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Intergenerational Continuity in Depression: The Importance of Time-Varying Effects, Maternal Co-morbid Health Risk Behaviors and Child's Gender

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Abstract

Intergenerational continuity in depressive symptoms is well established between mother and child, but there are still important facets of this relationship that are underexplored. We examine intergenerational continuity in depressive symptoms between mother–child dyads as a flexible function of child age and account for the potential moderating role of maternal co-morbid health risk behaviors. Using prospective, self-report data collected yearly from 413 mother–child dyads (210 mother–son dyads and 203 mother–daughter dyads) between child ages 12–17, the results indicate that the effect of maternal depressive symptoms on daughters' depressive symptoms steadily increases throughout adolescence whereas the effect of maternal depressive symptoms on sons' depressive symptoms is relatively small, stable, and non-significant during mid-adolescence before increasing in effect in later adolescence. A positive interactive effect between maternal depressive symptoms and intimate partner violence is observed for sons and maternal depressive symptoms and substance use for daughters. A negative interactive effect of maternal depressive symptoms and substance use is observed among sons. Overall, this study identifies particular

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Data Sharing Declaration The data for this study are not currently available to the public, but provisions to deposit the Rochester Intergenerational Study data at the Inter-University Consortium for Political and Social Research are currently underway.

Compliance with Ethical Standards

Conflict of Interest The authors declare that they have no conflict of interest.

Ethical Approval All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards. Additionally, all research conducted was in compliance with the research procedures set forth by the University at Albany.

Informed Consent Informed consent was obtained from all participants in this research and was approved by the Institutional Review Board at the University at Albany.

subgroups for whom intervention programming is most beneficial and suggests targeting health risk behaviors of mothers to lessen the impact of maternal depressive symptoms on offspring.

Keywords

Depression; Intergenerational continuity; Substance use; Intimate partner violence; Gender

Introduction

Adolescents who suffer from depressive symptoms are at a heightened risk for a host of negative consequences including poor academic performance, peer difficulties, involvement in antisocial and problem behavior, adverse mental and physical health outcomes, and suicidal behavior (Naicker et al. 2013; Ritakallio et al. 2008). As children move through childhood and adolescence, the likelihood of experiencing depressive symptoms and, ultimately a clinical diagnosis of depression, increases dramatically. In 2015, over three million adolescents aged 12–17 experienced at least one depressive episode in the past year, representing 19.5% of females aged 12–17 and 5.8% of males aged 12–17 (National Institute of Mental Health 2017). Moreover, research indicates that 20% of adolescents reported a lifetime prevalence of depressive symptoms (Bansal et al. 2009). Clearly, depressive symptoms affect a nontrivial percentage of the adolescent population and serve as a major public health concern.

Although there is no single cause of depressive symptoms among children and adolescents, one of the most salient factors is the presence of parental depressive symptoms. Moreover, women are more likely to suffer from depressive symptoms than men (National Institute of Mental Health 2017), and there is evidence that continuity in depressive symptoms is stronger between mother and child compared to father and child (Pilowsky et al. 2014). Not only do children of mothers with depressive symptoms display a heightened risk for developing depressive symptoms themselves (e.g., Goodman et al. 2011; Mikkonen et al. 2016), but among these children, the course of depressive symptoms tends to be more severe compared to the children of mothers with no depressive symptoms (Lieb et al. 2002).

Despite strong evidence of intergenerational continuity in depressive symptoms between mother and child, there are still three facets of this continuity that we know comparatively little about. The first facet is timing of continuity, and, more specifically, the degree to which maternal manifestations of depressive symptoms at different points in the child's life course are related to manifestations of depressive symptoms among offspring. Most often, the lifetime prevalence of maternal depressive symptoms is considered as a predictor of offspring depressive symptoms at a particular developmental period, for example, during childhood or adolescence (e.g., Hammen et al. 2008). Other research uses maternal depressive symptoms at one point in time to predict concurrent offspring depressive symptoms or offspring depressive symptoms later in the life course (e.g., five or ten years later; see Hammen et al. 2012). Yet to be conducted is a systematic assessment of the degree to which a mother's depressive symptoms are associated with her child's depressive symptoms continuously across time. This investigation is particularly important given that

maternal depressive symptoms may wax and wane over time; thus, a one-time snapshot of maternal symptoms or a history of any depressive symptoms may not be sufficient to underscore the true nature of continuity in depressive symptoms across time. Moreover, there is considerable heterogeneity in the severity and type of depressive symptoms over the life course and during adolescence, in particular (Thapar et al. 2012). As such, the factors that underlie this continuity may vary in degree during this time. Therefore, it is worthwhile to directly examine the extent to which intergenerational continuity in depressive symptoms is stable or variable over time, and whether there are sensitive periods during adolescence in which offspring may be particularly vulnerable or resilient to the harmful effects of maternal depressive symptoms (Goodman and Gotlib 1999).

The role of mothers' co-morbid health risk behaviors is the second facet of intergenerational continuity in depressive symptoms that requires more investigation. Maternal health risk behaviors—including substance use, intimate partner violence, and involvement in crime—often co-occur with mothers' depressive symptoms, and each have been independently linked to maternal as well as child well-being (Folger et al. 2017; Whitaker et al. 2006). Little work has examined whether intergenerational continuity in depressive symptoms remains when accounting for these co-morbid health risk behaviors or whether these co-morbid health risk behaviors (or any factors) may exacerbate the harmful effect of a mother's depressive symptoms on her child's depressive symptoms (Goodman et al. 2011). To understand why the effect of maternal depressive symptoms on offspring depressive symptoms may be exacerbated when these symptoms co-occur with health risk behaviors, we draw upon the logic of the "Launch and Grow" model proposed by Garber and Cole (2010) and the Interpersonal Stress Model proposed by Hammen et al. (2004), which argue that the primary mechanisms that link maternal depressive symptoms to a child's depressive symptoms are the ensuing cascade of family stress and maladaptive family functioning. Since both maternal depressive symptoms and co-morbid health risk behaviors are known to promote family stress and poor family functioning, it is possible that the effects of maternal depressive symptoms on family functioning and child depressive symptoms may be intensified when a mother also abuses substances, is involved in intimate partner violence, and/or participates in criminal activities. Importantly, this causal pathway is speculative, and the role of moderators of intergenerational continuity of depressive symptoms, themselves, are largely unknown (Goodman et al. 2011). Therefore, we begin to contribute to this inquiry by focusing on the role of maternal co-morbid health risk behaviors as factors that can potentially intensify the harmful effect of maternal depressive symptoms on offspring's depressive symptoms

The final facet in need of greater examination is a focus on the role of child's gender in intergenerational continuity. Gender differences in depressive symptoms manifest in early adolescence, with girls demonstrating a greater risk for depressive symptoms and clinical diagnoses of depression compared to boys beginning around the age of 13 (Salk et al. 2016). Moreover, evidence suggests that the progression and patterns of depressive symptoms vary across gender in terms of the severity and duration of symptoms in childhood and adolescence, in particular (e.g., Ge et al. 2001), largely as a result of differential vulnerabilities to risk factors for depressive symptoms such as family stress and negative family functioning across gender (i.e., girls appear to be more vulnerable to these risk

factors and at a younger age; e.g., Cyranowski et al. 2000). Thus, it is not surprising that maternal depressive symptoms tend to elevate the risk for depressive symptoms in daughters more so than for sons (Chen et al. 2009). Given that intergenerational continuity is likely a function of child gender, we further explore gender differences in intergenerational continuity, particularly as it pertains to the potential for differences in timing (our first identified understudied facet) and the role of maternal co-morbid behaviors (our second identified understudied facet).

We address these three understudied and likely interrelated facets of intergenerational continuity in depressive symptoms between mother and child using prospective longitudinal data from 413 mother–child dyads from the Rochester Intergenerational Study (RIGS). Specifically, we examine intergenerational continuity in depressive symptoms from age 12 to age 17 while taking into account the importance of maternal co-morbid health risk behaviors and child gender. In the end, our efforts are intended to identify mother–child dyads who are particularly vulnerable and in need of intervention programming.

Intergenerational Continuity in Depressive Symptoms

Prior research consistently demonstrates that children of mothers who display depressive symptoms and/or have incurred a clinical diagnosis of depression are at a greater risk for depressive symptoms and/or clinical diagnoses of depression (for reviews see Goodman 2007; National Research Council 2009). Recent estimates suggest that there is a three- to five-fold increase in the likelihood of developing depressive symptoms if one's mother has a history depressive symptoms (Gotlib and Colich 2014), and a meta-analysis by Goodman et al. (2011) found an overall positive, albeit, small effect of maternal depressive symptoms on an array of offspring internalizing problems, including depressive symptoms.

The mechanisms underlying the positive relationship between maternal depressive symptoms and offspring depressive symptoms are varied and complex, including heritability and genetic factors, neuroregulatory functioning, family functioning and behaviors, and adverse environments (see Beardslee et al. 2012; National Research Council 2009 for reviews). While biological factors may account for nearly 45% of the intergenerational transmission in depressive symptoms (Chen and Yu 2015; Lau and Eley 2008), the majority of intergenerational continuity in depressive symptoms is a result of adverse environments resulting from parental depressive symptoms (Flint and Kendler 2014). Maternal depressive symptoms are predictive of heightened interpersonal stress, which may impact parenting quality, and this sequela leads to increased adolescent stress and lower perceived social competence, both of which are associated with adolescent depressive symptoms (i.e., the Interpersonal Stress Model; Hammen et al. 2004). Similarly, Garber and Cole (2010) found that maternal depressive symptoms yielded more family stress and strained parent-child relationships, which, in turn, predicted depressive symptoms in adolescent children (i.e., the "Launch and Grow" Model).

In research examining intergenerational continuity in depressive symptoms between mothers and adolescent offspring, the general conclusion is that any maternal history of depressive symptoms (whether historical using retrospective reports or concurrent measurement) is associated with increased depressive symptoms in children. Moreover, intergenerational

continuity tends to be stronger among adolescent daughters compared to adolescent sons (e.g., Mason et al. 2017; for an exception see Mikkonen et al. 2016). Recent research examining the effects of maternal depressive symptoms measured at child age 11 found that maternal depressive symptoms were associated with concurrent depressive symptoms among both sons and daughters, but maternal depressive symptoms assessed at age 11 only predicted depressive symptoms among daughters in late adolescence (age 18; Mason et al. 2017). This latter finding highlights two important factors related to intergenerational continuity. First, continuity between mothers and sons does not mirror continuity between mothers and daughters. Second, it contradicts the premise that the timing of maternal depressive symptoms is irrelevant to continuity (see Hammen and Brennan 2003). Depressive symptoms are not consistent across the life course; they vary in timing and course, with more severe depressive symptoms emerging during life transitions and times of stress (Ge et al. 2001; Wickrama et al. 2008). As such, Mars et al. (2012) argue that it is more than just a history of parental/maternal depressive symptoms that matters when studying intergenerational continuity in depression; it is also the magnitude and recency of maternal depressive symptoms.

The timing of maternal depressive symptoms is also pertinent to the study of intergenerational continuity in depressive symptoms across child gender due to the varying etiology of depressive symptoms (i.e., the relevance of biological and environmental factors can wax and wane over time) and symptomology (i.e., severity and duration) during adolescence. Prospective, longitudinal studies demonstrate that depressive symptoms begin to increase around the age of 13 among girls but remain fairly constant among boys (Cole et al. 1999). Whalen et al. (2016) also found gender differences in the severity and course of depression from early childhood through adolescence and argued gender-specific sensitivities to adversities likely account for these differences in the prevalence and magnitude of depressive symptoms. This is in line with the Generic Elaborated Cognitive Vulnerability-Stress Model (Hankin and Abramson 2001), suggesting that the general pattern of depressive symptoms among girls varies from their boy counterparts because adolescent girls are more likely to incur independent negative life events (i.e., events that are outside the realm of family) at an earlier age than boys (Rudolph and Hammen 1999). Furthermore, negative life events are more strongly related to depressive symptoms in girls compared to boys because girls are more likely to use internal coping mechanisms such as rumination, which is associated with depressive symptoms (Nolen-Hoeksema et al. 1999), and encode these events in more detail (Seidlitz and Diener 1998), while boys are more likely to employ external coping behaviors such as aggression (see also the Gender Intensification Process; Hill and Lynch 1983). As a result, girls may be more vulnerable to depressive symptoms following negative life events, including maternal depressive symptoms and their sequelae, than boys during adolescence because of their coping style.

During adolescence, adolescents experience biological (e.g., puberty and associated hormonal changes which are linked to changes in mood and affect) and social changes (e.g., increasing independence; increasing pressure to conform to culturally-defined gender-based stereotypes), both known to increase vulnerability to depressive symptoms. In fact, adolescence is a particularly acute period of vulnerability, particularly for girls who tend to begin puberty at an earlier age and struggle with the requirements of a culturally-prescribed

feminine identity known for promoting internalizing behaviors (i.e., the Gender Intensification Hypothesis; Hill and Lynch 1983; see also the Vulnerability-stress Model; Cyranowski et al. 2000; Nolen-Hoeksema and Girgus 1994). Maternal depressive symptoms can intensify stress among adolescents, with negative interpersonal interactions (or a lack of any interactions at all) between mother and child, further enhancing the risk for depressive symptoms in offspring in line with the Launch and Grow model of Garber and Cole (2010; see also Hammen et al. 2004). Thus, it may be that the effect of maternal depressive symptoms on adolescent depressive symptoms varies over this period of the life course and across child gender as both maternal depressive symptoms and adolescent vulnerabilities vary.

Maternal Depressive Symptoms, Comorbid Health Risk Behaviors, and Intergenerational Continuity of Depressive Symptoms

Maternal depressive symptoms negatively affect familial environments (e.g., poor parenting quality and family stress), which, in turn, increase the likelihood of depressive symptoms in offspring (e.g., Garber and Cole 2010). However, other maternal health risk behaviors also related to depressive symptoms in offspring, often proposed to be related through the same mechanisms invoked by models of intergenerational continuity in depression (i.e., interpersonal stress and family functioning). This raises the question as to whether the co-occurrence of these health risk behaviors may exacerbate the negative effect of maternal depressive symptoms on child depressive symptoms.

