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Approach/Positive Anticipation, Frustration/Anger, and Overt Aggression in Childhood

K. Deater-Deckard, C. Beekman, Z. Wang, J. Kim, S. A. Petrill, L. A. Thompson, and L. S. DeThorne

Abstract

We examined mothers' ratings of children's affective and behavioral aspects of approach tendencies and links with overt aggressive behavior problems, while considering the genetic etiology of these processes. Approach/positive anticipation (AP), frustration/anger (FA), and overt aggression in 4–9 year olds were assessed using mothers' reports in a diverse national sample ($n = 992$) and a sample of same-sex twins ($n = 195$ pairs). AP and FA were positively correlated with each other and with overt aggression (r from .2 to .5), and these associations were very similar for boys and girls. AP and FA provided overlapping as well as independent statistical prediction of aggression. AP statistical prediction of aggression was substantially mediated by FA, an effect that was accounted for by underlying genetic and nonshared environmental influences.

Keywords

Temperament; aggression; genetics

Decades of research on the etiology of individual differences in overt aggressive behavior (i.e., physical or verbal hostility that serves to harm another) have pointed to the importance of temperament and the interplay of biological and environmental factors (Dodge, Coie, & Lynam, 2006). The literature implicates genetic and environmental influences, and negative affectivity—frustration/anger in particular—in explaining why some children tend to show more overt aggression than others. By comparison, less is known about how appetitive approach tendencies work in conjunction with frustration/anger in the etiology of overt aggression in childhood, and how genetic and environmental influences operate in these processes. The goal of the current study was to investigate the links between approach/positive anticipation, frustration/anger, and overt aggressive behavior in the transition to middle childhood, with an emphasis on the identification of overlapping and independent genetic and environmental influences.

Behavioral Approach and Aggression

Individual differences in overt aggressive behavior emerge in early childhood, and these differences between children become moderately to substantially stable by the time they start school (Olweus, 1979). Aggression is a major component of externalizing behavior problems (Achenbach, 1991) and is strongly associated with problems in social relationships and functioning at home and at school (Dodge et al., 2006). The emergence and persistence of high levels of overt aggressive behavior in early childhood is one of the strongest and most consistent predictors of antisocial behavioral disorders for boys and girls alike, with

current theory emphasizing gene-environment interplay operating through transactions between social learning and temperament factors. Decades of theory and research have established a body of evidence suggesting that children who show high levels of dispositional frustration/anger are also more overtly aggressive—a link that appears in early childhood and persists into adulthood. In addition, frustration/anger and overt aggression both are moderately to substantially heritable in childhood, and also include modest to moderate levels of nonshared (i.e., that which differentiates siblings) environmental variance (Goldsmith, Buss, & Lemery, 1997; Oniszczenko et al., 2003; Rhee & Waldman, 2002).

The link between frustration/anger (FA) and overt aggression may involve reactive negative affect—children who become frustrated and angry are more likely to lash out at others. However, FA also is an essential aspect of a behavioral approach motivational system that is sensitive to potential rewards in the environment and includes positive affect including interest and excitement (Pickering & Gray, 2001). Although there is less behavioral genetic research on approach/positive anticipation (AP) compared to negative affect and aggression, the work that has been done points to moderate heritability in childhood (Eid, Riemann, Angleitner, & Borkenau, 2003; Goldsmith et al., 1997). Molecular genetics research indicates a potential role for dopamine receptor genes in the etiology of reward or sensation seeking behavior (Auerbach, Faroy, Ebstein, Kahana, & Levine, 2001; Ebstein et al., 1998; Pickering & Gray, 2001).

One way to think about the relationship between FA and AP is to view them as negatively and positively valenced affective or arousal components of a broader behavioral activation motivational system (Pickering & Gray, 2001). Studies of temperament in early childhood provide some evidence to support this view (for a review, see Derryberry & Rothbart, 2001). FA and AP organize as separate negative and positive affective dimensions of temperament in the first year, becoming fairly stable by end of the second year. By early childhood, FA and AP are positively correlated with each other, and both are correlated with and predict (from early to middle childhood) overt aggressive behavior. Derryberry and Rothbart suggest that as components of a reward-sensitive behavioral approach system, FA and AP may serve to facilitate each other, and together facilitate the expression of overt aggressive behavior. Accordingly, children who are higher in AP tendencies also are more likely to become frustrated and angry when potential rewards are not obtained. The perceived impediments to a potential reward (be they objects or people) often become the targets of overt aggressive behavior. Accordingly, AP may represent appetitive arousal that motivates reward-seeking behaviors, and FA may represent negative affect that motivates actions to reduce or remove impediments to obtaining those rewards if and when obstructions arise.

