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Abstract

In light of evidence suggesting that maternal adaptation may impact early child emotional development, this study investigated the interactive effects of maternal psychosocial maladjustment and maternal sensitivity on child internalizing symptoms, with the aim of investigating the potentially protective function of maternal sensitivity. Families (N = 73to 106 across measures) took part in four assessments between ages 1 and 3 years. Mothers completed measures of parental stress, psychological distress, and marital satisfaction when their children were between 12 and 15 months. A composite score of maternal psychosocial maladjustment was derived from these measures. Maternal sensitivity was rated by trained observers at 12 months following a home visit. Child internalizing symptoms were assessed by both parents at 2 and 3 years. Hierarchical regressions revealed that increased maternal psychosocial maladjustment was related to more internalizing symptoms in children, however only among children of less sensitive mothers. In contrast, children of more sensitive mothers appeared to be protected. This was observed with maternal reports at 2 years, and both maternal and paternal reports at 3 years. These results suggest that young children may be differentially affected by their parents' emotional adjustment, while highlighting the pivotal protective role of maternal sensitivity in this process.

Keywords: maternal psychological maladjustment, maternal sensitivity, child internalizing symptoms, buffer effects.

Maternal psychosocial maladjustment and child internalizing symptoms: Investigating the modulating role of maternal sensitivity

Emotional adjustment is a defining feature and a core component of healthy child development. One of the manifestations of impaired emotional adjustment is the presence of internalizing symptoms, which encompass features related to depression, anxiety, social withdrawal, and somatic complaints (without medical explanation). Research has demonstrated that internalizing symptoms can be detected in early childhood. Indeed, high levels of internalizing problems, as operationalized, for instance, by symptoms of depression and anxiety, show prevalence rates up to 28% in toddlers (e.g., Wilens et al., 2002). Furthermore, longitudinal data has been used to investigate developmental trajectories of internalizing symptomatology. For instance, Côté et al. (2009) identified three different trajectory groups among children aged 1.5 to 5 years. Those groups included children with stable low symptom levels (29.9% of their sample), children with moderate but increasing levels (55.4% of their sample), and children with high and increasing levels (14.7% of their sample). Internalizing symptoms have also been identified as predictors of increased risk for psychopathology later in life (e.g., Bittner et al., 2007; Moffitt et al., 2007; Weissman et al., 2005), and are associated with disturbances in several domains including interpersonal relationships, academic performance, and substance abuse (Birmaher et al., 1996; Hammen & Rudolph, 2003). Overall, research suggests that internalizing symptoms are common among very young children, are not often transient, and constitute risk factors for subsequent maladjustment. Hence, it is critical to understand the risk and protective factors involved in the emergence of internalizing symptoms, starting in infancy.

One such factor is parental psychosocial maladjustment. This term is used to characterize parental distress or impairments, is often operationalized by factors such as mood, stressors, or marital strain (e.g., Tietjen & Bradley, 1985), and is a well-documented predictor of child

emotional outcomes such as internalizing symptoms. For instance, children of depressed mothers are more likely to present with increased internalizing symptoms by middle childhood than those of non-depressed mothers (e.g., Goodman et al., 2011). Maternal anxiety is also related to offspring internalizing symptoms (Barker, Jaffee, Uher, & Maughan, 2011). Furthermore, both family conflict and family aggression are associated with impaired child emotional development (e.g., Handal, Tschannen, & Searight, 1998; Harachi et al., 2006; McCloskey, Figueredo, & Koss, 1995; Richmond & Stocker, 2006). In addition, poor marital relationship quality/satisfaction has been identified as a risk factor for child emotional problems, whereas positive marital characteristics have been associated with fewer offspring symptoms (e.g., Cummings, Goeke-Morey, & Papp, 2003; Goeke-Morey, Cummings, & Papp, 2007). There are, thus, documented associations between parental maladjustment and child maladjustment, notably internalizing symptoms.

Nonetheless, the size of these associations is, at times, rather small, suggesting that they may be moderated by other variables (e.g., Goodman et al., 2011). Biological variables have received the most attention as moderators. Studies suggest, for instance, that children with more optimal biological functioning are protected against the development of internalizing symptoms in the context of marital conflict (Koss et al., 2014) or maternal psychological maladjustment (Wetter & El-Sheikh, 2012). In contrast, few studies have investigated whether environmental factors can also play such a protective role. This appears likely however, especially with high-quality parenting, which meta-analytic data suggest is a robust protective factor against the development of internalizing problems in children (McLeod, Weisz, & Wood, 2007).

Accordingly, high-quality parenting might also act as a moderator, and buffer the risk for child internalizing symptomatology otherwise associated with maternal maladjustment. In fact, it is well demonstrated that some mothers are able to provide good-quality care to their children in

spite of personal hardship (e.g., Campbell et al., 2004; Grant, McMahon, Reilly, & Austin, 2010a) – what is less clear is whether such care is sufficient to offset the otherwise negative impact of maternal psychosocial maladjustment on child emotional outcomes.

Maternal sensitivity as a protective factor

Child development is reliably related to the quality of provided parental care (Sroufe, 2005). One aspect of high-quality parental care is maternal sensitivity, defined as mothers' capacity to recognize, correctly interpret, and quickly and adequately respond to their children's signals (Ainsworth, Bell, & Stayton, 1974). Sensitive parenting is associated, for instance, with child language and cognitive development, attachment security, emotion regulation, and social competence (e.g., Bornstein, 2002; 2006).

