

Justus-Liebig Universität Gießen

**Major depression and mother-infant interaction:
Affect-related behaviors in presence of a major
depressive episode and after remission compared
to non-depressed control mothers**

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Dedicated to Corinna Reck and Christiane Hermann.

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Major depression and mother-infant interaction: Affect-related behaviors in presence of a major depressive episode and after remission compared to non-depressed control mothers

1. Introduction: Research targets and basic ideas

The past three decades have seen an accumulation of findings that led to the overwhelming conclusion that children with affectively ill parents are at increased risk for deviancies, either in behavior, or for developmental impairments or psychopathology (Beardslee et al., 1998; Weissman et al., 2006; Grigoriu-Serbanescu et al., 1991; Hammen et al. 1990; Radke-Yarrow et al., 1992; NICHD, 1999; for short introductions refer to the highly cited reviews of Downey and Coyne, 1990; Cummings and Davies, 1994; Goodman and Gotlib, 1999; and, Lovejoy et al., 2000). For example, a body of research concludes that children of depressed mothers are at heightened risk for internalizing and externalizing symptoms (see also Billings and Moos, 1983, Brennan et al., 2002; Murray, 1992; Zahn-Waxler et al., 1990), for psychological disorders in general (Lieb et al., 2002 Hammen, Burge, Burney and Adrian, 1990, Rutter and Quinton, 1984), for developing depression (Beardslee et al., 1998; Beardslee, Keller, Lavori, Staley and Sacks, 1993; Downey and Coyne, 1990, Orvaschel, 1990) or anxiety disorders (Weissman et al., 2006; Hammen and Shih, 2004), of developing conduct disorders (Conger, Patterson and Ge, 1995; Zahn-Waxler et al., 1990; Hammen, Burge and Stansbury, 1990; Harnish, Dodge and Valente, 1995), for impaired performance (Hay et al., 2001; Lyons-Ruth, Zoll, Connell and Grunebaum, 1986, Laucht et al., 2002), psychosocial impairments (Radke-Yarrow et al., 1992; Orvaschel, Weissman and Kidd, 1980), deficits in emotion regulation (Zahn-Waxler et al., 1984), attention problems (Dodge (1990), language and cognitive problems (Cogill et al., 1986; Murray, 1992; Sharpe et al., 1995; Murray et al., 1996; Hay et al., 2001 and Murray et al., 2003), and, finally, for developing impaired attachment relationships (Lyons-Ruth et al., 1986; Teti et al., 1995).

With respect to disorder prevalence rates in children Lavoie and Hodgins (1994) reported in their meta-analytic report that 61% of children of parents with a major depression are at risk for a psychiatric disorder of any type. In comparison with children of psychiatrically healthy parents a fourfold risk of developing an affective

disorder was reported (similarly, Weissman et al., 2006; found the risk tripled). Additionally, maternal unipolar depression characteristics such as severity or chronicity were found to predict increased risks for psychiatric disorders in children (Keller et al., 1986).

In accordance with these findings several authors (e.g. Beardslee et al., 1998) have recommended that a parental depression should alert practicing clinicians to take into account the possibility of heightened child risks and to include parental support in therapeutic actions.

This recommendation is accompanied by the fact that depression disorders are common and are considered a health problem of major public interest. The life-time prevalence in the general female population of 16 - 25% and the 12-month prevalence of 5 - 10% can be considered as high (Hasin et al., 2005; Kessler et al., 2003). A common feature of depression epidemiology is that depression rates amongst women are almost double compared to those of men (Weissman and Olfson, 1995) which is important given the fact that most parenting during the infant's first year is provided by women. Moreover, depressed individuals are at high risk of recurrence and chronicity which seem to be quite typical characteristics of depression (Angst et al., 1996). Accordingly, children of depressed mothers are considered as endangered by being frequently exposed to extended periods of parental depressive behaviors.

Furthermore, the WHO Global Burden disease report (Murray and Lopez, 1997) lists unipolar depression in developed countries as the leading cause of disability measured by the years lived with disability, compared to other psychiatric diseases, even non-psychiatric ones. In addition, according to Downey and Coyne (1990) this disability might have the potential to generalize to or be incorporated into parenting behaviors (e.g. a parenting style mainly characterized by flat affect). Moreover, the frequently reported low detection and treatment probability of depression disorders further supports the view that this constitutes an as-yet unresolved public problem (Ballestrem et al., 2005; Hasin et al., 2005; Kessler et al., 2003), a problem which - in the presence of effective mechanisms of risk transmission - might also affect children.

To date a major part of the research that was conducted on children of depressed

mothers - generally speaking the research in the field of parental mental illness - has been guided by the belief that early childhood exposures to parental behavior are predictive for later child behavior (e.g., Newport et al., 2002).

This notion may particularly apply to infants for whom maternal presence constitutes a large part of the environment (Baildam et al, 2000), and thus are particularly exposed to maternal depressive behaviors or disruptions in interpersonal relations, both of which have been suggested as longitudinally effective, e.g., with a subsequent later risk of psychopathology (Kendler et al, 2002; Johnston, 1996). Problematic interpersonal behaviors are part of the definition of many psychiatric disorders (Lyons-Ruth, 1995) and the lack of intimate relationships in depressed individuals, poor ability to adjust socially plus findings that depressed might actively induce rejections in their major interaction partner (Segrin and Abramson, 1994, Coyne, 1976b; and Coyne, 1976a) may affect a completely dependent major interaction partner, e.g. an infant.

Additionally, maternal depression has been linked to impairments in essential social roles, e.g., depressed mothers were reported to gain less satisfaction from mothering and were less warm and consistent, and felt less adequate in their role (Weissman, Paykel and Klerman, 1972; Bromet and Cornely, 1984). A substantial percentage of postpartum depressed mothers (29%) were reported to have impairments in their bonding with the infant (Brockington et al, 2001; 17% compared to 6% in healthy mothers, Reck et al. 2006). In accordance with reduced bonding, Campbell et al. (2004) reported that infants of depressed mothers frequently had an insecure or disorganized attachment to their mother, especially if the maternal depression was chronic and the parental behavior of the mother was rated as insensitive. This is well in line with the meta-analysis of Martins and Gaffin (2000) who concluded reduced secure and more avoidant and disorganized attachment in children of depressed mothers. Jennings et al. (1999) found that thoughts harming the infant are highly prevalent in mothers with depression (41% compared to only 7% in the population).

Regarding dyadic interaction detrimental effects of maternal depression on the way in which mother and infant interact are consistently reported, yet rigorous laboratory studies with a focus on major depression and its effects after remission are lacking. Detrimental effects of parental depression on offspring have been summarized using

terms such as “intergenerational transmission” or phrases such as “depression runs in families” (Hammen, 1991; 2009; Goodman and Gotlib, 1999; Rutter et al., 1999; Downey and Coyne, 1990).

The interactive or parenting behaviors of depressed mothers with their infants in either naturalistic or structured interactions have been characterized as affectively flat, disengaged, unresponsive, insensitive, ineffective, non contingent, negative and intrusive. They have been described as less synchronous and contingent (Campbell, Cohn and Meyers, 1995; Cohn and Tronick, 1983; Field, 1984; 1986; Lyons-Ruth et al., 1986; Pelaez-Nogueras et al., 1996; Stanley et al., 2004). Although these characteristics might reflect the symptomatic profile of depression, it might also imply consequences for a rapidly developing infant as a major interaction partner: For example, a number of publications in the last decade have suggested that dysfunctional interactive patterns might have a mediating potential on how a depression diagnosis unfolds its effects on adverse child outcomes, e.g., maladaptive parental behavior as mediator for child psychopathology (Johnson et al., 2001), neglectful or abusive parenting (Bifulco et al., 2002) or poor disciplinary practices as precursors of child adjustment problems (Conger, Patterson and Ge, 1995).

Thus, a parental diagnosis of depression has not only been found predictive for changes in dyadic interaction, but also predictive in terms of a mediating potential for adverse child effects.

A further stream of research suggests that the sole presence of maternal depression is predictive for adverse child outcomes. Conversely, the absence (i.e. after remission from maternal depression) was suggested to precede ameliorations in adverse child outcome (e.g., by the group of Weissman, refer to the review of Gunlicks and Weissman, 2008; or by Hammen and Brennan, 2003). Although a potential off-onset indicator (i.e. parental depression, present then child effects present or, vice versa, depression in remission, then child effects in amelioration) might be a promising indicator for behavioral transmission effects, the number of currently available studies has been regarded as insufficient (Gunlicks and Weissman, 2008). Beyond that, studies with mothers in major depression and after remission with infants below one year are completely lacking.

Regarding mechanisms it seems to be a consensus in the literature that

transmission-mechanisms from the depressed parent to the child are poorly understood. Moreover, the focus of research on potential pathways to child-risks tends to move away from a focus on genetic pathways, for example as an explanation for child risks of psychopathology. Genetic pathways have been harshly criticized for the inconsistency of effect estimators (Burbach and Bordin, 1986), for missing indicators of representativeness of samples (e.g., the studies reviewed by Sullivan et al, 2000) and for the non-specificity of child outcomes (e.g., maternal depression was found both predictive for both internalizing and externalizing child behavior). On the other hand, research into environmentally driven mediating factors is growing, driven by the concept of the potential adverse effects of child exposure, e.g. exposure to a maladaptive interpersonal milieu of the depressed parent, to altered affects, behaviors and cognitions of the depressed parent and to the associated dysfunctional parenting (e.g., Goodman and Gotlib, 1999; Rutter, 1990; Lovejoy et al., 2000; Gelfand and Teti, 1990).

The inclusion of infants into the present research is supported by the suggestions of Thapar and McGuffin (1994), Rutter and Sroufe (2000) and Scourfield et al. (2003), who suggest that genetic components might exert less influences if individuals are younger and that environmental factors (factors other than being exposed to maternal behaviors) may be less directly effective on the infant (e.g., factors such as family disruption or severe parental discord, Rutter and Quinton, 1984).

From a theoretical point of view, this research focuses on three major theoretical lines of why parental depression is predictive for adverse infant behaviors, why it might be predictive for changes in interactions and for impaired parenting.

First of all, the characteristics of the disorder which might be suitable to compromise or at least restrict parenting skills (Cummings and Davies, 1994; Downey and Coyne, 1990; Gelfand and Teti, 1990), e.g. by submitting a child under changed reinforcement conditions due to restricted maternal resources (theory of impaired parenting): Symptoms of depression, such as loss of interest and energy, flat affect and withdrawal might generalize to behaviors towards the child and thus alter reinforcement conditions for the infant. Flat affect is a common depression symptom, so the parent might be emotionally unavailable and thus less sensitive to the child's cues. As a consequence, infants of depressed mothers might withdraw from social interactions, and, develop deficient social skills; or, contrarily, use exaggerated

eliciting behaviors to overcome lowered parental responsiveness. If social interaction itself is assumed to be an effective transfer mechanism for an infant to gain knowledge (Winnicott, 1965, 1974, 1976; Vygotsky, 1978; Wertsch and Tulviste, 1992; see the summary table on pages 21 and 22) then early dysfunctional or low-level social interactions might predict later impairments in child performance.

Secondly, restricted maternal resources might compromise the modelling or shaping of infant behavior and affects. The idea that maternal affect in social interaction has influencing, or even controlling or regulating consequences has been formulated long ago (e.g., Stern, 1985). Contrariwise, if there is a markedly lowered level of maternal affect then infant regulation is predicted to fail. Gergely and Watson (1996; 1999) formulated a theory based on the idea that maternal affect-feedback modulates and shapes the infant's behavior. Quite similar regulation theories have been formulated by others such as Tronick and Gianino (1986), Field (1992, 1985; 1991), Beebe and Lachmann (1994) or Demos (1982). In detail, Gergely's theory predicts that affect mirroring - in the sense of multimodal parallelisms of affect - allows a mother to exert influences on, and to regulate the affects of her infant. Affect mirroring might allow for affect transmissions and the build up of representations on how to deal with affects. A depressed mother, however, due to flat affect, a reduced level of positive affect, and reduced responsiveness might mismanage infant affects and might fail in terms of the regulation of infant behavior and affects.

Affect regulation mechanisms are thought either to work by imitation (which has been demonstrated to occur in the first weeks of infancy by Meltzoff and Moore, 1989; 1983; 1997) or by affect contagion which has been extensively studied in adults (Joiner and Katz, 1999; Segrin and Dillard, 1992). Affect contagion as a transmission mechanism from mother to infant have been suggested by Field (1990, 1992).

On the other hand, the remission of depression has been theorized to be paralleled by a reduction of child disturbances (e.g., rates of behavior deviancies or psychopathology) under the term "transient child disturbance theory" (Gunlicks and Weissman, 2008; Downey and Coyne, 1990; Hammen et al., 1991): it predicts that dysfunctional interactions and child maladjustment might disappear when the maternal depression remits. Accordingly the present study hypothesizes that deviant mother-infant interactions disappear and affect mirroring normalizes after the remission of the maternal major depression. A sole focus on genetic transmission

approaches would not allow for the prediction of a temporal association of maternal depression and child deviancies.

All three theories imply the potential quality of early mother-infant interaction as pathway between maternal depression and adverse infant outcome which has long been theorized (Hossain et al., 1994, Rutter, 1990; Murray et al., 1996).

Based on the above mentioned theories a prospective, highly standardized, observer-blind, controlled, laboratory trial with repeated measurements was conducted: A total of 59 mothers and their infants, 24 with a clinical diagnosis of major depression, together with their infants, and 35 control dyads were videotaped during face-to-face interactions. Mothers were instructed to interact with the infant as they would normally do at home and were tested twice a) when the mother was in a major depressive episode (within a few days after admittance to the mother-infant ward of the Heidelberg University Clinic) and b) after the remission of the depression. Control mothers were retested after a comparable time period. Diagnoses were made strictly according to the criteria for major depression in the Diagnostic and Statistical Manual of Mental Disorder, Fourth Edition. Affect-related behaviors were coded during a highly standardized situation, the still-face procedure. This procedure consists of two free interaction phases interrupted by a phase of maternal still-face.

The present study - as part of an ongoing longitudinal study sponsored by the German ministry for education and research - has the aim of replicating and extending previous findings. With this study, we aim to overcome several shortcomings of the available studies (by gathering observational data instead of ratings, including individuals with a clinical diagnosis of maternal major depression instead of self-rated depressed mood, making observations of dyadic interaction during an episode of maternal major depression instead of including mothers with a history of depression, using a highly standardized laboratory setting instead of free home observations, including time patterns, e.g., reciprocal and bidirectional behaviors instead of using global measures, employing assumption-free, nonparametric univariate and multivariate statistical methods instead of high assumption-needy procedures, and, finally applying adjustments for decision errors due to multiple testing, a commonly neglected problem in this area of research).

2. Theoretical background

2.1. Depressive disorders

Depression is a common and highly prevalent mood disorder. It exceeds normal variations in affect by its duration, intensity, accompanying symptoms, and is usually accompanied by extensive functional (social) impairments.

2.1.1 Operationalization

Operationalized criteria for psychiatric diagnoses are state-of-the-art in modern clinical psychiatric and psychological research and an approach to solve the problem of unreliability of diagnoses (Spitzer, Endicott and Robins, 1978). The most important diagnostic classification systems are the ICD-10, the International Classification of Diseases (Dilling et al., 1994), and - but more relevant for research purposes - the DSM-IV, the Diagnostic and Statistical Manual of Mental Disorders (Conger, 1980). Both systems subsume depressive disorders under Mood Disorders. Other disorders in this category that are not referenced in this research include dysthymic, bipolar and cyclothymic disorders as well as mood disorders due to either a general medical condition or substance-induced.

A patient is classified as having a major depressive episode when a total of 5 or more of the 9 DSM-IV symptoms have been present during the last four weeks for a period of at least 2 weeks in duration. At least one of the symptoms “depressed mood” or “anhedonia” are required and both symptoms are indicated by subjective report or observation made by others. The presence of both symptoms is highly sensitive, having a sensitivity of 96% for detecting major depression when the DSM criteria are used (the “two-question test”, Whooley and Simon, 2000; NHS, 2002).

In order to diagnose a major depressive episode the so called clinical significance criterion has to be fulfilled, which means that those 5 or more symptoms have to cause clinically significant distress or impairment in social, occupational, or other important areas of functioning (historically the clinical significance criterion was added to reduce the rate of false-positive diagnoses, Spitzer, Endicott and Robins, 1978).

Further symptoms include a more than 5% change in weight in a month, sleep disturbance, psychomotor agitation or retardation that is observable by others, fatigue or loss of energy nearly every day, feelings of worthlessness or excessive or

inappropriate guilt, which might be delusional, impaired concentration or decision-making, and recurrent thoughts of death or suicidal ideation (Moses-Kolko and Roth, 2004).

The frequency and number of symptoms define the intensity of the disorder. The criteria of a major depression approximately equal those of the ICD-10, with the exception that the DSM-IV requires only one major symptom (depressed mood or anhedonia), whereas the ICD-10 requires both to occur at the same time.

However, the symptoms must not meet the criteria for a mixed episode and must not result from the effects of a substance (e.g., drugs or medication) or a general medical condition (e.g., hypothyroidism). Bereavement, such as the loss of a loved one, has to be excluded.

The DSM-IV and its text revision use “postpartum onset” as a modifier for mood disorders (Seyfried and Marcus, 2003). A postpartum onset refers to an episode of depression within 4 weeks after delivery.

With respect to pregnant or postpartum women there is a certain risk that depressive symptoms might be misleading since they might possibly overlap with general changes in or following pregnancy, for example disturbances in appetite and sleep, the loss of energy - including sexual energy, and increased somatic concerns. Thus, indicators that are associated with a pregnancy might increase the risk of a false-positive diagnosis and have to be controlled for (Coverdale et al., 1996).

The DSM-IV is strictly descriptive and avoids etiological assumptions for its categories. The PHQ - an abridged form of the DSM-IV - might be a preferred alternative since its accuracy was found to be sufficient (98% and a specificity of 80%, Loewe et al., 2004) compared with other screening tools, such as the Hospital Anxiety and Depression Scale (HADS) and the WHO Well Being Index (WBI-5).

2.1.2 Delimitations

Mothers with “postpartum blues” or “postpartum psychosis” do not fulfill the inclusion criteria of this study. “Blues” refers to certain symptoms of mood lability ranging from euphoria to tearfulness. They are experienced by 50-85% of new mothers following delivery and were found to end about two weeks after delivery (Nonacs and Cohen, 1998; Pitt, 1968). Postpartum blues currently is considered a benign condition, but

was reported to be predictive for an onset of depression (Reck et al., 2008). “Postpartum psychosis” refers to a rare form of bipolar disorder. It affects one women per 1000 and symptoms of rapidly changing mood and erratic behavior can begin as early as 48-72 hours after delivery. Clinical manifestations include psychotic thoughts, severe depression and mania (Brockington, 2004). This disorder is considered a psychiatric emergency and requires inpatient treatment (Nonacs and Cohen, 1998).

2.1.3 Epidemiology of depression

Depression is generally considered as a common disorder. Kessler et al. (1994) found a 12-month prevalence of 12.9% for women and a lifetime prevalence of 21.3%. Jacobi et al. (2004) report a DSM-IV major depression lifetime prevalence of 17.5% and a 12-month rate of 11.2%. Hasin et al. (2005), in a US American nationwide survey of over 40000 participants on the epidemiology of major depression according to DSM-IV, found a 12-month prevalence of 5.3% and a lifetime prevalence of 13.2% with a precision (confidence ranges) of well below 1%. Corresponding rates were found by Bijl et al. (1998) with a rounded lifetime prevalence for affective disorders of 19% and of 15% for major depression, with a rounded 12-month prevalence of 6 % and a rounded 1-month prevalence of 3%. Usually the female depression prevalence rates are twice as high as those of men.

2.1.4 Epidemiology of postpartum depression

As of 2009, two quantitative meta-analyses with prevalence estimates of postpartum depression were available, one published by O'Hara and Swain (1996), the other by Gaynes et al. (2005). They are based on different methodologies and have quite different qualities.

O'Hara and Swain (1996) summarized 59 studies, mixing diagnoses and mood self-ratings, i.e. half of these studies were based on interview measures, the other half on self-report measures of depression. They found a DSM-III-R major depression postpartum prevalence of 7.2% (page 40, with 95% confidence from 3.7 to 10.7%), based on 3 studies and only 208 subjects. 19 studies based on Spitzer's Research Diagnostic Criteria resulted in a rate that was not substantially different, i.e. of 10.5% (9.7 – 11.3%). The 3-month incidence rates after delivery were estimated as 6.5% for major depression. The authors estimated an overall average postpartum depression prevalence of 13%; however, this based on a study sample with high variance. The

highest depression rates (>16%) were found with self-report measures, such as the Goldberg or Pitt criteria or the CES-D. Rates of around 11% were found in studies with Beck's Depression Inventory or the Edinburgh Scale for postpartum depression. Although the authors conclude that depression definitions based on interview (compared to self-report) correspond with lower prevalence rates, their meta-analysis has severe methodological flaws.

First, standard meta-analytic methods according to classical textbooks have not been applied, e.g., Hedges and Olkin (1985), Rosenthal (1991) or Sutton et al. (2000). The calculation of an average rate can be regarded as statistical misconception, since the total patient count summed up across all the studies was used as unit of observation, instead of the study count. This increases the data basis from 59 (studies) to 12810 (study participants) which artificially overestimates precision (i.e. the confidence band) and exaggerates the weight of those studies that report a high prevalence based on self-report measures only, e.g. based on the Beck Depression Inventory, the CES-D and the Edinburgh Scale. There are only 3 studies based on DSM criteria. Other studies based on DSM-IV criteria (e.g., Kurstjens and Wolke, 2001; in 1329 mothers) reported a considerably lower prevalence rate of 7%, or 9% (802 women, Yonkers et al., 2001). An Australian research group (Matthey et al., 2003) found a 6 week postpartum DSM-IV depression rate of 6% in 408 mothers. Garcia-Esteve et al. (2003) found a postpartum major depression rate of 3% in 1201 women in Spain, similar to the 3%-rate of major depression observed by our study group (Reck et al., 2008; table 2).

Second, the prevalence estimate of O'Hara and Swain (1996) did not rely on a specific meta-analytic model (fixed or random effect) and no measures for publication bias were included (e.g., based on the concept that small studies display higher variances compared to studies with a high number of participants). Taken together these factors amount to a violation of major criteria in O'Hara and Swain's (1996) meta-analysis (the unit of observation, the estimation method, the calculation of precision, and a missing test for publication bias). Thus, the reported overall rates of postpartum-depression of 13% should be interpreted with caution.

The meta-analysis of Gaynes et al. (2005) included 30 studies published between 1980 and 2004. Contrary to the approach of O'Hara and Swain (1996) the meta-analytic model is explicitly given (random effects and the inverse variance-weighted

method). Combined rates for major depression in the 1st postpartum year from 1 to 6% (rounded) are given. Major plus minor depression rates were twofold and ranged between 7 and 13% as highest value (3rd month after delivery), and the rates of childbearing women were not different from those of non childbearing women.

Taken together, the statistical and methodological quality of the meta-analysis of Gaynes et al. (2005) can be regarded as superior compared to the meta-analysis of O'Hara and Swain (1996). Thus, the prevalence of the postpartum major depression ranges in the lower range of the percentage scale (1 - 6%).

2.1.5 Course of depressive disorders (in general)

The course of affective disorders is characterized by episodes, a high risk of recurrence, and a remarkable risk of chronification. The course is usually characterized as inter-individually heterogeneous (Angst, 1997) as well as heterogeneous in terms of estimates from meta-analyses (Piccinelli and Wilkinson, 1994). Consequentially, prognoses for a patient have a high degree of uncertainty. Heterogeneity might be due either to the chosen methods or the underlying characteristics. Uncontrolled, single group longitudinal studies, conducted in naturalistic settings appear to be the preferred method, e.g., for the investigation of course descriptors of depression (remission, recovery, relapse or recurrence, Frank et al., 1991). However, naturalistic study-settings usually have a heightened risk of uncontrollable covariates that might considerably contribute to heterogeneity of estimates. Moreover, single-group designs put any interpretation under risk: the characteristic of interest, the depression course, is accompanied by many other changes, e.g., the on- and offset of treatments or social changes. Additionally, the longitudinal studies reported below are predominantly based on initially hospitalized and heterogeneously treated patients (Angst, 1986), and an inclusion of treatment variables into the statistical analysis is lacking. Further sources of variance are the use of different outcome criteria, e.g., different definitions of course descriptors (definition of "episode", or, "remission") or the use of non-standard versus standard course descriptors, which complicates a comparison of different studies for depression course (Boland and Keller, 2005).

Thus, results obtained in naturalistic studies have to be viewed with caution: It is unknown to what degree the course reflects an either true course of the underlying

characteristic or mixed characteristics of the chosen sample, the variations in treatment and its efficacy or other environmental conditions.

Nevertheless, longitudinal naturalistic descriptions of a depression course have been seen as advantageous, changing the understanding of depression (Boland and Keller, 2005) from an disease viewed as episodic to a disease viewed as lifelong. Piccinelli and Wilkinson (1994) included in their meta-analysis 51 naturalistic studies that were based on standardized diagnostic criteria. They concluded that the one-year full recovery rate was 64% and the one-year recurrence rate 26%. They reported a two to five year recurrence rate of about 50%, which extended to 76% within 10 years. A heightened likelihood of recurrence was found after 4 - 6 months after the initial recovery (NIMH, 1985). Thus, for the majority of major depressed patients "recurrence after recovery" seems to be the rule (Mueller et al., 1999). A German WHO study (Kuehn et al., 2002) found a similar recurrence rate of 33% after one year. Keller et al. followed patients with an episode of unipolar major depression for a total of 18 years and found a 70% rate of recovery from the first index episode after one year (Boland and Keller, 2005; Katz and Klerman, 1979). A failure to recover in the first year seemed to increase the risk of non-recovery: 20% had still not recovered after 2 years (Keller, Shapiro, Lavori and Wolfe, 1982) and 12% after 5 years (Keller et al., 1992). Mueller et al. (1996) report a recovery failure of 7% after 10 years and of 6% after 15 years.

Solomon et al., (1997) reported that episodes have a median duration of about 20 weeks, even for subsequent episodes. This seems well comparable to Angst (1986) who reported a 23-week duration. The time until recovery was reported to be relatively stable (Solomon et al., 1997), with an increasing number of episodes, but the rate of recovery declined over episodes. A full recovery was followed by an average latency of 180 weeks until recurrence. Patients with residual symptoms had an 87% recurrence rate and a shorter recurrence latency of 33 weeks. Moreover, the number of previous episodes allowed the prediction of a risk for chronification; after the second episode the risk of recurrence is 75%, after the third episode over 90% (NIMH, 1985; Keller et al., 1992). Thus, the risk of having a recurrence increases with each successive recurrence (e.g., by 16%, Solomon et al., 2000).

10 - 25% of major depressed patients are expected to experience chronification defined as non-remitting episodes for at least two years (Angst, 1997; Keller, 1994;

Kuehn et al., 2002). A longer index episode seemed to be predictive for a risk of developing a chronic course (Ruppe, Keller and Wolfersdorf, 1996). Taken together, major depression is highly recurrent with a high risk of chronification (Klerman and Weissman, 1992).

Persisting dysthymic symptoms after a patient has recovered from major depression and the presence of a non-psychiatric disorder, such as alcoholism, drug dependence, or anxiety, and even old-age were discussed as factors favoring an increased risk of relapse or recurrence (NIMH, 1985). Late depression onset (after the age of 50) approximately doubled the risk for chronicity (18% vs. 8% in case of an early onset, Angst, Kupfer and Rosenbaum, 1996).

Other risk predictive factors include previous episodes, a longer duration of depression before intake, the persistence of subsyndromal symptoms (threefold shorter time to recurrence, Mueller et al., 1999), a higher intensity of the index-episode and a history of non-affective psychiatric disorder (Keller, 1994).

Suicide poses a special problem in affective disorders. In deviation from the rate of 15% which is generally quoted (Hautzinger, 1997; Hawton, 1992; Chen, Lan, Yang and Juang, 2005) Inskip et al. (1998) found in their meta analysis a lower lifetime suicide risk (6%) in patients who were clinically diagnosed with depression. Sokero et al. (2005) found a risk of 8% in an 18-months follow-up. Outpatients seem to be at lower risk (Simon and VonKorff, 1998). However, suicide attempts, e.g. an another attempt or a suicide completion have been found to be predictive for an unfavorable course of depression (Oquendo et al., 2002).

Further risk increasing factors are: being female (double risk compared to men, Angst and Merikangas, 1997), being of lower age (Bland, Newman and Orn, 1997; Barth, 2005; Wittchen, Knäuper and Kessler, 1994), low socioeconomic status (e.g., the meta-analysis of Lorant et al., 2003) and low educational level (Bijl et al., 2002), having a family history of mental illness, suffering from major life changes, negative life events or stress (daily hassles or difficult relationship), having had adverse early childhood experiences (parental divorce, trauma, loss of parent), having never been married, being divorced or separated, being unemployed for more than 6 months, low self esteem, heightened perfectionism and sensitivity to loss and rejection and insomnia or chronic sleep problems. Further risk-increasing factors include lack of

social support, critical life events (Reck et al., 1999), problems in partnership (Backenstrass, 1998), interpersonal problems (Mundt et al., 1996) and personality factors (Mundt et al., 1997).

Moreover, psychopathological factors are of high importance (Kessler et al., 1994; Wittchen et al., 1994).

Highly predictive, however, are single depression symptoms. Horwath, Johnson, Klerman and Weissman, 1992; and Eaton, Badawi and Melton (1995) investigated precursor signs for the one-year incidence of major depression in subjects not meeting the criteria of a major depression. 50% of first onsets had previous depressive symptoms, and specific depressive symptoms were precursor signs, such as 2 or more weeks of sad mood (7-fold increased risk ratio), sleep (7.6-fold) or concentration problems (6.1-fold), thoughts of death or wishes for death (6.8-fold) or weight changes (3-fold increased risk ratio). The most predictive precursor sign for major depression, however, were feelings of worthlessness or guilt, which increased the onset risk of a major depression ten-fold.

Comparable results were found in the case of incomplete recovery from major depression (Judd et al., 2000). Patients having residual symptoms had faster relapses to major and minor episodes and a higher recurrence frequency. Consequently, periods of well-being were shorter compared to patients who were symptom-free.

2.1.6 Course of depressive disorders (with application to the mother-infant dyad)

Several aspects of the course of maternal depressive disorders have been suggested to be associated with child development, e.g., concurrent maternal depressive symptoms in the presence of child psychopathology or, conversely, reductions in child behavior problem rates in the case of a remission of maternal depression. It was suggested that depression severity, timing and chronicity are more closely associated with impairment of functioning in children than the diagnosis itself (Keller et al., 1986). The proximity of maternal depression that is concurrent with child behavioral problems and the parallelism of the offset of both are thought to be better explained by interpersonal and reciprocal mechanisms rather than a biological or genetic substrate.

For example, Hammen, Burge and Adrian (1991) found episodes of unipolar maternal depression and child depression in temporal proximity, i.e. either overlapping or one month after maternal remission. Only a few onsets of child depression were reported to be unrelated to maternal episodes. Thus, according to Hammen maternal depression, possibly as stressor for the child due to unavailability and lack of support, may parallel or antecede child depression.

Brennan et al. (2000) reported that both depression chronicity and severity were equally predictive for heightened problems in child behavior and lower language reception. The time pattern of maternal depression symptoms, however, did not predict lowered child language performance, yet heightened rates of problematic child behavior appeared to be paralleled by maternal depression episodes.

In contradiction to the results of their 1991 study, Hammen and Brennan (2003) found depression severity better predictive for risk of depression in offspring than depression chronicity.

Several reports found that both depression severity and depression chronicity are better predictive for adverse outcomes in children than the depression diagnosis itself (e.g., Keller et al., 1986).

Chronicity and timing of maternal depression might be regarded as critical since they might impair development e.g., of attachment, regulation of infant-emotion, or social competence (reviewed by Goodman and Gotlib, 1999). However, usually depression chronicity and severity are often confounded, since a severe depression is usually associated with a longer duration.

2.2. Theoretical background - models explaining associations between maternal depression and interpersonal behavior of mother and infant

2.3. General remarks

A range of mechanisms or prediction models have been proposed with respect to the question of why a maternal diagnosis of depression might indicate or precede adverse child outcomes, measured in terms of parent-child interaction, of child performance, or incidence of child psychopathology.

The models range from approaches that implicitly assume a transfer of “hardware” from parent to infant (e.g., genetic transmission models, Sullivan, Neale and Kendler,

2000; or in the sense of a transfer of insufficient serotonergic transporters and heightened sensitivity to life events, Kendler et al., 2005) to models that imply dysfunctions in maternal neuroregulation during pregnancy (e.g. the review of Goodman and Gotlib, 1999), deviations in family interactions (Fendrich, Warner and Weissman, 1990; Keitner and Miller, 1990; Katz, 1999), environmental or social interactions (Field, 1992; Murray and Cooper, 1997) and, finally, observational infant-learning in presence of depressive maternal symptoms and impaired parenting (Downey and Coyne, 1990) characterized by flat affect or loss of energy. Parenting practices of depressed mothers might also be impaired, e.g. include the use of coercive techniques and might increase the incidence of conduct and behavior problems (Downey and Coyne, 1990). The depression timing, chronicity, and severity, parental availability and functioning of other family members, infant gender, and infant temperament have been suggested as factors that moderate the predictive value of parental depression (Goodman and Gotlib, 1999; Gunlicks and Weissman, 2008).

Moreover, the linkage between maternal depression and adverse child outcomes might not be unidirectional in the sense that maternal depression antecedes child problems. Maladaptive child behaviors themselves might contribute to maternal depression (Johnson et al., 2001). It is also possible that a third factor causally relates both to maternal depression and child problems (e.g. marital discord, substance abuse or personality disorders, and Rutter, 1990).

2.4. Genetic pathways as a major explanation of adverse effects on children of depressed mothers - a general critique

Intrafamilial transmission of depression symptoms or psychopathology through genetic pathways has long been considered as one of the most important factors on risks for the offspring (Kendler et al., 1997; Plomin, 1990). Today, it seems to be consensus in the literature that depressive disorders in offspring are both hereditary and environmentally driven (McGuffin et al., 1996; Kendler and Prescott, 1999; Sullivan, Neale and Kendler, 2000). Classical intrafamilial aggregation studies either predicted the incidence of depressive disorders in children of depressed parents (Weissman et al., 2006), or incidence rates depending on variations of genetic similarity, e.g., dicygotic or monozygotic twins (Kendler and Prescott, 1999; Thapar and McGuffin, 1994; 1996). For example, in a large Swedish twin series, Kendler et

al. (2006), found a depression heritability of 29% in males and 42% in females. The absolute magnitude of heritability, in particular high rates of resilient, i.e. completely unaffected children (Rutter, 1990), is still the subject of intense discussions: On one side Sullivan, Neale and Kendler (2000) conclude in their review that the intrafamilial aggregation of depression “mostly or entirely” (page 1552) results from genetic influences and that findings might be considered as consistent over samples and methods with negligible contribution of environmental effects. Taking the opposing view, Rice, Harold and Thapar (2002) doubt these presumed effects and criticize the empirical evidence for the extraordinary heterogeneity of heritability estimates in twin and adoption studies. Estimates of heritability (e.g. for major depression) were found to range between 0 and 100% (Sullivan, Neale and Kendler, 2000; figure 1 on page 1555). Consequently, Rice et al. deny that intrafamilial aggregation studies allow for the conclusion that depressive child symptoms or other risks in association with parental depression might be based on a genetic aetiology. Technically, genetic research designs are in principle “correlational” (i.e. based on concordance measures in various ways; e.g., Weissman et al., 2006; Kendler et al., 2006; Thapar and McGuffin, 1996). Moreover, there is a high degree of unspecificity: children of depressed parents are not only reported to have a heightened risk for depression but are also at risk for any type of psychopathology such as anxiety or disruptive disorders, or personality or substance abuse disorders (Johnson et al., 2001; Beardslee et al., 1998).

Finally, there are considerable methodological problems with the samples commonly used in twin studies; the sample size of large twin studies alone does not guarantee representativeness and representativeness of samples is rarely shown: This might be considered as important, since twin prevalence is particularly low. Twins are present in about 1 in 40 births (dizygotic) or 1 in 250 births (monozygotic, Fraga, Ballestar and Paz, 2005). If these rates are combined with the current prevalence rates for major depression (DSM-IV, i.e. with a prevalence rate of 3 to 5%), this makes the prevalence of depression in twins particularly low ($1/40 \times 0.05 \times 100\% = 0.125\%$ for dizygotic and $1/250 \times 0.05 \times 100\% = 0.020\%$ for monozygotic twins). Those rates approach a zero rate and indicate a very limited population whose representativeness for a general genetic liability of depression in the whole population might be questioned. Moreover, measures for twin concordances are

usually based on the assumption of “equal environments”, i.e. that both twins are comparably exposed to environmental events. This, however, can only be quantified retrospectively (and thus is prone for error). Also non-differing and selective environmental variables do not prove the equal environment assumption, since the absence of between-twin differences may result from error variance, from insensitive indicators or from the wrong statistical test (equivalence should be shown with “equivalence tests”, e.g. Wellek, 2002; which none of studies of the meta-analysis of Sullivan, Neale and Kendler, 2000, did). Twins can also be considered a population that is frequently included in research activities as the increasing establishment of twin register suggest and any interview with twins is clearly based on the implicit assumption to find concordances, since interviewed twins may not be blind to research hypotheses (and this may increase concordance rates). But rarely, if at all, specific hypotheses and corresponding tests for biasing factors were included or are at least discussed.

Taken together, the over reliance on the magnitude of genetic transmission mechanisms can easily be questioned due to heterogeneity (as in the meta analysis of Sullivan et al., 2000), as well as the unspecificity of child outcomes and due to the research methodology.

Despite the intense discussion about genetic pathways (Kendler and Baker, 2007; Kendler, 1996; Rice, Harold and Thapar, 2002; Thapar and McGuffin, 1996), there are workgroups that suggest that concurrent maternal depressive behavior is better predictive for aversive behavior in children than depression symptoms (Hammen, Burge and Adrian, 1991; Hammen and Brennan, 2003) and that depression remissions are followed by reductions in frequencies of the children's diagnoses as well as depressive, internalizing, and externalizing symptoms (Weissman et al., 2006). Animal studies (Francis et al., 2002) have suggested the effectiveness of cross-fostering methods or adoption methods, i.e. offspring educated by non-genetic, unrelated parents and thus non-genomic pathways. Fearful and stress responses of the hypothalamic-pituitary adrenal axis were higher in offspring “adopted” by low-caring parents, compared to offspring “adopted” by high caring parents. However, offspring of low-caring parents that were adopted by high caring new parents did not differ from normal offspring. Francis et al. concluded that stress reactivity and fear of novelty can be transmitted from one generation to the next via parenting behavior,

i.e. via a non-genomic mechanism of inheritance. Similarly, Cohn et al. (1990) argued that maternal depression has a negative impact on early mothering behavior which would be expected from clinical descriptions of depressive symptoms. Flat affect and social withdrawal or irritability characterizes the interactions of most mothers in these studies. Moreover, the infants of these negative and withdrawn mothers are likewise atypical, showing only limited engagement with the environment, clearly suggesting the influence of negative maternal affect on infant behavior.

2.5. Models for behavior-related transmission mechanisms

A range of behavioral models is currently available regarding the predictive value of maternal depression. These models can be classified as follows a) if they assume transmissions over behavior (all models below in table 1 and table 3, pages 21 and 22), and b) with respect to the direction of effect, e.g. if they assume that adverse effects on infants are preceded by maternal behavior or if effects derive from mutual behaviors, i.e. models focusing on interactive exchanges.

The present research focuses on three major prediction models. First, Lovejoy et al. (2000; page 562) and Downey and Coyne (1990; page 61) who suggested a **theory of impaired parenting** (table 1 below) as explanation for the link between the diagnosis of maternal depression and changes in interactional behavior or as precedent of child problems. The theory suggests that the exposure to restricted parenting resources, e.g., the loss of energy and flat effect, may change reinforcement conditions for the infant, e.g. maternal responsiveness, emotional rewards and overall level of (affective) stimulation (for details see page 25).

Secondly, in accordance with depression-associated changes in affect, Gergely and Watson (1996) suggested, based on their theory of affect mirroring as social biofeedback, that flat affect in depressed mothers leads to failures to use this affect to regulate (= control) infant affects (**theory of distorted affect mirroring**). Failures in infant regulation may be suitable to precede adverse infant outcomes, e.g., deviant externalizing or internalizing behavior. Gergely and Watson's theory predicts that a depressed mother may show deficits in affect mirroring and thus fail in the task of infant regulation (details on page 28).

Correspondingly, based on the remission of maternal depression a reduction of infant deviancies have been predicted (**transient child disturbance theory**). The theory

predicts that child maladjustment and dysfunctional interactions may disappear when the maternal depression remits (Gunlicks and Weissman, 2008; Downey and Coyne, 1990), i.e. may be regarded as a reversal of the impaired parenting theory mentioned above. Accordingly, the present study expects deviant mother-infant interactions to disappear and affect mirroring to normalize after the remission of the maternal major depression.

Table 1: Theories of intergenerational transmission effects of maternal depression to the infant (genetic pathways excluded); the models are explained in detail in the next chapter

Theory	Authors	Basic idea	Implied mediating mechanisms
theory of impaired parenting , symptom based approach, exposure model, e.g. working by instrumental or observational learning: missing response-contingent positive reinforcement	Lovejoy, Graczyk, O'Hare and Neuman, 2000; Lewinsohn, 1969; 1974	the exposure to a depressed caregiver's behaviors poses the infant under risk, e.g. depressed or withdrawn infant behavior may be anteceded by missing response-contingent positive reinforcement	effects of the symptom profile of maternal depression, i.e. child under-stimulation, affect negativity, social withdrawal
theory of distorted mirroring , affect mirroring as social biofeedback fails in presence of maternal depression	Gergely (Gergely and Watson, 1996; 1999)	multimodal mirroring of infant affects may allow a mother to regulate (intensify or de-escalate) affects of her infant	deviant or failing mirroring (missing biofeedback), may lead to dysfunctional regulation capacities of an infant, to distorted representations of inner states and emotion expressions

An extensive range of alternative transmission models are given below. Most of these relate more or less directly to the models above (e.g., Meltzoff's model of mirroring) and will be used for explorative model tests in later chapters. For example, mood contagion models (Coyne, 1985; 1976; details in table 2 below) allow for the prediction that depression may be witnessed as aversive by a major interaction partner (e.g. the infant), leading to negative mood, rejection and withdrawal, and - in both interactants - to a spiral of negativity. A alternative model of Patterson (1982) focuses on coercive behaviors of depressed parents that is due to unsuccessful interactions which may increase the risk of child negative behaviors. Moreover, Hammen's interpersonal stress model (1991; 2004) allows for the prediction that dysfunctional interpersonal behavior of a depressed parent creates stressful life events and thus may increase the risk of child depression. Further models

conceptualize the role of mirroring of affects (see above) in achieving infant regulation or mutual regulation (e.g., Stern, 1985; Beebe and Lachmann, 1994; Tronick and Gianino, 1986; Demos, 1982) or the role of dyadic interaction as knowledge transfer, e.g., with respect to the acquisition of social behaviors (e.g. Vygotsky, 1978; Bruner, 1974; Winnicott, 1965). A final class of models conceptualizes the role of maternal emotional availability as guidance for the child (e.g., Emde, 1980; 2000), or as emotional cue (Feinman and Lewis, 1983). Both models are based on the assumption that depression symptoms such as flat affect and emotional unavailability may constitute a deficient social rewarding system and thus may promote infant maladjustment.

Table 2: Further theories of intergenerational transmission effects of maternal depression to the infant (genetic pathways excluded); table continued on next page; note that models may overlap; the models are explained in detail in the subsequent chapters

Theory	Authors	Basic idea	Implied mechanism of how maternal depression may unfold effects on an infant
mood contagion approach , aversive effects of depression on a major interaction partner	Coyne, 1985; 1976; Coyne and Gotlib, 1983; Downey and Coyne, 1990; Segrin and Abramson, 1994; or the meta analysis of Joiner and Katz, 1999	interaction with a depressed partner may lead to negative mood and thus provokes rejection, e.g., hostility, depression, anxiety	depression may be witnessed as aversive, leading to negative mood, rejection and a withdrawn infant
mirroring and modeling concepts	Meltzoff et al. (1988; 1989; 1992; 1994; 1997); Bandura (1977; 1985), Field et al, 1983	infants imitate by observing caregiver behaviors; behaviors may be stimulated, facilitated or inhibited, very early imitation as intentional reproduction of a model influences the acquisition of social skills and the expression of emotions	infants might use the mother as major model, the interactional style of a depressed mother (reductions and negativism) may be mirrored, i.e. low motor activity levels, withdrawn behavior, unresponsiveness, low levels of maternal involvement and warmth

Theory	Authors	Basic idea	Implied mechanism of how maternal depression may unfold effects on an infant
model of coercive processes , Patterson's theory of coercive family processes	Patterson, 1982	unsuccessful interactions with difficult children causes mothers to react either with withdrawal or hostility, which itself may increase the risk of child negative behavior	depressive behavior has been viewed as one form of coercion: depressed individuals control aversive or aggressive behavior of others by emitting dysphoric expressions
Hammen 's intergenerational interpersonal stress model of depression	Hammen, 1991; Hammen and Shih, 2004; Hammen and Brennan, 2002; Hammen and Shih, 2004; Hammen and Brennan, 2001; Hammen, Shih, Altman and Brennan, 2003; Hammen, 2003	dysfunctional interpersonal behavior is a key mediator in how maternal depression unfolds effects on the child	maternal depression is associated with dysfunctional interpersonal behavior and increases child maladaptation, which itself generates stressful interpersonal life events and increases depression
affective attunement models (synonymously: affective mirroring or empathic responsiveness)	Stern (1985)	sharing of subjective experiences with the aim to expose the infant to new stimuli, attunement in dyadic interaction may connect infant's inner states with experiences of the outer world	a depressed caregiver's inability to read and mirror infant states might predict interaction deficits, skill deficits in infants and unregulated infant states
regulation model I , mutual regulation theories, theories of disruptions in mutual engagement (Tronick's model of mutual regulation, Field's model of attunement)	Tronick and Gianino (1986), Field (1992, 1985; 1991)	maternal unresponsiveness and failure to read infant cues leads to mismatching of positive affects, thus to poorly coordinated interactions	a depressed mother fails to give her infant regulatory help, positive interactions do not occur, the infant may perceive interactions as non-rewarding, self-directed behavior may dominate
regulation model II , bidirectional regulation via dyadic interaction	Beebe and Lachmann (1994)	mother-infant interactions are "co-constructed", i.e. both interactants use self- and interactive regulation to structure interaction, disruptions are mutually regulated by both interactants	depression associated disinterest and neglect of social interaction, lack of affect mirroring, and ongoing disruptions in interactions may impair infant skills (e.g., pre-symbolic competencies or interactive regulation skills)

Theory	Authors	Basic idea	Implied mechanism of how maternal depression may unfold effects on an infant
regulation model III , affective parental components form or modify infant affects	Demos (1982)	the affective component in mother-infant interaction is important in child development and in the modification of pre-existing child affects	lack of parental affect may lead to unformed and thus unorganized child behaviors
social interaction as experience mediator for the child, interaction as scaffolding or help to structure the infant's environment	Vygotsky (1978; Wertsch and Tulviste, 1992)	social interactions mediate infant development, e.g., based on the idea that mental processes have social origins	a non-responsive, withdrawn caregiver may fail as "skill mediator"; scaffolding behavior, interactive support in social mastery processes may be missing and may result in developmental impairments in child functioning and social skills
social interaction as sensitizer , social interaction as pre-speech communication	Bruner (1974; 1976)	interactions sensitize the infant to communicate states and needs, non-dysfunctional early interactions predict language development	a depressed, e.g., withdrawn and unresponsive caregiver, may delay speech acquisition; force an infant to use communication alternatives (e.g., excessive crying or hostile behaviors)
dyadic interaction as facilitating environment	Winnicott (1965)	interactions are essential for infant development (mental representations, self concept)	disruptions in interaction or dysfunctional interaction lowers the effect of a facilitating environment, and delay mental representations and a concept of the self
emotional availability as reciprocal reward system for infant and parent	Emde (1980; 2000)	early mother-child interactions may create internal object relations or working models which may guide later child behavior	depression associated symptoms such as flat affect and emotional availability and thus missing social rewards may impair infant development and promote infant maladaptation
social referencing as behavioral transmission mechanism	Feinman and Lewis, 1983; 1992, Sorce, Emde, Campos and Klinnert, 1985;	maternal emotional cues may effectively regulate infant behavior, e.g., prototypical facial expressions	behavior regulation may fail, the infant may adopt the depressed interaction partner's state (e.g. withdrawal, non-responsiveness) via mood contagion mechanisms

Theory	Authors	Basic idea	Implied mechanism of how maternal depression may unfold effects on an infant
multifactor model of Goodman and Gotlib	Goodman and Gotlib, 1999	explains adverse child outcomes, i.e. the transmission of risks to children of depressed mothers	(a) heritability of depression, (b) innate dysfunctional neuroregulation, (c) negative cognitions, behaviors, and affect of a depressed mother; (d) the stressful environmental context

2.6. Unidirectional models in detail: Behavior-related pathways of transmission - models with a focus on an exposure related risk

2.6.1 The impaired parenting or exposure model (symptom based approach)

Adverse effects of maternal depression on offspring have been predicted based on the model of impaired parenting (Lovejoy, Graczyk, O'Hare and Neuman, 2000). These impairments are thought to result from restricted maternal resources. Models of this type usually suggest that depressed mother is the infant's dominating environment (Murray and Cooper, 1997) and that the symptom profile of maternal depression may be experienced by the child as under-stimulating, e.g. due to general motor retardation, reduced energy level, flat affect and general emotional unavailability. The children are exposed to negative maternal affect and behaviors, in particular hostility, withdrawal, inconsistent parenting practices, social withdrawal and non-contingent responsiveness.

Compromised or at least impaired parenting of depressed individuals is covered by a large body of literature (Downey and Coyne, 1990; Beardslee, Versage and Gladstone, 1998; Goodman and Gotlib, 1999). Impaired parenting issues of depressed mothers have been discussed under two aspects: first, the restriction of parenting resources that are directly related to the symptoms of the disorder (e.g., Lovejoy et al., 2000) and, secondly, an increase of negative (usually summarized as distorted) affects (e.g. Goodman and Gotlib, 1999).

First, a maternal depression may affect parenting by reducing the effort that is invested into dyadic interaction e.g. due to reduced energy levels, self-absorption and motor retardation (e.g., also refer to criteria of major depression, page 8). These behavior restrictions have been claimed to interfere with behaviors necessary for maintaining mother-infant interaction (Downey and Coyne, 1990). For example

depressed mothers were found to speak slower, speak less, with less volume, in a monotonous tone, with larger silence periods and take longer to respond (Segrin and Abramson, 1994). They were found to use less eye contact in interaction. As individuals, depressed persons, were reported to have social skill impairments (Segrin, 2000). Depression has been reported as debilitating, in particular in terms of social functioning (Hirschfeld et al., 2000). Lower involvement in dyadic interaction and reduced responsiveness suggest “an individual who does not find interaction to be rewarding and whose enthusiasm for social interaction is near zero” (Segrin and Abramson, 1994; page 658).

Secondly, parenting of depressed individuals has been characterized by distorted affects (e.g., the generally flattened affect plus outbursts in hostility or intrusions) which may affect emotion exchanges between mother and child. Weissman, Paykel and Klerman (1972) not only found reduced emotional involvement, impaired communication, but also increased hostility and resentment in depressed mothers. Burbach and Borduin (1986) described them as having difficulties with their role as parent. Depressed were reported to experience negativity toward parenting demands, elicit rejection and hostility towards their child and be less competent than other parents (Davenport et al., 1984; Webster-Stratton, 1998; Gordon et al., 1989; Hammen et al., 1987; Panaccione and Wahler, 1986; Segrin and Abramson, 1994). Depressed mothers consider themselves having a lower self-efficacy (Weaver, Shaw, Dishion and Wilson, 2008). Living and interacting with a depressed individual has been characterized to be negative, conflictful and stressful (Coyne et al., 1987). They were found to engage in rejective parenting (Trentacosta and Shaw, 2008) defined as hostile, controlling engagement to noncompliant child behavior associated with predominantly negative, aversive, or, even coercive interactions (e.g., Patterson, 1982). Depressed mothers were found to have problems in the use of directives in guiding child behavior (Kochanska and Kuczynski, 1989). Paulson, Dauber and Leiferman (2006) found anticipatory guidance to be disrupted in presence of depression, e.g. in areas such as sleep habits, discipline, and less positive enrichment activity for the child.

Observational studies in infants and children (e.g. for a review refer to Lovejoy, Graczyk, O'Hare and Neuman, 2000), similarly, found a large range of parenting difficulties in depressed mothers, such as reductions in positivity and heightened

hostility. In accordance with the model of restricted parental resources, depressed mothers were reported to be less responsive, be less synchronous, and generally to interact less positively with their children (Cohn et al., 1990; Field et al., 1990; Goodman and Brumley, 1990).

Thus, a range of authors noted that maternal depression may be experienced as aversive by a major interaction partner (Goodman and Gotlib, 1999; Field et al., 1984; 1988; Campbell, Cohn and Meyers, 1995; Cohn et al., 1990).

If the “exposure model” is added it may be predicted that adverse effects on an infant increase with exposure time. And, since the total daily “exposure time” for an infant has been shown to be usually high (Baildam et al., 2000), social competencies of an infant of a depressed mothers are expected to be restricted. Thus, predominant components of the maternal disorder, together with exposure characteristics may predict that children of depressed mothers are at heightened risk for adverse outcome, e.g., heightened levels of negativity, heightened activity levels, or cognitive impairments such as retarded development of receptive language (e.g., due to under-stimulation).

However, depression associated factors, such as marital discord, are not expected to exert direct effects on 3 month olds (Weindrich, Laucht, Esser and Schmidt, 1992). The symptom based approach has been harshly criticized by Lovejoy, Graczyk, O'Hare and Neuman, 2000) predominantly due to its unidirectionality and neglect of child factors (e.g., child temperament) and bidirectional interpersonal processes. In particular dysfunctional interpersonal processes have repeatedly been claimed either to maintain or exacerbate maternal depression (e.g. in the sense of Coyne 1976; Joiner and Coyne, 1999; Joiner and Katz, 1999; e.g., Coyne claimed that depressed individuals elicit rejection feedback in the interaction partner which itself is predicted to maintain or exacerbate the depression). Moreover, unidirectional approaches or behaviorist theories in child development, i.e. parenting conceptualized as teaching and learning by reinforcement or observation (Maccoby, 2000; WHO, 2004), were increasingly replaced by concepts of mutual exchange or dyadic interaction; based on the idea of a highly skilful infant (Trevvarthen, 1974; Dornes, 2004).

Lewinsohn (1969; 1974; Coyne, 1986; Lewinsohn et al., 2005) assumed that depressed or withdrawn behavior may be anteceded by a lack of response-

contingent positive reinforcement and that depressed individuals may lack both the availability and ability to make use of reinforcing behaviors. Lewinson also made the assumption that low response-contingent reinforcement may stimulate depressive behaviors, e.g., it may explain lowered behavior rates. Moreover he assumed that the amount of response-contingent positive reinforcers an individual gets depends on the existence of reinforcing events, their availability to the individual and the skill of the individual to use them. Thus, depressed individuals may have received or are still receiving a low rate of response-contingent reinforcers.

2.6.2 Maternal affect mirroring as social biofeedback, Gergely

Gergely and Watson developed a model that describes how infant behavior may be controlled by using affect mirroring as biofeedback during interaction (1996; 1999). Gergely theorizes how a mirroring mother may be able to regulate affects of her infant (Dornes, 2004). He essentially suggests that the maternal affect feedback modulates infant behavior. Affects, however, are not expected to be mirrored exactly, but in a multimodal (face, voice) and modulated manner, i.e. the mother has all degrees of regulation at her disposal and modulation may include affect intensification or de-escalation until the infants' emotional state parallels the maternal state. Gergely includes several sub-processes in his theory which he assumes are necessary for infant affect modulation: One is markedness, i.e. the mother signals an "as if" congruence or an exaggeration with the affect expression of her infant. Another process is referential decoupling and anchoring, i.e. the ability of an infant, to conclude that exaggerated maternal expressions mirror his own expressions. The affect is "decoupled" (detached) from the emitting subject and is "anchored" (learned, or referenced to own inner states). Gergely conceptualizes social biofeedback as a prototypical learning situation: The infant expresses inner states; those expressions are followed by maternal regulation and are mirrored more or less exactly (in modulated form). This allows the infant to connect inner states with corresponding expressions of affect. Gergely's theory, predicts that maternal mirroring of infant affects fosters emotional development, e.g., of categories of emotional states and the ability to self-regulate emotions.

Preconditions for affect mirroring are that infants are well capable of contingency perception (Rochat and Morgan, 1995; Rochat, Neisser and Marian, 1998; Markova and Legerstee, 2006; Bigelow and Rochat, 2006; Gergely and Watson, 1999;

Watson, 2001) and contingency maximization, emotion perception, and the ability of basic emotional expressions.

Gergely's proposed model can be summarized as follows: Maternal affect mirroring has three essential functions: it allows for a regulation of infant affects, affect transmission, and it leads to representations or connections of inner states and emotional expressions in the infant, and, finally, it predicts that infant competence of regulability and mentalization (e.g., the awareness of emotional states of others) are extended via affect mirroring behavior of the caretaker (Fonagy, Gergely and Target, 2007).

Aspects of deviant or pathological styles of affect mirroring: Gergely and Watson (1996; page 1202-3) differentiate deviant mirroring styles, i.e. two possible forms where maternal affect mirroring fails: lack of markedness and lack of congruence. Lack of markedness is not viewed as equivalent to ignoring affects. According to Gergely, markedness includes a signal that affects are being mirrored (the "as if" congruence). If an affect-display is not marked, the infant presumably fails to carry out referential decoupling (i.e. to detach the affect display from the mother and refer it to himself). The affect display will be attributed to the parent as her real emotion and will not be anchored to the infant. Since the infant emotion-states are established, a deficient self-perception and self-control of affect may result. Gergely lists as an example (page 1202) the exact matching of negativity between mother and infant, where the maternal style is realistic or unmarked and the infant may attribute mirrored negativity to the caregiver rather than to himself, and a down-modulation of infant negativity may fail and escalations of infant negativity may occur, possibly with impairments in the long-term built-up of infant regulation capacities.

A second form of deviant mirroring styles combines non-congruence (with the affect of the infant) with markedness, e.g. states of maternal hostility expressed by use of positive affects expressions. Gergely suggests this could lead to distorted representations of inner states of an infant and to deviant emotion expressions (Gergely and Watson, 1996; page 1203). In particular, over-controlling or defensively distorted parental perceptions of the infant's affect may favor such a mirroring style.

2.6.3 Mirroring as mediator for behavior transmission

Concepts of mirroring and modeling, e.g., the classical theories of Bandura (1977; 1985) have been expanded to infants and neonates by Meltzoff et al. (1988; 1989; 1992; 1994; 1997; Dornes, 2004). In general these theories assume that mirroring importantly promotes social development through observational learning, which may stimulate, facilitate, or inhibit infant behaviors. In particular, Meltzoff (1983) bases his theory on imitational capacities in neonates on the observation that from their first day, neonates respond to parental facial expressions, although their repertoire in the first days is limited to few behaviors such as the imitation of mouth opening, tongue and lip protrusion (Meltzoff and Moore, 1983). Field et al. (1982) found that affect expressions are imitated by few day old infants. Meltzoff and Moore (1977) suggested that (even in newborns) imitation is an intentional reproduction of a model: Infants have been reported to correct their imitative efforts in case of errors (Meltzoff and Moore, 1977; 1983; 1994) and even novel acts can be imitated (Fontaine, 1984; Meltzoff and Moore, 1994; as well as absent targets (Heimann and Schaller, 1985; Legerstee, 1991; 1977; 1989; 1992; 1994). Moreover, infants have been reported to recognize that they are being imitated and developmental change occurs in imitation processes (Field et al., 1986; Jacobson, 1979; Meltzoff and Moore, 1992; Meltzoff and Moore, 1997; Meltzoff and Moore, 2002).

Thus, before spoken language emerges imitation may play a role in the acquisition of social skills, and emotion expression may be formed through imitation, e.g., imitations of maternal vocalizations or facial expressions. Imitation is considered to be a social response with implications for infant development of nonverbal communication and speech. Accordingly, the neonatal capacity for imitation may be an effective mechanism for the intergenerational transmission of maternal behavior and characteristics. Meltzoff et al. basically assume that certain adult gestures will be responded to with matching responses by young infants (not necessarily over the same channel, e.g., by imitation of mimic gestures, mouth or head movements). Thus, the behavioral repertoire and knowledge about objects in early infancy is thought to grow after perception of behavior of the interaction partner

Particularly infants who are in close proximity to the mother might use her as major model. Those infants may either imitate motor activity levels (e.g., in the case of a depressed mother her unresponsiveness and slowed pace), emotional displays

(flattened emotions, Field, 1984; Cohn et al., 1986), or verbal behavior (lack of positivity in expressions). Field (1984) in particular referenced Bandura's concept to explain negativism and withdrawal both in the maternal and the infants' interactions and suggested that the depressed mother's interactional style may be mirrored in the infant's behavior and be reflected in the low general level of infant activity. Maternal modeling as risk mechanism for transmission, e.g., for depressed affect, reduced facial expressions, or reduced level of motor behavior has been suggested by a range of other authors (e.g. Teti et al., 1995; Field, 1984, 1985; Lyons-Ruth et al., 1986; Lovejoy et al., 2000; Downey and Coyne, 1990). Harnish, Dodge and Valente (1995) suggested that poor quality mother-child interactions predict heightened risks of child externalizing behavior, since the mother's negative interactional style acts as model whilst the child's knowledge of rules of social contingency is lacking.

2.7. Further models

A few interesting model alternatives are available. However, up to today no competitive model comparison is available, e.g., to allow for a data-driven preference of one model. Moreover, several models of those listed below involve the above-listed characteristics of a depressed parent, i.e. components of parenting impairment, or problems or weaknesses in exchange processes between mother and infant.

2.7.1 Models of coercivity in interactions

A well-known model regarding problems in dyadic exchange processes is Patterson's (1982; 1980) theory of coercive family processes. Patterson describes coercive cycles of interaction. According to his theory, mothers react either with withdrawal or hostility after repeated unsuccessful interactions with difficult children, and, use coercion as the dominating mode of controlling their children, which itself may increase the risk of child negative behavior. In the long-term, the theory predicts that both interactants will be reinforced by their maladaptive behavior (e.g. the child compels rewards by using misbehavior), which may promote coercive cycles (Patterson, DeBaryshe and Ramsey, 1989; Patterson, 1982). Thus, children and parents behave aversively, escalating, and circular exchanges dictate the probability of negative enforcement (for example, harsh and inconsistent discipline, coupled with low warmth and involvement were found to predict child misconduct). Patterson's model describes how interactants coerce reactions from each other in dysfunctional ways. Following Patterson's theory of "coercive process" Hops et al. (1987) and

Biglan et al. (1985) assume that depressive behavior is one form of coercion: depressed individuals try to control aversive or aggressive behaviors of others by emitting dysphoric expressions. The effectiveness of the coercion itself may serve as reinforcement for dysphoric behavior.

2.7.2 Mood contagion approaches

Coyne et al. developed an interpersonal model with depressiogenic effects on the interactants (e.g., refer to Coyne's essential papers on depression, Coyne, 1985; or, 1976; Coyne and Gotlib, 1983; Downey and Coyne, 1990; Segrin and Abramson, 1994; or the meta analysis of Joiner and Katz, 1999). Coyne's theory (1976) claims that a depressed individual induces a negative mood in the interaction partner during interaction. This negative mood, then, is then thought to provoke rejection, e.g., hostility, depression, or anxiety. Thus the negativity induction in the counterpart of the depressed individual is assumed to mediate rejection. It is noteworthy that it is not behavior itself that is predicted to cause rejection but rather, a negative mood. The rejection itself can maintain or exacerbate the depression. Moreover, Coyne's theory is phrased very general and makes no statements regarding the age of the interaction participants: age is neither presumed to be a mediating factor (i.e. a necessary precondition), nor a moderating factor. Accordingly, in caregiver-infant interaction with a caregiver who is afflicted with major depression, infants have been found to be unwilling to interact, or to show signs of non-compliance (Mohan, 2004) and to show signs of indifference or rejection (Colletta, 1983). Segrin and Dillard (1992) also reported this rejection-effect (operationalized via rating scale) in their meta-analysis. However, the negative mood induction hypothesis was found to be u-shaped: Increasing depression severity was found to be associated with heightened levels of negative mood in others, but the highest levels of depression severity were associated with low negative mood.

Two further models with a focus on the risks associated with the exposure to a mother with depression are the models of Hammen (Hammen and Shih, 2004) and Goodman and Gotlib (1999).

Hammen's intergenerational interpersonal stress model of depression (Hammen, 1991; Hammen and Shih, 2004; Hammen and Brennan, 2002; Hammen and Shih, 2004; Hammen and Brennan, 2001; Hammen, Shih, Altman and Brennan, 2003; Hammen, 2003) suggests that maternal depression is associated with

dysfunctional interpersonal behavior, which itself is a key mediator for effects such as child maladaptation such as impaired social competences, inadequate social problem solving skills or poor coping with social stressors. Child maladaptation itself may then contribute to the generation of stressful interpersonal life events and increase the risk of depression in children and adolescents. The offspring is either exposed to maternal interpersonal difficulties with others or to poor parenting. Maladaptive parenting, i.e. parenting perceived as negative and stressful, is viewed as an important characteristic of families in which a depressed mother lives. A maternal depression may include the modeling of inadequate social behavior, and the mother may cope poorly with social stressors. Although the theory was developed with 8-16 year olds age is phrased neither as mediator nor as moderator. According to Hammen, the model is intergenerational since maternal depression is used as the key element in predicting depression in offspring. The quality of interpersonal relationships, which themselves may increase family stress, is assumed to be one key transmission mechanism. Depressed behavior is experienced as stressful and high stress in a depressed mother's interpersonal relationships with other family members predicts poor parenting quality, low child social competence, and high interpersonal stress in the mother-child relationship. Thus, maternal depression is assumed to exert its effects largely through maternal interpersonal stress and a low parenting quality (although direct effects from maternal depression on child maladaptation are not denied).

The multifactor model of Goodman and Gotlib (1999). Their "integrative" model intends to explain the transmission of risk to children of depressed mothers and lists factors which presumably precede the childhood risk for psychopathology. Goodman includes four possibly effective mechanisms by which maternal depression affects the functioning of the child, such as (a) heritability components of depression, (b) innate dysfunctional neuroregulation, (c) negative cognitions, behaviors, and affect of the mother; and finally (d) the stressful environmental context in which the child lives. According to this model any mother-child dyad can be characterized with zero to 4 risk components that can act interdependently. Any one or more of the factors mentioned above increase child vulnerabilities which includes a wide range of changes in child functioning, such as neuroregulatory changes, i.e. changes of the hypothalamic-pituitary adrenocortical axis, changes in developmental performance

indicators or emotional regulation, as well as behavioral or interpersonal indicators, e.g., inadequate social skills or lowered impulse control of children of depressed mothers. Since the model implies a dynamic system, child vulnerabilities may lead to an increased maternal stress, may lower parenting efficacy and the quality of mother-child interactions. The final pathway of Goodman's model, subsequent to the acquisition of child vulnerabilities, is the emergence of disorders.

2.8. Models with a focus on bidirectional pathways

In the literature, current perspectives of bidirectional dyadic interaction are discussed within three interdisciplinary streams a) the developmental-psychological perspective (predominantly intradyadic processes of affect mirroring), b) the interactional perspective of preverbal/affective exchanges and c) the intersubjective perspective of preverbal and affective exchanges. For each perspective the core messages of theory, the interactive process of affective exchange and the hypothesized effects of intradyadic mechanisms on the development of infant emotions will be described with application to the case of a current episode of major depression in the primary caregiver.

2.8.1 Interaction as facilitating environment for development - Winnicott

Winnicott (1965; WHO, 2004) conceptualized dyadic interaction as facilitating environment. He postulated that infants were already sensitive to caregiver emotions and interactions. In particular, he suggested that these interactions and the mother-infant relationship may promote infant development, since they form mental representations of the world and a concept of the self in the infant. Winnicott conceptualized care-giving as a period of heightened maternal awareness on the state of the infant, on expressed infant emotion and behaviors. Heightened awareness is claimed to allow the caregiver to sensitively adjust and respond to the infant. This responsive care giving was claimed to be essential for emotional infant development and the internalization of external objects via early interaction with the parent. Conversely, impaired infant development is predicted in the case of an insensitive or neglecting caregiver. Unsatisfactory maternal care may lead to interruptions in child behavior, to ongoing interpersonal impingements, and interfere with recoveries from these processes.

2.8.2 Dyadic interaction as affective attunement and as preconditions to develop intersubjectivity

Stern (1985 2008) suggested that mirrored affect and the process of affect attunement in dyadic interaction is a central mechanism of transmission with respect to infant development. Mirroring changes qualitatively during the first infant year from exact imitation to affective attunement, i.e. mirroring of affects based on different modalities. These so called transmodal matching abilities are suggested to be essential for the development of intersubjectivity. Attunement involves resonance of different (intermodal) channels between parent and infant. This involves processes such as parental matching of the infant's internal feeling state (although not in an identical manner with an exact imitation of behavior), moreover, as well as the cross-modal affective expressions of both interactants, and a signaling that marks those shared affect states.

Stern characterizes the interactive process as an exchange of experiences without reference to an external object. His basic assumption is that infants are able to capture the quality of the interaction partners' state via different behavior dimensions. Maternal preconditions for optimal "attunement" include the reading of infant's state and infant behavior, skills to perform a corresponding behavior and an infant that is able to read parental responses. Stern's position has been extended by Meltzoff (1988; Meltzoff and Moore, 1989) and Trevarthen (1974) to very young infants (≤ 2 months). Non-attunement and skill deficits in sharing intersubjective states, however, might be predictive for subsequent interaction deficits and the development of psychopathology (Stern, 1985; page 210) or developmental impairments, e.g., in language development. The importance of Stern's concept is the assumption that inner states and experiences of the outer world are getting connected during dyadic interaction, that infant abilities of affective regulation are shaped (i.e. affective self regulation develops from interpersonal regulation of affect), and that the relation between inner affective state and own emotional expressions are established, infant affects are mirrored and regulated.

2.8.3 Intersubjective perspective - Trevarthen's theory of an innate intersubjectivity

Theories of intersubjectivity usually postulate that the infant - from birth on - is capable of self awareness, and able to participate in the affective state of his interaction partner as well as share (at least in part) his perspective or his affective

state. Trevarthen's theory suggests that an infant is born with a receptive awareness of subjective states in others (Trevarthen and Aitken, 2001). Beginning with the 6th week, intersubjectivity involves reciprocal regulation between infant and caretaker based on mimics, vocalizations and gestures, but - most importantly - in a multimodal manner: not exact imitations, but rather a specific regulation target is suggested to be essential. The infant is thought to act intentionally and to play an active role, e.g., in emotion communication, in imitating the caretaker, or provoking caretaker reactions with emotion expressions. It is proposed, that not social interaction but the infant's intrinsic motivation for affective exchanges favours infant development (Trevarthen and Aitken, 2001). In contrast, interactionistic theories usually state that the origin of infant self-awareness results from dyadic interaction, particularly affective mirroring and regulation experiences with a primary caretaker. Trevarthen defines intersubjectivity as an ability to not only express intentions and affects, but to apprehend and read these intentions and thus participate in those affects (Trevarthen and Aitken, 2001; Murray, 1991; Trevarthen, 2001). Proposed components of intersubjectivity are reciprocal coordination of affect, i.e. bidirectional exchanges of affect, and the adjustment of self awareness. Trevarthen further assumes intentionality of both partners, i.e. the assumption of intrinsic motive states to express own affect and read affects. Joint attention and mirroring, as well as recursive affects, are necessary processes of intersubjectivity.

2.8.4 Concepts of bidirectional regulation via dyadic interaction (Beebe and Lachmann)

Beebe and Lachmann (1994) conceptualized a bidirectional model of intradyadic processes of regulation for the first year of life. They suggested that mother-infant interactions are co-constructed, i.e. both interactants use self-regulation and interactive regulation to structure the interactive process. Interaction is thought to be best conceptualized by three principles: a) continuously ongoing bidirectional regulations (e.g., via affect mirroring, affect matching or rhythmic timings), b) ongoing repair of disruptions during interaction, and, finally, c) interaction is characterized by heightened affective moments, particularly observable in facial mirroring of affect. In accordance with Stern (1985), affective mirroring is not conceptualized as exact imitation but as parallelisms of affect, where both interactants may use different communication channels (e.g., facial, verbal affect expressions). Affective exchanges are (in accordance with Stern, 1985) thought to be a basis for pre-symbolic self and

object representations. Ongoing regulations presumably generate predictive expectations in the infant with respect to regulation patterns. According to Beebe and Lachmann (1994), interactive regulation favors infant competencies for self-regulation and interactive regulation and the competence for pre-symbolic communication.

2.8.5 Additional regulatory models - Tronick's model of mutual regulation

First published under the title "transmission of maternal disturbance to the infant" (Tronick and Gianino, 1986) Tronick's model suggests that maternal depression and the associated unresponsiveness and failure to read infant cues lead to a state that is labeled as "mismatching" or "poorly coordinated interactions" (page 9). Dyads with a healthy, i.e. non-depressed mother are expected to match themselves affectively using bidirectional processes. Both interactants are assumed to be guided (Weinberg, Tronick, Cohn and Olson, 1999) by their expressive displays (e.g., facial expressions, gestures, and vocalizations). Tronick assumes that the interaction quality depends on both the ability to regulate and express affects and to be able to respond emotionally. A depressed mother, however, may fail to give her infant regulatory help. Initially, the infant may reinforce attempts to attract the mother's attention and repair (re-establish) the interaction. But the infant - in the case of having a depressed mother - is predicted to be unable to initiate positive interactions, may turn to self-stimulatory behavior. Unregulated parent-infant interactions and the infant's self-directed behavior may dominate interaction styles. These interaction styles when coupled with an unreliable mother might favor the development of child psychopathology.

2.8.6 Additional regulatory models: Field's model of attunement

This model is a variant of the previous model proposed by Tronick. It is based on observations that infants appeared to imitate behaviors of a depressed mother (Field, 1984), for example, less vocalizations or a reduced frequency of expressions of affect. The attunement model postulates that the mother modulates her behaviors to those of the infant in order to provide adequate stimulation. In healthy dyads highly synchronized interactions should be expected. However, in the case of an affectively unresponsive or emotionally unavailable or depressed mother, unsynchronized and disorganized behaviors with disturbed affect and a lack of regulation skills might result.

2.8.7 Interaction as preverbal communication - Bruner

Interactionistic perspectives usually define early social interactions as precedents of human competencies, e.g., gain of cognition, language, knowledge of self and others. Thus, dyadic interaction is suggested to initiate and favor infant development, especially in a very asymmetric manner, since the behavior of the primary caretaker is thought to be the primary determinant of development.

