Objective: Research suggests that early life adversity may affect subsequent parenting. Animal studies investigating mechanisms of transmission have focused on biological factors; whereas research in humans has emphasized cognitive and psychosocial factors. We hypothesized that neuropsychological and physiological factors would act as mediators between maternal retrospective reports of early life experiences (ELE) and current parenting. Method: We recruited a community sample of 89 mothers and their infants (2–6 months of age). Maternal ELE consisted of self-reports of consistency of care and childhood maltreatment. Diurnal salivary cortisol samples were collected as the measure of hypothalamic-pituitary-adrenal (HPA) function. Executive function measures included attentional set-shifting and spatial working memory. Maternal sensitivity was assessed through videotapes of mothers interacting with their infants. Results: A series of path analyses indicated that maternal ELE was indirectly related to maternal sensitivity via two pathways: one through HPA function, and the other through HPA function and spatial working memory. There was no direct path between maternal ELE and parenting. Conclusion: These findings provide support for the notion that mediators linking early life experiences to parenting in humans may be similar to physiological mechanisms found in animal models. As maternal care is associated with numerous infant outcomes, our findings may have broad relevance to understanding the risk associated with parenting and adverse outcomes in infants. A greater understanding of mechanism is important to informing interventions targeted at disrupting maladaptive trajectories of parenting. J. Am. Acad. Child Adolesc. Psychiatry, 2012;51(7): 673–682. Key words: early life experiences, parenting, diurnal cortisol, executive function

Parenting is affected by multiple factors, including early life experiences. Despite evidence that this relationship exists, less is known about pathways influencing the development of parenting. In humans, proposed mechanisms have been social and cognitive in nature or have included psychosocial risk factors; whereas in animals the focus of mechanism has been physiological. Understanding how early life experience effects mediate parenting has broad relevance for outcomes in infants. The purpose of this article is to propose a model of physiological and neuropsychological factors as mediators linking maternal early experiences to maternal sensitivity.

Early Experience and Parenting in Humans
There is much evidence to support the notion that the way one was parented in childhood influences later parenting. Proposed mechanisms to date include mediators such as training and observational learning, presence of antisocial behaviors, and parental social competence. Attachment theorists argue that early experience influences parenting via internal working models. These models are mental representations of the world and oneself within it; they develop early in life and tend to remain stable into adulthood, influencing later interpersonal relations. Others studies have found that psychosocial factors are significant mediators.
doubtedly, biology subserves both mental representations and behavior; yet, neurobiological explanations have largely been overlooked.

Early Experience and Maternal Behavior in Non-Human Animals

Studies in animals have established that early experiences are related to later maternal behaviors via alterations in biological systems. In rodents, maternal behavior deficits are related to the degree of maternal deprivation received as a neonate and to natural variations in maternal care. These behavioral differences are accompanied by biological variations including changes to the hypothalamic–pituitary–adrenal (HPA) axis. Maternal care patterns are also related across generations in monkeys and have been associated with serotonergic functioning and increased cortisol levels. Do similar mechanisms mediate the association between early experience and parenting in humans? We hypothesized that two variables, HPA axis and executive function, are likely candidates.

Early Life Experiences: Influence on HPA and Executive Functions

Early experiences produce long-lasting changes in both stress reactivity and diurnal levels of HPA function. Variations in diurnal cortisol profiles are reported in children exposed to maternal stress, in maltreated children, and in anxiously attached children. Adults who experienced low parental care, maltreatment, and multiple forms of early adversity also show alterations in HPA function. In addition, cortisol increases in response to a stressor have been found in insecurely attached infants and disorganized infants.

Our second pathway from maternal early experiences to current parenting is via alterations in executive function. Maternal deprivation in rodents is associated with deficits in attention and spatial learning. In humans, the quality of early parenting is related to attentional processes in children. Lower levels of maternal sensitivity were related to poorer working memory, impulse control, and cognitive flexibility.

In our model, we contend that variations in executive function performance likely occur through alterations in HPA function. A direct pathway from early experiences to executive function is conceivable but presumably involves a change in a biological substrate, such as the PFC. Executive function is primarily mediated by the frontal lobes, specifically the PFC. Chronic elevations in glucocorticoids may cause damage to prefrontal and limbic regions. The two types of corticosteroid receptors are distributed abundantly in the PFC, thus rendering it sensitive to high levels of glucocorticoids. Glucocorticoids have been linked to impairments in spatial working memory and executive function in both animals and humans.

