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Mediational Pathways Through Which Positive and Negative Emotionality Contribute to Anhedonic Symptoms of Depression: A Prospective Study of Adolescents

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Abstract

This study takes a developmental psychopathological approach to examine mechanisms through which baseline levels of positive emotionality (PE) and negative emotionality (NE) prospectively predict increases in anhedonic depressive symptoms in a community sample of 350 adolescents (6th–10th graders). Dependent stressors mediated the relationship between baseline levels of NE and anhedonic depressive symptoms after controlling for initial symptoms. Supportive relationships mediated the relationship between baseline levels of PE and anhedonic depressive symptoms, after controlling for baseline symptoms. In addition, NE × PE interacted to predict later anhedonic depressive symptoms, such that adolescents with low levels of PE and high levels of NE experienced the greatest increase in anhedonic depressive symptoms. Last, supportive relationships interacted with baseline PE to predict prospective changes in anhedonic depressive symptoms, such that adolescents with low PE and low supportive relationships experienced the greatest increase in anhedonic depressive symptoms. Results are discussed in terms of current theoretical models of the link between temperament and depression.

Keywords

Depression; Temperament; Mediation; Stress

The number of individuals diagnosed with depressive disorders has increased dramatically throughout the last several decades (Kessler et al. 1994). Depressive disorders are recurrent and chronic conditions (Keller et al. 1992; Kovacs 1998) that often exhibit onset in childhood and adolescence (Lewinsohn et al. 1994; Petersen et al. 1993). For example, one prospective longitudinal study indicates that rates of depression increase dramatically for individuals between the ages of 15 and 18 (from 3% to 17%), and girls' depression rates doubles that of males (Hankin et al. 1998). Further, the average age of onset for depression has rapidly decreased, such that individuals are becoming depressed at an earlier age (Lewinsohn et al. 1994). Given these facts it is important to identify risk factors for the development of depression, especially among youth. One promising approach that has gained increasing attention is the study of how individual differences in temperament can function as vulnerability to psychopathology (e.g., Tackett & Krueger 2005). In particular, temperament

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traits in children and adolescents have been hypothesized to function as distal risk factors for depression (Compas et al. 2004; Hankin & Abramson 2001; Krueger 1999).

Longitudinal studies indicate that temperament and emotionality, measured in childhood, predict the later development of depressive disorders (Block et al. 1991; Caspi et al. 1996; Krueger 1999; Lonigan et al. 2003; vanOs et al. 1997), yet the pathways through which temperament leads to depressive symptoms are less studied. It is likely that individuals with certain temperamental traits are at increased risk to develop depressive symptoms, although this process probably occurs through different pathways. Indeed, the concept of equifinality indicates that the same developmental outcome may result from different starting points and through various processes. Developmental psychopathologists (e.g., Cicchetti & Rogosch 2002; Frick 2004) have noted that psychopathology in adolescence likely results from different pathways across individuals rather than the same pathway for all adolescents. Studying potentially different developmental pathways to depression is important because this would enhance understanding the etiology and variation in trajectories of temperament and mood disorders.

Accordingly, it is important to study how temperament, individual experiences, and the relationship between the two may influence the pathways through which individuals develop depression. The present study used a prospective longitudinal design with a sample of adolescents to examine the processes through which temperament contributes to elevations of depressive symptoms over time. The primary goal of this study was to investigate various theoretically motivated pathways through which temperament may serve as a vulnerability to depressive symptoms among adolescents at a time when the rates of depression are known to rise dramatically and the sex difference in depression emerges.

Temperament

Temperament refers to individual patterns in emotional and behavioral reactivity that appear early in life, are stable across time and situations, and have a biological basis (Rothbart & Bates 2006). Researchers have proposed numerous frameworks to classify the dimensions of temperament (for reviews, see Rothbart & Bates 2006; Strelau 1998). Although early work in this area focused on identifying narrow dimensions of temperament based on behavioral traits (e.g., Thomas & Chess 1977), recent work has focused on identifying broad higher order traits (e.g., Ahadi et al. 1993; Anthony et al. 2002; Halverson et al. 2003; Rothbart et al. 2001; Shiner & Caspi 2003). Compas and colleagues (2004) note that this shift in classification of temperament can facilitate the synthesis of findings across studies, can simplify comparisons of temperament across the lifespan, and higher order traits can be more clearly linked to underlying neurobiological systems, compared to lower order traits. Thus, the current study used higher order temperament traits to understand pathways through which temperament leads to later depressive symptoms.

Across prominent theories of temperament traits, the higher order factors of neuroticism and extraversion consistently emerge. For example, Shiner and Caspi (2003) conducted a comprehensive literature review in which they delineated a taxonomy to conceptualize higher-order factors of child and adolescent temperament. They proposed a four-factor model consisting of the following factors: Extraversion, Neuroticism, Agreeableness, and Conscientiousness. In their recent review, Rothbart and Bates (2006) included Negative Emotionality and Surgency/Extraversion as broad factors of temperament in infancy and childhood. Relatedly, Rothbart et al. (2001) have found support for a three-factor model of child temperament that includes the dimensions of Extraversion (Surgency), Negative Affectivity, and Effortful Control. In addition, a similar factor structure has been found cross-culturally for children in China and Japan (Ahadi et al. 1993; Kusanagi 1993; see also

Halverson et al. 2003). Some research has shown age and sex differences in both negative and positive emotionality, such that adolescent girls exhibit less positive emotionality and greater negative emotionality than adolescent boys (e.g., Jacques & Mash 2004; Lonigan et al. 1999). Given this broad consensus on the higher order structure of temperament, we examined and focused on the temperament traits of negative emotionality (NE, or neuroticism) and positive emotionality (PE, or extraversion). We chose not to investigate additional higher order temperament traits (e.g., Effortful Control, Agreeableness), because previous studies have consistently linked NE and PE to depressive symptoms, and examining pathways to depressive symptoms was the main goal of this study.

Temperament and Risk for Depression

Clark and Watson (1991) proposed the tripartite model of depression and anxiety in which they conceptualized risk for depression based on the two broad temperament dimensions of negative emotionality and positive emotionality (see also Clark 2005). The tripartite model proposes that NE is a nonspecific factor that is common to both depression and anxiety and that low PE specifically characterizes depression (Clark & Watson 1991; Clark et al. 1994). Results from several studies of child samples have supported the tripartite model (Chorpita & Daleiden 2002; Lonigan et al. 1999; Lonigan et al. 1994). In addition, longitudinal studies support the link between the main effects of NE and PE and later depressive symptoms (Block et al. 1991; Caspi et al. 1996; Krueger 1999; Lonigan et al. 2003; Lonigan et al. 1999; van Os et al. 1997). In addition, Joiner and Lonigan (2000) found that NE and PE interacted to predict diagnoses of depression in youth psychiatric inpatients, such that changes over time in depression were associated with the combination of low levels of PE and high levels of NE.