Substance use—There is a clear association between substance use and depressive symptoms (e.g., Lai et al. 2015) with evidence of shared etiologies (e.g., genetic predisposition) and various causal pathways (e.g., Swendsen and Merikangas 2000). Though directionality is difficult to ascertain, some evidence suggests that depressive symptoms can lead to substance use, consistent with the self-medication hypothesis (Khantzian 1985). However, this evidence varies depending on substance type (e.g., cigarettes vs. marijuana; Wilkinson et al. 2016; Womack et al. 2016). Conversely, substance use may precede depressive symptoms, with studies demonstrating a greater risk for subsequent depressive symptoms among cannabis users (Lev-Ran et al. 2014) and individuals suffering from an alcohol use disorder (Boden and Fergusson 2011). Substance use itself may also interact with pre-existing vulnerabilities to mood disorders (Quello et al. 2005) or the positive association between substance use and depressive symptoms may be bidirectional (Pacek et al. 2013) and developmentally specific (Brière et al. 2014). Nonetheless, depressive symptoms and substance use often co-occur.

Parental substance use is related to offspring internalizing and externalizing problem behaviors, including depressive symptoms (Chassin et al. 1999). Substance use is also associated with reduced quality of parenting behaviors, such as erratic responses to child needs (Patterson et al. 1992), and it can create stressful environments where material resources and social support are deflected away from the child (Dishion et al. 1999; Fals-Stewart et al. 2004). As such, parental substance use in the form of excessive alcohol and/or drug use are linked to depressive symptoms in children via associated adverse environments (Anda et al. 2002). The question remains if intergenerational continuity in depressive

symptoms persists when accounting for parental substance use and if the relationship between parental substance use and offspring depressive symptoms endures when accounting for maternal depressive symptoms. Moreover, with the frequent co-occurrence of these behaviors, it is possible that the co-occurrence of these behaviors enhances the other's harmful effect on offspring depressive symptoms.

Intimate partner violence—Involvement in intimate partner violence increases a woman's risk for depressive symptoms (Devries et al. 2013), and it is associated with more severe depressive symptoms, particularly in women without a history of depressive episodes (Bonomi et al. 2006). Alternatively, women with depressive symptoms or have a history of depressive symptoms are at greater risk for partner conflict and intimate partner violence (Capaldi et al. 2012), including both perpetration and victimization (Keenan-Miller et al. 2007). Overall, the relationship between depressive symptoms and interpersonal violence appears to be bidirectional for women, indicating a troubling cycle that may perpetuate itself in the absence of intervention (Hammen 2009; Helweg–Larsen et al. 2008).

Children exposed to intimate partner violence are at an increased risk for depressive symptoms (McFarlane et al. 2003). More specifically, maternal interpersonal trauma, which includes intimate partner violence, is related to child socio-emotional functioning, which affects the likelihood of depressive symptoms (Folger et al. 2017). The relationship between maternal involvement in intimate partner violence and offspring depressive symptoms is also accounted for by a number of other factors related to poor family functioning including child perception of family cohesion (Owen et al. 2009).

Acknowledging the comorbidity of depressive symptoms and intimate partner violence, Bair-Merritt et al. (2015) found that the effect of intimate partner violence exposure on youth internalizing symptoms was partially accounted for by maternal depressive symptoms. Furthermore, evidence suggests that maternal depressive symptoms and intimate partner violence likely interact to affect offspring behaviors. Silverstein and Colleagues (2006) reported that the effect of maternal depression on child school and social functioning is compounded by child exposure to in-home violence. Moreover, Breslend et al. (2016) found that interparental conflict exacerbates parental depressive symptoms, leading to more internalizing problems in children. However, it is not known whether the combination of maternal intimate partner violence and maternal depressive symptoms interact and to further promote depressive symptoms, specifically, in offspring, especially adolescent children.

Criminal behavior—Numerous studies demonstrate a relationship between depressive symptoms and antisocial/criminal behavior (e.g., Douzenis et al. 2005; Ge et al. 1996). A history of conduct disorders may explain this link, with a conduct disorder diagnosis predicting subsequent illegal behavior in women (Bardone et al. 1996). Moreover, conduct disorders are also a known risk factor for later depressive symptoms (Stringaris et al. 2014). Although the relationship between depression and criminal behavior may be spurious, with evidence that less than 5% of crime is directly related to depressive symptoms and, among those with depression, only 15% of crimes were related to current symptoms (Peterson et al. 2014), it is possible that the combination of these two health risk behaviors may interact to negatively affect the next generation.

Knowledge of, or witnessing, parental involvement in criminal behavior serves as an acute stressor, affecting the well-being and emotional adjustment of a child (Dallaire and Wilson 2010). In fact, research suggests a link between parental criminality and prepubertal depressive symptoms (Zalsman et al. 2006; see also Jaffee et al. 2002). Further, maternal arrest and conviction have powerful impacts on adolescent offspring (Shlafer et al. 2012). Dallaire et al. (2015) found that both arrest and conviction increase the risk for internalizing and externalizing problem behaviors in offspring (see also Silberg et al. 2012). Similarly, children whose parents are involved in criminal behavior and witness their parent's arrest or incarceration exhibit greater levels of anxiety and depressive symptoms (Dallaire and Wilson 2010). Moreover, Murray and Farrington (2008) found that parent-child separation resulting from criminal participation predicts enduring internalizing problems in male children.

Few known studies have examined the role of parental criminal behavior within the context of intergenerational continuity in depression. Kerr et al. (2013) examined the effects of adoptive mother depressive symptoms and antisocial behaviors on early childhood internalizing behaviors while accounting for the biological mother's depression history and found that both adoptive mother depressive symptoms and antisocial behavior were a predictor of internalizing behaviors. On the other hand, Sellers et al. (2014) found that maternal depressive symptoms led to an increased risk of depressive symptoms in adolescent offspring via mothers' antisocial behavior (including criminal behavior). Therefore, we continue with this line of inquiry and query whether intergenerational continuity in depressive symptoms remains when accounting for maternal criminal behavior and examine whether maternal involvement in criminal behavior exacerbates the effect of maternal depressive symptoms on adolescent depressive symptoms.

Current Study

In this study, we examine the time-varying relationship between maternal depressive symptoms and child depressive symptoms from age 12 to 17. We focus on general depressive symptoms instead of clinical diagnoses of depression given that many depressive episodes fail to result in a clinical diagnosis and subclinical levels of depressive symptoms negatively affect individual functioning similar to depressive symptoms that meet diagnostic criteria (Gotlib et al. 1995). Moreover, we focus solely on intergenerational continuity during adolescence because we want to utilize self-report data of depressive symptoms by mothers and their offspring since maternal reports of child depressive symptoms may be colored by one's own depression experiences (Goodman 2007). Younger children are likely too young to reliably assess their own depressive symptoms (Goodman 2007). In assessing the time-varying relationship between maternal and child depressive symptoms, this study further advances the study of intergenerational continuity in depressive symptoms by (1) accounting for the independent and potentially conditioning effects of maternal co-morbid health risk behaviors that often co-occur with depressive symptoms and (2) examining how child gender moderates these relationships.

Although this inquiry is largely exploratory in nature, we draw upon various theoretical models (i.e., the Gender Intensification Hypothesis, Hill and Lynch 1983; the Generic Elaborated Cognitive Vulnerability-Stress Model, Hankin and Abramson 2001; the

Interpersonal Stress Model, Hammen et al. 2004; and the Launch and Grow Model, Garber and Cole 2010) and expect that the effect of maternal depressive symptoms on a child's depressive symptoms will strengthen as adolescence progresses, particularly for daughters who are more susceptible to maternal depressive symptoms (see Hankin and Abramson 2001; Hill and Lynch 1983). Moreover, we hypothesize that the effect of maternal depressive symptoms on a child's depressive symptoms will remain when accounting for maternal co-morbid health risk behaviors, but the effect of maternal depressive symptoms will be exacerbated by co-morbid health risk behaviors, particularly for female offspring who are more likely to internalize stressful environments.

Method

Sample

The sample for this analysis is drawn from the Rochester Intergenerational Study (RIGS), the intergenerational extension of the Rochester Youth Development Study (RYDS). Only a brief summary of these studies is provided since detailed information about the two longitudinal, companion studies are presented elsewhere (Henry et al. 2015). The RYDS data is originally comprised of a birth cohort of 1000 adolescents (25% of the population), representative of the 7th and 8th grade public school population in Rochester, New York in 1988. Adolescents at a high risk for antisocial behavior were overrepresented by oversampling males and adolescents who resided in high-crime areas of the city based on police arrest data for 1987. These adolescents averaged 13.6 years of age at the start of the study, 73% were male, 68% were black, 17% were Hispanic, and 15% were white. The sample also represents the full socioeconomic spectrum found in an urban population (Farnworth et al. 1994), but it over represents poor families as 33% of the head of households were unemployed at the start of the study and 40% were on welfare. These adolescents were followed through adulthood from 1988 to 2006, and completed three phases of semi-annual (1988–1992; Phase I), annual (1994–1996; Phase II) and biannual (2003–2006; Phase III) interviews.

Beginning in 1999, RIGS identified the oldest biological child of the RYDS participants and added new firstborns to the sample in each subsequent year. Both the RYDS participant and the other primary caregiver (if it is the mother) completed annual interviews since the inception of RIGS (continuing until the child turns 18/turned 18) and the child completed annual interviews once he/she turned eight. Data collection is ongoing, but currently, there are prospective longitudinal data on 529 parent child dyads. The current analysis uses data from 413 parent–child dyads (210 mother–son dyads and 203 mother–daughter dyads), which includes all dyads where the child was at least 12 years of age in 2016, the last year from which data is available for analysis. The children are relatively evenly split by sex (51% male and 49% female), and 66% of children are black, 9% are Hispanic, 14% are mixed race, and 11% are white. Almost all mothers (96%) reported living with their child during adolescence. This generates a sample of 1,196 mother–son dyad years and 1194 mother–daughter dyad years for analysis. All data collection procedures were approved by the State University of New York at Albany's Institutional Review Board.

Measures

Maternal depressive symptoms—Maternal depressive symptoms were assessed at each yearly interview using 19-items adapted from the Center for Epidemiological Studies-Depression scale (CES-D scale; Radloff 1977). This widely used, self-report measure of depressive symptoms was created for community samples and asks about the frequency of depressive symptoms in the past two weeks. The scale demonstrates acceptable reliability in all groups studied (Radloff 1991), and among this sample, the reliability of maternal measures of depressive symptoms across child ages 12–17 is .83–.94. Higher scores on this scale indicate more numerous and frequent depressive symptoms. A full list of all the items included in this measure, as well as other measures, are presented in the Appendix.

Child's depressive symptoms—Beginning at the age of 12, child's depressive symptoms were assessed using 13-items based on the CES-D scale (Radloff 1977), subsequently adapted for adolescents by removing items likely to be irrelevant and rewording items to make them more understandable and applicable to the present population. This scale has been shown to have acceptable reliability among junior high school and high school students (Radloff 1991), and among this sample, the reliability for child's depressive symptoms for each year from child age 12–17 is .86–.90.

Maternal substance use—In each year interview, mothers were asked if they drank alcohol since the date of the last interview. If the response was affirmative, a set of follow-up questions asked whether or not she had gotten drunk since the date of the last interview, and if so, how many times. Similarly, mothers were also asked if they had used any drugs, including marijuana or other non-prescribed drugs, since the date of the last interview. With an affirmative response, a follow-up question asked how many times she had used marijuana or another illegal drug, respectively, since the date of the last interview. Since our focus is on problematic substance use by mothers, we combined the number of times a mother self-reported being drunk and the number of times that a mother self-reported drug use to create a frequency measure of problem substance use. It should be noted that marijuana use was the only type of drug used in this sample of women. Given the extreme skew of the distribution, we added a constant of one and took the natural log. Additional analyses were conducted by separating the frequency of excessive alcohol use and the frequency of illegal drug use and are presented at the end of the results section.

Maternal intimate partner violence—If a mother reported that she was married, living with a significant other, or had been in a relationship with a significant other for at least one month, then she was asked a set of questions to assess the prevalence and frequency of intimate partner violence using the Conflict Tactics Scale (CTS; Straus 1979), a widely used scale to measure intimate partner violence with documented reliability and validity across samples (Straus 1990). Nine items in the CTS create a physical violence scale. These nine questions were asked in terms of both perpetration and victimization, ascertaining the prevalence of these events since the date of last interview. Because the frequency of physical violence is highly correlated with severity ($r = .57$, $p < .01$; see also Holtzworth-Munroe and Stuart 1994; Straus et al. 1996) and the most common pattern of intimate partner violence among women is mutual violence (Carney et al. 2007), our measure of maternal intimate

partner violence is based on the frequency of perpetrating behaviors and violent victimization. Additional analyses were also conducted using the frequency of intimate partner violence victimization and the frequency of intimate partner violence perpetration, respectively, as the unique measure of maternal intimate partner violence and the results are presented at the end of the Results section.

Notably, for each dyad-year of analysis, only 65% of both mother-son dyad-years and 64% of mother–daughter dyad-years had a mother with a significant other and eligible to answer questions from the CTS (Straus 1979). Therefore, we use the two part-predictor method outlined by Dziak and Henry (2017) to include all available dyad-years to estimate the independent and moderating effects of maternal intimate partner violence on child's depressive symptoms. In this method, both an indicator of whether the mother was in a relationship or not (based on eligibility to answer the CTS scale questions), as well as the frequency of intimate partner violence conditional on being in a relationship, are considered as simultaneous predictors of the dependent variable.

Maternal involvement in crime—In each year interview, mothers were asked whether or not they were involved in 14 different criminal behaviors since the last interview. These items are a subset of the delinquency index (Huizinga et al. 1991) appropriate for adult respondents. Given the overlap in items and skip patterns in the data that prevent the construction of a variety or frequency measure for the majority of mothers, we use a binary indicator of involvement in crime, which represents whether or not a mother responded affirmatively to involvement in any of the 14 criminal behaviors in the past year.

Control variables—We also include a set of control variables in all analyses to limit the potential that the effect of maternal depressive symptoms on child's depressive symptoms is spurious. We include a continuous measure of the mother's age at birth and whether or not the mother lives with her child. Additionally, we include an indicator of financial stress as reported by the mother annually. This is a count of the number of different financial hardships faced by the mother/mother's household in the past year including reduced food intake as a result of a lack of money/food stamps, falling behind in rent or house payments, and not having enough money to pay utility bills. Finally, we include a set of controls for the race/ethnicity (black, Hispanic, and mixed race with white serving as the reference group) of the child. Descriptive information for each variable included in the study is presented in Table 1.