One of the pioneers of interaction related theories - particularly with a focus on language acquisition - is Jerome Bruner. Bruner (1974, 1976) focused on early social interactions as pre-speech mother-infant communication. Pre-speech interactions are claimed as central, since they may sensitize the infant to communicate inner states and needs, e.g., through the use of reciprocal preverbal conversations with gaze behaviors and vocalizations. Two processes are suggested: The regulation of attention via gaze focusing of both interactants beginning with the 2nd month with accompanying voices on both sides and measures of directing the infant's attention by introducing objects. Non-dysfunctional early interactions are conceptualized as central in language development. In contrast, the theory also allows the prediction that early dysfunctional interactions, i.e. in the case of a withdrawn and unresponsive caregiver, may lead to a delayed speech acquisition and an infant using alternatives methods to communicate affective states, e.g., by excessive crying.

2.8.8 Social interaction as experience mediator - Vygotsky

Vygotsky (1978; Wertsch and Tulviste, 1992; WHO-report, 2004), similarly, claimed that social interactions mediate infant development, i.e. he suggested that the development of mental processes have social origins. This theory is in contrast to classical theories which focused on child maturation only. Vygotsky focused on the role of social interaction in child development, mostly in an asymmetric manner: The mother attributes meaning and interprets infant behavior. Child functioning results from the mastery of social processes in which the caregiver mediates experiences for the child by instructions, by structuring information and linguistic support, and thus by extending the child's capacity. Basically, the child's relations to the environment are conceptualized as social relations. Vygotsky's theory is a suitable basis for the prediction that in the case of parental depression, a non-responsive, withdrawn caregiver may fail as "skill mediator": Lacking scaffolding and lack of interactive support in social mastery processes might result in developmental impairments such

as in child functioning or social skills.

2.8.9 Maternal emotional availability as reciprocal reward system

Emde et al. favor special maternal qualities during dyadic interaction that may act as transmission mechanism. In particular they suggest that the **emotional availability** of a caregiver might be relevant for child development (Emde, 1980; 2000; Frankenburg, Emde and Sullivan, 1984; Biringen, 2000; Weifel et al., 2005; Bornstein, 2006), i.e. they believe that the affective components of early mother-child interactions are a reciprocal reward system that helps to create the infant's internal object relations or working models. It is suggested that a parent facilitates contingencies between infant action and result. Emde focused on the emotional tone of interactions (rated by observers) as a quality measure of the parent-child relationship. Biringen (2000) conceptualized emotional availability in terms of parental sensitivity and non-intrusiveness, child responsiveness and involvement. Emotional features, such as parental emotional signals and understanding of infant signals, are suggested to define the quality and health of parent-child interactions. Accordingly, active caregiver engagement is expected to predict infant expressions. Expressing a range of emotions is assumed to be an important incentive for engagement for both caregiver and infant. Former theories of emotional availability used the term "supportive maternal presence" and the communication of emotions had been assigned an eminent role in healthy child adaptations. The predictive value of maternal depression, then, is conceptualized as an interfering condition for emotional availability (page 98, 105).

Similarly, Demos (1982) suggested that the affective component in mother-infant interaction in particular is important in child development and in the modification of pre-existing child affects. Maturation plus caregiver-child interactions are suggested to transform unorganized child behaviors and a repertoire of functions into dispositions and also shape affective behaviors. Accordingly, Demos stresses the role of affect in development. Child affects are organized and thus regulated in mother-infant transactions but the pre-existence of a maternal affective component is essential.

2.8.10 Social referencing as behavioral transmission mechanism

The term "social referencing" describes the possibility of effectively regulating infant behavior or the regard for an object through parental messages of emotion; e.g.,

happiness or fear. Social referencing has been defined as infant behavior regulation by emotional messages that a caregiver provides (Walden and Kim, 2005). Social referencing allows infants to be guided in novel situations or situations of uncertainty through maternal emotional cues, e.g., prototypical facial expressions to signal information about environmental events or a novel object to resolve uncertainty (Dornes, 2008; Walden and Ogan, 1988). The classical paradigm is the visual cliff situation, where an infant's locomotion over a plexiglass-covered table is guided by face-to-face maternal expressions (Sorce et al., 1985). Other examples are the guidance in the presence of a stranger or the inhibition of toy use (Camras and Sachs, 1991; Feinman and Lewis, 1983; Sorce, Emde, Campos and Klinnert, 1985). According to Feinman (1982), expressions of maternal emotions are not only responses that indicate internal states of a mother, but are also indicators for the child's regulatory behavior. Conversely, the ability of an infant to read maternal cues and the ability of a mother to emit readable cues may be characterized as relevant for this "behavior transmission mechanism". In the case of a depressed mother transmissions from mother to infant or vice versa may completely fail if maternal emotional cues are weak or are lacking. This may result in a non-responsive, under-stimulated, or highly eliciting infant who is constantly searching for social cues. Social referencing has been extensively studied over various infant ages (e.g., Sorce, Emde, Campos and Klinnert, 1985; Striano and Rochat, 2000; Moses, Baldwin, Rosicky and Tidball, 2001) and although classical social referencing studies included 12-month old infants, some authors suggest that younger infants are capable of using components of social referencing (information seeking, e.g., Hornik and Gunnar, 1988). For example, 6-9 months olds and older infants were examined by Walden and Ogan (1988). They reported that infants of all age groups responded to maternal social signaling. However, before effective infant locomotion has been developed (the infants of this research are well below one year), the value of a social referencing paradigm is very limited. But other social referencing situations have been created that rely on the reading and interpreting of the maternal focus of attention, or by developing special attention to facial expressions (e.g., Striano and Rochat, 2000; Feinman and Lewis, 1983; Walden and Baxter, 1989; Walden and Ogan, 1988). For example, it has been shown that infants are well capable of reacting to and discriminating between various emotional expressions, either facial or vocal, in the early months of life (Walker-Andrews, 1997; Bushnell, 1982).

Some authors also suggested that the theory of social referencing may have the implication of the mood contagion hypothesis of Coyne (1976; Joiner and Katz, 1999), i.e., it is basically assumed that a socially referring infant adopts the emotional state of the a (e.g. depressed) interaction partner (Slaughter and McConnell, 2003). Accordingly, an infant interacting with an emotionally flat mother with affectionless tone of voice and reduced emotional displays may be predicted to adopt negative emotions and withdrawn behaviors. Moreover, infant exploration behavior out of dyadic interaction may be inhibited since maternal depression may be witnessed as adverse; conversely, maternal expressions of pleasure seem to favor active infant exploration. Other implications of social referencing include associative learning, i.e. that the infant may learn to associate own and maternal emotional expressions, or the theory that infants evaluate objects using the emotional state of their interaction partner (Baldwin and Moses, 1996). Some authors found referencing to be higher effective, if a caretaker increases the level of affect expressions (Camras and Sachs, 1991).

2.9. Empirical evidence for impaired parenting in presence of maternal depression

A number of studies have shown that maternal depression is a suitable predictor for deviancies in mother-infant interaction.

The specific approaches (see table 3), however, vary considerably. Interaction behavior is either quantified based on ratings or coded using observational techniques. The predominant type of design, however, is a controlled cohort design using a group of dyads with a depressed mother and a healthy control group (mother and infant). The observations predominantly are made in the laboratory in standardized situations (e.g., using a standardized routine like the still-face procedure), and in some cases, at home.

Basically, the primary target of almost all studies listed below was to show detrimental effects of maternal depression on interactive behaviors. Specific theories that were addressed are affect mirroring approaches (Field, 1984; Field et al., 1988, 1990), parental disability or depression-symptom based approaches (Cohn et al., 1990, 1986; Bettes, 1988; Livingood, Daen and Smith, 1983; Hops et al., 1987; Fleming et al., 1988; Lyons-Ruth et al., 1986; Lyons-Ruth et al., 2000; Panaccione and Wahler, 1986; Davenport et al., 1984; Breznitz and Sherman, 1987).

The most frequently cited publications are those of Field (e.g., 1984), Cohn et al. (1990), Stein et al. (1991) and Stanley, Murray and Stein (2004). All four have shown maternal depression to be highly predictive for deviancies in indicators of interaction (these four major articles can be found in the table 3 below, refer to numbers 8, 6, 26 and 27).

All available studies are tabulated in table 3 with a detailed focus on study-design, sample sizes, findings and the authors' assumptions regarding the underlying mechanisms. They will be summarized more comprehensively on page 52.

Table 3: Studies examining the predictive value of parental depression on various indicators of maternal and infant outcome, e.g., vocalizations, positive engagement

	Study	Sample	Child age	Definition of depression	Observation situation	Results	Line of interpretation	Assumed underlying mechanisms	Limitations
1	Bettes (1988)	10 unipolar depressed mothers, 26 control dyads, community sample	3-4 months	BDI > 10	3-15 min face-to-face interaction in the home	Mothers with depression responded with higher latencies to the infant, they vocalized with longer and more variable pauses.	Postpartum depressed mothers are less responsive, indicating "time outs" and periods of disengagement and thus are likely to miss a certain necessary "inter-stimulus interval for infant conditioning"	A low response likelihood of a depressed mother imposes a risk on the infant since they are deprived of social cues, disturbances in interactive abilities may predict child psychopathology	Only a self-report screening measure for depression detection was used.
2	Breznitz and Sherman (1987)	Unipolar= 14, control = 18 (mothers)	3 years	RDC based on SADS-L	unstructured interaction	Depressed mothers vocalized lesser, responded less quickly, their infants spoke less, made lengthier vocalization pauses.	Depressed women may have a reduced speech capacity, children of depressed mothers may be exposed to different patterns of socialization, e.g., depressed individuals keep social interaction to a minimum	Depressed behave differently due to general motor retardation, reduced energy and social withdrawal. Children of depressed adapt to maternal level of speech productivity as a result of missing reinforcement.	small sample sizes
3	Campbell, Cohn, Flanagan, Popper and Meyers (1992)	70 unipolar, 59 healthy mothers	2 months	RDC	unstructured interactions during feeding and play sessions	Depressed were rated as less positive engaged, having more negative affect, there were no differences on infant social engagement. Depression severity was positively related to negativity.	Depressed women seem to be less competent as mothers (page 44, right column).	depressed mothers may express a sensitivity deficit, lack warmth and withdraw from their children	
4	Campbell, Cohn and Meyers (1995)	67 unipolar, 63 healthy mothers	2, 4, 6 months	SADS	home observations using standardized ratings during mother-infant interaction	No differences between depressed and healthy mothers were reported, neither during feeding, nor in face-to-face interactions, or during play, women whose depression lasted more than 6 months had less positive and more negative infants.	The duration of exposure to maternal depression may be an important factor, i.e. a protracted depression may exert different effects compared to a transient one.	The course of maternal depression may be predictive for the quality of the mother-infant relationship (page 355, right column).	

	Study	Sample	Child age	Definition of depression	Observation situation	Results	Line of interpretation	Assumed underlying mechanisms	Limitations
5	Cohn, Matias, Tronick, Connell and Lyons-Ruth (1986)	13 unipolar depressed mothers, community sample, low SES, control group missing	6-7 months	CES-D	6 min face-to-face and 40 min naturalistic interaction	Depressed were found to display high frequencies of disengagement paralleled by disengaged infants, high frequencies of intrusion while infants were looking away or disengaged even if mothers were positive, depressed were less responsive, flatness of affect correlated negatively with play behavior	Maternal intrusion seemed to be accompanied by infant gaze avoidance, maternal disengagement by high rates of infant protest. Depressed maternal behavior seemed to be characterized by detachment, impatience and insensitivity (page 40).	Adverse effects of maternal depression may be mediated by distorted or deviant interacting characteristics (page 41), maternal hostility may limit contact to infant, and opportunities to learn are fewer	Only a self-report screening measure for depression detection was used. A control group is missing.
6	Cohn, Campbell, Matias and Hopkins (1990)	24 unipolar depressed mothers, 12 controls drawn from the community	2 months	RDC based on SADS	3 min face-to-face interaction	Depressed (nonworking only) were slightly more negative and their babies were less positive. Using explorative regression mother negativity correlated with infant negativity (in the group of depressed mothers only).	Study supports previous studies, i.e. depressed mothers interact more negatively, their babies less positively).	A carry over effect for infant negativity due to reduced maternal responsiveness is discussed. This is assumed to result in a reduced involvement in persons and objects.	Sample size due to stratifications of design according to gender and working status too small.
7	Cohn, Campbell and Ross (1991)	33 unipolar, 33 healthy mothers	2, 4 and 6 months	SADS-L	home observations using standardized ratings during mother-infant interaction	No differences between depressed and healthy mothers were reported. Results for the whole sample were given instead: eliciting behavior of 6 month old infants predicted secure attachment (using explorative logistic regression). Failure in elicitation predicted avoidant behavior.	Positive emotions in mother-infant interactions predict later attachment.	Not applicable since no depression-related results were given.	Depression-related results are missing, although depression diagnosis is a major part of the study design.
8	Field (1984)	12 unipolar, 12 controls	3 months	BDI > 15	3 min face-to-face interaction during mother's simulation of looking normal and of looking depressed	Infants of depressed mothers had less positive and more negative facial expressions, vocalized lesser and protested more frequently. Mothers had less positive, more frequent negative facial expressions, spent less time looking at the child and provided less tactile stimulation.	Infants of depressed mothers behaved affectively different compared to infants of healthy mothers, even displaying little changes if the behavior of the mother changed.	Infants of depressed mothers may be accustomed to an unavailable mother, they may be under-stimulated, or assuming that infants are able to detect quality of maternal affect; they may mirror the maternal affects.	Sample sizes are small. Only a self-report screening measure for depression detection was used.

	Study	Sample	Child age	Definition of depression	Observation situation	Results	Line of interpretation	Assumed underlying mechanisms	Limitations
9	Field, Healy, Goldstein, Perry, Bendell, Schanberg, Zimmermann and Kuhn (1988)	40 unipolar, 34 control mothers, sample with lower socio-economic background	3-6 months	BDI \geq 12	3 min face-to-face interaction with mother and with a stranger	Depressed (both mother and infant) had lower activity levels, less facial expressions, and vocalized less. Infants of depressed mothers had lower ratings when interacting with a stranger on activity, vocalizations and contingent responsivity.	Depressed style of interaction seems to generalize from the depressed mother to strangers.	Genetic, prenatal factors or temperament might be responsible for impairments in infant behavior. Alternatively social learning processes, such as imitation, or limited stimulation effects may be responsible.	Only a self-report screening measure for depression detection was used.
10	Field, Sandberg, Garcia, Vega-Lahr, Goldstein and Guy (1985)	12 unipolar, 12 control mothers	3-5 months	BDI > 11	10 min face-to-face free play	Both infants of depressed and their mothers showed less activity and less facial expressions. Depressed mothers showed less imitating behavior and contingent responsivity.	Depressed mothers and their infants seem to show suboptimal interaction.	None discussed.	Sample sizes relatively small. Only a self-report screening measure for depression detection was used.
11	Field, Healy and Leblanc (1989)	7 unipolar depressed, 9 healthy mothers	3 months	BDI \geq 9	3 min face-to-face interaction	Dyads with a depressed mother spent significantly more time in anger, were less engaged and spent less time in play. Their infants spent more time in protest and less time in play. Both depressed mothers and their child spent more time matching for anger / protest, for disengagement / looking away, and spent less time in play, compared to healthy mothers and their infants.	Distressed dyads seem to share negative states more often if they are distressed, suggesting a contagion effect of negative mood.	The sharing of negative states may reflect a transmission of negative affect from mother to child.	Small sample sizes. Only a self-report screening measure for depression detection was used.
12	Field, Healy, Goldstein and Guthertz (1990)	24 unipolar, 24 healthy mothers	3 months	BDI \geq 9	3 min face-to-face interaction	Depressed mothers and their infants paralleled negative behavior states more often, yet positive states to a lesser degree.	The result reflect both a dominance of negativity in depressed mothers and a dominance of parallel occurring negativity both in the depressed mother and her infant.	In distressed mother-infant pairs a greater reciprocity of negative affect is discussed. Infant develops a depressed style of interacting.	Only a self-report screening measure for depression detection was used.

	Study	Sample	Child age	Definition of depression	Observation situation	Results	Line of interpretation	Assumed underlying mechanisms	Limitations
13	Field, Morrow and Adelstein (1993)	30 unipolar, 30 healthy mothers		BDI \geq 12	3 min face-to-face interaction	Depressed mothers and their infants were rated as being more negative and less positive. Depressed mothers rated their own infants as more negative and less positive compared with an independent observer.	Depressed mothers coded their infants more negatively compared to non-depressed mothers who agreed with the independent observer.	Depressed mothers may have a distorted or biased perception of their infants. Mothers may transfer their own negative view of self.	Only a self-report screening measure for depression detection was used.
14	Fleming, Ruble, Flett and Shaul (1988)	56 mothers from community; correlational study	3 days, 1, 3 and 16 months	10-item mood scale	At 3 days, feeding situation; at 1, 3, 16 months, naturalistic interaction for 10 min.	High scoring depressed mothers were found to score lower on affectionate or engaged contact time. Depression was associated with less engaged behavior only 1 month after birth; after 16 months no differences were found.	Self-rated and mild depression is associated with reduced feelings and less affectionate behavior towards the infant	Early care taking activities are associated with less affectionate contact behavior, physical contact is reduced in postpartum depressed mothers, depression reduces feelings of maternal adequacy with subsequent reduction of optimal mothering (page 79, right column)	Only a self-report screening measure for depression detection was used. Screening measure is unspecific towards depression.
15	Goodman and Brumley (1990)	25 depressed and 23 control mothers (plus 53 schizophrenic)	3 months to 5 years	major depression according to DSM III criteria	5 minute play session	Dyads with a depressed mother showed no differences in rated interaction (maternal affect, tenseness, hostility or responsiveness, page 37). Differences emerged only in schizophrenic mothers.	Depressed women do not have a lower quality of parenting in general, but do show higher variability as a group	Depressed mothers may have impaired parenting quality due to restricted social functioning	

	Study	Sample	Child age	Definition of depression	Observation situation	Results	Line of interpretation	Assumed underlying mechanisms	Limitations
16	Gordon, Hammen, Adrian, Jaenicke, Hiroto and Burge (1989)	12 mothers with treated chronic or recurrent unipolar depression, 12 with bipolar disorder and 12 medically ill mothers	8-16 years	RDC based on SADS-L	5 minute interaction during achievement and during conflict resolution task	Compared to non-depressed mothers depressed mothers were less positive, were mostly negative, behaved in a disconfirmatory and critical manner that was paralleled by very unproductive comments	unipolar depressed mothers display a negative interaction style	Depression disrupts the maternal role, chronic stress may be confounded with the affective disorder, especially chronic stress apparently was associated with less confirming, positive behavior and criticalness with mood; depressed mothers judges the child negatively which may lead to critical or hostile behavior, reduced sensitivity may lead to disconfirming behavior, stress reduced tolerance to child with leading to negative maternal reactions	Small sample sizes, high refusal-to-participate rates
17	Hoffman and Drotar (1991)	11 unipolar, 11 healthy mothers	2 months	BDI > 10	20 minutes unstructured interaction	Depressed mothers and their infants scored lower on positivity in interaction, affect expression and responsivity. Maternal stimulation and infant activity was not different.	Depression may have a selective effect on social affect-related interactions since no differences in overall activity and stimulation were found.	Children may mirror maternal behavior.	Only a self-report screening measure for depression detection was used.
18	Hops, Biglan, Sherman, Arthur, Friedman and Osteen (1987)	27 unipolar, 27 control mothers	3-16 years	RDC based on SADS	Naturalistic in-home interaction of all family members, 20 minutes with the mother, 10 minutes with other family members	No heightened rate of aversive interactions in association with parental depression was found.	Some single effects only emerged in association with marital discord	Third variable mechanisms may account for effects normally attributed to maternal depression	

	Study	Sample	Child age	Definition of depression	Observation situation	Results	Line of interpretation	Assumed underlying mechanisms	Limitations
19	Hossain, Field, Gonzalez, Malphurs and Del Valle (1994)	12 unipolar and 14 non-depressed mothers, fathers were included	3-6 months	BDI \geq 12	3 min face-to-face interaction	There were few, almost no differences between healthy and depressed mothers (but fathers differed from depressed mothers)	Dyads with a depressed parent display "interactional difficulties"	Depression may stress the interaction partner, infant behavior in interaction with a depressed mother may not generalize to other individuals	Small sample size. Only a self-report screening measure for depression detection was used.
20	Livingood, Daen and Smith (1983)	25 unipolar, 25 control mothers, community sample	2 days	BDI > 10	15 -min feeding session	Depressed were found to score lower on unconditional positive regard, no other differences (e.g., vocalization, infant touching) emerged, marital adjustment had no effect.	Postpartum blues did appear to disorganize women in their maternal role.		Limited usability of study since depression was quantified 2-3 days after delivery, which increases the risk assessing postpartum blues instead of postpartum depression.
21	NICHD (1999)	92 chronically depressed, 460 depressed, 663 non-depressed mothers	6, 15, 24, 36 months	CES-D \geq 16	15 minutes play interaction at home as well as in the laboratory	Mothers with chronic depression were rated as less sensitive when interacting with their child, in mother-infant interaction there were no differences in negative or positive mood, children had lower scores on measures of performance and higher scores on aversive behavior.	Infants of depressed mothers show impairments in performance and higher aversive behavior.	Maternal sensitivity moderated the effects between maternal depression and child development.	Only a self-report screening measure for depression detection was used.
22	Murray, Hipwell, Hooper, Stein and Cooper (1996)	29 mothers with major depression, 20 healthy control mothers	2 months	RDC	5 minute face-to-face interaction, 5 minutes interaction with a stranger	No data were reported (although interactional parameters obtained) with respect to differences between depressed and non-depressed mothers.	Environmental factors, not depression, were predictive of adverse child outcome		

	Study	Sample	Child age	Definition of depression	Observation situation	Results	Line of interpretation	Assumed underlying mechanisms	Limitations
23	Panaccione and Wahler (1986)	N= 33, correlational study	M = 4.3 years	BDI	120 min naturalistic in-home interaction in four 30-min sessions	Higher depression scores correlated with high maternal aversive behavior, child behavior was better predictive for maternal behavior than maternal depression or coercive interactions	Child behavior appeared to act as stimulus for maternal behavior; depression appeared to act in the background.		Correlational study only, control group missing. Only a self-report screening measure for depression detection was used.
24	Pickens and Field (1993)	27 depressed mothers, 33 healthy control mothers	3 months	BDI \geq 13	3-minute face-to-face interaction	Infants of depressed mothers spent less time showing interest, and showed more sadness and anger compared to infants of non-depressed mothers.	Infants of depressed mothers mirror maternal behavior (page 987).	Infants of depressed mothers may be distressed or upset and thus show heightened anger, and, high frequencies of infant non-interest may be due to lowered maternal interest.	Only a self-report screening measure for depression detection was used.
25	Radke-Yarrow, Nottelmann, Belmont and Welsh (1993)	49 unipolar and 45 non-depressed mothers	1.5 - 3.5 years	RDC based on SADS-L	5 hours naturalistic observation on 2 days	Depressed mothers expressed more negativity towards their children and their children synchronously spent comparable time in negative affects.	Children of depressed mothers are synchronous with respect to negativity.		
26	Stanley, Murray and Stein (2004)	72 unipolar, 50 healthy dyads	3 months	DSM-III, SCID	6 minute interaction in face-to-face play in the still-face paradigm	Depressed dyads showed no differences in face-to-face interaction, depressed mothers were lower positively but higher negatively contingently responsive, i.e. had higher infant-negating sequences	Depression is associated with a reduction in mother-infant relationship quality.		
27	Stein, Gath, Bucher, Bond, Day and Cooper (1991)	49 unipolar and 49 non-depressed in the first year postpartum	19 months	Present state exam (stand. interview)	9 minutes of structured play between mother and child	Depressed mothers displayed less facilitation with their children, their children had lower affective sharing and less social behavior	Depressed mothers show reduced interaction quality which seemed to generalize to strangers or even after maternal remission	Depression reduces with maternal parenting capacity, marital and social difficulties may moderate or mediate effects, child temperament may aggravate maternal depression	Only a self-report screening measure for depression detection was used.

	Study	Sample	Child age	Definition of depression	Observation situation	Results	Line of interpretation	Assumed underlying mechanisms	Limitations
28	Weinberg and Tronick (1998)	13 mothers with major depression, 30 healthy control mothers	3 months	SCID	2-minute face-to-face interaction, 2-minute still-face, 2-minute face-to-face interaction with an unfamiliar research assistant	Depressed mothers talked less to their infants, touched less, were more invasive and disruptive, showed more anger toward their infants, their infants were less interested, expressed more anger, sadness, and tended to fuss and cry more.	Maternal depression appeared to compromise parenting and was associated with impaired maternal and infant social and emotional functioning.	Maternal unresponsiveness is assumed to disrupt "mutual regulation".	Small sample size of depressed mothers
29	Zlochower and Cohn (1996)	15 unipolar and 20 non-depressed mothers	4 months	RDC	3 min face-to-face interaction	Depressed mothers showed longer and more variable vocalization pauses during interaction with their infants.	Depressed mothers fail to coordinate their vocal behavior, are less responsive and thus less predictive for their infant.	Depressed mothers use timing mechanisms that are less predictive.	
30	Jameson, Gelfand, Kulcsar and Teti (1997)	29 unipolar depressed and 14 non-depressed mothers	13-29 months	formerly depressed (1 year), lifetime diagnosis of DSM III, major depress., dysthymic or adjustment disorder		Depressed mothers showed lower interactive coordination, repaired interruptions less often, and their infants were less likely to maintain the interaction.	Depressed mothers show reduced quality of interaction.	Maternal depression may compromise continuous interpersonal adjustment and participation in partnership.	A mixed sample of depressed and remitted patients and a small control sample were used. An unclear definition of interactive coordination was applied.
31	Field, Hernandez-Reif, Diego, Feijo, Vera, Gil and Sanders (2007)	14 depressed and 14 non-depressed mothers	4 months	CES-D score>16, SCID		Infants of depressed mothers and the mothers as well were less active (fewer positive and negative behaviors), and were less distressed during the still-face condition.	Depressed mothers are unresponsive and emotionally unavailable, their infants become accustomed to flat affect		Only a self-report screening measure for depression detection was used. Cut-off criteria for SCID not given.

	Study	Sample	Child age	Definition of depression	Observation situation	Results	Line of interpretation	Assumed underlying mechanisms	Limitations
32	Weinberg, Beeghly, Olson and Tronick (2008)	14 mothers with major depression, 14 with panic disorder and 48 control dyads	3 months	Structured Clinical Interview for DSM-III-R Axis I Disorders	6 minute interaction in face-to-face play in the still-face paradigm	Dyads with major depression did not differ compared to control dyads	No significant diagnostic group effects, probably due to the low risk sample, depression may unfold effects only in high risk samples		

BDI=Beck Depression Inventory; CES-D = Centre for Epidemiological Studies-Depression Scale; SES = socio-economic status; RDC = Research Diagnostic Criteria; SADS = Schedule of Affective Disorders and Schizophrenia, L= Lifetime; DSM = Diagnostic and Statistical Manual of Menial Disorders.

Bettes, 1988, investigated vocal interactions and found that depressed mothers were not able to mirror vocal behavior to their infant, i.e. they were found to respond more slowly, pause more frequently and act much more variably. They included 10 depressed and 26 non-depressed mothers. The infants were 3-4 months old. Primary target parameters were intonations of the mother, particularly their timing compared to infant vocalization. Participants were taped with a simple recorder at home for 3 to 15 minutes. Depression was identified using Beck's depression inventory and a score greater or equal to 10. Dependent measures were average duration of utterance (vocalizations with short pauses), variability of utterance duration, average pause duration, and variability of pause duration. Multiple univariate comparisons revealed no differences between depressed and normal mothers or infants for vocalization frequencies or mean durations. However, depressed mothers were found to need twice as long to respond to their infant, their vocalizations were longer and were more variable. Timing measures, such as pause duration or length of vocalization, showed positive correlations of about 0.40 to the Beck sum-score. Taken together, depressed mothers were found to vocalize and pause more briefly if preceded by vocalizations of the baby. The latter led the authors to conclude a failure of depressed mothers to modify their behavior and make use of vocal qualities in response to their infant.

Cohn, Matias, Tronick, Connell and Lyons-Ruth (1986) investigated 13 depressed mothers and their 6-7 month-old infants. Depression was identified via cut-off on the Centre for Epidemiological Studies Scale. Mothers and their infants were observed during face-to-face interactions in both structured and naturalistic observations. The study consists of a single group with one point of measurement. Repeated measurements and a control group of healthy mothers are missing. Despite the small sample size, the study contributed to several aspects that are rarely observed in the laboratory: remarkably high frequencies of disengagement paralleled by disengaged infants, high frequencies of intrusion while infants were looking away or disengaged even if mothers were positive. All in all depressed mothers were less responsive and their flatness of affect correlated negatively with play behavior.

Stanley, Murray and Stein (2004) found that depressed mothers are generally less positively responsive and have higher frequencies of infant-negating sequences. They compared 72 mothers with major depression (DSM-III) with 50 dyads with a healthy mother. The infants were 2-3 months old. Essentially, the authors compared

both groups in a variety of interaction parameters, such as infant engagement with the mother or the environment, infant protest or avoidance of the mother. Additionally, sequences of infant action and maternal affirmation were defined as a measure of contingency and were corrected for baseline spontaneous frequencies. Other classical behaviors were also obtained, such as a smile, frown, pout, and yawns, or positive, neutral, or negative vocalizations, crying, or return of the mother's gaze. Interobserver reliabilities were taken via double coding of interaction sequences and may be regarded as satisfactory, since Cohen's kappas ranged from 0.67 to 0.75. At the infant age of 2 months, depressed mothers were found to be less positively contingent. Moreover, a higher rate of contingently negating the infants' behavior was observed. Interestingly, confounders such as social class, infant gender, or chronicity of depression did not modify the association between depression and responsivity. After 3 months, interactional measures were recorded in the still-face paradigm where no differences between depressed and healthy dyads were found.

Cohn, Campbell, Matias and Hopkins (1990) compared 24 depressed and 22 non-depressed mothers in interaction with their 2-month old infants. Depression within the last month was diagnosed using standardized interviews. Mother-infant interactions were videotaped in a face-to-face play of mother and her infant with the infant sitting in a reclining chair. Measures were then derived by coding the videotape using measures of interaction developed by Cohn and Tronick (1987, such as affective expressions or gaze behaviors of mother and infant. Additionally, expressions of mother and child were scored: "negative" (e.g., sadness, maternal intrusion), "look away", "attend", "low positive", "high positive" (facial and vocal expressions as well). Both video-coders were blind to diagnosis with an excellent agreement ranging above a Cohen's kappa of 0.8. Contrary to the title of the study ("... interactions of postpartum depressed ..."), both groups were stratified by gender and according to working and non-working status of mothers; resulting in a complete change of study design with one of the subgroups containing 4 subjects. However, results with respect to adverse depression related main-effects were rare, yet several effects in subsamples of non-working depressed mothers were found (e.g., they were more negative than non-working non-depressed mothers and their infants were found to be less positive).

Field, Healy, Goldstein and Guthertz (1990) found divergences in parallel behavior of

a depressed mother and infant, i.e. depressed mothers matched ("paralleled") their infants more frequently on negative states and less frequently on positive states. The conclusions are based on 24 depressed mothers compared to 24 non-depressed mothers with their 3-month-old infants. Depression was identified with Beck's depression inventory using a cut-off above 9 as lower limit for a minor depression. The infant was sitting in a reclining chair face-to-face with the mother and both were videotaped after being instructed to play. The mother was rated on a scale of 1 to 4 for several characteristics such as "anger" (i.e. angry speaking, roughly pulling the child), "disengagement" (neutral affects, no interaction with child), "elicit" (trying to get the infant's attention), and "play" (positive affects, smiles, vocalizations). Similar ratings were used for the facial and vocal expressions and gaze of the child, such as protest, looking away, attending and playing. The mother and her infant were defined as matching if anger in the mother and protest in the child occurred; thus parallel behavior was defined as follows: mother scored in anger while infant protests, mother in disengagement while infant looks away, mother elicits while infant attends. Interrater reliabilities ranged well above a Cohen's kappa of 0.8. Compared to the normal group, depressed mothers were generally found to spend significantly more time being scored angry, were less engaged and spent less time in play. Similar results were found with their infants. Infants of depressed mothers spent more time in protest and less time in play. Depressed dyads were found to have more parallel time of maternal anger while infant protests, disengagement while infant looks away and less time in play. Taken together, the authors interpreted their results as a predominance of negative states, i.e. in the sense of a contagion effect due to reciprocity of negativity in distressed / depressed dyads - an effect similar to that reported in dissatisfied marriages.

The study of Field (1984), in spite of an inclusion of mothers identified by maternal self-report, is regarded as pivotal for the present research since it is cited without exception by all depression-related mother-infant studies published after 1984. In deviation to previous and subsequent studies, Field reported an impressive list of effects associated with maternal depression. She compared 12 mothers receiving a Beck score of above 16 with 12 healthy mothers with a Beck score lesser than or equal to, 4. Mother and infant were videotaped in a laboratory setting where mother and infant interacted face-to-face. There were three phases lasting 3 minutes each: A baseline play situation, another phase in which the mother was instructed to simulate

unavailability to her infant (mother simulated tiredness, being unable to play with child, speaking flat and uninterested with expressionless face and minimized body movement and touch). And there was a final play phase labelled as "reunion". Measures of vocalization, of positive or negative facial expressions, duration of infant looking away, protesting and looking wary (suspicious) were taken (Cohen's kappa of one half of the subjects ranged between 0.83 and 0.97). Field reports an impressive and extraordinarily high number of results, especially in the infant; infants of depressed mothers were found to be affectively different, i.e. to express less positive, and more negative facial expressions, to vocalize less, protest more. They also engaged in less motor activity. On the other hand, mothers had a similar pattern; they had less positive, more frequent negative facial expressions, spent less time looking at the infant and used less tactile stimulation. After the phase of maternal unavailability, infants of depressed mothers had more negative facial expressions, vocalized less and looked away from the mother more frequently compared to infants of non-depressed mothers. This led the author to conclude a kind of generalizing effect in infants of depressed mothers: since they displayed little changes in their behavior, they might be accustomed to an unavailable mother, and develop or mirror a depression-like style of interaction.

Hoffman and Drotar (1991) generally replicated Field's (1984) effect of reduced positivity of depressed mothers and they concluded that depressed mothers have a less optimal interactional style. Their sample, however, was quite small: 11 depressed to 11 non-depressed mothers in a free play situation. The infants were 2-months old. Depression was identified by self-report based on both Beck's depression inventory and Lubin's Depression Adjective Check List. Mothers scoring higher than 10 on both scales were defined as depressed; mothers scoring lower than or equal to 5 were defined as non-depressed. Dependent measures were ratings of maternal affect expression, infant stimulation, ease of interaction in the sense of maternal withdrawal or intrusion, and infant regard in an unconditional and positive way. The authors found that depressed mothers interact less optimally and oscillate between withdrawal and intrusion, and found their infants to be less positive and with reduced affect. The authors concluded that depression may be associated with deficits in both mother and - due to mirroring processes - in the infant, even if mothers were mildly to moderately depressed.

Field, Healy, Goldstein, Perry, Bendell, Schanberg, Zimmermann and Kuhn (1988)

reported that infants of depressed mothers, who were found to be less positive and active, behaved the same way even with other interaction partners. This led Field to conclude that deviant infant behaviors may generalize beyond the depressed mother. They compared 40 depressed with 34 non-depressed mothers with a lower socioeconomic background. The infants were 3-6 months old. Depression was identified with Beck's depression inventory, classifying mothers with a score below 9 as non-depressed and mothers with a score above 12 as depressed. The mothers and their infants were videotaped in a 3 minute face-to-face play followed by a play sequence where the mother was replaced by a stranger. Dependent measures included standards to quantify aspects of mother-infant interaction, such as maternal or infant activity, infant gaze, head turning, facial expressions, vocalizations, maternal imitative behavior and contingent responsiveness to the infant. Both depressed mothers and their infants differed from healthy mothers in almost all ratings. Both were reported to be less active, gaze less, and use less facial expressions and vocalizations, and the infants were found to continue expressing similar low reactions in presence of a stranger. Beyond the well-known reduction in positivity and activity in infants of depressed mothers, Field and colleagues concluded that a depression-like interaction behavior generalizes beyond interactions with the depressed mother.

Field, Sandberg, Garcia, Vega-Lahr, Goldstein and Guy (1985) similarly found deviant indicators of mother-infant interaction in mothers scoring high for depression and having pregnancy problems (e.g., unplanned pregnancy or marital problems). They compared two groups of 12 mothers who scored low compared to 12 high scoring mothers. High scorers had the highest depression self-ratings on the Beck-scale. Mother and child were videotaped during a 10 minute play session and the tapes were rated for classical codes of infant and mother behavior, such as activity, gaze, facial expressions, vocalizations, and for the mother taking initiative and showing responsiveness. The 3-5 month old infants were found to be less active in interaction, to gaze less, and to use fewer facial expressions. Field and colleagues concluded that a simple screening for pregnancy problems may well be predictive for impaired mother-infant interaction.

In a longitudinal and correlational study, Fleming, Ruble, Flett and Shaul (1988) investigated associations between maternal mood and responsiveness. 56 mothers and their infants were observed at 0, 1, 3, and 16 months after birth. Depression was identified using the Mood State inventory. Social support and attitude of the mother,

i.e. feelings of adequacy as a mother, of care-taking, and attachment to the baby were also obtained. The mothers were videotaped while bottle-feeding or nursing their babies and a sequence of 10 minutes was coded for affect expressions, care-taking, vocalizing and orientation toward the infant. The infant was coded for cries, non-cry vocalization and activity. The (highly explorative) statistical model was a hierarchical regression. Maternal mood after birth and self-rated feeling of adequacy, care-taking, and attachment towards the infant were highly and positively correlated. Additionally, the mothers were split according to their mood score before birth. Thus 29 depressed and 28 non-depressed mothers were compared for total time spent in each coded behavior. High scoring depressed mothers were found to score lower on affectionate or engaged contact time. Depression after birth, however, was rarely associated with disengaged behavior, i.e. only after 1 month after birth. No other differences in behaviors were found between depressed and non-depressed mothers. In total, the authors concluded that self-rated and mild depression is associated with reduced feelings and less affectionate behavior towards the infant. However, by 16 months, no differences were found, i.e. depression did not seem to be associated with long-term effects on mother-infant interaction.

Campbell, Cohn and Meyers (1995) published one of the first longitudinal studies to test for cumulative exposure of infants to maternal depression and associated effects on mother-infant interaction. Campbell and colleagues compared 67 depressed mothers with a healthy group of 63 mothers. They were videotaped in interaction in their home at infant ages of 2, 4 and 6 months. Depression was identified through interview based on the Schedule for Affective Disorders and Schizophrenia. Mother and infant were coded as having “neutral”, “positive” or “negative” engagement. No differences were reported with respect to the primary question; depressed compared to non-depressed mothers did not differ, even if infant gender or maternal working status were included in the statistical model. However, 20 mothers with a chronic depression that lasted over 6 months revealed differences compared with 27 women with subclinical symptoms and 19 women who had remitted; the chronically depressed mothers, as well as their infants, were found to be less positively engaging. The authors concluded that a depression itself may not necessarily be associated with differences in interaction, but the distinction between a transient and a chronic depression seems to be a promising predictor regarding deviant mother-infant interaction.

Field, Morrow and Adelstein (1993) coded behavior of the infants both by observers and mothers. Primary interests were differences in association with depression, but the authors also hypothesized deviations of the depressed mothers' ratings of their own infants compared to ratings of independent observers (maternal ratings were hypothesized to be more negative due to negative maternal perceptions). They included 32 healthy and 28 depressed mothers; all were black and had a low economic status. The authors labelled mothers with a score more than 12 on Beck's Depression Inventory as depressed, and mothers lower than 9 as non-depressed. Mother and child were videotaped while playing in a face-to-face play setting for 3 minutes. The videos were coded by observers unaware of group assignment, as well, based on a simple 3-point system of "negative", "neutral" and "positive" and based on the time spent in these behaviors. Comparably to other studies, infants of depressed mothers were found to be more negative and less positively engaged. This was found by both independent observers and the mother. However, depressed mothers coded their own infant more negatively than the independent observer. On the other hand, the non-depressed mothers almost agreed with the observer, whilst depressed mothers rated themselves more positively than the observer. Field and colleagues concluded that depressed mothers may have "distorted" perceptions because they rate the behavior of their own children as more negative and their own as more positive.

Livingood, Daen and Smith (1983) compared 2 groups of 25 postpartum depressed and 25 healthy mothers and tested for the level of maternal stimulation and quality of stimulation, e.g., unconditional positive regard. Livingood and colleagues labelled mothers with a Beck depression score of more than 10 as depressed. The usability of the study, however, is limited because depression was quantified 2-3 days after delivery, which increases the risk of assessing postpartum blues instead of depression. Observers rated maternal unconditional positive regard and measured the time the mothers stimulated their infant during feeding sessions. Gaze, vocalization, and touching the infant were coded. Surprisingly, depression was not predictive for differences in maternal stimulation behavior. Depressed mothers, however, scored lower on unconditional positive regard.

Cohn, Campbell and Ross (1991) reported that infant reactivity to maternal still-face differed in infants of depressed compared to control infants. Moreover, negative reactivity in the still-face period predicted a less secure attachment in infants of

depressed mothers. Cohn et al. compared 33 women with postpartum depression compared to 33 healthy women at 2, 4, 6 and 12 months. Depression was identified via interview using the Schedule for Affective Disorders and most of the depressed mothers (86%) were classified as having a major depression. Mothers and their infants were videotaped at home for a 3 minute phase of play and a still-face phase of 2 minutes duration. It was hypothesized that a high eliciting frequency of the child in a still-face situation at the age of 4 or 6 months predicts higher attachment behavior after 12 months. Coders unaware of the maternal depression status coded infant eliciting behavior and facial and vocal affective expressions and direction of gaze. Negative eliciting of the infant was merely counted if the infant grimaced or cried, or counted as positive eliciting if the infant smiled or intended to play during the still-face period. Attachment was quantified using a stranger sitting face-to-face to the child instead of the mother. The children were classified with respect to attachment quality by two observers as avoidant or secure. The authors ran logistic regressions for each point of measurement (2, 4, or 6 months) to predict attachment classification at the 12th month using the infants' eliciting behavior. Cohn and colleagues reported results for the whole group only, i.e. that positive eliciting behavior of 6 month olds predicted secure attachment for 12th month, but not negative eliciting behavior. Although the groups were formed via clinical interview, clear-stated results with respect to differences between depressed and non-depressed mothers could not be found in the paper.

Field, Healy and Leblanc (1989) reported that dyads with a depressed mother shared negative states more often and positive states less often. The study is based upon 7 depressed and 9 non-depressed mothers only, using the Beck Depression Inventory with a cut-off score of 9, videotaped in a face-to-face play setting. Sharing states were defined if mother and infant displayed similar states, such as mother in anger and infant protesting, or, mother disengaged and infant looking away, mother eliciting and infant attending, and finally, both interacting positively. Field and colleagues found that depressed dyads showed parallel behavior much more frequently in maternal anger while the infant was protesting, more disengagement while the infant was looking away, and rated less positively while playing. Depressed mothers were reported to be generally less positively in their play and to display more anger and disengagement, while infants of depressed mothers generally protested more. The authors concluded that dyads with a depressed mother share negative states more

often and positive states to a lesser degree. Field et al. interpreted this as a transmission effect of maternal negativity to the infant; either a contagion effect of negative mood or an effect of reciprocity in negativity - similar to that reported in distressed and dissatisfied marriages.

Zlochower and Cohn (1996) found that maternal depression was predictive for longer and more variable pauses in vocalization with their infant. The authors compared 15 depressed and 20 non-depressed mothers in measures of vocal expressions. The infants were 4 months old. Depression was identified using Spitzer's Research Diagnostic Criteria. Chronic depressed mothers, i.e. longer than 6 months, especially had longer and more variable vocalization onsets compared to non-depressed mothers. The author concluded that depressed mothers may fail in their vocal coordination and are thus less responsive and less predictive in their behavior for their infant. All of this may contribute to asynchronous interaction between depressed mothers and their child.

Campbell, Cohn, Flanagan, Popper and Meyers (1992) found that depressed mothers were less positive and engaged more negatively with their infant. They scored maternal affect, engagement and responsiveness in dyadic interaction of 70 depressed and 59 non-depressed mothers. Participants were visited at home after 2, 4, and 6 months after birth and were videotaped. Depression was identified using Spitzer's Research Diagnostic Criteria and was repeatedly assessed at 9, 12, 18 and 24 months. Depressed women were rated as showing less positive and more negative affect; they were more negative with their infants. However, overall social behavior ratings did not differ to those of normal control mothers. Depressed mothers reported a higher feeling of being overwhelmed and rated their infant as having a higher temperament. Depression severity, however, did not predict a reduction in maternal positive engagement or infant social behavior (contrary to previous episodes, delivery or pregnancy complications, bad spousal relationships, "not being prepared for the baby", or missing help by the spouse). Depression chronicity of 6 months, however, was best predicted by 2-month infant negativity and limited spousal support.

Weinberg and Tronick (1998) found that maternal depression may be predictive for impaired parental behavior even after remission. They compared 30 normal control mothers with 30 mothers with major depression. Diagnoses were assigned via

Structured Clinical Interview for DSM-III Axis I Disorders. The majority of the mothers were given medication. Mothers and infants were videotaped in the laboratory when the infants were 3-months old. Behaviors and facial expressions were coded using Tronick and Weinberg's Infant and Maternal Regulatory Scoring System. When the mothers appeared in the laboratory, the clinical group did not differ compared to control subjects either on depressive symptomatology (using the CES-D), or in maternal self-esteem (using the Maternal Self-Report Inventory). Nonetheless, Weinberg and colleagues found that formerly depressed mothers talked less with their infants, touched less, were rated as more invasive and disruptive and showed more anger towards their infants. Their infants were less interested, expressed more anger and sadness, tended to fuss and cry more and vocalized less with the stranger. The authors concluded that even though the mothers have been in treatment and symptom-free, social and functional disadvantages of depressed mothers persisted.

Murray, Hipwell, Hooper, Stein and Cooper (1996) found that maternal depression was associated with heightened insensitive behavior which was found to predict later infant impairments in several cognitive indicators. Depression was identified based on the Edinburgh postnatal depression scale and Spitzer's Research Diagnostic Criteria. 58 depressed and 42 control mothers were selected. A subsample of 29 depressed mothers and 20 controls were analyzed at an infant age of 2 months in a face-to-face interaction play of 5 minutes with the mother, followed by 5 minutes with a stranger. The mothers were rated for sensitivity and responsiveness, acceptance, warmth and engagement with her infant. The infant was rated for positive active engagement, attention, vocalizations, affect and for distressed behavior. The study generally aimed at performance deficits in children of depressed mothers and deficits mediated by maternal parenting behavior. Measures of child performance were taken, such as Piaget's tasks of object performance, the Bayley Scales of Mental Development and the McCarthy Scales of Children's Abilities at infant ages of 18 and 60 months. Interestingly, Murray and colleagues found no effect of depression on measures of infant performance, nor did she report direct effects of depression on 2-month interactional measures. Only general variables such as maternal insensitivity, low infant stimulation and low social class predicted poor child outcome at the age of 5 years.

The study by the American National Institute of Child Health and Development

(NICHD, 1999) found that mothers with chronic depression were rated as having lower sensitivity when interacting with their child. After 3 years, their children were found to score lower on measures of language performance and child cooperation. 1215 mothers were longitudinally followed at 6, 15, 24 and 36 months after delivery. Healthy, depressed and chronic depressed mothers were compared using videotaped play sessions of 15 minutes. Maternal sensitivity was operationalized based on ratings of supportive presence, positive regard, and intrusiveness or hostility. Child behavior was rated for negative and positive mood. Depression was identified via maternal self report using Radloff's Centre for Epidemiological Studies Scale. The 5 points of measurement were used to classify the mothers according to their depression score: 663 having never been depressed, i.e. at all points of measurement below 16 points, 460 as sometimes depressed, i.e. 1 to 4 points at 16 or above, and 92 as chronically depressed (4 or 5 times at 16 or above). With respect to measures of mother-infant interaction, no differences were found, even if demographic variables were accounted for. However, children of depressed mothers had lower school readiness and verbal comprehension scores. Children of chronically depressed mothers had lower scores of language expression, lower scores of cooperation and higher problem scores. If maternal sensitivity was included, depression-related effects were lower. This led the authors to conclude that maternal sensitivity may moderate (i.e. in parts be predictive for) the effects on children.

Pickens and Field (1993) found that maternal depression was predictive for lower interest, sadness and anger in infants. They compared 27 depressed (scoring ≥ 13 according to Beck's depression inventory), 33 non-depressed (scoring between 3 and 10), and 24 low scoring subjects (scoring between 0 and 2). The infants and their mothers were videotaped during a 3 minute play session. Codes for facial expressions of the infants were assigned for interest, joy, anger, distress, sadness, surprise, and gaze-aversions. Maternal expressions were assessed for negativity and positivity using Fields' Interaction Rating Scale. Infants of depressed mothers were found to spend less time showing interest, and, more sadness and anger compared with infants of non-depressed mothers. The authors concluded that the heightened infant anger may indicate that depressed mothers are distressed, whereas the low infant interest results from the lowered maternal interest.

Contrary to expectation, Hops, Biglan, Sherman, Arthur, Friedman and Osteen (1987) found no heightened aversive interactions in families with a depressed parent

(refer to the beginning of the discussion chapter), even though the depressed individual expressed high rates of dysphoric affect. 27 families with a depressed mother were compared to 27 control families without any parent who was classified as depressed. In home observations affect codes were assigned, e.g., for caring, happiness, irritation, sarcasm, or whining. Children of depressed mothers were found to have higher irritability, whilst depressed mothers were scored as being less caring.

Hossain, Field, Gonzalez, Malphurs and Del Valle (1994) found maternal depression to be predictive for lower "interaction ratings" and interactional difficulties. The study included 26 depressed mother-infant pairs compared with 14 non-depressed dyads. Depression was identified with a Beck score above the cut-off of 12. All participants in the study were of lower socio-economic status. The 3-6 month old infants were videotaped in face-to-face play for 3 minutes. Mother, father and infant were observed in pairs and scored for physical activity, gaze, facial expressions and vocalizations. This study is one of the rare studies which included fathers. Although control mothers and fathers did not differ, differences between depressed mothers compared with fathers were found. Differences between depressed and non-depressed mothers, however, were not addressed with statistical tests but the descriptive statistics revealed few to zero descriptive differences of non-depressed mothers in measures of interaction.

Lyons-Ruth, Zoll, Connell and Grunebaum (1986) found increased scores of hostility toward the infant when observing mother and infant at home. Mothers were coded for sensitivity, warmth, verbalizations, physical contact caretaking, hostility, and disengagement. The one-year old infants were coded for security of attachment and verbal and motor development. Interestingly, the study found that maternal intelligence, irrespective of depression, explained most of the variance of maternal sensitivity and warmth, whereas the maternal depression, although self-reported, predicted neither maternal sensitivity nor warmth during interactions. However, maternal depression, intelligence and verbal communication scores predicted infant motor and language development scores at one year. Higher depression scores predicted lower infant development.

Panaccione and Wahler (1986) found that maternal depression predicted aversive behavior in interactions with the child. The study included 33 mother-child pairs with low socio-economic backgrounds. Maternal depression was identified using the Beck

Inventory. Mothers and their 4-year-old children were observed at home using a standardized coding system for facial and verbal behavior, for violations of home rules, maternal instructions, child non-compliances, verbal complaints and protest of the child, for maternal positive facial affect or verbally expressed affect, for social aversive behavior such as shouting, or even slapping. Panaccione and Wahler reported that higher depression scores correlated with high maternal aversive behavior, i.e. expressions of aversion or antagonism to the child. Child behavior was found better predictive for subsequent maternal behavior. Maternal ratings did not correlate with infant behavior but were best predicted by maternal depression.

Breznitz and Sherman (1987) found maternal depression predictive for impaired verbal interactions with 3-year old children. They included 14 depressed and 18 non-depressed mothers and observed verbal interactive behavior. Depression was identified with Spitzer's Research Diagnostic Criteria. All dyads were observed in unstructured interaction during play and normal family routine time in a homelike apartment in the researchers' laboratory. Depressed mothers vocalized less, and responded less quickly. Their infants were found to speak less and to make lengthier vocalization pauses compared with children of healthy mothers. Deficits in the verbal behavior of children of depressed mothers were interpreted as an adaptation to the maternal level of motor retardation, reduced energy, social withdrawal, and a lack of reinforcement resulting from the non-welcoming mother.

Radke-Yarrow, Nottelmann, Belmont and Welsh (1993) found correlated affects between mothers and infants. Dyads with a depressed mother spent more time in negative affects with heightened synchronous negativity. The children were 1.5 - 3.5 years old. Maternal depression was identified using Spitzer's Research Diagnostic Criteria. The mothers' and children's affects were coded for sadness, anxiety, irritability and anger, negativity, or joy. Unipolar depressed mothers were found to express more overall negative affect towards their children over time, and their children spent comparable time in negative affects. Maternal and child affects were significantly correlated. Expressions of maternal negativity were much more extended in unipolar depressed mothers or mothers with increasing depression severity.

Stein, Gath, Bucher, Bond, Day and Cooper (1991), in a high-ranking published article, reported a generally lower interaction quality in dyads with a depressed mother. Stein and colleagues compared 49 mothers with depression in the first

postpartum year with 49 healthy control mothers. Depression was identified using the Present State Examination; a standardized interview. The infants were tested at 19 months when their behavior was recorded in a structured play situation. Codes were assigned for verbal and nonverbal interactions, and for facilitating activities, e.g. activities for child interest maintenance. Codes were also given for affective sharing. Dyads with a depressed parent were found to have lower affective sharing, lower interaction quality and a less facilitating mother. Those effects were more pronounced if the mother showed a chronification of depression. The children were found to show less interest and have stronger negative responses. Interestingly, chronic social and marital difficulties, together with postpartum depression, were found to predict reduced interactional quality. The predictive weights of social and marital difficulties were higher than those of depression. Moreover, indicators of reduced interactional quality were found to persist even after the depression remitted.

Gordon, Hammen, Adrian, Jaenicke, Hiroto and Burge (1989) included in their study 12 mothers with chronic or recurrent unipolar depression (yet with both conditions already treated), compared to 12 mothers with bipolar disorder and a medically ill control group, i.e. hospitalized or ill for non-psychiatric reasons. Depression was identified via interview, i.e. with Spitzer's Research Diagnostic Criteria. Mother and child were observed during unstructured verbal interaction, specifically a discussion of a topic of mutual disagreement. Although the children were older than those previously reported (8-16 years), the study adds to the knowledge that unipolar mothers in particular were the most negative of all groups, behaved disconfirmatory, in a critical manner and preferably used low productive comments.

2.10. Summary and methodological critique with respect to the predictive value of maternal depression in indicators of mother-infant interaction

The majority of the studies above suggested a detrimental effect of maternal depression on both maternal and infant behavior. A minority of studies failed to report effects (e.g., Campbell et al., 1995; Cohn et al., 1991; to a major extent the studies of Hops et al., 1987; Livingood et al., 1983; and with respect to interaction raw data, Stanley et al., 2004, page 11). Interestingly, the latter studies defined depression with a clinical interview, in contrast to studies with large depression-associated differences (e.g., Field, 1984) that identified depression via maternal self-report.

Nevertheless, several dyadic interaction indicators differentiated between depressed

mothers-infant dyads and healthy control dyads: depressed mothers were found having a negative interaction style, i.e. they were more non-responsive to their infant, disengaged, and displayed lower positivity and higher negativity in general. They were even intrusive, with less facial expressions. They were found to spend less time playing with their infants when instructed to do so, and more time in anger (Field et al., 1988). Depressed mothers were also found to show less unconditional positive regard or to facilitate less (Stein, Gath, Bucher, Bond, Day and Cooper, 1991), to behave in a disconfirmatory and critical manner, and to be asymmetrically contingent, i.e. they behaved less positively and shifted to a negative-contingent responsiveness (Stanley, Murray and Stein, 2004). There are additional findings that maternal negativity correlates with infant negativity (Cohn, Campbell, Matias and Hopkins, 1990).

The infants were found to be less active, use less vocalizing or to even speak less frequently, and to be less contingent responsive; their positive affective behavior also appeared less parallel to maternal behavior (e.g., Field, 1984, 1988, 1985, 1989).

Accordingly, both interactants were found to be negative in a synchronous manner Radke-Yarrow, Nottelmann, Belmont and Welsh (1993) and to spend more time in matching their affective states for anger / protest, and disengagement / looking away, and spend less time in play as compared to healthy mothers and their infants.

However, there are also a few null findings, i.e. studies that reported that none of those differences above occurred (e.g. Campbell, Cohn and Meyers, 1995; Cohn, Campbell and Ross, 1991; Hoffman and Drotar, 1991; Hossain et al., 1994); Stanley, Murray and Stein, 2004; Weinberg et al., 2008)

Taken together, however, the association between depression and parameters of dyadic interaction in studies seems to be well supported in the majority of findings.

There are, however, several critical points, a few are listed in advance (also refer to the chapter “deduction of hypotheses”): From a methodological point of view it can be stated that the studies listed above are quite heterogeneous, with respect to primary target parameters, coding systems, samples included, or statistical methods applied. This variety of indicators, designs, and test statistics clearly eliminates the possibility of an application of meta-analysis methods (Hedges and Olkin, 1985; Sutton et al., 2000).

Regarding the primary target parameters, a range of operationalizations were used to quantify “dyadic interaction”. This raises the question whether dyadic interaction can be regarded as a homogeneous construct. Some operationalizations include maternal responsiveness, maternal or infant engagement, overall maternal positivity, infant negativity, and maternal intrusion. Additionally, within single parameters, different descriptors were used, e.g., behavior durations, frequencies or conditional probabilities of behavior sequences (even within the same group of researchers, e.g., the Field group or the Oxford-group; Stanley, Murray and Stein, 2004). In the majority of studies, the characteristics were observation-coded or rated by independent observers (e.g., the group around Campbell et al., 1992, 1995) based on the assumption that a depressed mother may be biased with respect to child descriptions (refer to the discussion of maternal information bias due to depression by Field, Morrow and Adelstein, 1993; Field, 1992; De Los Reyes and Kazdin, 2005; Youngstrom, Izard and Ackerman, 1999).

Moreover, quite different coding systems were used, either self-constructed or ad-hoc systems (Bettes, 1988; Field, 1984; Cohn and Campbell, 1992), with unpublished psychometric criteria. Those studies predominantly reported interrater concordance (this, however, can only be used as a measure for objectivity and further psychometric properties such as reliability or validity were omitted in these reports). Coefficients of the frequently used interaction coding system of Tronick are missing (Tronick's Monadic Phase coding system, Tronick, Als and Brazelton, 1980; Weinberg and Tronick, 1998). Practically none of the publications included behavioral indicators for interaction based on reference values, such as population referenced cut-off values or standardized measures. In some rare cases, standardized coding systems were used (Hops et al., 1987) and psychometric criteria were given.

Furthermore, the studies are well heterogeneous with respect to sample characteristics: sometimes only highly selected participants, e.g. with low socio-economic status were included (Field, Healy and Leblanc, 1989; Field, Morrow and Adelstein, 1993). There are some tendencies to use post-hoc stratifications with respect to gender without any control of statistical decision errors (Cohn et al., 1990).

A range of studies that defined depression via self-report measures selected participants from community samples. Depression groups formed by clinical interview (13 of 29 in table 3 on page 43) are in the minority and in most cases a two-stage

strategy was applied, i.e. mothers who were pre-selected by a depression self-report measure were then included based on a clinical interview. It is well-known that the depression screenings, e.g., BDI or CES-D, provide high specificity (true negative results, i.e. confirmed by clinical interview) but low sensitivity (low rate of depression cases verified by clinical interview). Thus group-forming by depression self-report screenings (e.g., BDI or CES-D) seems to be a critical issue. For example, the sensitivity of the Beck scale has been reported 57 - 95% (Meakin, 1992), thus the grouping may not always reflect clinical depression. Given the misclassification rates of 6% false positive and 36% false negative predictions of the self-report screening test (CES-D) reported by Myers and Weissman (1980) only a very low sensitivity of 64% (Bayes formula, Bortz, 2005) and a positive predictive value of 41% may be concluded. Both the sensitivity of 64% and the positive predictive value of 41% support Myers and Weisman's conclusion of a critical relationship between self-report and diagnosis of depression. Current screening measures, such as the Hospital Anxiety and Depression Scale (HADS), the WHO (five) Well Being Index (WBI-5), and the Patient Health Questionnaire (PHQ) are reported with high sensitivities (Loewe et al., 2004; Doering et al., 2007).

The heterogeneity also refers to the stability of results. The above-mentioned list of preferred indicators of dyadic interaction is not a constant. Nevertheless, heightened negativity, reduced positivity, engagement and responsiveness appear to be the most reported results.

2.11. Empirical evidence suggesting that parent-child interactions mediate between maternal depression and adverse child effects

The idea that parenting behaviors may work as mediating process between a maternal depression diagnosis and adverse child outcomes was based on the observation of dysfunctional interactions in dyads with a depressed mother, and, of consistent observations of heightened risk in children of depressed mothers (e.g. developmental risks such as cognitive impairments, deviations from normal attachment, difficult infant temperament or externalizing child problems, for extensive reviews see Cummings and Davies, 1994; Downey and Coyne, 1990; Goodman and Gotlib, 1999; and, Goodman, 2007).

Partly based on the critique of genetic pathways as major pathways for transmission (refer to page 17), current research has focused on the idea that being exposed to

behavior of a depressed mother, to her negative affect, her parenting and cognitions might be a predictive link between maternal depression and child risks.

Two studies in particular may be considered as pivotal for this research with respect to the question of how parenting may mediate offspring depression: This is Johnson, Cohen, Kasen, Smailes and Brook (2001) and Bifulco, Moran, Ball, Jacobs, Baines, Bunn and Cavagin (2002). Both are summarized in table 4 below and in greater detail on page 69; all currently available studies, which put several mediating mechanisms under test, are listed in table 4.

Table 4 Studies with a focus on parenting as mediator between maternal depression and adverse child outcome (modified according to Goodman, 2007)

Author	Sample	Suggested mediator	Primary target parameter	Main results
Johnson, Cohen, Kasen, Smailes and Brook (2001)	593 families followed over 18 years.	Maladaptive parental behavior, e.g., inconsistent enforcement, low amount of time spent with the child, low affection displayed, poor communication and harsh punishment.	Prevalence of child psychopathology	Maladaptive parenting was found to mediate between parental psychiatric disorder and child psychopathology. The effect was not specific for depression.
Bifulco, Moran, Ball, Jacobs, Baines, Bunn and Cavagin, (2002)	276 mothers and their children.	Measures of parenting quality, i.e. offspring neglect or abuse.	Risk of psychiatric disorder.	Dysfunctional parenting, i.e. offspring neglect or abuse, mediated between maternal depression and the risk of a psychiatric disorder.
Burt, VanDulmen, Carlivati, Egeland, Sroufe, Forman, Appleyard and Carlson, 2005	184 families; mothers and children assessed at 4, 6, 7, 8, 16 and 18 years.	Measures of the home environment (e.g., emotional responsivity, emotional climate, provision for active stimulation), and measures of family functioning (conflict, lack of constructivism).	Symptom scores for child psychopathology and child behavior problems.	Home environmental measures and intra-family conflict scales mediated between maternal depression and child psychopathology.
Davies and Windle (1997)	443 adolescents and their mothers; three follow-ups at ages of 16-17 years	Measures of discord, e.g., high rated distress, conflict, low intimacy and satisfaction.	Conduct problems (scale for delinquent activity) and adjustment.	Family discord as strong mediator between maternal depression and adolescent conduct disturbances or, to a lesser degree, adolescent depression.
Lundy (2002)	15 parents, infants 6 months.	Synchrony in mother-infant interactions.	Quality of mother-infant attachment.	Maternal depression was found to be linked to reduced infant attachment mediated by mother-infant synchrony.
Snyder (1991)	10 families, children 4-5 years.	Harsh, inconsistent, or ineffective discipline.	Conduct problems.	Negative disciplinary tactics mediated between maternal negative mood and child conduct problems.
Ghodsian, Zayicek and Wollcind (1984)	131 mothers and their children assessed at 4, 14, 27, 42 months.	Frequency of physical punishment.	Child behavior problems.	Association between depression and child problem scores was found. It was reduced if child punishment was taken into account, thus suggesting a mediating effect of punishment.

Author	Sample	Suggested mediator	Primary target parameter	Main results
Murray, Kempton, Woolgar and Hooper (1993)	59 mothers, infants assessed at 2 months.	Maternal speech characterized by negative affect and lack of focus on the infant.	Cognitive development at 15 months.	Quality of mother-infant communication mediated the link between depression and child performance at 15 months.
Goodman and Brumley, 1990	25 depressed and 23 control mothers, children assessed at 3 & 60 months	Mother-child interaction quality (e.g., affect, responsiveness, stimulation).	Child IQ and social competencies.	Parenting practices, and not diagnosis, predicted child outcome.
Hamish, Dodge and Valente, (1995)	376 mothers and children at 6 year	Mother-child interaction quality (e.g. maternal sensitivity, responsiveness).	Externalizing problems.	Mother-child interaction quality (sum score of joy in interaction, maternal sensitivity, responsiveness, maternal directness, involvement, command clarity and child compliance) was found to partially mediate child externalizing problems.
McCarty and McMahon (2003)	224 mothers and children at 11-12 years.	Cold, hostile, or difficult mother-child relationship, lack of maternal social support.	Disruptive behavior problems.	Difficult mother-child relationships were reported to mediate externalizing child behavior. Lack of maternal social support was found to mediate internalizing behavior.
Hammen, Burge and Stansbury (1990)	64 mothers and children aged 8-16 years.	Overall positivity or constructivism in interaction.	Child behavior problems.	Maternal depression, low positivity and low child positivity in interaction predicted an unfavorable child outcome, such as child behavior problems and low social competence.
Zahn-Waxler, Iannotti, Cummings and Denham (1990)	22 depressed and 22 healthy mothers and children at 2 and 5 years.	Deficits in child rearing practices (lack of promotion of social behavior, warmth, or lack of modulated control).	Child externalizing behavior.	Children of depressed mothers were more likely to have maladaptive, dysregulated behavior. Dysregulated 2-year behavior in interaction with maternal diagnosis was found to predict externalizing behavior when the children were 5 years old. Depressed mothers who used so called "proactive parenting" had a lower likelihood of having children with externalizing behavior.
NICHD (1999)	1215 mothers and children assessed at 1, 6, 15, 24, 36 months.	Maternal sensitivity.	Infant performance.	Scores for school readiness and verbal comprehension.
Conger, Patterson and Ge (1995)	75 families and children at 12 years.	Disrupted parental discipline practices.	Child adjustment problems.	The link between depression and child adjustment problems was mediated by poor maternal discipline practices.
Laucht, Esser and Schmidt (2002)	22 children of postpartum depressed mothers and 116 control children assessed at 3 months and 2, 4, 8 years.	Disturbances in the mother-child interaction (low vocal activity, low reactivity and low warmth).	Child performance and externalizing behavior.	Depressed mothers with low vocal activity, low reactivity and low warmth had externalizing and lower performing children.

Johnson, Cohen, Kasen, Smailes and Brook (2001) focused on the role of so-called maladaptive parenting as mediator between parental psychiatric disorder and child psychopathology. They observed 593 families over 18 years. Parental and child disorders were identified via structured interview to meet DSM-IV criteria. Maladaptive parenting was quantified via structured interview and referred to the use of negative parenting, e.g., low amount of time spent with the child, low affection displayed, harsh punishment, inconsistent enforcement and poor communication. Johnson and colleagues found that these behaviors were associated with a heightened risk of psychiatric disorder in children. In particular, depression and other parental disorders (anxiety, personality or substance abuse disorders) were positively associated with high prevalence rates of maladaptive parenting. Even in the absence of a parental disorder, the presence of maladaptive parenting was found to favor high risks of child psychopathology, such as depression, anxiety, personality or substance abuse disorders. Johnson and colleagues concluded that both conditions suggest a mediating model, i.e. depression favoring maladaptive parenting and, on the other hand, maladaptive parenting favoring heightened child risks. Moreover, the model was not reported to be restricted to depression only. Other parental disorders were associated with comparable risks of child psychopathology too, thus pointing to an unspecific association; Johnson and colleagues concluded that parental disorders may serve as a marker for maladaptive parental behavior which in itself increases child risks (but not necessarily the disorder itself).

Burt, VanDulmen, Carlivati, Egeland, Sroufe, Forman, Appleyard and Carlson (2005) contributed to the question of how parental psychopathology may be transmitted to offspring. They included 276 families, assessed depression via self-report, i.e. Beck and CES-D scales, included observation scales of parenting and family environment (HOME scale), family functioning measures (Self-Report Family Inventory), child behavior problems (Child Behavior Checklist), and finally, they assessed child psychiatric symptoms via structured clinical interview (K-SADS). Children were assessed at the ages of 4, 6, 7, 8, 16 and 18 years. The data pointed to a mediation model, yet restricted to males only; moderate correlations ($r \approx 0.3$) were found between depression measures and both intra-family conflict scales and child psychopathology symptom scores. Regression models based on a classical test for mediation (Sobel's test) indicated that family conflict scores mediated between maternal depression and child behavioral problems or child psychopathology. Thus

child behavioral problems and child psychopathology appeared to have a higher predictive value in the case of family conflict, which itself seemed to be favored by maternal depression. On the other hand, the absence of family conflict or negative parenting behavior appeared to reduce both child behavior problems and the risk of child psychopathology.

Bifulco, Moran, Ball, Jacobs, Baines, Bunn and Cavagin(2002) assessed maternal psychiatric disorders and disorders of the offspring in 276 mothers and their children. Mothers with poor social function or low self-esteem were selected and were compared to mothers without. Aspects of parenting such as neglect, antipathy, control and abuse were assessed based on interviews, as well as maternal and child psychopathology. Children of a group of “vulnerable” mothers (e.g. of mothers with depression) were reported to have a fourfold risk of a disorder. The combined effects of maternal depression and inadequacy of caretaking, as well as poverty were best predictive for heightened risks of child disorders. Maternal depression alone was not predictive because similarly to Johnson et al. (2001) the effects were found to be completely mediated by parenting, i.e. offspring neglect or abuse mediated between maternal depression and the risk of a psychiatric disorder.

Davies and Windle (1997) reported an association between maternal depressive symptoms and adolescent reports of depressive symptoms and child conduct problems. Although the study is correlative only, self-reported family discord was reported to mediate between maternal depression and emotional adjustment. The results, however, were restricted to a subsample of adolescent girls aged 16-17. Davies and Windle included 443 adolescents and their mothers in their study, and assessed them in three 6-month follow-ups according to maternal depressive symptoms (CES-D), measures of family discord (Family Cohesion Scale and Marital Satisfaction Scale), e.g., high rated distress, conflict, low intimacy and satisfaction, as well as conduct problems (Scale for delinquent activity) and adjustment (CES-D). Family discord was reported to be a strong mediator between maternal depression and adolescent conduct disturbances and, although to a lesser degree, adolescent depression. On the other hand, if family discord was partialled out, this markedly lowered the association between maternal depressive symptoms and offspring adjustment (depression, risk for delinquency and alcohol problems). Moreover, Davies and Windle explicitly tested if maternal depression had a direct effect on offspring problems. However, the partialling out of maternal depression scores left

the link of family discord and child problems almost unchanged and indicated that parental depression may be only a distal marker of offspring problems.

Lundy (2002) tested the question of whether measures for mother-infant synchrony, e.g., for positivity, mediated the link between maternal depression and the classification for infant attachment. Lundy included 15 parents and their 6 month old infants. Depression was assessed by self-report (CES-D). The frequencies of parent-child synchrony were derived from videotaped interactions. Attachment quality was assessed via parental self-report when the infant was 13 months old. Maternal depression was found to be negatively associated with infant attachment quality, whilst marital satisfaction was found to be positively associated. Based on a classical model for the test of mediation, a hierarchical regression model, maternal depression was found to be linked to reduced infant attachment via mother-infant synchrony. Thus high mother-infant synchrony predicted secure attachment. Maternal depression itself predicted low mother-infant synchrony frequencies, but the effect of maternal depression on infant attachment was found to be mediated by mother-infant synchrony.

Snyder (1991) reported that the link between maternal mood (depression, hostility, anxiety) and child conduct problems was mediated by maternal disciplinary practices. The children were 4-5 years old. The study included 10 mother-child pairs. Conduct problems were assessed based on the Child Behavior Checklist. Maternal distress was assessed via self-report (Family Event List and Multiple Affect Adjective Checklist), and maternal discipline via a videotaped interaction in the investigator's laboratory. Codes were assigned for reciprocate maternal behavior either reacting to preceding aversive child behavior or for cessation of aversive maternal behavior although the child behaved aversively. Conduct problems were derived from the videotaped interaction by counting child aversive behavior. According to the sample selection, reciprocate aversive (39%) but also cessation of behavior (49% of observation time) were frequent. Moreover, based on structural equations, Snyder concluded that negative disciplinary tactics mediated between maternal negative mood and child conduct problems.

Ghodsian, Zayicek and Wollcind (1984) investigated the link between depression and conduct problems. They assessed 131 children at 4, 14, 27, and 42 months after birth and their mothers. Child conduct problems were assessed with a self

constructed rating scale and maternal depression with a psychiatric interview. Ghodisan and colleagues found heightened problem scores in children of depressed mothers. Moreover, child problem scores were higher if they were preceded by previously assessed maternal depression. Depressed mothers were found to use physical punishment more frequently (however, only at 27 months), which was associated with high non-compliance, followed by heightened child problem scores. However, high children problem scores did not appear to precede occurrences of maternal depression. The association between depression and child problem scores was found to be reduced if child punishment was taken into account, thus suggesting a mediating effect of punishment with respect to the link of maternal depression and child problem behavior.

Murray, Kempton, Woolgar and Hooper (1993) included 29 mothers with one postpartum depression episode, identified via interview; 20 healthy mother-infant pairs and 10 mother-infant pairs with a depression history. The infants were 2 months old at the first assessment where measures of vocalizations, such as length and incidence of repetitions were taken. The infants were observed during play interactions. At 9 and 18 months, cognitive measures were taken (e.g., the Bayley, 1969). Murray et al. found the speech of postpartum depressed mothers to be more negatively affected, less infant-focused and less engaged, as compared with healthy mother-infant pairs. Mothers with a depression history were found comparable with controls. Based on regression analyses, Murray and colleagues concluded that the level of infant-focused speech when the infant was 2 month old mediated between maternal depression and the infant's cognitive index when the infant was 18 months old.