Thus, there is theoretical emphasis and some empirical evidence implicating FA and AP in the etiology of overt aggression. However, there are a number of important questions that remain unanswered. The prior research has relied on non-representative volunteer samples, and has not examined potential sex differences in these links. In addition, we do not know from the literature whether the AP-aggression and FA-aggression links are independent or overlap with each other—and to the extent that there is overlap, whether FA statistically mediates the effect of AP on aggression or AP mediates the effect of FA on aggression. Furthermore, if FA and AP reflect distinct but inter-related components of an approach motivational system, there should be evidence of overlapping genetic influences underlying their “phenotypic” links with each other and with overt aggression, because these observed links are thought to reflect underlying neurological systems in which genetic factors figure prominently. At the same time, there should be some statistical independence in these links, since FA and AP are etiologically and functionally distinct. Although there is established evidence that these two temperament dimensions are heritable, to our knowledge there has not been a test of genetic overlap between them, or in their links with aggression.

Using two samples of 4–9 year olds, we tested three hypotheses to address these gaps in knowledge. First, we predicted that FA and AP would be moderately positively correlated with each other and with overt aggression, and that this effect would replicate in two large samples of 4–9 year olds. We also explored whether these associations differed for boys and girls—given that boys are more likely to show higher levels of overt aggressive behavior than girls (Crick & Grotpeter, 1995). Second, we expected FA and AP to show some overlapping as well as some independent statistical prediction of overt aggressive behavior—reflecting distinct yet interrelated components of a behavioral approach motivation system. We also conducted exploratory tests of statistical mediation, by comparing FA as a mediator (AP → FA → aggression) to AP as a mediator (FA → AP → aggression). Third and finally, the overlapping and independent prediction of overt aggression at the phenotypic level of analysis also would be evident at the genetic level of analysis, with significant overlapping and independent genetic covariance between FA, AP, and overt aggression.

Methods

Sample

Participants were from two independent research projects—the Western Reserve Reading Project (WRRP, an on-going longitudinal twin study; Petrill, Deater-Deckard, Thompson, DeThorne, & Schatschneider, 2006), and the Study of Early Child Care and Youth Development (SECCYD, a portion of which is a public longitudinal dataset; NICHD Early Child Care Research Network, 2004). We examined data from the first and second annual assessments in the WRRP, and from the 54-month and 1st grade assessment in the SECCYD. Parents and children completed an informed consent/assent procedure and received honoraria. Procedures were conducted in compliance with our universities' Institution Review Boards, and APA ethical standards.

WRRP—The data from the WRRP included 195 twin pairs for whom we had complete data for this study. All were same-sex pairs (61% female; 44% genetically identical). Cheek swabs were taken to determine zygosity using DNA analysis. The majority (~90%) of the parents consented to the genotyping. However, for the minority of cases where parents did not consent to genotyping, zygosity was determined using a highly reliable parent questionnaire on twins' physical similarity (Goldsmith, 1991). For some analyses, the sample of twins was arbitrarily divided into two sub-samples (referred to as “twin 1” and “twin 2”) so that no two co-twins were in the same sub-sample; this was done as a test for internal replication of effect sizes and to address the non-independence of the sibling data. Parental education levels varied markedly and were very similar for mothers and fathers on average, with 1–2% high school or less, 39% some college, 30% bachelor's degree, 25% some post-graduate education or a degree, and 5% not specified. Most were Caucasian (92%) and lived in two-parent households (6% single mothers). The children were 6.1 years old on average ($SD = .88$ years, from 4.3 to 7.9 years) at the time of the first assessment.

SECCYD—We used data from the public datasets of the NICHD (National Institute of Child Health and Development) Study of Early Child Care and Youth Development or SECCYD (<http://www.nichd.nih.gov/research/supported/seccyd/datasets.cfm>). Data collection began in 1991 in 9 states (Arkansas, California, Kansas, New Hampshire, North Carolina, Pennsylvania, Virginia, Washington, and Wisconsin) and included 1,364 children (51.7% male) and their families when the children were one month of age. The sample included participants from five racial categories: white (80.4%), black (12.9%), other (4.7%), Asian (1.6%), and American Indian, Inuit, or Aleutian (0.4%). Parent education and socioeconomic levels were very widely distributed although the sample was somewhat more

affluent on average compared to the US population. We analyzed data for 992 children (each child from a different family) for whom there was complete data, using data from the 54-month and 1st-grade questionnaires.

Procedures

In the WRRP, during two annual home visits, mothers (and some fathers) completed a questionnaire regarding their children's behavior problems. Before or after the second home visit, parents were invited to complete a mailed questionnaire regarding each child's temperament. In the SECCYD, mothers completed questionnaires regarding their children's temperament and behavior problems at 54 months of age during a visit to the laboratory. Mothers completed the behavior problems questionnaire again during a visit to the laboratory when their children were in first grade.

