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Developmental inter-relations between early maternal depression, contextual risks, and interpersonal stress, and their effect on later child cognitive functioning

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Short title: Impact of risks on cognitive functioning

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Abstract

**Background:** Maternal depression and contextual risks (e.g., poverty) are known to impact children’s cognitive and social functioning. However, few published studies have examined how stress in the social environment (i.e., interpersonal stress) might developmentally inter-relate with maternal depression and contextual risks to negatively affect a child in these domains. This was the purpose of the current study. **Method:** Mother-child pairs (n= 6979) from the Avon Longitudinal Study of Parents were the study participants. Mothers reported on depression, contextual risks and interpersonal stress between pregnancy and 33 months child age. At age 8, the children underwent cognitive assessments and the mothers reported on the children’s social cognitive skills. **Results:** Maternal depression, contextual risks, and interpersonal stress showed strong continuity and developmental inter-relatedness. Maternal depression and contextual risks directly predicted a range of child outcomes, including executive functions and social cognitive skills. Interpersonal stress worked indirectly via maternal depression and contextual risks to negatively affect child outcomes. **Conclusion:** Maternal depression and contextual risks each increased interpersonal stress in the household, which, in turn, contributed to reduced child cognitive and social functioning.
Introduction

Maternal depression can negatively impact a range of cognitive functions\textsuperscript{[1]} and verbal abilities,\textsuperscript{[2]} as well as children’s abilities to regulate their own emotions and behaviours\textsuperscript{[3]}. The effects of maternal depression are often attributed to impaired parenting. That is, depressed mothers can show decreased sensitivity in interactions with a child, and a lack of contingency in the response to the child’s actions\textsuperscript{[4,5]}. Such developmental circumstances are thought to impede the ability of a child to achieve developmental milestones in the cognitive\textsuperscript{[6]} and socio-emotional domains\textsuperscript{[5]}.

Similar to maternal depression, contextual risks can negatively impact children’s cognitive functioning, including executive functions such as attention\textsuperscript{[7]}, inhibitory control\textsuperscript{[8]}, IQ\textsuperscript{[9]}, and language development\textsuperscript{[10]}. Contextual risks are defined as factors that affect an individual’s basic living conditions (e.g., poverty and housing inadequacy). The effect of contextual risks on child cognitive functioning is believed to work through parents having lower levels of education and fewer resources with which to engage their children in cognitively stimulating interactions\textsuperscript{[11]}.

Still, it may be that the effect of both maternal depression and contextual risks on children is at least partially explained by factors relating to the immediate social environment a child experiences\textsuperscript{[12]}. This is particularly important since, early in the life course, the mother constitutes the primary social environment for the child\textsuperscript{[13]}. With regard to depression, Hammen’s Stress Generation Model\textsuperscript{[14]} posits that depressed persons can generate interpersonal stress (i.e., interpersonal conflict and low levels of social and practical support)\textsuperscript{[15]}, which can then work in a bidirectional manner to increase and maintain depression\textsuperscript{[16]}. Three studies have shown that the interpersonal stress of depressed mothers can negatively affect the wellbeing of
adolescents\textsuperscript{16,17} and children\textsuperscript{18}. Still, these studies largely did not assess the relative impact of contextual risks. This is an important limitation since maternal depression co-occurs, both cross-sectionally\textsuperscript{19} and longitudinally\textsuperscript{18}, with interpersonal stress and contextual risks\textsuperscript{20}. Therefore, both contextual risks and interpersonal stress may affect the development of a child via depression in a mother. For example, contextual risks (e.g., income) are reported to be more strongly associated with low levels of maternal sensitivity in depressed compared with non-depressed mothers\textsuperscript{21}.

