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Parent-and Observer-Rated Positive Affect in Early Childhood: Genetic Overlap and Environmental Specificity

Megan Flom*, Manjie Wang*, Kendra J. Uccello, and Kimberly J. Saudino

Department of Psychological & Brain Sciences Boston University Boston, MA

Abstract

The sources of individual differences in both observed and parent-rated positive affect (PA) were examined in a sample of 304 3-year-old twin pairs (140 MZ, 164 DZ). Based on model-fitting analyses, individual differences in observed PA were attributed to moderate genetic and high nonshared environmental factors, but not shared environmental factors. In contrast, shared environmental effects accounted for over half of the variance in parent-rated PA and genetic and nonshared environmental effects were more modest. The genetic correlation across the two measures was high, indicating substantial overlap between genetic factors influencing the two. It was these overlapping genetic effects that fully explained the phenotypic correlation between both measures. There was no significant covariance between the environmental influences on parent rated and observed PA. Thus, the two measures of PA in early childhood have common genetic underpinnings, whereas environmental influences are measure-specific. Measurement implications are discussed.

Keywords

positive affect; observer-rated; parent-rated; genetics; temperament; early childhood

Introduction

Temperamental positive affect (PA) refers to a biologically-based tendency to experience positive emotions (Watson & Naragon, 2009). Children with higher levels of PA are more inclined to experience and express cheerful emotions. PA plays an important role in promoting favorable developmental outcomes, and is a resilience factor for psychological problems (Carr, 2011). Hence, understanding the factors that contribute to individual differences in PA in early childhood may provide insight on the early malleability of such protective characteristics.

Corresponding author address: Megan Flom, mflom@bu.edu; 617-353-1102, 64 Cummington, Mall, Room 100 Boston, MA, 02215.

*Joint first authors presented alphabetically

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Ethical approval: All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

Informed consent: Informed consent was obtained from all individual participants included in the study.

Behavioral genetics research, such as twin studies, are a valuable tool for informing on genetic and environmental sources of individual differences in behavior. Twin studies of PA in infancy and early childhood yield an interesting pattern of results. In contrast to most temperament dimensions, which typically show moderate genetic effects and little or no shared environmental influences, the pattern is reversed for early PA (see Saudino & Wang, 2012 for a review). In infancy and early childhood, the heritability of PA is often low or near zero and shared environmental influences (i.e., environmental influences or experiences that are shared by family members and enhance familial resemblances, such as family demographics, one's rearing neighborhood, shared friends, family resources) account for between 30 to 79% of the variance (Cohen, Dibble, & Grawe, 1977; Goldsmith, Lemery, Buss, & Campos, 1999; Goldsmith, Buss, & Lemery, 1997; Saudino, 2005). That is, in very young children familial resemblance in PA is largely due to family-wide shared environmental factors—not shared genes.

However, in middle childhood and beyond, the preponderance of evidence suggests that PA is moderately to highly heritable (.33-.79) and the influence of shared environments is negligible (Allan, Mikolajewski, Lonigan, Hart, & Taylor, 2014; Bartels & Boomsma, 2009; De Neve, Christakis, Fowler, & Frey, 2012; Lykken & Tellegen, 1996; Mullineaux, Deater-Deckard, Petrill, Thompson, & DeThorne, 2009; Nes, Røysamb, Tambs, Harris, & Reichborn-Kjennerud, 2006; Stubbe, Posthuma, Boomsma, & De Geus, 2005). An exception to this pattern is state-like measures of self-reported positive mood averaged over short intervals (e.g., momentary ratings using the experience sampling method, 15-minute epochs, or multiple days), which demonstrate little to no genetic effects in adolescents and adults (Jacobs et al., 2013; Menne-Lothmann et al., 2012; Riemann, Angleitner, Borkenau, & Eid, 1998; Zheng, Plomin, & von Stumm, 2016). Most of the variance in these measures of current positive mood is due to nonshared environmental influences (i.e., experiences that are unique to individuals within a family such as differential parental treatment, accidents, illness and measurement error), (Menne-Lothmann et al., 2012; Zheng et al., 2016). Thus, sources of individual differences in PA may differ when conceptualized as a trait or a state, nonetheless, the bulk of evidence suggests that PA as a trait tends to be substantially heritable in older samples.

