

Trajectories of Positive and Negative Affect Across Adolescence: Maternal History of Depression  
and Adolescent Sex as Predictors

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## CHAPTER I

### Introduction

Substantial research has identified positive (PA) and negative (NA) affect as central correlates and predictors of depression. Diminished PA specifically characterizes depression and elevated NA is associated with both depression and anxiety (Clark & Watson, 1991; Joiner & Lonigan, 2000). Longitudinal studies have shown that such atypical affect in childhood predicts increased risk for depression in adolescence and adulthood (Caspi et al., 1996; van Os et al., 1997). In adolescence, during which rates of depression increase and sex differences in depression prevalence emerge (e.g., Dalsgaard et al., 2020; Hankin et al., 1998; Kessler et al., 2001; Thapar et al., 2012), lower PA and elevated NA are associated with greater risk for subsequent depression (Lindahl et al., 2013). Given the importance of PA and NA in relation to depression, the purpose of the present study was to examine how these constructs change across development and whether certain risk factors are associated with such change.

Over the course of childhood, PA increases and NA declines (Olino et al., 2011), whereas in adolescence, these trends are largely reversed (Griffith et al., 2021; Larson et al., 2002; Moneta et al., 2001; Weinstein et al., 2007), such that PA declines and NA increases. A longitudinal study of working- and middle-class European American children in 5th through 8th grade at baseline (10- to 14-years-old) found that scores on measures of daily emotional states, using a bipolar index (e.g., unhappy to happy) became less positive and more negative until 10th grade (Larson et al., 2002). Such scales are difficult to interpret, however, as they treat NA and PA as opposite ends of the same affective continuum rather than as orthogonal constructs.

Another longitudinal study followed students in Grades 6, 8, 10, and 12 (ages 12 to 18) for 4 years and found that PA declined quadratically across adolescence such that participants' reports of "feeling good" declined across adolescence with steeper decreases in early adolescence (Moneta et al., 2001). Similarly, a one-year study of youth in grades 8 and 10 using Ecological Momentary Assessment (EMA) found that PA declined, and NA remained relatively stable from grades 8 through 11 (Weinstein et al., 2007). Finally, another one-year longitudinal study reported mean-level declines in PA but periodic increases and decreases in NA for youth ages 9 to 17 (Griffith et al., 2021).

These results provide consistent evidence of declining trajectories for PA but differing trajectories of NA across adolescence. PA tends to decline across adolescence and shows steeper decreases in early relative to late adolescence. Conversely, NA does not show a consistent pattern of change in adolescence, perhaps indicating greater individual variability in levels of NA throughout this developmental period.

Some of the variability in trajectories of NA may be attributable to characteristics of the measures. Twenge and Nolen-Hoeksema (2002) found that self-report measures of depressive symptoms often show declines after the first wave in longitudinal studies, but such declines may not reflect changes in the construct itself. Such artifacts might bias estimates of longitudinal trajectories of depression-related variables (see also Ge et al., 2001; Long et al., 2020; Shrout et al., 2018). Therefore, in the current study, we controlled for this initial decline.

Several variables are associated with adolescent affect. Among Swedish adolescents, PA was associated with higher life satisfaction ( $r = .43, p < .001$ ), psychological well-being ( $r = .4, p < .001$ ), environmental mastery ( $r = .48, p < .001$ ), self-acceptance ( $r = .4, p < .001$ ), personal

growth ( $r = .32, p < .001$ ), and purpose in life ( $r = .21, p < .01$ ); NA exhibited similar but inverse associations with many of these constructs (Garcia et al., 2013). Sleep is associated with affect as well, as sleep-deprived adolescents and adults showed diminished PA for 9 of 12 PA items as compared to rested individuals, but not elevated NA (Talbot et al., 2010).

Affect also is related to environmental factors. For instance, Fox et al. (2010) noted that among adolescent females, negative life events were positively associated with NA ( $r = .47, p < .001$ ) but not significantly related to PA. Additionally, Luebke and colleagues (2014) found that PA was associated with greater family expressiveness of positive emotions ( $.49, p < .01$ ) and maternal warmth ( $.47, p < .01$ ), and negatively associated with family expressiveness of negative emotions ( $-.20, p < .05$ ) and maternal psychological control ( $-.30, p < .01$ ). In contrast, NA was negatively associated with maternal warmth ( $-.49, p < .01$ ) and positively associated with family expressiveness of negative emotions ( $.56, p < .01$ ) and maternal psychological control ( $.41, p < .01$ ). Thus, PA and NA are associated with a range of factors, including environmental factors like parenting and negative life events.