Measures

Frustration/anger and approach/positive anticipation—In the WRRP, mothers completed the Child Behavior Questionnaire-Short Form (CBQ-SF; Putnam & Rothbart, 2006) for each twin separately. The instrument includes 36 items rated on a 7-point Likert-type scale (1 = extremely untrue of your child to 7 = extremely true of your child) that yields 15 subscales ($\alpha > .6$). We examined two of these subscales—frustration/anger (FA) and approach/positive anticipation (AP). We examined the same subscales in the SECCYD, although in that study the mothers completed the full-length CBQ.

Aggression—In both studies, we used the mother-rated Aggression scale score from the Child Behavior Checklist (CBCL; Achenbach, 1991), which includes 20 items rated on a 0–2 point Likert-type scale. The CBCL Aggression score is a widely used and validated measure of child overt aggressive behavior. Because these scores were so stable over time, in an effort to yield the most reliable score possible we averaged two assessments of mothers' ratings (in SECCYD, $r = .70$ between 54-month and 1st-grade scores; in WRRP, $r = .72$ between first and second annual wave scores).

Results

Descriptive Statistics

Descriptive statistics are shown in Table 1. Aggression was widely distributed in both samples, but negatively skewed to the low end of the scale. However, the distributions and means for both samples were consistent with the literature regarding community samples of school-age children (Achenbach, 1991). The aggression scores were very similarly distributed in the two twin sub-samples. The children in the WRRP had slightly lower aggression scores (about one-third SD difference) and slightly less variability in scores compared to children in the SECCYD—perhaps because the SECCYD children were two years younger than the WRRP children on average.

FA and AP also varied widely and were similarly distributed in both samples, as well as in both twin sub-samples within the WRRP. The mean for FA was close to the middle (4) of the 7-point scale on the CBQ, with a standard deviation of about one point. By comparison, the AP scores were more heavily skewed to the higher end of the 7-point scale. The mean was just above 5 on the scale, and the standard deviation was two-thirds to three-quarters of one point.

There was little evidence to suggest gender differences in average levels of FA, AP, or aggression. Pearson correlations were estimated between the study variables and child sex, coded as female = 1 and male = 2. We estimated correlations because doing so provides a

direct estimate of effect size while yielding a t -test for $r > 0$ that is the same as a t -test of group means. In the SECCYD, these correlations were $-.05$ (n.s.) with aggression, $-.09$ ($p < .01$) with FA, and $-.04$ (n.s.) with AP. In the WRRP, these correlations were estimated separately for each twin: for twin 1, $r(193) = .05$ (n.s.) with aggression, $-.12$ (n.s.) with FA, and $-.10$ (n.s.) with AP; for twin 2, $r(193) = .07$ (n.s.) with aggression, $-.02$ (n.s.) with FA, and $-.09$ (n.s.) with AP. This pattern suggests that boys were slightly higher in FA, AP, and overt aggression, but that sex differences were modest in magnitude. We also found little evidence of age differences based on correlations; this was tested only in the WRRP because age did not vary in the SECCYD. For twin sub-sample 1, the correlations with age were $r(193) = .05$ (n.s.) with aggression, $-.03$ (n.s.) with FA, and $.03$ (n.s.) with AP; for sub-sample 2, $r(193) = .02$ (n.s.) with aggression, $.08$ (n.s.) with FA, and $-.16$ ($p < .05$) with AP.

Hypothesis 1

Next, we tested our first hypothesis—that FA, AP, and overt aggression would all be positively correlated. The correlations are presented in Table 1, and the hypothesized pattern was found in both studies. All were significant, positive in direction, and small to moderate in magnitude (.26 to .44 for FA and AP, .20 to .34 for AP and aggression, and .45 to .52 for FA and aggression). These correlations did not differ for boys and girls. In the SECCYD, the correlations were estimated separately for boys ($n = 503$) vs. girls ($n = 489$): .45 (boys) vs. .43 (girls) for FA and AP; .34 vs. .35 for AP and aggression; and .51 vs. .53 for FA and aggression. In the WRRP, the correlations were not as consistent across sexes, but none of the differences replicated across the two sub-samples. For sub-sample 1 boys ($n = 77$) vs. girls ($n = 118$), the correlations were .20 vs. .30 for FA and AP; .35 vs. .27 for AP and aggression; and .47 vs. .45 for FA and aggression. For sub-sample 2 boys ($n = 79$) and girls ($n = 116$), the correlations were .22 vs. .36 for FA and AP; .20 vs. .22 for AP and aggression; and .57 vs. .36 for FA and aggression. Thus, the statistical associations between the three study variables were largely consistent across the two independent samples, across the two twin sub-samples within the WRRP, and across subgroups of boys and girls.

