



Published in final edited form as:

*J Res Pers.* 2014 February ; 48: 51–60. doi:10.1016/j.jrp.2013.11.002.

## Examining the etiological associations among higher-order temperament dimensions

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### Abstract

A multivariate independent pathway model was used to examine the shared and unique genetic and environmental influences of Positive Affect (PA), Negative Affect (NA), and effortful control (EC) in a sample of 686 twin pairs ( $M$  age = 10.07,  $SD$  = 1.74). There were common genetic influences and nonshared environmental influences shared across all three temperament dimensions and shared environmental influences in common to NA and EC. There were also significant independent genetic influences unique to PA and NA and significant independent shared environmental influences unique to PA. This study demonstrates that there are genetic and environmental influences that affect the covariance among temperament dimensions as well as unique genetic and environmental influences that influence the dimensions independently.

### Keywords

Positive Affect; Negative Affect; Effortful control; Childhood; Twin study

## 1. Introduction

Individual differences in the expression of various temperament traits play a substantial, yet complicated role in normal and abnormal development in childhood (e.g., Eisenberg, Fabes, Guthrie, & Reiser, 2000; Nigg, 2006; Rettew, Copeland, Stanger, & Hudziak, 2004). Behavioral genetics studies have been used to decompose the etiology of childhood temperament traits into their genetic and environmental components (e.g., Lemery-Chalfant, Doelger, & Goldsmith, 2008; Mullineaux, Deater-Deckard, Petrill, Thompson, & DeThorne, 2009). However, most of these studies only consider the genetic and environmental components unique to individual temperament traits and not the overlapping genetic and environmental components shared among traits. This study fills this gap in the literature by examining both the unique and shared etiological influences of core temperament dimensions in children.

Research on temperament is necessary because of the integral role of temperament in the development of adult personality as well as child and adult psychopathology (Nigg, 2006; Tackett, 2006). There is now considerable agreement that temperament and personality are highly overlapping constructs in children (e.g., Caspi & Shiner, 2006; De Pauw & Mervielde, 2010; Grist & McCord, 2010). Therefore, the transition from child temperament

to adult personality does not concern the influence of temperament on the development of personality but rather concerns developmental continuity of temperament/personality (however, see Rothbart, 2011, for an opposing viewpoint). There is ample empirical evidence of an association between temperament traits and psychopathology (e.g., Eisenberg et al., 2005; Gjone & Stevenson, 1997; Leve, Kim, & Pears, 2005; Lonigan, Vasey, Phillips, & Hazen, 2004), as well as theoretical models that attempt to explain these relations between temperament/personality and psychopathology (e.g., vulnerability, pathoplasty models; Tackett, 2006). Understanding the etiological underpinnings of temperament may help clarify the developmental continuum of temperament/personality and the relations this construct shares with psychopathology.

### 1.1. The structure of temperament

Several different theoretical models have been developed to describe the structure and biological underpinnings of temperament (e.g., Buss & Plomin, 1984; Goldberg, 1990; Rothbart & Bates, 2006). Most models describe temperament hierarchically, consisting of at least three core dimensions at or near the top of the hierarchy. These include two reactive dimensions, Positive Affect (PA) and Negative Affect (NA), and a regulatory dimension, effortful control (EC; Mervielde, De Clercq, De Fruyt, & Van Leeuwen, 2005; Rothbart & Bates, 2006). The reactive dimension of PA is associated with approach behavior and the expression of positive emotions and is consistent with characteristics such as enthusiasm, pleasurable engagement, and sociability. The reactive dimension of NA is associated with withdrawal behavior and the expression of negative emotions and is consistent with characteristics such as nervousness, anger, guilt, and sadness (Watson & Clark, 1984). The regulatory dimension of EC acts as a moderator of the reactive dimensions (Lonigan et al., 2004). This construct is associated with suppressing reactive behavior and emotions. High levels of EC are synonymous with high levels of attentional control (i.e., focusing and shifting attention; Derryberry & Reed, 2002). Further, EC is thought to be necessary for planning and goal-directed behavior (Caspi, Roberts, & Shiner, 2005; Nigg, 2006; Rothbart & Bates, 2006). These three dimensions of temperament have been consistently extracted in factor analytic studies involving children and adolescents, using both other- and self-report measures (Gartstein & Rothbart, 2003; Muris, Meesters, de Kanter, & Timmerman, 2005; Rothbart, Ahadi, Hershey, & Fisher, 2001; see Nigg, 2006; Putnam, Ellis, & Rothbart, 2001, for reviews). Further, these terms are highly overlapping with the terms extraversion (PA), neuroticism (NA), and conscientiousness (EC) that are used in personality research (De Pauw & Mervielde, 2010; Mervielde et al., 2005), so much so that for consistency and clarity, we use the term temperament when discussing either child temperament or child personality studies throughout the rest of the current study.

Whereas several theories of temperament operate under the assumption that the higher-order dimensions are orthogonal and have attempted to create measures reflecting this orthogonality (e.g., Costa & McCrae, 1985; Goldberg, 1992), evidence has accumulated that most temperament factors covary (e.g., Digman, 1997; Musek, 2007; Zawadzki & Strelau, 2010). For example, Musek (2007) examined correlations across the Big Five personality dimensions (e.g., extraversion, agreeableness, conscientiousness, neuroticism, and openness to experience [openness]) in two adult and one adolescent Slovenian sample. In one of the adult samples and the adolescent sample, all of the dimensions were significantly correlated, with absolute values of correlations ranging from .17 to .54. In the other adult sample, most, but not all, dimensions were significantly correlated as well. Relevant to the focus on temperamental PA, NA, and EC in the current study, Musek found that extraversion and neuroticism were correlated at  $-.35$ , and conscientiousness (similar to EC) was correlated with extraversion at  $.36$  and with neuroticism at  $-.37$  in the adolescent sample. The overlap among temperament/personality traits has led several researchers to propose a hierarchical

structure of temperament comprising two higher-order dimensions, labeled  $\alpha$ , under which NA and EC as well as agreeableness are subsumed, and  $\beta$ , under which PA as well as openness are subsumed (e.g., Digman, 1997). Others have proposed the presence of a single higher-order factor of temperament/personality, labeled the general factor of personality (GFP; e.g., Musek, 2007; Rushton, Bons, & Hur, 2008). Support for both of these hierarchical models has been found using exploratory and confirmatory factor analysis (CFA) in adults as well as children and adolescents (e.g., Digman, 1997; Musek, 2007; Rushton et al., 2008; Wang, Chen, Petrill, & Deater-Deckard, 2013; Zawadzki & Strelau, 2010). Behavioral genetics studies focusing on the covariance between PA, NA, and EC can help refine temperament models by clarifying the nature of the genetic and environmental influences on this covariance.

