Exploring Depressive Personality Traits in Youth: Origins, Correlates, and Developmental Consequences

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Abstract

Research suggests that depressive personality (DP) disorder may represent a persistent, trait-based form of depression that lies along an affective spectrum ranging from personality traits to diagnosable clinical disorders (Klein & Bessaha, 2009). A significant gap in this area of research concerns the development of DP and its applicability to youth. The present research explored the construct of DP traits in youth. Specifically, this study examined the reliability, stability, and validity of the construct, potential origins of DP traits, and the developmental consequences of DP traits. A sample of 143 youth (M age = 12.37 years, SD = 1.26) and their caregivers completed semi-structured interviews and questionnaires on two occasions, separated by a 12 month interval. The measure of DP traits was reliable and moderately stable over time. Providing evidence of construct validity, DP traits were associated with a network of constructs, including a negative self-focus, high negative and low positive emotionality, and heightened stress reactivity. Moreover, several potential origins of DP traits were identified, including a history of family adversity, maternal DP traits, and maternal depression. Consistent with hypotheses regarding their developmental significance, DP traits predicted the generation of stress and the emergence of depression (but not nondepressive psychopathology) during the pubertal transition. Finally, depression predicted subsequent DP traits, suggesting a reciprocal process whereby DP traits heighten risk for depression, which then exacerbates these traits. These findings support the construct of DP traits in youth, and suggest that these traits may be a useful addition to developmental models of risk for youth depression.

Keywords
depressive personality; development

The construct of depressive personality (DP) or temperament dates back to the ancient Greeks, and was frequently discussed in the classical descriptive psychopathology (Kraepelin, 1921; Schneider, 1958) and psychoanalytic (Bemporad, 1976; Berliner, 1966; Laughlin, 1967) literatures. During the past two decades, there has been a resurgence of interest in DP (Huprich, 1998; Klein, 1990; Phillips, Gunderson, Hirschfeld, & Smith, 1990), and depressive personality disorder (DPD) was included in the Appendix of the fourth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM; American Psychiatric Association, 1994) as a condition requiring further study.
Few epidemiological data have been reported, but the best estimates suggest a prevalence of approximately 2% in community samples of adults (Ørstavik, Kendler, Czajkowski, Tambs, & Reichborn-Kjennerud, 2007) and 22%–26% in outpatient mental health settings (Klein, 1990; McDermut, Zimmerman, & Chelminski, 2003). Much of the empirical research on DPD focuses on nosological issues, chiefly its association with Axis I mood disorders (Klein & Bessaha, 2009; Ryder, Bagby, Marshall, & Costa, 2005). This research indicates significant associations, but the degree of overlap is relatively modest. Although longitudinal data are limited, DP traits appear to be moderately stable over periods of up to 10 years (Laptook, Klein, & Dougherty, 2006). Young adults with DPD have an increased risk of later developing dysthymic disorder (Kwon et al., 2000). Overall, the relationship between DPD and Axis I mood disorders remains controversial; however, many investigators believe that DPD is best conceptualized as lying at the milder end of a continuum with Axis I depressive disorders (Akiskal, 1989; Klein, 1999).

A Developmental Perspective on Depressive Personality

Minimal theory or research on DPD has considered the applicability of this construct prior to late adolescence or has explored its etiology. Unfortunately, by neglecting to consider early signs and potential precursors of DPD, researchers have overlooked the opportunity to better understand the development of this disorder. In particular, it is reasonable to consider whether these atypical traits emerge early in life, perhaps crystallizing into adult forms of pathology over the course of development.

Supporting this view, theories of normative personality development increasingly take the perspective that there is significant overlap between early temperamental traits and adult personality dimensions (for reviews, see Caspi & Shiner, 2006; Shiner, 2005). Although early theories of temperament highlighted the genetic and biological origins of individual differences, more recent conceptualizations acknowledge social and environmental contributions (Rothbart & Bates, 2006). Likewise, adult dimensions of personality are believed to have their roots in both genetic and environmental influences (Caspi & Shiner, 2006; Shiner, 2005). Although specific manifestations of personality may differ somewhat across development, consistent with the idea of heterotypic continuity (Cicchetti & Cohen, 2006), there is growing consensus that a common structure and overarching dimensions (e.g., negative emotionality, constraint, extroversion) underlie individual differences throughout the lifespan (for a review, see Caspi & Shiner, 2006). Moreover, research shows that early temperamental traits predict personality and competence in adolescence and adulthood (Shiner, Masten, & Roberts, 2003).

This gradual convergence in theories of normative individual differences in temperament and personality sets the stage for the question of whether extremes in these dimensions, conceptualized in terms of personality pathology or disorder in adults, similarly characterize individual variations in youth. Traditionally, personality disorders are viewed as a chronic and persistent form of pathology that cannot be identified or diagnosed until late adolescence or early adulthood, when personality is believed to become consolidated and stable. Indeed, this view is endorsed by the DSM, as reflected in the proposed criteria for DPD. According to a developmental psychopathology perspective, however, it may be possible to detect early signs of dysfunction before the emergence of a diagnosable disorder (Cicchetti & Cohen, 2006); youth with these early signs may be at risk for the development of clinically significant psychopathology later in life (Caspi & Shiner, 2006). Indeed, based on growing evidence that personality pathology emerges during childhood, it has been suggested that developmental research should consider the role of early temperamental traits in the development of personality disorders (Caspi & Shiner, 2006; Crick, Murray-Close, & Woods, 2005; Crick,
The goal of the present research was to explore the early emergence of DP traits. Four specific issues were addressed: (1) the reliability, stability, and construct validity of DP traits in youth; (2) the developmental origins of DP traits; (3) the developmental consequences of DP traits; and (4) the influence of depression on subsequent DP traits. To assess DP traits, we adopted an interview measure developed in adults (Klein, 1990) that assessed the seven key criteria of DPD as outlined by Akiskal (1983): (a) quiet, introverted, passive, and nonassertive; (b) gloomy, pessimistic, serious, and incapable of fun; (c) self-critical, self-reproaching, and self-derogatory; (d) skeptical, hypercritical, and hard to please; (e) conscientious, responsible, and self-disciplined; (f) brooding and given to worry; and (g) preoccupied with negative events, feelings of inadequacy, and personal shortcomings. These criteria were deemed to be developmentally appropriate for youth in light of research suggesting that the broad dimensions of individual differences underlying these criteria (i.e., negative emotionality/neuroticism, constraint, (lack of) positive emotionality/extroversion, and (lack of) agreeableness) are quite comparable across developmental stages (Caspi & Shiner, 2006). Moreover, the interview items and examples tapped manifestations of these criteria that were considered appropriate in youth.

**Construct Validity of Depressive Personality Traits**

The first goal was to examine whether DP traits in youth were associated with a network of characteristics that one would expect to cohere with this personality type. Based on the idea that DP traits may reflect the intersection between normative individual differences and pathological variations in cognition, emotion, and behavior, we selected three relevant domains of developmental competence in which to assess construct validity: sense of self, emotionality, and the ability to cope with stress.

Individuals with DPD show a tendency to engage in harsh self-judgments and excessive self-blame, dwelling on their shortcomings and failures, and comparing themselves unfavorably to others. They have low self-esteem and may feel excessively guilty (American Psychiatric Association, 2000; Klein & Bessaha, 2009). Although most youth show increasing levels of introspection, self-consciousness, and social comparison across development, particularly during the adolescent transition (Arnett, 1999; Higgins, 1991), some youth may show a more extreme preoccupation with negative aspects of the self, as reflected in this dimension of DP. Thus, as one form of construct validity, we examined whether DP traits in youth were associated with a negative self-focus, characterized by a tendency to engage in negative self-evaluation and social comparison.

DPD also is marked by persistent and pervasive feelings of gloominess and joylessness; individuals with DPD are unable to experience enjoyment and are pessimistic about their future (American Psychiatric Association, 2000; Klein, 1990). Learning how to down-regulate negative affect and to up-regulate positive affect are critical developmental tasks (Chaplin & Cole, 2005); when children’s mastery of these tasks is compromised, they may experience persistent high levels of negative emotionality and low levels of positive emotionality. It has been proposed that individuals with DPD experience both types of emotional disruption (Clark, Watson, & Mineka, 1994; Shea & Hirschfeld, 1996). Indeed, research demonstrates that DPD in adults is negatively correlated with positive affect and extroversion, and is positively correlated with negative affect and neuroticism (Klein & Shih, 1998). As a second form of construct validity, therefore, we examined whether DP traits in youth were associated with higher levels of negative affect and low levels of positive affect (i.e., low positive affect, high anhedonia).
Although not a defining feature, individuals with DPD are likely to show heightened stress reactivity. The development of adaptive cognitive, emotional, behavioral, and physiological responses to stress is likely guided by both learning and temperamental characteristics (Compas et al., 2004). For individuals who are prone to brooding, pessimism, and feelings of inadequacy, stressful events may take on heightened meaning and present a greater sense of threat. Moreover, high levels of self-blame and preoccupation with one’s shortcomings may cause individuals to take excessive responsibility for negative events, attributing events to their own wrongdoings rather than to external circumstances. Individuals who are passive, doubt their self-worth, and are pessimistic about the future also may be less likely to engage in active efforts to cope with stressors and the accompanying negative emotions. Indeed, if individuals’ psychological resources are allocated toward brooding about negative emotions, past negative experiences, and possible future negative events, they may feel overwhelmed by stressors, lacking the resources to respond purposefully and effectively. Thus, DP traits may be linked to more intense emotional reactions and maladaptive responses to stress. Consistent with these ideas, adults with DPD endorse more negative attributions about stress and higher stress reactivity than do those without DPD (Klein, 1990). More generally, negative emotionality contributes to greater appraisals of threat and maladaptive responses to stress in youth (Eisenberg, Fabes, Nyman, Bernzweig, & Pinuelas, 1994; Lengua & Long, 2002). As a third form of construct validity, therefore, we examined whether DP traits in youth were associated with heightened stress reactivity, as reflected in inaccurate cognitive appraisals of stress (i.e., overestimation of the stressfulness of events and one’s personal contribution to events), heightened negative emotional reactivity (i.e., sadness, fear, anger, and guilt), and maladaptive responses to stress (i.e., fewer purposeful, adaptive responses and more involuntary, dysregulated responses).