Despite an abundance of studies documenting the association between NE and PE and the development of depressive symptoms, little research has considered the mechanisms through which temperament can lead to psychopathology. For example, there may be direct linear effects, mediation effects, or moderation effects (e.g., temperament × environment interactions, temperament × temperament interactions) (for a review, see Rothbart & Bates 2006). Clark et al. (1994) proposed four etiological models to describe the relation between temperament and psychopathology: (1) the *vulnerability model* proposes that temperament predisposes an individual to be at risk for the development of a disorder; (2) the *pathoplasty model* postulates that temperament shapes the course of a disorder without necessarily having a direct etiological role (i.e., mediation); (3) the *scar model* hypothesizes that the experience of a disorder can cause changes in temperament; and (4) the *spectrum or continuity model* posits that depression represents an extreme endpoint of a trait, suggesting that both temperament and the disorder have the same underlying processes. These models are not mutually exclusive (Clark et al. 1994). It is likely that some combination of them best represents the link between temperament and the development of psychopathology. The current study was theoretically motivated by the pathoplasty and vulnerability models and investigated how adolescents with varying levels of NE and PE may seek out specific environments, shape their current environment, and influence reactions from others, potentially in order to understand better the processes by which these traits contribute to depressive symptoms.

Developmental Pathways

Cicchetti and Rogosch (2002) note that depression in adolescence likely results from diverse processes across different individuals rather than from all individuals following the same pathway to depression. Relatedly, Pickles and Hill (2006) emphasize the importance of studying developmental pathways and identifying mechanisms that may influence an individual's developmental trajectory. In addition, they note that pathways can provide a framework to consider the differential effects of mechanisms on outcomes.

The present study investigated two separate mediating mechanisms that may lead from initial levels of NE and PE to depressive symptoms. In particular, we propose that stressors will mediate the relationship (or pathway) between NE and depressive symptoms and supportive relationships (i.e., perceived emotional support from peers and parents) will mediate the relationship between PE and depressive symptoms.

Stressors as Mediational Pathway

Previous research provides theoretical and empirical support for the selection of stressors as the meditating mechanism between NE and depression (e.g., Lakdawalla & Hankin 2008; Hankin & Abramson 2001; Kendler et al. 2003; van Os & Jones 1999). This study used Hankin and Abramson's (2001) elaborated cognitive vulnerability transactional stress model to test potential mediating mechanisms through which initial levels of NE may serve as a vulnerability to depressive symptoms. This model posits that the relationship between NE and depression may be explained by an increased likelihood of encountering more negative events, or stressors, over time. Consistent with this hypothesis, studies have found that NE is an independent predictor of exposure to stressful events (Bolger & Zuckerman 1995; Fergusson & Horwood 1987; Kendler et al. 2003; Magnus et al. 1993; van os & Jones 1999). In addition, longitudinal research with adults has shown that stressors partially mediate the association between baseline NE and later depressive symptoms (Lakdawalla & Hankin 2008; van os & Jones 1999), but no research has investigated this pathway among adolescents.

It is important to note that the stress literature has conceptualized stressors as independent events or dependent events. Typically, dependent events are considered to be at least partially caused by an individual's behavior or characteristics (e.g., arguments with friends), while independent events occur outside of an individual's control and can be considered "fateful" (e.g., death of a parent). Research has shown that adults and adolescents with elevated depressive symptoms encounter more dependent than independent stressful events, and individuals who generate dependent stressors experience increases in depressive symptoms (e.g., Hankin et al. 2007; Rudolph & Hammen 1999; for a review, see Hammen 2005). Consistent with the stress literature, we chose to categorize stressors as independent or dependent in order to examine specifically the relationship among temperament, depression, and each type of stressor as theory suggests that dependent, but not independent, events would result from negative emotionality.

Supportive Relationships as Mediational Pathway

To our knowledge, there is no empirical research that has examined the mediating role of supportive relationships in the link between PE and depression in children and adolescents, and relatively few studies have done so with adults (Finch & Graziano 2001; Joiner 1997). For example, Finch and Graziano (2001) found that extraversion exerted an indirect effect on depression via social support satisfaction in a sample of undergraduate students.

Despite the lack of empirical investigation, there are theoretical reasons to hypothesize that supportive relationships may be one developmental pathway accounting for the association between PE and depression. Several studies have established a link between levels of PE and social support (Asendorpf & van Aken 2003; Asendorpf 1998; Shiner 2006). Indeed, it is theorized that one of the core features of PE is an individual's tendency to evoke and enjoy social attention (Ashton et al. 2002). Children who exhibit high levels of Surgent Engagement (a trait with characteristics common to high PE) are more likely to have friends and be socially accepted, both concurrently and across time (Shiner 2000). Deficits in interpersonal relationships (e.g., peer relationship problems, lack of social support) are associated with the development and maintenance of depression in childhood and adolescence (Hammen & Rudolph 2003; Rudolph & Asher 2000), and peer difficulties predict future depression (Boivin

et al. 1995; Panak & Garber 1992). Thus, we posited that youth with low PE may have less supportive relationships, which in turn, may contribute to later depressive symptoms.

Additional Pathways

Another way to conceptualize the relationship among temperament, psychosocial risk factors, and depression is to identify specific groups that may develop depressive symptoms through different pathways. Thus, we examined if temperament interacted with either dependent stressors, independent stressors, or supportive relationships to predict changes in depressive symptoms. There is a growing body of theoretical and empirical work in children and adults that suggests the effect of temperament on depression may be moderated by responses to stress (e.g., Lengua et al. 2000; Mroczek & Almeida 2004; for reviews, see Compas et al. 2004; Grant et al. 2003). However, no empirical research to our knowledge has examined the moderating effect of stress on the relationship between temperament and depression in adolescents. Similarly, relatively little research has examined if supportive relationships moderate the association between temperament and depression. Although a study with adults (Nezlek & Allen 2006) found that social support moderated the relationship between daily well-being and daily negative events, the source of support (i.e., friends vs. family) dramatically affected this association. Thus, it is clear that further research is needed to explore the relationship among temperament, supportive relationships, and depression, especially among youth.

The Present Study

This study takes a developmental psychopathological approach to understand better the etiology and variation in developmental trajectories that lead to prospective changes in anhedonic depressive symptoms. Specifically, data from a prospective longitudinal study were used to examine the mechanisms through which temperament contributes to the development of later anhedonic depressive symptoms. We chose to use anhedonic depressive symptoms as our outcome, rather than the broader construct of depressive symptoms, because there is an accumulation of literature that shows depressive symptoms, as measured by the CDI, may be more a measure of negative emotionality than of depression (Chorpita et al. 2005; Stark & Laurent 2001). This issue is especially important to the current investigation because negative emotionality is used to predict changes in depressive symptoms, measured by the CDI. Thus, to clarify the independence of the CDI as a measure of depressive symptoms and not broad negative emotionality, we chose to use anhedonic depressive symptoms as our outcome.