Adolescent sex also has been found to relate to trajectories of PA and NA across adolescence. Although determining differences in the relation of depression to biological sex and gender may be important for understanding mechanisms of onset and change, few studies clearly differentiate between these constructs (including our own). Consequently, we report findings from the literature based on which construct seems to have been assessed, though we acknowledge ambiguity is present for many studies. Sex differences in the rates of depressive symptoms and disorders emerge in early adolescence and persist across adolescence (e.g., Salk et al., 2017) such that girls exhibit more depression than boys. Such differences in depression

may result from sex differences in hormonal and physiological changes associated with puberty, cognitive vulnerabilities, or familial and social relationships (Hyde et al., 2008; Rapee et al., 2019).

Sex differences in PA and NA also are apparent in adolescence, although findings vary across studies. Moneta et al. (2001) showed that girls reported lower levels of “feeling good” relative to boys during adolescence. Conversely, Larson et al. (2002) reported that girls showed higher average affect than boys across adolescence, although both girls and boys reported decreases in positive emotional states over time.

Griffith et al. (2021) also identified distinct trajectories of change in PA and NA for girls versus boys. Whereas girls showed consistent declines in PA across adolescence, boys exhibited an initial increase followed by a decline in PA. Additionally, girls demonstrated a curvilinear change in NA across adolescence (decreases, then increases, followed by decreases), whereas boys exhibited quadratic change (initial declines followed by increases) in NA (Griffin et al. (2021). Weinstein et al. (2007) reported that boys showed a greater decline in PA during adolescence than girls and decreases in NA over time for girls, but stable NA scores for boys. Thus, sex differences in the trajectories of PA and NA have been observed, although the results have been inconsistent across studies. Variability in the measures of affect used and sample characteristics likely contribute to this heterogeneity.

Exposure to maternal depression in infancy and childhood is a known risk factor for depression in adolescence (e.g., Côté et al., 2018; Spence et al., 2002) and is associated with blunted PA and elevated NA in at-risk offspring (Davis et al., 2020; Goodman et al., 2011; Olino et al., 2011). A meta-analysis of maternal depression and outcomes in children (mean age =



7.13,  $SD = 5.08$ ) revealed small but significant associations between maternal depression and lower PA and higher NA in their offspring (Goodman et al., 2011). Davis et al. (2020) showed that 12-month-old children exposed to maternal depression exhibited higher NA at 26 months. Similarly, children exposed to maternal depression in infancy exhibited lower PA from infancy through age 9 (Olino et al., 2011).

Diminished positive emotionality in infancy and childhood may be a source of vulnerability for depression that persists into adolescence (Durbin et al., 2005; Meehl, 1975; Olino et al., 2011). Thus far, however, only cross-sectional studies have examined relations between maternal history of depression and affect in their adolescent offspring. In a sample of 8- to 17-year-old children, Dietz et al. (2008) found that children whose first- and second-degree relatives had a history of depression exhibited lower PA in interactions with their mothers than did children with no family history of depression.

Characterizing trajectories of affect and their predictors is important because it may guide early interventions for depression. Knowledge about such trajectories could inform the timing of preventive interventions by identifying developmental periods in which youth are particularly at risk. For example, an inflection point for PA, in which trajectories shift from increasing to decreasing, may indicate a particularly salient time for intervention. Additionally, understanding predictors of these trajectories may improve identification of high-risk populations who could benefit most from timely intervention. Girls and boys may experience a shift in PA from increasing to decreasing at about the same time, but girls may experience a steeper decline as compared to boys.

Finally, distinct trajectories as a function of different predictors may suggest which types of interventions are warranted. For instance, greater increases in NA across adolescence among offspring of mothers with a depression history but no differences in trajectories of PA may suggest that this population would benefit more from interventions that target negative emotionality. Characterizing these features of affect trajectories may enhance large-scale interventions for teens, but for individual clinicians and patients, tailoring treatment to the patient's own affective experiences will remain an important component of treatment.

The present study examined the longitudinal trajectories of PA and NA in youth from grade 6 through 12. First, we hypothesized that PA would decrease across adolescence (see Larson et al., 2002; Moneta et al., 2010; Weinstein et al., 2007). Second, we expected that youth with a maternal history of depression (high-risk) would show lower average PA and higher average NA than offspring of mothers with no depression history (low-risk) (Dietz et al., 2008; Goodman et al., 2011; Olino et al., 2011). We also examined potential differences in the shape and magnitude of growth of PA and NA across adolescence as a function of adolescent risk and sex.

