

Emotion

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Maternal Depression Impairs Child Emotion Understanding and Executive Functions: The Role of Dysregulated Maternal Care Across the First Decade of Life

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The long-term negative effects of maternal depression on child outcome are thought to be mediated in part by deficits in caregiving; yet, few studies utilized longitudinal cohorts and repeated observations to specify these links. We tested the impact of deficits in maternal regulatory caregiving across the first decade of life on children's emotional, social, and cognitive outcomes at 10 years. A community birth cohort was repeatedly assessed for maternal depression across the first year and again at 6 and 10 years. At 9 months, 6 years, and 10 years patterns of regulatory caregiving were assessed during mother-child interactions; at 6 and 10 years children underwent psychiatric diagnosis; and at 10 years children's emotion recognition (ERc), executive functions (EF), and social collaboration (SC) were evaluated. Depressed mothers displayed deficits to regulatory caregiving across development and their children exhibited more psychiatric disorders, lower SC, and impaired ERc. Structural equation modeling demonstrated both direct paths from dysregulated caregiving at 6 and 10 years to impaired child EF and ERc and mediated paths via child psychiatric disorder on all 3 outcomes. Effects of 9-month caregiving were only indirect, via child disorder, differentiating infants on risk versus resilient trajectories. Patterns of maternal caregiving were individually stable over time. Our findings demonstrate disruptions to core regulation-based abilities in children of depressed mothers beyond infancy, contribute to discussion on risk and resilience in the context of a distinct early life stress condition, and underscore late childhood as a period of specific vulnerabilities that should become a focus of targeted interventions.

Keywords: maternal depression, emotion recognition, executive function, regulation, mother-child interaction

Although the prevalence of major depressive disorder (MDD) in women of childbearing years is continuously on the rise, rendering maternal depression the most prevalent psychiatric disorder and one with a direct impact not only on the suffering individual but also on her offspring, very few studies have followed children of depressed mothers from birth and over lengthy periods. MDD in the months following childbirth is estimated at 7% to 13% of women in high income countries and up to 30% in the developing

world (Parsons, Young, Rochat, Kringelbach, & Stein, 2012); women are twice more likely to experience MDD than men (Kessler, 2003); more than 10% of women aged 18 to 39 years experience depression at any given time; and lifetime prevalence of MDD in women is estimated at more than 20% (Kessler, Petukhova, Sampson, Zaslavsky, & Wittchen, 2012). Studies have repeatedly shown that exposure to maternal MDD bears long-term negative consequences for children, including increased psychopathology, behavior problems, and social maladjustment (Feldman et al., 2009; Goodman et al., 2011; Halligan, Murray, Martins, & Cooper, 2007; Rohanachandra, Prathapan, & Wijetunge, 2018; Shaw, Connell, Dishion, Wilson, & Gardner, 2009; Yan & Dix, 2014). Yet, the mechanisms underlying the transmission of vulnerability from depressed mothers to their offspring are far less clear, and it has been suggested that studies should move beyond the assessment of psychopathology to study targeted, theoretically derived child outcomes, consider developmental stages beyond infancy, and observe patterns of maternal care over time to examine their contribution to maladaptive outcomes (Feldman, 2015b; Goodman et al., 2011).

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In the current study, we recruited a large community birth cohort of women with no contextual risk and followed a select sample overrepresented for maternal MDD across the first decade of the child's life. Guided by an evolutionary frame, we observed mother-child interactions in the home environment in infancy, middle childhood, and late childhood for multiple theoretically based patterns of maternal care that facilitate regulation. At 10 years, we examined the effects of maternal MDD on three child regulation-based outcomes: emotion recognition (ER), executive functions (EF), and social collaboration (SC) as mediated by deficits in regulatory caregiving over time. Three overarching goals guided our study. First, we aimed to test the longitudinal impact of maternal depression in of itself on development, apart from frequently occurring comorbidities which are typically included in longitudinal studies on maternal MDD, such as single parenting, teenage mothering, premature birth, or poverty. Second, we focused on late childhood, an age rarely studied in relation to maternal depression, and on three regulation-based abilities that have not been studied in combination in relation to maternal MDD. Finally, we hoped to specify the contribution of the mother's regulatory function at different developmental nodes (infancy, early childhood, late childhood) to children's emotional, social, and cognitive outcomes and, by doing so, contribute to the discussion on regulation—a key construct across multiple scientific domains.

The Mother's Regulatory Caregiving and Maternal Depression

Although the term *regulation* still lacks a clear definition, several features constitute its core definition. Across multiple fields, regulation adapts a system's perspective. It describes how various components of the system dynamically coalesce into a functional whole; how higher and lower elements hierarchically organize over time; and how components from within the system integrate online with those in the immediate environment (Cole, Martin, & Dennis, 2004; Fogel, 1993; Gross, 2013; Heatherton & Wagner, 2011; Ochsner, Silvers, & Buhle, 2012; Ojeda et al., 2006; Thelen & Smith, 1994). Disruptions to early regulatory processes are known to underpin later regulatory difficulties, including psychopathology (Lougheed & Hollenstein, 2012; Woltering, Granic, Lamm, & Lewis, 2011), mood disorders (Joormann & Gotlib, 2010), social maladjustment (Eisenberg, Valiente, & Eggum, 2010), attention problems (Shiels & Hawk, 2010), and anger management (Robertson, Daffern, & Bucks, 2012). Conceptual models suggest that regulatory processes mature on top of each other from simple to complex and that parent-child coregulation supports maturation of higher-order regulatory skills, including attention modulation, emotion recognition, social participation, and self-control (Eisenberg, 2000; Feldman, 2009, 2015a,b; McRae et al., 2012; Pons, Lawson, Harris, & De Rosnay, 2003; Tucker, Derryberry, & Luu, 2000).

The mother's consistent, well-adapted behavior serves an important regulatory role, and through external-regulatory, synchronous, and age-appropriate caregiving mothers support the development of children's regulatory skills (Feldman, 2007a, 2015a). Patterns of regulatory caregiving evolve over time. Whereas some aspects of the mother's regulatory behavior remain constant, such as acknowledging the child's signals, adapting to his or her level

of arousal, monitoring the child's physical and mental state and regulating communications accordingly, and providing a "supportive presence" that is safe and secure, other components become more important as children grow, for instance, expansion of social bids in ways that promote symbolic expression, creating a context that enhances creativity, and providing appropriate limits and direction to the social dialogue. In late childhood and adolescence, regulatory caregiving must also integrate recognition of the child's perspective, empathy to his or her emotional experiences, and the capacity to assume a mental stance that verbalizes the child's inner experiences. Such regulatory caregiving, in which mother adapts her behavior to the child's expanding abilities, supports a host of child regulation-based outcomes, including emotion recognition, self-control, social participation, behavior adaptation, and executive functions (Bernier, Carlson, Deschênes, & Matte-Gagné, 2012; Feldman, 2007a, 2010, 2015a, b; Morris, Silk, Steinberg, Myers, & Robinson, 2007; Moss et al., 2011; Spinrad et al., 2007; Wilson, Havighurst, & Harley, 2012).

Depressed mothers fail to provide age-appropriate regulatory parenting to their children. Mothers with MDD are more withdrawn and less sensitive, show a restricted range of affective expression, create a tense and constricted atmosphere, express more negative emotions, have difficulty maintaining physical closeness, and shift unpredictably between moments of withdrawal and anger (Feldman et al., 2009; Field, 2010; Granat, Gadassi, Gilboa-Schechtman, & Feldman, 2017; Jameson, Gelfand, Kulcsar, & Teti, 1997; Malphurs, Raag, Field, Pickens, & Pelaez-Nogueras, 1996). Such inconsistent style impairs the physiological foundations that support the maturation of self-regulatory competencies (Feldman, 2015a; Murray & Cooper, 1997). Yet, research on maternal MDD and parenting behavior focused on infancy and very few studies addressed the mother's style in preschool (Apter-Levi et al., 2016), later childhood (Priel, Djalovski, Zagoory-Sharon, & Feldman, 2019), or adolescence (Gordon et al., 1989).