In addition to such direct links, maternal sensitivity has begun to be identified as a risk buffer against biological (e.g., Spangler, Johann, Ronai, & Zimmerman, 2009) and environmental adversity (Rochette & Bernier, 2014). Importantly for our purposes, sensitivity also appears to play a protective function against suboptimal maternal characteristics. For instance, harsh maternal discipline has been found to relate to increased aggressive behavior in children, but only for those whose mothers also displayed low sensitivity (Alink et al., 2009a). Moreover, maternal sensitivity has been found to modulate the link between maternal prenatal anxiety and infant mental development at 7 months of age, such that infants whose mothers were anxious displayed poorer mental development only when their mother was also less sensitive (Grant et al., 2010a). It was also reported that maternal prenatal anxiety was associated with increased child negative affect following a stressful episode at 7 months of age only for infants of less sensitive mothers (Grant, McMahon, Reilly, & Austin, 2010b). Furthermore, positive mother-child relationships have been identified as a protective factor in the context of family conflict (Alink, Cicchetti, Kim, & Rogosch, 2009b) as well as in the presence of interparental violence (Davies, Winter, &

Cicchetti, 2006; Manning, Davies, & Cicchetti, 2014). All in all, there is emerging evidence that maternal sensitivity may play not only a direct positive role in child emotional development, but also an indirect role, by attenuating the effects of otherwise negative maternal influences on child functioning.

The current study

Building and expanding on previous results (e.g., Alink et al., 2009a, b; Grant et al., 2010a, b), the current study focused on child internalizing symptoms as assessed by both mothers and fathers at two different ages. Provided that components of maternal mental health (e.g., Goodman et al., 2011) and relational stress (e.g., Goeke-Morey et al., 2007; Richmond & Stocker, 2006) have both been previously associated with offspring socio-emotional development and given that maternal maladjustment has been found to be more strongly related to child internalizing symptoms than paternal maladjustment (see Connell & Goodman, 2002, for a review), the present study's objectives were 1) to assess the links between child internalizing symptoms at 2 and 3 years of age and a combination of facets of maternal maladjustment in the personal, parenting, and marital spheres, and 2) to investigate whether maternal sensitivity may buffer the transmission from maternal to child maladjustment. It was expected that high levels of observed maternal sensitivity would buffer the risk stemming from maternal psychosocial maladjustment in the prediction of child internalizing symptoms, as reported by mothers and fathers.

Method

Participants

Our low-risk community sample was comprised of 106 mother-infant dyads living in a large Canadian metropolitan area. Families were drawn from random birth lists of the Ministry of Health and Social Services. Criteria for participation were full-term pregnancy and the absence of

any known disability or severe delay in the infant. Socio-demographic information was gathered when infants were 8 months old. At that time, mothers were between 22 and 45 years old (M = 31.7), had 16.1 years of education on average (varying from 10 to 18 years), and their average family income lied in the \$60,000 to \$79,000 bracket, representative of the average family income in Canada for the years of data collection (i.e., \$74,600). Fathers were between 25 and 50 years old (M = 33.9) and had 15.7 years of education on average (varying from 11 to 19). Both maternal and paternal education levels were similar to average education levels in the province of Quebec, with 63.5% of mothers and 62.8% of fathers having at least a college degree (63.3% of parents hold college degrees in Quebec). Offspring gender was spread almost evenly (51 boys and 55 girls). Most of the sample (95.7%) was Caucasian and the majority of the parents were either married or living together (86.8%). Finally, 19% of children had no siblings.

Procedure

Data were collected in the family homes through four visits which lasted an average of 90 minutes. When children were aged 12 months (T1), the home visit was modeled after the work of Pederson and Moran (1995), and aimed at challenging the mother's capacity to divide her attention between several competing demands, thus reproducing the natural conditions of daily life when caring for an infant. The home-visit protocol was thus purposely designed to create a situation where maternal attention was being solicited by both the research tasks and the infant's demands, which placed the dyad in a challenging situation, likely to activate both the infant's attachment system and the mother's caregiving system in response. Visits included a brief interview with the mother, a developmental assessment of the infant, and a 20-min free-play period. Observations performed throughout this home visit were used to assess maternal sensitivity, as described below. After this first visit (T1), as well as when children were aged 15 months (T2), 2 years (T3), and 3 years (T4), parents were given questionnaires that they later

returned via mail (see measures below). Parents were invited to fill out these questionnaires independently, once the research assistant was gone, and were each provided with a prepaid envelope at each assessment.

Measures

Maternal psychosocial maladjustment. At T1, mothers completed the Parenting Stress Index (PSI-Short Form; Abidin, 1995), which is a 36-item self-report questionnaire designed to asses parents' perceived stress in relation to their child and their parenting role. Items are rated on a Likert scale ranging from 0 to 5. The total average score was retained. The PSI shows excellent internal consistency and convergent validity with respect to prenatal stress and to other indices of postnatal stress (Abidin, 1995; Teti, Nakagawa, Das, & Wirth, 1991). Internal consistency was also excellent for our sample (Cronbach's $\alpha = .98$).