## CHAPTER II

### Method

#### Participants

Participants were 240 youth and their mothers. Adolescents (mean age = 11.86 years,  $SD = 0.57$ ) were evaluated annually beginning in the sixth grade. The adolescent sample was 82% White, 14.7% African American, and 3.3% Hispanic, Asian, or Native American. Participants were asked to indicate if they were a boy or girl; 54.2% of participants identified as a girl. Mean socioeconomic status in the sample (Hollingshead, 1975) was 41.84 ( $SD = 13.25$ ), which represents lower middle (e.g., retail sales, childcare workers) to middle class (e.g., teachers, nurses).

#### Procedures

Parents of children in grade 5 from public schools in the greater metropolitan area of a medium size city in the midsouth region of the United States were invited to participate in a study about parents and children. We sent parents a letter describing the study, along with a brief health history questionnaire inquiring about 24 medical conditions (e.g., diabetes, heart disease, depression) and 34 medications (e.g., Prozac, Elavil, Valium). Of the 1,495 mothers who indicated an interest in participating, 587 endorsed either a history of depressive symptoms, use of antidepressants, or no history of psychopathology. We conducted phone interviews with these mothers. Of the 587 families screened, we excluded 238 because they did not report having had sufficient symptoms to meet criteria for a depressive disorder (38%), they had other psychiatric disorders that did not also include a depressive disorder (19%), either the mother or

the target child had a serious medical condition (14%), the family was no longer interested (21%), the target child was not in 5<sup>th</sup> grade (6%), or the family had moved out of the area (2%). Using the Structured Clinical Interview for *DSM* diagnoses (SCID; Spitzer, Williams, Gibbon, & First, 1990), we conducted in-person interviews with the remaining 349 mothers. Based on the SCID, 149 families then were excluded because (a) the mother reported a history of either a psychiatric diagnosis that did not also include a mood disorder or had a serious and disabling medical condition (e.g., cancer, multiple sclerosis), or (b) the child had a serious or chronic medical illness or a pervasive developmental disorder. The final sample of 240 families consisted of 185 mothers who had experienced a depressive disorder during the child's life (high risk) and 55 mothers who were life-time free of psychopathology (low risk).

## **Measures**

### ***Positive and Negative Affect***

Adolescents completed the child version of the Positive and Negative Affect Schedule (PANAS-C; Laurent et al. 1999; Watson, Clark, & Tellegen, 1988). The PANAS-C is a 27-item measure comprised of adjectives describing different moods. Adolescents rated how much they had experienced each affect over the past week. Each item was rated on a 5-point Likert scale from 1 (not at all) to 5 (very much) and has displayed good internal consistency ( $\alpha_{PA} = .89$ ,  $\alpha_{NA} = .92$ ; Laurent et al., 1999). In the current sample, alphas ranged from .83 to .92 for PA and from .84 to .91 for NA across time points. Participants completed the PANAS-C in grades 6, 7, 8, 9, 11, and 12 (data collection was reduced in the 10<sup>th</sup> grade year due to a gap in funding).

### ***Youth Risk***

We assessed mothers' history of depression using the Structured Clinical Interview for DSM diagnoses (SCID; Spitzer, Williams, Gibbon, & First, 1990). We used risk as a dichotomous, time-invariant predictor in each growth curve model. Children of mothers with a history of depression comprised the high-risk group and children of mothers with no history of depression were in the low-risk group.

### **Data Analytic Plan**

We conducted analyses using the lavaan package in R for Structural Equation Modeling (Rosseel, 2012; R Core Team, 2021). We assessed missing data using Little's MCAR test and to address missingness, we used full information maximum likelihood (FIML) estimation, a method that allows utilization of partial data under the assumption that missingness is random. We first fit unconditional growth curve models, then identified the best fitting unconditional model using multiple fit indices, and finally incorporated predictors of latent factors.

*Affect Growth Models.* Following procedures outlined by Cole et al. (in press) to examine trajectories of growth in continuous variables, we conducted a series of latent growth curve analyses testing the latent intercept (i.e., No Growth), linear trend (i.e., Linear Growth), and quadratic trend (i.e., Quadratic Growth) of PA and NA (taken separately). This analytic approach allowed us to isolate features of affect trajectories and evaluate their relation to additional variables. Previous work has outlined an initial decline in self-reported scores (Twenge & Nolen-Hoeksema (2002), Shrout et al., (2018), and Long et al., (2020)), suggesting the potential for an artifact from measurement. Therefore, we implemented two different strategies to account for this decline. One involved an orthogonal latent change score approach; the other involved dropping the initial wave of data. We centered all latent variables at Time 1. We defined good

fit as root mean square error of approximation (RMSEA)  $<.06$ , comparative fit index (CFI)  $>.95$ , and standardized root mean square residual (SRMR)  $<.08$ , whereas we defined “acceptable fit” as RMSEA  $<.08$  and CFI  $>.90$  (Hu & Bentler, 1999).