Emotion Recognition and Maternal Depression

Emotion recognition (ERc) is a key component of emotion understanding—a multifaceted construct that has been implicated in the development of children's social and cognitive competencies (Vollmer & von Salisch, 2017). Emotion understanding involves both ERc and knowledge abilities that target personal and interpersonal emotions and includes understanding of the internal and external triggers of emotions, various qualities of emotions, consequences and function of emotions, cultural rules and norms, and the capacity to manage emotions (Castro, Cheng, Halberstadt, & Grünh, 2016). The term *emotion recognition* covers the perception and labeling of emotional expressions and the ability to use relevant contextual cues for perceiving and naming emotions. It is therefore not surprising that children with better ERc enjoy greater peer acceptance (Arsenio, Cooperman, & Lover, 2000), display more competent social behavior (Domitrovich, Cortes, & Greenberg, 2007), and exhibit better behavioral adjustment (Denham et al., 2002; Izard et al., 2008; Schultz, Izard, & Bear, 2004).

Children's ERc abilities undergo significant development across childhood. By 7 months infants are able to detect common emotions in both face and voice and by the end of the first year infants begin to use others' facial expressions to interpret external events

(Grossmann, 2010). At 4 years, children can accurately label prototypical displays of happiness, sadness, and anger (Widen & Russell, 2003), can identify common emotion-eliciting situations (Denham & Couchoud, 1990a), and can talk about causes and consequences of emotions (Denham, Cook, & Zoller, 1992). By preschool, children become aware of equivocal emotions, conflicting expressive and situational cues of emotions, and the use of personalized information about emotional reactions (Denham, 2007; Denham & Couchoud, 1990b, 1990a). By middle childhood, children can identify prototypical emotions at levels comparable with adults (Durand, Gally, Seigneuric, Robichon, & Baudouin, 2007); yet, they have difficulty recognizing less intense emotions (Thomas, De Bellis, Graham, & LaBar, 2007). Late childhood is the stage when children improve their ability to detect low-intensity emotions and learn to recognize and understand mixed emotion (Larsen, To, & Fireman, 2007).

Parents play a key role in helping children recognize, regulate, and appropriately express emotions. Parents model emotional expression and teach emotional responses during parent-child interactions (Eisenberg, Cumberland, & Spinrad, 1998). With increasing age, parent-child conversations become the context for the communication of appropriate expression of emotions in social situations, beliefs about emotions and their consequences, understanding others' emotional reactions, and accepted strategies of emotion regulation (Thompson, 2011). When parents encourage and support the child's emotional learning, children acquire an increasingly sophisticated emotional literacy and ERc skills (Wilson et al., 2012).

Depressed mothers are less effective in scaffolding emotional development (Hoffman, Crnic, & Baker, 2006). Maternal MDD across the first year predicts less emotional expression and regulation at 9 months (Granat et al., 2017), reduced ERc at 4 years (Maughan, Cicchetti, Toth, & Rogosch, 2007), and maternal depressive symptoms at age 4 predict lower emotion regulation at age 7 (Blandon, Calkins, Keane, & O'Brien, 2008). During preschool, maternal depressive symptoms are associated with less accurate emotional labeling (Székely et al., 2014), impaired ERc (Raikes & Thompson, 2006), and biased perception of sadness (Martin, Williamson, Kurtz-Nelson, & Boekamp, 2015), whereas in later childhood maternal depressive symptoms are linked with errors in identifying emotional intensity (Joormann, Gilbert, & Gotlib, 2010) and biased ERc (Burkhouse et al., 2016). Yet, although studies emphasized the role of maternal behavior in supporting ERc (Doan & Wang, 2010; McElwain, Halberstadt, & Volling, 2007; Taumoepeau & Ruffman, 2006, 2008), we are aware of no study that tested the relations between maternal behavior and ERc in late childhood.

Executive Functions and Maternal Depression

Executive functions (EF) is an umbrella term that taps the goal-oriented control functions of the prefrontal cortex (PFC: Best, Miller, & Jones, 2009). EF comprise a set of cognitive control processes that regulate lower level perceptual and motor responses to orient behavior toward a goal, evaluate risk, flexibly adapt habits, make future plans, and cope with novelty (Snyder, Miyake, & Hankin, 2015). Although no consensus exists on whether EF is a unitary construct or a set of interrelated skills (Best & Miller, 2010), studies of preschoolers and school-age children indicate

that EF consists of related but separable components (Best et al., 2009) which are supported by common mechanisms (Miyake et al., 2000). Three fundamental components of EF have been defined: shifting, inhibition, and updating working memory (Best et al., 2009). Planning is also considered a critical aspect of goal-directed behavior (Best et al., 2009) and involves both identification of a goal and charting the steps to achieve it (Miyake et al., 2000). By late childhood, most tasks that evaluate children's EF abilities involve the coordination of several processes (Best & Miller, 2010).

Similar to ERc, EF skills undergo significant maturation across childhood. During infancy and preschool, core components of EF emerge and set the stage for the development of higher-order cognitive processes that undergo maturation well into adulthood (Garon, Bryson, & Smith, 2008). In the first three years, EF abilities develop separately but begin to integrate during preschool (Garon et al., 2008) and continue their integrated development across late childhood and adolescence (Best & Miller, 2010). Testing the maturation of EF from 5 to 17, it was found that the magnitude of EF improvement was greatest in early childhood (5-7 years), became moderate in late childhood (8-15 years), and diminished in adolescence (15-17 years; Best, Miller, & Naglieri, 2011). Importantly, EF abilities are surprisingly malleable (Zelazo & Carlson, 2012) as the PFC shows plasticity and sensitivity to early caregiving. The slow maturation of the PFC renders EF skills open to favorable environmental inputs but also susceptible to early adversity (Kolb et al., 2012). Thus, whereas individual differences in EF have been viewed as mainly genetic (Friedman et al., 2008), research has increasingly recognized the importance of environmental provisions and failures for the development of children's EF (Hughes, 2011).

Maternal depression interferes with maturation of children's EF and results of a meta-analysis show that maternal MDD uniquely predicts impaired EF performance (Snyder, 2013). Early exposure to maternal depression is related to compromised child EF at age 6 (Hughes, Roman, Hart, & Ensor, 2013), and maternal depressive symptoms in infancy, independent of later exposure, predict poor EF at school entry (Wang & Dix, 2017). Yet, other studies indicate that the associations between maternal depression and children's EF are mediated by other factors, such as low SES (Rhoades, Greenberg, Lanza, & Blair, 2011) or offspring depression (Micco et al., 2009).

Although maternal depression may not directly impact on EF, maternal regulatory behavior may be a key factor mediating the effects of maternal depression on child EF as parenting quality was found to predict child EF (Bernier et al., 2012; Camerota et al., 2015; Cuevas et al., 2014; Devine, Bignardi, & Hughes, 2016; Fay-Stammach, Hawes, & Meredith, 2014; Sarsour et al., 2011). Consistent with the distinction between different subtypes of depression (e.g., Leckman et al., 1984; Schatzberg et al., 1983), evidence supports the notion that postpartum depression is not a unitary phenomenon but a heterogenic one (Kohlhoff, Charles, Sharpe, & Matthey, 2015; Phillips, Sharpe, Matthey, & Charles, 2010; Putnam et al., 2015). Maternal depression may manifest in different ways, and thus the concrete deficits in the maternal regulatory caregiving associated with maternal depression at different nodes may mediate the effects of maternal depression on EF.

