Developmental Pathways to BPD-Related Features in Adolescence:

Infancy to Age 15

Laura E. Brumariu
Adelphi University, Derner School of Psychology

Margaret Tresh Owen
The University of Texas at Dallas, School of Behavioral and Brain Sciences

Nazly Dyer
University of Houston-Downtown, Institutional Data Analytics

Karlen Lyons-Ruth
Harvard Medical School at Cambridge Health Alliance, Department of Psychiatry

Address correspondence to Dr. Brumariu, Adelphi University, Gordon F. Derner School of Psychology, 158 Cambridge Ave., Garden City, NY 11530; email:lbrumariu@adelphi.edu

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Abstract

The self-damaging behaviors central to borderline personality disorder (BPD) become prominent in adolescence. Current developmental theories cite both early family processes and childhood dysregulation as contributors to BPD, but longitudinal data from infancy are rare. Using the NICHD Study of Early Child Care and Youth Development database (SECCYD; n = 1364), path models evaluated parent and child contributors from infancy/preschool, middle childhood, and adolescence to adolescent BPD-related features. In addition, person-centered latent class analyses investigated whether adolescent BPD-related features were more strongly predicted by particular patterns of maladaptive parenting. Path modelling identified unique influences of maternal insensitivity and maternal depression on BPD-related features, first, through social-emotional dysregulation in middle childhood, and second, through continuity from infancy in maternal insensitivity and depression. LCA results indicated that early withdrawn parenting was particularly predictive of BPD-related features in adolescence. Results suggest multiple points of intervention to alter pathways toward adolescent borderline psychopathology.

**Keywords:** borderline personality disorder, risky self-damaging behavior, adolescence, maternal sensitivity, maternal depression
Adolescence is a critical time of transition from dependence on the family to more autonomous functioning in the community. This transition involves expansion of activities into a variety of new domains, including intimacy/sexuality and work or career-related studies (e.g., Kobak, Zajac, Abbott, Zisk, & Bounoua, 2017). Notably, a variety of risky self-damaging behaviors are also initiated during adolescence (e.g., DuRant, Smith, Kreiter, & Krowchuk, 1999). These increase in frequency over the adolescent years (Brener & Collins, 1998) and tend to cluster together (Pena, Matthieu, Zayas, Masyn, & Caine, 2012). Further, these behaviors significantly threaten adolescent health and well-being, result in high usage of public health services at enormous costs, and place long-term constraints on further growth and development (e.g., Youngblade, Curry, Novak, Vogel, & Shenkman, 2006). Studies indicate that risky behaviors such as unsafe sex, self-harm, and illicit substance use are key risk factors for incident disability-adjusted life years (DALYS), contributing from 2% to 4% to DALYS among 10- to 24-year-olds globally (Gore et al., 2011). Suicidality is of particular concern because it is among the primary reasons for use of costly emergency and inpatient services (Peterson, Zhang, Santa Lucia, King, & Lewis, 1996), and suicide is the 2nd leading cause of death among 15-24 years-old in the U.S. (Centers for Disease Control and Prevention, 2017). When risky self-damaging behaviors are clustered together, the morbidity, mortality, and social costs are likely to be much higher.

Clustering of these risky self-damaging behaviors also characterizes the adult psychiatric diagnosis of borderline personality disorder (BPD) (American Psychiatric Association (APA), 2013). Importantly, as noted in the DSM-5 criteria for BPD, individuals who engage in multiple types of risky self-damaging behavior also tend to display particular forms of emotional and
social dysregulation, including labile moods, inappropriate intense anger, and conflicted and unstable relationships, as well as identity diffusion, feelings of emptiness, and fears of abandonment (APA, 2013). Not surprisingly, adolescents with elevated BPD features show serious impairments in functioning and high rates of psychiatric service use, whether or not they meet full criteria for BPD (Chanen & Kaess, 2012). Thus, BPD-related features in adolescence, features that include both risky self-damaging behaviors and particular forms of social-emotional dysregulation, are of particular interest, because together they index a prominent form of psychiatric disorder in adulthood. In the current study, the very rich NICHD SECCYD database (NICHD Early Child Care Research Network, 2001) was leveraged to develop a composite index of BPD-related features at age 15, including risky self-damaging behaviors, suicidal ideation, and relevant forms of social and emotional dysregulation, from the extensive set of validated measures of adaptive and maladaptive functioning included in that study.

The developmental trajectories toward adolescent BPD-related features have been a topic of considerable interest, as reviewed below (e.g. Michonski, Sharp, Steinberg, & Zanarini, 2013; Sharp, Steinberg, Temple & Newlin, 2014; Stepp et al. 2014; Vaillancourt et al., 2014; Wolke, Schreier, Zanarini, & Winsper, 2012). In particular, increased understanding of the developmental precursors of adolescent BPD-related features in infancy, preschool, and middle childhood would facilitate earlier identification and treatment of children at risk, before the increase in morbidity and mortality evident in adolescence.

Theories of the etiology of BPD include complex transactions between biological vulnerabilities and family environment, including characteristics of the child (e.g., impulsivity, negative affectivity) and the family environment (e.g., ineffective parenting, disorganized attachment, invalidation of emotions) (Crowell, Beauchaine & Linehan, 2009; Gunderson &
The influence of family environment has been theorized to begin early in development by a number of authors (Fonagy & Luyten, 2009; Cole, Llera, & Pemberton, 2009; Gunderson & Lyons-Ruth, 2008; Carlson, Egeland, & Sroufe, 2009). In one of the more detailed theoretical accounts, Fonagy and Luyten (2009) have speculated that the early caregiving context and disruptions in attachment relationships lead to poor self-other differentiation that perpetuates a combination of low thresholds for activation of the attachment system under stress, in concert with low thresholds for deactivation of the capacity for controlled mentalization. These low thresholds infringe upon one’s capacity to navigate and understand interpersonal relationships. In a number of contemporary theories (see Winsper, 2018), the negative effects of poor early parental regulation, combined with child temperamental vulnerability, impede the development of emotional regulation ability, in particular (Linehan 1993; Crowell et al. 2009; Cole et al., 2009). By middle childhood, difficulties in emotion regulation also lead to disturbed peer relationships. These difficulties in middle childhood are viewed as further linking earlier risk factors to the development of BPD features in adolescence. This general developmental model provides one potential account to explain the difficult interpersonal cycles, affective dysregulation, and high levels of impulsivity, as well as the painful inner experiences of aloneness and identity diffusion of individuals experiencing BPD. Thus, theoretically, BPD-related features are viewed as rooted in family processes that affect early social-emotional regulation, rather than emerging in adolescence primarily in response to new developmental stressors. In the current work, videotaped assessments of parenting sensitivity from infancy to adolescence were available and included in longitudinal modelling to assess the contributions of parenting over time to BPD-related features at age 15.
While prior theoretical models have not focused on a potential contribution of maternal depression, the developmental psychopathology literature points to maternal depression as a contributor to particular self-damaging behaviors in adolescence, as well as to BPD in adulthood. Maternal depression is associated with an increased risk of suicidal ideation in offspring (Hammerton, Zammit, Thapar & Collishaw, 2016), as well as with disturbed eating behaviors (Tafà et al., 2017). In addition, children from adverse family backgrounds, including a history of maternal depression, are at increased risk for behavioral and emotional dysregulation at age 11 (Winsper, Zanarini, & Wolke, 2012). Finally, studies have shown significant effects of parental depression on offspring substance use (Campbell, Morgan-Lopez, Cox, & McLoyd, 2009). These effects of parental depression may be understood theoretically as operating through both biological and social mechanisms (e.g. Crowell et al., 2009). Family association studies have shown that first-degree relatives of those with BPD are more likely to have affective disorders, as well as increased rates of BPD (Zanarini, Barison, Frankenburg, Reich, & Hudson, 2009). In addition, parental depression has repeatedly been associated with impaired parenting, including both less parental structure and support and increased parental hostility (Goodman & Gotlieb, 2002; Lyons-Ruth, Wolfe, Lyubchik, Grogan, & Steingard, 2002). Thus, it is important to model how maternal depression at particular developmental stages may contribute to pathways to BPD-related features in adolescence. The SECCYD database also includes repeated assessments of maternal depression over time, so that longitudinal path models in the current study also assess its contributions.