At T1 also, mothers completed the short version of the Psychiatric Symptoms Index (Ilfeld, 1976, 1978). This self-report questionnaire assesses psychological symptoms including depression, cognitive disturbance, anxiety, and anger, and yields one global score of psychological distress. Mothers completed the 14 items on a Likert scale ranging from 0 to 4. This instrument presents good internal consistency (Ilfeld, 1976; Préville, Potvin, & Boyer, 1995) and content validity with DSM-IV diagnostic criteria (Okun, Stein, Bauman, & Silver, 1996) and with other instruments measuring depression and anxiety (Sakakibara, Miller, Orenczuk, & Wolfe, 2009). Internal consistency for our sample was good (Cronbach's $\alpha = .88$).

At T2, mothers completed the short Dyadic Adjustment Scale (DAS; Spanier, 1976; four-item version – DAS-4; Sabourin, Valois, & Lussier, 2005). The DAS-4 is a four-item questionnaire that assesses individuals' degree of satisfaction with regards to their current romantic relationship with a 1-6 Likert scale. As described by Sabourin et al. (2005), the DAS-4 shows very good internal consistency (α consistently above .80), excellent temporal stability over

a 1-year period for men (r = .87) and women (r = .83), and high predictive validity with regards to couple dissolution, and it is less subject to socially desirable responding than longer versions of the DAS. Internal consistency for our sample was also good (Cronbach's $\alpha = .88$).

Given the inter-correlations among these three measures (.28 < r < .40, p < .01), a composite score of maternal psychosocial maladjustment was derived by standardizing and averaging the total scores of the three measures (reverse-coding marital satisfaction). This allowed for the use of a psychometrically stronger predictor, while reducing Type-I error probability in the context of the examination of four dependent variables (maternal and paternal reports of child symptoms at 2 and 3 years).

Maternal sensitivity. Maternal sensitivity was assessed at T1 using the Maternal Behavior Q-Set (MBQS; Pederson & Moran, 1995), a 90-item measure designed to assess the quality of maternal behaviors during in-home mother-infant interactions. A trained research assistant noted maternal behaviors throughout the visit and rated the MBOS immediately afterward, based on the entire observation period. Items describing potential maternal behaviors were sorted into nine clusters, ranging from very similar to very unlike the observed mother's behaviors. The observer's sort was then correlated with a criterion sort representing the prototypically sensitive mother, which is provided by the developers of the instrument. Sensitivity scores can thus vary from -1 (least sensitive) to 1 (prototypically sensitive). The MBOS is significantly correlated with other measures of maternal behavior, such as the HOME Inventory and the Ainsworth scales (see Pederson & Moran, 1995), and shows good temporal stability (Bailey, Moran, Pederson, & Bento, 2007; Tarabulsy et al., 2008). Its construct validity is demonstrated by meta-analytic data showing its excellent predictive capacity with respect to child attachment security (van IJzendoorn, Vereijken, Bakermans-Kranenburg, & Riksen-Walraven, 2004). MBQS scores also relate to subsequent child cognitive and socio-emotional

functioning (Bernier, Carlson, Deschênes, & Matte-Gagné, 2012; Bordeleau, Bernier, & Carrier, 2012; Lemelin, Tarabulsy, & Provost, 2006).

To maximize the reliability of these observations, research assistants attended a 2-day training workshop, during which they reviewed several videotapes of mother—infant interactions so as to practice coding the MBQS. After the workshop, the assistants performed their first few home visits with a more experienced colleague, and they completed the MBQS together. When the junior home visitors were ready to rate maternal behavior, the first two or three visits were followed by a debriefing session either with the PI or with an experienced graduate student to review the salient elements of the visit before scoring the MBQS. The assistants then went on to rating the MBQS autonomously. Thirty percent of the visits were conducted by two research assistants who completed the MBQS independently. In total, ten different assistants coded maternal sensitivity. Inter-rater reliability was always estimated between two coders (i.e., the two assistants who conducted the home visit together for a given family). Mean agreement between the pairs of raters was very good, *ICC* = .87.

Child internalizing symptoms. At T3 and T4, mothers (N = 106 and 94, respectively) and fathers (N = 73 and 78, respectively) completed the internalizing problems subscale of the Child Behavior CheckList, 1.5-5 year version (CBCL; Achenbach & Rescorla, 2000). This subscale is comprised of 36 items, with possible scores ranging from 0 to 72. Test-retest reliability for this subscale is .90, inter-parent agreement is .59, and one-year stability is .76 (Achenbach & Rescorla, 2000). CBCL scores also show good convergent validity with other child socio-emotional ratings such as the Richman Behavior Checklist (Richman, 1977) and the Toddler Behavior Screening Inventory (Achenbach & Rescorla, 2000; Mouton-Simien, McCain, & Kelly, 1997). Internal consistency for overall internalizing problems was good for the current sample (Cronbach's α = .78, .77, .71, and .83 for maternal ratings at T3 and T4 and paternal

ratings at T3 and T4, respectively). Maternal and paternal reports were positively correlated (r = .55 at T3 and r = .52 at T4, p's < .01). Child internalizing symptoms scores were normally distributed and did not require transformation. Mothers and fathers who did not return the CBCL questionnaire were not different from those who did in terms of socio-demographic characteristics (i.e., ethnicity, income, and education; all p's > .10). Moreover, mothers who did not return the CBCL questionnaires did not differ from mothers who did on sensitivity scores (p > .10). Means and standard deviations for all main study variables are reported in Table 1.

