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Maternal caregiving and girls' depressive symptoms and antisocial behavior trajectories: An examination among high-risk youth

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Abstract

Past research has identified parental depression and family-of-origin maltreatment as precursors to adolescent depression and antisocial behavior. Caregiving experiences have also been identified as a factor that may ameliorate or accentuate adolescent psychopathology trajectories. Using the unique attributes of two geographically diverse, yet complementary longitudinal research designs, the present study examined the role of maternal caregiver involvement as a factor that promotes resilience-based trajectories related to depressive symptom and antisocial behaviors among adolescent girls. The first sample comprises a group of US-based adolescent girls in foster care (*n* = 100; mean age = 11.50 years), all of whom have had a history of childhood maltreatment and removal from the home of their biological parent(s). The second sample comprises a group of UK-based adolescent girls at high familial risk for depression (*n* = 145; mean age = 11.70 years), with all girls having a biological mother who has experienced recurrent depression. Study analyses examined the role of maternal caregiving on girls' trajectories of depression and antisocial behavior, while controlling for levels of co-occurring psychopathology at each time point across the study period. Results suggest increasing trajectories of depressive symptoms, controlling for antisocial behavior, for girls at familial risk for depression, but decreasing trajectories for girls in foster care. A similar pattern of results was noted for antisocial behavior trajectories, controlling for depressive symptoms. Maternal caregiver involvement was differentially related to intercept and slope parameters in both samples. Results are discussed with respect to the identification of

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family level promotive factors aimed at reducing negative developmental trajectories among high-risk youth.

Keywords

Maternal caregiving; depression; antisocial behavior; foster care; trajectories

Depression and antisocial behavior problems among youth constitute an area of significant clinical, social, and economic concern (Greenberg et al., 2003; Welsh, Schmidt, McKinnon, Chattha, & Meyers, 2008). Recent estimates suggest that depression will become the second leading medical cause of disability in the world by 2020 (World Health Organization, 2001), and that the prevalence rate is rising among young people (Collishaw, Maughan, Goodman, & Pickles, 2004). There is also evidence highlighting increasing rates of antisocial behavior problems among children and adolescents internationally (Ford, 2008). Depression and antisocial behavior often co-occur (Angold, Costello, & Erkanli, 1999), yet most research in the field of developmental psychopathology continues to focus on single problem behaviors with considerably less attention given simultaneously to multiple problem domains. Further, when co-occurring symptoms are considered, research and clinical efforts are typically focused on samples of boys (e.g., Capaldi, 1992; Drabick, Beauchaine, Gadow, Carlson, & Bromet, 2006; Loeber, Farrington, Stouthamer-Loeber, & Van Kammen, 1998), with few studies focused on the co-occurrence of antisocial behavior and depressive symptoms in girls.

Identification of the pathways and processes through which depressive symptoms and antisocial behaviors develop and are maintained, with consideration of both problem behaviors in a single model, can provide information about etiological pathways while simultaneously informing researchers as to modifiable targets for the development of intervention programs aimed at remediating problematic outcomes among at risk youth. The primary goal of this study is to examine trajectories of depressive symptoms and antisocial behavior in two samples of at-risk adolescent girls: a US sample of girls who experienced childhood maltreatment and subsequent placement in foster care, and a UK sample of girls with mothers who have experienced recurrent depression. Using a multiple problem framework, antisocial behavior problems are considered when predicting depressive symptom trajectories and depressive symptoms are considered when predicting antisocial behavior trajectories. Given the high-risk nature of the populations in this study, of particular relevance to the two samples is the role of maternal caregiver involvement as a potential resilience-promoting factor in reducing trajectories of psychopathology across the 2-year study period.

Links between Antisocial Behavior and Depressive Symptoms

The etiology, prevalence rates, and long-term outcomes for depressed and antisocial youth are well illustrated in the extant literature (Angold et al., 1999; Boylan, Vaillancourt, Boyle, & Szatmari, 2007; Lahey, Loeber, Burke, Rathouz, & McBurnett, 2002; Wiesner, 2003). Youth with co-occurring indices of psychopathology experience a range of poorer outcomes over time compared to youth with single, phenotype specific problems (e.g., depression or

antisocial behavior), including suicidality, substance use, and related health problems (Fite, Colder, Lochman, & Wells, 2008). For example, Angold, Costello, and Erkanli (1999) found that after controlling for other comorbidities, conduct disorder was about seven times more common in depressed than in non-depressed adolescents (Angold, Costello, & Erkanli, 1999), with recent evidence suggesting that this odds ratio reduced from 7 to 2.4 when controlling for oppositional defiant behavior (Copeland, Shanahan, Erkanli, Costello & Angold, 2013), thereby converging with the present study's focus on broad antisocial behaviors. Similarly, Kovacs, Paulauskas, Gatsonis, and Richards (1988) estimated that approximately one-third of youth with a major depression diagnosis also met criteria for an externalizing diagnosis.

Several theoretical models have been proposed to explain the co-occurrence of depression and antisocial behavior problems among adolescent youth. Patterson and Capaldi (1990) propose a failure model, whereby antisocial behavior problems lead to depression due to the negative consequences that behavioral problems have for youth development, including academic failure, peer rejection, and increased family conflict. Antisocial behavior problems may interfere with the ability to develop competency skills, resulting in negative reactions and rejection from peers (e.g., Capaldi & Stoolmiller, 1999). Such children may also evoke hostile and rejecting parenting (Reid, Patterson, & Loeber, 1982), leading to decreased feelings of self-worth and self-competence. This combination of low self-competence and negative reactions from others may cause pervasive failures in adjustment (e.g., academic failure, inability to build social support networks, and relationship failures), making a child vulnerable to depressive symptoms (Biederman, Faraone, Mick, & Lelon, 1995; Capaldi, 1991, 1992; Patterson & Stoolmiller, 1991). Recent research suggests support for the failure model as a primary pathway to co-occurring problems; for example, a longitudinal study using offspring of women in the National Longitudinal Study of Youth found that, of children who developed depressive symptoms, all had moderate or high levels of pre-existing oppositional symptoms (Boylan, Vaillancourt, & Szatmari, 2012).

A less common but nonethess important pathway to co-occurring problems is from depressive symptoms to antisocial behavior problems. Depressive symptoms may lead depressed youth to seek associations with deviant peers, possibly as a means of attaining social acceptance. Children showing depressive symptoms may find that their choice of friends is limited – depressive symptoms have been linked to ongoing problems in social relationships (Capaldi & Stoolmiller, 1999; Rudolph et al., 1997) – and may be more easily accepted by deviant peers. Alternatively, depressed youth may "act out" underlying depressive symptoms by externalizing their feelings and dysregulated mood in a manner more consonant with antisocial behavior. This model proposes that depression can also precede antisocial behavior (Capaldi, 1992; Ritakallio et al., 2008). The purpose of this report is not to disentangle the directionality of effects, but rather, given the extant literature on the co-occurrence of antisocial behavior and depression, to examine trajectories of each problem behavior while controlling for concurrent levels of the other.

A Focus on Girls

Despite evidence of a high prevalence rate of co-occurring antisocial behavior and depression among youth, there is a paucity of research on multiple problem behaviors among adolescent girls, with most longitudinal studies focusing on the co-occurrence of depression and antisocial behavior problems among boys (Angold et al., 1999). However, females have significantly higher lifetime prevalence rates of depression than males, with 21% of women meeting criteria for lifetime depression vs. 13% of males (Kessler et al., 1994). The origin of sex differences in depression can be traced to adolescence, at which time an elevated increase in depressive symptoms has been shown in girls as compared to boys (Angold & Costello, 2001; Ge, Lorenz, Conger, Elder, & Simons, 1994; Hankin et al., 1998), and where rates of depression among girls are higher than rates for boys (Thapar, Collishaw, Pine, & Thapar, 2012). This adolescent-onset sex difference has been shown across ethnic groups and sampling criteria (Grant & Compas, 1995; Hyde, Mezulis, & Abramson, 2008).

