

Maternal depression and co-occurring antisocial behaviour: testing maternal hostility and warmth as mediators of risk for offspring psychopathology

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Background: Disruption in the parent–child relationship is a commonly hypothesized risk factor through which maternal depression may increase risk for offspring psychopathology. However, maternal depression is commonly accompanied by other psychopathology, including antisocial behaviour. Few studies have examined the role of co-occurring psychopathology in depressed mothers. Using a longitudinal study of offspring of mothers with recurrent depression, we aimed to test whether maternal warmth/hostility mediated links between maternal depression severity and child outcomes, and how far direct and indirect pathways were robust to controls for co-occurring maternal antisocial behaviour. **Methods:** Mothers with a history of recurrent major depressive disorder and their adolescent offspring (9–17 years at baseline) were assessed three times between 2007 and 2010. Mothers completed questionnaires assessing their own depression severity and antisocial behaviour at Time 1 (T1). The parent–child relationship was assessed using parent-rated questionnaire and interviewer-rated 5-min speech sample at Time 2 (T2). Offspring symptoms of depression and disruptive behaviours were assessed using the Child and Adolescent Psychiatric Assessment at Time 3 (T3). **Results:** Maternal hostility and warmth, respectively, mediated the association between maternal depression severity and risk for offspring psychopathology. However, the effects were attenuated when maternal antisocial behaviour was included in the analysis. In tests of the full theoretical model, maternal antisocial behaviour predicted both maternal hostility and low warmth, maternal hostility predicted offspring disruptive behaviour disorder symptoms, but not depression, and maternal warmth was not associated with either child outcome. **Conclusions:** Parenting interventions aimed at reducing hostility may be beneficial for preventing or reducing adolescent disruptive behaviours in offspring of depressed mothers, especially when depressed mothers report co-occurring antisocial behaviour. **Keywords:** Hostility, warmth, mediation, depression, antisocial behaviour, disruptive behaviour.

Introduction

Maternal depression is a well-established, common and potent risk for offspring depression and disruptive behaviour disorder (DBD) (Brennan, Hammen, Katz, & Le Brocque, 2002; Wickramaratne & Weissman, 1998). Genetically sensitive designs suggest heritable factors only partially account for intergenerational transmission of risk, and that noninherited factors also have an important role (Harold, Elam, Lewis, Rice, & Thapar, 2012; Harold et al., 2010; Lewis, Rice, Harold, Collishaw, & Thapar, 2011; Silberg, Maes, & Eaves, 2010; Tully, Iacono, & McGue, 2008). Mothers who suffer from depression are reported to show more hostility and less warmth compared with healthy controls; such difficulties in parenting may be one mechanism through which maternal depression exerts effects on offspring (Goodman & Gotlib, 1999; Marmorstein & Iacono, 2004; McCarty & McMahon, 2003). What is not well understood is the extent to which difficulties in

parenting reflect parental depression per se or other commonly co-occurring associated maternal psychopathology, such as antisocial behaviour (ASB).

Hostility and warmth are distinct constructs of the mother–child relationship, with hostility being defined as maternal anger, criticism, negativity and disapproval directed towards the child. In contrast, warmth is defined as supportive parenting and positive maternal affect whereby the mother demonstrates affection and interest in their child (Connor & Rueter, 2006; Rueter & Conger, 1998). Although distinct constructs of the mother–child relationship, they are also correlated (Rueter & Conger, 1998). Because the mother–child relationship is potentially modifiable, greater understanding of the role of the mother–child relationship as a risk mechanism for offspring of mothers with depression could inform intervention and prevention strategies.

Disruptions in the mother–child relationship have been found to mediate the association between maternal depression severity and offspring depression (Davies & Windle, 1997) and externalizing problems (McCarty & McMahon, 2003). However,

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not all studies support the mediating role of parenting on risk for child psychopathology (Frye & Garber, 2005; Keenan-Miller, Hammen, & Brennan, 2010). Maternal ASB has also been associated with increased risk for offspring psychopathology, particularly disruptive behaviours (Harold et al., 2010, 2012; Thornberry, Freeman-Gallant, Lizotte, Krohn, & Smith, 2003). There is consistent evidence that this association may be mediated, in part, by disruptions in the mother-child relationship (Harold et al., 2010, 2012; Hovee et al., 2009).

