Title: Does father-child conflict mediate the association between fathers’ postnatal depressive symptoms and children’s adjustment problems at 7 years old?

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Word count: 5,531

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Financial support: This research received no specific grant from any funding agency, commercial or not-for-profit sectors.
**Background:** Paternal depressive symptoms are associated with children’s emotional and behavioural problems, which may be mediated by negative parenting. But there is no research on the influence of paternal depressive symptoms on children’s emotion regulation and limited literature investigating fathers’ parenting as a mediator in the pathway between paternal depressive symptoms and children’s externalising and internalising problems. We aimed to investigate the mediating role of father-child conflict (at 3 years) in the association between postnatal paternal depressive symptoms (at 9-months) and children’s emotional and behavioural (at 7 years) (aim 1). We also examined whether mediation pathways were more pronounced for boys or for girls (aim 2).

**Methods:** Secondary data analysis was conducted on the Millennium Cohort Study, when children were 9-months, 3-years and 7-years-old (n=3,520). Main study variables were measured by self-report questionnaires. Fathers completed the Rutter Scale (depressive symptoms) and the parent-child relationship questionnaire (father-child conflict), while mothers completed the Strengths and Difficulties questionnaire and the Social behaviour questionnaire (child emotional and behavioural problems, emotion regulation). We used structural equation modelling to estimate direct, indirect and total effects of paternal depressive symptoms on child outcomes, mediated by father-child conflict whilst adjusting for relevant covariates (maternal depressive symptoms, child temperament, marital conflict, and socio-economic factors such as poverty indicator and fathers’ education level). Multi-group and interaction analysis was then conducted to determine the differential effect by gender of the association between paternal depressive symptoms on child outcomes via father-child conflict.

**Results:** Father-child conflict mediated the association between paternal depressive symptoms and emotion regulation problems (SIE – CI: -0.03 - -0.01, p<0.001; STE–CI: -0.03 - -0.01, p<0.05) (aim 1). Father-child conflict mediated a larger proportion of the effect in boys (SIE CI: -0.03- -0.01, p<0.001, STE–CI: -0.05- -0.00, p=0.063) than it did in girls (SIE–CI: -0.02- -0.01, p<0.001, STE–CI: -0.04 - 0.01, p=0.216) (aim 2).

**Conclusions:** Father-child conflict may mediate the association between postnatal paternal depressive symptoms and children’s emotion regulation problems. Paternal depressive symptoms and father-child conflict resolution may be potential targets in preventative interventions.

**Key words:** Paternal depressive symptoms, parenting, father-child conflict, behavioural problems, emotion regulation.
Introduction

Postnatal depressive symptoms in fathers are associated with behavioural and emotional problems in children (Dave et al., 2008, Fletcher et al., 2011, Ramchandani et al., 2005, Ramchandani et al., 2008b), but there is limited understanding about the underlying mechanisms that explain these associations. Childhood behavioural and emotional problems are associated with poor outcomes during adulthood, including academic underachievement, psychiatric problems, relationship difficulties, substance abuse and dependency on services (Caspi et al., 1996, Fergusson et al., 2005). This causes considerable burden on public services and has huge costs on society (Scott et al., 2001, Snell et al., 2013). Improved understanding of how paternal depressive symptoms influence children’s behavioural and emotional outcomes during childhood may improve theoretical understanding about the transmission of risk from parents to children and might provide targets for interventions involving fathers (Garfield, 2015, Ramchandani and Murphy, 2013).

Using longitudinal data from the Millennium Cohort Study (MCS), Malmberg and Flouri (2011) found that paternal depressive symptoms at 9 months old predicted behavioural problems in children when they were 3 years old via lower overall quality (lower warmth and higher conflict) in father-child relationship. Using another large cohort study (Longitudinal Study of Australian Children: LSAC study), Giallo et al. (2014b) reported that paternal depressive symptoms during infancy were associated with children’s emotional and behavioural problems at 4-5 years old, and the association was mediated via increased hostile parenting. Child gender did not appear to moderate this association. In both studies, fathers’ parenting and children’s outcomes were measured at the same time-point, therefore causality cannot be assumed. Finally, a study of fathers with children aged 5-9 years old found inconsistent discipline practices mediated the association between paternal depressive symptoms and their sons’ hyperactivity, but not their daughters’ (Dette-Hagenmeyer and Reichle, 2014). Although there was a longitudinal element to this study, paternal depressive symptoms and parenting were measured simultaneously and the measure of child outcomes was collected 6-months later, making the analysis almost cross-sectional.

