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EMPIRICAL RESEARCH

Correlates of Childhood vs. Adolescence Internalizing Symptomatology from Infancy to Young Adulthood

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Abstract In light of its associations with child and adolescent health and well-being, there remains a need to better understand the etiological underpinnings and developmental course of internalizing symptomatology in children and adolescents. This study leveraged intensive longitudinal data ($N = 959$; 49.6 % females) to test the hypothesis that internalizing symptoms in childhood may be driven more strongly by family experiences whereas internalizing symptoms in adolescence may derive more uniquely from familial loading for affective disorders (i.e., maternal depression). We evaluated the relative contributions of (a) family experiences (b) maternal depression, and (c) peer influences in testing this hypothesis. The results indicated that family predictors were more strongly correlated with childhood (relative to adolescent) internalizing symptoms. In contrast to previous findings, maternal depression also exhibited stronger associations with childhood internalizing symptoms. Although often overlooked in theories concerning potential differential origins of childhood vs. adolescent internalizing symptomatology, peer experiences explained unique variation in *both* childhood and adolescent internalizing problems.

Keywords Environment · Internalizing symptomatology · Peer victimization · Psychopathology · Puberty

Introduction

Internalizing symptomatology refers to an empirically-derived cluster of symptoms that indicate problems in regulating intropunitive emotions and moods, including problems related to anxiety, fear, shyness, low self-esteem, sadness, and depression (Achenbach and Edelbrock 1978; Crawford et al. 2001; Graber and Sontag 2009; Kovacs and Devlin 1998; Ollendick and King 1994; Zahn-Waxler et al. 2000). From a developmental psychopathology perspective, which has as its goal understanding the processes and pathways that lead to developmental success or limitation and to improve the lives of individuals at risk for mental health problems (Cicchetti 1984; Sroufe and Rutter 1984), research efforts that aim to identify and better understand the antecedents, developmental course, and sequalae of internalizing symptomatology are of particular importance.

A unique feature of internalizing symptomatology is its well documented rise during the adolescent period, particularly for girls (Kovacs and Devlin 1998; Zahn-Waxler et al. 2000, Zahn-Waxler 2000). Empirical efforts to illuminate potential causal factors contributing to this rise have been diverse, including a focus on biological, cognitive, and social factors (Graber and Sontag 2009; Zahn-Waxler et al. 2000; Zahn-Waxler 2000). The rise in internalizing symptoms during adolescence also raises the possibility that different etiological factors may underlie internalizing problems that begin prior to adolescence as compared to those that begin during adolescence. Such a possibility is also consistent with the developmental chronology of anxiety

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and depressive symptoms, in which early childhood appears to be the high-risk period for the onset of anxiety symptoms and late childhood or early adolescence to be the high-risk period for the onset of depressive symptoms (Brady and Kendall 1992; Kovacs and Devlin 1998). Given that anxiety and depressive symptoms are moderately to highly correlated both with each other (Brady and Kendall 1992; Seligman and Ollendick 1998) as well as with higher-order internalizing syndrome constructs (e.g., Achenbach and Edelbrock 1978), it is not unreasonable to consider the possibility that, in addition to a large pool of common risk or etiological factors, there may also be unique developmental antecedents and correlates (e.g., family vs. peer relationships) that are differentially associated with internalizing symptomatology in childhood and adolescence. A third stream of evidence for the possibility that different developmental factors may underlie internalizing problems that begin prior to adolescence and those that emerge during adolescence comes from studies investigating distinct correlates of *depressive problems or symptoms* that begin in childhood (prepubertal or early-onset) relative to those that begin in adolescence (pubertal or postpubertal onset; Harrington et al. 1996, 1997; Silberg et al. 1999).

Despite the possibility that childhood and adolescent internalizing symptomatology may reflect distinctive developmental phenomena, large scale, multi-domain developmental investigations of internalizing problems in youth have rarely directly examined this possibility. Indeed, internalizing problems have historically received less theoretical attention and large-scale empirical scrutiny than have longitudinal and classificatory analyses of childhood and adolescent *externalizing* symptomatology and antisocial behavior (Cicchetti and Natsuaki 2014; Ollendick and King 1994; Rubin and Mills 1991), such as Moffitt's (1993) seminal taxonomy of life-course persistent and adolescence-limited antisocial life-course patterns (e.g., Roisman et al. 2010). As such, there remains an important basic and applied need to leverage high-quality prospective data to further explore the possibility of developmental heterogeneity in internalizing symptoms across childhood and adolescence (Cicchetti and Natsuaki 2014). In an effort to address this need, the objective of the current inquiry was to use prospective, multi-informant data from the NICHD Study of Early Child Care and Youth Development (SECCYD) to examine unique and overlapping correlates of childhood and adolescent internalizing symptomatology. Below we briefly review empirical work with *depressive problems or symptoms* that has provided a key stimulus for the broader notion that internalizing symptomatology that begins in childhood and adolescence may reflect distinct developmental phenomena. Using this work as a point of departure, its limitations are discussed and its relevance to the current study is highlighted.

The notion that internalizing symptomatology in childhood and adolescence may reflect distinct developmental phenomena demarcated by timing of occurrence can be traced to research conducted by Harrington and colleagues (1997). They found that a family history of mania or hypomania was more common in postpubertal depressed cases whereas prepubertal cases of depression tended to have lower familial rates of depression, higher rates of criminality among first-degree relatives, and higher rates of retrospectively reported maternal criticism/hostility directed toward the child (i.e., an "environmental risk factor"). Harrington et al. (1997) thus suggested that postpubertal onset depressive disorders may have a higher heritability (i.e., higher familial loading for depression) than prepubertal onset depressive disorders—which in contrast may be more associated with adverse family environments (although see Kovacs and Devlin 1998 for a different interpretation of these findings). Subsequent to the Harrington et al. (1997) finding, a growing body of behavior-genetic research (Murray and Sines 1996; Scourfield et al. 2003; Silberg et al. 1999; Thapar and McGuffin 1996) has provided evidence that family discord and shared environmental factors are more strongly associated with depressive symptoms in childhood whereas genetic factors are more strongly associated with depressive symptoms in adolescence. Nevertheless, findings of developmental change in the etiology of depression symptoms have not been unequivocal and some studies have failed to find an increasing heritability of depressive symptoms in adolescence (e.g., Gjone et al. 1996; O'Connor et al. 1998a, b).

