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Maternal Mental Illness, Mother-Infant Interactions and Maternal Cognitive Functioning

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General Abstract

The influence of maternal mental illness on children's physical, cognitive and emotional development is an area of concern to researchers and health professionals. The literature review considers the issue of parenting by mothers diagnosed with a mental illness, with particular reference to the impact of maternal mental illness on the quality of mother-infant interactions. Studies of mothers diagnosed with depression or schizophrenia have found important differences in the quality of their interactions, as compared to mothers with no psychiatric history (Cohn, Campbell, Matias, & Hopkins, 1990, Murray, Fiori-Cowley, Hooper, & Cooper, 1996, Riordan, Appleby, & Faragher, 1999). The review proposes that these differences may be at least partly explained by the cognitive impairments associated with serious mental illness.

The empirical paper reports an exploratory study examining the relationship between maternal mental illness, the quality of mother-infant interactions and maternal cognitive functioning. The study replicates previous research by demonstrating that mothers with mental illness are significantly less sensitive when interacting with their infants, as compared to mothers with no mental illness. Poor cognitive functioning in mothers with mental illness was demonstrated, but only on Speed of Memory Processing. The presence of maternal mental illness was found to be a significant predictor of maternal sensitivity. When cognition (Speed of Memory Processing) was taken into account, the strength of this relationship was reduced, suggesting that the relationship between maternal mental illness and mother-infant interaction may be mediated by level of cognitive function.

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Literature Review*

Mother-infant Interaction in Mothers with Mental Illness and its Relationship to Cognitive Function

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Abstract

The impact of maternal mental illness on children's physical, cognitive and emotional development has been of interest to many authors, with concerning findings being documented (Cleaver, Unell, & Aldgate, 1999, Goodman & Brumley, 1990, Henriksson & McNeil, 2004, Tronick, 1989). This paper will review the literature, with particular reference to the impact of maternal mental illness on the quality of mother-infant interactions. Important differences in these interactions have been observed in comparative studies of mothers with and without mental illness (Cohn, Campbell, Matias, & Hopkins, 1990, Murray, Fiori-Cowley, Hooper, & Cooper, 1996, Riordan, Appleby, & Faragher, 1999). However, the mechanisms underlying these differences are poorly understood.

This paper proposes that poorer quality mother-infant interactions in cases where the mother has a mental illness may in part be mediated by the mother's cognitive functioning. A variety of cognitive impairments have been demonstrated in adults with mental illness (Addington, Saeedi, & Addington, 2005, Fossati et al., 1999, Porter et al., 2003). This literature will be discussed with reference to its methodological limitations. The relationship between maternal mental illness, mother-infant interactions and maternal cognitive functioning is proposed as an avenue for future research.

Introduction

This paper considers the issue of parenting by mothers diagnosed with a mental illness, with particular reference to early mother-infant interactions. The importance of these interactions for infant development will be discussed, and literature describing the quality of mother-infant interactions when the mother has been diagnosed with depression or schizophrenia will be reviewed. In particular, it is hypothesised that maternal cognitive functioning may influence the quality of mother-infant interaction in mothers with mental illnesses. This will be proposed as an avenue for future research.

Defining Mental Illness

The term "mental illness", rather than alternatives such as "mental health problems" or "mental disorder" will be used throughout this review for consistency with the published literature. Mental illnesses are generally diagnosed both clinically and for research purposes using DSM-IV (American Psychiatric Association, 1994) or ICD-10 (World Health Organisation, 1992) criteria. This medical model of diagnostic psychiatry has been criticised (e.g. Bentall, 1990, Horwitz, 2002). However, these diagnostic terms will be used within this review.

Schizophrenia

Schizophrenia is a relatively common severe mental illness, affecting approximately 1% of the population, with an annual incidence of about 10-15 per 100 000 people (Turner, 1998). Characteristic symptoms include delusions, hallucinations,

disorganised speech and behaviour, and so-called negative symptoms such as flattened mood and avolition (American Psychiatric Association, 1994). For a diagnosis of schizophrenia under DSM-IV criteria, signs of disturbance must be present for at least 6 months, including 1 month of symptoms, and may include prodromal or residual symptoms (American Psychiatric Association, 1994). Schizophrenia is often chronic and persistent, with periods of remission and relapse.

The diagnostic category of schizophrenia is heterogeneous. Within DSM-IV there are many sub-divisions, including "paranoid type", "disorganised type", and "catatonic type" (American Psychiatric Association, 1994). Patients only need display two or more of the key symptoms for diagnosis, therefore there will be differing presentations within a group of patients diagnosed with schizophrenia. Some authors have criticised the concept of schizophrenia as a discreet disease entity. Bentall (1990) questions the reliability and validity of schizophrenia as a scientific concept, citing evidence that patients can often receive different diagnoses across the course of their illness, when seen by different professionals, and that the nature of symptoms, severity, duration, course and response to medication varies considerably from one patient to another; emphasising the heterogeneity of schizophrenia and questioning the predictive validity of the diagnosis. Bentall has therefore argued for a symptom-based approach; we should be seeking to understand and treat individual psychotic symptoms instead of studying a syndrome that exhibits questionable validity and reliability. Others suggest that different subtypes of schizophrenia with differing symptom profiles and cognitive functioning may be related to different patterns of neurological impairment (Pantelis and Brewer (1995). Further research may enable the subdivision of schizophrenia, not only according to

overt symptoms but also according to the pattern of neurological impairment and its relationship to cognitive, social and emotional functioning. Alternatively, the interactions between genetic and environmental factors thought to give rise to schizophrenia may prove so unique to the individual that attempts to further categorise patients with schizophrenia may always be confounded by heterogeneity. A symptom- based approach, as advocated by Bentall, may therefore offer a useful framework for understanding the relationship between psychotic symptoms and cognition. Some researchers have adopted this approach, by investigating the links between specific symptoms such as thought disorder, and aspects of cognition such as verbal learning and verbal memory (e.g. Subotnik et al., 2006). However, this literature review takes a more traditional approach, referring to patients diagnosed with "schizophrenia" in accordance with the majority of the published literature on both cognitive function and mother-infant interactions.

Depression

Depression is common, but often under-reported (Hale, 1998), making true prevalence difficult to gauge. It is estimated that 1 in 5 women and 1 in 10 men will develop a clinically significant depressive episode during their lifetime (Blehar & Oren, 1995), with the prevalence of postnatal depression ranging from 8-15% (O'Hara, 1997). Symptoms include persistent low mood, diminished interest or pleasure in activities, weight loss, sleep disturbance, fatigue and poor concentration or decision-making (American Psychiatric Association, 1994). For a diagnosis of Major Depressive Disorder, an individual must have experienced at least one depressive episode, with no evidence of mania or hypomania (American Psychiatric

Association, 1994). Depressive illness ranges from mild to severe, and may represent a single episode or a recurrent condition.

Comorbidity

It is important to note the issue of comorbidity with respect to mental illnesses. For example, there is a high prevalence of depression amongst patients diagnosed with schizophrenia (Sim, Mahendran, Siris, Heckers & Chong, 2004), with estimates ranging from 7% to 75%. High levels of comorbid anxiety disorders have also been demonstrated in patients diagnosed with depression (Zimmerman, Chelminski, Zizook, & Ginsberg, 2006). Comorbid anxiety and depression is associated with a more chronic course of depression, and may influence the type of medication prescribed (Zimmerman et al. 2006) because some medications are considered effect in reducing anxiety as well as improving depressive symptoms. Comorbid depression in schizophrenia has been associated with poorer quality of life (Sim, et al. 2004), particularly regarding measures of physical and psychological health. Comorbid mental illnesses may therefore result in a poorer prognosis, and may influence the type of treatment offered by professionals.

This literature review focuses on depression and schizophrenia over other mental illnesses, reflecting the published literature. Additional discussion of other mental illnesses is beyond the scope of this review. However, the high prevalence of depression amongst individuals diagnosed with schizophrenia means that any consideration of the latter would be advised to include the former, so as to provide a more complete picture of the potential difficulties these patients may experience.

An Overview of Parenting by Mothers with Mental Illness

Children whose parents are diagnosed with a mental illness have been studied for several reasons. Researchers investigating potential genetic components to mental illnesses have frequently studied such children. For example, the desire to identify early indicators and risk factors for schizophrenia has prompted the study of children whose parents have that diagnosis (e.g. Henriksson & McNeil, 2004). Concerns have also been expressed regarding the impact of parental mental illness on parenting capacity. Parenting skills may be affected, and in extreme cases children's physical needs may be neglected, and they may experience adverse social situations such as poverty (Cleaver, Unell, & Aldgate, 1999). Another area of research focuses on the impact of parental mental illness on children's emotional, social and cognitive development, and the mechanisms through which this occurs. The majority of published research focuses on mothers, and this bias is reflected here. However, it is important not to disregard the role of fathers in infant development, or to overly criticise mothers. Some authors have cautioned against the potential "mother-bashing" tone of this research (Downey & Coyne, 1990, p. 50).

Notwithstanding these concerns, the developmental, social, physical and mental health outcomes for children of mothers with mental illness have been documented. For example, Henriksson and McNeil (2004) studied 84 children born to mothers with a diagnosis of schizophrenia or affective psychosis, from birth until they were 4 years old. Children were scored according to their attainment of developmental milestones, sensory difficulties, language disorders, need for medical treatment, malformations, biological dysfunctions, and the degree to which they demonstrated

disturbed behaviour. Compared to a control group of children whose mothers had no psychiatric history, children of mothers with schizophrenia showed significantly increased rates of delayed walking, visual impairment, language disorders, enuresis and disturbed behaviour (Henriksson & McNeil, 2004). Children of mothers with affective psychosis also demonstrated increased incidence of delayed walking, but no other abnormality. It is not clear whether these observations represent evidence for genetic risk factors for schizophrenia or the influence of social and/or environmental factors on child development. A high risk of psychiatric disorders has also been recorded in children whose parents experience mental illness. Estimates of prevalence vary, but rates of psychiatric disturbance in children of parents with unipolar depression exceed 40% (Gotlib & Goodman, 2002). Problems include depression, anxiety, conduct disorder, and alcohol and drug dependency (Gotlib & Goodman, 2002). The authors highlight the relatively weak genetic component in unipolar depression. For example, in a meta-analysis of twin studies of depression, genetics accounted for 36% of the variance (Sullivan, Neale, & Kendler, 2000). This provides evidence for the role of environmental influences in the development of these difficulties. It should be noted that heritability influences include gene-environment interaction as well as purely genetic effects (Bebbington, 2004).

Rutter & Quinton (1984) proposed four sets of mechanisms through which parental mental illness may impact upon children. A direct biological explanation is potential genetic transmission of mental illness itself. Secondly, environmental factors arising from poor parental mental health, such as neglect or abuse may influence child development. Thirdly, the mental illness may have indirect effects, through disruption of parenting and parent-child relationships. Finally, parental mental

illness may be associated with marital difficulties, unemployment, and poverty, and this may impact upon the child's welfare. The author's own research offers some support for this model. They studied families of psychiatric patients over a 4-year period and identified significantly higher rates of marital discord and previous marital failure compared to a control sample (Rutter & Quinton, 1984). At the initial assessment, marital discord was associated with child behavioural disturbance (as rated by the child's teacher) in boys but not in girls. Persistent marital discord over the 4-year follow-up was associated with continual behavioural disturbance in boys, and the development of such disturbance in girls. The authors concluded that boys were more immediately affected by marital discord, whereas girls were only affected if discord persisted (Rutter & Quinton, 1984).

More recently, Goodman and Gotlib (1999) proposed a model for understanding the mechanisms through which children of mothers with depression come to be at risk of abnormal development. They also proposed four mechanisms: heritability of depression, innate dysfunctional neuroregulatory mechanisms, exposure to the mother's negative/maladaptive cognitions, behaviours and affect, and exposure to a stressful environment (Goodman & Gotlib, 1999). This model is similar to Rutter and Quinton's (1984) in highlighting the influence of stressful child-rearing environments associated with maternal depression, as well as the role of genetics. Downey and Coyne (1990) cautioned against attributing children's problems directly to maternal depression, an alternative hypothesis being that both are caused by pre-existing conditions such as a stressful family environment. They highlighted the difficulty in examining the interaction between maternal depression, contextual influences, and child outcomes. Researchers have attempted to evaluate the relative

contributions of stressful circumstances and depression to specific child outcomes. For example, Murray, Fiori-Cowley, Hooper, and Cooper (1996) examined the relative contributions of maternal depression and adverse life events to the quality of mother-infant interactions. Mothers were videoed interacting with their 2-month old infants, and scored for sensitivity to their infant, intrusive behaviour, affirming and negating behaviour. Using multiple regression analysis, they found that both depression and adversity had a significant effect on maternal sensitivity, and maternal affirming and negating behaviour. Once adversity had been controlled for, depression continued to have a significant impact on maternal sensitivity and negating behaviour (Murray et al., 1996).

Goodman and Brumley (1990) argued that parenting practice is the major determination for the way in which maternal mental illness influences child development. They studied 53 mothers with schizophrenia, 25 with depression and 23 mothers with no psychiatric history, to investigate the relationship between quality of mother-infant interaction and child social and intellectual development using the Home Observation for the Measurement of the Environment inventory (HOME, Bradley & Caldwell, 1979), the Bayley Scales of Infant Development (Bayley, 1969, as cited in Goodman & Brumley, 1990), and videoed mother-infant interactions. The children were aged between 3 months and 5 years. Quality of the childrearing environment (measured by the HOME) was significantly poorer for mothers with schizophrenia compared to the control group. Mothers with depression obtained interim scores. During interactions with their infants, mothers with schizophrenia were rated as displaying significantly less affectional involvement and responsiveness than the control group, with the mothers with depression again

obtaining interim scores (Goodman & Brumley, 1990). Moreover, maternal affectional involvement and responsiveness was the only variable found to have a significant effect on child IQ ($p < .02$, $R^2 = .06$) (Goodman & Brumley, 1990). Hierarchical regression analyses showed that maternal diagnosis itself did not account for a significant proportion of the variance in child outcome, but parenting variables (measured by the HOME inventory) did. For intellectual development, the significant variables were maternal "avoidance of punishment and discipline", and the quality of the environment. For social development, the significant variable was "maternal responsiveness". It was the mother's interactions with her child that were important in determining outcome, irrespective of diagnosis.

Mother-Infant Interactions

For the first 3-4 months of life, an infant's contact with their caregiver occurs mainly through face-to-face interactions, at times of feeding, dressing and play (Vasta, Haith, & Miller, 1999). These early interactions are considered essential for the infant's social, cognitive and emotional development (e.g. Murray et al., 1996, Tronick, 1989), as well as playing a vital role in the formation of an attachment bond between the infant and its caregiver (Belsky & Isabella, 1988).

Studies of early interactions focus on the mother as the main caregiver. Typically, mother-infant interactions are captured using video cameras and subjected to detailed analysis, often using coding systems. One procedure involves the mother and infant sitting opposite each other, with the mother instructed to play with her baby in her usual manner. One video camera records the mother's face and another one records

the infant's (Vasta et al., 1999). An alternative procedure positions the camera behind the mother to record the infant, while her face is reflected in a mirror positioned adjacent to the infant (Murray, Cooper, Wilson, & Romaniuk, 2003). Importantly, both procedures allow for simultaneous recording of both mother and infant facial expressions.

Using these procedures, several important features of mother-infant interaction have been revealed. Newborn babies display a behaviour cycle consisting of movement between states of interest and attention, and states of inattention and avoidance (Vasta et al., 1999). Mothers appear to recognise this cycle and adjust their behaviour accordingly, concentrating their interactive behaviour (such as talking and smiling) during their infant's attentive state (Vasta et al., 1999). When the infant moves into a state of inattention, the mother's interactive behaviours reduce (Vasta et al., 1999). This behaviour-state matching (Field, Healy, Goldstein, & Guthertz, 1990) establishes a pattern of interaction between mother and infant in which they are both "on" or "off" at the same time, referred to as interactional synchrony (Kaye, 1982).

These interactions are believed to form the basis of an effective communication system between mother and infant (Vasta et al., 1999). The infant is actively communicating, for example through facial expressions and gestures (Tronick, 1989), and the mother interprets these messages and responds accordingly. Another important feature of these interactions is turn-taking (Vasta et al., 1999), where the mother waits for a response from her infant before "answering", for example by

imitating the infant's behaviour. This turn-taking may represent early conversational dialogues between mother and infant (Vasta et al., 1999).

Early mother-infant interactions are also thought to be important for the development of emotion regulation. Tronick (1989) describes an example of an infant looking away from its mother and sucking its thumb. This may communicate to the mother that her infant needs to calm down and disengage from the interaction for a short period. The mother then waits for the infant to look back at her before continuing with their interaction (Tronick, 1989). By taking such action, the infant may be able to learn to regulate their own internal state. This looking away behaviour has been shown to reduce infant heart rate during stress, and thumb-sucking can calm infants (Tronick, 1989). In summary, infants develop a range of self-directed behaviours, enabling them to manage emotions and distress, and to regulate their emotional state (Tronick, 1989).

Mother-infant interactions have been disrupted for research purposes using the "still face" (Cohn & Tronick, 1983) or "blank face" technique (Murray, 1986). Mothers are instructed to interact with their infant as usual, but when signalled switch to an unresponsive state, displaying no facial expression. Infants aged 3 months initially attempt to gain their mother's attention, but when this fails they display negative emotions and use self-regulatory behaviours (Tronick, 1989). Murray used two techniques to disrupt mother-infant interaction, creating an "interruption" condition and the "blank face" condition. In the "interruption" condition, mother and infant were interacting as usual, when another adult entered the room and engaged the mother in conversation for 30 seconds, during which time the mother turned away

from her infant. In the "blank face" condition, the mother was instructed to cease interacting and sit still and expressionless for 45 seconds, whilst looking at her infant. During periods of normal interaction, infants directed their gaze at their mother, smiled, made active mouth movements and displayed relaxed or raised eyebrows. During the interruption condition, the infant's communicative behaviours (e.g. smiles) reduced but there were no signs of distress, or displacement activity. However, during the blank face condition the infant appeared disturbed, and communicative behaviours were sustained, even intensified. There was also an increase in frowning, grimacing, and displacement activities such as lip-biting. Murray refers to this as "protest" behaviour (Murray, 1986, p. 172). This was followed by apparent withdrawal, with the infant looking away from the mother, and drooping their head, but briefly glancing back at their mother's face. There were also more expressions of negative affect. Infants appear disturbed and distressed by the lack of responsiveness and emotional expression displayed by their mothers.