Analytic Plan

To explore time-varying effects of maternal depressive symptoms on offspring depressive symptoms from offspring age 12 to 17, we estimated a series of time varying effect models. Time-varying effect models are flexible, semi-parametric models that estimate change in an outcome (e.g., child's depressive symptoms), and the relationship between predictors (e.g., mother's depressive symptoms) and an outcome, as a function of time (Tan et al. 2012; Vasilenko et al. 2014). An optimal, nonparametric age trend representing both change in the behavior over time and the relationship between predictors and outcome over time is utilized to produce an effect size curve based on the strength of the relationship between the

predictors and outcome at each point in development. As a result, the time-varying effect model affords us the opportunity to uncover nuanced changes in child's depressive symptoms over time, as well as a nuanced assessment of critical periods of time in which children are most vulnerable to a mother's depressive symptoms and co-morbid behaviors (Vasilenko et al. 2017). For instance, the age-varying effect of maternal depressive symptoms on child depressive symptoms is expressed as

$$CD_{ij} = \beta_0(t) + \beta_1(t) * MD_{ij} + \beta_2(C) + \varepsilon_{ij},$$

where CD_{ij} (child's depressive symptoms) and MD_{ij} (maternal depressive symptoms) are measured for mother–child dyad i at time t and C is a vector of time stable control variables. Unlike other common methods for assessing change over time (e.g., growth models), the time-varying effect model does not require an *a priori* constraint on the shape of the relationship between a time-varying predictor and an outcome. Rather, the time-varying effect model only assumes that there is a smooth change pattern, and utilizes splines (in this case a p-spline) to model change in a process, as well as the changing relationship between a predictor and an outcome over time. Therefore, the time-varying effect model provides the opportunity to allow the data to speak to the relationship investigated rather than placing a parametric constraint on the change processes. Although this method has been used to assess changes in various outcomes such as alcohol use, smoking urges and drug use using longitudinal panels (Vasilenko et al. 2014; Epstein et al. 2017; Shiyko et al. 2012), this approach has not yet been applied to the assessment of continuity in depressive symptoms between mother and child and, therefore, provides an opportunity to uniquely examine the interplay between these variables.

The analysis proceeded in three steps. First, we estimated the relationship between maternal depressive symptoms on offspring depressive symptoms from child ages 12 to 17 net of controls. Next, we included each of the three co-morbid health risk behaviors in the aforementioned model to determine if maternal depressive symptoms were uniquely associated with a child's depressive symptoms when accounting for these co-morbid health risk behaviors. Finally, we added an interaction between maternal depressive symptoms and each co-morbid health risk behavior to ascertain whether or not the presence of the health risk behavior exacerbated the effects of maternal depressive symptoms on adolescent depressive symptoms. Each model was estimated separately for mother–son dyads and mother-daughter dyads. All models were estimated in SAS v.9.3 using the TVEM SAS macro (Li et al. 2014). The results are presented in Figs. 1–8 as time-varying coefficients, the appropriate display of effects for a time-varying effect model (see Evans-Polce et al. 2015), with the y-axis set to similar scales for each covariate across gender.

Results

Figure 1 demonstrates the time-varying relationship between maternal depressive symptoms and offspring depressive symptoms. Among mother-son dyads, the relationship between maternal depressive symptoms is small, relatively stable in early adolescence before becoming non-significant in mid-adolescence (approximate age 15). Beginning at age 16,

the relationship between maternal depressive symptoms and sons' depressive symptoms is significant and appears to increase, with the largest effect observed at age 17. Among mother–daughter dyads, the relationship between maternal depressive symptoms and daughters' depressive symptoms steadily increases in strength as the daughter progresses through adolescence.

Figure 2 presents the time-varying effects of maternal depressive symptoms and co-morbid health risk behaviors among mother-son dyads. Importantly, all covariates are included in the same model. The relationship between maternal depressive symptoms and sons' depressive symptoms weakens during early adolescence and it is not significant during mid adolescence (approximate ages 14–15) when accounting for maternal co-morbid health risk behaviors. However, maternal depressive symptoms continue to be associated with depressive symptoms among sons net of co-morbid maternal behaviors in later adolescence. With respect to the relationship between maternal health risk behaviors and sons' depressive symptoms across adolescence, neither maternal substance use nor maternal involvement in crime are related to sons' depressive symptoms during adolescence when accounting for maternal depressive symptoms and the other health risk behaviors. Maternal involvement in intimate partner violence, on the other hand, is positively related to depressive symptoms among sons when controlling for maternal depressive symptoms and other health risk behaviors, but this relationship appears to be limited to early to mid-adolescence (approximate ages 13–14).

Figure 3 presents the same time-varying effects curves as Fig. 2 but for mother-daughter dyads. The positive, increasing relationship between maternal depressive symptoms and daughter depressive symptoms remains unchanged after controlling for each co-morbid health risk behavior. As with mother–son dyads, neither maternal substance use nor maternal involvement in crime are related to daughters' depressive symptoms across the investigated period of adolescence when accounting for maternal depressive symptoms and each of the other co-morbid health risk behaviors. Maternal involvement in intimate partner violence is negatively related to depressive symptoms in daughters in mid- to later-adolescence (approximate ages 15–17), although the magnitude of this effect is quite small.

Figure 4 presents the results of the models including the interactive effects of maternal depressive symptoms and co-morbid health risk behaviors on offspring depressive symptoms. Figure 4a–c present the results for mother-son dyads, and, contrary to hypotheses, Fig. 4a indicates that maternal substance use weakens the effect of maternal depressive symptoms on sons' depressive symptoms (approximate ages 13–16). Alternatively, there is a positive interactive effect between maternal depressive symptoms and maternal intimate partner violence on sons' depressive symptoms in mid-adolescence (approximate ages 15–16); however, this exacerbating effect is rather small in magnitude. There is also some evidence to suggest that maternal involvement in crime enhances the effect of maternal depressive symptoms on sons' depressive symptoms at age 16, but again this interactive effect is quite small in magnitude.

Figure 4e, f indicate that neither maternal involvement in crime nor maternal intimate partner violence affect the relationship between maternal depressive symptoms and

daughters' depressive symptoms, respectively. Maternal substance use, on the other hand, intensifies the effect of maternal depressive symptoms on daughters' depressive symptoms in later adolescence (approximate ages 15–17), but this exacerbating effect is small in magnitude.

Given that we found that the presence of maternal co-morbid health risk behaviors conditions the relationship between maternal depressive symptoms and offspring's depressive symptoms, we further probed these significant interactive effects to determine if the conditioning effects found vary across levels of maternal depressive symptoms assessed at one standard deviation below the mean, the mean, and one standard deviation above the mean (see Appendix). Among mother–son dyads, maternal substance use seems to weaken the effect of maternal depressive symptoms on sons' depressive symptoms similarly across each level of maternal depressive symptoms. Moreover, the positive, interactive effect between maternal depressive symptoms and intimate partner violence is also consistent across levels of maternal depressive symptoms. Among mother–daughter dyads, though, the positive synergistic effect of maternal substance use is stronger in magnitude at lower levels of maternal depressive symptoms (when measured at 1 standard deviation below the mean compared to the mean and 1 standard deviation above the mean).

Additional Analyses

Since two of our measures of maternal co-morbid health risk behaviors combined various types of behaviors (i.e., maternal substance use included excessive alcohol use and drug use and maternal intimate partner violence included violent victimization and perpetration), additional analyses were performed to assess whether the specific components of our substance use and intimate partner violence measures were driving the previous findings.

With respect to maternal substance use, we re-estimated the previous models but included both maternal excessive alcohol use and drug use in the model instead of the global measure of substance use. Notably, the main effects of maternal depressive symptoms, maternal intimate partner violence, and maternal involvement in crime remain unchanged; therefore, the time-varying effects figures for these covariates when including both maternal excessive alcohol use and drug use are not presented a second time. Figure 5 presents the results of the main effects of maternal excessive alcohol use and drug use when accounting for maternal depressive symptoms and the other co-morbid health risk behaviors. Similar to the results with the global measure of substance use, neither excessive alcohol use nor drug use by mothers is related to sons' depressive symptoms during the investigated period of adolescence. Among daughters, whereas maternal substance use was unrelated to depressive symptoms throughout adolescence, there is some evidence that maternal excessive alcohol use is negatively related to depressive symptoms among daughters in early adolescence only (age 12). Maternal drug use is unrelated to daughters' depressive symptoms.

Figure 6 displays the results examining the interactive effects between each measure of maternal substance use and maternal depressive symptoms. Among mother–son dyads, we see that both maternal excessive alcohol use and drug use weaken the effect of maternal depressive symptoms on sons' depressive symptoms during mid-adolescence, but the negative interactive effect of between maternal alcohol use and maternal depressive

symptoms is much weaker and for a shorter period of adolescence compared to the interactive effect of maternal drug use and maternal depressive symptoms. Recall that among mother–daughter dyads there was a positive, interactive effect between maternal substance use and maternal depressive symptoms during mid- to late-adolescence. However, the positive, interactive effect between maternal excessive alcohol use and maternal depressive symptoms is only significant around the age of 17 and the positive, interactive effect between maternal depressive symptoms and drug use is only significant around the age of 15. This suggests that it is likely the combination of these two forms of substance use that exacerbates the effect of maternal depressive symptoms on daughters' depressive symptoms.

Unlike the two components of maternal substance use investigated, it was not possible to include both intimate partner violence victimization and intimate partner violence perpetration in the same model due to issues of collinearity. Therefore, we replaced the global measure of intimate partner violence with violent victimization and perpetration, respectively. Again, the independent effects of maternal depressive symptoms and the other co-morbid health risk behaviors remain unchanged when using the different measures of intimate partner violence; therefore, the results are not presented again. Figure 7 presents the main effects of intimate partner violence victimization and perpetration among mother–son and mother–daughter dyads. Among mother–son dyads, only maternal intimate partner violence perpetration is related to depressive symptoms among sons (approximate ages 13–14). Among mother–daughter dyads, both maternal intimate partner violence victimization and perpetration are negatively related to daughters' depressive symptoms during mid-adolescence (approximate ages 15–16), but the effect of maternal intimate partner victimization is stronger than the effect of perpetration.

The results previously presented demonstrated that the global measure of maternal intimate partner violence enhanced the effect of maternal depressive symptoms on sons' depressive symptoms during mid-adolescence. Similarly, both intimate partner violence victimization and perpetration among mothers enhance the positive effect of maternal depressive symptoms among sons, but the exacerbating effect is stronger for intimate partner violence perpetration than victimization (see Fig. 8a, b). Parallel to the findings for the global measure of intimate partner violence among daughters, neither intimate partner violence victimization nor perpetration interacted with maternal depressive symptoms to affect daughters' depressive symptoms.

We made additional efforts to further explore the main and interactive effects of co-morbid health risk behaviors on offspring depressive symptoms. For instance, mothers were asked if their child witnessed any instances of intimate partner violence, but this binary indicator of intimate partner violence was consistently unrelated to offspring depressive symptoms. Efforts were also made to examine whether maternal arrests were related to offspring depressive symptoms, but given the extremely low prevalence of arrests among our sample of mothers (<1%), the models would not converge.

Discussion

Continuity in depressive symptoms between mothers and offspring is well established (for a review see National Research Council 2009), and we extend upon this line of inquiry and explore three understudied facets of intergenerational continuity in depressive symptoms in order to add rich nuance to this complex relationship. First, we explore whether the magnitude of intergenerational continuity in depressive symptoms varies throughout adolescence. Second, we assess the role of comorbid health risk behaviors in intergenerational continuity of depressive symptoms. Third, we account for the potential moderating role of a child's gender in both intergenerational continuity in depressive symptoms and the manner in which co-morbid maternal health risk behaviors are related to intergenerational continuity in depressive symptoms. To address these issues, we employed time-varying effects models to a community-based sample of mother-child dyads where maternal depressive symptoms, maternal co-morbid health risk behaviors, and child's depressive symptoms were measured each year between child ages 12 and 17. Overall, the results indicate a dynamic relationship of continuity between mothers and their offspring during adolescence, which further varies by child gender. While the relationship between maternal depressive symptoms and son's depressive symptoms is small, relatively stable, and at times non-significant during mid-adolescence, it increases in effect during late adolescence. Among female offspring, on the other hand, the positive, significant relationship between maternal depressive symptoms continuously increases in effect throughout adolescence. Moreover, the effect of maternal depressive symptoms varies in the presence of co-morbid health risk behaviors with differential effects (both protective and exacerbating) across health risk behavior. Maternal substance use weakens the harmful effect of maternal depressive symptoms on the depressive symptoms of sons during mid-adolescence whereas maternal substance use exacerbates the harmful effect of maternal depressive symptoms among daughters. Additionally, intimate partner violence exacerbates the harmful effect of maternal depressive symptoms on the depressive symptoms among male offspring. We now focus on the interpretation of these findings.

First, the relationship between maternal depressive symptoms and a child's depressive symptoms are not consistent across adolescence, and this reinforces the notion that adolescent vulnerabilities to the mechanisms underlying intergenerational continuity in depressive symptoms, whether genetic or environmental, vary during this period of the life course marked by reduced dependence on one's family of origin and increased self-sufficiency. In fact, the Generic Elaborated Cognitive Vulnerability-Stress Model proposed by Hankin and Abramson (2001) stresses this very fact as biological, maturational, and social changes that characterize this period of the life course also make individuals more susceptible to negative responses, including depressive symptoms, to stressful life events. Moreover, the effect of maternal depressive symptoms increases as offspring approach late adolescence. This finding, which is consistent across gender, highlights a particular period of vulnerability for both male and female offspring.

Later adolescence is characterized by efforts to achieve autonomy and more frequent episodes of negative life events related to peers and significant others in contrast to negative life stress situated primarily in the family realm (Hankin and Abramson 2001). However, it

is also during this time that adolescents are more negatively affected by stressors related to maternal depressive symptoms. Perhaps youth in late adolescence are more likely to model their behavior after mothers who they see responding to life's challenges with depressive symptoms, or offspring are simply more socially aware and affected by maternal mental health. It may also be that the genetic underpinning linking maternal depressive symptoms to child's depressive symptoms is stronger at this point in the life course. In any case, this finding of increased vulnerability to maternal depressive symptoms as adolescence progresses appears to contradict the arguments of Goodman et al. (2011) that with increased age (maturity), adolescents are less vulnerable to maternal depressive symptoms because they are less dependent on mothers and can cognitively/emotionally better understand, process, and cope with the manifestations of maternal depressive symptoms. Given that this suggestion made by Goodman and colleagues (2011) was based on the relationship between maternal depressive symptoms in childhood and offspring depressive symptoms in adolescence, it would be worthwhile for future research to examine a more extensive portion of a child's life course (spanning childhood and adolescence) to more accurately compare how intergenerational continuity in depressive symptoms change over time.