Duggal et al. (2001) were the first to *prospectively* evaluate the possibility of different developmental pathways to depressive and anxious symptomatology in childhood and adolescence using data ($n = 168$) drawn from the Minnesota Longitudinal Study of Risk and Adaptation (MLSRA). A particular strength of their study was the availability of data on observed early family relationships during the first 3.5 years of life that could be used to evaluate more directly the idea that early family adversity would be more strongly associated with depressive problems occurring in childhood rather than adolescence (e.g., Harrington et al. 1996, 1997; Thapar and McGuffin 1996). Duggal et al. (2001) found that psychosocial factors (supportive early care, parenting support, abuse, and early maternal stress) accounted for 13 % of the variance in childhood depressive symptomatology even after accounting for the effects of maternal depression (which accounted for 6 % of the variance in childhood depressive symptoms). In contrast, maternal depression was more strongly associated with *adolescent* depressive symptomatology, accounting for 10 % of the variance in depressive symptomatology while psychosocial variables accounted for 9 % of the variance. When comparing extreme groups (i.e., using

clinical cutoffs), both abuse and early family stress were more strongly associated with childhood than to adolescent depressive symptomatology. Moreover, maternal depression was the only covariate to distinguish the adolescent depressive symptomatology group from controls (i.e., never depressed). These findings were in line with the above work suggesting a higher familial loading for adolescent depression and provided preliminary support for the notion of distinct depressive subgroups defined by their timing of onset (childhood vs. adolescent).

Despite a number of strengths of this study—especially concerning prospective measurement of family adversity—Duggal et al. (2001) did not control for stability in depressive and anxious symptomatology in their continuous analyses of child and adolescent depression. In addition, demarcation of the timing of depressive symptomatology (i.e., childhood or adolescence) was made on the basis of *age* rather than *pubertal status.* Pubertal status and the dynamic changes in hormonal status associated with the pubertal process have been shown to be stronger predictors than chronological age of the gender disparity in unipolar depressive disorders that emerge during adolescence and to the emergence of major depressive disorder (Angold et al. 1998) and negative (depressive and aggressive) affect (Brooks-Gunn and Warren 1989). Moreover, adolescence is often operationalized as a broad interval of maturation encompassing physical, mental, and socioemotional development that results in entry into the social world of adults (Graber and Brooks-Gunn 1996), whereas puberty encompasses a more specific set of processes involved in physical and reproductive functional reorganization that permit greater precision in measurement (Dorn et al. 2006). Finally, in addition to the conceptual and methodological advantages of demarcating childhood and adolescence on the basis of pubertal status, puberty (and school events) are frequently studied as key transitions signaling the entry into adolescence (Graber and Brooks-Gunn 1996).

In addition to the limitations of the Duggal et al. (2001) study, a limitation common to much of the existing work on the possibility of distinctive pathways to child- and adolescent internalizing problems has been that both family and peer researchers have had a tendency to construct models of depressive and internalizing symptomatology that have omitted high quality variables from the others' domain of inquiry, limiting an understanding of the potentially unique effects of family and peer experiences on internalizing problems. In particular, family experience researchers exploring distinctive pathways to child- and adolescent internalizing symptomatology have often neglected to consider the role of peer influences, especially those occurring during later childhood and adolescence (although see, for example, Criss et al. 2009). This is surprising in light of both theoretical work surrounding the importance of peer group socialization (Harris 1995) and peer relationship processes (Rose and Rudolph 2006) for development and empirical evidence that experiences of peer victimization in childhood and adolescence are associated reliably with both concurrent and later depressive and internalizing symptomatology, as well as other forms of maladjustment (Criss et al. 2009; Bowes et al., 2015; Kretschmer et al. 2015; Rose and Rudolph 2006). These findings have been substantiated by both cross-sectional (Hawker and Boulton 2000) and longitudinal (Reijntjes et al. 2010) meta-analytic studies that generally have found modest associations between victimization and internalizing problems. Moreover, given the increasing developmental salience of establishing and maintaining positive peer relations (i.e., peer competence) during later childhood and early adolescence (Sroufe and Rutter 1984), children's friendship quality might also be expected to influence the development of internalizing symptomatology either directly or via its effects on experiences of peer victimization (e.g., Hodges et al. 1999; Waldrip et al. 2008). Increased understanding of whether peer victimization experiences and children's and adolescents' perceived quality of peer friendships track differentially with internalizing symptomatology in childhood or adolescence has the potential to be of both basic and applied value and build upon the extensive body of research that has shown clear evidence for the association of peer victimization with depressive and internalizing symptomatology across childhood and adolescence.

The Current Study

Despite good reason to believe that childhood vs. adolescent internalizing symptomatology may represent distinct developmental phenomena with unique etiological underpinnings, little research to date has evaluated this possibility directly (although see Sterba et al. 2007). As such, the objective of the present study was to provide the first large-sample, prospective examination of unique and overlapping correlates of internalizing problems that occur prior to and following pubertal onset by leveraging data from the NICHD Study of Early Child Care and Youth Development (SECCYD). Data from the SECCYD are particularly well-suited for this purpose as the study includes multi-informant data on children's internalizing symptoms, high quality assessments of the family, multi-informant data on children's peer experiences collected over an 18-year period, and measures of maternal depression. In addition, gold-standard measures of children's pubertal development, rated by trained health-care professionals, are available in the SECCYD dataset, thereby allowing for a more precise demarcation of childhood- and adolescent internalizing symptomatology (Susman et al. 2010).

Drawing in part on both the aforementioned work with major depressive disorder and depressive symptomatology (e.g., Harrington et al. 1996, 1997; Silberg et al. 1999) as well as the Duggal et al. (2001) study, we selected key early family experience variables from the SECCYD dataset including repeated measurements of observed maternal sensitivity, family income-to-needs ratio, father absence, the frequency of negative life events, and the perceived quality of the marital relationship by primary caregivers. Also consistent with Duggal et al. (2001), we used maternal reports of depression as a proxy variable reflecting, in part, genetic/familial loading for depressive symptomatology. Finally, we selected peer variables in the SECCYD (mother and teacher reports of peer victimization and child-reported friendship quality) that have been repeatedly identified in the literature as correlates of both depressive and internalizing symptomatology.