Hypothesis 2

Our second hypothesis was that FA and AP would provide some overlapping as well as independent statistical prediction of overt aggressive behavior. For the first part of the hypothesis, we used simultaneous regression in which we statistically predicted overt aggression from FA and AP. Separate equations were estimated for the SECCYD sample, WRRP twin sub-sample 1, and WRRP sub-sample 2. In the SECCYD, FA ($\beta = .45, p < .001$) and AP ($\beta = .17, p < .001$) accounted for 31% of the variance ($p < .001$) in overt aggression. Of this 31%, 8% overlapped across FA and AP, and 23% was attributable to their additive independent effects. Thus, FA and AP provided overlapping as well as independent statistical prediction of overt aggressive behavior in the SECCYD. A very similar pattern was found in WRRP sub-sample 1. FA ($\beta = .40, p < .001$) and AP ($\beta = .21, p < .01$) accounted for 24% of the variance in overt aggression, $F(2, 192) = 31.23, p < .001$. Of this 24%, 4% overlapped across FA and AP, and 20% was due to their additive independent effects. A distinct pattern was found in WRRP sub-sample 2, because FA ($\beta = .47, p < .001$) but not AP ($\beta = .07$, non-significant) accounted for 23% of the variance in overt aggression, $F(2, 192) = 30.33, p < .001$. Of this 23%, nearly all (22%) was attributable to the independent effect of FA.

Given that some of the statistical prediction of aggression overlapped between FA and AP, did one temperament dimension statistically mediate the effect of the other? We used correlational and regression procedures described by Baron and Kenny (1986). The conditions for testing statistical mediation were met in both samples—in preceding analyses,

the three variables in question (FA, AP, and aggression) were significantly correlated with each other; FA and AP were significant predictors of aggression; and FA and AP significantly predicted each other in separate univariate regression equations.

We estimated two sets of equations—one set testing FA as a mediator (AP → FA → aggression) and a second set testing AP as a mediator (FA → AP → aggression). For the first set (FA as mediator), we used hierarchical regression with aggression as the outcome and AP as the statistical predictor (step 1), then entered FA as the mediator (step 2). For the second set of equations (AP as mediator), aggression was the outcome and FA was the predictor (step 1) then AP was included as the mediator (step 2). These equations were estimated separately for SECCYD, WRRP sub-sample 1, and WRRP sub-sample 2. All of the step 1 and step 2 equations were statistically significant ($p < .001$ in SECCYD, $p < .01$ in WRRP), and unless indicated otherwise, the standardized regression coefficients also were significant ($p < .001$ in SECCYD, $p < .01$ in WRRP).

A summary of the standardized regression coefficients for unmediated and mediated effects is presented in Figure 1. None of the mediated effects was 0 and all but one remained significant, suggesting partial rather than full mediation. At the same time, direct estimates of the statistical significance of the mediated effects using the Sobel test statistic indicated that all but one were statistically significant at $p < .05$ (two-tailed, ranging from 2.49 to 3.61)—no doubt due to the high statistical power arising from the large samples. Therefore, we examined effect sizes using the standardized regression coefficients and magnitude of reduction in explained variance, for interpreting the partial mediation effects. As Figure 1 shows the regression path from AP → aggression decreased substantially with the inclusion of FA as mediator—a pre/post-mediation difference in β of .20 (SECCYD), .10 (WRRP sub-sample 1), and .13 (WRRP sub-sample 2). This represented an 83% (SECCYD), 60% (WRRP sub-sample 1), and 88% (WRRP sub-sample 2) reduction in explained variance in AP → aggression when the mediating effect of FA was included in the equation. In contrast, the regression paths for FA → aggression decreased only modestly with inclusion of AP as mediator—a pre/post-mediation difference in β of .06 (SECCYD), .05 (WRRP sub-sample 1), and .02 (WRRP sub-sample 2). This was equivalent to a 22% (SECCYD), 20% (WRRP sub-sample 1), and 8% (WRRP sub-sample 2) reduction in explained variance in FA → aggression when the mediating effect of AP was included in the equation. To summarize, there was statistically significant overlapping variance in FA and AP in the prediction of aggression, and there was evidence of a relatively substantial partial mediation effect for AP → FA → aggression compared to a relatively modest partial mediation effect for FA → AP → aggression.

Hypothesis 3

Our third hypothesis was that the overlap in statistical prediction of aggression from AP and FA at the “phenotypic” level of analysis (hypothesis 2) would also be seen at the behavioral genetic analysis, evidenced as substantial overlapping genetic influence. To test this hypothesis, we conducted behavioral genetic analyses to test for underlying genetic and non-genetic sources of variance and covariance linking FA, AP, and aggression. Only data from the WRRP twin study were used for these analyses. We computed twin intra-class correlations and cross-correlations, which are shown on the left side of Table 2.

The intra-class correlations on the diagonals were substantial for MZ twins (.81 to .82) and moderate for DZ twins (.38 to .55), suggesting evidence of heritable variance in all three study variables. The cross-correlations (off-diagonal correlations) were consistently higher for MZ twins than for DZ twins: .36 vs. .07 for FA-AP, .41 vs. .26 for FA-aggression, and .29 vs. .04 for AP-aggression. This suggested that there was potential overlap in the genetic variance between the three variables.