We also built upon prior research suggesting that a child's gender moderates intergenerational continuity in depressive symptoms (e.g., Mason et al. 2017) and confirmed its importance as the patterns of continuity in depressive symptoms evince varying trends across the gender of offspring. Using contemporaneous measurements of depressive symptoms, the effect of maternal depressive symptoms on sons' symptoms varies in strength and significance across adolescence whereas the effect of maternal depressive symptoms on daughters' depressive symptoms is consistently positive and becomes stronger over the course of adolescence. Numerous theoretical models that seek to understand continuity in depression explicitly acknowledge that adolescents vary in susceptibility to the risk factor of maternal depressive symptoms across gender (e.g., Vulnerability-stress Model; Cyranowski et al. 2000; and the Generic Elaborated Cognitive Vulnerability-Stress Model; Hankin and Abramson 2001). These models also note that the negative consequences of maternal depressive symptoms are tied to the negative life events and emotional upheaval associated with puberty. Research suggests that early puberty and maturity are associated with problem behaviors and depressive symptoms in females but not males (Angold et al. 1998; Stattin and Magnusson 1990), and these findings are invoked to explain the higher prevalence of depressive symptoms among females compared to males during adolescence (Angold et al. 1998). In all likelihood, both the emotional changes associated with puberty and environmental adversity interact with maternal depressive symptoms at a younger age for females and are compounded over time with other negative life events related to peers, school, and romantic relationships. Moreover, the prevailing socialization schema for girls tends to promote internalizing behaviors (including depressive symptoms) when confronting stressful situations (Hill and Lynch 1983). On the other hand, early puberty and negative life events tend to be less strongly related to depressive symptoms in sons (Angold et al. 1998; Rudolph and Hammen 1999). Maturity, on the other hand, appears to be strongly related to depressive symptoms in males (Angold et al. 1998); thus, younger males may be less inclined to make sense of and be affected emotionally by negative life events until emotional maturity is nearly complete during later adolescence (Hankin and Abramson 2001). Thus, it

is likely that male offspring are less strongly influenced by maternal depressive symptoms until later adolescence when a certain level of maturity is reached, on average (as evidence by chronological age in the study). Pleck (1975) also notes that while younger adolescents adhere to gender-typed norms more stringently (e.g., hegemonic masculinity which eschews internalizing behaviors such as depressive symptoms in favor of instrumental, externalizing behaviors such as aggression; Horwitz and White 1987), older adolescents act more in line with needs, resulting in more depressive symptoms as a result of stressful environments such as ones related to maternal depressive symptoms.

This work also examined whether or not the presence of health risk behaviors frequently associated with maternal depressive symptoms account for intergenerational continuity in depressive symptoms. Among daughters, the positive, increasing relationship between maternal depressive symptoms and daughters' depressive symptoms remained even when accounting for these behaviors that are also known to increase the likelihood of adolescent depressive symptoms. Among sons, the relationship between maternal depressive symptoms and sons' depressive symptoms in early and mid-adolescence weakened. However, these co-morbid health risk behaviors did not account for the relationship between maternal depressive symptoms and depressive symptoms in sons from age 16 forward. Together, this suggests that the stress or adverse environments associated with these health risk behaviors do not account for intergenerational continuity in depressive symptoms of mother–child dyads, particularly in later adolescence.

Moreover, we investigated the potential interactive effects of maternal depressive symptoms and co-morbid health risk behaviors. The effect of maternal depressive symptoms on sons' depressive symptoms is conditioned by maternal substance use, but in an unexpected, negative direction. Although contrary to our hypothesis, this finding may make sense given the sample used for this analysis—the majority of whom are African American. Research suggests that African American males develop more autonomy at an earlier age (Smetana et al. 2004), which is protective against depressive symptoms. The stronger sense of autonomy among African American males may be reinforced by a home life that is particularly problematic with the co-occurrence of maternal depressive symptoms and substance use. Rates of substance use in African American families tend to be low as many families adhere to abstinence models of substance use, particularly for alcohol and drug use among women (Wallace and Muroff 2002). As such, maternal substance use in tandem with maternal depressive symptoms among African Americans families may signal a particularly problematic home environment causing sons to seek greater autonomy, which is protective against depressive symptoms. It may also be that the combination of maternal internalizing behaviors (i.e., depressive symptoms) and non-normative externalizing behaviors signals to young males that one's mother needs help and protective actions are taken, weakening the effect of maternal depressive symptoms. While our sample for analysis does not allow for the comparison of this interactive effect between African-American mother–son dyads and mother–son dyads of other race-ethnicities due to too few cases that allow for the re-estimation of our models, future research should attempt this comparison in order to more thoroughly understand the nature of these relationships and how the co-morbidity of these two risk behaviors may contribute to emotional competence and resilience (see Luthar et al. 2000).

Maternal involvement in intimate partner violence, on the other hand, appears to exacerbate the effect of maternal depressive symptoms on the depressive symptoms of sons in mid-adolescence, although this effect is weak and only of practical significance at extreme levels of involvement in intimate partner violence. While we cannot explicitly account for the reasons for this exacerbating effect during mid adolescence, it may be that increased pressure to be masculine and independent during mid-adolescence is met with visible signs of maternal violent victimization, perhaps as a result of hegemonic masculinity, or emasculation of one's partner by maternal perpetration of intimate partner violence. Interestingly, when separating the global measure of intimate partner violence into violent perpetration and violent victimization, respectively, the interactive effect between maternal depressive symptoms and intimate partner violence perpetration was stronger. As such, it may be that sons experience inner conflict as masculine socialization during adolescence is met with maternal violence perpetration (and victimization). As a result, adolescent males may be more willing to question what it means to be masculine and seemingly "emotionless" (Pleck 1975; Way et al. 2014) and respond to associated maternal depressive symptoms with confusion, internalization, and depressive symptoms of their own (Way et al. 2014).

The combination of maternal excessive alcohol use and drug use likely produces adverse environments and stressful situations for daughters and, unlike their male counterparts, this substance use compounds the effect of maternal depressive symptoms on depressive symptoms of daughters. However, the level of substance use required to produce an effect of practical significance is quite high. Overall, the sum of these findings suggests that for mother-daughter dyads, daughters are negatively affected by maternal depressive symptoms alone and largely unaffected by the co-morbid health risk behaviors.

Our findings have significant implications for treatment and prevention efforts seeking to stymie intergenerational continuity in depressive symptoms. First, more routine access to depression prevention programming should be offered to all mothers in order to limit the negative effects of depressive symptoms on the next generation. Moreover, *effective* prevention programs such as Family Talk (Beardslee et al. 2007) should specifically target mother-daughter dyads at the onset of adolescence and focus on treating depressive symptoms early in both generations before the negative effects of maternal depressive symptoms have time to escalate in effect and consequence as adolescence progresses. Examples of effective intervention programs for youth at the onset of depressive symptoms include Interpersonal Psychotherapy-Adolescent Skills Training (Young et al. 2006) and Teaching Kids to Cope programs (Puskar et al. 2003). Moreover, other cognitive behavioral interventions for adolescents (Gregory Clarke's Cognitive-Behavioral prevention Intervention; Clarke et al. 1995; Penn Resiliency Program; Gillham et al. 2007) could mitigate the negative effects of maternal depressive symptoms as they specifically seek to teach individuals how to effectively cope with life stressors including maternal depressive symptoms and other negative life events. Importantly, these programs could be adapted to emphasize the development of coping skills for mothers and daughters in order to disrupt the continuity in maladaptive coping strategies that daughters observe in their mothers (e.g., rumination; see Papadakis et al. 2006). Mother-son dyads would also benefit from these same interventions, particularly in later adolescence. Young male adolescents also are likely

to benefit from maternal participation in programs targeting antisocial behaviors in addition to depressive symptoms, such as Cognitive Behavioral Therapy, which is shown to be effective in the reduction of various antisocial behaviors including substance use (McHugh and Barlow 2010), criminal participation (Wilson et al. 2005), and intimate partner violence (Eckhardt et al. 2013).

A unique aspect of this study is that we used prospective longitudinal data collected at yearly intervals to examine variation in the intergenerational continuity of depressive symptoms during a significant portion of adolescence. Ideally however, we would have extended this study over a larger portion of the child's life course, particularly during childhood and during the transition from adolescence to young adulthood. Unfortunately, this was not possible with the current data given the different measurement of depressive symptoms in children prior to age 12 and the lack of information on maternal depressive symptoms past the child age of 17. Furthermore, while we view the assessment of intergenerational continuity in depressive symptoms as advantageous because non-clinical levels of depressive symptoms still have deleterious consequences (Gotlib et al. 1995), it would be worthwhile to replicate this analysis with clinical diagnoses of depression. Moreover, we recognize that the reliance on self-report measures of health behavior/symptom frequency is wrought with its own limitations including reduced precision. Therefore, this type of analysis would also benefit from using symptoms and diagnoses assessed via a diagnostic interview. Additionally, our measure of the frequency of substance use produced an extremely skewed distribution. While we added one and took the natural log of this measure to address the skewed distribution, we urge caution and careful consideration when interpreting these findings.

This study is also limited in that it only represents mother-child dyads from one birth cohort in one city in the United States. Although intergenerational continuity in depressive symptoms is thought to be place invariant, the predominant environment (notably not all mother-child dyads live in Rochester for part or the duration of the study period) and adversity of the sample (poverty) may affect the generalizability of these findings. Moreover, the sample's racial/ethnic makeup is predominantly minority (African American and Hispanic). While this is a positive element of the study because these are understudied populations with respect to intergenerational continuity in depressive symptoms, we suggest that replication with other populations (both minority and non-minority) is warranted to confirm and validate these findings, particularly as it is unclear whether these findings would extend to non-minority populations.

Conclusion

Depressive symptoms during adolescence are common and pose substantial consequences for health and development (National Institute of Mental Health 2017). This study acknowledges the varying importance of maternal depressive symptoms, a key risk factor for child onset and escalation of depressive symptoms, on adolescent depressive symptoms and further underscores that intergenerational continuity in depressive symptoms is conditioned by child gender. In addition, this research highlights specific periods of the life course of adolescents in which prevention and intervention efforts should target (mid to late

adolescence for males and all of adolescence for females). Furthermore, the results suggest that these prevention and intervention efforts should target maternal co-morbid health risk behaviors, such as substance use and intimate partner violence, in order to limit continuity in depressive symptoms. Given that these findings present only a part of the story on intergenerational continuity in depressive symptoms, additional research is warranted to continue to understand the complex relationships between maternal depressive symptoms and offspring depressive symptoms, especially those that focus on the causal pathways from various etiological perspectives, as these pathways may also vary across gender. Only through additional and rigorous evaluations of intergenerational continuity in depressive symptoms will prevention efforts be truly efficacious in their efforts to reduce depressive symptoms among adolescents and limit the negative consequences associated with this psychopathology.

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References

Anda RF, Whitfield CL, Felitti VJ, Chapman D, Edwards VJ, Dube SR, Williamson DF. Adverse childhood experiences, alcoholic parents, and later risk of alcoholism and depression. Psychiatric Services. 2002; 53(8):1001–1009. DOI: 10.1176/appi.ps.53.8.1001 [PubMed: 12161676]

Angold A, Costello EJ, Worthman CM. Puberty and depression: The roles of age, pubertal status and pubertal timing. Psychological Medicine. 1998; 28(1):51–61. DOI: 10.1017/S003329179700593X [PubMed: 9483683]

Bair-Merritt MH, Ghazarian SR, Burrell L, Crowne SS, McFarlane E, Duggan AK. Understanding how intimate partner violence impacts school age children's internalizing and externalizing problem behaviors: A secondary analysis of Hawaii Healthy Start Program evaluation data. Journal of Child & Adolescent Trauma. 2015; 8(4):245–251. DOI: 10.1007/s40653-015-0066-8 [PubMed: 26889302]

Bansal V, Goyal S, Srivastava K. Study of prevalence of depression in adolescent students of a public school. Industrial Psychiatry Journal. 2009; 18(1):43.doi: 10.4103/0972-6748.57859 [PubMed: 21234162]

Bardone AM, Moffitt TE, Caspi A, Dickson N, Silva PA. Adult mental health and social outcomes of adolescent girls with depression and conduct disorder. Development and psychopathology. 1996; 8(4):811–829. DOI: 10.1017/S0954579400007446

Beardslee WR, Gladstone TRG, O'Connor EE. Developmental risk of depression: Experience matters. Child and Adolescent Psychiatric clinics of North America. 2012; 21(2):261–278. DOI: 10.1016/j.chc.2011.12.001 [PubMed: 22537726]

Beardslee WR, Wright EJ, Gladstone TRG, Forbes P. Long-term effects from a randomized trial of two public health preventive interventions for parental depression. Journal of Family Psychology. 2007; 21(4):703–713. DOI: 10.1037/0893-3200.21.4.703 [PubMed: 18179342]

Boden JM, Fergusson DM. Alcohol and depression. Addiction. 2011; 106(5):906–914. DOI: 10.1111/j.1360-0443.2010.03351.x [PubMed: 21382111]

Bonomi AE, Thompson RS, Anderson M, Reid RJ, Carrell D, Dimer JA, Rivara FP. Intimate partner violence and women's physical, mental, and social functioning. American Journal of preventive Medicine. 2006; 30(6):458–466. DOI: 10.1016/J.AMEPRE.2006.01.015 [PubMed: 16704938]

Breslend NL, Parent J, Forehand R, Compas BE, Thigpen JC, Hardcastle E. Parental depressive symptoms and youth internalizing and externalizing problems: The moderating role of interparental conflict. Journal of Family Violence. 2016; 31(7):823–831. DOI: 10.1007/s10896-016-9817-z [PubMed: 27795614]

Brière FN, Rohde P, Seeley JR, Klein D, Lewinsohn PM. Comorbidity between major depression and alcohol use disorder from adolescence to adulthood. Comprehensive Psychiatry. 2014; 55(3):526–533. DOI: 10.1016/J.COMPPSYCH.2013.10.007 [PubMed: 24246605]

Capaldi DM, Knoble NB, Shortt JW, Kim HK. A systematic review of risk factors for intimate partner violence. Partner Abuse. 2012; 3(2):231–280. DOI: 10.1891/1946-6560.3.2.231 [PubMed: 22754606]

Carney M, Buttell F, Dutton D. Women who perpetrate intimate partner violence: A review of the literature with recommendations for treatment. Aggression and Violent Behavior. 2007.