Evidence suggests that poor emotion regulation in children may be associated with increased externalising and internalising behavioural problems (Eisenberg et al., 2010). Difficulty with regulating anger and impulsivity (under-regulation) has been linked with externalising problems, whereas inability to control cognition and attention (over-regulation involved in rumination and negative bias) have been associated with internalising
problems (Gross, 1998). Studies have also focused on the effects of maternal depression on children’s emotion regulation, and have reported that children of depressed mothers have poorer emotion regulation compared to children of non-depressed mothers (Silk et al., 2006). This may be due to the environmental influence of depressed mothers’ inability to parent sensitively or children modelling mothers’ maladaptive emotion regulation strategies (Eisenberg et al., 2001, Hoffman et al., 2006). Although there have been studies on the importance of fathers to the development of emotion regulation among children (Cabrera et al., 2007, Kiel and Kalomiris, 2015, Wilson et al., 2014), to our knowledge there are no studies that examine the association between paternal depressive symptoms and children’s emotion regulation.

There are some studies that provide evidence for the potential mechanisms by which paternal depressive symptoms may influence children’s emotional and behavioural problems. One hypothesis proposes that paternal depressive symptoms may influence children’s development via fathers’ impaired parenting (Ramchandani and Psychogiou, 2009). Depression in parents has been associated with negative parent-child interactions (Psychogiou and Parry, 2014, Sethna et al., 2015) and a meta-analysis of 28 studies reported paternal depressive symptoms to be associated with increased negative (intrusive, hostile, harsh, controlling, and critical) and decreased positive parenting behaviours (sensitive responding, accepting, warm, affectionate, and supporting) towards children (Wilson and Durbin, 2010). One study found that fathers’ disengaged parenting when their infants were 3-months old predicted externalising problems at 1 year in their sons, but not daughters (Ramchandani et al., 2013). Another meta-analysis consisting of 6 studies reported that father-child conflict mediated the association between paternal depressive symptoms and children’s emotional problems (Kane and Garber, 2004). However, the studies in this meta-analysis included children with a wide age range (from 3 to 14 years) and most studies were cross-sectional. To infer mediation, it is necessary to study the exposure of paternal depressive symptoms, fathers’ parenting and children’s outcomes longitudinally (Selig and Preacher, 2009). Thus, the variables need to be measured at different time-points. Additionally, given that there were only 6 studies on father-child conflict, mediation requires further investigation.

Although studies have found a link between paternal depressive symptoms and children’s behavioural and emotional problems (Davé et al., 2008, Ramchandani et al., 2005, Ramchandani et al., 2008b), there are mixed findings about the influence of paternal depressive symptoms regarding child gender-specific pathways of risk transmission. Postnatal depressive symptoms in fathers 8 weeks after child birth were associated with children’s
psychopathology at 3 and 7 years old in the UK Avon Longitudinal Study of Parents and Children (ALSPAC) (Ramchandani et al., 2005, Ramchandani et al., 2008b); sons of fathers with high depressive symptoms were reported to display more conduct problems compared to daughters. In contrast, findings from the LSAC suggested that the daughters of fathers with high depressive symptoms in the first post-natal year were more likely to have emotional and conduct problems when they were 4-5 years old, whereas sons were more likely to exhibit hyperactivity problems and lower levels of prosocial behaviour (Fletcher et al., 2011). Exploring gender-specific pathways may help explain the possible father to child transmission of risk which may subsequently enable us to identify whether girls and/or boys are more vulnerable to their fathers’ depressive symptoms. This could influence the content and direction of interventions with depressed fathers.