### Family Origins of Depressive Personality

The second goal was to explore the potential origins of DP traits. Theory and research implicate both genetic and environmental contributions to individual differences in temperament and personality (Caspi & Shiner, 2006). Consequently, we examined three potential family risk factors that might reflect these types of contributions: a history of family adversity, maternal DP traits, and maternal depression.

Family adversity (e.g., interparental conflict, long-term parent-child separations, chaotic family living circumstances), maternal DP traits, and maternal depression may contribute to the emergence of DP traits through several pathways. First, these family risk factors may reflect a shared genetic liability to affective disturbance. Second, family risk may be transmitted through explicit or implicit socialization processes. For example, through exposure to severe adversity or to maternal DP traits and depression, youth may develop the belief that they have little control over their lives and that others are not consistent or trustworthy sources of support, leading to negative views of the self and to a critical and gloomy perspective on others and the future (Geiger & Crick, 2001; Rudolph, Kurlakowsky, & Conley, 2001). Youth with disrupted family relationships also may fail to develop the capacity for effective regulation of emotions, leading to a more intense and dysregulated experience of negative emotions (Geiger & Crick, 2001). Third, youth may learn to model parental cognition and behavior in ways that create a vulnerability to DP traits. For example, youth of mothers with DP traits may model their negative preoccupations, brooding, or passivity.

Although relatively little research has explored the origins of DPD, there is some evidence for genetic influences on the transmission of DP traits. In a large community sample of twins, Ørstavik et al. (2007) found significant additive genetic effects on liability to DP traits. Interestingly, heritability was greater among women than men, and some of the genes contributing to DP traits differed between the sexes. In addition, there is strong evidence for a
shared familial liability between DPD and mood disorders. A number of studies have found an increased rate of mood disorders in the relatives of adults with DPD (Klein, 1990; Klein & Miller, 1993; Kwon et al., 2000; McDermut et al., 2003). Finally, several studies have reported increased DPD or DP traits in the adolescent offspring and adult relatives of adults with major depressive disorder (Klein et al., 1988; Klein, 1999). These findings support both the familial transmission of DP traits, in part attributable to genetic effects, and the familial co-aggregation of DP traits and mood disorders.

Because all three of the proposed family risk factors could operate through multiple pathways, we were not able to determine whether associations between family risk and youth DP traits reflect genetic or environmental contributions. Thus, the goal was to identify potential risk factors for future in-depth study. We also examined whether links between maternal psychopathology and youth DP traits were specific to depression versus nondepressive disorders. Finally, we examined whether these family risk factors made a unique contribution to youth DP traits versus clinical depression by adjusting for youths’ history of depression.

### Developmental Consequences of Depressive Personality Traits

The third goal was to investigate the developmental consequences of DP traits. In particular, we were interested in the idea that DP traits would portend depression in the context of the transition through adolescence. Drawing from developmental theories regarding the linkage between personality traits and psychopathology (Caspi & Shiner, 2006; Compas, Connor-Smith, & Jaser, 2004), we hypothesized that DP traits would increase the likelihood that youth select and shape their environments in ways that heighten risk for subsequent depression through the process of stress generation.

Youth with DP traits may generate greater stress in their lives for several reasons. Their gloomy and self-critical nature may interfere with their ability to develop relationships and with their efforts to achieve competence in other life domains (e.g., academic success). Moreover, their criticism and skepticism of others may elicit negative reactions, creating conflict in their relationships. As noted earlier, these youth may overestimate the stressfulness of events and show difficulties responding adaptively to problems. These tendencies may cause them to overreact to everyday hassles, thereby exacerbating their consequences. For example, interpreting a minor negative comment by a friend as overly threatening and showing an exaggerated emotional reaction may create tension within the friendship. Likewise, overreacting to minor setbacks in schoolwork may interfere with learning and achievement. Youth with DP traits also may select into less adaptive relationships, perhaps because of a lack of choices or because they feel that they are not worthy of close and supportive relationships. Collectively, these difficulties may create high levels of stress that heighten risk for depression.

This emerging process of stress generation and depression may be exacerbated during the adolescent transition. During this challenging stage of development, youth must negotiate significant physical, psychological, and social reorganization (Graber, 2003). Those with prior vulnerabilities, such as DP traits, may encounter particular difficulty navigating these complex changes. Because these youth may enter the adolescent transition with a compromised sense of self, disruptions in their emotionality, and impaired coping abilities, they are likely to face greater challenges. Adolescence also presents youth with a variety of choices, such as selection of friends and romantic partners and decisions about one’s desired level of school engagement. Youth with DP traits may make poor choices, fostering the selection of riskier social contexts. Thus, we hypothesized that the contribution of DP traits to stress generation and depression would be particularly salient during the transition to adolescence. Moreover, these adverse consequences may be accentuated in youth who undergo pubertal maturation prior to their peers, as earlier maturation is associated with a greater likelihood of generating stress, entering

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*Dev Psychopathol. Author manuscript; available in PMC 2010 October 1.*
into risky contexts, and experiencing disruptions in mood and behavior relative to later maturation (Rudolph, 2008; Weichold, Silbereisen, & Schmitt-Rodermund, 2003).

To test this hypothesized process, we investigated whether DP traits predicted heightened depression in youth with more advanced development (i.e., older, more advanced pubertal status, earlier pubertal timing) relative to youth with less advanced development (i.e., younger, less advanced pubertal status, later pubertal timing). Moreover, we examined whether heightened stress generation accounted for these depressive consequences of DP traits. In this context, we also examined the discriminant validity of DP traits for predicting subsequent depression versus nondepressive psychopathology, consistent with research showing that adults with and without DPD do not differ in rates of nondepressive disorders (Klein, 1990).

**Effect of Depression on Depressive Personality Traits**

The final goal was to examine whether depression significantly predicted subsequent DP traits. Despite stability in temperament and personality across development, research also suggests significant change, particularly during earlier stages of life (Shiner, 2005). Because personality traits are still under construction during childhood, experiencing significant psychopathology may leave a developmental “scar” (Casi & Shiner, 2006; Rohde, Lewinsohn, & Seeley, 1994). In particular, youths’ early affective experiences may influence the development of DP traits. During a depressive episode, youth experience declines in self-worth, as well as emotional and social disruptions. It is possible that experiencing an episode of depression early in life, prior to the consolidation of youths’ sense of self and others and the maturation of emotion regulation abilities, leaves a lasting scar in the form of persistent DP traits.

**Study Overview**

In sum, this study examined the origins, correlates, and consequences of DP traits in youth. Each set of analyses explored the possibility of sex differences. This is important in light of evidence that some of the genetic influences contributing to DP traits may differ as a function of sex, and that the heritability of DP traits may be greater in females than males (Ørstavik et al., 2007). However, because there has been little research on sex differences in the origins, correlates, and consequences of DP traits, no firm hypotheses were made. Nonetheless, drawing from theory and evidence regarding sex differences in the etiology, mediating processes, and consequences of depressive disorders (Hankin & Abramson, 2001; Rudolph, 2009), it was thought that DP traits may be particularly strongly associated with interpersonal dysfunction (e.g., heightened reactivity to interpersonal stress and interpersonal stress generation) in girls relative to boys. In the context of a two-wave longitudinal study, youth and their caregivers completed semi-structured interviews to assess DP traits, psychopathology, lifetime family adversity, recent stress, and stress reactivity. Participants also completed questionnaire measures of negative self-focus, emotionality, responses to stress, and puberty.

**Method**

**Participants**

Participants were 143 youth (77 girls, 66 boys; M age = 12.37 years, SD = 1.26; 78.3% White, 21.7% minority) and their female caregivers (88.1% biological mothers, 11.9% other) recruited from several Midwestern towns. Families represented a range of socioeconomic classes as reflected in income level [15.2% below $30,000; 50.0% $30–59,999; 21.0% $60,000–89,999; 13.8% over $90,000]. These youth were selected from 167 families participating in a longitudinal study of depression based on the availability of DP traits data.¹ Youth were selected for the longitudinal study based on school-wide screenings with the Children’s Depression Inventory (Kovacs, 1992). We selected potential participants (4th – 8th graders; n
with a range of CDI scores, over-sampling slightly for youth with severe symptoms (15.8% of the screening sample, 20.3% of targeted youth, and 24.1% of recruited youth had scores > 18). Participants from the screening sample were recruited for the longitudinal study based on several factors, including CDI scores, a maternal caregiver in the home, and proximity (within one hour) to the university, until the targeted sample was successfully recruited.

Youth whose families did and did not consent to participate in the study did not differ in sex, \( \chi^2(N = 468, df = 1) = .39, ns \), ethnicity (white versus minority), \( \chi^2(N = 468, df = 1) = .02, ns \), or CDI scores, \( t(280) = 1.11, ns \). Participants (\( M = 12.41, SD = 1.19 \)) were slightly younger than nonparticipants (\( M = 12.65, SD = .89 \)), \( t(275) = 2.28, p < .05 \). Reasons for nonparticipation included being busy or not interested (\( n = 229 \)), having moved or being unreachable (\( n = 40 \)), chronic rescheduling (\( n = 5 \)), and failing to meet eligibility criteria (\( n = 27 \)). Exclusion criteria included having a non-English speaking maternal caregiver or a severe developmental disability that interfered with the assessment. Relevant data were available for 140 (98%) of the original sample at Wave 2 (\( W_2 \)).