Chassin L, Pitts SC, DeLucia C, Todd M. A longitudinal study of children of alcoholics: Predicting young adult substance use disorders, anxiety, and depression. Journal of Abnormal Psychology. 1999; 108(1):106–119. DOI: 10.1037/0021-843X.108.1.106 [PubMed: 10066997]

Chen J, Yu J. Sex differences in genetic and environmental influences on adolescent depressive symptoms: A meta-analytic review. Depression Research and Treatment. 2015.

Chen M, Johnston C, Sheeber L, Leve C. Parent and adolescent depressive symptoms: The role of parental attributions. Journal of Abnormal Child Psychology. 2009; 37(1):119–130. DOI: 10.1007/s10802-008-9264-2 [PubMed: 18712594]

Clarke GN, Hawkins W, Murphy M, Sheeber LB, Lewinsohn PM, Seeley JR. Targeted prevention of unipolar depressive disorder in an at-risk sample of high school adolescents: A randomized trial of a group cognitive intervention. Journal of the American Academy of Child & Adolescent Psychiatry. 1995; 34(3):312–321. DOI: 10.1097/00004583-199503000-00016 [PubMed: 7896672]

Cole DA, Martin JM, Peeke LA, Seroczynski AD, Fier J. Children's over- and underestimation of academic competence: A longitudinal study of gender differences, depression, and anxiety. Child Development. 1999; 70(2):459–473. DOI: 10.1111/1467-8624.00033 [PubMed: 10218266]

Conner KR, Pinquart M, Gamble SA. Meta-analysis of depression and substance use among individuals with alcohol use disorders. Journal of Substance Abuse Treatment. 2009; 37(2):127–137. DOI: 10.1016/J.JSAT.2008.11.007 [PubMed: 19150207]

Cyranowski JM, Frank E, Young E, Shear MK. Adolescent onset of the gender difference in lifetime rates of major depression: A theoretical model. Archives of General Psychiatry. 2000; 57(1):21–27. DOI: 10.1001/archpsyc.57.1.21 [PubMed: 10632229]

Dallaire DH, Wilson LC. The relation of exposure to parental criminal activity, arrest, and sentencing to children's maladjustment. Journal of Child and Family Studies. 2010; 19(4):404–418. DOI: 10.1007/s10826-009-9311-9

Dallaire DH, Zeman JL, Thrash TM. Children's experiences of maternal incarceration-specific risks: Predictions to psychological maladaptation. Journal of Clinical Child & Adolescent Psychology. 2015; 44(1):109–122. DOI: 10.1080/15374416.2014.913248 [PubMed: 24871820]

Devries KM, Mak JY, Bacchus LJ, Child JC, Falder G, Petzold M, Watts CH. Intimate partner violence and incident depressive symptoms and suicide attempts: A systematic review of longitudinal studies. PloS Medicine. 2013; 10(5):e1001439.doi: 10.1371/journal.pmed.1001439 [PubMed: 23671407]

Dishion TJ, Capaldi DM, Yoerger K. Middle childhood antecedents to progressions in male adolescent substance use: An ecological analysis of risk and protection. Journal of Adolescent Research. 1999; 14(2):175–205. DOI: 10.1177/0743558499142003

Douzenis A, Ferentinos P, Lykouras E. Depression and criminality. Archives of Hellenic Medicine. 2005; 22(6):535–543.

Downey G, Coyne JC. Children of depressed parents: An integrative review. Psychological Bulletin. 1990; 108(1):50–76. DOI: 10.1037/0033-2909.108.1.50 [PubMed: 2200073]

Dziak JJ, Henry KL. Two-part predictors in regression models. Multivariate Behavioral Research. 2017; 52(5):551–561. DOI: 10.1080/00273171.2017.1333404 [PubMed: 28622056]

Eckhardt CI, Murphy CM, Whitaker DJ, Sprunger J, Dykstra R, Woodard K. The effectiveness of intervention programs for perpetrators and victims of intimate partner violence. Partner Abuse. 2013; 4(2):196–231. DOI: 10.1891/1946-6560.4.2.196

Epstein M, Hill KG, Roe SS, Bailey JA, Iacono WG, McGue M, et al. Time varying effects of families and peers on adolescent marijuana use: Person–environment interactions across development. Development and psychopathology. 2017; 29(3):887–900. [PubMed: 27417425]

Evans-Polce RJ, Vasilenko SA, Lanza ST. Changes in gender and racial/ethnic disparities in rates of cigarette use, regular heavy episodic drinking, and marijuana use: Ages 14 to 32. Addictive Behaviors. 2015.

Fals-Stewart W, Kelley ML, Fincham FD, Golden J, Logsdon T. Emotional and behavioral problems of children living with drug-abusing fathers: Comparisons with children living with alcohol-abusing and non-substance-abusing fathers. Journal of Family Psychology. 2004; 18(2):319–330. DOI: 10.1037/0893-3200.18.2.319 [PubMed: 15222839]

Farnworth M, Thornberry TP, Krohn MD, Lizotte AJ. Measurement in the study of class and delinquency: Integrating theory and research. Journal of Research in Crimean and Delinquency. 1994; 31(1):32–61. DOI: 10.1177/0022427894031001002

Flint J, Kendler KS. The genetics of major depression. Neuron. 2014; 81(3):484–503. DOI: 10.1016/J.NEURON.2014.01.027 [PubMed: 24507187]

Folger AT, Putnam KT, Putnam FW, Peugh JL, Eismann EA, Sa T, Ammerman RT. Maternal interpersonal trauma and child social-emotional development: An intergenerational effect. Paediatric and Perinatal Epidemiology. 2017; 31(2):99–107. DOI: 10.1111/ppe.12341 [PubMed: 28140478]

Garber J, Cole DA. Intergenerational transmission of depression: A launch and grow model of change across adolescence. Development and Psychopathology. 2010; 22(4):819–830. DOI: 10.1017/S0954579410000489 [PubMed: 20883584]

Ge X, Best KM, Conger RD, Simons RL. Parenting behaviors and the occurrence and co-occurrence of adolescent depressive symptoms and conduct problems. Developmental Psychology. 1996; 32(4):717–731. DOI: 10.1037/0012-1649.32.4.717

Ge X, Conger RD, Elder GH. Pubertal transition, stressful life events, and the emergence of gender differences in adolescent depressive symptoms. Developmental Psychology. 2001; 37(3):404–417. DOI: 10.1037/0012-1649.37.3.404 [PubMed: 11370915]

Gillham JE, Reivich KJ, Freres DR, Chaplin TM, Shatté AJ, Samuels B, Seligman MEP. School-based prevention of depressive symptoms: A randomized controlled study of the effectiveness and specificity of the penn resiliency program. Journal of Consulting and Clinical Psychology. 2007; 75(1):9–19. DOI: 10.1037/0022-006X.75.1.9 [PubMed: 17295559]

Goodman SH. Depression in mothers. Annual Review of Clinical Psychology. 2007; 3(1):107–135. DOI: 10.1146/annurev.clinpsy.3.022806.091401

Goodman SH, Gotlib IH. Risk for psychopathology in the children of depressed mothers: A developmental model for understanding mechanisms of transmission. Psychological Review. 1999; 106(3):458–490. DOI: 10.1037/0033-295X.106.3.458 [PubMed: 10467895]

Goodman SH, Rouse MH, Connell AM, Broth MR, Hall CM, Heyward D. Maternal depression and child psychopathology: A meta-analytic review. Clinical Child and Family Psychology Review. 2011; 14(1):1–27. DOI: 10.1007/s10567-010-0080-1 [PubMed: 21052833]

Gotlib IH, Colich NL. Children of depressed parents. In: Gotlib I, Hammen C, editorsHandbook of depression. 3. New York: Guilford Press; 2014. 240–258.

Gotlib IH, Lewinsohn PM, Seeley JR. Symptoms versus a diagnosis of depression: Differences in psychosocial functioning. Journal of Consulting and Clinical Psychology. 1995; 63(1):90–100. [PubMed: 7896995]

Hammen C. Adolescent depression: Stressful interpersonal contexts and risk for recurrence. Current Directions in Psychological Science. 2009; 18(4):200–204. DOI: 10.1111/j.1467-8721.2009.01636.x [PubMed: 20161119]

Hammen C, Brennan PA. Severity, chronicity, and timing of maternal depression and risk for adolescent offspring diagnoses in a community sample. Archives of General Psychiatry. 2003; 60(3):253.doi: 10.1001/archpsyc.60.3.253 [PubMed: 12622658]

Hammen C, Brennan PA, Keenan-Miller D. Patterns of adolescent depression to age 20: The role of maternal depression and youth interpersonal dysfunction. Journal of Abnormal Child Psychology. 2008; 36(8):1189–1198. DOI: 10.1007/s10802-008-9241-9 [PubMed: 18473162]

Hammen C, Hazel NA, Brennan PA, Najman J. Intergenerational transmission and continuity of stress and depression: depressed women and their offspring in 20 years of follow-up. Psychological Medicine. 2012; 42(5):931–942. DOI: 10.1017/S0033291711001978 [PubMed: 22018414]

Hammen C, Shih JH, Brennan PA. Intergenerational transmission of depression: Test of an interpersonal stress model in a community sample. Journal of Consulting and Clinical Psychology. 2004; 72(3):511–522. DOI: 10.1037/0022-006X.72.3.511 [PubMed: 15279534]

Hankin BL. Depression from childhood through adolescence: risk mechanisms across multiple systems and levels of analysis. Current Opinion in Psychology. 2015.

Hankin BL, Abramson LY. Development of gender differences in depression: An elaborated cognitive vulnerability–transactional stress theory. Psychological Bulletin. 2001; 127(6):773–796. DOI: 10.1037/0033-2909.127.6.773 [PubMed: 11726071]

Helweg–Larsen M, Harding HG, Kleinman KE. Risk perceptions of dating violence among college women: The role of experience and depressive symptoms. Journal of Social and Clinical Psychology. 2008; 27(6):600–620. DOI: 10.1521/jscp.2008.27.6.600

Henry KL, Thornberry TP, Lee RD. The protective effects of intimate partner relationships on depressive symptomatology among adult parents maltreated as children. Journal of Adolescent Health. 2015; 57(2):150–156. DOI: 10.1016/j.jadohealth.2015.02.015 [PubMed: 25912653]

Hill JP, Lynch ME. Girls at Puberty. Boston, MA: Springer; 1983. The intensification of gender-related role expectations during early adolescence; 201–228.

Holtzworth-Munroe A, Stuart GL. Typologies of male batterers: Three subtypes and the differences among them. Psychological Bulletin. 1994; 116(3):476–497. DOI: 10.1037/0033-2909.116.3.476 [PubMed: 7809309]

Horwitz AV, White HR. Gender role orientations and styles of pathology among adolescents. Journal of Health and Social Behavior. 1987; 28(2):158–170. DOI: 10.2307/2137129 [PubMed: 3611702]

Huizinga D, Esbensen FA, Weiher AW. Are there multiple paths to delinquency? The Journal of Criminal Law and Criminology. 1991; 82(1):83–118. DOI: 10.2307/1143790

Jaffee SR, Moffitt TE, Caspi A, Fombonne E, Poulton R, Martin J. Differences in early childhood risk factors for juvenile-onset and adult-onset depression. Archives of General Psychiatry. 2002; 59(3): 215–222. DOI: 10.1001/archpsyc.59.3.215 [PubMed: 11879158]

Keenan-Miller D, Hammen C, Brennan P. Adolescent psychosocial risk factors for severe intimate partner violence in young adulthood. Journal of Consulting and Clinical Psychology. 2007; 75(3): 456–463. DOI: 10.1037/0022-006X.75.3.456 [PubMed: 17563162]

Kerr DCR, Leve LD, Harold GT, Natsuaki MN, Neiderhiser JM, Shaw DS, Reiss D. Influences of biological and adoptive mothers' depression and antisocial behavior on adoptees' early behavior trajectories. Journal of Abnormal Child Psychology. 2013; 41(5):723–734. DOI: 10.1007/s10802-013-9711-6 [PubMed: 23408036]

Khantzian EJ. The self-medication hypothesis of addictive disorders: focus on heroin and cocaine dependence. America Journal of Psychiatry. 1985; 142(11):1259–1264.

Lai HMX, Cleary M, Sitharthan T, Hunt GE. Prevalence of comorbid substance use, anxiety and mood disorders in epidemiological surveys, 1990–2014: A systematic review and meta-analysis. Drug and Alcohol dependence. 2015; 154:1–13. DOI: 10.1016/J.DRUGALCDEP.2015.05.031 [PubMed: 26072219]

Lau JY, Eley TC. Disentangling gene-environment correlations and interactions on adolescent depressive symptoms. Journal of Child Psychology and Psychiatry. 2008; 49(2):142–150. [PubMed: 18211276]

Lev-Ran S, Roerecke M, Le Foll B, George TP, McKenzie K, Rehm J. The association between cannabis use and depression: a systematic review and meta-analysis of longitudinal studies.

Psychological Medicine. 2014; 44(4):797–810. DOI: 10.1017/S0033291713001438 [PubMed: 23795762]

Li R, Tan X, Huang L, Wagner AT, Yang J. TVEM (time-varying effect model) SAS macro users' guide (Version 2.1.1). University Park; PA: 2014.