A limited number of studies have examined the combined effect of maternal depression and contextual risks on children’s cognitive functioning, and findings are mixed. For example, certain studies report that maternal depression and contextual risks independently affect child cognition\textsuperscript{4,6,8,22}, whereas other studies suggest that the effect of maternal depression is particularly pronounced when co-occurring with contextual risks\textsuperscript{13,23}. Some studies have related the effect of maternal depression on children’s development to impaired mother-infant interactions\textsuperscript{5,13,21}. Yet, as stated above, these studies have largely not considered the additional impact of interpersonal stress.

The present study examined the extent to which maternal depression, contextual risks, and interpersonal stress early in development can inter-relate to negatively impact children’s cognitive functioning and social cognitive skills. We investigated four developmental pathways. Two pathways (paths A and B) tested the bidirectional relationships between maternal depression and interpersonal stress suggested by Hammen’s Stress Generation Model\textsuperscript{24}. The remaining two pathways (paths C and D) investigated the effect of contextual risks increasing either maternal depression or interpersonal stress, each of which then increases the other (i.e., interpersonal stress or maternal depression, respectively) and, in turn, affects children’s cognitive
and social functioning. Pathways C and D followed research that has reported contextual risks to prospectively associate with maternal depression and interpersonal stress\(^{18,20,21}\); these two pathways tested the degree to which contextual risk can affect child development through associating with higher levels maternal symptoms of depression, and interpersonal stress.

**Materials and Methods**

**Sample**

The Avon Longitudinal Study of Parents and Children (ALSPAC) is an ongoing population-based study designed to investigate the effects of an array of influences on the development and health of children. Pregnant women residing in the former Avon Health Authority in South-West England, who had an estimated date of delivery between 1 April 1991 and 31 December 1992, were invited to participate in the study. This resulted in a cohort of 14,541 pregnancies, of which 13,988 singletons/twins were alive at 12 months of age\(^{25}\). The overall ALSPAC sample has been found to be representative of the UK population as a whole\(^{26}\). Ethical approval for the current study was obtained from the ALSPAC Law and Ethics Committee and the Local Research Ethics Committees. More information on the ALSPAC sample is available from the website: [http://www.bris.ac.uk/alspac/](http://www.bris.ac.uk/alspac/).

**Risk measures**

Mothers completed questionnaires at multiple time points during their pregnancy and their child’s infancy and childhood. The early risk factors examined here were drawn from questionnaires completed between birth and approximately 3 years of child age. Our previous research has demonstrated the validity of the risk measures specified below\(^{27,28}\).

**Maternal depression.** Maternal depression was assessed at 32 weeks in pregnancy and
repeatedly postnatally (at 8 weeks, 8 months, 21 months, and 33 months) with the Edinburgh Postnatal Depression Scale\cite{29}. EPDS is a widely used 10-item self-report questionnaire that has been shown to be valid both in and outside of the postnatal period\cite{29}. We used a measure of summed symptoms, where higher scores corresponded to higher levels of depressive symptoms. Continuous measures of psychopathology have shown high reliability and validity, even compared with categorical (i.e., clinical) measures\cite{30}.

*Contextual risk.* Measures of contextual risks were drawn from the family adversity measure\cite{31}, and obtained from maternal questionnaires at 18 weeks in pregnancy and at 33 months postnatally. Contextual risks were grouped into three time points: pregnancy, 0-2 years, and 2-4 years. We assessed seven contextual risks resulting in a cumulative risk index ranging from 0 to 7. Indication of any risk, at any time point, was scored as 1. The seven risks were: (1) inadequate basic living conditions (e.g., not having a working bath/shower, no hot water, no indoor toilet and/or no working kitchen) (assessed 8 weeks in pregnancy, and at 2, 8, 21, 33, and 47 months postnatally); (2) inadequate housing conditions (e.g., indication of crowding) (assessed at 8 weeks in pregnancy and at 21 and 33 months postnatally) and/or homelessness (assessed at 18 weeks in pregnancy and at 2, 8, 21, and 33 months postnatally); (3) housing defects (e.g., indication of mold, roof leaks, and rats, mice or cockroaches) (assessed at 18 weeks in pregnancy; and 8, 21, and 33 months postnatally); (4) poverty coded via the Registrar General’s social class scale\cite{32} (assessed at 32 weeks in pregnancy and at 8, 21, and 33 months postnatally); (5) single caregiver status (e.g., not cohabiting and not in a relationship) (assessed at 32 weeks in pregnancy and at 6, 21, 33, and 47 months postnatally); (6) early parenthood (one parent being 19 years or younger) (assessed at 18 weeks in pregnancy); and (7) low parental educational attainment (e.g., did not finish mandatory schooling) (assessed at 32 weeks in
pregnancy and at 21 and 33 month).