This study had five main hypotheses. First, it was hypothesized that baseline levels of PE would predict anhedonic symptoms of depression, such that adolescents who have lower levels of PE would report higher prospective levels of anhedonic depressive symptoms. Second, it was hypothesized that baseline levels of NE would predict anhedonic symptoms of depression, such that adolescents with higher levels of NE would exhibit greater increases in anhedonic depressive symptoms. Third, it was hypothesized that baseline levels of NE and PE would interact to predict anhedonic depressive symptoms, such that adolescents with both high levels of NE and low levels of PE would report the greatest increase in anhedonic depressive symptoms. Fourth, two separate mediation models were proposed. In the first model, it was hypothesized that supportive relationships would partially mediate the relationship between PE and anhedonic symptoms of depression. In the second model, it was hypothesized that stressors would partially mediate the relationship between NE and anhedonic symptoms of depression.

Finally, we explored whether sex moderated any of these hypotheses. More girls than boys exhibit depressive disorders starting in early adolescence (Angold et al. 2002; Costello et al. 2003; Hankin et al. 1998). The 2:1 sex difference in rates of depression stabilizes in middle to

late adolescence (ages 15–18) and is maintained throughout adulthood (Hankin & Abramson 1999). Investigating potential moderation by sex is an important goal as such knowledge informs etiological theories on the development of depression during adolescence generally as well as the emergence of sex differences in depression more specifically (Hankin et al. 2008).

Method

Participants

Participants were 6th–10th graders who were recruited from five Chicago area schools. Briefly, 390 adolescents provided active parental consent and were willing to participate, and 356 youth completed baseline questionnaires. The 34 students who were willing to participate but did not complete baseline measures were absent from school and unable to reschedule. Present analyses are based on 350 adolescents who provided complete data at baseline. There is a 98% retention rate of participants from baseline to Time 2; data are available from 345 students at Time 2. The age range was 11–17 (*M*=14.5; *SD*=1.40) and 57% of the sample was female. Approximately 53% of the sample was White, 21% were African-American, 13% were Latino, 6% were Asian or Pacific Islander, and 7% were bi– or multi–racial.

Procedures

Students participated in this study with active parental informed consent. Permission to conduct this investigation was provided by the school districts and their institutional review boards, school principals, the individual classroom teachers, and university institutional review board. Trained research personnel visited classrooms in the schools and briefly described the study to adolescents, and letters describing the study were sent home to parents. Specifically, students and parents were told that this study was about adolescent mood and experiences, and participation would require completion of questionnaires over time. Students, who agreed to participate and had returned active parental consent, read and signed their own informed assent form after having the opportunity to ask any questions about the study. Adolescents completed a battery of questionnaires during class time and were debriefed at the end of the study. Participants completed questionnaires at two time points over a 5-month period. Adolescents were compensated $10 for their participation at each wave in the study.

Measures

Adolescent Depressive Symptoms—Adolescents completed the 27-item Children's Depression Inventory (CDI; Kovacs 1985) at each assessment period to assess levels of depressive symptoms. Each CDI item is rated on a scale from 0–2, such that higher scores indicate greater symptom severity. The CDI has demonstrated reliability (alpha=0.89) and validity (e.g., Kazdin et al. 1983). The mean for the CDI at Time 1 was 12.80 (SD=8.64) and the mean at Time 2 was 15.31 (SD=12.82). At Time 1 CDI scores ranged from 0– 47, and at Time 2 scores ranged from 0– 52.

Although the CDI is one of the most commonly used measures of depressive symptoms, there is a growing concern about the construct validity of the CDI and its specificity as a measure of depression. Specifically, it has been suggested that the CDI may lack independence and discriminant validity from neuroticism, or negative emotionality (e.g., Chorpita et al. 2005). Thus, to clarify the independence of the CDI as a measure of depressive symptoms and not broad negative emotionality, based on past work (e.g., Chorpita et al. 1998; Joiner et al. 1996), particular anhedonic items from the CDI (nos. 4, 12, 15, 20, 21, 22; Chorpita et al. 1998) were selected to examine the relative specificity of the CDI as a measure of depressive symptoms. Although low levels of PE have been conceptualized as strongly related to anhedonia (Clark et al. 1994), it is important to note that in our sample, baseline PE and Time 1 and Time 2 anhedonia were only moderately correlated at −0.22 and −0.37, respectively,

suggesting that PE and anhedonic depressive symptoms do not overlap too strongly and mollify potential concerns about these constructs being redundant. The mean for the anhedonic CDI items at Time 1 was 2.55 (SD=1.83) and the mean at Time 2 was 2.63 (SD=1.97). At Time 1 anhedonic CDI scores ranged from 0– 10, and at Time 2 scores ranged from 0–11. Anhedonic items on the CDI at Time 1 were significantly correlated with the full CDI at Time 1 and Time 2, r=0.36, p<.01 and r=0.33, p<0.01, respectively. In sum, all analyses were conducted with the anhedonic items from the CDI as the outcome.

Adolescent Temperament—Adolescents completed the Dimensions of Temperament Scale-Revised (DOTS-R; Wills et al. 1998) at Time 1 to measure their levels of positive emotionality and negative emotionality. The DOTS-R uses a 4-point Likert scale that ranges from “not at all” to “very much” and is a reliable and valid questionnaire that taps various constructs, including positive and negative emotionality. There are 11 items on the Negative Emotionality subscale (alpha for current study=.84) and 13 items on the Positive Emotionality subscale (alpha for current study=.87).

Adolescent Supportive Relationships—Adolescents completed the Network of Relationships Inventory (NRI; Furman 1998) at Time 2 to measure perceived levels of support. The NRI is a 12-item self-report questionnaire with responses measured on a 5-point Likert scale. The NRI has demonstrated reliability (alphas>.80 for subscales and broadband scales) and validity (Furman 1998).

Adolescent Stressors—Adolescents completed the Adolescent Life Events Questionnaire (ALEQ; Hankin & Abramson 2002) at Time 2. The ALEQ is a checklist of 57 negative events (both independent and dependent events) that typically occur to adolescents. Examples include parental divorce (independent event) and arguments with friends (dependent event). Adolescents were asked to indicate if an event happened to them over the last 20 weeks that covered the prospective follow-up between Time 1 and 2. These ratings were then transformed into dichotomous count of stressors (0=no event; 1=event occurred). The ALEQ has demonstrated reliability and validity (Hankin & Abramson 2002; Hankin 2008; Hankin et al. (2009)). Test-retest reliability is good, with r’s ranging from 0.42 over 5 weeks to 0.35 over 2 years.

The 57 different negative life events included in the ALEQ can be categorized into independent versus dependent types of events. Independent events are those that are believed to befall individuals and are not expected to be related to a person’s characteristics (e.g., close friend moves, death of a relative), whereas dependent events are those that the individual’s characteristics or behaviors may have contributed to the occurrence of these events (e.g., fight with friend, romantic break-up). To create variables for independent and dependent events, the list of 57 stressors was categorized independently by the authors with 100% agreement. This resulted in 37 of these as dependent and 13 as independent. The remaining 7 events were not clearly dependent or independent, and so they were not included in these categorized variables. Examples for the dependent category of event include: “You had an argument with a close friend,” “Your parents grounded you,” “Your boyfriend/girlfriend criticized you,” “A boyfriend/girlfriend broke up with you but you still wanted to be with them,” “Did poorly on, or failed, a test or class project,” and “You didn’t complete the required homework for class.” For independent, examples include: “A close friend moved away,” “A close family member couldn’t work due to injury or illness,” and “close family member (parent, sibling) died.” The ALEQ was given at both time points with scores at T2 covering the prospective follow-up from Time 1 to 2. For the present study, the following variables, dependent and independent stressors, at both time points, were used in analyses to test our hypothesis that negative emotionality would predict dependent stress generation better than independent, and in turn, anhedonic depressive symptoms.