Based on the logic of the prior empirical work reviewed above (e.g., Harrington et al. 1997; Silberg et al. 1999) suggesting that there may be differences in the etiology of depressive symptomatology over development, we hypothesized that family environment influences would be associated more strongly with internalizing symptomatology occurring prior to pubertal onset in childhood whereas maternal depression, reflecting a genetic liability to internalizing symptomatology, would be associated more strongly with internalizing symptomatology occurring following pubertal onset in adolescence. Additionally, we were also interested in evaluating whether peer psychosocial influences would exert a unique influence on internalizing symptoms in childhood and adolescence after accounting for the effects of family experience factors and maternal depression. Based on the literature discussed above chronicling the reliable association between peer victimization and internalizing symptoms, we anticipated that peer victimization would be associated with higher levels of internalizing symptomatology, and in particular internalizing symptomatology occurring during adolescence. Moreover, we anticipated this association in adolescence *even after accounting for effects of family experiences and maternal depression*. Similarly, based on theoretical work articulating the importance of positive peer relationships in children and adolescent's development, we anticipated that friendship quality would be negatively associated with elevated internalizing symptomatology. Once again, because of the increasingly salient role that peer relationships play as children develop into adolescence, we anticipated this promotive effect to be more pronounced for internalizing symptomatology occurring during adolescence. That said, in some contrast to peer victimization, we were less certain how robust its association with internalizing symptoms might be in the context of our other predictors. In evaluating these questions, we address a limitation of prior research by controlling for the stability of internalizing symptomatology across childhood and adolescence.

Methods

Participants

Families were recruited for the NICHD SECCYD in 1991 from hospitals located in or near Little Rock, AR; Orange County, CA; Lawrence, KS; Boston, MA; Pittsburgh, PA; Philadelphia, PA; Charlottesville, VA; Seattle, WA; Morganton, NC; and Madison, WI. During selected 24-h sampling periods, 8986 women who gave birth were screened, 5416 of whom met the eligibility criteria for the study. Families were excluded if: (a) the mother was younger than 18 years of age, (b) the family planned to move, (c) there was a multiple birth, (d) the infant had a known disability or remained in the hospital more than 7 days, (e) the mother acknowledged substance abuse, (f) the mother did not speak English, (g) the mother lived more than an hour from the laboratory site or in an extremely unsafe neighborhood, as determined by local police. From that group, 1364 families became study participants upon completing a home interview when their infants were one month old. Additional details about recruitment and selection procedures are available in prior publications from the study (see NICHD Early Child Care Research Network [ECCRN 2005]) and from the study web site (https://www.nichd.nih.gov/research/supported/Pages/seccyd.aspx). Data were collected longitudinally on the SECCYD sample through age 15 years; and follow-up studies led by researchers at the University of California Irvine and the University of Washington provided age-18 data (see Booth-LaForce and Roisman 2014). Specifically, for the age-18 follow-up interviews, SECCYD youth assessment data was collected at the University of California Irvine and both youth and parent assessment data were collected at the University of Washington. Accordingly, in the current study, youth self-report internalizing data were pooled. Note that, while large, demographically diverse, and methodologically rich, the NICHD SECCYD was not designed to be a nationally representative study.

Analytic Sample

The analytic sample for the current report consists of 959 children who participated in any of the study's repeated physical assessments of pubertal status and for whom a categorical measure of the timing of pubertal onset could be estimated (see below for a detailed description of this variable). We conducted attrition analyses examining the

full sample and analytic sample on child sex, single-parent status in early childhood, total income after the birth of the child, and child race/ethnicity. The full and analytic sample did not differ on any of these demographic variables. In addition, the full and analytic samples did not differ on our index of the highest level of internalizing symptomatology (see below) across time points with the exception of our Kindergarten [$t(1073) = -2.02$, $p < 0.05$)], Grade 3 [($t(1079) = -3.55$, $p < 0.01$)], and Grade 6 [$t(1038) = -2.45$, $p < 0.05$)] measures. Note that effect sizes for these differences were all small by Cohen's standards (ds 0.17, 0.32, and 0.25, respectively). Lastly, the full and analytic samples did not differ on any of our composite substantive covariates of interest with the exception of negative life events [$t(1152) = -1.97$, $p < 0.05$] and teacher reports of peer victimization [$t(215.84) = -4.03$, $p < 0.01$]. Follow-up analyses indicated these differences were due to our Grade 3 measure of negative life events [$t(1026) = -2.14$, $p < 0.05$] and Grade 3 [$t(208.29) = -4.21$, $p < 0.01$] and Grade 6 [$t(119.87) = -2.38$, $p < 0.05$] measures of teacher-reported peer victimization. Similar to above, effect sizes for these differences were all generally small in magnitude (ds 0.21, 0.41, and 0.28 respectively). Note that for all differences, participants in the analytic sample demonstrated *higher* levels of internalizing symptomatology, negative life events, and teacher-reported peer victimization than those who were not. As these were the only observed differences between youth with pubertal timing data and those without, we assumed data were missing at random.

Measures

Measures are presented in four sets corresponding to their function and order of entry in the analyses discussed below: Variables used to create separate composite (dependent) measures of (1) child and adolescent internalizing symptomatology and variables used to composite measures of (2) maternal depression, (3) family experience, and (4) peer experience. In all cases we selected variables that were measured multiple times by multiple informants using standard assessment tools to maximize validity and reliability in our measurement.

Child and Adolescent Internalizing Symptomatology

Participant internalizing symptomatology from childhood to late adolescence was assessed using the internalizing scale of the Child Behavior Checklist obtained using the parent (CBCL) and teacher-report (TRF) versions (Achenbach 1991a; Achenbach and Edelbrock 1986; Achenbach et al. 1987). Participant self-reported internalizing symptomatology also was assessed in adolescence (ages 15 and 18 years) using the Youth Self Report (YSR) version of the Child Behavior Checklist (Achenbach 1991b). Following Duggal et al. (2001), for cases in which data were obtained concurrently from two different informants (e.g., mother and teacher), the highest internalizing symptomatology rating for a given informant at that assessment point was used as an index of the most severe level of internalizing symptomatology experienced. Because there are some differences in item content between the CBCL forms/versions for 2–3 year olds and that for 4–18 year olds, we used standardized *T* scores, which were averaged over time yielding mean internalizing symptomatology composites for the time periods prior to and after pubertal onset (see below for a description of demarcation of pubertal timing estimates) for each individual. Maternal reports on the CBCL were used from the following time points: 24, 36, and 54 months, Kindergarten, Grades 1, 3, 4, 5, and 6; and ages 15 and 18 years. Teacher reports were used from the following assessment points: Kindergarten and Grades 1, 2, 3, 4, 5, and 6. Youth self-reports from the age 15 and 18 assessment points were also used. The internalizing symptomatology scale demonstrated adequate reliability across time and had a standardized coefficient α averaging 0.84 for maternal reports, 0.86 for teacher reports, and 0.90 for youth self-reports across all assessments. Note that we also created average composite measures of childhood internalizing symptomatology within informant to use in sensitivity analyses (discussed below).

Maternal Depressive Symptomatology

Self-reported maternal depressive symptom average composites were created using every assessment point at which the Center for Epidemiologic Studies Depression Scale (CES-D; Radloff 1977) was acquired in the SECCYD: 1, 6, 15, 24, 36, 54 months; Grades 1, 3, 5, and 6; and ages 15 and 18 years. Although the CES-D was developed initially to assess the severity of depressive symptoms, it is now often used to estimate the prevalence of depression or screen for depressive symptomatology across many populations and settings (Santor and Kazdin 2000). The CES-D demonstrated adequate reliability across time with a standardized coefficient α averaging 0.90.