Lieb R, Isensee B, Höfler M, Pfister H, Wittchen HU. Parental major depression and the risk of depression and other mental disorders in offspring: A prospective-longitudinal community study. Archives of General Psychiatry. 2002; 59(4):365–374. DOI: 10.1001/archpsyc.59.4.365 [PubMed: 11926937]

Luthar SS, Cicchetti D, Becker B. The construct of resilience: A critical evaluation and guidelines for future work. Child Development. 2000; 71(3):543–562. DOI: 10.1111/1467-8624.00164 [PubMed: 10953923]

Mars B, Collishaw S, Smith D, Thapar A, Potter R, Sellers R, Thapar A. Offspring of parents with recurrent depression: Which features of parent depression index risk for offspring psychopathology? Journal of Affective Disorders. 2012; 136(1–2):44–53. DOI: 10.1016/j.jad.2011.09.002 [PubMed: 21962850]

Mason WA, Chmelka MB, Trudeau L, Spoth RL. Gender moderation of the intergenerational transmission and stability of depressive symptoms from early adolescence to early adulthood. Journal of Youth and Adolescence. 2017; 46(1):248–260. DOI: 10.1007/s10964-016-0480-8 [PubMed: 27055682]

McFarlane JM, Groff JY, O'Brien JA, Watson K. Behaviors of children who are exposed and not exposed to intimate partner violence: An analysis of 330 Black, White, and Hispanic children. Pediatrics. 2003; 112(3):e202–e207. http://pedia-trics.aappublications.org/content/112/3/e202.short. [PubMed: 12949313]

McHugh RK, Barlow DH. The dissemination and implementation of evidence-based psychological treatments: A review of current efforts. American Psychologist. 2010; 65(2):73–84. DOI: 10.1037/a0018121 [PubMed: 20141263]

Mikkonen J, Moustgaard H, Remes H, Martikainen P. Intergenerational transmission of depressive symptoms—The role of gender, socioeconomic circumstances, and the accumulation of parental symptoms. Journal of Affective Disorders. 2016; 204:74–82. DOI: 10.1016/j.jad.2016.06.036 [PubMed: 27341423]

Murray J, Farrington DP. Parental imprisonment: Long-lasting effects on boys' internalizing problems through the life course. Development and Psychopathology. 2008; 20(1):273–290. DOI: 10.1017/S0954579408000138 [PubMed: 18211738]

Naicker K, Galambos NL, Zeng Y. Social, demographic, and health outcomes in the 10 years following adolescent depression. Journal of Adolescent Health. 2013; 52(5):533–538. DOI: 10.1016/J.JADOHEALTH.2012.12.016 [PubMed: 23499382]

National Institute of Mental Health. Major depression among adolescents. 2017. https://www.nimh.nih.gov/health/statistics/prevalence/major-depression-among-adolescents.shtml

National Research Council. Preventing mental, emotional, and behavioral disorders among young people: Progress and possibilities. Washington, DC: National Academies Press; 2009.

Nolen-Hoeksema S, Girgus JS. The emergence of gender differences in depression during adolescence. Psychological Bulletin. 1994; 115(3):424–443. DOI: 10.1037/0033-2909.115.3.424 [PubMed: 8016286]

Nolen-Hoeksema S, Larson J, Grayson C. Explaining the gender difference in depressive symptoms. Journal of personality and Social Psychology. 1999; 77(5):1061–1072. DOI: 10.1037/0022-3514.77.5.1061 [PubMed: 10573880]

Owen AE, Thompson MP, Shaffer A, Jackson EB, Kaslow NJ. Family variables that mediate the relation between intimate partner violence (IPV) and child adjustment. Journal of Family Violence. 2009; 24(7):433–445. DOI: 10.1007/s10896-009-9239-2

Pacek LR, Martins SS, Crum RM. The bidirectional relationships between alcohol, cannabis, co-occurring alcohol and cannabis use disorders with major depressive disorder: Results from a national sample. Journal of Affective Disorders. 2013; 148(2–3):188–195. DOI: 10.1016/J.JAD.2012.11.059 [PubMed: 23260381]

Papadakis AA, Prince RP, Jones NP, Strauman TJ. Self-regulation, rumination, and vulnerability to depression in adolescent girls. Development and Psychopathology. 2006.

Patterson G, Reid J, Dishion T. Antisocial boys. Eugene, OR: Castalia Publishing Company; 1992.

Peterson JK, Skeem J, Kennealy P, Bray B, Zvonkovic A. How often and how consistently do symptoms directly precede criminal behavior among offenders with mental illness. Law and Human Behavior. 2014; 38(5):439–449. [PubMed: 24730388]

Pilowsky DJ, Wickramaratne P, Poh E, Hernandez M, Batten LA, Flament MF, Weissman MM. Psychopathology and functioning among children of treated depressed fathers and mothers. Journal of Affective Disorders. 2014; 164:107–111. DOI: 10.1016/j.jad.2014.04.012 [PubMed: 24856562]

Pine DS, Cohen E, Cohen P, Brook J. Adolescent depressive symptoms as predictors of adult depression: moodiness or mood disorder? American Journal of Psychiatry. 1999; 156(1):133–135. DOI: 10.1176/ajp.156.1.133 [PubMed: 9892310]

Pleck JH. Masculinity-femininity. Sex Roles. 1975; 1(2):161–178. DOI: 10.1007/BF00288009

Puskar K, Sereika S, Tusaie-Mumford K. Effect of the Teaching Kids to Cope (TKC©) Program on outcomes of depression and coping among rural adolescents. Journal of Child and Adolescent Psychiatric Nursing. 2003; 16(2):71–80. DOI: 10.1111/j.1744-6171.2003.tb00350.x [PubMed: 12873069]

Quello SB, Brady KT, Sonne SC. Mood disorders and substance use disorder: A complex comorbidity. Science & Practice Perspectives. 2005; 3(1):13–21. http://www.ncbi.nlm.nih.gov/pubmed/18552741. [PubMed: 18552741]

Radloff LS. The CES-D scale: A self-report depression scale for research in the general population. Applied Psychological Measurement. 1977; 1(3):385–401. DOI: 10.1177/014662167700100306

Radloff LS. The use of the Center for Epidemiologic Studies Depression Scale in adolescents and young adults. Journal of Youth and Adolescence. 1991; 20(2):149–166. DOI: 10.1007/BF01537606 [PubMed: 24265004]

Ritakallio M, Koivisto AM, von der Pahlen B, Pelkonen M, Marttunen M, Kaltiala-Heino R. Continuity, comorbidity and longitudinal associations between depression and antisocial behaviour in middle adolescence: A 2-year prospective follow-up study. Journal of adolescence. 2008; 31(3):355–370. DOI: 10.1016/j.adolescence.2007.06.006 [PubMed: 17692369]

Rudolph KD, Hammen C. Age and gender as determinants of stress exposure, generation, and reactions in youngsters: A transactional perspective. Child Development. 1999; 70(3):660–677. DOI: 10.1111/1467-8624.00048 [PubMed: 10368914]

Salk RH, Petersen JL, Abramson LY, Hyde JS. The contemporary face of gender differences and similarities in depression throughout adolescence: Development and chronicity. Journal of Affective Disorders. 2016; 205:28–35. DOI: 10.1016/j.jad.2016.03.071 [PubMed: 27391269]

Seidlitz L, Diener E. Sex differences in the recall of affective experiences. Journal of Personality and Social Psychology. 1998; 74(1):262–271. DOI: 10.1037/0022-3514.74.1.262 [PubMed: 9457787]

Sellers R, Harold GT, Elam K, Rhoades KA, Potter R, Mars B, Collishaw S. Maternal depression and co-occurring antisocial behaviour: testing maternal hostility and warmth as mediators of risk for offspring psychopathology. Journal of Child Psychology and Psychiatry. 2014; 55(2):112–120. DOI: 10.1111/jcpp.12111 [PubMed: 23808575]

Shiyko MP, Lanza ST, Tan X, Li R, Shiffman S. Using the time-varying effect model (TVEM) to examine dynamic associations between negative affect and self confidence on smoking urges: differences between successful quitters and relapsers. Prevention Science. 2012; 13(3):288–99. DOI: 10.1007/s11121-011-0264-z [PubMed: 22246429]

Shlafer RJ, Poehlmann J, Donelan-McCall N. Maternal jail time, conviction, and arrest as predictors of children's 15-year antisocial outcomes in the context of a nurse home visiting program. Journal of Clinical Child & Adolescent Psychology. 2012; 41(1):38–52. DOI: 10.1080/15374416.2012.632345 [PubMed: 22233244]

Silberg JL, Maes H, Eaves LJ. Unraveling the effect of genes and environment in the transmission of parental antisocial behavior to children's conduct disturbance, depression and hyperactivity. Journal of Child Psychology and Psychiatry. 2012; 53(6):668–677. DOI: 10.1111/j.1469-7610.2011.02494.x [PubMed: 22141405]

Silverstein M, Augustyn M, Cabral H, Zuckerman B. Maternal depression and violence exposure: Double jeopardy for child school functioning. Pediatrics. 2006; 118(3):e792–e800. http://pediatrics.aappublications.org/content/118/3/e792.short. [PubMed: 16950968]

Smetana JG, Campione-Barr N, Daddis C. Longitudinal development of family decision making: Defining healthy behavioral autonomy for middle-class African American adolescents. Child Development. 2004; 75(5):1418–1434. DOI: 10.1111/j.1467-8624.2004.00749.x [PubMed: 15369523]

Stattin H, Magnusson D. Pubertal maturation in female development. Lawrence Erlbaum Associates, Inc; 1990.

Straus M. The conflict tactics scales and its critics: An evaluation and new data on validity and reliability. In: Straus M, Gelles R, editorsPhysical violence in American families: Risk factors and adaptations to violence in 8145 families. New Brunswick, NJ: Transaction Publishers; 1990. 49–73.

Straus MA. Measuring intrafamily conflict and violence: The conflict tactics (CT) scales. Journal of Marriage and Family. 1979.

Straus MA, Hamby SL, Boney-McCoy S, Sugarman DB. The revised conflict tactics scales (CTS2) development and preliminary psychometric data. Journal of Family Issues. 1996; 17(3):283–316. DOI: 10.1177/019251396017003001

Stringaris A, Lewis G, Maughan B. Developmental pathways from childhood conduct problems to early adult depression: findings from the ALSPAC cohort. The British Journal of Psychiatry. 2014; 205(1) http://bjp.rcpsych.org/content/205/1/17.short.

Sullivan PF, Neale MC, Kendler KS. Genetic epidemiology of major depression: Review and meta-analysis. American Journal of Psychiatry. 2000; 157(10):1552–1562. DOI: 10.1176/appi.ajp.157.10.1552 [PubMed: 11007705]

Swendsen JD, Merikangas KR. The comorbidity of depression and substance use disorders. Clinical Psychology Review. 2000.

Tan X, Shiyko MP, Li R, Li Y, Dierker L. A time-varying effect model for intensive longitudinal data. Psychological Methods. 2012; 17(1):61–77. DOI: 10.1037/a0025814 [PubMed: 22103434]

Thapar A, Collishaw S, Pine DS, Thapar AK. Depression in adolescence. The Lancet. 2012; 379(9820):1056–1067. DOI: 10.1016/S0140-6736(11)60871-4

Vasilenko SA, Linden-Carmichael A, Lanza ST, Patrick ME. Sexual behavior and heavy episodic drinking across the transition to adulthood: Differences by college attendance. Journal of Research on Adolescence. 2017.

Vasilenko SA, Piper ME, Lanza ST, Liu X, Yang J, Li R. Time-varying processes involved in smoking lapse in a randomized trial of smoking cessation therapies. Nicotine & Tobacco Research. 2014; 16(Suppl 2):S135–S143. DOI: 10.1093/ntr/ntt185 [PubMed: 24711627]

Wallace JM, Muroff JR. Preventing substance abuse among African American children and youth: Race differences in risk factor exposure and vulnerability. The Journal of Primary Prevention. 2002; 22(3):235–261. DOI: 10.1023/A:1013617721016

Way N, Cressen J, Bodian S, Preston J, Nelson J, Hughes D. “It might be nice to be a girl… Then you wouldn’t have to be emotionless”: Boys’ resistance to norms of masculinity during adolescence. Psychology of Men & Masculinity. 2014; 15(3):241–252. DOI: 10.1037/a0037262

Weissman MM, Wolk S, Goldstein RB, Moreau D, Adams P, Greenwald S, Wickramaratne P. Depressed adolescents grown up. JAMA. 1999; 281(18):1707–1713. DOI: 10.1001/jama.281.18.1707 [PubMed: 10328070]

Whalen DJ, Luby JL, Tilman R, Mike A, Barch D, Belden AC. Latent class profiles of depressive symptoms from early to middle childhood: predictors, outcomes, and gender effects. Journal of Child Psychology and Psychiatry. 2016; 57(7):794–804. DOI: 10.1111/jcpp.12518 [PubMed: 26748606]

Whitaker RC, Orzol SM, Kahn RS. Maternal mental health, substance use, and domestic violence in the year after delivery and subsequent behavior problems in children at age 3 years. Archives of General Psychiatry. 2006; 63(5):551–560. DOI: 10.1001/archpsyc.63.5.551 [PubMed: 16651512]

Wickrama KAS, Conger RD, Lorenz FO, Jung T. Family antecedents and consequences of trajectories of depressive symptoms from adolescence to young adulthood: A life course investigation.

Journal of Health and Social Behavior. 2008; 49(4):468–483. DOI: 10.1177/002214650804900407 [PubMed: 19181050]

Wilkinson AL, Halpern CT, Herring AH. Directions of the relationship between substance use and depressive symptoms from adolescence to young adulthood. Addictive Behaviors. 2016; 60:64–70. DOI: 10.1016/j.addbeh.2016.03.036 [PubMed: 27100470]

Wilson DB, Bouffard LA, Mackenzie DL. A quantitative review of structured, group-oriented, cognitive-behavioral programs for offenders. Criminal Justice and Behavior. 2005; 32(2):172–204. DOI: 10.1177/0093854804272889

Womack SR, Shaw DS, Weaver CM, Forbes EE. Bidirectional associations between cannabis use and depressive symptoms from adolescence through early adulthood among at-risk young men. Journal of Studies on Alcohol and Drugs. 2016; 77(2):287–297. https://doi.org/10.15288/JSAD.2016.77.287. [PubMed: 26997187]

Young JF, Mufson L, Davies M. Efficacy of interpersonal psychotherapy-adolescent skills training: An indicated preventive intervention for depression. Journal of Child Psychology and Psychiatry. 2006; 47(12):1254–1262. DOI: 10.1111/j.1469-7610.2006.01667.x [PubMed: 17176380]

Zalsman G, Brent DA, Weersing VR. Depressive disorders in childhood and adolescence: An overview: Epidemiology, clinical manifestation and risk factors. Child and Adolescent Psychiatric Clinics of North America. 2006; 15(4):827–841. DOI: 10.1016/j.chc.2006.05.002 [PubMed: 16952763]

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Appendix

Table 2

Individual items included in measures for analysis

Maternal depressive symptoms (adapted CES-D)	Responses: 0 (Never), 1 (Seldom), 2 (Sometimes), 3 (Often), 4(Always)
	1. Feel bothered by things that don t usually bother you?
	2. Not feel like eating or have a poor appetite?
	3. Feel you had trouble keeping your mind on what you were doing?
	4. Feel depressed?
	5. Feel that everything you did was an effort, or that everything was difficult for you to do?