To test for overlapping and independent genetic, shared environmental (i.e., leading to family member similarity), and nonshared environmental (i.e., leading to family member differences) statistical effects, a multivariate Cholesky decomposition was used to partition the variances of, and covariances between, approach/positive anticipation, frustration/anger, and aggression (Neale & Cardon, 1992). An advantage of this model is that it allows for the variables to be placed in an order that represents the statistical mediation found in the phenotypic analyses described above (i.e., AP → FA → aggression). 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. In this model, the pathways between latent variables representing genetic variance or covariance across twins are set at 1 for monozygotic (MZ, identical) twins and .5 for dizygotic (DZ, fraternal) 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.

So as to not need to arbitrarily designate a “twin 1” and “twin 2”, we used double-entered data and adjusted degrees of freedom (−9) in the model accordingly. The ACE model fit well, $\chi^2(15) = 5.97, p = .980, AIC = -24.03, RMSEA = .005$. The CE model (in which the A paths are set to 0) did not fit well, $\chi^2(21) = 73.70, p = .000, AIC = 31.70, RMSEA = .230$. The chi-square difference between the CE and ACE model (6 df) was 67.73, $p < .001$, far exceeding the critical value of 12.59, suggesting that model fit deteriorated significantly if the A paths were removed. We then tested an AE model, in which the C paths were set to 0. This model fit the data well, $\chi^2(21) = 13.20, p = .902, AIC = -28.80, RMSEA = .037$. The chi-square difference between the AE and ACE model (6 df) was 7.23, *n.s.*, well below the critical value of 12.59. In the AE model, dropping the C paths from the ACE model did not lead to a significant deterioration in model fit. The AE model also had the largest negative AIC and the most degrees of freedom; therefore, the AE model was the most parsimonious and best fitting model to the data.

Standardized path estimates (with 95% confidence intervals) are shown in Figure 2, and were consistent with the hypothesis. The paths for “C” and “c” (shared environment) were fixed at 0 in the best fitting AE model, and are not shown. The path estimates show evidence for a statistically significant common genetic factor (A1) accounting for heritable variance in AP, FA, and aggression. Furthermore, even after controlling for this overlapping genetic covariance, there remained significant genetic covariance between FA and aggression (A2)—and even with all of the overlapping genetic covariance with FA and AP controlled, there remained independent genetic variance in aggression (A3). The same pattern was found for nonshared environment covariance (E1, E2, and E3). However, the paths representing overlapping nonshared environment covariance were modest (from .10 to .19) whereas the paths representing genetic covariance were modest to moderate in magnitude (.19 to .37).

Discussion

Developmental theories about reward-seeking behavior and overt aggression have implicated behavioral approach/positive anticipation (AP) and frustration/anger (FA) as important aspects of the etiology of aggressive behavior problems in childhood. However, much remains to be learned about the overlapping and independent links between these two aspects of temperament and aggression—at the phenotypic and behavioral genetic levels of analysis. We tested three hypotheses in an effort to further our understanding of these connections.

Our first hypothesis was that AP, FA, and overt aggression would be positively correlated with each other. Findings from two independent studies with participants spanning 4–9 years of age were consistent with this hypothesis. In addition, these associations were very similar for boys and girls. Though boys typically show somewhat higher levels of overt aggressive behavior compared to girls (Crick & Grotpeter, 1995), the current findings suggest that the sexes are more alike than different when it comes to the associations between AP, FA, and aggression. If replicated, this finding implies that the role of an approach motivational system in the expression of overt aggression operates in a similar way for males and females.

The second hypothesis was that AP and FA would provide independent as well as some overlapping statistical prediction of aggressive behavior. There was support for this hypothesis. Results from the regression analyses showed that in both the WRRP and SECCYD studies, anger and approach together accounted for variance in overt aggression—although each provided some independent statistical prediction as well. This finding suggests that FA and AP are distinct but related in their links with aggressive behavior problems, perhaps as components of a broader behavioral approach motivation system that can promote aggression in some children (Derryberry & Rothbart, 2001). We found that children with higher levels of FA or AP were more likely to exhibit overt aggressive behavior. More importantly, these appeared to be additive effects whereby children who were high in both AP and FA were the most likely to exhibit overt aggression and those who were low in AP and FA were the least likely to do so.

We also explored whether FA or AP operated as statistical mediators of each others' links with aggression. As summarized in Figure 1, the results suggested partial statistical mediation. With the inclusion of FA as a statistical mediator (AP → FA → aggression), there was a 77% average reduction in explained variance from AP → aggression. In contrast, including AP as a statistical mediator (FA → AP → aggression) resulted in a relatively modest 15% average reduction in explained variance from FA → aggression. Thus, although both FA and AP contributed to the statistical prediction of overt aggression, much of this predictive effect was overlapping and most of this overlap could be attributed to FA as a statistical mediator.