Results

Preliminary Analyses

Descriptive statistics for the main variables are presented in Table 1. Low to moderate correlations were observed among all key variables. Examination of the correlation matrix shows a significant negative correlation between positive emotionality and negative emotionality at Time 1. In addition, negative emotionality at Time 1 and positive emotionality at Time 1 were significantly correlated with anhedonic depressive symptoms at Time 2. Also, positive emotionality at Time 1 was significantly correlated with supportive relationships at Time 2. Last, negative emotionality at Time 1 was significantly correlated with independent and dependent stressors at Time 2.

Does Baseline Temperament Predict Anhedonic Depressive Symptoms?

We started with an omnibus regression analysis with all variables (i.e., sex, initial anhedonic depression, PE, NE) and all higher order interactions (i.e., all 2 way interactions and the 3-way interaction of NE × PE × Sex). Baseline anhedonic depressive symptoms scores were always entered first to control for initial symptoms in order to examine prospective changes in symptom levels. This procedure enabled prediction of residual change scores in anhedonic depressive symptoms. Results are presented in Table 2. In particular, the 3-way interaction was not significant, so that was cut from analyses, β=−0.01, *ns*. Also, the 2-way interactions of PE × Sex and NE × Sex were non-significant, so these were trimmed from the analyses, β=0.06, *ns*, and β=0.12, *ns*, respectively. These non-significant interactions suggest that sex did not moderate any of the significant associations between temperament and prospective changes in anhedonic depressive symptoms, so effects appear to apply equally to both boys and girls.

The findings presented next are for the regression analyses that investigated our key hypotheses after all non-significant interactions were eliminated. Specifically, we examined if sex and baseline levels of NE and PE predicted prospective elevations in anhedonic depressive symptoms 5 months later (Time 2). As predicted, adolescents' baseline levels of NE significantly predicted prospective elevations in anhedonic depressive symptoms at Time 2, after controlling for baseline anhedonic depressive symptoms, β=0.31, p<0.001. In addition, adolescents' baseline levels of positive emotionality significantly predicted anhedonic depressive symptoms at Time 2 after controlling for baseline anhedonic depressive symptoms, β=−0.18, p< 001. A significant NE × PE interaction also emerged, such that adolescents with high levels of NE and low levels of PE at baseline experienced the greatest increase in anhedonic depressive symptoms at Time 2, β=−0.10, p<0.05.

To examine the form of the NE × PE interaction, anhedonic depressive symptoms scores were calculated by inserting specific values for NE and PE scores (i.e., 1 SD above and below the sample's mean) into the regression equation summarized in Table 2. Results are shown in Fig. 1. Relatively low levels of anhedonic depressive symptoms were seen among (1) adolescents who exhibited low levels of both NE and PE; (2) adolescents who exhibited low levels of NE but high levels of PE; and (3) adolescents who exhibited high levels of both NE and high PE. Consistent with our hypothesis and the tripartite theory, adolescents who exhibited high levels of NE but low levels of PE demonstrated the greatest prospective elevations in anhedonic depressive symptoms. Follow-up analyses were conducted to examine if the slope of the relationship between NE and anhedonic depressive symptoms significantly differed from 0. Analyses indicated that residual change scores in anhedonic depressive symptoms varied as a function of level of NE for those adolescents who reported low levels of PE, t(346)=3.80, p<0.001 and for adolescents experiencing high levels of PE t(346)=3.35, p<0.001.

Mediation Analyses

Given that the NE × PE interaction predicted prospective changes in anhedonic depressive symptoms, we then conducted mediation analyses (Baron & Kenny 1986) to see if either stressors (independent or dependent) or supportive relationships would mediate the longitudinal association between the baseline NE × PE interaction and later elevations in anhedonic depressive symptoms. Separate mediation analyses were conducted. The first analysis examined if independent or dependent stressors would account for the relationship between Time 1 NE × PE and Time 2 anhedonic depressive symptoms. The second analysis tested if supportive relationships would account for the relationship between Time 1 NE × PE and anhedonic depressive symptoms. Statistical procedures described by Baron and Kenny (1986) were used to test if the conditions for mediation had been satisfied. Specifically, the following conditions must be met to satisfy criteria for mediation: (1) the predictor variable is significantly correlated with the outcome variable; (2) the predictor variable is significantly correlated with the mediator; (3) the mediator is significantly correlated with the outcome variable, after controlling for the predictor variable; and (4) the association between the outcome variable and the predictor variable is decreased once the mediator is included in the analysis. Once the conditions for mediation were tested, procedures outlined by MacKinnon and colleagues (MacKinnon et al. 2002) were used to examine the significance of the indirect effect.

Condition 1, that NE × PE would predict prospective changes in anhedonic depressive symptoms, was demonstrated above. However, the next condition, that NE × PE would predict the mediator, was not supported as the NE × PE interaction was not significantly associated with dependent stressors, β=0.02, *ns*, independent stressors, β=0.21, *ns*, or supportive relationships, β=0.14, *ns*. Thus, no further mediation analyses were conducted to examine the link between NE × PE at Time 1 predicting changes in anhedonic symptoms of depression at Time 2. Although mediation analyses could not be examined for NE × PE, we continued to test for mediation and examined if stressors mediated the association between baseline NE and later anhedonic symptoms of depression and if supportive relationships mediated link between baseline PE and later anhedonic symptoms of depression, as these were the a priori hypothesized developmental pathways posited to account for the association between baseline temperament and later elevations in anhedonic depressive symptoms.

Do Stressors Mediate the Relationship Between Baseline NE and Anhedonic Depressive Symptoms?

Condition 1: *Baseline negative emotionality predicts anhedonic depressive symptoms at Time 2.* As demonstrated earlier, this condition was met in that Time 1 NE predicted Time 2 anhedonic depressive symptoms after controlling for Time 1 anhedonic depressive symptoms.

Condition 2: *Baseline negative emotionality predicts stressors.* Next, we tested the association between Time 1 negative emotionality (the predictor variable) and Time 2 stressors (the mediator) after controlling for Time1 anhedonic depressive symptoms. Results indicated that Time 1 NE significantly predicted dependent stressors at Time 2 after controlling for Time 1 anhedonic depressive symptoms, β=0.24, p<0.01. However, and consistent with our hypothesis, baseline NE did not significantly predict independent stressors at Time 2, β=0.11, *ns*, thus further tests were not conducting using independent stressors as the mediator.