We tested five models for both PA and NA. The first three were conventional growth models: (1) intercept only, (2) intercept and linear trend, and (3) intercept, linear, and quadratic trend. Only the quadratic model for NA provided a moderately good fit across indices (see Table 1), whereas the quadratic model for PA did not achieve good fit across indices. Next, using the quadratic model for NA and PA, we tested two other models, designed not only to test intercept, linear, and quadratic trends but also to control for the wave 1 to wave 2 drop described by Twenge and Nolen-Hoeksema (2002). Model 4 accounted for this effect by incorporating a fourth latent variable representing the change from wave 1 to wave 2. In contrast, model 5 dropped wave 1 from the analysis. Both models fit the data well for PA and NA. Model 4, however, generated several out-of-range parameter estimates. Consequently, we focused on Model 5 for both PA and NA.

*Predicting Affect Growth.* After identifying growth models with adequate fit for both positive and negative affect, we introduced risk and sex concurrently as time-invariant predictors of the latent growth factors. This approach allowed us to assess associations of risk and sex with affect trajectories concurrently. We allowed risk and gender to covary.

## Chapter III

### Results

#### Descriptive Statistics

Descriptive statistics and correlations (Table 1) are presented in the supplementary materials. High-risk adolescents exhibited significantly lower PA in grades 6 (40.49 vs. 43.26,  $p = .019$ ) and 7 (33.60 vs. 36.81,  $p = .03$ ), and elevated NA in grade 6 (21.77 vs. 19.58,  $p = .02$ ). Girls and boys did not differ significantly on PA across timepoints; girls exhibited significantly higher NA than boys in grades 9 (19.45 vs. 16.71,  $p = .005$ ) and 11 (20.41 vs. 16.61,  $p < .001$ ). Missing data at each time point ranged from 14% to 24%. Patterns of missing data suggested that data were missing completely at random through examining Little's MCAR test ( $X^2_{207} = 237.55$ ,  $p = 0.07$ ).

#### Unconditional Univariate Growth Models for PA and NA

The model for PA exhibited a significant latent intercept (coefficient = 34.62,  $p < .001$ , 95% CI [33.48, 35.75]), latent slope (coefficient = -1.40,  $p = .002$ , 95% CI [-2.30, -.051]), and latent quadratic trend (coefficient = 0.24,  $p = .006$ , 95% CI [0.07, 0.41]). The NA model exhibited a significant latent intercept (coefficient = 18.27,  $p < .001$ , 95% CI [17.50, 19.04]) and quadratic trend (coefficient = .22,  $p = .001$ , 95% CI [0.10, 0.35]); the linear trend was not significant (coefficient = -.65,  $p = .055$ , 95% CI [-1.30, 0.01]).

#### Conditional Models

Next, using the unconditional models for PA and NA that omitted wave 1, we tested conditional models that concurrently included adolescent risk and gender as time-invariant

predictors of the latent intercept, slope, and quadratic trends. Both models fit the data well (see Table 2).

In Figure 1, we plot trajectories of affect based on the output from conditional models. Figure 1a shows trajectories of PA for adolescents at low and high risk for depression. The intercept of PA scores for the high-risk group was significantly lower as compared to the low-risk group (coefficient = -3.51,  $p = .008$ , 95% CI [-6.11, -0.91]). In both groups, PA initially declined and then increased. Figure 1b shows trajectories of PA for girls and boys. Both girls and boys declined in PA in early adolescence with increases beginning in 11<sup>th</sup> grade for girls and in 9<sup>th</sup> grade for boys. No significant differences in either the intercept or slope emerged between girls and boys for PA.

Figure 1c shows the NA trajectories by risk group. Adolescents experienced slight declines in NA from grade 7 through grades 8 and 9 for high-risk and low-risk youth, respectively, followed by increases in NA through 12<sup>th</sup> grade. Figure 1d depicts the NA trajectories for girls and boy. Girls reported increases in NA from 7<sup>th</sup> through 12<sup>th</sup> grade, with significant differences in both linear (coefficient = 2.07,  $p = .002$ , 95% CI [0.77, 3.37]) and quadratic (coefficient = -0.29,  $p = .025$ , 95% CI [-0.55, -0.04]) slopes as compared to boys. Boys showed declines in NA from grades 7 through 9 followed by increases through grade 12.