Goodman and Brumley (1990) found that mother-child interaction mediated between maternal diagnosis and child social and performance indicators. They included 25 depressed, 53 schizophrenic, and 23 healthy control mothers. Diagnoses were assigned by interview according to DSM-III. Assessments were made when the children were 3 months and 5 years old. Mother-child interaction was quantified with a standardized interaction rating system e.g., for affective quality (joy, anger, tenseness) and mutuality (reciprocity). The child's home was assessed for emotional and verbal responsivity and stimulation level, and the children for IQ and social behavior. Schizophrenic mothers were found to have the lowest score on affect involvement and responsivity compared with healthy mothers. Depressed mothers'

scores lied in between both groups. The same pattern occurred for the scores of child-rearing environment (e.g., maternal responsiveness and provision of child stimulation); i.e., the lowest values for schizophrenics, the highest for control mothers and mothers with depression were in between. Maternal affect and involvement during 3-month interaction predicted the 60-month child IQ as did maternal diagnosis, but the indicators of parenting quality, especially responsiveness, had higher predictive values, i.e. indicators explained more child variance of IQ and social behavior, whereas an extensive list of covariates, e.g., maternal education, infant age, and disorder severity, were not predictive.

Harnish, Dodge and Valente (1995) reported that the quality of mother-child interaction together with socioeconomic status mediated between maternal depression and child behavior problems or child externalizing behavior. 386 mother-child pairs were included, in which the children were 6 years old. Externalizing behavior was rated by teachers on behavior checklists (CBCL), and maternal depression via self-report (CES-D). Measures of interaction such as joy, maternal sensitivity, responsiveness, maternal directiveness, involvement, clarity of commands and child compliance were taken during three 5-minute mother-child-play and teaching interactions. An overall quality of interaction measure was derived by summarizing measures that loaded on a single factor. Harnish and colleagues found that maternal depression was associated with both high externalizing scores and low overall maternal interaction quality. Children of parents with a lower socio-economic status had higher externalizing behavior. The mediating effects of interaction quality remained even if the effect of social economic status was controlled for. The direct effect of maternal depression on child behavior problems remained significant in every model. Accordingly, Harnish and colleagues concluded that quality of interaction, together with socio-economic status, partially mediated the link between maternal depression and child conduct problems.

Hammen, Burge and Stansbury (1990) found child behavior problems were not only related to both maternal depression and mother-child interaction quality, but also to child characteristics. Children between 8 and 16 years old were assessed in half-year intervals. 14 children of unipolar mothers, 12 of bipolar depressed mothers, 14 of medically ill mothers (e.g., diabetes), and 24 children of healthy mothers were included. Diagnoses were made with the Schedule of Affective Disorders and

Schizophrenia. Child behavior was rated by the mothers using the Child Behavior Checklist. Mother-child interaction was quantified during a discussion topic with ratings for positivity and constructivism. Hammen et al. found that maternal depression, plus behaviors such as low positivity and less favourable child characteristics, e.g., low positivity in interaction, predicted an unfavourable child outcome (e.g. child behavior problems and low social competences). Hammen et al. concluded that both mother and child contribute to negative reactions in their counterpart.

Zahn-Waxler, Iannotti, Cummings and Denham (1990) compared 2-year old children of 22 depressed mothers with children of 22 healthy mothers and subsequently assessed problem behavior when the children were 5 years old. Maternal depression was diagnosed via structured interview (SADS-L). 22 mothers were diagnosed with depression; mostly major depression or remitted major depression. Measures of interaction were taken during a 35-minute videotaped play session including a separation and reunion period. Child aggression was rated for interpersonal or undirected aggression or aggression towards objects. Maternal parenting was rated for giving structure, for social promoting, warmth, control, or force. Child problem behavior was assessed with the Child behavior checklist. Children of depressed mothers were found to be more likely of having maladaptive behavior; e.g., dysregulated or out-of-control behaviors. Dysregulated behavior in interaction coupled with a maternal diagnosis was found to predict externalizing behavior when the children were 5 years old. However, depressed mothers who used so called "proactive parenting" (e.g., exertion of modulated and respectful control, or problem anticipation) had a lower likelihood of having children with externalizing behavior.

The NICHD (1999) study of the American National Institute of Child Health and Development found that language impairments in children of depressed mothers was mediated by the degree of maternal sensitivity in interactions. 1215 mothers were longitudinally followed at 6, 15, 24, and 36 months after delivery. Maternal depression was identified via maternal self report using Radloff's Centre for Epidemiological Studies Scale with a cut-off of above 16; 663 mothers who had never been depressed, 460 who were depressed at some points of measurement and 92 chronically depressed mothers were compared. The mother-child dyads were videotaped during play sessions of 15 minutes. Maternal sensitivity was scored (e.g.,

supportive presence, positive regard, and intrusiveness or hostility). The children were rated for negative and positive mood. At 36 months the mother rated her child for social withdrawal, depression, sleep problems, somatic problems, aggression, and destruction, as well as for adaptive social behavior, i.e. for expression, compliance, and disruption. With respect to measures of mother-infant interaction, no differences between depression groups in negative or positive mood were found, even if demographic variables were controlled for. However, children of depressed mothers (either chronically depressed or sometimes depressed) had lower scores for school readiness and verbal comprehension. Children of chronically depressed mothers had lower scores of language expression, lower scores of cooperation, and higher problem scores. All depression-related effects, however, disappeared if maternal sensitivity was included, which led the authors to conclude a mediating effect of maternal sensitivity.

Conger, Patterson and Ge (1995) found that the link between maternal depression and child adjustment problems was mediated by poor maternal discipline practices. 451 seven year olds and their parents were included. Parental depression was assessed with the CES-D and Lubin Checklist, and parental discipline with an interview (punishment and consistency ratings). Direct observation was coded via a family process code system, adjustment of the child with standardized ratings of antisocial behavior, school achievement, peer ratings and ratings of child depression. A maximum-likelihood-estimation found that the link between depression and child adjustment problems was mediated by poor maternal discipline practices. Interestingly, the direct path between depression and deviant child behavior was reported to be insignificant, thus pointing to a completely indirect effect.

Laucht, Esser and Schmidt (2002) found performance impairments in children of postpartum depressed mothers; they also found increased externalizing behaviors. 22 postpartum depressed (DSM-III-R) and 116 healthy mothers and their children were followed for 8 years. 2-8-year-old children of postpartum depressed mothers were reported as having an IQ 10 points lower than control children and a 3-fold higher externalizing rate. This link between depression and child performance deficits and increased disorder risk was found to be moderated by maternal vocal activity, reactivity and warmth; depressed mothers with low vocal activity, low reactivity and low warmth had children who externalized and had a lower performance. On the other hand, depressed mothers who engaged in these three activities had children

comparable with children of healthy mothers. Laucht and colleagues concluded that child deviancies may be mediated by disturbances in the quality of mother-child interaction.

2.12. Summary with respect to the predictive value of parenting indicators

Although the available studies listed above are heterogeneous, they render some impression of the nature of the link between maternal depression and adverse child outcomes. A range of studies reported that the manner in which the psychiatrically ill parent and child interact may mediate adverse outcomes and may antecede intergenerational transmission effects. In a community sample, Johnson et al. (2001) identified maladaptive parental behavior as mediator for child psychopathology. Bifulco et al. (2002) found that aspects of parenting quality, such as child neglect or abusive parenting, mediated the link between maternal depression and psychiatric child disorder. Burt et al. (2005) found that family functioning measures, i.e. a high rate of conflict and home environmental measures (e.g., low responsiveness, low emotional climate) worked as mediator.

Moreover, these studies show certain mediating factors that may be summarized as high degrees of interpersonal negativity (e.g. harsh, punishing behavior, high distress, and conflict), as well as low interactional quality (low emotional responsivity), or displays of low maternal affect, high controlling maternal behavior or discipline. All these studies favor more or less the conclusion that parenting or interactional measures might be suitable mediators through which a parental disorder might manifest itself in adverse child outcomes.

3. The timing of depression (coincidence in time of maternal disorder and child deviancies)

3.1. Problem

The idea that parental unipolar maternal depression precedes or parallels maladjusted behavior or psychopathology of a child has been suggested by the groups of Hammen (1991) and Weissman (e.g. the review of Gunlicks and Weissman, 2008). Weissman et al. (2006) found that the remission from maternal depression (as investigated in the present study) regularly was accompanied by lowered rates of psychopathology in their children. Moreover, children who initially were asymptomatic developed symptoms if maternal depression remission was not reached after 3 months.

The amount of literature covering this subject is remarkably small and mostly correlative. In 2009, the number of available reports lists a maximum of approximately 10 publications (Gunlicks and Weissman, 2008). All of these will be presented briefly in the next chapter.

However, more than the half of the above-mentioned reports are by-products of medication trials with the aim of testing the efficacy of maternal depression treatments where children were assessed additionally (Weissman et al., 2006); Byrne et al., 2006; Clark et al., 2003; Forman et al., 2007; Murray et al., 2003; Verduyn et al., 2003). Two studies are simply open studies regarding treatments for maternal depression, i.e. simple pre-post treatment assessments without any control or reference group (Modell et al., 2001; Verdelli et al., 2004). Two studies followed improved parental depression in association with child outcomes and the type of treatment was not specified but simply labelled as “outpatient psychiatric treatment” (Lee and Gotlib, 1989; 1991; Timko and colleagues, 2002).

Moreover, all the reports are heterogeneous; e.g., with respect to sample size, child age, inclusion of mother and father, inclusion of mothers with the diagnosis of either major or minor depression, or even the allowance for co-morbidity. With respect to this research, i.e. infants and toddlers, reports with a focus on the timing between maternal depression and child behavior are practically nonexistent for the year 2010.

According to Hammen, Burge and Adrian (1991), parallelisms of maternal psychopathology and deviant child behavior may possibly point to non-genetic

transmission mechanisms. They suggested that depressed behavior rather than the biological substrate ("acting depressed rather than having depression", page 341) may be associated with maladaptive child behavior.

Besides the lack of sufficient epidemiological data of incidence rates in infants, the exact mechanisms explaining how a transmission may work are far from clear. Theoretically the mechanisms tabulated on page 21 may account for this phenomenon, i.e. for a child psychopathology transmitted through behavior or behaviorally induced adverse child outcomes.

With respect to the timing of depression and depending on the research group, following mechanisms are favored to account for these effects. For example, Hirsch et al. (1985) favor the parental disability hypothesis, i.e. depression associated skill deficits, maternal unavailability, or unresponsiveness (approaches also listed by Downey and Coyne, 1990; Burbach and Borduin, 1986; Goodman, 2007; or Conrad and Hammen, 1989). Thus children may react to maternal symptoms and subsequently develop emotional or behavioral problems due to maternal disturbances in parenting style which distresses children (behavioral model, Dumas and Serketich, 1994; Dumas et al., 1989; Conrad and Hammen, 1989; Roizen et al., 1996; Johnston, 1996; Woodward et al., 1998; Biederman et al., 1995).

The psychological distress hypothesis (e.g., Lyons-Ruth et al., 1986) suggests that child maladjustment may derive from psychological distress associated with maternal psychopathology. For example, symptomatic mothers who report higher levels of child behavior problems tend to be more aversive and controlling towards their children.

Three other approaches, although not followed with this research, are the hypotheses that the parental psychopathology may distort or bias the way the child's behavior is perceived (perceptual model, Fergusson, Lynskey and Horwood, 1993; Chilcoat and Breslau, 1997; Najman et al., 2000; Boyle, 1997). Distorted perceptions may affect rating data delivered by the depressed mother herself (this research, however, is based on observational data). Other explaining models comprise hypotheses of shared genetics (shared genes model, Sanger, MacLean and Van, 1992; Webster-Stratton, 1998; Jensen et al., 1990; Dumas and Serketich, 1994), or approaches where external factors may adversely affect mother and child (e.g., depression associated marital problems; Webster-Stratton, 1998; or heightened aversive and

controlling parental behavior; Dumas and Serketich, 1994).

Two further hypotheses deal with the stability of child effects over time. The transient disturbance hypothesis predicts that child maladjustment may be present only when the maternal depression is acute; a remission would be accompanied by child improvements. Contrary to this, the prolonged disturbance hypothesis would predict carry-over effects, i.e. that child maladaptation would continue after maternal recovery from depression.

3.2. Available studies

The available studies are predominantly constructed based on two basic designs. The first is a retrospective approach; depressed mothers are either classified based on their depression course, e.g., non-remitted, remitted and never-depressed women. Another research strategy is that different treatment groups (e.g., interpersonal therapy compared to mothers on a waiting list) are longitudinally observed and changes in infant parameters (e.g. externalizing and internalizing behavior) are compared with the extent of maternal remission.

Weissman and colleagues (2006) classified depressed mothers by their depression course. She showed an association between a change in maternal depression and child diagnoses (such as anxiety, disruptive behavior disorders and major depressive disorder). In 151 mother-child pairs, they found that a remitting maternal depression (DSM-IV) was followed by a lower rate of child psychiatric diagnoses (-11%, Kiddie-SADS), whereas in mothers who did not experience remission, the rate increased (+8%). Children whose mother completely remitted at 3 months had no psychiatric diagnoses at all. In addition, children whose mothers remitted from major depression over the 3-month period experienced a decrease in internalizing and externalizing child symptoms compared with those mothers who did not remit. Thus, a non-response to the treatment of maternal depression seemed to be followed by an increase in incidence of psychiatric child diagnoses. All findings were statistically controlled for child age as well as for other factors such as the sex of the child, child baseline symptoms, household income, maternal depression severity, and the treatment status of the child. The findings may point to the timing of maternal depression diagnosis and risk for child psychiatric diagnosis as well as suggest an environmental influence (i.e. the remission of maternal depression) on child psychopathology.

Hammen, Burge and Adrian (1991) analyzed temporal associations of maternal diagnoses and child diagnoses in 8 to 16-year old children over 3 years. The study was based on the hypothesis that maternal depression might be linked to symptom expression in children due to missing interpersonal sensitivities and skills on the part of the mother, in addition to the exposure of the child to life stressors including the maternal depression itself. The study has a remarkably small sample size of unipolar mothers (n=16) because other groups (e.g., of bipolar depression, chronic medical illness or healthy mothers) were formed. Maternal and child diagnoses were obtained via interview. The study registered onsets of child depression within a time frame of one month after the onset of the maternal depressive episode or during persistent maternal depression. A hierarchical regression successfully predicted that both maternal symptoms, together with stressful events and initial maternal depression, predicted the onset of child depressive symptoms. Hammen and colleagues concluded that maternal and child diagnoses are temporally associated, however with bidirectional influences, and offer two explanations; affect contagion according to Coyne (1985) or the effects of maternal depression on the quality of interactions due to withdrawn, unresponsive, irritable and impatient behavior.

Lee and Gotlib (1989; 1991) assessed children of mothers who initiated treatment for major depression. The sample sizes of the unipolar depressed mothers, however, was small (n=16). 10 mothers with another psychiatric illness, 8 with a medical condition, and 27 children of non-depressed mothers served as controls. The children were 7-13 years old. 44 dyads were included in a 10-month follow-up. Both self-rated and interviewer-rated internalizing and externalizing child symptoms showed no improvement in association with a maternal remission, i.e. adjustment difficulties tended to continue. The authors concluded that the presence of maternal psychopathology might be predictive for child adjustment rather than the diagnostic status per se (which was found to be non-predictive).

Cox, Puckering, Pound and Mills (1987, 2002) included mothers and their 2-year old children. A comparison of three groups (chronically depressed mother for 6 months, remitted by 6 months, or never depressed) revealed that children of remitted mothers were reported to be less disturbed (e.g., in ratings of peer-interaction) than children in the group of the chronically depressed. However, children of remitted mothers were even more impaired in comparison with controls, i.e. never-depressed mothers, which - according to the authors - points to some improvement in child functioning,

but with persisting impairments.

Alpern and Lyons-Ruth (1993) followed children from 18 months up to 4 and 6 years of age with respect to child behavior problems. Compared to those of never-depressed mothers, the behavior scores increased in children with chronically depressed mothers. Children of chronically depressed mothers were more hostile, whereas children of remitted mothers were rated as more withdrawn or anxious at school.

Moos and colleagues published a 10-year follow-up of 121 initially depressed parents (Timko, Cronkite, Berg and Moos, 2002; Billings and Moos, 1985). Groups of stably remitted, partially remitted, and non-remitted patients and controls were compared after 1, 4, and 10 years. The parents were asked whether their children had psychiatric symptoms or behavioral problems (e.g., discipline problems with peers or school deficits). Behavioral problems and child disturbances were heightened in children of partially remitted and non-remitted parents. Initial parental depression as well as current depression (together with family measures of conflict and cohesion) significantly predicted child behavioral problems and psychological distress, i.e. both higher initial or current parental depressive symptoms predicted poorer child adaptation. In the 1-year follow-up, children of parents with remitted depression were less impaired compared with children of parents with stable depression, but still had more impairment compared with children of never-depressed parents. After 4 years, children of remitted parents were comparable to children of never-depressed parents. Children of non-remitted parents had the lowest functioning scores. After a 10-year follow-up, children of stably remitted parents functioned comparably with children of non-remitted parents, although they did worse than children of never-depressed parents in indicators of psychological problems. The authors concluded that - irrespective of the course of parental depression - children of depressed mothers are at risk of developing problems.

Billings and Moos (1985) reported that depressed parents in remission continuously rated their children with lower functioning scores (table II on page 159). There was no decrease in psychological symptoms (e.g., anxiety or depression) in children whose mothers were in remission.

Byrne et al. (2006) studied 172 four to sixteen year old children of parents who did not respond to a depression treatment, and 88 children of parents who did respond

as a comparison group. After a 2-year follow-up, the children of those mothers responding to depression-treatment had a larger reduction in emotional symptoms as well as in the ratio of behavioral problems and symptom count (table 2, page 242). Although the authors conclude that a parental symptom reduction may be associated with reductions in child behavioral problems, the absolute amount of change was small. Children of treatment responders had no increased improvement in conduct problems or hyperactivity scores.

Clark et al. (2003) randomized major depressive women to mother-infant psychotherapy (n=13), or interpersonal psychotherapy (n=15) and compared both with a waiting-list group (n=11). Infants were between 1 and 24 months old and mother-child interactions were obtained by independent observers and the mothers as well. Maternal remissions in both therapy groups were paralleled with improved scores for infant adaptability, but lower scores for 'infant reinforces parent'. The total amount of change, however, was small, and infant cognitive or motor scores were not related to the maternal treatment. Other parameters, such as scores for infant mood, hyperactivity, dysregulation, social skills, and reciprocity were completely unaffected by the treatment-related changes.

Forman et al. (2007) randomized 60 major depressive women and their infants to interpersonal psychotherapy and a waiting list group (n=60) and compared them with 56 non-depressed mothers. Depression was assessed by clinical interview (SCID). The basic idea was the question of whether the remission of maternal major depression (Hamilton scores below 7) is associated with improved child measures, e.g., infant emotionality, attachment security, scores for child temperament or behavior problems. Although treatment-related reductions in parenting stress were obtained, the initial responses to depression therapy did not predict child outcomes. All in all, none of the child measures corresponded in any way to remissions in maternal depression. Independently of the remission of depression, self-rated negative parental affect and child-related parenting stress moderately predicted ($r < 0.5$) child negative affect and child behavior problems.

Modell et al. (2001) longitudinally followed 24 major depressed mothers and their 4-15 year old children. During antidepressant treatment and a retest interval of 4-8 weeks, self-reported depression scores decreased by 50%. This decrease correlated with a decrease of total child behavior problems (parental rating), predominantly due

to decreased conduct and learning problems, and lowered impulsive-hyperactive scores.

Murray, Cooper, Wilson and Romaniuk (2003) included 193 mothers with major depression (assessed by clinical interview plus EPDS above a cut-off of 12) and their newborns and randomly assigned them to four therapy groups; routine primary care as control group, a group with non-directive supportive counseling, a cognitive-behavioral therapy group and a fourth group with psychodynamic therapy. The children were assessed after 4, 18 and 60 months. Maternal management of infant behavior (feeding, sleeping, crying; or at 18 months, excessive temper tantrums – also reported by the teacher at 60 months) and mother-infant relationship problems (infant demands for attention, separation problems) were assessed by the mother. Although the study found no long-term effects (e.g., for attachment, or cognitive development at the 60-month follow-up), short term and post-treatment effects showed all 3 active treatments to be associated with fewer problems in the mother-infant relationship compared with mothers in routine primary care. Maternal sensitivity, rated by observers in mother-infant face-to-face interactions, showed no significant associations with the treatments. Although it increased from 2 to 4.5 months postpartum, a controlling for baseline made the differences between treatment-groups disappear. Accordingly, the study left the question open of whether the depression remission was associated with an overall reduction in child problems.

Verdeli, Ferro, Wickramaratne, Greenwald, Blanco and Weissman (2004) submitted a very small sample of 12 major depressed mothers to interpersonal psychotherapy (the mothers used outpatient clinic services, e.g. for a depression treatment). The study was uncontrolled, with a single treatment-group. After weekly therapy (45-min sessions for 12 weeks), both the mother's and children's depressive symptomatology and global functioning improved (both rated by a clinician). Interestingly, the improvement of maternal depression predicted improvements in child functioning, but not a reduction in symptoms.

Verduyn, Barrowclough, Roberts, Tarrier and Harrington (2003) randomized 119 mothers with major depression or dysthymia either to cognitive-behavioral therapy, to a support group, or to no depression treatment at all. The children were 36 months old on average. The study's usability, however, is limited since there was an attrition rate of over 60% per group due to refusal or withdrawal. There were no significant

differences between groups with respect to child problem behavior (internalizing and externalizing symptoms) from either pre-treatment of maternal depression to post-treatment or after 6 and 12 months. All in all, the study did not show that an effective depression treatment had any relation to improved child problem behavior.

Pilowsky, Wickramaratne and Ardesheer (2008) analyzed 123 mothers with major depression and their children (6-17 years) every 3 months over a period of one year. The children's symptoms were obtained via interview. Children of Citalopram-treatment non-responders had increases in psychiatric symptomatology, whereas mothers with a decreasing depression severity had children with less psychiatric symptoms. Previous reductions in depression symptomatology predicted later reductions in child symptoms, i.e. reductions in maternal HAMD scores preceded changes in child symptoms.

Foster et al. (2008) found in 114 mother-child that a remission from maternal major depressive (after treatment with Citalopram) was associated with increased ratings on 'acceptance/warmth' (according to the Children's Report of Parenting Behavior). Remission from maternal depression correlated with less internalizing and externalizing child behaviors, even after controlling for marital status, education and occupation. However, there was no relation between remission and psychosocial functioning of the child. The ratings on 'acceptance/warmth' were found to partially mediate the relation between depression-remission and lower internalizing symptoms according to the "Child Behavior Checklist" possibly pointing to the role of maternal functioning or parenting as mediator between remission children's psychiatric symptoms.

3.3. Conclusion and summary with respect to the suggested relevance of parallelism of maternal depression and adverse child outcome

Due to their heterogeneity the above presented studies do not allow a conclusion. They are too heterogeneous with respect to sample size, inclusion criteria of participants (e.g., refer to the wide age ranges), type of study (observational, controlled, uncontrolled), and study intention (e.g., some studies were byproducts of anti-depression medication trials).

However, the studies appeared to be consistent in the author's conclusion that remission from parental depression was paralleled by reductions in child symptoms or in the prevalence of child disorders. Most of the children then remained symptom-

free. Some authors also suggested that a reduction in postpartum depression had some effects on infant behavior, although based on too few data with infants. All in all Hammen et al. (1991) stress the potential of transmissions through behavior, which may be one mechanism of how maternal psychopathology and deviant child behavior may be connected, although genetic transmission mechanisms are not denied.

4. Looking over the fence: Parallelisms to other streams of research

4.1. Research in parental depression and parallelisms to research of emotional infant-neglect and emotional infant-deprivation

Extreme deviations in maternal emotional care have been studied under the term 'emotional neglect' or 'emotional maltreatment' (Kaplan et al., 1999). Emotional neglect has been established as subcategory of child maltreatment and has to be differentiated from other forms, e.g., physical or cognitive neglect (Alkema, 2006). Allin et al. (2005) cite emotional neglect as the most prevalent type of child maltreatment and central feature in all forms of maltreatment (see also Connell-Carrick, 2003; Iwaniec et al., 2007; Harrington et al., 2002; Claussen and Crittenden, 1991).

Official reports for child abuse and neglect (e.g. the 2007 DHHS report; Harrington et al., 2002) show that emotional child neglect occurs more than twice as physical abuse in officially registered cases in the US (56% vs. 23%). In Germany, it is estimated that that 5-10% of children are exposed to emotional neglect (Bmfsj, 2007). The 4th National Incidence Study of Child Abuse and Neglect (NIS-4, Sedlak et al., 2010) found an incidence rate of – however officially – registered cases of emotional neglect of 2.6 per 1000 children (page 3-4, 3-9). Mullen et al., 1996) found a rate of 5.6% in a community sample.

Emotional neglect is defined as parental behavior with remarkable similarities to that reported of depressed mothers. It has been characterized both by indifference and hostility (Iwaniec, 2003) , e.g. by omission, refusal of or delay in psychological care or inadequate affection to child's needs for affection or emotional support, with the potential to trigger emotional and behavior problems in children (Alkema, 2006). According to Glaser (2002) emotional neglect does not require physical contact (Claussen and Crittenden, 1991).

According to the American Professional Society on the Abuse of Children (1995) it

may include physical unavailability, unresponsiveness, withdrawal of attention, hostility and rejection of a child. It may include exposure of a child to inappropriate or inconsistent interactions and failure to provide cognitive stimulation (Ilwaniec, Larkin and McSherry, 2007, Glaser, 2002).

Parental depression was found to be the most frequent disorder in neglectful mothers (Connell-Carrick, 2003; Chaffin et al., 1996; Christensen et al., 1994; Coohey, 1998; Gaudin et al., 1993; Burke, 2003; Kinard, 1996) together with other correlates of child neglect such as increased infant temperament, infant gender, young child age, poverty and other factors of four major risk classes of demographic variables: such as family relations, parental and child characteristics (Brown et al., 1998), substance abuse (Ondersma, 2002) and parental obsessive compulsive disorders (Schumacher et al., 2001). Famularo et al. (1986) and Kaplan et al. (1983) found parental depression to be overrepresented in child maltreatment cases, although Chaffin, Kelleher and Hollenberg (1996) state that the rate of major mental illnesses is increased only in presence of substance abuse disorders.

In particular the risk factors that apply to the present research, i.e. maternal depression plus very young child age, e.g., under 3 years, have been found to be associated with heightened risks for neglect (Connell-Carrick, 2003). Accordingly, the early identification of parental depression has been recommended as strategy to minimize developmental child risks of (emotional) neglect and abuse (Kinard, 1996). Several authors have stated that emotional neglect or abuse compared to physical maltreatment is stronger as predictor with respect to a wide range of child problems such as externalizing and internalizing problems (see also next chapter, Kaplan et al., 1999, Mullen et al., 1996).

More extreme variants of neglect or maternal unavailability were studied with animals and published under the term “deprivation research” (e.g. see the review of Rutter, 1991). According to Bowlby (1951) - although his primary interests were children with prolonged maternal unavailability due to institutional care - the key aspect of deprivation is not the physical absence of a caregiver, but the lack of a warm, intimate and continuous relationship with the mother. Deprivation may be most effectively in infancy, not in childhood or later (e.g., refer to the review of Rutter, 1991, 1979). Bowlby was one of the first to point out that impacts of a parent with a maternal mental illness on children may resemble the effects of a maternal

deprivation (cited according to Newport, Stowe and Nemeroff, 2002) . It is interesting to note that Bowlby also postulated that long-time effects of deprivation stem from deprivation in infancy not from later childhood, are non-genetically, and, environmentally mediated (cited according to Rutter, 1991, page 332).

An extensive range of animal studies show that experimentally induced variations in maternal availability are associated with drastic effects on the offspring, for example as shown in separation studies with monkeys (Newport et al., 2002). Although these studies usually test for extreme conditions, Maestripieri and Carroll (1998) claimed them to be useful for the study of mechanisms in adaptation processes of offspring on variations in maternal care.

Classical experiments of deprivation are for example those of Harlow (e.g., reviewed by Suomi, 1991, and, Kraemer, 1997). Rhesus monkeys that were reared under a total lack of parenting in the only physical presence of a nonresponsive and mechanical surrogate mother, later displayed deviant peer-related social behavior, e.g., increased self-directed behavior, hyper- or hypo-emotional behaviors and failure to use facial expressions of peers in learning tasks. Kraemer et al. (1991) also reported depression-like responses and an inability to cope with the separation environment.

Although Novak and Harlow (1975) found that isolation effects were reversible if monkeys had access to peers, these animals turned out to be lower resistant to stress in case of environmental challenges, such as brief social separations, e.g. shown by heightened cortisol peaks and self-related behavior.

Francis et al. (1999) reported that offspring of rats that was exposed to a lower-quality maternal care, i.e. that was cross-fostered to a preselected and low-caring step-mother, indicated higher stress (cortisol) reactions and higher fearfulness under novelty conditions. Female offspring that were partially deprived of their environment (5 hr per day) showed reduced caring and interactive behaviors as adults and to their own offspring: they were less searching for tactile behaviors, and hyper-active in the open field (see also Lovic, Gonzalez and Fleming, 2001) .

4.2. Developmental consequences of emotional neglect / deprivation

The developmental consequences of emotional neglect (e.g. increased externalizing, and internalizing behaviors as well as impaired performance, Egeland et al., 2002,

Egeland, 2009) are remarkably similar to those discussed in association with maternal depression (e.g. Downey and Coyne, 1990). Infants of neglecting caregivers were found to be predominantly passive and emotional unresponsive during interactions. They were also found being increasingly non-compliant and impulsive (Egeland et al., 1983). Fagan and Dore (1993) characterized the child behaviors emotional negative with poor social interaction skills (see also DiLalla and Crittenden, 1990; Egeland et al., 1983; George and Main, 1979; Reidy, 1977, Egeland, 2009), or, with decreased interaction rates (Bousha and Twentyman, 1984). The risk for the infant of being classified as anxious-avoidant was increased, also being more angry, showing lack of persistence, and displaying little positive affect compared to children not classified as emotionally neglected (DiLalla and Crittenden, 1990). The behavior has been characterized as emotional instable and unresponsive (Kairys et al., 2002; Glaser, 2002), and with poor emotional regulation (Gaensbauer, 1982). As well as in infants of depressed (e.g. Laucht and Schmidt, 2005) Dietrich et al. (1983) found a lower mental and motor performance in neglected infants.

4.3. Summary: Comparison of maternal depression findings and the definition and findings of emotional child-neglect

According to the above-listed findings, the interactive behaviours of depressed mothers show remarkable parallels to the above-cited definitions of emotional neglect. These depression-related findings (the emotional unresponsiveness; the withdrawal, disrupted by hostility and rejection towards a child) are in accordance to the definition of an emotional neglect. Moreover, parental depression was found to be the most frequent disorder in neglectful mothers. The early identification of parental depression has been recommended to minimize developmental child risks of emotional neglect. Moreover, the same correlates are cited under which both maternal depression and emotional child neglect have been suggested to unfold their adverse effects, e.g. poverty or a conflictful family environment. However, it is unclear to what degree maternal depression and emotional neglect overlap. At least with respect to psychiatric child risks depression has been shown as non-necessary but a sufficient and a risk-increasing condition for neglect and to evoke the suspected subsequent effects on children (Johnson et al., 2001). Here, a parental psychiatric diagnosis (mostly depression and anxiety) was predictive for maladaptive parenting, e.g., low affection to the child, poor communication, inconsistent enforcement rules, low educational aspirations, and, low amount of time spent with the child. Moreover,

the parental diagnosis was – technically – a non-necessary factor (but a quite sufficient starting point for neglect behaviors and child maltreatment). Johnson et al. (2001) have shown that only these maladaptive parenting behaviors (emotional neglectful behavior) were suitable mediators for increased child risks with respect to a psychiatric disorder.

5. Hypotheses

5.1. Deduction of hypotheses based on limitations of existing research

The present study was designed to replicate and extend prior findings concerning the impact of maternal major depression on mother-infant interaction. It fills an important research gap concerning very few results with mothers diagnosed with major depression (previous studies preferred self-rated depression) and much fewer studies that observed affect-related interactive behaviors.

The present research fills a further gap since it maximizes the proximity between the depression episode and the observation of mother-infant interaction. It addresses a further completely unfilled gap in current infant-research, i.e. the question of what happens to observable interactions after the remission of a major depression.

Following strategies were derived from above-listed streams in the current literature:

The enlisted publications (see page 43) have found maternal depression to be predominantly predictive for compromised mother-infant interaction, e.g., a generally negative, hostile, non-responsive, or even intrusive interaction style with reduced unconditional positive regard or less facilitative behavior with asymmetrical contingencies; i.e., less positive with a shift to a negative-contingent responsiveness. Surprising parallelisms were observed in infants of depressed mothers. They were found to be less active, use less vocalizing, and to be less contingent responsive; their positive affects were found to be less parallel to maternal behavior. However, not all studies found relations and the study heterogeneity with respect to depression type, chronicity, timing, samples and indicators is impressive.

Longitudinal studies have concluded that children of depressed mothers are at risk of internalizing, externalizing behaviors or developing performance problems.

A further line of research suggests that dysfunctional mother-infant interaction acts as a mediator between a maternal diagnosis of depression and deviant child behaviors

and impaired development. The most influencing studies are those of Johnson et al. (2001), Bifulco et al. (2002) and Burt et al. (2005). They found that parenting quality in terms of maladaptive parental behavior or high degrees of interpersonal negativity had the quality of a mediator between maternal depression and psychiatric child disorder (although long-term outcomes are targeted in a later study of our study group, the possibility of long-term child risks underscores the validity of this research).

Another line of research suggests that the sole presence of an episode of maternal depression is predictive for adverse effects in children, i.e. risks for child disorders have been considered high in the presence of parental depression. Conversely, the absence of such depression (i.e. in case of a remission of maternal depression) has been suggested to precede ameliorations in adverse child effects.

Based on these major streams of research, this research follows the suggestion of several working groups (e.g., Stanley et al., 2004; Murray et al., 1999) that the exposure to maternal depression and deviant mother-infant interaction might be a suitable precedent and predictor of adverse child effects (e.g. a heightened risk of child disorder diagnoses or impaired child performances). However, it was also predicted that the remission of depression may lead to improvements in child outcome.

For the deduction of hypotheses several further assumptions are added: for example, that a mother is an infant's most important environment in the first months of life, emotionally as well as physically. Furthermore, very early infant learning strategies based on mirroring have been shown to be effective (refer to the empirical result survey), and finally, several relevant authors in the field of depression-related interaction (Field, 2002; Lovejoy et al., 2000; Stanley et al., 2004) have suggested that both mirroring itself and operant learning may be the key transmission mechanism based on which a maternal depression unfolds effects onto the offspring.

Based on the above, two approaches were followed.

The first approach is based on a scenario in which the operant learning environment for an infant is altered in the presence of maternal depression. A depressed mother may be restricted in her function and thus in her parenting resources (**theory of impaired parenting**, refer to the theory survey on page 21 and 22), essentially due

to characteristics of her depression-symptom profile. She may be impaired due to loss of energy and due to flat affect; both characteristics may completely alter the infant's operant learning environment and reinforcement conditions (e.g., Lovejoy and colleagues, 2000).

Secondly, several authors assume that maternal affect can control or regulate infant affects based on mirroring of infant-affects (e.g. Gergely and Watson, 1996). Accordingly, due to flat affect, a depressed mother may fail to use her affects to regulate infant affects. Unregulated affects have been operationalized as lack of affect parallelisms, predominantly of positive affects ("affect-mirroring"). Moreover, failures in infant regulation are theorized by various authors as precedents of adverse infant outcome, e.g. externalizing behavior. In particular, the theory of Gergely ("affect mirroring as social biofeedback", Gergely and Watson, 1996) connects flat maternal affects with deficient affect-mirroring of a depressed mother and a subsequent failure to provide infant regulation (**distorted affect-mirroring theory**).

Both theories together (impaired parenting and distorted affect-mirroring) allow for the prediction of reductions in affect-mirroring and for deviant mother-infant interactions when a mother is diagnosed with major depression.

Both theories are connected with a present maternal depression. Accordingly, a reduction of deviancies in interaction has been predicted in the case of a remission of maternal depression (**transient child disturbance theory**). The theory predicts that dysfunctional interactions and child maladjustment may disappear when the maternal depression remits (Gunlicks and Weissman, 2008; Downey and Coyne, 1990). Accordingly, the present study expects that deviant mother-infant interaction will disappear and the mirroring of affects will normalize after a remission of the maternal major depression.

Thus the main hypotheses of this research (details see below) directly derive from three theories: the theory of impaired parenting, the distorted affect mirroring theory and the transient child disturbance theory.

Hypotheses with confirmatory status (e.g., statistically tested with 1st-type error adjustment) refer to depression-related impairments in well-used indicators of mirroring (e.g., parallel occurring affects of mother and infant). Gergely's theory allows for a prediction of lowered affect mirroring when a mother is diagnosed with

major depression (hypothesis of a reduction of affect mirroring).

The impaired parenting (due to restricted resources) hypothesis allows for further predictions, e.g. of generally lower positive maternal affects and a prolonged latency until affect-mirroring re-occurs (hypothesis of a prolonged latency until positive affect-sharing in depressed dyads). Changes in reinforcement conditions under maternal depression allow for the prediction of a lack of affective infant-stimulating behavior (in accordance with the hypothesis of impaired parenting). Moreover, infants of depressed mothers may become accustomed to low-level affects and remain unaffected if maternal availability varies (exploratory hypothesis of non-affectedness of infants of depressed mothers in case of maternal unavailability). Conversely, if a major depression remits, its adverse effects on both interactants, e.g., reductions in both affect-mirroring and infant-negativity should recede (theory of transient disturbances).

As mentioned above, and since the present research also has a highly explorative character, other competing theories are also included and will be statistically tested in an exploratory manner (see chapter on exploratory hypotheses, page 99). Some of those theories may be regarded as variants of the impaired parenting theory with similar effects on infants of depressed mothers, e.g., Lewinsohn's lack-of-reinforcers theory (1974) and Coyne's model of rejection-due-to-negativity-in-interaction (1985). A learning-oriented model, such as the model of Lewinsohn, may be predictive in the case of stimulation deficits when a mother is depressed, and the model of Coyne (details see below) may be applied to predict the induction of negativity in infants due to dysfunctional maternal behavior.

Accordingly, negative behaviors of both the mother and infant are expected during interaction. However, the total level of behavior is expected to be lower. Both interactants are predicted to be restricted in their behavior repertoire; the mother due to loss of energy and flat affect, the infant due to a low level of stimulation.

A theory of Coyne focuses on the contagion effects of depressed moods and the respective prediction that a depressed interactant may induce rejection and (with increasing engagement) unwillingness to interact or reciprocal negativity (most probably circles of negativity).

Further theories (e.g. Hammen's) that are closely related to the theory of impaired

parenting allow for a deduction that engagement of depressed mothers (due to their dysfunctional interpersonal behavior) is experienced as stressful for their infants.

Moreover, depressed mothers may show interactive exhaustion, i.e. lower interaction maintenance, may be predicted to be less contingent responsive, and the predictability of their behavior may also be lower.

The operationalization of hypotheses will be given on page 97.

To test and explore these hypotheses the present study included mothers with a clinical diagnosis of major depression and their infants and compared them to a control group of mothers completely free of psychiatric disorders and their infants. Major depression according to DSM-IV (SCID, Wittchen, Wunderlich, Gruschwitz and Zaudig, 1997), or being free of psychiatric disorder according to the clinical interview, was an inclusion criterion for this study. Although the DSM-IV allows for the diagnosis of major depression, if in the last 4 weeks the criteria were present for at least 2 weeks, we included depressed mothers in an acute episode of depression (the videos were taken when the women were in an episode of depression). We notably followed the advice of the Hammen workgroup (1987; 1991; 2003): They propose that current symptoms are better predictive than a positive depression history, i.e., better predictive for deviant interactions or the incidence of adverse child outcome (e.g., internalizing or externalizing behavior).

All the dependent measures are operationalized as observable behaviors only. All codes of dyadic interaction were assigned in a standardized laboratory setting.

Moreover, infants with a large age range are included, i.e. predominantly between 2 and 8 months. This was mainly due to a low recruitment speed of participants but it also allows for additional tests which are completely lacking in the literature, e.g., depression-related cumulative exposition effects or increasing effects on the mother the younger infants are. Some authors (e.g., Radke-Yarrow et al., 1985; Stein et al., 1991) comment that the depressed parent may be conceptualized as the primary environment of an infant. If it is taken that the younger the infant is, the more amount of time is spent with the depressed parent, then transmission effects of maternal depression - if they exist - should be observable the younger the children are. There are several further advantages of a large infant age range, such as the ability to generalize over age groups, or, to calculate maternal-diagnosis by age interactions.

Furthermore, none of the theories listed in the survey (page 21 and 22) explicitly conceptualize infant age as a mediating factor. In the majority of previous studies, however, the effects of infant-age were not testable due to homogeneous age-groups.

Finally, to test for remission-associated effects, all mother-infant pairs were assessed twice; mothers with major depression (in episode) and again after an average of 2 months, after a complete remission from major depression.

It should be noted that the available studies are not only highly inconsistent (e.g. with respect to hypotheses, available measures such as ratings by the mother, by observers, or observed behaviors or settings, e.g., at home or in the laboratory). Their predominant goal was to test for the predictive value of maternal depression, not for a specific model of transmission. Specific tests for theories or head-to-head comparisons of different transmission theories are completely lacking.

Moreover, very few studies recorded observable behaviors, most of them applied ratings. Registering observable behaviors completely eliminates any possible discussion with respect to a possible bias of parental reports distorted by a depression diagnosis (e.g., due to distorted, negative maternal perceptions or tendencies to possibly over-report infant problems; refer to Field, Morrow and Adelstein, 1993; Lovejoy, 1991; Fergusson, Lynskey and Horwood, 1993; Richters and Pellegrini, 1989; Richters, 1992). Contrary to global rating-measures, a second-by-second observation of behavior allows the analysis of patterns over time, e.g., reciprocal behavior (such as cycles of negativity). In addition, only a few studies used a standard diagnostic interview for depression (e.g., Stein et al., 1991; Cohn et al., 1990; Stanley et al., 2004; Zlochower and Cohn, 1996).

Some interpretations of study results have also been questioned (e.g., by Rutter, 1990; page 61), since large-sized effects (e.g., Field et al., 1990) were derived from disadvantaged samples of single mothers with a lower socioeconomic status which is not the case in the present sample.

And finally, almost all previous studies used statistical analytical methods (e.g., analyses of variance or other general linear methods) without any tests for assumptions, e.g., for normality of residuals or the homogeneity of their variances. The assumption that these tests are robust is criticized here, in particularly because

distributions of behavior frequencies and durations are enormously skewed and far from being normal. In contrast to this, nonparametric univariate methods (Lehmann, 1998) and nonparametric multivariate methods (Wei and Lachin, 1984) avoid adverse effects due to unfulfilled assumptions (e.g., a lower detection sensitivity of statistical tests due to outliers). Moreover, in contrast to all preceding studies, a control procedure of statistical decision errors (1st type error adjustment; Altman et al., 2001) was applied to avoid the reporting of false-positive results.

5.2. Hypotheses, operationalized

5.2.1 Predictions based on the current episode of maternal depression, hypotheses with confirmative status

Based on the previous chapter and the above-mentioned theories, 7 confirmatory hypotheses were formulated, based on a study-wise error of $\alpha = 0.05$ and a comparison-wise error (adjusted 1st type error, Bonferroni, cited in Holm, 1979) of $\alpha_{\text{adjusted}} = 0.007$ ($\alpha = 0.05$, divided by 7 tests).

1st confirmative hypothesis: based on the social biofeedback theory of Gergely (Gergely and Watson, 1996; 1999) and the expectation of a depression- associated **reduction of affect mirroring**, we predict that depressed mothers will show impairments in the mirroring of affect (operationalized as parallel-occurring positive behaviors of mother and infant). When the caregiver displays smiles and vocalizes positively, her infant will simultaneously display joy, smile whilst gazing at her, or at least be attentive towards her. Depressed and healthy controls will be statistically compared based on an adjusted alpha of $\alpha = 0.007$ with a two-tailed exact Mann-Whitney-test (Lehmann, 1998; Mehta and Patel, 1997; Conover, 1980; Hollander and Wolfe, 1999) testing whether the two samples are drawn from one population and that their probability distributions are therefore equal. $H_0: F_1(x) = F_2(x + \lambda)$ versus $H_1: F_1(x) \neq F_2(x + \lambda)$, with F_1 and F_2 as distributions and λ as location parameter (which basically reduces to $H_0: \lambda = 0$ versus $H_1: \lambda \neq 0$).

2nd confirmative hypothesis: based on flat affect assumptions, dyads with a depressed mother will be characterized by frequent failures and delays in gaining a state of shared positive affect, i.e., they will need more time until affect mirroring occurs (hypothesis of a **prolonged latency until positive affect-sharing in depressed dyads**). Contrary to the previous hypotheses, it is not overlap time that is of interest but the time needed until parallel positivity occurs. Thus, since this

hypothesis refers to an event over time with the possibility that the event does not occur ("censored data"), time-to-event analyses are applied, i.e., estimators according to Kaplan & Meier (Cox and Oakes, 1984; Kalbfleisch and Prentice, 1980). The cumulative incidence rates of regained positivity overlap will be examined with a log-rank test to look for differences between depressed and healthy dyads using an adjusted alpha of $\alpha = 0.007$ (adjusted for 7 confirmatory tests). The hypotheses are $H_0: Z < |z_\alpha|$ versus $H_1: Z > |z_\alpha|$, with z_α as the upper α percentile of the standard

normal distribution and
$$Z = \frac{\sum_{j=1}^J (O_{1j} - E_{1j}) / \sqrt{\sum_{j=1}^J V_j}}$$
 with O as observed and E as expected frequencies on j distinct points on the time axis, and V as variance estimator, Peto and Peto, 1972).

3rd confirmative hypothesis: the idea that a depressed mother acts as a role model for flat affect and that the infant mirrors it (Meltzoff, 1988; Field, 1984), plus the theory that an infant of a depressed mother is exposed to a lack of response-contingent positive reinforcers, suggested the hypothesis of a **non-affectedness of infants of depressed mothers if the she is affectively unavailable**. The hypothesis predicts that infants of depressed mothers are not affected by the maternal still-face procedure, i.e., they are unaffected by a slight variation (off-on) of maternal care. Technically, this primary target parameter refers to a post minus pre difference in the overlap time of dyads. Again, an exact Mann-Whitney-test for inferiority of the depressed pair will be applied using a two-sided alpha of $\alpha = 0.007$ (again adjusted for 7 confirmatory tests, $H_0: F_1(x) = F_2(x + \lambda)$ versus $H_1: F_1(x) \neq F_2(x + \lambda)$, see page 97).

4th confirmative hypothesis: **lack of affective infant-stimulating behavior**: depressed mothers are expected to provide a low number of behavioral stimulators, i.e., be generally hypo-active when interacting with their infant. Technically, a lowered overall duration of stimulation behavior is expected (all parameters of table 21 intra-individually summed). Again, an exact Mann-Whitney-test for inferiority will be applied using a two-sided and adjusted alpha of $\alpha = 0.007$ ($H_0: F_1(x) = F_2(x + \lambda)$ versus $H_1: F_1(x) \neq F_2(x + \lambda)$, see page 97).

5th confirmative hypothesis: refers to the expectation of a deviant affective activity level in infants of depressed mothers (either too low or too high): infants of major

depressed mothers are expected to be **generally deviant in their affective activity level, i.e., either hypo-active or hyper-active**, that is, either lower or higher overall frequencies are predicted (parameters in table 20). Again, an exact Mann-Whitney-test for inferiority of the depressed pair will be applied using a two-sided and adjusted alpha of $\alpha = 0.007$ (overall effect in infant-frequencies, $H_0: F_1(x) = F_2(x + \lambda)$ versus $H_1: F_1(x) \neq F_2(x + \lambda)$, see page 97).

5.2.2 Predictions based on the remission of maternal depression, hypotheses with confirmative status

6th confirmative hypothesis: based on the hypothesis that parental impairments will decrease in association with the remission of depression, affect mirroring is predicted to increase at a greater rate in dyads with a depressed mother compared to dyads with non-depressed mothers, whose affect mirroring is expected to increase slightly, e.g., due to infant age (the comparison will be run with an exact Mann-Whitney-test for differences from in-episode to after-remission using a two-sided and adjusted alpha of $\alpha = 0.007$ (overall effect in time-proportions, $H_0: F_1(x) = F_2(x + \lambda)$ versus $H_1: F_1(x) \neq F_2(x + \lambda)$, see page 97).

7th confirmative hypothesis: based on the transient disturbance hypothesis, an overall reduction in negative child behaviors (heightened protest or - conversely - withdrawal rates) is expected after the maternal depression is remitted (tested with an exact Mann-Whitney-test over differences from in-episode to after-remission using a two-sided and adjusted alpha of $\alpha = 0.007$ (overall effect in time-proportions, $H_0: F_1(x) = F_2(x + \lambda)$ versus $H_1: F_1(x) \neq F_2(x + \lambda)$, see page 97)

5.2.3 Exploratory hypotheses based on the current episode of maternal depression

Several exploratory hypotheses will be evaluated based on an unadjusted 1st type error of $\alpha = 0.05$. Although multiple testing clearly increases the risk of false-positive predictions, the consequences of a beta error (falsely excluding existing risks for infants) are estimated to outweigh the risk of an alpha error (falsely rejecting null effects). However, result replication is highly necessary because all test results have hypothesis-generating status. Almost all hypotheses refer to between-group comparisons based on a single parameter or in a multivariate setting. Accordingly, an exact Mann-Whitney ($H_0: F_1(x) = F_2(x + \lambda)$ versus $H_1: F_1(x) \neq F_2(x + \lambda)$, see page 97) and Wei-Lachin's Multivariate Rank Analysis as multivariate extension of the Mann-

Whitney test (Wei and Lachin, 1984; Lachin, 1992) for two-group comparisons are applied ($H_0: F_{1j}(x) = F_{2j}(x)$ versus $H_1: F_{1j}(x) \neq F_{2j}(x)$ with F_h as the multivariate cumulative distribution function of the repeated observations, for details refer to Davis, 2000). Both tests demand zero to minimum requirements from the data with maximum power gain in case of non-normal or heteroscedastic (e.g. highly skewed) data distributions (as durations and behavior frequencies are expected to be).

1st exploratory hypothesis: **Heightened maternal negativity in specific aspects (hostility, withdrawal, exaggerations)**: Mothers with major depression are predicted to be more negative in specific aspects, that is, will engage negatively in certain behaviors much more frequently and for longer periods of time during dyadic interaction (such as being hostile, intrusive, withdrawn, non-infant focused and having exaggerated behaviors).

2nd exploratory hypothesis: heightened **infant** negativity in association with maternal depression: Infants of major depressed mothers are generally **more negative**, i.e., have higher frequencies of negative codes (being overall negative, withdrawn, or protesting, codes from table 20 intra-individually summed).

3rd exploratory hypothesis: Depression-associated excessive use of negative behaviors related to all behaviors: Both mothers with major depression and their infants are predicted to excessively use **more negative behaviors relative to their total behavior repertoire**, i.e., within their behavioral repertoire interactants are expected to use negative codes much more excessively than healthy dyads do (the individual probability of a negative behavior within the individual repertoire operationalized as total list of behaviors to be coded - codes from table 21).

4th exploratory hypothesis: Lowered effects of maternal affective unavailability in association with maternal depression: Infants of depressed mothers are expected to be **generally unaffected by maternal withdrawal or unavailability in the phase of the "still-face"**, i.e., with respect to all other behaviors no pre-post differences are expected to occur, whereas infants of control mothers show reduced behavior frequencies.

5th exploratory hypothesis: **Restrictions in behavior repertoire associated with major depression**: Mothers with major depression are predicted to have lower interaction skills. Since the applied behavior coding system of Tronick et al. claims to

cover an almost complete range of affect-related behaviors, a restricted usage of the full code range was hypothesized in dyads with a depressed mother, i.e. (in terms of variability) a lower intra-individual standard deviation of the behavior code range is predicted.

6th exploratory hypothesis: Depression-associated **behavior restriction in infants of depressed mothers**: Due to an expected restriction in the maternal behavior repertoire, it is expected that infants of depressed mothers will also have a lower (i.e. a restricted) behavior repertoire. Infant behavior restriction, too, is operationalized as width of usage of behavior codes.

7th exploratory hypothesis: **Reduction in general speed of interaction in dyads with a depressed mother**: A slower “production” of behaviors, i.e., a lower general interaction speed, is predicted in depressed dyads. Dyads with a depressed mother are expected to have lower overall behavior frequencies over time.

8th exploratory hypothesis: **Rejection-inducing effects of maternal depression**: Theories of mood contagion suggest that depressed individuals induce rejection in their interaction partner. This allows for the prediction that the longer the interaction has been going on, the steeper infant negativity will accumulate (e.g., infant protest and withdrawal). This effect is expected to be much pronounced in dyads with a depressed mother.

9th exploratory hypothesis. **Increased unwillingness to interact in infants of depressed mothers with increasing maternal engagement**: Again, based on the theory of mood contagion and subsequent effects, we predicted that infants of depressed mothers show avoiding behaviors more frequently, i.e., a higher rate of non mother-directed behaviors, more withdrawals, or non-mother focused behaviors, especially the more the mothers engage with their infant. Thus infants of depressed mothers are predicted to show a higher proportion of negative or non-mother focused activities compared with the overall rate of maternal engagement.

10th exploratory hypothesis: **Depression-associated reciprocal negative affects; negativity spirals**: Based on theories of mood contagion (plus the associated effects of reassurance-seeking behavior of the depressed individual and negativity induction in the interaction partner), it was predicted that dyads with a depressed mother are prone to interpersonal spirals of negativity. Based on the expectation of floor effects

in both maternal and infant negativity (infant protesting, withdrawals, hostility), negative interpersonal spirals or a negative reciprocity will be operationalized indirectly, i.e., as the expectation of lower occurrence of spirals of positivity. Thus, it is predicted that dyads with a depressed mother will be characterized by lower rates of interpersonal spirals of positivity during face-to-face interactions.

11th exploratory hypothesis: **Engagement of depressed mothers is experienced as stressful**: This hypothesis is based on interpersonal stress approaches, which suggest that parenting of depressed mothers is perceived as negative and stressful by their infants and that dysfunctional interpersonal behavior may mediate how maternal depression unfolds its effect on the child. Accordingly, this allows for the prediction that infants of depressed mothers may be much more negative in cases where maternal engagement takes place. In particular, increased infant protest or non-mother focusing and lowered positive infant engagement are expected during *high* engagement of depressed mothers compared with highly-engaging control mothers.

12th exploratory hypothesis: **Reduced interest or ability to maintain interaction**: According to symptom-based approaches, it is predicted that depressed mothers are more likely to show indications of “interactive exhaustion” or lowered interaction maintenance, i.e., they may show a downward trend in total engagement time over the observational period.

13th exploratory hypothesis: Contingency reduction in maternal responsiveness to infant behavior: Depressed mothers are predicted to be **less contingently responsive** to their infant or less sensitive to infant cues, i.e., show lower conditional responses in Alison-Liker estimators (a conditional measure that controls for spontaneous behavior rates; Allison and Liker, 1982).

14th exploratory hypothesis: **Deviancy in contingent reactions in infants of depressed mothers**: Infants of depressed mothers are predicted to behave either less responsive or over-responsive to maternal behavior, i.e., show deviant conditional responses in Alison-Liker estimators (conditional measures that control for baseline behavior rates; Allison and Liker, 1982)

15th exploratory hypothesis: Deviation in infant regulation: Based on theories of impaired infant regulation in association with maternal depression, it is predicted that

infants of depressed mothers will show **lower adjustability or controllability**, i.e., show longer offset latencies until cessation of protest or need longer until the mother is focused again.

16th exploratory hypothesis: **Reduction in the capacity to interact synchronously**. Dyads with a depressed mother are predicted to be less synchronous in terms of shared variance of two time series per dyad when behaviors are classified on a dimension of positivity and negativity.

17th exploratory hypothesis: **Reduction in predictability** of behavior both for depressed mothers and their infants. This hypothesis is based on regulation models that predict that dyads with a depressed mother are poorly coordinated and thus have generally less predictable behaviors. Predictability will be calculated using a Fisher-transformed Pearson correlation of mother and child's time-series lagged against each other.

18th exploratory hypothesis: **Reductions in overall responsiveness**: Based on the parenting impairment or resource restriction hypothesis (e.g. low energy), depressed mothers are expected to be less responsive in terms of time until a reaction occurs, whereas their infants are expected to be deviant-responsive to maternal behaviors due to the changed reinforcement conditions (e.g., they are expected to be either less responsive due to the imitation of maternal withdrawal behavior or due to the maternal hypo-stimulation or high eliciting behaviors resulting from heightened thresholds of maternal response where maternal depression is present).

5.2.4 Exploratory hypotheses based on the remission of maternal depression

19th exploratory hypothesis: Based on the transient disturbance hypothesis a normalization in infant reactions to interrupted communication following the remission of maternal depression is predicted. After remission, infants of formerly depressed mothers are predicted to respond to restrictions in maternal communication (still-face) no differently than healthy dyads would do. Technically, infant reactions from pre to post maternal still-face will be used and those reactions will be followed from depression to remission with the expectation of an increased infant reactivity after the maternal depression is remitted.

20th exploratory hypothesis: After maternal depression remission there is no longer a **lack of infant stimulation**. Based on the impaired parenting hypothesis and an

improvement following remission, depressed mothers are expected to improve in their number of emitted behavioral stimulators. Technically, an increase of lowered overall frequency of behavior to normal values is expected (all parameters in table 21 intra-individually summed per dyad).

21st exploratory hypothesis: Restoration of deviant infant activity level. Based on the mirroring hypothesis and hypothesis of under-stimulation due to the restriction of maternal resources, infants of depressed mothers are no longer **deviant in their activity level** following remission of maternal depression depressed mothers, i.e., are neither hypo-active (mirroring hypothesis) nor hyper-active (under-stimulation hypothesis), that is, a normalization of previously reduced or increased overall infant behavior frequencies is predicted.

22nd exploratory hypothesis: Based on theories that **depression is experienced as stressful** for the interaction partner, infant negativity is predicted to disappear following remission of maternal depression.

23rd exploratory hypothesis: Reduction of maternal negativity. Based on theories regarding the suggested **rejection inducing effect** of depression, it is predicted that suitable precursors for rejection, i.e., the occurrence of maternal negativity or hostility, are no longer expected to be different from controls given that the maternal depression is remitted.

24th exploratory hypothesis: Based on a recovered depression profile and the regain of functional resources after the depression is remitted, formerly depressed mothers are predicted not to **differ in their responsivity** in comparison with control mothers.

25th exploratory hypothesis: Based on a recovered maternal depression profile and the regain of functional resources after the depression is remitted, infants of formerly depressed mothers are predicted not to **differ in their responsivity** in comparison with control infants.

26th and 27th exploratory hypothesis: In association with the remission from depression, both mothers and infants are expected to recover from a **restricted behavior repertoire** in terms of lowered variability on a negative-positive dimension of available behavior codes. Thus following the remission, they are predicted to no longer have a lower level of interaction skills.

28th exploratory hypothesis: Following depression remission, mothers are expected to

regain a normal speed of interaction in terms of overall affect-related behavior frequencies over time.

29th exploratory hypothesis: Following remission of depression, mothers no longer provoke **rejection-inducing effects** in their infants, i.e., the incidence of negative affects is predicted to decrease.

30th exploratory hypothesis: Following remission of maternal depression, infants no longer show **unwillingness to engage**. Since mood contagion effects are expected to decrease after remission, infants no longer show avoiding behaviors, e.g., fewer mother-directed gazes, more frequent withdrawals or protest - especially if their mothers act highly positively (operationally, the rate of interaction avoidances is related to the overall rate of maternal positivity and this ratio is expected to decrease).

31st exploratory hypothesis: Following maternal remission from depression, fewer **reciprocal negative affects** are predicted to occur, i.e., less interpersonal spirals of negativity. Infants of remitted mothers no longer show higher rates of negative contingencies and likewise no lowered rates of positive reciprocals.

32nd exploratory hypothesis: Following remission of depression, maternal engagement is no longer **experienced as stressful** and infants of depressed mothers no longer show higher rates of protest or withdrawal when preceded by non-negative maternal engagement (e.g., positive or neutral engagement).

33rd exploratory hypothesis: Following maternal remission from depression there is no longer a **reduced interest or ability to maintain interaction**, i.e., remitted mothers no longer show indications of “interactive exhaustion” but show normal interaction maintenance, i.e., there is no longer a downward trend in total engagement time over the observational period.

34th exploratory hypothesis: Following maternal remission from depression there is no longer a **reduced contingency in maternal responsiveness** to infant behavior. Mothers are no longer less contingently responsive to their infants and no longer less sensitive to infant cues, i.e., they no longer show lower conditional responses in Allison-Liker estimators (conditional measures which control for baseline behavior).

35th exploratory hypothesis: Following maternal remission from depression, infants of depressed mothers are **no longer deviant-responsive**, i.e., no longer less

responsive or over-responsive to maternal behavior. Deviant conditional responses in Alison-Liker estimators are no longer present.

36th exploratory hypothesis: Following maternal remission from depression, infants of formerly depressed mothers recover in their **self-regulatory capacities** and show normalized values, i.e., values no different from those of control infants and no longer show latencies of consolability in terms of time until being soothed if crying or protesting.

37th exploratory hypothesis: After maternal depression has remitted there is no longer a **reduction in the capacity to interact synchronously**. Dyads with a depressed mother are no longer less synchronous in terms of shared variance of two time series.

38th exploratory hypothesis: After maternal depression has remitted there is no longer a reduction in **predictability** of affect-related behaviors of both mother and infant. Predictability will be descriptively calculated using the Pearson correlation of each mother and child's time series. Both groups will be compared based on the shared variances (calculated per dyad) and Fisher z-values (also calculated per dyad).

6. Methods

6.1. Narrative of study realization

Mothers who were hospitalized with their infants between September 2003 and November 2006 at the mother-infant treatment unit of the Psychiatric Clinic of the University of Heidelberg (Fricke, 2005; Fricke et al., 2006) and who received in-patient treatment were diagnosed according to DSM-IV-criteria with the Structured Clinical Interview (SCID, Wittchen, Wunderlich, Gruschwitz and Zaudig, 1997).

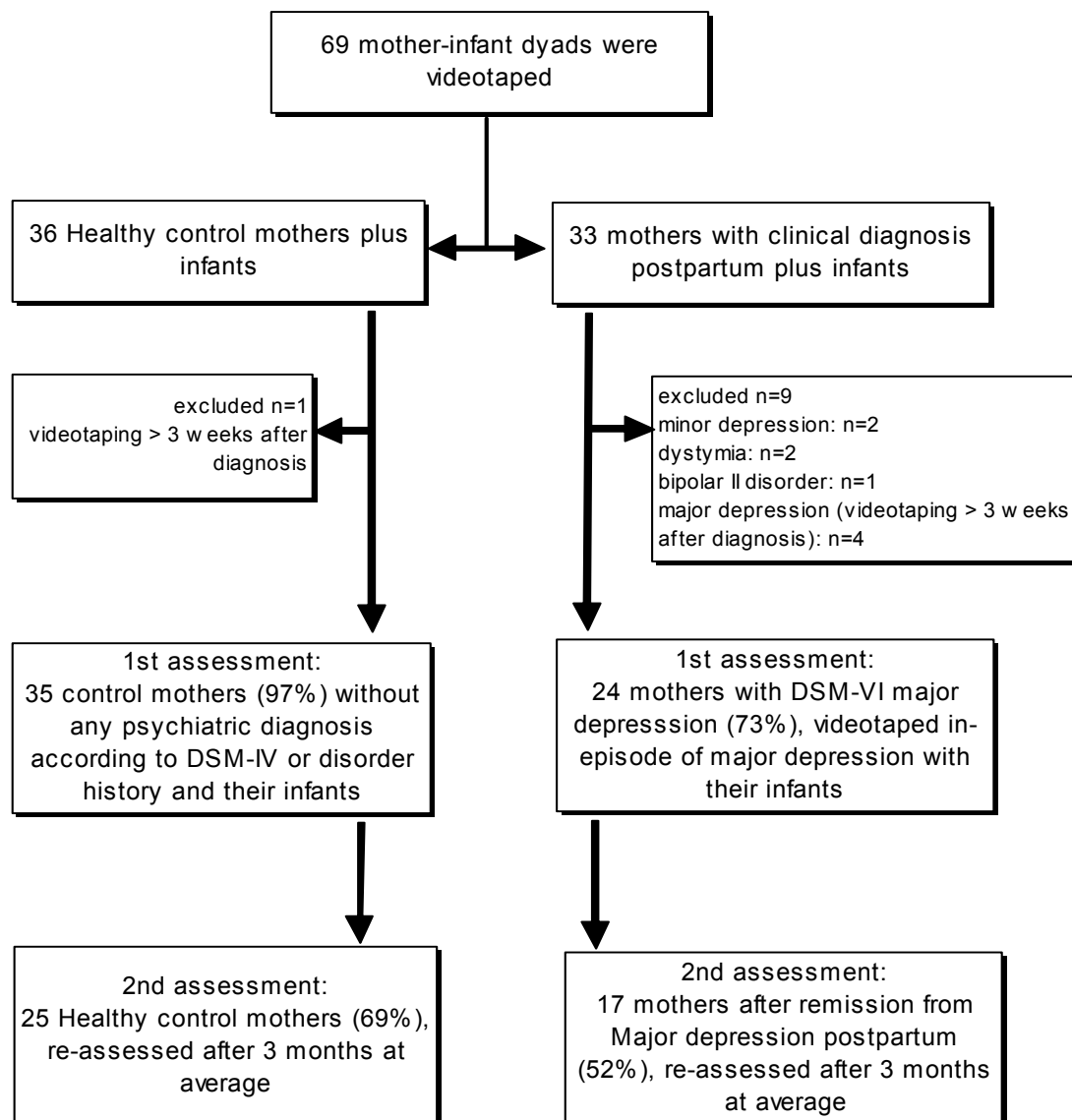
Mothers with major depression were asked to join an ongoing study where videotapes were taken of mother and infant. Control mothers were contacted in local maternity hospitals. A member of our research group approached the mothers, briefly described the study, and invited them to a videotaping session in our laboratory (for laboratory and procedure details please refer to page 116). Following criteria for inclusion and exclusion for the target group (table 5) were applied.

Table 5: Criteria for inclusion and exclusion

Inclusion Criteria
Diagnosis of major depressive episode according to DSM IV (at least 5 criteria fulfilled, with at least “depressed mood” or “diminished interest” criterion fulfilled)
Sufficient knowledge of the German language
Full-term infant, i.e. gestation time longer than 36 weeks
Healthy infant, without congenital abnormality
Exclusion Criteria
Maternal symptoms due to physiological effects, e.g., substance abuse
Lack of a clinical diagnosis according to the Structured Clinical Interview for the DSM-IV (SCID)
Bereavement
Manic, hypo-manic, or mixed episodes
Schizophrenia

We were finally able to recruit a sample of 59 mothers for the 1st assessment and 42 for the 2nd assessment as shown in the flowchart in figure 1. For the 1st videotaping 24 mothers in an acute episode of major depression and their infants were included. 35 mothers without any history of a psychiatric disorder and their infants were included as a control group. Although being unbalanced with respect to sample sizes, the groups allow for sufficient statistical power to detect differences in the primary target parameters (see power calculations, page 120). The clinical group was videotaped shortly (2-4 days) after being admitted to the hospital in an episode of depression.

Figure 1: Flow chart with initial and final sample sizes of mother-infant dyad recruitment including reasons for exclusion (e.g. time distance between diagnosis and video-take too long)



In the group of mothers with major depression (figure 2 and table 6) criteria with highest prevalence were symptoms such as fatigue or loss of energy, depressed mood, feelings of worthlessness, diminished interest and thoughts of death.

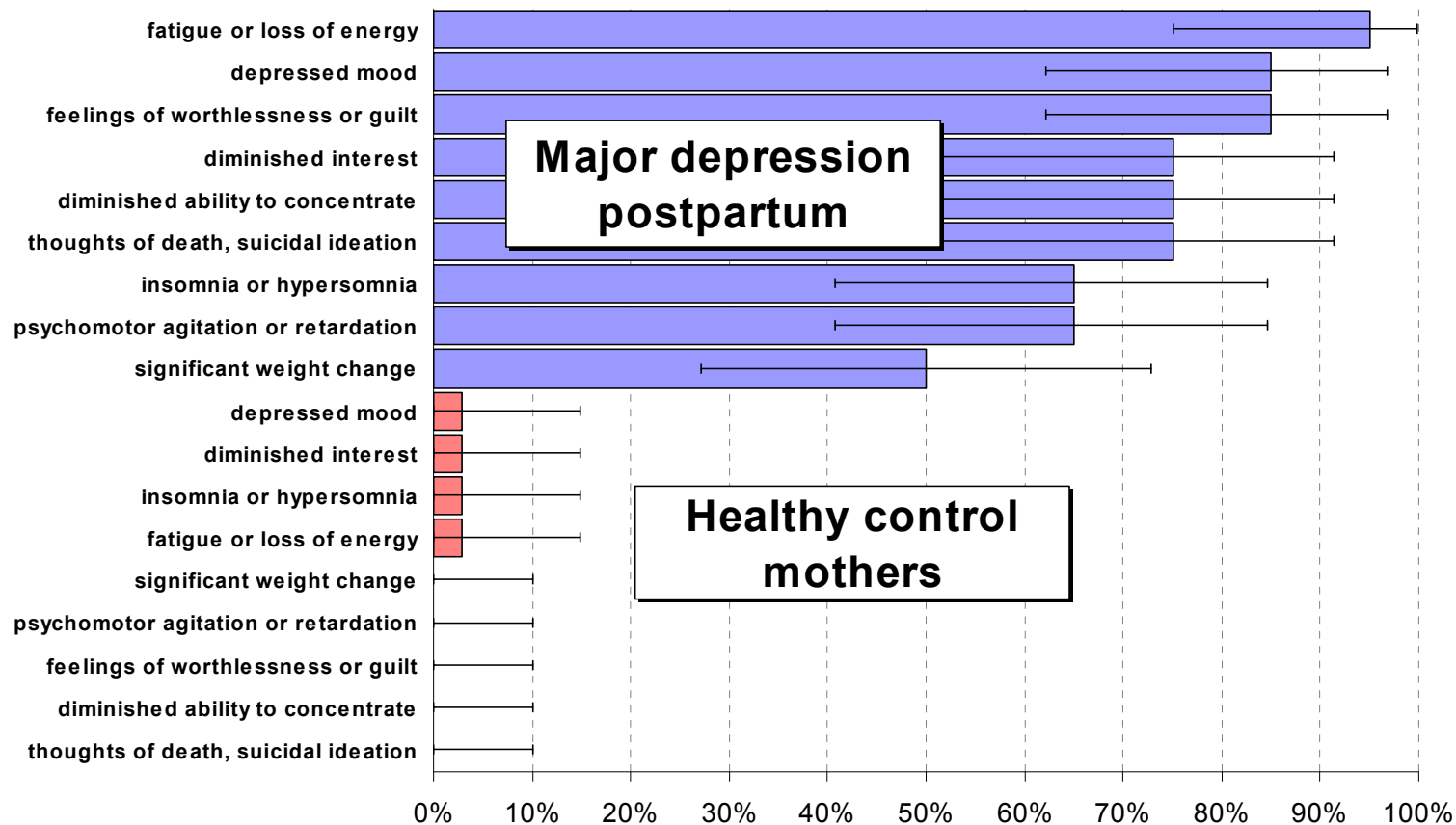


Figure 2: DSM-IV criteria for major depression in both groups sorted within each group by their rate of occurrence. Blue bars: sample of mothers with major depression, red bars: control mothers. All bars with 95%-confidence limits of estimation (Newcombe, 1998, estimation method 5 based on exact binomial tail areas). All data are for descriptive purposes only.

Table 6: Descriptions for DSM-V criteria, symptom prevalence and 95%-confidence intervals (CI)

	Major depression postpartum	subjects	lower 95% CI	upper 95% CI	cases	Healthy control mothers	subjects	lower 95% CI	upper 95% CI	cases
depressed mood	85%	17	62%	97%	20	3%	1	0%	15%	35
diminished interest	75%	15	51%	91%	20	3%	1	0%	15%	35
significant weight change	50%	10	27%	73%	20	0%	0	0%	10%	35
insomnia or hypersomnia	65%	13	41%	85%	20	3%	1	0%	15%	35
psychomotor agitation or retardation	65%	13	41%	85%	20	0%	0	0%	10%	35
fatigue or loss of energy	95%	19	75%	100%	20	3%	1	0%	15%	35
feelings of worthlessness or guilt	85%	17	62%	97%	20	0%	0	0%	10%	35
diminished ability to concentrate	75%	15	51%	91%	20	0%	0	0%	10%	35
thoughts of death, suicidal ideation	75%	15	51%	91%	20	0%	0	0%	10%	35

Note: data as available, for some patients only the overall SCID diagnosis was available

After an average of 3.0 months, mothers and infants were videotaped again (table 7), with an identical procedure. Both groups did not differ with respect to the interval between 1st and 2nd assessment ($p=0.126$, exact Mann-Whitney-test). Moreover, if an equivalence margin of ± 1 month is allowed then both groups can be considered equivalent with respect to their between-video-shoot distance ($p=0.042$, Mann-Whitney test modified for equivalence, i.e. test with shifted zero hypothesis, Wellek, 2002).

Table 7: Months between t1 and t2 video (t1 = mothers in episode, t2 = depressed mothers after remission, m = mean, sd = standard deviation, note: time based on date-differences, not all dates were available)

group	m	SD	min.	10th percentile	median	90th percentile	max.	cases
Major depression postpartum	3.5	2.7	0.7	1.2	2.4	6.6	11.2	17
Healthy control mothers	2.6	2.5	0.5	0.7	1.9	5.0	11.5	25
total	3.0	2.6	0.5	0.7	2.0	5.9	11.5	42

In addition, the groups did not differ in their cumulative chances of being reassessed (figure 3, log-rank test, $p=0.590$, Cox and Oakes, 1984).

