained model yielded −2LL (1906) = 1281.67, $\chi^2_\Delta(9) = 65.33$, $p = 0.00$, AIC = 47.33), see Figure 3. Similar to the longitudinal results, the best fitting model included additive genetic and nonshared environmental influences on both traits, with additive genetic influences accounting for the

covariation, −2LL (1905) = 1230.58; $\chi^2_\Delta(8) = 14.23$, $p = 0.08$, AIC = −1.77. From the standardized squared path coefficients given separately for girls and boys in Figure 3, we see that a larger proportion of the genetic influence on Internalizing was shared with parent-reported Effortful Control for boys than girls. For concurrent parent-reported Effortful Control and Externalizing, the full model also required separate estimates for girls and boys [−2LL(1897) = 1370.45, with the constrained model yielding −2LL (1906) = 1447.93, $\chi^2_\Delta(9) = 77.49$, $p = 0.00$, AIC = 59.49], the final reduced model did not include the influence of D on either trait, and covariation was due to both additive genetic and nonshared environmental effects [−2LL (1903) = 1371.68; $\chi^2_\Delta(6) = 1.24$, $p = 0.98$, AIC = −10.77], see bottom of Figure 3. Similar to the results with Internalizing, we see that more of the genetic influence on Externalizing was shared with parent-reported Effortful Control for boys than girls.

Moving to observer report of attentional control, we started with a model decomposing the relation between observed Attentional Control and Internalizing (see Figure 4). Comparing the full ADE-ADE model where estimates were allowed to vary by sex to a full model where estimates were constrained to be equal indicated that separate estimates for girls and boys were necessary, the fit of this full model was −2LL (1851) = 755.83, with the constrained model yielding −2LL (1860) = 796.26, $\chi^2_\Delta(9) = 40.43$, $p = 0.00$, AIC = 22.43. Although we tap attentional aspects of control by observer report rather than parent report of effortful control, we found that the best fitting reduced model was comparable, with additive genetic and nonshared environmental influences important for both traits, and additive genetic influences accounting for the covariation between them. The fit of the reduced model was −2LL (1859) = 760.53, $\chi^2_\Delta(8) = 4.70$, $p = 0.79$, AIC = −11.30. Similarly, estimates varied by sex for the observed Attentional Control—Externalizing model, with a fit of −2LL (1851) = 1018.43, with the constrained model yielding −2LL (1860) = 1077.53, $\chi^2_\Delta(9) = 59.10$, $p = 0.00$, AIC = 41.10. The best fitting model was the AE-AE model with additive genetic influences accounting for the covariation between traits [−2LL (1859) = 1022.91; $\chi^2_\Delta(8) = 4.49$, $p = 0.81$, AIC = −11.51].

Discussion

The purpose of this study was to consider the genetic and environmental architecture of multiple measures of effortful and attentional control (mother, father and observer report), symptoms of internalizing and externalizing psychopathology (mother and father report), and the association between these traits with a sample of twins in middle childhood.

Placing Results in Context

Our twin study results confirm a strong genetic component to parent-reported effortful and observed attentional control. Combined mother and father report on CBQ Effortful Control at two ages suggested broad sense heritability estimates ranging from 68–79%, with a higher heritability estimate of 83% obtained for observed Attentional Control and no influence of the shared environment on either trait. Moderate to high heritability affirms previous twin study results (see Introduction) and is consistent with molecular genetic studies. For example, the long repeat form of the dopamine D4 receptor gene and variants of the

monoamine oxidase A gene associated with lower enzyme expression (which synthesizes dopamine) have been associated with both activation in the anterior cingulate gyrus (Fan, Fossella, Sommer, Wu & Posner, 2003) and behavioral measures of executive attention (Fossella, Posner, Fan, Swanson & Pfaff, 2002).