In a second set of studies reported in the same volume, Murray and colleagues disrupted the synchronicity of mother-infant interactions (Murray, 1986). The mother's responses were relayed to the infant via a life-size video screen, with a 30 seconds delay. The infant's reaction was similar to the protest phase detailed above, but seemed more like confusion, rather than the distress seen in the blank face condition (Murray, 1986). This demonstrates the infant's sensitivity to the timing and contingency of the mother's response, as well as the quality of the interaction.

Mother-child interactions have been studied widely in the context of attachment theory (Bowlby, 1982). Bowlby (1969) believed that human attachment behaviour

has a biological basis, with mothers genetically predisposed to respond to infant signals, such as crying, smiling and grasping. Attachment refers to the bond between an infant and its caregiver (Ainsworth, Blehar, Waters, & Wall, 1978), usually considered to be the mother within attachment theory and research. The bond is formed and mediated through attachment behaviours, organised within a behavioural system (Marrone, 2000). When this system is activated, for example at times of distress the infant utilises attachment behaviours to increase proximity to their caregiver, and to attain comfort and assistance (Marrone, 2000).

The quality of the maternal response to the child has implications for the quality of attachment formed; that is the extent to which the child experiences the mother as emotionally available, safe and protecting (Lieberman & Pawl, 1988). Attachment theorists believe that one of the most important determinants in an infant's developmental pathway is the responsiveness of the caregiver (Marrone, 2000). Sensitive responsiveness includes recognising and responding to infant signals, facilitating achievement of the infant's goals, and supporting the achievement of a positive state (Marrone, 2000, Tronick, 1989). Tronick argues that through poorly coordinated interactions featuring intrusive or poorly timed maternal responses, infants develop a representation of themselves as ineffective, experience failure and negative affect, and their caregiver as unreliable and untrustworthy. This has implications for the mother-infant attachment relationship, and for the child's internal working models. These are mental representations of the self and caregivers, their communicative behaviour and interactions (Marrone, 2000), cognitive structures that influence the organisation of subjective experience and adaptive behaviour (Marrone, 2000). Disturbances in attachment have also been

shown to be a risk factor for later development (Green & Goldwyn, 2002) and for psychopathology in adulthood (Marrone, 2000).

Maternal Depression and Mother-Infant Interactions

Over the last 20 years, a number of studies have examined mother-infant interactions in cases where the mother is experiencing depression (Cohn, Campbell, Matias, & Hopkins, 1990, Field et al., 1990, Goodman & Brumley, 1990, Murray et al., 1996). It has been hypothesised that the sadness and withdrawal, which are characteristic of depression, may result in lower levels of positive affect expressed towards the infant and less contingent maternal responsiveness (Campbell, Cohn & Meyers, 1995). Both factors are important for the development of dyadic communication and affect regulation (Tronick, 1989), and for the development of a secure attachment relationship (Belsky & Isabella, 1988). Irritability and agitation associated with depression may also increase the likelihood of maternal negative affect, which is usually rare in mother-infant interactions (Downey & Coyne, 1990).

There is empirical support for these hypotheses. For example, Cohn et al., (1990) found that mothers with depression displayed more negative affect when interacting with their 2-month old infants, as compared to non-depressed mothers. They also displayed lower levels of positive affect, as did their infants. Murray and colleagues (1996) examined the quality of mother-infant interactions in 58 mothers with post-natal depression, as compared to 42 mothers with no symptoms or history of depression. Their infants were aged 2 months. They used a video recording procedure with a camera directed at the baby's face, and a mirror reflecting the

mother's face for the camera (Murray et al., 1996). As predicted, mothers with depression were significantly less sensitive to their infant's cues compared to non-depressed mothers, and expressed fewer affirmations of their behaviour such as imitating or smiling (Murray et al., 1996). In support of Cohn et al. (1990) they also found that mothers with depression expressed significantly higher levels of negation of the infant's behaviour, such as negative or discordant responses (Murray et al., 1996). The authors do not indicate whether these findings were shared with the participants, or discuss the ethical issues associated with doing so. Providing mothers with feedback about their interactional style may be inappropriate or unwelcome, unless this was discussed with the mother prior to videoing and her informed consent obtained. However, if the researcher has significant concerns about the implications of observed interactions for the infant's welfare or development, this places them in a difficult position. It would be advisable to consider this issue prior to any similar research, and to decide on a standard policy or procedure for that study, perhaps seeking advice from colleagues or ethics committees.

Field et al. (1990) assessed behaviour-state matching in mothers with and without a diagnosis of depression, when their infants were 3 months old. They used a coding procedure developed by Cohn and colleagues (Cohn, Matias, Tronick, Connell, & Lyons-Ruth, 1986). Four mother behaviour states were coded on a scale of 1 to 4: anger/poke (angry speech, pulling or poking infant), disengage (neutral, not interacting), elicit (attracting infant's attention) and play (positive- singing, smiling etc.). Infant behaviour states coded were: protest (negative- fussing, crying), look away (gaze directed away from mother), attend (gaze directed at mother), and play

(positive facial expressions, gaze directed at mother). Matching states were when the mother and infant's behaviour states were judged congruent, for example anger/poke and protest, play and play. Field and colleagues found that compared to non-depressed mothers, mothers with depression spent significantly more time in the anger/poke and disengaged states, and significantly less time in the play state. Their infants spent significantly more time in the protest state and less time in the play state. The total time spent in matched states was significantly lower for mothers with depression compared to those mothers without depression, indicating reduced interactional synchrony. In general, this study shows that both the overall nature and synchrony of the interaction were poorer in the depression group. This is of concern as the importance for infants of both the timing and quality of maternal responses has been demonstrated (Murray, 1986).

Maternal depression thus appears to have a negative impact on mother-infant interactions (Milgrom, Martin, & Negri, 1999), which in turn may affect infant cognitive, emotional and social development. Murray and colleagues (1996) studied mothers with postnatal depression interacting with their 2-month old infants. Ratings of maternal sensitivity and maternal remoteness at 2 months were found to be significant predictors of infant cognitive abilities at 18 months, as measured by the Bayley Scales of Infant Development (Murray et al., 1996). Tronick (1989) argued that infants develop a self-directed style of interacting in response to poorly coordinated interactions, relying on self-regulatory behaviours such as thumb-sucking. This may interfere with their interactions with people and objects in their environment, hindering their cognitive and social development (Tronick, 1989, Weinberg & Tronick, 1998).

Mother-Infant Interactions and Psychotic Illness

Parenting by mothers diagnosed with schizophrenia is of particular concern because of the recognised impact of the disorder on emotional responsiveness and social interaction (Riordan, Appleby, & Faragher, 1999). As demonstrated by Goodman and Brumley (1990), mothers with schizophrenia have been shown to be less affectionate and responsive compared to healthy mothers, and this style has been shown to have a significant effect on child IQ and social behaviour.

Riordan and colleagues (1999) assessed the mother-infant interactions of 26 women admitted to a psychiatric mother and baby unit, who had recovered from the acute phase of severe postpartum mental illness. 8 were diagnosed with schizophrenia and 18 with an affective disorder, either major depression, minor depression or bipolar disorder. Mothers were videotaped interacting with their 4-month-old infants, and the interactions scored using the Global Rating Scales of Mother-Infant Interaction (Murray, 1986). Mothers diagnosed with schizophrenia generally received lower scores, indicative of a less positive interactional style. Specifically, mothers with schizophrenia were significantly less sensitive and responsive, and significantly more demanding and intrusive than those with affective disorders. Additionally, their infants were significantly more avoidant and the interaction style was rated as significantly less mutually satisfying, more serious and less engaging (Riordan et al., 1999). However, this study had several limitations. Firstly, the study lacks comparison data from mothers with no psychiatric history, and the sample size is small, although the presence of significant findings suggests adequate statistical power. The authors acknowledged these points, but argue that the small sample size

reflects the relative rarity of such cases (Riordan et al., 1999). However, as reproductive rates amongst women with mental illnesses are comparable with the general population (Gregoire, 2001), this sample size may actually reflect recruitment difficulties, such as recruiting women who are in the process of adjusting to motherhood as well as managing their mental health. The authors also limited their study population by recruiting through one fairly specialist unit. The affective disorder group is heterogeneous (Riordan et al., 1999), and there is potentially a degree of overlap between the two groups. The authors did not indicate the influence of psychotic features in the affective group. Likewise, there may be evidence of depression in the schizophrenia group, blurring the distinction between the groups. Finally, recruiting from an inpatient unit is likely to bias the sample in favour of more severe cases where there may already be concerns surrounding the mother's parenting. However, the study provided an important indication that mothers with schizophrenia are less sensitive, responsive, and positive, and more demanding and intrusive, which is in accordance with the findings of Goodman and Brumley (1990) cited previously.

Riordan and colleagues (1999) suggested several possible explanations for their findings. These include the negative symptoms of schizophrenia (such as anhedonia and apathy), the influence of drug treatments, pre-morbid personality, the mother's own experiences of parenting, and social variables (Riordan et al., 1999). The authors did not expand upon how they thought these factors might influence mother-infant interactions, but stated that larger studies are required to identify predictor variables. This argument can also be applied to mothers with depression. Additional predictor variables for these patients may be the presence/absence of psychotic

features, and duration and severity of depressive illness. One factor which has received attention is the relationship between a mother's own experiences of parenting and her interactions with her infant. Using the Adolescent Attachment Interview (AAI), the Strange Situation paradigm to measure infant attachment (Ainsworth et al., 1978), and videotaped mother-infant interactions scored for maternal sensitivity, Ward and Carlson (1995) found that adolescent mothers who were classified as autonomous on the AAI were significantly more sensitive towards their infants at both 3 and 9 months, and that these mothers were also more likely to have infants classified as securely attached. Autonomous adult attachment is considered comparable to secure infant attachment, therefore this study illustrates a relationship between the mother's childhood attachment security, her sensitivity to her infant, and the infant's own attachment.

In summary, mother-infant interactions are thought to be important for infant emotional, social and cognitive development (Murray et al., 1986, Tronick, 1989, Vasta et al., 1999), and attachment (Belsky & Isabella, 1988). Experimental manipulations of these interactions reveal that infants are sensitive to both the timing and quality of the interaction, and appear distressed when these are disrupted (Murray, 1986). Repeated exposure to poorer interactions may have significant implications for infant development (Tronick, 1989). Studies have demonstrated poorer quality interactions in mothers with depression (Cohn et al., 1990, Murray et al., 1996) and with schizophrenia (Goodman & Brumley, 1990, Riordan et al., 1999), although the mechanisms underlying these differences are not well understood. Medication use, severity of illness, and the mother's own experiences of parenting are all potential influences. Another factor that may potentially impact upon

parenting and mother-infant interactions is the mother's cognitive functioning, but this relationship has received limited attention. In order to address this issue, this review will now describe what is known about cognitive functioning in adults with mental illness.

Cognitive Functioning and Mental Illness

Cognitive functions relate to the ability to perceive, transform or manipulate, and store information, and include perceptual abilities, visuo-spatial ability, praxis, constructional abilities, language, attention, memory processes and executive functions (Hodges, 2004). Memory can be further subdivided into several distinct capacities, including short-term or working memory, long-term memory, and verbal versus spatial memory (Hodges, 2004). Similarly, the term "executive function" refers to a diverse range of higher-order cognitive abilities, including initiation, planning, decision-making and cognitive flexibility (Spreen & Strauss, 1998).

The cognitive functioning of patients diagnosed with mental illness has been of interest to a number of researchers (Addington, Saeedi, & Addington, 2005, Keefe et al., 2004, Porter, Gallagher, Thompson, & Young, 2003). Studies have generally focused on patients diagnosed with either schizophrenia or major depression (unipolar), with fewer studies examining the cognitive profile associated with bipolar disorder, or major depression with psychotic features (Fleming, Blasey, & Schatzberg, 2004). While some studies have focused on exploring the profile of cognitive strengths and weaknesses associated with specific mental illnesses (e.g. Manoach et al., 2005, Porter et al., 2003), others have considered cognitive function

as a basis for understanding aetiology (Heinrichs & Zakzanis, 1998). Related to this, patients' relatives have also been studied with the aim of identifying neuropsychological vulnerability markers for specific mental illnesses (e.g. Chen et al., 1998). Further information about cognitive function has been published in studies investigating factors associated with psychosocial outcome (Addington & Addington, 1999, Prouteau et al., 2005) such as quality of life. Finally, neuropsychological functioning has been studied in relation to treatment outcomes, for example in assessing treatment efficacy (Goldman et al., 1993), and the possible role of cognitive deficits in symptom maintenance (Fossati et al., 1999).

In conclusion, research has demonstrated a variety of cognitive deficits in patients with serious mental illness. Such deficits have been shown to predict social functioning, and have come to represent targets for intervention (Green et al., 2004). Specifically, pharmacological treatments aimed at enhancing cognitive functioning are being developed, particularly for schizophrenia (Green & Nuechterlein, 1999). Research into the nature of any cognitive deficits is therefore an important area of research. However, some authors have cautioned that cognitive deficits are not evident in all patients (Kuperberg & Heckers, 2000). Indeed, some have argued that mental illnesses may actually be associated with high intelligence and creativity (David, 1999) rather than impaired cognitive function.

Methodological Issues

It is important to interpret this literature in the light of several methodological limitations. The first involves the selection of participants for studies. Most patient groups are selected using DSM-IV criteria (American Psychiatric Association, 1994)

to identify patients who represent a particular diagnostic group. However, this poses several difficulties. For example, the concept of schizophrenia as a discreet disease entity has been criticised (e.g. Bentall, 1990). A group of patients selected according to DSM-IV diagnostic criteria may still constitute a heterogeneous group of individuals with differing symptoms, severity and duration of illness, and level of functioning. Porter et al. (2003) also highlighted the issue of heterogeneity of patient samples with respect to depression. Many studies do not control for the subtype of depression, or for the presence of psychotic symptoms. This is important because differences in cognitive functioning between patients with depression with or without psychotic features have been observed (Basso & Bornstein, 1999a). Additionally many studies do not control for the effects of prescribed medications on cognitive functioning (Porter et al., 1999). Some of these issues may help to explain the conflicting findings evident in the literature.

Sample size is another important factor. Many studies used small samples; therefore the accuracy with which these reflect the wider population of individuals with mental illness can be questioned. It is also important to consider that many individuals diagnosed with mental illness show no cognitive deficits and some authors have linked mental illness with high intelligence and creativity (David, 1999). Most of the evidence for this view comes from case reports or biographies of artists, political figures or scientists. For example John Forbes Nash, a mathematician who developed a psychotic illness, was awarded the Nobel Prize for economics, and was the subject of a recent film "A Beautiful Mind". In addition, many studies have exclusion criteria, prohibiting the inclusion of patients with co-morbid diagnoses, alcohol or drug use, and medical conditions (for example, Harvey et al., 2004).

Whilst this is an attempt to control for extraneous variables, it may result in a research sample that does not accurately reflect the wider population. It is important to recognise these limitations when interpreting the research findings.

Tables 1 summarises studies of cognitive function in schizophrenia and depression. The table has been organised into sections according to domain of cognitive functioning (memory, working memory, attention and executive function). However, it is noted that some tasks will tap more than one cognitive domain, and could therefore feature in more than one section. For example, memory tasks such as the digit span also depend upon attentional processes. The original authors' papers and more general neuropsychological texts (e.g. Hodges, 2004) informed decisions regarding the placing of studies in this table.

Table 1

Studies of cognitive functioning in Schizophrenia and Depression

Memory			
Authors	Participants	Tests Used	Findings
Addington, Saeedi, & Addington, 2005	247 1st episode psychosis 66 HC	WMS-R[a] - Logical Memory I and II RAVLT [b] Rey Complex Figure	HC>psychosis All p<.0005 Rey NS
Basso & Bornstein, 1999 (a)	34 D + psychotic features (PF) 46 D no psychosis(NPF)	WMS-R[a] subtests : Visual Reproduction I Visual Reproduction II CVLT [c] total recall	 PF<NPF p< .01 PF<NPF p< .001 PF<NPF p< .001
Basso & Bornstein, 1999 (b)	46 recurrent D (RE) 20 D single episode (SE)	CVLT [c] - total recall acquisition slope long delay free recall short delay free recall long delay cued recall discrimination index	CVLT SE>RE $p \leq .05$ $p \leq .01$ $p \leq .001$ $p \leq .001$ $p \leq .0001$ $p \leq .01$

(table continues)

Table 1 (continued)

Authors	Participants	Tests Used	Findings
Fossati et al., 1999	20 D 14 SZ 20 HC	Free & cued selective reminding [d] (verbal learning test)	D + HC no difference SZ<HC+D
Nathaniel-James, Brown, & Ron, 1996	25 SZ 25 HC	RMT [e] -Words and Faces CVLT [c] - total free recall - Primacy/recency recall - short delay retention - long delay retention - serial vs semantic strategy use	NS SZ<HC $p<.007$ NS group difference SZ<HC $p<.03$ NS decline in recall SZ serial>semantic $p<.001$, HC NS
Nestor et al., 1998	18 SZ 21 HC	Word recall task -overall % word recall - recall pattern using connectionist model: effects of network size and word connectivity on % recall	 SZ<HC $p<.01$ SZ- recall significantly influenced by word connectivity ($p<.01$), rather than network size: abnormal recall pattern.
Porter et al, 2003	44 medication-free D patients	RAVLT [b] - word span	 NS

(table continues)

Table 1 (continued)

Authors	Participants	Tests Used	Findings
Porter et al, 2003 cont.	44 HC	- verbal learning	NS
		- distracter list	D<HC p= .009
		Paired-associates learning - No. levels completed	NS
		- No. completed 1st trial	D<HC p= .004
		Pattern recognition	D<HC p= .01
		Spatial recognition	D<HC p= .004
		Simultaneous/delayed matching to sample	Simultaneous: NS Delayed: D<HC p= .02
Weiland-Fiedler et al., 2004	28 D in remission 23 HC	CANTAB [f]:	
		Spatial Span	NS
		PRM [g]	
		non-verbal memory:	
		% correct	NS
		latency	NS
		DMS[h] visual recognition	
		% correct	NS
		latency	NS
		CVLT [c] total	NS