The difference in the magnitude of genetic and shared environmental effects across ages might indicate developmental change in the factors that influence PA as a trait. For example, shared environmental influences in early, but not later childhood or beyond, may reflect the influence of maternal personality and attachment security on PA in young children (Goldsmith, Lemery, et al., 1999). However, another potential explanation may have to do with the measurement of PA in young children. Twin studies of PA in infancy and early childhood rely predominately on parent ratings. It is possible that the shared environmental influences found in younger samples are a result of parents' difficulties in discriminating between PA of their twins. This may be due in part to the fact that younger twins are more likely to spend time together and may mutually influence each other's PA. If this is the case, shared environmental influences may not emerge with more objective measures of PA—particularly when the twins are assessed individually. Indeed, in a recent study of infant twins, the heritability of observed PA was .42 and .55 at 6 and 12 months of age,

respectively; whereas estimates of genetic influence for parent-reported PA were no higher than .18 (Planalp, Van Hulle, Lemery-Chalfant, & Goldsmith, 2017).

To our knowledge, there are no prior studies exploring whether parent ratings and objective measures tap the same underlying genetic and environmental sources of individual differences in PA in early childhood. The aforementioned infant study looked at genetic and environmental influences on parent-reported and observed PA in the first year of life, but did not examine sources of *covariance* between the two measures (Planalp et al., 2017).

Research of other temperament dimensions, such as activity level, inhibitory control, and shyness, has found that parent ratings and lab-based measures of the same dimension can tap different genetic and environmental effects (Gagne & Saudino, 2010; Saudino, 2009; Smith et al., 2012). It is possible, therefore, that such measure-specific effects may also emerge for PA. The present study aimed to address two novel questions. First, would a more objective measure of PA (i.e., behavioral observations) in early childhood also show shared environmental effects? Second, to what extent do the same genetic and environmental effects influence individual differences on behavioral observations and parent ratings of PA?

Method

Sample

The Boston University Twin Project sample was recruited from birth records supplied by the Massachusetts Registry of Vital Records. As is standard for twin research, twins were selected preferentially for higher birth weight and gestational age. No twins with birth weights less than 1,750 g or with gestational ages less than 34 weeks were included in the study. The sample included 304 same-sex twin pairs (140 Monozygotic [MZ] 51% male, and 164 Dizygotic [DZ] 55% male; mean age=3.05 years, $SD=.08$, Range=2.91-3.28 years). Ethnicity was generally representative of the Massachusetts population (85.4% Caucasian, 3.2% Black, 2% Asian, 7.3% Mixed, 2.2% Other). Socioeconomic status, according to the Hollingshead Four Factor Index (Hollingshead, 1975), ranged from low to upper middle class (range=20.5-66; $M=50.9$, $SD=14.1$). Zygosity was determined via DNA analyses using DNA obtained from cheek swab samples. In the cases where DNA was not available ($n=3$), zygosity was determined using parents' responses on physical similarity questionnaires which have been shown to be more than 95% accurate when compared to DNA markers (Price et al., 2000).

Procedure

All procedures were approved by the Boston University Institutional Review Board and informed consent was obtained for all participants. Within approximately 2 weeks of their third birthday, twins completed two 1.5-hour laboratory visits scheduled approximately 48 hours apart. During both visits, each member of the twin pair was assessed separately in either a test or a play situation. On the first day, one of the twins participated in a standardized test situation, in which the Mental Scale of the Bayley Scales of Infant Development-Second Edition (Bayley, 1993) was administered. Meanwhile, the other twin participated in a laboratory play situation in which episodes from the Laboratory Temperament Assessment Battery - Preschool Version (Lab-TAB; Goldsmith, Reilly,

Lemery, Longley, & Prescott, 1999) and three imitation tasks (Fenstermacher & Saudino, 2007) were administered. At the second visit, the situations were reversed for each twin. Each twin within a family was assessed by a different tester. Although testers administered tasks to the twins, they did not provide ratings of PA. Behavioral ratings of observed PA were obtained from videotaped data using raters who were specifically trained to code for PA (see below). As with testing, twins within a family were rated by different raters. The use of separate testers and raters for each twin within a twin pair ensured that twin resemblances for observed PA was not due to shared tester/rater variance (i.e., tester effects or rater bias would not contribute to estimates of shared environment). Parents (94% mothers) completed questionnaires on each twin's behavior and temperament.