**Procedures**

Families were recruited through phone calls to the primary female caregivers. Interested families completed a three- to four-hour initial assessment. Caregivers provided written informed consent, and youth provided written assent. Youth and their caregivers were interviewed separately and completed several questionnaires. Two different interviewers conducted the diagnostic and life stress interviews to avoid biases during the interviewing process. Using the same procedures, a follow-up interview was completed one year later (\( M \) time interval = 1 year, 1 week). To compensate families for their time, caregivers were given a monetary reimbursement and youth were given a gift certificate at each assessment.

**Measures**

**Assessment of Depressive Personality Traits**—Interviewers individually administered a 16-item assessment of DP traits (Klein, 1990). These items were originally developed to assess Akiskal’s (1983) criteria for DPD. Both youth and caregivers reported on traits in youth, and caregivers reported on traits in themselves. Based on conceptualizations of DPD described by Klein and colleagues (Klein, 1990; Klein & Shih, 1998), this measure was intended to capture stable personality traits rather than acute symptoms of depression. Interviewers first provided a general probe that indicated an interest in traits that youth and caregivers displayed throughout their lives. To avoid confounding acute symptoms of depression with DP traits, this probe specified that participants should consider traits expressed during nonsymptomatic periods. Interviewers then asked participants to indicate the extent to which they would describe youth or caregivers as someone who generally displayed a variety of traits (e.g., “Critical of yourself. Hard on yourself. Feels like you could or should do better than you do.” “Puts yourself down a lot.” “Thinks about things all the time that make you feel bad, unhappy, tense, or upset.” “Thinks a lot about bad things that have happened, things that you hadn’t done well, or feel like you’re not as good as other kids.” “Gloomy, as if everything goes wrong, and things won’t work out very well in your life.” “Complains a lot.”). Interviewers read each question aloud and asked follow-up questions with specific probes (e.g., requests for examples, clarifications). After gathering the relevant information, interviewers provided ratings of how typical each trait was of youth and caregivers on a scale of 1 (Not at all) to 3 (Definite). A best-estimate approach (Klein, Ouimette, Kelly, Ferro, & Riso, 1994) was used to integrate information across youth and caregiver reports on youths’ traits.

\(^1\)Missing data were due to the fact that the DP traits measure was added to the protocol after some families had already completed the first assessment.
A reliability analysis of the youth measure revealed that three items (quiet-introverted, extremely conscientious, and self-disciplining) showed low item-total correlations (average = .09; average for other items = .49). Moreover, deletion of these three items from the scale increased the alpha. Thus, these three items were omitted. Because these traits are not included in the DSM-IV criteria for DPD, their omission increased convergence with the DSM version of the construct (Klein & Bessaha, 2009). Scores were computed as the average of the 13 remaining items, with higher scores reflecting more DP traits. Comparable scores were computed for the caregiver measure. The measure had adequate internal consistency (α = .85 in youth; α = .83 in caregivers) and was moderately stable over time in youth (r = .56, p < .001) and caregivers (r = .70, p < .001). Research has established strong concurrent and discriminant validity of the construct of DPD, including showing that it is distinct from depressive disorders (Klein, 1990; Klein & Shih, 1998).

Not surprisingly, given the low base rates of DPD in community samples (Ørstavik et al., 2007), mean scores on the DP traits measure were fairly low (M = 1.31, SD = .32, Range = 1.26). Although there was no significant overall effect of sex, t(141) = .46, ns, there were significant Sex × Pubertal Status, β = .37, t(135) = 2.94, p < .01, and Sex × Pubertal Timing, β = .41, t(135) = 3.28, p < .01, interactions (the Sex × Age interaction was nonsignificant, β = .14, t(139) = 1.08, ns). Decomposition of these interactions revealed a similar pattern. Boys showed higher levels of DP traits than did girls when they were less advanced in their pubertal development and later in their pubertal timing, whereas girls showed higher levels of DP traits than did boys when they were more advanced in their pubertal development and earlier in their pubertal timing; the sex difference was marginally significant at less advanced pubertal development (M girls = 1.23, SD = .21; M boys = 1.39, SD = .37; t = 1.79, p < .10) and significant at earlier pubertal timing (M girls = 1.41, SD = .40; M boys = 1.21, SD = .20; t = −2.14, p < .05), whereas the other differences were nonsignificant.

**Assessment and Coding of Psychopathology**—Interviewers individually administered the Schedule for Affective Disorders and Schizophrenia for School-Age Children-Epidemiologic Version-5 (K-SADS-E; Orvaschel, 1995) to youth and caregivers to assess youth psychopathology. Interviewers included a faculty member in clinical psychology, a post-doctoral student in clinical psychology, several trained psychology graduate students, and a post BA-level research assistant. All interviews were coded through consultation with a clinical psychology faculty member or post-doctoral student. Consensual diagnoses were assigned using a best-estimate approach (Klein et al., 1994) to integrate information across youth and caregiver report.

For each period and type of psychopathology, interviewers used *DSM-IV* criteria (American Psychiatric Association, 2000) to assign ratings for each disorder on a 5-point scale: 0 = No symptoms, 1 = Mild symptoms, 2 = Moderate symptoms, 3 = Diagnosis with mild to moderate impairment, and 4 = Diagnosis with severe impairment. Ratings were assigned for both diagnosable episodes and subthreshold symptoms of psychopathology. Based on *DSM-IV* criteria, these ratings considered the number, severity, frequency, duration, and resulting impairment of the reported symptoms. Thus, subclinical symptoms (i.e., mild or moderate) reflected the presence of symptoms that failed to meet full *DSM-IV* criteria for the disorder (e.g., the youth had fewer than the required number of symptoms or had the required number of symptoms for less than the required duration). Ratings were summed across episodes and within major categories of psychopathology to create separate continuous symptom scores for (1) youths’ lifetime history of psychopathology (up until one year prior to the Wave 1 [W1] assessment) and (2) youths’ recent history of psychopathology (within the past year) at each assessment. Separate symptom summary scores were calculated for depressive disorders (e.g., major depression, dysthymia) and nondepressive disorders (e.g., anxiety and externalizing disorders). Higher ratings reflect more severe symptoms within a single diagnostic category.
the presence of symptoms from multiple categories, and/or multiple periods of psychopathology (for similar rating approaches, see Hammen, Shih, & Brennan, 2004; Rudolph, Flynn, Abaied, Groot, & Thompson, in press). Thus, these scores represent composite indexes of several different markers of severity.

This type of continuous index is consistent with contemporary conceptualizations, derived in part from taxometric analyses, that view psychopathology as best represented by dimensional continuums rather than discrete categories (Fergusson, Horwood, Ridder, & Beautrais, 2005; Hankin, Fraley, Lahey, & Waldman, 2005; Shih et al., 2006). Validity of the depression summary scores was established through significant correlations with self-report measures of depressive symptoms (average $r = .53, p < .001$). To establish the validity of the nondepressive psychopathology summary scores, a mean score was taken of all of the subscales on the Achenbach Child Behavior Checklist (Achenbach & Rescorla, 2001) with the exception of the subscale that included depressive symptoms. The nondepressive psychopathology score was significantly correlated with this composite score ($r = .49, p < .001$). Based on independent coding of audiotapes of 42 interviews, strong inter-rater reliability was found for the ratings (one-way random-effects intraclass correlation coefficient [ICCs] ≥ .91).

Of the 143 youth, 13% had a lifetime history of clinical depression (a rating of 3 or 4 for at least one episode) prior to the year before the W1 assessment; an additional 18% had a lifetime history of subclinical depressive symptoms (i.e., a rating of 1 or 2 for at least one episode). Forty percent had a lifetime history of a nondepressive disorder prior to the year before the W1 assessment; an additional 34% had a lifetime history of subclinical nondepressive symptoms. At W1, 13% met diagnostic criteria for clinical depression within the past year; an additional 17% experienced subclinical depressive symptoms. Forty percent met diagnostic criteria for a nondepressive disorder within the past year; an additional 36% experienced subclinical nondepressive symptoms.

Assessment of Negative Self-Focus—Youth completed an 8-item questionnaire assessing negative judgments about their abilities or worth, concerns about social evaluation, and a focus on social comparison (e.g., “I was disappointed in myself.” “I worried about what other people thought of me.” “I felt like other kids were better than me.”). They rated how much each item described them on a 5-point scale (1 = Not at all to 5 = Very Much). The measure had high internal consistency ($\alpha = .90$) and was relatively stable over time ($r = .57, p < .001$). Providing evidence of validity, the negative self-focus measure correlates significantly with a variety of associated constructs tapping negative self-appraisals, including heightened social-evaluative concerns, negative relational self-views, lower levels of perceived control, and a negative attributional style ($|r|s = .39 – .57, p < .001$). Scores were computed as the average of the 8 items, with higher scores reflecting more negative self-focus.

Assessment of Emotionality—Youth completed the Children’s Depression Inventory (Kovacs, 1992) and the Revised Manifest Anxiety Scale (Reynolds & Richmond, 1978). Prior factor-analytic research (Joiner, Catanzaro, & Laurent, 1996) has identified a subset of 11 items from these two measures that form a negative affect factor (e.g., sadness, worry, irritability, nervousness). The validity of this subscale was established in the original sample through correlations with scores on the Positive and Negative Affect Schedule (Joiner et al., 1996). In the present sample, the subscale had adequate internal consistency ($\alpha = .79$) and was relatively stable over time ($r = .58, p < .001$). Each item was standardized, and negative affect scores were computed as the average of the 11 items, with higher scores reflecting more negative affect.