(table continues)

Table 1 (continued)

Working Memory			
Authors	Participants	Tests Used	Findings
Basso &	34 D + PF	WMS-R [a] subtests:	
Bornstein, 1999	46 D + NPF	- Digit Span forward	PF<NPF $p<.01$
(a)		- Digit Span backward	PF<NPF NS
		Visual Span forward	PF<NPF NS
		-Visual Span backward	PF<NPF $p<.01$
Basso &	46 D (RE)	WMS-R [a] Digit Span	SE normal range, RE
Bornstein, 1999	20 D (SE)	forwards and backward	mildly impaired.
(b)			
Fossati et al.,	20 D, 14	WAIS [1] subtests:	
1999	SZ,	- Digit Span forward	SG+D<HC $p<.01$
	20 HC	- Digit Span backward	D<HC $p<.01$,
			SZ+HC NS
		-Visual Span forward	NS
		-Visual Span backward	D<SZ<HC $p<.001$
Harvey et al. 2004	22 D	n-back task [J]	
	22 HC	- overall	D<HC $p=.001$
		- level 1	D<HC $t=2.89$, $p=.006$
		- level 2	D<HC $t=2.29$, $p=.013$

(table continues)

Table 1 (continued)

Authors	Participants	Tests Used	Findings
Harvey, et al., 2004 cont.		- level 3	D<HC t= 2.79, p= .008
		- reaction time	NS
		WAIS subtests: [i]	
		- Digit Span forward	NS
		- Digit Span backward	D<HC t= 3.11, p= .003
		- Visual Span forward	NS
		-Visual Span backward	NS
Manoach, et al., 2005	16 SZ 12HC	Spatial working memory task (SPWM) Shape working memory task (SWM)	Both tasks SZ<HC Accuracy p<.001 Response latency p<.06 Both groups- accuracy reduced with increased WM load, but SZ<HC
Porter et al., 2003	44 D (medication-free) 44 HC	Spatial working memory: 6 shape problem errors 8 shape problem errors strategy score	 D<HC p= .005 D<HC p= .004 D<HC p= .005
Weiland-Fiedler et al., 2004	28 D in remission 23 HC	spatial working memory: strategy score :between errors	D<HC p= .028 NS

(table continues)

Table 1 (continued)

Executive Function			
Authors	Participants	Tests Used	Findings
Addington, et al., 2005	247 1st episode psychosis 66 HC	Letter fluency Category fluency Letter-Number span WCST [k] Trailmaking A & B	HC>psychosis All p<.0005
Basso & Bornstein, 1999 (a)	34 D + PF 46 D + NPF	Verbal fluency Trailmaking Test A Trailmaking Test B WAIS-R: Block Design	PF<NPF *p*< .001 PF<NPF NS PF<NPF *p*< .01 PF<NPF *p*< .05
Basso & Bornstein, 1999 (b)	46 recurrent D (RE) 20 D single episode (SE)	Verbal fluency Trailmaking Test A&B WAIS-R: Block Design	SE scores in normal range, RE mildly impaired. SE>RE but NS
Fossati, et al., 1999	20 D, 14 SZ, 20 HC	Category Fluency Letter Fluency Cognitive Estimates WCST [k] Delis Test [l]: overall No. attempted sorts	SZ<D<HC *p* <.001 SZ<HC *p*< .02 NS NS SZ+D<HC *p*= .002 SZ+D<HC *p*= .001

(table continues)

Table 1 (continued)

Authors	Participants	Tests Used	Findings
Fossati, et al.,		No. expected sorts	D+HC>SZ NS
1999, cont.		% correct sorts	SZ<D<HC NS
		No. sorts identified	NS
		Sort perseverations	SZ<D p= .01,
		Name perseverations	SZ<HC p= .05
			D+HC NS
		Correct rule names	SZ+D<HC *p*= .01
			SZ+D NS
		Rule Name Perseveration	NS
		Cued sorting	NS
Harvey, et	22 D	Trailmaking A	NS
al., 2004	22 HC	Trailmaking B	D<HC *p*= .038
		Verbal fluency	NS
		WCST [k]	NS except
			perseverative errors
			D<HC *p*= .015
		Stroop test:	
		word	NS
		colour	D<HC *p* = .008
		interference	D<HC *p* = .038

(table continues)

Table 1 (continued)

Authors	Participants	Tests Used	Findings
Nathaniel-James, et al., 1996	25 SZ 25 HC	Verbal fluency WCST [m] - categories - errors - perseverative errors - % perseverative errors Hayling test [n] - errors - response initiation	SZ<HC p<.005 SZ<HC p<.03 NS SZ<HC p<.03 SZ<HC p<.02 SZ<HC p<.001 SZ<HC p<.001
Porter et al., 1999	44 medication-free D patients 44 HC	Verbal fluency "Exclude letter" fluency VCPT [o] - omission errors - commission errors Tower of London	D<HC p= .037 D<HC p= .004 D<HC p= .04 D<HC p= .04 NS
Wang et al., 2005	77 SZ 53 HC	Word fluency	NS

Attention

Authors	Participants	Tests Used	Findings
Bozikas, et al., 2005	30 HC, 29 SZ 19 BP (in remission)	PCPT [p]	Group effect p =.0004 SZ<BP p = .037 SZ<HC p = .0008 BP<HC NS

(table continues)

Table 1 (continued)

Authors	Participants	Tests Used	Findings
Egeland, et al., 2003	53 SZ 50 recurrent D 50 HC	DS-CPT [q] Dichotic listening test Stroop: colour-word Stroop: interference Simple reaction time Sequential reaction time 3 composite measures: basal speed speeded attention non-speeded attention Vigilance decrement	SZ<HC, D<HC SZ<D+HC SZ<D<HC SZ<HC SZ+D<HC S+D<HC SZ+D<HC SZ<D<HC SZ<HC D<SZ
Porter, et al., 2003	44 medication-free D patients 44 HC	Digit symbol substitution	NS
Wang, et al., 2005	77 SZ 53 HC	Attentional Network Test [s] - alerting reaction time - alerting ratio score - orienting reaction time	 SZ<HC NS SZ,HC NS SZ<HC NS

(table continues)

Table 1 (continued)

Authors	Participants	Tests Used	Findings
Wang, et al., 2005 cont.		- orienting ratio score	SZ<HC p<.05
		- executive ratio	SZ<HC p<.01
		– executive reaction time	SZ<HC p<.01
		- mean reaction time	SZ<HC p<.01
		- % accuracy	SZ<HC p<.01
Weiland-Fiedler, et al., 2004	28 D in remission 23 HC	CANTAB [f]: RVIP [t] correct hits	D<HC p= .008
		RVIP [t] response latency	D<HC p= .017
		ID/ED [u] - total trials	NS
		- adjusted errors	NS
		- intrareversal trials	NS

Note. SZ- schizophrenia, BP- Bipolar disorder, D- depression, HC- healthy controls

[a] Wechsler Memory Scale Revised (Wechsler, 1987), [b] Rey Auditory Verbal Learning Test (Lezak, 1995), [c] California Verbal Learning Test (Delis, Kramer, Kaplin, & Ober, 1987, as cited in Nathaniel-James et al., 1996), [d] Free and Cued Selective Reminding- episodic memory task (Grober et al., 1988, as cited in Fossati et al., 1999), [e] Recognition Memory Test (Warrington, 1984), [f] Cambridge Automated Neuropsychological Test Battery, [g] Pattern Recognition Memory- CANTAB, [h] Delayed Match to Sample (Visual recognition memory)- CANTAB, [i] Wechsler Adult Intelligence Scale-Revised (Wechsler, 1981), [j] n-back task- working memory (Braver et al., 1997), [k] Wisconsin Card Sorting Test (Heaton, 1981), [l] Delis card sorting test (adapted by B. Pillon from Delis et al., 1992, as cited in Fossati et al., 1999), [m] Wisconsin Card Sorting Test- modified (Nelson, 1976, as cited in Nathaniel-James et al., 1996),), [n] Hayling test of sentence completion (Burgess & Shallice, 1994, as cited in Nathaniel-James et al., 1996), [o] Vigil Continuous Performance Test (Cegalis & Bowlin, 1991,

as cited in Porter et al., 2003), [p] Penn Continuous Performance Test (Kurtz et al. 2001), [q] Degraded stimulus continuous performance test (Nuechterlein, 1991), [r] Digit Symbol Substitution Task (Wechsler, 1981), [s] Attention network test (Fan, McCandliss, Sommer, Raz, & Posner, 2002, as cited in Wang et al., 2005), [t] Rapid Visual Information Processing -CANTAB, [u] Intradimensional/extradimensional shift (attentional set-shifting)- CANTAB.

Cognitive Functioning and Schizophrenia

Patients diagnosed with schizophrenia generally score between 1 ½ and 2 standard deviations below the mean on key dimensions of cognition (Keefe et al., 2004). Impairments in attention, memory, and executive functioning have been demonstrated. For example, patients with schizophrenia display sustained attention impairment (Bozikas et al., 2005, Egeland et al., 2003), and selective attention impairment indicative of executive dysfunction (Egeland et al., 2003). Poorer sustained attention has been demonstrated in first-degree relatives of patients with schizophrenia (e.g. Chen et al., 1998), leading some to propose this as a genetic vulnerability marker for the condition (Chen et al., 1998). These findings have been replicated, but not always achieving statistical significance (e.g. Laurent et al., 2000). In a meta-analysis of research into cognitive performance of relatives of patients with schizophrenia, Sitskoorn and colleagues concluded that the largest effect sizes were for verbal recall (d = .54) and trail-making task B (d =.51) (Sitskoorn, Aleman, Ebisch, Appels, & Kahn, 2004), rather than sustained attention.

Wang et al. (2005) explored attention in relation to attentional networks. According to Posner & Peterson (1990), there are 3 attentional networks: alerting (ability to maintain alert state, and to respond to a warning signal), orienting (the selection of

information from numerous sensory inputs) and executive control (ability to respond to one aspect whilst ignoring the dominant aspect of a stimulus). Wang and colleagues found that patients with schizophrenia demonstrated a large, highly significant deficit in the executive network, and a smaller significant deficit in the orienting network (Wang et al., 2005). There was no significant group difference in the alerting network. The authors related their findings to the functioning of the prefrontal cortex, and the anterior cingulate. These brain regions are components of the executive attention network, and are also rich in dopamine (Wang et al., 2005), a neurotransmitter long implicated in schizophrenia and targeted by pharmacological treatments (Bennett, 1998).

A variety of memory deficits have been found in patients experiencing schizophrenia, including impairments in source memory, semantic memory and episodic memory (Kuperberg & Heckers, 2000), verbal and visual memory (Addington et al., 2005), verbal recall (Nestor et al., 1998), and working memory (Addington et al., 2005, Manoach et al., 2005). Nathaniel-James, Brown, and Ron (1996) demonstrated significant immediate memory impairment suggestive of executive functioning deficits, with more intrusion errors and reduced semantic strategies use. Short-delay recall was significantly poorer, presumably due to poor encoding and/or retrieval. Longer-delayed recall was relatively unimpaired (Nathaniel-James et al., 1996), suggesting that patients may require longer to encode the words or to consolidate memory. Recognition memory was also relatively unimpaired, suggesting poor initial recall may reflect retrieval deficits, possibly due to insufficient strategy use. On measures of executive functioning, patients with schizophrenia performed significantly poorer than controls on the verbal fluency test,

the Wisconsin card-sorting test (WCST) measuring rule-following and set-shifting abilities (Hodges, 2004) and the Hayling test of response initiation and suppression (Nathaniel-James et al., 1996). A correlation analysis indicated a number of significant associations for the patient group, but not the control group. For example, word recall scores on the List A, Trial 1 on the California Verbal Learning Task (CVLT) (free recall) were positively correlated with WCST category scores, and negatively associated with WCST total errors made. However, the authors have not indicated whether multiple comparisons were controlled for. The authors proposed that frontal lobe functioning contributes to the memory impairment found in patients with schizophrenia, because the pattern of impairment observed was related to executive function, and is similar to the impairment observed in patients with frontal lobe lesions (impaired immediate recall, unimpaired delayed recall and recognition) (Nathaniel-James et al., 1996). The frontal lobes play an important role in memory by facilitating the encoding and retrieval of information from memory (Lezak, Howieson, & Loring, 2004) by setting and monitoring the criterion for the information to be recalled (Schacter, Norman, & Koutstaal, 1998). The memory deficits observed in this study were indicative of deficits in these abilities.

Fossati et al. (1999) studied executive functioning and verbal memory. Patients with schizophrenia and depression both demonstrated short-term memory impairment, through poor performance on digit span forward. The schizophrenia group demonstrated poorer free recall on the verbal learning task, but did not differ from controls on numbers of intrusive or perseverative errors, in contrast to Nathaniel-James et al. (1996). Both patient groups demonstrated executive impairments compared to the control group, although their pattern of task performance was

different (Fossati et al., 1999). The term executive functioning refers to a variety of differing processes and skills, and it may be these diagnoses are associated with different patterns of impairment. Alternatively, other aspects of the tasks may have affected performance. For example, verbal fluency is a timed task, so speed of processing or sustained attention could affect performance.

Research demonstrating executive functioning and attention deficits (e.g. Egeland et al., 2003, Fossati et al., 1999) indicate a role for the frontal lobes in schizophrenia. Many studies use particular tasks to tap specific executive abilities and corresponding brain regions. For example, Meyer-Lindenberg et al. (2002) used the WCST, and recorded decreased activation in the dorsolateral prefrontal cortex in patients with schizophrenia during performance of this task, as compared to control participants. These findings could account for the poorer WCST performance of patients with schizophrenia (Nathaniel-James et al., 1996). However, this approach has been criticised. The prefrontal cortex (PFC) does not function in isolation, and some authors have emphasised the role of cortical and sub-cortical connections between the PFC and other brain regions (Antonova et al., 2004). Neuropsychological tasks designed to assess cognitive functioning following head injury, brain surgery or degenerative disease were not designed to relate to specific brain structures (Antonova et al., 2004). Tasks generally employ several cognitive processes; different researchers use the same tasks to study different brain structures, and the converse is also true (Antonova et al., 2004). Therefore the relationship between a specific task, a cognitive process and a brain region is not straightforward.

Cognitive Functioning and Depression

Porter et al. (2003) demonstrated significantly poorer performance in patients with depression on measures of visuospatial memory and attention. This performance was suggestive of executive function impairment, with significantly more errors on the spatial working memory task and the continuous performance task (sustained attention), with less efficient use of search strategy as compared to controls (Porter et al., 2003). Significant correlations were found between severity of depression, as measured by the Hamilton Rating Scale for Depression (Hamilton, 1960) and several measures of learning and memory, including long-term recall and recognition, retroactive interference, and percentage correct (Porter et al., 2003). Weiland-Fiedler et al. (2004) also demonstrated a deficit in sustained attention in patients with depression. They studied individuals in remission, which they state is in contrast to the majority of studies, which focus on patients experiencing an acute episode of depression (Weiland-Fiedler et al., 2004). The remission group performed significantly poorer than the control group on measures of rapid visual information processing (RVIP), psychomotor speed, and spatial working memory. After correction for residual, subclinical depressive symptoms, only the difference in performance on the RVIP target sensitivity scores remained significant. This suggests that even subclinical depression may affect cognitive function. Weiland-Fiedler et al. (2004) concluded that patients in remission continue to experience neurocognitive disturbance, particularly in sustained attention. Alternatively, poorer performance on the RVIP may be the result of poorer processing speed, as this task is timed. Impaired processing speed has been demonstrated in patients with depression (Egeland et al., 2003). The task also has a working memory component,

as participants are required to hold the task rules in mind and use these to govern their responses. Finally, this task is relatively difficult, and some participants may experience anxiety, which could affect performance.

The pattern of memory functioning in patients with depression is somewhat controversial. Some authors (e.g. Austin, Mitchel, & Goodwin, 2001) state that memory impairments have been consistently demonstrated, particularly in episodic memory and learning (Goodwin, 1997), although some studies have found mixed results (Fossati et al., 1999). Research findings remain variable; possible explanations include severity, duration and subtype of depression, and the influence of psychotropic medications on functioning (Porter et al., 2003). Studies indicate that poorer cognitive performance is associated with recurrent rather then single-episode depression (Basso & Bornstein, 1999b), severity of depressive symptoms (Porter et al., 2003) and the presence of psychotic symptoms (Basso & Bornstein, 1999a). However, the factors contributing to this poorer performance is unclear. For example, poorer performance could be related to differences in the type of treatment received, or to neurochemical or neuroanatomical alterations in the brain that could take place over the course of the depressive illness.

Memory impairments may reflect disruptions to the limbic system, which plays an important role in memory and learning (Joseph, 1996). The hippocampus is a part of the limbic system involved in the encoding of new information (Joseph, 1996), and studies have found reduced hippocampal volume in patients with depression (e.g. Sheline, Wang, Gado, Csernansky, & Vannier, 1996). Additionally, memory encoding is associated with increases in blood flow to the hippocampus and anterior

cingulate in healthy participants, but not in patients with depression (Bremner, Vythilingam, Vermetten, Vaccarino, & Charney, 2004).

Depression has also been associated with executive function impairment (e.g. Fossati et al., 1999). Harvey et al. (2004) used a range of assessments including the n-back procedure (Braver et al., 1997), which requires participants to retain and update verbal information in working memory. Patients with depression scored significantly lower than controls on all levels of the n-back task, indicating a difficulty updating working memory. On the trail-making task, patients were significantly slower when shifting between letters and numbers, indicating a set-shifting deficit. Patients also made significantly more perseverative errors on the WCST, a measure of category switching. On the Stroop test, patients demonstrated significant interference effects, suggesting a deficit of inhibition. There were no significant differences between groups on verbal fluency, in contrast to Fossati et al.'s findings. Harvey et al. concluded that patients with depression show deficits in the updating of working memory, set-shifting and inhibition.