Although depression is a widely recognized public health concern in its own right, outcomes for adolescent girls with depressive symptoms are often further compounded by co-occurring delinquency problems. Official arrest record data show a 50% increase in girls' juvenile arrests, with girls now accounting for 30% of all juvenile arrests (Puzzanchera & Adams, 2011). Numerous studies have shown that depressive symptoms are more highly associated with delinquency-based behaviors in girls than in boys (e.g., Costello, Mustillo, Erkanli, Keeler, & Angold, 2003; Gallerani, Garber, & Martin, 2010; Roberts, Roberts, & Xing, 2007; Silberg, Rutter, D'Onofrio, & Eaves, 2003). For example, Fazel, Doll, and Långström (2008) found that 6-month prevalence of major depressive disorder among adolescent girls in the juvenile justice system was 29%, more than twice the rate of their male counterparts and four to five times the rate of the general population of girls. Similarly, Fagan and Western (2003) analyzed longitudinal data on Australian adolescents and found that delinquent behaviors increased the probability of depression only for female participants (not for males), and Wiesner and Kim (2006) reported that girls were more likely to exhibit comorbid depressive symptoms and delinquent behaviors than were boys (49.5% vs. 25.3%, respectively). One explanation for the increased rates of depression in girls with antisocial behavior problems is the "gender paradox", which suggests that the sex with the lower prevalence of a disorder has a higher likelihood of developing co-occurring problems originating from the low prevalence disorder (Loeber & Keenan, 1994; Loeber & Stouthamer-Loeber, 1998; Zoccolillo, 1992). Given the relative dearth of research on co-occurring depressive symptom and antisocial behavior problems among girls, as well as the deleterious outcomes for girls with co-occurring problems, we focus specifically on developmental trajectories in two samples of girls at high risk for problems due to factors potentially related to their prior (US) and present (UK) rearing/caregiving environments.

Associations between Caregiver Characteristics and Adolescent Psychopathology

Past research has identified that adolescent psychopathology may be explained by: (1) adverse rearing environments that promote psychopathological trajectories among offspring, (2) genetic factors passed on from biological parents to offspring, and (3) a combination of the two (gene-environment interplay; see Rutter, 2006). In this manuscript, we focus specifically on the rearing environment, while acknowledging that some of the associations identified between the rearing environment and youth psychopathology may result from genetic factors passed on from biological parents. In one of the samples examined in this manuscript, children resided with their biological mother; in the other sample, they were living in foster care and had been removed from the biological parent home. In each sample, the caregiving environment has been (and may continue to be) disrupted or is at higher-risk of being affected; in one sample, due to prior maltreatment of the child in the biological home, and in the second sample, due to recurrent depression in the biological mother.

The maternal caregiving environment has been identified as a consistent correlate of negative developmental outcomes for youth, both in relation to depression and antisocial behavior (Davies & Windle, 1997; McCarty & McMahon, 2003). Recent studies using genetically sensitive research designs where rearing parents and children are not genetically related have facilitated examination of associations between aspects of the rearing environment (e.g., maternal caregiving quality) and child psychological outcomes that are unconfounded by common genetic factors (known as passive gene-environment correlation, see Jaffee & Price, 2007; Harold et al., 2011). Results from these studies suggest two primary extensions from past research in this area. First, children at risk due to parental psychopathology may experience heterogeneous outcomes; studies suggest that children at risk for depression due to maternal depression, for example, may experience elevated symptoms of depression and/or antisocial behavior problems, rather than phenotype specific transmission (e.g., depression to depression; Silberg, Maes, & Eaves, 2010). Second, maternal caregiving may be a more consistent mediator of adverse outcomes for children in the case of antisocial behavior problems than depression (Harold, et al., 2011; Sellers et al., 2013).

The role of maternal caregiving in understanding risk and resilience mechanisms in relation to multiple adolescent problem behaviors was identified in a pioneering study by Ge, Best, Conger, & Simons (1996), who examined the associations among parental warmth, hostility, and disciplinary skills observed over 3 years across 4 groups of adolescents: (1) those with depressive symptoms, (2) those with conduct problems, (3) those with elevated conduct problems and depressive symptoms, and (4) those with neither depressive or conduct problems. Results suggested a differential pattern of association relative to the index of parenting considered, with parental hostility and harsh disciplinary practices more consistently associated with adolescent conduct problems than depressive symptoms. However, parental warmth and responsive parenting practices reduced the co-occurrence and long-term development of depressive symptoms and conduct problems in offspring (Ge et al., 1996). This work marks an important departure in the developmental history of

examining parenting effects on child outcomes, by partitioning specific parenting behaviors (e.g., warmth versus hostility) in examining long-term associations with youth internalizing and externalizing problems.

Building on this pattern of findings, this study used a measure of maternal caregiving that may be particularly salient when youth have previously lived with a caregiver who either suffered from clinical depression, or who maltreated the child: maternal caregiver involvement. Both childhood maltreatment and exposure to maternal depression have been identified as family-based risk factors for the development of psychopathology in girls (Leve & Chamberlain, 2007; Teicher & Samson, 2013; Trickett, Negriff, Ji, & Peckins, 2011). For example, Ryan and Testa (2005) found that, of the 10- to 16-year-olds in the Illinois child welfare system between 1995 and 2000, more than 50% had at least one report of delinquency, a 47% greater likelihood than their non–foster care peers. Studies using diagnostic interviews (e.g., the Casey Field Office Mental Health Study) have also indicated that youth in foster care tend to show high lifetime prevalence rates for disruptive disorders such as conduct disorder and oppositional defiant disorder, ranging from 21% to 48% (White, O'Brien, White, Pecora, & Phillips, 2008). Similarly, maternal depression has been identified as a risk factor for offspring depression and antisocial behavior problems (Lieb, Isensee, Hofler, Pfister, & Wittchen, 2002; Wickramaratne & Weissman, 1998).

Given the documented impacts of maltreatment and maternal depression on the development of offspring psychopathology, we utilize a resiliency framework to examine the potentially ameliorating (promotive) role of caregiver involvement on reducing trajectories of adolescent psychopathology (Cicchetti, 2013). As noted by Rutter (2000), understanding resilience in children and adolescents exposed to adversity is of considerable importance in guiding public policy aimed at the prevention of psychopathology. Learning about the protective mechanisms that promote resilience in the face of adversity is central to the prevention of psychopathology (Cicchetti, 2013; Masten, 2001; Rutter, 2000, 2007). Originating from investigations of poverty and response to trauma, resiliency research is thus highly germane to understanding outcomes for maltreated youth, who have previously experienced adversities (Cicchetti & Garmezy, 1993), and to understanding outcomes for youth living with a maternal caregiver who has suffered from recurrent depression. Masten's (2001) review of converging findings on resiliency highlighted that resilience occurs through ordinary processes involving the operation of basic human adaptational systems, even in the face of adversity. These adaptational systems can include family-level characteristics, such as close relationships with involved and caring adults. Through adaptational systems such as involved caregiving, interventions could enhance child resilience by directly adding sufficient positive experiences to the child's life to offset the adversity (Cicchetti, 2013; Garmezy, Masten, & Tellegen, 1984; Masten, 2001).