Additive genetic influences (69% and 68%, respectively) accounted for the majority of the variance in internalizing and externalizing in our sample of eight-year-old twins, with the remainder of the variance due to nonshared environment. In a recent review, Lemery and Doelger (2005) reported that shared environmental influences were significant (with a range of .25–.49) for depression in twin samples of young children, but samples including older children (7–12 years) showed much lower or no shared environmental influences. The strength of genetic influences and the presence of shared environmental influences were less clear when considering child anxiety, with a wide range of reported genetic influences (0–72%) and some studies finding significant shared environmental influences. For externalizing, the review concluded that genetic influences were significant (ranging from . 13–.71), with a significant shared environmental influence most often reported when using the Child Behavior Checklist (Achenbach, 1991), but not when using structured clinical interviews (Lemery & Doelger, 2005). This study was the first to report genetic and environmental influences utilizing the HBQ, which is a relatively new measure that corresponds with DISC-IV diagnoses (Lemery-Chalfant et al., in press).

Replicating previous work, significant negative correlations between measures of effortful/attentional control and internalizing and externalizing symptoms were found, longitudinally, concurrently and across reporters (Table 4). Correlations with Internalizing were smaller than correlations with Externalizing, again replicating previous work. Parent-reported Effortful Control may be less important for Internalizing, or alternatively, reporters such as parents and observers could be ill equipped to report reliably these symptoms because they are not usually observable. In fact, mothers underreport internalizing in comparison to psychiatric diagnosis (Clarke-Stewart et al., 2003). It has been suggested that children who are old enough to self report may be better than parents at reporting their own internalizing (Barrett et al., 1991; Ialong, Edelsohn, Werthamer-Larsson, Crockett, & Kellam, 1993; Renouf & Kovacs, 1994; Routh, 1990; Weissman, Wickramaratne, & Warner, 1987). Externalizing, on the other hand, includes easily detectable acting out and disruptive behaviors that lend themselves to being reported by parents and teachers (Edelbrock, Costello, Dulcan, Conover, & Kala, 1986; Loeber, Green, Lahey, & Stouthmaer-Loeber, 1989; Russo, Loeber, Lahey, & Keenan, 1994). Future studies should consider that internalizing might be better reported by the child, and externalizing by parents or other caregivers.

A measurement explanation for why temperament is correlated with internalizing and externalizing is not often considered. Measures of temperament may tap symptoms of psychopathology, and measures of symptoms may tap temperament. However, studies that dealt with measurement confounding by removing overlapping items and creating purified scales have found that reported relationships between temperament and internalizing and externalizing remain with purified scales (Lemery et al., 2002; Lengua, West, & Sandler, 1998; Oldehinkel et al., 2004).

We parsed the phenotypic associations into genetic and environmental components in a series of bivariate genetic models. We saw a strikingly similar pattern for all six bivariate models tested (Figures 2–4). Both longitudinally and concurrently with parent report and observer measures of effortful/attentional control, shared additive genetic influence accounted for the covariation between the temperamental control variables and internalizing or externalizing symptoms of psychopathology. Although no other studies in the literature have considered the genetic association between effortful/attentional control and symptoms of psychopathology, the few behavior genetic studies that have considered the relationship between reactive dimensions of temperament and symptoms have largely reported genetic mediation (see Introduction). However, genetic mediation is not necessarily the rule, with the environment being important for associations between temperament and other domains of development. For example, Volbrecht, Lemery-Chalfant, Aksan, Zahn-Waxler, and Goldsmith (in press) reported that shared environment accounted for the association between lab and questionnaire measures of positive affect and empathy-related helping behaviors during the second year of life.