Cognitive Functioning and Mental Effort

Some authors have proposed that reduced mental effort may underlie poorer cognitive functioning. Gorissen, Sanz, and Schmand (2005) used the Word Memory Test (WMT) to test mental effort in 64 patients with schizophrenia, 63 psychiatric patients without psychosis, 20 patients with acquired brain injury, and 20 healthy control participants. This task has been extensively validated for this purpose (Gorissen et al., 2005). They also administered a neuropsychological battery

assessing memory, attention and executive functioning. 72% of patients with schizophrenia and 25% of the non-psychotic psychiatric group failed the WMT, compared to 10% of the brain injury group and 10% of the control group. The difference between the first two groups was statistically significant. Patients with schizophrenia demonstrated the poorest performance on the neuropsychological measures, with the two remaining patient groups performing below the healthy control group. Effort, defined as performance on the WMT accounted for a significant proportion of the variance in all neuropsychological test scores ($p<.001$). Percentages ranged from 34% of the variance in word recognition scores to 14% of the variance on Trail Making part A. However, some patients with schizophrenia demonstrated cognitive impairments but no reduced effort, i.e. they passed the WMT (Gorissen et al., 2005). Insufficient effort may account for some of the cognitive deficits evident in patients with schizophrenia, but this cannot be said of all patients. Zakzanis, Leach, and Kaplan (1998) conducted a meta-analysis of studies of neurocognitive functioning in patients with depression, examining effect sizes from 22 studies, totalling 726 patients and 795 control participants. The largest effect sizes were for measures of encoding and retrieval from episodic memory, for example the Wechsler Memory Scale- Revised memory quotient effect size was d = -1.42. Intermediate effect sizes (e.g. d = -.63) were found for psychomotor speed and sustained attention. These data were then examined to determine whether any measures could discriminate between patients and controls. None could do so reliably. The authors then calculated effect sizes for effort-demanding tasks using data from Weingartner (1986). These tasks required participants to listen to lists of unrelated words and to respond with either semantically-related words (semantic, effort-demanding cognitive processing condition) or phonologically-similar words

(acoustic, superficial processing condition). Free recall was tested, and was poorer in patients with depression when words had been learnt under effort-demanding conditions (Weingartner, 1986). The effect size for effort-demanding processing was 2.63, which although large, had an overlap percentage of 10.7, so was considered to be almost a reliable discriminator (Zakzanis et al., 1999). They concluded that depression is associated with a "dysfunction of effortful encoding" of information (p. 111), which could underlie memory deficits.

Porter et al. (2003) highlighted the impact of motivation on neuropsychological test performance, stating that this may be particularly relevant to studies of depression because of the relationship between affect (mood) and drive (motivation). Motivation depends in part upon the ability to experience pleasure or reward, and must therefore be associated with our hedonic drive and affect (Austin et al., 2001). Low motivation is inherent in patients with depression, presumably linked to their lowered mood; therefore it has been argued that to study reduced motivation is to study depression itself (Austin et al., 2001).

The anterior cingulate plays an important role in motivation (Sewards & Sewards, 2003). Neuroimaging studies implicate the anterior cingulate in attentional effort, or motivated attention in the face of challenges to attentional performance (Sarter, Gehring, & Kozak, 2005). Disruptions to anterior cingulate function could account for the poor sustained attention (Egeland et al., 2003), and reduced cognitive effort (Zakzanis et al., 1999) observed in patients with depression. Smaller anterior cingulate volume has been observed in this patient group (Caetano et al., 2006). The anterior cingulate has also been implicated in the generation of affect, which is

closely linked to motivation (Porter et al., 2003). Using a negative mood induction procedure with a non-clinical sample, Teasdale et al. (1999) demonstrated that cognitive generation of negative affect was associated with activation of components of the medial prefrontal cortex, including the right anterior cingulate. The right thalamus and right inferior frontal gyrus were also activated. In another neuroimaging study, Mayberg et al. (1999) demonstrated that induced low mood was associated with increased cerebral blood flow to part of the anterior cingulate that lies beneath the corpus callosum, but decreased flow to the dorsolateral prefrontal cortex. Austin et al. (2001) state that this region has been implicated in the set-shifting deficits associated with depression (e.g. Harvey et al., 2004). The authors concluded that "cognitive deficits are intrinsic expressions of the brain changes associated with depression" (Austin et al., 2001, p. 204), and that affect and cognition may be anatomically linked via the complex and widely distributed neuroanatomical networks of the limbic system.

Cognition and Mental Illness- Section Summary

Cognitive impairments have been demonstrated in individuals diagnosed with schizophrenia or depression. Schizophrenia is associated with poorer sustained, speeded and selective attention (Egeland et al., 2003), deficits in the executive and orienting attentional networks (Wang et al., 2005), and impairments in verbal and visual memory, working memory, (Addington et al., 2005), and spatial working memory (Manoach et al., 2005). These may reflect failures of executive processes, evidenced by intrusion errors and reduced use of retrieval strategy (Nathaniel-James

et al., 1996). Impaired executive functioning has also been demonstrated (Fossati et al., 1999, Nathaniel-James et al., 1996).

Depression has been associated with impaired vigilance (Egeland et al., 2003), visuospatial memory (Porter et al., 2003), episodic memory (Goodwin, 1997) and executive functioning (Fossati et al., 1999). Some authors have argued for the role of cognitive effort in neuropsychological task performance (Gorisson et al., 2005, Zakzanis et al., 1998). This may be particularly relevant to depression because of the link between affect and motivation. These may be linked anatomically via the anterior cingulate (Sarter et al., 2005) and related structures of the dorsolateral prefrontal cortex.

Maternal Cognitive Functioning and Mother-Infant Interactions

Cognitive deficits in patients with schizophrenia have been linked to aspects of social functioning (Kuperberg & Heckers, 2000), such as performance of daily living tasks (Dickerson, Boronow, Ringel, & Parente, 1999), and social problem-solving skills (Addington & Addington, 1999). For a parent, interacting with one's infant is an important aspect of functioning involving processes and skills that overlap with those required for general social functioning. Cognitive deficits in attention, processing speed and other abilities may therefore impact upon a mother's ability to respond to her infant.

It is of interest to consider whether poor mother-infant interaction in mothers with depression and/or schizophrenia is partly explained by impaired cognitive function. However, this issue is complex. In support of a contribution of lowered cognitive

function, mothers with intellectual disabilities demonstrate impoverished interactions with their infants (Feldman, Case, Rincover, Towns, & Betel, 1989). These mothers have been found to be less affectionate, responsive, accommodating and contingently reinforcing in their interactions with their infants, as compared to non-intellectually disabled mothers (Feldman et al., 1989). Using the Bayley scales, maternal involvement was positively correlated with infant mental development ($r = .69$, $p < .05$), (Feldman, Case, Towns, & Betel, 1985). However, poor interaction may be associated with poor social understanding secondary to lowered cognitive function, rather than directly with low cognitive function itself. It may therefore be important to examine cognitive function, maternal socio-emotional function and mother-infant interaction in tandem in order to address this complex relationship. Despite the potential importance of this approach, it has not been empirically researched.

This line of investigation may be particularly important for the development of interventions for mothers diagnosed with mental illness (Craig, 2004). Parent training packages have been used with mothers with intellectual disabilities. For example, the Parent Education Project III study aimed to increase the affection and responsiveness displayed by mothers with learning disabilities towards their young children (Feldman et al., 1989). The results indicated that training led to an increase in observable maternal affection, praise and imitation of vocalisations. Two children who had showed signs of cognitive delay thereafter improved their language skills and their scores on the Bayley Scales. Training therefore appeared to have a positive impact on child development in these two cases. The appropriateness of generalizing findings from mothers with intellectual disabilities to mothers with mental illness may be limited. However, these data provide further evidence for a

link between maternal cognitive and socio-emotional function, mother-infant interactions and infant cognitive development.

Conclusions

Parenting by mothers diagnosed with a mental illness has long been an area of concern for health professionals. The developmental, social, physical and mental health outcomes for children of mothers with mental illness have been described (Goodman & Brumley, 1990, Gotlib & Goodman, 2002, Henriksson & McNeil, 2004). Studies of mothers with schizophrenia have found that their children display significantly increased rates of a variety of problems including language disorders, and disturbed behaviour (Henriksson & McNeil, 2004), with children of depressed mothers exhibiting fewer difficulties. High rates of psychiatric disturbance have also been reported in children of parents with mental illness (e.g. Gotlib & Goodman, 2002). However, it is not clear whether such findings are the result of genetic factors, environmental influences such as poverty, and family stress, or cognitive factors such as maternal executive function deficit. It is likely that a combination of factors are involved but to the author's knowledge there are no published studies examining maternal socio-emotional and cognitive factors contemporaneously with infant assessment. Considering that some authors (e.g. Goodman & Brumley, 1990) argue that parenting style is the major way in which maternal mental illness exerts its influence on child development, this is a cause for concern. Mothers with depression have been found to be significantly less sensitive to their infant's cues compared to non-depressed mothers, and express fewer affirmations of their behaviour such as imitating or smiling, and higher levels of negation of the infant's behaviour (Murray et al., 1996). Mothers with schizophrenia have been shown to be less sensitive and

responsive, and significantly more demanding and intrusive when interacting with their infants (Riordan et al., 1999).

In summary, the mechanisms underlying the relationship between maternal mental illness and poorer mother-infant interactions have not been thoroughly investigated. Several hypotheses have been proposed, including the mothers' own parenting experiences, and social variables (Riordan et al., 1999). Based on the evidence described in this review, it may be proposed that there is a relationship between maternal cognitive functioning and the quality of mother–infant interactions. There is evidence for cognitive impairment in patients with schizophrenia (Keefe et al., 2004) and depression (Porter et al., 2003), which is associated with poorer social functioning in schizophrenia (Dickerson et al., 1999). Thus it is feasible that cognitive difficulties could impact upon general parenting capacity, and more specifically upon the quality of a mother's interactions with her infant, providing one potential mechanism through which maternal mental illness could influence child development. This relationship is likely to be highly complex, and interpretation should be circumspect. Notwithstanding these caveats, this hypothesis may have the potential to offer a greater understanding of the relationship between maternal mental illness, cognitive function and mother-infant interaction than is currently available.

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Empirical Paper*

An Exploratory Study of the Relationship between Mother-Infant Interactions and Maternal Cognitive Functioning in Mothers diagnosed with Schizophrenia or Depression

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* Applying guidelines for publishers for submission to the Journal of Reproductive and Infant Psychology (Appendix 1).

Abstract

This paper explores the relationship between maternal mental illness, maternal sensitivity and cognitive functioning. The performance of 6 mothers with serious mental illness (SMI: schizophrenia or depression) and 12 mothers with no psychiatric history were compared using the Crittenden Care Index (Crittenden, 2004), subscales from the Parenting Stress Index (adapted from Abidin, 1995), and a computerised cognitive assessment battery (Cognitive Drug Research, 2002). It was hypothesised that the SMI group would demonstrate poorer quality of mother-infant interactions, and poorer cognitive functioning, as compared to the control group. Additionally, it was hypothesised that reduced cognitive function would mediate the relationship between maternal mental illness and poorer mother-infant interactions. In support of the first hypothesis, mothers in the SMI group were significantly less sensitive during interactions with their infants, and rated themselves as significantly less competent. They also performed significantly poorer on the Speed of Memory processing factor of the cognitive assessment. The presence of maternal mental illness was found to be a significant predictor of maternal sensitivity, but when cognition (Speed of Memory Processing) was taken into account, the strength of this relationship was reduced. This suggests that the relationship between maternal mental illness and mother-infant interaction may be mediated by level of cognitive function. These findings will be discussed with reference to previous research, and the limitations of the study will be considered.

Introduction

The impact of maternal mental illness on parenting capacity has received much interest within the literature. Reproductive rates among women with serious mental illness are comparable with the general population (Gregoire, 2001), and with the prevalence of postnatal depression ranging from 8-15% (O'Hara, 1997), a significant proportion of children may be expected to experience parental mental illness. The impact of these circumstances on children's physical, cognitive and emotional development has been documented (Goodman & Brumley, 1990, Henriksson & McNeil, 2004, Tronick, 1989). Parenting skills may be affected, and in extreme cases children's physical needs may be neglected, and they may experience social challenges such as poverty (Cleaver, Unell, & Aldgate, 1999).

One aspect of parenting which has received considerable attention is the impact of maternal mental illness on the quality of mother-infant interactions. Studies of mothers diagnosed with depression or schizophrenia have found important differences in the quality of interactions, as compared to mothers with no psychiatric history (Cohn, Campbell, Matias, & Hopkins, 1990, Murray, Fiori-Cowley, Hooper, & Cooper, 1996, Riordan, Appleby, & Faragher, 1999). Given the importance of early mother-infant interactions for many aspects of infant development (Tronick, 1989), any impairment could have potentially significant consequences. However, the mechanisms underlying the differences in the quality of mother-infant interactions have not been thoroughly researched (Riordan et al., 1999). The relative contributions of factors such as present symptoms, use of medication, and concurrent

life stress have not been specified. Another potential influence on the quality of mother-infant interactions is maternal cognitive functioning.

A variety of cognitive impairments have been demonstrated in individuals diagnosed with mental illnesses (e.g. Addington, Saeedi & Addington, 2005, Fossati et al., 1999), and these have been shown to relate to social functioning (Kuperberg & Heckers, 2000) in patients with schizophrenia. It is of interest to consider whether poorer quality mother-infant interaction in mothers with mental illness is at least partly explained by impaired cognitive function. Yet to the author's knowledge, no studies have attempted to explore this relationship. This line of research could further our understanding of the difficulties experienced by mothers with mental illness, and of the mechanisms through which maternal mental illness impacts upon the mother-child relationship. This research may also contribute to parenting programmes aimed at supporting mothers with mental illness to care for their infants more effectively and autonomously (Craig, 2004).

Mother-Infant Interactions

Rutter & Quinton (1984) proposed four sets of mechanisms through which parental mental illness may impact upon children. A direct biological explanation is that there may be genetic transmission of mental illness itself. Secondly, environmental factors arising directly from the parent's poor mental health may impact upon the child, and in extreme cases neglect or abuse may occur. Thirdly, the mental illnesses may have indirect effects, through disruption of parenting and parent-child relationships. Finally, associated factors including marital difficulties,

unemployment, and poverty can impact upon the child's welfare. Goodman and Brumley (1990) argued that parenting is the major mechanism through which maternal mental illness impacts upon children. Their research emphasised the importance of the quality of mother-infant interactions in influencing child outcomes, rather than maternal diagnosis per se. In their study, maternal responsiveness and maternal tenseness observed during mother-infant interactions were found to be significant predictors of child social behaviour, with maternal diagnosis (schizophrenia or depression) making no significant additional contribution (Goodman & Brumley, 1990).

Early interactions between a mother and her infant are important for infant social, cognitive and emotional development (e.g. Murray et al., 1996, Tronick, 1989), as well as playing a vital role in the formation of a secure attachment bond (Belsky & Isabella, 1988). Studies of mother-infant interactions typically use video recordings to capture the interaction, which is then subjected to a detailed analysis, often using coding systems. Mother and infant sit opposite each other, with the mother instructed to play with her baby in her usual manner. One video camera records the mother's face and another one records the infant's (Vasta, Haith, & Miller, 1999). Alternatively, one camera is positioned behind the mother to record the infant, while her face is reflected in a mirror positioned adjacent to the infant (Murray, Cooper, Wilson, & Romaniuk, 2003). Importantly, both procedures allow for simultaneous recording of both mother and infant facial expressions. Experimental disruption of these interactions revealed that infants are acutely sensitive to both the timing and quality of the maternal response. If mothers are instructed not to respond to their infants, referred to as the "blank-face" condition, they display initial protest

behaviour, including frowning and lip-biting, followed by withdrawal (Murray, 1986). Exposure to disrupted interactions on a longer-term basis may impact upon the infant's development, as they are thought to respond by developing a self-directed style of interacting, relying on self-regulatory behaviours such as thumb-sucking to regulate their internal state. This may interfere with their interactions with people and objects in their environment, hindering their cognitive and social development (Tronick, 1989, Weinberg & Tronick, 1998).

The impact of maternal depression on mother-infant interactions has been documented. For example, mothers with depression display more negative affect and less positive affect when interacting with their infants (Cohn et al., 1990), are less sensitive to their infant's cues compared to non-depressed mothers, and express fewer affirmations and more negations of their infant's behaviours (Murray et al., 1996). Disturbances in mother-infant interactions have been found to predict poorer infant cognitive development in women with postnatal depression (Murray et al., 1996). Maternal depression also affects the attachment relationship between mother and child, increasing the likelihood of insecure attachment development (Goldberg, 2000).

Mother-infant interactions have also been studied in cases where the mother has been diagnosed with schizophrenia. Studies have found mothers with schizophrenia to be less affectionate and responsive compared to mothers with no mental health problems, and this style has been shown to have a significant effect on child IQ and social behaviour (Goodman & Brumley, 1990). In a study comparing mothers with schizophrenia against mothers diagnosed with an affective disorder (major

depression, minor depression, or bipolar disorder), mothers with schizophrenia were rated as significantly less sensitive and responsive, and significantly more demanding and intrusive (Riordan et al., 1999). Their infants were significantly more avoidant and the interaction style was rated as less mutually satisfying, more serious and less engaging (Riordan et al., 1999).

The reasons underlying the poorer mother-infant interactions in mothers with mental illness have not been thoroughly explored. Contributing factors may include the influence of drug treatments, pre-morbid personality, the mother's own experiences of parenting, and social variables (Riordan et al., 1999). The occurrence of adverse life events (Murray et al., 1996), and the mother's attachment relationship with her own mother (Ward & Carlson, 1995) have been shown to influence mother-infant interactions, specifically maternal sensitivity to her infant. Additional influences may include the duration and severity of illness, and the nature of symptoms experienced (for example, anhedonia, apathy, or fatigue, or the presence of psychotic symptoms). This present study proposes for the first time that maternal cognitive functioning may also contribute to the poorer quality mother-infant interactions observed in mothers with mental illness. This relationship has not yet been explored within the research literature. Cognitive functioning has been linked to social functioning in schizophrenia (Kuperberg & Heckers, 2000), including performance of daily living tasks (Dickerson, Boronow, Ringel, & Parente, 1999), and social problem-solving skills (Addington & Addington, 1999). For a parent, interacting with one's infant is an important aspect of functioning involving processes and skills that overlap with those required for general social functioning. Cognitive deficits, for example in processing speed or attention may therefore impact upon a mother's

ability to respond to her infant. To provide further justification for this hypothesis, research examining the cognitive functioning of adults diagnosed with schizophrenia or depression is now discussed.