The Current Study

This study employs a unique sampling strategy where adolescent girls are at elevated risk for psychopathology due to: (1) having a history of maltreatment, or (2) having a parent with a history of recurrent depression. The first sample comprises a group of US-based adolescent girls in foster care, all of whom have had a history of childhood maltreatment and

removal from the home of their biological parent(s). Their current foster caregiving environment may facilitate decreases in adolescent psychopathology, relative to their biological parent environment. The second sample comprises a group of UK-based adolescent girls at high familial risk for depression, with all girls having a biological mother who has experienced recurrent depression. Thus, their current caregiving environment may continue to be risk-perpetuating. Uniquely, this two sample approach allows examination of depression and antisocial behavior trajectories among diverse samples of adolescent girls who all have exposure to a recognized risk (maltreatment or maternal depression) that is associated with both youth depression and youth antisocial behavior. The association between current maternal/carer caregiving quality can be examined in the two samples, where the UK sample of girls are fully genetically related to and living with their rearing mothers, and the US sample of girls are not living with their biological mother.

This study examined the role of responsive maternal caregiving behavior (caregiver involvement in the youth's life) on girls' trajectories of depressive and antisocial behavior symptoms, while controlling for levels of co-occurring psychopathology at each time point across the study period, using two three-wave longitudinal research designs. Both studies employ samples of similar aged adolescent girls (mean age = 11–12 years old at the start of the study), comparable measures of psychopathology (depressive symptoms, antisocial behavior problems), and maternal/carer caregiving practices (caregiver involvement). We hypothesized that: (1) antisocial behavior problems would be concurrently associated with depressive symptoms when examining trajectories of depressive symptoms, and depressive symptoms would be concurrently associated with antisocial behavior problems when examining trajectories of antisocial behavior; (2) the foster care sample would show declines in psychopathology over time due to their placement in an improved caregiving environment relative to their biological home, whereas the offspring of mothers with recurrent depression would show normative age-related increases in depression and antisocial behavior over time; and (3) maternal caregiver involvement would reduce depressive symptom and antisocial behavior problems (initial levels and trajectories) in both samples.

Methods

Participants and Procedures

Study 1 – Middle School Success (MSS)—The Study 1 sample was comprised of 100 girls living in foster care in the US. Originally, 145 girls who met the two study criteria (living in relative or nonrelative foster care in one of two counties containing major metropolitan areas in the Pacific Northwest, and in their final year of elementary school) were referred to the study by the child welfare system. Of these 145 girls, 27 girls refused to participate (either the girl, her caregiver, or her caseworker did not agree to the girls' participation), and an additional 18 girls were excluded because their eligibility status changed by the time they were contacted by the study staff for recruitment (e.g., moved out of the state, were pending reunification or adoption, or were in an incorrect grade level). Caseworkers and the foster caregivers provided informed consent for the remaining 100 girls, and the girls provided assent prior to participation. Both girls and caregivers were

compensated for participating. All procedures for the study were approved by the institution's Institutional Review Board.

The mean age of the girls was 11.54 years (*SD* = 0.48) at baseline. Sixty-three percent of girls were European-American, followed by 14% multiracial, 10% Latino, 4% African-American, and 4% Native American. According to child welfare records, 56% had at least one incident of physical abuse, 67% had at least one incident of sexual abuse, and 78% had at least one incident of neglect. Approximately 32% of girls experienced all three types of maltreatment. Sixty-eight percent of the girls were in nonrelative foster homes and 32% were in relative foster homes at baseline.

The girls and their caregivers completed a baseline (T1) assessment and follow-up assessments at 12 months (T2) and 24 months (T3) post-baseline. The retention rates were consistently high across the study period, ranging from 92% to 98%. The assessments included a structured in-person interview and questionnaires for each girl and her caregiver, an interview with the girl's caseworkers, and the collection of child welfare records. Assessments lasted approximately two hours and were conducted by trained interviewers. Participants were part of a longitudinal intervention trial in which girls were randomly assigned either to a behavioral support intervention condition (*n* = 48) or to a regular foster care control condition (*n* = 52; Chamberlain, Leve, & Smith, 2006). The intervention included parenting groups and girl groups, each focused on preventing the onset of behavior problems and health-risking behavior during the transition to middle school. Although an examination of intervention effects was not a primary focus of this study, intervention condition was included as a control variable in the analyses. Assessment staff members were blind to the intervention status of the girls.

Study 2 – Early Prediction of Adolescent Depression (EPAD)—The Study 2 sample was comprised of 145 girls who were the daughters of mothers with recurrent depression. The original sample included 337 parents who had a history of recurrent unipolar depression and their offspring (age 9–17 years). Participants were recruited predominantly from general practices across South Wales (78%), while the remainder of the sample was recruited through community volunteers (19%) and a variety of other resources (3%). A detailed description of the sample has been published previously (Mars et al., 2012; Sellers et al., 2013).

For this analyses we focused on female offspring (*n* = 197). One family was omitted due to a diagnosis of bipolar disorder in the affected parent between the first and second assessment. Families of 13 depressed fathers were omitted from the analyses. A further 38 families were excluded based on offspring age (age 17 years or above at any wave), in order to be comparable to the Study 1 sample age range. The remaining 145 families were eligible for inclusion in the study.

The mean age of the girls was *M* = 11.70 years (*SD* = 1.63) at baseline. Girls and their mothers completed a baseline assessment (T1) and follow-up assessments at approximately 15 months (T2: *M* = 13.00 years, *SD* = 1.57) and 27 months (T3: *M* = 13.95 years, *SD* = 1.51). The retention rate across the study period was high (> 90%). The assessments

included a structured in-person interview and questionnaires for each girl and her mother. Assessments lasted approximately two hours and were conducted by trained interviewers.

Measures

Youth depressive symptoms—In Study 1, youth-reported depressive symptoms were measured at T1, T2, and T3 using the Center for Epidemiological Studies-Depression scale (CESD; Radloff, 1977). The CESD is a 20-item self-report measure of depressive symptomatology with a typical clinical cutoff score of 16 or higher (Radloff, 1977). In this study, the percent of girls at or above the clinical threshold for depressive symptoms was 30% (T1), 27% (T2), and 20% (T3). Internal consistency was acceptable (α = .71–.78). In Study 2, youth-reported depressive symptoms were measured at T1, T2, and T3 using the child version of the Child and Adolescent Psychiatric Assessment (CAPA; Angold & Costello, 2000), which is a semi-structured interview based on DSM-IV symptoms. Youth completed the depression symptoms section about their symptoms over the last 3 months. A total depression severity score was derived using symptom totals from DSM-IV criteria (T1: *M* = 1.09, *SD* = 1.54; T2: *M* = 1.10, *SD* = 1.57; and T3: *M* = 1.27, *SD* = 1.85).

Youth antisocial behavior—In Study 1, youth-reported antisocial behavior was measured at T1, T2, and T3 using 23 items reflecting general delinquency that were developed using the diagnostic criteria for disruptive behavior disorder. Girls were asked to rate how many times they had committed various disruptive, antisocial, and delinquent acts. Sample items included ‘threatened to hit other kids’ and ‘skipped classes without an excuse’. The scale showed good internal reliability (α = .82–.84). Items were recoded as 0 (*never*) and 1 (*at least one time*) and then summed within wave to reflect the total level of antisocial behavior at that wave; T1: *M* = 10.02, *SD* = 4.20; T2: *M* = 8.88, *SD* = 4.34; and T3: *M* = 8.10, *SD* = 4.43. In Study 2, youth-reported antisocial behavior was measured at T1, T2, and T3 using the child version of the CAPA. Antisocial behavior scores were derived using symptom totals from DSM-IV criteria from the disruptive behavior scale (oppositional defiant disorder and conduct disorder); T1: *M* = 1.59, *SD* = 1.67; T2: *M* = 1.56, *SD* = 1.94; and T3: *M* = 1.49, *SD* = 1.94.