Given that the independent variable (maternal maladjustment) was based solely on maternal reports, maternal and paternal CBCL scores were kept separate rather than composited, with the aim of examining the robustness of the results to shared method variance, with an outcome methodologically independent of maternal reports (i.e., father CBCL).

Results

Preliminary analyses

Table 2 presents the zero-order correlations among all variables. We first examined the correlations between potential confounding variables (i.e., child gender, maternal age, maternal education, paternal age, paternal education, and family income) and main study variables. Child gender and family income were the only demographic variables that were associated with some of the outcome measures (i.e., reports of child internalizing symptoms at ages 2 and 3). In each subsequent main analysis, we accounted for the covariates that were marginally or significantly associated with the outcome. T-tests revealed no significant differences between maternal and paternal ratings of child internalizing symptoms at either time point (both p's > .05). No agerelated differences (2 vs. 3 years) were found for either maternal or paternal ratings (all p's > .05).

Main analyses

Multiple hierarchical regressions were then performed to assess the interactive effects of maternal psychosocial maladjustment and maternal sensitivity on child internalizing symptoms. Maternal and paternal reports, at 2 and 3 years of age, were submitted to distinct regression equations. We inserted variables in the following order: Block 1, covariates (when appropriate); Block 2, *maternal psychosocial maladjustment* and *maternal sensitivity*; Block 3, the multiplicative interaction term of *maternal psychosocial maladjustment* by *maternal sensitivity*. The results of the four regression models are shown in Table 3. The analyses revealed significant interaction effects of maternal psychosocial maladjustment and maternal sensitivity on maternal reports of child internalizing symptoms at 2 ($\beta = -.23$, p = .04) and 3 years ($\beta = -.27$, p = .02). The interaction between maternal psychosocial maladjustment and maternal sensitivity was also significant when predicting paternal reports of child internalizing symptoms at 3 years ($\beta = -.26$, p = .05), although not at 2 years ($\beta = -.07$, ns).

The significant interactions were explored both statistically, with post-hoc probing of moderation effects through analysis of simple slopes (Preacher, Curran, & Bauer, 2006), and graphically (Figures 1-3). The relations between maternal psychosocial maladjustment and child internalizing symptoms at 2 and 3 years were tested as a function of maternal sensitivity. Fitted regression lines were plotted at high (+ 1 SD) and low (-1 SD) values of maternal sensitivity. At 2 years, the results revealed a significant and positive slope for mother reports of offspring internalizing symptoms for children of less sensitive mothers (B = 1.44, SE = 0.61, t = 2.35, p = .02), whereas the slope for children of more sensitive mothers was not significant (B = -0.44, SE = 0.48, t = -0.91, ns). Likewise, at 3 years, post-hoc analyses yielded a significant and positive slope for mother reports of offspring internalizing symptoms for children of less sensitive mothers (B = 1.92, SE = 0.75, t = 2.52, p = .01), whereas the slope for children of more sensitive mothers was not significant (B = -0.49, SE = 0.50, t = -0.99, t = 0.99, t = 0.50. Finally, at 3 years, post-hoc

analyses yielded a significant and positive slope for father reports of offspring internalizing symptoms for children of less sensitive mothers (B = 1.66, SE = 0.80, t = 2.08, p = .04), whereas the slope for children of more sensitive mothers was not significant (B = -0.45, SE = 0.54, t = 0.83, ns).

In order to determine whether maternal psychosocial maladjustment and maternal sensitivity were associated with child internalizing symptoms at age 3, over and above 2-year symptoms, we conducted similar regression models, adding internalizing symptoms at 2 years as a control when predicting 3-year internalizing symptoms. Analyses yielded non-significant results with maternal reports (β = -.05, ns; Table 4). In contrast, there was a significant interaction effect when predicting change in paternal reports of child internalizing symptoms between ages 2 and 3 (β = -.19, p = .03; Table 4). Decomposition of this interaction effect (Figure 4) revealed that maternal psychosocial maladjustment was positively related to an increase in child internalizing symptoms as rated by their fathers, but again, only for children of less sensitive mothers (B = 1.67, SE = 0.75, t = 2.24, p = .03), whereas the relation for children of more sensitive mothers was not significant (B = -0.45, SE = 0.49, t = -0.92, ns).

Taken together, the results indicate that maternal psychosocial maladjustment was generally related to higher child internalizing symptoms, however only among children of less sensitive mothers.

Discussion

Aiming to investigate familial characteristics likely to precipitate or impede the early development of internalizing symptoms, we examined whether the relation between maternal psychosocial maladjustment and toddlers' internalizing symptoms was moderated by maternal sensitivity. The results revealed significant positive relations between maternal psychosocial maladjustment and child internalizing symptoms for children of less sensitive mothers, as

reported by mothers when children were aged 2 years and by both parents when children were aged 3 years. Contrastingly, the relations were not significant for children whose mothers were rated as relatively more sensitive. Hence, poor maternal adjustment was related to increased levels of internalizing symptoms in children as early as 2 and 3 years of age, for children of less sensitive mothers; in contrast, children of more sensitive mothers appeared to be protected. Furthermore, when looking at changes between the ages of 2 and 3, results revealed that worse maternal psychosocial maladjustment during infancy was related to a subsequent increase in children's internalizing symptoms as reported by their fathers, again only for children of less sensitive mothers.