## 1.2. Genetic and environmental influences on temperament

Twin studies can be used to identify the proportion of genetic and environmental influences associated with temperament dimensions. Additive genetic influences (or heritability,  $h^2$ ) are those that children inherit from their parents. Shared environment influences include aspects of the environment that make twins more similar ( $c^2$ ). Nonshared environment influences include environmental factors unique to each twin (as well as measurement error;  $e^2$ ). These genetic and environmental influences are population statistics, providing information about sample-level variance and are therefore not specific to an individual.

Numerous twin studies have examined the univariate etiology of PA, NA, and EC in children and adolescents. Most studies, conducted across multiple measurement approaches, including self- and other-report, as well as direct assessment, report genetic estimates, in terms of proportions of phenotypic variance accounted for, ranging from around .20 to around .60 and nonshared environmental influences ranging from around .40 to around .80 (e.g., Anokhin, Golosheykin, Grant, & Heath, 2011; Goldsmith, Buss, & Lemery, 1997; Lemery-Chalfant et al., 2008; Mullineaux et al., 2009; Rettew et al., 2006; Spengler, Gottschling, & Spinath, 2012; see Saudino, 2005 for review). There is also mixed evidence of shared environmental influences for PA. For example, in a study using an actigraph to monitor activity level in a laboratory setting for 463 seven- to nine-year-old twin pairs, Wood, Saudino, Rogers, Asherson, and Kuntsi (2007) reported genetic effects of .36, shared environmental influences of .39, and nonshared environmental influences of .25. Isen, Baker, Raine, and Bezdjian (2009) examined the related trait of novelty seeking using a self-report measure in a sample of 605 nine- and ten-year-old twin pairs and found no genetic influences, shared environmental influences of .29, and nonshared environmental influences of .71. In contrast, there is little to no evidence of shared environmental influences in univariate studies of NA or EC. A study by Mullineaux et al. (2009) is one exception, as they found no genetic influences, a shared environmental influence of .57, and a nonshared environmental influence of .33 for father-reported temperament. In contrast, they found genetic estimates of .71, nonshared environmental estimates of .31 and no shared environmental estimates for mother-reported temperament. However, roughly half as many fathers completed questionnaires than did mothers (father-report was available for 98 twin pairs, mother-report was for 197 twin pairs). Further, the authors posited that rater bias may have accounted for discrepancies between mother- and father-report. Although they were unable to determine whether fathers' or mothers' ratings showed bias, mother-ratings of NA were more consistent with the values given in other studies than were father-ratings of NA.

Multivariate twin studies that include PA, NA, and EC in the same model provide a useful approach for exploring the levels of common and unique genetic, shared environmental, and nonshared environmental overlap among PA, NA, and EC, (Caspi et al., 2005; Saudino, 2005). However, only a few studies have moved beyond univariate twin study designs to

examine the overlap among multiple temperament dimensions. Deater-Deckard, Petrill, and Thompson (2007) used a multivariate Cholesky decomposition model to examine the covariance between observer-rated lower-order traits subsumed under EC and NA (i.e., task persistence for EC and anger/frustration for NA) and parent- and teacher-rated conduct problems in a sample of 259 twin pairs ( $M$  age = 6.09 years,  $SD = .69$ ). They reported a non-significant genetic correlation of .43 and a significant nonshared environmental correlation of .49 between task persistence and anger/frustration. Deater-Deckard et al. suggested that the non-significant genetic overlap was a function of the small sample size and not a lack of a genetic association between their measures of EC and NA. Gagne and Goldsmith (2010) examined the genetic and environmental influences between anger (a lower-order NA trait) at 12 and 36 months and lab-assessed EC at 36 months in a sample that ranged from 423 to 500 twin pairs. In a model containing lab-assessed anger, they reported no significant genetic overlap. A significant shared environmental correlation of  $-.73$  was found between anger and EC at 36 months and a significant nonshared environmental correlation of .22 was found between anger at 12 months and EC at 36 months. In a model containing parent reports of children's anger, they found significant genetic correlations between EC at 36 months and anger at 12 ( $r_g = -.26$ ) and 36 months ( $r_g = -.56$ ), respectively. They also found a significant nonshared environmental correlation of .22 between EC and anger at 36 months. To our knowledge, no other studies of children and adolescents have examined the common genetic and environmental influences across multiple temperament dimensions.

A recent study by Wang et al. (2013), although not a multivariate study, is informative about potential multivariate relations among temperament dimensions. Wang et al. examined the genetic and environmental influences for observer-reported  $\alpha$  and  $\beta$  scales, independently, in a sample of 1056 twins 3.5- to 12-years old. Traits related to NA and EC were subsumed under  $\alpha$  with agreeableness, and traits related to PA were subsumed under  $\beta$  with openness. For  $\alpha$ , Wang et al. found significant genetic ( $a^2 = .36$ ), shared environmental ( $c^2 = .27$ ), and nonshared environmental ( $e^2 = .37$ ) influences. For  $\beta$ , they found significant shared environmental ( $c^2 = .47$ ), and nonshared environmental ( $e^2 = .43$ ) influences but no genetic influences. Their findings suggest that the common phenotypic relation between NA and EC is a product of genetic, shared environmental, and nonshared environmental influences. However, the nature of the models tested (i.e., univariate  $\alpha$  and  $\beta$  behavioral genetics models tested independently) precluded tests of unique genetic and environmental estimates of PA, NA, or EC as well as tests of common genetic and environmental influences for PA with NA and EC.