A bidirectional link between EF and lower-level regulatory processes has been proposed, such that ERc and attention support

EF and vice versa (Blair & Ursache, 2011). These lower-level abilities mature in the context of the mother-infant coregulation and require sensitive caregiving for their development. Indeed, the link between maternal depression and child EF was found to be fully mediated by the mother's parenting (Baker & Kuhn, 2018). In a 9-year longitudinal study, depressed mothers' insensitive style toward toddlers predicted EF at age 9 (Wang & Dix, 2017), and harsh-intrusive parenting partially mediated the association between maternal depressive symptoms at 15 and 24 months and EF at 4 years (Gueron-Sela et al., 2017). These findings suggest that the effects of maternal depression on EF in late childhood may be mediated by the mother's regulatory caregiving across childhood.

Social Collaboration in Late Childhood and Maternal Depression

The mother's regulatory caregiving supports maturation of children's social engagement and collaborative capacities from infancy to adolescence (Feldman, 2007b, 2010). By late childhood/early adolescence, child social collaboration during interactions with the parent serves as an important index of social-regulatory skills. Children's social collaboration with mother and father from infancy to adolescence were found to predict the ability to resolve conflicts with peers in adolescence (Feldman, Bamberger, & Kanat-Maymon, 2013), and are associated with psychological adjustment at age 13 (Feldman, 2010) and autonomic regulation at 10 years (Feldman, Rosenthal, & Eidelman, 2014). In the context of chronic early life stress, children's social collaboration skills at 10 years index resilience, are associated with lower hair and salivary cortisol and higher oxytocin, and mediate the effects of early life stress on psychopathology (Halevi et al., 2017; Ulmer-Yaniv, Djalovski, Priel, Zagoory-Sharon, & Feldman, 2018). Children's emotion regulation abilities from birth and across the first decade of life were found to predict social collaboration at 10 years (Feldman, 2015a), indicating that collaboration is a regulation-based skill, built not only on temperamental sociality but also on the maturation of regulatory competencies across childhood.

Maternal depression compromises child social collaboration. Infants and children of depressed mothers were found to be more withdrawn and less socially collaborative during interactions (Apter-Levi et al., 2016; Feldman et al., 2009; Ulmer-Yaniv et al., 2018), underscoring the impact of maternal depression on children's social abilities. These findings mirror the greater loneliness and social withdrawal observed in adolescent offspring of depressed mothers described in research using self-reports and clinical assessments (Goodman & Tully, 2006).

The Current Study

In the current decade-long study, we examined the effects of maternal MDD on three child regulation-based outcomes at 10 years—emotion recognition, executive functions, and social collaboration—as mediated by patterns of regulatory caregiving at three time-points across the first decade: infancy, early childhood, and late childhood. At each age, we targeted maternal behaviors that provide age-specific external-regulatory function to supports the maturation of children's regulation-based abilities (Feldman, 2015a,b, 2017) and addressed a panel of social, emotional, and cognitive outcomes that are important for children's functioning in late childhood.

Three hypotheses were formulated. First, we hypothesized that depressed mothers would show deficits in regulatory caregiving at each developmental node (infancy, early childhood, late childhood), and their children would exhibit compromised emotional, social, and cognitive outcome at 10 years, that is, impaired ERc, EF, and SC. Second, we expected that the mother's regulatory caregiving would show individual stability over time, consistent with previous research (Feldman, 2010; Feldman et al., 2013), and would mediate the effects of maternal depression on 10-year outcomes. Models on continuity in caregiving and its effects on long-term outcome chart two separate mechanisms. The first, a *sensitive period* mechanism, postulates that infancy caregiving patterns directly impact outcome (Feldman, 2015b); the second, a *step-by-step* mechanism (Feldman, 2007b, 2015a), suggests that the infancy patterns initiate a cascade of small-step iterations that chart a trajectory, which, in turn, impacts outcome through the continuity of caregiving. To tease apart these mechanisms and specify age-related effects, we examined the specific pathways leading from maternal regulatory caregiving at each time-point to each of the three 10-year outcomes. Finally, the presence of a full-blown psychiatric disorder in the child was hypothesized to mediate the relations between deficits in maternal caregiving and child outcome. Children of depressed mothers display significantly more psychiatric disorders compared with their peers (Apter-Levy, Feldman, Vakart, Ebstein, & Feldman, 2013; Goodman et al., 2011); however, whereas some children exposed to maternal depression show psychiatric disorders, others are more resilient (Priel et al., 2019). ERc is impaired in children experiencing various psychopathologies, including mood disorders, anxiety, ADHD, and conduct disorders (Collin, Bindra, Raju, Gillberg, & Minnis, 2013); EF is lower in children with psychiatric disorders beginning in middle childhood (Hatoum, Rhee, Corley, Hewitt, & Friedman, 2018; Martel et al., 2017; Trentacosta & Fine, 2010; White et al., 2017); and SC mediates the effects of caregiving on child psychopathology in the context of early adversity (Halevi et al., 2017). We thus expected maternal caregiving to impair ERc, EF, and SC both directly and as mediated by the increased prevalence of child psychiatric disorders.

Method

Participants

We recruited a large low-risk community cohort and longitudinally followed a group overselected for maternal depression as compared to healthy controls in order to tease apart the effects of maternal depression on parenting and child outcomes from other comorbid conditions frequently included in research on maternal depression. Figure 1 presents a flowchart of the recruitment process and measures at each time point from birth to 10 years.

Birth and first year. The initial cohort included 1,983 women recruited on the second postbirth day in three maternity wards who completed measures of anxiety and depression. Only mothers who were healthy, completed high-school, were older than 21 years, were married or cohabiting, and above poverty line, and whose infants were term, healthy, and singleton were included. At 6 months, we mailed depression and anxiety questionnaires to women who were in the high (BDI >11; Beck, Ward, Mendelson, Mock, & Erbaugh, 1961) and low (BDI <9) ends of the depressive

