

**MATERNAL MOOD AND CHILD ADJUSTMENT PROBLEMS:
MUTUAL RISK AND RESILIENCE**

by
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for the degree of Doctor of Philosophy

at

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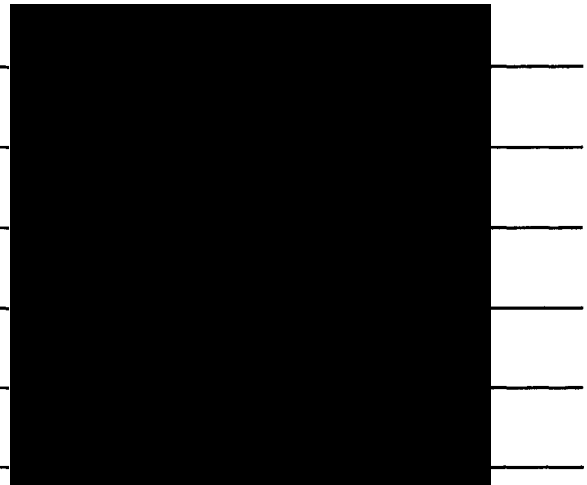
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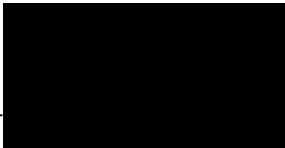
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ABSTRACT

Depressed mood in mothers and adjustment problems in children are common conditions that share mutual influences. Previous research is inconclusive about whether maternal depression tends to precede or follow child adjustment problems and whether their antecedent-consequence conditions vary across domains of child functioning, dimensions of maternal mood, and between frequent fluctuations in functioning and stable underlying symptomatology. Two studies were undertaken to study mutual influences and temporal relations between maternal depressed mood and adjustment problems in children. In Study 1, maternal depressive symptoms and internalising and externalising child symptoms were studied in three waves of panel data collected over four years from a nationally representative sample of Canadian families ($N = 20,428$). Cross-lagged panel correlations showed that maternal depressive symptoms tended to precede, more than follow, child aggression and hyperactivity but tended to follow, more than precede child emotional problems. Logistic regression showed bidirectional risk between maternal mood and child adjustment, after earlier symptoms were statistically controlled. In Study 2, 30 mothers provided daily reports during eight consecutive weeks on their mood and their child's inattention/impulsiveness and opposition/defiance (623 pooled observations). Pooled time-series analyses showed synchronous fluctuations in difficult child behaviour and maternal distress. Time-lagged models indicated that antecedent-consequence conditions in maternal mood and externalising child behaviour varied according to the type of maternal mood (e.g., depression, frustration, fatigue) and child behaviour (e.g., hyperactivity, defiance) involved and the severity of child behaviour. Results were interpreted in the context of mechanisms that mediate risk and resilience in the intergenerational transfer of psychopathology. Findings implicated the emotional functioning of mothers in the aetiology of child maladjustment and the deleterious impact of disruptive child behaviour on depressed mood in mothers, underscoring the role of family influences in the assessment and treatment of common psychological problems.

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INTRODUCTION

Depression in mothers and child emotional and behavioural problems are common behavioural health problems that tend to coexist in families. Maternal depression puts children at risk for abnormal development and adjustment problems and, conversely, emotional and behavioural problems in children have detrimental effects on maternal functioning. In prevention programs that aim to reduce either the risk to children attributed to depression in mothers or the risk to mothers attributed to disruptive child behaviour, and in clinical interventions for psychological problems in mothers or in children, it is advantageous to consider how psychopathology is shared within families and how problems in one family member can influence the health of other family members. The existence of transactional, bidirectional influences between maternal and child functioning is well established, but because they have rarely been studied together in longitudinal studies, relatively little is known about their typical temporal sequence.