The current results are consistent with a growing body of literature targeting interaction effects and the suggestion that negative child outcomes are most likely due to a combination of risk factors rather than the presence of a single one (e.g., McMahon, Barnett, Kowalenko, & Tennant, 2006; Sameroff, Gutman, & Peck, 2003). In fact, most developmental theorists agree that the factors which influence child development are intertwined in complex ways, with general consensus that the nature of the interplay between these factors is interactive (Bronfenbrenner, 1979; Collins, Maccoby, Steinberg, Hetherington, & Bornstein, 2000). The current results suggest that children may be differentially affected by their parents' own emotional adjustment, according to the quality of care they receive. Indeed, maternal psychosocial maladjustment and offspring internalizing symptoms were not related for children whose mothers were relatively more sensitive.

This is consistent with existing literature showing links between high levels of maternal sensitivity and optimal child outcomes in the domains of self-regulation (e.g., Grossmann & Grossmann, 1991; Kopp, 1982; Sroufe, 1995) and socio-emotional functioning (e.g., Leerkes, Blankson, & O'Brien, 2009). The protective effect observed here might occur through the

promotion of competent emotion regulation by highly sensitive mothers. Indeed, while young children have limited capacity to efficiently self-regulate, responsive and sensitive mothering entails efficient external regulation of child negative affect, which progressively provides young children with the opportunity to internalize the regulatory skills initially taught by the caregiver, and gradually, to become able to use them independently (Calkins, 2004). In this way, highly sensitive caregivers support the development of emotion regulatory skills in the child, which can act as a protective factor in the face of environmental stress/adversity, in this case that triggered by maternal psychosocial difficulties and its familial consequences (e.g., Cole, Martin, & Dennis, 2004). Other potential mechanisms underlying the current results may include the fostering of secure attachment bonds via high levels of maternal sensitivity. Indeed, responsive and sensitive behaviors from the caregiver favor the development of secure attachment (e.g., DeWolff & Van IJzendoorn, 1997), which in turn is a well-documented protective factor against child internalizing problems (Groh, Roisman, Van IJzenndoorn, Bakermans-Kranenburg, & Fearon, 2012). Overall, mothers who manage to remain highly sensitive to their child despite poor psychosocial adjustment might favor the development of key relational and emotional skills in their child, hence protecting children against the development of emotional difficulties. Contrastingly, less sensitive mothers are likely to see their child develop insecure attachment ties, and may fail to provide successful external regulation in response to their child's distress during stressful situations, thereby impeding children's capacity to learn appropriate emotion-regulation tools that would protect them against internalizing problems.

Another factor that may explain the specific pattern of results observed here pertains to dual risk (e.g., Sameroff, 1983). Indeed, being exposed to solely one risk factor (i.e., either higher levels of maternal psychosocial maladjustment or lower levels of maternal sensitivity) might not be sufficient in and of itself to significantly increase children's risk of internalizing symptoms in

a low-risk sample like this one. However, being exposed to a combination of both risk factors, albeit at moderate levels, might be sufficient to impair children's emotional adjustment.

Furthermore, the current results support the idea that maternal sensitivity is an important protective factor for children not only in stressful environmental circumstances, broadly, but also in the face of maternal struggles (and, in this specific context, psychosocial maladjustment). Several factors might explain why mothers' maladjustment may affect their offspring. One core hypothesis is that of genetic transmission. Indeed, over and above the risk conveyed by environmental and relational factors, research has identified a genetic basis to emotional maladjustment. For instance, children of depressed or anxious mothers are more likely to develop mood disorders, and this is partly accounted for by shared genetic characteristics (see Eley, 1999 for a review). Furthermore, children presenting with certain genetic profiles are at increased risk of developing early signs of emotional disturbances when living in detrimental environmental contexts (e.g., Gotlib, Joorman, Minor, & Hallmayer, 2008; Pluess et al., 2011). Based on those transmission paradigms, most often derived from mid/high-risk samples, we assumed that similar transmission processes could apply to low-risk samples such as ours, and would allow us to test our hypotheses pertaining to individual differences within normal ranges of emotional dysregulation. Although the current study cannot tease apart environmental from genetic transmission of emotional difficulties, it does suggest that when mothers manage to show high sensitivity to their child in spite of their own emotional struggles, such high-quality parenting is a key environmental element that can offset the otherwise likely transmission of emotional difficulties from mother to child, at least in low-risk samples.

One noteworthy aspect of the findings is that significant results were found with mother reports at both 2 and 3 years, but only at 3 years for father reports. One may argue that lower mood (although assessed several months earlier) altered mothers' perceptions and led them to

overestimate their child's internalizing symptoms, thus creating spurious relations between measures. However, no significant differences were found between maternal and paternal ratings of internalizing symptoms at either time point (refer to Table 1 for means and standard deviations). A different explanation pertains to our sample size, which was considerably smaller for father than mother reports (especially at 2 years), thus necessitating a greater population-level effect size for the interaction to be detected as significant with our study parameters. Another potential explanation is that mothers typically spend more time with their child during their first few years of life than fathers do. This might make mothers more aware of their child's non-verbal or ambiguous cues, compared to fathers. As toddlers become more verbally sophisticated, and able to express their emotional states through language (i.e., between 2 and 3 years of age in the current context), fathers might more easily pick up on those early signs of internalizing symptoms. Such greater awareness may be facilitated further by fathers' increased parental involvement across toddlerhood (e.g., Bailey, 1994). Thus, one might speculate that the phenomenon may have been present at 2 years already, but more easily detected by fathers at age 3, given toddlers' growing capacity to describe their feelings verbally, along with many fathers' enhanced involvement and thus familiarity with their child. This might also explain why analyses pertaining to changes between the ages of 2 and 3 years revealed significant interactive effects for father reports only.