Cognitive Functioning and Schizophrenia

Patients diagnosed with schizophrenia generally score between 1 ½ and 2 standard deviations below the mean on key dimensions of cognition (Keefe et al., 2004). For example, Addington et al. (2005) found that patients with schizophrenia performed significantly poorer than controls on measures of letter fluency, category fluency, verbal and visual memory, working memory, attention, early information processing, visual-constructional ability, visuomotor sequencing, psychomotor speed, and the Stroop test. Egeland et al. (2003) studied attention in patients with schizophrenia and depression and concluded that patients with schizophrenia display impairments in speed and selective attention, the latter indicating executive dysfunction, whereas patients with depression display impairments in speed and vigilance. Sustained attention deficits have also been demonstrated in this client group (Bozikas et al., 2005). Poorer sustained attention has also been demonstrated in first-degree relatives of patients with schizophrenia (e.g. Chen et al., 1998), leading some to propose that this may represent a genetic vulnerability marker for the condition (Chen et al., 1998). However, in a meta-analysis of research into cognitive performance of relatives of patients with schizophrenia, Sitskoorn and colleagues concluded that the largest effect sizes were for verbal recall (*d* .54) and trail-making task B (*d* .51) (Sitskoorn, Aleman, Ebisch, Appels, & Kahn, 2004), rather than sustained attention

A variety of memory deficits have been found in patients experiencing schizophrenia, including impairments in source memory, semantic memory, episodic memory (Kuperberg & Heckers, 2000), and working memory (Manoach et al., 2005). Nathaniel-James, Brown, and Ron (1996) demonstrated impaired immediate memory but not delayed recall or recognition memory in patients with schizophrenia. The same study demonstrated executive impairment, and found some correlations between memory and executive function. The authors concluded that the pattern of memory impairment observed in patients with schizophrenia is similar to that found in patients with frontal lobe lesions, and argue that frontal lobe functioning contributes to the memory impairment found in patients with schizophrenia (Nathaniel-James et al., 1996). Other studies have also demonstrated executive impairment in patients with schizophrenia (e.g. Fossati et al., 1999), implicating frontal lobe dysfunction. Consistent with this hypothesis, studies have indicated that patients with schizophrenia experience reduced blood flow to the frontal cortex, or hypofrontality (Gazzaniga, Ivry, & Mangun, 1998). However, almost every cortical and sub-cortical brain structure has been implicated in schizophrenia (Antonova, Sharma, Morris, & Kumari, 2004), therefore the neurobiology relating to the aetiology and symptom expression lacks specificity. For example, studies have found lower overall brain volume (Mellers, 2004), smaller temporal lobes, and enlarged ventricles (McGrath et al., 2003). Dopaminergic mechanisms have also been implicated, supported by the therapeutic effects of neuroleptic medications, which act upon the dopaminergic D2 receptor (Bennett, 1998).

Cognitive Functioning and Depression

A variety of cognitive impairments have been demonstrated in patients with depression (Egeland et al., 2003, Fossati et al., 1999, Rose & Ebmeier, 2006). Memory impairments are often reported by patients, and have been demonstrated empirically (Austin, Mitchel, & Goodwin, 2001), particularly in episodic memory and learning (Goodwin, 1997). Impairments in working memory and executive functioning (Fossati et al., 1999, Harvey, Le Bastard, et al., 2004, Moritz et al., 2002) have also been demonstrated. For example, Porter et al. (2003) compared the neurocognitive functioning of medication-free patients diagnosed with major depressive disorder, with demographically-matched control participants. Significant differences between the groups were found on measures of visuospatial memory, with patients significantly less accurate on pattern and spatial recognition tasks, and significantly slower on pattern recognition (Porter et al., 2003). Additionally, patients demonstrated poorer performance on attention and executive function tests, making significantly more errors of omission and commission on the continuous performance test (sustained attention), significantly more errors on a spatial working memory task, and making less efficient use of search strategy as compared to controls (Porter et al., 2003), suggestive of executive deficits. Harvey, Le Bastard, and colleagues (2004) administered a variety of executive function tasks and concluded that patients with depression show deficits in the updating of working memory, set-shifting and inhibition. Fossati et al. (1999) also demonstrated executive impairments, finding that patients with depression generated significantly fewer words on measures of verbal category fluency, and were impaired in their ability to generate and identify accurate card sorts on a card-sorting task.

Although a variety of cognitive impairments have been demonstrated in patients diagnosed with depression, attempts to specify the profile of cognitive strengths and weaknesses associated with depression have proved inconclusive (Rose & Ebmeier, 2006). Methodological limitations associated with this line of enquiry may in part explain this outcome. For example, several authors have highlighted the heterogeneity of samples of patients diagnosed with depression, and caution that factors including use of antidepressant medication, and severity, duration and subtype of depression may impact upon cognitive functioning (Porter et al., 2003). Studies indicate that poorer cognitive performance is associated with recurrent rather then single-episode depression (Basso & Bornstein, 1999b), severity of depressive symptoms (Porter et al., 2003) and the presence of psychotic symptoms (Basso & Bornstein, 1999a). However, the factors contributing to the poorer performances of these patient groups are unclear. For example, poorer performance could be related to differences in the type of treatment received, or to neurochemical or neuroanatomical alterations in the brain that could take place over the course of the depressive illness.

Some authors have suggested that reduced cognitive effort may underlie the poor cognitive test performance of patients with depression (Zakzanis, Leach, & Kaplan, 1998). Porter et al. (2003) highlighted the impact of motivation on neuropsychological test performance, stating that this factor may be particularly relevant to studies of depression because of the relationship between affect (mood) and drive (motivation) (Porter et al., 2003). Motivation depends in part upon the ability to experience pleasure or reward, and must therefore be associated with our hedonic drive and affect (Austin et al., 2001). Low motivation is inherent in patients

with depression, presumably linked to their lowered mood; therefore it has been argued that to study reduced motivation is to study depression itself (Austin et al., 2001).

In summary, impairments in memory, attention and executive function have been demonstrated in patients with schizophrenia and in patients with depression. The pattern of neurocognitive performance associated with these two conditions is similar, with impairments in speed and sustained attention (Egeland et al., 2003), short-term memory (Fossati et al., 1999) and working memory and executive function (Moritz et al., 2002) common to both. Differences between the two patient groups have been found on measures of selective attention (Egeland et al., 2003), and whilst Fossati et al. demonstrated executive impairments in both groups, their pattern of performance across a number of executive tasks was different. However, other studies have directly compared patients with schizophrenia and those with depression and have found no differences on measures of executive function and working memory (Moritz et al., 2002). Similar brain regions have also been implicated in both conditions (Hafner et al., 2005), including the prefrontal cortex and the anterior cingulate (Cleare, 2004, Wang et al., 2005).

Cognitive Functioning and Mother-Infant Interactions

Schizophrenia and depression are associated with a variety of cognitive impairments in memory, attention and executive functioning (Addington et al., 2005, Egeland et al., 2003, Harvey, Le Bastard, et al., 2004, Porter et al., 2003). Cognitive functioning has been found to relate to social functioning in patients with

schizophrenia (Addington & Addington, 1999, Dickerson et al., 1999, Kuperberg & Heckers, 2000). As a mother, interacting with one's infant is an important aspect of functioning involving processes and skills that overlap with those required for general social functioning. It is therefore of interest to consider whether poor mother-infant interactions in mothers with mental illness are at least partly explained by impaired cognitive function. In support of this hypothesis, research with mothers with intellectual disabilities suggests that cognitive functioning can impact upon mother-infant interactions. Studies have shown these mothers to be less affectionate, responsive, accommodating and contingently reinforcing in their interactions with their infants, as compared to non-intellectually disabled mothers (Feldman, Case, Rincover, Towns, & Betel, 1989). However, this may reflect poor social understanding secondary to intellectual impairment, rather than a direct influence of cognitive function. In addition, the validity of generalising from mothers with global intellectual impairment to mothers with schizophrenia or depression can be questioned. Notwithstanding these caveats, this research suggests that maternal cognitive functioning may have a negative impact upon a mother's interactions with her infant, which in turn may have potentially significant consequences (Goldberg, 2000, Murray et al., 1996, Tronick, 1989).

The clinical relevance of this line of enquiry is two-fold. Firstly, it may contribute to our understanding of the mechanisms underlying the poorer quality mother-infant interactions observed in cases where the mother has been diagnosed with a mental illness. As a result, this research could then assist professionals working with this client group, through educating colleagues and potentially mothers themselves about the relationship between maternal mental health, cognitive functioning and mother-

infant interactions. This research may also inform the development of training packages for mothers (Craig, 2004, Mowbray, Oyserman, & Bybee, 2000) aimed at improving the quality of mother-infant interactions. Training packages for mothers with learning disabilities have shown positive results, with increases in observable maternal affection, praise and imitation of child vocalisations, and improvements in child cognitive development (Feldman et al., 1989). Again, generalising findings from this client group to mothers with mental illness must be approached with caution.

The first aim of this study is to replicate previous research by demonstrating significant differences in the quality of mother-infant interactions in mothers diagnosed with a serious mental illness (SMI), as compared to a control group. It is hypothesised that mothers with SMI will also demonstrate significantly poorer cognitive functioning compared to the control group. The two groups will also be compared with respect to self-reported parental stress and parental competence. The second aim is to extend the current literature by examining the relationship between maternal mental illness, maternal cognitive functioning and the quality of mother-infant interactions.

Method

Design

An independent measures design was used. The independent variable was the presence or absence of maternal mental illness. The dependent variables were

cognitive function (as measured by a cognitive assessment battery), quality of mother-infant interaction and perceived mother-infant relationship.

Data for this study were collected as part of a wider study conducted by a Perinatal Mental Health Service.

Participants

A total of 6 mothers diagnosed with a serious mental illness (SMI) were recruited through a local Perinatal Mental Health Service. Fourteen suitable mothers were initially identified, one was later considered too unwell to be approached, one recovered and was discharged, and one could not be contacted. Eleven were approached, four declined to participate, one agreed but then could not be contacted, and six were recruited for the study. All had received a primary diagnosis of either schizophrenia (n = 2), or a depressive disorder (mild, moderate or severe depressive episode, recurrent depressive disorder or major depression) (n = 4) according to ICD-10 diagnostic criteria (World Health Organisation, 1992). Diagnosis was performed by the Consultant Psychiatrist as part of routine clinical practice, prior to recruitment for the study. Current symptoms were recorded using the Brief Psychiatric Rating Scale- Expanded (Lukoff, Nuechterlein, & Ventura, 1986). The team selected this measure to form part of their follow-up research; it was not included in this study. The median BPRS total score was 35.5, with a range of 30-55. Mothers were not excluded from the sample for the presence of other mental health problems (for example, social anxiety), as high levels of co-morbidity are a feature of this client group. However, women were excluded from the study for the

following reasons: history of head injury or learning disability, primary diagnosis of personality disorder, significant alcohol or drug use likely to impair cognitive functioning, current eating disorder, or history of Electro-Convulsive Therapy (ECT) treatment. All mothers were currently living with the father of the index infant.

Table 1

Socio-demographic information for the two groups

	Control Group (n = 12)	SMI Group (n = 6)
Infant Age (weeks) [a]	11.5 (5-17.5)	10.5 (7- 12)
Infant Gender [b]	66.7% male (n =8)	83.3% male (n =5)
Maternal Age (years) [a]	34 (25-40)	33.5 (29-39)
Maternal Educational Level	50% level 5	33.3% level 5
(PSI) [b]	25% level 4	0% level 4
	25 % level 2/3	66.7% level 2/3
Baby Order [b]	50% first baby (n =6)	83% first baby (n =5)

Note. [a] median and range reported, Mann-Whitney test performed. [b] χ^2 test (Fisher's Exact). Educational level (as measured by the Parenting Stress Index (PSI): 5 = achieved post-graduate qualification, 4 = obtained undergraduate level degree, 3 = attended college or university, 2 = completed compulsory education.

Twelve mothers were recruited for the control group, two for each participant in the SMI group. They were recruited through the National Childbirth Trust (NCT) (n =6), or through colleagues and acquaintances (n =6). None were well know to the researcher. Mothers were selected according to infant age, to ensure they were comparable with the age of the infants of mothers in the SMI group. All mothers were married or co-habiting with their infant's father. Demographic information for both groups can be found in Table 1. Exploratory analyses showed that there were no significant differences between the two groups on any of the demographic variables (all $p > .1$).

Mothers in the control group were screened for the presence of current mental health symptoms using the Psychosis Screening Questionnaire (PSQ, Bebbington & Nayani, 1995) and the Edinburgh Postnatal Depression Scale (EPDS, Cox, Holden, & Sagovsky, 1987). Descriptions of these measures can be found in Appendix 2. All participants screened negative for psychosis and scored below the clinical threshold on the EPDS (median 2.5, range 0-8). They were also asked about previous psychiatric support. Nine mothers had received no such support, two mothers had received counselling more than five years previously (one for bereavement, one for university-related stress), and one mother experienced occasional panic attacks during pregnancy for which she had not sought treatment and had ceased at the time of the study.

Measures

Crittenden Care Index (Crittenden, 2004).

The CARE index has been developed based on attachment theory and is proposed to evaluate adult-infant patterns of interaction. It differs from other measures by assessing the specific relationship or dyad, rather than individuals (Crittenden, 2004). Mother and infant are videotaped while engaging in playful interaction. The infant faces the video recorder whilst mother faces the child, with a mirror positioned so that her face is visible on camera. They are recorded for 3-5 minutes. Professionals trained in using the coding system then view this tape. Mothers and infants are rated on seven aspects of interactional behaviour: facial expression, verbal expression, position and body contact, affection, turn-taking, control and choice of activity (Crittenden, 2004). These ratings then contribute to three adult scales: Sensitive, Controlling and Unresponsive, and four infant scales: Cooperative, Difficult, Compulsive and Passive.

Sensitive maternal behaviour is any behaviour that pleases the infant, engages them in the interaction, or reduces their distress or discomfort (Crittenden, 2004), for example soothing vocalisations, humorous games, or responding to physical needs. Examples of controlling maternal behaviour include manipulating the infant's body against their will, grabbing toys from them or forcing them to hold or play with specific toys. Unresponsive behaviours include not responding to infant distress, or avoiding eye contact. A Cooperative infant is positively engaged with interaction, and is concentrating or playful (Crittenden, 2004). Difficult infants may display anger or blank facial expressions, with few vocalisations. Compulsive infant behaviour is used to prevent adult hostility, the infant feels compelled to comply with the adult (Crittenden, 2004). Passive infants are disengaged, avoiding eye contact or adult attempts to play. Further details of the scoring system are included

in Appendix 3. Coders generally achieve 80-85% agreement (Crittenden, 2004). The CARE index has been used in several studies using this client group (Crittenden, 2004).

Parenting Stress Index (Adapted with permission, from Abidin, 1995)

The Parenting Stress Index (PSI) is a self-report questionnaire measuring sources of stress within the parent-child system. The full PSI consists of 120 items, which contribute to seven Parental Domains and six Child Domains, with an additional Life Stress scale. The majority of items are scored on a 5-point scale, ranging from "strongly agree" to "strongly disagree". There are ten multiple-choice items, and the life stress questions are answered "yes" or "no". For each subscale, higher scores indicate more stress. The PSI has adequate internal consistency, test-retest reliability, and concurrent, construct, predictive and discriminant validity (Abidin 1990). It has been validated for use across samples varying in ethnicity and socioeconomic status (Hutcheson & Black, 1996). The PSI is designed for parents of children aged between 1 month and 12 years.

For the purposes of this study the PSI was adapted with permission from the publishers (Appendix 4). The full PSI is lengthy, so the following subscales were selected as being of most interest to the present study. The child subscales of Reinforces Parent, Mood, and Demandingness were administered, together with the parental subscales of Competence, Role Restriction and Attachment. The Life Stress scale was also included.

Computerised Cognitive Test Battery (CDR, 2002).

The cognitive test battery was produced by Cognitive Drug Research (CDR, 2005). CDR Ltd. assesses human cognitive function through producing cognitive testing batteries for clinical trials (CDR, 2002). Tests were selected by the Perinatal Research Team to produce a concise battery that represented the major domains of cognitive function, and reflected the cognitive abilities shown in previous research to be potentially impaired in patients with mental illness (e.g. Egeland et al., 2003, Fossati et al., 1999, Harvey, Le Bastard et al. 2004). Due to a lack of existing studies it was not possible to determine which domain would be most sensitive to both the presence of SMI and mother-infant interaction. It was therefore considered reasonable to administer a range of cognitive tests.

The computerised battery was also chosen for its ease of administration, and the reliability and accuracy of computerised recording. The battery is computerised, and was administered using a laptop computer, enabling data collection to take place in participants' homes. Responses are indicated by pressing "yes" and "no" buttons. The investigator gives standardised verbal instructions. The battery also contains visual analogue scales for participants to rate their mood and fatigue. This computerised battery has been used in many studies with this client group (e.g. Ferguson, Wesnes, & Schwartz, 2003), as well as in dementia drug trials (Wesnes, McKeith, Edgar, Emre, & Lane, 2005).