	6. Feel hopeful for the future? (reverse coded)
	7. Feel that you were just as good as other people? (reverse coded)
	8. Think your life has been a failure?
	9. Feel fearful?
	10. Sleep restlessly?
	11. Feel happy? (reverse coded)
	12. Feel that you talked less than usual?
	13. Feel lonely?
	14. Feel that people were unfriendly?
	15. Feel you enjoyed life? (reverse coded)
	16. Have crying spells?
	17. Feel sad?
	18. Feel people disliked you?
	19. Feel that you could not “get going”?
Child depressive symptoms (adapted CES-D)	Responses: 0 (never), 1(almost never), 2(sometimes), 3 (often)
	1. Feel you had trouble keeping your mind on what you were doing?
	2. Feel depressed or very sad?
	3. Feel hopeful about the future?
	4. Feel bothered by things that don’t usually bother you?
	5. Not feel like eating because you felt upset about something?
	6. Feel that everything you did was an effort, or that everything was difficult for you to do?
	7. Feel scared or afraid?
	8. Toss and turn because you couldn’t sleep?
	9. Feel that you talked less than usual?
	10. Feel nervous or stressed?
	11. Feel lonely?
	12. Feel people disliked you?
	13. Feel you enjoyed life? (reverse coded)
Maternal intimate partner violence—perpetration (CTS)	Responses: 0 (Never), 1(once), 2(twice), 3(3–5 times), 4(6–10 times), 5(11–20 times), 6 (more than 20 times)
	1. Thrown something at partner?
	2. Pushed, grabbed or shoved partner?
	3. Slapped partner?
	4. Kicked, bit or hit partner with a fist?
	5. Hit or tried to hit partner with something?
	6. Beaten partner up?
	7. Choked partner?
	8. Threatened partner with a knife or gun?
	9. Used a knife or fired a gun at partner?
Maternal intimate partner violence—victimization (CTS)	Responses: 0 (Never), 1(once), 2(twice), 3(3–5 times), 4(6–10 times), 5(11–20 times), 6 (more than 20 times)
	1. Thrown something at you?

	2. Pushed, shoved or grabbed you?
	3. Slapped you?
	4. Kicked, bit or hit you with a fist?
	5. Hit or tried to hit you with something?
	6. Beaten you up?
	7. Choked you?
	8. Threatened you with a knife or gun?
	9. Used a knife or gun at you?
Maternal criminal behavior	Responses: 0 (No), 1 (Yes)
	1. Driven while under the in uence od drugs, beer, wine or liquor?
	2. Gone into or tried to go into a building to steal or damage something?
	3. Tried to buy or sell things that were stole, including illegal or bootleg copies of CDs and DVDs?
	4. Forged a check, used counterfeit money, or cashed or used bad checks on purpose?
	5. Stolen or tried to use a credit card, bank or ATM card, phone card, or account numbers so you could buy services or things for either yourself of someone else?
	6. Stolen or tried to steal a car or other motor vehicle?
	7. Tried to steal or actually stolen money or things worth $50 or less?
	8. Tried to steal or actually stolen money or things worth over $50?
	9. Shoplifted or taken something from a store on purpose?
	10. Sold marijuana, reefer, or weed?
	11. Sold hard drugs such as crack, heroin, cocaine, LSD or acid?
	12. Used a weapon or force to make someone give you money or things?
	13. Taken part in illegal gambling?
	14. Attacked someone with a weapon or with the idea of seriously hurting or killing them?

Table 3

Correlation matrix between maternal depressive symptoms and child depressive symptoms

	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.
1. Maternal depressive symptoms age 12	1											
2. Maternal depressive symptoms age 13	.58	1										
3. Maternal depressive symptoms age 14	.57	.61	1									
4. Maternal depressive symptoms age 15	.54	.55	.56	1								
5. Maternal depressive symptoms age 16	.50	.53	.58	.60	1							
6. Maternal depressive symptoms age 17	.47	.54	.50	.54	.55	1						
7. Child depressive symptoms age 12	.13	.07	.18	.11	.11	.11	1					
8. Child depressive symptoms age 13	.13	.11	.17	.06	.08	.07	.42	1				

	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.
9. Child depressive symptoms age 14	.19	.19	.26	.17	.13	.15	.47	.59	1			
10. Child depressive symptoms age 15	.18	.14	.25	.18	.12	.16	.44	.49	.60	1		
11. Child depressive symptoms age 16	.18	.14	.17	.18	.17	.13	.38	.44	.53	.60	1	
12. Child depressive symptoms age 17	.19	.15	.22	.15	.16	.22	.46	.46	.56	.65	.67	1

Table 4

Correlation matrix between all covariates averaged across child age

	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.	14.	15.
1. Maternal depressive symptoms	1														
2. Child depressive symptoms	.19	1													
3. Maternal substance use	.18	.04	1												
4. Maternal excessive alcohol use	.20	.04	.64	1											
5. Maternal drug use	.13	.03	.87	.03	1										
6. Maternal intimate partner violence	.18	.05	.13	.16	.07	1									
7. Maternal intimate partner victimization	.17	.05	.12	.14	.07	.80	1								
8. Maternal intimate partner perpetration	.16	.05	.12	.14	.06	.95	.58	1							
9. Maternal crime	.15	.01	.35	.37	.25	.11	.10	.10	1						
10. Mother age at birth	−.03	−.07	−.11	−.11	−.08	−.07	−.03	−.08	−.04	1					
11. Child lives with mother	−.04	−.04	−.07	−.05	−.05	−.08	−.06	−.08	−.03	.09	1				
12. Maternal financial stress	.26	.07	.11	.11	.08	.07	.06	.07	.05	−.05	−.02	1			
13. Black	−.13	−.04	−.08	−.11	−.04	.01	−.03	.03	.00	−.10	−.03	−.03	1		
14. Hispanic	.10	.03	−.05	−.00	−.06	.02	.06	−.00	−.03	−.06	.01	.03	−.48	1	
15. Mixed race	.01	−.02	.08	.06	.08	−.03	−.02	−.03	.01	.04	.01	−.03	−.53	−.13	1

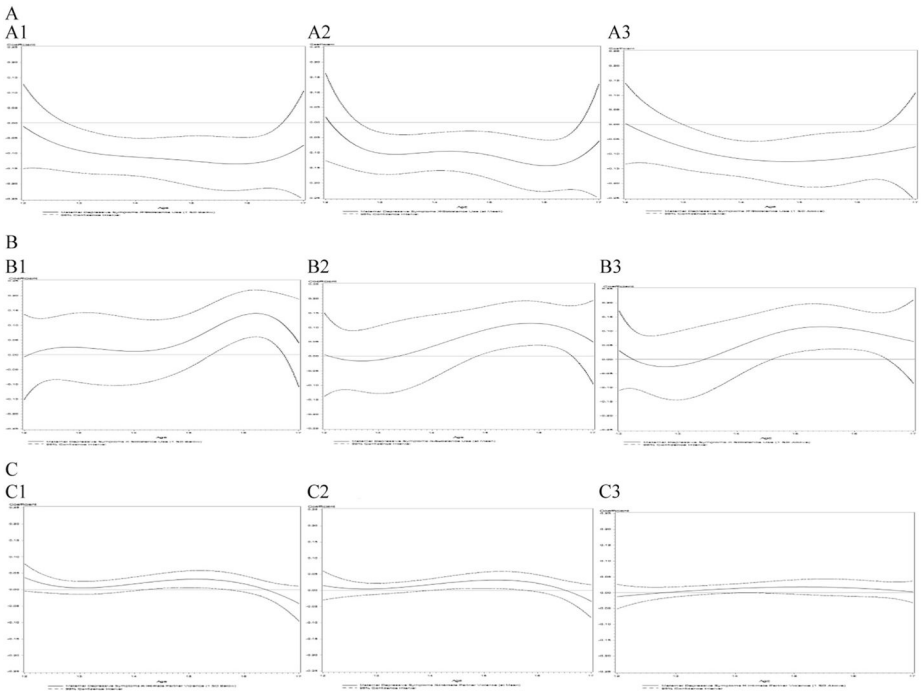

Fig. 9.

Probing significant interaction terms. These figures present models probing the significant interaction terms by presenting the figures of the interaction coefficients and 95% confidence intervals when covariates are centered at a one standard deviation below the mean, the mean, and one standard deviation above the mean. **a** Maternal depressive symptoms x maternal substance use among sons. **a1** Evaluated at 1 SD below mean, **a2**evaluated at mean, **a3** evaluated at 1 SD above mean. **b** Maternal depressive symptoms x maternal substance use among daughters. **b1** Evaluated at 1 SD below mean, **b2** evaluated at mean, **b3** evaluated at 1 SD above mean. **c** Maternal depressive symptoms x maternal intimate partner violence (IPV) among sons. **c1** Evaluated at 1 SD below mean, **c2** evaluated at mean, **c3** evaluated at 1 SD above mean

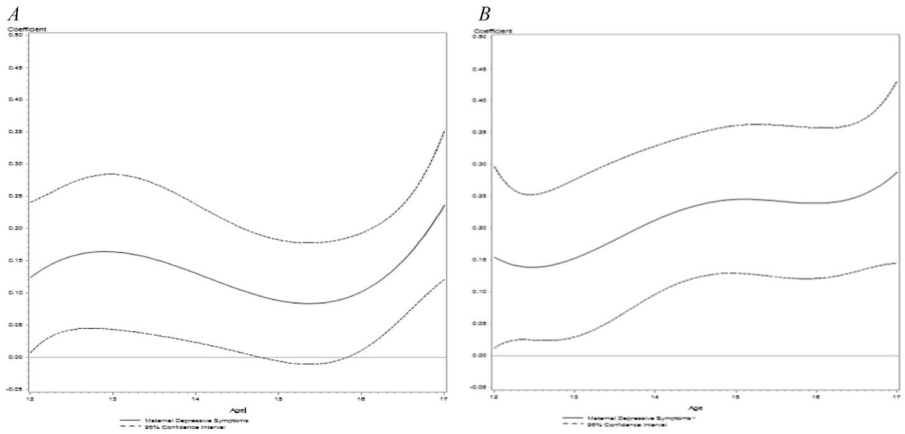

Fig. 1.

Effects of maternal depressive symptoms on child's depressive symptoms during adolescence by child gender. These figures present the coefficient curves and 95% confidence intervals for sons and daughters, respectively, across child ages 12–17. **a** Sons, **b** daughters

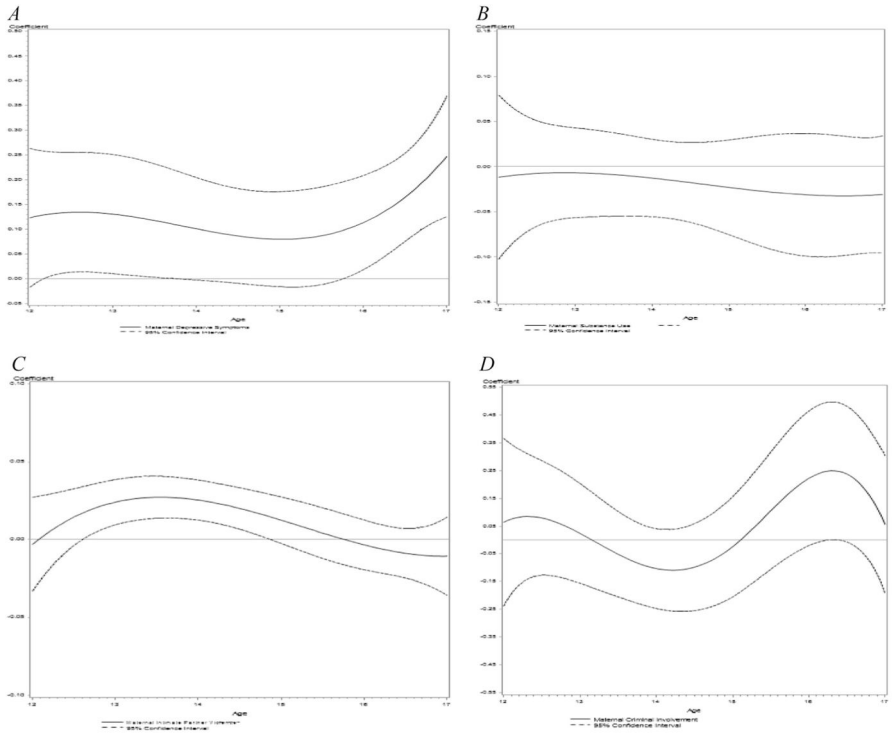

Fig. 2.

Effects of maternal health risk behaviors on son's depressive symptoms during adolescence. These figures present the coefficient curves and 95% confidence intervals for sons across ages 12–17 when accounting for maternal depressive symptoms and maternal co-morbid health risk behaviors. **a** Maternal depressive symptoms, **b** maternal substance use, **c** maternal intimate partner violence, **d** maternal criminal behavior

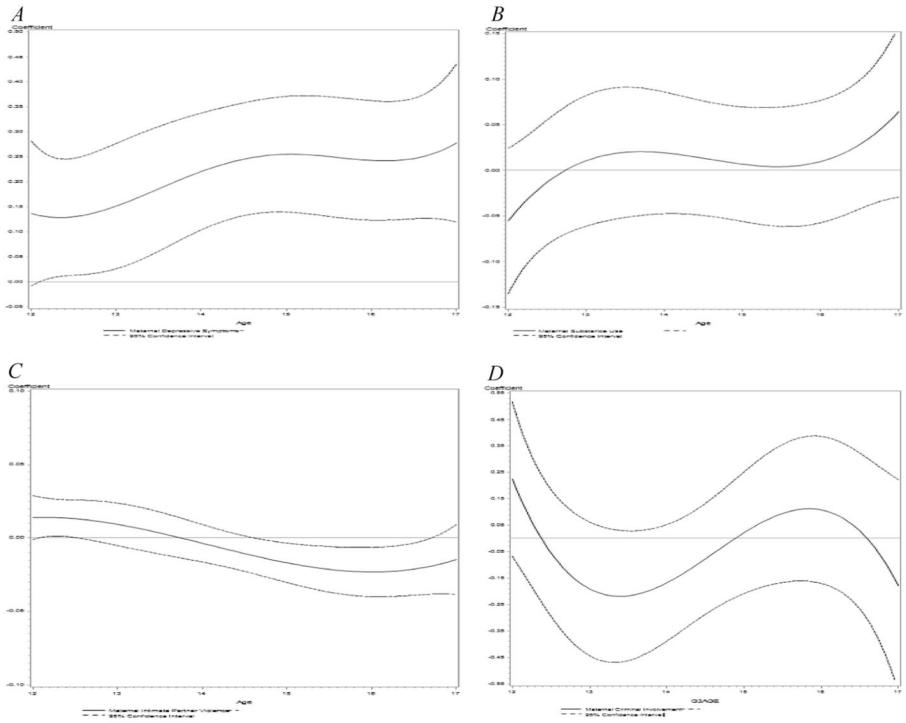

Fig. 3.