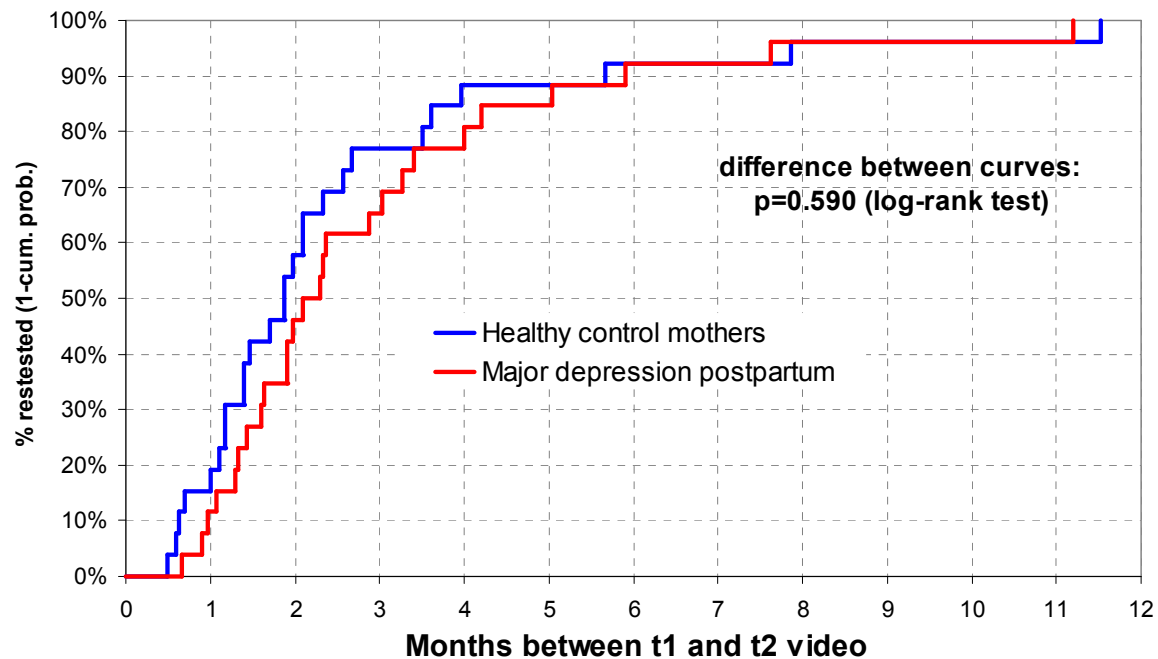


Figure 3: Cumulative percentage of dyads reassessed (estimation according to the method of Kaplan and Meier, 1958). Y-axis: 1 minus cumulative probability in percent, test for between-group comparison of reassessment: log-rank test (Cox and Oakes, 1984).

At the 2nd assessment, a total of 42 dyads could be reassessed: 25 control dyads and 17 dyads with a mother who had completely remitted from major depression. At 2nd assessment the groups did not differ both in the overall count (table 8) and in single criteria of depression (see figure 4; last column in table 9, page 113, exact Chi²-test).

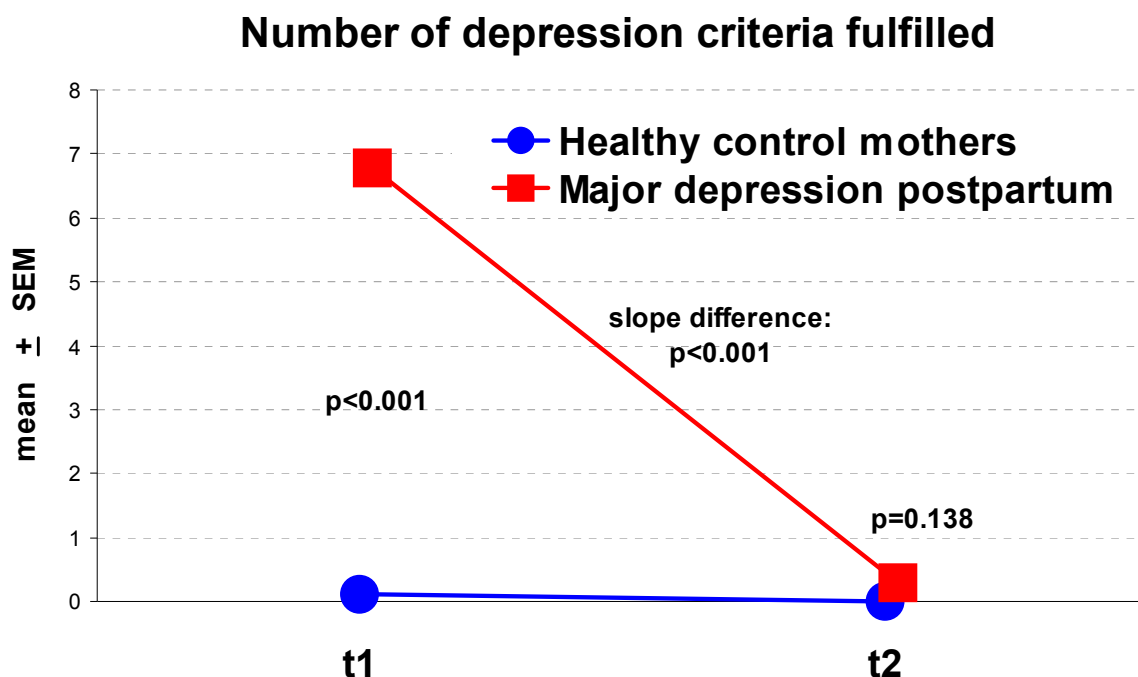


Figure 4: Depression-interview criteria fulfilled (t1 = mothers in episode, t2 = depressed mothers after remission, averages plus minus standard error of the mean, SEM). Test: Mann-Whitney test, exact, one-sided. All data as available (for some patients only the overall SCID diagnosis was available).

Please note that the figure 4 simply reflects the inclusion criteria (in the depression group only mothers with full remission were included at the 2nd point of measurement).

Table 8: Descriptive statistics (m = mean, se = standard error)

		Number of depression criteria fulfilled						
		m	se	min.	10th percentile	median	90th percentile	cases
Healthy control mothers	t1	0.1	0.1	0.0	0.0	0.0	0.0	36
	t2	0.0	-	0.0	0.0	0.0	0.0	25
Major depression postpartum	t1	6.8	0.3	5.0	5.0	7.0	8.7	24
	t2	0.3	0.2	0.0	0.0	0.0	1.0	14

Note: data as available, for some patients only the overall SCID diagnosis was available

If an equivalence margin of ± 1 depression criterion is set, then at the 2nd assessment both groups can be considered equivalent ($p < 0.001$, Mann-Whitney test modified for equivalence; Wellek, 2002).

Moreover, there were no differences with respect to single criteria of depression (table 9).

Table 9: DSM-IV single criteria of the clinical interview (SCID, -- not present, (+) present but not criterion-like, ++ present), statistical test for between group comparisons: exact Chi²-test, all tests exploratory, i.e. for descriptive purposes only.

t1: Depressed in episode						t2: After Remission							
		Major depression postpartum n = 24		Healthy control mothers n = 35		p-value		Major depression postpartum (n=17)		Healthy control mothers (n=25)		p-value	
depressed mood	--		0%	34	97%	p<0.001	11	92%	24	100%	p=0.333		
	(+)	3	15%		0%		1	8%		0%			
	++	17	85%	1	3%			0%		0%			
diminished interest	--	3	15%	34	97%	p<0.001	9	75%	24	100%	p=0.031		
	(+)	2	10%		0%		3	25%		0%			
	++	15	75%	1	3%			0%		0%			
significant weight change	--	5	25%	34	97%	p<0.001	11	92%	24	100%	p=0.333		
	(+)	5	25%	1	3%			0%		0%			
	++	10	50%		0%		1	8%		0%			
insomnia or hypersomnia	--	4	20%	34	97%	p<0.001	11	92%	24	100%	p=0.333		
	(+)	3	15%		0%			0%		0%			
	++	13	65%	1	3%		1	8%		0%			
psychomotor agitation or retardation	--	5	25%	35	100%	p<0.001	10	83%	24	100%	p=0.102		
	(+)	2	10%		0%		1	8%		0%			
	++	13	65%		0%		1	8%		0%			
fatigue or loss of energy	--	1	5%	34	97%	p<0.001	11	92%	24	100%	p=0.333		
	(+)		0%		0%		1	8%		0%			
	++	19	95%	1	3%			0%		0%			
feelings of worthlessness or guilt	--	2	10%	35	100%	p<0.001	11	92%	24	100%	p=0.333		
	(+)	1	5%		0%		1	8%		0%			
	++	17	85%		0%			0%		0%			
diminished ability to concentrate	--	1	5%	35	100%	p<0.001	10	83%	24	100%	p=0.105		
	(+)	4	20%		0%		2	17%		0%			
	++	15	75%		0%			0%		0%			
thoughts of death, suicidal ideation	--	4	20%	35	100%	p<0.001	11	92%	24	100%	p=0.333		
	(+)	1	5%		0%			0%		0%			
	++	15	75%		0%		1	8%		0%			

-- not present

(+) present but not criterionlike

++ present

Note: data as available, for some patients only the overall SCID diagnosis was documented.

Differences between subjects who dropped out and subjects who could be assessed twice were also analyzed (table 10 and table 11): Mothers, who could be reassessed, compared with those who were not reassessed, did not statistically differ.

Table 10: Subjects who dropped out compared to subjects who could be assessed twice. All tests exact and exploratory, statistical test: Mann-Whitney-U test (m = mean, sd = standard deviation, n = number of cases with available data). Total sample size: n = 59.

	not re-assessed n = 17							re-assessed n = 42							descriptive test
	m	sd	min	md	max		n	m	sd	min	md	max		n	
maternal age (years)	31 ±	6	(24	30	41)		15	33 ±	4	(25	33	40)		42	p=0.159
T1 age child (months)	4.4 ±	2.7	(1	4	11)		17	3.8 ±	2.3	(1	3	9)		42	p=0.437
child number	1.4 ±	0.6	(1	1	3)		17	1.6 ±	0.9	(1	1	4)		42	p=0.511

Table 11: Subjects who dropped out compared to subjects who could be assessed twice. All tests exact and exploratory (statistical test: Fisher-test for 2 x 2 - tables, Chi²-test for r x c - tables). Total sample size: n= 59 (data as available).

	not re-assessed n = 17		re-assessed n = 42		descriptive test
no comorbidity	12	71%	32	76%	p=0.745
comorbidity	5	29%	10	24%	
low / intermed. secondary level	6	35%	15	36%	p=0.547
qualific. for univ. entrance	6	35%	9	21%	
university degree	5	29%	18	43%	p=1.000
no antidepressants	3	50%	7	41%	
antidepressants	3	50%	10	59%	p=1.000
no neuroleptics	5	83%	15	88%	
neuroleptics	1	17%	2	12%	p=1.000
no benzodiazepine	6	100%	15	88%	
benzodiazepine	0	0%	2	12%	p=1.000
no phase prophylactic	6	100%	16	94%	
phase prophylactic	0	0%	1	6%	

The proximity in time between depression diagnosis and videotaped interactions is shown in table 12 below. The median is 0 days. The interquartile range is also 0. The 10th to 90th percentile is between -7 (video-shoot before clinical interview) and +6 days (video-shoot after clinical interview). Both groups do not differ (p=0.85, Mann-Whitney test; compare median values in table 12). Moreover, with an equivalence margin of ± 7 days, both groups can be considered equivalent (p<0.001, Mann-Whitney test modified for equivalence; Wellek, 2002).

Table 12: Days between depression diagnosis and video-shoot compare median values

	10th percentile	25th percentile	median	75th percentile	90th percentile	cases
Healthy control mothers	0	0	0	0	0	31
Major depression postpartum	-8	-7	0	4	17	21
total	-7	0	0	0	6	52

Note: Sample size: n=59. Values not available: days between SCID-diagnosis and video-shoot n=7. Positive values denote that video recording was after the clinical interview (SCID). Negative values denote that the recording was before the interview.

6.2. Sample descriptions

The mothers were on average 32 years old at the time of delivery (table 13; standard deviation 5 years) and had on average 1.6 children (standard deviation 0.8). The infants were on average 3.9 months old (standard deviation 2.4 months) with an interquartile age range between 2 and 5 months and a total age range between 1 and 11 months.

Table 13: Sample descriptors (m = mean, n = number of cases). Data as available

	m	sd	min	25th	md	75th	max	n
maternal age (years)	32	5	(24	29	33	36	41)	57
T1 age child (months)	3.9	2.4	(1	2	3	5	11)	59
child number	1.6	0.8	(1	1	1	2	4)	59

Note: m = mean, sd = standard deviation, min/max = minimum/maximum value.

Almost all of the mothers were living with a partner (98%, table 14). All mothers were Caucasian. The gender ratio of the infants was 56% versus 44% and not deviant from an equal distribution ($p=0.36$, Chi²-goodness of fit test). 36% had a low or an intermediate secondary level education. 25% of the mothers had the qualification for university entrance, 39% a university degree. 57% of mothers with major depression received antidepressants. The postpartum incidence rate of depression was 50%, i.e. those in the depressed group had a depression after birth and for the first time. 63% of them had co-morbid diagnoses (anxiety, compulsive disorder, eating disorder, personality disorder). 61% received medication as part of the treatment of their depressive disorder, 57% with antidepressants for at least 6 months after remission, 13% with neuroleptic medication, and 9% with benzodiazepines for less than 2 weeks. Both study groups did not differ in partner status, infant gender and education level.

Table 14: Descriptive statistics of samples (statistical test: Fisher-test, Agresti, 1990; or exact Chi²-test for larger contingency tables, data as available from the clinical documentation).

		total n = 59	Healthy control mothers n = 35	Major depression postpartum n = 24	descriptive test
partner	not living with partner	1 2%	1 3%	0 0%	p=1.000
	living with partner	57 98%	34 97%	23 100%	
male infant gender (y/n)	female	26 44%	14 40%	12 50%	p=0.594
	male	33 56%	21 60%	12 50%	
depression history	no depression history	47 80%	35 100%	12 50%	p<0.001
	depression history	12 20%	0 0%	12 50%	
comorbidity (y/n)	no comorbidity	44 75%	35 100%	9 38%	p<0.001
	comorbidity	15 25%	0 0%	15 63%	
education	low / intermed. secondary level	21 36%	12 34%	9 38%	p=0.378
	qualific. for univ. entrance	15 25%	7 20%	8 33%	
	university degree	23 39%	16 46%	7 29%	
antidepressants	no antidepressants	10 43%	0	10 43%	-
	antidepressants	13 57%	0	13 57%	
neuroleptics	no neuroleptics	20 87%	0	20 87%	-
	neuroleptics	3 13%	0	3 13%	
benzodiazepine	no benzodiazepine	21 91%	0	21 91%	-
	benzodiazepine	2 9%	0	2 9%	
phase prophylactic	no phase prophylactic	22 96%	0	22 96%	-
	phase prophylactic	1 4%	0	1 4%	
any medication given	no	9 39%	0	9 39%	-
	yes	14 61%	0	14 61%	

6.3. Laboratory environment and procedure including Tronick's face-to-face still-face paradigm

Mother and infants were videotaped in Tronick's face-to-face still-face paradigm (Tronick, Brazelton and Als, 1978). The still-face paradigm has been applied in a range of studies with the intention of quantifying mother-infant interaction in a standardized way and of assessing how both interactants react before and after maternal responsiveness is artificially restricted, i.e. a variation in caring behavior or mother-infant communication is artificially induced (the mother is asked to keep a "still face" and to totally interrupt any communication with her infant).

The mother and infant were guided into a video room equipped with an infant seat, which was fixed on a table and an adjustable swivel stool for the mother. The mother was seated on the stool facing the infant and was asked to play with her infant as she would normally do. There were two cameras. One focused on the mother, the other on the infant. The babies were placed in the infant seat in a relatively upright position. The height of the desk allowed the adult to talk to and look at the baby from a distance of 0.5 m at the maximum. A camcorder on a tripod was put behind and to one side of the mother, allowing for a simultaneous recording of both the baby and the mother.

The instructions for the still-face paradigm were as follows: "During the next few minutes you should have a good time with your child. Please behave as you do at home, don't do anything special. Do not use a pacifier or toys. Please don't take your child out of the baby seat, nor move the table. After two minutes, I will knock at the window. Please stop immediately, turn away from your child, count to 10 in your mind, then turn again to your child. From now on, please don't look at your child, but focus on a point behind him/her. Please don't talk to your child, don't touch your child nor take your child out of the baby-seat. Please remain in this position for two minutes until you hear a knock at the window. Afterwards, please play normally again with your child for two minutes."

Effects associated with a maternal still-face have been reported by Field, Vega-Lahr, Scafidi and Goldstein (1986), Gusella, Muir and Tronick (1988), Mayes and Carter (1990), Toda and Fogel (1993), Tronick, Brazelton and Als (1978), Weinberg and Tronick (1996) and Stanley, Murray and Stein (2004). One particular reported effect was that in normal infants, positivity, activity as well as monitoring of the mother are

reduced, followed by heightened frequencies of eliciting behavior or protest, or monitoring of the environment, behaviors that were reported to continue after interruption of the period of maternal still-face. Only a few studies used this standardized provocation procedure (maternal still-face) in mothers with major depression - a procedure that Cohn and Tronick (1983) or Weinberg, Olson, Beeghly and Tronick (2005) described as a stressor for both mother and child.

The mother and her infant were videotaped in 3 phases of 2 minute each. First, a 2-minute face-to-face play sequence for which we instructed the mother to play with the infant "as she would do at home". Then, a 2-minute still-face sequence for which the mother was instructed to keep a still face, to look in the direction of her infant, but to focus on a point on the infant's chair behind and above his or her head. The mother was instructed not to smile, talk or touch her infant. In the last phase of 2-minute duration, the mother was instructed to play with her infant once again.

The signals from both infant camera and maternal camera were combined to a single "split-screen image". On one half of the image we recorded a simultaneous view of the mother's front, her face, hands, and upper body; and on the other half of the image the infant's entire body.

6.4. Tronick's Still-face paradigm – reasons for its application

Tronick's still-face paradigm has been extensively applied in numerous studies with depressed mothers to elicit current impairments in mother-infant interactions, or, to predict future impaired child developments (refer to table 3 on page 43). The still-face situation has been regarded as a situation where the mother is emotional unavailable. The unavailability-effect has been shown by the affect-related observational data of the still-face period. Other situations such as leaving the room, or, simulating a depression-like behavior (Cohn and Tronick, 1983) were hypothesized to be less effective on infant behavior (Field, 1992) compared to the emotional unavailability situation.

We applied the still-face paradigm since several authors reported a specific response in infants of depressed (e.g., Field, 1992). These infants were found to be unimpressed by the unavailability situation. They continued to show less positive behavior, less motor activity, gaze aversion, distress brow, and crying. Accordingly, Field et al. (2007) concluded that a maternal unavailability situation seems to be less distressing to the infants of depressed versus those of non-depressed mothers.

Moreover, we applied the still-face paradigm to test for the effects of the repeated measurement from in-episode to after-remission. Infants of depressed mothers have been claimed to generalize over situations, e.g., infant-withdrawal and depression-like infant-affects were found to continue when interacting with a non-depressed adult (Field, 1992, 1988). Also, Pelaez-nogueras et al. (1994) found that children of depressed developed a depressed style of interaction which also generalized to other interaction partners.

6.5. Study design and flow chart of study phases

Because we observed two independent groups running over three phases (figure 5) and used several dependent measures (table 20 on page 126, as well as table 21 and 22), the design can be considered as a multivariate parallel-group design with repeated measurement (Winer, Brown and Michels, 1991) on two factors (phases within one assessment and the two assessments themselves). The unit of the observation is the dyad, i.e. the mother and infant (in a total of 59 dyads).

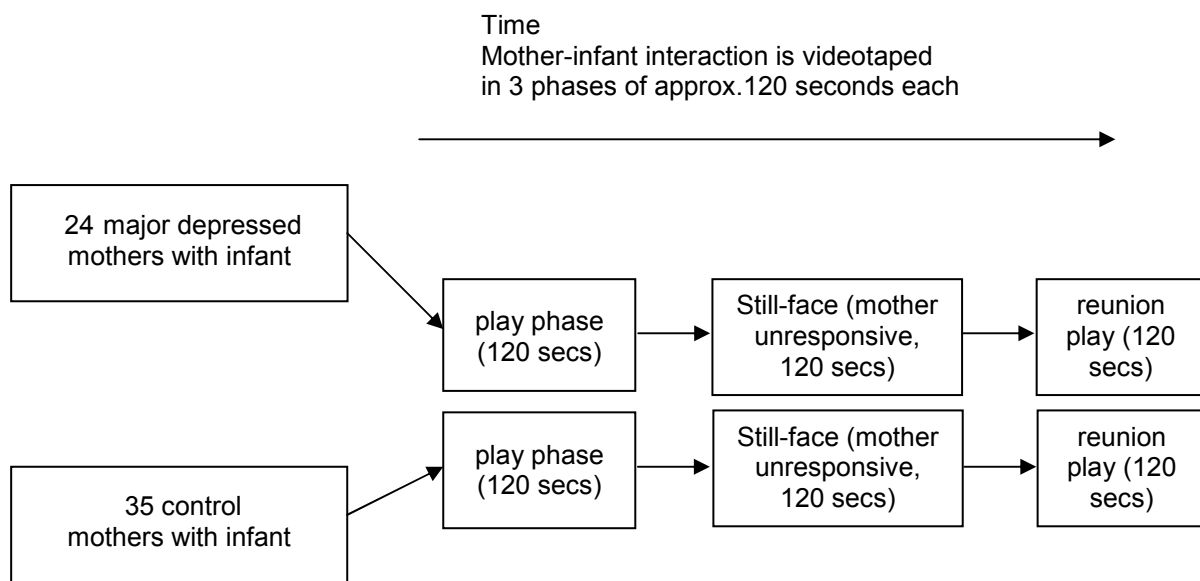


Figure 5: Study phases (this scheme was repeated twice, i.e. in episode and after remission, twice for control dyads)

No stratifications by sex or infant-age were made due to sample size and subsequent power restrictions (i.e. a high risk of false-negative effects). Nevertheless, effect-distorting variables will be analyzed by analyses of covariance, by recently developed multivariate matching techniques based on the propensity score method (Rubin and Thomas, 1996; D'Agostino, 1998) and another method for the correction of selection bias such as Heckman's two-stage procedure (Heckman 1979; 2000; Winship and

Mare 1992; Lung-Fei, 1983).

6.6. Definition of group allocation - assessment of major depression

Maternal major depression was assessed using the Structured Clinical Interview for DSM-IV, Axis I (SCID-I, Wittchen, Wunderlich, Gruschwitz and Zaudig, 1997; First, Spitzer, Gibbon and Williams, 1996). Interviewers were trained student assessors (according to Ventura et al., 1998) so that experienced as well as new interviewers were able to achieve and maintain sufficient reliability and diagnostic accuracy. The Structured Clinical Interview is a semi-structured instrument (Spitzer, Williams, Gibbon and First, 1992), that allows for a reliable and valid assessment and diagnosis of Axis I syndromes and disorders based on the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV).

A range of studies is available with measures for interrater or retest agreement (corrected for chance agreement; Cohen's kappa, see table 15 below), which pointed to at least sufficient (0.61; Zanarini et al., 2000) to excellent reliabilities (0.93; Skre et al., 1991).

Table 15: SCID reliabilities (SCID = Structured Clinical Interview)

Authors	Sample size	Kappa for Major Depressive Disorder
Skre et al., 1991	N=54	0.93
Zanarini et al., 2000	N=27	0.80
Zanarini et al., 2000	N=52	0.61
Segal et al., 1995	N=40	0.90
Williams et al., 1992	N=592	0.64
Zanarini and Frankenburg, 2001	N=45	0.90
Zanarini and Frankenburg, 2001	N=30	0.73

Instrument validities are mostly given in reference to or as agreement with a "gold standard". Currently, however, there is no diagnostic "gold standard" in psychiatry, with the exception that the SCID itself has often been used as kind of standard, e.g., to check for accuracy of clinical diagnoses or self-ratings. Spitzer suggested that the "best estimate diagnosis" should be used as reference to check for diagnostic accuracy. This implies a longitudinal diagnostics assessment by experts, use of all available patient data, e.g. from family members, medical records, and also involves having clinical staff observe the patient. Based on these procedures, the SCID was reported to have a higher validity compared with standard interviews (Basco et al., 2000; Fennig et al., 1994; Kranzler et al., 1996, 1995).

6.7. Quantitative sample size considerations - Power analyses

Sample sizes and their respective power are calculated based on either the expectation of impairments under major depression compared to controls, or improvements after remission (relative to those in control dyads; accordingly). The power calculation is based upon the following parameters (short survey in table 16; refer to details on page 128 or 126).

Table 16: Basic survey of primary target parameters used for the power calculation

parameter	coding	data stream / window	operationalization	unit of observation
"affect mirroring"	codes regarding positive (e.g. facial) affect expressions	codes occurring at the same time, both interactants	proportion of overall observation time	dyad
onset of "affect mirroring"	onset of parallel affect expressions (positivity only)	onset of codes occurring at the same time	incidence of cases over time who met criterion ("affective mirroring")	dyad
infant stimulation behavior	all affect-related behaviors emitted by the mother	codes emitted by one interactant only	proportion of overall observation time	mother
infant activity	all affect-related behaviors emitted by the infant	codes emitted by one interactant only	proportion of overall observation time	infant

Note: For details of parameter derivation see also table 22 on page 128 (for primary target parameters), table 20 and table 21 on page 126 for affect-codings.

The respective critical values are based on differences of means. Regarding the absolute effect to be expected several sources were used, e.g., Field (1984), and, Field, Healy, Goldstein, Perry, Bendell, Schanberg, Zimmermann and Kuhn (1988), and finally, Field, Healy and Leblanc (1989). They found that the rates of affect-expressions of control subjects almost doubled those of dyads with a depressed mother. Moreover, Field, Healy and Leblanc (1989, Figure 3 on page 365) found in controls that positive affects were parallel at approximately 21% (time proportions of 0.21) of time and 7% of time in dyads with a depressed mother (time proportions of 0.07). Thus, an expected difference in time spent in positive affects might be at least 0.14 (in units of time proportions). Field, Healy, Goldstein, Perry, Bendell, Schanberg, Zimmermann and Kuhn (1988) found a difference of 0.25 in favour of control dyads (in units of time proportions where affect-codes of mother and infant were parallel). Standard deviations were displayed ranging as large as the some difference of means (e.g. SD = 0.20).

These effect measures, however, seem to rely on very optimistic expectations and may reflect an overestimation. In a first step a mean difference of 0.20 with a

comparable standard deviation of 0.20 has been used (rounded middle between 0.14 and 0.25, see above). This mean difference and standard deviation result in a Cohen's $D = 1$, since $D = (\mu_1 - \mu_0) / \sigma = 0.20 / 0.20 = 1$.

Clearly, this value exceeds the classical Cohen-limit of a large-sized difference of 0.8. Therefore, all power calculations were confronted with a classical Cohen's D of 0.8 and $D = 0.5$.

Regarding the improvement after the depression remission a change in affect expression proportions of 0.20 is expected (using the same difference as the between-group difference; for references see above), based on a known standard deviation of 0.20. Again these values were confronted with classical Cohen's D of 0.8 and 0.5 to compare resulting powers.

Calculations were made based on an adjusted type I error of 0.007.

All listed effect measures (and the smaller ones based on Cohen's D of 0.8 and 0.5) basically reduce to two power scenarios (refer to table 17):

In case of a Cohen's D of 0.8 a power of 0.59 can be expected (right column in table 17). The power of 0.84 (Cohen's D = 1, data from the literature) might include a risk of being erroneously too high and represent an overestimation. The follow up measures have a power of 0.64 if the literature data is used (it reduces to only 0.4 in case of a Cohen's D = 0.8).

Table 17: Basic scenarios for the sample size calculation (except for hypothesis 2; here, a time to event-analysis was applied, see below; n_0 and n_1 = sample of control and clinical group, α = type I error)

target parameter	unit	expectation	measure	mean diff.	SD	Cohen's D	method	n_0	n_1	α	power
interaction time with positive affect codes occurring	proportion of total observation time	lower under major depression	between-group difference	0.20	0.20	1	t-test	34	25	0.007	0.84
replication of the calculations above with classical Cohen's D limits (D = 0.8 and D = 0.5)						0.8	t-test	34	25	0.007	0.59
						0.5	t-test	34	25	0.007	0.19
improvement of interaction time with positive affect codes occurring	proportion of total observation time	higher under major depression; not changed in controls	between-group difference of post minus pre differences	0.20	0.20	1	t-test	24	18	0.007	0.64
replication of the calculations above with classical Cohen's D limits (D = 0.8 and D = 0.5)						0.8	t-test	24	18	0.007	0.40
						0.5	t-test	24	18	0.007	0.12

Note: For details of parameter derivation see also table 22 on page 128 for primary target parameters, table 20 and table 21 on page 126 for original behavior codings.

Sample sizes were calculated based a two-Sample t-test power analysis using SAS software (SAS, 2005). Al-Sundugchi (1990) suggested that the power for the Mann-Whitney test may be calculated based on standard t-test formulas. For the explicit confirmatory hypotheses and details of the 1st- type error-adjustment see page 97. Hypotheses and the respective expectations are listed below.

Hypothesis 1 (**lowered affective mirroring¹ in presence of major depression**):

Table 18: Estimation results, also confronted with classical Cohen's D limits of 0.8 and 0.5 (refer to column 4)

method	mean difference	sd	Cohen's D	n ₀	n ₁	α	power	evaluation of effect measure
independent t-test (SAS proc power)	0.20	0.20	1	34	25	0.007	0.84	probably too optimistic
			0.8	34	25	0.007	0.59	based on a Cohen limit of D = 0.8
			0.5	34	25	0.007	0.19	based on a Cohen limit of D = 0.5

Based on the expectation that controls are simpler to recruit and thus better obtainable (assumed ratio of 1.3, i.e. ratio of controls versus major depressed), and the expectation of lowered interaction time with parallel positive affects (between-group difference of means of 0.20 and a known standard deviation of 0.20, according to data of the literature), resulting in a critically optimistic and possibly inflated Cohen's D = 1, group sample sizes of 34 control dyads and 25 with major depression achieve a power of 0.84 to detect a difference of 0.20. However, when confronted with a classical Cohen's limit of D = 0.8, the power reduces to 0.59 (in case of a D = 0.5 it reduces to 0.19).

Under hypothesis 2, a **much slower onset of affective mirroring** is expected in dyads where the mother is diagnosed with major depression. This hypothesis includes a time-to-event analysis, the sample-size estimation for the log-rank test was applied and the procedure recommended by Lachin and Foulkes (1986) was used (based on the validated software NNPar). A two-sided log-rank test with an overall sample size of 59 subjects (of which 34 are in the control group and 24 are in the depressed group) achieves a power of 0.63 at an adjusted 0.007 significance level to detect (no data from the literature were available) a difference in proportions (this time "proportions" refer to the incidence of cases who reach the criterion, 0.75 of the control subjects and only 0.50 of the dyads with a major depressed mother are expected to reach the criterion of sharing affects at the same time) assuming no loss of follow-up during the observation of interest (1st assessment, i.e. depressed in episode).

Hypothesis 3 (**infants of depressed mothers are less affected if the mother is**

¹ The term "affective mirroring" refers to that interaction time where positive emotions occur at the same time in both interactants; it is included as proportion, i.e. relative to the total interaction time.

unavailable) predicts that the infants of depressed mothers are not affected by the maternal still-face procedure (no change and a zero mean of differences whilst infants of control subjects show increases). Based on the group-ratio of 1.3 mentioned above (i.e. ratio of controls versus major depressed), group sample sizes of 25 depressed and 34 controls (assuming a critically high and possibly inflated Cohen's $D = 1$) achieve 0.84 power to detect an average change difference of 0.20 in affect mirroring proportions (pre-post maternal still-face situation) with known group standard deviations of 0.20 and with an adjusted significance level (α) of 0.007 based on a t-test (also refer to the previous validation table). However, when confronted with a classical Cohen's limit of $D = 0.8$, the power reduces to 0.59 (in case of a $D = 0.5$ it reduces to 0.19).

Hypothesis 4 (**lack of infant stimulating behavior under major depression**) predicts that major depressed mothers have lower stimulation durations (in units of time proportions). Based on group allocations (ratio of 1.3 controls versus major depressed mothers) and the expectation that the stimulation proportions are (relatively) lower by an average of at least 0.20, then group sample sizes of 25 depressed and 34 controls (under probably too optimistic and possibly inflated conditions of $D = 1$) a power of 0.84 based on known group standard deviations of 0.20 and with an adjusted significance level (α) of 0.007 (two-sided t-test). However, when confronted with a classical Cohen's limit of $D = 0.8$, the power reduces to 0.59 (in case of a $D = 0.5$ it reduces to 0.19).

Hypothesis 5 (**deviant activity level in infants of depressed mothers, i.e. either hypo-active or hyper-active**) predicts that infants of major depressed mothers have activity levels that are too low or too high in the sense of time proportions when the infant is active. Based on the aforementioned group allocations (ratio of 1.3 controls versus major depressed mothers) and the expectation of a deviancy of activity-levels of at least 0.20 from control infants, group sample sizes of 25 depressed and 34 controls achieve a power of 0.84 (again assuming a critically high and possibly inflated Cohen's $D = 1$) based on known group standard deviations of 0.20 and with an adjusted significance level (α) of 0.007. However, when confronted with a classical Cohen's limit of $D = 0.8$, the power reduces to 0.59 (in case of a $D = 0.5$ it reduces to 0.19).

Hypothesis 6 (disappearance of impaired affect mirroring after remission):

Table 19: Estimation results, also confronted with classical Cohen's D limits of 0.8 and 0.5 (refer to column 4)

method	mean difference ²	sd	Cohen's D	n ₀	n ₁	α	power	evaluation of effect measure
independent t-test (SAS proc power)	0.20	0.20	1	24	18	0.007	0.64	probably too optimistic
			0.8	24	18	0.007	0.40	based on a Cohen limit of D = 0.8
			0.5	24	18	0.007	0.12	based on a Cohen limit of D = 0.5

The hypothesis predicts a reinstatement of affect mirroring after depression remission. Based on the expectation that dyads with a depressed mother show an increase (+0.20) compared to controls (± 0), based on the standard deviation of above (assuming a critically high and possibly inflated Cohen's D = 1), and based on a group-ratio of 1.3 (ratio of controls versus major depressed with comparable attrition rate) group sample sizes of 18 depressed and 24 controls achieve a power of 0.64 with an adjusted significance level (α) of 0.007, based on a two-sided t-test. Again, when confronted with a classical Cohen's limit of D = 0.8 the power reduces to 0.40 (in case of a D = 0.5 it reduces to 0.12).

Hypothesis 7 (infant disturbances transient after major depression remits) predicts an overall reduction in negative child behaviors after depression remission. Based on the expectation that infants of depressed mothers show a marked negativity decrease (protest, withdrawal, -0.20) compared with controls (± 0). Based on the standard deviation of above (assuming a critically high and possibly inflated Cohen's D = 1) group-ratio of 1.3 (ratio of controls versus major depressed), group sample sizes of 18 depressed and 24 controls achieve a power of 0.64 with a standard deviations of 0.20 and with an adjusted significance level (α) of 0.007, again, based on a two-sided t-test (also refer to the previous validation table). Again, when confronted with a classical Cohen's limit of D = 0.8 the power reduces to 0.40 (in case of a D = 0.5 it reduces to 0.12).

6.8. Dependent measures**6.8.1 Operationalization of behavior measures**

The behavior of both mother and infant was coded in separate runs by trained and

² post minus pre changes when compared between both groups

independent observers who were blind to both the study hypothesis and the maternal diagnosis. The observers used a standardized video coding system, the “Noldus Observer” (Noldus, 2004) and assigned predefined behavior codes (see table 20) with an accuracy of 1/10 second to videotaped behavior on an event coding basis (Roberts and Forehand, 1978). Any change of behavior resulted in a new assignment, so that behavior on- and offsets, as well as behavior duration could be registered. The Noldus Observer allows for the viewing of a video and assign behavior codes in real-time (“baby protests”, “mother is withdrawn”, “baby looking at environment / baby’s focus not on mother”). Whenever the behavior of the baby or the mother changes (e.g., attends to mother), a code combined with the time of occurrence can be assigned. It is possible to reverse the video at modified speed (e.g., low speed) to check for behaviors which occur with high density.

The infant and maternal behaviors were coded using the “Infant and Caregiver Engagement Phases” (Tronick, 1978; Weinberg and Tronick, 1998); a derivate of the Monadic Phases System (Cohn and Tronick, 1987; Tronick, Als and Brazelton, 1980; Matias R, Cohn JF and Ross S, 1990). Two coders unaware of both maternal diagnosis and clinical hypotheses independently assigned codes for the infant (table 20 and table 21), for behavior including gaze behavior (looks at mother, at objects and scans), vocalizations (neutral, positive, crying), interacting with an object (e.g., own hands), or a person, pick-me-up and other gestures, maternal facial affects as well as her vocalizations.

Table 20: Basic dependent measures for the infant: behavior codes, applied with the behavioral coding system “Noldus observer” after videotaping three 2 minute phases of the face-to-face still-face paradigm.

	Codes for the infant given in each of the 3 phases of the still-face paradigm	Codes assigned	Status of parameter in statistical analysis
1	Infant shows negative engagement (e.g., cries, shows negative facial expressions, protests, is withdrawn)	ineg	exploratory
2	Infant is protesting	ipro	exploratory
3	Infant is withdrawn	iwit	exploratory
4	Infant looks at objects (e.g., looking at own hand, seat, camera or environment with a neutral facial expression)	inon	exploratory
5	Infant is attending to caregiver (e.g., gazing at mother)	ineu	exploratory
6	Infant shows positive engagement (e.g., facial expression of joy, smiles, looks at mother)	ipos	exploratory

(List of dependent variables continued in table 21.)

Table 21: Basic dependent measures for the caregiver, applied with the behavioral coding system “Noldus observer” after videotaping three 2 minute phases of the face-to-face still-face paradigm.

	Codes for the mother given in each of the 3 phases of the still-face paradigm	Codes assigned	Status of parameter in statistical analysis
7	Caregiver shows negative engagement (e.g., mother withdrawn, intrusive or hostile)	cneg	exploratory
8	Caregiver is rated hostile or intrusive	chos	exploratory
9	Caregiver is withdrawn	cwith	exploratory
10	Caregiver is non-infant focused (focuses on environment, own clothes, etc.)	cnon	exploratory
11	Caregiver is infant-focused without vocalization (e.g., looks at infant with neutral face)	cneu	exploratory
12	Caregiver is infant-focused, neutral face, with vocalization	cpvc	exploratory
13	Caregiver shows positive engagement (e.g., mother is gazing at infant, is interested, smiles, vocalizes, laughs)	cpes	exploratory
14	Caregiver exaggerates positive engagement (e.g., exaggerated laughter, exaggerated play)	cexg	exploratory

(List of dependent variables continued in table 22 overleaf.)

Additional measures of behavior-state matching or measures quantifying latencies until behavior overlaps occur were derived. Two of them (positivity overlap and the pre-post still-face changes, measures 15 and 16) will be used in confirmatory statistical tests; the other derived measures will be used in exploratory analyses.

Table 22: Derived measures for the confirmative statistical tests (refer to table 20 and maternal codes in table 21) in time stream, predominantly matching behaviors or overlaps, i.e. parallel occurring behavior of mother and infant

Dependent variable	Label	Behavior	Combination of abbreviations of table 20 and table 21	Status of parameter in statistical analysis
15	Positive affect sharing, multimodal mirroring of positive affect,	Infant is attending to caregiver or is positively engaging, paralleled by a caregiver who is positively engaging or vocalizing. Overall time in percent of total observation time will be used for statistical analysis.	ineu, ipos, cpvc, cpos	1 st confirmatory hypothesis
16	Regain of multimodal mirroring / affect sharing	Cumulative probability until dyads regain affect mirroring, time until criteria for previous parameter are met again.	ineu, ipos, cpvc, cpos	2 nd confirmatory hypothesis
17	Non-affectedness of infants if a mother is unavailable	Changes in multimodal mirroring / affect sharing before and after the still-face phase.	Operationalization like 1 st primary parameter, refer to line 15 above	3 rd confirmatory hypothesis
18	Caregiver initiates stimulation of the infant	All positive infant engagement behaviors emitted by the mother, all parameters in table 21 intra-individually summed.	cpvc, cpos, cexg	4 th confirmatory hypothesis
19	Infant activity level	Sum of infant engagement behaviors.	ineg, ipro, ineu, ipos	5 th confirmatory hypothesis

6.8.2 Interrater reliabilities

Interobserver reliabilities were assessed by using a randomly drawn subsample of 17 videos ($\approx 30\%$, randomly drawn by coders) for which behavior codes were assigned by two independent coders. Interrater reliability was calculated by using Cohen's kappa (Cohen, 1960; Landis and Koch, 1977) with 95% bootstrapped confidence ranges (R, statistical package, 2005; module "r-cran-psy").

We found kappa values between 0.75 and 0.83 for infant behavior codes (table 23 below - some behaviors were not testable due to floor effects) and 0.67 and 0.82 for maternal codes (table 24). According to Landis and Koch (1977) kappas above 0.60 may be labeled as substantial, thus, pointing to a sufficient interrater reliability in our case.

Table 23: Reliability measures for double-coded infant videos; some reliabilities not calculable, e.g., for negative behaviors w (e.g., due to floor effects).

	Cohen's Kappa, rater 1 compared to rater 2	lower bound of 95%- confidence	upper bound of 95%- confidence	p-value
infant negative (ineg)				
infant protesting (ipro)	0.75	0.69	0.81	p<0.001
infant withdrawn (iwit)				
infant looks at objects (inon)	0.75	0.67	0.81	p<0.01
infants' attention to caregiver (ineu)	0.83	0.78	0.88	p<0.001
infant social pos. engagement (ipos)	0.85	0.77	0.91	p<0.001

Table 24: Reliability measures for double-coded infant videos; some reliabilities not calculable, e.g., for negative behaviors (e.g., due to floor effects).

	Cohen's Kappa, rater 1 compared to rater 2	lower bound of 95%- confidence	upper bound of 95%- confidence	p-value
caregiver neg. engagement (cneg)				
caregiver hostile / intrusive (chos)				
caregiver withdrawn (cwit)				
caregiver non-infant focused (cnon)	0.82	0.53	1.00	p<0.001
caregiver social monitor and no vocs (cneu)	0.72	0.66	0.80	p<0.001
caregiver social monitor, pos. vocs (cpvc)	0.79	0.74	0.86	p<0.001
caregiver social pos. engagement (cpos)	0.73	0.65	0.80	p<0.001
caregiver exagg. pos. engagement (cexg)	0.67			

6.8.3 Intersystem validity

Concurrent validities between of the currently used system (ICEP / Monadic Phases Coding System) and a competitive system, the Maximally Discriminative Facial Movement, are reported by Matias, Cohn and Ross (1990). Generally speaking, both systems have been used to quantify categories of infant affective behavior. They are not specialized in facial action only, as is the system of Izard (e.g., 1990). Intersystem correlations in the sense of concurrent validity have been reported e.g., with respect to negative engagement between $r=.55$ and $r=.85$, for attending behavior of $r=.77$, and, for positivity of $r=0.85$, all denoting an at least sufficient convergent validity of the applied coding system.

6.9. The advantages of behavior observations – avoidance of problems derived from the depression-distortion hypothesis (depression-related biased parental perceptions)

A range of authors suggested that behavior observations have the advantage that there is no need for a mother as informer for her child. Thus, the registration of behaviors completely avoids any discussion such as those regarding the “depression-distortion hypothesis” (e.g. Gartstein et al., 2009; Richters and Pellegrini, 1989) versus the “depression-realism hypothesis” (Richters, 1992). This debate is

still ongoing. A range of authors still questions the validity of ratings obtained by depressed mothers, e.g., the accuracy of maternal reports about the behavior of their children. In particular depressed mothers were found to over-report problem behaviors in their children (e.g. externalizing behavior in boys and internalizing behaviors in girls) compared to independent observers.

For example, Friedlander et al. (1986) reported that depressed mothers over-estimate child behavior problems and thus lower the validity of a standardized inventory child behavior inventory. Schaughency and Lahey (1985) found that an increasing depression severity was associated to higher child ratings of externalization behavior. Accordingly, the authors concluded that maternal ratings of child externalizing problems might be flawed without the assessment of maternal depression severity. Breslau et al. (1988) reported that depressed mothers tend to overstate symptoms of their children. Panaccione and Wahler (1986) concluded that maternal ratings showed no correlation with child behaviors. Gartstein et al. (2009) found that depressed mothers tend to over-report externalizing behaviors of boys and internalizing behaviors of girls.

Richters (1992) defined minimum requirements for a test of the depression-distortion hypothesis, for example, the availability of an external gold-standard measure that allows for a decision if the maternal ratings are “correct”; then, the availability of maternal ratings, and, finally a depression score to show that larger deviations occur with higher depression severity.

Richters (1992) argues that most of the studies have no justification to support a depression-related distortion of the maternal view on her child: many did not analyze depression-related disagreements of cross-informant data, i.e. data of different assessment types, i.e., between mothers and criterion-raters. Moreover, they did not show that the applied criterion ratings (e.g. the independent observer or a teacher) were superior over the maternal ratings. An additional criterion that Richters omitted is the question of the relevance of the cross-informant deviation: the mother and independent observer may well disagree but either below a threshold of relevance or below measurement error of the instruments. It seems that up to now no test for the depression-distortion hypothesis has included any criterion of relevance. The few studies that fulfilled the Richters criteria did not reveal cross-informant differences that increased with higher severity of a maternal depression. On the contrary,

Richters claims that the evidence favours the depression-realism hypothesis, i.e., that depressed mothers are well capable to rate their children and agree with independent informers. Richters found 6 studies that both fulfilled these criteria and found no distorted maternal views, thus favoured the depression-realism hypothesis (Angold et al., 1987; Conrad and Hammen, 1989; Weissman and Wickramarante, 1987; Billings and Moos, 1985; Richters and Pellegrini, 1989; Ivens and Rehm, 1988).

Nevertheless, the discussion is still ongoing, e.g. with Gartstein et al.'s (2009) finding that depressed mothers tend to over-report externalizing behaviors of boys and internalizing behaviors of girls. Further recent studies increasingly tend to favour the depression-distortion hypothesis, in terms of an over-reporting of negative child-behaviors and under-reporting of positive behaviors in presence of maternal depression (see also e.g., Chilcoat and Breslau, 1997; Chi and Hinshaw, 2002; Fergusson et al., 1993; Najman and Williams, 2000; Briggs-Gowan et al., 1996).

6.10. Statistical methods: Focus on nonparametric methods instead of parametric ones

Hypotheses concerning group contrasts constituted the main focus of the statistical analysis. Data distributions in the population were completely unknown. Moreover, behavior frequencies and time proportions are rarely normally distributed (which was confirmed in several pilot data in our study group). Thus, nonparametric tests, such as the exact Mann-Whitney-U test were applied (Mann and Whitney, 1947, Lehmann, 1998; SPSS, 2005; module "exact tests") and its multivariate generalization for two-group comparisons (the Wei-Lachin Multivariate Rank Analysis, Wei and Lachin, 1984; Lachin, 1992; open source computer program by Davis, 2000). This test is applicable even when values are missing (at random) and – contrary to a multivariate analysis of variance – avoids a list wise deletion of an observation when missing values occur.

Classical statistical methods such as the analysis of variance were not applicable since distributions were both skewed (e.g., due to floor effects) and variances were well heterogeneous. Parametric tests require a range of assumptions to be fulfilled, amongst them a Gaussian distribution of data residuals (normality assumption), the homogeneity of variances over groups, or the assumption of linearity. Statistically every additional test, e.g., for deviancies from normality or for homogeneous variances, can be considered as a pre-test and has the potential to increase the risk

of erroneous decisions (α -inflation).

Although there have been doubts regarding the robustness of rank tests and their efficiency Pitman showed already in 1948 that nonparametric tests have excellent efficiency with clear advantages in case of a violation of the normality assumption. In the last years rank tests have taken their place as strong competitors of classical normal theory methods (Lehmann, 1998).

To test for differences in changes, the exact two-sided Mann-Whitney-test was applied to pre-post differences. Thus, repeated measurements were analyzed in a 2 x 2 design based on 2 groups (depressed, healthy) and 2-phases (pre- and post maternal still-face), and extended to 2 periods for the re-assessments (mothers in-episode, after remission).

The experiment-wise error was adjusted based on the Bonferroni method to control for an α -inflation (i.e. for an increased risk of false-positive results due to multiplicity, Bortz, 2005). The experiment-wise error of $\alpha = 0.05$ was adjusted by $\alpha = 0.05 / k$ (Holm, 1979) where k denotes the number of confirmative statistical tests (in our case we had $k=7$ confirmative hypotheses). All exploratory hypotheses were run in a strictly descriptive or exploratory manner without adjustment for the 1st type error level.

To test for associations in $r \times c$ tables (e.g., if demographical characteristics were compared) the exact Chi²-test (Bortz, 2005) was used. For equivalence testing, the Mann-Whitney test modified for equivalence was applied (Wellek, 2002). To compare groups based on time-to-event data and “censored data” (possibility of missing events after observation period), e.g., the Kaplan-Meier estimator was applied (Kaplan and Meier, 1958). Between-group comparisons were made based on the log-rank test (Kalbfleisch and Prentice, 1980; Cox and Oakes, 1984, SPSS procedure *k-m*).

Contrasts or shifts between groups were additionally given based on the Hodges-Lehmann estimator of shift and 95% exact confidences (Lehmann, 1998; R, 2005; package “coin”).

Lag-sequential analyses (Allison and Liker, 1982; Bakeman and Gottman, 1986; Gottman and Roy, 1990; Gottman, 1979, Sackett, 1979) have been applied to test for conditional response and control for baseline rates. Standard z -values were used to

compare conditional responsiveness.

In addition the lag_0 or lag_1 cross-correlation coefficient was calculated as measure for association of behaviors of intradyadic time stream (after behavior codes were scored on a positivity-negativity dimension). The lag_0 correlation was used to indicate mother-infant parallelisms or synchrony, the lag_1 correlation to test for predictability.

A principal component analysis with VARIMAX rotation (PCA, Bortz, 2005) was applied to compare groups based on factor values as composite measures. A Scree-plot was used to show sufficiency of Eigenvalues and variances explained by the resulting factors.

Several further bias correction methods were applied, e.g., methods based on inequality assumptions of groups, e.g., matched-pair methods based on propensity score methods (see page 201), i.e. to control for a large number of confounders without necessity of a large range of matching scenarios (Rosenbaum and Rubin, 1985; Joffe and Rosenbaum, 1999; Rubin and Thomas, 1996; Braitman and Rosenbaum, 2002).

Also a classical method for the analysis of confounders, the analysis of covariance (page 204) was used for explorative reasons (Winer, Brown and Michels, 1991; Winer, 1971; SPSS procedure “univariate”, Norusis, 2008).

And, finally, correction methods for selection bias were applied, Heckman’s two stage procedure (e.g., refer to Heckman’s Nobel-Prize lecture, 2000; or, 1979) to control for and eliminate effects of potential biases (page 209).

Descriptive statistics are also given, i.e. continuous data is displayed as averages plus standard error, minimum, maximum and percentiles (10th and 90th and median values) are also provided. 95%-confidence limits of estimation is given for binary data (percentages, e.g. depression symptom prevalence) and are calculated according to Newcombe’s (1998) estimation method 5 based on exact binomial tail areas.

7. Results

7.1. Primary target parameters – confirmative hypotheses

7.1.1 1st confirmative hypothesis: reduction of affect mirroring and disappearance of impaired affect mirroring after remission (6th confirmative hypothesis)

The first confirmative hypothesis referred to parallelisms of affect which was expected to be impaired in presence of major depression. Parallel positive behaviors (labeled as “affect-mirroring”) in general occurred 20-35% of the time during interaction (figure 6). In dyads with a depressed mother, however, values were only slightly lower (red curves). Statistically, dyads with a depressed mother and dyads with a healthy mother did not differ with respect to affect-mirroring ($p=0.12$, 1st confirmative statistical test, left side of the chart, all other p-values have exploratory status).

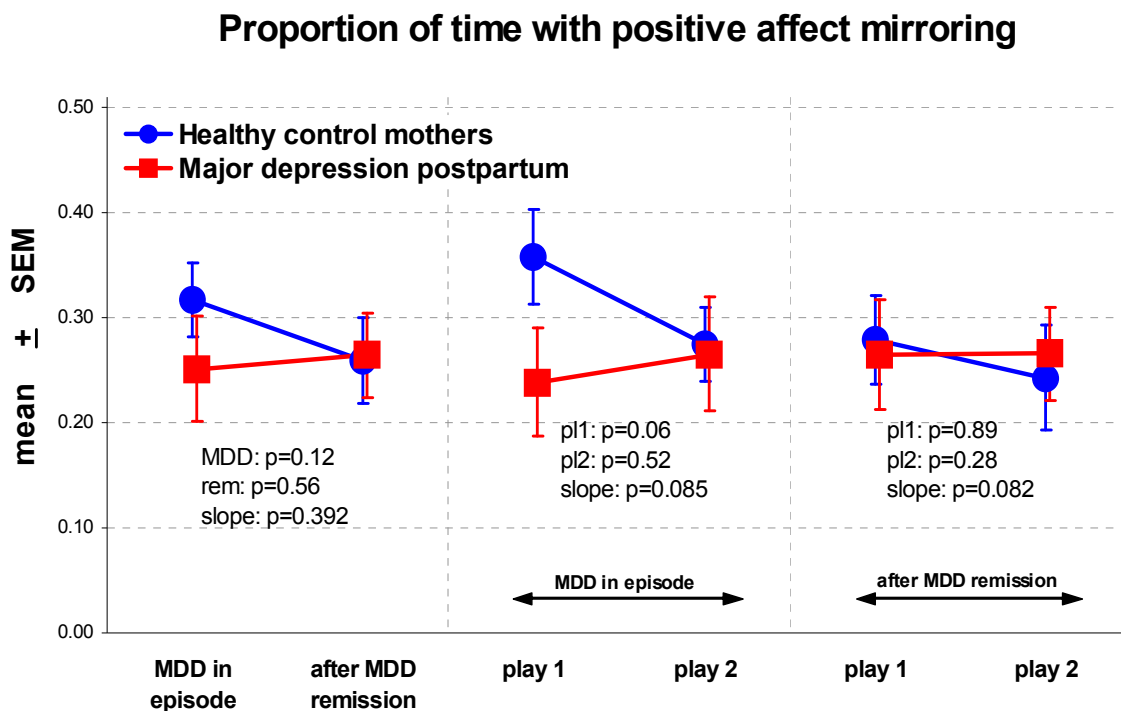


Figure 6: Proportion of time with affective mirroring (pl1 = p-value for play 1 phase, pl2 respectively, MDD = major depressive disorder. For operationalization of affect mirroring, i.e., parallel occurring system-codes: “ineu”, “ipos”, “cpvc”, “cpos”, refer to table 20, table 21 and 22 on page 126), statistical tests: exact Mann-Whitney-test (Lehmann, 1998), two-sided p-values

The time proportions in major depressed mothers were only slightly lower (0.25 versus 0.32 in control dyads). The nonparametric measure of contrast (the between-group shift) showed a contrast of -0.08 (table 25, 4th data line, with 95%-confidence between -0.19 and 0.03) and includes the value zero, thus pointing to an almost

negligible between-group contrast. Thus, the first statistical test failed to fall below the confirmatory limit of $\alpha = 0.007$, the zero hypothesis could not be rejected and the descriptive contrast measure pointed to a zero-contrast. Dyads where the mother was in major depression generally did not show lower values in affective mirroring (in the sense of parallel occurring affect codes).

The courses (middle part of figure 6) were slightly different because dyads with a mother who was in an episode of depression had reduced affect sharing in the beginning, i.e. before the still-face ($p=0.06$, exploratory test). Afterwards, both group averages seem well comparable, but the group courses were not different (slope comparison: $p=0.085$), although proportions of mirrored affects tended to move in opposite directions: towards lower proportions in dyads with a healthy mother and towards higher proportions in dyads with a depressed mother.

Table 25: Contrast measures for the difference in proportions of affective sharing (bold line: the contrast of the 1st primary hypothesis), Hodges-Lehmann shift estimators and exact 95%-confidences (Lehmann, 1998), all data are time-proportions contrasted between depressed mothers and control mothers

two group comparisons with Hodges-Lehmann shift estimators				
		median shift	lower 95% confidence	upper 95% confidence
MDD in episode	play 1	-0.12	-0.27	0.00
	play 2	-0.04	-0.14	0.09
	differences	0.11	-0.01	0.23
	combined	-0.08	-0.19	0.03
after MDD remission	play 1	-0.01	-0.14	0.11
	play 2	0.07	-0.07	0.18
	differences	0.11	-0.01	0.20
	combined	0.04	-0.09	0.14

Note: A median shift does not refer to the arithmetic difference of two medians.
The term "difference" refers to last phase (play 2) minus first phase (play 1).
The term "combined" refers to both phases (play 1 and 2), averaged.

With respect to the predicted disappearance of impaired affect mirroring after the remission of maternal depression (refer to the 6th confirmative hypothesis), there were no differences between groups as shown by the courses ($p=0.392$, compare slopes in left part of figure 6). Postpartum depressed mothers remained almost stable with respect to their in-episode values as compared with their after-remission values (0.26 to 0.27, proportions of interaction time), healthy control mothers changed only slightly (from 0.32 to 0.25 of total interaction time).

Thus, the statistical test with respect to the disappearance of lower affect-mirroring

failed to fall below the confirmatory limit of $\alpha = 0.007$ and the zero hypothesis could not be rejected. All in all there was no indication of a reduced affect mirroring in dyads with a depressed mother and subsequently no disappearance after the remission of depression.

7.1.2 2nd confirmative hypothesis: prolonged latency until positive affect-sharing in depressed dyads

Based on flat affect assumptions, dyads with a depressed mother are expected to be characterized by failures and delays in gaining a state of shared positive affect or affect mirroring. The second confirmatory hypothesis expects that the time until parallelisms in positive affect occur is significantly longer in dyads with a depressed mother.

For each case the latency until first occurrence of affect mirroring was recorded, as well as so-called “censored” cases (Cox and Oakes, 1984), i.e. cases without occurrence of affect-sharing until end of observation time. Calculations were made according to the method of Kaplan and Meier (1958). This method combines events with the time of occurrence and calculates cumulative chances of events over time (SPSS procedure “k-m”, see also Cox and Oakes, 1984; Kalbfleisch and Prentice, 1980).

For the first observation (depressed mothers in episode, figure 7, upper part) healthy dyads in particular had the shortest latency until a positivity overlap occurred (red non-dotted curve compared to blue non-dotted curve). However, differences between healthy dyads and dyads with a depressed mother were not confirmatory significant (no p-value was below $p \leq 0.007$, play phase, $p = 0.995$, reunion phase, $p = 0.034$, log-rank Test (Cox and Oakes, 1984), although there was a tendency for control dyads with higher latencies to regain parallel affects ($p = 0.034$, figure 7, upper part, compare red and blue solid lines).

Thus, with regard to the 2nd confirmatory hypothesis there were no differences with respect to the expected prolonged regain of positivity mirroring.

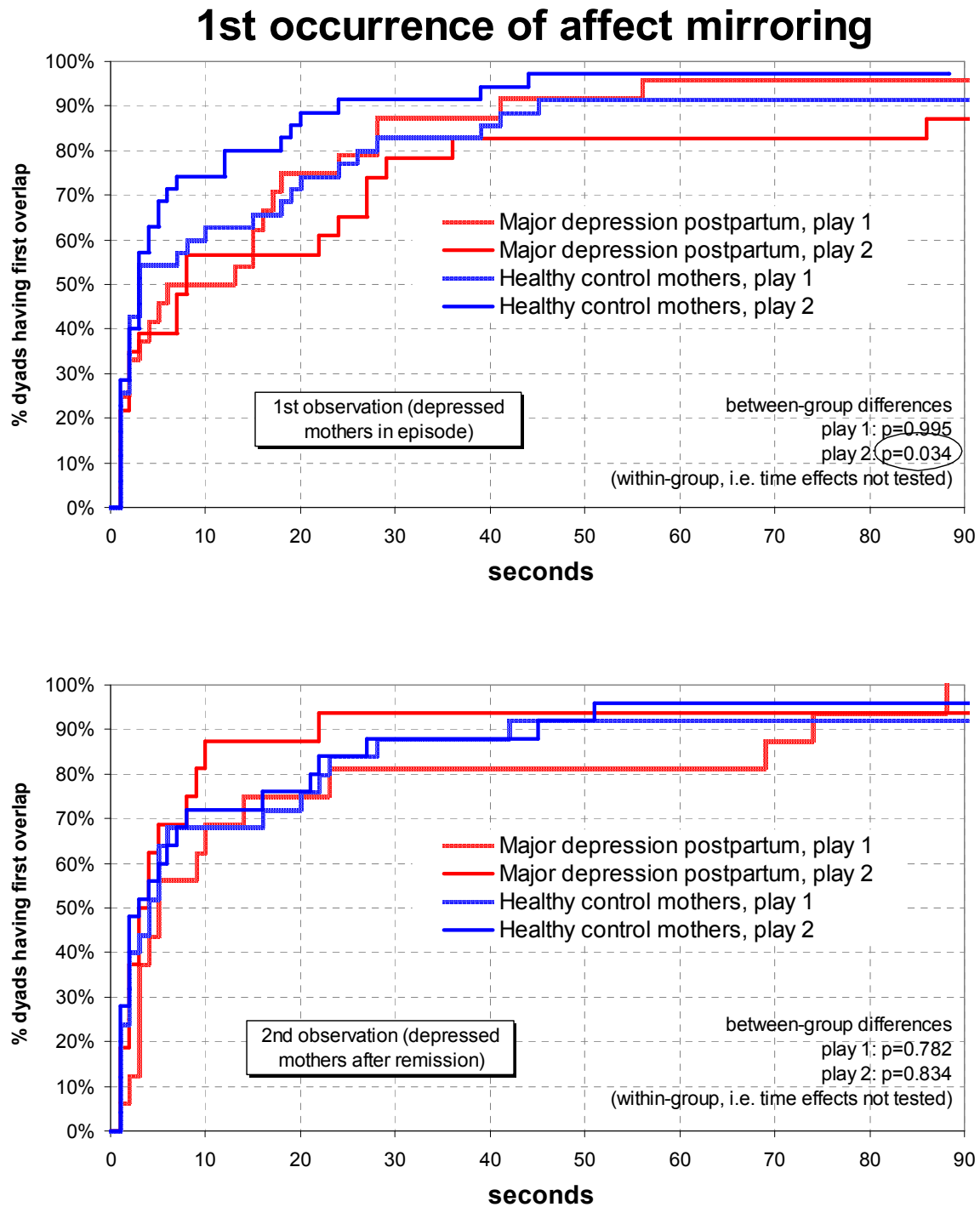


Figure 7: Latency in seconds until 1st onset of affect mirroring, method according to Kaplan and Meier (Cox and Oakes, 1984), test for between-group differences: log-rank test, y-axis: 1 minus cumulative probability of the Kaplan-Meier estimator in %

In the second observation (depressed mothers after remission, figure 7, lower part), there were again no statistically significant differences between diagnosis groups ($p=0.782$ and $p=0.834$, for the 1st and 2nd play phase, respectively).

Even if average latencies are analyzed descriptively (figure 8) diagnosis groups did not differ in their courses (depressed mothers in episode, $p=0.084$).

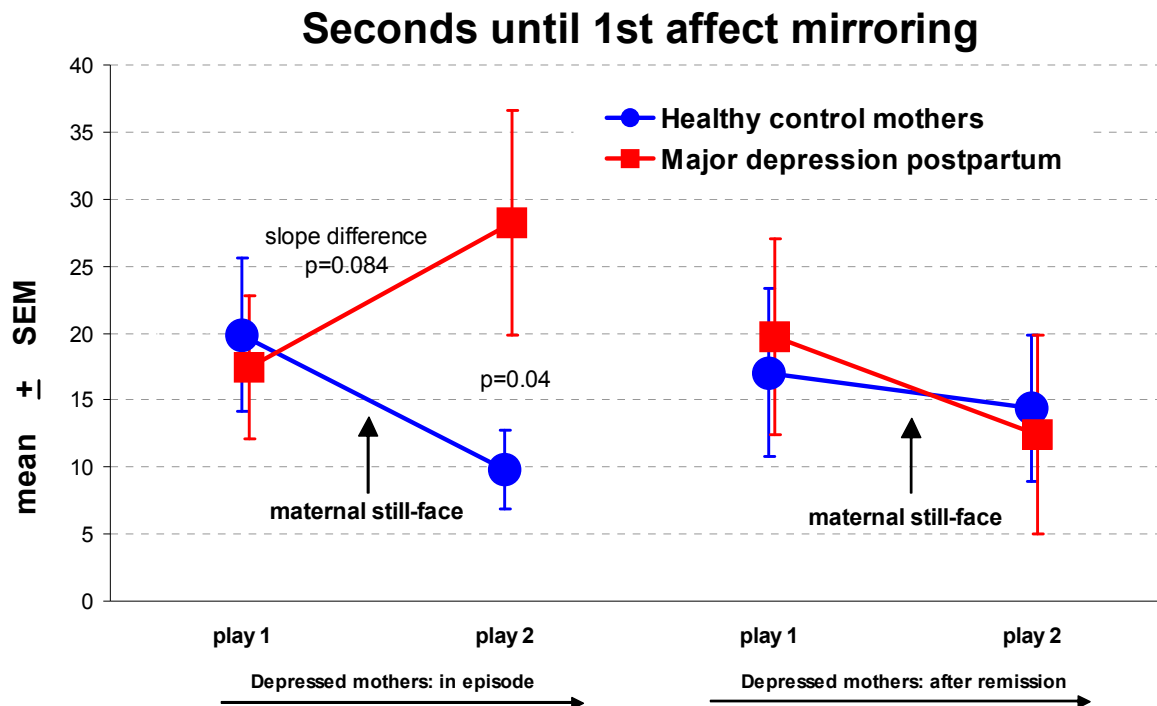


Figure 8: Seconds until positivity overlap reoccurs, test for latencies until parallel occurring positive behaviors reoccur, statistical test: exact and one-sided Mann-Whitney test, upper limit for p-value to reject zero hypothesis of no difference: $\alpha_{\text{adjusted}} = 0.007$

Thus, dyads with a depressed mother did not differ in general from dyads with a healthy mother with respect to a prolonged regain of positive affect mirroring.

7.1.3 3rd confirmative hypothesis: non-affectedness of infants of depressed mothers if the mother is unavailable

The theory that a depressed mother acts as role model for flat affect and the infant mirrors it resulted in the prediction that infants of depressed mothers react non-affectedly if the mother is unavailable, i.e. a brief maternal unavailability is predicted not to change their constantly lower level of affective sharing. Results of this hypothesis, i.e. p-values have already been presented on page 134 (middle part of figure 6). Both groups tend to be affected differently: dyads with a depressed mother tended to increase their affective mirroring after the still-face, whereas control dyads tended to restrict their affective mirroring (Mann-Whitney test for differences between post minus pre values: $p=0.085$, which is far beyond the confirmatory decision limit of $\alpha=0.007$). The contrast measure for both courses moving in opposite directions is 0.11 (-0.01 to 0.23, Hodges-Lehman estimators of shift). Thus, dyads with a depressed mother were not affected differently by the still-face phase, i.e. by the brief phase of maternal unavailability in comparison with control dyads.

7.1.4 4th confirmative hypothesis: lack of affective infant-stimulating behavior

Based on the parenting impairment hypothesis aspects of the maternal depression profile (e.g., low energy levels and reduced or flat affect) allowed for the prediction that these mothers have lower activity levels (in affect expression) and thus lower levels of infant stimulation.

Total stimulation time (in proportions to observation time) is shown in figure 9, ranging between 60 and 80% of observation time and with stimulation frequencies of 6 to 9 per minute.

Contrary to expectation depressed mothers (1st observation, courses on the right in figure 9) did not engage with at a lower overall stimulation level ($p=0.48$). Thus, dyads with a depressed mother did not display a significantly lowered rate of affective infant stimulation. Hence, there is no indication of a lack of affective infant stimulation in association with a current episode of depression.

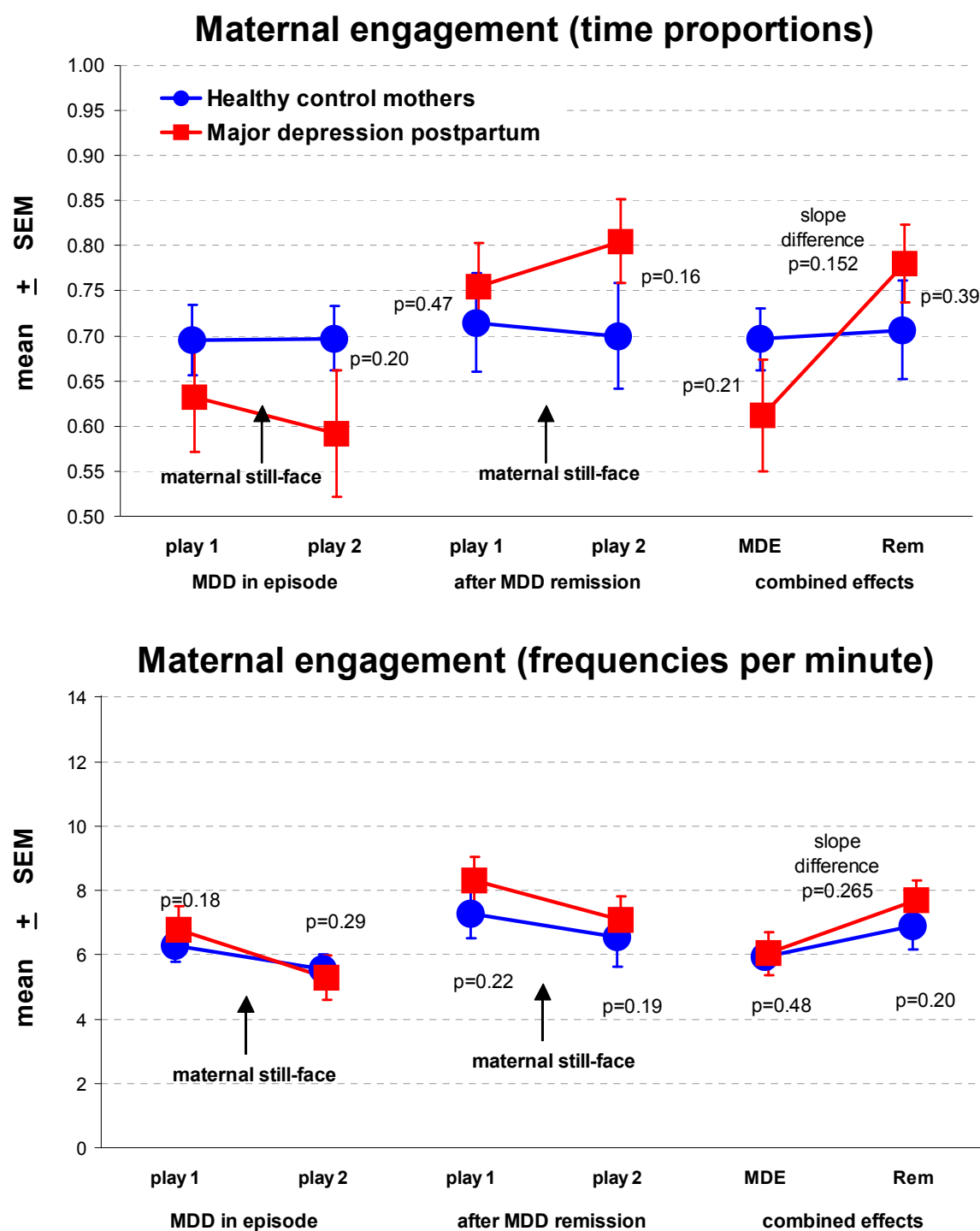


Figure 9: Overall maternal engagement, as proportions of observation time (upper chart) and frequency per minute (lower chart). Occurrence of behavioral codes “cpvc, cpos, cexg” either counted per minute or seconds summed up and related to observation time, groups compared with a Mann-Whitney-U-Test (Bortz, 2005), one-sided

7.1.5 5th confirmative hypothesis: deviant affective activity level in infants of depressed mothers (activity either too low or too high)

Infants of mothers with major depression were predicted to be generally deviant in their affect activity level, i.e., either hypo-active or hyper-active, that is, either lower or higher overall frequencies of affect codes are expected. The data revealed the total amount of infant engagement took up 35 to 55% of observation time (Figure 10), with

stimulation frequencies of 6 to 9 per minute. However, infants of depressed mothers showed no generally lower activity levels ($p=0.25$, refer to the course on the right in figure 10, upper part, confirmative hypothesis 5, all other p -values are exploratory).

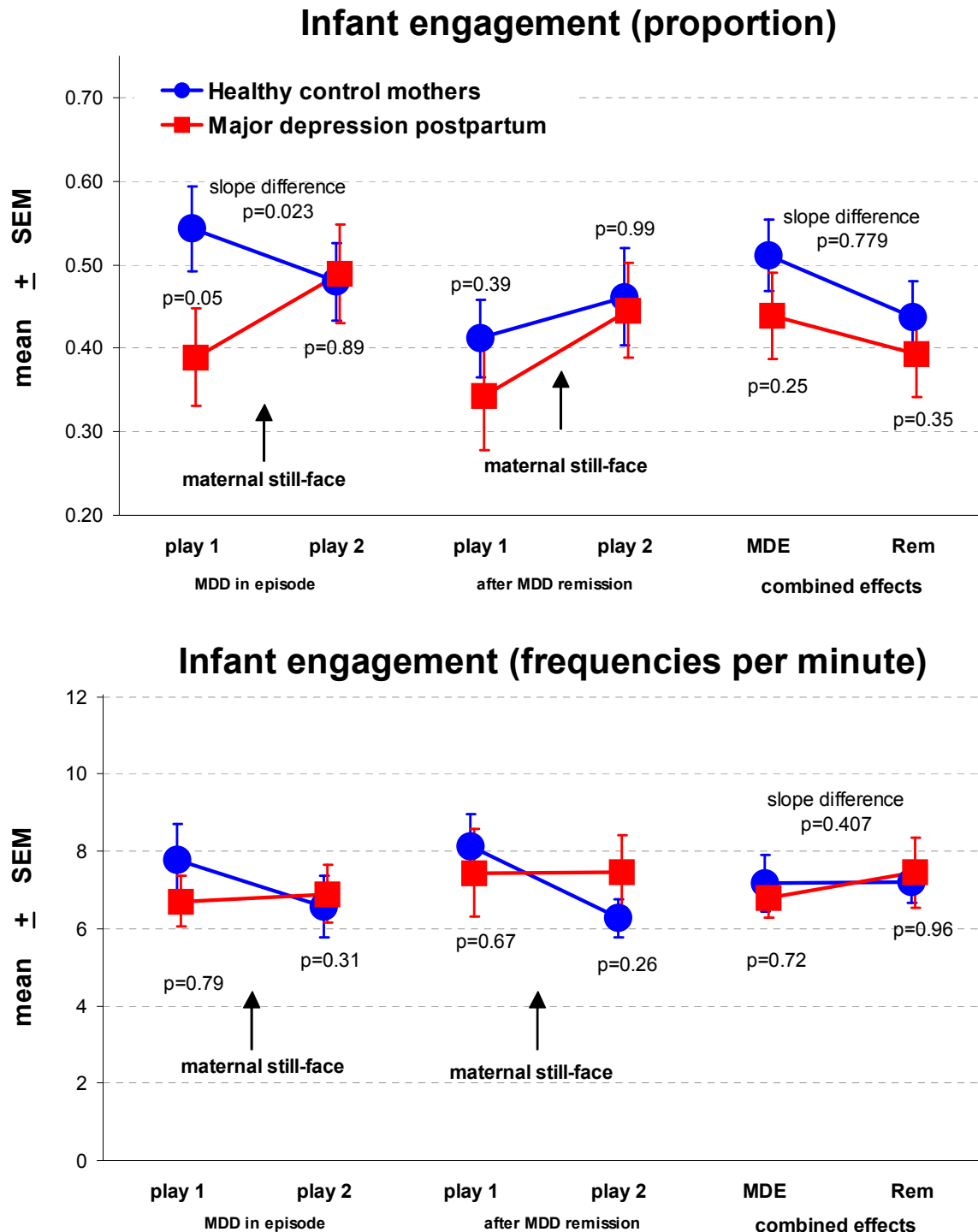


Figure 10: Overall infant engagement, as proportions of observation time (upper chart) and frequency per minute (lower chart). Occurrence of behavioral codes of infant protest, neutral/attending to the mother or positive engagement (behavior codes “ipro”, “ineu”, “ipos”) either counted per minute or as relative observation time. Groups compared with a Mann-Whitney-U-Test (Lehmann, 1998), two-sided

Thus, infants of mothers with major depression did not differ in their affect-related

activity levels, i.e. they were neither hypo-active nor hyper-active, when compared to infants of healthy control mothers.