The finding that shared genes account for the association between measures of control and symptoms of psychopathology is important in that it encourages 1) identification of these common genes; 2) isolating environmental triggers that are unique to psychopathology; and 3) testing of mediational and moderational models that take into account the genetic association. Revisiting Rothbart and Bates (2006) putative models representing the association between positive aspects of temperament and psychopathology from the Introduction, results are consistent with the *Protective model*, in which a person has a predisposition (i.e., high effortful/attentional control) to deal effectively with stressful environments, and that this predisposition thus moderates the influence of stress on symptoms. The results do not prove moderation, but allow for the possibility. The results discourage the pursuit of mediational models suggesting that effortful control is linked to symptoms through some aspect of the environment (i.e., because effortful control was linked to both internalizing and externalizing symptoms through shared genetic influences).

Sex Differences in the Magnitude of Genetic and Environmental Influences

The univariate results indicated that the magnitude of genetic and environmental influences significantly differed for girls and boys for T2 parent-reported Effortful Control. Specifically, both girls and boys had strong genetic influences, with both additive and nonadditive/dominant influences important for girls, and only nonadditive genetic influence important for boys (Table 2). The classic twins-reared-together design has low power for distinguishing between these two types of genetic influence (Rietveld, Posthuma, Dolan & Boomsma, 2003), so in general, the broad sense heritability (i.e., genetic influences combined) of 79% for girls and 71% for boys is emphasized. T1 and T2 parent-reported effortful control were highly correlated (Table 3), and the intraclass correlations revealed similar patterns of twin similarity (Table 1). However, estimates for girls and boys at T1 were not significantly different, perhaps because of lower power at T1, with only a sub-sample participating in the earlier assessment.

More sex differences emerged with the bivariate models. There was a smaller genetic influence for boys on T2 parent-reported Effortful Control, but a larger proportion of boys' genetic influence than girls' on Internalizing and Externalizing was shared with T2 parent-reported Effortful Control (Figure 3). Boys also had less of a genetic influence than girls on observed Attentional Control, Internalizing, and Externalizing in the models decomposing these associations (Figure 4). A few other studies have found differences in genetic and environmental influences by sex. However, the two reported twin studies of effortful control in the literature did not have the sample sizes necessary for testing sex differences; Goldsmith et al. (1997) regressed out any effects of sex on parameter estimates, and Yamagata et al. (2005) combined males and females and did not consider sex.

The literature on internalizing and externalizing symptoms in children is much larger. The majority of studies of internalizing and externalizing in children did not find significant sex differences in the magnitudes of genetic and environmental influence (Bartels et al., 2003; Gjone & Stevenson, 1997b; Hudziak et al., 2003; Jaffee, Moffitt, Caspi, Taylor & Arseneault, 2002; McGue, Iacono, Legrand & Elkins, 2001; Van Den Oord, Boomsma, & Verhulst, 2000; van der Valk, van den Oord, Verhulst & Boomsma, 2003). However, Kuo, Lin, Yang, Soong and Chen (2004) found larger genetic influence on externalizing for girls than boys, and Derks, Hudziak, van Beijsterveldt, Dolan and Boomsma (2004) reported larger genetic influence on aggression in girls, with Eley, Lichtenstein, and Stevenson, (1999) finding stronger genetic influence on nonaggressive antisocial behaviors for girls. In contrast, Silberg et al. (1994) found larger genetic influence on externalizing in boys, and Nadder, Silberg, Eaves, and Maes (1998) reported larger genetic influences on oppositional defiant and conduct disorder symptoms in boys using a parent telephone survey. For internalizing, fewer sex differences have been reported. Eley and Stevenson (1999) found larger genetic influences on self-report of depression in boys, and Derks et al. (2004) reported larger genetic influence on withdrawn behaviors in boys. In contrast, Scourfield et al. (2003) reported larger genetic influences on depression for girls with parent report but not self-report measures.

In conclusion, our study was the first to consider sex differences in the magnitude of genetic and environmental effects for effortful and attentional control. Some studies have reported significant sex differences in estimates of heritability for internalizing and externalizing, but the results are sometimes contradictory. As a generalization, the ranges of the parameter estimates by sex for these studies were very similar, suggesting that significant sex differences are relatively small in magnitude (see Lemery & Doelger, 2005, for a review). Thus, future studies of sex differences will need large sample sizes to resolve the outstanding issues.