The following sections of this thesis include a review of the relevant epidemiological literature on maternal depression and children behavioural problems and research that has attempted to parse mechanisms of risk and resilience shared by mothers and children. These mechanisms are relevant to conceptualising mutual influences on maternal and child functioning and their antecedent-consequence conditions. The objectives and research hypotheses

of the thesis are outlined thereafter. Two studies were undertaken to examine the magnitude and temporal course of bidirectional influences between maternal mood and child behaviour. In each of these studies, antecedent-consequence conditions of the two constructs were studied using complementary but distinct methodological approaches. The results of these studies follow with implications for further research and public health and clinical interventions for families.

Epidemiology of Maternal Depression

Depression afflicts some 10 to 20 percent of women at any point in time (Kringlen, Torgersen, & Cramer, 2001) and about a third of all women at some point during their lifetime (Kendler & Prescott, 1999). Depression is a highly recurrent condition with over 80 percent of depressed individuals experiencing more than one depressive episode (Belsher & Costello, 1988). More than half of all depressed individuals have been found to relapse within two years of recovery (Keller & Shapiro, 1981), and individuals with three or more previous depressive episodes have a relapse rate as high as 40 percent within 12 to 15 weeks after recovery (Keller et al., 1992).

Although depression is a common and recurrent disorder, there is wide variability in the extent to which it interferes with social functioning over time. Some people have only a single episode whereas others are chronically handicapped by repeated episodes (Duggan, Sham, Minne, Lee, & Murray, 1998). Data also suggest that individuals who have experienced a depressive

episode do not normally return to complete asymptomatic functioning following the episode, but rather, continue to experience subclinical levels of distress (Duggan et al., 1998). Part of the reason for such stability in depressive illness is its pervasive effect on several domains of functioning. Some 60 percent of women diagnosed with major depression also meet diagnostic criteria for a comorbid condition – most typically an anxiety condition such as Panic Disorder or Generalised Anxiety Disorder (Clayton, 1987).

Unfortunately, less than half of all women who show symptoms of depressive illness receive specialist treatment, and those who do receive care are misdiagnosed approximately 30 to 50 percent of the time (Kessler et al., 1994). Approximately 70 percent of the prescriptions for antidepressants are given to women, often with improper diagnosis and monitoring (McGrath, Keita, Stickland, & Russo, 1990). Therefore, many women and mothers suffer from untreated or poorly managed depressive illness.

Clinical and subclinical levels of depressive symptoms are associated with considerable burden to families and society. The World Health Organisation estimates that by 2020, depression will carry the second highest disease burden, second only to heart disease (Murphy & Lopez, 1996). Depression causes severe impairment of social and physical functioning and is often a major precipitating factor in suicide. Depression is associated with medical costs, disability, self-care and adherence to medical regimens, and increased morbidity and mortality from medical illness (Katon & Sullivan, 1990). Even depressive symptoms that fall short of a diagnosis of a depressive

disorder are an additional contributor to disability (Judd, Akiskal, & Paulus, 1997). Studies also indicate that depression is particularly common in women during childbearing years and among women with young children (Brown & Harris, 1978).

Epidemiology of Child Behavioural Problems

Like depressive illness in adults, emotional and behavioural problems are among the most common chronic health problems among children. Surveys of community samples of children have shown point prevalence rates from 18 to 22 percent for one or more psychiatric disorders (e.g., Breton, Bergeron, Valla, Berthiaume, Gaudet, & Lambert, 1999; Offord, 1995; Offord, Boyle, Fleming, Blum, & Grant, 1989; Rutter, Cox, Tupling, Berger, & Yule, 1975; Zubrick, Silburn, Burton, & Blair, 1995). In the Isle of Wight studies of the 1970s – still one of the most comprehensive sets of epidemiological studies of childhood disorders – Rutter and his colleagues found that 7 to 14 percent of children exhibited at least one behaviour disorder and that the likelihood of a disorder increased markedly as a function of multiple family stressors (Rutter et al., 1975; Rutter, Tizard, Yule, Graham, & Whitmore, 1976). Their research showed that, in isolation, any one family stressor was not associated with an increased likelihood of child behaviour problems, but when two or more stressors were present, the risk of child behaviour problems increased as much as two- to four-fold. The family stressors included overcrowding in the home, large family size, father criminality, and maternal depression or “neurotic