Caregiver Involvement—Both studies included a measure of the level of caregiver involvement in the girl’s life, measured at T1. In Study 1, youth reported on how much time they spent with their caregiver talking and doing things they enjoy (e.g., sports, hobbies or games) in a variety of settings (weekdays, weekends), in terms of actual minutes and hours. The total number of minutes doing activities and talking together was summed across four items. Higher scores indicated greater caregiver involvement. The scale showed acceptable reliability (α = .70). In Study 2, mothers completed a 12-item self-report questionnaire assessing maternal involvement and warmth towards her daughter. (e.g., active interest; interested in what child does; enjoy having child around; pay a lot attention; like to spend time with child). Each item was scored on a 5-point Likert-type scale, ranging from, ‘Almost never true’, ‘Rarely true’, ‘Sometimes true’, ‘Almost always true’, and ‘Always’. A total score was created (*M* = 44.14, *SD* = 5.13), with high scores indicating more time/interest in the youth’s life. Internal reliability was excellent (α = .94).

Covariates—Two covariates were included in the analytical models for Study 1, due to the specificities of the foster care sample: intervention condition (0 = control group, 1 = intervention group), and age at first placement. Age at first placement was coded from official child welfare records. Girls were first placed in foster care at 7.63 years (*SD* = 3.14) on average, and had spent approximately 2.90 years (*SD* = 2.25) in foster care prior to study entry.

Analytical Approach

The distribution of child depression and antisocial behavior symptoms was positively skewed in both studies. Therefore, maximum likelihood estimators (MLR) with standard errors that are robust to non-normality (which incorporates full information maximum likelihood [FIML]) were used (Muthén & Muthén, 1998–2012). In addition, in Study 1, square root transformations were used to transform the depression and antisocial behavior scores prior to conducting latent growth curve modeling.

Two models were examined to test the study hypotheses: the first set of analyses considered trajectories of depressive symptoms as the outcome, and the second set of analyses considered antisocial behavior trajectories as the outcome. In the first model, we ran a linear latent growth curve model (LGM) to examine developmental trajectories of depressive symptoms over time with antisocial behaviors as time-varying covariates. Girls' depressive symptom scores at T1, T2, and T3 were used to estimate two latent growth factors (intercept and slope) of depressive symptoms over time. Antisocial behavior at each of the corresponding time points was included as a time-varying covariate to take into account its proximal concurrent influence on depressive symptoms. The intercept factor loadings were all fixed at 1 and the slope factor loadings were fixed at 0, 1, and 2 (Study 1) and 0, 1.3, and 2.3 (Study 2) for T1, T2, and T3, respectively, to reflect the amount of time between assessments. In the event that a linear model did not fit the data well, a spline model was tested. In the spline model, loadings were fixed at 0 and 1 for T1 and T3, respectively and the middle slope factor loading for T2 was freely estimated.

After the basic LGM was examined, a second LGM that included T1 caregiver involvement as a predictor of depressive symptom intercept and slope factors was tested. Caregiver involvement was centered and included in the model as a time-invariant covariate. Study 1 also included the two covariates specific to that sample in the models (intervention condition and age of first foster care placement).

In our second set of analytical models, we examined antisocial behavior trajectories from T1 to T3 while including depressive symptoms as a time-varying covariate, and T1 caregiver involvement as a predictor. The inclusion of time-varying covariates allows for the unique contribution of these variables on the outcome variable to be estimated while taking into account co-occurring symptoms. Analyses were conducted using M*plus* version 7.1 (Muthén & Muthén, 1998–2012).

Due to the longitudinal nature of the study designs, and the presence of a modest amount of missing data, we examined whether the data were missing at random using Little's Missing Completely at Random (MCAR) test in SPSS. The MCAR test was significant for both

studies: Study 1, (χ^2 (17) = 28.84, p = .04), and Study 2, (χ^2 (70) = 94.97, p = .03), indicating that the data may not be missing completely at random. In both studies, we then compared the data for participants with and without missing data. In Study 1, girls who had complete data (n = 91) were not significantly different from girls with missing data (n = 9) on any of the study variables included in this analyses with the exception of T1 antisocial behavior: girls with missing data reported significantly higher levels of antisocial behavior at T1 compared to girls with complete data (t(98) = −2.55, p = .012). In Study 2, girls who had complete data (n = 102) did not differ significantly from girls with missing data (n = 43) on any of the study variables included in this analyses. We used the FIML approach in M*plus* to accommodate missing data across both studies, to provide unbiased estimates of model coefficients.

Multiple indices were used to provide a comprehensive assessment of model fit, including chi-square values (χ^2), comparative fit index (CFI), Tucker Lewis Index (TLI), and root mean square of approximation (RMSEA). Goodness of fit was determined in accordance with Hu & Bentler (1999): CFI and TLI > 0.90, and RMSEA < 0.08.

Results

Descriptive Results

Tables 1 and 2 show the means, standard deviations, and correlations among study variables for Study 1 and Study 2, respectively. As seen in the tables, there were significant associations over time within behavior: depressive symptoms at T1, T2, and T3 were intercorrelated in both studies (with the exception of T1 and T3 depressive symptoms for Study 2), and antisocial behavior symptoms at T1, T2, and T3 were intercorrelated in both studies. In addition, depressive symptoms and antisocial behavior symptoms were correlated with each other both within and across time, with the exception of T1 depressive symptoms with T3 antisocial behavior symptoms for both studies. In addition, there was evidence that caregiver involvement was associated with both symptom sets. In Study 1, caregiver involvement was significantly, and inversely, associated with T1 depressive symptoms (r = −.26, p < .01), and in Study 2 caregiver involvement was significantly, and inversely, associated with T1 and T2 antisocial behavior symptoms (r = −.28, p < .001; r = −.26, p = .004), respectively. These bivariate associations suggested partial support for the study hypotheses, which were then tested using a series of LGM analyses.

Depressive Symptom Trajectories with Antisocial Behaviors as Time-Varying Covariates

Study 1—The analyses first tested an LGM where the intercept factor loadings were all fixed at 1 and the slope factor loadings were fixed at 0, 1, and 2 for T1, T2, and T3, respectively. However, the model fit the data quite poorly (χ^2(10) = 20.61, p = .02, CFI = .79, TLI = .75, RMSEA = .10). In order to accommodate potential nonlinearity for some individuals in the sample, a spline model was fitted. As was the case with the linear model, all intercept factor loadings were fixed at 1. The slope loadings were fixed at 0 and 1 for T1 and T3, respectively and the middle slope factor loading for T2 was freely estimated. The spline model showed a significantly better fit (χ^2(9) = 9.12, p = .43, CFI = 1.00, TLI = 1.00, RMSEA = .01); (nested χ^2 (1) = 11.49, p < .001) and was used in the remaining analyses.

The means of the intercept and slope factor, the average initial levels at T1 and change rates across all individuals in the sample, were .64 ($p < .01$) and .00 (ns), respectively. This suggests that only the initial level of depressive symptoms were significantly different from zero. The nonsignificant slope factor mean suggests that on average, there was no significant change in girls' depressive symptoms over time. The intercept and slope had variances of .14 ($p < .01$) and .10 ($p < .01$), respectively. The significant intercept and slope factor variance indicate that there is substantial individual variability in the initial level (T1) as well as in the change rates of depressive symptoms over time. In addition, girls' antisocial behavior at each time point was significantly and positively associated with their depressive symptoms (.44, $p < .01$), suggesting proximal influence of girls' antisocial behavior on their depressive symptoms that is above and beyond the trajectory processes. The covariance between the intercept and slope factor (−.10, $p < .01$) and covariances among time-varying covariates were also significant (.03, .03, and .04 at T1, T2, and T3, respectively, all $ps < .01$).