Condition 3: *Dependent stressors predict anhedonic depressive symptoms at Time 2, after controlling for negative emotionality.* Regression analyses were performed to determine if dependent stressors at Time 2 would predict anhedonic depressive symptoms at Time 2, after controlling for negative emotionality and anhedonic

depressive symptoms at Time 1. Results indicated that this condition for mediation was met. Dependent stressors significantly predicted Time 2 anhedonic depressive symptoms while controlling for negative emotionality and anhedonic depressive symptoms at Time 1, β=0.21, p<0.01.

Condition 4: *The association between anhedonic depressive symptoms and negative emotionality is decreased once dependent stressors are included in the analysis.* Last, we examined the extent to which the prospective association between baseline negative emotionality and Time 2 anhedonic depressive symptoms was reduced by the inclusion of Time 2 dependent stressors in the model. The association between negative emotionality and Time 2 anhedonic depressive symptoms was reduced by 12% once dependent stressors were included (β from 0.41, p<0.01 to 0.36, p<0.01). The Sobel test (Sobel 1982) indicated that dependent stressors significantly mediated the prospective link between Time 1 negative emotionality and Time 2 anhedonic depressive symptoms, z=4.26, p<0.001. Figure 2 shows path coefficients (standardized betas) for the mediation model for the prospective association between negative emotionality and anhedonic depressive symptoms, as mediated by dependent stressors.

Do Supportive Relationships Mediate the Relationship Between Baseline PE and Anhedonic Depressive Symptoms?

Condition 1: *Baseline positive emotionality predicts anhedonic depressive symptoms at Time2.* As shown in Table 2, Time 1 PE predicted Time 2 anhedonic depressive symptoms after controlling for Time 1 anhedonic depressive symptoms.

Condition 2: *Baseline positive emotionality predicts supportive relationships.* The association between Time 1 PE and the mediator, supportive relationships, was examined next. Time 1 PE significantly predicted supportive relationships at Time 2 after controlling for Time 1 anhedonic depressive symptoms, β=0.22, p<0.01, thus meeting the second condition for mediation.

Condition 3: *Supportive relationships predict anhedonic depressive symptoms at Time 2, after controlling for positive emotionality.* Based on the significant results from the previous two conditions for mediation, regression analyses were performed to determine if supportive relationships at Time 2 would predict anhedonic depressive symptoms at Time 2, after controlling for positive emotionality and anhedonic depressive symptoms at Time 1. This condition for mediation was met, in that supportive relationships significantly predicted Time 2 anhedonic depressive symptoms while controlling for positive emotionality and anhedonic depressive symptoms at Time 1, β=−0.26, p<0.01.

Condition 4: *The association between anhedonic depressive symptoms and positive emotionality is decreased once supportive relationships are included in the analysis.* To meet the final condition for mediation, we examined how much of the association between baseline positive emotionality and Time 2 anhedonic depressive symptoms was reduced by the inclusion of supportive relationships at Time 2. The association between positive emotionality and Time 2 anhedonic depressive symptoms was reduced by 19% once supportive relationships were included (β from −0.30, p<0.01 to −0.26, p<0.01), indicating partial mediation. The Sobel test (Sobel 1982) indicated that supportive relationships significantly mediated the prospective link between Time 1 positive emotionality and Time 2 anhedonic depressive symptoms, z=−3.19, p<.001. Path coefficients (standardized betas) for this effect are shown in Fig. 3.

Additional Moderation Analyses

We conducted additional moderation analyses to identify whether there were adolescents in specific groups, based on the risk factors assessed in this study, who might be at increased risk for prospective elevations in anhedonic depressive symptoms. Specifically, we tested if baseline temperament interacted with psychosocial risk factors (i.e., supportive relationships, dependent stressors, or independent stressors) to predict changes in anhedonic depressive symptoms. Three separate regression models were run in which interaction terms were entered in each equation. Similar to previous analyses, we included all variables and higher order interactions, eliminating all non-significant interactions to present current findings. Baseline anhedonic depressive symptoms scores were always entered first to control for initial symptoms in order to examine prospective changes in symptom levels.

Results from linear regression analyses indicated a significant PE × Supportive relationships interaction, such that adolescents with low levels of PE and low levels of supportive relationships reported the greatest prospective increase in anhedonic depressive symptoms, β=1.53, p<0.001. Results are shown in Fig. 4. Simple slope analyses indicated that residual change scores in anhedonic depressive symptoms varied as a function of level of PE for those adolescents who reported low levels of supportive relationships, $t(346)=-4.52$, p<0.001 and for adolescents experiencing high levels of supportive relationships $t(346)=-4.75$, p<0.001. All additional 2– and 3–way interactions were non-significant.

Discussion

Numerous prospective longitudinal studies have demonstrated a link between temperament and psychopathology (e.g., Caspi et al. 1996), yet relatively little research has examined the mechanisms through which temperament dimensions lead to the development of depressive symptoms. The primary goal of this study was to examine theoretically motivated mediating mechanisms through which levels of positive emotionality and negative emotionality contribute to increases in anhedonic depressive symptoms.

Results indicated that baseline levels of NE and PE were associated with anhedonic depressive symptoms at Time 2, after controlling for baseline anhedonic depressive symptoms. More importantly, support was found for pathways through which baseline temperament predicted changes in anhedonic depressive symptoms. First, dependent stressors partially mediated the link between baseline NE and later anhedonic depressive symptoms after controlling for baseline levels of anhedonic depressive symptoms. Second, supportive relationships partially mediated the relationship between baseline PE and anhedonic depressive symptoms after controlling for baseline depressive symptoms. These findings held for both boys and girls. In addition to finding mediation, results indicated the presence of a significant NE × PE interaction, such that adolescents with low levels of PE and high levels of NE experienced the greatest prospective increase in anhedonic depressive symptoms. Results revealed one additional significant interaction: baseline PE interacted with supportive relationships at Time 2 to predict changes in anhedonic depressive symptoms, such that individuals with low PE and low levels of supportive relationships experienced the greatest increase in anhedonic depressive symptoms.

This study builds upon earlier theoretical and empirical work that investigates the link between temperament and psychopathology. In particular, the mediation model that used dependent stressors as the mediator examined one aspect of Hankin and Abramson's (2001) elaborated cognitive vulnerability-transactional stress model of depression. Specifically, it examined the piece of Hankin and Abramson's model that proposes that individuals with increased levels of temperamental/personality risk (i.e., negative emotionality) will experience an increase in stressors, which in turn will lead to elevations in depressive symptoms. Support for the

mediation pathway in the present study indicated that adolescents with high levels of NE report more dependent negative events in their lives over time, and this exposure to greater stressful experiences, in turn, contributed to elevated levels of anhedonic depressive symptoms.

Relatedly, we identified another developmental pathway through which baseline temperament leads to prospective changes in depressive symptoms. Specifically, the mediation model in which supportive relationships partially mediated the relationship between baseline PE and anhedonic depressive symptoms highlights the importance of interpersonal factors in the development of depressive symptoms during adolescence. Although a large body of theoretical and empirical evidence indicates that interpersonal relationships play a significant role in risk for psychopathology, and particularly depression (for a review, see Rudolph et al. 2008), to our knowledge, no study to date has examined the mediating role of supportive relationships in the link between positive emotionality and depressive symptoms.