Particular child behaviors have also been proposed as possible early precursors of BPD-related features. In the infancy period, disorganized attachment has been shown to contribute to prediction of BPD features in early adulthood (Carlson et al., 2009; Lyons-Ruth, Bureau, Holmes,
In infancy, the term ‘disorganized’ refers to the apparent lack of a consistent way of organizing attachment responses to the parent when under stress (Main & Solomon, 1990). Starting at age three, controlling attachment patterns also emerge in which the child actively attempts to control or direct the parent’s attention and behavior in either a punitive or caregiving manner (Main & Cassidy, 1988). Lyons-Ruth et al. (2013) found that disorganized-controlling attachment patterns observed at eight years of age added to prediction of BPD features at age 19. These controlling patterns are also of interest because Kobak et al. (2017) found that, among male adolescents, controlling-caregiving patterns of interaction with parents at age 13 predicted increased levels of risky self-damaging behavior by age 15. Finally, Lyons-Ruth, Brumariu, Bureau, Hennighausen, and Holmes (2015) found that elevated BPD features, as well as increased incidence of suicidality/self-injury specifically, were associated with controlling-caregiving patterns of interaction with mothers at age 19. Both disorganized attachment at age 15 months and controlling forms of attachment at age three years were assessed in the SECCYD and are included in the longitudinal path models assessed in the current study.

Major theoretical models (e.g. Crowell et al., 2009; Fonagy & Luyten, 2009; Gunderson & Lyons-Ruth, 2008) also suggest that peer interactions indicating heightened risk for later BPD are likely to be identifiable by middle childhood. Regarding child behavioral precursors in middle childhood, the Avon Longitudinal Study of Parents and Children (ALSPAC) found that increased experiences of bully-victimization between 4 and 10 years of age were precursors to age 11 affective dysregulation, impulsivity, and interpersonal dysfunction, assessed by interview (Wolke et al., 2012). These are quite similar to the forms of behavioral and emotional dysregulation observed as criteria for BPD in adulthood and are thought to be likely precursors of BPD-related features in adolescence (Wolke et al.). In addition, other forms of childhood
dysregulated behavior between 4 and 8 years (i.e., negative emotionality, externalizing problems, and hyperactivity) also predicted age 11 affective dysregulation, impulsivity, and interpersonal dysfunction (Winsper, et al. 2012). In the McMaster Teen Study, childhood relational aggression by boys at ages 10 to 14, and both physical and relational aggression by girls at age 14, were related to emotional dysregulation at age 14 (Vaillancourt et al., 2014).

The ALSPAC Study also found that being bullied and victimized in middle childhood (7-10 years) was specifically associated with self-harm at 16 to 17 years (Lereya et al., 2013). Similarly, Klomek et al. (2009) found that children involved in bullying, both bully/victims and chronic victims, were at increased risk for suicidality/self-injury in preadolescence. Finally, studies have confirmed that peer victimization is often associated with substance use (Earnshaw et al., 2017) and disordered eating (Lee & Vaillancourt, 2018). Thus, peer experiences of bully-victimization in middle childhood contribute to emotional and behavioral dysregulation. In the SECCYD study, several relevant indices of social-emotional dysregulation in middle childhood were available, including intense anger, suicidal ideation, impulsivity, peer conflict, and victimization. Based on the above literature, a composite measure of middle childhood social-emotional dysregulation was included in the longitudinal model as one potential mediator of the effects of early insensitive parenting and disturbed attachment on adolescent BPD-related features.

In addition to general models emphasizing maladaptive parenting, increasing empirical work has looked more specifically at the particular forms of parenting disturbances related to the emergence of BPD-related features in adolescence. A variety of studies have linked aspects of parenting to adolescent self-damaging behavior. Exposure to harsh parenting has emerged as one factor related to alcohol use and risky sex (Alati et al., 2014; Guilamo-Ramos et al., 2012).
Other studies have found a relation between lack of parental support and self-damaging behaviors such as suicidal ideation, substance use, and disturbed eating (Hammerton et al., 2016; Tafà et al., 2017).

Several relevant studies have also used direct observation in short- or long-term longitudinal designs to assess the contributions of particular aspects of early parenting and attachment quality to the development of BPD features in adulthood. Carlson et al. (2009) reported a relation between observed harsh parenting at age three and the extent of BPD features assessed at age 20. Lyons-Ruth et al. (2013) found that observed maternal withdrawal in infancy was a potent predictor of both suicidality/self-injury specifically and also of overall BPD features at age 19. Using expressed emotion in parental speech samples, Belsky et al. (2012) found that exposure to harsh treatment in the family environment through age 10 predicted affective dysregulation, impulsivity, and interpersonal dysfunction at age 12. Winsper et al. (2016) meta-analysis showed an approximately three-fold increase in risk of BPD-related features in children and adolescents exposed to maternal hostility or verbal abuse.

Importantly, Stepp et al. (2014) demonstrated reciprocal associations between parenting behavior and adolescent BPD symptoms, in that low parental warmth and harsh punishment predicted subsequent increases in adolescent BPD features. In turn, adolescent BPD features predicted subsequent decreases in parental warmth and increases in harsh punishment. In a narrative review of the parenting behaviors of mothers who themselves have BPD, Stepp, Whalen, Pilkonis, Hipwell, and Levine (2011) suggested that mothers with BPD may oscillate between extreme forms of hostile control and passive aloofness in their interactions with their children. Therefore, the existing literature suggests that both withdrawing (lack of parental support) and harsh or hostile parenting, either in oscillation or in different developmental
periods, may make contributions to the emergence of BPD-related features in adolescence. To address this question of whether particular aspects of insensitive parenting may be differentially important in pathways to BPD, latent class analysis was also used to identify distinct classes of parents exhibiting qualitatively different profiles of early parenting. Those early parenting classes were then assessed for how different patterns of parenting might relate to BPD-related features in adolescence.