Consistency in Findings Across Reporters

The findings for strong genetic and no shared environment components underlying the variance in effortful/attentional control were consistent across parental and observer report, despite the differences in measures and contexts. Parent report was a composite of scales measuring frequency of attentional focusing and inhibitory control across a variety of situations over the past six months. Observer report was exclusively a measure of attentional

control including adaptation to change, attention to tasks, and persistence items averaged across a single, four-hour home visit. Thus, parents and observers were reporting from different contexts. Further, parents usually have prolonged exposure to a limited comparison group of children, while observers are exposed to a large comparison group of other children. In contrast to our findings, previous studies with different but related variables have reported greater genetic variance for parent report and greater shared environmental variance for observer report (Deater-Deckard, 2000; Ghodsian-Carpey & Baker, 1987; Leve, Winebarger, Fagot, Reid, & Goldsmith, 1998; Miles & Carey, 1997; Plomin & Foch, 1980).

Limitations

One issue inherent in twin studies is that of generalizability–whether the experiences of twins can be generalized to singletons. Angold, Erkanli, Silberg, Eaves, and Costello (2002) found no differences in depression scores for twins and single born children between the ages of 8 and 17, while Moilanen et al. (1999) found nonsignificant trends suggesting lower rates of emotional and behavioral problems in twins. Studies with larger, population-based samples usually find no differences between twins and singletons in internalizing and externalizing (Lytton & Gallagher, 2002). Thus, most literature supports the generalizability of twin data to singletons.

Another potential limitation of twin studies is violation of the equal environments assumption (EEA), or the assumption that the environment influencing the behavior being studied is no more similar for MZ twins than DZ twins (Neale & Maes, 2004). If the EEA is violated, the interpretation of twin data is confounded. In response to skepticism, the EEA has been tested with large samples of twins. Borkenau, Riemann, Angleitner, and Spinath (2002) measured twin similarity of childhood experience through self-report and found that these measures were unrelated to similarities in personality, which supports the EEA. Cronk et al. (2002) tested the EEA for mother reports of child and adolescent mood and behavioral disorders. They compared models controlling for environmental similarity in twin pairs to models that did not control for environmental similarity. Controlling for environmental similarity did not greatly affect the best fitting models, which again supports the EEA. Thus, combined with earlier approaches to testing the EEA (Loehlin & Nichols, 1976), the recent literature supports validity of the EEA.

Conclusion

The regulatory temperament dimension of effortful control has consistently been identified as one pathway that protects children from psychopathology (Rothbart & Bates, 2006). In this study, we document high heritability of mother and father report of CBQ parent-reported Effortful Control and observer report Attentional Control in middle childhood. We have demonstrated, for the first time, that genetic influences accounted for the covariation between measures of control and mother and father report of internalizing and externalizing symptoms, both longitudinally and concurrently. These results encourage a search for genes that influence both temperament and psychopathology, and are consistent with effortful and attentional control providing trait resilience, or as Rothbart and Bates' (2006) suggested with their protective model, a predisposition to effectively dealing with stressful environments.