In addition to mediation, moderation provides another way to conceptualize a developmental pathways perspective to understanding the relationship among temperament, psychosocial risk factors, and depressive symptoms. The current study found evidence for two interactions, allowing us to identify specific groups that are at increased risk for experiencing prospective elevations in depressive symptoms. The interaction between baseline PE and supportive relationships at Time 2 emphasizes the role of interpersonal relationships in risk for anhedonic depressive symptoms. Results showed that adolescents with low PE and low supportive relationships showed the greatest elevation in anhedonic depressive symptoms at Time 2. These results indicate that perceived low levels of supportive relationships may be a particularly potent stressor for adolescents with low levels of positive emotionality. Building upon results of the current study, it will be important for future studies to investigate what constructs may mediate the relationship between PE × supportive relationships and anhedonic depressive symptoms. For example, adolescents with low levels of PE and low levels of supportive relationships may engage in greater levels of excessive reassurance seeking in those few relationships they do have, ultimately contributing to increase in depressive symptoms (e.g., Haeffel et al. 2007; for review see Van Orden & Joiner 2006).

Evidence for the interaction between baseline levels of NE and PE expands upon Clark & Watson's (1991) tripartite model of anxiety and depression and empirical studies that have investigated how levels of NE and PE are associated with increased risk for depression and anxiety (e.g., Joiner & Lonigan 2000; Krueger 1999; Caspi et al. 1996). Despite the significance of the NE × PE interaction term, it is important to note that it explained only 1% of the variance in changes in anhedonic depressive symptoms, substantially lower than the variance explained by the NE main effect and somewhat lower than the variance explained by the PE main effect. We located only one prior study that found support for an interaction between NE and PE in predicting depression, and this study did so with an inpatient sample of youth (Joiner & Lonigan 2000). To our knowledge, the current study is the first to find that levels of NE and PE interact to predict the development of depressive symptoms in a community sample of adolescents. Given the known biases in psychiatric samples (e.g., Goodman et al. 1997), the replication of this effect and extension to a general sample of youth is an important contribution to the current literature. In addition, future research should investigate potential mediators for the relationship between NE × PE and anhedonic depressive symptoms and also examine the specificity of this interaction to symptoms of depression versus anxiety

Despite evidence indicating that stressors mediated the link between baseline NE and anhedonic depressive symptoms and that supportive relationships mediated the link between baseline PE and anhedonic depressive symptoms, neither stressors nor supportive relationships mediated the relationship between the baseline NE × PE interaction term and Time 2 anhedonic depressive symptoms. This is of particular interest because it suggests that the mechanisms

through which adolescents with high levels of NE coupled with low levels of PE experience increases in depressive symptoms differ from the mechanisms through adolescents with either high NE or low PE experience increases in depressive symptoms. Importantly, adolescents with high levels of NE, those with low levels of PE, and those with the combination of high NE and low PE exhibit increases in depressive symptoms, yet this appears to occur through different mechanisms. The processes through which the latter group experiences increases in depressive symptoms have yet to be identified. Variation in pathways to a common outcome is consistent with the process of equifinality, in which individuals begin at different points (i.e., individuals with high NE, individuals with low PE, and individuals with both high NE and low PE), and can arrive at a common outcome (i.e., increases in depressive symptoms) through a diverse set of processes.

There are several strengths to this study. First, and most important, the study integrated theoretical and empirical evidence drawn from the disparate temperament and depression literatures to investigate developmental mediating pathways. Specifically, examination of mediating mechanisms through which temperament leads to increases in anhedonic depressive symptoms can move the field forward in its understanding of risk for depression and possible prevention techniques. In addition, the study used longitudinal data from a relatively large (n=350) community sample of adolescents, increasing power and the generalizability of the results. Last, prospective longitudinal research has shown that rates of depression increase dramatically for individuals between the ages of 15 and 18 (from 3% to 17%) (Hankin et al. 1998), especially for girls. Because this study examined 11 to 17 year-olds (M=14.5 years), we were able to investigate prospective elevations in depressive symptoms and potential moderation of mediating pathways during a time when adolescents are at most risk.

Despite these strengths, the study had several limitations. First, only self-report methods were used. This may be considered problematic, especially for assessing stressors (e.g., Monroe 2008). Yet, tempering this concern, research by Lewinsohn et al. (2003) has directly compared self-report and interviewer-based measures of stressors and found that the majority of self-reported stressors were verified in the more comprehensive stressor interview, and the interview-identified stressors showed the same associations with depression as did the self-reported stressors. Thus, this research suggests that interviewer-based and self-report measures of negative life events yield comparable results in the prediction of depression. Second, Time 2 temperament was not examined in this study, which precluded testing addition etiological models of temperament and depression, such as the scar model. Future research would benefit from including assessment of temperament at multiple time points, along with depressive symptoms, to examine the vulnerability, pathoplasty and scar models. Last, the present study was limited because we did not obtain clinical diagnoses of depression; rather, a subset of depressive symptoms was used as the outcome measure. Yet, research with youth suggests that depression is dimensionally structured (Hankin et al. 2005), not qualitatively defined, so use of a continuous measure of depressive symptoms is likely an appropriate first step in this program of research. Given the strong theory and replicable set of findings based on adolescent reports, the next step will be to use multiple informants and diagnostic interviews to assess clinically significant depressive episodes and the more severe end of the depression dimension.

Conclusion and Future Directions

As Tackett (2006) noted in a recent review, in contrast to adult models of personality and psychopathology, developmental researchers have not yet integrated adequately and summarized research on temperament and psychopathology in children and adolescents, making it difficult to achieve a comprehensive understanding of this complex relationship. This study adds to the current literature because it integrated prominent theories of temperament in childhood and adolescence to aid in the conceptualization of temperament

dimensions and their relationship to depression. It also incorporated several theoretically motivated temperament-psychopathology models in order to inform the hypotheses and interpretations of results.

Specifically, results from the mediation analyses provided support for the pathoplasty model, indicating that adolescents' pre-existing temperamental characteristics can partially influence the development of anhedonic depressive symptoms. Also, results from the NE × PE interaction and the PE × supportive relationships interaction suggested that particular groups are at increased risk for experiencing anhedonic depressive symptoms, providing support for the vulnerability model. The current study was not designed to test the scar model or the spectrum model, and it will be important for future studies to investigate the extent to which these models explain the association between temperament and depression. As Tackett (2006) and other researchers have suggested (e.g., Compas et al. 2004), it is unlikely that any single model will fully explain the relationship between temperament and psychopathology.

In sum, this study used a prospective longitudinal design to investigate theoretically motivated mediating mechanisms to identify pathways that lead from baseline levels of temperament to increases in anhedonic depressive symptoms. In addition, this study examined if the relationship between temperament and depressive symptoms varied across several psychosocial risk factors. Future research should investigate how multiple factors may influence the temperament-psychopathology relationship, and if this relationship varies across disorders and/or across individuals. Specifically, it will be important to identify other mediators in the relationship between temperament and depression. This way, we can better understand the processes through which temperament leads to depression, hopefully increasing the effectiveness of prevention and intervention efforts.