The battery consists of the following tests, administered in the order listed:

i) Practice Choice Reaction Time

Either the word YES or the word NO was presented on the computer screen. Participants were instructed to press the corresponding button as quickly as possible. This task yields measures of accuracy, reaction time, and number of outliers.

ii) Word Presentation and Immediate Recall

Fifteen words were presented one at a time on the screen, for the participant to remember. Immediately following presentation, participants were given one minute to write down as many of the words as they could remember. Accuracy of recall and number of errors are recorded.

iii) Picture Presentation

Twenty pictures were presented one at a time on the screen for the participant to remember.

iv) Simple Reaction Time

Participants were instructed to press the "yes" button whenever they saw the word YES presented on the screen.

v) Digit Vigilance

A single digit number (target) was presented on the right hand side of the screen, and remained there throughout the task. A series of digits were then presented one at a time in the centre of the screen. Participants were instructed to press the "yes" button as quickly as possible, whenever the number in the centre matched the target number. This yields measures of target detection, speed, and number of false alarms.

vi) Choice Reaction Time

This task is the same as the practice choice reaction time task.

vii) Rapid Visual Information Processing (RVIP)

A series of digits were presented one at time in the centre of the screen. Participants were instructed to press the "yes" button as quickly as possible whenever they saw either three even numbers in succession, or three odd numbers succession. This yields measures of target detection, speed, and number of false alarms.

viii) Spatial Working Memory

A picture of a house with nine windows appears on the screen. Four are illuminated and five are dark. The house remains on the screen for 15 seconds, and the participant is instructed to remember the position of the lit windows. The researcher confirms this by asking the participant to indicate the positions on a

diagram. A series of houses are then presented one at a time on the screen, each with one window illuminated. The participant is instructed to press the "yes" button as quickly as possible if they think that this window was illuminated in the original house, and to press "no" as quickly as possible if it was not. This task yields a percentage accuracy score and a mean reaction time for correctly identifying those windows that were illuminated in the original house, a percentage accuracy score and a mean reaction time for correctly identifying those that were not illuminated in the original house, and a sensitivity index (calculated according to Frey & Colliver, 1973).

ix) Numeric Working Memory

Five digits were presented, one at a time, for the participant to remember. Then a series of digits were presented, one at a time. For each digit, the participant was instructed to press the "yes" button as quickly as possible if it was the same as one of the five they were asked to remember, and to press "no" if it was not. This task yields a percentage accuracy score and a mean reaction time for correctly identifying to-be-remembered digits, a percentage accuracy score and a mean reaction time for correctly identifying new digits, and a sensitivity index.

x) Delayed Word Recall

Participants were given one minute to write down as many words as they could recall from the list presented previously. Accuracy of recall, and the number

of errors are recorded. Time elapsed between immediate and delayed recall was approximately 15 minutes.

xi) Delayed Word Recognition

The original words, plus 15 distracter words were presented one at a time in a randomised order. For each word, participants were instructed to press the "yes" button if they recognised it as being from the original list, and to press "no" as quickly as possible if it was not. This yields measures of accuracy, speed, and a sensitivity index.

xii) Picture Recognition

The original pictures, plus 20 distracters were presented one at a time in a randomised order. For each picture, participants were instructed to press the "yes" button if they recognised it as being from the original set, and to press "no" as quickly as possible if they did not. This yields measures of accuracy, speed, and a sensitivity index.

xiv) Factor Scores

The scores yielded by each test collapse onto five factors (Wesnes, Ward, McGinty, & Petrini, 2000); Power of Attention, Continuity of Attention, Quality of Episodic Memory, Quality of Working Memory, and Speed of Memory. Further details of how these are calculated can be found in Appendix 12. Briefly however,

the Power of Attention factor is composed of reaction times, the Continuity of Attention factor is a measure of response accuracy on the attentional tasks, Quality of Episodic Memory reflects the accuracy for memory tasks, Quality of Working Memory reflects performance on working memory tasks, and Speed of Memory Processing reflects participant response speed on the memory tasks. These factors were used to reduce the data and simplify the analysis. This methodology has been used in other studies (e.g. Scholey & Kennedy, 2004). The RVIP is not included in the factors and must therefore be analysed separately.

Procedure

After obtaining approval from the local regional ethics committee (Appendix 5), suitable mothers for the clinical group were identified through the Perinatal Service. They were initially approached by a member of the clinical team, usually their key-worker or the Consultant Psychiatrist, and offered information about the study (Appendix 7). They were then given three days to consider this before deciding whether or not to take part. If they wished to participate, the mother's contact details were passed to the researcher, who then arranged to visit the mother at home, to take written consent and collect the data. Written informed consent was taken in the presence of a member of the clinical team, who accompanied the trainee for safety reasons and to assist with data collection. A copy of the consent form can be found in Appendix 8. Mothers were offered the opportunity to have an additional adult such as a family member present. Only mothers considered well enough to give informed consent were approached. A member of the clinical team who knew the mother well made this decision.

Mothers for the control group were recruited through the NCT, colleagues or acquaintances of the researcher. After obtaining permission from the NCT, information about the study was e-mailed or posted to local group leaders, who distributed this to group members. Information sheets were also passed to colleagues and acquaintances (Appendix 9). Mothers then contacted the researcher to express their interest in the study. A time was then arranged for the researcher to visit the mother at home to take consent (Appendix 10) and collect the data.

After confirming that the mother understood the procedure and was still happy to participate, the 5-minute mother-infant interaction video was recorded. Infants were placed in a bouncy chair or similar, and the mother sat opposite. A mirror was positioned adjacent to the infant to reflect the mother's facial expressions, and the video camera was positioned behind the mother, facing the infant and the mirror. Mothers were asked to play with their infant in their usual way and were permitted to use toys if they wished. This procedure has been used in other mother-infant interaction studies (Murray et al., 1996). The 40-minute cognitive test battery was then administered before completion of the remaining measures (PSI for clinical group, PSI, PSQ, and EPDS for the control group). Mother were thanked for their participation, and offered the opportunity to ask questions of the trainee and to receive a summary of the research findings upon completion of the study. They were also offered a copy of their video. The video recordings were rated using the Crittenden Care Index (2004) by Maddalena Miele-Norton (St Mary's Hospital, London). She is fully trained in the methodology, having obtained a certificate of reliability, and was blind to the participant's group.

Data Analysis

Non-parametric tests for two-independent samples (Mann-Whitney) were used due to the small sample size, and the unequal groups. The significance level was set at $p < .05$. Due to the small sample size, it was considered that the study may lack sufficient statistical power to detect differences on all measures. It was therefore of interest to report trends approaching significance, with $p < .09$. This provides an indication as to the direction of results that may show significance with increased power. The second hypothesis was tested by a hierarchical regression model. This allowed for the determination of the proportion of variance in mother-infant interaction associated with presence or absence of SMI, and the extent to which this relationship was mediated by level of cognitive function.

Results

Group Differences

To address the first hypothesis a series of Mann-Whitney tests were performed. The median score and range for each measure is indicated in Table 2. Significant group differences were found for measures of mother-infant interaction, perceived maternal competence, and cognitive function.

(i) Mother-infant interaction and perceived maternal competence: On the CARE index variables there were significant differences between the two groups on the Maternal Sensitivity index, and the Infant Cooperative scale. Examination of the median scores indicates that mothers in the control group were rated as significantly

more sensitive, and their infants were rated as significantly more cooperative. Consistent with this finding, there was a significant difference between the groups on self-reported Parental Competence, indicating that the mothers with mental illness perceived themselves as significantly less competent than the control group.

(ii) Cognitive function: The groups were comparable on all measures of cognitive functioning, except for Speed of Memory Processing, where higher scores were indicative of poorer performance in the mothers with SMI. There was a non-significant trend for the SMI group to perform below the control group on the RVIP target detection.

Table 2

Comparison of the 2 groups on the CARE index, Parenting Stress Index, and the Cognitive Factor Scores.

Measure	Control Group	SMI Group	Group Differences
CARE Index			
Maternal Sensitivity	8.5 (3-13)	5 (4-9)	p= .041
Maternal Control	4 (1-11)	7.5 (4-9)	ns
Maternal Unresponsiveness	0.5 (0-5)	1.5 (0-5)	ns
Infant Cooperative	7.5 (3-13)	3 (3-8)	p= .018
Infant Compulsive	0 (0-7)	1 (0-4)	ns

(table continues)

Table 2 (continued)

Measure	Control Group	SMI Group	Group Differences
CARE Index cont.			
Infant Difficult	2 (0-7)	4 (2-5)	ns
Infant Passive	3 (0-6)	3.5 (1-8)	ns
Parenting Stress Index			
Parent- Competence	24.5 (16-28)	32.5 (22-59)	p= .013
Parent- Attachment	12 (9-17)	13.5 (8-21)	ns
Parent- Role restriction	18.5 (14-28)	21.5 (8-29)	ns
Life stress	7.5 (4-31)	18 (4-29)	ns
Infant- Reinforces parent	11 (6-15)	11.5(6-21)	ns
Infant- Demandingness	17(14-25)	17(10-27)	ns
Infant- Mood	10.5(5-14)	9.5 (7-19)	ns
Cognitive Measures			
Power of Attention	1153.1 (1058.4-1353.7)	1163.8 (1047.7-1954.2)	ns
Continuity of Attention	92.5 (90-95)	92.5 (89-94)	ns

(table continues)

Table 2 (continued)

Measure		Control Group	SMI Group	Group Differences
	Quality of Working Memory	1.9 (1.7-2)	1.9 (1.2-1.9)	ns
	Quality of Episodic Memory	233.3 (171.7-283.3)	200.8 (158.3-238.3)	ns
	Speed of Memory	3304 (2953-3682.5)	4145 (3180.5-4643.3)	p= .018
	RVIP % targets detected	81.25 (25-100)	50 (0-75)	ns trend p=.08
	RVIP Mean Reaction Time	447.3 (339.5-802.3)	505.3 (430.6-570.5)	ns
	RVIP No. False Alarms	0.5 (0-5)	1 (0-5)	ns
	RVIP Standard Deviation	102.9 (27.9-282.6)	156.5 (86.8-284.1)	ns

Note. For the CARE Index scales, a higher score indicates higher frequency or intensity of observations of behaviours indicative of that style. For the Parenting Stress Index, higher scores indicate poorer ratings. For the Cognitive Factors, higher scores indicate better performance, with the exception of Power of Attention and Speed of Memory, where higher scores equate to poorer performance. Median and range of scores are reported, and Mann-Whitney tests were used.

The Relationship between Mother-Infant Interaction and Cognitive Function

Due to the small sample size, this investigation was limited to those measures of mother-infant interaction and cognitive function that were found significantly to differentiate between the two groups at the univariate level, namely Maternal Sensitivity and Speed of Memory Processing. Maternal Sensitivity was chosen over Infant Cooperative because adult sensitivity is the central construct within the CARE index (Crittenden, 2004). Measures of maternal sensitivity have been included in many studies, with poorer maternal sensitivity repeatedly demonstrated in mothers with depression (Cohn et al., 1990), Murray et al., 1996) and with schizophrenia (Goodman & Brumley, 1990, Riordan et al., 1999). The PSI parental competence measure was not included in this analysis because the focus of this study was the relationship between maternal mental illness, maternal cognitive functioning and the quality of mother-infant interactions, rather than perceived parental competence.

In accordance with guidelines published by Baron and Kenney (1986), for Speed of Memory Processing to act as a mediator of mental illness, three criteria must be met: (i) both the mediator (Speed of Memory Processing score) and the predictor (presence/absence of SMI) must be related to the dependent variable (maternal sensitivity); (ii) there must be a relationship between the predictor (presence of SMI) and the mediator (Speed of Memory Processing); and (iii) after controlling for the effects of the mediator variable (Speed of Memory Processing), the relationship between mental illness and maternal sensitivity should be significantly reduced. As illustrated by Table 3, Spearman's Rho correlations (one-tailed) confirmed these criteria, indicating that reduced maternal sensitivity was associated with presence of

SMI, and with higher Speed of Memory Processing scores (indicative of worse performance). Speed of Memory Processing score was also significantly increased in mothers with SMI.

Table 3

Spearman's Rho correlations between the three variables of interest.

	Presence of SMI	Maternal Sensitivity	Speed of Memory Processing
Presence of SMI	-	-	-
Maternal Sensitivity	-.493*	-	-
Speed of Memory Processing	.568**	-.567**	-

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

A hierarchical multiple regression analysis was performed to indicate the proportion of variance in maternal sensitivity associated with presence of SMI, and the proportion additionally associated with Speed of Memory Processing. Multiple regression assumes multivariate normality of all variables, and usually requires larger samples. However, multiple regression is also a relatively robust method and

can be used if the residuals rather than the variables themselves are normally distributed (Howell, 1992). The standardised and unstandardised residuals were explored for the regression analysis and found to be normally distributed (as per Frost, Myers, and Newman, 2001).

Within the regression, the dependent variable was Maternal Sensitivity. Predictor variables were entered in the following order: model one: group – control (1) vs. SMI (2); model two: group, Speed of Memory Processing – raw score (higher scores indicate worse performance). Both model one ($F(1,17) = 4.92$, $p = .041$) and model two ($F(2,17) = 3.74$, $p = .048$) were significant. As can be seen from Table 4, group was a significant independent predictor of maternal sensitivity, accounting for 23.5% of the variance. Group, together with Speed of Memory Processing accounted for 33.3% of the variance in maternal sensitivity, but the additional contribution of Speed of Memory Processing (~10%) did not significantly increase the sensitivity of the model as a whole. Rather, the inclusion of this variable reduced the significance of the relationship between maternal sensitivity and presence/absence of mental illness, indicating that the relationship between group and maternal sensitivity was mediated by Speed of Memory Processing.

Table 4

Hierarchical multiple regression of predictors of maternal sensitivity

Model	R^2	R^2 change	F change	*p* change
1. Group	.235	.235	4.92	.041
2. Group, SMP	.333	.098	2.20	.159

Table 5

Correlation coefficients for the hierarchical regression analysis

	Model	B	SE B	β	*P* value
1.	Group	-2.750	1.239	-.485	.041
2.	Group	-.959	1.699	-.169	.581
	SMP	-.003	.002	-.445	.159

This suggests that cognitive impairment may be present in women with serious mental illness, in the domain of Speed of Memory Processing, and that as hypothesised this factor may mediate the relationship between presence/absence of SMI and reduced maternal sensitivity.

Discussion and Conclusions

This study replicates previous research (Cohn et al., 1990, Murray et al., 1996, Riordan et al., 1999) by demonstrating that mothers with SMI obtain significantly lower ratings of maternal sensitivity, as compared to mothers with no mental illness. Cognitive deficit was also demonstrated in the domain of Speed of Memory Processing. These findings are in partial agreement with the literature demonstrating reduced cognitive function in individuals with SMI (e.g. Addington et al., 2005, Porter et al., 2003); whilst impairment was demonstrated in the domain of Speed of Memory Processing, no group differences were found on the remaining cognitive factors, in contrast to what might be predicted on the basis of previous research. The present study offers an important extension to this literature by suggesting that the relationship between presence of SMI and poorer quality of mother-infant interaction is mediated Speed of Memory Processing. If replicated, this represents an alternative perspective to our understanding of mother-infant interaction in mothers with SMI, and perhaps provides a basis for the development of existing intervention strategies.

Mothers with SMI were rated as significantly less sensitive to their infants compared to control group mothers, and their infants were rated as significantly less cooperative than the offspring of mothers in the control group. Although patterns of mother-infant behaviours were not analysed at an individual level, the sensitive mother-cooperative infant is a recognised pattern (Crittenden, 1988). Therefore, it could be predicted that the control group infants would be rated as more cooperative, because their mothers were rated as significantly more sensitive. Additionally, mothers in the control group rated themselves as significantly more competent than

the mothers with SMI. Lower perceived competence ratings in the mothers with SMI suggest that they may have been aware of their lack of sensitivity in the mother-infant interaction task. Lower perceived competence may also have been reflective of the higher percentage of first-time mothers in the SMI group. Whilst the difference between the two groups in terms of the number of primiparous women was not significant, this issue should be considered in future studies. Moreover, the issue of whether self-reports of competence reflect actual parental competence is unknown, as this was not assessed. Self-report measures are influenced by the mother's emotional and cognitive state (Melhuish, Gambles, & Kumar, 1988). It may be that mothers in the SMI group lacked confidence in their parenting ability, or interpreted their parenting with the negative cognitive bias typically associated with depression (Beck, 1976). The potential discrepancy between self-report and objective measures has been highlighted. For example, Frankel and Harmon (1996) found that although mothers with depression in their study made significantly more negative evaluations about their parenting ability compared to mothers in the control group, their interactions and attachment relationships with their children were unimpaired. This suggests that mothers with depression may believe themselves to be less competent than they really are. The potential impact of this belief on mother-infant interactions was not considered in this study, although according to the principles of self-efficacy (Bandura, 1977), an individual's behaviour is strongly influenced by their belief in their ability to perform that behaviour. Mothers who believe that they are not very competent may behave in a less competent manner. This has received some support from Teti and Gelfand (1991). They found that the poorer the mother's sense of self-efficacy regarding her maternal role, the poorer her interactions with her infant.

The finding that mothers with SMI performed significantly poorer that controls on Speed of Memory Processing could be considered consistent with previous research demonstrating impaired processing speed in patients with depression and patients with schizophrenia (Egeland et al., 2003). However, the degree to which Speed of Memory Processing reflects more general processing speed is not clear. Speed of Memory Processing is derived from the reaction times to the memory tasks, and is therefore though to reflect the speed of retrieval from memory. If Speed of Memory Processing were analogous to more general processing speed, one would expect the Power of Attention factor to also be impaired in the SMI group, because this too is composed of reactions times (simple and choice reaction times, and digit vigilance reaction times). The fact that there were no group differences on this factor suggests that it is the reaction times specifically for memory tasks that are poorer in this sample of mothers with SMI. This would suggest some impairment in memory processing.

It is somewhat surprising not to find evidence of other cognitive impairments in mothers with SMI, as could be predicted from previous research (Addington et al., 2005, Egeland et al., 2003, Porter et al., 2003). Scores on the RVIP and the Quality of Working Memory factor demonstrated non-significant differences in the predicted direction, with the SMI group performing poorer than controls, but performance was comparable on the other three factors (Power of Attention, Continuity of Attention, and Quality of Episodic Memory). The lack of significant differences between the groups on these measures could be due to a number of reasons. Firstly, not all patients experience cognitive impairments (Kuperberg & Heckers, 2000). Secondly, several studies administering comprehensive neuropsychological assessments, whilst

demonstrating impairments in patients with mental illness, have also demonstrated comparable performance of patients and controls on a number of tasks, including measures of memory and executive function (Fossati et al., 1999, Nathaniel-James et al., 1996). Thirdly, the small sample size may have limited the study's power to accurately reflect group differences. A larger sample may have detected additional significant group differences, for example on the Quality of Working Memory factor or the RVIP, which indicated a trend for poorer performance in the SMI group. In addition, a more detailed measure-by-measure analysis, rather than using only the five factor scores may have revealed more trends. However, this would significantly increase the likelihood of making a Type 1 error, through running multiple comparisons on a small sample. Several studies use only the five factors to assess cognitive functioning (Scholey & Kennedy, 2004, Wesnes et al., 2000), with some combining the two "quality of memory" factors, resulting in four factors (Wesnes et al., 2000). However, the extent to which these factors relate to scores derived from other cognitive measures is unclear, which is important when making comparisons between this study and the findings of others that have used alternative methods of assessment. Future studies may utilise cognitive assessments that are more commonly used in studies of mental illness, such as the California Verbal Learning Test, Wisconsin Card-Sorting Test and Verbal Fluency (Quraishi & Frangou, 2002), or the Cambridge Automated Neuropsychological Test Battery (Cambridge Cognition, 2006), which would facilitate comparison with the published literature. Notwithstanding these caveats, the present study is of interest because it demonstrates the mediation of the relationship between maternal mental health and maternal sensitivity by a cognitive factor, specifically Speed of Memory Processing.