This study aims to address these gaps in the current literature using a large representative sample of fathers from the MCS in the UK. The primary objective is to test the proposed model (Figure 1) and investigate whether father-child conflict mediates the association between paternal depressive symptoms and children’s emotional and behavioural outcomes. The exposure of paternal depressive symptoms during infancy has been identified as a potential sensitive period where paternal depressive symptoms may influence children’s later outcomes (Ramchandani et al., 2008a). The mediator variable father-child conflict was measured at 3 years old during the preschool year when fathers’ become more involved in parenting their children (Bruce and Fox, 1999, Grossmann et al., 2002, MacDonald and Parke, 1986). Previous literature has associated father-child conflict with paternal depression and negative outcomes in children (Kane and Garber, 2004, 2009). Additionally, using the MCS dataset, a recent study found an association between paternal depressive symptoms and father-child conflict, but not father-child warmth or fathers’ involvement in parenting activities, suggesting that father-child conflict may be an important construct that is influenced by paternal depressive symptoms (Nath et al., 2015). Children’s outcomes were measured at 7 years old. This is a developmentally challenging period as children learn new behaviour and emotion management skills and problems with adjustment at this age predict poor outcomes in adulthood (Fergusson et al., 2005, Pianta et al., 1995). Given that maternal depression, marital conflict, child temperament, child gender, and family socio-economic status may be associated with fathers’ depressive symptoms, parenting and children’s emotional and behavioural outcomes (Flouri et al., 2014,
Goodman, 2004, Gutierrez-Galve et al., 2015, Hanington et al., 2012, Hanington et al., 2010, Kiernan and
Huerta, 2008, Malmberg and Flouri, 2011), we controlled for these factors in our models. Our secondary
objective was to test the moderating influence of child gender on any potential associations. We predicted that
higher father-child conflict at 3 years old would mediate the association between higher paternal depressive
symptoms at 9-months old and children’s increased behavioural-emotional outcomes at 7 years. We expected
that child gender would moderate this mediation, but did not expect a specific direction given the mixed
literature.

Methods

Participants

This secondary data analysis was conducted using the first (S1), second (S2) and forth (S4) sweeps of the
Millennium Cohort Study (MCS), when children were 9 months, 3 years and 7 years old. The MCS is a large-
scale survey of infants (n=19,519) born in four constituent countries of the United Kingdom (Dex and Joshi,
2005). The sample design allowed for over-representation of families living in areas with high rates of child
poverty or high proportions of ethnic minorities in England and the three smaller countries in the UK (Northern
Ireland, Wales, and Scotland). Full details of the survey, objectives, content of survey and sampling strategy can
be found in the documentation attached to the data deposited with the UK Data Archive and elsewhere (Hansen,
2014, Plewis and Ketende, 2006). MCS had informed consent from participants and ethical approval (Hansen,
2012). Our work was a secondary analysis of anonymised data that is publically available on the website
(http://discover.ukdataservice.ac.uk/series/) requiring no direct contact with the individual participants, so
further ethical approval was not required.

The first wave (S1) of data was collected from 2001-2002 on 18,533 families, with a total of 18,819 infants aged
between 9-11 months. The same sample were then invited to follow-up with 15,590 families in the second wave
(S2) when the children were approximately 3 years old and 13,857 in the fourth wave (S4) when children were
approximately 7 years old.
The MCS collected data from main respondents (usually mothers) and partner respondents (fathers, step fathers, same sex partners). For the current study, the sample was limited to biological fathers (partner respondents) and mothers (main respondents). Fathers who were main respondents were excluded to simplify analysis as main and partner questions were not identical. Part-time resident and step-fathers were also excluded due to insufficient numbers. A small sample of twins and triplets were excluded to avoid the need to include an extra level of analysis that would have accounted for intra-family variability. Thus, only one child per family (the first cohort member) was studied. See Figure 2 for details of eligibility, sample size at each stage and final sample used for main analysis.

[Insert Figure 2 here]

**Measures**

**Paternal depressive symptoms**

Rutter’s 9-item Malaise Inventory (S1 – 9 months old) was used as an indicator for depressive symptoms in S1 completed by fathers (Dex and Joshi, 2004, Rutter et al., 1970). This is the shortened version of the Rutter’s 24-item Malaise Inventory self-completion questionnaire measuring psychological distress (Bartley et al., 2004, Johnson, 2012, Rutter et al., 1970). The 9-item short form included items “feel tired most of the time”, “feel miserable or depressed”, “worried about things”, “often get into a violent range”, “suddenly become scared for no good reason”, “easily upset or irritated”, “constantly keyed up and jittery”, “every little thing gets on nerves and wears you out”, and “heart race like mad”. Scores from these were summed to create a continuous scale. This scale has been used in previous studies as an indicator of depressive symptoms (Kiernan and Huerta, 2008, Malmberg and Flouri, 2011). Using Cronbach’s alpha (α) coefficient the internal consistency of the scale was 0.75 for mothers and 0.71 for fathers which is similar to previous validation studies on the scale (Rodgers et al., 1999). The original scale has also shown acceptable validity (Area Under the Curve (AUC) = 0.74 with mental health problems, AUC = 0.77 with psychiatric diagnosis, AUC = 0.87 with depression) (Rodgers et al., 1999).