Maternal depression often co-occurs with additional psychopathology, including ASB, which is rarely taken into account despite evidence that the presence of multiple forms of psychopathology in mothers with depression confers heightened risk of psychiatric disorder in offspring (Sellers et al., 2012). Furthermore, there is preliminary evidence that depressed mothers with co-occurring ASB provide poorer quality care-giving environments compared with mothers with depression alone (Kim-Cohen, Caspi, Rutter, Tomas, & Moffitt, 2006). Therefore, co-occurring ASB in mothers with recurrent depression may account for the association between maternal depression and mother-child relationship difficulties. We are not aware of any previous studies assessing the unique effect of both maternal depression and co-occurring ASB symptoms simultaneously on child outcomes via the mother-child relationship.

To address these issues, this study employed data from a high-risk longitudinal study of adolescent offspring of mothers with recurrent depression using a multiinformant design, to consider the role of maternal warmth and hostility. We hypothesized that maternal warmth and hostility would mediate the effects of maternal depression severity on child DBD; and that direct and indirect effects of maternal depression severity would be attenuated when adjusting for co-occurring maternal ASB.

Methods

Participants

The current analyses utilized data from the 'Early Prediction of Adolescent Depression' (EPAD) study, a study of the offspring of parents with recurrent depression. The sample and procedure have been detailed elsewhere (Mars et al., 2012; Sellers et al., 2012), and are therefore described briefly.

Parents were recruited predominantly from primary care in South Wales, UK on the basis of treatment for at least two episodes of depression (confirmed at interview). Families were excluded if the depressed parent was biologically unrelated to the child, or the child had moderate-to-severe intellectual disability (IQ < 50).

The sample at baseline consisted of 337 parents with recurrent depression and their adolescent offspring. Two families were excluded due to a bipolar

diagnosis in the affected parent after the first assessment. Fourteen families were excluded as the child was not living at home throughout the study. Depressed fathers were also excluded ($n = 22$).

The eligible sample in this study thus consisted of 299 mothers at baseline (age $M = 41.2$ years, range 26–55 years) and their children (age $M = 12.3$, range 9–17 years; 58.2% females). T2 assessments were conducted approximately 16 months later ($SD = 2.64$). T3 assessments took place a further 12 months later ($SD = 1.48$). Retention rates were high (95.6% at T2 and 93.3% at T3; 91.3% participated in all assessments). Families were interviewed at their home (99%) or University Hospital of Wales, and provided informed consent (assent if child under 16 years). Ethical approval was provided by the Multi-Regional Ethics Committee.

Measures

Maternal depression. Maternal diagnoses of recurrent depression were confirmed at baseline using a life history calendar approach (Belli, 1998; Caspi et al., 1996) and the Schedule for Clinical Assessment in Neuropsychiatry (Wing et al., 1990). In addition, mothers completed the Beck Depression Inventory (BDI) (Beck, Rush, Shaw, & Emery, 1979) to assess the severity of depression symptoms at T1 ($\alpha = .91$).

Antisocial behaviour. Mothers reported on their own current ASB at T1 using the 34-item Adult Self Report (ASR) questionnaire (Achenbach & Rescorla, 2003) with items coded 0 (absent), 1 (somewhat or sometimes true) or 2 (very true). The item 'I have never been arrested' was excluded as it was the only item referring to lifetime behaviour ($\alpha = 0.75$).

Child psychopathology. Mother and child versions of the Child and Adolescent Psychiatric Assessment (Angold & Costello, 2000) were used to assess child symptoms of depression and DBD (oppositional deviant disorder/conduct disorder) in the preceding 3 months. The total number of DSM-IV symptoms (for each disorder) in the child at T3 was calculated by combining mother and child reports; where a symptom was reported by mother *or* child, the symptom was considered as present. Symptom totals were created for child depression and DBD. Interrater reliabilities for child symptoms were excellent (average $\kappa = .94$).