In support of previous findings (Cohn et al., 1990, Murray et al., 1996, Riordan et al., 1999), the presence of maternal mental illness (group) was found to be a significant predictor of maternal sensitivity, accounting for 23.5% of the variance. The inclusion of Speed of Memory Processing weakened the relationship between maternal sensitivity and maternal mental illness, suggesting that this relationship was mediated by speed of memory function. This supports the study's second hypothesis, by suggesting that the relationship between maternal mental illness and mother-infant interaction may be mediated by level of cognitive function.

Speed of Memory Processing may affect maternal sensitivity because infants are sensitive to the timing of their mother's responses to them. Murray (1986) demonstrated this by using a video procedure similar to that of the present study, but the mother's responses to her infant's behaviours were relayed to the infant via a life-size video screen, with a 30 seconds delay. The infants seemed confused by this disruption of the synchronicity typically present in mother-infant interactions, and displayed protest behaviours such as grimacing and lip-biting. Impairments in processing speed may mean that mothers respond more slowly to their infants, disrupting synchronicity and contingent reinforcement. Poorer interactional synchrony (Field, Healy, Goldstein, & Guthertz, 1990), and higher levels of negation of the infant's behaviour, including discordant responses (Murray et al., 1996) have been demonstrated in mothers with depression. Tronick (1989) argued that infants respond to such poorly coordinated interactions by developing a self-directed style of interacting, relying on self-regulatory behaviours such as thumb-sucking. This may interfere with the infants' interactions with people and objects in their environment, hindering their cognitive and social development (Tronick, 1989, Weinberg & Tronick, 1998). This behaviour may also reflect a less cooperative interactional

style on the part of the infant, which could explain why in the present study, infants of mothers in the SMI group were rated as significantly less cooperative than infants of mothers in the control group. This less cooperative, self-directed style may mean that it is more difficult for mothers to interact with their infants in a sensitive manner, and a cycle of worsening mother-infant interaction could potentially develop.

These findings are of relevance to professionals working with mothers with serious mental illness, suggesting that maternal level of cognitive functioning should be considered when mother-infant interactions are being evaluated. Any interventions aimed at improving the quality of mother-infant interactions should recognise the potential impact of cognitive functioning. Significant impairments in cognitive function may also affect the mother's ability to utilise any feedback given regarding her interactions with her infant, or to implement any strategies or suggestions offered by professionals. Given that speed of memory functioning was found to mediate the relationship between maternal mental illness and maternal sensitivity, cognitive remediation strategies may potentially have a positive impact on maternal sensitivity. It should be stressed that this proposal is currently speculative and requires empirical investigation.

The finding that level of cognitive function, in the domain of memory processing speed may mediate the relationship between presence of SMI and poorer quality mother-infant interaction is novel, and thus the basis for this conclusion requires consideration. In particular, this study was conducted with a small sample of mothers with SMI. The small sample size could be considered a limitation of this

study, but significant results were obtained in the predicted direction, suggesting sufficient statistical power. As may be anticipated, recruiting mothers with mental illness - particularly in the early post-partum period when many healthy women may be expected to be reluctant to participate in research - proved difficult, an issue acknowledged by others (Riordan et al., 1999), and reflected in the small samples of similar studies. For example, Riordan and colleagues included 8 women with schizophrenia and 18 with affective psychosis in their study (Riordan et al., 1999), but they did not have a control group of mothers with no mental illness. A control group is important as there may be considerable variation in the quality of mother-infant interaction in the general population, and this may be due to a number of factors including socio-economic status, a factor that was controlled for in the present study. Another possible limitation of the present study is the inclusion of participants with differing diagnoses, but this has also been a feature of other studies. For example, Riordan and colleagues' affective disorder group included patients with major depression, minor depression, and bipolar disorder (Riordan et al., 1999), and Weinberg and Tronick (1998) included mothers diagnosed with depression, panic disorder and obsessive-compulsive disorder in their study of mother-infant interactions. Heterogeneity of patient samples has been criticised (e.g. Porter et al., 2003), but may be justified in difficult-to-recruit samples; the influence of different mental illnesses may be examined in subsequent studies with larger sample sizes once a general effect is highlighted. Moreover, there are documented similarities between patient groups with respect to pattern of cognitive function, suggesting that the mediation may hold across diagnoses. Studies of cognitive functioning have found similar patterns of performance, demonstrating impairment in executive functioning, attention and memory in both patients with schizophrenia (Addington et

al., 2005, Nathaniel-James et al., 1996, Wang et al., 2005) and depression (Harvey, Le Bastard, et al., 2004, Porter et al., 2003): thus they do not consistently demonstrate significant differences between individuals with depression and those with schizophrenia. For example, Moritz and colleagues found that whilst both patients with depression and patients with schizophrenia performed poorer than a control group on measures of working memory and executive function, they did not differ from each other (Moritz et al., 2002). In addition, similar brain abnormalities have also been associated with both diagnoses (Häfner et al., 2005).

It is important to note that the model presented (Presence/absence of SMI, and Speed of Memory Processing) only accounts for 33.3% of the variance, leaving 66.6% unaccounted for. It is possible that other cognitive domains assessed account for a degree of the variance, and future studies with larger patient populations may have the power to demonstrate this. For the present study, a conservative approach was taken by only including those measures in the regression that significantly differentiated between the two groups at the first stage of statistical analysis, and the model is convincing in that it demonstrates a statistically significant effect in the expected direction. There are also many other factors not considered by this study, which may play a role (e.g. severity of illness, medication use, maternal experiences of their own parenting). However, the specific hypothesis of the current study was to demonstrate an effect on mother-infant interaction of level of cognitive function and presence of mental illness. Both these factors encompass a whole range of sub-variables, suggesting that it was appropriately conservative to enter into the model only those variables that showed significant group differences (e.g. maternal

sensitivity and speed of memory processing). Further research may examine the role of those variables not considered here.

This study relied on current psychiatric diagnosis to determine group membership, but future studies may also utilise a more structured interview such as the Structured Clinical Interviews for DSM-IV (SCID: First et al., 1997) for confirmation of diagnosis, as has been used in other studies (Egeland et al., 2003, Manoach et al., 2005). This may also be used for screening control groups. However, the increased time required to complete the interview, and the degree to which this interview is acceptable to control group mothers must be considered.

Finally, this study did not consider the possible influence of psychotropic medications on cognitive functioning. For example, tricyclic antidepressants may impair cognitive and psychomotor function (Lane & O'Hanlon, 1999), as can benzodiazepines (Peart, 2000), and older antipsychotic medications may cause psychomotor slowing (Palmer & Heaton, 2000) and impair reaction times (Blyler & Gold, 2000). This is particularly relevant to the present study, which demonstrated impaired memory processing speed in mothers with SMI. In contrast, some medications are regarded as potentially cognitive enhancing. These include a class of anti-depressants known as selective serotonin re-uptake inhibitors (SSRIs) (Rose et al., 2006), and many of the newer anti-psychotic medications (Harvey, Green, Keefe, & Velligan, 2004). However, the precise effects of antipsychotic medications on cognitive function remain controversial (Keefe, Seidman et al., 2004), and Lane and O'Hanlon (1999) argue that there is evidence of differential effects of SSRIs on cognitive function. The potential impairing or enhancing effects of medications

represent a potential confounding variable in cognitive functioning research, as it may mask the true nature of cognitive impairment. However, despite the lack of control for medications in studies of cognition in mental illness, a consistent profile of impairment has been demonstrated. In not considering the impact of medications on cognitive function, this study is in accordance with many studies that simply report the type of medications used by their participants (for example, Bozikas, et al., 2005, Harvey, Le Bastard, et al., 2004), without evaluating its impact on cognitive functioning. Studies that have controlled for the effects of medications have found no significant influence of benzodiazepines (Fossati et al., 2003, Moritz et al., 2002) or the anti-depressant Escitalopram (Rose, Simonotto, Spencer, & Ebmeier, 2006) on cognitive performance in individuals with mental illness. However, for future research it may be advisable to record participants' medication type and dosage. This information may then be subjected to statistical analysis in order to evaluate any relationship with cognitive functioning, the procedure already adopted by Fossati et al. (2003). In addition, researchers may wish to ask participants whether they are experiencing any side effects from their medication, such as headaches or fatigue, as these may also affect task performance.

In summary, this study found that mothers with SMI were rated as significantly less sensitive during interactions with their infant, as compared to mothers with no mental illness. Perhaps accordingly, their infants were rated as significantly less cooperative than the offspring of mothers in the control group. Mothers in the SMI group also rated themselves as significantly less competent than the control group, but the relationship with actual competence and with mother-infant interaction is likely to be complex and was not determined by the present study. This study also

demonstrated poorer cognitive functioning in mothers with SMI, but only for a measure of Speed of Memory Processing; a possible confound with medication should be investigated. Notwithstanding these factors, it is of interest that the relationship between maternal mental illness and maternal sensitivity was mediated by Speed of Memory Processing. If replicated, this finding may serve to extend our understanding of the nature of mother-infant interaction in mothers diagnosed with a mental illness, and perhaps pave the way towards the further development of intervention strategies designed to improve the quality of mother-infant interactions, promoting greater self-efficacy in mothers and protecting vulnerable infants.

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List of Appendices

Appendix 1

Guidelines for publication in the Journal of Reproductive and Infant Psychology

Journal of Reproductive and Infant Psychology

Instructions for Authors:

Click here to check your article status

Note to Authors:** please make sure your contact address information is clearly visible on the **outside** of all packages you are sending to Editors.

Journal of Reproductive and Infant Psychology welcomes reports of original research and creative or critical review articles which make an original contribution. Articles should not currently be submitted for publication elsewhere.

Topics of interest to the journal include medical, behavioural, cognitive, affective, dynamic, psychological, societal and social aspects of: fertility and infertility; menstruation and menopause; pregnancy and childbirth; antenatal preparation; motherhood and fatherhood; neonatology and early infancy; infant feeding; early parent-child relationships; postnatal psychological disturbance and psychiatric illness; obstetrics and gynaecology including preparation for medical procedures; psychology of women.

The journal also publishes brief reports, comment articles and special issues dealing with innovative and controversial topics. A review section reports on new books and training material.

Studies of both human and animal subjects are welcome.

Papers should be sent in the first instance to the Editor:

Co-Editors:

Dr. O.B.A. van den Akker, Reader in Health Psychology, Psychology Group, Life & Health Sciences, Aston University, Aston Triangle, Birmingham, B4 7ET, UK

Dr. Margaret Redshaw, National Perinatal Epidemiology Unit, University of Oxford, Oxford, OX3 1LF, UK

Associate North American Editor: **John Worobey**, Department of Nutritional Sciences, Rutgers University, New Brunswick, NJ 08903-0270, USA

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Associate Continental European Editor: **Prof. Dr Axel Schoelmerich**, Fakultaet fur Psychologie, Ruhr-Universtat, Bochum, D-44780, Bochum, Germany

Authors are encouraged to submit manuscripts electronically. Electronic submissions should be sent as email attachments using a standard word processing program, such as MSWORD or PDF. If email submission is not possible, please send an electronic version on disk along with three paper copies together with one set of high quality figures for reproduction.

Contributions should be as concise as possible and should not normally exceed 5000 words or the equivalent lineage including tables and figures. The title should be brief but precise. Each paper should be accompanied by an abstract of not more than 200 words.

Papers should be typed on A4 or equivalent paper, on one side, double spaced with margins of not less than 3.5 cm. Sheets should be numbered consecutively at the head. The top copy and two good copies should be submitted.

Papers are refereed anonymously. The author's name and address should therefore appear under the title on a separate page. The title and abstract should appear on the first page of text. Authors who wish to ascertain in advance the criteria on which submissions are judged may obtain a copy of the blank referee's form from the editors.

Tables should be typed double spaced on separate sheets, or spaced sufficiently to be distinct in the case of small tables. They should be numbered in sequence in arabic numerals and referred to in the text as 'Table 1' etc. Large tables of more than six lines should be titled in order to make the contents comprehensible independently of the text.

Diagrams, graphs, drawings and half-tone illustrations should be on a separate sheet labeled 'Fig. 1' and so forth. Each sheet should carry at the top the title of the article. Where possible they should be submitted as artwork ready for photographic reproduction, larger than the intended size. Where more than one figure is submitted, they should as far as possible be to the same scale. When submitting articles on disk (see below) figures should be supplied as separate TIFF or EPS files if possible.

References in the text should cite the author's name followed by the date of publication unless there are more than two authors where only the first author's name should be given followed by **'et al.'**. References should be listed at the end of the paper in alphabetical order by first author, but including all authors, in the following format with titles of articles, books and journals given in full.

Berryman, J.C. and Windridge, K.C. (1993) Pregnancy after 35: a preliminary report on maternal-fetal attachment. **Journal of Reproductive and Infant Psychology, 11:** 169-174.
Berryman, J.C., Thorpe, K.J. and Windridge, K.C. (1995) **Older Mothers: conception, pregnancy and birth after 35**. Pandora, London.
Reid, M. (1990) Prenatal diagnosis and screening. In Garcia, J., Kilpatrick, R. and Richards, M.P.M. (Eds), **The Politics of Maternity Care**, pp. 300-324. Oxford University Press, Oxford.

SI units should be used for all measurements. Imperial measurements may be quoted in brackets. Where studies involve small numbers of subjects, both numbers and percentages of groups should be given.

Authors are advised to avoid sexist sentiments and language, except insofar as these form part of a study.

After notification of acceptance of a paper, authors should, if possible, send a copy of the final version in PC format as a word-processed document on a 3.5" or 5.25" floppy disk (Apple Macintosh formats can be accepted, but not Amstrad's Locoscript on CF2 disks) to the accepting editor.

Manuscripts and correspondence concerning publication of articles will only be kept for 3 years by the Editors.

Page proofs will be sent to the author submitting each article. Correction of typographical errors only will be permitted at this stage. Textual alterations may be charged to authors in exceptional circumstances.

Early Electronic Offprints: Corresponding authors can now receive their article by e-mail as a complete PDF. This allows the author to print up to 50 copies, free of charge, and disseminate them to colleagues. In many cases this facility will be available up to two weeks prior to publication. Or, alternatively, corresponding authors will receive the traditional 50 offprints. A copy of the journal will be sent by post to all corresponding authors after publication. Additional copies of the journal can be purchased at the author's preferential rate of £15.00/$25.00 per copy.

For further guidance on general aspects of manuscript preparation authors should consult APA or BPS Manuals for Contributors.

Appendix 2

Descriptions of the Edinburgh Postnatal Depression Scale, the Psychosis Screening Questionnaire, and the Brief Psychiatric Rating Scale

Edinburgh Postnatal Depression Scale (Cox et al., 1987)

This ten-item self-report scale was used to screen for depressive symptoms in the control group. Participants are required to read 10 statements and underline the response that best fits their experience. The maximum score is 30 with a clinical threshold of 12 (Warner, Appleby, Whitton, & Faraghen, 1996). Cox et al. (1987) calculated sensitivity at 85%, specificity 77%, positive predictive value 83%, split-half reliability .88 and α=.87.

Psychosis Screening Questionnaire (Bebbington & Nayani, 1995)

This was used to screen for the presence of psychotic symptoms in the control group. The researcher uses the 5 probe questions plus secondary questions, to ask participants about hypomania, thought insertion, paranoia, strange experiences and hallucinations (Johns et al. 2004). In the original study, sensitivity was calculated at 96.9%, specificity 95.3%, positive predictive value 91.2%, and negative predictive value 98.4% (Bebbington & Nayani, 1995). It has been used with both clinical and non-clinical populations (e.g. Johns et al. 2004).

Brief Psychiatric Rating Scale- Expanded (Lukoff, Nuechterlein, & Ventura, 1986)

This recorded current symptoms in the clinical group and was completed by the participant's key-worker (Community Psychiatric Nurse or Consultant Psychiatrist). The BPRS-E consists of 24 constructs, such as "hallucinations" and "unusual thought content", each rated on a 7 point scale ranging from "not present" to "extremely severe". The BPRS-E yields a total score by summing the scores for each item. Factor analysis of the BPRS-E is conflictual, some authors proposing four or five (Ventura et al., 1993) or six (Mogge, LePage, Del Ben, & Murphy, 2002) solutions. Therefore, only total scores were used. The BPRS is widely known and used both clinically (Varner, Chen, Swann, & Moeller, 2000) and within research studies, and provides a sensitive measure of psychiatric symptoms with good inter-rater reliability (Roncone, et al., 1999). It is sensitive to symptom change (Ventura et al., 1993), important because a follow-up study was planned by the service.

Appendix 3

Details of the Crittenden Care Index Scoring (summarised from Crittenden, 2004).

Scoring (see Crittenden, 2004 for full details of the scoring system)

Each of the seven aspects of behaviour (e.g. facial expression) has two points allocated to it, out of a total of 14. Each of the number/letter combinations (items) on the scoring sheet represents descriptions and examples of behaviour (see the CARE index manual for these descriptions). There are four items for each of the seven scales. Mothers and infants are scored according to which of these descriptions best fits the observed interaction. Two points are allocated for each scale; these can be allocated to one item or split across two, whichever best reflects the observed interaction. The points for each scale are added to yield the seven subscale scores, three for adults (Sensitivity, Control, and Unresponsiveness) and four for infants (Cooperative, Compulsive, Difficult, and Passive). The sum of the subscale scores for adults and, separately, for infants is 14.

Maternal scoring

The top line of the scoring chart yields the mother's scores. For each of the seven aspects of interactional behaviour, the first item (e.g. 1) corresponds to the

Sensitivity score. The second and third items contribute to the Control score. There are two types: pseudo-sensitive, covertly hostile (the "a" items) and overtly hostile (the "b" items). The forth item contributes to the Unresponsive score. Each aspect of interactional behaviour has two points allocated to it. Two points can be given to one item, or divided between two items. The sum of the Sensitivity, Control and Unresponsive scores will be 14.

Example

Facial Expression: Mother

Score item 1 for attentive, responsive facial expressions.
Score item 2a for incongruous or unchanging facial expressions.
Score item 2b for hostile or angry expressions.
Score item 3 for impassive expressions.