**Fathers’ parenting: Father-child conflict**

Fathers’ parenting was measured using The Child-Parent Relationship Scale reported by fathers (CPRS; Short form, (Johnson, 2012, Pianta and Steinberg, 1992). In this study, father-child conflict refers to the communication and relationship between the parent and the child which is measured by 8 self-report items on a
5-point Likert scale (ranging from 1=definitely does not apply to 5= definitely applies). All items were summed to create a continuous scale. Items include “child and I always seem to be struggling with each other”, “child uncomfortable with physical affection or touch by me”, “child easily becomes angry with me”, “child remains angry/resistant after discipline”, “dealing with my child drains my energy”, “when child wakes up in bad mood, I know we’re in for a long and difficult day”, “child’s feelings towards me can be unpredictable or change suddenly” and “child is sneaky or manipulative with me”. This scale has been used by other studies as an indicator of parenting (Kiernan and Huerta, 2008, Mensah and Kiernan, 2011, Nath et al., 2015) and the items originate from attachment theory, Attachment Q-set and literature on parent-child relationships to form a subscale looking at negative approach towards father-child relationship (conflict). Higher scores on the scale indicated higher conflict in relationship. Fathers’ reports are comparable to mothers’ reports and have been validated against observational data on parent-child interactions which has shown conflict ratings on the CPRS to correlate with observational coding of hostility (Driscoll and Pianta, 2011). The scale had adequate internal consistency (α=0.73).

Child behavioural and emotional problem
The Strengths and Difficulties Questionnaire (SDQ; (Goodman, 2001)) completed by mothers was used to assess child emotional and behavioural problems (S4, 7 years old). The SDQ is validated for children aged 3-16 years old and was developed as a clinical tool to identify psychopathology. There are 25 items in total consisting of five continuous subscales: emotional symptoms, conduct problems, hyperactivity, peer problems and prosocial behaviour. The scores for each subscale range between 0-10. All sub-scales were used in the analysis. Higher scores indicate greater problems on the emotional, conduct, hyperactivity, and peer problems, whereas higher scores on the prosocial scale indicated more prosocial behaviour. The internal consistencies from the MCS were: conduct problems α= 0.55, emotional problems α=0.68, hyperactivity α= 0.78, peer problems α=0.59 and prosocial α=0.71. These are similar to internal consistencies reported in other studies using non-clinical samples of children (Muris et al., 2003, Niclasen et al., 2012). The scale has also been reported to have sufficient validity against diagnostics of DSM-IV disorders (specificity=96%, sensitivity=49%) (Goodman, 2001) and Child Behaviour Chick List (CBCL; r=0.76) (Stone et al., 2010).

Child emotion regulation
The MCS team selected items from the Child Social Behaviour Questionnaire (CSBQ) (Hogan et al., 1992, Johnson, 2012) that were completed by mothers and used to generate three continuous sub-scales; 1) self-regulation defined as children’s ability to adapt to situations independently, 2) emotion dysregulation defined as children’s inability to deal with difficult/frustrating situations, and 3) cooperation defined as children’s ability to cooperate with others. The self-regulation and the reverse of emotion dysregulation scales were significantly correlated (r=0.32, p<0.001) and were summed to create an emotion regulation scale for the analysis. The cooperation subscale was not used because it does not belong to the emotion regulation construct. Higher scores on the emotion regulation scale indicated more adaptive emotion regulation. The internal consistency of the scale was α= 0.72. This scale devised by the MCS team has been used in other studies to measure emotion regulation (Flouri et al., 2014).

Family context covariates

Maternal depressive symptoms (S1- 9 month) were measured using the Rutter Malaise Inventory as above (Dex and Joshi, 2004, Johnson, 2012, Rutter et al., 1970). Children’s temperament (S1- 9 months) was measured with mothers’ reports on the Carey Infant Temperament Scale (Carey and McDevitt, 1978). Fourteen items from the original scale were selected by the MCS team to measure regularity (4 items), approach withdrawal (3 items), adaptability (2 items) and mood (5 items), and has also been used in other studies as an indicator of child temperament (Flouri and Malmberg, 2012, Kiernan and Huerta, 2008). Items were on a 5-point scale (almost never, rarely, usually does not, often, almost always). All scores were on a continuous scale ranging from (14 – 70) consisting of the total score of all items. Higher scores indicated easier infant temperament and lower scores indicated more difficult temperament. The internal consistency of the scale was α=0.66. Marital conflict (S1- 9 months) was measured using the modified version of the Golombok Rust Inventory of Relationship State (Rust et al., 1990). The original 28-item questionnaire had high content validity and reliability of Cronbach’s alpha = 0.91 (men) and 0.87 (women). The MCS selected seven items at S1 and S2, and three items at S4 (Johnson, 2012). These were summed to create continuous scales where higher scores indicate higher levels of relationship conflict.