Family Experience Indicators

Maternal Sensitivity

Maternal sensitivity was assessed in the context of mother-child interactions that were videotaped during 15-min semi-structured situations at 6, 15, 24, 36 and 54 months; Grades 1, 3, and 5; and age 15. At each assessment point, the children were videotaped while engaging in tasks at the zone of proximal development while primary caregivers

provided assistance at the younger ages; at older ages (Grade 3 and older), joint tasks, including discussion tasks, were used. Tasks were designed to be developmentally appropriate. Psychometric properties for composite measures of observed maternal sensitivity at each assessment point were adequate (internal consistencies of the sensitivity composite measures for mothers averaged 0.79 [range 0.70–0.85] across assessments; for detailed information on the SECCYD sensitivity assessments see Belsky et al. 2007b; Haltigan et al. 2013; NICHD ECCRN 2001, 2004, 2008). Maternal sensitivity scores were standardized and averaged to create composite measures of observed maternal sensitivity.

Family Income-to-Needs Ratio

Family financial resources were operationalized in terms of an income-to-needs ratio, computed separately for every assessment point at which relevant data were acquired (1, 6, 15, 24, 36, 54 months; Grades 1, 3, 4, 5, 6; age 15). The income-to-needs ratio at each assessment point was calculated from US Census Bureau tables as the ratio of family income to the poverty threshold for each household size at that time point. Higher scores on this composite reflect greater income-to-needs. Scores were averaged across assessment points to create composite measures of family financial resources.

Father Absence

Primary caregivers indicated whether the study child's father was living in the home at each assessment wave through age 15. The scoring was reversed (0 = father in home, 1 = father not in home) and average composite measures of father absence (i.e., the father was *not* living in the household) were computed from 1 month of age to 15 years (1, 3, 6, 9, 12, 15, 24, 36, 42, 46, 50, 54, 60, and 66 months; Kindergarten-Fall [F], Kindergarten-Spring [S]; Grades 1F, 1S, 2F, 2S, 3, 4, 5, 6, 7; ages 14 and 15).

Negative Life Events

Mothers completed the Life Experiences Survey (LES; Sarason et al. 1978) at 54 months, Grades 3 and 5, and age 18. This 57-item questionnaire asks mothers to identify from a list those life events that have happened to them over the past year, and to rate, on a 7-point scale (from +3 = *very positive* to 0 = *neutral*, to –3 = *very negative*) the impact the event has had on their lives. Events include routine happenings (e.g., "child started school") to major events (e.g., "major change in financial status") to catastrophic events (e.g., "death of a parent"). This measure provides an overview of the stressful events that have befallen the child's family and may have an impact on the child's well-being, as well as on the quality of parenting. Composite (average) measures of negative life events were created from the assessment points noted above.

Marital Quality

Composite measures of marital quality were created using the 6-item intimacy subscale of the Personal Assessment of Intimacy in Relationships Inventory (Schaefer and Olson 1981) which was completed at the following assessment points: 1, 36, and 54 months; Grades 1, 3, 5, 6; and at the age 15 and 18-year assessment points. Samples items on this measure include *My partner listens when I need to talk* and *My partner understands me*. Subscale scores were computed as an average of the six item responses. Reliability was adequate across time with an average standardized coefficient $\alpha = 0.87$). Scores were standardized and averaged across assessment points with higher scores reflecting higher levels of emotional intimacy in the marital relationship.

Peer Psychosocial Indicators

Peer Victimization: Mother and Teacher Report

At Grades 3, 4, 5, and 6, mothers and teachers were asked to complete a questionnaire designed to measure the study child's peer-related behaviors. This questionnaire consisted of 43 items, which were adapted from the Child Behavior Scale (Ladd and Profilet 1996), the Peer Victimization Scale (Kochenderfer and Ladd 1996) and the Relational Aggression scale (Crick et al. 1996). Respondents were asked to rate the child's behavior with peers on a 3-point scale (0 = *Not True*, 1 = *Sometimes True*, 2 = *Often True*). For purposes of the current project, the peer victimization subscale (7 items) from this measure was used. Peer victimization scores were computed at each time point as the average of these 7 items. Both mother and teacher report measures of peer victimization demonstrated adequate reliability across time with a standardized coefficient α averaging 0.90 for maternal reports and 0.89 for teacher reports. Scores at each time point were then averaged across assessment points within informant to create separate composite measures of mother and teacher-reported peer victimization. Higher scores reflected higher levels of mother- and teacher-reported peer victimization.

Friendship Quality

Study children completed a modified version of the Friendship Quality Questionnaire (Parker and Asher 1993) designed to assess their perceptions of their friendship with

their very best friend at Grades 3, 4, 5, 6, age 15, and age 18 years. Children rated how true 20 statements (28 statements at grade 6, and ages 15 and 18) were of their relationship with their best friend on a 5-point scale, from 1 = *Not at all true* to 5 = *Really true*. These statements are grouped into six subscales including companionship and recreation, validating and caring, help and guidance, intimate disclosure, conflict and betrayal, and conflict resolution. In the current report, a friendship quality total score at each time point was computed as a weighted average of the items. Child reports of friendship quality demonstrated adequate reliability across time with a standardized coefficient α averaging 0.90. Friendship quality total scores at each assessment were standardized and averaged across assessment points to create friendship quality composites, with higher scores reflecting higher levels of friendship quality.

Pubertal Development

Starting at age 9½ years, all SECCYD children were asked to participate in an annual health and physical development assessment. A primary component of the assessment was a physical examination of the child. Pubertal status was assessed using Tanner staging. Tanner staging for girls was based on instructions from the American Academy of Pediatrics Manual, Assessment of Sexual Maturity Stages in Girls (Herman Giddens and Bourdony 1995), augmented with breast bud palpation. For boys, Tanner staging was based on Tanner's original criteria (adapted from Tanner 1962; Marshall and Tanner 1970). The majority of the exams were conducted by nurse practitioners; however, some were administered by pediatric endocrinologists, depending upon staff employed at each data collection site. All clinicians were experienced with Tanner staging of children in the evaluated age groups. Additional details regarding the measurement of pubertal development in the SECCYD can be found in Belsky et al. (2007a).