We then tested the prediction model by adding the intervention status, age at first placement, and caregiver involvement as time-invariant predictors to the model described above, to examine the extent to which these factors were related to the intercept and slope factor of girls' depressive symptom trajectories (Table 3). Again, the model fit the data well ($\chi^2(21) = 13.04$, $p = .91$, CFI = 1.00, TLI = 1.13, RMSEA = .00). The means of the intercept and slope factor in the prediction model were .66 ($p < .01$) and .05 (ns), respectively, suggesting that the mean of the slope factor remained nonsignificant in the prediction model. The intercept (.12, $p < .01$) and slope (.08, $p < .01$) factor variances were significant, indicating significant individual variances both in the initial level and in the change rates of girls' depressive symptoms even in the presence of the time-invariant predictors. The covariance between the intercept and slope factor (−.08, $p < .01$), the time-varying effects of antisocial behaviors (.44, $p < .01$), and covariances among time-varying covariates (.03, .03, and .04 at T1, T2, and T3, all $ps < .01$) remained significant in the prediction model.

Results also indicated that caregiver involvement was negatively associated with the initial level (−.01, $p = .01$) and positively associated with the slope factor (.02, $p < .01$). This suggests that girls who spent more time talking and doing activities with caregivers were more likely to have lower initial levels of depressive symptoms. However, they tended to show greater increases in depressive symptoms over time, likely a statistical artifact given the significant inverse association between intercept and slope factors in the model. Predictors in the model explained approximately 12% of the variance in the intercept factor ($p = .047$) and 21% of the variance in the slope factor ($p = .034$).

Study 2—The analyses first tested an LGM where the intercept factor loadings were all fixed at 1 and the slope factor loadings were fixed at 0, 1.3, and 2.3 (average increase in age in years across waves) for T1, T2, and T3, respectively. A linear growth model fit the data well ($\chi^2(11) = 9.74$, $p = .55$, CFI = 1.00, TLI = 1.02, RMSEA = .00).

The means of the intercept and slope factor, the average initial levels at T1 and change rates across all individuals in the sample, were .48 ($p < .01$) and .09 ($p = .28$), respectively, suggesting that only the initial level of depressive symptoms were significantly different

from zero. The nonsignificant slope factor mean again suggests that on average, there was no significant change in girls' depressive symptoms over time. The intercept and slope had variances of .64 (p = .07) and .37 (p = .02), respectively, indicating that there is substantial individual variability in the rates of change in depressive symptoms over time. In addition, girls' antisocial behavior at each time point was significantly and positively associated with their depressive symptoms (.36, p < .01), consonant with results presented for Study 1. The covariance between the intercept and slope factor (−.23, p = .11) and covariances among time-varying covariates were also significant (1.60, .95, and 2.05 at T1, T2, and T3, respectively, all *ps* < .01).

We then tested the prediction model by adding caregiver involvement as a time-invariant predictor to the model described above to examine the extent to which this factor was related to the intercept and slope factor of girls' depressive symptom trajectories (Table 4). Again, the model fit the data well with ($\chi^2(12) = 10.32$, p = .58, CFI = 1.00, TLI = 1.02, RMSEA = .00). The means of the intercept and slope factor in the prediction model were .48 (p < .01) and .08 (ns), respectively, suggesting that the mean of the slope factor remained nonsignificant in the prediction model. The slope (.31, p = .02) factor variance remained significant, indicating significant individual variance in the change rates of girls' depressive symptoms even in the presence of maternal involvement. There was no significant association for the initial status (.64, p = .07). The covariance between the intercept and slope factor (−.28, p < .05), the time-varying effects of antisocial behaviors (.35, p < .01), and covariances among time-varying covariates (1.60, .97, and 2.06 at T1, T2, and T3, all *ps* < .01) also remained significant in the prediction model. Results also indicated that caregiver involvement was not associated with the initial level (−.01, p = .79) or with the slope factor (.01, p = .68).

Antisocial Behavior Trajectories with Depressive Symptoms as Time-Varying Covariates

Study 1—The reverse model was tested by using antisocial behavior at each time point to estimate two latent growth factors (intercept and slope) of antisocial behavior trajectories over time and by including girls' depressive symptom scores at each time point as time-varying covariates. The intercept factor loadings were all fixed at 1 and the slope factor loadings were fixed at 0, 1, and 2 for T1, T2, and T3, respectively. The resulting model fit the data reasonably well ($\chi^2(11) = 17.72$, p = .09, CFI = .94, TLI = .93, RMSEA = .080).

The means of the intercept and slope factor were .82 (p < .01) and −.05 (p < .01), respectively, indicating that both were significantly different from zero. The negative slope factor mean suggests that on average, there were significant decreases in girls' antisocial behaviors over time. The intercept and slope had variances of 0.03 (p < .01) and .01 (ns), respectively. These variances represent the individual variability in the initial level and slope. The significant intercept factor variance and nonsignificant slope factor variance indicate that there is substantial individual variability in the initial level (T1) only. In addition, girls' depressive symptoms at each time point were significantly and positively associated with their antisocial behaviors (.14, p < .01), suggesting proximal influence of depressive symptoms on antisocial behaviors above and beyond the trajectory processes.

While the covariance between the intercept and slope factor was nonsignificant, covariances among time-varying covariates were significant (.05, $p < .01$).

We then added the intervention status, age at first placement, and caregiver involvement as time-invariant predictors to the model to examine the extent to which these factors were related to the intercept and slope factor of girls' antisocial behavior trajectories (Table 3). However, the model did not fit the data well ($\chi^2(23) = 37.60$, $p = .03$, CFI= .85, TLI = .87, RMSEA = .08). The means of the intercept and slope factor in the prediction model were .77 ($p < .01$) and −.04 (ns) respectively. The mean of the slope factor was no longer significant once the time-invariant predictors were included. The intercept factor variance remained significant (.03, $p < .01$), indicating that significant individual variances in the initial level of girls' antisocial behavior in the presence of the time-invariant predictors. The slope factor variance (.01, ns) and the covariance between the intercept and slope factor (.00, ns) remained nonsignificant in the prediction model. Furthermore, the time-varying effects of depressive symptoms (.14, $p < .01$) and covariances among time-varying covariates (.05, $p < .01$) also remained significant in the prediction model. Results also indicated that none of the predictors were significantly related to the growth factors of girls' antisocial behavior trajectories.

Study 2—The analyses first tested an LGM where the intercept factor loadings were all fixed at 1 and the slope factor loadings were fixed at 0, 1.3, and 2.3 for T1, T2, and T3, respectively. A linear growth model was an excellent fit to the data: ($\chi^2(11) = 12.99$, $p = .29$, CFI = 0.98, TLI = .97, RMSEA = .04).

The means of the intercept and slope factor, the average initial levels at T1 and change rates across all individuals in the sample, were 1.15 ($p < .01$) and −.07 ($p = .31$), respectively, suggesting that only the initial level of antisocial behavior symptoms were significantly different from zero. The nonsignificant slope factor mean suggests that on average, there was no significant change in girls' antisocial behavior over time. The intercept and slope had variances of 1.49 ($p < .01$) and .39 ($p < .01$), respectively. The significant intercept and slope factor variance indicates that there is substantial individual variability in the initial level (T1), as well as change rates in antisocial behavior symptoms over time. In addition, girls' depressive symptoms at each time point were significantly and positively associated with antisocial behavior (.43, $p < .01$), suggesting proximal influence of girls' depressive symptoms on antisocial behavior that is above and beyond the trajectory processes, replicating results across all models tested. The covariance between the intercept and slope factor (−.38, $p = .01$) was significant. Only the covariances among time-varying covariates at T3 were significant (.75, .40, and 1.61 at T1, T2, and T3, respectively, $ps < .01$).