Although the correspondence between child temperament/personality and adult personality is not exact because of the developmental nature of this construct (i.e., temperament/personality; Roberts & DelVecchio, 2000), multivariate studies with adults can still inform what is to be expected in child studies. There are few multivariate adult twin studies of personality (e.g., Jang et al., 2006; Krueger, 2000). Krueger (2000) conducted principal components analysis (PCA) of 11 purported lower-order personality traits in a sample of 2490 adults ( $M$  age = 39.80 years,  $SD = 6.54$ ). He found that, across phenotypic, genetic, and nonshared environmental covariance matrices, traits clustered together into dimensions consistent with PA, NA, and EC factors. Although not explicitly tested, Krueger indicated that this result might suggest little to no genetic or nonshared environmental overlap among personality dimensions. In a sample of 1910 adult twin pairs from multiple countries (i.e., Canada, Germany, and Japan), Jang et al. (2006) fit a common factors model independently to  $\alpha$  and  $\beta$  factors. For the  $\alpha$  factor, neuroticism (similar to NA) and constraint (similar to EC) demonstrated both unique and overlapping genetic and nonshared environmental influences. Whereas extraversion (similar to PA) demonstrated unique and overlapping genetic influences on the  $\beta$  factor, these results do not provide information about the etiological relations found among all three core temperament dimensions. The paucity of

child and adult studies examining higher-order temperament/personality factors from a multivariate perspective highlight a glaring need for studies identifying unique and shared genetic and environmental aspects of temperament dimensions.

### 1.3. Current study

The purpose of this study was to examine the common genetic and environmental influences on the three higher-order temperament dimensions, NA, PA, and EC. Researchers have demonstrated covariances across these temperament factors using multiple measures and approaches (e.g., Deater-Deckard et al., 2007; Zawadzki & Strelau, 2010). Therefore, we hypothesized that there would be common variance among these factors to be accounted for. Further, because the preponderance of evidence indicates that temperament factors can be accounted for primarily by genetic and nonshared environment effects (e.g., Mullineaux et al., 2009; Rettew et al., 2006; Saudino, 2005), we hypothesized that the covariance among the temperament factors would be accounted for by genetic effects and nonshared environmental influences and that there would be no significant shared environmental covariance. Given that the covariance across temperament factors is typically reported as moderate (e.g., Digman, 1997), we expected to find unique etiological influences as well. Based on the reviewed literature (e.g., Anokhin et al., 2011; Deater-Deckard et al., 2007; Mullineaux et al., 2009), we hypothesized that unique genetic and nonshared environmental influences would be found for all three temperament dimensions. Given the equivocal findings for shared environmental influences regarding PA (e.g., Isen et al., 2009; Mullineaux et al., 2009), we expected that if unique shared environmental influences emerged, they would be for PA only.

## 2. Method

### 2.1. Participants

Participants for this study were part of the Florida State Twin Registry (Taylor, Hart, Mikolajewski, & Schatschneider, 2012; Taylor, James, Reeves, & Bobadilla, 2006) who were recruited through the Florida Twin Project on Reading (Taylor & Schatschneider, 2010). Using a statewide database of school registration information, pairs of children who had the same last name, birth date, and school were identified as potential twin pairs and sent a recruitment packet including a letter describing the Florida State Twin Registry, a parent consent form, and an assessment of zygosity (Lykken, Bouchard, McGue, & Tellegen, 1990). Those families who agreed to be part of the Florida State Twin Registry and had same-gender twins were targeted for the current study. They were sent a packet containing a cover letter, consent and assent forms, and a questionnaire booklet for one parent (or guardian) to complete. Of the 1624 packets sent out, 150 were returned by the post office as undeliverable, and 693 families participated. Upon returning the questionnaire booklet, parents received a \$30 gift card for their participation. Two families were not included because the zygosity of the twins could not be determined, and five families were missing data on all variables included in this study.

The final sample for this study included 686 same-gender 7- to 13-year-old ( $M = 10.07$ ,  $SD = 1.74$ ) twin pairs (180 families with monozygotic [MZ] female twins; 168 families with dizygotic [DZ] female twins; 167 families with MZ male twins; 171 families with DZ male twins). Parent-reported racial composition of the sample was 70% White, 12% African American, 9% mixed race, 2% Asian, and 5% other (with 2% failing to provide information about race). Parent-reported ethnic composition was 24% Hispanic and 71% non-Hispanic (with 5% not reporting ethnicity). Parents reported their family income on a scale ranging from 1 (indicating 'less than \$10,000') to 6 (indicating '\$90,000 or greater'). Parent-reported mean household income roughly corresponded to the '\$50,000 to \$69,000' response option

and the modal reported income was \$90,000 or more. Income was not reported by 3% of the sample. Additional details regarding this sample have been reported elsewhere (Mikolajewski, Allan, Hart, Lonigan, & Taylor, 2013).

## 2.2. Measures

**2.2.1. Positive Affect and Negative Affect**—The Positive and Negative Affect Schedule (PANAS; Watson, Clark, & Tellegen, 1988) was used to measure parent-rated positive and Negative Affect in the twins. The PANAS consists of 10 descriptive terms that are markers of Negative Affect, and 10 terms that are markers of Positive Affect (shown in Table 1). Parents rated the extent to which their twins experienced each emotion in general using a 5-point scale ranging from “very slightly or not at all” to “extremely.” The use of parent-rated affect has been supported by previous research (Ebesutani, Okamura, Higa-McMillan, & Chorpita, 2011). The PANAS has been used as both a trait measure of temperament and a state measure of mood. When used as a trait measure, ratings are made using a “general” timeframe. The use of the PANAS as a trait measure of PA and NA has been supported by previous research in children and adults (e.g., Lonigan, Phillips, & Hooe, 2003; Watson & Clark, 1997). Items on the Positive Affect scale were used as indicators of the Positive Affect factor and items on the Negative Affect scale were used as indicators of the Negative Affect factor. The scale reliabilities were adequate ( $\alpha = 0.86$  and  $0.85$ , respectively).