Another noteworthy aspect of the findings is the fact that among less sensitive mothers, maternal maladjustment, although assessed prior to child outcomes and early in the children's life (i.e., at 12 and 15 months), was associated with toddlers' internalizing symptoms later on (i.e., ages 2 and 3). Although the current correlational design does not allow for causal inference, these longitudinal results raise the possibility of lingering effects of maternal psychological maladjustment onto the development of early childhood internalizing symptoms, and reiterate the

importance of studying such relations very early on. Furthermore, maternal sensitivity was also rated prior to child outcomes, which suggests that a maladaptation cascade, stemming from the combination of poor maternal psychological adjustment and low sensitivity, might unfold over early childhood, and perhaps have increasingly deleterious consequences as children fail to acquire age-appropriate emotion regulation strategies. Indeed, a key principle of developmental psychopathology is that successfully mastered developmental tasks provide children with tools to negotiate the developmental tasks that become salient at later ages (Sroufe & Rutter, 1984). Longer-term longitudinal designs are needed to test the speculation that what was observed here is the beginning of a developmental cascade entailing the development of emotional difficulties among children whose mothers suffer from psychosocial maladjustment and have difficulty providing sensitive care.

Besides, one may notice the lack of direct associations between our three main variables: maternal psychosocial maladjustment, maternal sensitivity, and child internalizing symptoms. Those results are, however, less unexpected than they may seem. For instance, it is not unusual in the literature to find non-significant associations between maternal maladjustment and maternal sensitivity, especially among community samples (e.g., Kaitz, Maytal, Devor, Bergman, & Mankuta, 2010; van Doesum, Hosman, Riksen-Walraven, & Hoefnagels, 2007). Likewise, reported associations between maternal psychosocial maladjustment and child internalizing symptoms vary substantially in magnitude (e.g., Elgar, McGrath, Waschbusch, Stewart, & Curtis, 2004; Gravener et al., 2012; Mezulis, Hyde, & Clark, 2004), as do the links between maternal sensitivity and child internalizing symptoms (e.g., Ciciolla, Gerstein, & Crnic, 2014; Gershoff, 2002).

This study is not without limitations. First, only parental reports were used for both predictor and outcome variables. However, this limitation is partially compensated for by the fact that measurements were not taken at the same time points and in fact, maternal reports of predictor and outcome variables were not correlated (hence ruling out shared method variance as a key hypothesis). Moreover, the dependent variables were measured with the CBCL, which is a widely used, reliable tool that is well established in both research and clinical domains. Nonetheless, non-parental reports of child internalizing symptoms (e.g., by daycare providers) would have provided rich independent information. As mentioned above, the design did not allow for teasing apart genetic and environmental factors in the mother-child transmission of emotional maladjustment that was observed in dyads with a relatively less sensitive mother; geneticallyinformed designs entailing careful observational assessment of sensitivity are needed to investigate this question. One should also keep in mind the correlational nature of the design, which entails that results could potentially be interpreted in alternative ways, for instance that maternal optimal psychosocial adjustment protects children of less sensitive mothers against the development of child internalizing symptoms, or that higher levels of child internalizing symptoms influence maternal psychosocial maladjustment in mothers with lower sensitivity.

Furthermore, given the low-risk status of this community sample, the current results may not generalize to higher-risk populations. Indeed, a good deal of research suggests that mothers presenting with significantly higher levels of maladjustment than those found in this sample (e.g., clinically depressed mothers) may not be able to display high levels of sensitivity (e.g., Field, 2010). However, one may speculate that in such high-risk samples, where levels of maternal sensitivity are expected to be lower, low sensitivity would remain a risk-enhancing variable when combined to low maternal adjustment. Nonetheless, the current results also suggest that individual variations within the normal range for both maternal psychosocial maladjustment and

maternal sensitivity might be sufficient to associate, to some extent, with early internalizing symptoms.

Additionally, the current results should be interpreted with caution provided the relatively low proportion of variance that was explained by the models, meaning that other factors come into play. Hence, future research should continue to investigate relational moderators that can act as buffers of early environmental risk, including paternal sensitivity and other proximal caregiving markers (e.g., attachment security). This appears to be an important endeavor, given convincing data showing that parental sensitivity and parent-child attachment security can be enhanced by relatively brief, evidence-based intervention (for a review, see Bakermans-Kranenburg, van IJzendoorn, & Juffer, 2003). Such interventions have, in fact, been found to increase maternal sensitivity even among mothers presenting with very high levels of psychosocial maladjustment (Moss et al., 2011). Thus, regardless of the degree of genetic and environmental contributions involved in the mother-child transmission of emotional maladjustment, the current results combined with those of intervention studies suggest that parental sensitivity might be a key, malleable vehicle through which the transmission could be attenuated, thus contributing to break the intergenerational cycle of maladjustment.