Results

Analytic Plan

Prior to conducting focal analyses, we first determined each individual's estimated onset of puberty using latent transition analysis applied to the Tanner staging assessments. Each individual's timing of pubertal onset was then used to create separate composite measures of internalizing symptomatology which began prior to and following pubertal onset. Next, zero-order intercorrelations among study variables were computed. We also tested whether the magnitude of family experience (i.e., maternal sensitivity, father absence, family income-to-needs, negative life events, and marital quality) and maternal depression associations with prepubertal vs. postpubertal internalizing symptomatology were significantly different.

Our primary substantive analyses consisted of two hierarchical stepwise regression models examining the predictive significance of family experience, maternal depression, and peer experience variables for child and adolescent internalizing symptomatology. In the first model, internalizing symptomatology in childhood was predicted from *childhood* measures of family experience, maternal depression, and peer experience predictor variables (Model 1). In a second model, *adolescent* internalizing symptomatology was predicted from *across-time* (i.e., childhood and adolescent) composites of family experience, maternal depression, and peer experience predictor variables (Model 2). Note that for Model 2, the use of across time predictor composites was designed to maximize the reliability and precision of predictor variable sets by leveraging all of the available data in the SECCYD in predicting adolescent internalizing symptomatology.

For each of the regression models, the order of entry of each of the three blocks of predictor variable sets was theoretically guided by the original logic of the aforementioned empirical work with depressive problems. Specifically, because prior research (e.g., Harrington et al. 1996, 1997; Murray and Sines 1996; Thapar and McGuffin 1996) suggests that family experience variables should be more strongly associated with internalizing symptomatology occurring prior to pubertal onset (i.e., in childhood) these variables were entered in the first block of Model 1, followed by maternal depression, and finally peer psychosocial influences. Similarly, because prior work (e.g., Duggal et al. 2001) suggests that maternal depression should be more strongly associated with internalizing symptomatology occurring following pubertal onset (i.e., in adolescence), this variable was entered in the first block of Model 2, followed by family experience, and finally peer psychosocial influences. In each model, we controlled for the stability of internalizing symptomatology in a final block. As such, we were able to ascertain the robustness of focal predictors for internalizing symptomatology in childhood and adolescence in relation to the addition of other predictors in each model.[1]

Finally, we conducted a series of sensitivity analyses to determine whether: (1) our substantive results from Models

[1] In light of the well documented finding that the initiation of puberty is associated with a rise in depressive and internalizing symptomatology among girls relative to boys (Nolen-Hoeksema, 2001; Zahn-Waxler et al., 2000) we also conducted interaction analyses to examine whether child sex moderated any of the focal associations between our predictor sets composited across time and internalizing symptomatology occurring in childhood and adolescence. None of the interaction effects were significant.

Table 1 *N*s and percentages of children starting puberty by age in the SECCYD

Age of pubertal onset	Girls ($N = 476$)		Boys ($N = 483$)	
	N	%	*N*	%
≤9.5	103	21.6	33	6.8
9.5–10.5	182	38.2	56	11.6
10.5–11.5	124	26.1	208	43.1
11.5–12.5	63	13.2	120	24.8
12.5–13.5	4	0.8	55	11.4
>13.5	–	–	11	2.3

1 and 2 differed as a function of whether internalizing symptomatology was operationalized by informant (i.e., mother, teacher, or youth self-report [adolescence only]) relative to our index of the highest level of internalizing symptomatology; and (2) substantive results for internalizing symptomatology in adolescence using *across time* composites of predictor variable sets differed when predictor sets were operationalized based on developmental timing (i.e., childhood predictor composites and adolescent predictor composites). Because few effects from these models were substantively different than our core models presented above, they are summarized in the electronic supplement to this manuscript.

Estimation of Pubertal Timing

As reported in previous work using this same dataset (Belsky et al. 2007a), a categorical version of the timing of pubertal onset was estimated using latent transition analysis (LTA; Collins and Flaherty 2002; Muthén and Muthén 1998–2006). Separate models were conducted for boys and girls. The data modeled indicated, at each age of measurement, whether the child exhibited any evidence of pubertal development (i.e., yes/no) on (1) physical exam of genitals (for boys) or breast (for girls) development and (2) physical exam of pubic hair development. LTA analyses were conducted using *M*Plus (version 4.1: Muthén and Muthén 1998–2006), which uses maximum likelihood (ML) estimation under the assumption of data missing at random (MAR). The weighting given to any particular indicator was equal across time points (i.e., measurement invariance) and children, once categorized as having initiated puberty, could not revert to a no-initiation state. That is, the LTA models constrained the weights of the measures listed above to be equal at each age and constrained children who had "started puberty" at one age to remain "in puberty" at later ages. Results from this analysis are presented in Table 1.

Table 2 Intercorrelations among childhood composites of family experience, peer, and maternal depression predictor variables and achenbach system measures of childhood and adolescent internalizing symptomatology

	1	2	3	4	5	6	7	8	9	10	11
1. Childhood internalizing	–										
2. Adolescent internalizing	0.48*	–									
3. Maternal sensitivity childhood	−0.28**	−0.10**	–								
4. Income-to-needs childhood	−0.21**	−0.11**	0.44**	–							
5. Father not in home childhood	0.20**	0.12**	−0.44**	−0.38**	–						
6. Negative life events childhood	0.15**	0.22**	0.09**	−0.07*	0.04	–					
7. Emotional intimacy childhood	−0.27**	−0.23**	0.14**	0.19**	−0.15**	−0.25**	–				
8. Peer victimization MR childhood	0.42**	0.33**	−0.29**	−0.18**	0.18**	0.16**	−0.21**	–			
9. Peer victimization TR childhood	0.30**	0.13**	−0.29**	−0.18**	0.19**	0.06	−0.06	0.49**	–		
10. Friendship quality score childhood	−0.09**	−0.10**	−0.03	−0.05	−0.03	−0.03	0.02	−0.09**	−0.08*	–	
11. Maternal depression childhood	0.45**	0.31**	−0.37**	−0.33**	0.33**	0.30**	−0.49**	0.31**	0.20**	0.02	–

Note All predictors are average childhood composites of the target variable. The maternal sensitivity and emotional intimacy composites are the average of the standardized scores for each assessment in childhood. For internalizing symptomatology in childhood and adolescence, composites are average *T* scores using the highest informant at each available time point

MR mother report, *TR* teacher report. *N*s range 902–959

*$p < 0.05$; ** $p < 0.01$

Table 3 Sample descriptive statistics and intercorrelations among across time composites of family experience, peer, and maternal depression predictor variables and achenbach system measures of childhood and adolescent internalizing symptomatology