Infant scoring

Infants are scored in the same way, using the second line of the chart. For each of the seven aspects of interactional behaviour, the first item contributes to the Cooperative score, the second to the Compulsive score, the third to the Difficult score, and the forth to the Passive score. Each of the seven scales is again allocated two points, which can be assigned to one item or divided between two. The total again will be 14.

Example

Score item 1 for infant's who attend to the activity, with no avoidance of the mother.

Score item 2a for rigid, masked expressions, avoidance of eye contact.

Score item 2b for angry facial expressions.

Score item 3 for inattentive or vacant expressions.

Appendix 4

Appendix 4

PAR
Psychological
Assessment
Resources
16204 N. FLORIDA AVENUE
LUTZ, FLORIDA 33549
Tel: (813) 968-3003
Fax: (813) 968-2598
www.parinc.com

Sent Via Email: a.e.mayers@soton.ac.uk

December 15, 2005

Dr. Alain Gregoire
Perinatal Research Team
The Lodge/Tatchbury Mount
Calmore Southampton
SO40 2RZ England

Dear Dr. Gregoire:

In response to your recent request, permission is hereby granted to you to reproduce up to a total of 40 copies of the 66 items in the Reinforces Parent, Demandingness, Mood, Competence, Attachment, Role Restriction and Life Stress subscales of the Parenting Stress Index (PSI) for use in your study on "Cognition and Mother-Infant Interactions". If additional copies are needed, it will be necessary to write to PAR for further permission.

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DATE: 23 12 05.	DATE:
	PAYMENT RECEIVED:

SIGNATURE OF QUALIFIED INDIVIDUAL REQUIRED:

I hereby certify that I am qualified to use and interpret the results of these tests as recommended in the *Standards for Educational and Psychological Testing*, and I assume full responsibility for the proper use of all materials used per this Agreement.

BY:

Printed Name: A. G. Kinmons

Appendix 5

Appendix 5

SOUTHAMPTON & SOUTH WEST HAMPSHIRE
RESEARCH ETHICS COMMITTEES (A
1ST Floor, Regents Park Surgen
Park Street, Shirley
Southampton
Hampshire
SO16 4RJ

Tel: 023 8036 2466
023 8036 3462
Fax: 023 8036 4110

Email: GM.E.hio-au.SWHRECA@nhs.net

23rd September 2005

Dr Alan Gregoire
Consultant and Honorary Senior Lecturer in Psychiatry
Perinatal Mental Health Research
Maples Building
Tatchbury Mount
Calmore
Southampton
SO40 2RZ

Dear Dr Gregoire

Study title: The effect if cognitive function in women with and without mental health problems on mother/infant interaction.
REC reference: 024/04/t
Protocol number: n/a
EudraCT number: n/a

Amendment number: 2
Amendment date: September 2005

The above amendment was reviewed at the meeting of the Sub-Committee of the Research Ethics Committee held on 16th September 2005.

Ethical opinion

The Committee noted that the study title had been changed for the information sheets and consents form, but still felt this to be over complicated. They would suggest the following simplified title "How mothers relate to their babies" with the full study title also being included. Revised copies of the information sheets and consent forms should be submitted for the file.

The members of the Committee present gave a favourable ethical opinion of the amendment on the basis described in the notice of amendment form and supporting documentation.

Approved documents

The documents reviewed and approved at the meeting were:

Notice of Substantial Amendment – amendment 2 dated September 2005
Psychosis Screening Questionnaire (PSQ Bebbington & Nayani 1995)
Edinburgh Post Natal Depression Scale (EPDS)
GP Letter – version 2
Information Sheet (Control) – version 2
Information Sheet (SMI) – version 3
Consent – version 3
CV for Joanna Steadman

Membership of the Committee

The members of the Ethics Committee who were present at the meeting are listed on the attached sheet.

SL32 Favourable opinion of amendment
Version 3, June 2005

Research governance approval

All investigators and research collaborators in the NHS should notify the R&D Department for the relevant NHS care organisation of this amendment and check whether it affects research governance approval of the research.

Statement of compliance

The Committee is constituted in accordance with the Governance Arrangements for Research Ethics Committees (July 2001) and complies fully with the Standard Operating Procedures for Research Ethics Committees in the UK.

024/04/t	Please quote this number on all correspondence

Yours sincerely

Mrs Sharon Atwill
Committee Co-ordinator

E-mail: GM.E.hio-au.SWHRECA@nhs.net

Copy to: Andy Mayers
Research Co-ordinator
Research Department
Maples Building
Tatchbury Mount
Calmore
Southampton
SO40 2RZ

Enclosures *List of names and professions of members who were present at the meeting and those who submitted written comments*

Southampton and South West Hampshire
Research Ethics Committees (A)

Attendance at Sub-Committee of the REC meeting on 16 September 2005

Committee Members:

Name	*Profession*	*Present?*	*Notes*
Mr Edward Carter	Vice-Chair	Yes	
Dr A Moorman	LREC member	Yes	

Appendix 6

Appendix 6

University of Southampton

School of Psychology

University of Southampton
Highfield Southampton
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Fax +44 (0)23 8059

18 August 2005

Joanna Steadman
Department of Clinical Psychology
University of Southampton
Highfield
Southampton SO17 1BJ

Dear Joanna,

Re: An exploratory study of cognition in mothers with mental illness and its relationship to mother-infant interaction

I am writing to confirm that the above titled ethics application was approved by the School of Psychology Ethics Committee on 18 August 2005.

Should you require any further information, please do not hesitate in contacting me on 023 8059 3995.

Please quote approval reference number CLIN/03/87.

Yours sincerely,

Kathryn Smith
Secretary to the Ethics Committee

Appendix 7

Information Sheet for the SMI group

Hampshire Partnership NHS
NHS Trust

Perinatal Mental Health Researc
The Lodg
Tatchbury Mour
Calmor
Southampto
SO40 2R

Tel: 023 8087 434
Fax: 023 8087 436

The Effect of Cognitive Function in Women with and without Mental Health Problems on Mother/Infant Interaction

Short title: How mothers relate to their babies

Subject Information – please read carefully

You are being invited to take part in a research study. Before you decide, it is important for you to understand why the research is being done and what it will involve. Please take time to read the following information carefully and discuss it with friends, relatives or your GP, if you wish. Ask if there is anything that you do not understand or if you would like more information. Take time to decide whether or not you wish to take part. Should you decide to take part in the study, you should keep this document in a safe place in case you need to look at it again.

A group called Consumers for Ethics in Research (CERES) publish a leaflet entitled 'Medical Research and You'. This leaflet gives more information about medical research and looks at some questions you may want to ask. A copy may be obtained from CERES, PO Box 1365, London N16 0BW.

Thank you for reading this.

What is the purpose of this study?

We are trying to understand what particular difficulties and needs women who have suffered mental health problems in the past have in looking after young children.

Why have I been chosen?

You have been asked to take part because you have had difficulties with your mental health in the past. The Southampton Perinatal Mental Health Service is interested in understanding how this might influence your thoughts and how you and your baby interact (talk and play with each other).

Do I have to take part?

Participation in this trial is voluntary. It is up to you to decide whether to take part. If you decide to take part, you will be given this information sheet to keep and be asked to sign a consent form. If you decide to take part, you are still free to withdraw at any time and without giving a reason. This will have no influence on your future medical care.

What will happen to me if I take part?

If you agree to take part, you will be asked to undertake some simple procedures on two occasions: within the next few days and again when your baby is six month's old. This will involve videoing you and your baby for about 5 minutes, the completion of a scale that measures your perceptions of parenting, and some tests that will evaluate how you are thinking (that test will only be done once). You will be asked to complete a form that measures how you perceive your relationship with your baby and a form about your current mental health will also be completed by your doctor. Information relevant to the study will also be obtained from your medical records by the researcher.

You may wish to have a copy of the recording of you and your baby. If so please ask the researcher to make a copy for you. We may also wish to contact you, one year after the study, to see if there is any change in our observations; we will ask your permission for this.

What will happen to the videotape/DVD?

A copy of the recording will be kept up to 15 years after the study. This may be used for education and training purposes within the perinatal service. You will be asked to give your permission for this on the consent form. Also, with your agreement, it may also be used in future research. This will be coded so you or your baby will not be identified by name.

What are the disadvantages of taking part?

You have to give up about 50 minutes of your time for the first session, much less for the second session.

What are the possible benefits of taking part?

By taking part in this study you will be helping us to understand the needs and difficulties of women in your situation.

Will my taking part in the research be kept confidential?

Your identity in this study will be treated as confidential. If you agree to take part in the research, you agree that any of your medical records may be inspected by the research team. They may also be looked at by people from the Ethics Committee and from regulatory authorities to check that the study is being carried out correctly. Your name and records will not be made known publicly.

During the study certain personal information will be collected. This information will be general personal information (e.g. initials, date of birth) and medical information (e.g. medical history, physical and mental health condition). This information will be collected and processed in compliance with the applicable privacy laws.

However, if the data highlights information which may be of concern to the health and safety of a participant or her child, the patient's details will be decoded and appropriate care initiated or child protection policies followed.

You have the right to request disclosure of any personal data, that is maintained in an identifiable form and the right to request rectification of any data that is not correct and/or complete.

Who has reviewed the study?

This study has been reviewed and approved by the Southampton and South West Local Ethics Committee.
In case you need additional information concerning the study and your rights and obligations, you may contact:

Investigators name: **Dr Gregoire** **Tel:** **023 8087 4348**

At any time during the study.

If you consent to take part, you will be given a copy of this information sheet to keep as well as a copy of your signed consent form.

Thank you for taking part in this study

Hampshire Partnership NHS
NHS Trust

Appendix 8

The Effect of Cognitive Function in Women with and without Mental Health Problems on Mother/Infant Interaction

Perinatal Mental Health Research
The Lodge
Tatchbury Mount
Calmore
Southampton
SO40 2RZ

Tel: 023 8087 4348
Fax: 023 8087 4360

Short title: How mothers relate to their babies
Consent Form for Study Participants

Centre identification.....

Patient identification for this study.....

Name of researcher.....

Please initial line to indicate agreement

1. I confirm that I have read and understood the Subject Information sheet for the above study, and I have had enough time and opportunity to ask questions. I have received sufficient information and received satisfactory answers to my questions. _____

2. I understand that my participation is voluntary and that I am free to withdraw at any time without giving a reason. _____

3. I agree to my GP being informed of my participation. _____

4. I agree that my medical records may be reviewed by research personnel and by members of the Ethics Committee and other regulatory bodies. _____

5. I understand that the information I provide to this study may be looked at by responsible individuals. Such individuals are those representing ethics committees or research governance, whose role is to protect the interests of research participants. I give permission for these individuals to have access to my information. I have been told that my identity will be kept confidential. _____

6. I consent to a short video recording made of my baby and myself. I understand that this video will be securely and confidentially stored (for a period of 15 years before being destroyed) according to guidelines set out by the NHS Central Office for Research Ethics Committees (COREC). _____

7. I have been given a copy of this signed and dated Subject Informed consent and Subject Information sheet. _____

8. I consent that a member of the research team contact me 1 year after study completion _____

9. I consent to take part in this study. _____

I would like to have a copy of my video ☐

Name of participant	Date	Signature
Name of person taking consent	Date	Signature
Name of researcher	Date	Signature

Hampshire Partnership NHS
NHS Trust

Perinatal Mental Health Researc
The Loc
Tatchbury Mo
Calm
Southamp
SO40 2

Tel: 023 8087 4
Fax: 023 8087 4

Appendix 9

Information Sheet for the Control Group

The Effect of Cognitive Function in Women with and without Problems on Mother/Infant Interaction

Short title: How mothers relate to their babies
Subject Information – please read carefully

You are being invited to take part in a research study, as part of the **"Healthy Controls Group"**, to compare to our group of mothers who are suffering from severe mental illness. Before you decide, it is important for you to understand why the research is being done and what it will involve. Please take time to read the following information carefully and discuss it with friends, relatives or your GP, if you wish. Ask if there is anything that you do not understand or if you would like more information. Take time to decide whether or not you wish to take part. Should you decide to take part in the study, you should keep this document in a safe place in case you need to look at it again.

A group called Consumers for Ethics in Research (CERES) publish a leaflet entitled 'Medical Research and You'. This leaflet gives more information about medical research and looks at some questions you may want to ask. A copy may be obtained from CERES, PO Box 1365, London N16 0BW.

It is important that you remember that you would be included in our healthy controls group. Thank you for reading this.

NB. We are looking to recruit mothers with a baby aged 6 months or younger.

What is the purpose of this study?
We are trying to understand what particular difficulties and needs women who have suffered mental health problems in the past have in looking after young children.

Why is a healthy control group needed?
In order to fully understand what we observe in the group of mothers with severe mental illness, we need to compare each of the study measures in a group of mothers who are not demonstrating those problems. This will help us identify the extent of those problems and may give some information about how we might help them.

Do I have to take part?
Participation in this trial is voluntary. It is up to you to decide whether to take part. If you decide to take part, you will be given this information sheet to keep and be asked to sign a consent form. If you decide to take part, you are still free to withdraw at any time and without giving a reason.

What will happen to me if I take part?
If you agree to take part, the researcher will arrange to visit you at home, at a time convenient to you, where you will be asked to undertake some simple procedures. This will involve videoing you and your baby for about 5 minutes, and completing some tasks on a computer that will evaluate how you are thinking. You will be asked to complete a form that measures how you perceive your relationship with your baby. The researcher will ask you questions about any history of mental health problems.

You may wish to have a copy of the recording of you and your baby. If so please ask the researcher to make a copy for you. We may also wish to contact you, one year after the study, to see if there is any change in our observations; we will ask your permission for this.

What will happen to the videotape/DVD?

A copy of the recording will be securely and confidentially stored (for a period of 15 years before being destroyed) according to guidelines set out by the NHS Central Office for Research Ethics Committees (COREC).

What are the disadvantages of taking part?

You have to give up about 1 and a half hours of your time for the session.

What are the possible benefits of taking part?

By taking part in this study you will be helping us to understand the needs and difficulties of mothers with severe mental illnesses.

Will my taking part in the research be kept confidential?

Your identity in this study will be treated as confidential. If you agree to take part in the research, you agree that any information you give may be studied by the research team. It may also be looked at by people from the Ethics Committee and from regulatory authorities to check that the study is being carried out correctly. Your name and records will not be made known publicly, although we will write to your GP, as a matter of courtesy, to confirm that you are taking part in study.

During the study certain personal information will be collected. This information will be general personal information (e.g. initials, date of birth) and medical information (e.g. medical history, and physical health condition); we will also need to confirm that you not suffering from mental illness. This information will be collected and processed in compliance with the applicable privacy laws.

You have the right to request disclosure of any personal data, that is maintained in an identifiable form and the right to request rectification of any data that is not correct and/or complete.

Who has reviewed the study?

This study has been reviewed and approved by the Southampton and South West Local Ethics Committee.
In case you need additional information concerning the study and your rights and obligations, you may contact:

Investigators name: **Dr Gregoire** **Tel:** **023 8087 4348**

At any time during the study.

If you consent to take part, you will be given a copy of this information sheet to keep as well as a copy of your signed consent form.

If you are interested in taking part in this study please contact

Jo Steadman, Trainee Clinical Psychologist on 023 8087 4348

(Perinatal Mental Health Service) or email me at

js1503@soton.ac.uk

Hampshire Partnership NHS Trust

Appendix 10

Perinatal Mental Health Researc
The Loc
Tatchbury Mo
Calm
Southamp
SO40 2

Tel: 023 8087 4
Fax: 023 8087 4

The Effect of Cognitive Function in Women with and without Mental Health Problems on Mother/Infant Interaction

Short title: How mothers relate to their babies

Consent Form for Study Participants

	Please initial line to indicate agreement
1. I confirm that I have read and understood the Subject Information sheet for the above study, and I have had enough time and opportunity to ask questions. I have received sufficient information and received satisfactory answers to my questions.	_____
2. I understand that my participation is voluntary and that I am free to withdraw at any time without giving a reason.	_____
3. I agree to my GP being informed of my participation.	_____
4. I understand that the information I provide to this study may be looked at by responsible individuals. Such individuals are those representing ethics committees or research governance, whose role is to protect the interests of research participants. I give permission for these individuals to have access to my information. I have been told that my identity will be kept confidential.	_____
5. I consent to a short video recording made of my baby and myself. I understand that this video will be securely and confidentially stored (for a period of 15 years before being destroyed) according to guidelines set out by the NHS Central Office for Research Ethics Committees (COREC).	_____
6. I have been given a copy of this signed and dated Subject Informed consent and Subject Information sheet.	_____
7. I consent that a member of the research team contact me 1 year after study completion	_____
8. I consent to take part in this study.	_____

I would like to have a copy of my video ☐

Name of participant	Date	Signature
Name of person taking consent	Date	Signature
Name of researcher	Date	Signature

Appendix 11

Calculation of the Cognitive Factor Scores

These calculations were performed by CDR Ltd. using Microsoft Office Excel software. All reaction time scores are mean reaction times for the task. Accuracy scores are percentage accuracy for the task. Sensitivity Indexes are a calculation combining the accuracy of detecting the target stimuli (original information) with the accuracy of correctly rejecting the distractor stimuli (new information) with an adjustment for chance. These were calculated as per Frey and Colliver (1973), again by CDR Ltd.

Power of Attention

Simple Reaction Time Score + Choice Reaction Time Score +Digit Vigilance Reaction Time Score

Continuity of Attention

(Digit Vigilance Accuracy Score * 0.45) + (Choice Reaction Time Accuracy score) – Digit Vigilance No. False Alarms Score

Quality of Working Memory

Spatial Working Memory Sensitivity Index + Numeric Working Memory Sensitivity Index

Quality of Episodic Memory

(Delayed Recall Accuracy Score (original information) + Delayed Recall Accuracy Score (new information) – 100) + (Delayed Picture Recognition Accuracy Score (original information) + Delayed Picture Recognition Accuracy Score (new information - 100) + ((Immediate Recall Accuracy – Immediate Recall Error Score) * 100/15) + (Delayed Recall Accuracy score * 15/100 – Delayed Recall Error score) * 100/15)

Speed of Memory Processing

Spatial Working Memory Reaction Time + Numeric Working Memory Reaction Time + Delayed Recall Reaction Time + Delayed Picture Recognition Reaction Time