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Table 1

Descriptive statistics

Variable	Mean	SD	Score Range
Parental Stress Index global average score	2.90	1.10	0-5
Psychiatric Symptom Index average score	1.77	.45	1 - 4
Dyadic Adjustment Scale average score	4.02	.89	1 – 6
Maternal psychosocial maladjustment composite	06	1.52	-5.68 - 3.04
Maternal sensitivity	.64	.27	-1.00 - 1.00
Child internalizing symptoms at 2 years - Mother	6.99	5.22	0-72
Child internalizing symptoms at 3 years - Mother	7.85	5.28	0-72
Child internalizing symptoms at 2 years - Father	7.17	5.55	0-72
Child internalizing symptoms at 3 years - Father	7.05	5.55	0-72

Note. SD = Standard deviation.

Bivariate correlations among all study variables

Table 2

	:	2.	္ပ	4.	5.	6.	7.	<u>.</u> ∞	9.	10.	11.	12.	13.	14.	15.
1. Child gender ^a	1	.05	08	.04	.00	08	.22t	03	08	.04	02	.14 ^t	.01	.19*	.03
2. Maternal age		1	.38**		.29**	.44**	.01	-:11	12	14	.20**	.04	08	09	01
3. Maternal education			!		.62**	.59**	27*	.05	15	27**	.22**	04	08	08	19
4. Paternal age				:	.20**	.29**	10	.03	.03	02	.07	01	08	09	02
5. Paternal education					;	.53**	12	.06	14	09	.20**	02	05	10	07
6. Family income						:	18	.08	32**	18	.24**	12	12	19*	06
7. Maternal PSI total score							ŀ	37**	.36**	.70**	03	.07	.13	.18	.12
8. Maternal DAS total score								1	35**	.47**	01	23 ^t	24*	08	17
9. Maternal IIfeld total score									1	.42**	.01	.31*	.30*	.30*	.19
MPM composite score										1	06	.12	.24*	.19	.12
 11. Maternal sensitivity 											:	24**	.02	15	05
12. CBCL 2 years - Mother												1	.58**	.55**	.54**
13. CBCL 3 years - Mother													1	.44**	.52**
14. CBCL 2 years - Father														ł	.67**
15. CBCL 3 years - Father															1

Ilfeld = Psychiatric Symptoms Index; MPM = Maternal Psychosocial Maladjustment; ^aBoys = 1, Girls = 2. $^{t}p < .10.^{*}p < .05.^{**}p < .01.$ Note. CBCL = Child Behavior CheckList - Child internalizing symptoms scores; PSI = Parental Stress Index; DAS = Dyadic Adjustment Scale;

Table 3

Summary of regression analyses for interactive effects of maternal psychosocial maladjustment and maternal sensitivity onto maternal and paternal reports of child internalizing symptoms at 2 and 3 years

Model and steps	β	t
Maternal reports of child internalizing symptoms at 2 years		
1. Child gender	.16	1.64
2. Maternal psychosocial maladjustment	.15	1.52
Maternal sensitivity	01	14
3. Maladjustment x Sensitivity	23	-2.14*
R^2 (adj.)		.06
df		(4, 105)
Maternal reports of child internalizing symptoms at 3 years		
1. Family income	16	-1.63
Child gender	.14	1.45
2. Maternal psychosocial maladjustment	.20	1.84^{t}
Maternal sensitivity	02	19
3. Maladjustment x Sensitivity	27	-2.34*
R^2 (adj.)		.11
df		(5, 94)
Paternal reports of child internalizing symptoms at 2 years		
Maternal psychosocial maladjustment	.26	2.12^{*}
Maternal sensitivity	.20	1.42
2. Maladjustment x sensitivity	07	49
R^2 (adj.)		.04
df		(4, 70)
Paternal reports of child internalizing symptoms at 3 years		
1. Maternal psychosocial maladjustment	.18	1.46
Maternal sensitivity	.13	1.00
2. Maladjustment x Sensitivity	26	-1.96 [~]
R^2 (adj.)		.02
df		(3, 76)

 $^{^{}t}p < .10. ^{*}p < .05. ^{\sim}p = .05.$

Table 4

Summary of regression analyses for interactive effects of maternal psychosocial maladjustment and maternal sensitivity onto maternal and paternal reports of child internalizing symptoms at 3 years when controlling for child internalizing symptoms at 2 years

Model and steps	β	t
Maternal reports of child internalizing symptoms at 3 years		
1. Family income	02	15
Child gender	.41	3.11**
Maternal reports of child internalizing symptoms at 2 years	.54	6.36**
2. Maternal psychosocial maladjustment	.06	1.01
Maternal sensitivity	04	62
3. Maladjustment x sensitivity	05	74
R^2 (adj.)		.79
df		(6, 80)
Paternal reports of child internalizing symptoms at 3 years		
1. Paternal reports of child internalizing symptoms at 2 years	.86	13.03**
2. Maternal psychosocial maladjustment	.10	1.38
Maternal sensitivity	.01	.18
3. Maladjustment x sensitivity	19	-2.30^*
R^2 (adj.)		.81
df		(4, 53)

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

Figure 1

Interactive effects of maternal psychosocial maladjustment and maternal sensitivity onto maternal reports of child internalizing symptoms at 2 years