Associations Among Maternal Sensitivity, HPA, and Executive Function

The second step in our model involves the association among maternal sensitivity, HPA, executive function. The HPA axis has been implicated in maternal behavior and attachment relations. Findings in rodents link attention and executive function to maternal behavior. In humans, emotional Stroop reaction time and self-report attentional strategies were related to maternal sensitivity. Furthermore, poorer working memory was associated with greater reactive negativity in mothers.

The Current Study

The goal of this study was to propose a model examining the impact of maternal reports of early experiences on maternal sensitivity through HPA and executive function. Specifically, we examined cognitive flexibility and spatial working memory as measures of executive function. Our selection of these two measures was guided by evidence that these measures may be sensitive to early experiences and related to parenting in animals or humans and may be related to HPA function. Mediation path analyses were conducted adhering to the current practices and recommendations, using a test of indirect effects and bootstrapping.

METHOD

Study Participants

Postpartum mothers were recruited through two primary sources: Ontario Early Years Centres in Toronto, Ontario, Canada; and the maternity ward at St. Joseph’s Healthcare, Hamilton, Ontario. The study was approved by the Research Ethics Board of St. Joseph’s Healthcare and the University of Toronto and written informed consent was obtained from each participant. A total of 89 women aged 24–42 years (mean = 31.8,
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SD = 4.1) participated in the study. All mothers completed the study. The women ranged from 2 to 6 months postpartum at the time of testing (mean = 4.0, SD = 1.4). Of the women, 71.1% were first-time mothers, and 52% of the women had female infants. The majority of mothers had completed some form of post-secondary education; 16.8% had completed graduate studies, 61.8% had finished college or university, 13.3% had completed some college or technical training, and 7.9% had completed high school. Family income ranged from $20,000 to more than $100,000, with 7.8% reporting family incomes less than $50,000, 34.9% of the mothers reporting incomes less than $70,000, and 57.3% reporting family incomes greater than $70,000. The majority of women were of white ethnicity (67.2%), with Asian women (13.2%) making up the next largest represented ethnic group. The remainder of the participants were Hispanic (12.0%), East Indian (3.8%), and mixed ethnicity (3.8%).

Procedure
Mothers were tested during two home visits spaced approximately 1 week apart. During the first home visit, the Montgomery-Asberg Depression Rating Scale (MADRS),40 the Hamilton Anxiety Scale (HAM-A),41 and the Edinburgh Postnatal Depression Scale (EPDS)42 were administered. In addition, mothers were administered self-report inventories assessing early life adversity and eliciting background information. Mothers were videotaped with their infants during a 20-minute free-play interaction, followed by a 10-minute divided attention task. Between visits 1 and 2, mothers were given a study pack consisting of standardized written instructions for salivary collection and 16 saliva-sampling tubes (salivettes, Sarstedt Canada, Inc., St. Laurent, PQ) for collection of diurnal cortisol samples across 2 consecutive days. During the second home visit participants were tested in the comfort of their homes. During this visit the National Adult Reading Test (NART)43 and CANTAB battery44 were administered.

Concepts and Measures
Early Life Experiences. Although there are multiple types of adversity in childhood,45,46 we concentrated on two types consistently shown to be associated with negative outcomes: maltreatment and parental loss.46 Our measures of early life experiences (ELE) consisted of retrospective reports of childhood maltreatment (Childhood Trauma Questionnaire [CTQ])47 and consistency of care in the family of origin (Life History Calendar).48 We examined the cumulative effect of early life experiences because several studies have demonstrated a positive relationship between the number of risk factors during childhood with psychosocial adjustment49 and physical health problems.50 This graded relationship between multiple early experiences and health outcomes has been theoretically linked to cumulative exposure on the stress system and associated areas.50 We endeavored to examine whether psychobiological measures may mediate between cumulative ELE and parenting quality.

The CTQ47 is a 28-item self-report questionnaire retrospectively assessing the frequency and severity of different types of childhood abuse experiences. Psychometric properties have been shown to be good.47,51 Cutoff scores in the manual for none to low, low to moderate, moderate to severe, and severe to extreme exposure are provided for each scale of five scales. We used moderate to severe cutoff scores for each subscale to classify women as positive for history of childhood maltreatment. Most women did not experience maltreatment, with 71.9% reporting none to moderate across the five subscales.