7.1.6 Disappearance of impairments in affect mirroring after remission (6th confirmative hypothesis)

With respect to the confirmative hypothesis regarding the disappearance of impaired affect mirroring after the depression remission, absolutely no differences between groups were found, as the p-values on page 134 have shown (test for divergent courses, $p=0.392$, compare slopes in left part of figure 6). Thus, no disappearance in impaired affect mirroring in association with the remission of maternal major depression could be observed.

7.1.7 7th confirmative hypothesis of a reduction in negative child behaviors (heightened protest or - conversely - withdrawal rates) after the maternal depression is remitted (transient disturbance hypothesis)

The transient disturbance hypothesis allowed for the prediction that specific negative child behaviors (e.g., those that hinder interaction such as protest or withdrawal) also remit after the maternal depression remits. For this hypothesis both behaviors are summed up (also, in a 2-factor principal component analysis both behaviors load on the same factor, i.e., the sum of two highly correlated behaviors with a comparable scale seemed to be justified, each behavior is nevertheless displayed in a later chapter). Rates and time proportions of negative child behaviors are only expected to be increased during an episode of major depression but expected to decrease with depression remission.

The data showed infant negativity with very low proportions of time in general (lower than 10 to 15%, upper part of figure 11) and average frequencies well below one per minute (lower part of the figure). There were no between-group differences, at any point of measurement, i.e., there were no higher values in infants of depressed mothers nor was there any amelioration after the maternal depression remitted ($p=0.92$ for proportions and $p=0.85$ in frequencies; on the left side of the figure 11).

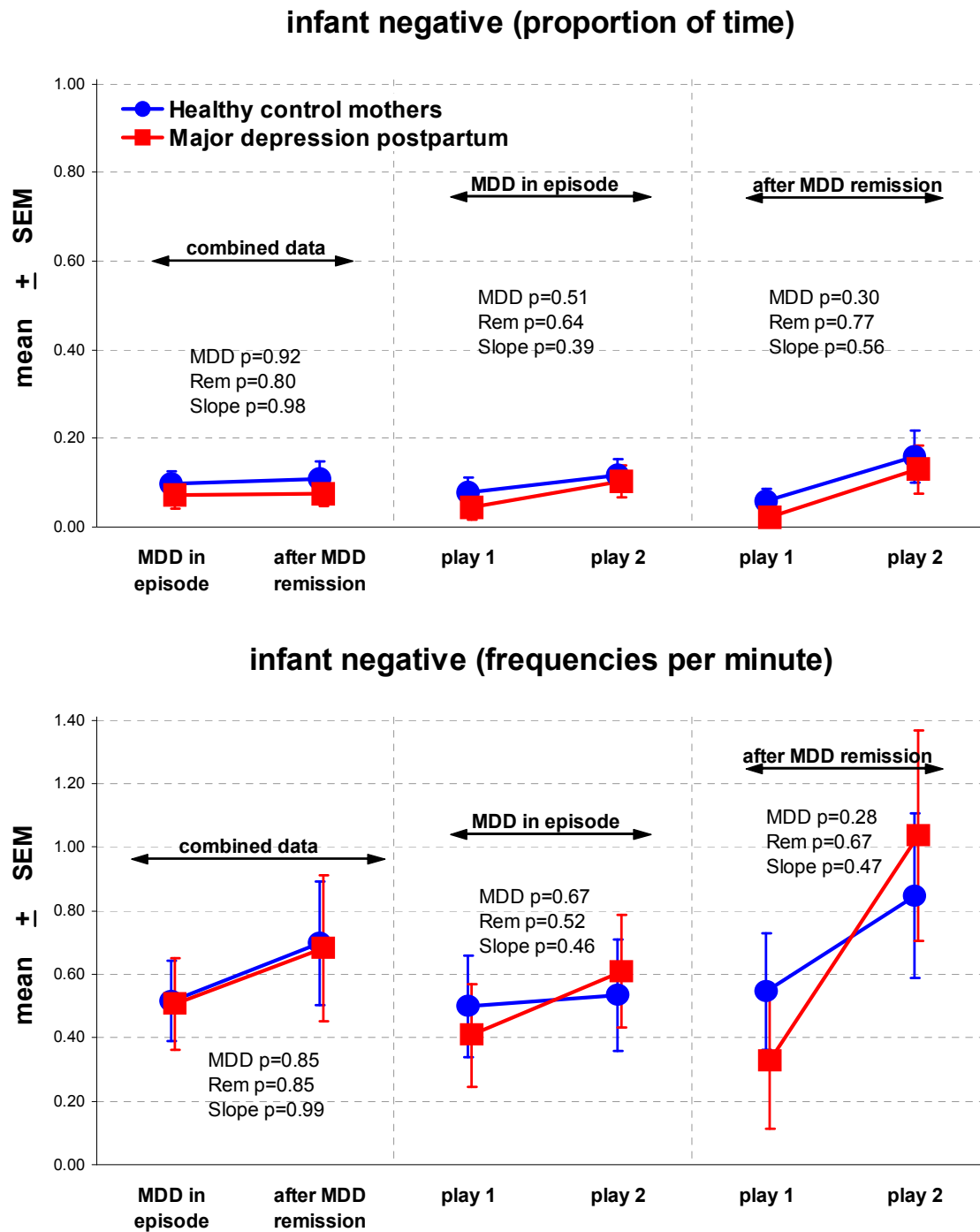


Figure 11: Infant negativity (protest, withdrawal), statistical test: exact one-sided Mann-Whitney test

7.2. Exploratory hypotheses on dyadic behavior in association with maternal major depression

7.2.1 Heightened maternal negativity in specific aspects (hostility, withdrawal, exaggerations), 1st exploratory hypothesis

Based on the hypothesis that depressed individuals are far more negative, specific negative maternal behaviors were analyzed with the expectation of heightened durations and frequencies in mothers with depression, e.g. of hostile behaviors towards the infant, maternal withdrawals, non-infant focusing or, on the contrary, exaggerated behaviors. However, the data showed that most behaviors primarily resulted in floor effects in both groups (figure 12, raw data in table 26) and non-significant test results.

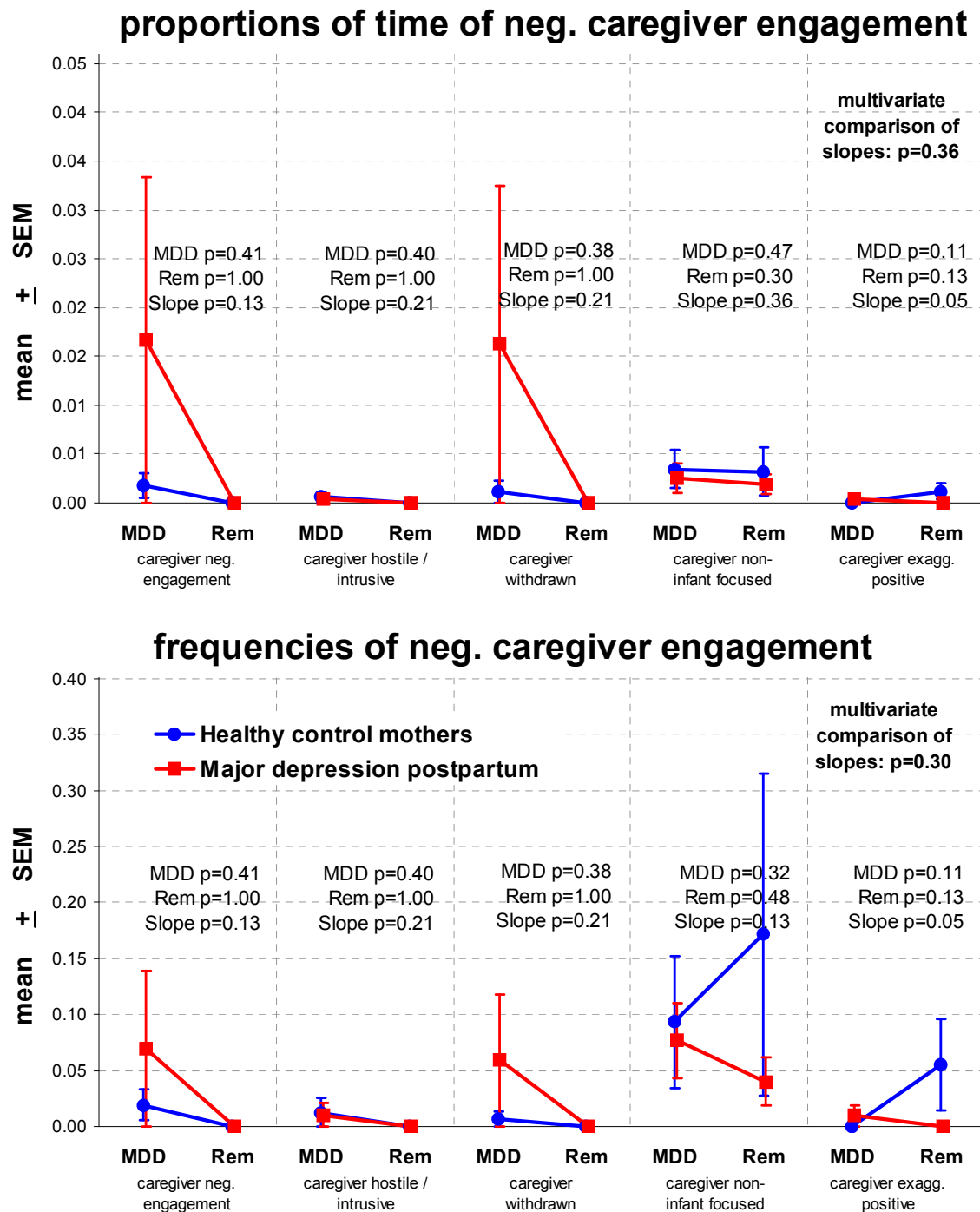


Figure 12: Caregiver engagement, as proportions of time (upper chart) and frequency per minute (lower chart). MDD = major depressive disorder, Rem.: Remission. Groups compared with a Mann-Whitney-U-Test (Bortz, 2005), one-sided. Caregiver exaggerations were added to this list due to results of principal component analysis (exaggerated behavior sorted into the same factor as negative or hostile behavior). Multivariate test for global difference (1st and 2nd play phase pooled): Wei-Lachin Multivariate Rank Analysis (Wei and Lachin, 1984; 1992; two-sided).

Thus, mothers with major depression did not show differences compared with control mothers in any of these measures. All these behaviors rarely occurred in the laboratory. Accordingly, depressed mothers did not exhibit higher frequencies in specific negative affects, nor was there any ameliorating or worsening trend from the depression episode (1st observation) to remission (2nd observation). Thus, mothers with major depression did not differ from healthy mothers in specific aspects of negative affects.

Other behavior aspects, e.g. attending or positive behaviors are shown in table 26, all tests exploratory). In accordance with previous results, none of these behaviors showed differences in favor of control dyads.

Table 26: Caregiver and infant behaviors in proportions of time (e.g. 0.30 = 30% of observation time, of 2 minutes for each play and still-face intervals), t1 refers to the 1st observation, i.e. mothers with postpartum depression in episode, whereas t2 refers to the 2nd observation, i.e. mothers with postpartum depression after remission; (1) test for global difference (1st and 2nd play phase pooled and depressed mothers compared to healthy dyads). Statistical test is a multivariate Wei-Lachin Multivariate Rank Analysis (Wei and Lachin, 1984; Lachin, 1992; two-sided), (2) differences between depressed and healthy dyads per phase: Mann-Whitney test, one-sided, (3) and (4) effect of maternal still-face: both groups compared in change values (simple differences) from 1st play to 2nd play phase (Mann-Whitney-Test, Lehmann, 1998), two-sided, (5) and (6) test for a trend from “in episode” to “after remission” (1st and 2nd play were averaged) compared to doubly tested healthy dyads (t1 and t2), m = mean, se = standard error. All comparisons are strictly exploratory.

proportions of time		Healthy control mothers (n=35)						Major depression postpartum (n=24)						(1) Global group difference	(2) differences per phase 1-3 (one-sided Mann-Whitney tests)	(3) changes 1st to 2nd play		(4) between groups	(5) means 1st to 2nd observation		(6) diverging trends from 1st to 2nd observaton
		1st play		still-face		2nd play		1st play		still-face		2nd play				healthy	depr.		healthy	depr.	
		m	se	m	se	m	se	m	se	m	se	m	se			m	m	m	m		
caregiver neg. engagement	t1	0.00	0.00	0.00	-	0.00	-	0.02	0.02	0.00	0.00	0.02	0.02	p=0.37	none	0.00	0.00	p=0.10	0.00	0.02	p=0.25
	t2	0.00	-	0.00	-	0.00	-	0.00	-	0.00	-	0.00	-	p=0.35	none	-	-	-	0.00	0.00	
caregiver hostile / intrusive	t1	0.00	0.00	0.00	-	0.00	-	0.00	-	0.00	-	0.00	0.00	p=0.40	none	0.00	0.00	p=0.15	0.00	0.00	p=0.42
	t2	0.00	-	0.00	-	0.00	-	0.00	-	0.00	-	0.00	-	p=0.35	none	-	-	-	0.00	0.00	
caregiver withdrawn	t1	0.00	0.00	0.00	-	0.00	-	0.02	0.02	0.00	0.00	0.02	0.02	p=0.26	none	0.00	0.00	p=0.15	0.00	0.02	p=0.42
	t2	0.00	-	0.00	-	0.00	-	0.00	-	0.00	-	0.00	-	p=0.35	none	-	-	-	0.00	0.00	
caregiver non-infant focused	t1	0.01	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	p=0.49	none	0.00	0.00	p=0.70	0.00	0.00	p=0.73
	t2	0.01	0.00	0.00	-	0.00	-	0.00	0.00	0.06	0.06	0.00	0.00	p=0.42	play 2: p=0.04	0.01	0.00	p=0.28	0.00	0.00	
caregiver social monitor and no vocs	t1	0.30	0.04	0.99	0.00	0.32	0.04	0.36	0.06	0.95	0.04	0.34	0.06	p=0.39	none	-0.02	0.02	p=0.85	0.31	0.35	p=0.57
	t2	0.30	0.06	0.96	0.04	0.31	0.06	0.27	0.05	0.93	0.06	0.21	0.05	p=0.31	none	-0.02	0.05	p=0.12	0.30	0.24	
caregiver social monitor, pos. vocs	t1	0.60	0.04	0.01	0.00	0.60	0.04	0.56	0.05	0.00	0.00	0.50	0.06	p=0.18	still: p=0.01	0.00	0.05	p=0.32	0.60	0.53	p=0.52
	t2	0.62	0.05	0.05	0.04	0.60	0.05	0.62	0.05	0.01	0.00	0.70	0.04	p=0.45	none	0.01	-0.07	p=0.06	0.61	0.66	
caregiver social pos. engagement	t1	0.10	0.02	0.00	0.00	0.11	0.03	0.09	0.02	0.01	0.01	0.10	0.02	p=0.39	none	0.00	-0.02	p=0.37	0.11	0.10	p=0.76
	t2	0.11	0.02	0.00	0.00	0.11	0.03	0.15	0.04	0.01	0.01	0.12	0.04	p=0.38	none	0.00	0.03	p=1.00	0.11	0.14	
caregiver exagg. positive	t1	0.00	-	0.00	-	0.00	-	0.00	0.00	0.00	-	0.00	-	p=0.15	none	0.00	0.00	p=0.23	0.00	0.00	p=0.10
	t2	0.00	0.00	0.00	-	0.00	-	0.00	-	0.00	-	0.00	-	p=0.29	none	0.00	0.00	p=0.25	0.00	0.00	
infant negative	t1	0.08	0.03	0.07	0.03	0.12	0.04	0.04	0.03	0.18	0.06	0.10	0.04	p=0.48	none	-0.04	-0.06	p=0.39	0.10	0.07	p=0.98
	t2	0.06	0.03	0.11	0.04	0.16	0.06	0.02	0.01	0.16	0.07	0.13	0.05	p=0.29	none	-0.10	-0.11	p=0.56	0.11	0.08	
infant protesting	t1	0.08	0.03	0.07	0.03	0.11	0.04	0.04	0.03	0.15	0.06	0.10	0.04	p=0.47	none	-0.03	-0.05	p=0.38	0.09	0.07	p=0.99
	t2	0.06	0.03	0.11	0.04	0.16	0.06	0.02	0.01	0.16	0.07	0.13	0.05	p=0.29	none	-0.10	-0.11	p=0.56	0.11	0.08	
infant withdrawn	t1	0.00	-	0.00	-	0.01	0.01	0.00	-	0.03	0.03	0.01	0.01	p=0.41	none	-0.01	-0.01	p=0.81	0.00	0.00	p=0.77
	t2	0.00	-	0.00	-	0.00	-	0.00	-	0.00	-	0.00	-	p=0.35	none	-	-	-	0.00	0.00	
infant non-mom focused	t1	0.46	0.05	0.48	0.04	0.45	0.05	0.59	0.06	0.45	0.06	0.46	0.06	p=0.12	play 1: p=0.03	0.01	0.14	p=0.07	0.46	0.53	p=0.64
	t2	0.58	0.05	0.49	0.06	0.50	0.06	0.67	0.06	0.50	0.07	0.56	0.06	p=0.39	none	0.08	0.11	p=0.42	0.54	0.62	
infant attending to caregiver	t1	0.38	0.05	0.29	0.03	0.32	0.04	0.29	0.05	0.26	0.05	0.33	0.06	p=0.18	none	0.07	-0.05	p=0.09	0.35	0.31	p=0.97
	t2	0.26	0.04	0.29	0.05	0.22	0.04	0.23	0.04	0.19	0.05	0.23	0.04	p=0.42	none	0.04	0.00	p=0.25	0.24	0.23	
infant social pos. engagement	t1	0.09	0.02	0.02	0.01	0.06	0.02	0.07	0.02	0.02	0.01	0.07	0.02	p=0.40	none	0.03	0.00	p=0.68	0.08	0.07	p=0.81
	t2	0.11	0.03	0.02	0.01	0.09	0.03	0.11	0.03	0.01	0.01	0.09	0.04	p=0.40	none	0.02	0.01	p=0.49	0.10	0.10	

Table 26 Table continued from previous page: Caregiver and infant behaviors in frequencies per minute (refer to previous page for a table description)

frequencies per minute		Healthy control mothers (n=36)			Major depression postpartum (n=33)			(1) Global group difference	(2) differences per phase 1-3 (one-sided Mann-Whitney tests)	(3) changes 1st to 2nd play		(4) between groups	(5) means 1st to 2nd observation		(6) diverging trends from 1st to 2nd observation
		1st play m se	still-face m se	2nd play m se	1st play m se	still-face m se	2nd play m se			healthy m	depr. m		healthy m	depr. m	
caregiver neg. engagement	t1	0.04 0.03	0.00 -	0.00 -	0.05 0.04	0.00 -	0.10 0.07	p=0.21	none	0.04	-0.05	p=0.18	0.02	0.08	p=0.97
	t2	0.00 -	0.00 -	0.00 -	0.00 -	0.00 -	0.00 -	p=0.37	none			-	0.00	0.00	
caregiver hostile / intrusive	t1	0.02 0.02	0.00 -	0.00 -	0.00 -	0.00 -	0.06 0.04	p=0.25	none	0.02	-0.06	p=0.08	0.01	0.03	p=0.96
	t2	0.00 -	0.00 -	0.00 -	0.00 -	0.00 -	0.00 -	p=0.37	none			-	0.00	0.00	
caregiver withdrawn	t1	0.01 0.01	0.00 -	0.00 -	0.05 0.04	0.00 -	0.05 0.05	p=0.19	none	0.01	0.01	p=0.57	0.01	0.05	p=1.00
	t2	0.00 -	0.00 -	0.00 -	0.00 -	0.00 -	0.00 -	p=0.37	none			-	0.00	0.00	
caregiver non-infant focused	t1	0.18 0.12	0.01 0.01	0.01 0.01	0.05 0.03	0.02 0.02	0.06 0.04	p=0.33	none	0.17	0.00	p=0.28	0.10	0.06	p=0.89
	t2	0.33 0.28	0.00 -	0.00 -	0.04 0.02	0.02 0.02	0.05 0.03	p=0.34	play 2: p=0.04	0.33	-0.02	p=0.09	0.17	0.04	
caregiver social monitor and no	t1	3.19 0.36	0.68 0.05	3.28 0.32	2.95 0.31	0.76 0.16	2.95 0.34	p=0.24	none	-0.10	0.01	p=0.88	3.23	2.95	p=0.08
	t2	3.02 0.36	0.60 0.07	2.91 0.37	3.69 0.51	0.65 0.07	2.93 0.38	p=0.26	none	0.11	0.76	p=0.24	2.96	3.31	
caregiver social monitor, pos. vocs	t1	4.36 0.27	0.00 -	4.05 0.30	4.07 0.37	0.00 -	3.79 0.38	p=0.26	none	0.32	0.28	p=0.77	4.21	3.93	p=0.07
	t2	4.68 0.37	0.02 0.02	4.50 0.50	5.51 0.45	0.00 -	4.77 0.37	p=0.13	play 1: p=0.05	0.17	0.74	p=0.35	4.59	5.14	
caregiver social pos. engagement	t1	2.08 0.30	0.08 0.04	1.60 0.26	2.19 0.28	0.21 0.16	1.85 0.28	p=0.24	none	0.48	0.33	p=0.62	1.84	2.02	p=0.92
	t2	2.64 0.43	0.05 0.05	2.15 0.50	2.90 0.57	0.14 0.09	2.23 0.40	p=0.31	none	0.49	0.67	p=0.41	2.39	2.57	
caregiver exagg. pos. engagement	t1	0.00 -	0.00 -	0.00 -	0.01 0.01	0.00 -	0.07 0.06	p=0.03	none	0.00	-0.06	p=0.54	0.00	0.04	p=0.03
	t2	0.11 0.08	0.00 -	0.00 -	0.00 -	0.00 -	0.00 -	p=0.45	none	0.11	0.00	p=0.16	0.05	0.00	
infant negative	t1	0.51 0.16	0.72 0.17	0.52 0.17	0.34 0.12	0.58 0.19	0.52 0.14	p=0.38	none	-0.01	-0.17	p=0.42	0.51	0.43	p=0.98
	t2	0.53 0.18	1.02 0.30	0.81 0.25	0.31 0.15	0.62 0.19	0.76 0.23	p=0.47	none	-0.29	-0.45	p=0.62	0.67	0.54	
infant protest	t1	0.51 0.16	0.72 0.17	0.49 0.17	0.34 0.12	0.57 0.19	0.46 0.14	p=0.36	none	0.02	-0.11	p=0.44	0.50	0.40	p=0.94
	t2	0.53 0.18	1.02 0.30	0.81 0.25	0.31 0.15	0.62 0.19	0.76 0.23	p=0.47	none	-0.29	-0.45	p=0.62	0.67	0.54	
infant withdrawn	t1	0.00 -	0.00 -	0.03 0.03	0.00 -	0.01 0.01	0.06 0.06	p=0.47	none	-0.03	-0.06	p=0.93	0.01	0.03	p=0.96
	t2	0.00 -	0.00 -	0.00 -	0.00 -	0.00 -	0.00 -	p=0.37	none			-	0.00	0.00	
infant non mom-focused	t1	6.37 0.74	4.64 0.42	5.47 0.68	6.30 0.69	3.90 0.49	5.06 0.51	p=0.46	none	0.90	1.24	p=0.98	5.92	5.68	p=0.87
	t2	6.36 0.73	5.21 0.57	5.14 0.58	5.73 0.67	3.49 0.48	5.20 0.57	p=0.47	still: p=0.02	1.23	0.53	p=0.40	5.75	5.47	
infants attention to caregiver	t1	5.87 0.68	4.28 0.41	5.26 0.61	4.92 0.46	3.92 0.51	4.69 0.47	p=0.28	none	0.61	0.23	p=0.56	5.57	4.80	p=0.45
	t2	5.44 0.51	5.25 0.71	4.40 0.41	4.97 0.57	3.16 0.51	4.61 0.53	p=0.47	still: p=0.02	1.04	0.36	p=0.51	4.92	4.79	
infant pos. engagement	t1	1.91 0.49	0.49 0.19	1.30 0.41	1.38 0.23	0.32 0.10	1.50 0.29	p=0.23	none	0.61	-0.13	p=0.09	1.60	1.44	p=0.66
	t2	2.51 0.59	0.73 0.31	1.44 0.26	2.48 0.55	0.34 0.12	1.81 0.49	p=0.36	none	1.07	0.67	p=0.89	1.97	2.15	

7.2.2 Expectation of heightened infant negativity in association with maternal depression, 2nd exploratory hypothesis

When compared to control infants, infants of depressed mothers were predicted to behave much more negatively in specific aspects (refer to page 144). However, a comparison of infant behaviors such as protest, infant withdrawal, or a combined measure of both (i.e. at least one behavior) again revealed absolutely no differences between both groups (figure 13; raw data already presented in table 26, on page 147).

Floor effects occurred in infant-behaviors (e.g. protest) and, again, no between-group differences emerged. Thus, infants of depressed mothers did not behave much more negatively when compared with infants of depression-free control mothers.

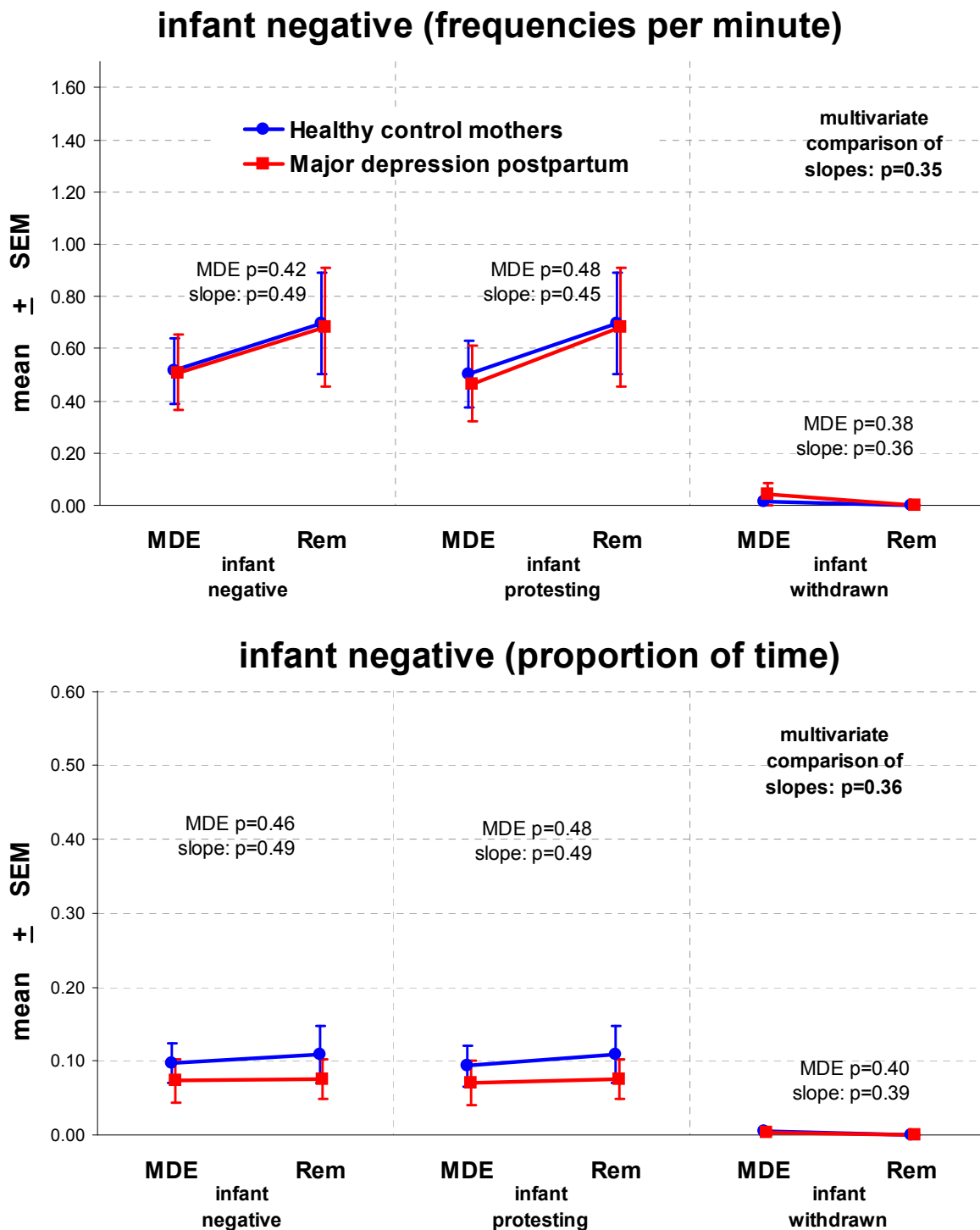


Figure 13: Infant behaviors classified as “negative”, upper chart: frequency per minute, lower chart proportions of time. Groups compared with a Mann-Whitney-U-Test (Lehmann, 1998), one-sided (MDE = major depressive episode, Rem = remission, “slope” refers to the comparison of pre-post differences between groups), multivariate test for global difference (1st and 2nd play phase pooled): Wei-Lachin Multivariate Rank Analysis (Wei and Lachin, 1984; 1992; two-sided)

7.2.3 Depression-associated excessive use of negative behaviors related to all behaviors, 3rd exploratory hypothesis

Based on the depression profile, dyads in which the mother is depressed were expected to focus predominantly on the negative behaviors in relation to their total range of behaviors. Note that in contrast to previous analyses the total level of “affect production” is included as well. The applied coding system allowed for an assignment of 8 different maternal behavior codes and 6 for the infant (table 20 and 21 on page 127). Also healthy mothers and their infants are predicted to use negative codes, but much more rarely in comparison with their usage of positive behavior codes. Thus “excessive use” is defined in relative terms, i.e., relatively to all behaviors displayed.

The data, however, revealed massive floor effects of negative behaviors when related to all behaviors, predominantly due to the rare occurrence of negative maternal behaviors. Accordingly the proportions of maternal and infant negative behaviors compared with the sum of their total behaviors resulted in floor effects and, as a consequence, dyads with and without a depressed mother did not differ in their focus on negativity (see column (1) in table 27). Thus, mothers with major depression and their infants did not differ in any way from healthy controls in their usage of negative behavior compared to all other behaviors.

Table 27: Proportions negative maternal behaviors compared to all other maternal behaviors displayed, t1 refers to the 1st observation, i.e. mothers with postpartum depression in episode, whereas t2 refers to the 2nd observation, i.e. mothers with postpartum depression after remission; (1) test for global difference (1st and 2nd play phase pooled and depressed mothers compared to healthy dyads). Statistical test is a multivariate Wei-Lachin Multivariate Rank Analysis (Wei and Lachin, 1984; Lachin, 1992; two-sided), (2) differences between depressed and healthy dyads per phase: Mann-Whitney test, one-sided, (3) and (4) effect of maternal still-face: both groups compared in change values (simple differences) from 1st play to 2nd play phase (Mann-Whitney-Test, Lehmann, 1998), two-sided, (5) and (6) test for a trend from “in episode” to “after remission” (1st and 2nd play were averaged) compared to doubly tested healthy dyads (t1 and t2), m = mean, se = standard error. All comparisons are strictly exploratory.

		Healthy control mothers (n=35)						Major depression postpartum (n=24)						(1) Global group difference	(2) differenes per phase 1-3 (one- sided Mann- Whitney tests)	(3) changes 1st to 2nd play		(4) between groups	(5) changes 1st to 2nd observation		(6) diverging trends from 1st to 2nd observaton
		1st play		still-face		2nd play		1st play		still-face		2nd play				healthy m	depr. m		healthy m	depr. m	
caregiver negative compared to all behavior	t1	0.01	0.01	0.00	-	0.00	-	0.01	0.01	0.00	-	0.01	0.01	p=0.41	none	0.00	0.00	p=0.41	0.00	0.01	p=0.88
	t2	0.01	0.01	0.00	-	0.00	-	0.00	-	0.00	-	0.00	-	p=0.29	none	0.00	0.00	p=0.25	0.00	0.00	
infant negative compared to all behavior codes	t1	0.06	0.02	0.09	0.03	0.08	0.03	0.04	0.02	0.11	0.03	0.06	0.02	p=0.38	none	0.00	0.00	p=0.77	0.07	0.05	p=0.83
	t2	0.05	0.02	0.12	0.03	0.10	0.03	0.02	0.01	0.11	0.04	0.13	0.04	p=0.36	none	-0.10	-0.10	p=0.69	0.08	0.07	

7.2.4 Lowered effects of maternal affective unavailability in dyads with a major depression, 4th exploratory hypothesis

Based on the prediction that infants of depressed mothers are unaffected by maternal withdrawal or unavailability (e.g. due to an adjustment to low behavior frequencies in the presence of maternal depression) both groups were compared with respect to their response to the maternal "still-face". In an extension to the results reported above (this was the parameter affect mirroring, see page 138 and figure 6, page 134), both groups are now compared with respect to specific affect codes (see figure 14). Infants exposed to a withdrawn and depressed mother were expected to be unaffected by the artificial episode of maternal unavailability (i.e. the still-face situation). Differences in infant behavior frequencies and durations from pre- to post maternal still-face were calculated and submitted as dependent variables to statistical tests.

The infant responses (as pre- minus post-still-face differences on the y-axis) are displayed in figure 14. It shows that infants of depressed mothers showed no differences in reaction (i.e. in changes from pre- to the post still-face situation), either in presence of maternal depression (MDE) or following remission ("Rem").

Moreover, the nonparametric multivariate test revealed no general differences (neither for MDE, nor for the remission, nor for the course from in-episode to after-remission). Thus, infants of depressed mothers showed no differences in reaction to a brief period of simulated maternal unavailability when compared with control dyads.

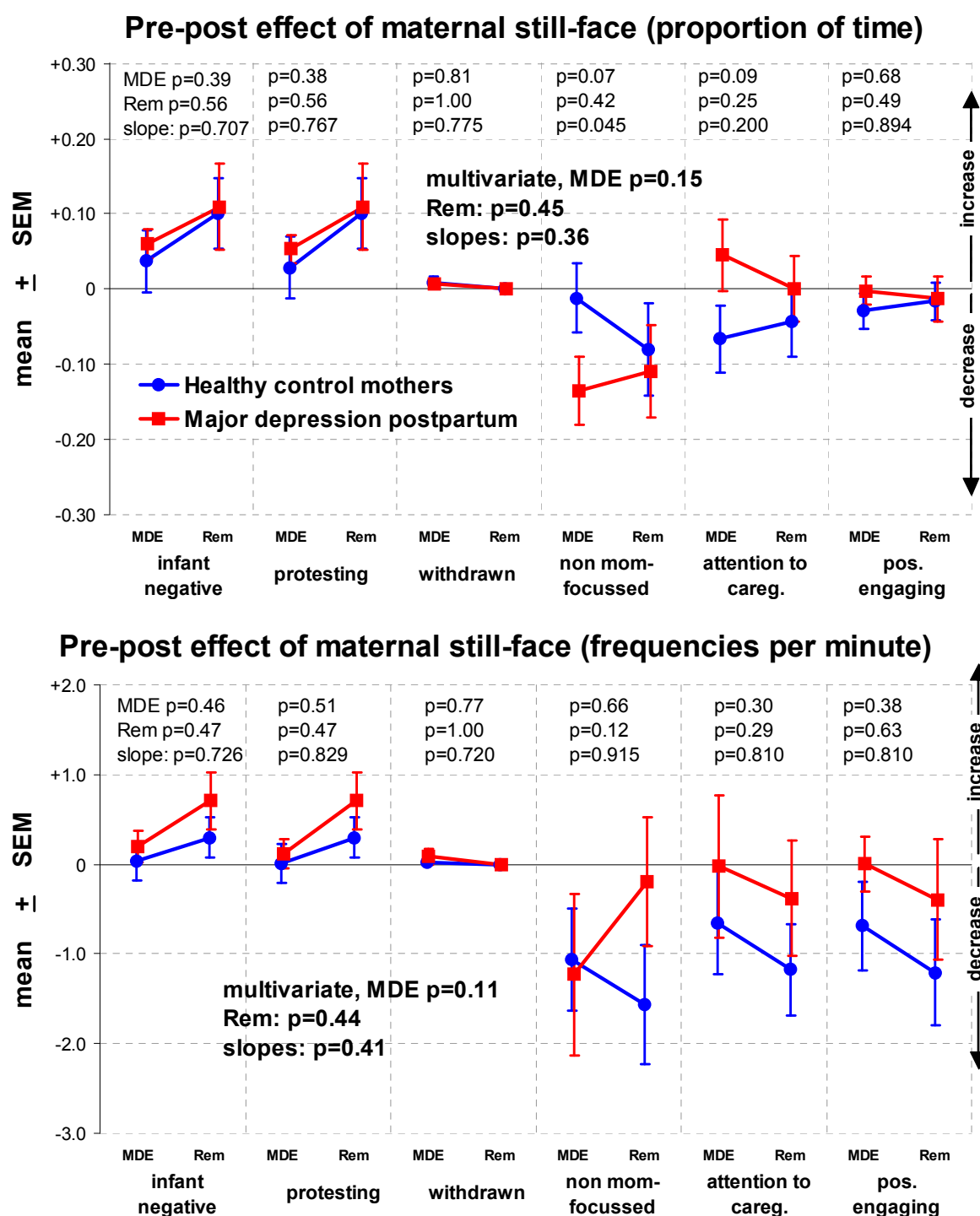


Figure 14: Infant behaviors as differences from pre- to post-phase of maternal unavailability (still-face) in frequencies per minute or as durations (time proportions). Positive values denote an increase while negative values denote a decrease from the period from before to after the phase of maternal still-face. Statistical test: Mann-Whitney test, two-sided (Lehmann, 1998). Multivariate test: Wei-Lachin Multivariate Rank Analysis (Wei and Lachin, 1984). All p-values are strictly exploratory.

7.2.5 Restrictions in behavior repertoire associated with major depression, 5th and 6th exploratory hypotheses

Individuals with major depression have regularly been claimed to have lower interaction skills and, in cases where behavior is mirrored by the infant or reinforced by the mother, their infants, are also predicted to have lower interaction skills. Here

and technically, “interaction skills” were operationalized as usage of the full range of the available interaction codes (see all codes tabulated on page 126). They were sorted in ascending order on a negative-positive dimension (also refer to Lovejoy et al., 2000), and scores were assigned to the behavior categories (refer also to table 39 on page 178). For each individual the standard deviation was calculated as a measure of how variably the different code categories were used. For example, an infant using protest only would be given the standard deviation of zero. Similarly, a mother who is coded as withdrawn only, irrespective of how her infant acted, would show a small variability. On the contrary, a mother who switches from vocalizations to infant monitoring and to withdrawal and then back to vocal engagement would be given a higher variability because she uses a wider spectrum of different behavior codes. Now, these variability data are given in figure 15 below. However, the data revealed no differences between dyads with and without a depressed mother. Numeric differences, i.e. in absolute values, were small. Thus, dyads with a depressed mother did not display lower variability of behaviors. Consequently, there was no indication of a lower behavioral repertoire or reduced interaction skills in dyads with a mother in major depression.

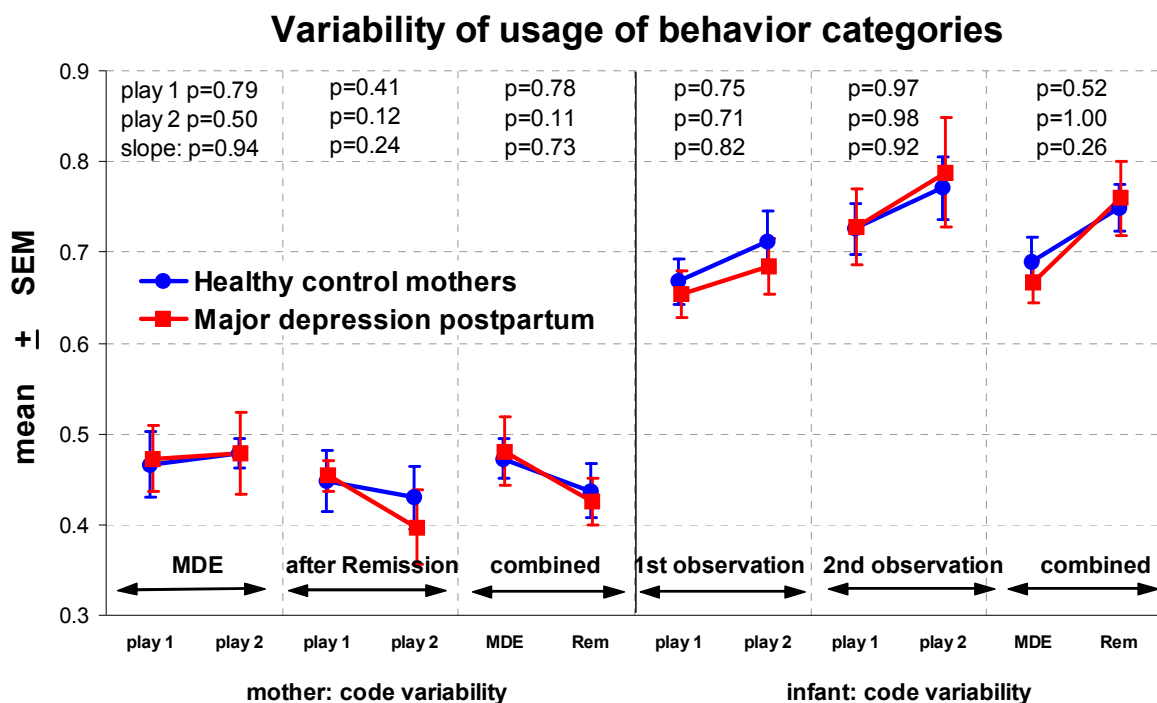


Figure 15: Variability over behavior categories. Categories were ranked from negative to positive (left side: mothers, right side: infants). Higher values reflect a more variable use of the sorted code categories. The still-face phase is located between play 1 and 2, but not displayed due to floor effects in maternal behavior, mostly due to the given instruction to the mothers. Two group differences tested with Mann-Whitney test (Lehmann, 1998), all comparisons are strictly exploratory.

7.2.6 Reduction in general speed of interaction in dyads with a depressed mother, 7th exploratory hypothesis

With respect to the maternal depression profile (e.g. loss of energy and flat affect) a generally lower “production” of affect-related behaviors was expected in the presence of major depression. This was operationalized as occurrence of any maternal affect-engagement (vocalizations, positive or even exaggerated or hostile behavior towards the infant). Any engagement by the infant was also counted (vocalizations, paying attention to the mother, and even protesting behavior). Codes such as withdrawal, non mother-focusing or neutral maternal behaviors were excluded and not counted as engagement. The results are shown in figure 16.

Behavior production rates of about 4 to 7 per minute were found. However, deviations in neither depressed mothers nor their infants occurred; thus, there were no indications of a reduced interaction speed in association with maternal depression, neither for the mother, nor for the infant.

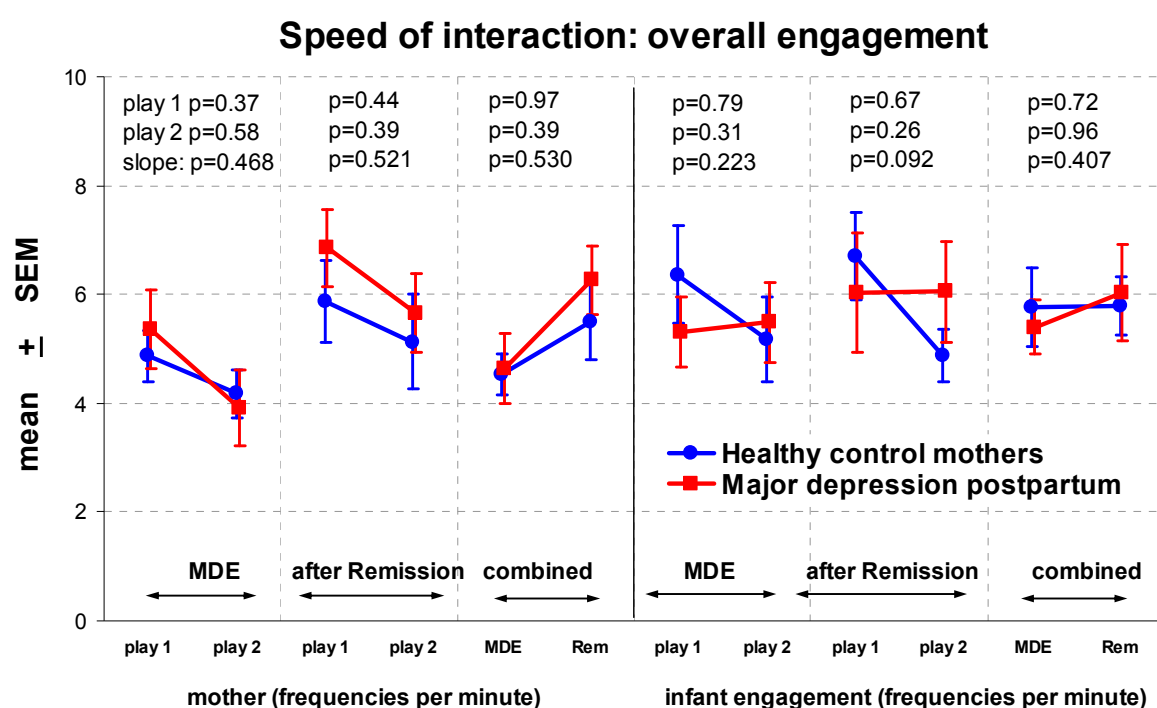


Figure 16: Speed of interaction as engagement frequencies per minute (averages and standard error) in healthy dyads and dyads with a depressed mother (left side: mothers, right side: infants). Higher values reflect a more variable use of the sorted code categories. The still-face phase is located between play 1 and 2, but not displayed due to floor effects in maternal behavior, mostly due to the given instruction to the mothers. Two group differences tested with Mann-Whitney test (Lehmann, 1998), all comparisons are strictly exploratory.

The overall maternal engagement time (figure 17 with durations) was well over 60% of the total observation time; for infants it was between 40 and 50%. None of the

differences in figure 17 reached exploratory significance. Thus, dyads with a depressed mother did not differ in their interaction speed in terms of behavior production frequencies or time.

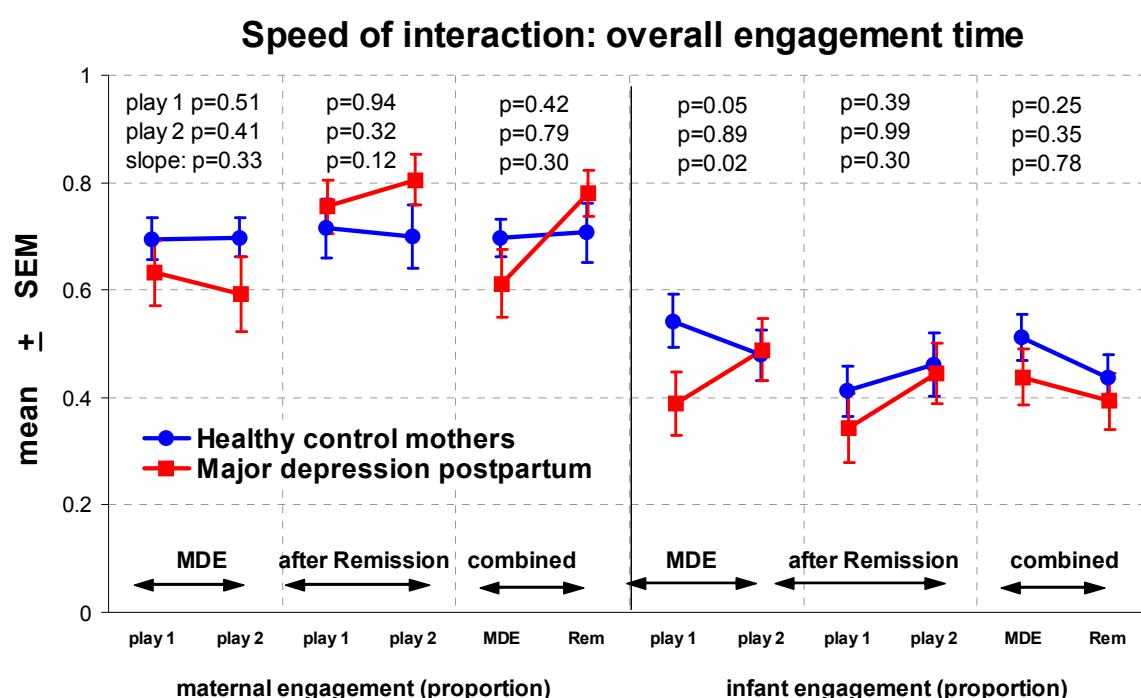


Figure 17: Speed of interaction as engagement time proportions (averages and standard error, e.g. 0.6 = 60% of total time) in healthy dyads and dyads with a depressed mother (left side: mothers, right side: infants). Higher values reflect a more variable use of the sorted code categories. The still-face phase is located between play 1 and 2, but not displayed due to floor effects in maternal behavior, mostly due to the given instruction to the mothers.

7.2.7 Rejection-inducing effects of maternal depression, 8th exploratory hypothesis

With respect to theories that suggest that a depression has contagious effects and that a depressed individual is supposed to induce rejection in the interaction partner, it was predicted that aversive infant behavior (such as protest, withdrawal from interaction, non-maternal focusing) may particularly accumulate with ongoing mother-infant interaction when a mother is depressed. This accumulation of child negativity is predicted to result in a steeper linear slope in infants of depressed mothers compared to infants of non-depressed mothers. Technically, and for exploratory reasons, slopes were calculated within each dyad based on a simple linear regression of time (SPSS procedure "regression") on the cumulative value of events (e.g. the sum of infant protest over time). For example, a slope value of one denotes the accumulation of one negative behavior every second, a slope of 1/30 every 30 seconds. Particularly in infants with a depressed mother, the negativity is expected to increasingly cumulate with increasing interaction time.

Slope averages are shown in figure 18 (infant withdrawal could not be calculated due to floor effects). As can be seen in the figure no differences in the cumulative increase of non-mother focused behavior or protest emerged. Thus, there was no higher accumulation of negativity in infants of depressed mothers with increasing time of interaction.

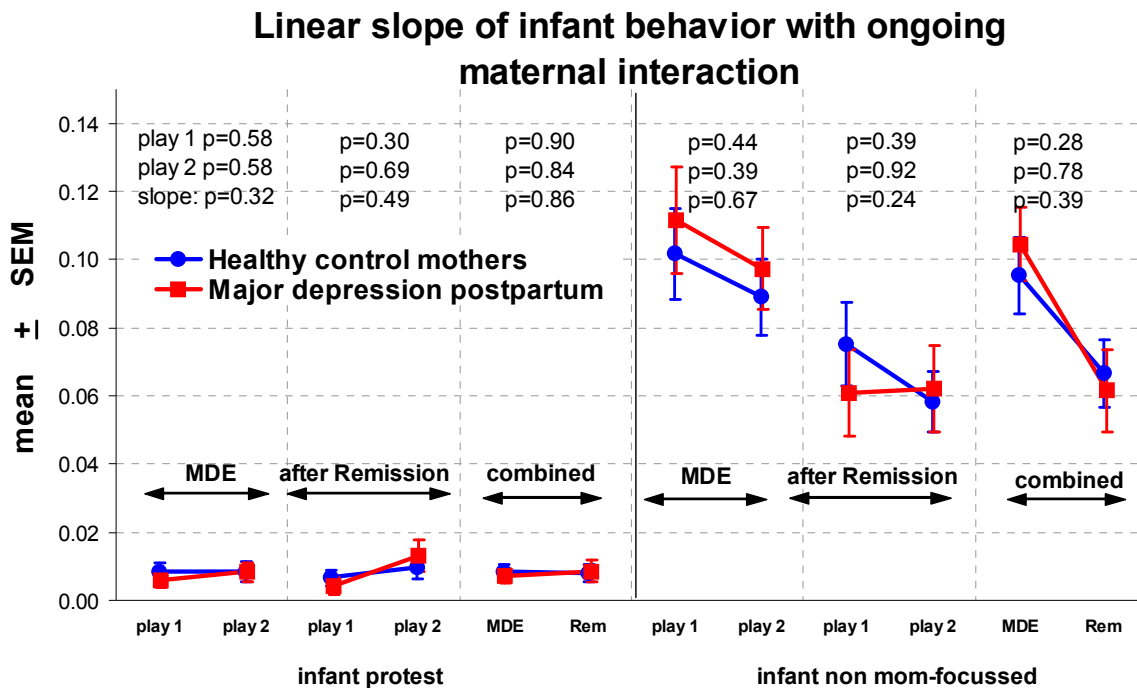


Figure 18: Regression slopes of infant protest and non-mother directed behaviors, with increasing interaction time (a slope of 0.10 refers to one infant-behavior within 10 seconds), data are given as means and standard error. Statistical tests: Mann-Whitney test, both for differences at point of measurement and pre-post-differences (one-sided, Lehmann, 1998)

7.2.8 Increased unwillingness to interact in infants of depressed mothers with increasing maternal engagement, 9th exploratory hypothesis

Based on theories of mood contagion and associated effects the prediction was derived that infants of depressed mothers will display higher negative behaviors (protest, withdrawal) or interaction avoiders (non-mother focusing) despite their mother's being coded with heightened engagement.

Accordingly, higher rates of such avoiders were expected, especially if their mothers acted highly positively. Technically, the relation of non-mother-directed infant behaviors (protest, withdrawal, non-mother focusing) compared with maternal positive behaviors was calculated, and computed as a simple ratio per dyad. Maternal engagement is operationalized based on positive behavior codes, i.e., with or without vocalizations, even if coded as exaggerated behavior. The higher the individual ratio is, the higher the level of infant negativity in spite of maternal

engagement (e.g. a ratio of one denotes one infant avoiding behavior per maternal engagement code).

However, as shown in figure 19, infants of depressed mothers had no heightened ratios of interaction-avoidant behaviors per maternal positivity (compare p-values for the first interaction period and the changes to the 2nd period; see p-values of slopes). There were indeed outliers, particularly in infants of depressed mothers (denoted by huge standard errors).

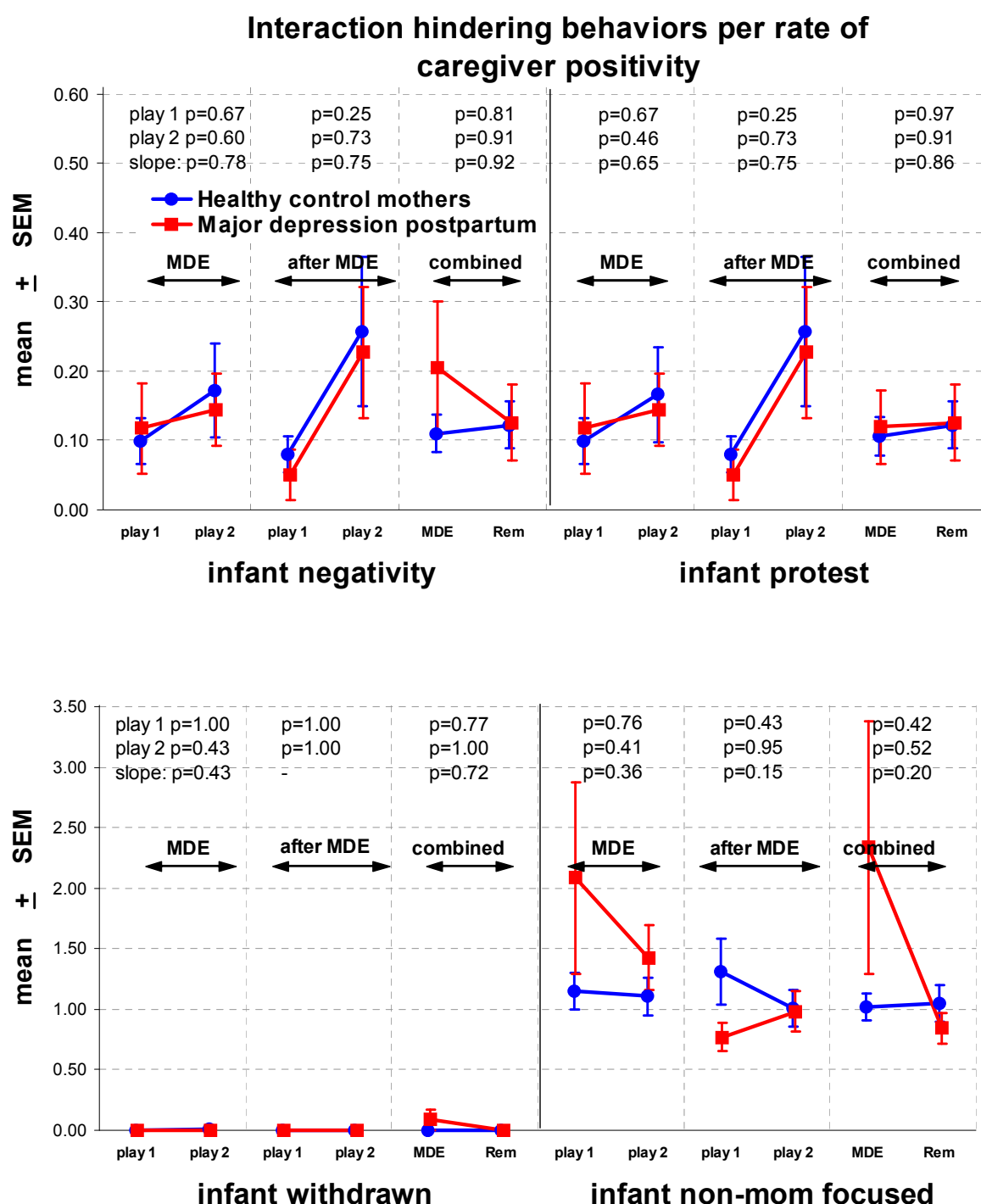


Figure 19: Ratio of interaction involving hindering or avoidant behaviors per rate of caregiver-positivity. Two group differences tested with Mann-Whitney test (one-sided). All comparisons are strictly exploratory.

In general there were no differences between groups in ratios of infant protest and non-maternal focusing behavior as related to maternal engagement. Thus, infants of depressed mothers did not show higher frequencies of interaction avoiders when controlled for maternal positivity: despite high maternal engagement, negative infant behaviors were not increased in infants of depressed.

7.2.9 Depression-associated reciprocal negative affects; negativity spirals or depression-associated reduction in positive reciprocals, 10th exploratory hypothesis

Based on theories of mood contagion, it was expected that dyads with a depressed mother would engage in a reciprocal level of negativity, i.e., be prone to interpersonal spirals of negativity. Until today interpersonal spirals have not been measured in mother-infant interaction. In descriptive terms, the simplest form of a reciprocal behavior is a 3 way pattern of interaction (for example, the mother is withdrawn, then infant protests, then the mother becomes hostile, or: the infant is withdrawn, then the mother exaggerates positively, then infant protests). Three-way patterns were simply counted in a first step. This resulted in a raw-extraction of a total of 2554 three-way patterns of dyadic exchange (see table 28 on page 160).

Table 28 primarily shows that exchanges of positivity were found to either follow or precede infant negativity (ranks 1 and 2). Reciprocal positive behaviors were found with rates of 11.4% and 9.6%, respectively (ranks 3 and 4). Reciprocal negative behaviors were rare (ranks 13 and 15) depending upon who initiated these behaviors: Rates were higher (3.2%) when the infant started first (compared to 0.3% when the mother started first).

The table 28 reveals almost negligible differences between dyads with and without a depressed mother, for example compare columns (5) and (6). Differences between groups were negligible even when the depression was remitted, e.g., compare columns (7) and (8).

Table 28: Descriptions of **three way sequences of dyadic exchange**; patterns were simply counted, (table columns 1-3, for statistical tests refer to table 29). The patterns were sorted by their overall occurrence (table column 4) and their occurrence within groups (columns 5 - 8). Measurements t1 = MDE (major depressive episode), t2 = after remission of MDE, inf. = infant, pos. = positive, neg. = negative). Behavior codes were previously aggregated as: "inf. neg.", infant negative (e.g. infant protesting, withdrawn, non-mother-focused), or maternal behaviors ("mom. neg."), e.g. mother hostile, withdrawn or neutral, versus interaction directed behaviors: "inf. pos." infant attending or positive, "mom. pos.": mother: positively engaging with or without vocalizations)

(1)		(2)		(3)		(4)		(5)		(6)		(7)		(8)	
rank	behavior sequence					occurrences	%	t1, Major depression postpartum	%	t1, Healthy control mothers	%	t2, Major depression postpartum	%	t2, Healthy control mothers	%
1	inf. pos.	-->	mom pos.	-->	inf. neg.	376	14.7%	89	15%	109	14%	72	15%	106	15%
2	inf. neg.	-->	mom pos.	-->	inf. pos.	374	14.6%	87	15%	119	15%	72	15%	96	14%
3	mom pos.	-->	inf. pos.	-->	mom pos.	291	11.4%	69	12%	72	9%	66	14%	84	12%
4	inf. pos.	-->	mom pos.	-->	inf. pos.	244	9.6%	56	9%	69	9%	55	12%	64	9%
5	mom neg.	-->	inf. neg.	-->	mom pos.	176	6.9%	31	5%	77	10%	23	5%	45	7%
6	mom pos.	-->	inf. neg.	-->	mom neg.	174	6.8%	43	7%	51	6%	25	5%	55	8%
7	inf. neg.	-->	mom neg.	-->	inf. pos.	172	6.7%	45	8%	51	6%	29	6%	47	7%
8	inf. pos.	-->	mom neg.	-->	inf. neg.	145	5.7%	30	5%	62	8%	22	5%	31	5%
9	mom neg.	-->	inf. pos.	-->	mom pos.	140	5.5%	37	6%	41	5%	27	6%	35	5%
10	mom pos.	-->	inf. neg.	-->	mom pos.	124	4.9%	28	5%	27	3%	27	6%	42	6%
11	mom pos.	-->	inf. pos.	-->	mom neg.	112	4.4%	27	5%	47	6%	15	3%	23	3%
12	inf. neg.	-->	mom pos.	-->	inf. neg.	85	3.3%	16	3%	30	4%	15	3%	24	4%
13	inf. neg.	-->	mom neg.	-->	inf. neg.	82	3.2%	24	4%	28	3%	8	2%	22	3%
14	inf. pos.	-->	mom neg.	-->	inf. pos.	51	2.0%	8	1%	21	3%	13	3%	9	1%
15	mom neg.	-->	inf. neg.	-->	mom neg.	8	0.3%	4	1%	3	0%	0	0%	1	0%
						2554	100.0%	594	100%	807	100%	469	100%	684	100%
positive circles (pattern 3 & 4, above)						535	20.9%	125	21%	141	17%	121	26%	148	22%
negative circles (pattern 13 & 15, above)						90	3.5%	28	5%	31	4%	8	2%	23	3%

In order to run statistical tests the 3-way patterns of table 28 were counted per dyad (which ensures that the units of observation are independent). These data were then related to the duration of observation and are given as frequencies per minute in figure 20 and table 29 (page 162).

However, in accordance with previous findings (figure 20), dyads with a depressed mother did not show heightened occurrence of negative circles. Negative circles were very rare (in both groups) as the floor effects in the left part of figure 20 show.

Three-way patterns of positivity, however, were countable. This allowed for an indirect test of the hypothesis of a lesser occurrence of positivity circles in presence of maternal depression. Nevertheless, simple two-group comparisons showed that those dyads did not have lower rates of positive 3-way patterns (figure 20, table 29 and figure 21).

Thus, dyads with a depressed mother could not be characterized by heightened circling of negativity. Likewise, there were no impairments in circling positive behaviors.

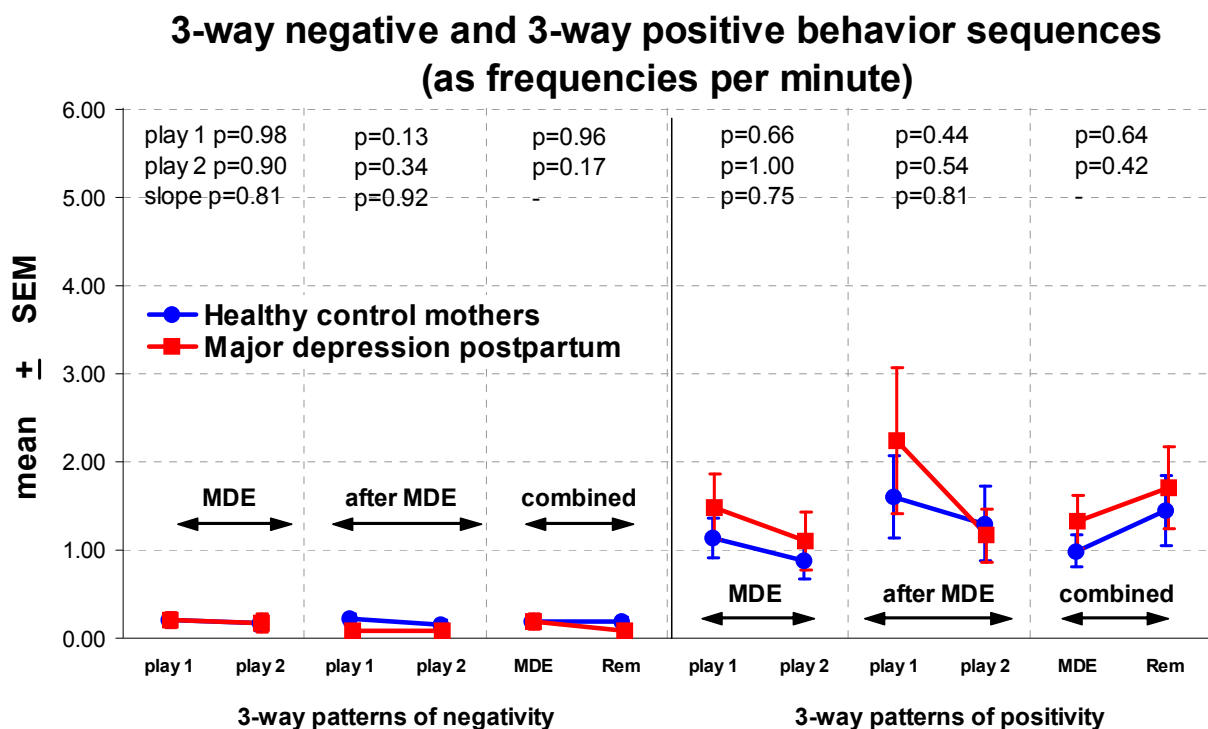


Figure 20: Three-way sequences of behaviors per minute, group means of patterns counted per dyad and related to the observation duration (displayed as frequencies per minute). For negative circles see ranks 13 and 15 (table 28, page 160), for circles of positivity see ranks 3 and 4 (table 28, page 160). Two group differences tested with Mann-Whitney test (one-sided, Lehmann, 1998). All comparisons are strictly exploratory

Table 29: Frequency of occurrence per minute of three way sequences of behaviors (unit of observation: dyad), t1 refers to the 1st observation, i.e. mothers with postpartum depression in episode, whereas t2 refers to the 2nd observation, i.e. mothers with postpartum depression after remission; (1) test for global difference (1st and 2nd play phase pooled and depressed mothers compared to healthy dyads). Statistical test is a multivariate Wei-Lachin Multivariate Rank Analysis (Wei and Lachin, 1984; Lachin, 1992; two-sided), (2) differences between depressed and healthy dyads per phase: Mann-Whitney test, one-sided, (3) and (4) effect of maternal still-face: both groups compared in change values (simple differences) from 1st play to 2nd play phase (Mann-Whitney-Test, Lehmann, 1998), two-sided, (5) and (6) test for a trend from “in episode” to “after remission” (1st and 2nd play were averaged) compared to doubly tested healthy dyads (t1 and t2), m = mean, se = standard error. All comparisons are strictly exploratory.

A	→	B	→	C	Healthy control mothers (n=35)						Major depression postpartum (n=24)						(1) Global group difference	(2) differences per phase 1-3 (one-sided Mann-Whitney tests)	(3) changes 1st to 2nd play		(4) between groups	(5) changes 1st to 2nd observation		(6) diverging trends from 1st to 2nd observaton
					1st play m	se	still-face m	se	2nd play m	se	1st play m	se	still-face m	se	2nd play m	se			health m	depr. m		healthy m	depr. m	
1	inf. pos.	mom pos.	inf. neg.	t1	0.90	0.14	0.00	-	0.66	0.13	1.02	0.19	0.12	0.12	0.83	0.17	p=0.24	none	0.26	0.09	p=0.65	0.78	0.98	p=0.42
				t2	1.10	0.21	0.03	0.03	1.01	0.21	1.03	0.23	0.06	0.06	1.04	0.23	p=0.46	none	0.10	-0.01	p=0.68	1.05	1.04	
2	inf. neg.	mom pos.	inf. pos.	t1	0.79	0.13	0.03	0.03	0.91	0.14	0.88	0.16	0.04	0.04	0.90	0.24	p=0.42	none	-0.10	0.01	p=0.53	0.83	0.87	p=0.20
				t2	1.13	0.20	0.00	-	0.82	0.19	1.04	0.23	0.00	-	1.22	0.21	p=0.36	play 2: p=0.05	0.30	-0.17	p=0.27	0.98	1.13	
3	mom pos.	inf. pos.	mom pos.	t1	0.58	0.14	0.00	-	0.45	0.11	0.82	0.19	0.00	-	0.62	0.18	p=0.20	none	0.17	0.19	p=0.73	0.50	0.73	p=0.76
				t2	0.90	0.25	0.00	-	0.79	0.26	1.16	0.40	0.00	-	0.72	0.19	p=0.39	none	0.10	0.45	p=0.77	0.84	0.94	
4	inf. pos.	mom pos.	inf. pos.	t1	0.56	0.11	0.00	-	0.43	0.11	0.66	0.19	0.04	0.04	0.49	0.16	p=0.47	none	0.15	0.16	p=0.84	0.48	0.59	p=0.79
				t2	0.71	0.23	0.07	0.07	0.51	0.18	1.08	0.45	0.00	-	0.45	0.15	p=0.47	none	0.20	0.62	p=0.64	0.61	0.77	
5	mom neg.	inf. neg.	mom pos.	t1	0.56	0.13	0.03	0.03	0.51	0.13	0.29	0.09	0.00	-	0.34	0.11	p=0.09	none	0.00	-0.04	p=0.88	0.56	0.31	p=0.67
				t2	0.46	0.15	0.00	-	0.51	0.12	0.41	0.14	0.05	0.05	0.23	0.08	p=0.26	play 2: p=0.04	-0.06	0.18	p=0.18	0.49	0.32	
6	mom pos.	inf. neg.	mom neg.	t1	0.38	0.09	0.00	-	0.38	0.11	0.47	0.12	0.08	0.08	0.43	0.09	p=0.10	none	-0.01	-0.04	p=0.48	0.38	0.48	p=0.05
				t2	0.58	0.12	0.03	0.03	0.49	0.15	0.45	0.10	0.06	0.06	0.24	0.09	p=0.26	none	0.09	0.21	p=0.86	0.54	0.34	
7	inf. neg.	mom neg.	inf. pos.	t1	0.26	0.06	0.13	0.05	0.41	0.09	0.38	0.11	0.19	0.09	0.51	0.14	p=0.28	none	-0.15	-0.21	p=0.79	0.33	0.48	p=0.54
				t2	0.42	0.13	0.23	0.07	0.39	0.15	0.41	0.14	0.16	0.08	0.33	0.12	p=0.41	none	0.03	0.08	p=0.60	0.41	0.37	
8	inf. pos.	mom neg.	inf. neg.	t1	0.38	0.10	0.25	0.08	0.37	0.09	0.31	0.11	0.11	0.08	0.26	0.08	p=0.17	still: p=0.05	0.00	0.06	p=0.56	0.38	0.28	p=0.57
				t2	0.28	0.06	0.15	0.06	0.27	0.07	0.25	0.09	0.16	0.08	0.29	0.10	p=0.41	none	0.01	-0.04	p=0.70	0.27	0.27	
9	mom neg.	inf. pos.	mom pos.	t1	0.28	0.07	0.00	-	0.32	0.07	0.36	0.09	0.04	0.04	0.40	0.12	p=0.28	none	-0.03	-0.08	p=0.64	0.29	0.39	p=0.94
				t2	0.38	0.11	0.03	0.03	0.31	0.12	0.48	0.17	0.00	-	0.27	0.09	p=0.48	none	0.08	0.20	p=0.76	0.35	0.38	
10	mom pos.	inf. neg.	mom pos.	t1	0.18	0.06	0.00	-	0.20	0.07	0.27	0.10	0.00	-	0.32	0.13	p=0.23	none	-0.01	-0.04	p=0.29	0.19	0.29	p=0.80
				t2	0.37	0.09	0.00	-	0.47	0.11	0.39	0.12	0.00	-	0.41	0.13	p=0.41	none	-0.10	-0.02	p=0.48	0.42	0.40	
11	mom pos.	inf. pos.	mom neg.	t1	0.32	0.09	0.03	0.03	0.34	0.07	0.26	0.08	0.12	0.12	0.24	0.10	p=0.23	none	-0.01	0.03	p=0.90	0.33	0.24	p=0.82
				t2	0.21	0.08	0.03	0.03	0.24	0.07	0.36	0.11	0.00	-	0.06	0.04	p=0.40	play 2: p=0.03	-0.03	0.30	p=0.02	0.23	0.21	
12	inf. neg.	mom pos.	inf. neg.	t1	0.23	0.06	0.00	-	0.20	0.07	0.20	0.09	0.00	-	0.13	0.05	p=0.25	none	0.02	0.08	p=0.80	0.22	0.16	p=0.77
				t2	0.23	0.07	0.00	-	0.27	0.09	0.18	0.06	0.06	0.06	0.23	0.09	p=0.41	none	-0.04	-0.06	p=0.94	0.25	0.21	
13	inf. neg.	mom neg.	inf. neg.	t1	0.19	0.06	0.08	0.05	0.15	0.07	0.18	0.06	0.32	0.07	0.13	0.07	p=0.46	still: p=0.01	0.03	0.05	p=0.93	0.18	0.15	p=0.19
				t2	0.20	0.06	0.15	0.06	0.16	0.05	0.09	0.06	0.13	0.09	0.09	0.05	p=0.24	none	0.03	0.00	p=0.87	0.18	0.09	
14	inf. pos.	mom neg.	inf. pos.	t1	0.12	0.06	0.16	0.06	0.10	0.04	0.06	0.03	0.11	0.08	0.04	0.03	p=0.23	none	0.02	0.02	p=0.42	0.11	0.05	p=0.22
				t2	0.06	0.04	0.12	0.07	0.05	0.03	0.19	0.09	0.17	0.09	0.09	0.05	p=0.38	none	0.01	0.09	p=0.78	0.05	0.14	
15	mom neg.	inf. neg.	mom neg.	t1	0.02	0.01	0.00	-	0.01	0.01	0.04	0.04	0.00	-	0.04	0.04	p=0.44	none	0.01	-0.01	p=0.32	0.02	0.04	p=0.40
				t2	0.02	0.02	0.00	-	0.00	-	0.00	-	0.00	-	0.00	-	p=0.41	none	0.02	0.00	p=0.41	0.01	0.00	
negative circles (pattern 13 & 15)				t1	0.21	0.06	0.08	0.05	0.16	0.08	0.21	0.09	0.32	0.07	0.17	0.11	p=0.48	still: p=0.01	0.03	0.05	p=0.81	0.19	0.19	p=0.30
				t2	0.22	0.06	0.15	0.06	0.16	0.05	0.09	0.06	0.13	0.09	0.09	0.05	p=0.22	none	0.05	0.00	p=0.92	0.19	0.09	
positive circles (pattern 3 & 4)				t1	1.14	0.22	0.00	-	0.87	0.21	1.48	0.37	0.04	0.04	1.10	0.32	p=0.37	none	0.32	0.35	p=0.75	0.99	1.32	p=0.99
				t2	1.60	0.46	0.07	0.07	1.30	0.42	2.24	0.82	0.00	-	1.17	0.30	p=0.39	none	0.30	1.07	p=0.81	1.45	1.70	

Even in specific 3-way sequences (figure 21, see below x-axis) dyads with a depressed mother did not differ from controls.