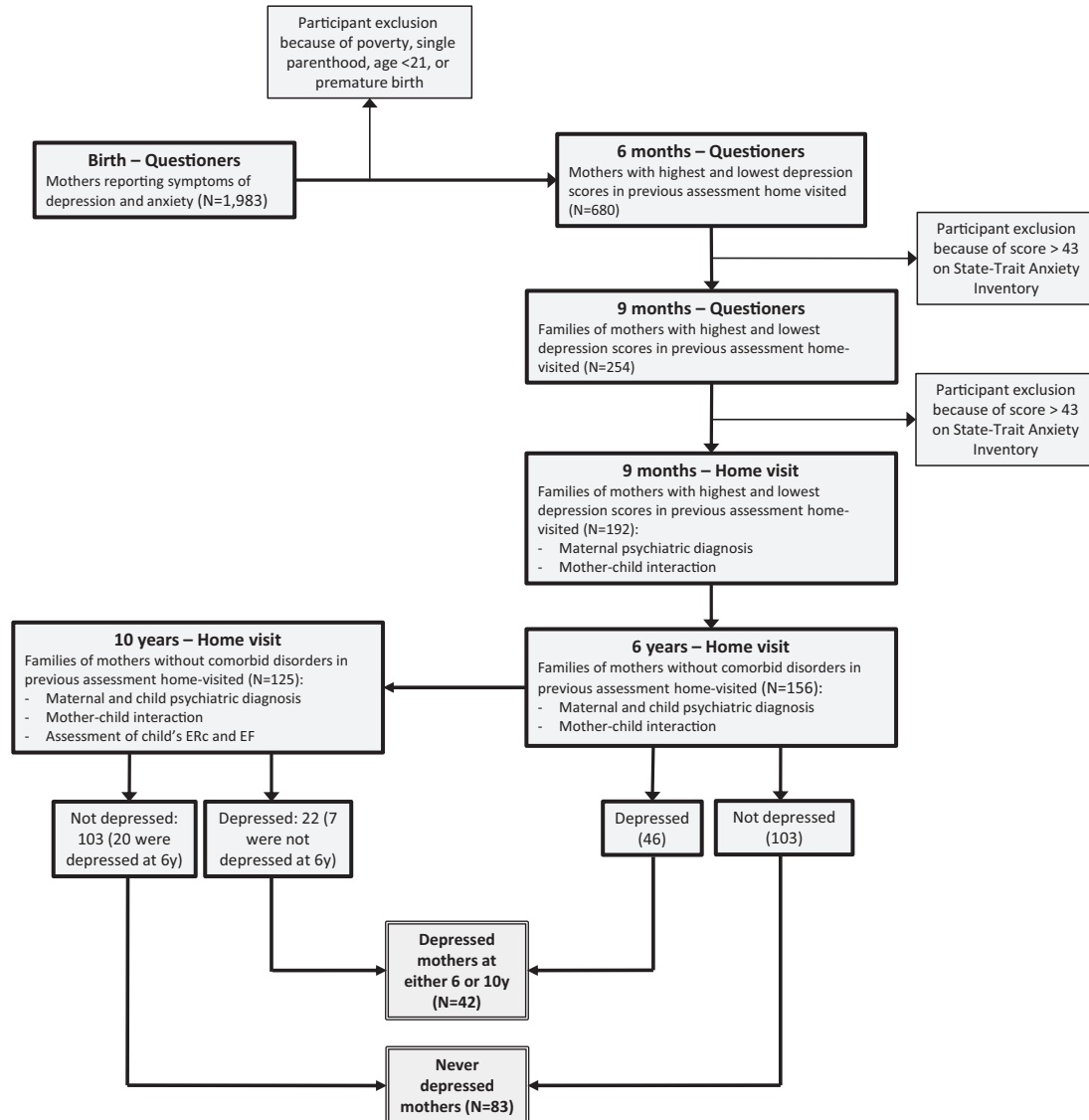


Figure 1. Flow chart of sample recruitment and study measures from birth to ten years.

symptoms continuum at birth. Of the 900 women we mailed questionnaires, 680 women responded and completed questionnaire and sent them by mail (75.5%). At 9 months, we again sent depression and anxiety questionnaires to women at the high- and low-end of the depression continuum at 6 months and of 350 women approached, 254 responded (72.5%). Mothers experiencing high anxiety symptoms (State-Trait Anxiety Inventory Score >43), at either 6 or 9 months were excluded from the study.

Nine months. Of the responding mothers at 9 months, 192 (75.5%) were visited at home. During the 9-month visit mothers underwent psychiatric diagnosis using the SCID-I (First, Spitzer, Gibbon, & Williams, 1997) and mother-child interaction was videotaped and coded (see below).

Six years. Of the 192 families visited at 9 months, 156 (81.2%) were revisited at 6 years (child age 6.33 ± 1.25 , mothers' age 38.66 ± 4.4). Visits were conducted between 4 and 7 p.m. In these 156 families, 80% of the parents had college degree, 91.4%

were married, and 89% of mothers were employed. Forty-six mothers (29.6%) were defined as chronically depressed. These mothers showed high depressive symptoms (BDI >11) at birth and at 6 and 9 months, received MDD diagnosis at 9 months and 6 years, and reported being depressed throughout the child's first six years. Thus, the depressed group at 6 years presents a group of mothers who were continuously depressed across the child's first years of life. One hundred three mothers (66%) were defined as controls. These mothers showed no elevated symptoms at any time-point (birth, 6 months, 9 months, 6 years), and did not receive any Axis-I diagnosis. Seven mothers were excluded because of clinical anxiety (State-Trait Anxiety Inventory Score >43; $n = 3$) or subclinical depression as evaluated using the BDI ($n = 4$). During this visit, mothers were again diagnosed with the SCID-I (First et al., 1997), child was diagnosed with the DAWBA (Goodman, Ford, Richards, Gatward, & Meltzer, 2000), and mother-child interaction videotaped.

Ten years. Of the families visited at 6 years, 125 (80.1%) were located and visited at home at 10 years (child age 9.63 ± 0.65 , range = 9–11 years, mothers' age 39.06 ± 5.64) and visits were again at 4–7 p.m. Attrition was mainly related to inability to locate families. There were no significant differences in demographic variables or psychopathology rates and distribution between those who dropped out and those who continued at 6 and 10 years. Of the 125 mothers, 22 mothers were diagnosed as experiencing MDD at this time point and 103 were not. At 10 years, mothers were diagnosed again with the SCID-I (First et al., 1997), children were diagnosed again with the DAWBA (Goodman et al., 2000), mother–child interactions were videotaped, and children's EF and ERc were assessed (see below).

Measures

Maternal psychiatric diagnosis. The Structured Clinical Interview for *Diagnostic and Statistical Manual of Mental Disorders*, fourth edition (*DSM-IV*) Axis I Disorders (SCID-I; First et al., 1997) was administered at 9 months, 6 years, and 10 years. At 10 years, 15 of the 46 depressed mothers at 6 years (42.8%) were still diagnosed with MDD, whereas 20 (57.1%) were not. Of the nondepressed mothers at 6 years, 83 mothers (92.2%) remained without a diagnosis at 10 years, whereas 7 (7.7%) received MDD diagnosis. We defined the maternal depression group as mothers receiving MDD diagnosis at 6 years, 10 years, or both (42.8%). This resulted in 42 depressed mothers and 83 control mothers (mothers without diagnosis throughout the first decade of the child's life). Diagnoses at all time points were conducted by a clinical psychologist supervised by child psychiatrist with case conferred every few weeks and reliability exceeding 85%.

Child psychiatric diagnosis. The Development and Well-Being Assessment (DAWBA) is a well-validated structured interview and questionnaire that generates ICD-10 and *DSM-IV* diagnoses in children 4–16 years (Goodman et al., 2000). Diagnoses were conducted by a clinical psychologist supervised by a child psychiatrist blind to any other information with case conferred every few weeks and reliability exceeding 85%. The group of child disorder included children receiving Axis-I diagnosis at 6 years, 10 years, or both (52.8%), with 31.8% having more than one disorder at one or both time points. No difference was found between the number of comorbid disorders at 6 years and 10 years. Diagnoses at 6 years included: affective disorders (25.6%), ADHD (9.6%), developmental delay/ASD (3.2%), and tic-disorder (0.8%). Diagnoses at 10 years included: affective disorders (20%), ADHD (19.2%), developmental delay/ASD (1.6%), and tic-disorder (3.2%). Only the prevalence of ADHD increased significantly from 6 to 10 years ($p < .01$).

Mother–child interaction. At 9 months, mother–child interactions included 6 min of free play. Mothers were instructed to play with the infants freely as they typically do. At 6 years, interactions included 10 min of play with age-appropriate toys that elicit creative-symbolic play (e.g., tea set, cars, little figures etc.). At 10 years, mothers and children engaged in two well-validated discussion paradigms for 7 min each. The first involved a typical conflict in their relationship, the second involved planning “the best day ever” to spend together. These paradigms and their coding were validated in our lab in several studies of healthy and high-risk children at this age (Feldman, 2010; Feldman et al., 2013, 2014;

Halevi et al., 2017; Ulmer-Yaniv et al., 2018). Interactions were coded by coders blind to all other information including maternal and child psychiatric status using the Coding Interactive Behavior Manual (CIB; Feldman, 1998). The CIB is a well-validated rating system for social interactions in infants, children, adolescents, and adults that includes multiple scales for parent, child, and dyad, aggregated into theoretically derived composites with good psychometric properties (Feldman, 2012). Coding was conducted by trained coders, blind to other information, and reliability on 20% of the interactions exceeded 93% and 90% on all codes ($k = .84$, range .78–.95 and $k = .82$, range = .78–.96) at 6 and 10 years, respectively.