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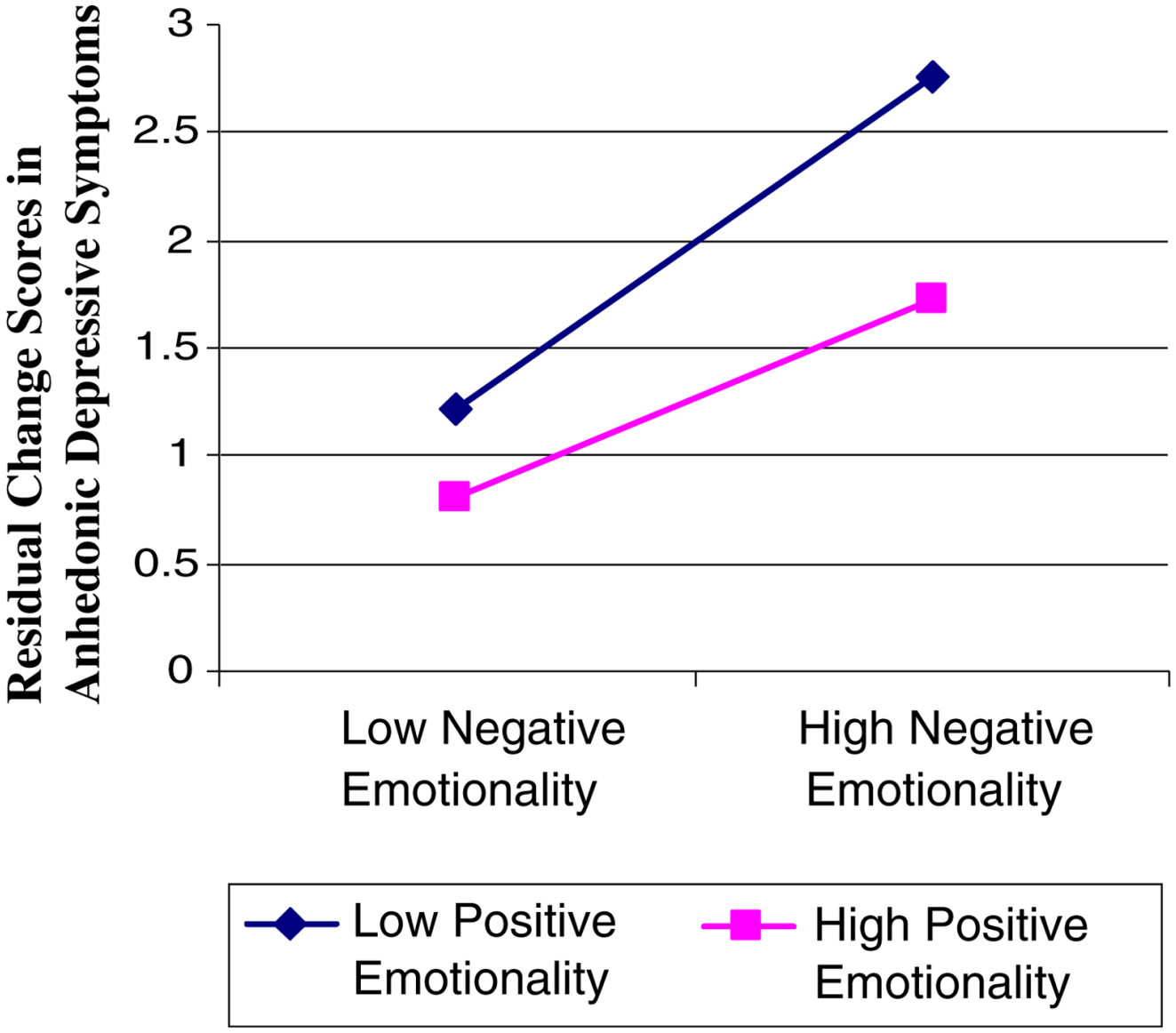

Fig. 1.
Positive emotionality × negative emotionality predicts increases in anhedonic depressive symptoms

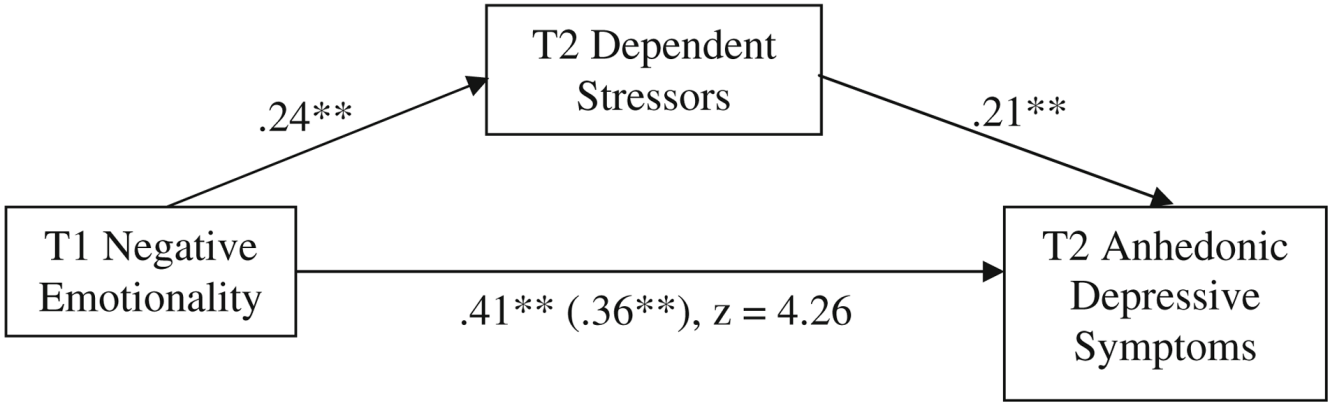

Fig. 2.
Mediation model for the association between negative emotionality and anhedonic depressive symptoms by dependent stressors. Values on paths are standardized betas.