3-way behavior sequences (frequency per minute)

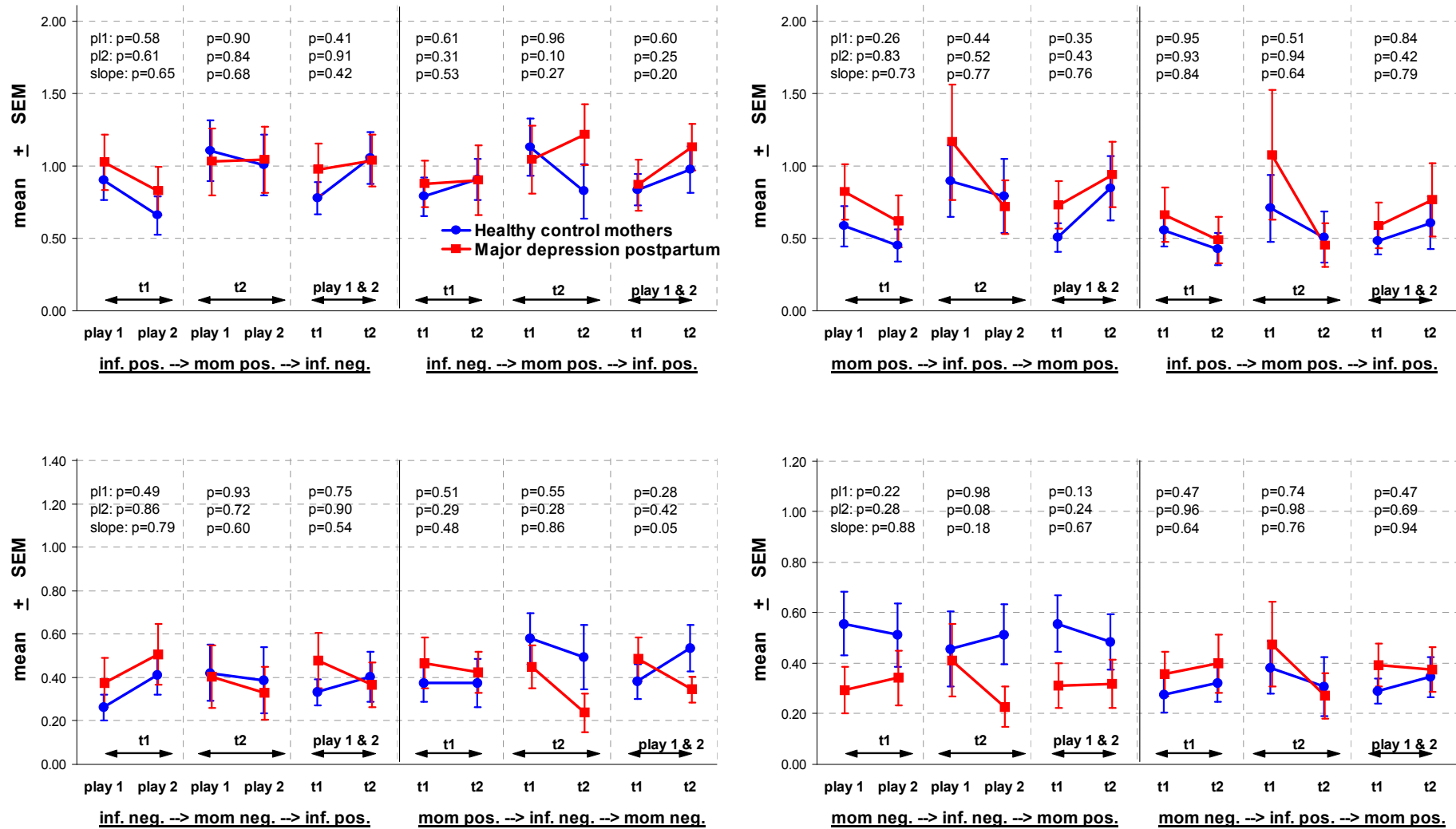


Figure 21: Frequency of occurrence per minute of specific three way sequences of behaviors (see x-axis of charts, detailed description see figure 20).

7.2.10 Engagement of depressed mothers is experienced as stressful, 11th exploratory hypothesis

Interpersonal stress approaches suggest that parenting behaviors of depressed mothers is generally perceived as negative and stressful by their children. Moreover, dysfunctional interpersonal behaviors have been suggested as one of the key mediators with regards to how the effects of depression are transmitted from mother to child. Accordingly, this allowed for the prediction that infants of depressed mothers may be much more negative, even when a generally high level of maternal engagement is present.

In particular, increased infant-protest or non-mother focusing and lowered positive infant engagement can be predicted if highly engaging depressed mothers are compared with highly engaging non-depressed mothers.

An exploratory median split (see table 30 and 31) for the general rate of maternal affective engagement was introduced and mothers with high and low levels of engagement, irrespectively of depression diagnosis, were compared.

Table 30: Median of overall affective engagement per minute; codes for maternal positivity, for positive vocalizations and exaggerated maternal behavior were counted and expressed as frequency per minute (the phase of the still-face was excluded since maternal engagement was restricted per instruction), MDE = mothers in depression, Rem = depressed mothers after remission

mother (frequencies per minute)	mean	median	min.	max.	cases
1st assessment (MDE and controls)	6	6	0	13	59
2nd assessment (Rem and controls)	7	7	2	14	41

Note that this median split - irrespectively of the chosen cut-off - still maintains a ranking order of maternal engagement, i.e., high versus low.

Table 31: Healthy control dyads and dyads with a mother diagnosed with major depression; data after a median split of general engagement (codes used: mother positive with or without vocalizations, or exaggerated positivity), t1 = 1st observation of controls and clinical group (mothers in depression), t2 = 2nd observation (depressed mothers after remission)

	1st assessment (MDE & controls)		2nd assessment after MDE Remission	
	cases	%	cases	%
Healthy control mothers & low-engag.	20	34%	11	27%
Healthy control mothers & high-engag.	15	25%	14	34%
Major depression postpartum & low-engag.	12	20%	4	10%
Major depression postpartum & high-engag.	12	20%	12	29%
total	59	100%	41	100%

As dependent measures infant-protest (figure 22), infant non-mother-focusing and

infant overall positivity were used. Due to floor effects infant withdrawal frequencies were not submitted to exploratory statistical tests.

However, contrary to the prediction, the infants of over-engaged mothers with major depression (red lines with red dots) did not interact differently, i.e., the infants did not interact with heightened protesting or less positive engagement frequencies. All in all, there were no disadvantageous effects for infants of depressed mothers who highly engaged with their children.

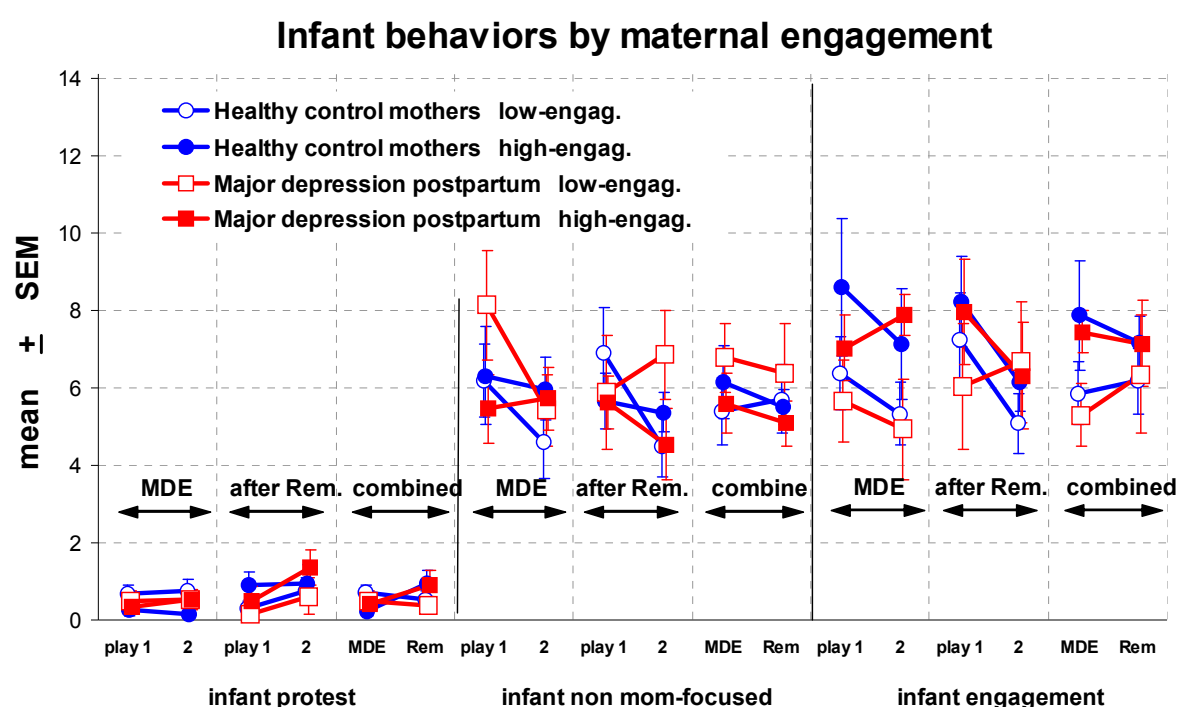


Figure 22: Mean frequencies per minute and standard error of the mean (SEM) of infant protest (left), non-mother focused behavior (middle) and overall infant engagement (right part of the figure) in association with overall maternal engagement (median split half of maternal engagement (codes used: mother positive, positive vocalization, mother exaggerated positive). All statistical tests are strictly explorative, p-values according to an exact rank analysis of variance (Mehta and Patel, 1997) have been calculated for absolute and for difference values, 4 groups), not displayed (none reached $p \leq 0.05$).

7.2.11 Reduced interest or ability to maintain interaction, 12th exploratory hypothesis

Symptom-based approaches of depression (e.g. loss of energy, motor retardation, or anhedonia, i.e., the loss of interest or pleasure to interact) allowed for the prediction that depressed mothers might be characterized by higher "interactive exhaustion", i.e., by a lowered ability to maintain interaction over time. Accordingly, depressed and non-depressed mothers were compared with respect to how their affective engagement changed over the observation period. The data show that mothers received engagement-codes for about 60-75% of the total observation time (figure

23, upper part) with a behavior initiation frequency of 6-8 per minute (figure 23, lower part). With increasing time depressed mothers basically remained on the same levels (red lines from play 1 to 2). There were high variabilities and no differences between groups (neither at points of measurements nor for the comparison of changes).

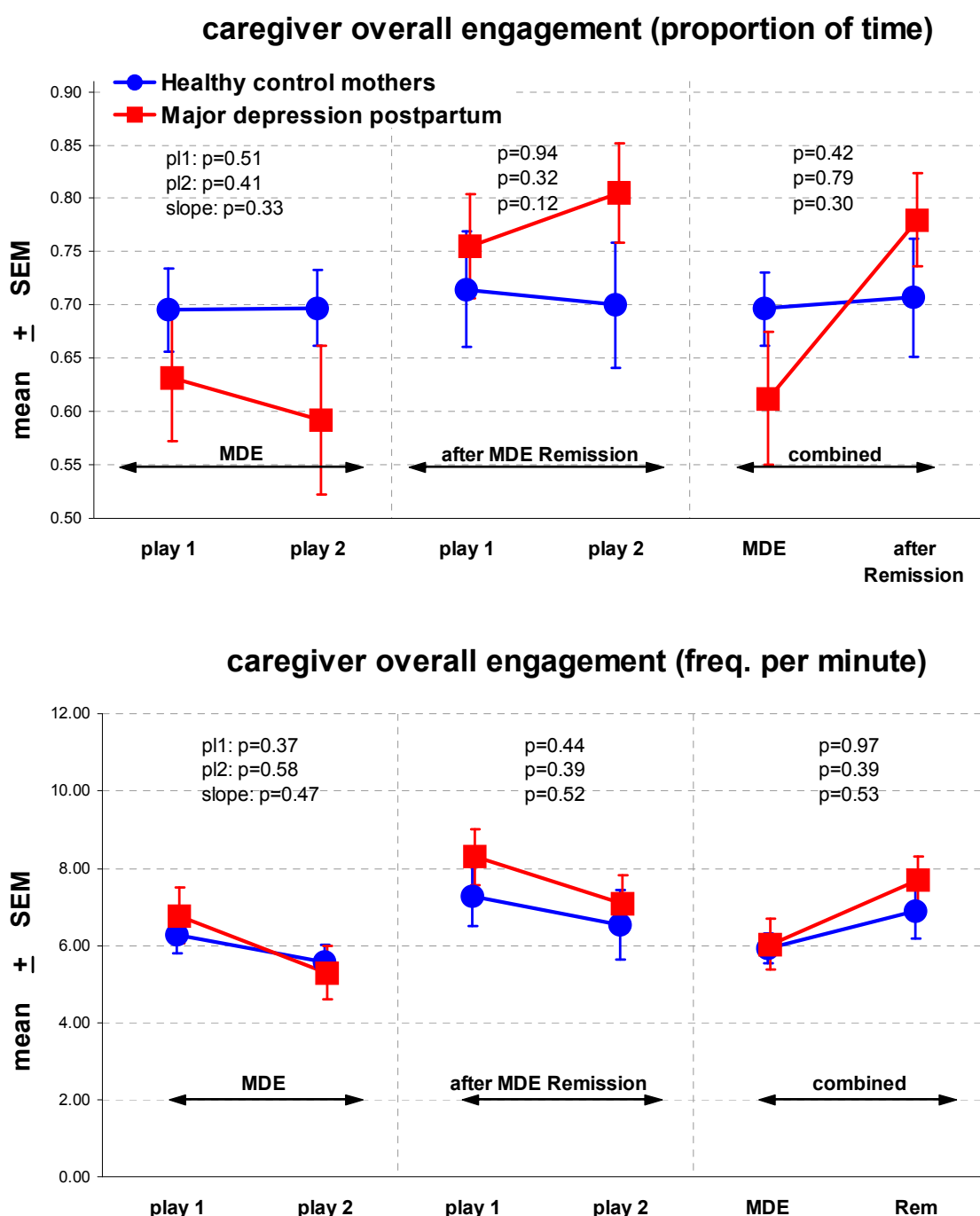


Figure 23: Proportion of total time and frequencies per minute of caregiver overall affective engagement (behavior codes used: "cpo", "cpvc", "cexg", dependent variables according to table 21 on page 127, "slope" refers to the comparison of pre-post differences). Exploratory statistical test for between-group differences: Mann-Whitney-U-Test (Lehmann, 1998).

Even if the production of affect codes were analyzed minute by minute (figure 24), there was no downward trend in mothers with major depression. Both the proportions and the per-minute frequencies of engagement of depressed mothers remained on the same level, i.e. about 70% of the observation time and about 7 engagements per minute. Curves were even not different if intra-individual changes are compared (compare point-to-point courses and refer to p-values of slopes in figure 24). Even the multivariate test failed to show that depressed mothers reduce their engagement in comparison with control mothers.

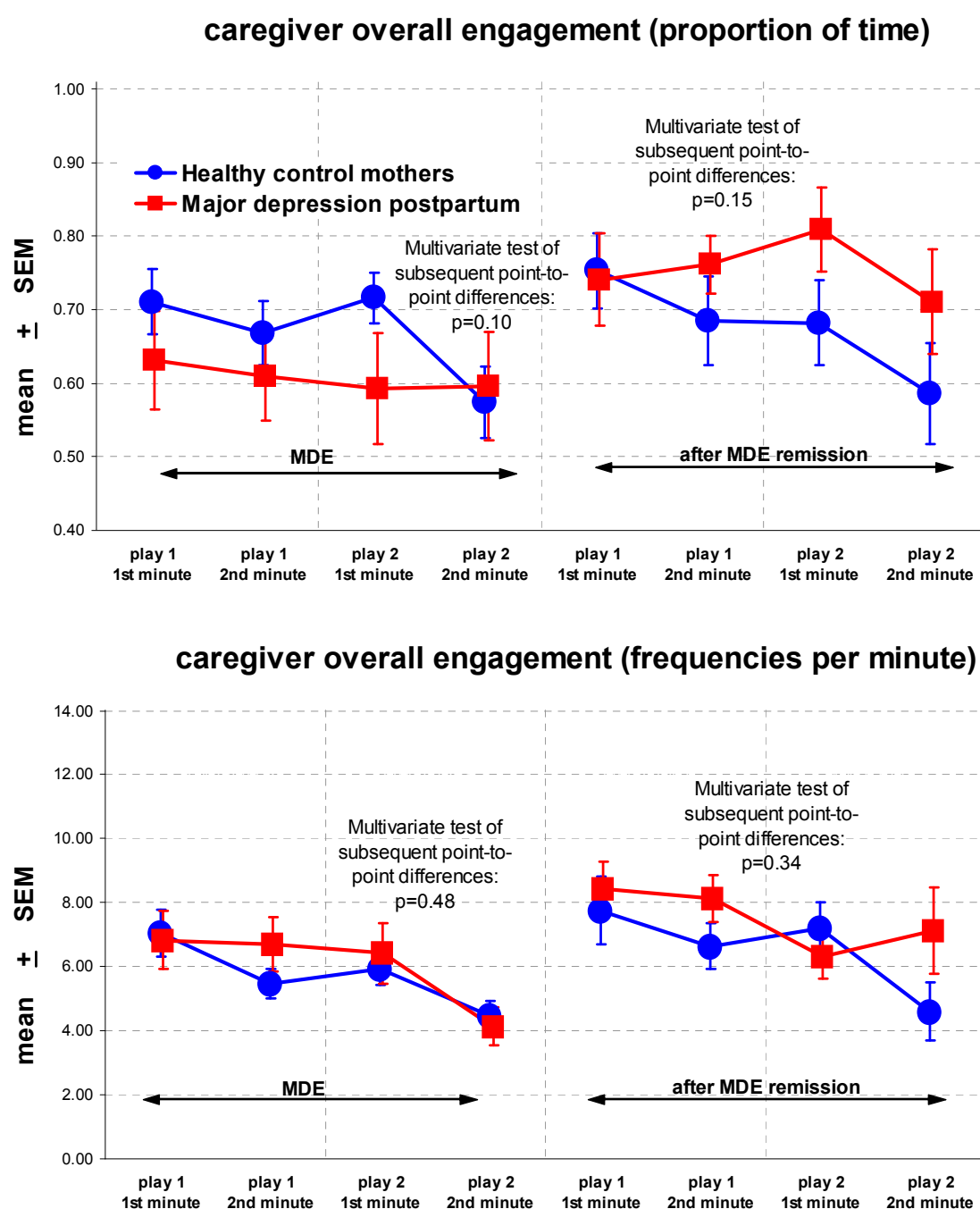


Figure 24: proportion and frequency of caregiver engagement (behavior codes of table 21 on

page 127 "cpo", "cpvc", "cexg"), statistical tests: Mann-Whitney-U-Test (Lehmann, 1998) and the nonparametric Multivariate Rank Analysis (Wei and Lachin, 1984)

7.2.12 Contingency reduction in maternal responsiveness to infant behavior, 13th exploratory hypothesis

Based on theories of restricted resources and associated reductions in responsiveness in depressed individuals, it was predicted that depressed mothers would have a lower contingent response to their infants, i.e., would respond less to infant cues. In contrast to previous analyses, however, unspecific baseline-frequencies are controlled for by the application of the lag-sequential analysis (Allison and Liker, 1982; Bakeman and Gottman, 1986; Gottman and Roy, 1990; Gottman, 1979, Sackett, 1979). This method allows for a calculation of conditional responses (e.g. baby protesting then mother vocalizes positively) in which the baseline rates are controlled for (in this example for the overall rate of maternal vocalization). Statistically Allison-Liker coefficients quantify the conditional probability of a behavior sequence that exceeds the unconditional (baseline) probability. The lag-sequential method for the analysis of interactional data has been extensively applied in marital research for interactional patterns (e.g. refer to the review of Gottman, 1998; Biglan et al., 1985; Margolin and Wampold, 1981).

Standard z-values with zero mean and a standard deviation of one were calculated per dyad and for specific behavior sequences. These z-values then were used to compare conditional responsiveness of depressed mothers in comparison with control mothers. A z-value above zero denotes responsiveness exceeding the spontaneous rate, whilst a zero z-value of zero indicates a baseline phenomenon of behavior near the overall rate. Following Bakeman and Gottman (1986) and due to low reliability expectations of rare sequences only frequent sequences were included. For example, top behavior sequences (see table 32, ranks 1-4) referred to positive or neutral maternal behavior after the infant had been previously non-mother focused or had received a neutral code.

Some descriptive analyses were done initially. They showed that mothers with major depression (refer to bold %-column in table 32) did not have markedly deviant response patterns to their infants. On the contrary, differences in responsiveness compared with control mothers were small and almost negligible.

Even if these response patterns are counted per dyad (table 33), and then related to the total observation time (i.e. transformed into frequencies per minute) and averaged

within each group, mothers with major depression did not differ (refer to bold column, p-values under (1) in table 34, page 171), i.e. did not have different response sequences if baseline behaviors were controlled.

Table 32: Maternal responsiveness to prior infant behavior, **absolute frequencies of behavior patterns** (for descriptive purposes only). Some codes are not displayed due to floor effects (e.g. codes for maternal hostility), inf. = infant, mom = mother, vocs = vocalizations, n = cases in n = 1962 sequences, i.e. patterns where a maternal behavior followed an infant behavior (t1 = 1st observation, mothers with depression in episode), t2 = after remission

rank	sequences with maternal response	n	%	t1 & Healthy control mothers	%	t1 & Major depression postpartum	%	t2 & Healthy control mothers	%	t2 & Major depression postpartum	%
1	inf. non mom-focussed → mom positive vocs	348	20%	105	16%	82	18%	98	20%	63	18%
2	inf. looking at mom → mom positive vocs	328	14%	131	20%	70	15%	70	14%	57	16%
3	inf. non mom-focussed → mom neutral	270	15%	83	13%	57	12%	77	15%	53	15%
4	inf. looking at mom → mom positive	255	13%	76	12%	70	15%	65	13%	44	12%
5	inf. looking at mom → mom neutral	180	7%	78	12%	43	9%	35	7%	24	7%
6	inf. non mom-focussed → mom positive	142	7%	40	6%	37	8%	36	7%	29	8%
7	inf. positive → mom positive	138	8%	36	6%	24	5%	42	8%	36	10%
8	inf. positive → mom positive vocs	123	6%	32	5%	35	8%	29	6%	27	8%
9	inf. positive → mom neutral	59	2%	16	2%	19	4%	10	2%	14	4%
10	inf. protesting → mom positive vocs	48	4%	19	3%	4	1%	18	4%	7	2%
11	inf. protesting → mom neutral	29	2%	10	2%	7	2%	11	2%	1	0%
12	inf. protesting → mom positive	11	1%	4	1%	2	0%	4	1%	1	0%

Table 33: Maternal responsiveness to prior infant behavior as **frequencies per minute** (for descriptive purposes only), counted per dyad and then averaged per group (still-face phase excluded, pre- and post-still-face interval were aggregated); m = mean, se = standard error; dyads where the respective pattern did not occur were given the value zero, t1 = 1st observation (mothers with postpartum depression in episode), t2 = 2nd observation (mothers with postpartum depression after remission); (1) and (2) detail the differences between depressed and healthy dyads for t1 and t2 (Mann-Whitney test), (3) shows the means of differences between t1 and t2 and (4) is the statistical test for a trend from t1 to t2

rank	frequencies per minute			Healthy control mothers (n=35)				Major depression postpartum (n=24)				(1) between groups at t1	(2) between groups at t2	(3) changes t1 to t2		(4) diverging trends from t1 to t2 (5)
				t1		t2		t1		t2				healthy m	depr. m	
				m	se	m	se	m	se	m	se					
1	inf. non mom-focused	→	mom positive vocs	0.75	0.12	1.00	0.11	0.83	0.17	0.94	0.16	p=0.41	p=0.30	+0.25	+0.11	p=0.77
2	inf. looking at mom	→	mom positive vocs	0.93	0.10	0.71	0.13	0.81	0.16	0.87	0.17	p=0.14	p=0.26	-0.22	+0.05	p=0.20
3	inf. non mom-focused	→	mom neutral	0.58	0.11	0.77	0.11	0.57	0.09	0.78	0.13	p=0.24	p=0.38	+0.19	+0.21	p=0.90
4	inf. looking at mom	→	mom positive	0.52	0.10	0.63	0.13	0.69	0.14	0.66	0.15	p=0.21	p=0.46	+0.11	-0.03	p=0.48
5	inf. looking at mom	→	mom neutral	0.54	0.10	0.36	0.07	0.41	0.08	0.34	0.09	p=0.30	p=0.43	-0.18	-0.07	p=0.72
6	inf. non mom-focused	→	mom positive	0.27	0.06	0.36	0.06	0.36	0.13	0.41	0.08	p=0.47	p=0.34	+0.09	+0.04	p=0.65
7	inf. positive	→	mom positive	0.25	0.06	0.42	0.11	0.27	0.07	0.56	0.20	p=0.37	p=0.48	+0.16	+0.29	p=0.50
8	inf. positive	→	mom positive vocs	0.23	0.06	0.29	0.07	0.35	0.10	0.43	0.15	p=0.24	p=0.49	+0.06	+0.08	p=0.52
9	inf. positive	→	mom neutral	0.12	0.05	0.09	0.05	0.18	0.07	0.23	0.10	p=0.13	p=0.06	-0.02	+0.05	p=0.93
10	inf. protesting	→	mom positive vocs	0.14	0.04	0.19	0.06	0.04	0.02	0.11	0.04	p=0.06	p=0.27	+0.05	+0.07	p=0.64
11	inf. protesting	→	mom neutral	0.07	0.03	0.12	0.04	0.07	0.03	0.01	0.01	p=0.39	p=0.02	+0.05	-0.06	p=0.17
12	inf. protesting	→	mom positive	0.02	0.01	0.04	0.04	0.02	0.01	0.01	0.01	p=0.49	p=0.40	+0.02	-0.01	p=0.84

Descriptively (refer to table 32 and table 33), depressed mothers did not react much more neutral, nor did they act less positively than the control mothers. On the contrary, they were found to respond to their infants in a way that was completely comparable to the control mothers. Finally, maternal response behaviors of table 32 were controlled for baseline frequencies with the lag-sequential method (detailed description on page 168), i.e. analyzed for their conditional response (table 34, the SPSS macro is available on request by the author). The table 34 displays z-values - intraindividually calculated and averaged within groups.

All in all, dyads with a depressed mother did not differ in their baseline-corrected reactivity. Thus, with respect to behavior frequencies there were no findings to indicate that mothers with major depression had any contingency reductions, or were less responsive to their infants.

Table 34: Maternal response patterns, z-values, results of the lag-sequential analysis (Allison and Liker, 1982), z-values above zero denote more heightened rates than expected by baseline rates alone, m = mean, se = standard error, statistical test: one-sided Mann-Whitney-U test (Lehmann, 1998 two-sided in the case of testing diverging trends), p-values lower than or equal 0.05 denote an exploratory difference, t1 refers to the 1st observation, i.e. mothers with postpartum depression in episode, whereas t2 refers to the 2nd observation, i.e. mothers with postpartum depression after remission; (1) and (2) shows differences between depressed and healthy dyads for t1 and t2: Mann-Whitney test, one-sided, (3) shows means of the differences between t1 and t2 and (4) is the test for trend (to test if both groups diverge) from t1 to t2, based on pre-post differences, m = mean, se = standard error

rank	results of the lag-sequential analysis z-values			Healthy control mothers (n=35)				Major depression postpartum (n=23)				(1) between groups at t1	(2) between groups at t2	(3) changes t1 to t2		(4) diverging trends from t1 to t2 (5)
				t1		t2		t1		t2				healthy m	depr. m	
				m	se	m	se	m	se	m	se					
1	inf. non mom-focussed	→	mom positive vocs	-0.05	0.17	0.32	0.18	0.25	0.32	0.16	0.20	p=0.16	p=0.22	+0.37	-0.09	p=0.21
2	inf. looking at mom	→	mom positive vocs	0.12	0.19	-0.28	0.24	-0.20	0.26	-0.04	0.22	p=0.35	p=0.17	-0.40	+0.16	p=0.12
3	inf. non mom-focussed	→	mom neutral	0.31	0.20	0.61	0.22	0.39	0.18	0.98	0.17	p=0.33	p=0.16	+0.29	+0.59	p=0.95
4	inf. looking at mom	→	mom positive	0.05	0.21	0.54	0.24	0.57	0.27	0.71	0.28	p=0.07	p=0.27	+0.49	+0.14	p=0.60
5	inf. looking at mom	→	mom neutral	-0.17	0.18	-0.28	0.21	-0.43	0.18	-0.71	0.22	p=0.18	p=0.10	-0.11	-0.28	p=0.48
6	inf. non mom-focussed	→	mom positive	-0.33	0.17	-0.79	0.23	-0.57	0.32	-1.08	0.23	p=0.26	p=0.12	-0.47	-0.51	p=0.58
7	inf. positive	→	mom positive	0.59	0.25	0.52	0.20	0.07	0.27	0.74	0.26	p=0.25	p=0.20	-0.08	+0.67	p=0.07
8	inf. positive	→	mom positive vocs	-0.11	0.14	-0.16	0.17	0.06	0.31	-0.54	0.16	p=0.49	p=0.06	-0.06	-0.60	p=0.19
9	inf. positive	→	mom neutral	-0.29	0.11	-0.51	0.17	-0.13	0.19	-0.20	0.19	p=0.25	p=0.12	-0.22	-0.07	p=0.24
10	inf. protesting	→	mom positive vocs	0.20	0.09	0.17	0.15	-0.13	0.15	0.39	0.20	p=0.03	p=0.23	-0.03	+0.53	p=0.15
11	inf. protesting	→	mom neutral	-0.03	0.10	0.11	0.14	0.25	0.15	-0.11	0.13	p=0.26	p=0.10	+0.14	-0.36	p=0.36
12	inf. protesting	→	mom positive	-0.21	0.07	-0.28	0.10	-0.11	0.15	-0.30	0.11	p=0.44	p=0.39	-0.07	-0.19	p=0.44

7.2.13 Deviancy in contingent reactions in infants of depressed mothers, 14th exploratory hypothesis

Based on expectations of impaired parenting of depressed mothers, restricted maternal resources, reduced responsiveness in care-giving and the associated infant under-stimulation, it was predicted that infants of depressed mothers would exhibit deviant responsiveness, i.e., be either less responsive to maternal behavior (e.g. non-mother focusing) or overly responsive (for maternal responsiveness refer to the previous chapter). A description of the patterns that occurred (table 35) predominantly shows gaze behaviors of the infant both to and away from the mother (ranks 1-4).

However, infants of depressed mothers (% column with percentages marked bold) had only small deviations in comparison with infants of control mothers (table 35 and table 36).

Thus, an initial description showed a quite comparable infant-responsiveness irrespective of a maternal diagnosis of major depression.

Table 35: Infant responsiveness in response to prior maternal behavior, **absolute frequencies of behavior patterns** (behavior sequences counted for descriptive purposes only, frequencies sorted by their rank of occurrence), inf. = infant, mom = mother, vocs = vocalizations, n = overall sequence count

rank	sequences with infant response		n	%	t1 & Healthy control mothers		t1 & Major depression postpartum		t2 & Healthy control mothers		t2 & Major depression postpartum	
						%		%		%		%
1	mom positive vocs →	inf. looking at mom	458	22%	158	23%	96	19%	118	22%	86	22%
2	mom positive vocs →	inf. non mom-focussed	340	17%	107	16%	90	18%	92	17%	51	13%
3	mom neutral →	inf. looking at mom	331	14%	108	16%	88	18%	77	14%	58	15%
4	mom neutral →	inf. non mom-focussed	267	10%	113	16%	58	12%	56	10%	40	10%
5	mom positive vocs →	inf. positive	165	7%	52	8%	34	7%	40	7%	39	10%
6	mom positive →	inf. non mom-focussed	153	8%	38	6%	31	6%	43	8%	41	11%
7	mom positive →	inf. looking at mom	152	6%	40	6%	45	9%	34	6%	33	9%
8	mom positive →	inf. positive	87	5%	22	3%	18	4%	29	5%	18	5%
9	mom neutral →	inf. positive	49	2%	12	2%	15	3%	12	2%	10	3%
10	mom neutral →	inf. protesting	45	3%	19	3%	7	1%	17	3%	2	1%
11	mom positive vocs →	inf. protesting	33	2%	8	1%	10	2%	11	2%	4	1%
12	mom positive →	inf. protesting	12	1%	4	1%	1	0%	6	1%	1	0%
13	mom non inf.-focussed →	inf. looking at mom	6	0%	3	0%	2	0%	0	0%	1	0%
14	mom withdrawn →	inf. non mom-focussed	5	0%	1	0%	4	1%	0	0%	0	0%
15	mom exaggerated →	inf. non mom-focussed	3	1%	0	0%	0	0%	3	1%	0	0%
16	mom non inf.-focussed →	inf. non mom-focussed	2	0%	2	0%	0	0%	0	0%	0	0%
17	mom withdrawn →	inf. looking at mom	1	0%	0	0%	1	0%	0	0%	0	0%
18	mom non inf.-focussed →	inf. positive	1	0%	0	0%	0	0%	0	0%	1	0%
19	mom neutral →	inf. withdrawn	1	0%	1	0%	0	0%	0	0%	0	0%
20	mom positive vocs →	inf. withdrawn	1	0%	1	0%	0	0%	0	0%	0	0%

Table 36: Infant responsiveness to prior maternal behavior as **frequencies per minute**, counted per dyad and for descriptive purposes only (still-face phase excluded, pre- and post-still-face interval were aggregated). Dyads in which the respective pattern did not occur were given the value zero, t1 refers to the 1st observation, i.e. mothers with postpartum depression in episode, whereas t2 refers to the 2nd observation, i.e. mothers with postpartum depression after remission; (1) is the test for global difference (t1 and t2 were pooled) and depressed were compared to healthy dyads, based on the Wei-Lachin Multivariate Rank Analysis (Wei and Lachin, 1984; Lachin, 1992; two-sided), (2) shows differences between depressed and healthy dyads for t1 and t2: Mann-Whitney test, one-sided, (3) shows means of the differences between t1 and t2 and (4) is the test for trend (to test if both groups diverge) from t1 to t2, based on pre-post differences, m = mean, se = standard error

rank	infant responsivity patterns (per minute)			Healthy control mothers (n=35)				Major depression postpartum (n=24)				(1) global group difference	(2) group differ in t1, in t2	(3) changes t1 to t2		(4) diverging trends from t1 to t2 (5)
				t1		t2		t1		t2				healthy m	depr. m	
				m	se	m	se	m	se	m	se					
1	mom positive vocs	→	inf. looking at mom	1.11	0.12	1.15	0.13	0.97	0.17	1.27	0.21	p=0.18	none	+0.03	+0.30	p=0.22
2	mom positive vocs	→	inf. non mom-focussed	0.75	0.10	0.89	0.13	0.98	0.17	0.76	0.10	p=0.42	none	+0.14	-0.22	p=0.13
3	mom neutral	→	inf. looking at mom	0.68	0.10	0.67	0.15	0.72	0.12	0.77	0.15	p=0.34	none	-0.00	+0.05	p=0.36
4	mom neutral	→	inf. non mom-focussed	0.67	0.12	0.46	0.09	0.58	0.15	0.45	0.12	p=0.27	none	-0.21	-0.13	p=0.99
5	mom positive vocs	→	inf. positive	0.37	0.09	0.38	0.09	0.37	0.10	0.63	0.22	p=0.46	none	+0.01	+0.25	p=0.81
6	mom positive	→	inf. non mom-focussed	0.27	0.06	0.40	0.10	0.29	0.09	0.61	0.15	p=0.41	none	+0.12	+0.32	p=0.80
7	mom positive	→	inf. looking at mom	0.28	0.06	0.33	0.08	0.45	0.10	0.51	0.09	p=0.11	none	+0.05	+0.06	p=0.51
8	mom positive	→	inf. positive	0.15	0.05	0.27	0.07	0.18	0.05	0.30	0.12	p=0.48	none	+0.12	+0.11	p=0.93
9	mom neutral	→	inf. positive	0.06	0.03	0.08	0.04	0.14	0.06	0.12	0.06	p=0.19	none	+0.01	-0.02	p=0.81
10	mom neutral	→	inf. protesting	0.12	0.04	0.16	0.06	0.04	0.02	0.03	0.02	p=0.08	still: p=0.05	+0.05	-0.01	p=0.31
11	mom positive vocs	→	inf. protesting	0.06	0.03	0.11	0.04	0.10	0.03	0.06	0.03	p=0.44	none	+0.05	-0.04	p=0.27
12	mom positive	→	inf. protesting	0.02	0.02	0.06	0.03	0.01	0.01	0.01	0.01	p=0.17	none	+0.04	+0.00	p=0.22

As in the previous chapter, infant contingency was tested based on the lag-sequential method to control for spontaneous rates of behavior. Behavior sequences with sufficient occurrence rates were analyzed for their conditional response. These measures are shown in table 37.

Some differences emerged: infants of depressed mothers were found to less frequently focus on the mother (bold lines in table 37). However, given the number of statistical tests and the high risk of α -Inflation (high risk of false-positive results) plus the borderline p-values (the bold-marked p-values in table 37 are mostly near $p=0.05$) these results appear to be weak.

All in all, with respect to behavior contingencies the evidence was not sufficient to claim that infants of depressed mothers had deviant contingency reactions.

Table 37: Infant response patterns, z-values, results of the lag-sequential analysis (Allison and Liker, 1982), z-values above zero denote more heightened rates than expected by baseline rates alone, m = mean, se = standard error, statistical test: one-sided Mann-Whitney-U test (Lehmann, 1998 two-sided in the case of testing diverging trends), p-values lower than or equal 0.05 denote an exploratory difference, t1 refers to the 1st observation, i.e. mothers with postpartum depression in episode, whereas t2 refers to the 2nd observation, i.e. mothers with postpartum depression after remission; (1) and (2) shows differences between depressed and healthy dyads for t1 and t2: Mann-Whitney test, one-sided, (3) shows means of the differences between t1 and t2 and (4) is the test for trend (to test if both groups diverge) from t1 to t2, based on pre-post differences, m = mean, se = standard error

rank	results of the lag-sequential analysis z-values		Healthy control mothers (n=35)				Major depression postpartum (n=24)				(1) between- groups at t1	(2) between- groups at t2	(3) changes t1 to t2		(3) diverging trends from t1 to t2 (5)
			t1		t2		t1		t2				healthy m	depr. m	
			m	se	m	se	m	se	m	se					
1	mom positive vocs	→ inf. looking at mom	0.29	0.21	0.34	0.22	-0.37	0.26	-0.05	0.36	p=0.02	p=0.15	+0.05	+0.32	p=0.52
2	mom positive vocs	→ inf. non mom-focussed	-0.38	0.19	-0.14	0.21	0.24	0.23	-0.46	0.38	p=0.04	p=0.09	+0.24	-0.70	p=0.08
3	mom neutral	→ inf. looking at mom	-0.07	0.18	0.11	0.22	0.36	0.26	0.41	0.19	p=0.08	p=0.15	+0.18	+0.05	p=0.54
4	mom neutral	→ inf. non mom-focussed	0.36	0.17	-0.08	0.23	-0.15	0.21	-0.05	0.25	p=0.05	p=0.48	-0.44	+0.10	p=0.22
5	mom positive vocs	→ inf. positive	0.34	0.16	0.00	0.14	0.01	0.24	0.41	0.36	p=0.08	p=0.13	-0.33	+0.40	p=0.67
6	mom positive	→ inf. non mom-focussed	0.06	0.15	0.13	0.21	-0.20	0.21	0.50	0.37	p=0.14	p=0.22	+0.07	+0.69	p=0.42
7	mom positive	→ inf. looking at mom	-0.26	0.18	-0.52	0.19	0.09	0.27	-0.28	0.31	p=0.13	p=0.26	-0.26	-0.36	p=0.73
8	mom positive	→ inf. positive	0.30	0.16	0.51	0.22	0.39	0.25	-0.09	0.29	p=0.45	p=0.05	+0.21	-0.48	p=0.24
9	mom neutral	→ inf. positive	-0.60	0.13	-0.42	0.11	-0.42	0.19	-0.37	0.28	p=0.26	p=0.47	+0.19	+0.05	p=0.94
10	mom neutral	→ inf. protesting	0.27	0.11	0.37	0.17	0.24	0.19	0.08	0.15	p=0.45	p=0.09	+0.10	-0.16	p=0.51
11	mom positive vocs	→ inf. protesting	-0.28	0.11	-0.25	0.17	0.05	0.11	0.02	0.13	p=0.07	p=0.09	+0.03	-0.04	p=0.98
12	mom positive	→ inf. protesting	-0.01	0.09	0.02	0.11	-0.20	0.08	-0.14	0.07	p=0.10	p=0.22	+0.03	+0.06	p=0.78

7.2.14 Deviation in infant regulation, 15th exploratory hypothesis

Some authors suggested that infants of depressed mothers have lowered self-regulation capacities.

Usually, infant self-regulation is defined as an infant's ability to regain control and focus on the mother after a period of intense emotions (e.g., Gillespie and Seibel, 2006; Shonkoff and Phillips, 2000), or consolability and self-quieting activity in the case of rapid excitement or irritability (Lundqvist-Persson, 2001). Tronick and colleges (see page 37 of this manuscript), for example, assume that infants of depressed mothers do not have these capacities since their mothers fail to give them regulatory help, e.g. since they might be unable to read infant cues or unable to initiate positive interaction.

Based on these suggestions the infant-ability to regain control and re-focus on the mother was compared between both groups. It was hypothesized that infants of depressed might need longer to cease crying or protesting, or might need longer to cease self-stimulatory behavior and need longer to re-focus the mother again.

In order to test the hypothesis of deviant regulation in infants of depressed mothers, the latency of terminating crying or protesting behavior and the latency until non-mother directed behaviors are terminated and the mother is focused again were compared between mothers with and without major depression.

Since the focus of interest is the time until 1st termination of behavior (infant protest, infant being non-mother focused, i.e., offset latencies), a time-to-event method, the estimator of Kaplan and Meier was used (Kalbfleisch and Prentice, 1980; also refer to the original publication of Kaplan and Meier, 1958). Differences between groups were tested based on the log-rank test (Cox and Oakes, 1984). In the case of between-group comparisons, the corresponding chi-square statistic is evaluated with one degree of freedom. Graphically the proportion of subjects experiencing the event are displayed with "stair curves", subjects without event (e.g. continuously crying infants) are counted as "censored", i.e., they are included with their whole observational time.

As displayed in figure 25 latencies of termination of protest and non-mother directed infant behavior show rapidly falling curves with an increasing proportion of infants who quickly ceased protest or stopped averting their focus. However, the exploratory log-rank tests revealed no differences between groups in the sense that infants of

depressed mothers did take longer to terminate their crying or to re-focus again.

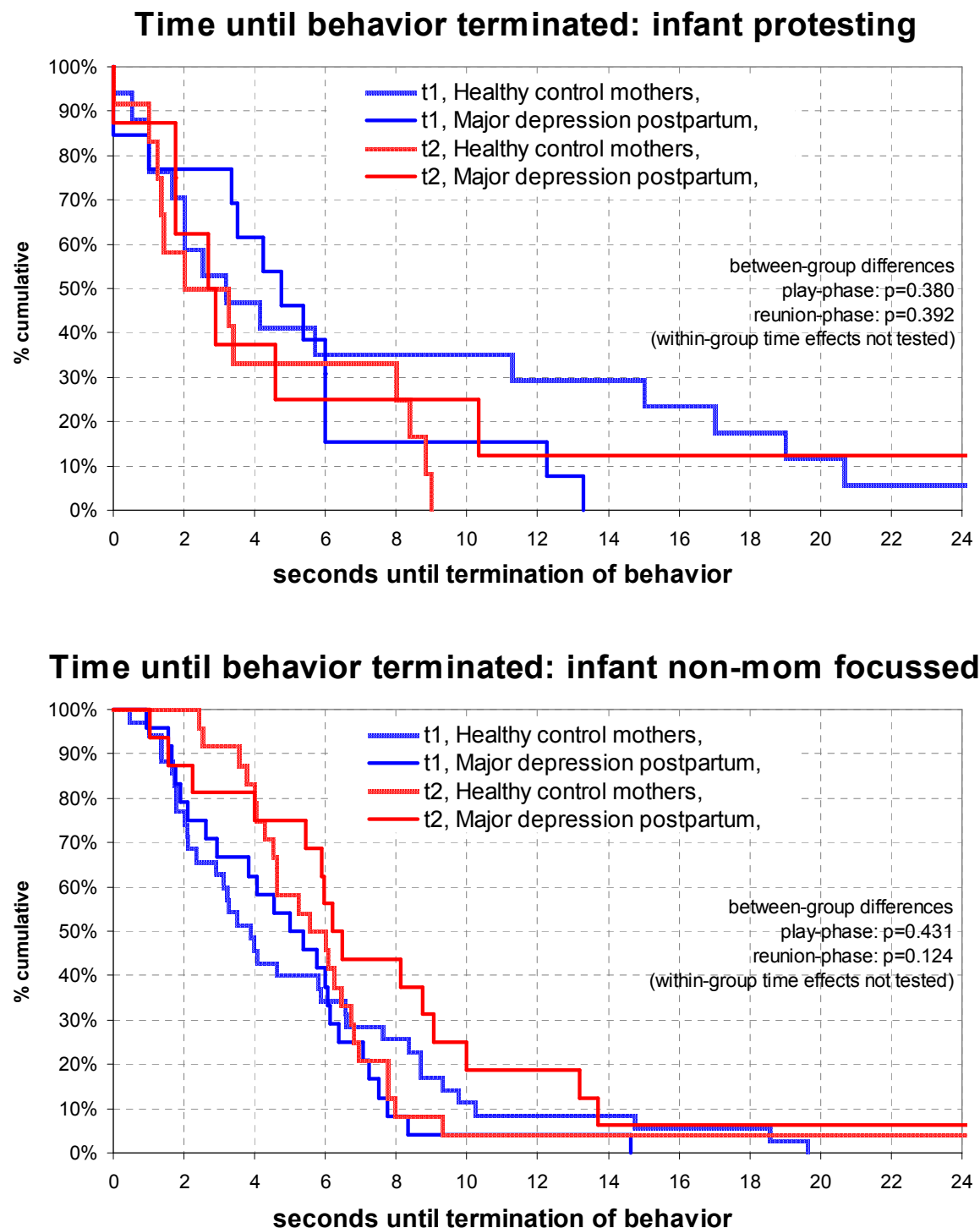


Figure 25: Latency until behavior offset, method according to Kaplan and Meier (details in Cox and Oakes, 1984), test for between-group differences: log-rank test, all p-values are strictly descriptive, the data is not stratified by phases (e.g. 1st and 2nd play phase, still-face phase excluded from the analysis due to the restriction of maternal behavior by the given instruction). Note that infant-withdrawal could not be tested due to floor effects.

Even if infant behavior is controlled by the maternal behavior which occurred parallel to the infant protest, no differences occurred, as shown in table 38. A regression according to Cox (Cox and Oakes, 1984) showed that almost none of the included parameters of maternal behavior (rows 1-3 in table 38, refer to p-values), or the interaction of maternal diagnosis and maternal engagement (rows 1-3, refer to p-values), or the diagnosis alone (row 7) were sufficiently able to predict the termination of infant behavior (in this case, infant protest).

Table 38: Combined predictors for the termination of infant protesting, regression according to Cox and Oakes (1984) for the prediction of events over time controlled by group (depression, health) and parallel occurring maternal behavior. 1st column included predictors, 2nd and 3rd column parameters of regression, 4th column p-value of predictive value of included parameter (exploratory predictive if $p \leq 0.05$). Last 3 columns refer to strength of association: odds ratio as risk ratio of behavior termination, inclusive 95%-confidence intervals

infant protesting: predictors for behavior offset (Cox-regressions, Cox, 1984)	coef- ficient	standard error	p- value	odds ratio	lower 95%-CI	upper 95%-CI
1. group x caregiver social monitor and no vocs	-0.08	0.20	0.67	0.92	0.63	1.35
2. group x caregiver social monitor, pos. vocs	0.03	0.06	0.54	1.03	0.93	1.15
3. group x caregiver social pos. engagement	0.37	0.31	0.24	1.44	0.78	2.67
4. caregiver social monitor and no vocs	0.05	0.10	0.64	1.05	0.87	1.27
5. caregiver social monitor, pos. vocs	-0.06	0.07	0.43	0.95	0.82	1.09
6. caregiver social pos. engagement	0.13	0.46	0.77	1.14	0.46	2.82
7. group	0.07	0.29	0.80	1.08	0.61	1.91

infant non-mom focussed: predictors for behavior offset (Cox-regressions, Cox, 1984)	coef- ficient	standard error	p- value	odds ratio	lower 95%-CI	upper 95%-CI
1. group x caregiver social monitor and no vocs	0.03	0.02	0.24	1.03	0.98	1.08
2. group x caregiver social monitor, pos. vocs	0.00	0.02	0.76	1.00	0.97	1.04
3. group x caregiver social pos. engagement	0.03	0.05	0.56	1.03	0.94	1.13
4. caregiver social monitor and no vocs	0.04	0.02	0.07	1.04	1.00	1.08
5. caregiver social monitor, pos. vocs	0.05	0.02	0.01	1.05	1.01	1.10
6. caregiver social pos. engagement	0.02	0.04	0.63	1.02	0.95	1.10
7. group	-0.09	0.21	0.66	0.91	0.61	1.37

To sum it up, infants of depressed mothers did not take longer to cease of protest or return their focus back to the mother. Neither maternal diagnosis nor maternal engagement pointed to lower adjustability, less controllability, or a deviant regulation (based on the definitions of above) in infants of depressed mothers.

7.2.15 Reduction in the capacity to interact synchronously, 16th exploratory hypothesis

Based on the expectation of reduced affect sharing in dyads with a depressed mother and the associated restriction of resources, depressed mothers have been predicted to have a reduced capacity to act synchronously with their child on a positive-negative dimension of affect (e.g. see Lovejoy et al., 2000).

Technically, synchrony was operationalized as intradyadic predictability, i.e. shared variance of mother and infant on a negative-positive scale of behavior codes (see table 39).

Table 39: Scores applied on a negativity-positivity dimension (1 = negative, 2 = neutral, 3 and 4 = positive). Codes in detail on page 126, table 20 and table 21.

infant code	score assigned	maternal code	score assigned
infant negatively engaging	1	caregiver negatively engaging	1
infant is protesting	1	caregiver hostile or intrusive	1
infant is withdrawn	1	caregiver withdrawn	1
infant not-mom focused	2	caregiver non-infant focused	2
infant attends to caregiver	3	caregiver infant focused	3
infant positively engaging	4	caregiver is infant focused with vocalization	4
		caregiver positively engaging	4
		caregiver exaggerated positively engaging	4

Over the behavior stream of each dyad the lag₀-cross-correlation coefficient according to Pearson was calculated as a measure for association on a positivity-negativity dimension. A mother-infant dyad interacting almost totally positively would be given a correlation of 1. A dyad where the infant protests or is withdrawn while the mother is acting positively the whole time (e.g. vocalizing) would be given a correlation of minus one (due to opposite affects).

For the comparison of groups, two measures were derived for descriptive purposes: the intradyadic proportion of shared variance of mother-infant scores (i.e. the squared correlation coefficient between the maternal and the infant series) and the corresponding Fisher-z standardized correlation coefficient as an interval scaled measure. Both are shown in table 40.

The results show that neither the shared variances nor the standardized association measure differed between the groups. The proportions of shared variance are remarkably small. Both measures revealed floor effects (well below 10%) and generally pointed to a negligible synchronicity of affective interaction.

Table 40: Measures for synchrony, Fisher-z standardized cross-correlation of dyadic data stream (the table displays averaged z-values and average % shared variance of each mother-infant pair), t1 refers to the 1st observation, i.e. mothers with postpartum depression in episode, whereas t2 refers to the 2nd observation, i.e. mothers with postpartum depression after remission; (1) test for global difference (1st and 2nd play phase pooled and depressed mothers compared to healthy dyads). Statistical test is a multivariate Wei-Lachin Multivariate Rank Analysis (Wei and Lachin, 1984; Lachin, 1992; two-sided), (2) differences between depressed and healthy dyads per phase: Mann-Whitney test, one-sided, (3) and (4) effect of maternal still-face: both groups compared in change values (simple differences) from 1st play to 2nd play phase (Mann-Whitney-Test, Lehmann, 1998), two-sided, (5) and (6) test for a trend from “in episode” to “after remission” (1st and 2nd play were averaged) compared to doubly tested healthy dyads (t1 and t2), m = mean, se = standard error. All comparisons are strictly exploratory.

		Healthy control mothers (n=35)				Major depression postpartum (n=24)				(1) global group difference	(2) group differ per phase 1-3	(3) changes 1st to 2nd play		(4) between groups	(5) changes t1 to t2		(6) diverging trends from t1 to t2 (5)
		1st play		2nd play		1st play		2nd play				healthy	depr.		healthy	depr.	
		m	se	m	se	m	se	m	se			m	m		m	m	
1: Fisher-z standardized corr.	t1	0.15	0.03	0.09	0.03	0.10	0.05	0.10	0.03	p=0.35	none	0.04	0.06	p=0.81	0.11	0.08	p=0.92
	t2	0.18	0.03	0.14	0.04	0.14	0.04	0.15	0.03	p=0.46	none	0.00	0.06	p=0.40	0.16	0.14	
2: % shared variance	t1	4.1	0.9	3.6	0.8	6.6	1.8	2.4	0.8	p=0.33	none	3.45	0.38	p=0.11	3.8	5.0	p=0.39
	t2	5.0	1.6	5.2	1.4	4.4	1.3	3.4	1.2	p=0.48	none	1.47	0.44	p=0.71	5.2	3.7	

Thus, out of two chosen measures of synchrony, the proportion of common variance and the Fisher-z standardized correlation coefficients did not differ between groups. Thus, the conclusion seems to be warranted that dyads with a mother in major depression did not interact with impairments in synchrony of affects.

7.2.16 Reduction in predictability, 17th exploratory hypothesis

Infant regulation models (e.g. the models of Gergely, page 28, or Tronick, page 37 of this manuscript) predict that infants of depressed mothers have a lower degree of regulation, particularly due to transmissions of maternal disturbance or irritability to the infant (as suggested by Tronick and Gianino, 1986). This may result in mismatching of affects, poorly coordinated interactions (and, accordingly, in low affect-predictability) in case of maternal depression.

Technically, predictability was calculated based on the Pearson correlation of mother and child's time series scored on a positivity-negativity scale and lagged against each other. A high correlation denotes that prior behaviors of one interactant predict the behaviors of the second one. A low correlation of lagged series of mother and infant denotes a low predictability of subsequent behaviors. Higher coefficients and thus higher predictability are hypothesized in healthy dyads and lower predictability in dyads with a depressed mother.

The time series of mother and infant were classified on a negativity-positivity scale (see table 39, page 178). The time series of one interactant was then shifted one lag against the time series of the other interactant. With these scores a simple Pearson correlation per dyad was computed (basically the same procedure from the previous chapter plus the shift of one series against the other). A sufficient correlation coefficient, then, may indicate that scores of one interactant may be predicted by the preceding behavior of the interaction partner. In the cases where the time series of the caregiver is one lag ahead, her behavior may be tested how it predicts subsequent infant behavior (e.g. infant protest by preceding maternal hostile behavior). On the other hand, if the time series of the infant is shifted one lag ahead, then maternal behavior may be tested for predictability.

Again, two measures were derived for exploratory reasons: The Fisher-z standardized correlation coefficient and the percentage of common variance of infant and maternal behavior codes. Both measures were calculated per dyad and then aggregated within each group and submitted to statistical tests. As shown in table 41 both the percentages of shared variance and the Fisher-z standardized correlation coefficients were remarkably small and below 0.20. Both groups did not differ in their predictability of maternal on infant behavior (table 41).

By contrast, there were some weak indications that infant behavior predicted maternal behavior to a lesser degree in depressed mothers (table 41, refer to (1), $p=0.01$, Wei-Lachin Multivariate Rank Analysis).

Table 41: Measures for predictability of maternal behavior, infant lagged ahead of mother series (and vice versa), Fisher-z standardized correlation coefficient and % shared variance calculated per dyad and then within groups, t1 refers to the 1st observation, i.e. mothers with postpartum depression in episode, whereas t2 refers to the 2nd observation, i.e. mothers with postpartum depression after remission; (1) test for global difference (1st and 2nd play phase pooled and depressed mothers compared to healthy dyads). Statistical test is a multivariate Wei-Lachin Multivariate Rank Analysis (Wei and Lachin, 1984; Lachin, 1992; two-sided), (2) differences between depressed and healthy dyads per phase: Mann-Whitney test, one-sided, (3) and (4) effect of maternal still-face: both groups compared in change values (simple differences) from 1st play to 2nd play phase (Mann-Whitney-Test, Lehmann, 1998), two-sided, (5) and (6) test for a trend from “in episode” to “after remission” (1st and 2nd play were averaged) compared to doubly tested healthy dyads (t1 and t2), m = mean, se = standard error. All comparisons are strictly exploratory.

infant lagged ahead mother series		Healthy control mothers (n=35)						Major depression postpartum (n=24)						(5) play 1 & 2 pooled		(1) global group difference	(2) group differ in 1st or 2nd play	(3) changes 1st to 2nd play						(6) changes from t1 to t2					
		1st play			2nd play			1st play			2nd play			healthy	depr.			healthy		depr.				healthy		depr.			
		m	se	n	m	se	n	m	se	n	m	se	n	m	n	m	n	m	n	m	n		m	n	m	n	m	n	
1: Fisher-z standardized corr.	t1	0.17	0.06	25	0.50	0.20	28	0.06	0.10	17	-0.12	0.11	17	0.35	31	0.02	22	p=0.01	play 2: p<0.01	0.40	22	-0.20	12	p=0.12	0.10	19	0.00	13	p=0.43
	t2	0.14	0.10	20	0.17	0.10	17	-0.05	0.14	14	0.08	0.15	12	0.14	20	0.00	15	p=0.44	none	0.00	17	0.10	11	p=0.41					
2: % shared variance	t1	10.3	2.5	25	23.6	5.5	28	11.4	3.8	17	15.4	4.6	17	17.0	31	15.0	22	p=0.11	none	15.6	22	10.9	12	p=0.59	0.5	19	0.3	13	p=0.80
	t2	16.0	4.2	20	13.4	3.9	17	14.6	5.8	14	17.4	5.5	12	14.2	20	14.4	15	p=0.49	none	-4.2	17	1.8	11	p=0.72					

mother lagged ahead infant series		Healthy control mothers (n=35)						Major depression postpartum (n=24)						(5) play 1 & 2 pooled		(1) global group difference	(2) group differ in 1st or 2nd play	(3) changes 1st to 2nd play						(6) changes from t1 to t2					
		1st play			2nd play			1st play			2nd play			healthy	depr.			healthy		depr.				healthy		depr.			
		m	se	n	m	se	n	m	se	n	m	se	n	m	n	m	n	m	n	m	n		m	n	m	n	m	n	
1: Fisher-z standardized corr.	t1	0.27	0.09	28	0.00	0.29	30	0.25	0.09	20	0.26	0.12	16	0.17	33	0.26	22	p=0.39	none	-0.50	25	0.00	14	p=0.62	-0.20	21	0.20	14	p=0.59
	t2	0.20	0.09	20	0.12	0.42	19	0.43	0.27	14	0.00	0.33	14	0.16	22	0.23	15	p=0.36	none	0.00	17	-0.50	13	p=0.32					
2: % shared variance	t1	18.1	4.5	28	25.7	6.5	30	15.6	3.8	20	16.6	5.5	16	21.5	33	16.7	22	p=0.17	none	7.0	25	5.6	14	p=0.64	1.1	21	-0.5	14	p=0.84
	t2	13.0	4.4	20	30.9	8.9	19	13.4	7.1	14	28.1	8.9	14	21.1	22	20.7	15	p=0.44	none	23.6	17	13.2	13	p=0.40					

Note: reference values for a Fisher-z standardized value: e.g. $r = 0.40$ (correlation coefficient) results in a $z=0.42$, a $r=0.70$ in $z=0.87$ and a $r=0.80$ in $z=1.10$

All in all, infants of depressed mothers were not found to have a lower predictability in their affect-related behaviors. However, there was a very weak effect of lowered predictability of maternal behavior when maternal depression was present.

7.2.17 Reductions in overall responsiveness, 18th exploratory hypothesis

Based on the parenting impairment and resource restriction hypothesis (e.g. low energy) depressed mothers are expected to be less responsive in terms of a latency until a reaction occurs. Their infants, however, are expected to be deviant responsive to maternal behaviors, i.e. either react too slow due to withdrawal or too high e.g. due to irritability.

Responsiveness was operationalized as latency, i.e. as the number of seconds that elapsed between two-way patterns of behavior (e.g. infant non mother-focused, then mother using positive vocalizations). Seconds between patterns were counted and averaged per dyad if the patterns occurred several times. Since some patterns were very rare (e.g. patterns with infant withdrawal), only patterns with sufficient sample size were tested for differences between controls and dyads with a depressed mother.

The table 42 shows predominant patterns. It includes patterns with predominantly positive or neutral maternal reactions towards infant behavior. The table shows maternal latencies of behavior toward their infants ranging between 2 and 6 seconds on average.

Mothers with major depression had no remarkably increased latencies. There were no relevant differences between groups (e.g. refer to p-values table 42 under (1) and compare group means, i.e. seconds between patterns).

Moreover, depressed dyads did not show a different course from pre- to post-still-face (compare results under (3) in table 42).

Table 42: **Maternal response in seconds** after respective infant behavior (inf. = infant, mom = mother, two-way behavior patterns with sufficient rate of occurrence), t1 refers to the 1st observation, i.e. mothers with postpartum depression in episode, whereas t2 refers to the 2nd observation, i.e. mothers with postpartum depression after remission; (1) test for global difference (1st and 2nd play phase pooled and depressed mothers compared to healthy dyads). Statistical test is a multivariate Wei-Lachin Multivariate Rank Analysis (Wei and Lachin, 1984; Lachin, 1992; two-sided), (2) differences between depressed and healthy dyads per phase: Mann-Whitney test, one-sided, (3) and (4) effect of maternal still-face: both groups compared in change values (simple differences) from 1st play to 2nd play phase (Mann-Whitney-Test, Lehmann, 1998), two-sided, (5) and (6) test for a trend from “in episode” to “after remission” (1st and 2nd play were averaged) compared to doubly tested healthy dyads (t1 and t2), m = mean, se = standard error. All comparisons are strictly exploratory.

mother responding within seconds			Healthy control mothers (n=35)						Major depression postpartum (n=24)						(1) global group difference		(2) group differ in 1st or 2nd play		(3) changes 1st to 2nd play					(5) play1 & 2 pooled				(6) changes from t1 to t2				
			1st play			2nd play			1st play			2nd play			p	none	healthy		depr.		p	healthy		depr.		p	healthy		depr.		p	
			m	se	n	m	se	n	m	se	n	m	se	n			m	n	m	n		m	n	m	n		m	n	m	n		m
inf. non mom- focussed	→ mom positive vocs	t1	3.2	0.5	23	3.2	0.4	22	3.1	0.5	18	2.9	0.7	16	p=0.45	none	0.2	14	0.3	12	p=0.74	3.1	31	3.0	22	0.80	20	1.20	12	p=0.68		
		t2	2.4	0.4	19	2.2	0.2	19	2.5	0.4	13	2.1	0.3	10	p=0.34	none	-0.3	15	-0.9	9	p=0.21	2.2	23	2.2	14							
inf. looking at mom	→ mom positive vocs	t1	2.4	0.2	30	3.7	1.1	26	2.2	0.3	15	2.5	0.6	16	p=0.04	none	1.6	22	0.3	13	p=0.39	2.9	34	2.3	18	0.8	21	-0.5	9	p=0.79		
		t2	2.1	0.4	18	2.7	1.1	14	4.0	1.9	12	1.9	0.4	10	p=0.37	none	0.6	11	-2.6	9	p=0.70	2.3	21	2.9	13							
inf. non mom- focussed	→ mom neutral	t1	2.8	0.6	18	3.5	0.8	20	3.9	0.9	18	3.9	1.0	14	p=0.04	none	1.5	13	1.7	11	p=0.86	3.3	25	4.1	21	-1.5	16	-0.4	15	p=0.42		
		t2	5.4	1.3	20	2.3	0.3	15	3.8	1.1	14	3.6	1.0	10	p=0.48	none	-2.1	13	0.9	8	p=0.42	4.4	22	4.0	16							
inf. looking at mom	→ mom positive	t1	3.2	0.5	21	3.1	0.6	12	2.5	0.4	15	3.1	0.5	14	p=0.20	none	0.5	11	0.7	11	p=0.65	3.2	22	2.7	18	1.2	9	0.4	11	p=0.25		
		t2	1.9	0.3	16	1.7	0.2	12	2.6	0.4	12	2.3	0.7	10	p=0.19	none	-0.2	10	-0.6	8	p=0.39	1.8	18	2.4	14							
inf. looking at mom	→ mom neutral	t1	2.3	0.5	15	3.6	0.7	19	2.2	0.3	12	3.1	0.7	13	p=0.36	none	0.5	10	0.9	9	p=0.51	3.4	24	2.5	16	-0.4	13	-0.3	7	p=0.47		
		t2	3.5	0.7	14	3.5	1.3	9	2.6	0.6	8	2.5	0.4	6	p=0.33	none	-1.5	6	-0.3	3	p=0.89	3.9	17	2.6	11							
inf. non mom- focussed	→ mom positive	t1	6.0	1.9	12	3.4	0.9	14	6.2	2.5	10	4.9	1.3	7	p=0.49	none	-3.5	9	0.2	5	p=0.26	4.0	17	6.6	12	-2.1	8	1.6	4	p=0.17		
		t2	4.6	1.5	11	3.5	0.7	13	4.2	1.0	6	3.6	1.3	10	p=0.45	none	0.8	5	0.4	4	p=0.54	4.3	19	3.6	12							
inf. positive	→ mom positive	t1	2.4	0.6	14	2.1	0.4	8	1.9	0.5	11	2.0	0.5	6	p=0.46	none	0.2	7	-0.5	5	p=0.56	2.5	15	1.7	12	-0.4	8	-0.6	7	p=0.64		
		t2	3.0	0.8	13	1.8	0.4	6	2.2	0.8	9	1.7	0.3	5	p=0.47	none	-1.1	5	0.0	3	p=0.88	2.7	14	2.0	11							
inf. positive	→ mom positive vocs	t1	1.4	0.2	12	1.6	0.5	8	1.8	0.3	10	2.6	0.5	8	p=0.13	play 2:	-0.3	5	0.5	6	p=0.57	1.6	15	2.0	12	-0.9	7	0.3	6	p=0.20		
		t2	1.6	0.2	11	3.0	0.9	9	1.8	0.3	6	2.0	0.3	7	p=0.46	none	1.6	6	0.4	5	p=0.58	2.0	14	1.8	8							

Thus, there were practically no differences in the sense that depressed mothers were lower in responsiveness. On the contrary, in terms of descriptive data, mothers with major depression responded to their infants in latencies comparable to control mothers.

Moreover, infants of depressed mothers were predicted to be deviant responsive, i.e. to be either hypo-reactive and, or a hyper-reactive. The table 43 includes infant patterns. Again predominantly positive or neutral infant reactions on maternal behavior emerged (negative two-way patterns had to be omitted due to floor effects). Infant latencies ranged between 1 and 6 seconds on average and

infants of depressed mothers were not remarkably deviant from control infants. Again, there were practically no relevant differences between groups (refer to p-values under (1) in table 43 and compare groups in mean values of seconds between patterns). Again, infants of depressed mothers did not show any different course from pre- to post-still-face in response patterns (compare results under (3) in table 43).

Table 43: Infant response in seconds after respective maternal behavior (inf. = infant, mom = mother, two-way behavior patterns with sufficient rate of occurrence), t1 refers to the 1st observation, i.e. mothers with postpartum depression in episode, whereas t2 refers to the 2nd observation, i.e. mothers with postpartum depression after remission; (1) test for global difference (1st and 2nd play phase pooled and depressed mothers compared to healthy dyads). Statistical test is a multivariate Wei-Lachin Multivariate Rank Analysis (Wei and Lachin, 1984; Lachin, 1992; two-sided), (2) differences between depressed and healthy dyads per phase: Mann-Whitney test, one-sided, (3) and (4) effect of maternal still-face: both groups compared in change values (simple differences) from 1st play to 2nd play phase (Mann-Whitney-Test, Lehmann, 1998), two-sided, (5) and (6) test for a trend from "in episode" to "after remission" (1st and 2nd play were averaged) compared to doubly tested healthy dyads (t1 and t2), m = mean, se = standard error. All comparisons are strictly exploratory.

infant responding within seconds		Healthy control mothers (n=35)						Major depression postpartum (n=24)						(1) global group difference		(2) group differ in 1st or 2nd play		(3) changes 1st to 2nd play				(5) play1 & 2 pooled				(6) changes from t1 to t2				
		1st play			2nd play			1st play			2nd play							healthy	depr.		healthy	depr.		healthy	depr.					
		m	se	n	m	se	n	m	se	n	m	se	n	m	n	m	n	m	n	m	n	m	n	m	n	m	n	m	n	
mom positive vocs →	inf. looking at mom	t1	4.0	0.6	30	4.8	1.1	30	3.5	0.6	19	4.4	1.4	16	p=0.11	none	1.0	26	0.7	15	p=0.70	4.3	34	3.8	20	0.40	24	0.20	12	p=0.57
		t2	3.7	0.6	22	3.4	0.6	19	3.6	0.7	14	3.5	0.7	14	p=0.48	none	-0.5	17	-0.1	13	p=0.75	3.5	24	3.6	15					
mom neutral →	inf. looking at mom	t1	2.8	0.5	23	3.1	0.4	22	4.1	0.9	13	2.9	0.7	16	p=0.41	still: p=0.01	0.5	17	-0.9	12	p=0.10	2.8	28	3.3	17	-0.8	14	1.7	10	p=0.09
		t2	3.9	0.7	15	3.4	0.6	13	1.4	0.2	12	3.2	0.8	9	p=0.35	play 1: p<0.01, still: p<0.01	-0.4	11	0.1	6	p=0.61	3.5	17	2.3	15					
mom positive vocs →	inf. non mom- focussed	t1	3.6	0.4	28	2.9	0.5	21	3.1	0.8	20	5.6	1.5	17	p=0.39	none	-1.1	17	2.0	15	p=0.17	3.3	32	4.2	22	0.3	21	-2.4	15	p=0.91
		t2	2.9	0.6	21	3.8	1.2	19	6.4	3.9	14	5.2	2.8	11	p=0.26	none	1.7	16	3.1	9	p=0.17	3.4	24	6.7	16					
mom neutral →	inf. non mom- focussed	t1	2.3	0.3	18	3.7	0.7	20	3.0	0.5	14	3.9	0.9	12	p=0.36	none	0.4	12	0.6	9	p=0.62	3.2	26	3.1	17	1.1	13	1.2	8	p=0.61
		t2	1.9	0.3	13	2.5	0.5	12	1.3	0.1	8	2.6	0.6	8	p=0.36	none	0.8	9	1.4	6	p=0.72	2.0	16	1.8	10					
mom positive vocs →	inf. positive	t1	4.6	1.4	15	4.1	1.2	13	3.6	1.2	10	3.6	0.7	8	p=0.38	none	1.7	10	0.5	6	p=0.87	5.0	18	3.5	12	-1.3	9	0.8	8	p=0.66
		t2	5.2	1.9	13	4.1	2.2	6	2.6	0.5	8	2.0	0.5	5	p=0.44	none	-1.4	4	0.1	3	p=0.29	5.4	15	2.4	10					
mom positive →	inf. non mom- focussed	t1	2.0	0.5	17	2.1	0.5	10	1.2	0.1	10	3.0	0.9	7	p=0.41	none	-0.9	7	2.0	5	p=0.09	1.8	20	1.7	12	-0.9	5	-0.9	6	p=0.93
		t2	1.3	0.1	10	1.8	0.3	9	2.2	0.5	9	1.7	0.3	7	p=0.22	play 1: p=0.03	-0.2	5	-0.4	6	p=0.85	1.6	14	2.0	10					
mom positive →	inf. looking at mom	t1	2.6	0.7	13	2.0	0.5	14	2.0	0.4	11	3.0	0.9	11	p=0.21	none	-0.6	6	0.2	6	p=0.63	2.1	21	2.4	16	0.1	9	-1.6	8	p=0.02
		t2	1.6	0.2	12	2.0	0.3	8	3.2	0.7	9	3.5	0.7	10	p=0.06	play 1: p=0.02	-0.3	5	0.1	6	p=0.85	1.7	15	3.3	13					
mom positive →	inf. positive	t1	3.4	1.2	9	2.4	1.2	5	1.4	0.2	5	2.4	0.5	8	p=0.24	none					-	2.9	12	1.9	10	0.3	6	0.4	5	p=0.85
		t2	1.3	0.1	8	1.5	0.2	9	2.2	0.4	6	1.5	0.4	5	p=0.48	play 1: p=0.02	0.5	4	0.0	3	p=0.47	1.3	13	1.9	8					

Thus, no differences in infants of depressed mothers emerged in comparison with infants of control mothers. Infants of depressed mothers did not seem to be deviant responsive. In terms of descriptive data, they responded comparably to infants of control mothers.