Maternal regulatory caregiving. We selected specific theoretically driven codes from the CIB to create the maternal regulatory caregiving at each age according to the developmental needs of the child at this stage. Consistent with theory (Feldman, 2009), the maternal regulatory caregiving factor included an increasingly expanding repertoire to match the child's increasing abilities. Thus, the maternal factor at 6 years included all the 9-month codes plus additional codes that match the child's 6-year abilities, and the 10-year maternal factor included all the 6-year codes plus additional codes relevant to 10-year-old children.

At 9 months *maternal regulatory caregiving* included the following averaged codes: maternal adaptation–regulation to infant's state and signals, maternal acknowledgment of the infant's non-verbal communications, maternal calmness, maternal supportive presence, maternal warm vocalizations, appropriate range of affective expressions, and maternal enthusiasm and involvement in building and maintaining a positive social atmosphere (Cronbach's $\alpha = .78$).

At 6 years, the *maternal regulatory caregiving* factor included the same variables as 9 months, in addition to the following codes: mother's expressing positive affect through verbal and nonverbal channels, mothers organization of the interaction, and maternal elaboration of the child's verbal communications, topics, and imaginary play (Cronbach's $\alpha = .79$).

At 10 years, the *maternal regulatory caregiving* factor included, in addition to the 6-year factor also the following codes: mothers' limit-setting and maternal empathy and understanding of the child's mental state, desires, and perspective (Cronbach's $\alpha = .77$).

Child social collaboration was coded from the mother-child interactions at 10 years and included the following averaged codes: child motivation to engage socially and remain on task, child collaboration with the mother's suggestions, questions, and discussion, child vocalization (including both amount and tone of vocal output), and child attention is maintained on mother and topic of conversation (Cronbach's $\alpha = .78$).

Child executive functions. Children were administered the Stockings of Cambridge test from the computerized Cambridge Neuropsychological Testing Automated Battery (CANTAB, Cambridge Cognition, Cambridge, U.K.). The Stockings of Cambridge is a spatial planning task that is similar to the Tower of London task, a derivative of the classic Tower of Hanoi (Shallice, 1982), with the advantage of having an automated data collection. Task completion requires the coordination of multiple EF processes that are supported by the development of core EF processes including response inhibition and working memory (Asato, Sweeney, & Luna, 2006). In this task, the computer screen is split into a top half

and a bottom half. Each half contains three rows of “black holes.” Three holes are in the first row, two in the second row, and one in the third row. Three colored balls (blue, red, green) are placed in predetermined position in the displays. The balls in the lower display are in different locations from those in the upper display. The child is told that the upper display is the model and the goal is to make the lower display match the upper display by moving the balls to different locations in a minimum number of moves possible. The child must move the balls in the lower display to copy the pattern shown in the upper one. The balls may be moved one at a time by touching the required ball, then touching the position to which it should be moved. Only balls at the top of a stack can be moved. The starting position of the balls differs on each trial so that the solution can be reached after a minimum of two, three, four, or five moves. The problem is terminated if the participant makes more than double the number of moves that are necessary for the simplest solution. The test ends after the computer terminates three problems in a row. The number of problems solved in the minimum possible moves (maximum 12) was used here to index EF.

Emotion recognition. The *Emotion Recognition Toolbox* is a computerized program developed at our lab. The paradigm tests the recognition and knowledge of emotions that appear in various levels of intensity and the recognition of both single and multiple emotions. It includes 15 brief 30-s movies that demonstrate happy, sad, angry, and neutral emotions of low or high intensity. After each movie, the child is asked by the computer whether each emotion appeared in the movie and once the child pressed yes or no another set appeared and asked if these emotions also appeared. The proportion scores for each emotion were similar (anger: 82%, happy: 85.6%, and sad 83.4%). The final score was the proportion of correct recognition of emotion. Split-half reliability between trials were $r = .91$, $r = .93$, and $r = .88$, for anger, happy, and sad.

Ethical considerations. The study was approved by the IRB of Bar Ilan University for the project “developmental outcome of maternal depression” and all families signed informed consent. Procedures were conducted according to ethical guidelines and explained to participants before study. Participants received a small gift for participation.

Statistical Analysis

Chi-square and t tests were used to compare the study variables between children of depressed and nondepressed mothers. Pearson correlations tested the relationships among our main variables. For a comprehensive model on the direct and mediated paths leading from maternal MDD to children’s ERc and EF as mediated by child disorder and maternal regulated behaviors, we conducted a path analysis using lavaan 0.6–3 package (Rosseel, 2012) in R 3.5.2 (R Core Team, 2014; RStudio, 2015). After, we tested our proposed model. Path analysis was based on maximum likelihood estimations and indicators of model fit were chi-square values, root mean square error of approximation (RMSEA), comparative fit index (CFI), with Tucker-Lewis index (TLI) values >0.95 considered good fit (Hu & Bentler, 1999). To assess the significance of the mediation effects, we used a procedure recommended by Hayes (2013) and calculated the 95% confidence intervals of 5,000 bias-corrected and accelerated bootstrapping analyses (Hayes,

2013; MacKinnon, Lockwood, & Williams, 2004). In cases in which the value zero is not included in the confidence interval, this indicates a significant effect at $\alpha < .05$. Preacher and Hayes (2008) contemporary statistical approach suggests that it is enough to show an association between the independent and mediator variables and between the mediator and dependent variables to infer a mediation model. After testing that our less than 10% missing data were missing at random, we chose to impute the mean value of the variable. The results before and after imputation were not significantly different and held the same patterns. Lastly, to ease the visual presentation of our model, although all paths were tested, only the significant paths are shown.

Results

To maximize sample size and address child outcomes in the context of exposure to maternal MDD, we combined into the *depressed* group children exposed to maternal MDD at birth-to-six years ($N = 46$), at 10 years ($N = 22$), or both ($N = 15$), resulting in a total group of 53 depressed mothers. No significant differences ($p > .05$) were found in any study variable between children exposed to maternal MDD at 6 or at 10 years. As hypothesized, children of depressed mother were more likely to display a full-blown psychiatric disorder at both 6 years ($\chi^2_{(1)} = 10.7, p < .01$) and 10 years ($\chi^2_{(1)} = 5.75, p < .05$).

Results of the t tests on the main study variables according to group are presented in Figure 2 and mainly support our first hypothesis. Depressed mothers displayed lower regulatory caregiving at 9 months, 6 years, and 10 years. Children of depressed mothers showed significantly less social collaboration and lower emotion recognition, but no differences were found between the group in children’s EF.

Pearson’s correlations among study variables appear in Table 1. As seen, maternal regulatory caregiving was individually stable from 9 months to 10 years. Maternal regulatory caregiving at 6 years longitudinally correlated with EF and ERc and maternal regulatory caregiving at 10 years was related to social collaboration and ERc. Children’s EF and ERc were also inter-related.

Next, we used path analysis to test our conceptual model on the pathways linking maternal depression and children’s ERc, social collaboration, and EF as mediated by maternal regulatory behaviors (hypothesis 2) and child psychiatric disorder (hypothesis 3). The overall model, presented in Figure 3, provided excellent fit to the data: $\chi^2_{(9)} = 6.611, p = .687, RMSEA = .000$ with lower 90% CI = .000 and higher 90% CI = .08, PCLOSE = .838, CFI = 1.00, TLI = 1.066.