Although they are based on correlational data, these findings are consistent with the theory that AP is indicative of appetitive, reward-sensitive behavior that for some children may increase FA—perhaps as a result of people or circumstances interfering with their ability to obtain those rewards—that in turn can increase the likelihood of overt aggression. But would this pattern in the observable (i.e., phenotypic) level of analysis be found in the latent behavioral genetic level of analysis? Previous studies have found moderate to substantial genetic and nonshared environmental variance in aggressive, FA, and AP behaviors (Goldsmith et al., 1997; Oniszczenko et al., 2003; Rhee & Waldman, 2002). Whether and how genetic influences on AP and FA also account for genetic variance in overt aggression has, to our knowledge, not been examined. Therefore, our third hypothesis was that the statistical mediation found at the phenotypic level of analysis would also be seen in the genetic variances of AP, FA, and aggression.

There was support for this hypothesis (Figure 2). Significant portions of the genetic variance in aggression overlapped with the genetic variance in FA and AP (i.e., genetic correlation). This suggests that when specific genetic influences on overt aggression are identified, the expectation is that at least part of their additive genetic effect will operate through influences on stronger AP and FA tendencies—genetic influences that likely involve the production and use of dopamine neurotransmitter (Pickering & Gray, 2001).

At the same time, it is important to emphasize that with the genetic influences involving AP and FA statistically controlled, there remained substantial genetic variance in aggressive behavior. This is heritable variance that could be accounted for by different sets of genes. For instance, FA and overt aggression can arise from thwarted reward-seeking behavior, but also can arise from fear (i.e., “defensive” anger leading to aggression). Though moderately correlated, fear and anger are distinct negative emotions, with fear being fundamental to the behavioral inhibition system involved in withdrawal from potential punishment (Gray, 1987). The underlying gene-environment and temperament processes involved in defensive anger and aggression are likely to be distinct from those involved in offensive instrumental anger and aggression (Posner & Rothbart, 2007, p.127).

In addition, although we had no hypothesis for the effect, we found similarly clear evidence in the quantitative genetic modeling for overlapping nonshared environmental influences—non-genetic influences that do not contribute to sibling similarity that accounted for some of the covariation between AP, FA, and aggression. This could reflect, in part, systematic child-specific error variance in mothers’ ratings across the three constructs, since nonshared environmental variance includes random measurement error. However, it also is plausible that certain aspects of the environment that promote sibling differentiation operate on the affective and behavioral aspects of an approach motivational system, such as twin differences in parent-child and peer relationships and interaction dynamics. With respect to aggression and antisocial behaviors more generally, candidates for nonshared environment processes include harsh reactive parenting and peer rejection and affiliation (Bullock, Deater-Deckard, & Leve, 2006; Mullineaux, Deater-Deckard, Petrill, & Thompson, in press). However, much remains to be learned about whether such nonshared environmental processes can be identified and replicated across studies (Reiss, Neiderhiser, Hetherington, & Plomin, 2000; Turkheimer & Waldron, 2000).

Although the use of independent samples and internal replication within the WRRP are strengths, there are limitations to the study that should be considered when interpreting the findings. Only mothers’ reports were used. In addition, although the WRRP sample was socioeconomically diverse, it was not representative of families in the US with school-age children in regard to ethnicity and parent education levels. However, replication of effects from the SECCYD suggests this may be a minor concern. With respect to the behavioral genetic design, the twin design is underpowered for detecting shared environmental effects—though it is noteworthy that the “C” and “c” parameters all had effect sizes near zero, suggesting that even with a very large sample these paths would not have been significant.

In conclusion, the results from the current study suggest that the distinct yet inter-related temperament dimensions of FA and AP dispose some children to overt aggressive behavior problems. The statistical mediation results suggest an underlying process involving a behavioral activation system in which reward-seeking behavior, when impeded, increases the likelihood of FA and overt aggression for some children. The results suggest that intervention efforts to reduce aggression should be concerned with differentiating children with a combination of strong appetitive approach tendencies and low frustration tolerance from children who show only one of these tendencies—it is the former group of children who are likely to be more aggressive. Furthermore, interventions that target management or regulation of frustration and anger may be more effective if they also address management of approach behavior. Finally, the finding of both genetic and nonshared environmental influences is important, although the specific genetic and environmental factors that account for these effects remain to be identified. However, such an endeavor holds the promise of clarifying mechanisms of change that will improve our understanding of “what works” for improving interventions for aggressive behavior problems.