**2.2.2. Effortful control**—The Persistence/Low Distractibility scale from the Effortful Control Scale (ECS; Lonigan & Phillips, 2001) was used to measure parent-rated EC in the twins. The ECS is a 24-item measure consisting of two scales (Persistence/Low Distractibility and Impulsivity). The presence of two factors was verified in the present analysis using CFA in Mplus version 5.1 (Muthén & Muthén, 2008). A two-factor model of the ECS demonstrated adequate overall fit ( $\chi^2 = 997.57$ ,  $df = 251$ ,  $p < .001$ , CFI = .89, RMSEA = .05) and demonstrated significantly improved fit as compared to the fit of a one-factor model ( $\Delta\chi^2 = 218.36$ ,  $df = 1$ ,  $p < .001$ ). The 12 items on the Persistence/Low Distractibility scale were used in the current study as indicators of the Effortful Control factor because this scale was determined to reflect EC more accurately than the Impulsivity scale or than a composite scale comprising both scales. In previous studies, the Persistence/Low Distractibility scale demonstrated stronger correlations with other measures of EC than did the Impulsivity scale (e.g., Verstraeten, Vasey, Claes, & Bijttebier, 2010). Further, there is some disagreement as to whether less effortful and more automatic types of control, such as impulsivity, actually measure EC (e.g., Eisenberg et al., 2005). Abbreviated versions of items on this scale are presented in Table 1. Parents rated the extent to which each item described their children on a 5-point scale ranging from “not at all” to “very much.” Higher scores on the scale reflected less EC and lower scores reflected more EC. The reliability of this scale was adequate ( $\alpha = 0.88$ ).

## 2.3. Data analysis

By convention, fraternal or DZ twins share approximately 50% of their segregating alleles, and identical or MZ twins share 100% of their genes. By taking this into account, twin studies can estimate the additive genetic ( $h^2$  or A), shared environmental ( $c^2$  or C), and nonshared environmental ( $e^2$  or E) influences on traits. Intraclass correlations (ICCs) were calculated for each item/indicator and compared across zygosity. When ICCs among MZ twins are higher than those among DZ twins, it indicates genetic influences. When MZ ICCs are less than twice the magnitude of DZ ICCs, it indicates shared environmental influences. Finally, if MZ ICCs are less than 1.00, it indicates nonshared environmental influences (and error).

A multifactorial independent pathway model (Neale & Cardon, 1992) was fit to the residualized raw data to examine the genetic and environmental influences contributing to the covariation of PA, NA, and EC. The raw data was used rather than summary statistics such as correlation matrices because means and variances can be more accurately estimated, leading to more accurate covariance estimates. As a first step, three factors were created. The use of factors (as opposed to scale scores) provides estimates for further analyses that are random-error free (see Gayan & Olson, 2003). Recoding PA items by  $-1$  to reflect a positive relation with NA and EC was done for clarity in understanding the results of this study. For example, conceptually, it is difficult to understand what a negative value representing a genetic covariance means. Next, the independent pathway model was simultaneously applied to the factors, and four sets of biometric factors were estimated. The first set ( $A_1$ ,  $C_1$ , and  $E_1$ ) represented the common genetic and environmental influences contributing to the covariance among PA, NA, and EC. The three remaining sets of biometric factors represented the independent genetic and environmental influences unique to each factor, after accounting for the first set of biometric factors. All sets of A components were set to 1.0 within MZ twin pairs and .5 within DZ twin pairs. All sets of C components were set to 1.0 across MZ and DZ twins, and E components were allowed to freely vary. Finally, residual item-level variance was set to equality across all twins.

### 3. Results

Sample means, standard deviations, and distribution statistics for PA, NA, and EC scales are presented in Table 2. Item scores were normally distributed (skew values were all below 2; kurtosis values were all below 4). Correlations among factor scores are reported in Table 3.<sup>1</sup> To reduce potential bias of age and gender, data were residualized on age, age-squared, gender, and an age-by-gender interaction, as recommended by McGue and Bouchard (1984).<sup>2</sup> These corrected data were used for all analyses. Factor and item-level ICCs (presented in Table 1) were higher among MZ twins across all items, indicating genetic influences. However, given that MZ correlations were less than 1 and not always twice the magnitude of DZ correlations, there was also evidence for nonshared and shared environmental influences.

CFAs were first conducted using Mplus version 5.1 (Muthén & Muthén, 2008) to ensure that the three-factor measurement model demonstrated adequate fit. This model was compared to one- and two-factor theoretical models (see Table 4). The one-factor model (i.e., the GFP model; Rushton et al., 2008) allowed all temperament items to load on a single factor. The two-factor model (i.e., the  $\alpha$  and  $\beta$  model; Digman, 1997) allowed the NA and EC items to load on one factor and the PA items to load on a separate factor. A sandwich estimator was used to control for nesting of children within families. Model fit indices for the three-factor model indicated that the model demonstrated modest overall model fit ( $\chi^2 = 2091.67$ ,  $p < .05$ , CFI = .83, RMSEA = .05). Further, this model demonstrated significantly improved model fit over the one- and two-factor models.<sup>3</sup> Therefore, the three-factor model was accepted as the best-fitting model of temperament. Factor loadings were acceptably high, ranging from .38 to .82, with the exception of one item on the EC scale (i.e., item 18, “often gets lost in work”), which had a loading of .18. To verify that items were properly loading on their respective factor, a three-factor exploratory factor analyses (EFA) was conducted in Mplus and factor loadings were examined. Model fit indices for the three-

<sup>1</sup>Correlations between scale items are available upon request.

<sup>2</sup>There were minimal effects of age, age-squared, gender, and age-by-gender across items, with  $R^2$  ranging from .00 to .05, and an average  $R^2$  of .02.

<sup>3</sup>One- and two-factor hierarchical models were also fit to the data. Model fit for these models are not reported because these models are essentially equivalent to the three-factor model. These models did not improve on the three-factor correlated traits model, as the EC factor did not load significantly on the hierarchical factor in the one- and two-factor models.

factor EFA indicated that the model demonstrated modest overall fit ( $\chi^2 = 1694.87, p < .05$ , CFI = .87, RMSEA = .05). Further, all items loaded significantly on their hypothesized factor, with higher loadings than any non-hypothesized factor, and cross-loadings were acceptably low (<.26). Although the data did not provide exemplary fit to the measurement model, no modifications to the structure of the model, such as allowing correlated residuals within or across factors, were used because altering a model based on modification indices alone takes advantage of chance variance in the data to increase model fit, and EFA confirmed that the items were loading on their correct factors (Kline, 2011).