Five significant paths were identified. Whereas the first two show direct pathway from maternal regulatory behavior at 6 and 10 years on outcome, the last three converge through child disorder.

Pathways Mediated by Maternal Regulatory Behavior

Maternal MDD–maternal regulatory behavior 6 years–EF and ERc. The first path linked maternal depression with lower maternal regulatory behavior at 6 years, and maternal regulatory

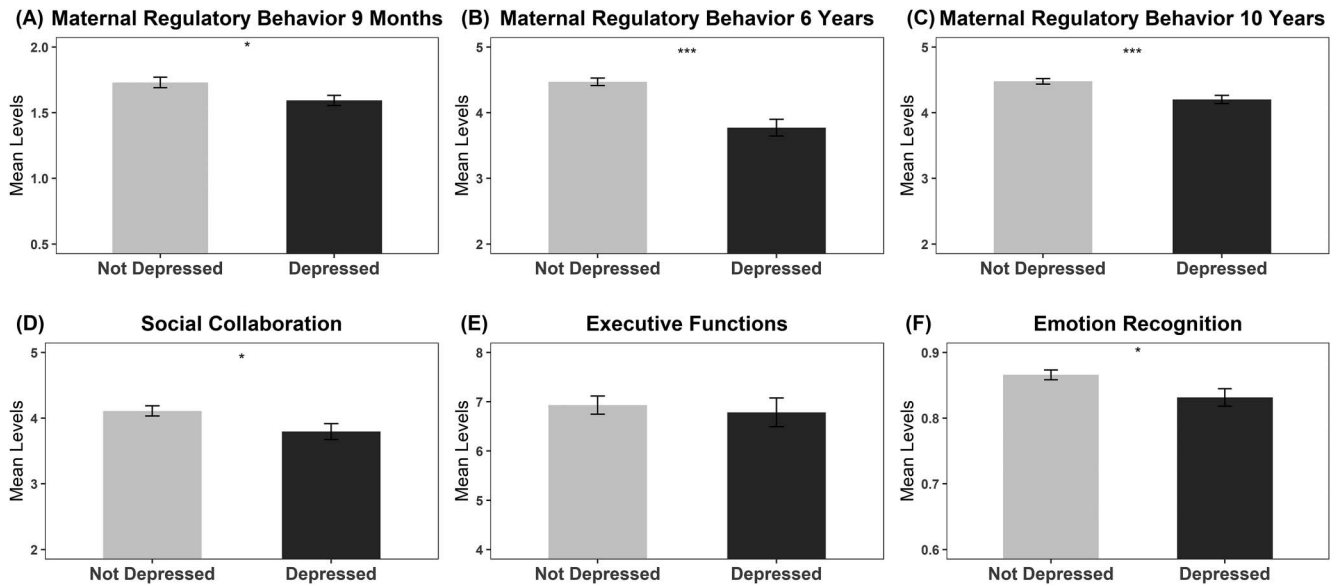


Figure 2. Group differences in study variables. Children of depressed mother experienced less regulatory behaviors at 9 month, $t_{(123)} = 2.18, p < .05$, Cohen's $d = .44$, 6 years, $t_{(123)} = 5.8, p < .001$, Cohen's $d = 1.02$, and 10 years, $t_{(123)} = 3.73, p < .001$, Cohen's $d = .7$. In addition, their children showed less social collaboration, $t_{(123)} = 2.26, p < .05$, Cohen's $d = .42$, and less ERc, $t_{(123)} = 2.44, p < .05$, Cohen's $d = .44$. No significant differences were found in EF, $t_{(123)} = .45, p > .05$, Cohen's $d = .08$. * $p < .05$. *** $p < .001$.

behavior were linked with greater EF and ERc skills (EF: 95% CI [-.181, -.016], ERc: 95% CI [-.267, -.034]).

Maternal MDD–maternal regulatory behavior 10 years–ERc. The second path linked maternal depression with lower maternal regulatory behavior at 10 years and such maternal regulation was associated with higher ERc (95% CI [-.141, -.004]).

Pathways Mediated by Maternal Regulatory Behavior and Child Psychiatric Disorder

Maternal MDD–maternal regulatory behavior 9 months–child disorder–EF, social collaboration, ERc. The third path linked maternal depression with lower maternal regulatory behavior at 9 months, which led to child disorder. Diagnosed children had lower EF, ERs, and SC. Test of mediation indicated that this indirect path were significant (EF: 95% CI [-.026, -.001], SC: 95% CI [-.027, -.001], ERc: 95% CI [-.024, -.001]).

Maternal MDD–maternal regulatory behavior 6 years–child disorder–EF, social collaboration, ERc. The fourth path was identical to the third but linked maternal depression with lower

regulatory behavior at 6 years. This path converged to the first path (EF: 95% CI [-.058, -.002], SC: 95% CI [-.057, -.002], ERc: 95% CI [-.049, -.002]).

Maternal MDD–maternal regulatory behavior 10 years–child disorder–EF, social collaboration, ERc. The final path showed the same pattern as the previous two but operated via the effects of maternal regulatory behavior at 10 years (EF: 95% CI [-.038, -.001], SC: 95% CI [-.041, -.002], ERc: 95% CI [-.035, -.001]).

Discussion

Although depression in women of childbearing years is highly prevalent, rendering millions of children exposed to its toxic effects, very few studies followed children of clinically depressed mothers from birth and across lengthy periods, integrating repeated observations of caregiving with assessment of age-specific regulation-based outcomes. Particularly lacking are studies that focus on developmental periods beyond infancy, extend to abilities beyond global psychopathology, and test theoretically derived

Table 1
Pearson Correlations Between Main Study Variables

Variable	1	2	3	4	5
1. Maternal regulatory behavior–9 months					
2. Maternal regulatory behavior–6 years	.18*				
3. Maternal regulatory behavior–10 years	.20*	.23**			
4. Social collaboration	.08	.08	.30**		
5. Executive functions	-.05	.24**	.09	.11	
6. Emotion recognition	.08	.37**	.28**	.14	.25**

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

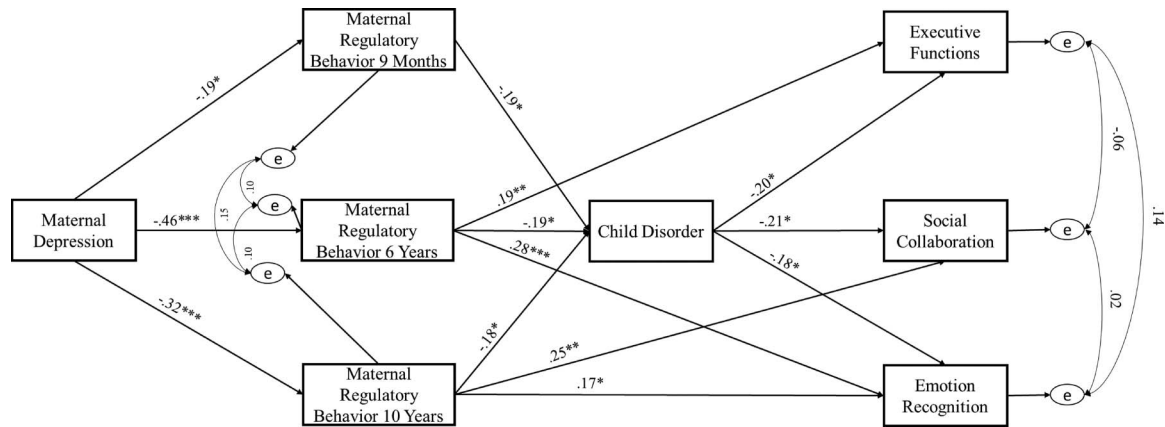


Figure 3. Path analysis model. Model fit: $\chi^2_{(9)} = 6.611, p = .687, RMSEA = .000$, with lower 90% CI = .000 and higher 90% CI = .08, PCLOSE = .838, CFI = 1.00, TLI = 1.066. * $p < .05$. ** $p < .01$. *** $p < .001$.