**Aims of the Current Study:** In the current study, we leverage the unusually comprehensive longitudinal database from the NICHD SECCYD ($n = 1364$). The SECCYD is one of the only large-scale longitudinal studies to include repeated videotaped assessments of mother-child interaction from infancy to age 15, as well as videotaped assessments of early attachment behaviors. As noted, the database also includes assessments of a number of other parental and child factors thought to be theoretically central to the emergence of BPD-related features at age 15. In middle childhood the SECCYD database provides assessments of bully-victimization, peer conflict, intense anger, impulsivity, and suicidality. Finally, although no validated BPD scales for children and adolescents were available at the time of the SECCYD, a range of risky or self-damaging behaviors and their associated forms of social and emotional dysregulation were assessed at age 15 using well-validated measures of substance abuse, risky sexual behavior, disturbed eating, suicidal thoughts, peer conflict, bully/victimization, impulsivity, and uncontrolled, intense anger.

The first aim of the study was to evaluate a path model of longitudinal contributors to BPD-related features at age 15. Based on the literature reviewed above, we expected that early maternal insensitivity and depression in the infancy and preschool periods would lead to difficulties in emotion regulation and conflicted peer relations in middle childhood. In turn, such
difficulties in middle childhood would add to the likelihood of the child exhibiting BPD-related features in adolescence. In addition, if maternal depression or insensitivity were not limited to early childhood but continued into middle childhood and adolescence, such continued parental dysfunction would be likely to contribute further to BPD-related features at age 15.

Thus, the following hypotheses were evaluated through path modeling: 1.) Insecure parenting, maternal depression, and child disorganized attachment behavior in the first five years of life will contribute to BPD-related features in adolescence; 2.) The influence of early parenting insensitivity and depression will be mediated in part through the continuation of these maladaptive processes through middle childhood and adolescence; 3.) The influence of early parenting insensitivity and depression will be also be mediated in part through their influence on the emergence of social-emotional dysregulation in middle childhood.

The second aim of the study was to supplement path models based on the continuous index for maternal sensitivity with person-centered latent class models. The central parenting variable in the SECCYD was a composite measure of maternal sensitivity-insensitivity, with sensitivity defined as “being aware of and correctly interpreting the child’s signals, and responding appropriately and promptly” (Ainsworth et al., 1974, p. 129). However, maternal insensitivity can be manifested in varied ways, including harsh parenting and withdrawn parenting, that may make different contributions to BPD-related features. Person-centered models are better able to identify distinct groups of parents who differ in their profiles of parental behavior. Evidence suggests that both hostile/harsh and withdrawn parenting profiles are relevant for the development of BPD-related features (Lyons-Ruth et al., 2013; Winsper et al., 2013). By age 54 months, the SECCYD parental interaction assessments included six scales, only three of which were included in the single composite score for sensitivity (see Methods below).
Hypotheses for the LCA were the following: 1.) There would be several distinct patterns of early parenting in the sample that varied in hostility and withdrawal, and 2). Both hostile and withdrawn patterns would predict later social-emotional dysregulation in middle childhood and BPD-related features at age 15.

Method

Participants

Participants were enrolled in the (NICHD) Study of Early Child Care and Youth Development (SECCYD). Children and their families were recruited from 10 different sites across the United States when the target child was an infant (see NICHD Early Child Care Research Network, 2001, for recruitment details). For the present study, data were utilized from all phases of data collection: phase 1 (birth to age 3 years, $n=1364$), phase 2 (54 months through first grade, $n=1226$), phase 3 (second through sixth grades, $n=1061$), and phase 4 (age 15 years, $n=1009$). Mothers provided demographic data.

Of the recruited sample, 48.3% of target children were female and 19.6% were ethnic minority children (Caucasian, $n=1097$, African-American, $n=176$, and other ethnicity, $n=91$). Mothers’ levels of education ranged from 7 to 21 years $M(SD)=14.23$ (2.51). Approximately 10.3% ($n=139$) had not completed high school, 21% ($n=287$) completed high school or obtained a GED, 33.4% ($n=455$) completed some college, and 35.3% ($n=482$) had a college or graduate degree. Presence or absence of the mother’s partner in the home was examined at 1 mo., 36 mo., and 15 years, and the variable ‘single parenting’ indexed the number of those three assessments in which the partner was living in the home. Approximately 8.7% ($n=118$) did not have a partner in the home at any of these time points; 22.2% ($n=303$) had a partner at one time point, 24.2% ($n=330$) had a partner at two time points, and 44.9% of mothers ($n=613$) had a partner at all three
time points. Family income-to-needs ratio was assessed at ages 1 mo., 36 mo., 54 mo., grade 3, grade 5, grade 6, and 15 years. Income-to-needs ratios were highly correlated ($r$’s range from .49-.89, $p < .001$), therefore, a mean score across age was used in analyses, $M = 3.80$.

At age 15, measures of adolescent outcomes were obtained for 984 youth (72% of the original recruitment sample). Compared with adolescents who did not participate at age 15, adolescents who participated were more likely to have families with higher income-to-needs ratios ($t(833.05) = 7.30, p < .001, Ms = 2.87$ and 4.15, respectively). Compared with mothers who were no longer participants at age 15, mothers who did participate had more education ($t(1361) = 5.07, p < .001, Ms = 13.68$ and 14.45, respectively). There were no differences in retention by children’s ethnicity (White versus not-white: $\chi^2 (1) = 1.72, p = .19$). Girls were more likely to have data at age 15 than boys, ($\chi^2 (1) = 5.05, p < .05, 75\%$ of recruitment sample versus 69.5%, respectively).

**Measures**

**BPD-related features at age 15 (BPDRF-15).** A composite measure of BPD-related features at age 15 was created using well-validated measures assessing substance abuse, risky sexual behavior, disturbed eating, suicidal thoughts, disturbed peer relationships, impulsivity, and uncontrolled, intense anger (see Table 1 for descriptive statistics for individual indicators). To the extent possible, indicators were chosen to represent the BPD-related criteria represented in the adult DSM-5 criteria in order to maximize the relevance of findings to the understanding of BPD. Regarding indicators of risky behaviors, substance abuse was scored from the teen’s reported use of alcohol, marijuana, and tobacco on the Risky Behavior Questionnaire (adapted from Conger & Rand, 1994). Disordered eating was captured using the Eating Attitudes Test (Garner, Olmsted, Bohr, & Garfinkel, 1982), indexing bingeing and restricting behavior. Risky
sexual behavior was scored using teen responses on the Risky Behavior Questionnaire. Self-reported impulsivity was measured with scores on the eight-item Weinberger Adjustment Inventory (Weinberger & Schwartz, 1990; alpha = .82; sample item “I’m the kind of person who will try anything once, even if it’s not that safe.”)

Suicidal thoughts or behavior at age 15 were measured from the three suicidal thoughts/behavior items in the Youth Self Report (YSR; Achenbach, 1991a). In middle childhood (see below), these items were from the Child Behavior Checklist (CBCL; Achenbach, 1991b), administered to the study children’s mothers in grades 3, 4, 5, and 6. Across these multiple assessments, there were 105 children with at least one report of suicidal thoughts and/or behavior.