Measures

Observational Measure of PA.—An observational measure of PA was obtained by coding the frequency of smiles and/or laughter during three episodes (i.e., “Dinky toys”, “Snack delay”, and “Gift”) from the Lab-TAB. In “Dinky toys” the child is given two chances to choose a toy from a container of approximately 20 toys (untimed); in “Snack delay” the child is told to wait to eat an M&M candy placed under a clear plastic cup for six trials of different lengths (20s, 30s, 0s, 40s, 10s, 60s); and in “Gift” the child is given a gift and told to wait for the tester to finish their work (2 minutes) before they open it (see Gagne & Saudino, 2010). Smiles and/or laughter were rated as either 0 (behavior absent) or 1 (behavior present) for a total of 24 possible behavioral segments. PA coding segments for Dinky toys included the two instruction phases as well as two choice trials ($n=4$); Snack Delay included a practice trial in addition the six test trials ($n=7$); and Gift segments were based on the number of timed 10 sec intervals before opening ($n=13$). To adjust for variations in total number of behavioral segments during which PA was observed (e.g., not all children waited the full time period before opening the gift), a proportional measure of PA was calculated as the percentage of observed segments in which the child either smiled or laughed. This proportion was calculated only when children had more than 50% of the segments within a given episode (over 97% of the sample had data for all segments within an episode). An overall PA composite was created by averaging the proportion scores from the three episodes. Reliability coding was completed for 10% of the sample and the intraclass correlation between raters was .78.

Parent Rating Measure of PA.—The Pleasure subscale of the Toddler Behavior Assessment Questionnaire (TBAQ; Goldsmith, 1996) provided a parent-rating measure of PA. This subscale consists of 10 questions regarding the child's frequency of smiling, laughing, or squealing with joy in specific situations (e.g., playing with favorite toys). Parents indicated on a 7-point scale how frequently the child demonstrated the behavior during the previous month (1 = “Never” to 7 = “Always”). In our sample, internal consistency, as indicated by Cronbach's alpha, was .75.

Twin Design—The twin method involves comparing genetically identical (MZ) twins with fraternal (DZ) twins who share approximately 50% of their segregating genes. Genetic influences are implied when cotwin similarity covaries with the degree of genetic relatedness. Intraclass correlations typically serve as indices of cotwin similarity. When MZ

twins are more similar to one another than DZ twins, genetic influences are implied. To evaluate genetic and environmental contributions to the phenotypic correlation between observed and parent-rated PA, cross-twin cross-variable correlations were calculated. The cross-twin cross-variable correlation involves correlating Twin A's observed PA score with Twin B's parent-rated PA score, and vice versa. An MZ cross-twin correlation greater than the DZ cross-twin correlation implies genetic contributions to *covariance* between the measures. Because twin correlations can be inflated by variance due to gender (McGue & Bouchard, 1984), all scores were residualized for gender effects.

Model-Fitting Analyses—To explore genetic and environmental sources of variance and covariance, a bivariate Cholesky model (Neale & Cardon, 1992; Saudino, 2012) was fit to the data¹. The model as depicted in Figure 1, includes additive genetic (A), shared environmental (C) and nonshared environmental (E) variances that are common to both PA measures; and effects that are unique to observed PA (a, c, e). We entered observed PA last in the model because we were interested in seeing if it provided any additional information above and beyond parent ratings; however, the ordering of variables does not affect estimates of variance and covariance. To evaluate for the possibility of non-additive genetic effects (D), we also fit ADE models to the data. Models were fit to raw data using a maximum likelihood pedigree approach implemented in Mx structural equation modeling software (Neale, Boker, Xie, & Maes, 2006). This approach allows the inclusion of participants with incomplete data.

Using this model, we are able to estimate the magnitude of genetic and environmental contributions (and their 95% confidence intervals [CI]) to the variances of each PA measure. In addition, this model estimates genetic and environmental correlations (and 95% CI) between measures. The genetic correlation (r_g) indicates the extent to which genetic effects on parent-reported PA covary with genetic effects on observed PA, *independent of the heritability of each measure*. The genetic factors that influence two measures (or one measure across time) can covary perfectly even if the genetic factors on each measure contribute only slightly to the phenotypic variance. Thus, r_g can be 1.0 even when the heritability of each measure is modest. Conversely, two measures may be substantially heritable, but the genetic correlation would be zero if the genetic effects on the two measures do not overlap. Similar logic applies to shared (r_c) and nonshared (r_e) environmental correlations between PA measures.