Biometric model fitting was conducted using Mx (Neale, Boker, Xie, & Maes, 2003). Statistical significance was tested by using likelihood based 95% confidence intervals, which are advantageous compared to standard error-based confidence intervals because they allow for more accurate, asymmetric estimates (Neale & Miller, 1997). Individual item responses were missing for 5–29 participants; missing data were handled by using Full Information Maximum Likelihood estimation. Univariate heritability ( $h^2$ ), shared environment ( $c^2$ ) and nonshared environment ( $e^2$ ) estimates for each factor are presented in Table 5. Results indicate moderate to high heritability for PA, NA, and EC factors. Shared environmental influences were low to moderate, and nonshared environmental influences were low for all three factors. Factor loadings and residuals for each item are presented in Table 1.

Path values and confidence intervals for the independent pathway model are shown in Fig. 1. Model fit indices included a  $-2\log$ -likelihood of 107,421.09 ( $df = 43,340$ ), Akaike's information criterion (AIC) of 20741, and Bayesian information criterion (BIC) of  $-87814$ . Individual paths weights shown in the figure are statistically reliable at  $p < .05$  when confidence intervals do not include 0. The first set of biometric factors ( $A_1$ ,  $C_1$ , and  $E_1$ ) represents the genetic and environmental influences that are common among the PA, NA, and EC factors. To provide greater clarity, genetic, shared environmental, and nonshared environmental correlations (see Table 6) as well as the bivariate heritability, or the proportions of phenotypic variance accounted for by genetic influences, as well as the proportions of phenotypic variance accounted for by shared and nonshared environmental influences were calculated (see Table 6). The proportions of variance accounted for by genes were calculated as the product of the square roots of a pair of factors and their univariate heritability estimate, over their phenotypic correlation. The common genetic factor ( $A_1$ ) indicated that there were significant genetic influences underlying the covariance among the three factors (path estimates = .69, .58, and .33, respectively). The common shared environment factor ( $C_1$ ) indicated that there were significant common shared environmental influences underlying the covariance among NA and EC (path estimates .58 and .43), but not PA (path estimate .07, non-significant). The common nonshared environment factor ( $E_1$ ) indicated that there were significant nonshared environmental influences underlying the covariance among all three factors (path estimates .23, .15, and .33).

The second set of biometric factors ( $A_2$ ,  $C_2$ , and  $E_2$ ) represents the independent genetic and environmental influences unique to the PA factor. Pathways to  $A_2$  and  $C_2$  were both significant, indicating unique genetic influences accounting for 30% ( $.55^2$ ) of variance and unique shared environmental influences accounting for 15% ( $.39^2$ ) of variance in the PA factor. The third set of biometric factors ( $A_3$ ,  $C_3$ , and  $E_3$ ) represents the independent genetic and environmental influences unique to the NA factor. The pathway to biometric factor  $A_3$  was significant, indicating unique genetic influences accounting for 52% ( $.72^2$ ) of variance in the NA factor. The final set of biometric factors ( $A_4$ ,  $C_4$ , and  $E_4$ ) represent the independent genetic and environmental influences unique to the EC factor. None of the pathways to these biometric factors were statistically significant.

## 4. Discussion

This study is the first to model simultaneously common and unique genetic and environmental influences in the three core temperament dimensions, PA, NA, and EC using an independent pathways model. The independent pathways model was selected based on evidence that PA, NA, and EC are best conceptualized as distinct temperament dimensions. Genes partially explained the covariance between these three temperament dimensions. However, the variance accounted for by common genes varied widely across temperament dimension. Nonshared environmental influences also accounted for some of the covariance between low PA, high NA, and low EC. Only NA and EC had shared environmental influences in common. The environment contributed to the pattern of NA and EC such that levels of these traits expressed in children included patterns that could be detrimental, such as high NA and low EC. Accounting for the genetic and environmental influences overlapping in PA, NA, and EC, the genetic and environmental influences unique to the dimensions were only partially consistent with past studies. We found no statistically significant unique genetic or environmental influences for EC. The lack of statistically significant genetic effects may indicate a lack of power as the path estimate was similar in magnitude to the significant path estimates of genetic effects for PA and NA. In contrast, the lack of statistically significant environmental influences may indicate that environmental effects for EC are completely overlapping with environmental influences for PA and NA as the path estimate was at zero for the shared environmental effect unique to EC. There were unique genetic effects for both PA and NA and unique shared environmental influences for PA only, which was consistent with past research.

### 4.1. Genetic influences on the covariance between temperament dimensions

In this study, genes partially accounted for the covariance among PA, NA, and EC. Only two previous studies have examined genetic and environmental influences on the covariation between multiple temperament dimensions in children (although neither examined PA, NA, and EC together). Gagne and Goldsmith (2010) found significant genetic influences accounting for the covariance between parent-reported anger (a component of NA) and EC. Deater-Deckard et al. (2007) found no significant genetic relations between EC and NA. However, they believed that their small sample size (i.e.,  $N = 259$  twin pairs) most likely accounted for the lack of a significant genetic influence. Several studies have examined the genetic influence of more general temperament/personality factors (i.e., GFP; Rushton et al., 2008;  $\alpha$  and  $\beta$  factors; Digman, 1997) in adults and children (Rushton et al., 2008; Rushton et al., 2009; Wang et al., 2013; Zawadzki & Strelau, 2010). Most have reported heritability estimates for these more general factors to be around 50%. Our study provided additional evidence that the core temperament dimensions covary, and that a common set of genetic effects help account for this covariation.

### 4.2. Environmental influences on the covariance between temperament dimensions

The covariance among PA, NA, and EC was partially accounted for by nonshared environmental influences (i.e., twins' unique experiences). These findings are consistent with past studies that have included more than one temperament dimension or that have considered temperament globally. Deater-Deckard et al. (2007) reported significant nonshared environmental influences, as indicated by a correlation of .49 between the lower-order traits of NA and EC in a model that also included conduct problems. Gagne and Goldsmith (2010) reported significant nonshared environmental influences accounting for the covariance between anger and EC across lab tasks and parent report. Researchers who have examined more general temperament/personality factors also have uncovered nonshared environmental influences (Rushton et al., 2008; Rushton et al., 2009; Wang et al., 2013). Because our study included more than just EC and NA, we can conclude that

nonshared environmental influences generalize across the core dimensions of temperament. Further, the use of latent variables instead of scale scores to measure temperament allowed us to reduce the effect of measurement error that typically confounds nonshared environmental influences (Plomin, 2011).