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Abbreviations

FA	frustration/anger
AP	approach/positive anticipation

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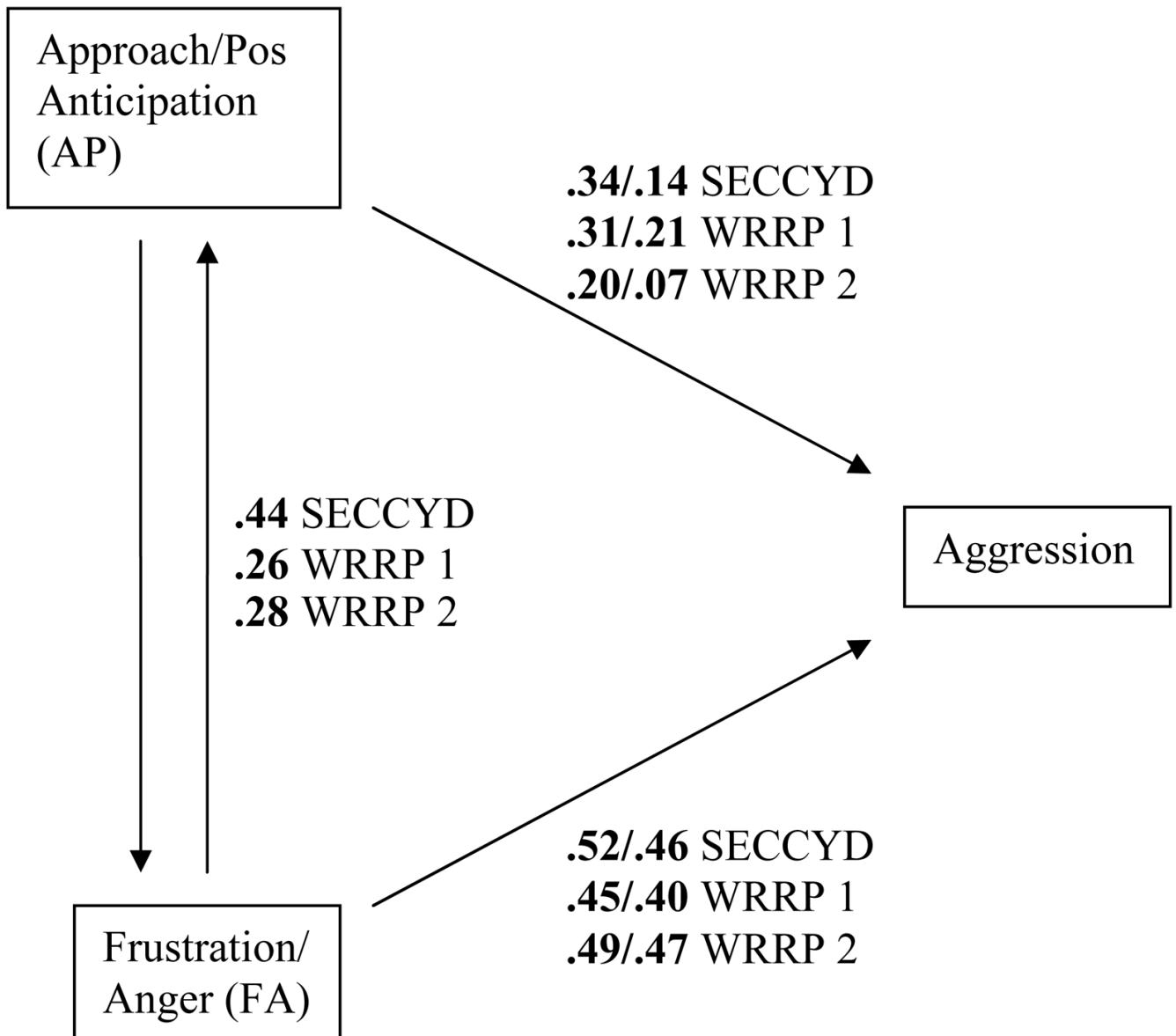


Figure 1.

Summary of the standardized path estimates from tests of statistical mediation. In separate regression models, frustration/anger (FA) was tested as a mediator of approach/positive anticipation (AP) → aggression, and AP as a mediator of FA → aggression. Standardized regression coefficients for the prediction of aggression are shown prior to/following inclusion of statistical mediator (e.g., .34/.14), separately for the SECCYD sample and WRRP sub-samples 1 and 2. Within each sample, the path coefficient between AP and FA was the same regardless of which variable was being tested as the mediator, so only one number is shown.