The overall fit of a model was assessed by calculating twice the difference between the negative log-likelihood (−2LL) of the model and that of a saturated model (i.e., a model in which the variance/covariance structure is not estimated and all variances and covariances for MZ and DZ twins are estimated). The difference in −2LL is asymptotically distributed as χ^2 with degrees of freedom equal to the difference in the number of parameters in the full model and that in the saturated model. In addition to the full model estimating all genetic and environmental sources of variance and covariance, a reduced model dropping all non-

¹Rater bias models were not fit to the data because they require that both twins within a twin pair be rated by the *same* rater (Saudino, 2017). As indicated above, by design, the study used separate raters and testers to ensure that rater/tester covariance did not inflate estimates of shared environment for our observed measure.

significant paths in the full model was fit to the data. The fit of this more parsimonious reduced model was compared to that of the full model.

Results

Descriptive Statistics

Table I lists means and standard deviations for the PA measures by gender and zygosity. We evaluated mean differences for gender and zygosity using generalized estimating equations (GEE) implemented in the SAS GENMOD procedure to account for dependence in the data due to the fact that our sample was comprised of pairs of twins. GEE are an extension of the standard generalized linear models that allow modeling of correlated data (Liang & Zeger, 1986; Zeger & Liang, 1986). For both measures of PA, the main effects of gender and zygosity, and the interaction between gender and zygosity, were nonsignificant.

Correlations

Observed PA was modestly correlated with the parent-rated PA ($r = .18$, CIs: .09, .26). As seen in Table II, the MZ intraclass correlations exceeded those for DZ twins for both PA measures, suggesting genetic influences on each. The DZ correlation for observed PA was less than one-half the MZ correlation suggesting possible non-additive genetic effects. For parent-rated PA, DZ correlations exceeded one-half the MZ correlation, implying shared environmental effects on this measure. The cross-twin cross-variable correlation was higher for MZ than DZ twins, indicating that genetic influences may contribute to the phenotypic correlation between the two measures. The relatively lower cross correlation for DZ twins indicated potential non-additive genetic sources of covariance.

Model-Fitting Analyses

Model-fit statistics are presented in Table III. As indicated by the lower AIC, the ACE model fit the data better than the ADE model. Furthermore, D could be dropped from the model without a significant decrement in fit. For the ACE model, neither all A nor all C could be dropped from the full model without worsening fit. However, it was possible to further reduce the ACE model by dropping all nonsignificant paths (i.e., all shared environmental variance on observed PA, unique genetic variance on observed PA, and nonshared environmental covariance) without a significant decrement in fit (see Figure 1). Although the reduced model provides a more parsimonious fit to the data, to avoid artificial inflation of parameter estimates (i.e., in reduced models, nonsignificant shared environmental variance is absorbed into genetic variance), we focus on the estimates from the full model.

As seen in Table IV, both measures of PA were moderately heritable, with genetic factors explaining 26% (CI: 5%-47%) of the variance in parent-reported PA and 33% (CI: 10%-46%) of the variance in observed PA (i.e., .18 shared with parent report + .15 unique to observed = .33). The nonshared environment explained the remaining variance in observed PA, whereas parent-reported PA was also influenced by both shared environmental (47%) and nonshared environmental (28%) factors. The majority of genetic variance (59%) for observed PA was shared with parent-reported PA, and it is these overlapping genetic effects that fully explain the phenotypic correlation between the two. The genetic correlation

indexing the degree to which the genetic effects covary across methods was .74 (CI: .07-1.00). Environmental factors did not significantly contribute to the relation between parent-reported and observed PA. The nonshared environment, though influencing both PA measures, was measure-specific (i.e., the nonshared environmental factors contributing to observed PA are different than those influencing parent-reported PA).

Discussion

This research establishes both etiologic similarities and differences between observed and parent-reported PA at age 3. Both methods were heritable and there was significant and substantial genetic covariance across methods. The modest phenotypic correlation between the two methods was fully due to these common genetic effects. Said another way, any agreement between parent-reported and observed PA was due to the fact that they share common genetic underpinnings. The two methods differ in terms of environmental effects. Parent-reported PA showed substantial family-wide shared environmental influences, but this was not the case for observed PA. The environments that influenced observed PA were of the nonshared variety.

As with other research using parent-reports in infancy and early childhood, the heritability of PA in our young sample was modest (Goldsmith, Lemery, et al., 1999; Goldsmith et al., 1997). In addition to replicating prior findings with parent reports, we extend this in two important ways. First, by providing novel support for the heritability of an observed measure of PA in early childhood. Second, by exploring the etiologic overlap between reported and observed PA. Despite operationalizing PA differently (global trait versus specific behaviors) and assessing PA in different contexts (home versus lab), we find that these methodological differences do not result in different genetic effects.