Youth completed subscales, adapted from the Mood and Anxiety Symptom Questionnaire (MASQ; Watson et al., 1995), assessing their experience of anhedonia (8 items; e.g., “I did not enjoy anything.” “I did not feel like doing things that I usually like to do.”) and positive affect...
(5 items; e.g., “I felt really happy.” “I felt like I had a lot to look forward to, like good things would happen to me.”). They rated how much each item described them on a 5-point scale (1 = Not at all to 5 = Very Much). The subscales had high internal consistency (αs = .87 and .85, respectively) and were relatively stable over time (rs = .60 and .41, respectively, ps < .001). Scores were computed as the average of the items within each subscale, with higher scores reflecting more anhedonia and positive affect.

Assessment of Life Stress—The Youth Life Stress Interview (Rudolph & Flynn, 2007), a semi-structured interview that uses the contextual threat method (Brown & Harris, 1978; Rudolph & Hammen, 1999), was administered to determine the nature and intensity of episodic life stress experienced by youth during the year prior to each interview. Specific, detailed probes are used to elicit objective information separately from caregivers and youth about youths’ experience of episodic events in several life domains (e.g., school, peer group, parent-child relationships). First, the interviewer provides a general probe regarding the occurrence of stressful events in the past year. Following the general probe, questions are asked about the occurrence of specific events within a variety of domains (e.g., failure of a test, a friend moving away, an argument with a parent). Based on detailed follow-up questions concerning the timing, duration, and context of the stress, interviewers prepare a narrative summary of each event. This information is presented to an independent team of coders with no knowledge of the youth’s diagnosis or subjective reaction to the events.

Integrating information from youth and caregivers, coders assigned two ratings on a 5-point scale: (a) the objective stress or negative impact of the event for a typical child in those circumstances (events that were rated “no negative stress/impact” were excluded from analyses), and (b) the dependence of each event, or the extent to which the youth contributed to the event’s occurrence. Following previous protocol (Davila, Hammen, Burge, Paley, & Daley, 1995; Rudolph et al., 2000), events with dependence ratings of 3 or higher were categorized as dependent to reflect the fact that the youth was at least an equal partner in determining the occurrence of the event. Given the present focus on stress generation, separate composite scores were calculated for dependent stress within the family, peer, and noninterpersonal (e.g., academic) domains by summing the impact ratings across relevant events. To assess reliability, 160 life events were coded by two independent teams. High reliability was found for ratings of objective episodic stress (ICC = .90), dependence (ICC = .96), and event content (Cohen’s κ = .92).

Assessment of Cognitive Appraisals and Emotional Reactivity—Cognitive appraisals and emotional reactivity were assessed in the context of the episodic life events reported by youth. Immediately following youths’ report of each event, they provided ratings on a five-point scale (1 = Not at All to 5 = Very Much) of event stressfulness (“How stressful or how much of a problem was [event]?”) and of their contribution to the event’s occurrence (“How much do you think that [event] was caused by something that you did?”). To examine youths’ stress estimations, standardized residual scores for each event were computed by regressing youths’ appraisals of stressfulness onto the team’s objective stress rating. Higher scores on this variable represent an overestimation of stress. Likewise, to examine youths’ dependence estimations, standardized residual scores for each event were computed by regressing youths’ appraisals of their contribution to the event’s occurrence onto the team’s objective dependence rating. Higher scores on this variable represent an overestimation of dependence. This approach (i.e., calculation of standardized residuals) is typical in studies designed to compare objective and subjective ratings (e.g., Cole, Martin, Peeke, Serocynski, & Hoffman, 1998; Krackow & Rudolph, 2008). Average stress-estimation and dependence-estimation scores were calculated separately for interpersonal and noninterpersonal events by taking the mean of the residualized scores across the relevant events. Scores could not be
calculated for mother-only reported events or in cases where the sample size for calculating the standardized residual scores was less than three.

For each event, youth also provided ratings on a five-point scale (1 = Not at All to 5 = Very Much) of the extent to which they felt sad, scared/worried/nervous, angry/mad, and guilty following the event. Average emotional reactivity scores were calculated separately for interpersonal and noninterpersonal events by taking the mean of all four emotion ratings across the relevant events.

**Assessment of Responses to Stress**—The Responses to Stress Questionnaire (RSQ; Connor-Smith et al., 2000) was used to assess youths’ effortful coping versus involuntary, dysregulated responses to stress. This measure distinguishes engagement with, and disengagement from, stressors. It includes four subscales, which demonstrated high internal consistency in the present sample: effortful engagement (e.g., problem solving, emotion regulation, cognitive restructuring; 21 items, \( \alpha = .86 \)), effortful disengagement (e.g., denial, avoidance, wishful thinking; 12 items, \( \alpha = .78 \)), involuntary engagement (e.g., rumination, emotional and physiological arousal; 15 items, \( \alpha = .93 \)), and involuntary disengagement (e.g., inaction, emotional numbing; 12 items; \( \alpha = .89 \)). Convergent validity and retest reliability have been established for these subscales (Connor-Smith et al., 2000).

Youth reported how much they engaged in each type of response to peer stressors (e.g., fighting with other kids, having problems with a friend) on a 4-point scale (1 = Not at All to 4 = A Lot). Consistent with previous research (Connor-Smith et al., 2000; Flynn & Rudolph, 2007), to correct for base-rate differences in the endorsement of responses to stress (Compas, Connor-Smith, Saltzman, Thomsen, & Wadsworth, 2001), proportion scores were calculated as the total score for each subscale divided by the total score on the RSQ. Higher scores reflect higher levels of each type of response to stress. The four subscales were relatively stable over time (average \( r = .54, ps < .001 \)).

**Assessment of Lifetime Family Adversity**—The lifetime adversity section of the Youth Life Stress Interview (Rudolph & Flynn, 2007) was administered to youth and their caregivers at \( W_1 \). This interview assesses youths’ exposure to severe negative family life events and circumstances across their lifetime (up until one year prior to the \( W_1 \) interview) using the same type of contextual threat method as the assessment of recent episodic stress. First, a general probe was used to assess exposure to particularly stressful events and circumstances, emphasizing that the focus was on very serious rather than everyday problems. Second, specific probes were used to assess the occurrence of the following types of adversity: death of a close family member, extended separation from parents, parental separation or divorce, exposure to severe marital conflict, and severe chronic illness of a close family member. Follow-up questions were asked as needed to assess the context of the event. Using a best-estimate approach (Klein et al., 1994) to integrate information from youth and caregivers, an independent rating team provided a consensual rating on a 10-point scale that reflected the overall level of adversity experienced by youth. For reliability purposes, two independent teams of raters coded a subset of 40 interviews. High reliability was found for ratings of adversity (ICC = .99).

**Assessment of Maternal Psychopathology**—Interviewers individually administered the nonpatient version of the Structured Clinical Interview for the DSM (SCID IV-NP; First, Spitzer, Gibbon, & Williams, 1996) to caregivers to assess maternal psychopathology. Each period and type of psychopathology was coded using the same procedures as used for the K-SADS. Again, separate continuous symptom scores were calculated for (1) mothers’ lifetime history of psychopathology (up until one year prior to the \( W_1 \) assessment) and (2) mothers’ recent history of psychopathology (within the past year) at the \( W_1 \) assessment. Validity of the
depression summary scores was established through significant correlations with the anhedonia subscale of the MASQ (Watson et al., 1995) (average $r = .35$, $p < .001$). Questionnaire data were not available to assess the validity of the nondepressive psychopathology composites, but similar ratings have been used in prior research (e.g., Hammen, Burge, Daley, Davila, Paley, & Rudolph, 1995). Strong inter-rater reliability was found for the ratings (ICCs ≥ .83).

Of the 126 biological mothers, 37% had a lifetime history of clinical depression (a rating of 3 or 4 for at least one episode) prior to the year before the W₁ assessment; an additional 12% had a lifetime history of subclinical depressive symptoms (i.e., a rating of 1 or 2 for at least one episode). Fifty percent had a lifetime history of a nondepressive disorder prior to the year before the W₁ assessment; an additional 35% had a lifetime history of subclinical nondepressive symptoms. At W₁, 20% met diagnostic criteria for clinical depression within the past year; an additional 14% experienced subclinical depressive symptoms. Forty-two percent met diagnostic criteria for a nondepressive disorder within the past year; an additional 32% experienced subclinical nondepressive symptoms.

Assessment of Pubertal Timing

Pubertal maturation: Two measures of pubertal status were administered. First, youth and caregivers completed the Pubertal Development Scale (Petersen, Crockett, Richards, & Boxer, 1988). This measure includes five questions that assess stage of growth spurt, body hair, skin changes, voice changes and facial hair (boys), and breast development and menarcheal status (girls) using a 4-point scale (1 = No development to 4 = Development is complete). Onset of menarche is rated using a dichotomous response (1 = No, 4 = Yes). Reliability and validity are well-established (Brooks-Gunn, Warren, Rosso, & Gargiulo, 1987). Because strong correlations were found between youth and caregiver reports ($r = .88$, $p < .001$, for girls, and $r = .72$, $p < .001$, for boys), composites were formed for each item by averaging across informants ($α = .86$ for girls and boys). When information was available from only one informant, this information was used.