The LHC provides a visual representation of every year of the person’s childhood, from birth to age 16 years. Record is made of where the individual was living and who else was living in the house during each year. Our measure included whether the mother had lived with both biological parents for the first 16 years of life (consistency of care). With respect to the LHC, 66.3% of participants reported consistent care, living with both biological parents.

To assess the impact of adverse ELE, a cumulative measure combining the two types of ELE was created. Using the standard cut-offs in the CTQ manual (none to moderate = 0, moderate to extreme = 1) and the inconsistent care measure on the LHC (consistent care = 0, inconsistent care = 1), women were stratified into one of three groups. Women were classified as having experienced both maltreatment and inconsistent care (n = 14), as either having experienced one form of early adversity (n = 27) or having experienced neither (n = 48).

Salivary Cortisol Sampling. To measure the diurnal cortisol rhythm, six samples were collected after awakening at 0800 h, 0830 h, 1000 h, 1600 h, 1800 h, and 2100 h. Samples were collected over 2 consecutive days using salivettes. All subjects were provided with detailed verbal and written explanations of the procedure for sample collections. Given that numerous substances affect cortisol,52 subjects were asked to refrain from brushing their teeth, smoking, eating, and drinking 60 minutes before taking each sample. In addition, previous research has demonstrated that salivary cortisol levels decrease within 15 minutes after breast-feeding53, therefore, all women were instructed either to collect their samples before breast-feeding or to wait 1 hour after feeding before collecting the sample.

Cortisol sample compliance was good, with a range of 1.1% to 8.9% of any individual sample missed on one of the 2 days. A missing value was interpolated with the mean of sample concentrations on either side.
of the missing value. The primary cortisol measure used in the model was the trapezoidal area under the curve (AUC$_C$) to yield an overall measure of diurnal cortisol secretion controlling for time of awakening. The AUC$_C$ is used as a measure of total hormonal output, and is one of the best methods of assessing diurnal cortisol because it incorporates different aspects of time course information of repeated measures into a single measure that can be easily related to other variables in the model.

Cortisol Assay. Samples were stored in the subject’s freezer until they were transferred to the laboratory and frozen at $-20^\circ$C until assayed. On the day of the assay, salivettes were centrifuged for 10 minutes, at 3,000 rpm at 4$^\circ$C. All samples were assayed in duplicate using a high-sensitivity salivary cortisol enzyme immunoassay kit (Salimetrics, State College, PA) for quantitative measurement of salivary cortisol. Samples from each participant were assayed in the same batch. The interassay variability was 10.6%; the intra-assay variation was 8.3% for samples with low values and 6.9% for samples with high values.

CANTAB. The testing period occurred over a 2-hour period, during which time the National Adult Reading Test (NART) and CANTAB battery were administered. CANTAB tasks were run on a Dell Latitude 810 laptop computer with a Microvitec 501 touch-sensitive screen. Participants were introduced to the apparatus by way of a motor screening task. All participants completed two subtests from the CANTAB, attentional set-shifting and spatial working memory. For practical reasons (to accommodate the dyads’ schedule), participants were assessed at various times of day. Time of day may be related to executive function performance and set-shifting and spatial working memory. For practical reasons, we recorded and assessed it statistically as a potential covariate.

Attentional Set-Shifting (ID/ED). This task includes a series of visual discrimination derived from the Wisconsin Card Sorting Test. Participants are presented with two dimensions, shapes, and lines and are trained to discriminate between dimensions on the basis of trial and error feedback. The test consists of nine stages, progressing in complexity from simple discrimination (two purple shapes) through to extradimensional shifts (ED; shifting attention from the purple shapes to the white lines). The performance index for this task included the number of errors on the extradimensional shift stage (ED shift errors), a measure of cognitive flexibility.

Spatial Working Memory. This is a self-ordered search task measuring a subject’s ability to retain spatial information and to manipulate remembered items in working memory and assesses heuristic strategy. The goal of this task is to collect blue tokens hidden inside a series of boxes. The number of boxes gradually increases in difficulty, from four to eight boxes. Subjects are instructed to search through boxes for the hidden tokens without returning to a box that has already contained a token. Efficiency of a strategy is computed by counting the number of times that the subject begins a new search with the same box for six- and eight-box problems. Higher scores are indicative of less efficient use of this strategy. The performance index was the strategy score.