Maternal warmth and hostility. Maternal warmth and hostility (T2) were assessed by two methods; a self-report questionnaire measure (Iowa Youth and Families project (IYFP) family interaction rating scales) (Melby et al., 1993) and an interviewer-rated 5-min speech sample of expressed emotion (EE) (Caspi et al., 2004).

Mothers completed a 10-item questionnaire (IYFP) containing two subscales: hostility (4-items) and lack

of warmth (6-items) towards the child. Each item was coded 1–7 with higher scores indicating increased hostility or low warmth (hostility: $M = 12.42$, $SD = 4.20$, $\alpha = .89$. Warmth: $M = 12.61$; $SD = 6.80$ $\alpha = .93$). Warmth items were reverse coded, so that higher scores were indicative of higher warmth.

A measure of EE was also used (Caspi et al., 2004). Mothers were asked to describe their child and encouraged to speak freely. Prompt questions were utilized to elicit responses. Two additional prompts were included (e.g. ‘What are your child’s strengths and good points?’). Interviews were recorded and coded by the researchers who had undergone in-house training supervised by a consultant child psychiatrist. This study utilized the global measures of warmth and hostility (each coded 0–5). High scores indicated high hostility or warmth (hostility: $M = .78$, $SD = .90$. Warmth: $M = 3.49$; $SD = .98$). Thirty interviews were randomly selected to assess interrater reliability and were coded by a second interviewer, blind to the initial coding. Ratings were compared between the first and second rater. Interrater reliability for warmth and hostility showed substantial agreement [warmth intraclass correlation (ICC) = 0.69; hostility ICC = 0.79].

The two measures of warmth were moderately correlated ($r = .30$, $p \leq .001$), as were those of hostility ($r = .36$, $p \leq .001$). A composite score of maternal warmth and a composite score of maternal hostility were created from the IYFP and EE. The subscales of hostility and warmth from each measure of the mother–child relationship were first standardized using z-score transformations and then added together: the z-scores of hostility from each measure of the mother–child relationship were added together to create a composite measure of maternal hostility as were the z-scores of warmth.

Analysis strategy

To test whether the mother–child relationship served as an indirect mechanism between maternal psychopathology and offspring symptoms of depression and DBD, the association between maternal depression severity or ASB (T1) and offspring psychopathology (symptoms of depression and DBD) at T3 was initially examined. Analyses then examined indirect associations among maternal depression severity (T1), maternal hostility and warmth (T2) and offspring symptoms of DBD and depression (T3), after adjusting for maternal ASB (T1).

Examination of indirect pathways (mediating pathways) was assessed according to criteria outlined by MacKinnon and colleagues (MacKinnon, Krull, & Lockwood, 2000; MacKinnon, Lockwood, Hoffman, West, & Sheets, 2002). If the independent and dependent variables are each related to the proposed intervening variable, the significance of the indirect association between the independent and dependent variables can then be assessed statisti-

cally (MacKinnon, Lockwood, & Williams, 2004). Indirect effects were estimated with bias-corrected bootstrapping and were considered significant if the 95% confidence intervals did not include zero (Hayes, 2009; Preacher & Hayes, 2008). Bias-corrected bootstrapping is a robust way of testing indirect effects (MacKinnon et al., 2004).

Families with missing data were more likely to have higher offspring depression and DBD symptoms at baseline, and higher maternal depression severity and ASB scores (Table S1). The Little’s test of missing data indicated that data were not missing completely at random ($\chi^2(39) = 71.36$, $p = .001$) (Little, 1988). The fraction of missing data varied from 1.7% (maternal ASB) to 31.2% (maternal hostility/warmth). Multiple imputation with data augmentation was used to generate values for missing data with variables relevant to the final model using NORM 2.03 (Schafer, 1997, 1999), regarded as the most robust multiple imputation method (Allison, 2001). Ten derived data sets were generated and subsequently tested within Mplus 6.12 (Muthén & Muthén, 1998).