Shared environmental influences were only common to NA and EC. This finding was somewhat unexpected, given that most studies do not find shared environmental influences for NA or EC (e.g., Anokhin et al., 2011; Deater-Deckard et al., 2007; Rettew et al., 2006). However, most studies are also univariate studies of individual temperament dimensions. Shared environmental influences emerge when MZ twin correlations are less than twice the size of DZ twin correlations. Many univariate temperament studies using parent ratings find MZ twin correlations twice the size of DZ twin correlations, with DZ twin correlations near zero or negative (e.g., Emde et al., 1992; Goldsmith et al., 1997; Saudino, Cherny, & Plomin, 2000). These findings are often explained by contrast effects, in which parents magnify differences between twins, which favors MZ twins, given that they are presumably more alike than DZ twins (e.g., Saudino et al., 2000). Contrast effects would seem more likely to emerge in univariate twin studies of individual traits, as parents are directly comparing twins on a single trait, typically on a single measure. In contrast, cross-trait comparisons are not as straightforward and would therefore be less likely to be influenced by contrast effects. Contrast effects at the multivariate level may be especially unlikely in the current study, given that NA ratings are based on single-word adjectives whereas EC ratings are based on children's behavior in more specific situations. Other studies examining cross-trait covariances between NA and EC provide some support for the notion that cross-trait covariances are less susceptible to contrast effects. Gagne and Goldsmith (2010) reported shared environmental influences for the covariation between anger (as measured by lab tasks) and EC measured at 36 months. Wang et al. (2013) reported shared environmental influences in a sample of 3.5- to 12-year-old children for an  $\alpha$  factor, which contained items measuring NA and EC as well as agreeableness. Therefore, there appear to be important environment effects that are common to twins within a family that affect the relation between NA and EC.

Based on the covariance patterns in this and other studies, children and adolescents with high NA also tend to have low EC (e.g., Deater-Deckard et al., 2007; Musek, 2007; Zhou, Lengua, & Wang, 2009). Further, this study demonstrates that the majority of this covariance is accounted for by common shared environmental influences. This finding supports several causal models that have been proposed regarding the relations between NA and EC. For instance, it has been proposed that EC moderates the influences of harmful stimuli or situations on the development of NA through either successful or unsuccessful implementation of strategies designed to regulate behavior or emotions (e.g., Lonigan & Vasey, 2009; Tortella-Feliu, Balle, & Sesé, 2010). It also has been proposed that NA moderates the implementation of EC (Suveg, Hoffman, Zeman, & Thomassin, 2009). In this model, higher levels of NA, caused by environmental stressors, limit the ability to employ regulatory strategies (Suveg & Zeman, 2004; Suveg et al., 2009). It is possible that both theorized processes operate together to increase the association between NA and EC (Lonigan, Phillips, Wilson, & Allan, 2011).

The high covariance between high NA and low EC is problematic because this indicates that children and adolescents who are at the greatest risk for experiencing intense negative emotions are the very children who are limited in their capacity to constrain these feelings (Eisenberg et al., 2000; Muris & Ollendick, 2005; Oldehinkel, Hartman, Ferdinand, Verhulst, & Ormel, 2007). Indeed, this combination (high NA and low EC) has been implicated as a risk factor in both internalizing and externalizing behavior problems (e.g., Lonigan & Vasey, 2009; Oldehinkel et al., 2007; Valiente et al., 2003). If the findings of

shared environmental influences between EC and NA are replicated, researchers could explore environmental factors such as parenting styles that have been shown to affect EC or NA to determine if they are general factors for both temperament dimensions and whether there are intervention or prevention strategies that could ameliorate conditions leading to both high NA and low EC jointly. Behavioral genetics research may be able to aid in this identification using twin study designs aimed at explaining shared environmental variance (e.g., measured environment mediation models; see Purcell & Koenen, 2005, for a review of this approach). Although it should be noted that exploring potential shared environmental impacts may be important even in the absence of replication, as gene by environment interactions have been demonstrated in twin studies of temperament, even in the absence of shared environmental influences (Krueger, South, Johnson, & Iacono, 2008).

#### 4.3. Independent genetic and environmental influences for the temperament dimensions

Univariate analyses of the core temperament dimensions revealed genetic influences across all three dimensions. Although this finding is consistent with reported correlations in past studies, the magnitudes of these influences were elevated compared to the magnitudes of these influences in most prior studies (e.g., Deater-Deckard et al., 2007; Goldsmith et al., 1997; Saudino, 2005). In contrast to the findings of previous studies (e.g., Goldsmith et al., 1997; Spengler et al., 2012), there were shared environmental influences present for NA and EC. Finally, nonshared environmental influences, smaller in magnitude than those reported in past studies (e.g., Deater-Deckard et al., 2007; Goldsmith et al., 1997), were found across PA, NA, and EC. The use of latent variables in this study may account for the higher than normal genetic influences as well as the findings of shared environmental influences for NA and EC. In twin studies not involving latent variables, the error variance is accounted for in the nonshared environmental estimate. However, when biometric models are conducted using latent variable analysis, the residuals of the manifest variables instead capture error variance, meaning that the proportion of variance accounted for by genetic, shared environmental, and nonshared environmental influences are less biased by error. Therefore, our study may provide a more accurate picture of the univariate genetic and environmental estimates than prior twin studies that did not use latent variable modeling.