Maternal Sensitivity Ratings. Mothers were videotaped individually with their infants for a period of 30 minutes in their homes. After 20 minutes of free-play interaction, mothers were presented with a short questionnaire while the infant was still in their presence, thereby producing an ecologically valid divided attention task, so that each mother would have to allocate her attentional capacity between completing the questionnaire and responding to any infant cues during the remaining 10 minutes of video recording. Maternal sensitivity was later coded using the Ainsworth Maternal Care Scales (MCS). The MCS consists of four rating scales (sensitivity, accessibility, cooperation, and acceptance); ratings are assigned along a nine-point Likert scale, with higher scores indicating higher quality of maternal interactions. The four scales were highly correlated, with Pearson correlation coefficients ranging from $r = 0.87$ to 0.96. The scales were averaged to create a single score of sensitivity. A primary coder (A.G.) classified all cases, and a second coder independently coded 20 cases; inter-rater reliability for the average sensitivity score was $k = 0.82$. The MBQS has been validated against infant attachment security with an effect size of $r = 0.48$.

Covariates: Depression and Anxiety Measures. To control for the impact of symptoms of depression and anxiety we administered the EPDS, the MADRS, and the HAM-A; all instruments have been shown to have good reliability and to be valid measures of maternal depression and anxiety.

Covariates: Sociodemographic Factors. To examine demographic variables as possible covariates, we included the following measures as dummy variables: parity and feeding status (i.e., breast versus bottle). In addition, demographic information was obtained on maternal age, education, and household income. To obtain an indicator of SES, family income was divided into seven categories ranging in increments from the lowest (i.e., less than $10,000) to the highest (i.e., $100,000 or more) income categories.

Statistical Analyses. Before the primary analyses, cortisol data were excluded if values were greater than three standard deviations from the mean. Square-root transformations were applied to correct the moderate positive skew in the data; this transformation provided the best correction. Area under the curve for diurnal cortisol was calculated using the trapezoidal method. Total hormonal output (AUC$_C$) for diurnal cortisol profile was calculated separately for days 1 and 2, and the mean was taken. Distributions of the CANTAB data were compared using t-tests.
variables were analyzed before statistical analyses; ED shifting errors was log transformed.

For our main analyses, we performed path analysis with maximum likelihood estimation (MLE), using AMOS 7.0.62 Model fit was tested with multiple indices including the $R^2$ goodness of fit, the root mean square error of approximation (RMSEA), and the Comparative Fit Index (CFI). CFI values greater than 0.95 and RMSEA values less than 0.06 indicate excellent model fit.63,64 CFI and RMSEA are among the measures least affected by sample size. 64

Three models were proposed to examine the direct and indirect relations between maternal ELE and sensitivity. First, we tested whether there was a direct effect between maternal ELE and parenting (model 1: Direct Effect of ELE on Maternal Sensitivity). For this model, we estimated the direct path between maternal ELE and parenting and the paths between maternal ELE and HPA and executive function. We then tested our hypothesized model (model 2: Indirect Effect via HPA Function) examining the indirect effect between ELE and maternal sensitivity via HPA function (ELE to HPA to EF to maternal sensitivity). Subsequently, we tested whether the relation between ELE and parenting was indirectly related through executive function (model 3: Indirect Effect via Executive Function); we expected that this model would not result in a good fit, because we contend that although ELE may be related to executive function, this association likely occurs through changes in HPA function. Depression has been linked to HPA function,65 executive function,66 and parenting67; therefore depression measures were included as covariates in all models. Time of awakening was also included as a control variable to account for varying wake times on our diurnal measure of cortisol.

RESULTS
Preliminary and Descriptive Analyses
Demographic data and mood scores were compared between the three ELE groups using one-way analyses of variance (ANOVA; maternal and infant age, EPDS, MADRS, HAM-A) and $R^2$ tests (parity, breast-feeding status, education, and household income). There were no significant differences between groups with respect to age, parity, breastfeeding status, education, and household income. There were no significant differences between groups with respect to age, parity, breastfeeding status, education, depression (EPDS, MADRS), or anxiety (HAM-A) (Table 1). However, there was a significant difference between groups for household income [$R^2(12, n = 89) = 23.76, p < .05$]. The means for the mood measures were all within the normal range.