Lack of control of anger or inappropriate intense anger was measured from three relevant items on the YSR at age 15: “gets in many fights,” “physically attacks people,” and “temper tantrums/hot temper.” In middle childhood, lack of control of anger or inappropriate intense anger was measured from the same items on the CBCL. One hundred eighty-five children had reports of many fights and physical attacks on others.

Unstable and conflicted relationships were assessed based on repeated child reports of victimization on the Peer Social Support, Bullying, and Victimization questionnaire (Kochenderfer & Ladd, 1996), available at age 15 and in grades 3, 5, and 6, and on scores on the conflict and betrayal subscale of the Friendship Quality Questionnaire (Clark & Ladd, 2000), administered to children at age 15 as well as in grades 3, 4, 5, and 6. An overall composite measure for BPD-related features at age 15 (BPDRF-15) was created by standardizing the scores of the 10 indicators above and then averaging the $z$ scores of the 10 indicators ($\alpha = .70$).

Social-emotional dysregulation in middle childhood (SEDR-MC). In middle childhood, many forms of risky self-damaging behavior are not yet apparent (e.g. risky sex, risky driving,
substance use, disordered eating). Thus, the middle childhood composites were comprised primarily of aspects of social-emotional dysregulation (see Table 1 for descriptive statistics for individual indicators). For grades 3, 5 and 6, four relevant indicators of social-emotional dysregulation were available, including anger, suicidality, conflict, and victimization, as described above. At grade 4, anger, suicidality, and conflict were available, but not victimization. For grade 1, only anger and suicidality were available, so grade 1 was not included in the analyses. For middle childhood, the three or four available indicators were composited at each age by standardizing the scores and then averaging the z scores of the indicators to form a composite score for social emotional dysregulation. Social emotional dysregulation scores for each grade showed good stability from grades 3 to 6, with r’s ranging from .41 to .55, all p < .001. The four composite scores for grades 3, 4, 5, and 6 were then averaged into a single composite score to reflect overall middle childhood SEDR-MC (α = .79).

Maternal sensitivity. Maternal sensitivity was indexed using composites of observational ratings based on videotaped mother-child interaction tasks at 6, 15, 24, 36, and 54 months; at grades 1, 3, and 5; and at 15 years. At ages 6, 15, and 24 months, the sensitivity composite was the sum of 4-point ratings of three items: sensitivity to non-distress, positive regard, and intrusiveness (reverse scored). At age 54 months and grades 1, 3, and 5, the sensitivity composite was the sum of 7-point ratings of supportive presence, respect for autonomy, and hostility (reverse scored). At age 15, the sensitivity variable was a composite of six 7-point ratings for validation/agreement, engagement, inhibiting relatedness (reversed), hostility/devaluing (reversed), respect for autonomy, and valuing/warmth. Interrater reliability for each rating item above ranged from .68 to .89 (n’s 195 to 259). The composite variables had reliabilities ranging from .83 to .91 (α’s from .79 to .88).

Maternal sensitivity variables across infancy/early childhood were significantly
associated, $rs = .30$ to .52, $p$'s $< .001$, as were maternal sensitivity variables across middle childhood, $rs = .37$ to .42, $p < .001$. Because we were interested in maternal sensitivity at three key developmental periods (infancy/preschool; middle childhood; and adolescence), maternal sensitivity composite variables were averaged over each developmental period, $\alpha = .75$ for infancy/preschool years; $\alpha = .65$ for middle childhood. Sensitivity at age 15 was a single score.

**Maternal interaction at 54 months.** In addition to the widely used SECCYD sensitivity composites described above, the six mother-child interaction ratings assessed at 54-months were used for person-centered latent class analyses (LCA) to identify patterns of early parenting. The rating items at 54 months included the three items used to generate the sensitivity composites above, as well as three additional items assessing cognitive stimulation, quality of assistance, and confidence. All six items were used to more specifically characterize maternal behavior for the LCA. Interrater reliability ($n = 242$) ranged from .78 to .87 for the three additional items not reported above.

**Maternal depression.** Mothers completed the Center for Epidemiologic Studies Depression Scale (CES-D, Radloff, 1977) at 1 month, 6 months, 15 months, 24 months, 36 months, 54 months, 1st grade, 3rd grade, 5th grade, 6th grade, and age 15 years ($\alpha$s = .88 to .92). The CES-D is a widely used 20-item, self-report scale used to measure current levels of depressive features, with well-established reliability and validity (Radloff, 1977). Items are rated on a 4-point scale according to how often they were experienced over the past week. Correlations of scores across time points ranged from .35 to .58, $ps < .001$. Similar to maternal sensitivity, composite scores were created for the infancy/early childhood period (1 to 54 months, $rs = .39$ to .58, all $ps < .001$, $\alpha = .84$), and the middle childhood period (1st to 6th grades, $rs = .45$ to .58, all $ps < .001$, $\alpha = .80$). Age 15 was indexed by a single score.
**Attachment insecurity and disorganization.** Attachment was assessed at 15 months with the Strange Situation Procedure (SSP; Ainsworth, Blehar, Waters, & Wall, 1978) and at 36 months with the modified Strange Situation Procedure (SSPm; Cassidy, Marvin, & MacArthur Working Group, 1992). During the SSP, the mother leaves and rejoins the infant twice, first leaving the infant with a female stranger, then leaving the infant alone to be rejoined by the stranger. The procedure is designed to be mildly stressful in order to increase the activation of the infant’s attachment behavior. Videotapes were coded for secure, avoidant, ambivalent, disorganized, and unclassifiable attachment behavior (Ainsworth et al., 1978; Main & Solomon, 1990). Intercoder agreement among three trained coders was 83% (K = .69; N = 1201), with 59.6% secure, 13.4% avoidant, 6% ambivalent, and 18.4% disorganized. The 3.5% in the unclassifiable group were combined with the disorganized category.

The SSPm at 36 months also consists of two separations and reunions, with the second separation longer than in infancy (5 min.) to be more stressful for older children (Cassidy, Marvin, & MacArthur Working Group, 1992). For the four category ABCD system, intercoder agreement among three trained coders was 75.7% (K = .58; N = 867), with 61.5% secure, 4.8% avoidant, 17.3% ambivalent, and 16.4% disorganized/controlling. A number of studies have provided evidence for adequate psychometric properties of the SSP and the SSPm (van IJzendoorn, Schuengel, & Bakermans-Kranenburg, 1999). Following precedent (van IJzendoorn et al., 1999), to capture overall quality of attachment at each age we used a three-level scale (1 = secure, 2 = organized avoidant and ambivalent, and 3= disorganized or controlling).

**Plan of Analysis**

Missing data ranged from 0 to 34.6%. For control analyses and descriptive statistics, only participants with data on the relevant variables were included and pairwise tests were used
to retain maximum power (see n’s Tables 1 and 2). To retain power for the main analyses, both bivariate correlations involving SEDR-MC and BPDRF-15 and subsequent path modelling of the direct and indirect paths from predictor variables to SEDR-MC and BPDRF-15 were conducted using full information maximum likelihood (FIML) for analysis and estimation of missing data (AMOS software). FIML provides a less biased estimate and is more efficient in handling missing data than are more conventional methods (Schafer & Graham, 2002).