Socioeconomic Status (SES)

Households were classed as living in poverty if their income was equal to or less than 60% of the median household income for the UK (dichotomous scale), the definition of poverty set by the UK government.
Paternal education was reported by fathers and was categorised into two groups: no qualification or school level, degree and higher degree (NVQ level or equivalent, undergraduate and postgraduate degree).

**Statistical analysis**

Structural Equation Modelling (SEM) using Stata for Windows version 13 was used to test the mediation model illustrated in Figure 1, i.e., whether ‘father-child conflict’ (3 years) mediated the association between ‘paternal depressive symptoms’ (9 months) and ‘child outcomes’ at 7 years old (conduct problems, emotional problems, hyperactivity, peer problems, prosocial and emotion regulation). The model estimated standardised direct, indirect and total effects, as well as adjusting for a number of relevant covariates (maternal depressive symptoms, marital conflict, child temperament, child gender and SES) (model 1). Outcomes that were significantly associated with paternal depressive symptoms in model 1 were taken forward into a multi-group SEM analysis. This investigated whether child gender moderated the associations between ‘paternal depressive symptoms’ and ‘child outcomes’ mediated by ‘father-child conflict’ while controlling for maternal depressive symptoms, marital conflict, child temperament and SES (model 2). The SEM mediation model was run again using interaction terms to further investigate whether gender interacted with the exposure or mediator to influence the outcome child variables while controlling for maternal depressive symptoms, marital conflict, child temperament and SES (model 3). Finally, we further controlled for marital conflict in subsequent sweeps to increase the validity of any associations found in model 3 (model 4).

As the sample was stratified, sampling weights were used in all analyses to adjust for the disproportionate number of participants from ethnic minority and low socio-economic status backgrounds initially recruited into the sample at S1. Weights aimed to return the sample to the structure of the UK population and also to account for the effect of attrition and multi-stage cluster sampling strategy used by the MCS. The use of the weights is recommended by the MCS team and available with the dataset. Missing data were not analysed. Only data with complete cases on variables of interest across time-points were included in the analysis. In order to check that the results in the ‘complete cases’ model were robust to the effects of attrition, missing data were imputed from all variables included in the analysis. A sensitively analysis was conducted to check that results were broadly replicated when missing data were imputed.
Results

Descriptive statistics

Table 1 shows descriptive statistics for the study main measures (exposure, mediator and outcomes). Logistic regression analyses were used to test for gender differences. Compared to boys, girls had lower odds of conflict with their fathers (OR: 0.99, 95% CI: 0.98-0.98, p=0.011), conduct problems (OR: 0.84, 95% CI: 0.80-0.87, p=<0.001), hyperactivity (OR: 0.85, 95% CI: 0.83-0.88, p=<0.001), and peer problems (OR: 0.92, 95% CI: 0.89-0.96, p=<0.001), but higher odds of prosocial problems (OR: 1.25, 95% CI: 1.21-1.30, p=<0.001) and adaptive emotion regulation (OR: 1.54, 95% CI: 1.41-1.68, p=<0.001). There was no significant associations between child gender and fathers depressive symptoms (OR: 0.98, 95% CI: 0.95-1.01, p=1.135) or child emotional problems (OR: 1.03, 95% CI: 0.99-1.07, p=0.100).

The predictor (paternal depressive symptoms), mediator (father-child conflict) and outcomes (child emotional and behavioural problems) were significantly correlated, a necessary requirement for mediation to occur (Table 2).

Attrition and missingness

Attrition (i.e. fathers who did not take part in Sweeps 2 (3 years) and 4 (7 years)), and missingness (i.e fathers who did not provide complete answers to survey questions) were associated with low socio-demographic factors (Table 3). Fathers had a higher odds of dropping out by both follow-up sweeps if they were below the 60% median of the poverty indicator (S2 –OR: 3.14, 95% CI: 2.63-3.74, p=<0.001; S4 - OR: 2.31, 95% CI:1.96-2.72, p=<0.001) and had lower odds if they were educated (S2 –OR: 0.45 , 95% CI: 0.35–0.50, p=<0.001; S4 - OR: 0.63, 95% CI:0.53-0.75, p=<0.001). Fathers with higher depressive symptoms also had higher odds of dropping out (S2 –OR: 1.08, 95% CI: 1.06-1.11, p=<0.001; S4 - OR: 1.07, 95% CI: 1.04-1.10, p=<0.001).