longitudinal mediators of outcomes. Here, we examined how disruptions to the environment-expected regulatory caregiving mothers provide at various developmental nodes longitudinally impair three child regulation-based abilities in late childhood; emotion recognition, executive functions, and social collaboration. We found that by 10 years, children exposed to maternal MDD had lower emotion recognition and social collaboration, and while no mean-level differences were observed in EF, the deficits in regulatory caregiving mediated the effects of maternal depression on EF. Regulatory patterns of maternal care were individually stable across the first decade and their impact on outcome was both direct and mediated by the child's psychiatric disorder, combining two mediating pathways in the transmission of vulnerability from depressed mothers to their offspring, a within-system path and a contextual path, consistent with dynamic systems' models (Feldman & Eidelman, 1998; Fogel, 1993; Thelen & Smith, 1994). Finally, we found that maternal caregiving in infancy did not have a direct impact on outcome 10 years later, but exerted indirect effects, through continuity in caregiving over time and via increasing the child's propensity to develop a psychiatric disorder by early or late childhood. Such findings contribute to the discussion on continuity and change over lengthy developmental epochs, a core issue in understanding human development (Sroufe & Jacobvitz, 1989). Overall, our study highlights the critical role of the mother's age-specific external-regulatory function for maturation of children's regulation-based abilities and charts direct and mediated paths by which regulatory maternal care facilitates the development of core abilities during late childhood.

Our findings describe how emotional, social, and cognitive regulation-based functions develop in children of depressed mothers during late childhood in relation to caregiving experienced across the first decade of life, providing a window into the ingredients required at each developmental stage for maturation of these skills. Our results are consistent with theories on the development of regulatory processes (Cole et al., 2004; Feldman, 2009, 2015a; Gross, 2013) and show how regulatory abilities depend on the integration of components from within the system, such as psychopathology, with those in the immediate environment, including mother's depression and caregiving patterns, to chart trajectories of risk and resilience. In infancy, the maternal external-regulatory

function is essential to support the foundation of self-regulation and expresses in care that is safe and calm, provides supportive presence, acknowledges the child's communications, adapts to changing infant state, builds and maintains positive arousal, and can flexibly shift across affective states (Cole et al., 2004; Feldman, 2007). Mothers engage in moment-by-moment adaptations to their infant's cues (Cohn & Tronick, 1988; Feldman, 2003), and such online adaptation builds the brain's regulatory structures, including the social brain and neuroendocrine systems that support stress management and affiliation throughout life (Feldman, 2016, 2017). Perturbations in this expected social environment, as seen, for instance, in the still-face paradigm that simulates both the unpredictability and lack of social behavior typical of the depressed mother, reliably elicit alterations in infant physiology, emotions, and self-regulation as a function of changes in the maternal affect (Cole et al., 2004; Pratt, Singer, Kanat-Maymon, & Feldman, 2015; Tronick, 1989). Although numerous studies have shown that depressed mothers fail to provide the careful, time-bound external regulation to their infants (Beebe et al., 2008; Field, 2010; Granat et al., 2017; Paris, Bolton, & Weinberg, 2009; Pratt, Goldstein, Levy, & Feldman, 2017), nearly no research exists on how such style develops over time. Our findings indicate that the infancy regulatory caregiving style did not exert a direct impact on outcome but operated through two pathway, first by initiating an individually stable style, which, like expanding ripples, incorporated increasingly complex abilities into the maternal regulatory caregiving as children mature. Second, the depressed mothers' limited regulatory caregiving in infancy was directly linked to increased prevalence of child psychiatric disorders at 6 and 10 years, which predicted compromised outcome.

After infancy, when children begin to communicate verbally, the mother's external-regulatory function must expand to support more complex dialogues. At 6 years, to regulate social interactions, mothers must learn to elaborate and expand the child's symbolic play and creative initiations and organize interactions to provide order while maintaining the positive atmosphere. In late childhood, when the context of interactions shifts from toy play to conversations, the external-regulatory style must include limit-setting during conflict discussion but also empathy to the child's position and perspective. These more mature and verbal regulatory caregiving

encourage the formation of the child's independent viewpoints, strengthen the sense of self, broaden emotional literacy, and promote verbal strategies for the modulation of negative arousal. As seen, the depressed mother's limited ability to provide regulatory caregiving at the verbal stages was directly predictive of compromised outcome; regulatory caregiving at 6 years predicted both EF and ERc at 10 years, and regulatory caregiving at 10 years predicted concurrent ERc. Our findings are novel by focusing on the mother's regulatory role across the entire first decade, specifying age-specific regulatory patterns, and detailing how regulatory caregiving behaviors mature on top of each other from simple nonverbal matching to complex verbal and interpersonal communications.

Children of depressed mothers showed lower emotional recognition abilities, and such skills were related to poor regulatory caregiving at both 6 and 10 years, to child psychiatric disorder, and to EF abilities. Similarly, social collaboration was compromised in children of depressed mothers. According to Gergely and Unoka's (2008) model, the development of higher cognitive functions, such as ERc and EF, are intimately linked with the mother-child relationship. Through synchronized, reciprocal, and attentive interactions, the child forms the capacity for mentalization, which sets the stage for maturation of higher emotional and cognitive functions. When maladaptive patterns become dominant, they undermine the child's ability to develop mental capacities, impairing ERc abilities. Children of depressed mothers develop an avoidant social style at a very early age, and the limited social engagement continues throughout development (Field, Healy, Goldstein, & Guthertz, 1990). Such withdrawal limits their mentalization capacity, directly impacting higher cognitive and emotional abilities.

Although the child's socially engaged orientation is biologically based, it is also shaped by sensitive and synchronous parenting and shows individual stability over time and across relationships with mother, father, and close friends (Feldman, 2010; Feldman, Gordon, Influss, Gutbir, & Ebstein, 2013). Coregulatory, synchronous parenting prepares children to function competently within the social world, acquire a social repertoire, master appropriate social skills, and develop a collaborative rather than withdrawn style (Feldman, 2010; Feldman & Masalha, 2010). Social collaboration in childhood was found to predict greater competence in the peer group, better emotion regulation and socialization, and lower psychopathology (Feldman & Masalha, 2010). Social withdrawal, on the other hand, is associated with a range of negative outcomes, including anxiety, low self-esteem, depressive symptoms, internalizing problems, peer rejection and victimization, poor friendship quality, poor teacher-child relationships quality, academic difficulties, school avoidance (Rubin, Coplan, & Bowker, 2009), and less emotional knowledge (Schultz, Izard, Ackerman, & Youngstrom, 2001). Children of depressed mothers show increased social withdrawal during mother-child interactions, mirroring the mother's withdrawn style and this has been shown in infancy (Feldman et al., 2009; Field, 2010), preschool (Apter-Levy et al., 2013; Jameson et al., 1997; Pratt, Apter-Levi, et al., 2015), and middle childhood (Lee et al., 2013). Social withdrawal impairs children's emotional development (Rubin et al., 2009; Schultz et al., 2001) and is associated with disruptions in FFC maturation in terms of function, white matter, and myelination (Liu et al., 2012; Maki-nodan, Rosen, Ito, & Corfas, 2012; Sánchez, Hearn, Do, Rilling, & Herndon, 1998). Our findings uniquely demonstrate the effects of

maternal depression on decreased social collaboration, as measured by reduction in the child's initiating, engaged, mutual, and alert social style.