Results

Correlational analysis

Means, standard deviations and intercorrelations of the study variables are presented in Table 1. Mothers’ depression severity was associated with maternal symptoms of ASB ($r = .43$, $p \leq .001$) at T1, with maternal hostility ($r = .26$, $p \leq .001$) and warmth ($r = -.17$, $p \leq .05$) at T2, and with offspring symptoms of depression ($r = .14$, $p \leq .05$) and DBD ($r = .19$, $p \leq .001$) at T3. Maternal hostility was associated with offspring depression and DBD symptoms ($r = .18$, $p \leq .01$ and $r = .51$, $p \leq .001$ respectively). Maternal warmth was associated with offspring DBD symptoms ($r = -.24$, $p \leq .001$), but not offspring depression ($r = -.13$, $p \geq .05$).

Analysis of indirect pathways

Offspring DBD. As noted, a significant association was observed between maternal depression severity (T1) and child symptoms of DBD at T3 ($\beta = .17$, $p = .01$). A significant association was also observed between maternal ASB (T1) and child DBD symptoms at T3 ($\beta = .15$, $p = .02$).

Analysis of indirect effects (Figure 1, panel a) revealed a significant indirect association between depression severity and child DBD via hostility (standardized 95% bias-corrected bootstrapped CI = .039, .197). However, after adjusting for maternal ASB, this indirect association was no longer significant (standardized 95% bias-corrected bootstrapped CI = -.006, .146). Rather, an indirect association was apparent between maternal ASB and child DBD via maternal hostility (Figure 1, panel b; standardized 95% bias-corrected bootstrapped CI = .088, .226).

Table 1 Intercorrelations, means and standard deviations for study variables

	1	2	3	4	5	6
1. T1 maternal depression severity	–					
2. T1 maternal antisocial behaviour	.43**	–				
3. T2 maternal hostility ^a	.26**	.37**	–			
4. T2 maternal warmth ^a	–.17*	–.27**	–.41**	–		
5. T3 child depression symptoms	.14*	.04	.18*	–.13	–	
6. T3 child disruptive behaviour symptoms	.19**	.15*	.51**	–.24**	.39**	–
Mean	16.41	7.70	–.01	.04	1.99	3.01
Standard deviation	(11.19)	(4.98)	(1.63)	(1.57)	(2.11)	(2.66)

^aStandardized z-scores of EE and IYPF measures of the parent–child relationship.

* $p < .05$; ** $p < .001$.

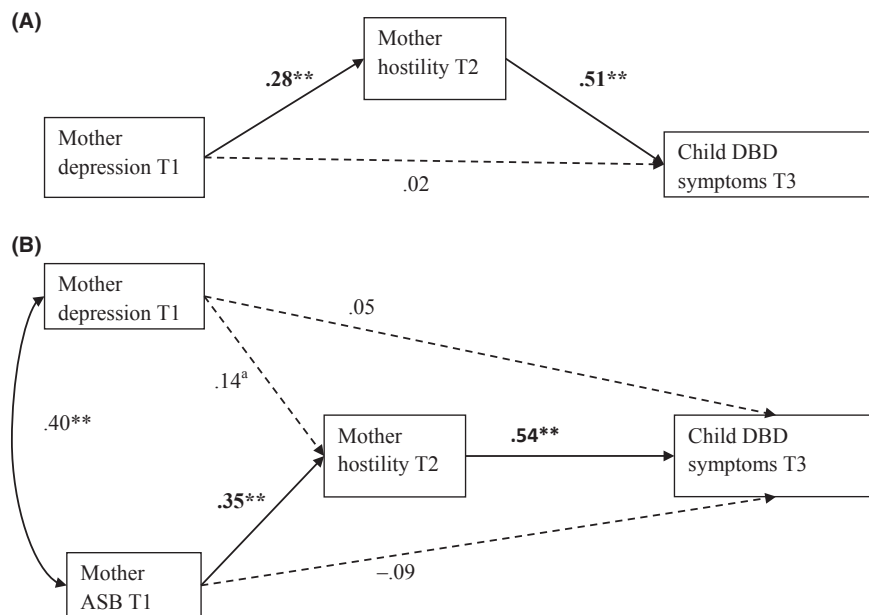


Figure 1 Mediating model showing the effect of maternal depression severity on offspring disruptive behaviour disorder (DBD) via maternal hostility (panel a) after adjusting for maternal antisocial behaviour (ASB) (panel b). ** $p \leq .001$; * $p \leq .05$; † $p \leq .10$