## Chapter IV

### Discussion

Three main findings emerged from the current study. First, we characterized trajectories of change for PA and NA across adolescence. Second, maternal history of depression explained variability in the intercept but not longitudinal trends of PA. Third, we found differences in linear and quadratic trends of NA but not PA as a function of adolescent sex.

First, we observed slight declines in PA from 7<sup>th</sup> to 9<sup>th</sup> grade, followed by minimal increases in PA through 12<sup>th</sup> grade. Similarly, NA exhibited small decreases from 7<sup>th</sup> to 9<sup>th</sup> grade and larger increases through 12<sup>th</sup> grade. Omission of PA and NA data from grade 6 allowed us to avoid modeling the artifactual initial decline in self-report measures identified by Twenge and Nolen-Hoeksema, 2002. These findings align with other studies that reported declines in PA in early adolescence (Griffith et al., 2021; Larson et al., 2002; Moneta et al., 2001; Weinstein et al., 2007).

There was considerable heterogeneity in the overall trajectories of PA across adolescence, however. The trajectory of PA found here aligns with findings reported in earlier studies (e.g., Larson et al., 2002; Moneta et al., 2001) that used composite measures of PA and NA on a single bipolar scale, whereas we evaluated these constructs as orthogonal dimensions. Although the trajectories looked similar, comparisons of these findings should be made with caution because of the different conceptualizations and measurement of affect.

Weinstein and colleagues identified modest declines in PA from 8<sup>th</sup> to 11<sup>th</sup> grade and relative stability in NA. Their use of ecological momentary assessments over a week likely

captured different aspects of affective change as compared to the measure used here. Thus, some of the variability may be attributable to differences in the assessment methods as well as in the constructs assessed. The current study included intervals of one year between assessments about affect in the past 2-weeks, which may have captured more stable, trait-like affect constructs. In contrast, studies with shorter inter-assessment intervals and momentary assessment of affect using EMA to evaluate affect multiple times a day over a week, likely tap more state-like affect. Future research employing state-trait models may help to clarify which construct is assessed and how these distinct affect constructs relate to depression in adolescents. The method used in the current study was most like that of Griffith and colleagues (2021), who showed linear decreases in PA across adolescence. Differences between the PA trajectories found here and those reported by Griffith et al. may be due, in part, to differences in the timing and number of assessments and in the methods of measurement of affect used across studies. The NA trajectories found here differed from those reported by Weinstein and colleagues (2007), who found relative stability in NA, and by Griffith et al. (2021), who showed curvilinear change in NA.

Second, the current study expanded upon previous research by assessing the trajectories of PA and NA as a function of risk (i.e., maternal history of depression). High-risk adolescents showed diminished PA across adolescence, which aligns with longitudinal studies of younger, at-risk children (Olino et al., 2011) and cross-sectional studies of adolescent offspring of mothers with a history of depression (Dietz et al., 2008). The current study found persistent deficits in PA across adolescence in offspring of mothers with a history of depression. Future studies should explore the trajectories of affect in conjunction with biological and

environmental factors related to maternal depression to identify additional predictors of individual differences in these trajectories. Maternal depression is associated with blunted responses to happy faces among emotionally healthy children (Morgan et al., 2019), reduced reward-related ventral striatum activation in depressed and never-depressed, at-risk daughters (Sharp et al., 2014), attenuated responses to social reward (Olino et al., 2015), and reduced putamen volume in children with and without depression (Pagliaccio et al., 2020). Identifying specific predictors of deficits in PA may provide insights into etiological mechanisms and potential treatment targets.

Third, we identified differences in trajectories of NA as a function of adolescent sex. Compared to boys, girls experienced earlier increases in NA, such that NA increased for girls beginning in 7<sup>th</sup> grade, whereas for boys it declined from 7<sup>th</sup> through 9<sup>th</sup> grade before increasing. These results partially mirror those of Griffith and colleagues (2021) who found that girls exhibited increases in NA around age 12, although we did not observe a decline in NA among girls after age 15. Additionally, our results revealed a quadratic trajectory of NA in boys across adolescence, with decreases in early adolescence followed by increases starting in 9<sup>th</sup> grade. Several factors may explain this earlier increase in NA. Girls typically begin puberty two years earlier than boys, and with earlier physical changes, girls may experience earlier self-surveillance and body shame than boys (Hyde & Mezulis, 2020). Additionally, peer sexual harassment is more frequent for girls than boys and may emerge earlier in adolescence due to the earlier start of puberty. Indeed, negative life events in adolescence may affect girls more strongly than boys, as such events account for greater variance in depressive symptoms for girls than boys (Spence et al., 2002). Although multiple studies have identified gender and sex as

predictors of adolescent affect, future research should examine control variables and potential moderators to further account for the variability of observed results.