Accounting for the genetic and environmental influences shared with PA and NA, there were no statistically significant unique genetic or environmental influences affecting EC. However, similar to Deater-Deckard et al. (2007), the lack of a significant path estimate for the unique genetic influence of EC ( $h^2 = .64$ ) is likely because of a lack of power to detect a significant pathway, given the high path estimate. This may have occurred because EC was not measured as well as the other constructs, as evidenced by the variability in factor loadings, ranging from .18 to .83. In contrast, the lack of unique environmental influences on EC does not appear to be a power issue, given that the point estimate for both  $c^2$  and  $e^2$  were zero. This finding is consistent with theories that consider a defining feature of EC to be its role as the modulating dimension of temperament (e.g., Rothbart & Bates, 2006). Further, there may be important implications for future EC research in that there appear to be no environmental risk factors for EC that are independent of those factors that also affect PA and NA. Given that PA and NA are identified earlier than EC (Rothbart & Bates, 2006), researchers may be able to identify and ameliorate environmental risk factors that affect PA and NA and in turn influence the development of EC.

There were unique genetic components for PA and NA, and unique shared environmental influences for PA. The findings of unique genetic estimates for PA and NA are consistent with findings in past behavioral genetics studies (e.g., Mullineaux et al., 2009). The finding of unique shared environmental influence on PA is consistent with past research, for which the preponderance of evidence suggests that if there are unique shared environmental

influences in temperament, they lie in the PA domain (e.g., Isen et al., 2009; Wood et al., 2007; see Saudino, 2005 for a review).

The unique shared environment influence on PA provides an interesting avenue for future research, as this finding suggests that there may be familial factors that increase children's similarity in their level of PA. Researchers have suggested that high levels of PA may be the defining feature in distinguishing between individuals who are modestly capable in coping with the multitude of stressors that accompany everyday life versus individuals who are particularly adept at dealing with these common stressors (e.g., Shiner, 2000; Shiner, Masten, & Tellegen, 2002). Our findings suggest that there are aspects of the environment that affect children's level of PA independent of the impact these factors have on children's other temperament characteristics. This study also provides an important clue for identifying environmental variables affecting PA, as some environmental variables should be unique to PA and should influence children within the family unit.

#### 4.4. Limitations

There were several limitations of the current study to consider when interpreting results. Parent ratings were used to assess temperament. There are assumptions that underlie behavioral genetics studies, such as MZ twins sharing 100% of their genes and DZ twins sharing 50% of their genes, and that these percentages are unvarying across the lifespan. Epigenetic and other post-genomic research (e.g., Charney, 2012) is being conducted that may limit the tenability of these assumptions. The use of parent reports could bias results by increasing or decreasing shared environmental influences as well as increasing genetic influences (e.g., Burt, 2009; Saudino et al., 2000). The inclusion of different or multiple ratings of temperament such as child self-report or teacher-report may have led to different etiological estimates. However, agreement among raters is typically only modest (Achenbach, McConaughy, & Howell, 1987), which limits the amount of variance explained when multiple ratings are combined (Burt, McGue, Krueger, & Iacono, 2005). Further, child reports can be problematically biased themselves, because of the increased error and unreliability present in child reports (Burt, 2009). Future research using different reports (i.e., teacher-report, self-report) should be conducted to examine whether the findings obtained in this study are robust to rater effects. Although the sample in this study was equally distributed across gender and was racially and economically diverse, the sample size was not large enough to examine the potential influences of important moderators such as socioeconomic status and gender. Finally, whereas we analyzed the etiology of three core temperament dimensions, there are other suggested models of temperament that include other dimensions that may be important. For example, the PANAS, which we used to assess PA and NA is concerned with measuring positive and negative emotions using one-word adjectives. Other measures of temperament include descriptions of common behaviors, such as behaviors characteristic of approach when measuring PA and behaviors characteristic of withdrawal when measuring NA (e.g., Buss & Plomin, 1984; Rothbart & Bates, 2006). Future research should explore whether the same etiological patterns obtained in this study emerge when different measures are used.

#### 5. Conclusions

This study was the first to examine the genetic and environmental influences common to three core temperament dimensions, PA, NA, and EC. Our findings that genetic influences accounted for variable, and in the case of NA, small amounts of the variance within these dimensions indicates that common genes may be more influential for some temperament dimensions than for other temperament dimensions. Therefore, examining temperament as a global factor may lead to a loss of important information. Given the negative sequelae associated with the combination of high NA and low EC, the finding of common shared

environmental influences suggests that identifying what environmental factors account for this covariance is integral for more effective intervention and prevention efforts. For PA, there appear to be unique environmental influences that could potentially be targeted as protective factors.

## Acknowledgments

Preparation of this work was supported, in part, by grants from the National Institute of Child Health and Human Development (P50 HD052120) and the Institute of Education Sciences, U.S. Department of Education (R305B04074). Views expressed herein are those of the authors and have neither been reviewed nor approved by the granting agencies.

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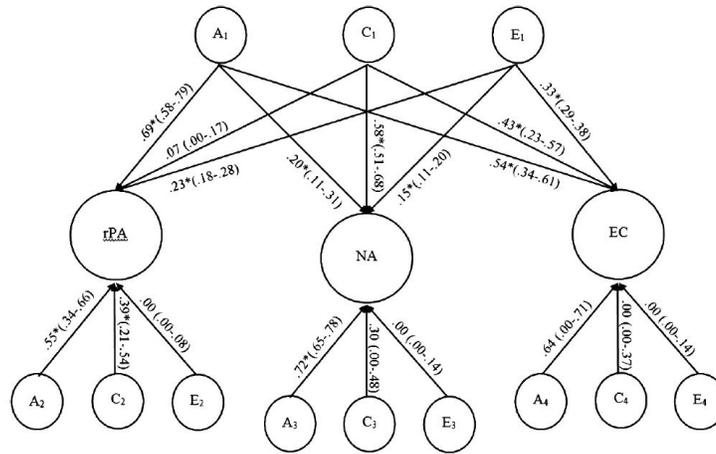
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**Fig. 1.** Independent pathway model representing Positive Affect, Negative Affect, and Effortful Control factors. Standardized path coefficients (and 95% confidence intervals) are provided. The rPA (reversed Positive Affect) factor is comprised of the positive affect items from the PANAS. The NA (Negative Affect) factor is comprised of the negative affect items on the PANAS. The EC (Effortful Control) factor is comprised of the persistence/low distractibility scale on the ECS. A = additive genetic influence; C = shared environmental influence; E = nonshared environmental influence.

Table 1

Item descriptions, intraclass correlations, factor loadings, and residuals.