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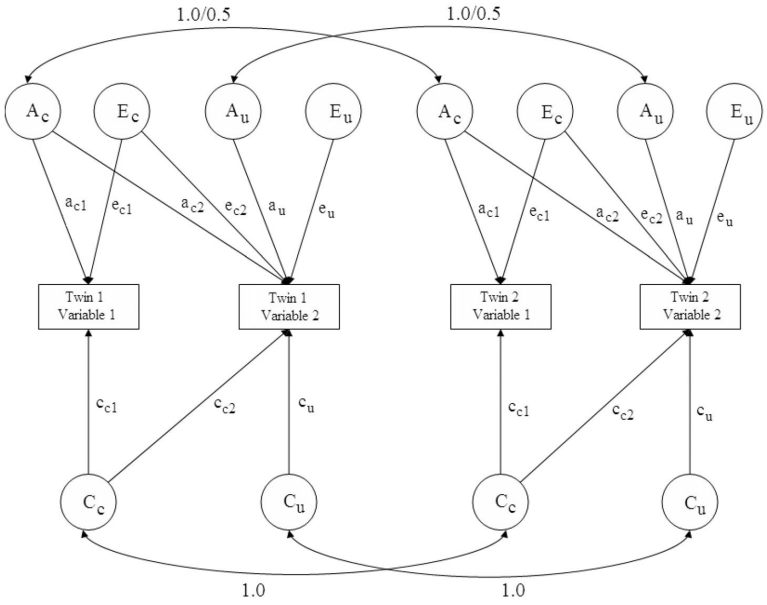
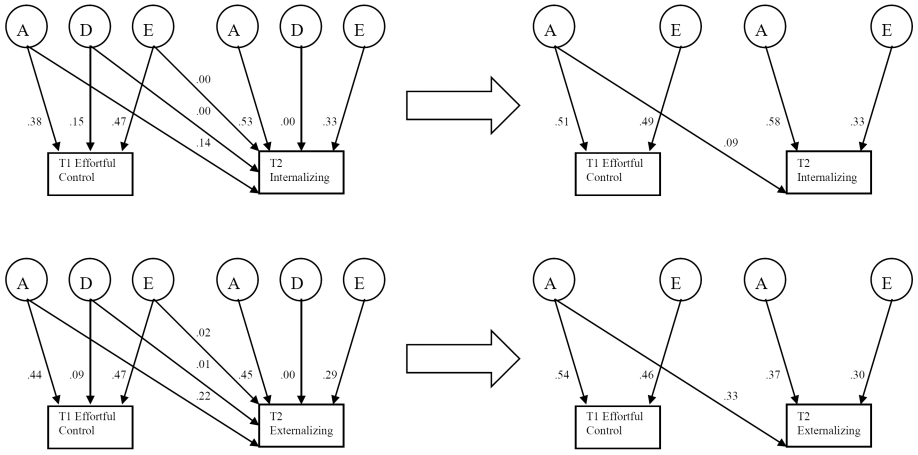


Figure 1.

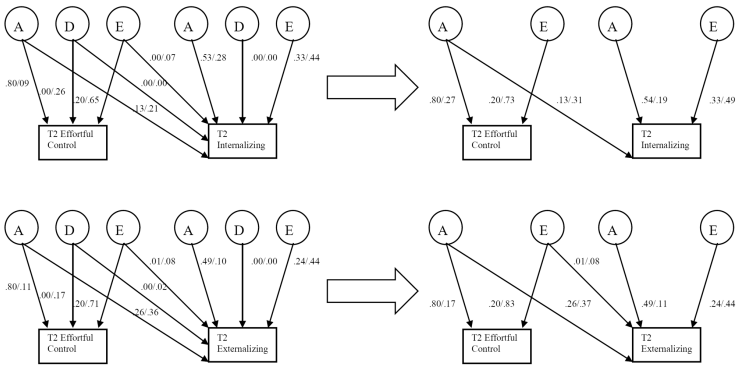
Example bivariate genetic model that decomposes the variance/covariance into additive genetic (A_c, A_u), shared environmental (C_c, C_u), and nonshared environmental (E_c, E_u) latent factors. All path estimates are freely estimated, but set equal across twin 1 and twin 2 (arbitrarily assigned), and also MZ and DZ twin pairs. The correlations between the A_c latent factors and the A_u latent factors are set to 1.0 for MZ and 0.5 for DZ twin pairs, the correlation between both the C_c and C_u factors are set to 1.0 for both MZ and DZ pairs. A_c, C_c, and E_c are factors common to both measured variables, whereas A_u, C_u, and E_u are unique factors that represent residual variance in variable 2 not accounted for by the common factors.