### ***Strengths, Limitations, and Future Directions***

Strengths of the current study include its multi-year, longitudinal design, and the focus on affect in low- and high-risk adolescents. Nevertheless, limitations in the current study suggest avenues for future research. First, the sample was 82% White, which limits the generalizability of the findings to more diverse racial and ethnic groups who often experience high levels of discrimination and associated stress. A recent study using ecological momentary assessment found that African American adolescents experienced an average of 5.21 instances of racial discrimination each day (English et al., 2020). A meta-analytic review reported that perceived racial or ethnic discrimination among adolescents was associated with higher levels of depressive symptoms ( $r = .26$ ), diminished positive well-being ( $r = -.14$ ), and lower self-esteem ( $r = -.17$ ) (Benner et al., 2018). Adolescent offspring of mothers with a history of depression who also regularly experience the additional burden of discrimination may be particularly vulnerable to depression.

Second, missing data ranged from 14% to 24% across timepoints. This level of missingness can be adequately addressed when data are missing at random, which appeared to be the case in the current study. Nevertheless, our data analytic approach reduced the risk of bias despite this limitation.

Third, we examined a relatively limited set of factors that may predict trajectories of adolescent affect. As noted by Kujawa and colleagues (2020), many RDoC-related factors likely interact to influence the developmental trajectory of affect. Greater granularity in the

assessment of risk is important. For offspring of mothers with histories of depression specifically, comorbid psychopathology, timing of first onset of depression, and frequency of recurrence of depressive episodes also may be related to differences in trajectories over time. Similarly, identifying contributors of mothers' history of depression and current depressive symptoms may yield greater insight into associations between maternal depression and adolescent affective health. For instance, a 13-year longitudinal study found that maternal postnatal depression was only associated with depressive symptoms in adolescent offspring when mothers experienced later bouts of depression (Halligan et al., 2007). Thus, examining maternal history of depression in conjunction with current maternal depression may be most informative for understanding associations with adolescent affect. Using integrated data from multiple samples may address such questions from a dimensional perspective and improve the validity of findings regarding predictors of the trajectories of adolescent affect.

Fourth, although the current results largely align with prior studies that used observational measures of adolescent affect, our exclusive use of self-report measures is a limitation. If adolescent self-reported affect is an indicator of vulnerability to depression, then understanding the relation between self-reported and observed affect during tasks and interactions with others (e.g., parents; peers) may be valuable. Olinio and colleagues (2011) studied children's affective displays in response to developmentally appropriate tasks intended to elicit affect. Dietz and colleagues (2008) observed affect during mother-child, conflict resolution tasks. Use of similar behavioral measures in addition to self-reported affect may help identify intervention targets to ameliorate lower levels of PA in at-risk youth.

### **Clinical Implications**

This study contributes to the literature showing a relation between maternal depression and alterations in positive valence systems in offspring. Importantly, not all high-risk children develop depression; maternal depression may predict greater risk of adolescent depression in offspring only with other vulnerabilities such as blunted or average reward responses (Kujawa et al., 2019). Effective coping may be protective, as at-risk offspring with better coping show higher positive mood and lower depressive symptoms (Jaser et al., 2011), and adaptive responses to peer stress in offspring of depressed mothers are related to reduced risk of depression (Monti et al., 2017).

Several promising interventions target positive valence systems. Behavioral activation (BA) therapy aims to increase engagement in reward activities (Curry & Meyer, 2016). Recent meta-analyses have reported positive findings for BA but also highlight study heterogeneity and recommend conducting additional large-sample trials to evaluate the generalizability of the findings (Martin & Oliver, 2019; Tindall et al., 2017). Positive Affect Treatment (PAT) is a neuroscience-based intervention that targets PA in adults (Craske et al., 2019). PAT produced greater improvements in PA and reductions in NA as compared to negative affect treatment, at both post-test and 6-month follow-up. Adapting PAT for adolescents would provide another potentially valuable intervention in addition to BA, both of which should be studied in at-risk youth.