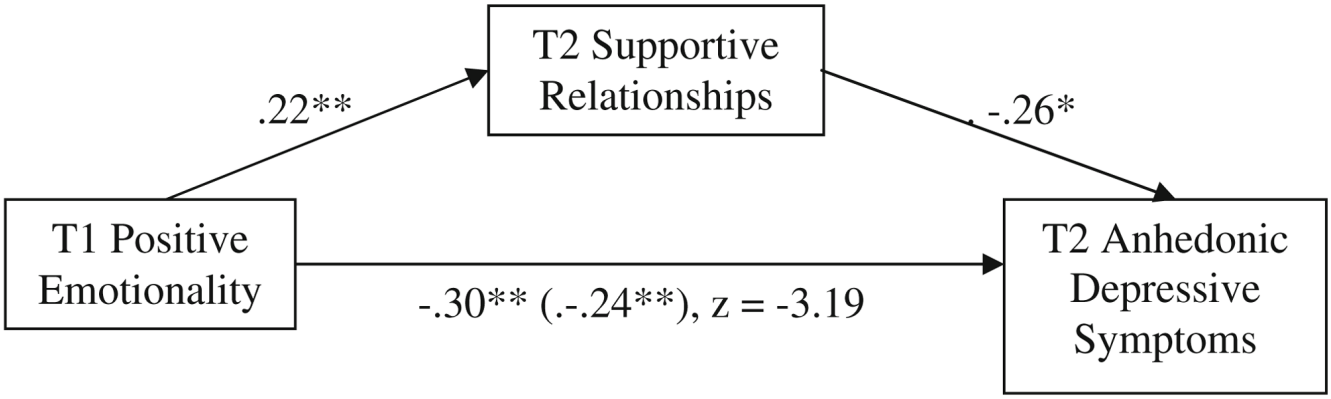

Fig. 3.
Mediation model for the association between positive emotionality and anhedonic depressive symptoms by supportive relationships. Values on paths are standardized betas

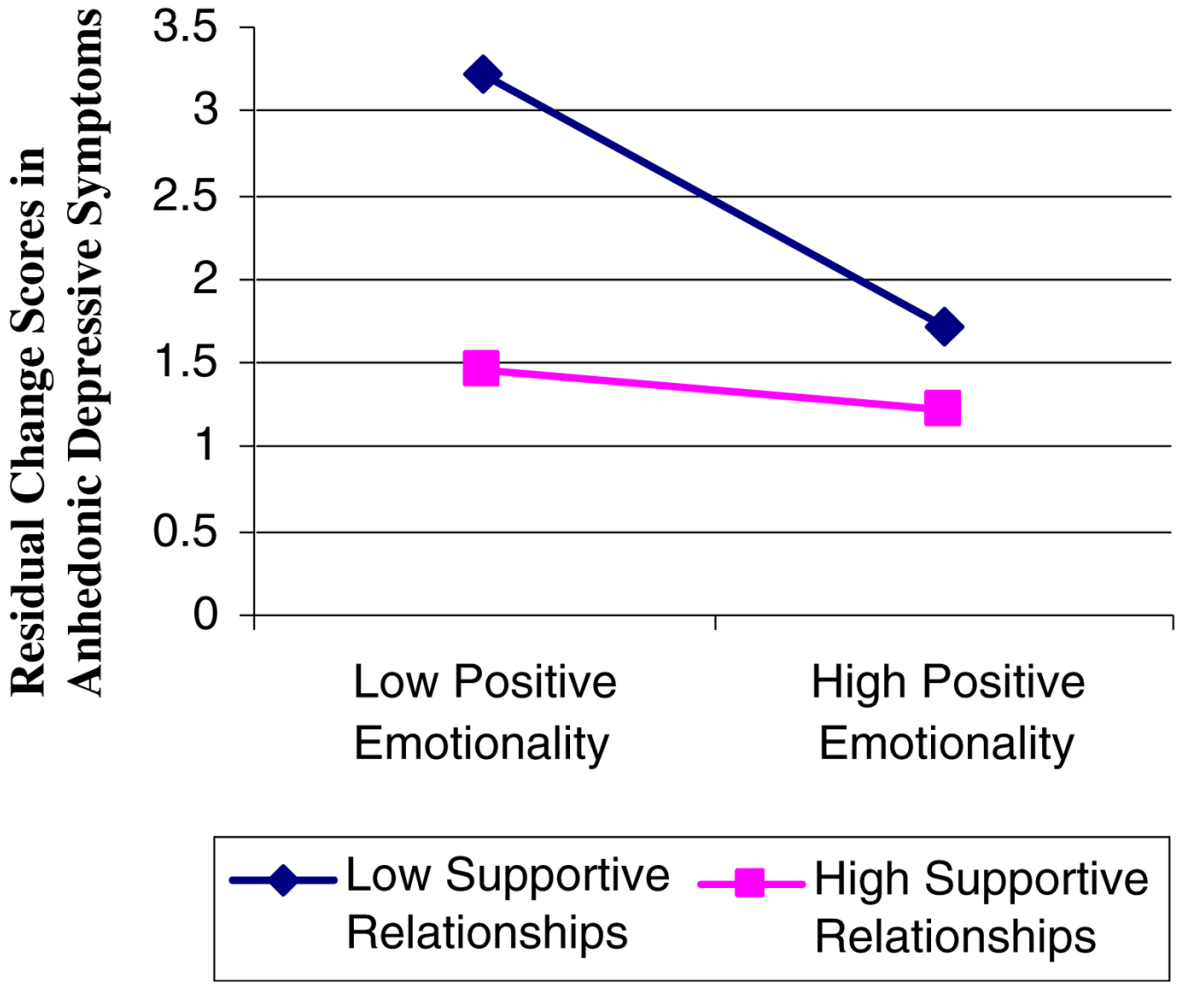

Fig. 4.
Positive emotionality × supportive relationships predicts increases in anhedonic depressive symptoms

Table 1

Descriptive Statistics and Correlations among Main Measures (*N*=350)

Measure	1	2	3	4	5	6	7	8
1. NE1	1							
2. PE1	−0.41***	1						
3. CDI1	0.22***	−0.22***	1					
4. CDI2	0.48***	−0.37***	0.38***	1				
5. Dep stress2	0.27***	−0.17**	0.15**	0.35***	1			
6. Ind stress2	0.21***	−0.14*	0.05	0.24***	0.76***	1		
7. Support Rel2	−0.27**	0.25***	−0.20***	−0.38***	−0.35***	−0.22***	1	
8. Sex	0.16**	0.14*	0.04	0.16**	0.18**	0.13*	−0.18**	1
M	2.26	3.01	2.55	2.63	43.49	17.77	17.54	
SD	0.52	0.52	1.83	1.97	13.24	5.93	2.70	

NE1 = Negative emotionality at Time 1; PE1 = Positive Emotionality at Time 1; CDI1 = Anhedonic items on the CDI at Time 1; CDI2 = Anhedonic items on the CDI at Time 2; Dep stress2 = Dependent stressors at Time 2; Ind stress2 = Independent stressors at Time 2; Support Rel2 = Supportive Relationships at Time 2; Sex was coded such that males=0 and females=1

* p<0.05

** p<0.01

*** p<0.001

Table 2

CNE × PE Interaction Predicts Changes in Anhedonic Depressive Symptoms

Predictors in set	t for within set predictors	df	β	ΔR^2
Main effect variables				
CDI (baseline)	5.60**	1, 343	0.26	0.15
Sex	2.45*	1,342	0.11	0.02
NE	6.30**	1, 341	0.31	0.15
PE	−3.61**	1, 340	−0.18	0.03
2-Way Interaction Terms				
NE × PE	−2.14*	5,339	−0.10	0.01

CDI (baseline) = Anhedonic items on the CDI at baseline; NE = Negative Emotionality at Time 1; PE = Positive Emotionality at Time 1 Model F=37.04**; R^2 =35.3

* p<0.05

** p<0.01