When indirect effects via maternal warmth were examined (Figure 2, panel a), there was a significant indirect association via warmth (standardized 95% bias-corrected bootstrapped CI = .001, .090). After adjusting for maternal ASB, there was no additional direct or indirect association between maternal depression severity and child DBD (standardized 95% bias-corrected bootstrapped CI = –.018, .056). Instead, the indirect association between maternal ASB and offspring DBD via maternal warmth (Figure 2, panel b) was significant (standardized 95% bias-corrected bootstrapped CI = .016, .117).

Offspring depression. Maternal depression severity (T1) was associated with offspring depression symptoms at T3 ($\beta = .14$, $p = .04$). Maternal ASB (T1) was not significantly associated with child depression at T3 ($\beta = .04$, $p = .59$). The direct association between maternal depression severity and child depression symptoms was attenuated when maternal hostility was considered (Figure 3 panel a), and revealed a significant indirect effect via hostility (standardized 95% bias-corrected bootstrapped CI = .001, .082).

However, after adjusting for maternal ASB, there were no direct or indirect effects via parenting of maternal depression severity (standardized 95% bias-corrected bootstrapped CI = –.007, .066) on child depression symptoms (Figure 3, panel b). There was, however, a significant indirect association between maternal ASB and offspring depression via maternal hostility (standardized 95% bias-corrected bootstrapped CI = .014, .143; Figure 3, panel b).

Combined model. Given the significant associations between maternal warmth and hostility and between child depression and DBD (Table 1), a final model assessed unique influences of each construct of the mother–child relationship (hostility and warmth) on each child outcome when considered simultaneously (Figure 4). Maternal depression severity did not significantly predict either warmth or hostility, nor were there any significant direct associations between maternal depression severity and child symptoms of depression or DBD. Maternal ASB symptoms were associated with both warmth and

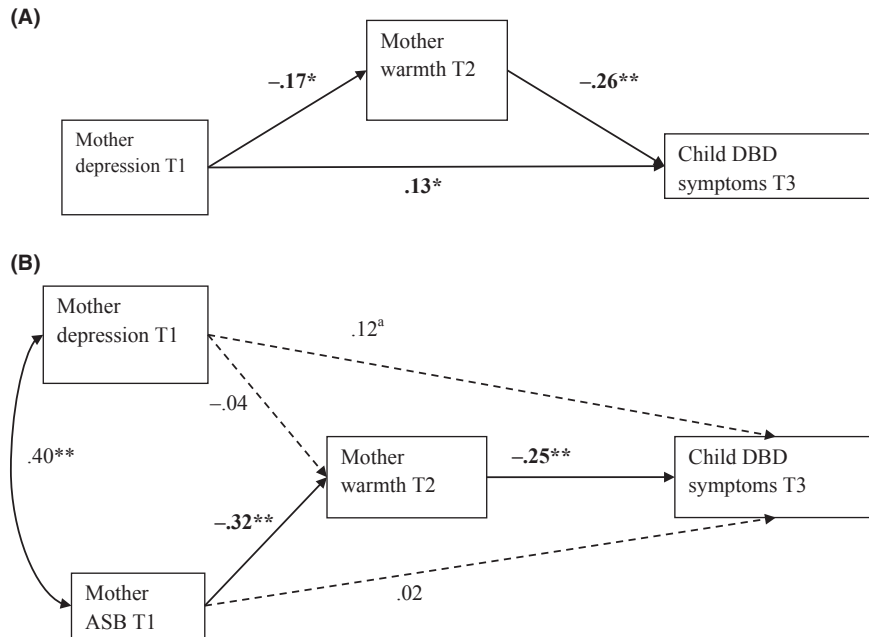


Figure 2 Mediating model showing the effect of maternal depression severity on offspring disruptive behaviour disorder (DBD) via maternal warmth (panel a) after adjusting for maternal antisocial behaviour (ASB) (panel b). ** $p \leq .001$; * $p \leq .05$; $\dagger p \leq .10$