We then tested the prediction model by adding caregiver involvement as a time-invariant predictor to the model described above to examine the extent to which this construct was related to the intercept and slope factor of girls' depressive symptom trajectories (Table 4). Again, the model fit the data well ($\chi^2(12) = 14.07$, $p = .30$, CFI = 0.98, TLI = 0.97, RMSEA = .03). The means of the intercept and slope factor in the prediction model were 1.15 ($p < .01$) and −.07 ($p = .35$), respectively, suggesting that the mean of the slope factor remained nonsignificant in the prediction model. The intercept (1.59, $p < .01$) and slope (.38, $p < .01$)

factor variance remained significant, indicating significant individual variance in the change rates of girls' depressive symptoms even in the presence of maternal involvement. The covariance between the intercept and slope factor (−.36, $p < .01$), the time-varying effects of depression symptoms (.42, $p < .01$), and covariances among time-varying covariates (.78, .43 and 1.61 at T1, T2, and T3, $ps < .01$) remained significant in the prediction model. Results also indicated that caregiver involvement was associated with the initial level (−.07, $p = .02$), but not with the slope factor (.01, $p = .58$) of antisocial behavior.

Discussion

This study employed two geographically diverse, yet complementary longitudinal samples to examine the role of maternal caregiver involvement on adolescent girls' depressive symptoms and antisocial behavior trajectories, while controlling for co-occurring symptom levels across each respective study period (3 time points). Both samples were comprised of adolescent girls at differential risk for psychopathology due to aspects of their caregivers and caregiving environments. The first sample was comprised of US adolescent girls in foster care, all of whom were prior victims of child maltreatment and who were not living with their biological parents at the start of the study as a direct result of this prior maltreatment. The second sample included UK adolescent girls at elevated risk for depression, all of whom had mothers who had experienced an episode of recurrent depression and who were living with their mother at the start of the study.

Our first hypothesis, that antisocial behavior problems would be concurrently associated with depressive symptoms when predicting trajectories of depressive symptoms, and depressive symptoms would be concurrently associated with antisocial behavior problems when predicting trajectories of antisocial behavior, was supported in both samples. There were significant associations between depressive symptoms and antisocial behavior at each time point, regardless of whether depressive symptom trajectories or antisocial behavior trajectories were modeled. These significant pathways provide additional support for the presence of co-occurring antisocial behavior and depressive symptoms problems among early adolescents (Capaldi & Stoolmiller, 1999; Essex et al., 2006; Ingoldsby, Kohl, McMahon, & Lengua, 2006; Mezulis, Vander Stoep, Stone, & McCauley, 2011); and builds the evidence base on co-occurring problems specifically among high-risk girls. Most prior studies used community samples, varied in terms of their measures and symptom versus cutoff value approach to categorize participants, and included either boys only, or both boys and girls. In comparison, our samples were comprised of high risk girls, who tended to show higher rates of psychopathology than prior community-based samples. In our samples, 20–30% of the sample showed clinical levels of either problem behavior at any given time point. By comparison, among sixth-grade boys, approximately 18% of the sample had elevated conduct problems, 15% had elevated depressive symptoms, and 11% were elevated on both domains of psychopathology (Capaldi & Stoolmiller, 1999). Among 5th grade youth, 14% had conduct problems, 12% had depressive symptoms, and 14% showed co-occurring problems (Essex et al., 2006). Our findings uniquely add to the existing literature in this area by showing significant associations between antisocial behavior and depressive symptoms among girls during the important developmental period of early adolescence (when rates of depression increase among girls, but not boys), while respectively controlling

for prior levels of antisocial behavior and depression as modeled in the trajectory analysis. Despite differences in the nature of the two samples (e.g., different index of familial risk; different countries), similar magnitudes of association were identified, suggesting the robustness of this pattern of associations.

Our second hypothesis, that the foster care sample would show declines in problem behavior over time (because the children have been removed from a maltreating environment and placed in a more nurturing foster care environment), whereas the offspring of mothers with recurrent depression would show increases in problem behavior over time (because they continue to reside with the affected biological mother, and because genetic influences on risk for depression become more pronounced across development (Rice, Harold & Thapar, 2002), was only partially supported. Examination of the mean levels of depressive symptoms and antisocial behavior *did* evidence decreases in both dimensions of psychopathology over time for the girls in foster care, whereas the offspring of depressed mothers evidenced increases in depressive symptoms over time. However, when the full models that considered the presence of co-occurring behavior problems (and of caregiver involvement) were examined, none of the models evidenced significant increases or decreases in psychopathology over time. This distinction is important to note; prior studies that have examined the trajectories of depression or antisocial behavior over time but have *not* considered the co-occurring influences of multiple problem behaviors may inadvertently misrepresent developmental increases or decreases in problem behaviors. Multiple forms of psychopathology may work together to magnify or reduce developmental trends in a single domain of psychopathology. For example, in addition to evidence for the positive association between depression and antisocial behavior across adolescence (Kofler et al., 2011), when youth exhibit quite high levels of clinical depression, to the extent that they do not have the motivation to leave the house, there is a natural reduction in the expression of antisocial behavior and delinquency. However, in both of the current samples, the associations between depressive symptoms and antisocial behavior were positive, perhaps due to the at-risk nature of our samples. The nature of the association between depressive symptoms and antisocial behavior has implications for our etiological models, as well as for the targeting of interventions that may intend to reduce psychopathology in one domain, but have unintended consequences on a related domain of psychopathology.

Fergusson, Lynskey, and Horwood (1996) build on the hypothesis that symptoms of psychopathology among youth likely co-occur by suggesting that common explanatory factors also underlie association with co-occurring outcomes. For example, while depression and antisocial behavior problems likely co-occur in affected youth, both might share a common underlying influence (e.g., shared genetic or shared environmental influences). While studies support the conclusion that there may be genetic overlap between co-occurring indices of psychopathology (e.g., antisocial behavior and depression; see Rowe, Rijsdijk, Maughan, Eley, & Hosang, 2008), it is also recognized that heritable characteristics only partially account for intergenerational transmission of risk, and that non-inherited factors have an important role (Harold et al., 2011; Kerr et al., 2013; Silberg, Maes, & Eaves, 2010; Tully, Iacono, & McGue, 2008).

Uniquely, the complement of study designs employed in this study offers a further substantive attribute that advances past research in this area: examination of maternal caregiving influences on adolescent girls' depressive and antisocial behavior trajectories among girls living with their biological mother (UK sample), and among girls *not* living with their biological mother (US foster care sample). Our hypothesis examined whether maternal caregiver involvement would serve as a factor to promote resilience among girls previously exposed to adverse caregiving qualities (maltreatment or maternal depression). Noted associations between caregiver involvement in the foster care sample cannot be consistently explained by common genetic factors underlying biological mothers' parenting behavior and adolescent symptoms of psychopathology, a confound of past research (see Moffitt, 2005) that has received significant recent research attention (see Harold et al., 2011; 2013). (Note, however, that there could still be genetic contributions to the associations between girls' psychopathology and caregiver involvement in the foster sample for those girls who were placed in relative foster care (e.g., a biological aunt, uncle, or grandparent), but this was only present for approximately one-third of the sample, and the degree of genetic relatedness between the female caregiver and girl was still often zero because the female caregiver was not the biological relative). In contrast, residence with the biological mother was a criterion for study inclusion in the offspring of depressed mothers' sample, and therefore, associations between maternal caregiver and child could be attributed to shared generic factors.