The associations between deficits in regulatory caregiving and disruptions to children's EF performance at 10 years is similarly novel. Animal studies have shown causal links between social deprivation in early life and impaired EF performance along with abnormalities in brain regions that support EF (Hostinar, Stellern, Schaefer, Carlson, & Gunnar, 2012). Rat pups experiencing disruptions to maternal care exhibit lower levels of plasticity-supporting neurotrophins, which are most pronounced in the PFC (Roceri et al., 2004), and show compromised performance on EF-related tasks, such as rule shifting (Lovic & Fleming, 2004). In rodents, chronic stress exposure leads to dendritic alterations in the medial PFC and orbital frontal cortex which are associated with impaired attention set-shifting performance (Liston et al., 2006). In nonhuman primates, early social deprivation is associated with reduced cognitive performance (Sánchez et al., 1998) and abnormalities in PFC and its associated areas (Sánchez, Ladd, & Plotsky, 2001). This is consistent with human studies showing that adopted children exhibit reduced glucose metabolism in the orbitofrontal gyrus and infralimbic PFC (Chugani et al., 2001) and reduced EF performance (Hostinar et al., 2012). Our findings add the links between the experience of limited and inconsistent caregiving across the first decade and poor EF performance in late childhood.

Both maternal regulatory behavior and child psychiatric disorder mediated the relations between maternal depression and child outcome. Whereas the child's psychiatric disorder is likely influenced to some extent by the transmission of genetic vulnerability, longitudinal studies on high-risk children, whether because of biological, maternal, or contextual factors, indicate that maternal caregiving and child dispositions mutually influence each other over time to shape development, particularly with regard to outcomes that define regulation-based components of the system which are open to environmental influences (Feldman, 2015a, 2015b; Feldman & Eidelman, 2009). Our findings indicate that although at 6 and 10 years the deficits in maternal caregiving directly impacted outcome, child psychiatric disorder mediated the effects of caregiving on all three outcomes, suggesting that whether a child is on a risky or a more resilient trajectory is an important determinant in adaptive functioning. Our model may contribute to the discussion on resilience, among the key topics in developmental research much in need of further conceptualization and longitudinal studies. Resilience—defined as positive outcome despite early adversity (Masten & Monn, 2015)—implies that some children growing up in adverse contexts, such as exposure to maternal MDD, are able to thrive despite harsh caregiving, and our findings highlight some potential components of such thriving. It is possible that some depressed mothers still provide elements of the age-appropriate regulatory caregiving at some points across childhood, and interventions may thus target these specific maternal behaviors at the appropriate ages. Similarly, although significantly more children of depressed mothers develop psychiatric disorders during childhood, many children do not, and because child disorder mediated the effects of depression on all three outcomes, the nondisorder group may fare better in terms of key cognitive, social, and emotional functions. These findings underscore the need to develop interventions that teach mothers the

specific regulatory behaviors needed at each stage or interventions that seek to provide these relational ingredients by other benevolent adults in the child's life, such as fathers, grandparents, or teachers/mentors.

Our findings highlight late childhood—a time when children are mature enough to possess emotional literacy and cognitive control abilities yet have not reached adolescence and full self-regulatory maturity—as a period that requires much further empirical attention. Late childhood is a period of opportunities for interventions, before the onset of puberty and its storms, yet it is also a period when symptoms consolidate and predict psychopathology in adolescence and adult life. Children's behavior problems in late childhood were found to predict elevated risk for alcohol, nicotine, and cannabis use (King, Iacono, & McGue, 2004), ADHD, and mood disorders in adolescence (Masi, Pisano, Milone, & Muratori, 2015). Similarly, psychological symptoms in late childhood increase the risk for ADHD, substance use, suicidality, and poor functioning in young adulthood (Holtmann et al., 2011). Thus, our findings on the increased risk for psychiatric disorders at 10 years among children exposed to maternal MDD should raise concern. It is worth noting that because children's EF and ERc were evaluated only at 10 years, it was not possible to examine the bidirectional hypothesis; that early EF and ERc disruptions predict psychopathology at 10 years. Such longitudinal analysis is important in light of recent models that emphasize the role of early EF impairments in the emergence of psychiatric disorders (Wakschlag et al., 2018).

Our decision to focus on maternal depression and exclude comorbid cases of depression and anxiety stemmed from the fact that although these two populations share some common features, they differ in many aspects, and our goal was to specify the effects of maternal depression per se on child development. For example, data from the Netherlands Study of Depression and Anxiety on 1783 participants found that individuals suffering from comorbidity of anxiety and depression exhibit more severe depression, longer durations of depressive episodes, and earlier onset compared with those with depression only (Lamers et al., 2011). A large survey of 74,045 participants across 24 countries found that individuals with comorbidity of anxiety and depression experience more severe functional impairments and suicide ideations (Kessler et al., 2015). Other studies showed differences in EEG patterns between the two groups (Nusslock et al., 2018) and associations between immune dysregulation, cortical thinning, and cortico-limbic dysfunction in cases of comorbidity of depression and anxiety but not in depression only (Gaspersz, Nawijn, Lamers, & Penninx, 2018). Finally, in relation to parenting, a study comparing the effects of being raised by a depressed, anxious, comorbid, and healthy mother found distinct differences between the impact of maternal depression and comorbidity on children's emotional and behavioral functioning (Prenoveau et al., 2017). Our findings, therefore, provide a first step and should be examined in cases of comorbidity to test the differential effects of these two conditions.

Our findings have clear implications for interventions. First, as our study included only low-risk families, omitting cases of poverty, single parenting, premature birth, or teenage mothering, impairments to child ERc, SC, and EF in children with more cumulative risk is probably greater (Sameroff, Seifer, Barocas, Zax, & Greenspan, 1987), specifying these outcomes as goals for intervention. As maternal depression is among the most underdiagnosed psychiatric disorders in the community (Ko, Farr, Dietz, &

Robbins, 2012), awareness to its long-term toxic effects on a host of child functions is paramount. Second, our study pinpoints components of the mother's external-regulatory caregiving at various stages that should be targeted in parenting interventions, particularly interventions that employ active modeling, such as video-feedback interventions (Klein Velderman, Bakermans-Kranenburg, Juffer, & Van Ijzendoorn, 2006). Finally, the findings that child psychiatric disorders mediated risk and resilience highlight the need to monitor very carefully children who consolidate symptoms upon school entry and provide special treatment for those on a risk trajectory. Findings also highlight the importance of engaging fathers when mothers are depressed, and prior research has indeed shown that in the context of chronic maternal depression, sensitive and involved fathering can mediate some of the negative effects on child psychopathology (Vakrat, Apter-Levy, & Feldman, 2018b) and on children's social collaboration (Vakrat, Apter-Levy, & Feldman, 2018a).

Several study limitations should be considered. First, as we focused on the long-term effects of maternal depression apart from other comorbidities, our findings need replication in higher risk samples. Second, as both parents shape their children's well-being and regulatory outcomes, the omission of fathers is an important limitation. Third, although we used an EF task that requires coordination of multiple processes, our findings need replication using additional EF tasks. Fourth, our sample size did not enable separating children exposed to maternal MDD at 6 and at 10 years, and larger studies should examine the effects of maternal depression at different developmental stages. Finally, the sample included only married/cohabiting couples from a medium-to-high SES. Although these criteria were chosen to control for potential confounds, it limits the ability to generalize the results to other populations. Future studies should broaden the study to include other populations, such as single parents and families from other backgrounds and social status. Much further research is required to follow children of depressed mothers as they go through other developmental challenges, including the pubertal transition, leaving home, forming pair-bonds, and eventually nurturing their own children and how disruptions to self-regulatory processes continue to impair their lives, with the hope that better recognition of specific regulatory difficulties in such children may lead to the construction of age-specific targeted interventions.

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