	1	2	3	4	5	6	7	8	9	10	11
1. Childhood internalizing	–										
2. Adolescent internalizing	0.48**	–									
3. Maternal sensitivity 6MO—age 15	−0.29**	−0.11**	–								
4. Income-to-needs 1MO—age 15	−0.22**	−0.11**	0.44**	–							
5. Father not in home 1MO—age 15	0.20**	0.13**	−0.43**	−0.37**	–						
6. Negative life events 54MO—age 18	0.17**	0.25**	0.09**	−0.06	0.04	–					
7. Emotional intimacy 1MO—X18	−0.25**	−0.25**	0.14**	0.18**	−0.13**	−0.28**	–				
8. Peer victimization MR G3–G6	0.44**	0.39**	−0.28**	−0.18**	0.20**	0.17**	−0.21**	–			
9. Peer victimization TR G3–G6	0.27**	0.19**	−0.32**	−0.20**	0.20**	0.05	−0.05	0.47**	–		
10. Friendship quality score G3—age 18	−0.13**	−0.16**	0.05	−0.01	−0.07*	−.02	0.04	−0.15**	−0.14**	–	
11. Maternal depression 1MO—age 18	0.46**	0.34**	−0.36**	−0.33**	0.32**	0.34**	−0.48**	0.32**	0.18**	−0.02	–
Mean	51.44	52.41	−0.02	3.85	0.25	3.37	−0.06	0.30	0.17	−0.01	9.61
Standard deviation	5.92	7.42	0.68	2.89	0.36	2.51	0.76	1.61	0.24	0.71	6.09
N	948	923	959	958	959	955	949	955	936	954	959

Note All predictors are average composites of the target variable. The maternal sensitivity and emotional intimacy composites are the average of the standardized scores for each assessment. For internalizing symptomatology in childhood and adolescence, composites are average *T* scores using the highest informant at each available time point

MR mother report, *TR* teacher report. *Ns* range 902–959

*$p < 0.05$; **$p < 0.01$

Intercorrelations among Study Variables

Zero-order correlations among prepubertal (*childhood*) composites of family experience, maternal depression, and peer experience predictor variables are reported in Table 2 and zero-order correlations of *across time* composites of family experience, maternal depression, and peer experience predictor variables and child and adolescent internalizing symptomatology, as well as summary descriptive statistics for study variables, are presented in Table 3. As is reflected in both correlation tables, there was moderate stability between internalizing symptoms in childhood and adolescence. Using equivalent transformations of Cohen's (1992) *d* effect size criteria to interpret *r* (small effect = 0.10, medium effect = 0.24, large effect = 0.37), intercorrelations among predictor variables, whether composited only in childhood or across time, were mostly small to medium in magnitude. Exceptions to this general pattern were correlations of larger magnitude between maternal sensitivity and income-to-needs, maternal sensitivity and father absence, income-to-needs and father absence, and mother and teacher reports of peer victimization. In addition, maternal depression was moderately correlated with each of the other predictors except teacher reports of peer victimization and child-reported friendship quality.

Given their focal role in prior work examining differential correlates of child and adolescent major depressive disorder, using the *Psych* package (Revelle 2015) in the R environment for statistical computing (R Core Team 2016), we tested whether the magnitude of the associations of family experience variables and maternal depression with internalizing symptomatology occurring in childhood and adolescence differed significantly using methods recommended by Steiger (1980) for dependent, overlapping correlations (Case A, see Steiger 1980). Paralleling our analytic approach described previously for core regression analyses (see below), in these preliminary analyses we leveraged the rich longitudinal data in the SECCYD to maximize the precision of our covariates by using the *across-time* composites of family experience and maternal depression variables. Note that the methods recommended by Steiger (1980) require a single sample size for computation. As such, listwise deletion was implemented when producing a correlation matrix among family experience variables, maternal depression, and internalizing symptomatology composites ($n = 911$). Of the family experience variables, maternal sensitivity ($r = -0.29$ vs. $r = -0.11$), family income-to-needs ($r = -0.22$ vs. $r = -0.11$), and father absence ($r = 0.21$ vs. $r = 0.12$) were all significantly ($p < 0.01$) more strongly associated with internalizing symptomatology occurring in childhood compared to adolescence. In contrast, the frequency of negative life experiences was more strongly associated with internalizing symptomatology in adolescence relative to

Table 4 Hierarchical regression analysis predicting internalizing symptomatology in childhood from childhood family experience, maternal depression, and peer psychosocial variables (model 1)

Step	Independent variables	R^2 change	*B* (SE)	β	Overall		
					R^2	*F*	df
1.	Maternal sensitivity	0.16**	−2.01 (0.30)	−0.25**	0.16	31.82	5, 848
	Income-to-needs		−0.06 (0.08)	−0.03			
	Father not at home		0.60 (0.59)	0.04			
	Negative life events		0.21 (0.07)	0.10**			
	Emotional intimacy		−1.49 (0.24)	−0.21**			
2.	Maternal sensitivity	0.07**	−1.29 (0.30)	−0.16**	0.23	41.94	6, 847
	Income-to-needs		0.01 (0.07)	0.00			
	Father not at home		−0.08 (0.57)	−0.01			
	Negative life events		0.05 (0.07)	0.02			
	Emotional intimacy		−0.59 (0.25)	−0.08*			
	Maternal depression		0.32 (0.04)	0.35**			
3.	Maternal sensitivity	0.08**	−0.67 (0.29)	−0.08*	0.31	42.80	9, 844
	Income-to-needs		0.01 (0.07)	0.00			
	Father not at home		−0.19 (0.55)	−0.01			
	Negative life events		−0.03 (0.07)	−0.01			
	Emotional intimacy		−0.42 (0.24)	−0.06			
	Maternal depression		0.29 (0.04)	0.31**			
	Peer victimization mother report		4.56 (0.63)	0.25**			
	Peer victimization teacher report		1.88 (0.74)	0.08*			
	Friendship quality		−0.42 (0.20)	−0.06*			
4.	Maternal sensitivity	0.08**	−0.79 (0.28)	−0.10**	0.39	54.69	10, 843
	Income-to-needs		−0.01 (0.07)	−0.00			
	Father not at home		−0.23 (0.51)	−0.01			
	Negative life events		−0.09 (0.06)	−0.04			
	Emotional intimacy		−0.27 (0.23)	−0.04			
	Maternal depression		0.23 (0.03)	0.25**			
	Peer victimization mother report		3.04 (0.61)	0.17**			
	Peer victimization teacher report		2.21 (0.70)	0.10**			
	Friendship quality		−0.26 (0.19)	−0.04			
	Adolescent internalizing		0.24 (0.02)	0.31**			

Note $N = 854$

*$p < 0.05$; ** $p < 0.01$

childhood internalizing symptomatology ($r = 0.25$ vs. $r = 0.16$, $p < 0.01$). Importantly, maternal depression was also significantly more strongly associated with internalizing symptomatology in childhood relative to internalizing symptomatology in adolescence ($r = 0.45$ vs. $r = 0.34$, $p < 0.01$).[2]

[2] Steiger (1980) recommended tests of separate, non overlapping (Case B; see Steiger 1980) dependent correlations between covariates composited within childhood and childhood internalizing symptomatology versus correlations of covariates composited within adolescence and adolescent internalizing symptomatology were not materially different than tests of dependent correlations noted above with the exception of associations between father absence and internalizing symptomatology ($r = 0.20$ [childhood] vs. $r = 0.13$ [adolescence], $p = 0.14$).