Second, youth and caregivers rated youths’ stage of development based on line drawings of the five Tanner stages (Morris & Udry, 1980). Girls were rated on breast development and pubic hair growth; boys were rated on genital development and pubic hair growth. Participants checked which of the drawings most closely matched the youth’s current stage of development. Validity of this measure has been established through significant associations with physician ratings based on physical exams (Morris & Udry, 1980; Schlossberger, Turner, & Irwin, 1992). In the present sample, strong correlations were found between girls’ and caregivers’ reports ($r = .83$, $p < .001$, for breast development; $r = .69$, $p < .001$, for pubic hair development). Moderate correlations were found between boys’ and caregivers’ reports ($r = .47$, $p < .01$, for genital development; $r = .65$, $p < .001$, for pubic hair development). Composites were formed for each of the two items by averaging across informants. When information was available from only one informant, this information was used. At W₁, participants represented a range of pubertal stages (35% Stage 2 or below; 43% between Stage 2 and Stage 4; 22% Stage 4 or above).

Confirmatory factor analyses in this data set yielded well-fitting measurement models using the seven items (five PDS and two Tanner) as indicators for a latent variable (Conley & Rudolph, 2009). Thus, a composite score was created by averaging the seven items, standardized within sex. Because prior research has used the Tanner stages alone as an index of pubertal development (Dorn et al., 1990; Morris & Udry, 1980), scores were calculated for a few participants with Tanner data only, or Tanner data plus one or two PDS items. To create an index of pubertal timing, residualized scores were computed separately for girls and boys by regressing pubertal status onto chronological age. Higher scores reflected earlier maturation relative to one’s agemates.
Results

Overview of Analyses

The first set of analyses examined the construct validity of the DP traits measure. It was hypothesized that DP traits would be associated with a negative self-focus, higher negative affect and lower positive affect, and heightened stress reactivity (i.e., inaccurate cognitive appraisals, emotional reactivity, and maladaptive responses to stress). The second set of analyses examined the potential origins of DP traits. It was hypothesized that a history of family adversity, maternal DP traits, and maternal depression (but not nondepressive psychopathology) would be associated with DP traits in youth. The third set of analyses examined the predictive validity of DP traits within a developmental context. It was hypothesized that DP traits would predict depression (but not nondepressive psychopathology) more strongly in youth who were more advanced in their development than in youth who were less advanced in their development; these analyses also examined whether stress generation accounted for these effects. The fourth set of analyses examined whether prior depression in youth predicted subsequent DP traits.

Establishing Construct Validity of Depressive Personality Traits

Correlations were computed among W_1 variables to examine the construct validity of DP traits. Table 1 presents these correlations across the total sample as well as separately for girls and boys. Fishers r-to-Z transformations were used to compared the size of the correlations across sex. Partial correlations also were computed that adjusted for youths’ lifetime history of depression. These analyses provided a stringent test of these associations by determining whether DP traits made an independent contribution that was not accounted for by a history of depression.

Negative self-focus—DP traits were significantly associated with a negative self-focus in girls and boys. This association was significantly stronger in girls than in boys, Z = 2.03, p < .05.

Emotionality—DP traits were significantly associated with higher levels of negative affect and lower levels of positive affect in both girls and boys; this association was significantly stronger in girls than in boys for negative affect, Z = 1.98, p < .05, but did not significantly differ for positive affect. DP traits were significantly associated with heightened anhedonia in girls but not in boys; this association was significantly stronger in girls than in boys, Z = 2.66, p < .01.

Stress reactivity—Within the interpersonal domain, DP traits were significantly associated with stress estimation, dependence estimation, and emotional reactivity in girls, and with emotional reactivity in boys. Within the noninterpersonal domain, DP traits were significantly associated with stress estimation and emotional reactivity in girls and were marginally associated with stress estimation in boys. DP traits were significantly associated with less effortful engagement and greater involuntary engagement and disengagement in girls, and with less effortful and greater involuntary engagement in boys. The size of these associations did not significantly differ across sex, except for noninterpersonal dependence estimation, Z = 2.04, p < .05, and marginally for involuntary disengagement, Z = 1.66, p < .10.

Summary—Overall, correlational analyses supported the validity of the DP traits construct. As expected, DP traits were associated with a negative self-focus, more negative affect and less positive affect, inaccurate cognitive appraisals, heightened negative emotional reactivity, and maladaptive responses to stress. Overall, the pattern of correlations was more consistent and somewhat stronger for girls, although there were only a few significant differences across
sex. Examination of the partial correlation coefficients (see Table 1) revealed that the observed associations held even after adjusting for youths’ lifetime history of depression, although some of the coefficients became smaller. Overall, however, these analyses suggest that DP traits make a unique contribution to these constructs that is not fully accounted for by a history of depression.

**Origins of Depressive Personality Traits**

Correlations were computed among W1 variables to examine the hypothesis that family risk would be associated with youth DP traits. Analyses for maternal DP traits and maternal psychopathology were conducted specifically for youth living with their biological mothers (n = 126); results for these variables were similar, although slightly weaker, when all mothers were included. Because of the low frequency of dysthymia, analyses focused on major depression. Moreover, because of the low frequency of past year substance use, analyses focused on lifetime history of substance use. Table 2 presents these correlations across the total sample as well as separately for girls and boys. Once again, Fishers r-to-Z transformations were used to compared the size of the correlations across sex.

Lifetime family adversity and maternal DP traits were significantly associated with youth DP traits in girls; lifetime family adversity was marginally associated with youth DP traits in boys. In girls, lifetime and past year maternal major depression but not nondepressive psychopathology were associated with DP traits. In boys, lifetime maternal major depression but not past year maternal major depression or nondepressive psychopathology was significantly associated with DP traits. The size of these associations did not significantly differ across sex. Examination of the partial correlation coefficients (see Table 2) revealed that the associations in girls remained significant for family adversity, maternal DP traits, and past year maternal major depression but not for lifetime history of maternal major depression after adjusting for youths’ lifetime history of depression. The associations in boys became nonsignificant for both lifetime family adversity and lifetime history of maternal major depression.

**Summary**—In girls, as anticipated, a history of family adversity, maternal DP traits, and maternal major depression, but not maternal nondepressive psychopathology, were associated with DP traits. The effect of lifetime history of maternal depression was accounted for by girls’ lifetime history of depression, suggesting that the contribution of lifetime maternal depression to girls’ DP traits is shared with its contribution to youth depression; however, the association remained significant for past year maternal depression. In boys, lifetime history of maternal major depression was significantly associated with DP traits, but this association was accounted for by boys’ lifetime history of depression.

**Developmental Consequences of Depressive Personality Traits**

**Prediction of psychopathology**—Hierarchical multiple regression analyses were conducted to examine whether W1 youth DP traits predicted W2 past year depression and nondepressive psychopathology, adjusting for W1 past year psychopathology. It was predicted that DP traits would predict subsequent depression but not nondepressive psychopathology, particularly for more developed youth relative to less developed youth. Separate regressions were conducted to examine depression and nondepressive psychopathology. For each regression, the main effects of W1 past year psychopathology, W1 mean-centered DP traits, W1 mean-centered development (i.e., age, pubertal status, or pubertal timing), and sex were entered at the first step; the two-way interactions were entered at the second step; the three-way (DP Traits × Development × Sex) interaction was entered at the third step. Significant interactions were interpreted by solving the unstandardized regression equation to predict W2 psychopathology from W1 DP traits at low (−1 SD), moderate (mean), and high (+1 SD)
levels of W₁ development (Aiken & West, 1991). To ensure that effects were not accounted for by youths’ lifetime history of depression, each regression was rerun adjusting for this variable.

For the prediction of depression, the three-way interactions were all nonsignificant, βs < .15, ts(127–131) < 1.63, ns; thus the regressions were rerun collapsing across sex (see Table 3). Each analysis yielded a significant main effect of DP traits; this main effect was qualified by significant DP Traits × Pubertal Status and DP Traits × Pubertal Timing interactions. Adjusting for lifetime history of depression, the regressions yielded similar DP Traits × Pubertal Status, β = .19, t(127) = 3.48, p < .01, and DP Traits × Pubertal Timing, β = .18, t(127) = 3.13, p < .01, interactions. As depicted in Figure 1a, DP traits predicted greater depression in youth with more advanced (β = .42, t(128) = 4.61, p < .001) but not less advanced (β = .03, t(128) = .38, ns) pubertal development. Similarly, as depicted in Figure 1b, DP traits predicted greater depression in youth with earlier (β = .45, t(128) = 4.74, p < .001) but not later (β = .07, t(128) = .88, ns) pubertal timing.

For the prediction of nondepressive psychopathology, the three-way interaction was significant for age, β = −.11, t(131) = −2.30, p < .05, but not for pubertal status, β = −.08, t(127) = −1.39, ns, or pubertal timing, β = .03, t(127) = .72, ns. However, follow-up of the significant three-way interaction for age revealed small and nonsignificant interactions for both girls, β = −.05, t(70) = −1.01, ns, and boys, β = .07, t(60) = 1.34, ns, making it difficult to interpret this effect. Thus, the regressions were rerun collapsing across sex (see Table 3). These analyses yielded nonsignificant main effects of DP traits and nonsignificant interactions.