Results examining our third hypothesis suggest a differential pattern of psychopathology-based trajectories for the two groups of adolescent children. Caregiver involvement played an important, yet very distinct role in the prediction of psychopathology in the two samples of girls. For the US foster care sample, caregiver involvement was a significant predictor of both the intercept and slope of depressive symptoms, but it did not predict either the intercept or slope of antisocial behavior. Conversely, in the UK sample, caregiver involvement predicted the intercept for antisocial behavior, but not depression, in the UK sample. In other words, the specific beneficial effects of caregiver involvement on girls' outcomes depended on the nature of risk to which the girls had been exposed. For girls who experienced childhood maltreatment, higher levels of caregiver involvement were associated with lower initial levels of depressive symptoms. In comparison, girls who had a mother with recurrent depression showed reduced antisocial behavior when their mothers were highly involved.

These differences could reflect the principles of multifinality (Cicchetti & Rogosch, 1996)] —a single protective factor may lead to multiple outcomes, depending on different risks the youth has experienced. In the case of the maltreated girls, the vast majority of whom experienced neglect as their primary maltreatment type (78%), having a caregiver who spends time with you could serve to lower depressive symptoms, and serves in contrast to the experiences in the home of origin. Importantly, this decrease comes at a time in development when girls begin to show normative increases in depression (Angold & Costello, 2001), and therefore, it is additionally meaningful from a clinical standpoint that these girls showed decreased depressive symptoms over time. In contrast, high caregiver involvement may have minimal effects on antisocial behavior, however, because firm and

consistent discipline has been identified as the key caregiver protective factor for delinquency among youth in foster care (Eddy & Chamberlain, 2000).

In the case of the offspring of depressed mothers, having a mother who is prone to depression who spends considerable time with her daughter may not protect against the daughter's own depressive symptoms, and may in fact exacerbate it; or that other types of factors beyond maternal involvement are needed to overcome a higher genetic or familial liability for depression (the mothers had recurrent depression, rather than antisocial behavior). However, caregiver involvement may instead protect against antisocial behavior when the sample is not at high risk for antisocial behavior, due to the ameliorative role of spending time with one's parent, rather than with deviant peers.

A similar pattern of findings has been identified for parental antisocial behavior (and specifically, father antisocial behavior). In an epidemiological sample of over a thousand children and their parents, Jaffee, Moffitt, Caspi, & Taylor (2003) found that the less time fathers lived with their children, the more conduct problems their children had, but only when the fathers engaged in low levels of antisocial behavior. In contrast, when fathers engaged in high levels of antisocial behavior, the more time they lived with their children, the more conduct problems their children had. This suggests that distance from an antisocial parent may be beneficial to the prevention of child antisocial behavior, while proximity may be detrimental. Similarly, having a depressed parent who is highly involved in the child's life may be detrimental to the development of child depressive symptoms. As noted by the increasing levels of girls' depression and the lack of protective effect for caregiver involvement on girls' depression in the UK sample, the compounding negative influence of caregiver involvement on adolescent depression could be the dual influence of both genetic and environmental exposure to risk for depression, or simply not enough to overcome familial liability to mood problems. By contrast, none of the girls in the foster care study were living with their biological mother at the first assessment.

Results derived from this study are consistent with findings from recent studies suggesting that not only might associations between specific indices of parent and child psychopathology (e.g., parent antisocial behavior predicting child antisocial behavior) be heterogeneous, such that a specific index of psychopathology in a parent may differentially predict one or more indices of psychopathology in offspring (e.g., conduct problems and/or depression, cf. Kerr et al., 2013), but that identified environmental mediators of this association may also be differentially linked to specific indices of psychopathology. Recent studies employing novel research designs that allow disaggregation of genetic (G) and environmental (E) factors underlying associations between parent and child psychopathology find differential results for the relative role of G versus E. For example, Silberg, Maes, and Eaves (2010) using an extended children of twins design, noted the role of parenting as a mediator in the case of parent antisocial behavior and both child conduct problems and depression using the children of twins design. Harold et al. (2011) echo this pattern of results using a sample of differentially related parents and children conceived through *in vitro* fertilization (IVF). Findings from this study suggest that genetically unconfounded associations between parent and child antisocial behavior are mediated by hostile parenting, while parenting does not mediate association between parent and child

depression. Findings from the present study add to this pattern of effects, such that the particular measure of parenting behavior employed was associated with intercept variance for antisocial, but not depressive symptoms.

Limitations and Recommendations for Future Research

Although this study offers several noteworthy advantages in examining the developmental trajectories of adolescent girls' depressive symptom and antisocial behavior trajectories relative to maternal caregiving among at-risk girls, several limitations also merit mention. First, caution should be employed in relation to employing direct comparisons regarding differences in the pattern of findings presented relative to risk group (maltreatment vs. depression), as differences may also be explained by differential measurement of theoretical constructs employed across each study (e.g., the index of maternal caregiving), as well as possible sample differences in relation to depressive symptoms and parenting/caregiver behaviors across internationally diverse samples (US and UK). However, these attributes may also be seen as relative study strengths. Second, in terms of direct comparisons between biologically related mother-child and biologically unrelated carer-child groupings and model associations, comparisons relative to the possible confounding role of passive *r*GE may not be consistently applied because approximately one-third of the girls in foster care were residing with a relative at the start of the study. In addition, some children were reunified with the biological parent(s) during the course of the study. Thus, we could not make stronger conclusions about the relative isolation of environmental components of caregivers versus genetically-transmitted aspects of behavior. However, none of the girls in foster care were living with their biological mother, thereby providing preliminary evidence applicable in addressing this confound of past research in this area (see Harold et al., 2011).

Third, the present study covers a relatively limited period of repeat assessment across the period of adolescence represented by each respective study (mean age = 11.50 and 11.70 years at T1 for Study 1 and Study 2, respectively). The trajectories of depressive symptoms and antisocial behavior may vary at earlier or later stages of adolescence, as may the role of caregiving, particularly as children adjust to school transitions, post-pubertal changes, and stronger peer influences. Replication and extension of the proposed theoretical model to additional ages and stages of adolescent development would therefore be informative. Fourth, the present study focuses exclusively on mothers and girls, yet emerging evidence has increasingly highlighted the importance of the father-child (and particularly the father-son and father-daughter relationships) in accounting for the transmission of parent to child psychopathology (Harold et al., 2013). Extending the present study objectives to also include fathers and sons would also significantly advance knowledge relative to the primary study hypotheses. Notwithstanding these limitations, the present study adds to the literature on the familial underpinnings of adolescent depression among at risk girls, while also considering the relative role of covarying antisocial symptoms, and vice versa, across two geographically diverse and high-risk samples representing two distinct domains of risk influence (maltreatment, maternal depression).

In terms of future directions, and given estimates of the trajectory of depression as an index of global disability (Murray & Lopez, 1996), it is incumbent on researchers to explore

mechanisms that underlie susceptibility, risk, and expression of depressive symptoms and depressive disorders across the lifespan. While evidence supports the conclusion that maltreatment affects brain development (Cicchetti, 2013), what about the role of maternal depression? This is an area of underexplored examination in the field of developmental psychopathology, yet examination of the neurobiological architecture that might underlie associations between exposure to environmental adversity marked by maternal (and paternal) depression and symptoms of child depression and antisocial behavior represents an important area of future research. Finally, study findings support the further study of prevention and intervention initiatives that target multiple domains of the family environment in ameliorating adolescent depressive symptoms and antisocial behavior. Specifically facets of the parenting/caregiving environment might be pursued as possible promotive factors among high-risk youth in the context of adolescent family-based caregiving experiences.