The sequence of analyses was as follows: First, we evaluated whether BPDRF-15 was predicted by SEDR-MC. Second, we evaluated whether the composite scores for maternal sensitivity and maternal depression across infancy/early childhood, middle childhood, and at age 15 were related to SEDR-MC and BPDRF-15. We also examined associations between mother-child attachment at 15 and 36 months and SEDR-MC and BPDRF-15. Third, we conducted path models to evaluate the direct and indirect pathways through which maternal sensitivity, maternal depression, and mother-child attachment predicted the emergence of SEDR-MC and BPDRF-15. Finally, to assess which profiles of early parenting disturbance were most predictive of SEDR-MC and BPDRF-15, the 54-months ratings were used for person-centered latent class analyses and latent classes were assessed via ANOVA for prediction of SEDR-MC and BPDRF-15.

Results

Control Variables and Descriptive Statistics

Preliminary analyses investigated whether demographic variables were related to BPDRF-15. Because family income-to-needs was highly related to maternal education ($r = .55$, $p < .001$), a single socioeconomic status (SES) score was created by averaging the standardized scores of the two variables. Both higher SES and single parenting were negatively correlated with BPDRF-15 ($SES \ r = -.18, \ p < .001$; single parenting $r = -.20, \ p < .001$). Gender was
unrelated to BPDRF-15 ($t(982) = -0.35, p > .05$). Ethnicity was related to BPDRF-15 ($F(2, 981) = 3.15, p < .05$), but the Bonferroni post-hoc analysis showed no significant differences among groups. Therefore, only SES and single parenting were included as covariates in the path analyses.

Descriptive statistics for key variables are shown in Table 2. Among the early childhood predictors, quality of attachment (security/insecurity/disorganization) at 15 and 36 months were modestly related ($r = .07, p < .05$). Quality of attachment at 15 months was also modestly associated with maternal sensitivity in early childhood ($r = .08, p < .01$), but not with early maternal depression. Quality of attachment at 36 months was significantly associated with both early sensitivity ($r = .22, p < .001$) and early maternal depression ($r = -.11, p < .001$). Early maternal depression was negatively related to early sensitivity ($r = -.31, p < .001$). (Associations at other ages are presented in the context of path analyses below).

### Bivariate Relations of Predictors with Outcomes

**Early attachment and later outcomes.** Quality of attachment at 15 months was significantly but weakly associated with BPDRF-15 ($r = .07, p < .05$) but not with SEDR-MC ($r = .01, p = ns$). Quality of attachment at 36 months was significantly associated with SEDR-MC ($r = .10, p < .05$) but not BPDRF-15 ($r = .03, p = ns$). Follow-up orthogonal comparisons indicated that these results were driven by attachment disorganization rather than by attachment insecurity (secure vs organized insecure, all ns; organized vs disorganized, 36 months to SEDR-MC, $F(1, 1006) = 5.49, p < .05, \eta^2 = .005$; 15 months to BPDRF-15, $F(1, 931) = 3.59, p =.06, \eta^2 = .004$).

**Maternal sensitivity and depression and later outcomes.** Maternal sensitivity at all three developmental periods was significantly related to both SEDR-MC and BPDRF-15
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(SEDR-MC: Infancy/preschool \( r = - .25 \), Middle Childhood \( r = - .27 \), Age 15 \( r = - .17 \), all \( p < .001 \); BPDRF-15: Infancy/preschool \( r = - .19 \), Middle Childhood \( r = - .17 \), Age 15 \( r = - .16 \), all \( p < .001 \)). The same was true of maternal depression (SEDR-MC: Infancy/preschool \( r = .24 \), Middle Childhood \( r = .33 \), Age 15 \( r = .22 \), all \( p < .001 \); BPDRF-15: Infancy/preschool \( r = .13 \), Middle Childhood \( r = .17 \), Age 15 \( r = .19 \), all \( p < .001 \)).

**SEDR in middle childhood and BPDRF-15.** Finally, bivariate correlations indicated that higher levels of SEDR-MC (grades 3, 4, 5, and 6) were associated with higher levels of BPDRF at age 15 \( (r = .26, p < .001) \). Thus, early forms of social-emotional dysregulation in middle childhood predicted increased BPD-related features in mid-adolescence.

**Path Modelling of Trajectories from Early Childhood to BPDRF-15**

The theoretical path model of predictors of BPDRF-15 is shown in Figure 1, as represented by both solid and dotted lines. A path analysis assessed the goodness of fit of the proposed model, including indirect effects from early maternal sensitivity, early maternal depression, and early attachment quality through middle childhood SEDR to BPDRF-15. Because attachment at 15 months was directly related only to BPDRF-15, we only included that direct pathway. Because single parenting and SES were also significantly related to BPDRF-15, we included those variables as covariates. Covariances among all predictor variables were included in the model.

The model showed good fit to the data \( (\chi^2 (36) = 194.97, p < .001; CFI = .95, IFI = .95; TLI=.89, RMSEA = .06). \) Not surprisingly, with all variables included in the model, several of the paths significant in bivariate correlations did not make independent contributions to prediction, including quality of attachment at 15 and 36 months, maternal depression in infancy/preschool, and family SES, as indicated by the dotted lines in Figure 1. Thus, we
trimmed the non-significant paths from the model (significant standardized path estimates shown in solid lines in Figure 1). The final model showed very good fit to the data ($\chi^2 (22) = 107.41, p < .001; \text{CFI} = .97, \text{IFI} = .97, \text{TLI} = .93, \text{RMSEA} = .05$) and was a better fit than the non-trimmed model (delta CFI = .02), based on adequate cut-off differences proposed by Meade, Johnson, and Braddy (2008).

Because the distributions of some variables in the model deviated from normality (maternal sensitivity in middle childhood, skewness = -1.35; kurtosis = 3.83; social-emotional dysregulation in middle childhood, skewness = -2.44; kurtosis = 12.43; BPD-related features at age 15, skewness = -1.75; kurtosis = 3.70), we also ran the models using a robust maximum likelihood estimator (MLR) that is less biased when facing departures from normality in the data (Savalei, 2010; Yuan & Bentler, 2000). All results were similar to those obtained using FIML (original model: $\chi^2 (36) = 179.10, p < .001; \text{CFI} = .91, \text{TLI} = .88, \text{RMSEA} = .054$; trimmed model: $\chi^2 (22) = 90.40, p < .001; \text{CFI} = .95, \text{TLI} = .93, \text{RMSEA} = .05$; delta CFI = .04).

Individual effect sizes in the model were also similar. Thus, the reported results were robust to these departures from normality. Because MLR does not model missing data, however, FIML was retained as the primary estimator.

**Assessment of Mediating Paths**

In the final model in Figure 1, significant infancy/preschool predictors were linked to BPDRF-15 via seven potential mediating paths (see Table 3). It should be noted that most of these paths did not represent links across three different developmental timepoints, so the results should not be interpreted as implying etiological effects. However, the assessment of whether maternal behavior in a later period was statistically mediating a link between maternal behavior in an earlier period and child effects in a later period adds important information to the model.
Therefore, the significance of the seven potential indirect paths were assessed using the confidence interval for the mediated effects method. This method creates asymmetrical confidence intervals based on the assumption of a non-normal distribution of the indirect product term. Evidence for mediation is assumed if the 95% confidence interval does not include zero (Tofghi & MacKinnon, 2011). Six of the seven potential paths were significant (Table 3).