Item description	MZ	DZ	Loading [95% CI]	Residual [95% CI]
Positive Affect	.84*	.48*		
interested	.73*	.40*	.65 [.63-.68]	.57 [.53-.61]
excited	.81*	.51*	.56 [.51-.60]	.69 [.64-.74]
strong	.66*	.30*	.48 [.45-.52]	.77 [.73-.81]
enthusiastic	.79*	.45*	.67 [.63-.68]	.56 [.52-.60]
proud	.86*	.64*	.53 [.52-.55]	.72 [.69-.74]
alert	.79*	.49*	.59 [.56-.60]	.65 [.60-.67]
inspired	.84*	.64*	.69 [.67-.71]	.53 [.49-.57]
determined	.70*	.30*	.70 [.68-.73]	.51 [.47-.56]
attentive	.73*	.30*	.65 [.64-.68]	.58 [.54-.62]
active	.84*	.40*	.48 [.45-.51]	.77 [.73-.82]
Negative Affect	.84*	.60*		
distressed	.66*	.43*	.57 [.55-.59]	.67 [.67-.70]
upset	.77*	.46*	.68 [.66-.70]	.54 [.50-.57]
guilty	.78*	.60*	.57 [.53-.59]	.67 [.66-.70]
scared	.73*	.51*	.67 [.64-.68]	.55 [.51-.58]
hostile	.76*	.44*	.56 [.54-.58]	.69 [.67-.71]
irritable	.71*	.35*	.61 [.59-.63]	.62 [.60-.65]
ashamed	.84*	.67*	.58 [.54-.62]	.67 [.63-.70]
nervous	.70*	.45*	.66 [.62-.68]	.57 [.54-.61]
jittery	.82*	.59*	.50 [.47-.52]	.75 [.70-.79]
afraid	.76*	.55*	.68 [.67-.70]	.54 [.51-.59]
Effortful control	.80*	.52*		
does not complete homework	.86*	.67*	.38 [.37-.41]	.85 [.84-.88]
difficulty completing assignments	.70*	.39*	.60 [.58-.62]	.64 [.60-.69]
hard to enjoy something else when doesn't get what s/he wants	.84*	.57*	.45 [.41-.47]	.80 [.78-.82]
has a hard time following instructions	.81*	.52*	.69 [.66-.71]	.52 [.50-.57]
gives up when activity or task is difficult	.60*	.30*	.65 [.61-.68]	.58 [.53-.63]
moves from one task to another without completing any of them	.72*	.48*	.75 [.74-.77]	.44 [.41-.47]
is distracted by little things	.75*	.41*	.70 [.68-.72]	.51 [.46-.53]
leaves projects or tasks unfinished	.80*	.55*	.83 [.81-.85]	.31 [.29-.34]
has a hard time concentrating on work because thinking about other things	.69*	.43*	.79 [.76-.80]	.38 [.35-.41]
starts many things that s/he doesn't finish	.80*	.59*	.80 [.79-.81]	.36 [.32-.38]

Item description	MZ	DZ	Loading [95% CI]	Residual [95% CI]
often gets lost in work	.84*	.59*	.18 [.16-.20]	.97 [.94-.97]
quits when frustrated with projects or tasks	.69*	.45*	.69 [.66-.71]	.52 [.49-.56]

\*  $p < .01$ .

**Table 3**

Correlations among Positive Affect, Negative Affect, and effortful control factors.

	Positive Affect	Negative Affect
Positive Affect	-	
Negative Affect	-.23*	-
Effortful control	-.49*	.42*

Higher scores on the effortful control scale reflect lower levels of effortful control.

\*  $p < .01$ .

**Table 2**

Descriptive statistics for Positive Affect, Negative Affect, and effortful control scales.

<b>Variable</b>	<b>Mean</b>	<b>SD</b>	<b>Skew</b>	<b>Kurtosis</b>
Positive Affect	39.13	6.32	-0.48	-.10
Negative Affect	18.23	6.94	1.06	.94
Effortful control	25.83	8.46	0.79	.53

Higher scores on the effortful control scale reflect lower levels of effortful control.

**Table 4**

Confirmatory factor analyses of one-, two-, and three-factor models of temperament.

Model	Y-B $\chi^2$	df	CFI	RMSEA	BIC	$\Delta \chi^2$
One-factor	5005.24	464	.54	.08	114645	1185.72***
Two-factor	3646.11	463	.68	.07	112369	502.87***
Three-factor	2091.67	461	.83	.05	109743	-

A significant  $\Delta \chi^2$  indicates that the model fit worse than the nested three-factor model comprising separate PA, NA, and EC factors.

\*\*\*  
p<.001.

**Table 5**

Univariate estimates for Positive Affect, Negative Affect, and effortful control factors [with 95% confidence intervals].

	$h^2$	$c^2$	$e^2$
Positive Affect	.79 [.63-.96]	.16 [.00-.28]	.05 [.03-.07]
Negative Affect	.56 [.42-.71]	.42 [.29-.55]	.03 [.01-.04]
Effortful control	.71 [.62-.81]	.18 [.06-.31]	.11 [.08-.14]

*Note.*  $h^2$  = univariate genetic;  $c^2$  = univariate shared environment;  $e^2$  = univariate nonshared environment.

**Table 6**

Genetic, shared environment, and nonshared environment correlations among Positive Affect, Negative Affect, and effortful control factors.

	<u>Biometric correlations</u>		<u>Bivariate heritability</u>	
	Positive Affect	Negative Affect	Positive Affect	Negative Affect
<i>Genetic</i>				
Positive Affect	-		-	
Negative Affect	.21	-	.66	-
Effortful control	.51	.17	.79	.26
<i>Shared environment</i>				
Positive Affect	-		-	
Negative Affect	.15	-	.17	-
Effortful Control	.16	.89	.06	.60
<i>Nonshared environment</i>				
Positive Affect	-		-	
Negative Affect	1.0	-	.18	-
Effortful control	1.0	1.0	.15	.14

Higher scores on the Effortful Control scale reflect lower levels of effortful control. The Positive Affect scale was reverse-coded for biometric analysis such that higher scores reflect lower levels of positive affect (i.e., a positive correlation between Positive Affect and Negative Affect indicates that individuals with less positive affect have more Negative Affect).