Note. A = additive genetic; D = nonadditive "dominant" genetic; E = nonshared environment influence; T1 = time one; T2 = time two. Numbers are squared standardized parameter estimates, representing the variance accounted for in the measured variable by the latent factor. Because the estimates are the same for twin 1 and twin 2, only estimates for twin 1 are displayed. Models on the left hand side are full models, whereas models on the right hand side are the final, reduced models.

Figure 2.

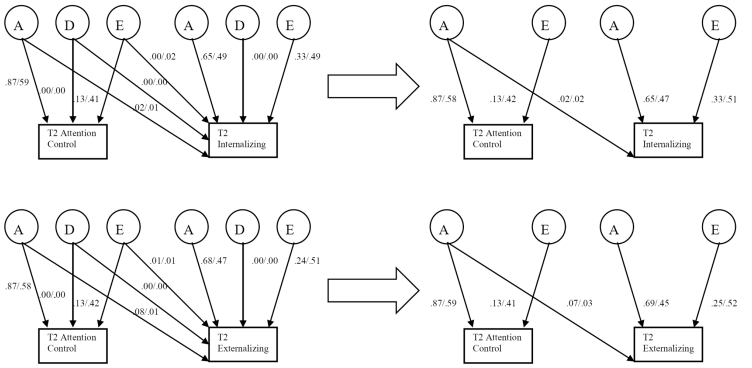
Longitudinal Models Decomposing the Relation between Effortful Control and the Internalizing or Externalizing Composite.



Note. A = additive genetic; D = nonadditive "dominant" genetic; E = nonshared environment influence; T1 = time one; T2 = time two. Numbers are squared standardized parameter estimates, representing the variance accounted for in the measured variable by the latent factor, with estimates given for girls first, then boys. Because the estimates are the same for twin 1 and twin 2, only estimates for twin 1 are displayed. Models on the left hand side are full models, whereas models on the right hand side are the final, reduced models.

Figure 3.

Concurrent Models Decomposing the Relation between Effortful Control and the Internalizing or Externalizing Composite.



Note. A = additive genetic; D = nonadditive "dominant" genetic; E = nonshared environment influence; T1 = time one; T2 = time two. Numbers are squared standardized parameter estimates, representing the variance accounted for in the measured variable by the latent factor, with estimates given for girls first, then boys. Because the estimates are the same for twin 1 and twin 2, only estimates for twin 1 are displayed. Models on the left hand side are full models, whereas models on the right hand side are the final, reduced models.

Figure 4.

Decomposing the Relation Between Observed Attentional Control and the Internalizing or Externalizing Composite.

Table 1

Means, Standard Deviations, Ranges, and Twin Intraclass Correlations.

	Pooled Sample		Females	Males	Female	Female	Male	Male	Opp-Sex
	M (SD)	Range	M (SD)	M (SD)	MZ	DZ	MZ	DZ	DZ
T1 P report Effortful Control	4.55 (.68)	1.83–6.32	4.70 (.67)	4.42 (.66)	.65	.27	.71	−.11	.04
T2 P report Effortful Control	4.62 (.77)	1.86–6.55	4.81 (.70)	4.43 (.79)	.75	.32	.67	.03	.04
T2 observed Attentional Control	4.02 (.60)	1.33–5.00	4.10 (.56)	3.94 (.62)	.82	.50	.82	.44	.52
T2 P report Internalizing	0.30 (.22)	0.00–1.53	0.30 (.22)	0.29 (.22)	.61	.45	.70	.33	.37
T2 P report Externalizing	0.30 (.25)	0.00–1.50	0.26 (.23)	0.35 (.26)	.60	.45	.69	.23	.35

Note: Means and standard deviations were computed separately for the Twin 1 and Twin 2 subsamples, averages are reported. T1 = time one; T2 = time two; MZ = monozygotic (identical) twins; DZ = dizygotic (fraternal) twins; P = parent report, which is a composite of mother and father report; Observed is a composite of observer 1 and observer 2 ratings.