*Maternal interpersonal stress.* Measures of maternal interpersonal stress were collected simultaneously with contextual risks (i.e., in pregnancy, 0-2 years and 2-4 years child age). We used five categories of interpersonal stress resulting in a cumulative index ranging from 0 to 5. Again indication of any risk, at any age-stage, was scored as 1. The five categories of interpersonal stress were (1) mother experiencing partner cruelty (e.g., any indication of emotional and/or physical abuse from partner) (assessed at 18 weeks in pregnancy and at 2, 6, 21, 33, and 47 months postnatally); (2) low partner affection towards the mother (e.g., partner shows no affection, does not hug/kiss, low intimate bond) (assessed at 12 weeks in pregnancy and at 8 and 33 months postnatally); (3) low partner social support (e.g., partner does not discuss feelings, lack of emotional support) (assessed at 18 weeks in pregnancy and at 2 and 8 months postnatally); (4) low practical support (i.e., no one could lend the mother £100 and/or the absence of someone the mother could turn to if in trouble) (assessed at 12 weeks in pregnancy and at 8 months postnatally); and (5) major family problems examined via social services data (e.g., a child in the household taken into extra-familial care or registration with at-risk register) (assessed at 18 weeks in pregnancy and at 21, 33, and 47 months postnatally).

*Child cognitive functioning at age 8*

*Verbal and performance Intelligence Quotient (IQ).* Verbal and performance IQ were measured with the Wechsler Intelligence Scale for Children (WISC) (3rd UK edition) using an edition consisting of alternate items for all subtests except for the coding subtest, which was administered in full-length. Scores were age-normed in accordance with standard procedures.[33] WISC-III has been widely used in research and clinical work, and has shown high construct
Attention and inhibition. Attentional and inhibitory control was measured by the Test of Everyday Attention for Children (TEA-ch)\textsuperscript{35}. We used the Sky Search Task to measure selective attention, and inhibition was measured in the Opposite Word Task. In the Sky Search Task the child was given a printed display containing rows of pairs of spacecrafts and instructed to identify pairs of two identical crafts by circling them on the sheet (20 target pairs among 108 distractor pairs). The task was self-paced and selective attention was calculated as the time spent per identified target (the total time spent on the task divided by the number of identified space crafts). We adjusted for motor speed by subtracting time spent per identified spacecraft in a display that contained no distractors. The Sky Search task has shown high convergence validity with other commonly used measures of attention\textsuperscript{35}. Moreover, the task has been used to distinguish between healthy children and children with attention deficit disorders\textsuperscript{35}. The Opposite Word Task required the child to inhibit a pre-learned response. The child was presented with a list of 24 numbers (“1” or “2”) in a random sequence and asked to read the numbers out as quickly as possible (same word condition). In a second run, the child was required to inhibit the prepotent response by saying “2” whenever “1” was printed and vice versa (opposite word condition). Reaction time in the opposite word condition was used as a measure of inhibitory control. The Opposite Word Task has also shown high convergence validity with other measures of inhibition, and performance on this task is significantly correlated with the Stroop task\textsuperscript{35}.