Correlations
Intercorrelations between the major variables are reported in Table 2. Maternal sensitivity was significantly related to AUCG diurnal cortisol ($r = −0.26, p < .05$), SWM strategy score ($r = −0.38, p < .01$), and ED shifting errors ($r = −0.26, p < .05$). Maternal sensitivity was also correlated with scores on the EPDS ($r = −0.23, p < .05$) and the MADRS ($r = −0.21, p < .05$), but not with HAM-A scores. Significant relations between maternal sensitivity, HPA function, and the CANTAB variables remained even when maternal depression was partialled out. Although there were significant correlations between ED errors and maternal sensitivity, there were low...
correlations between ED errors and maternal ELE and between ED errors and HPA function (Table 2). Cognitive flexibility (ED errors) was not a significant mediator in the relation between maternal ELE and maternal sensitivity when tested in a preliminary path analyses, providing a poor fit. Therefore, to increase model parsimony, we tested spatial working memory strategy only as a mediator. Before model testing, we examined correlations between the demographic variables, mood measures, and other model measures. As mentioned above, sensitivity was significantly correlated to depression scores; however, no other covariates correlated significantly with cortisol or SWM; therefore depression, household income, and time of awakening were controlled for in subsequent analyses.

Path Analyses

Model 1: Direct Effect of ELE on Maternal Sensitivity. In the first model, we tested whether there was a direct effect between maternal ELE and parenting. There was not a significant association found between maternal ELE and sensitivity ($\beta = 0.08$, SE = 0.14) and fit indexes suggest an overall poor fit of the model [$\chi^2 (8, n = 89) = 32.30, p = .0001, CFI = 0.10, RMSEA = 0.19$]. The absence of a significant direct pathway between maternal ELE and sensitivity indicates that there may be an indirect effect between ELE and maternal sensitivity and that HPA function and spatial working memory may act as mediators. Traditionally, the causal steps strategy popularized by Baron and Kenney (1986) has been used to test mediation\(^68\); however, these criteria have recently been disputed, with new approaches not contingent on the first step demonstrating a direct and significant association between the predictor and outcome variable.\(^39\) Instead, modern techniques include the use of bootstrapping to test for indirect effects over the causal steps approach and the Sobel test.\(^69\)

Model 2: Indirect Effect via HPA Function. In the second model, we tested the role of HPA and spatial working memory as mediators through a pathway from HPA function to maternal sensitivity, and through a pathway from HPA to spatial working memory to maternal sensitivity. This model fit the data well [$\chi^2 (5, n = 89) = 5.53, p = .36, CFI = 0.98, RMSEA = 0.04$]. The relative effects include the following (Figure 1): maternal ELE to HPA ($\beta = 0.27, SE = 0.13, p < .05$); HPA to maternal sensitivity ($\beta = -0.23, SE = 0.11, p < .05$); HPA to spatial working memory ($\beta = 0.24, SE = 0.11, p < .05$); and spatial working memory to maternal sensitivity ($\beta = -0.32, SE = 0.10, p < .01$).

As a further test of the indirect effect of HPA function and spatial working memory for

<table>
<thead>
<tr>
<th>Table 2</th>
<th>Means, Standard Deviations, and Intercorrelations of the Main Variables in the Primary Analyses</th>
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<tbody>
<tr>
<td>Variable</td>
<td>Mean (SD)</td>
</tr>
<tr>
<td>1. Maternal ELE</td>
<td>—</td>
</tr>
<tr>
<td>2. HPA function (diurnal AUC)</td>
<td>6.67 (7.77)</td>
</tr>
<tr>
<td>3. Cognitive flexibility (ED shift errors)</td>
<td>28.57 (6.08)</td>
</tr>
<tr>
<td>4. Spatial working memory (strategy score)</td>
<td>5.87 (2.05)</td>
</tr>
<tr>
<td>5. Maternal Sensitivity</td>
<td>—</td>
</tr>
</tbody>
</table>

Note: AUC = area under the curve; ED = extradimensional; ELE = Early life experience; HPA = hypothalamic–pituitary–adrenal; SWM = spatial working memory.

*p < .05, **p < .01.

FIGURE 1 Path analysis model testing hypothalamic–pituitary–adrenal (HPA) function and spatial working memory as mediators in the association between maternal early life experiences (ELE) and maternal sensitivity. Note: Standardized path coefficients of the nonsignificant direct effect model (Model 1, as indicated by dashed line) and the final selected model are shown (Model 2, as indicated by solid line). *p < .05, **p < .01.
model 2, we conducted a follow-up analyses using bias-corrected bootstrapping technique. We found that the indirect pathway was significant including links between maternal ELE and HPA function (95% CI = 0.064, 0.378, p < .01); HPA function and maternal sensitivity (95% CI = −0.420, −0.020, p < .05); and spatial working memory and maternal sensitivity (95% CI = −0.325, −0.511, p < .01). The pathway between HPA function and spatial working memory approached significance (95% CI = −0.018, 0.416, p = .07).