Table 2

Model Fit and Estimates of Genetic, Shared Environment, and Nonshared Environment Contributions to Effortful Control, Attentional Control, Internalizing and Externalizing Composites.

	Model	−2LL	df	Δ χ^2	Δdf	p	AIC	h^2	d^2	c^2	e^2
T1 parent report Effortful Control	ACE	1078.17	557					.63		.00	.37
	ADE	**1068.49**	**557**					**.00**	**.68**		**.32**
	AE			9.69	1	0.00	7.69				
	CE			27.81	1	0.00	25.81				
	E			60.69	2	0.00	56.69				
T2 parent report Effortful Control	ACE	1669.16	788					.68[a]		.10[a]	.22[a]
								.60[b]		.05[b]	.36[b]
	ADE	**1657.29**	**788**					**.41** [a]	**.38** [a]		**.21** [a]
								.00 [b]	**.71** [b]		**.29** [b]
	AE			14.14	2	0.00	10.14				
	CE			48.22	2	0.00	44.22				
	E			110.91	4	0.00	102.91				
T2 observed Attentional Control	ACE	1114.35	745					.63		.19	.18
	ADE	1118.08	745					.83	.00		.17
	AE			**3.73**	**1**	**0.05**	**1.73**	**.83**	**.00**		**.17**
	CE			41.65	1	0.00	39.65				
	E			228.12	2	0.00	224.12				
T2 parent report Internalizing Composite	ACE	−399.26	1118					.65		.04	.31
	ADE	−399.05	1118					.69	.00		.31
	AE			**0.20**	**1**	**0.65**	**−1.80**	**.69**			**.31**
	CE			33.14	1	0.00	31.14				
	E			175.94	2	0.00	171.94				
T2 parent report Externalizing Composite	ACE	−127.95	1118					.68		.00	.32
	ADE	−128.00	1118					.64	.04		.31

Model	−2LL	df	Δ χ^2	Δdf	p	AIC	h^2	d^2	c^2	e^2
AE			**0.05**	**1**	**0.82**	**−1.95**	**.68**			**.32**
CE			37.27	1	0.00	35.27				
E			166.13	2	0.00	162.13				

Note: T1 = time one; T2 = time two; Parent report is a composite of mother and father report; Observed is a composite of observer 1 and observer 2 ratings; −2LL = the fit statistic −2 times the log likelihood; df = degrees of freedom; Δχ = change in chi-squared from the best fitting full model to reduced model; p = probability; AIC = the fit index Akaike's Information Criterion; A = additive genetic; C = shared environmental; D = nonadditive/dominant genetic; E = nonshared environmental influences. The parameter estimates in the rightmost columns are given for the full ACE and ADE models, plus the best fitting reduced model (which is also bolded).

[a] parameter estimate for girls;

[b] parameter estimate for boys.

Table 3

Correlations Among Effortful Control, Attentional Control, and Internalizing and Externalizing Composites.

Variable	1	2	3	4	5
1. T1 parent report Effortful Control	-	.68**	.31*	−.20*	−.43**
2. T2 parent report Effortful Control	.69**	-	.22**	−.23	−.46**
3. T2 observed Attentional Control	.21	.28**	-	−.31*	−.38**
4. T2 parent report Internalizing Composite	−.24**	−.35**	−.08	-	.36**
5. T2 parent report Externalizing Composite	−.38**	−.39**	−.24	.40**	-

Note.

Correlations for girls are displayed above the diagonal and correlations for boys are displayed below the diagonal. Correlations were averaged between the Twin 1 and Twin 2 subsamples via Fisher's z transformation. An average N of the two subsamples was used to determine significance. T1 = time one; T2 = time two; Parent report is a composite of mother and father report; Observed is a composite of observer 1 and observer 2 ratings.

* $p < .05$.

** $p < .01$.