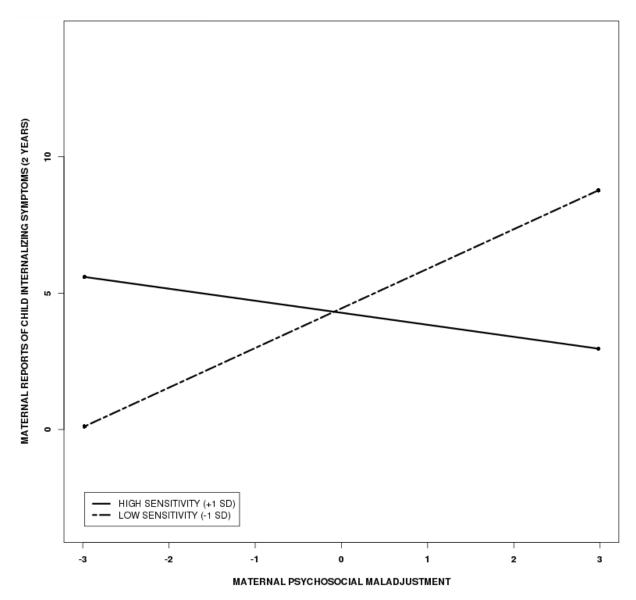


Figure 2

Interactive effects of maternal psychosocial maladjustment and maternal sensitivity onto maternal reports of child internalizing symptoms at 3 years

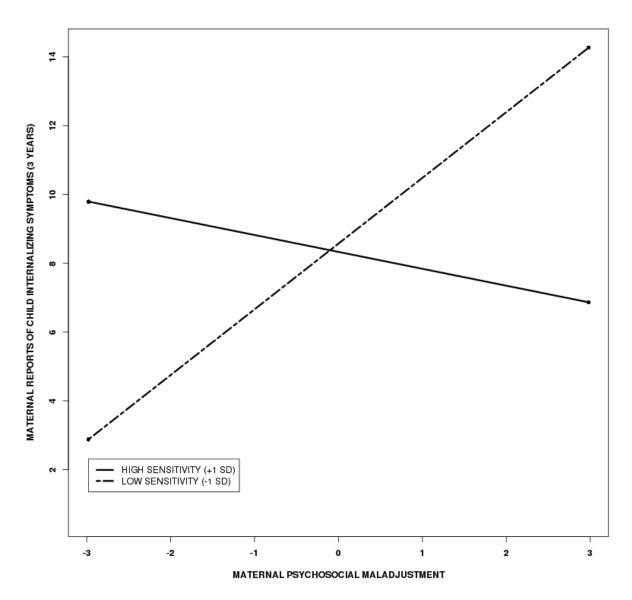


Figure 3

Interactive effects of maternal psychosocial maladjustment and maternal sensitivity onto paternal reports of child internalizing symptoms at 3 years

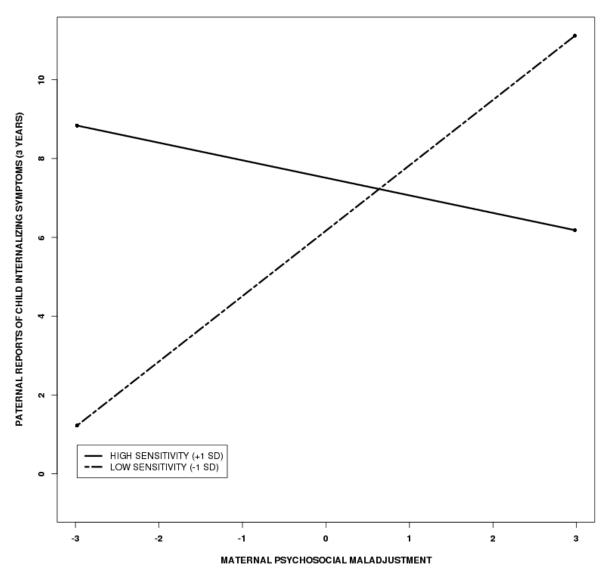
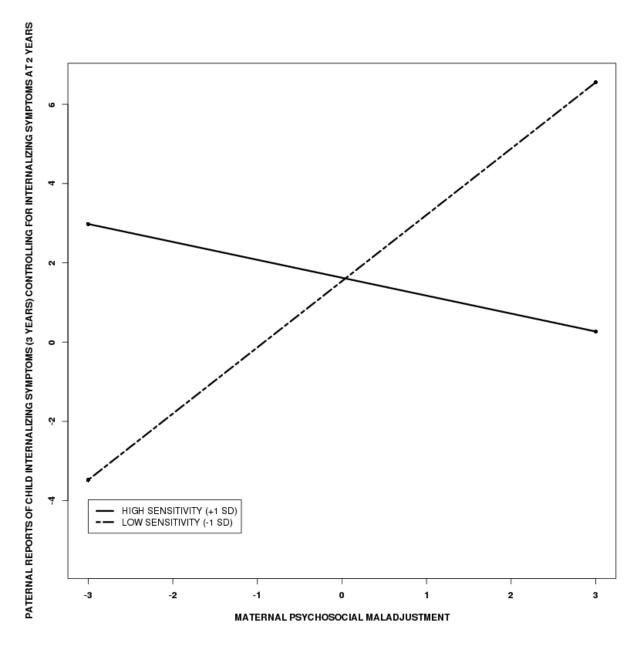


Figure 4

Interactive effects of maternal psychosocial maladjustment and maternal sensitivity onto paternal reports of child internalizing symptoms at 3 years controlling for child internalizing symptoms at 2 years

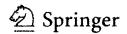




Disclosure of potential conflicts of interest

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