Child social cognitive skills. Social cognitive skills were evaluated using the Social Cognition Scale\textsuperscript{36}. The scale was completed by the mother who responded to questions exploring various aspects of her child’s social cognitive skills e.g., awareness of other people’s feelings and understanding of social norms. The scale contains 12 items. A score of 0 indicates
that an item is not true, 1 indicates that an item is sometimes true and 2 indicates that an item is very or often true. Higher scores are indicative of decreased social cognitive skills\(^\text{[36]}\). The scale has shown high re-test reliability and high construct validity when scores are compared with other measures of social cognitive competences\(^\text{[37]}\). Moreover, the scale has been found to discriminate children with conditions characterized by social cognitive deficits such as Autism Spectrum Disorders\(^\text{[37]}\).

**Attrition and Missing Data.**

Of the original 14,541 mothers enrolled in the study, we included only the children (and corresponding mothers) who had data for the verbal IQ task on WISC, since this task that had the highest number of completions \((n=6979)\). Within the selected subsample, complete data was available for maternal depression, contextual risks, and interpersonal stress. For performance IQ, 0.4\% of the children \((n = 30)\) were missing. For attention and inhibition, 3.5\% of the children \((n = 246)\) were missing. For social cognitive skills, 15\% of the children \((n = 1045)\) were missing. Because listwise deletion of cases with partial complete data can increase sample bias\(^\text{[38]}\), missing data on the outcomes was replaced using full information maximum likelihood\(^\text{[38]}\). In accordance with Little and Rubin’s theory\(^\text{[39]}\), Mplus includes respondents with missing data using full information maximum likelihood estimation, which treats missing data as missing at random and allows the use of all available data\(^\text{[40]}\).

We tested, in a multivariate logistic regression, the degree to which the study variables predicted exclusion from the sample. Odds ratios (ORs) showed that mothers who were excluded from the current analysis had higher symptoms of depression (OR=1.06; 95\% CI=1.05-1.07), and experienced higher levels of contextual risks (OR=1.28; 95\% CI=1.22-1.34) and
interpersonal stress (OR=1.09; 95% CI=1.05-1.13). Of note, all three risks (i.e., maternal depression, contextual risk and interpersonal stress) were included in the overall analyses, which helps decrease bias related to missing data\(^{[39]}\).

Analysis

The analysis proceeded in two steps. In the first step an autoregressive cross-lag (ARCL) model was estimated (see Figure 1, top portion). The ARCL model allows for the simultaneous estimation of four basic parameters of interest: (1) auto-regressions, i.e., continuity in maternal depression, contextual risks, and interpersonal stress; (2) cross-lags, i.e., the effect of maternal depression on subsequent contextual risks and interpersonal stress, and vice versa; (3) within-time covariance of maternal depression, contextual risks, interpersonal stress, and the cognitive outcomes; and (4) predictions from maternal depression, contextual risks, and interpersonal stress to later child cognitive functioning. The ARCL model provides a conservative estimate of child outcome predictions given the auto, the cross and within-time measures.

In the second step of the analysis we examined four indirect pathways that could lead to decreased cognitive functioning (see Figure 1). The first two pathways (A and B) investigated Hammen’s model\(^{[24]}\), which suggests a bidirectional inter-relation between maternal depression and interpersonal stress. The subsequent two pathways (C and D) investigated paths going from increased contextual risks to either maternal depression or interpersonal stress, which then in turn increased the other. In Pathway A, maternal depression increased interpersonal stress, which in turn, increased maternal depression which negatively affected child cognitive functioning. In Pathway B, interpersonal stress increased maternal depression, which in turn, increased interpersonal stress, leading to decreased cognitive functioning. In Pathway C, contextual risks increased maternal depression, which then increased interpersonal stress, which negatively
impacted child cognitive functioning. Finally, in Pathway D, contextual risks increased interpersonal stress, which then increased maternal depression, which negatively affected child cognitive functioning.