These mediated effects fell into three groups. Two paths indicated that early maternal insensitivity and early maternal depression were linked to SEDR-MC in part through the continuity of maternal insensitivity and depression into middle childhood. Second, two paths indicated that maternal insensitivity and depression in middle childhood were related to BPDRF-15 in part through the emergence of social and emotional dysregulation in middle childhood. The final two paths indicated that maternal insensitivity and depression in middle childhood were related to BPDRF-15 in part through the continuity of maternal insensitivity and depression to age 15.

**Alternative Path Model: Insensitivity as Mediator of Effects of Maternal Depression?**

In the current data, maternal depression was related to maternal sensitivity both in early childhood (standardized estimate = -.31, \( p < .001 \)) and in middle childhood (standardized estimate = -.10, \( p < .001 \)). The relation did not reach significance at 15 years (standardized estimate = -.05, \( p = .13 \)). Thus, we also tested an alternative model in which the effects of maternal depression were not independent but, rather, were mediated through maternal sensitivity. To test this model, we removed the direct paths from maternal depression to SEDR-MC and BPDRF-15 and added indirect paths through maternal sensitivity to SEDR-MC and BPDRF-15. This mediated model provided a relatively poor fit to the data, \( \chi^2 (23) = 515.79, p < .001, \) CFI = .80, IFI = .80, TLI = .61 and RMSEA = .13). Thus, results indicated that maternal
depression was not primarily affecting SEDR-MC and BPDRF-15 through decreases in maternal sensitivity.

**Do Particular Classes of Parenting Disturbance Predict SEDR-MC or BPDRF-15?**

Results of the path modeling indicated that pathways from maternal insensitivity to adolescent BPDRF-15 had their beginnings in the infancy/preschool period. Using the sensitivity composite from the SECCYD as a single continuous variable was parsimonious for the path modelling and provided a consistent metric for maternal behavior over all ages of the study. However, given the importance of maternal behavior beginning in the infancy/preschool period for later prediction of both SEDR-MC and BPDRF-15, and given the diverse parenting problems that have been associated with these difficulties in the literature, it was also important to generate a more fine-grained look at the aspects of maternal behavior most predictive of later outcomes. Thus, a latent class analyses was conducted including the six rating scales indexing quality of mother-child interaction at 54 months of age.

The LCA was run as an iterative model, starting with a single group and continuing until the model fit was no longer better and statistically different than the previous model. Model fit was assessed for 1-, 2-, 3-, 4-, and 5-group models. Based on the high entropy score (the closer to 1, the better the fit) and the significant LMR, which is used to determine whether there is statistically significant improvement over a model with one fewer classes (k-1) (Nylund, Asparouhov, Muthén, 2007), the four-class model provided the best fit to the data (BIC = 21882.03, Entropy = .90, LMR = P < .01; see Figure 2).

Descriptively, examination of the profiles in each class indicated the following characterizations: Class 1 was labeled “Optimal” and indexed sensitive, responsive, and involved parenting, as shown by the high ratings on all positive scales and low ratings on negative scales.
This class included 36.76% of parents ($n = 386$). Class 2, called “Sub-Optimal Support” indexed a parenting style in which hostility remained low, but mean ratings for supportive presence and respect for autonomy were approximately one scale point lower on average than the Optimal class, and scales for cognitive stimulation, quality of assistance and confidence were approximately 1.5 scale points lower. The Sub-Optimal Support parenting group accounted for 47.23% of parents ($n = 446$). Mothers in Class 3 were labeled “Withdrawn” because, while hostility remained low, the mother’s lack of supportive involvement was notable. Support for autonomy was 1.5 scale points lower than Optimal, but other rating items were even lower, with supportive presence and confidence approximately 2.5 scale points lower than in the Optimal group and quality of assistance and cognitive stimulation approximately 3 scale points lower than in the group labeled Optimal parenting. The Withdrawn parenting group accounted for 15.04% of parents ($n = 158$). Class 4, labeled “Hostile-Unsupportive”, made up the smallest group, accounting for only 4.76% of parents ($n = 50$). Mothers in the Hostile-Unsupportive group had scale scores for hostility that were 3 scale points above all other groups. In addition, their scale scores for respect for autonomy were lower than all other groups, indicating an intrusive or interfering quality to the mother’s behavior. Further, their scale scores for supportive presence, quality of assistance, and confidence were similar to those of the withdrawn group. Thus, mothers in this group combined high levels of hostility and low levels of respect for autonomy with low levels of assistance, stimulation and support.

Two one-way ANOVAs were then conducted using the four-class parenting categorization as the independent variable and SEDR-MC and BPDRF-15 as the outcome variables. For middle childhood, prediction of SEDR-MC from the four parenting classes was significant, $F(3, 979) = 10.64$, $p < .001$, $\eta^2 = .03$, and a post-hoc Tukey analysis revealed that
SEDNR-MC was significantly elevated among children whose mothers were in the Sub-optimal, Withdrawn, and Hostile parenting classes, compared to children whose mothers were in the Optimal parenting class (all ps < .001, M(SD) optimal parenting = -.11(.38), M(SD) suboptimal parenting = .02(.48), M(SD) withdrawn parenting = .09(.60), M(SD) hostile parenting = .19(.56)).

For BPDRF-15, prediction from the four classes was also significant, $F(3, 888) = 4.90, p < .01, \eta^2 = .02$ (see Figure 3). However, post-hoc Tukey analyses revealed that only adolescents of mothers in the Withdrawn class at 54 months had elevated BPDRF-15 compared to those of mothers in the Optimal parenting class, $M(SD)$s = .09(.58) and -.09(.48), respectively, $p < .01$. There was also a marginally significant difference between adolescents whose mothers were in the Sub-optimal parenting group, compared to those whose mothers were in the Optimal parenting group, $M(SD)$s = .01(.55) and -.09(.48), $p = .059$. No other comparisons were significant ($M(SD)$ hostile parenting = .12(.63)).

**Discussion**

A number of investigators have shown that a coherent clustering of BPD-related features can be identified in mid-adolescence (Sharp et al., 2014; Stepp et al., 2014; Vaillancourt et al., 2014). In addition, the public health costs of such behaviors are known to be considerable (e.g., Youngblad et al., 2006). However, we still know relatively little about the earlier developmental pathways leading to BPD-related features in adolescence. The general developmental model reviewed earlier posits that BPD-related features are rooted in family processes that affect early social-emotional regulation, rather than emerging in adolescence in response to new developmental pressures. Further, the negative effect of poor parental regulation, combined with child temperament vulnerability, impedes the development of emotional regulation ability, so that by middle childhood difficulties in emotion regulation also lead to disturbed peer
relationships. These difficulties in middle childhood further link early risk factors to the development of BPD features in adolescence. Thus, the first aim of the current study was to assess this general model and evaluate the direct and indirect paths through which earlier risk factors influenced the emergence of BPD-related features at age 15.