[Insert Table 1 here]
[Insert Table 2 here]
[Insert Table 3 here]
Covariates

All family and socio-economic covariates that were associated with higher paternal depressive symptoms, father-child conflict and child outcome, and therefore were controlled for in the analysis models (See online supplementary tables 1 and 2).

Mediation model

Table 4 (Model 1) shows that after adjusting for relevant covariates (maternal depressive symptoms, child temperament, marital conflict, child gender and family SES), higher father-child conflict mediated the association between high paternal depressive symptoms at 9-months and children’s increased conduct problems, and emotion dysregulation at age 7 years old. The estimated total effects of paternal depressive symptoms on children’s emotional, hyperactivity, prosocial behaviour and peer problems were not significant and therefore were not taken forward into model 2. Table 4 shows the coefficients for each outcome (Model 1).

[Insert Table 4 here]

Moderation by gender

Model 2 tested for child gender moderator effects (Table 4). This model included significant outcomes from model 1 (conduct problems and emotion regulation) and also adjusted for maternal depressive symptoms, marital conflict, child temperament and SES. Higher father-child conflict mediated the association between higher paternal depressive symptoms and boys increased conduct problems (Table 4, Model 2). This association was also marginally significant for emotion regulation. No significant effects were found for girls, which suggest that paternal depressive symptoms may have an effect via conflict on conduct and emotion regulation problems in their sons but not in their daughters.

After testing for interaction effects (Model 3), high father-child conflict still significantly mediated the association between higher post-natal paternal depressive symptoms and boys’ conduct problems and emotion regulation. Table 4 (Model 3) shows the standardised coefficients for each outcome according to gender interaction with the exposure paternal depressive symptoms and mediator father-child conflict. For conduct problems, child gender interacted with both paternal depressive symptoms and father-child conflict while for
emotion regulation, child gender interacted significantly with father-child conflict. After further adjusting the
model for marital conflict at all time-points (table 5, model 4), higher father-child conflict still significantly
mediated the association between high post-natal paternal depressive symptoms and emotion regulation, but the
association between paternal depressive symptoms and child conduct problems became non-significant. In this
model, child gender interacted significantly with father-child conflict, but not paternal depressive symptoms in
the association between paternal depressive symptoms and child emotion regulation. The model fit statistics
show that model 4 is the best fit. The final overall model (model 4) explained 59% of the variance ($R^2 = 0.59$).

**Sensitivity analysis**
The sensitivity analysis using imputed data replicated the findings in the main analysis in that all effects
remained significant. In fact, significant findings increased in the main analysis model 1 (see online
supplementary table 3). Therefore, this suggests that we have underestimated the effects of paternal depressive
symptoms on children’s emotional and behavioural problems via father-child conflict; therefore further analysis
was not undertaken using the imputed datasets on models 2, 3 and 4.

**Discussion**
As hypothesised, higher paternal depressive symptoms at 9 months were significantly associated with children’s
emotion regulation at 7 years old, via higher father-child conflict when children were 3 years old. Thus,
depressive symptoms in fathers may influence their mood. Therefore these fathers may struggle with conflict
resolution with their toddlers and this negative interaction may later impact on their children’s emotional
development during early school years. These findings extend previous studies investigating the association
between paternal depressive symptoms, fathers’ parenting and children’s emotional and behavioural problems
(Dette-Hagenmeyer and Reichle, 2014, Giallo et al., 2014b, Kane and Garber, 2009, Malmberg and Flouri,
2011, Ramchandani et al., 2005, Ramchandani et al., 2008b) and add to literature by using longitudinal data
from key time points. This study is also the first to our knowledge that has investigated the association between
paternal depressive symptoms and children’s emotion regulation. Our findings also extend the literature by
adding that father-child conflict may mediate this association path. Furthermore, we found that gender had an
interaction effect with father-child conflict and not fathers’ depressive symptoms. These findings suggest that
father-child conflict may be an important factor that could be targeted to reduce emotional and behavioural
problems in sons of fathers suffering from depression. Our findings that boys with poorer emotion regulation abilities had higher externalising problems is also in line with previous research (Eisenberg et al., 2010).