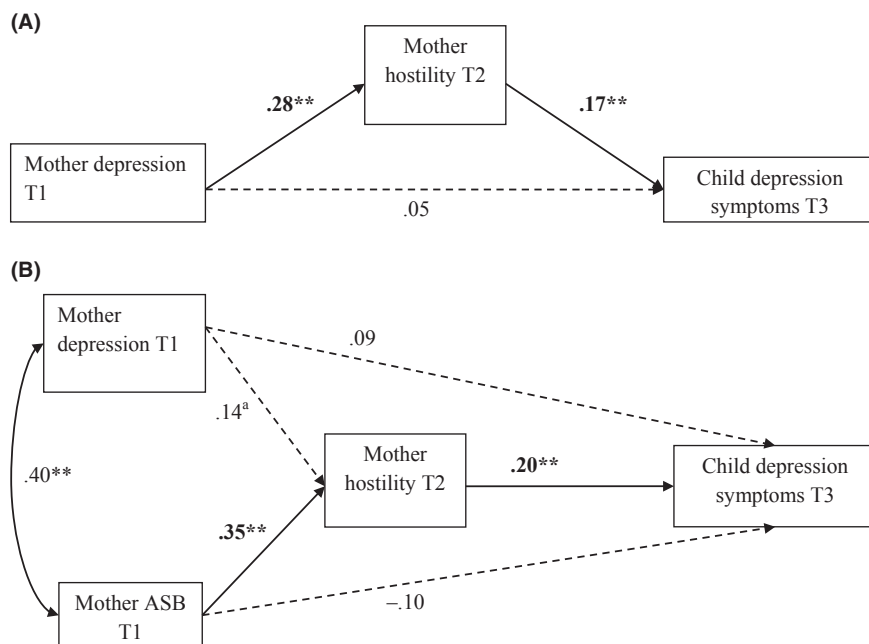


Figure 3 Mediating model showing the effect of maternal depression severity on offspring depression via maternal hostility (panel a) after adjusting for maternal antisocial behaviour (ASB) (panel b). ** $p \leq .001$; * $p \leq .05$; $\dagger p \leq .10$

hostility. Maternal hostility was a significant predictor of child symptoms of DBD, but not depression.

Given findings of nonsignificant paths from maternal warmth to either child outcome when maternal hostility was included in the model, a competing model constrained pathways from maternal warmth to child symptomatology to zero. Model fit statistics indicated a good fit to the data ($\chi^2(2) = 1.87$, $p = .393$; CFI = .99; RMSEA = .05).

We conducted post hoc exploratory subgroup comparisons using stacked modelling procedures (Bollen, 1989) to assess whether estimates differed significantly for boys and girls in relation to the final

model (Figures S1 and S2). Maternal ASB symptoms were associated with both warmth and hostility, and maternal hostility, in turn, was a significant predictor of child symptoms of DBD for both boys and girls. Maternal hostility predicted depression symptoms more strongly in boys than girls, as did maternal depression symptoms ($p \leq .05$). Maternal depression symptoms also predicted maternal hostility more strongly in boys than girls ($p \leq .05$). However, caution should be applied to the present pattern of findings as our sample size became small for subgroup comparisons.