PLEASE INSERT FIGURE 1

Indirect pathways were programmed in model constraint statements and were bootstrapped 10,000 times with bias-corrected confidence intervals. All analyses were carried out in Mplus version 5.1\textsuperscript{[41]}. Model fit was assessed through the Comparative Fit Index, the Tucker-Lewis Index (CFI and TLI; acceptable fit determined as $\geq .09$\textsuperscript{[42]}) and root mean square error of approximation (RMSEA; acceptable fit determined by $\leq .08$\textsuperscript{[43]}).

Results

Descriptive statistics

Correlations. Table 1 shows the correlations (and means and standard deviations) among the study variables. Firstly, maternal depression, contextual risks, and interpersonal stress were significantly correlated both within and between time points. Maternal depression and interpersonal stress were more highly correlated with each other, than they were with contextual risks. Correlations between the three risk factors (measured at age 2-4 years) and the outcome variables showed that, verbal and performance IQ were correlated with all three risks, but most highly correlated with contextual risks. Attention and inhibition were both highly correlated with maternal depression and contextual risks. Social cognitive skills were correlated with all three risk factors. All correlations indicated that higher levels of risks were associated with worse child outcomes.
Step 1: Autoregressive cross-lags

The ARCL model (Figure 2) with the five child outcomes (verbal IQ, performance IQ, attention, inhibition and social cognitive skills) showed acceptable fit to the data. $\chi^2(39) = 1016.984$, $p<.0001$; CFI = 0.958; TLI = 0.906; RMSEA = 0.060, 90% CI 0.057 -0.063; SRMR = 0.031.

PLEASE INSERT FIGURE 2

Interrelations among risks. Maternal depression, contextual risks, and interpersonal stress showed strong between-time continuity (i.e., auto-regressions). The cross-lagged predictions showed that maternal depression, contextual risk, and interpersonal stress largely predicted each other at subsequent time points. More specifically, maternal depression in pregnancy predicted both interpersonal stress and contextual risks at child age 0-2; maternal depression at child age 0-2 predicted subsequent (age 2-4) interpersonal stress, but not contextual risks; and contextual risks predicted interpersonal stress at subsequent time points.

Direct risk associations with cognitive outcomes at age 8. Three results are highlighted. Firstly, maternal depression significantly predicted all cognitive outcomes, but was most strongly associated with social cognitive skills. Secondly, contextual risks significantly predicted social cognitive functioning and cognitive outcome on all measures except for attention. In addition contextual risk was a particularly strong predictor of performance IQ and verbal IQ. Thirdly,
interpersonal stress predicted social cognitive skills only.

**Step 2: Indirect effects**

The indirect effects (Table 2) showed that two pathways were significantly associated with decreased performance IQ and social cognitive skills: (1) Pathway A, which tested the effect of maternal depression increasing interpersonal stress, which then increased maternal depression; and (2) Pathway D, which tested contextual risks increasing interpersonal stress which then increased symptoms of maternal depression.

**PLEASE INSERT TABLE 2**

**Discussion**

Within an integrated developmental model, which controls for a variety of potential confounds (i.e., auto regressions, cross-lags and within-time associations), we examined four developmental pathways through which maternal depression, contextual risks and interpersonal stress may impact on child cognitive and social cognitive skills. A strength of the current study is that we tested the developmental inter-relationships amongst risks that may affect child functioning in different manners[22]. Furthermore, we tested these developmental associations for a range of cognitive functions including executive functions and social cognitive skills. Our findings increase the current knowledge on risk-to-outcome association on child cognitive abilities in three main ways.

First, the present study examined the developmental inter-relationships of maternal depression, contextual risks, and interpersonal stress. Our findings suggest strong continuity
within each risk factor, and strong inter-relations among risks such that one risk factor (e.g., maternal depression during pregnancy) predicted other risk factors (e.g., interpersonal stress) at subsequent time points. Hence, this research lends an important perspective on how risk factors may developmentally maintain and reinforce each other. Importantly, we note that the inter-relations among risks suggest that risks that are distal to the child’s social environment (i.e., contextual risks), can work to maintain risks that are more proximal to the child’s social environment (i.e., maternal depression and interpersonal stress).