**Mediation by stress generation**—The next set of analyses examined whether the contribution of the DP Traits × Pubertal Status and DP Traits × Pubertal Timing interactions to depression were accounted for by the generation of stress (i.e., test of mediated moderation). Several conditions must be satisfied to demonstrate mediated moderation (Muller, Judd, & Yzerbyt, 2005). Condition 1 requires that the magnitude of the overall effect of the independent variable (i.e., DP traits) on the dependent variable (i.e., depression) depends on the moderator (i.e., puberty); Condition 1 was previously satisfied for pubertal status and timing (see Table 3). Condition 2 requires that the mediator (i.e., stress generation) accounts for the overall moderation effect. For this to be the case, either the effect of DP traits on stress generation depends on puberty and the average partial effect of stress generation on depression is significant (non-zero) and/or the partial effect of stress generation on depression depends on puberty and the average effect of DP traits on stress generation is significant (non-zero). As a result, the moderation of the residual direct effect of DP traits on depression is reduced compared to the overall moderated effect (Muller et al., 2005). To investigate Condition 2 in the present analyses, two regression analyses were conducted for each type of stress. The first regression examined whether the path from DP traits to stress generation was moderated by puberty (Condition 2a). The second regression examined whether stress generation or the Stress Generation × Puberty interaction predicted depression after adjusting for the main and interactive effects of DP traits and puberty (Condition 2b), and whether the overall interactive effect of DP traits and puberty on depression was reduced upon inclusion of stress generation and the Stress Generation × Puberty interaction (Condition 2c).

First, hierarchical multiple regression analyses were conducted to examine whether W₁ pubertal status or W₁ pubertal timing moderated the association between W₁ DP traits and

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2The two-way interactions also were examined separately for anxiety and externalizing psychopathology. For predicting anxiety, only the DP traits × Pubertal Status interaction was significant, β = −.07, t(131) = 2.56, p < .05. Decomposition of the interaction revealed that DP traits predicted greater anxiety in youth with less advanced (β = .10, t(131) = 2.35, p < .05) but not more advanced (β = −.05, t(131) = 1.20, ns) pubertal development. For predicting externalizing psychopathology, none of the main effects of DP traits or DP Traits × Development interactions were significant.
W₂ stress generation; separate analyses were conducted for family, peer, and noninterpersonal stress (Condition 2a). W₂ stress generation represented the level of dependent stress that was experienced between the two waves, making it an appropriate variable for mediation. W₁ depression, W₁ stress generation, and the mean-centered main effects of W₁ DP traits and W₁ puberty (either status or timing) were entered in the first step; the W₁ DP Traits × W₁ Pubertal Status or W₁ DP Traits × W₁ Pubertal Timing interaction was entered in the second step.

Pubertal status did not significantly moderate the effect of DP traits on family, peer, or noninterpersonal stress generation (βs ≤ 1.66, ns); moreover, the residual direct effect of the DP Traits × Pubertal Status interaction on depression was not reduced relative to the overall moderated effect (βs = .17 – .22, ps < .01). Because these conditions for mediated moderation were not met, specific results for the pubertal status analyses are not presented.

Pubertal timing moderated the effect of DP traits on family stress generation (significantly) and peer stress generation (marginally) but not noninterpersonal stress generation (see Table 4). As displayed in Figure 2a, DP traits predicted family stress generation in youth with earlier (β = .47, t(127) = 3.39, p < .01) but not later (β = −.03, t(127) = −.30, ns) pubertal timing. Similarly, as displayed in Figure 2b, DP traits predicted peer stress generation in youth with earlier (β = .26, t(128) = 1.94, p = .05) but not later (β = −.07, t(128) = −.64, ns) pubertal timing.

Next, hierarchical multiple regression analyses were conducted to examine whether stress generation or the Stress Generation × Pubertal Timing interaction predicted depression after adjusting for the main and interactive effects of DP traits and pubertal timing (Condition 2b). W₁ depression, W₁ stress generation, and the mean-centered main effects of W₁ DP traits and W₁ pubertal timing were entered in the first step; the W₁ DP Traits × W₁ Pubertal Timing interaction, the mean-centered main effect of W₂ stress generation, and the W₂ Stress Generation × W₁ Pubertal Timing interaction were entered in the second step.

For family stress, these analyses revealed a significant main effect of family stress generation and a nonsignificant Family Stress Generation × Pubertal Timing interaction (see Table 5). The residual effect of the DP Traits × Pubertal Timing interaction on depression was smaller than the overall moderated effect (see Table 3; Condition 2c) but remained significant after adjusting for family stress generation and the Family Stress Generation × Pubertal Timing interaction (see Table 5), suggesting that family stress generation partially mediated the interactive contribution of DP Traits and pubertal timing to depression. We examined two indexes to quantify the strength of mediation within the earlier timing group. First, we found a significant indirect effect (IE = .13, Z = 2.68, p < .01; Sobel, 1986). Second, the effect proportion (indirect effect/total effect; Shrout & Bolger, 2002) revealed that family stress generation accounted for 29% of the total effect of DP traits on subsequent depression in early-maturing youth.

For peer stress, these analyses revealed a nonsignificant main effect of peer stress generation and a nonsignificant Peer Stress Generation × Pubertal Timing interaction (see Table 5). Moreover, the residual effect of the DP Traits × Pubertal Timing interaction on depression (see Table 3) remained virtually unchanged after adjusting for peer stress generation and the Peer Stress Generation × Pubertal Timing interaction (see Table 5).

For noninterpersonal stress, these analyses revealed a nonsignificant main effect of noninterpersonal stress generation and a nonsignificant Noninterpersonal Stress Generation × Pubertal Timing interaction (see Table 5). Again, the residual effect of the DP Traits × Pubertal Timing interaction on depression (see Table 3) remained virtually unchanged after adjusting for noninterpersonal stress generation and the Noninterpersonal Stress Generation × Pubertal Timing interaction (see Table 5).
Summary—Evidence was obtained for the moderating influence of pubertal status and pubertal timing on the association between DP traits and depression, and the moderating influence of pubertal timing on the association between DP traits and family and peer stress generation. In each case, these paths were positive and significant in youth with more, but not less, advanced pubertal status or earlier, but not later, pubertal timing. Family stress generation, but not peer or noninterpersonal stress generation, partially accounted for the contribution of DP traits to subsequent depression in early-maturing youth.

Influence of Depression on Depressive Personality Traits

Hierarchical multiple regression analyses were conducted to examine whether \( W_1 \) past year depression predicted \( W_2 \) DP traits, adjusting for \( W_1 \) DP traits, and whether these associations differed for more developed youth relative to less developed youth. For each regression, the main effects of \( W_1 \) DP traits, \( W_1 \) mean-centered past year depression, \( W_1 \) mean-centered development (i.e., age, pubertal status, or pubertal timing), and sex were entered at the first step; the two-way interactions were entered at the second step; the three-way (Depression × Development × Sex) interaction was entered at the third step. Significant interactions were interpreted by solving the unstandardized regression equation to predict \( W_2 \) DP traits from \( W_1 \) depression at low (−1 SD), moderate (mean), and high (+1 SD) levels of \( W_1 \) development (Aiken & West, 1991). To ensure that effects were not accounted for by youths’ lifetime history of depression, each regression was rerun adjusting for this variable.

The three-way interaction was marginal for pubertal status, \( \beta = .31, t(122) = 1.80, p < .10 \), but not for age, \( \beta = .20, t(126) = 1.65, ns \), or pubertal timing, \( \beta = .09, t(122) = .63, ns \). However, follow-up of the significant three-way interaction for pubertal status revealed nonsignificant interactions for both girls, \( \beta = .17, t(65) = 1.63, ns \), and boys, \( \beta = -.16, t(56) = -1.24, ns \), making it difficult to interpret this effect. Thus, analyses were rerun collapsing across sex (see Table 6).

For age, these analyses revealed a significant main effect of \( W_1 \) depression, a nonsignificant main effect of age, and a nonsignificant Depression × Age interaction (see Table 6). Adjusting for lifetime history of depression, the regression yielded a significant effect of \( W_1 \) past year depression, \( \beta = .35, t(129) = 3.30, p < .01 \), but not lifetime history of depression, \( \beta = -.02, t(129) = -.23, ns \).

For pubertal status, these analyses revealed a significant main effect of \( W_1 \) depression, a nonsignificant main effect of pubertal status, and a nonsignificant Depression × Pubertal Status interaction (see Table 6). Adjusting for lifetime history of depression, the regression yielded a significant effect of \( W_1 \) past year depression, \( \beta = .34, t(125) = 3.05, p < .01 \), but not lifetime history of depression, \( \beta = -.01, t(125) = -.05, ns \).

For pubertal timing, these analyses revealed a significant main effect of \( W_1 \) depression, a nonsignificant main effect of pubertal timing, and a nonsignificant Depression × Pubertal Timing interaction (see Table 6). Adjusting for lifetime history of depression, the regression yielded a significant effect of \( W_1 \) past year depression, \( \beta = .33, t(125) = 2.95, p < .01 \), but not lifetime history of depression, \( \beta = .00, t(125) = .02, ns \).

Summary—Analyses revealed that recent depression predicted heightened levels of DP traits over time. This effect appeared to be independent of sex and developmental status.

Discussion

Investigating early precursors of DPD may help to elucidate the etiology of this disorder and to provide insight into possible strategies for early identification and intervention before these
traits undermine youths’ development and crystallize into more engrained and unmalleable characteristics. The goal of this research was to establish the validity of the construct of DP traits in youth and to investigate the origins and consequences of these traits. The findings provided support for the reliability, temporal stability, and validity of a measure of DP traits, and identified several family risk factors. Moreover, DP traits predicted a process of family stress generation and consequent depression, particularly during the pubertal transition.