Model 3: Indirect Effect via Executive Function. The third model was based on the assumption that maternal ELE may be related to maternal sensitivity indirectly through spatial working memory; and indirectly from spatial working memory to HPA function to maternal sensitivity. This model resulted in a relatively poor fit [χ²(5, n = 89) = 9.48, p = .09, CFI = .83, RMSEA = .10]. The path from maternal ELE to spatial working memory (β = 0.10, SE = 0.14, p = .48) was not significant.

DISCUSSION
This is the first study to examine the association between maternal early life experiences and current parenting via HPA and executive functions in humans, thereby supporting the notion that early experiences may be related to parenting via similar psychobiological mechanisms found in animal models. In particular, we found that higher levels of diurnal cortisol served as a mediator between maternal reports of adverse early experiences and greater insensitive parenting. In line with animal models, our model supports that early experiences may be associated with later parenting through HPA function.

In our second pathway, we found that early experiences were indirectly related to parenting through HPA and executive function. Specifically, higher levels of diurnal cortisol were related to poorer performance on the spatial working memory task, which in turn was associated with lower sensitivity. Previous studies have shown that higher levels of cortisol in response to a stress test impair cognitive flexibility and working memory. Although we did not administer a stress test in our study, it is possible that higher levels of diurnal cortisol may subvert executive function processes that are necessary to parent effectively. In our model we propose HPA function as a possible biological mediator; however, we recognize there are a number of other potential candidates.

This is the first study highlighting the associations between executive processes and maternal sensitivity early in the postpartum period. In particular, less sensitive parenting was associated with a poorer strategy in spatial working memory and less cognitive flexibility. Maternal sensitivity involves behaviors that are contingent, timely, and appropriate. These behaviors are complex, involving cross-situation adaptations. During interactions with their infants, the relevance of executive functions appears obvious; mothers must be able to recognize and attend to their infant cues and to integrate environmental demands with the needs of their infant, all requiring an ability to hold information on-line (working memory) and flexible strategizing (cognitive flexibility).

There are several potential limitations associated with the current study. First, retrospective reporting of early experiences are plagued with concerns and qualifiers. Retrospective reports have been found to be valid when serious and objective events are reported, e.g., abuse, and dating tools are used to support the memory, as in the Life History Calendar. We incorporated two measures of early experiences that, although retrospective, adhere to these principles. In addition, for the purposes of generalizability, we combined our ELE measures; however, our current research is examining more specific aspects and a broader range of adverse childhood experiences. There is also the possibility that a heritable factor may contribute to these findings; however, this cannot be assessed with the current design. Secondly, our sample was a relatively small, low-risk community sample; thus, relations established in this study may not generalize to dyads experiencing considerable adversity. Third, our maternal observation occurred at a single time point. Although we were able to achieve considerable variability in sensitivity ratings, future studies should follow dyads across a number of years. Assuming that parenting becomes more challenging as infants get older, perhaps greater discrepancies in maternal functioning will be evident later.

In summary, despite these limitations, findings of this study support the notion that parental stress (HPA function) and executive function
may be important factors associated with parenting in humans. This study provides evidence of the importance of parenting programs in addressing stress reduction and executive function to promote more sensitive caregiving. Elements of existing interventions include psychoeducational training and attachment-based components.75-77 These programs have shown moderate benefits in improving maternal responsiveness, child development, and attachment security.77,78

There are a number of implications for intervention development, augmentation, and assessment stemming from the findings of this study. First, although many parenting interventions focus on parenting skills alone, enhancing the executive skills of mothers, for example through mindfulness training,79,80 may serve as promising strategy to improve parenting. Outside the parenting context, mindfulness training has been associated with improved cognitive flexibility,79 increased working memory,80 and changes in cortisol function.81 Second, key executive functions assessed in this study, working memory (in this case, the ability to strategically provide alternative explanations for the infant’s behavior while overriding one’s internal emotion or thoughts) are potentially related to a mother’s ability to engage in mentalizing and the development of a reflective stance. Both mentalizing and reflective functioning are core components to the interventions Minding the Baby75 and The Mothers and Toddlers Program.82 Assessing a mother’s executive function before service delivery may help to inform the implementation and potential success of these interventions with mothers.

REFERENCES


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