Second, with regard to direct associations, the present study found that maternal depression and contextual risks negatively impacted almost all of the cognitive abilities assessed, as well as the children’s social cognitive skills. This finding supports previous research where maternal depression and contextual risks have been found to each independently affect child cognition\[^{8,10,22}\]. More specifically, however, we found that the main difference between maternal depression and contextual risks was that only maternal depression (which is more proximal to the child’s immediate social environment) was associated with child attention. This may support previous suggestions that the child's social environment is key to the development of attentional capacities\[^{44}\]. This finding is intriguing since attention, as measured here, has previously been associated with attention deficit disorder\[^{45}\], which has also been associated with risks relating to the social environment\[^{46}\].

Maternal depression and interpersonal stress were found to have a larger effect on the child’s social cognitive skills than contextual risks. Hence, social cognitive functioning, which is known to relate to autism\[^{47}\], depression\[^{48}\] and conduct problems\[^{49}\], appears to be particularly sensitive to disruptions in the immediate social environment of children (i.e., maternal depression and interpersonal stress). Of interest, we found that, compared to maternal
depression, contextual risks were most strongly associated with verbal IQ. The contextual risks index used in the current study includes factors relating to parental education and economic resources, which have previously been associated with below average language development in children\textsuperscript{[50]}. Our findings may support the notion that crystallized measures of intelligence, such as verbal IQ, are strongly associated with factors related to contextual risks, such as the decreased amount of cognitive stimulation related to the education and resources of the parents, as measured in the current contextual risks variable\textsuperscript{[51,52]}.

The third way the current research contributes to the extant knowledge on risk exposure and child cognitive abilities is through the examination of the four developmental pathways (i.e., indirect effects). In reminder, we investigated four developmental pathways through which maternal depression, contextual risks, and interpersonal stress could interrelate to affect children’s cognitive and social functioning. Indirect pathways give an important perspective on how risks developmentally inter-relate to affect child outcomes\textsuperscript{[48]}. Of note, we found that although interpersonal stress did not directly affect children’s cognitive functioning, it was implicated in two indirect pathways (A and D). First, in support of Hammen's Stress Generation Model\textsuperscript{[24]}, we identified a bidirectional relationship between maternal depression and interpersonal stress\textsuperscript{[16]} where maternal depression increased interpersonal stress, which, in turn increased maternal depression, which then impacted child performance IQ and social cognitive skills. The second significant developmental pathway was contextual risks increasing interpersonal stress, which increased maternal depression, which then associated with decreased performance IQ and social cognitive skills. This pathway underscores that contextual risks can affect factors related to the social environment (i.e., interpersonal stress and maternal depression), which can then affect the cognitive and social functioning of children. A recent
study found that unfavorable economic conditions in the first year of life had adverse impactions for children’s psychological development and behavioral problems\textsuperscript{[53]}. Future studies could look into whether such effects are related to the effect of contextual risks on risks that are more proximal to the social environment of the child.

\textit{Limitations.} The current study should be interpreted in light of five main limitations. First, measures of maternal depression, contextual risks, interpersonal stress, and the child’s social cognitive skills were based on maternal reports, introducing the problem of shared methods variance. Second, maternal reports on child social cognition may be affected by the fact that some mothers qualified for a clinical diagnosis of depression\textsuperscript{[18]} and may consequently rate their children more negatively. Still, a recent meta-analysis showed no difference in maternally rated child outcomes in depressed and non-depressed mothers\textsuperscript{[48]}. Third, the risk measures were collected at least 4 years prior to the cognitive assessments at age 8, therefore, unmeasured factors more proximal to the assessments may have influenced the cognitive outcomes. Still, it is important to note that, given the strong continuity of the risk variables over time, these risks undoubtedly still negatively affected the child at times proximal to the age 8 assessments. Fourth, the present study is correlational in nature and the indirect pathways should not be considered causative. Relatedly, all of the significant prospective associations (direct and indirect) between risk exposures and child outcomes were small in effect size, and hence should no be considered deterministic of a child’s cognitive or social functioning. Fifth, similar to most large longitudinal cohorts, the Avon Longitudinal Study of Parents and Children has faced attrition over time. As might be expected, mothers high in depressive symptoms with co-occurring contextual risk and interpersonal stress were more likely to have children who did not complete the cognitive assessments. Therefore, the present sample underrepresents families at
the highest risk. However, a recent study based on the Avon Longitudinal Study of Parents and Children cohort\textsuperscript{[54]} confirmed that although attrition affected the prevalence rates of antisocial behaviors and related disorders, associations between risks and outcomes remained intact (although conservative estimates of the likely true effects).