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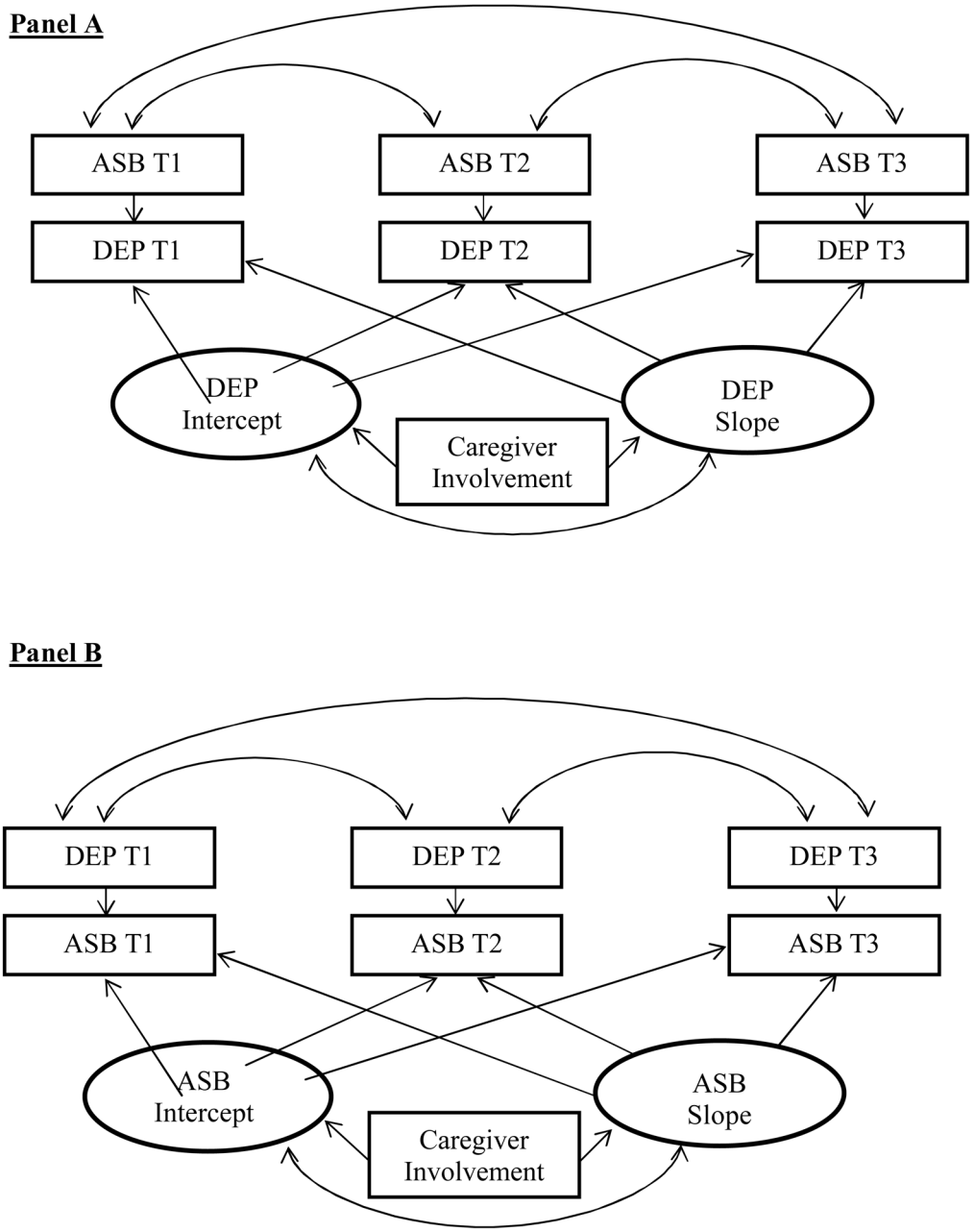

Figure 1.

Theoretical model (Panel A, depressive symptoms outcome adjusting for co-occurring antisocial behavior; Panel B, antisocial behavior outcome adjusting for co-occurring depressive symptoms).

Table 1

Means, Standard Deviations, and Correlations for Study 1

	1	2	3	4	5	6	7	8	9
1. Intervention condition									
2. Age at 1st placement	−.10								
3. T1 Caregiver involvement	.15	−.21*							
4. T1 Depression	−.08	.26*	−.26**						
5. T2 Depression	−.14	−.07	.06	.31**					
6. T3 Depression	−.02	.02	.09	.28**	.28**				
7. T1 Antisocial behavior	.01	.01	.03	.25*	.32**	.28**			
8. T2 Antisocial behavior	.02	−.01	−.01	.21*	.38***	.34**	.50***		
9. T3 Antisocial behavior	−.00	−.01	.09	.07	.27**	.43***	.42***	.68***	
Mean	n/a	7.65	8.01	12.85	12.51	11.65	10.02	8.88	8.10
SD	n/a	3.13	7.43	8.89	8.59	8.57	4.20	4.34	4.43

* $p < .05$,

** $p < .01$,

*** $p < .001$.

Table 2

Means, Standard Deviations, and Correlations for Study 2

	1	2	3	4	5	6	7
1. T1 Caregiver involvement							
2. T1 Depression	−.16						
3. T2 Depression	−.08	.34**					
4. T3 Depression	−.07	.14	.57**				
5. T1 Antisocial behavior	−.28**	.48**	.37**	.28**			
6. T2 Antisocial behavior	−.26**	.27**	.49**	.44**	.51**		
7. T3 Antisocial behavior	−.14	.10	.44**	.50**	.30**	.56**	
Mean	44.13	1.09	1.10	1.27	1.59	1.56	1.49
SD	5.13	1.54	1.57	1.85	1.67	1.93	1.94

** < .01.

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Table 3

Final models for Study 1 latent growth curve model predicting depressive symptom trajectories and antisocial behavior symptom trajectories

	Depressive Symptoms			Antisocial Behavior		
	Estimates	**SE**	***p***	**Estimates**	**SE**	***p***
Time varying covariates[a]	.440	.087	.000	.140	.037	.000
Effects on the intercept						
Intervention condition	−.056	.070	.427	.014	.046	.761
Age at 1st placement	.021	.011	.058	.001	.007	.892
Caregiver involvement	−.012	.005	.014	.003	.003	.351
Effects on the slope						
Intervention condition	−.002	.073	.982	−.002	.026	.936
Age at 1st placement	−.021	.012	.072	.000	.004	.985
Caregiver involvement	.015	.005	.005	.000	.002	.849
Factor means						
Intercept	.656	.170	.000	.767	.106	.000
Slope	.045	.160	.780	−.041	.059	.489
Factor variances						
Intercept	.118	.017	.000	.027	.010	.006
Slope	.077	.022	.001	.006	.005	.266

[a]Time varying covariates were constrained to be equal across all three time points

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Table 4

Final Models for Study 2 Latent Growth Curve Model Predicting Depressive Symptom Trajectories and Antisocial Behavior Symptom Trajectories

	Depressive Symptoms			Antisocial Behavior		
	Estimates	**SE**	***p***	**Estimates**	**SE**	***p***
Time varying covariates[a]	.354	.051	.000	.421	.076	.000
Effects on the intercept						
Caregiver involvement	−.006	.023	.787	−.073	.031	.019
Effects on the slope						
Caregiver involvement	.007	.016	.675	.011	.019	.580
Factor means						
Intercept0	.483	.120	.000	1.151	.152	.000
Slope	.080	.073	.272	−.063	.068	.353
Factor variances						
Intercept	.643	.356	.071	1.585	.275	.000
Slope	.308	.131	.018	.379	.106	.000

[a]Time varying covariates were constrained to be equal across all three time points

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