**Developmental Conceptualization of Depressive Personality**

Emerging theory and research point to the utility of adopting a developmental perspective on the emergence of both normative and atypical aspects of personality. According to this perspective, individual differences, whether normative or extreme variations of personality, share a similar structure and underlying dimensions across development although the precise expression of these variations may differ (Caspí & Shiner, 2006; Crick et al., 2007; Geiger & Crick, 2001). Elaborating on these ideas, Geiger and Crick (2001) proposed seven dimensions that underlie a variety of personality disorders, highlighting the importance of mapping these dimensions onto relevant developmental processes to clarify early precursors of adult personality disorders. Although the Geiger and Crick (2001) conceptualization did not include DPD, several of these dimensions are relevant to understanding developmental competencies that may go awry in youth with DP traits.

First, Geiger and Crick (2001) suggest that many personality disorders are characterized by the presence of a negative self-view. Specifically, they suggest that youth who show a deviation from normative developmental trajectories of self-representation either in terms of amount (e.g., a more extreme negative self-view) or timing (e.g., they do not show a gradual increase in self-concept during mid to late adolescence) may be at risk for the development of personality disorders characterized by an excessively negative self-view. This negative self-view is consistent with the symptoms of self-criticalness, negative self-talk, excessive self-blame, and preoccupation with one’s inadequacies in youth with DP traits. Moreover, examination of the validity of our measure revealed that DP traits were associated with a negative self-focus and with a tendency to overestimate one’s contribution to the occurrence of stressful interpersonal events, particularly in girls. Thus, youth with DP traits, especially girls, show difficulty negotiating the critical developmental task of establishing a positive sense of self.

Second, Geiger and Crick (2001) point to the role of intense, unstable, and inappropriate emotion, resulting from disruption in the maturation of effective emotion regulation abilities. This emotion dysregulation is marked by symptoms of gloominess, brooding, and worrying in youth with DP traits. Reflecting these disruptions in emotionality, our measure of DP traits was significantly associated with heightened negative affect and anhedonia (the latter, in girls only) and with diminished positive affect. These findings are consistent with those in adults linking DPD with higher levels of neuroticism and lower levels of extroversion. Thus, another core deficit in youth with DP traits appears to be dysregulation of both negative and positive emotions. Difficulty coping with intense arousal and emotions also likely helps to explain heightened stress reactivity in youth with DP traits. Specifically, DP traits were significantly associated with a tendency to overestimate the stressfulness of events and to experience heightened negative emotional reactivity, particularly in girls, and with less adaptive cognitive, behavioral, and self-reported physiological responses to stress.

Third, Geiger and Crick (2001) highlight the hostile world view endorsed by individuals with some types of personality disorders. Although a predominant characteristic of DPD involves a negative self-view, individuals with these traits also are skeptical and critical; they are overly judgmental toward others and complain excessively. The emergence of this hostile world view may reflect difficulty negotiating the early developmental task of establishing secure attachments with caregivers that provide the basis for positive and trusting perceptions of others.
and the world. The present study revealed that DP traits in youth were associated with family adversity and maternal depression; both of these forms of family risk potentially interfere with the development of a secure attachment (Geiger & Crick, 2001), thereby undermining youths’ views of others as supportive and trustworthy and views of the world as predictable and fair. Finally, Geiger and Crick (2001) suggest that certain personality disorders can be characterized on a continuum from rigidity to impulsivity. The role of this dimension is somewhat less clear in DPD. The original Akiskal criteria include symptoms that reflect high levels of constraint (e.g., extreme conscientiousness, self-discipline). However, reliability analyses from the present study revealed that these symptoms did not cohere with the overall DP construct. Research with adults has revealed a similar pattern, whereby items that tap constraint were uncorrelated with other symptoms and did not appear to be a core component of DPD (Klein, 1990; Klein & Shih, 1998); indeed, these items are no longer included in the proposed DSM criteria for DPD. It is possible that the constraint items on this measure tap adaptive aspects of this personality dimension (e.g., responsibility) rather than maladaptive and extreme tendencies (e.g., rigidity). Thus, perhaps excessive constraint to the point of rigidity would better characterize youth with DP. Alternatively, perhaps there are subgroups of youth with DP traits, some of whom show extreme conscientiousness and self-discipline and some of whom do not. Interestingly, in the present study DP traits were associated with a tendency to engage in fewer planful coping efforts and more involuntary, dysregulated responses to stress, perhaps stemming from the heightened emotion dysregulation and stress reactivity associated with DP traits. Some of these involuntary responses (e.g., impulsive action) actually seem to reflect lower rather than higher levels of constraint. Overall, therefore, more research is needed to determine the relevance of this dimension (rigidity versus impulsivity) to DP traits.

**Developmental Origins of DP Traits**

A second goal of this study was to identify possible family origins of DP traits, focusing on the role of three aspects of family risk: family adversity, maternal DP traits, and maternal lifetime and past year depression. Results revealed that each of these aspects of family risk was associated with DP traits in girls; only maternal lifetime history of depression was significantly associated with DP traits in boys. Moreover, these associations were not accounted for by shared variance with youths’ lifetime history of depression, with the exception of maternal lifetime history of depression in both girls and boys. These findings suggest that there is both shared and unique variance associated with the link between DP traits and mood disorders, and are consistent with research in adults that provides evidence for a shared family aggregation of DP traits and mood disorders (Klein, 1999; Klein & Miller, 1993; Kwon et al., 2000) as well as for genetic influences on the transmission of DP traits (Ørstavik et al., 2007). Moreover, our observed significant association between maternal and youth DP traits in girls but not in boys mirrors prior evidence for greater heritability among women than men (Ørstavik et al., 2007). DP traits were not significantly associated with maternal nondepressive psychopathology, consistent with research in adults suggesting that the relatives of individuals with DPD show more depressive but not nondepressive disorders (e.g., substance use, antisocial traits) than the relatives of individuals without DPD (Klein, 1990).

The present findings cannot disentangle the genetic versus environmental contribution of family risk to DP traits. It is possible that these traits reflect a genetic liability to mood disturbance that is expressed as DP traits, clinical depression, or both. A variety of psychosocial processes also may account for the link between family risk and DP traits. Consistent with Geiger and Crick’s (2001) developmental psychopathology perspective on vulnerability to personality disorders, family adversity, maternal DP traits, and maternal depression may undermine youths’ achievement of salient developmental tasks, creating the type of cognitive, emotional, and behavioral disturbances characteristic of youth with DP traits. For example,
these family risk factors may interfere with the development of secure attachment relationships, thereby hindering the emergence of a positive sense of self and others and effective coping abilities. Moreover, these risk factors may adversely affect biological systems responsible for the regulation of arousal and emotion, creating a vulnerability to emotion dysregulation. Further research is needed to determine more precisely the specific pathways through which family risk confers vulnerability to DP traits.

**Developmental Consequences of DP Traits**

A third goal of this study was to investigate the developmental consequences of DP traits. In support of our hypotheses, DP traits predicted subsequent depression, but not nondepressive psychopathology, particularly for youth with more advanced pubertal status and earlier pubertal timing. Consistent with frameworks suggested elsewhere (Caspi & Shiner, 2006; Klein, Durbin & Shankman, 2009), several explanations may account for this observed link between DP traits and subsequent psychopathology.

First, DP traits may lie on a spectrum that ranges from normative individual differences in cognition, emotion, and behavior at one pole, to clinically significant mood disorders at the other pole (Klein & Bessaha, 2009). One assumption of the spectrum, or common cause, model is that the various points on this spectrum (DP traits, DPD, Axis I mood disorders) differ quantitatively, rather than qualitatively. A related assumption is that the causal processes underlying each of these phenomena overlap. Although taxometric studies are necessary to test the hypothesis that DP traits, DPD, and Axis I mood disorders exhibit continuous variation rather than comprising discrete entities, our findings of familial co-aggregation between youth DP traits and maternal mood disorder suggest that DPD and mood disorders have common familial etiological influences.

A variant of the spectrum model holds that personality is a developmental precursor that crystallizes into psychopathology over time. This is consistent with the finding that DP traits predicted subsequent depressive symptoms, although longer follow-ups and larger samples are needed to determine whether DP traits predict the onset of DPD and Axis I mood disorders. Our longitudinal findings also are consistent with a model in which DP traits are viewed as a vulnerability factor for subsequent mood disorders. The precursor and vulnerability models are similar in that both posit that personality predicts later psychopathology, however the former assumes that personality and psychopathology have overlapping etiologies, whereas the latter presumes that the causal influences underlying personality and psychopathology are distinct. As noted above, our data on familial co-aggregation of DP traits and mood disorders suggest the existence of shared familial etiological influences, hence the present data appear to be more consistent with a precursor than a vulnerability conceptualization (Klein et al., 2009).

Both the precursor and vulnerability models raise the question of what processes are responsible for the transition between the risk state and manifest psychopathology. Caspi and Shiner (2006) suggest that maladaptive personality traits may cause youth to act in ways that elicit aversive responses from others, disrupt their environments, and foster the selection of risky social contexts, which then heighten risk for the emergence, maintenance, or exacerbation of psychopathology. Consistent with this idea, we found that DP traits predicted the generation of stress within the family and peer group, particularly in early-maturing youth. Thus, youth with DP traits appear to face particular interpersonal difficulties when they encounter the challenges associated with earlier pubertal maturation. Moreover, the generation of stress within the family partially accounted for the contribution of DP traits to depression in early-maturing youth, suggesting a dynamic process through which these traits heighten vulnerability to psychopathology. However, the significant residual direct effect of the DP Traits × Pubertal
Timing interaction on depression, even after accounting for family stress generation, suggests the need to investigate other mechanisms accounting for this developmental association.