Conclusion

The current study adds to the extant knowledge of how risk factors during pregnancy, infancy, and early childhood are developmentally associated with decreased cognitive functioning in childhood. Such developmental understanding of early risk exposure is critical for our understanding of risk-to-outcome relations, and may have important direct implications for early intervention strategies\textsuperscript{[48]}. Our results indicate that early maternal depression and contextual risks directly affect children’s cognitive and social functioning later in life. Moreover, we found that interpersonal stress contributed to developmental pathways that enhanced the effects of maternal depression and contextual risks on children’s cognitive outcome and social cognitive skills. Our results illustrate the importance of both contextual risks, and risks that are more proximal to the social environmental of the child (i.e., maternal depression and interpersonal stress), supporting the notion that the social environment represents an important developmental context during early development\textsuperscript{[55]}. Given the dependence of children on the mother early in development\textsuperscript{[9,44]}, environmentally focused interventions often address the circumstances of the mother as a proxy for the social environment of the child\textsuperscript{[57]}. The current research adds to the field by suggesting that helping mothers in high-risk environments manage their interpersonal stress may benefit the child by reducing the effect of maternal depression and contextual risks on the child’s cognitive and social functioning.
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References


32. HMSO. Office of Population and surveys, Standard Occupational classification (vol. 3); 1991.


41. Muthen LK, Muthen BO. Mplus. Syatistical analyses with latent variables. User’s guide. Los Angeles: Muthen & Muthen


Table 1. Correlations of the study variables across time of measurement. All correlations are significant unless indicated otherwise. Means and SD of the study variables are also reported.

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<td>5. C. risks (0-2 years)</td>
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<td>0.139</td>
<td>0.167</td>
<td>0.177</td>
<td>0.223</td>
<td>0.435</td>
<td>1.000</td>
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<td></td>
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</tr>
<tr>
<td>10. Verbal IQ</td>
<td>-0.034</td>
<td>-0.038</td>
<td>-0.051</td>
<td>-0.065</td>
<td>-0.102</td>
<td>-0.150</td>
<td>-0.033</td>
<td>-0.040</td>
<td>-0.046</td>
<td>1.000</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>11. Performance IQ</td>
<td>-0.032</td>
<td>-0.040</td>
<td>-0.057</td>
<td>-0.043</td>
<td>-0.067</td>
<td>-0.095</td>
<td>-0.024</td>
<td>-0.031</td>
<td>-0.034</td>
<td>0.499</td>
<td>1.000</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>12. Attention</td>
<td>0.012†</td>
<td>0.017†</td>
<td>0.026</td>
<td>0.011†</td>
<td>0.018†</td>
<td>0.025</td>
<td>0.004†</td>
<td>0.003†</td>
<td>-0.009†</td>
<td>-0.151</td>
<td>-0.270</td>
<td>1.000</td>
<td></td>
<td></td>
</tr>
<tr>
<td>13. Inhibition</td>
<td>0.013†</td>
<td>0.018†</td>
<td>0.027</td>
<td>0.014†</td>
<td>0.022†</td>
<td>0.032</td>
<td>0.007†</td>
<td>0.007†</td>
<td>0.001†</td>
<td>-0.200</td>
<td>-0.262</td>
<td>0.237</td>
<td>1.000</td>
<td></td>
</tr>
<tr>
<td>14. Social cognition</td>
<td>0.080</td>
<td>0.115</td>
<td>0.174</td>
<td>0.042</td>
<td>0.055</td>
<td>0.066</td>
<td>0.113</td>
<td>0.068</td>
<td>0.101</td>
<td>-0.125</td>
<td>-0.107</td>
<td>0.078</td>
<td>0.059</td>
<td>1.000</td>
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</table>