Consistent with other twin studies of PA in early childhood, our measure of parent-reported PA was significantly influenced by shared environmental factors (Cohen et al., 1977; Goldsmith, Lemery, et al., 1999; Goldsmith et al., 1997; Saudino, 2005). In fact, twin resemblance for parent-rated PA was due more to shared familial environments than to shared genes. As indicated earlier, shared environmental influences do not emerge for PA in older children (e.g., Allan et al., 2014; Mullineaux et al., 2009) and it has been suggested that the shared environment in early childhood may be a function of maternal personality and attachment security shaping early PA (Goldsmith, Lemery, et al., 1999). Given that we find no evidence of shared environmental influences for observed PA in the same children, we suggest that this has something to do with parent reports of PA in early childhood.

Because parent-reports of temperament use the same rater to assess both co-twins, it is possible that the shared environmental effects, and higher correlations for both MZ and DZ twins, reflect rater variance (Saudino, 2017). However, this would be the case whenever you have a parent rating both members of a twin pair, yet within the temperament literature the pattern of substantial shared environmental variance is unique for PA during early childhood (i.e., parent ratings of most other temperament dimensions show little evidence of shared environmental effects). Conversely, the twins in our study were assessed by different testers on different days, which could result in differences in PA between twins. This could explain

the larger influence of nonshared environmental experiences for observed PA, such that certain testers may elicit more positivity than others (tester effect) and/or that the twins could be in a better mood on one testing day or perhaps be more comfortable and thus happier in the lab on the second test day (testing day effect). We explored this by regressing out tester and test day variance from observed PA, and found that both tester and testing day combined accounted for less than 1% of the total variance, making this an unlikely explanation for the greater impact of the nonshared environment on this measure. Instead, the substantial nonshared environmental effects on observed PA likely reflects the importance of the twins' unique experiences in their expression of happiness when assessed individually in the lab. Although real-time measures of PA in adults have also found substantial nonshared environmental variance, in contrast to our observed measure, these studies do not yield evidence of genetic influences (Jacobs et al., 2013; Menne-Lothmann et al., 2012; Riemann et al., 1998; Zheng et al., 2016). Real-time measures may reflect momentary state-like effects and hence high estimates of nonshared environmental variance, but the significant genetic influences for our observed measure and the genetic covariance with parent-rated PA suggests that our observed measure is tapping PA more as a trait.

Given that shared rater variance is an unlikely explanation for the high shared environmental effects for parent-reported PA, parent ratings of PA in young twins might instead reflect contagion effects. That is, one twin's positive moods can influence the mood of his/her co-twin. In this case, parent reports of PA would be accurate, and the shared environmental influences would be a result of young twins' PAs influencing each other. Twins in our lab, on the other hand, were assessed individually, making contagion effects less likely to occur for our observed measure. There are hints of possible contagion effects on early PA from the MacArthur Longitudinal Twin Study (Emde et al., 1992; Plomin et al., 1993). Modest estimates of shared environmental influences were found for an observed measure of PA that combined episodes where twins were both together and apart; however, despite heritability estimates in the same range as our study, neither genetic nor environmental influences were significant. Though there may be an effect of emotion contagion on PA when twins are together, this concept could also apply to negative affect, yet greater familial similarity and shared environmental influences are rarely found in negative affect—even in young children (for review see Saudino, 2005). Given this, it is possible that emotion contagion is specific to PA (i.e., “laughter is contagious” and siblings are influenced by one another's positive affect, but may be more immune or less receptive to the other's negative affect). Though emotion contagion for PA may occur throughout development, it may be more evident in parent-reports of PA in early childhood because young co-twins spend more time together (i.e., there are more opportunities for twins to influence each other). Research that compares genetic and environmental sources of variance of observed PA for the same twins assessed both apart and together is needed to add resolution to the question of emotion contagion for PA.

It is also possible that there may be a parental bias when rating the positive affect of their young children. That is, parents may want view their children as equally happy. This bias may have a lesser effect in the ratings of PA for older children as parents gain a broader understanding of their children's unique temperaments in multiple contexts. However, we were not able to apply rater bias models to the data because they require two raters each

rating the same children, which was not possible given that we had separate raters for each twin in the lab. Indeed, this was part of our design in an effort to address concerns about rater variance contributing to twin resemblance.