First, in bivariate correlations, maternal insensitivity during each developmental period was significantly related to both social-emotional dysregulation in middle childhood and to BPD-related features at age 15. Further, in the multivariate path model, insensitivity during each developmental period continued to add independent prediction to the model with other variables controlled. Thus, it mattered how long the child experienced maternal insensitivity from infancy to adolescence in the prediction of BPDRF-15. In addition, mediation analyses indicated that maternal insensitivity in the infancy/preschool period contributed to BPDRF-15 primarily through the continuation of maternal insensitivity into the grade-school years. Insensitivity in middle childhood then further contributed to BPDRF-15 by contributing to the emergence of social and emotional dysregulation in middle childhood. In addition, maternal insensitivity in middle childhood signaled a risk for continuity of insensitivity into adolescence, which further added to prediction of BPDRF-15. The model, then, tracks the repeated additional contributions of maternal insensitivity at each developmental period from infancy to adolescence. Given the strong continuity in maternal insensitivity over time shown in Figure 1, these results suggest that efforts to prevent the emergence of both SEDR-MC and BPDRF-15 need to begin with efforts to improve maternal sensitivity in early childhood.

Maternal depression also emerged as a contributing factor to SEDR-MC and to BPDRF-15, consistent with previous work (Belsky et al., 2012; Hammerton et al., 2016; Wolke et al., 2012). However, few studies have modeled the contribution of maternal depression from infancy
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to adolescence, particularly in relation to BPD-related features. Results indicated, first, that there was strong continuity in maternal depression across the three developmental periods (Figure 1). In addition, early maternal depression increased SEDR-MC through the continuity of maternal depression into middle childhood. This higher level of SEDR-MC further acted to increase the level of BPD-related features at age 15. Finally, maternal depression in middle childhood also influenced the level of BPD-related features at age 15 through the continuity of maternal depression into adolescence. Notably, an alternative path model assessing whether effects of maternal depression on BPDRF-15 were mediated by maternal insensitivity did not provide a good fit to the data. Thus, in future studies, other mediators of the effect of maternal depression on adolescent BPD-features will be important to explore, including the effects of shared genes (Belsky et al., 2012).

Consistent with prior longitudinal reports (Carlson et al., 2009; Lyons-Ruth et al., 2013), children who exhibited attachment disorganization in infancy were more likely to exhibit increased BPD-related features in adolescence. In addition, bivariate correlations indicated that disorganized/controlling forms of attachment at 36 months were particularly relevant to pathways toward SEDR-MC, characterized by peer conflict, victimization, anger, and suicidal thoughts. Notably however, once maternal factors were entered into the model, neither attachment assessment made an additional contribution to prediction. Thus, while early disorganized and controlling attachment were part of a set of related predictors, maternal insensitivity and depression over time better accounted for the variance in SEDR-MC and BPDRF-15.

The relative advantage of maternal assessments may be due to the repeated assessment of maternal factors in the SECCYD, while child attachment was only assessed up to 36 months of
Since the SECCYD was conducted, assessments of controlling attachment have also been validated in middle childhood and adolescence (Bureau, Easterbrooks, & Lyons-Ruth, 2009; Brumariu et al., 2018; Obsuth Hennighausen, Brumariu, & Lyons-Ruth, 2014). As noted earlier, using these more recent assessments, Lyons-Ruth et al. (2013) found that controlling attachment in middle childhood was a significant predictor of BPD features at age 19. Further, Kobak et al. (2017) found that controlling/caregiving interaction with parents at 13 years of age was a significant predictor of increased risky behavior at 15 years. Finally, Lyons-Ruth et al. (2015) found that controlling-caregiving interaction with parents at age 19 was a concurrent correlate of both overall BPD features and suicidality. Thus, future studies are needed that assess disorganized/controlling forms of attachment in both middle childhood and adolescence in relation to bully/victimization, conflict with peers, intense anger, suicidal ideation, and risky behaviors.

Notably, consistent with the general developmental model, social-emotional dysregulation across grades 3, 4, 5, and 6, including intense anger, peer conflict, peer victimization, and suicidal thoughts, was predictive of BPD-related features at age 15. Further, SEDR-MC was an important mediator of the effects of both maternal (in)sensitivity and maternal depression on age 15 outcome. These findings are important because BPDRF-15 is characterized by a variety of risky behaviors not prominent in middle childhood, including substance abuse, unsafe sexual behavior, risky driving, and disordered eating, as well as increased self-injury and suicidality. Thus, it is important to identify precursors to these adolescent risky behaviors that can be assessed in the grade school years. Our findings suggest that suicidal thoughts, anger, peer conflict and peer victimization are relevant aspects of dysregulation in middle childhood that are linked to the more pervasive forms of risky self-
damaging behavior seen in adolescence. Therefore, preventive efforts in middle childhood might significantly decrease the incidence of BPD-related features in adolescence, with their serious public health consequences.

Finally, results of the person-centered LCA were notable in indicating that all less-optimal parenting classes were associated with increased SEDR in middle childhood, with hostile-unsupportive parenting showing the largest effect. This finding is consistent with the evidence reviewed earlier of the importance of harsh parenting on middle-childhood self-regulation, as well as poor peer relations. In contrast, in relation to BPDRF-15, only early withdrawn parenting was a significant predictor. Notably, a similar pattern of results emerged in the only other longitudinal study to separately analyze both hostile and withdrawing forms of early maternal behavior. Hostile maternal interaction was particularly predictive of social-emotional maladaptation in middle childhood (e.g. Lyons-Ruth, Easterbrooks, & Cibelli, 1997), but withdrawing forms of maternal interaction were more salient in predicting the risky, self-damaging features of BPD at age 19 (Lyons-Ruth et al., 2013).

The emergence of a similar pattern of results in the much larger SECCYD sample points to the important role of active parental support and regulation in the early years as a foundation for the child’s ability to effectively self-regulate. In addition, the current data indicate that the effects of withdrawn parenting on BPD-related features become particularly salient in adolescence compared to middle childhood, perhaps because the adolescent increasingly needs to self-regulate behavior in such important areas as sexuality, substance use, eating behavior, self-care, driving, and handling money. While the convergent results of these two studies point to the significant long-term effects of withdrawal during the preschool period, further work is needed to separately assess withdrawal and hostility as parenting components in middle childhood and
adolescence, as well as to evaluate how these different forms of maladaptive parenting might affect different facets of development over time. Notably, withdrawn patterns of maternal interaction have not been studied as extensively as parental hostility/harsh discipline, so that assessments of maternal withdrawal should be included in future work on developmental contributors to the dysregulations in affect and behavior characteristic of BPD.

The latent class results were also important in indicating that withdrawing patterns of maternal behavior may be more prevalent than hostile patterns of maternal behavior. Only 5% of the sample was classified in the Hostile Unsupportive group at 54 months, while 15%, or three times as many, were classified in the Withdrawn category. Finally, another 47% were rated as sub-optimal in support and assistance provided to the child, and even those smaller decrements in support were associated with a significant increase in social-emotional dysregulation in middle childhood. Thus, if we are to intervene effectively to prevent the risky self-damaging behaviors characteristic of BPD, withdrawing patterns of maternal behavior, marked by low levels of supportive presence, quality of assistance and cognitive stimulation, may need increased attention in intervention efforts.