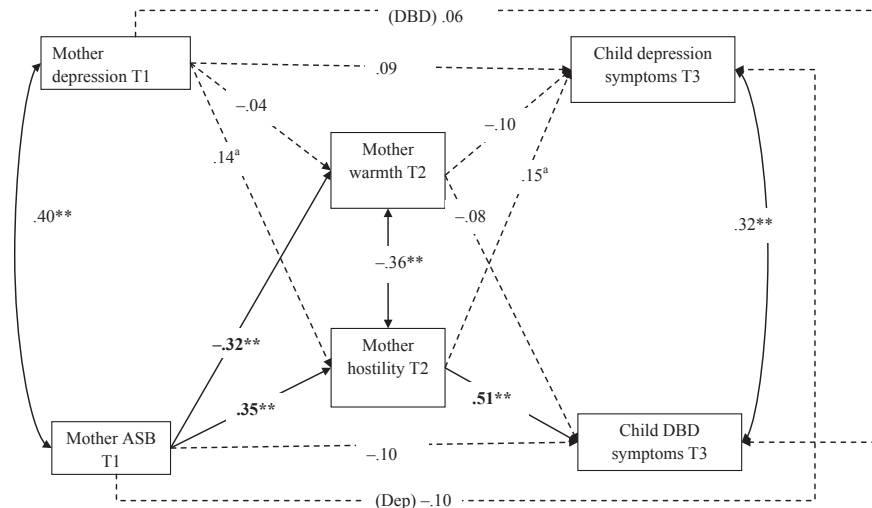


Figure 4 Estimated pathways between maternal depression and antisocial behaviour (ASB), constructs of the parent-child relationship (warmth and hostility) and child symptoms of depression and disruptive behaviour disorder (DBD). ** $p \leq .001$; * $p \leq .05$; † $p \leq .10$

Discussion

Maternal depression is associated with markedly increased risk for depression and DBD in offspring (Brennan et al., 2002; Wickramaratne & Weissman, 1998), and impaired mother-child relationships have been proposed as a possible mechanism for risk transmission (Davies & Windle, 1997; McCarty & McMahon, 2003). What has been unclear is the extent to which risk mechanisms are linked to co-occurring maternal antisocial behaviour or maternal depression per se.

Consistent with previous research, variation in maternal depression severity in this high-risk sample was associated with offspring depression and DBD symptoms (Goodman & Gotlib, 1999; Keenan-Miller et al., 2010; Marmorstein & Iacono, 2004; McCarty & McMahon, 2003). Mothers in this study also experienced additional psychopathology including ASB (Sellers et al., 2012), and mothers with increased depression severity had higher levels of ASB. When co-occurring ASB was taken into account, direct and indirect effects (via the mother-child relationships) of depression severity on child outcomes were attenuated. These findings are consistent with evidence that depressed mothers with co-occurring ASB symptoms may provide especially poor quality care-giving environments (Kim-Cohen et al., 2006), but provide additional evidence that depression severity alone may have less influence on child outcomes via mother-child relationships than previously thought (at least within high-risk clinical samples).

The final model in this study considered both maternal hostility and warmth and child symptoms of depression and DBD simultaneously. Findings here indicated that maternal ASB was especially predictive of offspring DBD symptoms and that associations were primarily mediated via maternal hostility. Low maternal warmth appeared to repre-

sent less of a risk for offspring DBD symptoms when hostility was accounted for. Influences of maternal ASB and hostility on child depression appeared less robust when child DBD was accounted for. These findings build on previous research suggesting that negative (rather than positive) aspects of parenting may have a stronger effect on risk for offspring psychopathology (Lovejoy, Graczyk, O'Hare, & Neuman, 2000), and that effects are likely to be stronger for offspring conduct disorder than internalizing problems (Combs-Ronto, Olson, Lunkenheimer, & Sameroff, 2009; McCarty & McMahon, 2003; Stubbé, Zahner, Goldstein, & Leckman, 1993). This study advances our understanding of intergenerational risk transmission in the context of maternal depression by confirming similar patterns in these high-risk dyads, but also highlights that parenting problems are not an inevitable consequence of depression. Instead parent-child mediating mechanisms, in particular hostility, may be better understood in the context of additional ASB that can co-occur with maternal depression.

Gender effects observed in this study were intriguing and will require further investigation. These results add to findings suggesting that specific family relationship factors, such as hostile parenting practices and interparental conflict, may have distinct influences on boys compared with girls during the period of adolescence (Grych, Harold, & Miles, 2003; Harold & Conger, 1997). However, other studies suggest that this pattern may be reversed during earlier stages of development (e.g. Lewis et al., 2011). Yet, other studies fail to find a moderating effect of child gender in young children (McCarty & McMahon, 2003). Further research is therefore needed to clarify these inconsistent findings, and to further examine the moderating effect of child gender across different developmental periods. In addition, caution should also be applied

to the present pattern of findings as our sample size became small for subgroup comparisons.