Finally, psychopathology may alter personality, leaving an enduring scar (Caspì & Shiner, 2006; Rhode et al., 1994). In this case, experiencing significant depressive symptoms and associated impairment early in life may undermine youths’ sense of self and disrupt their emotional development, leaving youth feeling ineffective and pessimistic and creating lingering emotion dysregulation. Thus, DP traits may in part reflect the residue of earlier depression. Supporting this pathway, the present findings revealed that depression predicted subsequent DP traits, regardless of youths’ age, pubertal status, or pubertal timing.

In addition to examining the longitudinal associations between DP traits and psychopathology, we also examined the stability of DP traits over time. DP traits were moderately stable across a one-year period. Interestingly, however, the stability of the DP measure was higher in mothers than in youth. This pattern is consistent with research showing increasing stability of personality traits across development (Roberts & DelVecchio, 2000), and suggests that interventions targeting DP traits may be more effective in early adolescence than in adulthood. Identifying factors that predict stability versus change in DP traits across development is an important direction for future research.

**Sex Differences in the Origins, Correlates, and Consequences of DP Traits**

The present study yielded mixed evidence for sex differences in the origins, correlates, and consequences of DP traits. With regard to the overall level of DP traits, boys showed higher levels earlier in development, particularly if they experienced later pubertal maturation, whereas girls showed high levels later in development, particularly if they experienced earlier pubertal maturation. For our validity analyses, the general pattern of correlations revealed more robust and consistent associations for girls than for boys in several domains (i.e., negative self-focus, negative affect, anhedonia, interpersonal stress estimation and dependence estimation, and emotional reactivity), but only a few of these differences were statistically significant. Similarly, we found more consistent associations between family risk and DP traits in girls than in boys, but again these differences were not statistically significant. Our sample size may have limited power to detect these differences; thus, further research is needed to determine whether these patterns replicate across studies. If they do, these findings may suggest that DP traits have different meanings and origins in girls and boys.

Analyses examining the consequences of DP traits also did not reveal statistically significant sex differences. However, follow-up analyses revealed that the DP Traits × Pubertal Status and DP Traits × Pubertal Timing interactions predicting depression were significant in girls, $\beta_s = .17$ and $.18$, $t(66) = 2.70$ and $3.23$, $p_s < .01$, but not in boys, $\beta_s = -.05$ and $-.02$, $t(57) = -.39$ and $-.12$, $ns$. Similarly, the DP Traits × Pubertal Timing interaction predicting family stress was significant in girls, $\beta = .33$, $t(65) = 3.21$, $p < .01$, but not in boys, $\beta = -.01$, $t(56) = -.07$, $ns$. Thus, future research should continue to investigate possible sex differences in the developmental consequences of DP traits.

**Implications and Future Directions**

The present research demonstrates that early DP traits can be measured in a reliable and valid fashion in youth, and points to the utility of a specific focus on the adolescent transition as a period of heightened risk for the expression of DP traits in the form of clinically significant depressive psychopathology. More generally, this study supports a developmental perspective on personality pathology, suggesting that early identification of, and intervention with, at-risk youth may help prevent a downward trajectory from DP traits to subsequent stress generation and depression. However, this study involved a short-term follow-up period, and therefore was
not able to address the question of whether DP traits in youth foreshadow clinical forms of DPD or mood disorders through late adolescence and adulthood. Moreover, additional research is needed to examine the mechanisms (i.e., genetic and socialization) through which family risk influences the emergence and progression of DP traits in youth, and the factors that predict stability versus change in these traits across development.

Acknowledgments

We thank Melissa Caldwell, Alyssa Clark, Colleen Conley, Alison Dupre, Julie Eisengart, Megan Flynn, Alison Groot, Elisa Krackow, and Kathryn Kurlakowsky for their contributions to this project, and the dedicated families who participated in this research. This research was supported by a National Institute for Mental Health Grant MH59711, a William T. Grant Foundation Faculty Scholars Award, and a University of Illinois Arnold O. Beckman Research Award to Karen D. Rudolph. Daniel N. Klein was supported by a National Institute for Mental Health Grant MH069942.

References


Figure 1.
Depressive Personality Traits × Puberty interactions predicting $W_2$ youth depression for (a) pubertal status and (b) pubertal timing.
Figure 2.
Depressive Personality Traits × Pubertal Timing interaction predicting (a) family stress generation and (b) peer stress generation.
Table 1
Correlates of Depressive Personality Traits

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<sup>a</sup> Correlations significantly differ for girls and boys, p < .05.

<sup>b</sup> p < .10.
<sup>*</sup> p < .05.
<sup>**</sup> p < .01.
<sup>***</sup> p < .001.
Correlations marginally differ for girls and boys, \( p < .10 \).

Note. Partial \( r \) adjus. for youths’ lifetime history of depression.
## Origins of Depressive Personality Traits

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*p < .10.

* *p < .05.

**p < .01.

***p < .001.

*Note. Partial r adjusts for youths’ lifetime history of depression.*
Table 3
Predicting W₂ Psychopathology From W₁ DP Traits, Development, and DP Traits × Development Interactions

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Step 1
W₁ Psychopathology   | .61 | 8.79*** | .57  | .89  | 22.99*** | .86  |
W₁ DP Traits         | .22 | 3.19*** |       | .06  | 1.53   |       |
W₁ Pubertal Status   | .13 | 2.23*   |       | .00  | −.04   |       |
Step 2
W₁ DP Traits × W₁ Pubertal Status | .18 | 3.22** | .03  | −.02 | −.46   | .00  |

Step 1
W₁ Psychopathology   | .60 | 8.90*** | .59  | .89  | 23.14*** | .86  |
W₁ DP Traits         | .22 | 3.30**  |       | .06  | 1.56   |       |
W₁ Pubertal Timing   | .19 | 3.44**  |       | .04  | 1.19   |       |
Step 2
W₁ DP Traits × W₁ Pubertal Timing | .19 | 3.29** | .03  | .00  | .02   | .00  |

* p < .05.
** p < .01.
*** p < .001.

Note. βs and ts represent standardized coefficients and t statistics at each step. ΔR² represents percent of variance accounted for at each step.
Table 4
Predicting W₂ Stress Generation From W₁ DP Traits, Pubertal Timing, and DP Traits × Pubertal Timing Interactions

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*p < .10.
* p < .05.
** p < .01.
*** p < .001.

Note. βs and ts represent standardized coefficients and t statistics at each step. ΔR² represents percent of variance accounted for at each step.
Table 5
Tests of Mediated Moderation to Predict $W_2$ Depression

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Step 2

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<td>.09</td>
<td>.18</td>
<td>3.15**</td>
<td>.03</td>
</tr>
<tr>
<td>$W_2$ Stress Generation</td>
<td>.26</td>
<td>3.46**</td>
<td></td>
<td>.02</td>
<td>.32</td>
<td></td>
</tr>
<tr>
<td>$W_2$ Stress Generation $\times W_1$ Pubertal Timing</td>
<td>.01</td>
<td>.12</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* $p < .05$.
** $p < .01$.
*** $p < .001$.

Note. $\beta$s and $t$s represent standardized coefficients and $t$ statistics at each step. $\Delta R^2$ represents percent of variance accounted for at each step.
Table 6
Predicting W\textsubscript{2} DP Traits From W\textsubscript{1} Depression, Development, and Depression × Development Interactions

<table>
<thead>
<tr>
<th>Step 1</th>
<th>β</th>
<th>t</th>
<th>ΔR\textsuperscript{2}</th>
</tr>
</thead>
<tbody>
<tr>
<td>W\textsubscript{1} DP Traits</td>
<td>.39</td>
<td>4.83***</td>
<td>.40</td>
</tr>
<tr>
<td>W\textsubscript{1} Depression</td>
<td>.33</td>
<td>4.13***</td>
<td></td>
</tr>
<tr>
<td>W\textsubscript{1} Age</td>
<td>.06</td>
<td>.85</td>
<td></td>
</tr>
<tr>
<td>Step 2</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>W\textsubscript{1} Depression × W\textsubscript{1} Age</td>
<td>.03</td>
<td>.46</td>
<td>.00</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Step 1</th>
<th>β</th>
<th>t</th>
<th>ΔR\textsuperscript{2}</th>
</tr>
</thead>
<tbody>
<tr>
<td>W\textsubscript{1} DP Traits</td>
<td>.37</td>
<td>4.52***</td>
<td>.39</td>
</tr>
<tr>
<td>W\textsubscript{1} Depression</td>
<td>.34</td>
<td>4.09***</td>
<td></td>
</tr>
<tr>
<td>W\textsubscript{1} Pubertal Status</td>
<td>−.03</td>
<td>−.43</td>
<td></td>
</tr>
<tr>
<td>Step 2</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>W\textsubscript{1} Depression × W\textsubscript{1} Pubertal Status</td>
<td>.04</td>
<td>.55</td>
<td>.00</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Step 1</th>
<th>β</th>
<th>t</th>
<th>ΔR\textsuperscript{2}</th>
</tr>
</thead>
<tbody>
<tr>
<td>W\textsubscript{1} DP Traits</td>
<td>.30</td>
<td>3.58***</td>
<td>.29</td>
</tr>
<tr>
<td>W\textsubscript{1} Depression</td>
<td>.33</td>
<td>4.02***</td>
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</tr>
<tr>
<td>W\textsubscript{1} Pubertal Timing</td>
<td>−.06</td>
<td>−.83</td>
<td></td>
</tr>
<tr>
<td>Step 2</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>W\textsubscript{1} Depression × W\textsubscript{1} Pubertal Timing</td>
<td>.03</td>
<td>.33</td>
<td>.00</td>
</tr>
</tbody>
</table>

***p < .001.

Note. βs and ts represent standardized coefficients and t statistics at each step. ΔR\textsuperscript{2} represents percent of variance accounted for at each step.