The limitations of this study should be acknowledged. First, our construct of observed PA included only frequency of smiling and laughing. Latency to smiling, intensity of affect, and duration were not included. Whether the same etiologic pattern would be found for these aspects of positive affect remains an empirical question. Replication in early childhood and beyond is necessary. Second, twin studies do not identify specific genes or environments, rather, they inform on the relative impact of each on variation in behavior. Identifying specific genes or environments relevant to PA is important in future work, particularly given that PA may be a buffer against psychopathology (e.g., Bartels, Cacioppo, van Beijsterveldt, & Boomsma, 2013; Wang & Saudino, 2015). Finally, our sample of just over 300 twin pairs is relatively small and consequently some confidence intervals are broad. For this reason, we focus on the overall pattern of results, rather than specific parameter estimates.

This study is the first to demonstrate genetic overlap between parent-reported and observed PA, as well as environmental specificity between the two. Parent-reported, but not observed PA, is influenced by the shared environment whereas observed PA is substantially influenced by nonshared environmental effects. These results, taken in the context of the literature, suggest the potential influence of parental bias in reporting positive emotions of young children, emotion contagion that may be particularly salient in early parent-reported PA, or both. Future research should consider methodological effects when measuring PA in early childhood and beyond, and continue to investigate how and why reported versus real-time PA overlap and differ from one another.

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Figure 1.

Bivariate Cholesky Full Model

Note. Significant path estimates are indicated in bold. 95% confidence intervals are presented in parentheses.

Table 1.

Means by Gender and Zygosity

PA Measure	Males		Females		Effect Size *	
	MZ	DZ	MZ	DZ	Gender	Gender x Zygosity
Parent-reported	55.84 (6.72)	56.40 (7.35)	56.59 (6.52)	55.70 (7.33)	.94	.87
<i>n</i>	135	175	135	141		
Observed	.34 (.24)	.37 (.22)	.39 (.23)	.40 (.22)	.81	.55
<i>n</i>	142	175	138	149		

Note. MZ=monozygotic; DZ=dizygotic.

* Cohen's d statistic.

Table II.

Twin Intraclass and Cross-Age Correlations (and 95% confidence intervals)

	MZ	DZ
Parent-reported PA	.72 (.64, .79)	.59 (.49, .67)
Observed PA	.37 (.23, .49)	.08 (−.08, .24)
Cross-twin cross-measure	.15 (.05, .25)	.01 (−.09, .12)

Table III.

Model-Fitting Results

	Overall Fit of Model ^a					Relative Fit of Model ^b		
	-2LL	df	χ^2	df	p	AIC	RMSEA	χ^2 df p
Saturated Model	3651.679	1162						
Full ACE Model	3658.894	1173	7.215	11	.781	-14.785	0	
AE Model	3678.421	1176	26.742	14	.021	-1.258	.054	19.527 3 .000
CE Model	3703.072	1176	51.393	14	.000	23.393	.092	44.178 3 .000
Best Fit Model ^c	3662.960	1177	11.281	15	.732	-18.719	0	4.067 4 .397
Full ADE Model	3676.803	1173	25.124	11	.009	3.124	.064	
AE Model	3678.431	1176	26.742	14	.021	-1.258	.054	1.628 3 .653
DE Model	3703.072	1176	51.393	14	.000	23.393	.092	26.268 3 .000

Note. -2LL=Likelihood Statistic. χ^2 =Chi-square fit statistic. df=Degrees of freedom. AIC=Akaike's Information Criterion.

^aOverall fit of the model is determined by the difference in -2LL of the model and that of a saturated model.

^bRelative fit of the model determined by the χ difference (χ^2) between full bivariate ACE model and reduced model.

^cBest Fit Model dropped all nonsignificant paths from the full model: all shared environmental variance on observed PA, unique genetic variance on observed PA, and nonshared environmental covariance.

Table IV.
Genetic and Environmental Variance Components (and 95% Confidence Intervals) From Full Model

	Overall Variance			Shared Variance			Unique Variance		
	a ²	c ²	e ²	a ²	c ²	e ²	a ²	c ²	e ²
Parent-reported PA	.26 (.05, .47)	.47 (.27, .63)	.28 (.21, .36)						
Observed PA	.33 (.10, .46)	.01 (.00, .19)	.65 (.53, .79)	.18 (.001, .44)	.01 (-.10, .02)	.00 (-.00, .04)	.15 (-.40, .40)	.00 (-.19, .19)	.65 (.53, .79)

Note. PA=positive affect; a²=additive genetic variance; c²=shared environmental variance; e²=nonshared environmental variance. Significant values are indicated in bold.