Unique Predictors of Childhood Internalizing Symptomatology

Results of the hierarchical multiple regression analysis predicting childhood internalizing symptomatology from childhood family experience, maternal depression, and peer experience variables (Model 1) are presented in Table 4. Maternal sensitivity, negative life events, and emotional intimacy in the marriage were all significant predictors of childhood internalizing symptomatology at entry in the first block, accounting for 16 % of the variance. Higher levels of maternal sensitivity and emotional intimacy in the marital relationship predicted lower levels, and negative life events higher levels, of internalizing symptomatology occurring in

Table 5 Hierarchical regression analysis predicting internalizing symptomatology in adolescence from across time maternal depression, family experience, and peer psychosocial variables (model 2)

Step	Independent variables	R^2 change	*B* (SE)	β	Overall		
					R^2	*F*	*df*
1.	Maternal depression	0.12**	0.43 (0.04)	0.35**	0.12	120.43	1, 886
2.	Maternal depression	0.03**	0.30 (0.05)	0.25**	0.15	25.12	6, 881
	Maternal sensitivity		−0.17 (0.42)	−0.02			
	Income-to-needs		0.04 (0.09)	0.02			
	Father not at home		0.38 (0.74)	0.02			
	Negative life events		0.40 (0.10)	0.14**			
	Emotional intimacy		−0.96 (0.35)	−0.10**			
3.	Maternal depression	0.09**	0.25 (0.05)	0.20**	0.24	30.11	9, 878
	Maternal sensitivity		0.57 (0.41)	0.05			
	Income-to-needs		0.03 (0.09)	0.01			
	Father not at home		−0.00 (0.71)	0.00			
	Negative life events		0.29 (0.10)	0.10**			
	Emotional intimacy		−0.72 (0.33)	−0.07*			
	Peer victimization mother report		6.90(0.88)	0.28**			
	Peer victimization teacher report		0.78 (1.07)	0.03			
	Friendship quality		−1.10 (0.32)	−0.10**			
4.	Maternal depression	0.07**	0.13 (0.05)	0.11**	0.31	39.30	10, 877
	Maternal sensitivity		0.87 (0.39)	0.08*			
	Income-to-needs		0.04 (0.08)	0.02			
	Father not at home		0.03 (0.67)	0.00			
	Negative life events		0.29 (0.09)	0.10**			
	Emotional intimacy		−0.64 (0.32)	−0.07*			
	Peer victimization mother report		4.53 (0.87)	0.18**			
	Peer victimization teacher report		0.44 (1.02)	0.01			
	Friendship quality		−0.90 (0.30)	−0.08**			
	Childhood internalizing		0.42 (0.04)	0.33**			

Note $N = 888$

*$p < 0.05$; **$p < 0.01$

childhood. When maternal depression was entered in the 2nd block, only maternal sensitivity remained a significant predictor. Higher levels of maternal depression significantly predicted higher levels of childhood internalizing symptomatology and accounted for an additional 7 % of the variance. When peer psychosocial influences were added on the third block, both maternal sensitivity and maternal depression remained significant predictors of childhood internalizing symptomatology, along with mother and teacher-reports of peer victimization (which predicted higher) and child-reported friendship quality (which predicted lower) levels of childhood internalizing symptomatology. Together, the peer psychosocial variables accounted for an additional 8 % of the variance. Lastly, when we controlled for internalizing symptomatology in adolescence in the 4th block, maternal sensitivity, maternal depression, and mother and teacher-reports of peer victimization remained significant predictors of childhood internalizing symptomatology. Not surprisingly, internalizing symptomatology in adolescence was significantly and modestly associated with internalizing symptomatology in childhood. Altogether, the final model accounted for 39 % of the variance in childhood internalizing symptomatology (31 % prior to the inclusion of adolescent internalizing symptomatology).

Unique Predictors of Internalizing Symptomatology in Adolescence

Results of the hierarchical multiple regression analysis predicting internalizing symptomatology in adolescence from *across time* family experience, maternal depression, and peer experience variables (Model 2) are reported in Table 5. On the first step, higher levels of maternal depression predicted higher levels of adolescent internalizing symptomatology, accounting for 12 % of the variance.

When family experience variables were added on the 2nd step, only negative life events and emotional intimacy in the marital relationship were significant predictors. Experiencing a greater number of negative life events predicted higher levels of internalizing symptomatology in adolescence whereas greater emotional intimacy in the marital relationship predicted lower levels of internalizing symptomatology in adolescence. Maternal depression remained significant in the 2nd step and the combined variables accounted for an additional 3 % of the variance. Mother reports of peer victimization and the child-reported friendship quality were both significant at entry on the 3rd step. Higher levels of mother-reported peer victimization predicted elevated levels of internalizing symptomatology in adolescence whereas higher friendship quality predicted lower levels of internalizing symptomatology in adolescence. Negative life events and emotional intimacy in the marital relationship remained significant predictors, as did maternal depression. Taken together, the peer psychosocial predictors accounted for an additional 9 % of the variance in internalizing symptomatology occurring in adolescence in the third step. Finally, when we controlled for childhood internalizing symptomatology in the 4th step, all of the family experience and peer psychosocial variables that were significant at step 3 remained significant. In addition, a small counterintuitive effect emerged with higher levels of maternal sensitivity predicting elevated levels of internalizing symptomatology in adolescence. As was the case in our analysis predicting internalizing symptomatology in childhood, there was a moderate and significant positive association between internalizing symptomatology occurring in adolescence with that occurring in childhood. The final model accounted for 31 % of the variance in internalizing symptomatology occurring in adolescence. (24 % prior to the inclusion of childhood internalizing symptomatology).