Implementing interventions that target parent-child interactions also may be important. A recent study testing a brief intervention for mothers with a history of depression showed that greater use of positive parenting behaviors produced an increase in observed positive affect in offspring (Cullum et al., 2022). On the other hand, mothers' aggressive responses to

adolescents' PA have been found to correlate with a larger volume of orbitofrontal cortex, dorsal anterior cingulate cortex, and in boys, larger amygdala volume (Whittle et al., 2009). Additionally, maternal dampening of adolescents' positive affective behaviors correlates with greater depressive symptoms and emotion dysregulation (Yap et al., 2008). These studies highlight the potential utility of family-based interventions, which have been found to prevent elevations in adolescents' depressive symptoms for up to 2-years (Compas et al., 2015).

In summary, the current study provided further evidence of lower levels of PA in adolescent offspring of mothers with a history of depression. Additionally, we found earlier increases in NA among girls as compared to boys. Plotting the trajectories of affect across adolescence may inform us about the best time to provide interventions that target positive and negative valence systems. Future prospective studies are needed that measure multiple contextual predictors of affect across the important developmental epoch of adolescence, which is a prime time for preventing affective disorders. Interventions that target adolescents' reward systems and interpersonal relationships (e.g., between mothers and offspring) may be especially likely to enhance positive affect in high-risk youth.

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**Table 1 - Correlations among sex, risk, NA, and PA in grades 6, 7, 8, 9, 11, and 12**

				Negative Affect						Positive Affect					
Variable	Mean (S.D.)	Sex	Risk	Grade 6	Grade 7	Grade 8	Grade 9	Grade 11	Grade 12	Grade 6	Grade 7	Grade 8	Grade 9	Grade 11	Grade 12
Sex	0.54 (0.50)	1													
Risk	0.77 (0.42)	0.06	1												
NA – Grade 6	21.27 (6.5)	0.01	0.14*	1											
NA – Grade 7	18.24 (5.85)	-0.04	0.05	0.39**	1										
NA – Grade 8	17.70 (5.45)	0.02	0.02	0.31**	0.54**	1									
NA – Grade 9	18.26 (6.02)	0.23**	0.14	0.25**	0.4**	0.41**	1								
NA – Grade 11	18.75 (6.93)	0.27**	0.16*	0.13	0.35**	0.38**	0.56**	1							
NA – Grade 12	21.16 (8.13)	0.12	0.18*	0.15*	0.37**	0.43**	0.43**	0.5**	1						
PA – Grade 6	41.12 (7.19)	-0.04	-0.16*	-0.33**	-0.08	-0.04	-0.19*	-0.02	-0.04	1					
PA – Grade 7	34.35 (8.57)	0.12	-0.16*	-0.03	0.09	0.02	0.06	0	-0.03	0.42**	1				
PA – Grade 8	34.46 (9.54)	0.13	-0.07	-0.04	0.02	-0.04	-0.03	-0.04	0.02	0.44**	0.55**	1			
PA – Grade 9	32.26 (8.98)	0.06	-0.07	-0.08	-0.08	-0.06	-0.02	0.03	-0.09	0.37**	0.47**	0.59**	1		
PA – Grade 11	32.62 (9.35)	-0.03	-0.05	-0.02	-0.01	-0.08	-0.01	-0.16*	-0.03	0.21**	0.4**	0.43**	0.47**	1	
PA – Grade 12	33.62 (8.93)	-0.07	-0.06	-0.01	-0.17*	-0.18*	-0.21**	-0.08	-0.19**	0.24**	0.24**	0.33**	0.43**	0.5**	1

\*p < .05. \*\*p < .01.