Even when responses were counted within specific time windows, i.e. 1, 2, and 5 seconds (as shown in figure 26) no lowered response-latencies were found.

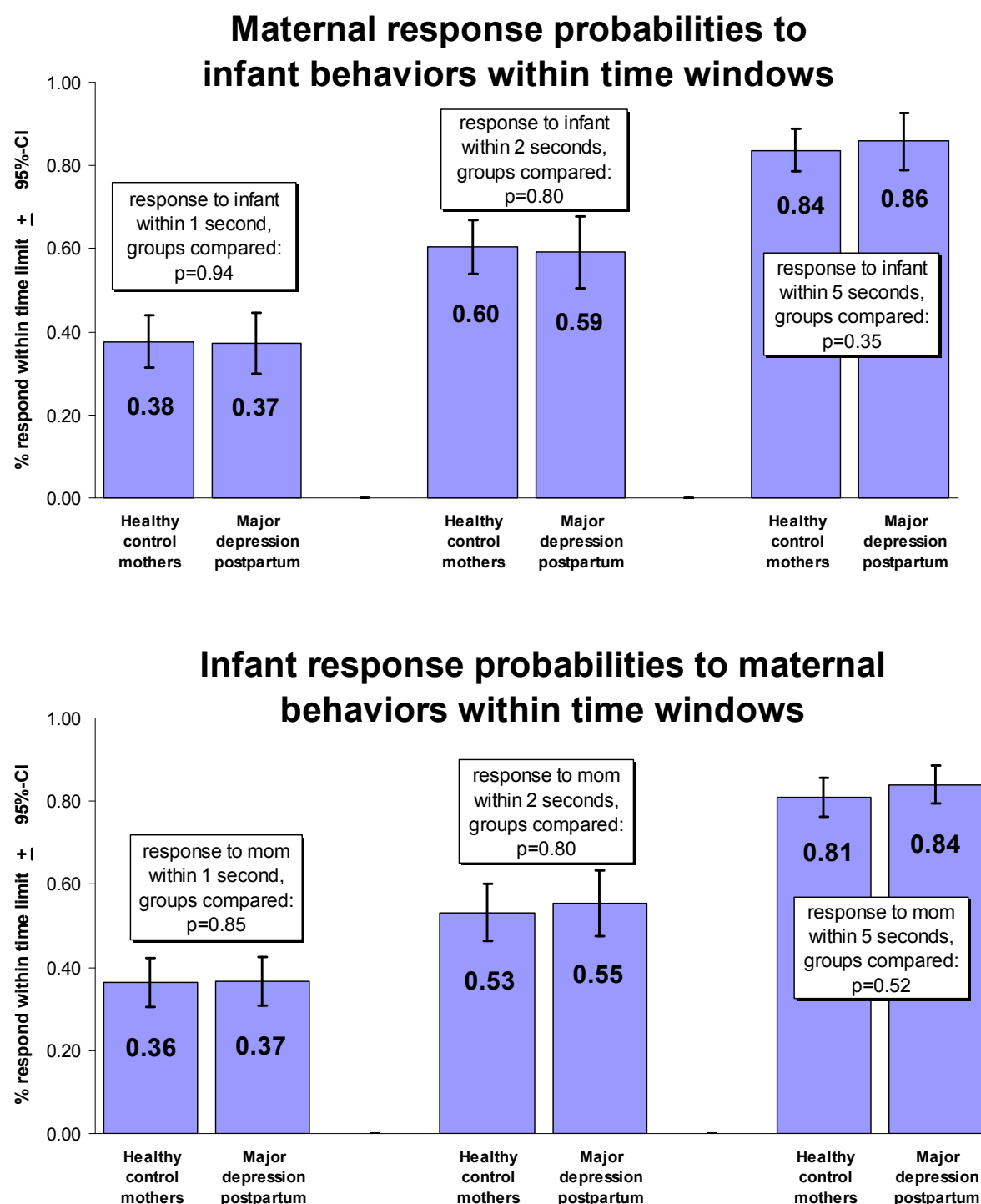


Figure 26: Averaged response probabilities of general response rates within time frames of 1,2, and 5 seconds (response latencies in seconds were calculated per dyad, intraindividually the proportion within or outside the respective time window were calculated then averaged per group, statistical nonparametric test: two-sided Mann-Whitney-U test, CI = confidence interval of respective mean)

Thus any indications of lowered responsiveness in interactive behaviors were not found in depressed mothers or infants.

Moreover, there were no remission-related effects as shown in figure 27. All changes from in-episode to after-remission did not differ between groups (see right part of the figure and refer to the “slope” p-values, “slope” means pre-post difference).

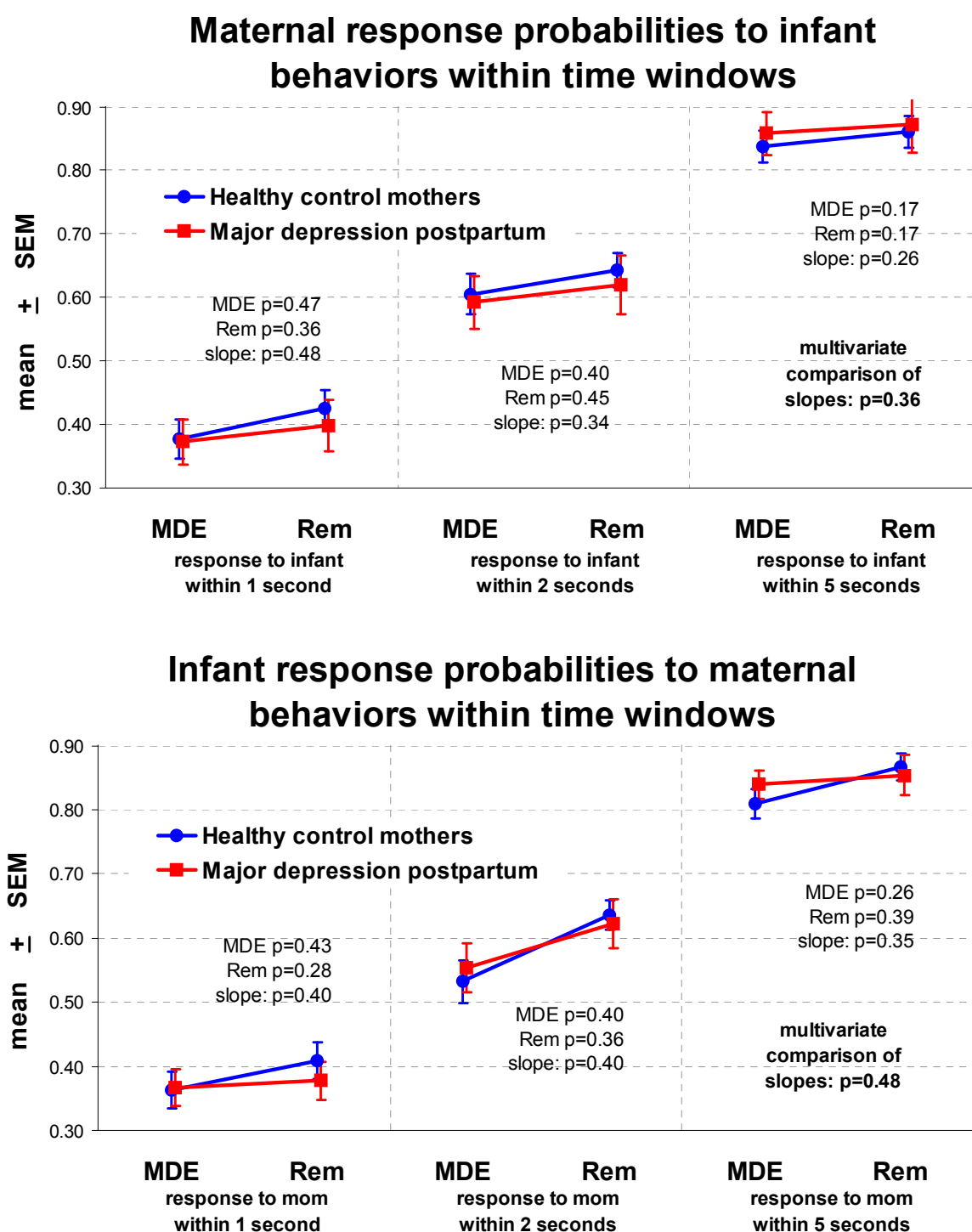


Figure 27: Averaged probabilities of general response rates from MDE to Remission, probabilities within time frames of 1,2, and 5 seconds (response latencies in seconds were calculated per response and dyad, the proportions within or outside of the respective time window were calculated per dyad, the chart above displays averages of those proportions within each group, statistical nonparametric test: two-sided Mann-Whitney-U test, the multivariate comparisons of slopes (i.e. differences from 1st to 2nd point of assessment, e.g. MDE to remission) were made with the Wei-Lachin Multivariate Rank Analysis, Wei and Lachin, 1984)

7.3. Dyadic behavior after the remission of the maternal major depression

7.3.1 Normalization of infant reactions to interrupted communication (19th exploratory hypothesis)

Based on the transient disturbance hypothesis, a normalization of infant behavior to an artificially interrupted communication after depression remission was predicted, in the sense that infants of a formerly depressed mother will respond to restrictions in maternal communication (in terms of the maternal still-face situation) no differently than when compared to controls. However, the changes in infant reactions to the still-face (data have already been displayed in figure 14 on page 152, refer to y-axis) from in-episode to after-remission were generally not different between both groups. The changes were tested with a multivariate test ($p=0.36$ for behavior time-proportion data and $p=0.41$ for frequencies, Wei-Lachin Multivariate Rank Analysis, differences from in-episode to the remission were used as dependent variables). Even if single infant behaviors were compared (compare p-values of changes, i.e. the comparison of slopes from depression to remission in figure 14), not one infant behavior after the mother remitted from depression pointed to any specific changes. Thus, the remission from major depression was not found to be predictive for a normalization of affect-related interactions, nor for how infants reacted on variations in maternal unavailability.

7.3.2 Reduction in the lack of infant stimulation after a remission of the maternal major depression, 20th exploratory hypothesis

The parental disability hypothesis allowed for the prediction that the depression remission is associated with improvements in the total level of infant stimulation. This level is no longer expected to differ from control mothers (data have already been presented in figure 9, page 140; see at the right of the chart).

There were no remission-specific changes as tests for difference values (post minus pre-values) show (refer to p-values on slopes, $p=0.15$ for proportions of maternal stimulation time and $p=0.26$ for stimulation frequencies per minute). Thus, irrespective of whether the depressed mothers were in-episode or post-remission, they neither differed from controls nor were their courses any different. The remission of depression was not associated with a restoration of overall maternal engagement and thus not with an improvement in the lack of stimulation behavior.

7.3.3 Restoration of deviant infant activity level after remission of maternal depression, 21st exploratory hypothesis

Based on the hypothesis that infants of depressed mothers are affectively under-stimulated, infants of depressed mothers were predicted to no longer be deviant in their activity level, i.e. no longer hypo-active or hyper-active. That is, a normalization of previously lower or higher overall infant behavior frequencies is expected. The data on infant activity levels (refer to figure 10 on page 141) showed no remission-specific changes ($p=0.779$ for time proportions of infant activity and $p=0.407$ for frequencies per minute, refer to p-values on slopes in figure 10 on page 141). Thus, irrespective of whether the mother is in-episode or post-remission, infants of depressed mothers did not have a different level of activity, i.e. there was no normalization in either hypo- or hyperactive behavior. Thus, a remission from maternal depression was not predictive for changes in infant affective engagement.

7.3.4 Reduction in infant negativity after maternal remission, 22nd exploratory hypothesis

Based on theories that predict that maternal depression is experienced as stressful for the interaction partner, infant negativity (e.g. protest or withdrawal) was predicted to disappear when the maternal depression disappears, i.e. after remission.

Infant protest, withdrawal or both combined are displayed in figure 13, page 149. All in all, infant negativity showed no remission-associated decreases compared with infants of non-depressed ($p=0.36$ for time proportions and $p=0.35$ for frequencies, multivariate comparison of slopes). In particular, neither overall infant negativity, or infant protest, or even infant withdrawal showed differences in courses. Thus, infants of depressed mothers showed no remission-associated changes (or even improvements) in negativity compared with dyads with a healthy mother.

7.3.5 Reduction of maternal negativity after remission of maternal depression, 23rd exploratory hypothesis

Coyne's theory of a rejection-inducing effect of depression allowed for the prediction that precursors with the potential to induce rejective infant behavior, e.g. maternal negativity, are no longer present after a remission from depression.

With respect to negative behaviors (results have been displayed in figure 12, page 145) differences in mothers whose depression remitted were not observed (multivariate tests $p=0.30$ for behavior frequencies and $p=0.36$ for negativity

durations). Even all univariate tests failed to show differences in mothers with major depression and control mothers. Thus, mothers who remitted from maternal depression did not differ in negativity, both in general and in particular, e.g. in negative engagement, hostility, withdrawal or non-infant focused behaviors. All in all, previously depressed mothers did not show any remission-associated recoveries from a presumably heightened level of negative affect displayed during interactions.

7.3.6 Recovery of deviant responsivity of mothers after the remission of depression, 24th exploratory hypothesis

Based on a recovered depression profile and the regain of functional resources after depression remission, previously depressed mothers were predicted to show no differences in their responsiveness in comparison with control mothers.

Data show (refer to figure 27 on page 186, upper part) that previously depressed mothers had no different course in their level of responsiveness if the depression remits. Neither the multivariate general measure of effect ($p=0.36$ and $p=0.48$, figure 27) nor specific time windows allowed for a differentiation of remitted mothers and healthy mothers (responses registered within a one second time window, upper part of figure 26, page 185, responsiveness within 1, 2 seconds or within a 5-second window, $p=0.94$, $p=0.80$, $p=0.35$, respectively). Even with respect to specific maternal response patterns (e.g. vocalizations or positive engagement after the infant lost the focus on the mother, table 42, page 183, p -values in last column), mothers did not act differently after remission when compared with healthy mothers. Thus, a remission of major depression was not associated with changes in maternal responsiveness in affect-related behaviors.

7.3.7 Recovery of deviant infant responsiveness after the remission of maternal depression, 25th exploratory hypothesis

The remission of maternal depression allowed for the prediction of a regain of functional maternal resources and responsiveness to reinforce the behaviors of their infants. Accordingly, the responsiveness in infants of previously depressed mothers was predicted to recover and as a result be not different to the responsiveness of control infants.

However, the data show (refer to figure 27 on page 186, lower part) that infants of previously depressed mothers had no different course in their responsiveness after the maternal depression remitted. Neither the multivariate general measure of effect

($p=0.48$) nor specific time windows of infant responsivity allowed for a differentiation of infants of remitted mothers and infants of healthy mothers (lower part of figure 27 on page 186; responses registered within a one second time window: $p=0.85$, within 2 seconds: $p=0.80$ and within a 5-second window: $p=0.52$). Moreover, even in specific patterns of infant responses (e.g. infant expresses a positive affect after a maternal positive vocalization, table 43 on page 184; refer to p-values in the last column) infants of remitted mothers did not have a different course in comparison with control infants. Thus, the remission of major depression was not associated with any changes in infant responsiveness.

7.3.8 Recovery of restricted maternal and infant behavior repertoire after the remission of maternal depression (26th and 27th exploratory hypothesis)

Since individuals with major depression have been claimed to have lower interaction skills, the remission of maternal depression was predicted to foster changes that results in skills comparable to those of healthy mothers.

Technically, skills were operationalized as usage of the full range of behaviors according to the current behavior coding system. Thus, a higher diversity of maternal behaviors should be accompanied by a larger individual standard deviation (codes were classified on a negative-positive dimension; refer to details on page 152).

Here, remission-associated effects are of primary interest. The data, however, do not indicate specific changes in maternal or infant indicators (figure 15, page 153), i.e. changes in association with the remission of maternal depression occurred. There was no increase in variability in mothers with major depression (refer to p-values of slopes from in-episode values to post-remission values in comparison with healthy dyads).

Thus, dyads with a depressed mother do not seem to recover from a previously restricted or lower variability of behavior categories in association with a depression remission (neither on the maternal nor on the infant side), and there seems to be no specific skill improvements.

7.3.9 Recovery of speed of interaction after the remission of maternal depression, 28th exploratory hypothesis

Based on the expectation that aspects of the maternal depression profile (e.g. loss of energy) are associated with a generally lower “production” of affects per time and

thus with a lower speed of interaction, the remission of depression therefore allowed for the prediction of a recovery. "Speed of behavior" or "behavior production" was operationalized as the occurrence of any maternal engagement per time unit (vocalizations, positive or even exaggerated or hostile behavior towards the infant per minute). Infant engagements were also counted and related to time (vocalizations, paying attention to the mother, and even protesting behavior). The data, however, show that neither depressed mothers (left side of figure 16, page 154) nor their infants (right side) interact at a deviant speed. Numeric differences were generally small (compare absolute level of mean values) and changes in maternal or infant speed did not show any association to the remission of maternal depression.

7.3.10 Reduction of rejection inducing effects in infants after the remission of maternal depression, 29th exploratory hypothesis

Since depressed individuals were predicted to induce rejection in their interaction partner, it was predicted that aversive infant behavior (protest, withdrawal) may accumulate in particular with ongoing mother-infant interaction when a mother is depressed. This heightened accumulation of child negativity and the associated steeper linear slope in infants of depressed mothers should no longer be present after the maternal depression remitted (slopes were calculated per dyad based on a linear regression of time with a cumulative value of, e.g., infant protest over time; a slope value of one denotes the accumulation of one negative behavior per second, a slope of 1/5 every 5 seconds, for more details see page 156). The results in figure 18 on page 156 (right side) showed no increasing infant negativity with ongoing interaction time in infants of depressed mothers and no recovery after the depression remission. Thus, in terms of cumulative infant-negativity with ongoing interaction time, there was no remission-associated recovery in infants of depressed mothers.

7.3.11 After remission of maternal depression, infants no longer show signs of unwillingness to interact, 30th exploratory hypothesis

Since mood contagion and associated effects allowed for the prediction of heightened negativity in the interaction partner infants of depressed mothers have been predicted to display higher frequencies of behaviors that hinder interaction (such as protest, withdrawal) or are suitable for avoiding interactions (non-mother focusing) despite the fact that their mothers are interacting positively.

In addition to the previous chapter the level of ongoing maternal positivity is explicitly included, i.e., infant hindrance and avoidance is related to the rate of maternal

positivity (as ratio) with the expectation of higher infant negativity despite positive maternal affect expressions when the mother is depressed.

The data (figure 19, page 158), however, showed neither higher ratios when mothers were in an episode of depression nor any specific changes in association with the remission (refer to slope-p-values in figure 19, page 158). Thus, there was no change in infants of depressed mothers with respect to interaction avoiding behaviors in the presence of highly positive maternal engagements.

7.3.12 Reduction of reciprocal negative affects after remission of maternal depression, 31st exploratory hypothesis

Based on theories of mood contagion of depressed individuals, it was expected that dyads with a mother in an episode of depression would be higher reciprocally negative and both interactants would be prone to interpersonal spirals of negativity. However, following the remission of maternal depression, reciprocal negativity should decrease and then become comparable to healthy dyads.

A test for this hypothesis was made indirectly, because there were floor effects in maternal and infant negativity. On the other hand there were high overall frequencies of positive behavior codes: accordingly, dyads with a depressed mother were predicted to recover from lowered rates of positivity spirals of after the maternal depression remitted.

The data, however, did not show changes in reciprocal patterns that were specific to maternal depression (refer to table 28 on page 160), i.e. there were no changes in rates of occurrence from in-episode to post-remission that differed from those of controls. If reciprocal patterns were counted per dyad and displayed as mean frequencies per minute (refer to figure 20 on page 161) no differences in courses emerged after the depression remitted (refer to the comparison of slopes in figure 20): None of the p-values indicated exploratory differences between groups. On the contrary, spirals of negativity were hardly observable. Thus, there were no indications that interpersonal spirals of negativity would decrease with the remission of maternal major depression, or, vice versa, the occurrence of spirals of positivity would increase.

7.3.13 Reduction of stressful effects of maternal engagement after the remission of depression, 32nd exploratory hypothesis

Since interpersonal stress approaches suggest that the parenting behavior of a

depressed mother is perceived as negative and stressful, a remission of depression allowed for the prediction of a reduction in stressful maternal behavior. Thus, infants of previously depressed mothers are predicted to cease reacting negatively in the case of heightened maternal engagement, i.e., infants of highly engaging mothers who have remitted from depression are expected to cease displaying higher rates of protest, non-mother focusing or lowered positive infant engagement. To differentiate between mothers with high versus low engagement a median split was introduced, which allowed for a comparison of high and low engaging mothers.

The data with respect to over-engaged mothers, however, revealed no overall differences between groups in association with the depression remission (refer to (figure 22, page 165, refer to the changes from in-episode to after remission). The rank analysis of variance found that changes were not specific for high engaging depressed mothers: Infants of these mothers did not behave differently from control infants ($p=0.16$ for changes in infant protest, $p=0.91$ for those that were non-mother focused and $p=0.85$ for infant engagement). Thus, remission-associated courses for highly engaging mothers with major depression were not different when compared with those courses of highly engaging control mothers. All in all, there were no indications of recovery effects in association with the remission of the maternal depression.

7.3.14 Recovery of interactive exhaustion after remission from maternal depression, 33rd exploratory hypothesis

Symptom based approaches of depression (e.g. loss of energy, motor retardation) allowed for a prediction of heightened “interactive exhaustion” in depressed mothers, i.e., a lowered ability to maintain interaction. Hence, the remission of maternal depression is expected to be associated with a recovery in exhaustive effects. Accordingly, interaction would be maintained over time at the same level as (or at least not differently to) healthy mothers.

However, the data (figure 23, page 166) reveal no clear changes following remission of maternal depression: there were no differences compared with healthy mothers ($p=0.30$ in time proportions and $p=0.53$ in frequencies, refer to slope p-values figure 23, page 166).

Even if the maternal affect behaviors are analyzed minute by minute (figure 24, page 167) mothers whose depression remitted showed no change in their maintenance

compared with controls. Even multivariate tests failed to show that remitted mothers had a different course in the emission of affects (refer to p-values of the Wei-Lachin Multivariate Rank Analysis). Thus, there were no signs of recovery from a presumed depression-related exhaustion in mothers who then remitted from major depression.

7.3.15 Recovery of reduced contingency in maternal responsiveness to infant behavior after remission from maternal depression, 34th exploratory hypothesis

Based on the symptom profile of maternal depression (e.g. loss of energy, motor retardation), an impairment in the maternal ability to respond contingently was predicted if the mother was in-episode. Accordingly, a subsequent recovery of normal values after remission from depression was predicted. The term “contingent response” was defined as a conditional response according to Alison-Liker estimators; these are conditional measures that control for baseline behavior (for details refer to page 168). The contingency data, however, showed no specific changes in mothers who remitted from depression. None of the exploratory p-values of the lag-sequential measures (z-value, i.e. the standardized conditional responses in table 34, page 171; refer to p-values last column) showed specific changes in remitting mothers compared with control dyads. Thus, there were no recovery effects in contingent responsiveness in association with maternal depression, i.e. no regain or recovery effects from in-episode to post-remission.

7.3.16 Recovery of deviant infant contingency after remission from maternal depression, 35th exploratory hypothesis

Based on the expectation of restricted maternal resources (e.g. loss of energy), reduced responsiveness and associated effects of infant under-stimulation, it was predicted that *infants* of depressed mothers would no longer respond to maternal behavior in a deviant manner after the remission (e.g. they would no longer be hypo- or hyper responsive compared to control infants). Technically, it is expected that infants of depressed mothers would no longer display any deviant conditional responses in the Alison-Liker estimators. However, the data with respect to this conditional measure (details on page 168) show no specific changes in infants whose mothers remitted from depression. None of the exploratory p-values of the lag-sequential measures (z-values of the conditional responses; refer to table 37, page 174, last column) showed differences in infants of remitting mothers compared with control infants. Thus, there were no specific recovery effects in infants of depressed

mothers, i.e. no regain or recovery effects in infant responsiveness from in-episode to post-depression remission.

7.3.17 Recovery in self-regulatory capacities in infants after remission from maternal depression, 36th exploratory hypothesis

Based upon the prediction that infants of depressed mothers have lower capacities of self-regulation (e.g. in the sense of regaining control after intense emotions or excitements or in the sense of refocusing on the mother), infants of depressed mothers were predicted to show a change towards a normalization in self-regulatory behaviors following remission of depression (compared to control infants). Technically, measures of latencies were derived until termination of crying or protesting and the time until the infant's focus is redirected toward the mother. These infant measures are predicted to recover with the remission of maternal depression.

In descriptive terms, however, both latencies (termination of crying and refocusing on the mother, see page 176, figure 25) were well comparable to control infants. Differences were so small as to be almost negligible. Neither for the depressed episode (for infant protest termination: $p=0.392$, mother-focusing: $p=0.380$) nor post-remission ($p=0.431$ and $p=0.124$, respectively) any differences emerged in reference to infants of depressed mothers having higher latencies.

Even if individual cumulative probabilities for the time-to-event estimate are used (Kaplan and Meier, 1958; an option of SPSS in the procedure "k-m" allows these values to be saved) and are followed from in-episode to post-remission, no differences emerged ($p=0.52$ for protest termination and $p=0.74$ for mother refocus, table 44).

Table 44: Cumulative probability estimate of non terminated behavior ("cumulative survival" of behaviors), t1 refers to the 1st observation, i.e. mothers with postpartum depression in episode, whereas t2 refers to the 2nd observation, i.e. mothers with postpartum depression after remission; (1) is the test for global difference (t1 and t2 were pooled) and depressed were compared to healthy dyads, based on the Wei-Lachin Multivariate Rank Analysis (Wei and Lachin, 1984; Lachin, 1992; two-sided), (2) shows differences between depressed and healthy dyads for t1 and t2: Mann-Whitney test, one-sided, (3) shows means of the differences between t1 and t2 and (4) is the test for trend (to test if both groups diverge) from t1 to t2, based on pre-post differences, m = mean, se = standard error. The estimates ("cumulative survival") were saved (for t1 and t2 as well) with the SPSS procedure "k-m" to test for trends from t1 to t2.

cumulative probability estimate of non terminated behavior	Healthy control mothers (n=35)				Major depression postpartum (n=24)				(1) global group difference	(2) group differ in t1, in t2	(3) changes t1 to t2		(3) diverging trends from t1 to t2 (5)
	t1		t2		t1		t2				healthy m	depr. m	
	m	se	m	se	m	se	m	se					
protest	0.47	0.07	0.46	0.09	0.42	0.09	0.50	0.11	p=0.44	none	-0.01	+0.08	p=0.52
non-mother focus	0.47	0.05	0.50	0.06	0.50	0.06	0.44	0.07	p=0.48	none	+0.02	-0.06	p=0.74

Thus, infants of depressed mothers neither differed from control infants in consolability (in terms of time until infant-protest terminates) nor in the time until mother-focusing re-occurred. Descriptively, changes from in-episode to post-remission were remarkably small (refer to column (3) in table 44) in both groups and infants of previously depressed mothers had no specific courses after the depression remitted. Thus there was no indication of a recovery in self-regulatory capacities in terms of regaining control after intense emotions or refocusing on the mother.

7.3.18 Recovery in the dyadic capacity to interact synchronously after remission from maternal depression, 37th exploratory hypothesis

Former hypotheses stated that depressed mothers would display reduced affect mirroring and thus a lower synchrony during interaction. Accordingly, a remission from maternal depression allowed for the prediction of a recovery in the ability to act synchronously (e.g. in terms of parallel occurring positive affects of both mother and infant). Synchrony was operationalized as shared variance of mother and infant, whose behavior codes were classified on a negative-positive scale (details in table 39 on page 178). However, the data show (see table 40, page 179, right column) that neither shared variance proportions ($p=0.39$) nor standardized correlations ($p=0.92$) indicated a specific recovery effect or trend in mothers whose depression remitted. In total, the measures for synchronous behaviors were small; i.e., there was less than 5% common variance between mother and infant, irrespective of diagnosis. Differences in comparison with control dyads were remarkably small. All in all, dyads with a depressed mother showed no recovery in indicators of synchrony. There were

no specific trends after remission. Thus, the remission of a major depression was not associated with measures of synchrony.

7.3.19 Recovery in affect-predictability after remission from maternal depression, 38th exploratory hypothesis

A final hypothesis tests for a prediction derived from regulation models (e.g. the model of Gergely, page 28, or Tronick et al., details on page 37 of this manuscript). These models predict that depressed mothers transmit their disturbance and irritability to the infant, resulting in poorly coordinated, mismatched and messy interactions. Poor coordination and messy behavior should be less predictable (at least according to the hypothesis), and this should be particularly the case when a mother is depressed. Therefore, a remission from depression may predict a recovery effect in the sense of an increase in predictability.

Technically, mother and infant codes were classified on a negativity-positivity scale (according to the scores given in table 39, page 178). Predictability was calculated based on the Pearson correlation of mother and child's time series scored on this positivity-negativity scale and lagged against each other. Accordingly, a high correlation allows for a prediction of subsequent behavior of the other interactant. All measures are given based on a z-standardization per dyad and in terms of shared variance per dyad. Both measures were then averaged per group for descriptive reasons.

The data show (refer to table 41 on page 181, last column) that neither standardized lag₁-correlations ($p=0.43$ for maternal, $p=0.59$ for infant predictability) nor shared variances ($p=0.80$ and $p=0.84$, respectively) indicate a specific recovery effect in the sense that a lack of affect-predictability returned to normal when the maternal depression remitted.

Thus, compared with controls, dyads with a depressed mother did not show either impairment in chosen indicators of affect-predictability or any recovery effects or specific trends associated with the depression remission. All in all the remission from major depression did *not* allow for improvements in predictability of affects.

7.4. Simple associations - correlations between infant and mother

Several descriptive analyses are added (table 45 and table 46), in particular with respect to the question of whether infant and maternal behavior of comparable types are related to each other (e.g. if infants and mothers are attentive towards each other, row 5 in table 45, or how both interactants engage positively, row 7). All in all, data show no relevant associations between mother and child. There were no differences between controls and dyads with a depressed mother. Even in dyads with a non-depressed mother, associations were only small. Correlations were well below $r=0.50$ (i.e. less than $0.50^2 \times 100\% = 25\%$ of variance was explainable).

Table 45: Pearson correlations for behavior classes of infant (1st column) and mother (3rd column). The explorative results refer to **proportions of behavior time**. All analysis made for descriptive purposes only. T1: depressed mothers in episode, t2 = after remission of depression, last column: between-group test for correlation coefficients (Bortz, 2005)

proportion of time			Major depression postpartum			Healthy control mothers			
t1: Pearson correlations			corr.	p-value	cases	corr.	p-value	cases	group correlation different?
1 infant negative	in corr. with	caregiver neg. engagement	r = -0.03	p=0.88	n = 24	r = 0.2	p=0.26	n = 35	p=0.2
2 infant protesting	in corr. with	caregiver hostile / intrusive	r = -0.03	p=0.90	n = 24	r = 0.32	p=0.06	n = 35	p=0.1
3 infant withdrawn	in corr. with	caregiver withdrawn	r = -0.04	p=0.84	n = 24	-	-	n = 35	
4 infant non mom-focussed	in corr. with	caregiver non-infant focused	r = 0.1	p=0.65	n = 24	r = 0.06	p=0.73	n = 35	p=0.56
5 infant attending to caregiver	in corr. with	caregiver social monitor and no vocs	r = -0.02	p=0.93	n = 24	r = -0.05	p=0.77	n = 35	p=0.54
6 infant social pos. engagement	in corr. with	caregiver social monitor, pos. vocs	r = 0.24	p=0.26	n = 24	r = -0.02	p=0.90	n = 35	p=0.83
7 infant positive or neutral	in corr. with	caregiver social pos. engagement	r = -0.38	p=0.07	n = 24	r = -0.28	p=0.10	n = 35	p=0.34
t2: Pearson correlations			corr.	p-value	cases	corr.	p-value	cases	group correlation different?
1 infant negative	in corr. with	caregiver neg. engagement	-	-	n = 16	-	-	n = 25	
2 infant protesting	in corr. with	caregiver hostile / intrusive	-	-	n = 16	-	-	n = 25	
3 infant withdrawn	in corr. with	caregiver withdrawn	-	-	n = 16	-	-	n = 25	
4 infant non-mom focussed	in corr. with	caregiver non-infant focused	r = -0.23	p=0.39	n = 16	r = -0.25	p=0.23	n = 25	p=0.52
5 infant attending to caregiver	in corr. with	caregiver social monitor and no vocs	r = 0.48	p=0.06	n = 16	r = 0.01	p=0.95	n = 25	p=0.93
6 infant social pos. engagement	in corr. with	caregiver social monitor, pos. vocs	r = -0.34	p=0.20	n = 16	r = 0.06	p=0.77	n = 25	p=0.12
7 infant positive or neutral	in corr. with	caregiver social pos. engagement	r = 0.48	p=0.06	n = 16	r = 0.02	p=0.94	n = 25	p=0.92

Note: not all comparisons for the between-group difference of correlation coefficients calculable due to zero variances in raw data (e.g. behavior did not occur)

Table 46: Pearson correlations for behavior classes of infant (1st column) and mother (3rd column), the results refer to **frequencies per minute**, all analysis for descriptive purposes only, t1: depressed mothers in episode, t2 = after remission of depression, last column: between-group test for correlation coefficients (Bortz, 2005)

frequencies per minute			Major depression postpartum			Healthy control mothers			group correlation different?
t1: Pearson correlations			corr.	p-value	cases	corr.	p-value	cases	
1 infant negative	in corr. with	caregiver neg. engagement	r = 0.07	p=0.73	n = 24	r = 0.15	p=0.39	n = 35	p=0.39
2 infant protesting	in corr. with	caregiver hostile / intrusive	r = 0.09	p=0.69	n = 24	r = 0.17	p=0.32	n = 35	p=0.39
3 infant withdrawn	in corr. with	caregiver withdrawn	r = -0.04	p=0.84	n = 24	-	-	n = 35	
4 infant non mom-focussed	in corr. with	caregiver non-infant focused	r = 0.33	p=0.11	n = 24	r = -0.09	p=0.61	n = 35	p=0.94
5 infant attending to caregiver	in corr. with	caregiver social monitor and no vocs	r = 0.13	p=0.54	n = 24	r = 0.09	p=0.62	n = 35	p=0.56
6 infant social pos. engagement	in corr. with	caregiver social monitor, pos. vocs	r = 0.58	p<0.01	n = 24	r = 0.33	p=0.05	n = 35	p=0.87
7 infant positive or neutral	in corr. with	caregiver social pos. engagement	r = -0.34	p=0.11	n = 24	r = -0.01	p=0.94	n = 35	p=0.11

t2: Pearson correlations			corr.	p-value	cases	corr.	p-value	cases	group correlation different?
1 infant negative	in corr. with	caregiver neg. engagement	-	-	n = 16	-	-	n = 25	
2 infant protesting	in corr. with	caregiver hostile / intrusive	-	-	n = 16	-	-	n = 25	
3 infant withdrawn	in corr. with	caregiver withdrawn	-	-	n = 16	-	-	n = 25	
4 infant non-mom focussed	in corr. with	caregiver non-infant focused	r = 0.31	p=0.24	n = 16	r = -0.16	p=0.46	n = 25	p=0.92
5 infant attending to caregiver	in corr. with	caregiver social monitor and no vocs	r = 0.38	p=0.15	n = 16	r = 0.34	p=0.10	n = 25	p=0.55
6 infant social pos. engagement	in corr. with	caregiver social monitor, pos. vocs	r = 0.24	p=0.37	n = 16	r = 0.26	p=0.20	n = 25	p=0.48
7 infant positive or neutral	in corr. with	caregiver social pos. engagement	r = 0.51	p=0.04	n = 16	r = -0.17	p=0.41	n = 25	p=0.98

Note: not all comparisons for the between-group difference of correlation coefficients calculable due to zero variances in raw data (e.g. behavior did not occur)

Moreover, there were no associations between maternal positive affects and the number of depression criteria (table 47).

Table 47: Maternal positive affects in correlation with the depression criteria count

Spearman's Rangkorrelation			coefficient	p-value	cases	evaluation
mother (frequencies per minute)	with	Number of depression criteria fulfilled	r = -0.01	p=0.932	n = 55	no correlation
maternal engagement (proportion)	with	Number of depression criteria fulfilled	r = 0.04	p=0.796	n = 55	no correlation

7.5. Adjustment for Covariates: Applying methods for bias correction**7.6. Derivation of composite measures to minimize error variance: Factorization based on a principal component analysis with VARIMAX rotation and between-groups tests based on factor values**

Based on the idea that an aggregation of variables might minimize error variance, or, possibly control for biases and thus better discriminate depressed from non-depressed mothers, a composite measure was introduced: Raw-measures of the whole sample were factorized based on a principal component analysis (PCA, Norusis, 2008). A Scree-plot showed that 4 factors (i.e. the “composite measures”) received sufficient Eigenvalues (i.e. above 2) and were VARIMAX-rotated. The PCA with orthogonal, i.e. zero-correlating, factors was chosen to receive only few factors, and the VARIMAX-rotation to maximize loadings of variables on single factors to simplify factor interpretation. The 4 factors explained 53% of the total variance of the observational measures, either in time proportions or in counts per minute (last column in table 48). In total an infant engagement factor (factor 1, 2nd column in table 48), a caregiver disengagement factor (factor 2) a caregiver engagement factor (factor 3) and an infant disengagement factor (factor 4) emerged.

Table 48: Factorization of observational measures at t1 (dyads with a mother in-episode of major depression and control dyads) based on a PCA with VARIMAX rotation. Note that only sufficient loadings of $|a| \geq .40$ are displayed.

principal component analysis with varimax rotation and a 4-factor solution (53 % of total variance explained)	factor 1 (14%)	factor 2 (14%)	factor 3 (13%)	factor 4 (12%)	type of measure
infant protesting	-0.743				prop. time
infant protesting	-0.628				freq. per min
infant social pos. engagement	0.608				freq. per min
infant attending to caregiver	0.598				freq. per min
caregiver social monitor, pos. vocs	0.583			0.458	freq. per min
infant non mom-focused	0.491				freq. per min
infant social pos. engagement	0.484				prop. time
caregiver withdrawn		0.836			prop. time
caregiver withdrawn		0.834			freq. per min
caregiver hostile / intrusive		0.671			freq. per min
caregiver non-infant focused		0.654			prop. time
caregiver hostile / intrusive		0.639			prop. time
caregiver non-infant focused		0.533			freq. per min
caregiver social monitor and no vocs			-0.675		freq. per min
caregiver social monitor and no vocs			-0.605	-0.510	prop. time
caregiver exagg. positive			0.590		freq. per min
caregiver exagg. positive			0.590		prop. time
infant attending to caregiver			0.584		prop. time
infant non mom-focused			-0.568		prop. time
caregiver social pos. engagement			0.458		prop. time
infant withdrawn				-0.797	freq. per min
infant withdrawn				-0.778	prop. time
caregiver social pos. engagement	0.425		0.485	0.518	freq. per min
caregiver social monitor, pos. vocs				0.439	prop. time

The resulting z-standardized factor-values were then saved (see SPSS procedure “factor”, Norusis, 2008) and used to test for differences between depressed dyads and controls in these new composite measures (table 49). However, the comparison based on factor values of the factor solution in table 48 showed that both groups did not differ (see p-values in last column) for any of these factors.

Table 49: Between-group comparison of factor values (z-values) derived with a principal component analysis (Table 48), test for the 2-group comparison: Mann-Whitney-U-test, two-sided (Lehmann, 1998), m = mean factor value, sd = standard deviation, md = median, n = number of cases

	Healthy control mothers n = 35						Major depression postpartum n = 24						descriptive test
	m	sd	min	md	max	n	m	sd	min	md	max	n	
factor 1	-0.04	+ 1.10	(-2.7	0.1	2.0)	35	0.06	+ 0.86	(-2.9	0.0	1.5)	24	p=0.53
factor 2	-0.08	+ 0.70	(-0.7	-0.2	2.7)	35	0.11	+ 1.33	(-0.6	-0.2	6.3)	24	p=0.21
factor 3	0.00	+ 0.84	(-1.6	-0.2	1.8)	35	0.00	+ 1.22	(-1.4	-0.2	4.5)	24	p=0.55
factor 4	0.06	+ 0.88	(-4.2	0.1	2.0)	35	-0.08	+ 1.16	(-4.6	0.0	1.2)	24	p=0.73

Thus, the idea to control error variances by an aggregation of different behaviors based on a factorization did not allow for an enhanced differentiation of dyads with and without a mother with major depression.

7.6.1 Bias correction methods based on inequality assumptions of groups: Matching methods based on propensity scores

It is well known that depression has a high potential for attracting a large range of co-effects, e.g., the disorder occurs in associations with lower social status, income or education, in association with child care stress, life stressors, or, lack of partner support (Beck, 1996; Goodman and Gotlib, 1999; Downey and Coyne, 1990). Accordingly, there is a high need to include these effects as potential confounders - as far as they are available - and to adjust for them or at least minimize their influence. Although the groups were shown not to be different with respect to various confounders (table 14, page 115) an effect on dependent measures can not be excluded (e.g. infant age on frequencies of affect expressions).

One method of confounder minimization is to parallelize groups using a case-control matching, e.g. based on maternal education, infant gender or infant age. However, if a large number of confounders is available, accordingly, there is a large range of matching scenarios with the possibility of differing results depending on the authors' choice. To avoid this, the propensity-score-method allows for an extension of the classical matched-pair design without any restriction on the number of covariates (Rosenbaum and Rubin, 1985, Joffe and Rosenbaum, 1999; Rubin and Thomas,

1996; Braitman and Rosenbaum, 2002).

Based on a logistic regression (Agresti, 1990), covariates such as maternal depression history, level of education, maternal age, existence of a partner, maternal comorbid diagnosis, infant gender, infant age and number of children are entered into the regression model. Now, the probability of how these confounders were associated with the allocation to the diagnosis group is calculated. In case of a logistic regression this is given as probability, the 'propensity score'. One after the other, a mother with major depression is matched to a control mother based on a comparable propensity score which was derived from the covariates (details in Rudner and Peyton, 2006). Those resulting pairs are submitted to a matched-pairs test, e.g. the Wilcoxon-signed-rank test. Rubin (1997) has shown that a matching based on a propensity score results in equivalent means and standard deviations of covariates. Since the covariates are now balanced by definition any resulting differences should be referable to the remaining variation, namely the contrast between depressed and control mothers.

22 dyads could be matched based on the propensity score based on infant gender and age, maternal depression history, comorbidity (anxiety), cohabitation with a partner, number of children, and maternal age.

The results are shown in table 50. However, even after the balancing for multiple covariates based on the propensity score method there were no differences between dyads with and without a depressed mother, or any trends.

Table 50: Proportion of time and frequencies per minute after propensity score matching (matching depressed to control dyads for inequality in multiple demographic criteria, e.g. Rubin and Thomas, 1996): Dyads are matched for maternal depression history, level of education, maternal age, existence of a partner, maternal comorbid diagnosis, infant gender, infant age and number of children, t1 = mothers with postpartum depression in episode, t2 = mothers with postpartum depression after remission; (1) and (2) differences between depressed and healthy dyads, statistical test: Wilcoxon-sign-rank test (a test for dependent observations since matched pairs are compared), two-sided, (3) descriptive statistics for changes between t1 and t2 and (4) test for a trend from t1 to t2, m = mean, se = standard error, n = number of cases

	Healthy control mothers (n=22)						Major depression postpartum (n=22)						(1) groups differ in t1	(2) groups differ in t2	(3) changes t1 to t2						(4) diverging trends from t1 to t2
	t1			t2			t1			t2					healthy			depr.			
proportion of time	m	se	n	m	se	n	m	se	n	m	se	n			m	se	n	m	se	n	
caregiver neg. engagement	0.00	0.00	22	0.00	-	16	0.02	0.02	22	0.00	-	15	p=0.65	-	0.00	0.00	16	0.00	-	15	-
caregiver hostile / intrusive	0.00	0.00	22	0.00	-	16	0.00	0.00	22	0.00	-	15	p=0.65	-	0.00	0.00	16	0.00	-	15	-
caregiver withdrawn	0.00	-	22	0.00	-	16	0.02	0.02	22	0.00	-	15	-	-	0.00	-	16	0.00	-	15	-
caregiver non-infant focused	0.00	0.00	22	0.00	0.00	16	0.00	0.00	22	0.00	0.00	15	p=1.00	p=0.71	0.00	0.00	16	0.00	0.00	15	p=1.00
caregiver social monitor and no vocs	0.26	0.04	22	0.30	0.08	16	0.38	0.06	22	0.25	0.05	15	p=0.22	p=0.72	0.05	0.09	16	-0.10	0.07	15	p=0.33
caregiver social monitor, pos. vocs	0.61	0.03	22	0.62	0.07	16	0.51	0.05	22	0.65	0.04	15	p=0.23	p=0.53	0.00	0.07	16	0.10	0.06	15	p=0.18
caregiver social pos. engagement	0.14	0.04	22	0.09	0.02	16	0.09	0.02	22	0.14	0.04	15	p=0.37	p=0.33	-0.03	0.05	16	0.06	0.04	15	p=0.06
caregiver exagg. positive	0.00	-	22	0.00	0.00	16	0.00	0.00	22	0.00	-	15	-	p=0.18	0.00	0.00	16	0.00	0.00	15	p=0.18
infant negative	0.06	0.02	22	0.09	0.04	16	0.08	0.03	22	0.07	0.03	15	p=0.76	p=0.21	0.02	0.03	16	0.00	0.04	15	p=0.03
infant protesting	0.06	0.02	22	0.09	0.04	16	0.08	0.03	22	0.07	0.03	15	p=0.76	p=0.21	0.02	0.03	16	0.01	0.04	15	p=0.03
infant withdrawn	0.00	-	22	0.00	-	16	0.00	0.00	22	0.00	-	15	-	-	0.00	-	16	-0.01	0.01	15	-
infant non-mom focussed	0.46	0.06	22	0.54	0.06	16	0.52	0.06	22	0.63	0.05	15	p=0.47	p=0.10	0.11	0.08	16	0.16	0.09	15	p=0.48
infant attending to caregiver	0.38	0.05	22	0.22	0.03	16	0.31	0.05	22	0.22	0.03	15	p=0.19	p=0.66	-0.19	0.06	16	-0.14	0.07	15	p=1.00
infant social pos. engagement	0.11	0.02	22	0.12	0.04	16	0.07	0.02	22	0.10	0.03	15	p=0.26	p=0.62	0.01	0.03	16	0.03	0.04	15	p=1.20
infant positive or neutral	0.08	0.02	22	0.06	0.03	16	0.07	0.02	22	0.04	0.01	15	p=0.78	p=0.44	0.01	0.03	16	-0.02	0.02	15	p=0.68
frequencies per minute																					
caregiver neg. engagement	0.02	0.02	22	0.00	-	16	0.08	0.08	22	0.00	-	15	p=0.65	-	-0.03	0.03	16	0.00	-	15	-
caregiver hostile / intrusive	0.02	0.02	22	0.00	-	16	0.01	0.01	22	0.00	-	15	p=0.65	-	-0.03	0.03	16	0.00	-	15	-
caregiver withdrawn	0.00	-	22	0.00	-	16	0.06	0.06	22	0.00	-	15	-	-	0.00	-	16	0.00	-	15	-
caregiver non-infant focused	0.13	0.09	22	0.00	0.00	16	0.08	0.04	22	0.00	0.00	15	p=0.67	p=0.71	-0.16	0.12	16	-0.09	0.04	15	p=1.00
caregiver social monitor and no vocs	2.83	0.36	22	0.30	0.08	16	3.03	0.41	22	0.25	0.05	15	p=0.76	p=0.72	-2.58	0.37	16	-2.47	0.51	15	p=0.53
caregiver social monitor, pos. vocs	4.09	0.29	22	0.62	0.07	16	4.04	0.45	22	0.65	0.04	15	p=0.86	p=0.53	-3.59	0.36	16	-3.32	0.56	15	p=0.66
caregiver social pos. engagement	2.02	0.34	22	0.09	0.02	16	1.98	0.36	22	0.14	0.04	15	p=0.83	p=0.33	-1.91	0.44	16	-1.98	0.48	15	p=0.93
caregiver exagg. pos. engagement	0.00	-	22	0.00	0.00	16	0.01	0.01	22	0.00	-	15	-	p=0.18	0.00	0.00	16	-0.02	0.02	15	p=0.18
infant negative	0.39	0.16	22	0.09	0.04	16	0.52	0.16	22	0.07	0.03	15	p=0.62	p=0.21	-0.32	0.18	16	-0.36	0.18	15	p=0.33
infant protest	0.39	0.16	22	0.09	0.04	16	0.47	0.16	22	0.07	0.03	15	p=0.76	p=0.21	-0.32	0.18	16	-0.29	0.17	15	p=0.88
infant withdrawn	0.00	-	22	0.00	-	16	0.05	0.05	22	0.00	-	15	-	-	0.00	-	16	-0.07	0.07	15	-
infant non mom-focussed	5.89	0.86	22	0.54	0.06	16	6.13	0.64	22	0.63	0.05	15	p=0.91	p=0.10	-5.31	1.11	16	-5.08	0.83	15	p=0.79
infants attention to caregiver	5.97	0.79	22	0.22	0.03	16	4.93	0.41	22	0.22	0.03	15	p=0.41	p=0.66	-6.09	1.01	16	-4.49	0.43	15	p=0.37
infant pos. engagement	2.11	0.55	22	0.12	0.04	16	1.42	0.30	22	0.10	0.03	15	p=0.58	p=0.62	-2.21	0.69	16	-1.42	0.36	15	p=0.53

7.6.2 Adjustment for confounders based on analyses of covariance

In addition to the confounder control methods of the two previous chapters major dependent variables of this study were tested after potential effects of confounding variables had been controlled for (e.g. the age of infants or maternal education level).

For this purpose, analyses of covariance (ANOCVA) were applied (Winer, Brown and Michels, 1991; Winer, 1971; SPSS procedure “univariate”, Norusis, 2008), which basically test for between-group differences over regression residuals i.e. after covariates have been partialled out. ANCOVAs, however, are based on a large range of assumptions: assumptions such as homogeneity of variance were tested with the Levene-Test (Bortz, 2005). Normality of residuals was checked graphically (i.e., they were plotted against normal values, “residual normal plots”, with the expectation that they would scatter around a 45° line, and “residual against predicted plot” with the expectation that they would scatter around a horizontal line). To test the homogeneity-of-slope assumption the interaction-term of covariate and independent was included in the model (if $p \leq 0.05$ the term was left in the model). The overall idea of applying analyses of covariance was that the inclusion of covariates may reduce the total amount of error variance and thus increase statistical power to detect impairments in dyads with a depressed mother.

Preliminary exploratory checks for suspected correlations of covariates with target parameters (table 51, 1st column) showed that infant age showed a few associations to mother-infant interaction. Existing correlations were small and less than 10% of explained variance, e.g., for affect mirroring ($r=0.29$), as well as for maternal engagement ($r=0.36$) and infant engagement ($r=0.33$). Correlations were moderate (less than 25% of common variance) for infant engagement ($r=-0.30$) and for the caregiver ($r=0.46$ or 0.44). The correlations ranged between 0 and 0.47, i.e. less than $0.47^2 \leq 25\%$ of variance were explainable. In addition, the maternal education showed a few small associations ($r \leq 0.35$). Most importantly, with few exceptions the covariate inclusion and elimination of respective linear effects did not produce notable between-diagnosis-groups effects (the differences in mean values were very small). All in all the inclusion of covariates did not help to increase presumably masked effects. The few emerging differences showed that depression-related differences were remarkably small in terms of estimated marginal means (averages of target parameters after confounders were eliminated).

Table 51: Results of the analysis of covariance to test for depression related differences after covariates have been controlled for; correlations (r) of covariates (see table header) with dependent variables (1st column) are given, r = Pearson correlation, p_r = exploratory p-value based on the t distribution with n-2 degrees of freedom, sl = homogeneity of slope assumption violated (interaction term of covariate with independent variable) ("+" yes, "-" no), co = effect of covariate, p_{cov} = groups difference (SPSS procedure univariate, with variable in 1st table row used as covariate, dummy coded if necessary), e = estimated marginal means of controls (e₀) and depressed mothers (e₁), all tests are strictly exploratory (i.e. without adjustment for 1st type error)

	covariate: infant age							maternal qualific. for university (y/n)							maternal age (years)							child number						
frequencies per minute	r	p _r	sl	co	p _{cov}	e0	e1	r	p _r	sl	co	p _{cov}	e0	e1	r	p _r	sl	co	p _{cov}	e0	e1	r	p _r	sl	co	p _{cov}	e0	e1
positive affect mirroring	0.29	0.03	-	+	0.43	0.86	1.08	0.05	0.72	-	-	0.66	0.83	1.15	-0.17	0.21	-	-	0.40	0.81	1.09	-0.05	0.71	-	-	0.88	0.83	1.12
maternal engagement	0.36	0.01	+	-	0.62	6.02	5.82	0.26	0.05	+	-	0.27	5.90	6.23	0.11	0.41	-	-	0.63	5.73	5.98	-0.17	0.21	-	-	0.49	5.93	5.93
infant engagement	0.33	0.01	+	-	0.48	7.41	6.68	-0.24	0.07	-	-	0.21	7.31	6.77	-0.29	0.03	+	-	0.06	7.41	6.67	-0.02	0.89	-	-	0.99	7.18	6.78
infant negative	0.03	0.82	-	-	0.48	0.53	0.52	-0.21	0.11	-	-	0.31	0.52	0.46	-0.10	0.48	-	-	0.79	0.54	0.51	0.21	0.10	-	-	0.40	0.51	0.54
infant protest	0.06	0.65	-	-	0.71	0.51	0.47	-0.17	0.21	-	-	0.41	0.50	0.43	-0.11	0.43	-	-	0.74	0.52	0.46	0.21	0.11	-	-	0.48	0.49	0.50
infant withdrawn	-0.15	0.26	-	-	0.09	0.01	0.05	-0.22	0.09	-	-	0.42	0.02	0.04	0.07	0.63	-	-	0.74	0.01	0.05	0.02	0.86	-	-	0.54	0.02	0.05
infant non mom-focused	0.20	0.13	-	-	0.23	5.86	6.16	-0.35	0.01	+	-	0.83	5.84	6.05	-0.22	0.09	-	+	0.01	5.89	6.26	-0.02	0.87	-	-	0.20	5.77	6.30
infants attention to caregiver	0.18	0.17	-	-	0.56	5.53	5.05	-0.25	0.06	-	-	0.20	5.51	5.04	-0.26	0.05	-	+	0.03	5.57	4.98	-0.07	0.62	-	-	0.88	5.41	5.03
infant pos. engagement	0.46	0.00	+	-	0.43	1.75	1.33	-0.03	0.80	-	-	0.29	1.62	1.47	-0.18	0.18	-	-	0.49	1.66	1.39	-0.05	0.71	-	-	0.60	1.61	1.44
caregiver neg. engagement	-0.02	0.87	-	-	0.67	0.02	0.07	-0.15	0.25	-	-	0.16	0.02	0.06	0.02	0.91	-	-	0.77	0.02	0.08	0.03	0.79	-	-	0.57	0.02	0.08
caregiver hostile / intrusive	-0.09	0.50	-	-	0.66	0.01	0.01	0.01	0.94	-	-	0.42	0.01	0.01	-0.02	0.86	-	-	0.61	0.01	0.01	-0.05	0.73	-	-	0.35	0.01	0.01
caregiver withdrawn	0.01	0.97	-	-	0.50	0.01	0.06	-0.19	0.15	-	-	0.16	0.01	0.05	0.03	0.84	-	-	0.86	0.01	0.07	0.06	0.66	-	-	0.73	0.01	0.06
caregiver non-infant focused	-0.04	0.78	-	-	0.88	0.09	0.08	0.03	0.84	-	-	0.68	0.09	0.07	0.21	0.12	-	-	0.20	0.08	0.08	0.09	0.51	-	-	0.95	0.09	0.08
caregiver social monitor and no vocs	0.47	0.00	+	-	0.13	3.35	2.72	0.10	0.47	-	+	0.03	3.30	3.04	0.00	0.98	-	-	0.09	3.28	3.05	-0.11	0.42	-	-	0.36	3.29	2.93
caregiver social monitor, pos. vocs	0.43	0.00	+	-	0.13	4.31	3.92	0.31	0.02	+	+	0.05	4.25	4.26	0.05	0.72	-	-	0.15	4.19	4.12	-0.22	0.10	-	-	0.95	4.28	4.05
caregiver social pos. engagement	0.13	0.33	-	-	0.54	1.90	2.06	0.17	0.20	-	-	0.96	1.84	2.14	0.19	0.16	-	-	0.39	1.73	2.03	-0.04	0.77	-	-	0.17	1.84	2.03
caregiver exagg. pos. engagement	-0.06	0.65	-	-	0.23	0.00	0.01	0.10	0.43	-	-	1.00	0.00	0.01	-0.21	0.12	-	-	0.09	0.00	0.01	0.07	0.58	-	-	0.85	0.00	0.01
frequencies of grouped sequences	r	p _r	sl	co	p _{cov}	e0	e1	r	p _r	sl	co	p _{cov}	e0	e1	r	p _r	sl	co	p _{cov}	e0	e1	r	p _r	sl	co	p _{cov}	e0	e1
positive affect mirroring	-0.10	0.46	-	-	0.68	0.32	0.25	0.03	0.82	-	-	0.16	0.32	0.26	-0.04	0.76	-	-	0.38	0.32	0.24	0.08	0.56	-	-	0.25	0.31	0.24
maternal engagement	-0.14	0.31	-	-	0.20	0.69	0.61	0.11	0.41	-	-	0.83	0.69	0.61	0.01	0.92	-	-	0.93	0.70	0.60	0.00	0.97	-	-	0.90	0.69	0.61
infant engagement	-0.30	0.02	-	+	0.90	0.50	0.46	-0.08	0.53	-	-	0.31	0.51	0.44	-0.06	0.65	-	-	0.16	0.51	0.43	0.26	0.05	-	-	0.20	0.50	0.44
infant negative	-0.19	0.15	-	-	0.85	0.09	0.08	-0.13	0.32	-	-	0.53	0.10	0.07	0.01	0.93	-	-	0.42	0.10	0.07	0.29	0.03	-	+	0.57	0.09	0.08
infant protesting	-0.18	0.17	-	-	0.78	0.09	0.07	-0.10	0.45	-	-	0.48	0.09	0.06	0.00	0.98	-	-	0.42	0.09	0.07	0.29	0.02	-	+	0.67	0.09	0.08
infant withdrawn	-0.08	0.57	-	-	0.48	0.00	0.00	-0.22	0.09	-	-	0.57	0.00	0.00	0.07	0.63	-	-	0.91	0.00	0.00	-0.05	0.72	-	-	0.35	0.00	0.00
infant non-mom focused	0.37	0.00	-	+	0.84	0.47	0.51	0.08	0.53	-	-	0.69	0.46	0.53	0.10	0.44	-	-	0.25	0.45	0.54	-0.17	0.19	-	-	0.35	0.47	0.53
infant attending to caregiver	-0.35	0.01	-	+	0.67	0.34	0.33	-0.01	0.97	-	-	0.18	0.35	0.31	-0.04	0.78	-	-	0.45	0.35	0.30	0.04	0.79	+	-	0.14	0.34	0.30
infant social pos. engagement	0.34	0.01	-	+	0.95	0.08	0.06	-0.04	0.74	-	-	0.43	0.08	0.07	-0.10	0.45	-	-	0.55	0.08	0.06	0.11	0.40	-	-	0.68	0.08	0.07
caregiver neg. engagement	0.00	1.00	-	-	0.51	0.00	0.02	-0.17	0.19	-	-	0.13	0.00	0.01	0.02	0.87	-	-	0.84	0.00	0.02	0.06	0.66	-	-	0.74	0.00	0.02
caregiver hostile / intrusive	-0.09	0.48	-	-	0.62	0.00	0.00	0.02	0.88	-	-	0.46	0.00	0.00	-0.03	0.85	-	-	0.61	0.00	0.00	-0.05	0.70	-	-	0.34	0.00	0.00
caregiver withdrawn	0.00	0.97	-	-	0.49	0.00	0.02	-0.18	0.17	-	-	0.13	0.00	0.01	0.02	0.86	-	-	0.86	0.00	0.02	0.06	0.63	-	-	0.77	0.00	0.02
caregiver non-infant focused	-0.11	0.39	-	-	0.70	0.00	0.00	0.00	1.00	-	-	0.79	0.00	0.00	0.19	0.16	-	-	0.23	0.00	0.00	0.11	0.42	-	-	0.96	0.00	0.00
caregiver social monitor and no vocs	0.18	0.16	-	-	0.36	0.32	0.35	-0.06	0.66	-	-	0.42	0.31	0.36	0.00	0.97	-	-	0.71	0.31	0.37	0.02	0.91	-	-	0.75	0.31	0.36
caregiver social monitor, pos. vocs	-0.19	0.14	-	-	0.20	0.59	0.53	0.16	0.23	-	-	0.77	0.60	0.53	-0.12	0.38	-	-	0.24	0.61	0.52	-0.16	0.23	-	-	0.29	0.61	0.53
caregiver social pos. engagement	0.07	0.58	-	-	0.83	0.11	0.09	-0.04	0.78	-	-	0.93	0.11	0.09	0.23	0.08	+	-	0.03	0.096	0.086	0.24	0.06	+	-	0.03	0.100	0.092
caregiver exagg. positive	-0.06	0.65	-	-	0.23	0.00	0.00	0.10	0.43	-	-	1.00	0.00	0.00	-0.21	0.12	-	-	0.09	0.00	0.00	0.07	0.58	-	-	0.85	0.00	0.00

r = simple Pearson correlations of covariate with dependent variable (1st column)
sl = slope, homogeneity of slope assumption violated (yes if "+")

p = exploratory p-value for correlation coefficient (1st p-value in list)
p = depressed group differs based on an ANCOVA model

Table 51 (continued) results of the ANCOVA models

	covariate: male infant gender (y/n)							partner							depression history						
frequencies per minute	r	p _r	sl	co	p _{cov}	e0	e1	r	p _r	sl	co	p _{cov}	e0	e1	r	p _r	sl	co	p _{cov}	e0	e1
positive affect mirroring	0.08	0.54	-	-	0.03	0.80	1.09	0.13	0.31	+	-	nd	0.83		0.16	0.22	+	-	0.60	1.04	
maternal engagement	0.01	0.94	-	-	0.27	5.88	5.96	0.04	0.74	-	+	nd	5.92		-0.01	0.92	-	+	0.78	6.11	
infant engagement	0.21	0.11	-	-	0.33	7.07	6.79	-0.03	0.81	-	+	nd	7.17		-0.06	0.67	-	+	0.88	6.91	
infant negative	-0.16	0.21	-	-	0.84	0.53	0.50	0.09	0.49	-	+	nd	0.52		0.03	0.84	-	+	0.84	0.48	
infant protest	-0.12	0.35	-	-	0.72	0.51	0.46	0.09	0.51	-	+	nd	0.51		-0.01	0.92	-	+	0.89	0.47	
infant withdrawn	-0.20	0.13	-	-	0.41	0.02	0.04	0.02	0.86	-	+	nd	0.01		0.20	0.12	-	+	0.77	0.02	
infant non mom-focused	0.21	0.11	-	-	0.60	5.65	6.27	-0.09	0.52	-	+	nd	5.68		0.06	0.63	-	+	0.79	6.09	
infants attention to caregiver	0.23	0.08	-	-	0.39	5.32	5.08	-0.10	0.46	-	+	nd	5.38		-0.03	0.85	-	+	0.70	5.05	
infant pos. engagement	0.18	0.18	-	-	0.43	1.54	1.43	0.07	0.60	-	+	nd	1.61		-0.10	0.46	-	+	0.85	1.61	
caregiver neg. engagement	0.12	0.36	-	-	0.85	0.02	0.08	0.02	0.86	-	+	nd	0.02		-0.09	0.50	-	+	0.11	0.11	
caregiver hostile / intrusive	0.16	0.23	-	-	1.00	0.01	0.01	0.02	0.86	-	+	nd	0.01		-0.09	0.49	-	+	0.71	0.02	
caregiver withdrawn	0.09	0.49	-	-	0.82	0.01	0.07	0.02	0.88	-	+	nd	0.01		-0.08	0.57	-	+	0.08	0.09	
caregiver non-infant focused	0.16	0.21	-	-	0.96	0.09	0.08	0.04	0.76	-	+	nd	0.09		-0.09	0.50	-	+	0.81	0.10	
caregiver social monitor and no vocs	-0.12	0.38	-	-	0.68	3.28	2.90	0.15	0.25	-	+	nd	3.29		-0.22	0.10	-	+	0.67	3.28	
caregiver social monitor, pos. vocs	-0.04	0.77	-	-	0.53	4.24	4.06	0.09	0.49	-	+	nd	4.26		-0.09	0.50	-	+	0.92	4.23	
caregiver social pos. engagement	0.05	0.72	-	-	0.23	1.84	2.06	-0.01	0.95	-	+	nd	1.86		0.08	0.55	-	+	0.84	2.01	
caregiver exagg. pos. engagement	-0.15	0.26	-	-	0.11	0.00	0.01	0.02	0.90	-	+	nd	0.00		0.26	0.05	-	+	1.00	0.00	
frequencies of grouped sequences	r	p _r	sl	co	p _{cov}	e0	e1	r	p _r	sl	co	p _{cov}	e0	e1	r	p _r	sl	co	p _{cov}	e0	e1
positive affect mirroring	-0.01	0.96	-	-	0.64	0.32	0.25	-0.02	0.86	+	-	nd	0.32		-0.10	0.43	+	-	0.44	0.26	
maternal engagement	0.08	0.54	-	-	0.65	0.69	0.61	-0.15	0.27	+	-	nd	0.69		0.01	0.92	+	-	0.10	0.58	
infant engagement	-0.10	0.46	-	-	0.28	0.51	0.44	0.08	0.55	+	-	nd	0.51		-0.15	0.26	+	-	0.62	0.46	
infant negative	-0.23	0.09	-	-	0.25	0.10	0.07	0.08	0.57	+	-	nd	0.10		-0.01	0.97	+	-	0.47	0.07	
infant protesting	-0.20	0.14	-	-	0.29	0.10	0.07	0.07	0.59	+	-	nd	0.09		-0.01	0.91	+	-	0.53	0.06	
infant withdrawn	-0.20	0.13	-	-	0.64	0.00	0.00	0.02	0.86	+	-	nd	0.00		0.06	0.63	+	-	0.57	0.00	
infant non-mom focused	0.17	0.20	-	-	0.26	0.45	0.53	-0.11	0.40	+	-	nd	0.46		0.10	0.43	+	-	0.52	0.52	
infant attending to caregiver	0.03	0.84	-	-	0.56	0.35	0.31	0.01	0.92	+	-	nd	0.35		-0.09	0.48	+	-	0.75	0.32	
infant social pos. engagement	0.01	0.93	-	-	0.71	0.08	0.07	0.07	0.59	+	-	nd	0.08		-0.14	0.28	+	-	0.66	0.08	
caregiver neg. engagement	0.11	0.42	-	-	0.89	0.00	0.02	0.02	0.88	+	-	nd	0.00		-0.08	0.57	+	-	0.07	0.03	
caregiver hostile / intrusive	0.16	0.23	-	-	1.00	0.00	0.00	0.02	0.86	+	-	nd	0.00		-0.09	0.50	+	-	0.79	0.00	
caregiver withdrawn	0.10	0.45	-	-	0.89	0.00	0.02	0.02	0.89	+	-	nd	0.00		-0.07	0.58	+	-	0.07	0.03	
caregiver non-infant focused	0.13	0.31	-	-	0.48	0.00	0.00	0.04	0.76	+	-	nd	0.00		-0.15	0.24	+	-	0.64	0.00	
caregiver social monitor and no vocs	-0.09	0.52	-	-	0.99	0.31	0.36	0.15	0.27	+	-	nd	0.31		-0.08	0.57	+	-	0.18	0.39	
caregiver social monitor, pos. vocs	0.07	0.60	-	-	0.82	0.60	0.53	-0.08	0.57	+	-	nd	0.60		0.03	0.81	+	-	0.08	0.49	
caregiver social pos. engagement	0.05	0.72	-	-	0.73	0.11	0.10	-0.14	0.30	+	-	nd	0.10		-0.01	0.92	+	-	0.78	0.09	
caregiver exagg. positive	-0.15	0.26	-	-	0.11	0.00	0.00	0.02	0.90	+	-	nd	0.00		0.26	0.05	+	-	1.00	0.00	

r = correlations of covariate with dependent variable (1st column)

sl = slope, homogeneity of slope assumption violated (yes if "+")

Note: Some marginal means could not be estimated.

p = exploratory p-value for correlation coefficient

p = depressed group differs based on an ANCOVA model

7.6.3 Effects of patient characteristics: medication, comorbidity, cohabitation with a partner, and the single criteria of the depression profile

Several potential confounders were tested within the group of depressed mothers only, such as the intake of antidepressant medication (13 by 24 mothers with major depression). Other medications, however, such as neuroleptic medication (3 mothers only), benzodiazepines (2 mothers) or phase prophylactic medication (1 mother) could not be compared with mothers who did not take the respective medication, mainly due to small sample sizes.

With respect to the intake of antidepressant medication, all exploratory p-values (table 52, right column) failed to show differences between mothers with major depression who took medication and those who did not. Thus, the intake of antidepressant medication showed no association with interactional parameters.

Table 52: Intake of antidepressants and interactional parameters at 1st observation, **left side frequencies** per minute, **right table side: proportions**, test (for exploratory purposes only): Mann-Whitney-U test, two-sided, m = mean, sd = standard deviation, n = sample size with available data, additionally (last table row) results of a multivariate method for a two-group discrimination are given, the logistic regression with backward elimination of non-predictive variables, “backward variable selection” (a p-value of $p \leq 0.05$ denotes that the two groups differ, df = degrees of freedom, χ^2 = test statistic of the final model)

	no antidepressants n = 10		antidepressants n = 13			no antidepressants n = 10		antidepressants n = 13		
	frequencies per minute					proportion of time				
	m	sd	m	sd		m	sd	m	sd	
positive affect mirroring	0.77	0.55	1.34	1.32	p=0.69	0.24	0.28	0.26	0.24	p=0.69
maternal engagement	5.49	2.29	6.19	3.93	p=0.71	0.51	0.31	0.67	0.29	p=0.18
infant engagement	5.94	1.98	7.29	2.79	p=0.17	0.40	0.31	0.47	0.22	p=0.34
infant negative	0.37	0.45	0.65	0.87	p=0.46	0.05	0.08	0.10	0.19	p=0.24
infant protest	0.37	0.45	0.57	0.88	p=0.72	0.05	0.08	0.09	0.19	p=0.40
infant withdrawn	0.00	0.00	0.08	0.28	p=0.38	0.00	0.00	0.01	0.02	p=0.38
infant non mom-focussed	5.56	3.26	6.57	2.73	p=0.46	0.53	0.33	0.51	0.22	p=0.80
infants attention to c.	4.35	1.61	5.43	1.98	p=0.08	0.29	0.26	0.32	0.23	p=0.66
infant pos. engagement	1.36	0.93	1.48	1.69	p=0.60	0.07	0.07	0.07	0.11	p=0.33
c. neg. engagement	0.17	0.53	0.00	0.00	p=0.25	0.04	0.13	0.00	0.00	p=0.25
c. hostile / intrusive	0.03	0.08	0.00	0.00	p=0.25	0.00	0.00	0.00	0.00	p=0.25
c. withdrawn	0.14	0.45	0.00	0.00	p=0.25	0.04	0.12	0.00	0.00	p=0.25
c. non-infant focused	0.05	0.14	0.11	0.18	p=0.26	0.00	0.01	0.00	0.01	p=0.79
c. social monitor and no vocs	3.04	1.53	2.91	2.22	p=0.76	0.40	0.26	0.34	0.29	p=0.53
c. social monitor, pos. vocs	3.83	1.58	4.20	2.44	p=0.71	0.46	0.28	0.57	0.24	p=0.31
c. social pos. engagement	1.80	1.20	2.16	1.96	p=0.98	0.06	0.06	0.11	0.11	p=0.47
c. exagg. pos. engagement	0.00	0.00	0.02	0.06	p=0.38	0.00	0.00	0.00	0.00	p=0.38
logist. regression to discriminate groups: Chi²=21.9, df=15, p=0.11						Chi²=31.2, df=15, p=0.01				

Note: The information regarding the intake of medication was not available for all study participants; the effects of logistic regression could not be attributed to specific dependent variables (upper table, on the right, refer to p-values in the last row)

Moreover, 15 mothers in the major depression group received co-morbid diagnoses (mostly generalized anxiety). An additional comparison of mothers with and without comorbidity revealed no exploratory differences (as shown in table 53). Thus, the

existence of a second disorder showed no association with interactional parameters (with the exception that infants of depressed mothers with a comorbid disorder displayed more negative affects, $p=0.04$).

Table 53: Maternal comorbidity and interactional parameters at 1st observation, **left side frequencies** per minute, **right table side: proportions**, test (for exploratory purposes only): Mann-Whitney-U test, two-sided, m = mean, sd = standard deviation, n = sample size with available data, additionally (last table row) results of a multivariate method for a two-group discrimination are given, the logistic regression with backward elimination of non-predictive variables, “backward variable selection” (a p -value of $p \leq 0.05$ denotes that the two groups differ, df = degrees of freedom, χ^2 = test statistic of the final model)

	no comorbidity (y/n) n = 9		comorbidity (y/n) n = 15			no comorbidity (y/n) n = 9		comorbidity (y/n) n = 15		
	frequencies per minute					proportion of time				
	m	sd	m	sd		m	sd	m	sd	
positive affect mirroring	1.22	1.17	1.05	1.02	p=0.79	0.33	0.28	0.20	0.22	p=0.27
maternal engagement	6.77	3.37	5.59	3.26	p=0.39	0.66	0.33	0.58	0.29	p=0.40
infant engagement	6.34	2.03	7.08	2.79	p=0.53	0.43	0.30	0.45	0.23	p=0.55
infant negative	0.17	0.28	0.71	0.81	p=0.04	0.01	0.02	0.11	0.18	p=0.06
infant protest	0.17	0.28	0.65	0.83	p=0.10	0.01	0.02	0.10	0.18	p=0.13
infant withdrawn	0.00	0.00	0.07	0.26	p=0.44	0.00	0.00	0.01	0.02	p=0.44
infant non mom-focussed	5.85	3.23	6.38	2.78	p=0.53	0.51	0.29	0.54	0.25	p=0.91
infants attention to c.	5.03	1.43	5.11	2.21	p=0.42	0.37	0.28	0.27	0.20	p=0.46
infant pos. engagement	1.32	1.29	1.51	1.43	p=0.72	0.06	0.06	0.08	0.10	p=0.95
c. neg. engagement	0.19	0.56	0.00	0.00	p=0.20	0.04	0.13	0.00	0.00	p=0.20
c. hostile / intrusive	0.03	0.08	0.00	0.00	p=0.20	0.00	0.00	0.00	0.00	p=0.20
c. withdrawn	0.16	0.47	0.00	0.00	p=0.20	0.04	0.13	0.00	0.00	p=0.20
c. non-infant focused	0.07	0.16	0.08	0.17	p=1.00	0.00	0.01	0.00	0.01	p=0.96
c. social monitor and no vocs	2.66	1.97	3.09	1.86	p=0.57	0.25	0.20	0.42	0.30	p=0.11
c. social monitor, pos. vocs	4.40	2.14	3.93	2.05	p=0.53	0.57	0.28	0.51	0.24	p=0.46
c. social pos. engagement	2.61	1.82	1.76	1.52	p=0.32	0.11	0.10	0.09	0.10	p=0.28
c. exagg. pos. engagement	0.03	0.08	0.00	0.00	p=0.20	0.00	0.00	0.00	0.00	p=0.20
logist. regression to discriminate groups: Chi²=9.9, df=15, p=0.83						Chi²=14.8, df=15, p=0.46				

logist. regression to discriminate groups: $\chi^2=9.9$, $df=15$, $p=0.83$

$\chi^2=14.8$, $df=15$, $p=0.46$

Note: Co-morbidity diagnosis not available for all participants.