Effects of maternal health risk behaviors on daughter's depressive symptoms during adolescence. These figures present the coefficient curves and 95% confidence intervals for daughters across ages 12–17 when accounting for maternal depressive symptoms and maternal co-morbid health risk behaviors. **a** Maternal depressive symptoms, **b** maternal substance use, **c** maternal intimate partner violence, **d** maternal criminal behavior

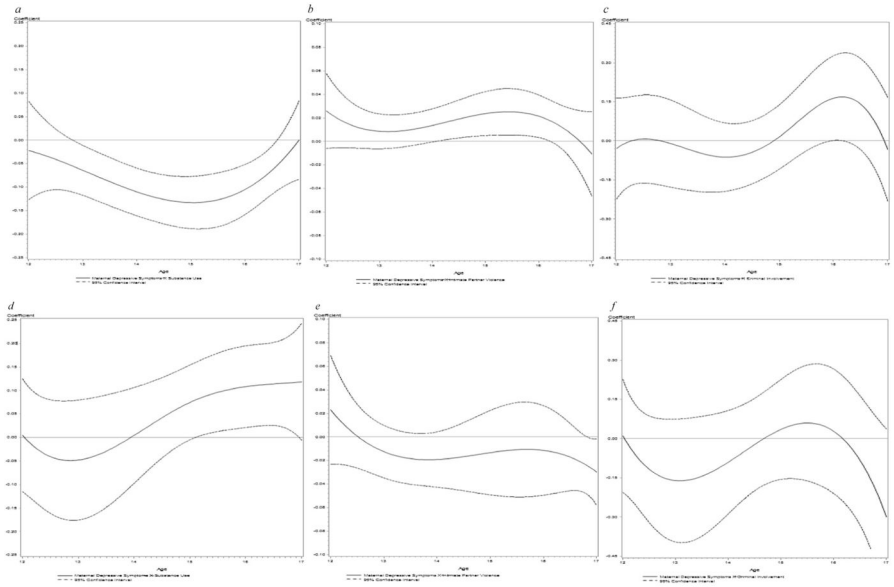

Fig. 4.

Interactive effects of maternal depressive symptoms and maternal co-morbid health risk behaviors on child's depressive symptoms. These figures present the coefficient curves and 95% confidence intervals for the interaction terms between maternal depressive symptoms and co-morbid health risk behaviors from child ages 12–17 by child gender. **a** Depressive symptoms × substance use among sons, **b** depressive symptoms × IPV among sons, **c** depressive symptoms × criminal behavior among sons, **d** depressive symptoms × substance use among daughters, **e** depressive symptoms × IPV among daughters, **f** depressive symptoms × criminal behavior among daughters

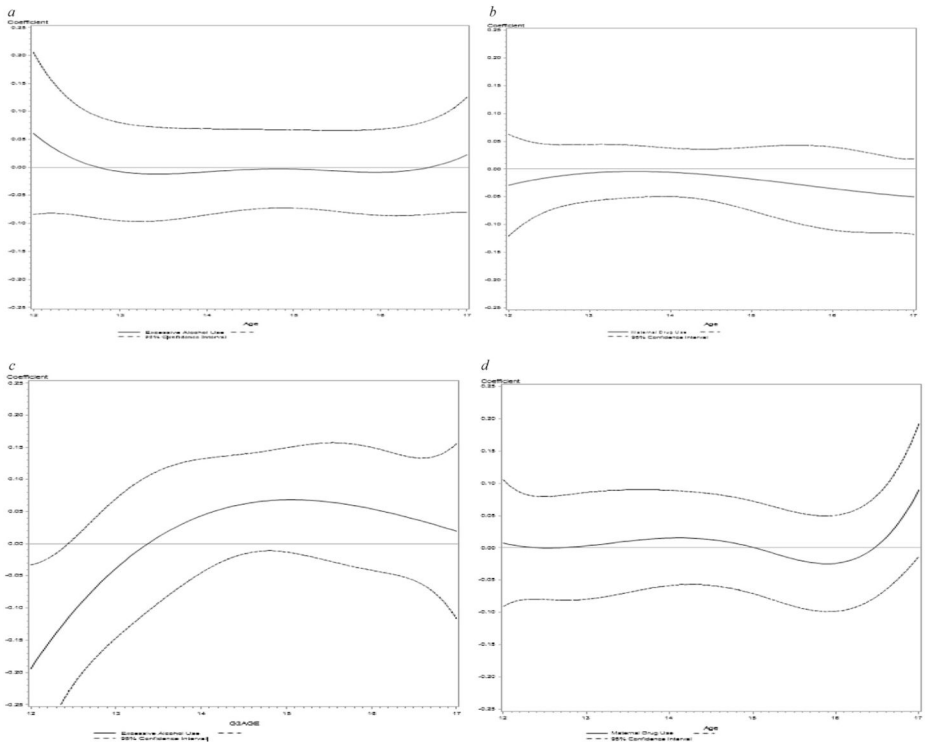

Fig. 5.

Effects of maternal excessive alcohol use and maternal drug use on child's depressive symptoms during adolescence. These figures present the coefficient curves and 95% confidence intervals when accounting for maternal depressive symptoms and maternal co-morbid health risk behaviors from child ages 12–17 by child gender. **a** Maternal alcohol use among sons, **b** maternal drug use among sons, **c** maternal alcohol use among daughters, **d** maternal drug use among daughters

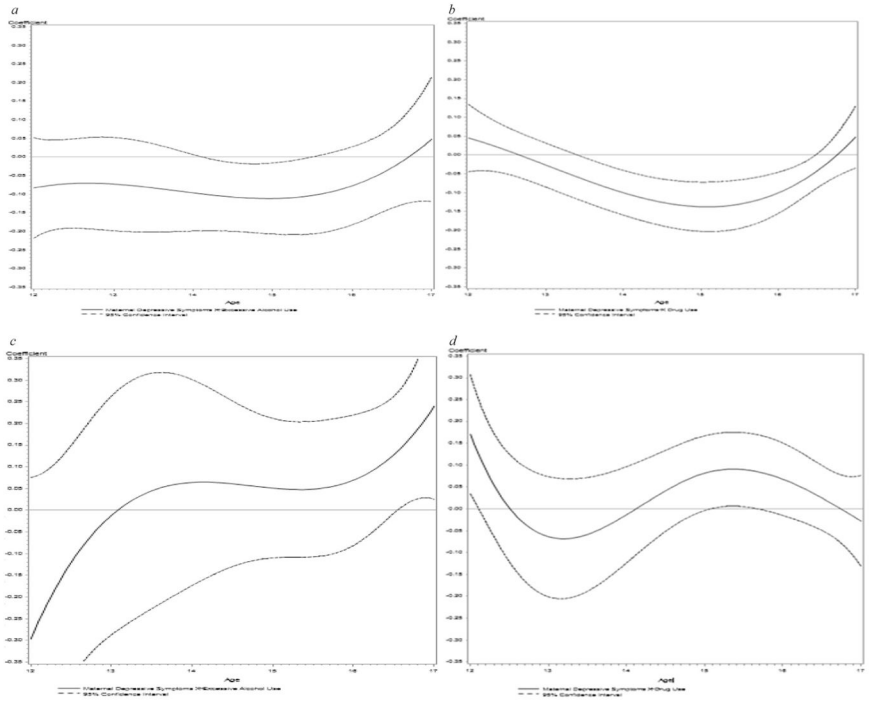

Fig. 6.

Interactive effects of maternal depressive symptoms and maternal excessive alcohol use and drug use on child's depressive symptoms. These figures present the coefficient curves and 95% confidence intervals for the interaction terms between maternal depressive symptoms and each type of maternal substance use from child ages 12–17 by child gender. **a** Maternal depressive symptoms × alcohol use among sons, **b** maternal depressive symptoms × drug use among sons, **c** maternal depressive symptoms × alcohol use among daughters, **d** maternal depressive symptoms × drug use among daughters

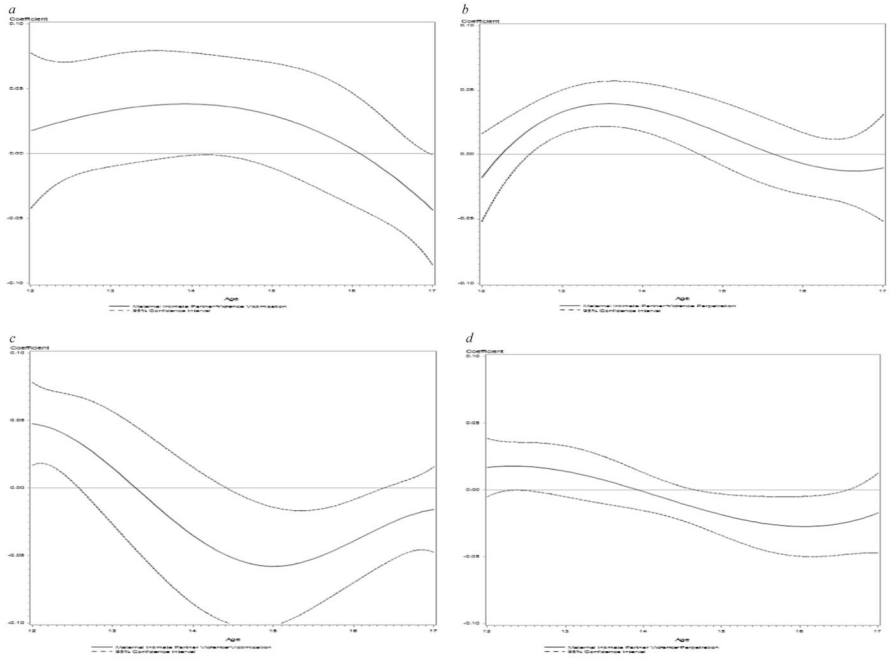

Fig. 7.

Effects of maternal intimate partner victimization and perpetration on child's depressive symptoms during adolescence. These figures present separate models estimating the coefficient curves and 95% confidence intervals for maternal intimate partner violence when accounting for maternal depressive symptoms and maternal co-morbid health risk behaviors from child ages 12–17 by child gender. **a** Maternal intimate partner violence victimization among sons, **b** maternal intimate partner violence perpetration among sons, **c** maternal intimate partner violence victimization among daughters, **d** maternal intimate partner violence perpetration among daughters

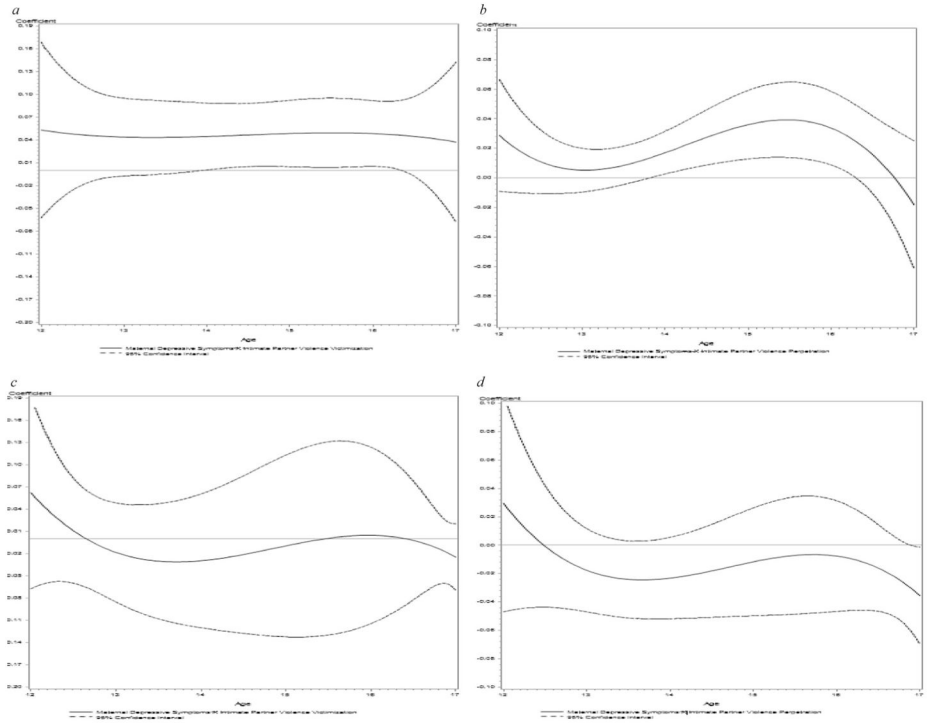

Fig. 8.

Interactive effects of maternal depressive symptoms and maternal intimate partner violence victimization and perpetration on child's depressive symptoms. These figures present separate models estimating the coefficient curves and 95% confidence intervals for the interaction terms between maternal depressive symptoms and each type of involvement in intimate partner violence from child ages 12–17 by child gender. **a** Maternal depressive symptoms × IPV victimization among sons, **b** maternal depressive symptoms × IPV perpetration among sons, **c** maternal depressive symptoms × IPV victimization among daughters, **d** maternal depressive symptoms × IPV perpetration among daughters

Table 1

Descriptive statistics (n = 413)

	Mother–son dyads n = 210			Mother–daughter dyads n = 203		
	Range	**Mean/Proportion**	**SD**	**Range**	**Mean/Proportion**	**SD**
Maternal depressive symptoms	0–4	0.85	0.55	0–4	.80	.62
Child depressive symptoms	0–3	0.81	0.63	0–3	1.10	.64
Moderators						
Maternal substance use	0–496	10.03	50.86	0–585	13.65	61.06
Maternal intimate partner violence	0–37	0.78	3.29	0–44	.875	3.87
Maternal crime	0,1	0.12	–	0,1	.08	–
Control variables						
Mother age at birth	10–32	18.63	2.90	13.2–30	18.89	2.96
Child lives with mother	0.1	.94	–	0.1	.97	–
Maternal financial stress	0–3	.40	.80	0–3	.33	.74
Black	0.1	.64	–	0.1	.68	–
Hispanic	0.1	.11	–	0.1	.07	–
Mixed race	0.1	.16	–	0.1	.13	–

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