While the SECCYD was unparalleled in its repeated observations of parent-child interaction and its rich assessment of maladaptive child behaviors over time, limitations of the study should also be noted. First, the SECCYD did not include a validated measure of BPD features in adolescence, since the study was conducted before such validated assessments were available. Thus, our measure of BPD-related features relied on items from other validated assessments of related constructs. In addition, assessments of maltreatment were not included, so the contributions of maltreatment to social-emotional dysregulation and BPD-related features could not be evaluated (Zanarini et al., 1997). Further, family psychiatric assessments were not
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conducted. Belsky et al. (2012) found that the link between exposure to harsh treatment through age 10 and emotional/behavioral dysregulation at age 12 was stronger among children with a family history of psychiatric illness. Thus, parental psychiatric assessments would be important to include in future work and might add to the current findings regarding the role of maternal depression. In addition, the effect of maternal depression on child dysregulation over time was independent of maternal sensitivity, suggesting a role for other mediators, including shared genetic factors, which could be explored in future work. Relatedly, the low-risk nature of the SECCYD sample may have underestimated the influence of some predictors, in that more extreme values may be more frequent in high-risk samples. Thus, these effects need to be replicated in more diverse samples with a higher risk of psychopathology.

Notably also, the interactions of fathers and children were not consistently assessed, so the role of fathers in these pathways remains an important topic for future work. As noted under Results, we assessed indirect effects that were based, in part, on cross-sectional associations rather than on three distinct temporal waves. Thus, etiological inferences should not be drawn. Replication of these mediated paths is needed using non-concurrent assessments in a multi-wave design. In addition, randomized intervention designs will be needed to fully assess etiological effects. Finally, we evaluated a relatively parsimonious model from a theoretical standpoint which posits that maternal insensitivity and depression in earlier periods contribute to later child and adolescent BPD-related features. However, bidirectional effects are equally important to test as it is likely that children exhibiting increased BPD-related features may affect mothers’ ability to remain sensitive to their children’s needs and may negatively affect mothers’ mood and capacity to self-regulate.

In summary, the findings point to a long developmental trajectory toward BPD-related
features in adolescence, with early maternal insensitivity and depression likely to continue into middle childhood and contribute to middle childhood social-emotional dysregulation. This increased dysregulation in middle childhood then increases the BPD-related features emerging in adolescence. In addition, the continuation of maternal depression and insensitivity into adolescence further increases the level of BPD-related features in adolescence.

Fortunately, these long-term developmental trajectories also point to several potential targets for intervention. First, there are now a number of evidence-based interventions for increasing early maternal sensitivity and decreasing disorganized attachment (for a review, see Facompré, Bernard, & Waters, 2018). The current results also suggest that such efforts should address maternal withdrawal, in particular, to increase active maternal support, regulation, and stimulation for the child (Yarger, Bronfman, Carlson, & Dozier, 2019). Second, school-based interventions are also indicated by the current findings to prevent the emergence of the disturbed peer relations and emotional dysregulation that forecast increased BPD features in adolescence. Finally, working with adolescents and their parents to improve sensitive parent-adolescent communication is indicated as an important intervention target, as is increased treatment of maternal depression.
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doi:10.1017/S0954579414000418


Table 1.

Descriptive Statistics for Individual Unstandardized Indicators of Social-Emotional Dysregulation in Middle Childhood and BPD-Related Features at Age 15

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<th>Variables</th>
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<th>M(SD)</th>
<th>Minimum</th>
<th>Maximum</th>
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<tr>
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<td>.02 (.12)</td>
<td>0</td>
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</tr>
<tr>
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<td>2</td>
</tr>
<tr>
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<td>5</td>
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<tr>
<td>Victimization</td>
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<tr>
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Table 2.

_Descriptive Statistics for Dependent and Independent Variables_

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<th>Minimum</th>
<th>Maximum</th>
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<td>10.48 (9.83)</td>
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<td>54</td>
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<tr>
<td>7. Insecurity/disorganization (15 months)</td>
<td>1191</td>
<td>1.59 (.78)</td>
<td>1</td>
<td>3(^1)</td>
</tr>
<tr>
<td>8. Insecurity/disorganization (36 months)</td>
<td>1140</td>
<td>1.55 (.76)</td>
<td>1</td>
<td>3(^1)</td>
</tr>
<tr>
<td>9. SEDR-MC</td>
<td>1096</td>
<td>.0012 (.49)</td>
<td>-.93</td>
<td>4.82(^2)</td>
</tr>
<tr>
<td>10. BPDRF-15</td>
<td>984</td>
<td>-.0008 (.54)</td>
<td>-.76</td>
<td>2.70(^2)</td>
</tr>
</tbody>
</table>

SEDR-MC = social-emotional dysregulation in middle childhood; BPDRF-15 = BPD-related features at age 15; \(^1\) Coded 1 = Secure; 2 = Organized insecure; 3 = Disorganized (Disorganized/controlling at 36 months); \(^2\) Composite scores for SEDR-MC and BPDRF-15 could be negative due to standardization.
Table 3.

*Proposed Mediated Paths from Earlier Predictors to Later Outcomes through Mediating Variables*

<table>
<thead>
<tr>
<th>Mediated Paths</th>
<th>Indirect effect estimate (SE)</th>
<th>CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Infancy/preschool maternal sensitivity → <strong>Maternal sensitivity</strong> middle childhood → SEDR-MC</td>
<td>-.023 (.006)</td>
<td>[-.034, -.012]</td>
</tr>
<tr>
<td>2. Infancy/preschool maternal sensitivity → SEDR-MC → BPDRF-15</td>
<td>-.004 (.002)</td>
<td>[-.009, 0]</td>
</tr>
<tr>
<td>3. Maternal sensitivity middle childhood → SEDR-MC → BPDRF-15</td>
<td>-.008 (.002)</td>
<td>[-.012, -.004]</td>
</tr>
<tr>
<td>4. Maternal sensitivity middle childhood → <strong>Maternal sensitivity at age 15</strong> → BPDRF-15</td>
<td>-.009 (.003)</td>
<td>[-.015, -.003]</td>
</tr>
<tr>
<td>5. Infancy/preschool maternal depression → <strong>Maternal depression middle childhood</strong> → SEDR-MC</td>
<td>.013 (.002)</td>
<td>[.01, .016]</td>
</tr>
<tr>
<td>6. Maternal depression middle childhood → SEDR-MC → BPDRF-15</td>
<td>.004 (.001)</td>
<td>[.003, .006]</td>
</tr>
<tr>
<td>7. Maternal depression middle childhood → <strong>Maternal depression at age 15</strong> → BPDRF-15</td>
<td>.005 (.002)</td>
<td>[.002, .008]</td>
</tr>
</tbody>
</table>

*Note:* SEDR-MC = social-emotional dysregulation in middle childhood; BPDRF-15 = BPD-related features at age 15. Pathways with confirmed mediation are in bold. A mediated pathway from infancy/preschool maternal depression through SEDR-MC to BPDRF-15 was not tested because the direct path from infancy/preschool maternal depression to SEDR-MC was not significant.