In addition to this, the single criteria of the depression profile were tested, i.e., of the Structured Clinical Interview for DSM-IV Axis I. Mothers who fulfilled the respective criterion (e.g. diminished interest or loss of energy) were compared to those who did not.

In short, it turned out that none of the interactional parameters showed any association with the depression criteria (tables not reported here): mothers with the criterion ‘depressed mood’ did not differ from those who did not qualify for this criterion. The presence of other criteria such as diminished interest, insomnia, psychomotor agitation or retardation, or loss of energy generally showed no differences in interactional parameters.

All in all, single criteria of the major depression diagnosis were not able to differentiate between both groups.

7.6.4 Correction methods for selection bias: Heckman's (1979) two stage procedure

7.6.4.1 Introduction

When there are certain chances that the drawn sample might not be a random sample of the population of interest, e.g. from the population of mothers with major depression, then this non-random selection process itself may “produce” outcomes that lead to biased inferences of the magnitude of effect of maternal depression. In a linear regression, for example, selection occurs when the dependent variable is associated with a third factor such as maternal education or infant age, and when this dependency is different for the depressed and the non-depressed group.

One approach to handle selection biases is Heckman's two-step correction procedure. For his research on methods for analyzing selective samples Heckman received in 2000 the Nobel Prize in economics (Nobel Prize lecture, 2000; Heckman, 1979; 1995; for a short statistical introduction refer to Smits, 2003; see also Winship and Mare, 1992; Lung-Fei, 1983; Bushway, Johnson and Slocum, 2007). The need for a correction procedure originally resulted from the idea that the effectiveness of social programs is mostly studied under the condition that samples are selected and that a control of the selection process may allow for less biased conclusions with respect to the independent variable (however, in this manuscript unbiased effects of the selected sample of mothers with major depression are of interest).

Basically, in a first step the Heckman procedure explicitly specifies the selection process (**selection model**) that is assumed to be responsible for the bias (e.g. the extent in which mothers with different education levels may have differing sample characteristics). For this purpose, a logistic regression model is applied (as suggested by Smits, 2003) in order to estimate how presumably biasing maternal characteristics, e.g. education, are associated with other sample characteristics, such as the age of the infant at 1st assessment, cohabitation with a partner, the number of children, or the maternal age. The logistic regression estimates how these variables are associated with maternal education which is assumed to bias results. However, the effects on education are not of final interest, since these data are available and can be controlled for. The final interest lies in the unmeasured characteristics and these, of course, are not available in the coefficients of the explanatory variables. Heckman assumes that unmeasured characteristics are available in the residuals after the removal of the effects of known factors. The residuals of the selection model

are used to build a selection bias control factor. According to Heckman, this factor reflects all unmeasured variables that are related to maternal education. Because a factor is included to control for effects on dyadic interactions by unmeasured characteristics (which are also related to maternal education), the other predictors are expected to be less biased by this effect. The individual residual values are added to the data file as an additional variable.

The second step of the Heckman procedure (**analysis of main interest / substantial model**) involves a linear regression analysis of the effects of maternal depression on parameters of dyadic interaction. The control factor for selection bias is used as an additional independent variable.

Since there is a control factor for the effect of the education-related unmeasured variables, which are also related to infant outcome, the variance of other predictors is expected to be reduced and the regression is expected to produce estimates with a lower bias.

There are several limitations concerning the Heckman two-step procedure. Besides the classical requirements of regression models (e.g. linearity of associations, assumption of normality and homoscedastic residuals), the selection model should include a minimum of one dependent variable that does not correlate to the dependent variable in the final equation in order to avoid multi-collinearity, and subsequent difficulties in estimating coefficients and difficulties with unreliable coefficients.

7.6.4.2 Suspected selection processes according to maternal education

In an initial model for bias control, the education of the mother was tested for any effects of selection on outcome parameters, i.e., the proportion of time with positive affect sharing. It is hypothesized that a higher maternal education is associated with increased affect mirroring in the population. Education levels were split into two: qualifications necessary for university entrance (23 cases) versus lower educational level (36 cases, table 54).

Table 54: Maternal education

	cases	%
no qualification for university entrance	23	39%
qualification for university entrance	36	61%
total	59	100%

All calculations were made with SPSS as suggested in detail by Smits (2003). The specification of the selection model, which is assumed to be responsible for bias, i.e. the effect of maternal education, is given in table 55 (estimation model) together with the calculation of Heckman's bias correction factor λ in table 56. It is derived from the residuals of the selection model and contains unmeasured characteristics that are not captured by the regression coefficients and thus - after the effects of observed factors have been eliminated - unknown factors.

Table 55: Specification of the selection process: associations with maternal education level (qualification necessary for university versus lower education levels), results of the logistic regression as precondition to calculate Heckman's correction factor λ .

selection model: associations to maternal qualific. for university (y/n)	coefficient in the regression	p-value	OR	lower 95%-CI	upper 95%-CI
infant age	0.005	0.970	1.005	0.776	1.301
child number	-0.312	0.433	0.732	0.335	1.597
male infant gender (y/n)	-0.231	0.703	0.794	0.243	2.598
group (depression diagnosis)	-0.112	0.856	0.894	0.266	3.003
maternal age (years)	0.198	0.007	1.219	1.055	1.409
Constant	-5.210	0.038	0.005		

N=57, % correctly classified=74%, Chi²=9.6, df=5, p=0.09. Maternal age missing in 2 cases.

Heckman's correction factor ($\lambda = 0.197$) is given in table 56.

Table 56: Heckman's correction factor λ to be included in the substantial regression model of table 57 (formula of the Inverse Mill's Ratio available in Smits (2003))

lambda = 0.197
cases = 59

Now, since the correction factor λ is known, it can be included in the substantial analysis, an OLS regression analysis estimated with the SPSS procedure "regression" (weights have been calculated according to Smits, 2003).

As shown in table 57 - after an inclusion of Heckman's bias correction factor into the model - the depression diagnosis itself, however, did not ($p=0.901$) allow for a differentiation in the primary target parameter (mother-infant affect mirroring).

Table 57: Substantial analysis with Heckman's correction factor Lambda as additional regressor in an WLS regression analysis (with the primary target parameter of the study, the proportion of time with positive affect mirroring as criterion)

substantial model: weighted least square regression including lambda	coefficient in the regression	t-value	p-value
(Constant)	-0.07	-0.08	0.935
LAMBDA	0.19	0.60	0.554
group (depression diagnosis)	0.01	0.13	0.901
maternal age (years)	0.01	0.36	0.718
comorbidity (y/n)	-0.14	-1.40	0.168
maternal qualific. for university (y/n)	0.04	0.55	0.583

Substantial model: weighted least square regression including lambda

Thus, even after the explicit introduction of a bias correction (maternal education), the depression diagnosis was not predictive for the interaction outcome (in terms of time proportions in affect mirroring).

7.6.4.3 Suspected selection processes according to infant age

In the second model, the age of the infants was used, and potential selection effects related to infant-age were subtracted. It is hypothesized that higher infant age and associated effects allow for a higher proportion of affect sharing, which may overlap a depression-related effect. Infant age was split into groups of ± 3 months (table 58), i.e. very young infants were compared with older ones (the split itself still maintained the ranking type of the infant age scale).

Table 58: Infant age below and above 3 months

Infant age	cases	%
infants < 3 months	21	36%
infants 3 months or above	38	64%
total	59	100%

The specification of the selection model in which the infant age is assumed to be responsible for bias is given in table 59. It shows that almost no characteristic was associated with infant age. However, these effects are not of final interest. On the contrary, the unmeasured characteristics are of interest (Heckman assumes that unmeasured characteristics are available in the residuals after the removal of the effects of the known factors).

Heckman's bias correction factor λ is given in table 60 and is of very small magnitude.

Table 59: Specification of the selection process: associations with infant age, results of the logistic regression as precondition to calculate Heckman's correction factor λ .

selection model: predictors for Infant age	coefficient in the regression	p-value	OR	lower 95%-CI	upper 95%-CI
maternal qualific. for university (y/n)	0.076	0.904	1.079	0.311	3.742
child number	-0.061	0.866	0.941	0.464	1.909
male infant gender (y/n)	-0.812	0.164	0.444	0.142	1.393
group (depression diagnosis)	0.337	0.571	1.401	0.437	4.489
maternal age (years)	-0.038	0.588	0.963	0.841	1.103
Constant	2.139	0.336	8.489		

N=57, % correctly classified=67%, Chi²=2.9, df=5, p=0.71. Maternal age missing in 2 cases.

table 60: Heckman's correction factor λ to be included in the substantial regression model of table 61 (formula of the Inverse Mill's Ratio available in Smits, 2003)

lambda = -0.002
cases = 59

Now, since the correction factor λ is known, it can be included in the substantial analysis (Smits, 2003; an OLS regression analysis estimated with the SPSS procedure "regression", the results of this regression are shown in table 61.

Table 61: Substantial analysis with Heckman's correction factor Lambda as additional regressor in an WLS regression analysis (with the primary target parameter of the study, the proportion of time with positive affect mirroring as criterion), standard errors are corrected by running the substantial analysis as a WLS regression

substantial model: weighted least square regression including lambda	coefficient in the regression	t-value	p-value
(Constant)	0.43	1.79	0.079
LAMBDA	0.00	0.01	0.989
group (depression diagnosis)	0.02	0.22	0.824
maternal age (years)	0.00	-0.33	0.743
comorbidity (y/n)	-0.10	-1.01	0.319
maternal qualific. for university (y/n)	0.01	0.12	0.907
model: p=0.89, F=0.33, goodness: R=0.18,			

Substantial model: weighted least square regression including lambda

As shown in table 61 above - after the correction for potential biasing effects of infant age, the depression diagnosis itself did not allow for a differentiation in the primary target parameter (p=0.824, mother-infant affect sharing).

7.7. Calculation of conditional power - futility of a sample size extension

Conditional power is a probability based on the idea that the data gathered thus far may not fully reflect the effects of the population and that an extended patient-recruitment will lead to statistically significant differences (Lachin, 2005). Many clinical trials analyze conditional power at a pre-specified point in the study, e.g., when 50% of the subjects were recruited. The interim results are used to decide

whether to terminate the study early (stopping for futility, e.g., in case of weak or zero effects), or, to calculate a sample size extension in case of emerging trends (sample size re-assessment). Technically, the conditional power is defined as the conditional probability that the study result will exceed a critical test statistic based on the data observed thus far (and not necessarily a significant one), plus the assumption about a trend to be observed in the ongoing study.

Suppose that a similar study such as the present one had been planned as a two-stage design, i.e., with an interim and a final analysis. An a-priori calculation for a group-sequential test of average values - for example - based on the O'Brien-Fleming error-spending function (O'Brien and Fleming, 1979; software NCSS, Hintze, 2007) shows that 2 times 30 (i.e. a total of 60 cases) cases for the first stage plus 2 x 30 cases for the second stage have 86% power to show a mean difference of 0.10 with a standard deviation of 0.20 (in terms of time proportions see the 1st primary target parameter of the present study). Accordingly, the 1st stage decision limit is $\alpha_1 = 0.0056$. Based on that limit the study could be stopped. The limit at the 2nd stage is $\alpha_2 = 0.048$ (power analysis for a group-sequential design, based on a one-sided test of means and the O'Brien-Fleming error-spending function; O'Brien and Fleming, 1979).

Approximately 2 times 30 cases in the study so far failed to fall below $\alpha_1=0.0056$ and revealed a p-value of $p_1=0.12$ instead. A conditional power analysis according to Chang (2007) revealed a conditional power value of less than 0.01 that a sample size extension of an additional 2 times 30 cases will result in a significant value after the 2nd stage (SAS macro "ConPower", macro 7.1, Chang, 2007). Thus the chances are less than 1% that an extension of the sample size will detect a difference. Accordingly, a reassessment of the necessary sample-size extension for a 2nd stage showed an unrealistic sample size to ensure a power of 80% ($n > 250$ per group, SAS macro nByCPower, macro 7.2, Chang, 2007). All in all, neither a small nor a large sample size extension increases the study-power sufficiently.

7.8. Comparison of the interactional data of the present study with a large reference-study (n=695)

For the evaluation of the representativeness of the present study, the data were compared with data of a reference study that delivered one of the largest data sets for interactions. This is the study of Tronick et al. (2005), which included 695 dyads and coded interactional behavior within a setting that was identical to the present study: Mothers and infants were videotaped in the still-face paradigm, the infants' and mothers' behavior were coded using the Infant and Caregiver Engagement Phases (ICEP; Weinberg and Tronick, 1998). The infants were on average 4 months old.

Although the initial study-intention referred to differences in interactions between cocaine-abusing mothers and mothers without a history of drug problems, the study generally failed to show relevant differences between these groups: although the abstract reports differences, there is no correction for multiple testing. If this is applied in table 5 a wide range of p-values does not allow for an interpretation in terms of significance. Moreover, the reported p-values in this table refer to apparently irrelevant differences of below 5% (compare mean values; there are differences marked as statistically significant concerning average differences of about 1%). Since the differences are small to negligible, the pooled data of both groups will be used for a comparison with the present interactional data (these data are presented in the study of Tronick in table 4, page 717).

Based on simple descriptive data, the comparison with the external data (figure 28) shows few to no differences between the present study-data (in red and blue) and the comparison study (in black). In fact, the profiles of both studies are almost identical.

Thus a comparison with a large external data-set (collected in an identical setting with identical dependent variables and with infants of comparable age, i.e. of about 4 months) shows that the data of the present study are well within comparable ranges and the present study appears to be well representative.

Time proportions: comparison with 695 dyads with 4-month olds (Tronick et al., 2005)

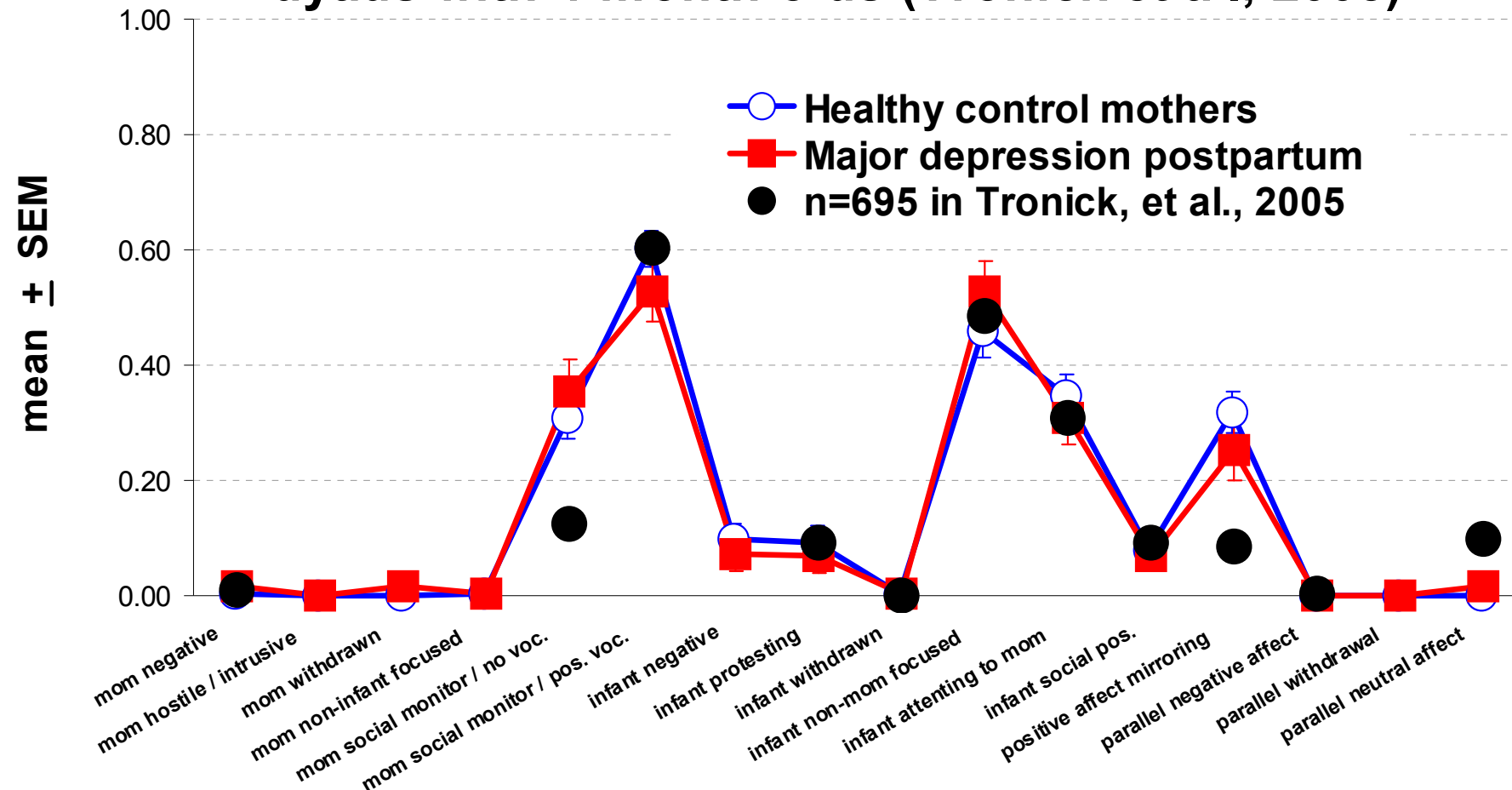


Figure 28: Time proportions of interactive behaviors from the study of Tronick et al. (2005, dots in black) in 695 dyads descriptively compared with the data of the present study (blue and red dots).

7.9. Multivariate and explorative test for equivalence of dyads with a mother with major depression and control dyads

Since almost all contrast zero-hypothesis could not be rejected it might be possible that dyads with a depressed mother are significantly equivalent compared to control dyads. According to Wellek (2002) an equivalence hypothesis may easily be tested: equivalence of two groups can be shown based on the interval-inclusion method. The construction principle is: First, calculate a point estimate of the between-group contrast, e.g., use the two-group mean difference, use Cohen's d (Cohen, 1988), or, better, an assumption-free non-parametric contrast measure such as the Wilcoxon-Mann-Whitney-coefficient, $\Theta = p(X>Y) + \frac{1}{2} p(X=Y)$ (Lehmann, 1998; Ahmad, 1996; Priebe and Cowen, 1999). Then, calculate the upper and lower confidence interval (CI) of this measure (e.g. the 95%-CI). Finally, equivalence can be concluded if this confidence-interval is included within predefined limits (interval inclusion method). For example, two groups may be considered as equivalent if the confidence interval of a Cohen's d is small, or, at least is positioned well *below* a "medium" sized effect (Cohen, 1988). Since Cohen characterized "medium-sized" with a $d = 0.50$, this may be written as

$$- 0.50 \leq d_{95\% \text{ lower CI}} < d < d_{95\% \text{ upper CI}} \leq + 0.50$$

Moreover, the Cohen limits can be transferred to the non-parametric measure of contrast, the Wilcoxon-Mann-Whitney-coefficient, based on

$$\Theta = \phi\left(\frac{d}{\sqrt{2}}, 0, 1\right),$$

with ϕ as the cumulative normal distribution with zero-mean and standard deviation of 1; d is Cohen's d and Θ ("theta") is the non-parametric measure of contrast (see above). Since a medium sized effect according to Cohen ($d = 0.50$) corresponds to a $\Theta = 0.64$ (note that $\Theta = 0.5$ corresponds to a zero-contrast and $\Theta = 0.36$ to $d = -0.50$) equivalence may be concluded for exploratory reasons if the confidence interval lies within the limits well below a medium sized effect, i.e.

$$0.36 < \Theta_{95\% \text{ lower CI}} < \Theta < \Theta_{95\% \text{ upper CI}} < 0.64$$

The contrast measures for the most important measures of this research are shown in figure 29 (page 219). Each point estimate is given as dot and the non-parametric confidence regions with vertical error bars. The more these dots are deviating from the zero-contrast-line of $\Theta = 0.5$ the greater is the between-group contrast. The figure shows that many of the confidence regions (e.g. positively mirrored affects, or, maternal or infant engagement, or, the other measures) are placed around the line of zero-contrast ($\Theta = 0.5$), most of them between upper and lower limit of a medium sized effect.

Moreover, the combined measure (the estimator of the nonparametric multivariate effect according to Wei and Lachin, 1984, Wei and Lachin, 1984, refer to the blue diamond at the right of figure 29) and its confidence band are well within the upper and lower boundaries of a medium sized effect and very close to a zero contrast ($\Theta = 0.55$, with 95%-CI between 0.50 and 0.59).

Thus, the combined measure allows for the conclusion that dyads with a mother with major depression and control dyads are significantly equivalent regarding their interactive behavior.

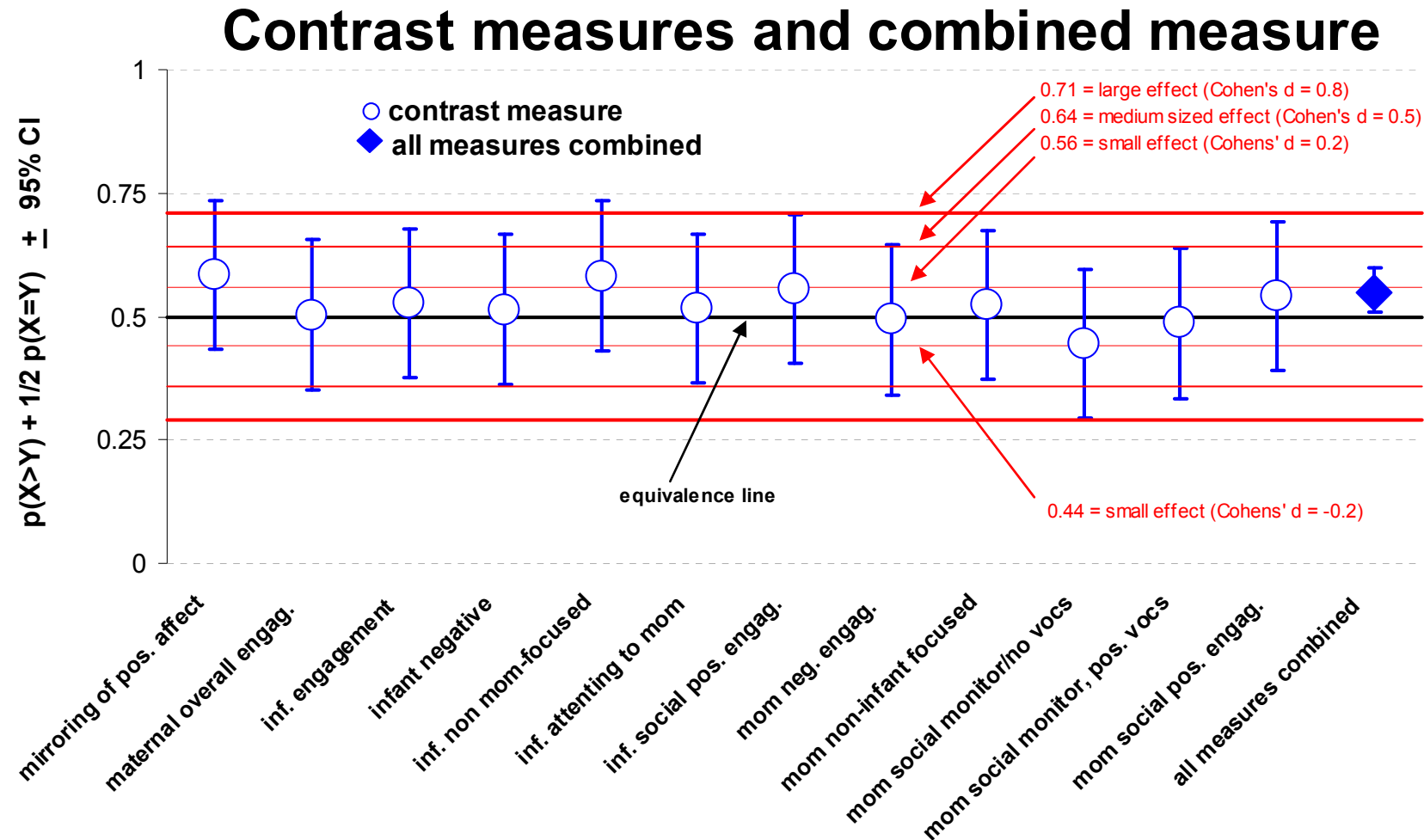


Figure 29: Assumption-free non-parametric contrast measures (blue dots, blue diamond = combined measure), Wilcoxon-Mann-Whitney-coefficients, $\Theta = p(X>Y) + \frac{1}{2} p(X=Y)$, Lehmann, 1998; Ahmad, 1996; Priebe and Cowen, 1999, and the 95%-confidence intervals (blue vertical bars). The combined measure (Wei and Lachin, 1984; Lachin, 1992) and the 95%-confidence interval is given as well. The limits in correspondence to Cohen's d are given with red lines. Calculation based on the open-source computer program of Davis (2000). Confidence interval for the combined effect was made according to Rosenthal (1991) based on the z -value of the global test for stochastic ordering

7.10. Simple effects of the initial level of affect-mirroring between mother and infant

In an additional analysis dyads were split into subgroups according their level of affect mirroring (i.e. parallel occurring positive affect-related behaviors) in the initial play period. The 33rd and 66th percentile were applied to form three subgroups (dyads with low, medium and high level of initial affect mirroring that are shown in the header of the table 62). All three “sharing-”groups were compared how the infants in the later play phase behaved. Exploratory comparisons (table 62 and 63) showed that infants in dyads, who previously had a high sharing level, later (in play 2) increasingly engaged (bold p-values), were less negative, were less non-focused on the mother, attended and engaged more. Thus, not diagnosis was predictive but behavior extremes in terms of low versus high affect mirroring.

Table 62: **Proportions of time** of infant and maternal behaviour depending on the amount of affect sharing in phase 1 (= affect mirroring, i.e. parallel-occurring positive affects of mother and infant, split into low, medium and high). Test for global group differences or differences in change values: rank analysis of variance (Lehmann, 1998); m = mean, SE = standard error. All comparisons are strictly descriptive and exploratory, p-values lower equal 0.05 or letters “a”, “b” or “c” denote an exploratory difference (see also below)..

proportion of time	low sharing (lowest 33% during play 1) (n=20)				mid sharing (mid 33% during play 1) (n=19)				high sharing (upper 33% during play 1) (n=20)				group differences (rank analysis of variance) play2	play 1 - 2 changes compared play 1 to play
	play 1		play 2		play 1		play 2		play 1		play 2			
	m	se	m	se	m	se	m	se	m	se	m	se		
maternal engagement (proportion)	0.53	0.05	0.56	0.07	0.62	0.06	0.63	0.06	0.86	0.03	0.78	0.04	p=0.06b	p=0.12
infant engagement (proportion)	0.26	0.06	0.39	0.06	0.42	0.05	0.44	0.05	0.75	0.04	0.62	0.07	p=0.03b	p=0.01b
infant negative	0.12	0.06	0.12	0.04	0.05	0.02	0.14	0.05	0.02	0.01	0.08	0.04	p=0.07b	p=0.26
infant protesting	0.12	0.06	0.11	0.04	0.05	0.02	0.12	0.05	0.02	0.01	0.08	0.04	p=0.10b	p=0.33
infant withdrawn	0.00	-	0.01	0.01	0.00	-	0.02	0.02	0.00	-	0.00	-	p=0.59	p=0.59
infant non-mom focused	0.69	0.06	0.55	0.06	0.60	0.05	0.45	0.06	0.27	0.04	0.36	0.07	p=0.17b	p<0.01bc
infant attending to caregiver	0.13	0.03	0.25	0.06	0.33	0.05	0.29	0.05	0.57	0.05	0.43	0.05	p=0.03b	p<0.01ab
infant social pos. engagement	0.02	0.00	0.03	0.02	0.05	0.01	0.03	0.01	0.18	0.04	0.13	0.03	p<0.01bc	p=0.16
infant positive or neutral	0.12	0.03	0.05	0.02	0.03	0.01	0.14	0.05	0.05	0.02	0.08	0.02	p=0.30	p=0.01ab
caregiver neg. engagement	0.02	0.02	0.02	0.02	0.00	0.00	0.00	-	0.00	-	0.00	-		p=0.70
caregiver hostile / intrusive	0.00	0.00	0.00	0.00	0.00	-	0.00	-	0.00	-	0.00	-		
caregiver withdrawn	0.02	0.02	0.02	0.02	0.00	0.00	0.00	-	0.00	-	0.00	-		p=0.23
caregiver non-infant focused	0.00	0.00	0.01	0.00	0.00	0.00	0.00	-	0.01	0.01	0.00	0.00	p=0.16	p=0.57
caregiver social monitor and no vocs	0.44	0.06	0.38	0.07	0.39	0.06	0.39	0.06	0.15	0.03	0.23	0.04	p=0.13	p=0.06b
caregiver social monitor, pos. vocs	0.48	0.05	0.50	0.07	0.54	0.05	0.54	0.06	0.73	0.03	0.64	0.04	p=0.32	p=0.23
caregiver social pos. engagement	0.05	0.01	0.06	0.02	0.09	0.03	0.10	0.04	0.15	0.03	0.16	0.04	p=0.02bc	p=0.83

Note: last 2 columns: for pair-wise comparisons based on exploratory Mann-Whitney-Tests and if p-values $p \leq 0.05$ then:
a = low versus mid-range affect sharing ($p \leq 0.05$), b = low versus high ($p \leq 0.05$), c = mid-range versus high range ($p \leq 0.05$), all tests are strictly exploratory

Table 63: Frequencies per minute of infant and maternal behaviour depending on the amount of affect sharing in phase 1 (= affect mirroring, i.e. parallel-occurring positive affects of mother and infant, split into low, medium and high). Test for global group differences or differences in change values: rank analysis of variance (Lehmann, 1998); m = mean, SE = standard error. All comparisons are strictly descriptive and exploratory, p-values lower equal 0.05 or letters "a", "b" or "c" denote an exploratory difference (see also below)..

frequencies per minute	low sharing (lowest 33% during play 1) (n=20)				mid sharing (mid 33% during play 1) (n=19)				high sharing (upper 33% during play 1) (n=20)				group differences (rank analysis of variance)	play 1 - 2 changes compared
	play 1		play 2		play 1		play 2		play 1		play 2			
	m	se	m	se	m	se	m	se	m	se	m	se		
maternal engagement (frequencies per minute)	5.30	0.59	4.26	0.70	6.83	0.75	5.34	0.49	7.31	0.72	6.74	0.70	p=0.04b	p=0.77
infant engagement (frequencies per minute)	4.60	0.58	5.82	0.96	7.28	0.64	6.60	0.68	10.17	1.32	7.69	1.18	p=0.56	p=0.03b
infant negative	0.51	0.21	0.81	0.18	0.62	0.23	0.56	0.29	0.26	0.14	0.32	0.17	p=0.01b	p=0.35
infant protest	0.51	0.21	0.71	0.17	0.62	0.23	0.51	0.29	0.26	0.14	0.32	0.17	p=0.02b	p=0.37
infant withdrawn	0.00	-	0.10	0.10	0.00	-	0.05	0.05	0.00	-	0.00	-	p=0.59	p=0.59
infant non mom-focused	7.10	1.12	5.64	0.67	6.03	0.45	5.28	0.63	6.26	1.19	5.09	0.99	p=0.55	p=0.92
infants attention to caregiver	3.51	0.61	4.50	0.77	5.72	0.45	5.15	0.49	7.19	1.01	5.59	0.85	p=0.63	p=0.02ab
infant pos. engagement	0.65	0.16	0.75	0.37	1.21	0.31	1.05	0.26	3.31	0.76	2.16	0.68	p=0.01b	p=0.70
infant positive (ineu, ipos, abs. freq.)	3.82	1.19	1.60	0.53	1.17	0.24	1.30	0.41	1.95	0.95	2.08	0.68	p=0.67	p=0.09b
caregiver neg. engagement	0.11	0.08	0.10	0.10	0.02	0.02	0.00	-	0.00	-	0.00	-		p=0.72
caregiver hostile / intrusive	0.04	0.04	0.03	0.03	0.00	-	0.00	-	0.00	-	0.00	-		
caregiver withdrawn	0.07	0.07	0.08	0.08	0.02	0.02	0.00	-	0.00	-	0.00	-		p=0.23
caregiver non-infant focused	0.11	0.05	0.12	0.07	0.09	0.05	0.00	-	0.20	0.20	0.00	-		p=0.72
caregiver social monitor and no vocs	3.62	0.40	2.94	0.43	3.59	0.50	3.68	0.46	2.16	0.44	2.82	0.43	p=0.39	p=0.02b
caregiver social monitor, pos. vocs	3.99	0.40	3.31	0.50	4.74	0.45	3.97	0.34	4.57	0.40	4.58	0.45	p=0.16	p=0.46
caregiver social pos. engagement	1.39	0.32	1.08	0.28	2.27	0.40	1.42	0.30	2.98	0.42	2.54	0.41	p=0.01b	p=0.39

Note: last 2 columns: for pair-wise comparisons based on exploratory Mann-Whitney-Tests and if p-values $p \leq 0.05$ then:
a = low versus mid-range affect sharing ($p \leq 0.05$), b = low versus high ($p \leq 0.05$), c = mid-range versus high range ($p \leq 0.05$), all tests are strictly exploratory

8. Discussion

8.1. What is already known

A range of publications have found maternal depression to be well predictive for compromised mother-infant interaction. Moreover, longitudinal studies have concluded that children of depressed mothers are at risk for internalizing or externalizing behaviors or at risk for developing performance problems. A further line of research suggests that dysfunctional mother-infant interaction may act as a mediator between a maternal diagnosis of depression and deviant child behaviors and impaired development. And finally, another line of research suggests that the sole presence of an episode of maternal depression is predictive for adverse effects in children. Conversely, the absence of such depression (i.e. in case of a remission of maternal depression) has been suggested to precede ameliorations in adverse child effects (e.g. the group of Weissman; see the review of Gunlicks and Weissman, 2008; or of Hammen and Brennan, 2003).

Gathering together these lines of research (i.e. the predictive value of maternal depression, children of depressed mothers are under risk, the interaction of depressed mothers tending to be a risk mediator, and finally, the parallelism in time of diagnosis and adverse child outcome), several working groups (e.g., Stanley et al., 2004; Murray et al., 1999; Field, 1992) suggested that the exposure of an infant to maternal depression and to deviant mother-infant interaction might be a suitable precedent and predictor for adverse child effects (e.g. a heightened risk of child disorder diagnoses or impaired child performances). It was also predicted that the remission of depression may lead to improvements in child outcome.

Although the available research covers a wide range of dependent measures of dyadic interaction, settings and study designs, two theoretical lines of research emerge from the literature concerning why maternal depression may contribute to these effects:

First, a depressed mother may be restricted in parenting resources (theory of impaired parenting), in particular due to flat affect and loss of energy, which may alter reinforcement conditions for the infant (e.g. Lovejoy et al., 2000).

Secondly, due to flat affect, a depressed mother may fail to use this affect to regulate

her infant's affects. Failures in infant regulation are theorized by various authors as precedents of adverse infant outcome, e.g. externalizing behavior. In particular, the theory of Gergely (affect mirroring as social biofeedback, Gergely and Watson, 1996) predicts that depressed mothers may show impaired affective mirroring and thus fail to provide infant regulation.

In the present research both theories together allowed for a prediction of deviant mother-infant interaction and reduced affect mirroring under the exposure to maternal depression.

On the other hand, a reduction of interaction deviancies has been predicted in the case of remission of maternal depression (transient child disturbance theory). The theory predicts that child maladjustment and dysfunctional interactions may disappear when the maternal depression remits (Gunlicks and Weissman, 2008; Downey and Coyne, 1990). Accordingly, this study expected deviant mother-infant interaction to disappear and affect mirroring to normalize after the remission of the maternal major depression.

8.2. Maternal major depression as predictor for deviancies in mother-infant interaction, in maternal or infant behavior

Based on Gergely's theory, this study derived the prediction of impaired affect mirroring in presence of a depressed episode. However, the data do not provide support that the diagnosis of major depression has predictive value in that domain. Surprisingly, dyads with a depressed mother did not differ from control dyads in their level of co-occurring positivity (labelled as affect-mirroring). There were also neither general reductions nor any deviancies in specific affects (e.g. in withdrawal, infant protest, maternal hostility, or the degree of positivity of mother and infant).

Although some authors suggested that affect mirroring is one transmission mechanism of how depression-related effects unfold from mother to infant (e.g., that infants of depressed mothers withdraw, be less responsiveness, or show heightened negativity; Field, 1984; Stern, 1985; Field et al., 2007), in this study a postpartum major depression did not affect the ability to share or mirror affects. The absence of reduced affect mirroring, together with floor affects in negativity, contradicts the findings of the authors above as well as others, including Stein et al. (1991) who found lowered affective sharing, or the findings of Cohn et al. (1986; 1990), who found correlated negativity between mother and infant, or Radke-Yarrow et al. (1993), who found

heightened synchrony in negativity in depressed dyads.

It also contradicts previous findings of lowered imitation behavior in children of depressed mothers, such as the findings by Field et al. (1985, 1987). Moreover, the floor effects in negativity in the present study are surprising, in particular when compared with other studies with identical settings that reported low positivity but heightened parallelisms in negativity in presence of maternal depression (Field, Healy and Leblanc, 1989; Field, Healy, Goldstein and Guthertz, 1990).

Conversely, in cases when mirroring behaviors (parallel occurring affects) were interrupted, in depressed mothers these behaviors are reinstated in a comparable way as in controls, i.e. depressed mothers did not excel by higher latencies to overcome interruptions. Descriptively, dyads with a depressed mother were almost identical compared to healthy dyads. Their latency until affective mirroring was re-initiated did not differ at any point of measurement. Thus neither mirroring of affect in general nor the onsets were found to be deviant under major depression.

Furthermore, infants of major depressed mothers did not differ from control infants in their reaction to maternal unavailability, i.e., they were not affected by the maternal still-face procedure any differently. This sharply contrasts to several studies that reported both significant and large still-face effects, as reported by the working group of Field et al. (1984; 2007), particularly in the sense that infants of depressed mothers were completely unaffected by maternal unavailability. For example, Field et al. (e.g., 2007) suggested that infants of depressed mothers generalize over situations, e.g. they may carry on behaving “depressed”, even when interacting with another non-depressed partner. But any depression-like behavior in infants of depressed mothers, however, could not be detected in the present data (some reasons why this might be the case are given below). The results of this study are well in line with Stanley, Murray and Stein (2004) who found no effects in the still-face data in association with a depression diagnosis (see page 11 of that publication). Other authors also completely doubted the predictive value of a maternal depression diagnosis, e.g. Campbell, Cohn and Meyers (1995, with the exception of chronic depression) or failed to find a general effect, e.g. Cohn, Campbell, Matias and Hopkins (1990) who only found effects after very specific splits into very small sub-groups. No differences between healthy and depressed mothers in a range of interaction parameters were also found by Hossain et al. (1994; table 2, page 353, results for mothers only).

Fleming et al. (1988; page 78) also concluded that depressed mothers, in spite of having difficulties in expressing emotions, are able to care adequately for their infants. Here, Fleming's findings of less affectionate behaviors in depressed dyads could not be replicated. Thus the interaction data of the present study, in association with the studies mentioned above, does not indicate that the parenting impairment hypothesis has predictive value for mothers with major depression.

It was further predicted that depressed mothers lack infant-stimulating behaviors and, vice versa, that their infants are generally deviant in their activity level (e.g. are hypo-active or hyper-active). However, both predictions did not find any support in the data: mothers in an episode of major depression did not display any impairment in stimulating behaviors and their infants' activity was not different from that of the controls. These results contradict a range of findings upon which this research was based, e.g., Field et al. (2007), who reported lower behavior frequencies (facial affect, motor and gaze activity) in association with maternal depression, or Bettes (1988), Breznitz and Sherman (1987) and Weinberg and Tronick (1998), who reported clear interactive impairments (e.g. impaired vocalization or engagement).

Taken together, the data of this study did not support the hypothesis of parenting impairments, since mothers with major depression did not show any restrictions in their resources, nor were there any deviations in infant behavior. Contrary to the conclusions of the studies cited above, these results might suggest the possibility that a maternal depression diagnosis does not stringently lead to certain detrimental behavior in either mother or infant. This conclusion is well in accordance with Hoffman and Drotar (1991), who did not find lower maternal stimulation and infant activity in association with maternal depression, or Fleming et al. (1988), who concluded that depressed mothers, although restricted in expressing emotions, are able to adequately care for their infants and respond to their needs.

Several exploratory hypotheses based on more specific predictions were added, e.g., the expectation of generally heightened negativity in mothers with major depression during interaction (e.g. hostile, intrusive or exaggerated behaviors or, vice versa, withdrawn, non-infant focusing behavior) or a relative level of negativity (i.e. related to the total level of behaviors).

However, contrary to the frequently cited studies of Field (1984) and Cohn et al. (1990; 1986), and to the findings of heightened negative responsiveness in

depressed mothers as reported by Stanley, Murray and Stein (2004), the present study did not indicate that mothers with major depression interact at an heightened level of negativity. Although the study design was a replication of a consistently used interaction paradigm (however, with a focus on acute major depression) maternal negativity was hardly observed in the present study. Floor effects dominated maternal behavior in both groups, including in the dyads with a depressed mother. Thus, contrary to expectation, the behavior of mothers with major depression was not characterized by negativity, e.g. by heightened hostility or even by withdrawal during interactions, even if maternal negativity was related to her total behavior level.

Since much literature describes depressed individuals as having lower social or interactive skills (e.g. Segrin, 2000; 1994), mothers with major depression were predicted to show a restricted behavior repertoire. Additionally, based on the expectation that a mother transfers behavior by modelling, their infants were predicted to show a restricted behavioral repertoire. Neither prediction, however, did not comply with the data nor were any trends observed. Neither mothers with major depression nor their infants showed any indications of restricted repertoire in the sense that they made less use of the available behavior categories.

Furthermore, based on the theory of restricted resources, a reduced general speed of interaction in the sense of a slower “production” of behaviors was predicted in depressed individuals, but could not be confirmed. The failure to find a reduced behavior speed contradicts our selection criteria of major depression, where “loss of interest” or “loss of energy” as a main criterion for major depression was almost entirely fulfilled. Again, this contradicts the parenting impairment and resource restriction hypothesis, and opens up the possibility that a diagnosis of major depression may not stringently generalize to or affect mother-infant interaction.

Moreover, mood contagion approaches (Joiner and Katz, 1999; or, for infant research: Field 1986) suggest that depressed individuals induce rejection in their interaction partner. Accordingly, the present study predicted that mother-rejecting or generally negative infant behaviors during interaction would increase with ongoing the duration of interaction. However, infants of depressed mothers had no marked accumulation in protest or non-mother focused behaviors during interaction time. A further aspect within the framework of mood contagion is the prediction that infants of depressed mothers are unwilling to interact when maternal engagement increases

and that reciprocal negative affects will occur. Both predictions, however, had no counterpart in the data, nor were there any trends. First of all, an increasing duration of interaction did not lead to heightened negativity in infants of depressed mothers, nor did heightened engagement of depressed mothers lead to signs of unwillingness to interact in infants. Finally, there were no indications of heightened reciprocities, e.g. spirals of negativity, in depressed dyads.

To sum up, it appears that mood contagion approaches in observational mother-infant data might not have the same level of predictive value which is proposed in reviews (Field, 1992; Joiner and Katz, 1999; Segrin and Dillard, 1992) and as given by Field et al. (1990) for interactions in the infant-domain. Note that these affect contagion theories are commonly formulated irrespective of the age of the interactants, i.e. age is not suggested to have either mediating or moderating status. Contrary to the conclusion of Field et al. (1990; page 12; here she explicitly referred to the mood contagion theory to explain heightened reciprocity of negativity), this study failed to show a heightened prevalence of negativity-spirals under maternal major depression.

A further line of research approaches (regarding how maternal depression may contribute to effects) was pursued based on interpersonal stress approaches (e.g. Hammen et al., 1991; 2004; 2002; this manuscript page 32). These approaches predict that engagement of depressed mothers is witnessed as stressful by their children. Accordingly, mothers with major depression, who - in addition - were highly affectively engaging, were predicted to elicit much more negativity in their children. Contrary to this, infants of depressed mothers were not found to protest more or be less positive. These findings contradict Hossain et al. (1994), who concluded that infants have better interactions with non-depressed individuals due to the stressing effects of depression.

Furthermore, according to symptom-based approaches, depressed individuals were predicted to have less interest in maintaining interaction ("interactive exhaustion hypothesis"). Moreover, they were assumed to be less contingently responsive. The latter was also predicted for their infants. Although mothers affectively engaged about $\frac{2}{3}$ of their total time with an initiation frequency of 6-8 per minute, depressed mothers basically remained on the same level with increasing observation time. They showed no signs of exhaustion nor any reduced reactions to infant signals, i.e. no prolonged

contingencies, which even applied to their infants. Thus three additional hypotheses which followed a symptom-based or parenting impairment approach as suggested by Lovejoy et al. (2000) failed to show any deviancies in association with maternal major depression (a series of reasons is given below).

Finally, predictions were tested based on the idea that maternal affect allows for a control of infant behavior; e.g. if depressed mothers show impaired infant regulation in the sense that their infants are less able to regain control or re-focus on the mother after intense emotions, e.g. with the cessation of protest or of non-mother focused behaviors. However, with respect to those latencies, infants of depressed mothers once again did not differ from the control infants, nor were any trends observable. It is surprising that approaches that predict that depressed mothers would fail to give their infant regulatory help (e.g. Tronick and Gianino, 1986; Tronick and Reck, 2009) did not correspond to this data.

Finally, both theories, i.e. resource restrictions and regulation distortion, allowed for the prediction of lowered synchrony or predictability. However, there were no differences in dyadic capacity to interact synchronously, nor were there any reductions in the predictability of affect-related behavior (which, by the way, was extraordinarily low even in the control group).

Theories of resource restrictions also allowed for the prediction of lowered maternal responsiveness in specific behaviors (e.g. the onset of vocalizations). Based on changed reinforcement conditions, infants of depressed mothers were predicted to be deviant in their responsiveness (e.g. responsiveness being either too low due to hypo-stimulation or highly eliciting due to maternal unresponsiveness). However, there were no indications of deviant infant responsiveness. Descriptively, infants of depressed mothers were quite comparable with infants of control mothers.

8.3. Predictive value of the remission from major depression for normalized mother-infant interactions

The hypothesis of transient child disturbance allowed for the prediction that deviant interactive behaviors may occur when being exposed to parental major depression and, conversely, it allowed for the prediction that a remission from depression will be paralleled by parental and child ameliorations. Based on the parental disability hypothesis (e.g., major depression characteristics of flat affect and loss of energy), it was predicted that impaired affect mirroring in dyads with a major depressed mother

will normalize, i.e., no longer differ from that of control mothers after the major depression is remitted. However, neither mirroring, nor any other indicator pointed to changes in infants whose mothers remitted from depression. On the contrary, prolonged disturbances in affect mirroring did not occur.

Accordingly, there were no signs of decreasing negativity in infants in association with the depression remission. Negativity was generally low, contrary to previous reports by Field et al. (1990, Field (1984), and Campbell et al. (1995), but otherwise well comparable to the low negativity rates found by Cohn et al. (1990). Rutter (1990) was one of the first who critically pointed out a large heterogeneity in the prevalence of child negativity in association with maternal depression. Surprisingly, mothers suffering from an episode of major depression and their infants do not differ from controls; and the remission from depression was not associated with improvements in affect mirroring or a decrease in infant negativity. Thus, transient disturbance approaches, e.g. as suggested by Hammen, Burge and Adrian (1991), Weissman et al. (2006) or Gunlicks and Weissman (2008) were not supported by this data. In addition, approaches that assume that adverse infant behavior generalizes beyond the maternal depression or continues into the period of remission (Field, 1992; page 51) did not find support in this data.

Moreover, infants of depressed did not respond differently, e.g. to a restricted maternal communication (still-face). Infants of remitted mothers behaved as control infants did; yielding no support for the transient or the prolonged disturbance hypothesis.

Furthermore, mothers showed no changes in their levels of infant stimulation, irrespective of whether they were currently experiencing an episode of major depression or not. The data showed no improvements in association with depression remission. Infants of depressed mothers showed no changes in activity levels; thus a remission-associated return to non-deviant behaviors (comparable to those of controls) was not observable. Again, the results neither support the hypothesis of transient child disturbances nor the hypothesis of a prolonged effect of child disturbance that may last beyond the remission.

There were also several additional approaches with respect to the predictive value of depression, that also had no counterpart in the data, e.g. that the remission from maternal depression ameliorates stressful effects of maternal behaviors. However,

since the data failed to show that interaction with a depressed mother is stressful for the partner (in the sense of accumulating infant negativity), no remission-related decrease in infant negativity could be observed, a further indication that the timing of the maternal depression might not be that crucial as suggested by Gunlicks and Weissman (2008).

A decrease in maternal negative behavior (according to Coyne's theory of a potential precursor of rejection) was also predicted in association with depression remission. However, maternal negativity was generally at a low level (not only in control subjects), and no effects in association with depression remission occurred. This rare prevalence of negativity is in sharp contrast to Field et al. (1990), who found mothers to be negative (e.g., anger) 21% of the observed time. The data more closely resemble the study of Cohn et al. (1990) with a prevalence of 4%. It is noteworthy that Field studied a minority group (mothers were economically disadvantaged and black) whereas Cohn's sample had a higher socio-economical status with a large proportion of intact families and a high standard of school education, as did this sample.

Surprisingly, even after depression remission no recovery effect emerged in terms of an increase of maternal responsivity or that infants responded differently. Descriptively, depressed mothers and their infants responded in a comparable manner as control mothers did, even when specific types of behavior sequences were considered (e.g. positivity exchanges only).

Furthermore, there were no indications that previously restricted behavior ranges widen after depression remission. Neither mothers nor infants showed increases in their bandwidth of behaviors. Thus there was no support for the hypothesis that a restricted repertoire that is associated with depression widens after the major depression remits and there were no effects such as a regain of a normal speed of interaction (since speed of interaction did not change).

A range of further effects also did not supported the view that the timing (in terms of a parallelism of maternal depression and adverse child effects) might be crucial (Gunlicks and Weissman, 2008).

Contrary to depression-contagion approaches, there was no disappearance of rejection-inducing effects, nor were any changes in indicators of infants'

unwillingness to interact, or any recovery of formerly reciprocal negative affects observed.

Other approaches suggest that extensive engagement of depressed mothers is stressful, and that this should no longer be the case after remission. However, high engagement in depressed mothers was not associated with heightened infant negativity and there were no trends towards reduction in association with remission.

Even the ability to maintain interaction did not change following remission, contrary to the hypothesis of restricted parental resources that may no longer be restricted after remission; the same held true for contingent responsiveness of both the mother and the infant. Thus there were no indications of any recovery effects, both in interaction maintenance and responsivity in terms of response latencies. Mothers with major depression responded to infant affects in a comparable manner than control mothers did.

Finally, infants of formerly depressed mothers were predicted to recover in their self-regulatory capacities and to show normalized values (e.g. in latencies of consolability), to recover in their ability to interact synchronously and to increase their lowered levels of behavior predictability. Again, the data did not show any effects that were indicative of a recovery of regulation capacities in both interactants.

8.4. Possible explanations

There is a number of reasons that could explain why maternal major depression failed to be predictive for impaired interaction and why the remission was not associated with a recovery in interactive functioning.

Many of the positive predecessor studies used behavior ratings to quantify 'interaction' (see the compressed literature view in table 3, page 43, in particular studies 15-17, 19-23). In this light, it is interesting to see that a range of authors who used observational data and a sample selection via clinical interview failed to find impairing effects of depression (e.g. Stanley et al., 2004; page 11; Campbell et al., 1995; Cohn et al., 1991). The interaction scorings and self-ratings of depressed mood of previous studies possibly capture different properties (e.g., anxiety or highly potential third variables) than those captured in the present study: here interview-based depression diagnoses and observed interactions were obtained. The Field's study group (e.g. 1984; 1988; 1985; 1989; 1990), however, stressed - without any

exception - the predictive value of maternal self-rated depression. Its results add considerably to the meta-analysis of Beck (1995), who concluded that maternal depression may well be predictive for effects, which clearly - in the case of depression - demanded intervention. In accordance with the present research the effects of Beck's meta-analysis shrink remarkably if observable behavior is coded and only those individuals with a clinical depression diagnosis are included.

A further reason for the failure to show depression-related effects might be that the applied behavior codings are insensitive for distinguishing between dyads with and without major depression. If a major depression diagnosis does indeed have predictive value (which would render this study a false-negative), behaviors compared to ratings may be less sensitive indicators for impairments in association with maternal depression. However, behavior codings showed clear advantages such as low susceptibility to observer bias in case of known diagnosis, sufficient reliability (e.g. between two raters), sufficient re-test reliability (Tronick et al., 2003; Moore, Cohn and Campbell, 2001) and validity of behaviors (e.g. for affect with facial coding systems, Matias, Cohn and Ross, 1990).

A further reason may be found in the setting. The still-face procedure has rarely been used as a psychometric instrument; it has been rather employed as a research tool. Its predictive value for later infant outcomes has rarely been tested and is still under test (e.g. Cohn, Campbell and Ross, 1991; Kogan and Carter, 1996; Moore, Cohn and Campbell, 2001). To date, the authors have published no manual with standardized measures or reference tables of values for a sufficient population.

It is also possible that the observational features of this coding system, plus the chosen setting, are non-exhaustive for relevant dimensions of parenting, and that mothers have periods when they are in control of the situation and can coordinate both their own and their child's activities.

Moreover, there is the possibility of third variable effects. Third variable-effects in association with maternal depression are extensively discussed by Downey and Coyne (1990). Possibly, the effects of depression on interaction show up only in specific care-giving environments (e.g. at home in the presence of a non-supportive partner) or they only show up in specific populations. For example, there are positive studies that included only depressed mothers from socio-economically disadvantaged backgrounds (i.e. "high risk individuals"; e.g., Cohn et al., 1986; Field,

1984; Field et al., 1990, page 8; Goodman and Brumley, 1990), of which the latter two included disadvantaged black mothers with low incomes; most of them being single parents.

Other factors may be less prevalent in the present sample of low risk mothers, e.g., insensitive parenting, marital discord, lack of social support and socio-economic disadvantage. Downey and Coyne (1990) and Hops et al. (1987) stressed the importance of marital discord, e.g., for the amount of affective irritability displayed by a depressed mother when interacting with her child. Vice versa, the presence of social support or lack of marital discord may be suitable to lower adverse effects on mother-infant interaction.

Moreover, maternal depression has been shown to be associated with the presence of life stress, child-caring stress and marital dissatisfaction (e.g. the meta-analysis of Beck, 1996). Thus there is the possibility that maternal depression may not be the key determinant for impaired interaction and - accordingly - may not mediate later infant risks, yet only be indicative but non-triggering for these risks (Rutter, 1990).

A further explanation of why depression-related effects did not emerge may lie in the presence or absence (in the present sample) of specific patterns of depression-associated personality traits or personality disorders both of which are consistently reported in depressed individuals (e.g. Boyce and Mason, 1996; Bagby, Quilty and Ryder, 2008).

Personality disorders were found to be highly prevalent among individuals with major mood disorders: 35-70% of patients with depression were reported having a personality disorder (Corruble et al., 1996; Farabaugh et al., 2005; Fava et al., 2002). Vice versa two-thirds with personality disorders had comorbid depressive episodes (Michels, 2010; Morey et al., 2010).

According to Bagby et al. (2008) personality factors may constitute pre-depression vulnerability factors (see Maier et al., 1992), or remission predictors (Canuto et al., 2009), or, they may reflect the current state of the depressive symptomatology, i.e. may moderate or complicate the expression of the depression, may constitute the lower range of a depression-spectrum (e.g. a chronic attenuated state of the disorder), or, they may constitute a second indicator for a common cause of the depressive episode (beneath the depression diagnosis).

Thus, it seems possible for the present study that maladaptive personality traits (either preceding or concurrent ones) differ from those studies that reported depression-related effects on mother-infant interaction. The problem could either be in the recruitment of mothers with specific personality traits in the area of Heidelberg or the state the mothers were in when the videos were taken. Possibly, mothers who managed to call for help and were subsequently hospitalized had less dysfunctional beliefs (at the time of their measurement), less reduced self-esteem, less interpersonal dysfunctions and less neuroticism and rigidity as is usually reported for depressed individuals (Quilty et al., 2008; Carter et al., 1999; Maier et al., 1992). Possibly, they had less attenuation in attentional bias towards mood-congruent information as expected (Gallardo et al., 1999), vice versa, had heightened demands for accomplishment and control (Nietzel and Harris, 1990; Jones et al., 2010), and heightened conscientiousness (Kronmueller and Mundt, 2006) as usually reported. It seems also possibly that - contrary to the prediction - heightened perfectionism, self-criticism and heightened sensitivity to it (Bagby et al., 2008; Blatt et al., 1995) triggered the mothers with major depression to interact according to their knowledge of normal interactive behaviors. It is possible that the study had fewer mothers with an avoidant personality disorder than is usually reported (Ramklint and Ekselius, 2003; Klein, 1999; Alpert et al., 1997), i.e. the study-mothers possibly had lesser social inhibition or social dysfunction, lesser tendency to avoid interpersonal contact and feelings of inadequacy.

Moreover, as Michels (2010) pointed out the state-trait dilemma that has consistently been discussed in the literature regarding personality disorders in association with depression (Allen and Potkay, 1981, 1983; Zuckerman, 1983; Fridhandler, 1986) may be called on as explanation, e.g. trait-personality factors relevant for deviancies in dyadic interaction (e.g. heightened harm avoidance, or, lowered cooperativeness, lowered optimism and persistence, Hansenne and Bianchi, 2009) might have temporarily been covered by the current state, i.e. the depressive episode, when interaction was videotaped, and these effects might have nullified effects on dyadic interaction in this study.

Moreover, Newton-Howes et al. (2006) found that depression in the presence of personality disorders minimized the chances for depression-remission. In this study, however, the quick remission speed of some of the study participants may be

indicative of the absence of impeding personality factors, and, thus may favour normal behavior in mother-infant interactions. Accordingly, Peselow et al. (1994) reported that recovered patients had lower trait scores e.g. for paranoid, antisocial, avoidant or compulsive disorders. Thus the selection for a sample that later displayed a total depression-recovery could have led to the inclusion of a specific subpopulation which nullified the effects.

On the other hand, it is also possible, that interviews by experienced clinicians could have better been resistant to the influence of personality factors and other third-variable factors (which may have been responsible for some of the huge effects reported in the literature), whereas self-rating measures for depression on which a great part of the literature is based on might have been more prone to the influence of personality characteristics and third variable influences.

It is also possible that preceding studies reported the effects of personality co-factors in presence of maternal depression where any effects on mother-infant interactions were mediated by dysfunctional personality factors (Carter et al., 1999) and the maternal depression was sufficient but not necessary for the effects. An explanation that - again - well fits, Rutter's notion (1990) that maternal depression might have only indicator function for child risks.

It could be further possible that the chosen time window of behavior observation (3 intervals of 2 minutes) was too narrow to allow for a generalization; a critique also listed by Rutter (1990). However, this study is in part a replication of predecessor studies which reported medium-sized to large effects within that window. Thus, the window clearly allows for a detection of effects. Moreover, if there is indeed an inverse relationship between the width of the sampling window and generalizability, then other easily obtainable measures, such as a self-report measure, would also have a very low generalizability (which is not the case for most clinical measures; for example, the Beck depression inventory can be completed within minutes, but this short period does not usually affect the generalizability of its content).

It is also possible that the type of depression or length of exposure is the relevant determinant. For instance, there are reports of deviant mother-infant interactions in low risk populations when the maternal depression is chronic, i.e. has lasted up to 6 months (e.g. Campbell, Cohn and Meyers, 1995; Murray, Fiori-Cowley and Hooper, 1996).

Biasing effects from being videotaped are also possible as factors with a risk of nullifying depression-related effects; e.g., in the sense that mothers guessed the study hypotheses (e.g. observations made in association with own depression diagnosis) and this affected their behavior. For example, Lundy et al. (1996) and Field (1992) reported a “faking feeling good syndrome” in depressed mothers when they were being observed. Nevertheless, this is sharply contrasted with many video-based studies that observed impairments in mothers with depression (e.g. Field, 1984; Stein et al., 1991; Stanley, Murray and Stein, 2004) and the settings of these studies (the still-face situation) have been fully replicated in this study.

It is also possible that mothers were already in unexpected remission after they were admitted to the hospital, plus, they received immediate support by physicians and staff. However, this idea brings the validity of the clinical interview and the delayed latency when a major depression usually remits into question. Wisner et al. (2006) found that remission in postpartum depressed mothers occurs after 4-8 weeks (25-75% cumulative remission rate, see also Noorlander et al., 2008), even under medication of proven effectiveness in randomized trials. Thus in this light it does not seem highly probable that the inclusion of mothers in an episode of depression is contaminated by a subsample with characteristics of an early remission.

Finally, it is possible that depression-related effects (e.g. with respect to increased risks of psychopathology in children of depressed mothers) derive from specific behaviors that have a low chance of being observed in front of a camera; e.g., physical maltreatment (Buist, 1998) or other maladaptive parenting behaviors such as harsh punishment or inconsistent enforcement rules. Johnson et al. (2001) found these maladaptive parenting behaviors (not the diagnosis per se) sufficiently predictive for the risk of child psychopathology, i.e. a prediction was possible irrespective of the parental psychiatric disorder (the latter, however, being well suitable to foster those maladaptive practices).

8.5. Limitations of the study

The present study clearly needs replication. It has major limitations, the most significant being the low availability of confounders and thus the lack of sufficient control of potential biasing factors (e.g. levels of sleep behavior of both mother and infant before assessment, or, more details about the socio-economic background, available support for the mother, etc.). Although it was shown to have no effect, a

certain limitation is the attrition of sample size for the follow-up data. This study is also limited because many mothers were already admitted to hospital and some were in treatment when the study began (nevertheless the medication may take some time to work, e.g. 4 - 8 weeks). Additionally, more information is needed with respect to the relevance of observational interaction data within the chosen window of assessment, i.e. behavior samples over a short period of several minutes. Moreover, the study results can be generalized only to a group of major depressed mothers who are predominantly urban and have a higher level of education. In light of these considerations, the findings presented here should be considered preliminary. The small samples, however, allowed for the detection of existing depression-associated impairments only in very optimistic conditions and thus erroneously inflated expectations with a sufficient statistical power. The power turned out to be insufficient under classical conditions in terms of Cohen's $D = 0.8$ or $D = 0.5$.

8.6. Strength of the study

The present study had a number of important strengths. First, it was a full replication of a wide range of predecessor studies with, however, an added follow-up measurement after the depression remission. The mothers were videotaped "in-episode" of major depression, i.e. the study only allowed for a very narrow gap between depression-interview and data sampling (in most cases at the same day, previous studies neglected this proximity between diagnosis and behavior observation). Moreover, the study applied a controlled, parallel group design: Interactions of dyads with a major depressed mother were contrasted with those of a non-depressed, completely healthy control group and a follow-up for both groups. The study applied rigorously defined measures based on observation only and in order to quantify mother-infant interaction (in the sense of "actions"). Third, maternal depression was based on a standard clinical interview, i.e. on a clinical diagnosis, not on mood or symptom self-ratings in brief questionnaires. Moreover, following the advice of Rutter (2005), this study did not rely on the same informant for measurement of both the independent (maternal depression) and dependent variable (behavior) which he suspects as one reason for inflated effects in the literature. In this study, both the depression diagnosis (interview) and behavior (independent raters unaware of the maternal diagnosis) had independent observers as informants. Furthermore, the study applied three different methods to control for potential

confounders and covariates (the analysis of covariance, the propensity score method, and a correction method for selection bias). In particular, infant age and gender, maternal education, and maternal age were controlled for and none of these methods allowed for an increased differentiation between dyads with and without a depressed mother.

8.7. What this study adds

This study indicates the possibility that major depression in mothers may not be a key predictor for deviancies in observable mother-infant interactions. There was no association between mother-infant interactions and the remission from depression. It raises the possibility that maternal emotional unavailability of a mother with major depression does not necessarily generalize to her parenting behaviors (e.g. by being incorporated) or that the effects of adverse parenting behaviors may not necessarily be transmitted to the infant. It has often been claimed that depressed mothers interact in less optimal ways, but these effects appeared either in rating data of dyadic interaction or when the maternal depression was derived by self-ratings. This study failed to find any effect on the disadvantage of depressed mothers or their infants or any improving (or worsening) effects after the remission of the mother's major depression. This may point to the relevance of other factors that are associated with maternal depression or for which major depression is indicative (possibly for socio-economically disadvantaged or non-supportive environments).

8.8. Clinical and research implications

To draw final conclusions for clinical practice a replication of this study is needed. Clinically, a parental diagnosis of major depression has been suggested to generally alert clinicians to potential risks for children, e.g. depression (Verdeli et al., 2004; page 56) or a generally higher risk of child psychopathology (Beardslee et al., 1998). If the low predictive value of maternal major depression can be replicated, then the effects of being socialized in early infancy with a maternal depressed parent may be weaker than usually predicted.

The present research raises possibility that maternal depression has an indicator function and is only predictive in combination with other factors, such as maladaptive parenting or social and socio-economic adversities. As Rutter (1990) suggests, it is also possible that offspring of depressed individuals may be less likely to be affected

if the parental disorder has a short duration, if the disorder is not associated with family discord and disorganization, and if it is not connected to impaired parenting.

The results, if they are generalizable and replicable, eventually narrow chances that patterns of mother-infant interaction have mediating capabilities, since according to Baron and Kenny (1986) a potential mediator (here: parameters of dyadic interaction) needs to correlate with both the initial (maternal depression) and with the final condition (long-term results, e.g., impaired child performance or internalizing or externalizing behaviors). Moreover, a correlation between initial (major depression) and final conditions (long-term infant behavior) is required. In this study, maternal major depression failed to correlate with dyadic interaction. Thus the conclusion seems warranted – given the replicability of this study – that dyadic interaction may not act as suitable mediator as suggested (Murray, 1997; among other factors such as genetics). Assuming that the results of this study are replicable and given that the mother constitutes the major environment for an infant, the assumption of an environmentally driven mediation of effects from a parental disorder on children is clearly weakened.

Moreover, indicators of dyadic interaction might not be sensitive indicators to changes, e.g. regarding responses to depression treatments. This is a possible explanation of why studies, e.g., for interpersonal therapy or other streams of research, were not able to show that the treatment for affective depression is predictive for the developing mother-child relationship (e.g. Forman et al., 2007; or Murray et al., 2003) .

Although the data presented here is the first one of this type (in particular data after the depression remission), the research implications with respect to the feasibility of this study indicate a negative cost-benefit ratio. The recruitment of major depressed mothers from the mother-infant ward of the Heidelberg clinic, two invitations to the laboratory, filming with two cameras and a split-screen technique, double-coding of the interaction data second-by-second, for both mother and infant separately, and the statistical analysis of the huge data set of more than 25,000 records produced by the coding system for 59 mother-infant pairs (i.e. 420 records per dyad at average) has turned out to be very time-consuming. In clinical practice observer-coding procedures of the present type will be feasible in only a very limited fashion.

8.9. Recommendations for future research

Future studies should consider the possibility that infants of depressed mothers may behave comparably or even equivalent to infants of depression-free control mothers. However, if there are, in truth, behavior deviancies in infants of depressed mothers, and this study is a false-negative one, ratings (compared with coded behaviors) might be easier to obtain and more sensitive in terms of a contrast-detection according to literature. Based on the notion of Rutter (1990) that indicators used in mother-infant research are well heterogeneous, a clear definition of presumably risk-predictive indicators should be derived from theories with a high degree of falsifiability (or refutability; note that falsifiability does not imply 'false', Popper, 1959); for example, indicators that intersect with relevant and predefined dimensions of parenting. Moreover, the applicability of the still-face paradigm may be questioned because psychometric criteria, retest reliability and concurrent and discriminant validity (of the paradigm itself) are not sufficiently available, even though they are urgently requested (e.g. Rutter, 1990; Stanley et al., 2004). Stanley also critically pointed to the setting in which infant deviations are observed: almost all cited theories in this research area do not include measurements of the test situation, i.e. they treat the observation situation as irrelevant for predictions. For future research, different settings (e.g., different standardized situations in the laboratory and different non-standardized at-home settings) should be used together with a few single but different parameters; e.g., behavior ratings, observational data and maternal self-ratings. Other factors should be included as well; for example, a co-morbidity with the depression diagnosis (e.g. generalized anxiety).

8.10. Abstract

A detrimental effect of maternal depression on the way mother and infant interact is consistently reported, yet there is a clear shortage of rigorous laboratory studies focusing on major depression and the effects after remission. Detrimental effects of parental depression on offspring have been summarized under terms such as "intergenerational transmission" or "depression runs in families". Although there is a consensus that depression is partly hereditary, a main focus on genetic pathways and a neglect of parental behaviors have been criticized for the inconsistency of heritability estimates and the non-specificity of adverse child-effects such as heightened externalization behaviors being consistently reported in children of

depressed parents. Moreover, genetic components have been suggested to be less effective if individuals are younger and factors other than being exposed to maternal behaviors may be less directly effective on the infant. In the last decade, a range of publications has suggested that dysfunctional interactive patterns of mother and infant may have mediator functions in how maternal depression affects infants in terms of adverse child outcomes. The present research pursued two basic models as to why maternal major depression may be predictive for detrimental effects. Both models focus on disturbances of affect exchanges. First, Gergely and Watson's (1996) model of "maternal affect mirroring as social biofeedback" proposes the expression of maternal affect has the quality of an infant-regulation mechanism. Accordingly, flat affect of a depressed mother is suggested to have disruptive quality on these processes and decreased affect-mirroring behaviors (in terms of lesser positive affect expressions in parallelism to infant affects) may increase the risk of a failed regulation or controllability of infant affects. Secondly, impaired-parenting approaches predict that a maternal major depression will restrict these resources and therefore change the reinforcement conditions for the infant. Conversely, transient child disturbance approaches suggest that adverse infant effects and dysfunctional mother-infant interactions will disappear after the remission of maternal major depression. Based on predictions such as those above, a prospective, highly standardized, observer-blind, controlled laboratory trial with repeated measurement was conducted: 59 mothers in total, 24 with a clinical diagnosis of major depression, together with their infants and 35 control dyads were videotaped during face-to-face play interactions in the laboratory. A power analysis revealed that only under very optimistic and probably inflated conditions these samples were sufficient to show the expected differences in the primary target variables (the power turned out to be insufficient under classical conditions in terms of Cohen's $D = 0.8$ or $D = 0.5$). Thus, the results of this study might have descriptive status only. In the laboratory the mothers were instructed to interact with the infant as they "would normally do at home" and were assessed twice: a) when the mother was in an episode of a major depression and still being in the "2-or-more-weeks" duration criterion for major depression and had already been admitted to the mother-infant ward of the Heidelberg University clinic (at a median of less than 1 day between diagnosis and videotaping) and b) re-assessed after the remission of the depression (17 of 24; 71%), at a median of 2 months. Control mothers were re-assessed after a

comparable period of time (25 of 35; 71%). Sample size attrition had no effect on primary or secondary parameters. Diagnoses strictly followed the criteria for major depression of the Diagnostic and Statistical Manual of Mental Disorder IV. Affect-related behaviors were coded during a “still-face” procedure (two free interaction phases interrupted by a phase of maternal “still-face”; i.e., a period of minimized maternal affect expressions). The results of the confirmative, 1st-type error-adjusted, comparisons showed the following: There was no depression-related impairment of affect mirroring (in terms of parallel occurring affect exchanges of mother and infant). Contrary to the literature, infants with a depressed mother were not differently affected by the short still-face period, i.e. by maternal unavailability compared with controls. Moreover, depressed mothers did not behave in an under-stimulating manner and their infants were neither hypo-active nor hyper-active. The ability to maintain interaction, and to respond was not decreased in mothers with major depression, as suggested by parenting impairment approaches. Contingent responses in their infants were not lower. After a full remission from depression, there were no changes in the level of affect mirroring. The remission from depression was not associated with changes in difficult infant behavior such as protest and withdrawal. Despite predictions of alternative approaches on depression-related effects, infants of depressed mothers did not show signs of rejection, or unwillingness to interact, or spirals of negativity; i.e., aspects predicted by depression-contagion approaches (as suggested by interpersonal models with depressiogenic effects on the interactants). Even high engagement of depressed mothers was not associated with heightened infant negativity, as predicted by interpersonal stress approaches. Infants of depressed mothers did not show less adjustability or controllability, as suggested by regulation approaches. The dyadic capacity to interact synchronously, the amount of behavior predictability and the degree of responsiveness in target dyads were not lower in comparison with controls and there were no remission-related changes. Thus, there was no sign that a clinical diagnosis of major depression has any predictive value with respect to observable affect-related behaviors, either in mothers or infants. Beyond that, a combined multivariate measure showed that they are significantly equivalent compared to controls. Moreover, there were no depression-specific changes after a remission: Maternal major depression did not appear to be predictive for failures in affect-mirroring or for impaired parenting behavior. Predictions of alternative approaches also showed no

reflections in the data, e.g. that maternal depression might be contagious, or might drive the dyad into spirals of negativity, or that maternal engagement of a depressed mother might be stressful. In fact, every theory-derived prediction was falsified by the data. The lack of predictability of a maternal major depression diagnosis is surprising, given the large range of publications concluding the well-predictive properties of depression (although self-rated). The low predictive value is discussed with respect to Rutter' (1990) notion that depression may not have causal but indicative function and the resilience in infants of depressed mothers may be considerable. The results open up the possibility that maternal major depression may not generalize on behavior towards an infant and may thus not be reflected in or transmitted to infant behavior.

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11. Availability of anonymized raw data and programming routines

Anonymized raw data and programming routines (SPSS syntax routines) are available by the author.

12. Ehrenwörtliche Erklärung

Ich erkläre: Ich habe die vorgelegte Dissertation selbständig und ohne unerlaubte fremde Hilfe und nur mit den Hilfen angefertigt, die ich in der Dissertation angegeben habe. Alle Textstellen, die wörtlich oder sinngemäß aus veröffentlichten Schriften entnommen sind, und alle Angaben, die auf mündlichen Auskünften beruhen, sind als solche kenntlich gemacht.

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