Discussion

In light of the well-established rise in internalizing symptomatology during adolescence, and inspired in part by work suggesting that major depressive disorders that begin in childhood and adolescence may represent unique developmental phenomena (e.g., Harrington et al. 1996, 1997; Silberg et al. 1999), the objective of the present inquiry was to provide the first large-sample examination of unique and overlapping correlates of internalizing symptomatology occurring in childhood and adolescence. Using multimethod, multi-informant data from the SECCYD, we tested whether family experiences (i.e., maternal sensitivity, family income-to-needs, presence of father in the home, quality of the marital relationship) were associated more strongly with childhood internalizing symptomatology (compared to internalizing symptomatology in adolescence) and whether maternal depression was associated more strongly with internalizing symptomatology in adolescence (compared to internalizing symptomatology occurring in childhood). Altogether, our comprehensive set of results demonstrated that both family influences and maternal depression tracked more strongly with internalizing symptomatology in childhood while the influence of peer relationship variables on internalizing symptomatology occurring in childhood and adolescence was relatively nonspecific. Thus, we did not find support for distinctive developmental correlates of childhood and adolescent internalizing symptomatology.

Despite finding that maternal sensitivity, family income-to-needs, and father absence more strongly tracked with childhood internalizing symptomatology (relative to that occurring in adolescence), the degree to which these variables alone accounted for variation in childhood internalizing symptomatology was modest. Moreover, zero-order associations between maternal depression—whether aggregated across time or within childhood and adolescence—and childhood and adolescent internalizing symptomatology suggested that maternal depression tracked more strongly with internalizing symptomatology occurring in childhood—a finding not in keeping with prior work on major depressive disorder suggesting that familial loading for depression is associated with pubertal rather than prepubertal depression (Harrington et al. 1997; Silberg et al. 1999). Additionally, unlike the results of Duggal et al. (2001), we found that maternal depression continued to remain a significant predictor of the most severe level of internalizing problems occurring in childhood even when family experience variables were taken into account.

A novel aspect and strength of the current study was the inclusion of *peer* relationship variables in predicting internalizing symptomatology occurring in childhood and adolescence while also controlling for stability in internalizing symptomatology itself. Inclusion of peer relationship variables along with family influences and maternal depression in predictive models of internalizing symptomatology is seldom seen in the literature and allows for a more comprehensive understanding of the relative predictive significance of these factors. The current findings demonstrate that peer psychosocial influences, especially maternal-reports of peer victimization, represent unique predictors of internalizing symptomatology, even after controlling for family influences and maternal depression (as well as stability in internalizing symptomatology itself).

Given the present results, it remains ambiguous as to whether a familial loading for internalizing symptomatology has any unique predictive significance for its timing of occurrence (i.e., prior to vs. following pubertal onset) or is

simply a more general marker of vulnerability that is distributed over childhood *and* adolescence. Future longitudinal work, including research informed by behavior-genetic designs (Thapar and McGuffin 1996), is needed to answer this question more definitively. Another possibility is that biological and/or genetic loading for depression could be associated with a distinct subclass of childhood-onset internalizing symptomatology. Additional person-centered methodological work with internalizing symptomatology (e.g., Sterba et al. 2007) could provide insight into this possibility.

There are limitations to the current study that delimit the reach of our findings. As has been noted in other reports using the SECCYD dataset, although it is a large national study, its sample is not nationally representative and is a normative-risk cohort. It remains possible that stronger associations between family experience variables, maternal depression, and internalizing symptomatology occurring in childhood and adolescence would have been uncovered had a high-risk cohort been studied in which clinically elevated levels of internalizing symptomatology might have been more commonly observed. A second limitation concerns the maternal depression variable that served as a proxy for genetic-loading (i.e., heritability) in the context of prior work examining differential correlates of childhood and adolescence-onset depression. We acknowledge that maternal depression reflects an imprecise marker of genetic loading for depression in offspring. Third, shared method variance between mother and teacher reports of peer victimization experiences and these same informants' reports of child internalizing symptomatology might partially explain the robust associations observed between maternal and teacher reports of peer victimization and internalizing symptomatology both in childhood and adolescence. Fourth, we exclusively relied on mother and teacher-reports of peer victimization. Other measurement approaches used to operationalize peer victimization, such as peer nomination procedures, are well-validated and their inclusion would have broadened our assessment battery for this construct. Fifth, it should be noted that the current study focused on internalizing symptomatology and it is conceivable that the current results may have been different if we had considered individual subscales (e.g., anxiety/depression) that comprise the broad-band internalizing dimension. That said, we believe this possibility is unlikely given the typically large associations observed between individual subscales of the internalizing dimension and the internalizing dimension itself.[3] Finally, the focus of the current study was on mean levels of internalizing symptomatology within childhood and adolescence as demarcated by pubertal status. The current analyses therefore do not address questions concerning growth in internalizing symptomatology across time nor were intended to directly address questions concerning 'onset' of clinically significant levels of problematic internalizing symptomatology.

[3] In the current study, we observed correlations in excess of 0.80 between the anxious/depressed and internalizing raw scale scores for teacher-reports and in excess of 0.90 for parent-reports. The correlation between these scales for youth self-reports at age 15 was 0.93 and at age 18 it was 0.92 (all $ps < 0.01$).

Conclusion

The current study contributes to the literature on internalizing symptomatology in at least two novel ways. First, it is the first large-scale prospective, multi-domain investigation to raise and address the possibility that there may be distinct developmental correlates of childhood and adolescent internalizing symptomatology. Using a variety of predictors from conceptually distinct domains (e.g., family, peer) and operationalized in multiple ways (e.g., observational, parent, teacher, and self-report), we were able to explain a good deal of the total variation in internalizing symptomatology occurring both in childhood and adolescence. In particular, we found relatively robust effects for peer victimization on internalizing symptomatology in both childhood and adolescence. Second, the current study provides a basic substantive contribution by explicitly testing a model of differential prediction of child and adolescent internalizing symptomatology that is based on earlier work with major depressive disorder (e.g., Harrington et al. 1997). We found little evidence indicating that specific conceptual subsets of predictors were uniquely associated with internalizing symptomatology in childhood or adolescence, as might have been expected based on this earlier work and subsequent work inspired by it (e.g., Duggal et al. 2001). This is not to suggest that that identifiable and meaningful patterns of distinct internalizing symptomatology in childhood and adolescence do not exist. Rather, in encouraging future research on this issue, we echo Cicchetti and Natsuaki (2014) and Shanahan et al. (2014), who point out that future work examining internalizing symptomatology will need to invoke an integrative perspective that investigates multiple co-active and interactive factors at different levels (e.g., family, school) of dynamic developmental systems in order to better understand the etiological underpinnings and developmental course of internalizing symptomatology. Such work, we believe, will yield potentially important implications for basic research into the nature of internalizing symptomatology as well as applied prevention and intervention efforts for youth with internalizing problems.

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Compliance with Ethical Standards

Conflict of Interest The authors declare that they have no competing interests.

Ethical Approval Approved by IRB review at the University of California, Irvine, the University of Minnesota, and the University of Washington.

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

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