**Table 2 – Means of Affect by Predictor**

Timepoint	Overall <sup>1</sup>	Sex			p-value <sup>2</sup>	Risk		p-value <sup>2</sup>
		Boys <sup>1</sup>	Girls <sup>1</sup>			Low <sup>1</sup>	High <sup>1</sup>	
Negative Affect								
Grade 6	21.27 (6.5)	21.19 (6.2)	21.34 (6.76)	0.98	19.58 (5.59)	21.77 (6.68)	0.018	
Grade 7	18.24 (5.85)	18.53 (6.08)	18.01 (5.67)	0.63	17.73 (5.57)	18.40 (5.94)	0.57	
Grade 8	17.70 (5.45)	17.6 (5.82)	17.79 (5.15)	0.54	17.5 (5.39)	17.77 (5.48)	0.75	
Grade 9	18.26 (6.02)	16.71 (4.95)	19.45 (6.51)	0.005	16.82 (5.00)	18.72 (6.26)	0.091	
Grade 11	18.75 (6.93)	16.61 (5.89)	20.41 (7.24)	<0.001	16.85 (4.03)	19.42 (7.60)	0.18	
Grade 12	21.16 (8.13)	19.96 (7.00)	21.97 (8.75)	0.18	18.82 (5.55)	22.04 (8.76)	0.051	
Positive Affect								
Grade 6	41.12 (7.19)	41.46 (7.39)	40.84 (7.02)	0.61	43.26 (6.04)	40.49 (7.39)	0.019	
Grade 7	34.35 (8.57)	33.21 (8.64)	35.27 (8.43)	0.081	36.81 (6.48)	33.60 (8.99)	0.03	
Grade 8	34.46 (9.54)	33.13 (9.88)	35.58 (9.14)	0.1	35.65 (7.81)	34.06 (10.04)	0.37	
Grade 9	32.26 (8.98)	31.67 (8.65)	32.71 (9.23)	0.54	33.34 (7.39)	31.91 (9.42)	0.28	
Grade 11	32.62 (9.35)	32.94 (8.85)	32.37 (9.76)	0.65	33.33 (8.61)	32.36 (9.62)	0.45	
Grade 12	33.62 (8.93)	34.35 (8.81)	33.14 (9.02)	0.3	34.54 (9.31)	33.28 (8.79)	0.34	

<sup>1</sup> Mean (SD)<sup>2</sup> Wilcoxon Rank Sum Test

**Table 3 - Unconditional Model Fit**

Fit Indices	Intercept	Linear	Quadratic	Twenge	Quadratic without Time 1
Positive Affect					
comparative fit index (CFI)	0.18	0.45	0.91	0.98	0.98
standardized root mean square residual (SRMR)	0.33	0.18	0.07	0.04	0.04
root mean square error of approximation (RMSEA)	0.23	0.21	0.1	0.04	0.06
Akaike's information criterion (AIC)	8448.18	8367.2	8233.29	8212.22	6636.51
Bayesian information criterion (BIC)	8476.03	8405.49	8285.5	8271.39	6684.71
Chi-square	268.8	181.82	39.9	14.84	11.08
degrees of freedom (df)	19	16	12	10	6
p-value	0	0	0	0.14	0.09
Negative Affect					
comparative fit index (CFI)	0.61	0.69	0.96	0.99	0.99
standardized root mean square residual (SRMR)	0.15	0.12	0.05	0.04	0.04
root mean square error of approximation (RMSEA)	0.15	0.15	0.06	0.03	0.04
Akaike's information criterion (AIC)	7622.84	7603.8	7536.6	7528.02	5980.58
Bayesian information criterion (BIC)	7650.69	7642.09	7588.81	7587.19	6028.77
Chi-square	124.35	99.31	24.11	11.53	7.8
degrees of freedom (df)	19	16	12	10	6
p-value	0	0	0.02	0.32	0.25

**Table 4 - Conditional Model Fit**

Fit Indices	Positive Affect	Negative Affect
comparative fit index (CFI)	0.99	1
standardized root mean square residual (SRMR)	0.03	0.04
root mean square error of approximation (RMSEA)	0.03	0.01
Akaike's information criterion (AIC)	7256.8	6587.76
Bayesian information criterion (BIC)	7343.82	6674.77
Chi-square	12.36	10.39
degrees of freedom (df)	10	10
p-value	0.26	0.41

**Table 5 - Conditional Model Results**

		<b>Positive Affect (PA)</b>								
		Intercept			Linear Slope			Quadratic Slope		
		Estimate	Standard Error	P-value	Estimate	Standard Error	P-value	Estimate	Standard Error	P-value
<b>Predictor</b>	Risk	-3.507	1.325	<b>0.008</b>	1.559	1.047	0.136	-0.226	0.2	0.258
	Sex	2.173	1.126	0.054	-0.317	0.909	0.727	-0.067	0.175	0.703
		<b>Negative Affect (NA)</b>								
		Intercept			Linear Slope			Quadratic Slope		
		Estimate	Standard Error	P-value	Estimate	Standard Error	P-value	Estimate	Standard Error	P-value
<b>Predictor</b>	Risk	0.723	0.935	0.439	0.409	0.766	0.593	0.003	0.15	0.982
	Sex	-0.828	0.794	0.297	2.071	0.661	<b>0.002</b>	-0.292	0.13	<b>0.025</b>

**Figure 1 - Conditional Model Trajectories**