Parental socialisation provides an environmental explanation for our findings (Eisenberg et al., 2001). Previous studies have shown that maternal depression negatively impacts on their daughters’ emotion regulation abilities but not sons’ (Silk et al., 2006). As emotion regulation abilities are learnt from socialisation with parents during early development, these results may indicate that same-gendered parents have a greater influence on their children in this process (Eisenberg et al., 2001). Compared to mothers, fathers have been reported to respond more harshly and provide less support towards their son’s emotional expressions, which according to role model theory may subsequently be imitated by boys (Brody and Hall, 2008, Brown et al., 2015, Chaplin et al., 2005, Fischer, 2000, Sanders et al., 2015). This may cause conflict interactions between fathers and their sons, which may lead to emotional and behavioural problems (Kane and Garber, 2004, 2009). An alternative explanation is that our findings could be due to genetic heritability or an interplay between gene-environment (Natsuaki et al., 2014, Ramchandani and Psychogiou, 2009). Sons of depressed fathers could be genetically predisposed to developing depressive symptoms and also exposed to the family environmental factors associated with paternal depressive symptoms (Gutierrez-Galve et al., 2015, Rutter, 2009, Rutter et al., 1997), which may increase father-child conflict and child emotional problems. However, more research is needed to support both of these explanations. As the current study could not control for genetic influences, this is something that could be addressed by future research.

We also found that higher paternal depressive symptoms at 9 months were significantly associated with children’s conduct problems at 7 years old, via higher father-child conflict when children were 3 years old after controlling for maternal depressive symptoms, child temperament, and family SES which was in line with previous literature (Ramchandani et al., 2005, Ramchandani et al., 2013, Ramchandani et al., 2008b). However, after further controlling for marital conflict at all time-points, the overall associations between paternal depressive symptoms and child conduct problems became non-significant. One explanation for this could be that marital conflict may have a stronger influence on children’s behavioural problems (Braithwaite et al., 2015, Hanington et al., 2012) compared to paternal depressive symptoms (predictor variable). Additionally, marital conflict and father-child conflict (mediator variable) are closely related variables that may jointly be influencing the association between paternal depressive symptoms and child outcomes within the model (Cummings and
Therefore, future studies should aim to differentiate and disentangle the effects of different conflictual relationships within the family in relation to child adjustment, perhaps with observational rather than self-report measures.

The current study has a number of strengths. First, the MCS is a unique dataset with a large representative sample of UK fathers (Hansen, 2014, Plewis and Ketende, 2006). Second, the MCS collected a large number of measures on mothers, fathers and children, most of which were well validated and reliable and we tested children’s outcomes longitudinally (Johnson, 2012). Third, our findings are further strengthened by child outcomes being reported from mothers, decreasing any variances produced by same informant reporter bias of predictors and outcomes. Finally, the MCS (like other cohort datasets) is subjected to attrition and missing data. However, research indicates that even when dropouts are taken into account, regression models with large cohort studies are still robust (Wolke et al., 2009). In addition, our analysis accounted for attrition/missing data by utilising sampling weights recommended by the MCS team (Ketende and Jones, 2011). This increased the representativeness and accounted for missing data/attrition rates that might have influenced or biased the results. We further conducted a sensitivity analysis using multiple imputation, a statistical method used in recent years to account for attrition in cohort studies (Niarchou et al., 2015, Sterne et al., 2009). This replicated our main finding, and if anything suggests that we have underestimated the effects.

There were also some limitations. Firstly, we lacked data on clinical diagnoses of depressive episodes using interview methods, which some might argue would be more informative. Parenting was also measured using self-reports, which might not be accurate due to biased reporting of positive parenting and inter-association with fathers reporting high depressive symptoms and higher conflict parenting. Parenting is often measured using observational methods of parent-child interactions (Aspland and Gardner, 2003). However, in both cases conducting studies using observational and interview methods with such a large sample size would be expensive, time-consuming, and impractical. Thus, the study of the influence of depressive symptoms is useful as results can later be tested experimentally using smaller clinical samples. Secondly, the coefficient effect sizes of the associations were small. Previous studies investigating mediation effects using large cohort studies have also reported small effect sizes of maternal and paternal depressive symptoms on child outcomes (Giallo et al., 2014a, Giallo et al., 2014b, Malmberg and Flouri, 2011). Given that the MCS consisted of a normal population of fathers, clinically relevant high levels of depressive symptoms would have been underestimated due to the
likelihood of depressed fathers being less motivated to participate and therefore might have resulted in small
effect sizes. A smaller scale study consisting of a clinical sample of depressed fathers may yield larger effect
sizes. This is something for future studies to investigate. However, given the huge challenges with recruiting
depressed fathers to participate in research with their children (Garber et al., 2011, Pilowsky et al., 2014, Sherr
et al., 2006), the findings from large cohort studies such as the MCS offers useful insight into the possible
associations in this field of limited literature (Fagan, 2014, Niarchou et al., 2015). These findings, if replicated
with a clinical population, could suggest targets for development of clinical interventions.