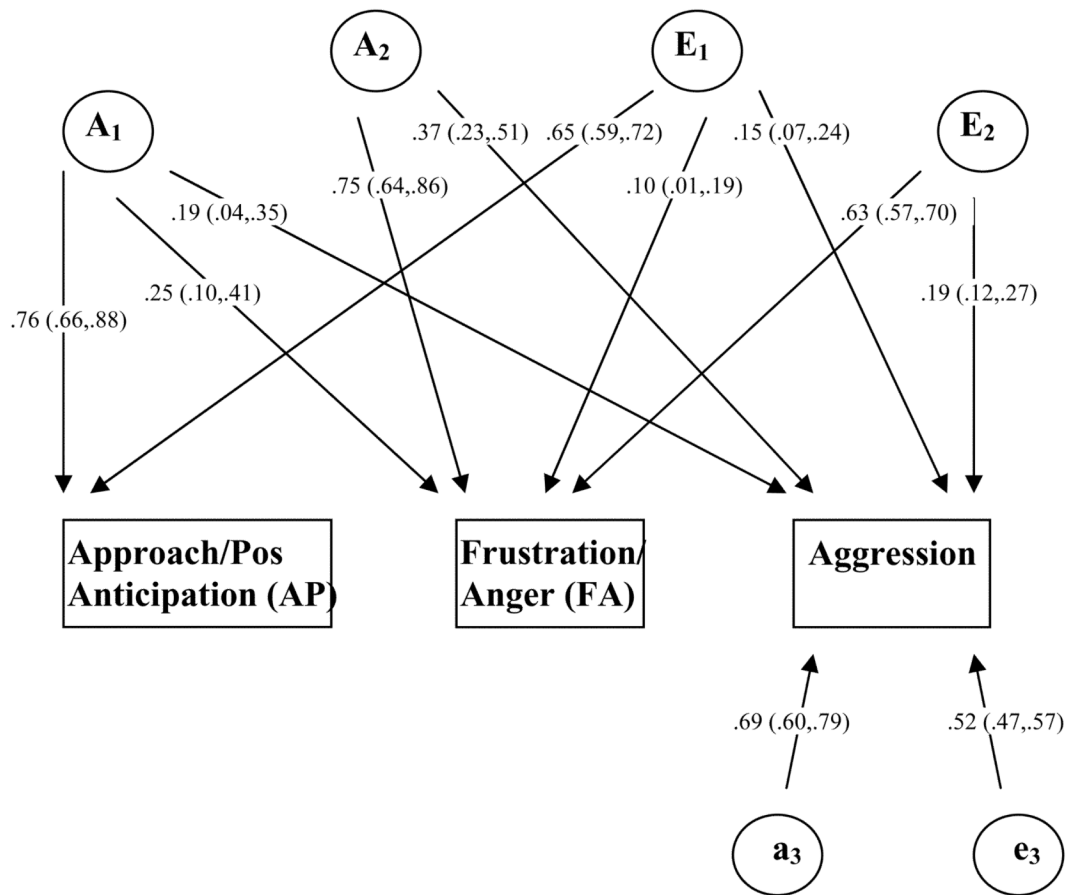


Figure 2.

Standardized path estimates for best fitting "AE" model. We used a multivariate Cholesky decomposition to partition the variances of, and covariances between, approach/positive anticipation (AP), frustration/anger (FA) and aggression, into genetic and non-genetic components. 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. Shared environment paths (C, c) are not shown, as they were fixed = 0 in the best fitting model because shared environment effects were negligible. The pathways between latent variables representing genetic variance or covariance across twins are set at 1 for monozygotic (MZ, identical) twins and .5 for dizygotic (DZ, fraternal) 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

Means (with standard deviations) and correlations (all correlations significant, two-tailed $p < .05$) in the Study of Early Child Care and Youth Development (SECCYD) and the Western Reserve Reading Project (WRRP)

SECCYD, n = 992			
	<u>FA</u>	<u>AP</u>	<u>Agg</u>
Frustration/Anger (FA)	4.74 (.83)		
Approach/Pos Anticip (AP)	5.21 (.63)	.44	
Aggression	7.80 (5.28)	.52	.34
WRRP, n = 390 (195 twin pairs)			
	<u>FA</u>	<u>AP</u>	<u>Agg</u>
	Sample 1	Sample 2	
Frustration/Anger (FA)	3.72 (1.14)	3.98 (1.22)	.61 .28 .49
Approach/Pos Anticip (AP)	5.14 (0.72)	5.12 (0.73)	.26 .58 .20
Aggression	6.20 (4.64)	5.85 (4.94)	.45 .31 .67

* Note: Diagonal includes twin intra-class correlations in italics. Correlations for twin sub-sample 1 are below diagonal, for sub-sample 2 above diagonal.

Table 2

Twin intra-class (italics, on diagonal) and cross-twin correlations (off diagonal) for monozygotic (MZ) and dizygotic (DZ) twins

	Intra-Class, Cross Correlations	
	MZ (81_{pairs})	DZ (114_{pairs})
Approach	<i>.81</i>	.38
Frustration/Anger	.36	<i>.82</i>
Aggression	.29	<i>.41</i>
		<i>.82</i>
		<i>.26</i>
		<i>.55</i>

Note: All estimates significant at two-tailed $p < .05$ unless noted (*ns*).