Mean       | 7.11  | 5.98  | 6.28  | 0.51  | 0.66  | 0.52  | 0.37  | 0.60  | 0.24  | 107.22| 99.67 | 5.20  | 17.45 | 2.83  |
SD         | 4.63  | 4.44  | 5.05  | 0.81  | 0.92  | 0.80  | 0.74  | 0.94  | 0.55  | 16.75 | 17.11 | 5.65  | 5.65  | 3.72  |

Mat. dep. = maternal depression, C. risks = contextual risks, Interp. Stress = interpersonal stress, IQ= Intelligence Quotient, † = n.s. (p > 0.05), SD = Standard Deviation.
Table 2. Indirect effects of maternal depression, contextual risks and interpersonal stress on the five child cognitive outcomes. Significant pathways are denoted by a star and highlighted in bold, significant effects on outcomes are highlighted in bold.

<table>
<thead>
<tr>
<th>Path</th>
<th>Preg.</th>
<th>0-2 years</th>
<th>2-4 years</th>
<th>Verbal IQ 95% CI</th>
<th>Performance IQ 95% CI</th>
<th>Attention 95% CI</th>
<th>Inhibition 95% CI</th>
<th>Social cognition 95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Est.</td>
<td>LL</td>
<td>UL</td>
<td>Est.</td>
<td>LL</td>
<td>UL</td>
<td>Est.</td>
<td>Low</td>
</tr>
<tr>
<td>Path A*</td>
<td>Mat. Dep.</td>
<td>Interp. stress</td>
<td>Mat. Dep.</td>
<td>-0.001</td>
<td>-0.002</td>
<td>0.000</td>
<td>-0.002</td>
<td>-0.003</td>
</tr>
<tr>
<td>Path B</td>
<td>Interp. stress</td>
<td>Mat. dep.</td>
<td>Interp. stress</td>
<td>-0.001</td>
<td>-0.003</td>
<td>0.000</td>
<td>0.000</td>
<td>-0.002</td>
</tr>
<tr>
<td>Path C</td>
<td>C. Risks</td>
<td>Mat. dep.</td>
<td>Interp. stress</td>
<td>0.000</td>
<td>-0.002</td>
<td>0.000</td>
<td>0.000</td>
<td>-0.002</td>
</tr>
<tr>
<td>Path D*</td>
<td>C. Risks</td>
<td>Interp. stress</td>
<td>Mat. Dep.</td>
<td>-0.003</td>
<td>-0.007</td>
<td>0.000</td>
<td>-0.005</td>
<td>-0.010</td>
</tr>
</tbody>
</table>

**Figures**

**Figure 1.** Schematic representation of the models used in the current study. The top portion of the figure illustrates the full ARCL model and highlight within it the indirect pathway A. Arrows represent predictions tested in the model, and grey scaling is used to highlight the risk variables tested within this pathway. The bottom portion of the figure illustrates the remaining three indirect pathways tested in the current study (B, C and D) in diagrams isolated from the full model due to space restrictions.
Figure 2. ARCL model showing interrelations among the three risk factors (maternal depression, interpersonal stress and contextual risks), and predictions from risks age 2-4 years to the five cognitive outcomes at age 8. Arrows represent predictions. Values are the standardized path coefficients. All included parameters are significant at p < 0.05.