We also assumed causal direction due to the data originating at different time points across the child’s life. We
are assuming linear relationships when in fact the interrelationships between parental mental health, parenting
and children’s development is likely to be complex and these factors may amplify and feedback on each other.
Therefore, our findings need to be replicated experimentally to draw firm conclusion about causal direction.
This could only be done in by using treatment trials for paternal depression which provide an opportunity to see
if father-child conflict and child emotional/behavioural problems are reduced among fathers who respond
compared to those who do not respond to treatment.

Finally, we acknowledge that there are more statistically advance techniques for testing mediation using Cross-
Lagged Panel Modelling (CLPM) and Latent Growth Mediation (LGM) Modelling (Selig and Preacher, 2009).
These techniques account for autoregressive controls, reverse causality and trends (slopes and intercepts)
between associations (Cole and Maxwell, 2003, Maxwell and Cole, 2007, Maxwell et al., 2011). These methods
have been used to investigate moderation and mediation in maternal depression, mothers’ parenting and
children’s behavioural outcomes (Beauchaine et al., 2005, Belsky et al., 2007, Eisenberg et al., 2005). However,
these statistical techniques require all variables to be available at all time-points (Selig and Preacher, 2009),
which was not the case in the MCS, restricting the analysis method we could utilise. Specifically with regards to
our research question, it would be important to investigate family context factors that may change over time
such as maternal and paternal depressive symptoms. Thus, future research work could expand the statistical
analysis of this paper by using longitudinal multivariate analysis with more statistically advanced techniques to
build on the findings of this paper once appropriate samples are available.
Despite some of the limitations, the findings of this study add to theoretical understanding of indirect effects of fathers’ postnatal depressive symptoms to their children’s outcomes. Postnatal paternal depressive symptoms were associated with boys’ emotion regulation problems at 7 years old via higher father-child conflict at 3 years old. This association still remained significant after accounting for maternal depressive symptoms, child temperament, SES and marital conflict (at all time points). There are some specific implications that can be taken from the current study to inform parenting interventions. For example, parenting interventions could help with managing conflict parent-child relationships between depressed parents and their children. This may have potential to break the intergenerational transmission of risk. Parenting interventions involving fathers have been found to lessen behavioural problems in children mainly due to the change in fathers’ parenting (Wilson et al., 2014), therefore involving depressed fathers in such interventions maybe beneficial. Like maternal depression, studies have shown paternal depression to have a cost on public health care services (Edoka et al., 2011), but intervention programs are still primarily targeted at mothers (Panter-Brick et al., 2014). In light of our findings we would encourage more research with fathers and involvement of fathers in parenting programs of depressed parents.
Required statements

Acknowledgements: We would like to thank the Millennium Cohort Study families and Millennium Cohort Study team at the Centre of Longitudinal Studies, Institute of Education, London, United Kingdom for the use of these data and to the UK Data Archive and Economic and Social Data Service for making them available.

Conflict of interest statement: None

Ethical standards: The authors assert that all procedures contributing to this work comply with ethical standards of the relevant national and institutional committees on human experimentation. More specifically, the Millennium Cohort Study had informed consent from participants and ethical approval. Our work was a secondary analysis of anonymised data that is publically available on the website (http://discover.ukdataservice.ac.uk/series/) requiring no direct contact with the individual participants, so further ethical approval was not required.

Contribution of each author statement:

Dr Selina Nath: Conception and design, data analysis, interpretation of data, write-up of article, revising it critically for important intellectual content and final approval of the version to be published.

Dr Ginny Russell: Conception and design, provided guidance with data analysis, interpretation of data and analysis, revising it critically for important intellectual content and final approval of the version to be published.

Prof Willem Kuyken: Interpretation of data and analysis, revising it critically for important intellectual content and final approval of the version to be published.

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Prof Tamsin Ford: Conception and design, interpretation of data and analysis, revising it critically for important intellectual content and final approval of the version to be published.
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