Findings should also be considered in light of certain limitations. First, although retention was excellent given the high-risk nature of the sample, families with missing data reported higher maternal ASB and depression severity scores, as well as higher child depression and DBD symptoms at baseline compared with those with complete data. Second, although this study was able to investigate risk factors for offspring psychopathology across adolescence, firm conclusions about effects at particular points during this developmental period are not possible due to the wide age range of children in the study. Third, there were too few fathers to consider the effects of paternal depression. Research suggests that depression in fathers has a significant and deleterious effect on relationships and child psychopathology (Connell & Goodman, 2002; Kane & Garber, 2004; Wilson & Durbin, 2010) and future research should test effects of paternal depression and co-occurring ASB on parent–child relationships. Future research should also consider that mothers with depression often present with other clinical problems including anxiety and substance misuse (Swendsen & Merikangas, 2000) that could potentially influence maternal hostility and warmth and offspring psychopathology. Finally, conclusions regarding causal direction require examination of possible effects of child symptomatology on parenting or maternal psychopathology. Transactional models of mutual influence between parents and children (Bell, 1968; Sameroff, 1975) have long been proposed with evidence that ASB is learnt through coercive patterns of social interactions (Granic & Patterson, 2006; Patterson, DeBaryshe, & Ramsey, 1989). Future research should therefore consider the possibility of child effects on parenting and on the course of parent psychopathology. Future research should also consider alternative pathways that may increase risk for psychopathology in high-risk offspring of mothers with recurrent depression including the presence of an antisocial father, parental discord and parental monitoring, as well as peer relations (Jaffee, Moffitt, Caspi, & Taylor, 2003; Kerr, Stattin, & Burk, 2010; Shelton & Harold, 2008).

Maternal depression is a well-documented and clinically significant risk for offspring depression and DBD (Brennan et al., 2002; Wickramaratne & Weissman, 1998). Evidence on mechanisms of risk transmission in high-risk families affected by recurrent maternal depression is important for informing the development and effective targeting of intervention and prevention programmes. There are a number of specific implications highlighted by this study in this regard. First, many intervention efforts have focused on treating parents' depressive illnesses (Weissman et al., 2006). Greater attention to

co-occurring parental problems, including ASB, in mothers with depression may yield additional benefits in the prevention of offspring psychopathology. Second, the majority of studies have focused on the prevention of offspring depression. This study highlights robust links with DBD in this sample. DBD is one of the most common reasons for referral of children to mental health services; offspring DBD is also an important risk factor for future depressive disorders (Silberg et al., 2010). Therefore, intervention/prevention strategies need to focus on risk mechanisms for DBD symptoms as well as depression in offspring of parents with recurrent depression. Third, the current findings suggest that parenting interventions reducing maternal hostility in mothers with depression may be especially beneficial for preventing or reducing disruptive behaviour problems in offspring, especially when depressed mothers also exhibit ASB. There is strong evidence that parenting interventions can effectively reduce disruptive behaviour problems in children and adolescents (Woolfenden, Williams, & Peat, 2001). These interventions may be of particular importance for families where a mother suffers from depression and also exhibits ASB.

Supporting information

Additional Supporting Information may be found in the online version of this article:

Figure S1 Estimated pathways between maternal depression and ASB, constructs of the parent–child relationship (warmth and hostility) and child symptoms of depression and DBD in male offspring.

Figure S2 Estimated pathways between maternal depression and ASB, constructs of the parent–child relationship (warmth and hostility) and child symptoms of depression and DBD in female offspring.

Table S1 Sample descriptive of complete and missing data at each follow-up.

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Key points

- Maternal depression is associated with disruptions in the mother–child relationship, which may negatively impact on child symptomatology. Mothers with recurrent depression can also present with antisocial behaviours, which could explain the association.
- Disruptions in the mother–child relationship mediated the association between maternal depression severity and risk for offspring symptomatology. However, maternal depression severity no longer predicted warmth or hostility after adjusting for maternal antisocial behaviour.
- In the final model, maternal antisocial behaviour predicted maternal hostility and warmth. Maternal hostility predicted offspring disruptive behaviours.
- Interventions aimed at reducing maternal hostility may help reduce risk for offspring disruptive behaviours, particularly where mothers also present with additional antisocial behaviour.

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