disorder” (Rutter et al., 1975). Similar rates have been found in Canada. The Ontario Child Health Survey (OCHS), a community survey of children four to 16 years of age, found that at any point in time, about one in five children could be diagnosed with a psychiatric disorder (Offord et al., 1989). The Survey reported a six month prevalence rate of 18.1 percent for one or more of four child psychiatric disorders (conduct disorder, hyperactivity, emotional disorder, and somatisation; Offord et al., 1989).

Unfortunately, many children (like adults) suffer from untreated or poorly managed emotional or behavioural disturbances. Despite the large percentage of children needing treatments for behavioural disorders, only a small minority can access them. In Canada, where there exists a comprehensive public health care system, fewer than one in five children who show signs of a mental health disorder have any contact with a mental health care provider during the previous six months (Offord et al., 1987). The utilisation rate of mental health services is far below the 60 percent of children in Canada who receive regular primary or ambulatory care during the same period (Offord et al., 1989).

As with depression in mothers, untreated adjustment problems in children cause significant burden on families and communities. Childhood disorders of mood, anxiety, or disruptive behaviours tend to coexist with many other problems, including poor school performance, interpersonal conflict, chronic health problems, substance misuse and suicidal behaviour (Elgar & Arlett, 2002; Elgar, Arlett, & Groves, 2003; Offord et al., 1989). Without adequate screening and treatment, the prognosis for most childhood disorders

is generally poor. Left untreated, disruptive and aggressive behaviour in childhood may persist and evolve into more sociopathic behaviours in adulthood and child emotional disorders place individuals at risk of depressive and anxiety disorders throughout the lifespan (Hofstra, Van der Ende, & Verhulst, 2000; Patterson, DeBaryshe, & Ramsey, 1989). One study found that children who showed elevated rates of behavioural problems at 4 to 16 years of age were 41 percent (4.6 times) more likely than healthy children to continue to show elevated rates of behavioural problems as adults 14 years later (Hofstra et al., 2000).

Maternal Depression Affects Child Health

Research indicates that many children are repeatedly exposed to maternal depression and consistently exposed to subclinical maternal depressive symptoms. There is ample cause for concern for the health of these children. Mood disorders are heritable, affect neuroendocrine and circulatory function during pregnancy, are generally incompatible with good parenting behaviour, and can cause significant life stress for the child (Cummings & Davies, 1994). Consequently, living with a depressed parent poses a risk for a number of developmental and adjustment problems in children (Downey & Coyne, 1990; Rutter, 1990), including emotional and behavioural disturbances and a wide range of social and achievement deficits (Billings & Moos, 1986; Goodman, Brogan, Lynch, & Fielding, 1993; Kurstjens & Wolke, 2001; Lytton, 1990). Studies found that depressive illness in the mother encroaches upon several

domains of child functioning including internalising and externalising problems, prosocial behaviour and cognitive functioning (Forehand, McCombs, & Brody, 1987; Lovejoy, Craczyk, O'Hare, & Neuman, 2000).

Indicative of such negative influences on child functioning, point prevalence rates of psychiatric disorder among children of depressed parents have been estimated as two to five times above normal (Beardslee, Keller, Lavori, Staley, & Sacks, 1993; Weissman, Prusoff, Gammon, Merikangas, Leckman, & Kidd, 1984) – ranging from 41 to 77 percent (Beardslee, Schultz, & Selman, 1987; Weissman et al., 1986). Among children of unipolar depressed parents, lifetime prevalence rates of psychiatric conditions, including depression, oppositional defiant or conduct disorder, anxiety disorders, and alcohol or drug use and dependency, exceed 40 percent (Rutter & Quinton, 1984). A meta-analytic review estimated that children of mothers with major depression have an approximately 40 percent chance of experiencing an episode of major depression before age 20 (Beardslee, Versage, & Gladstone, 1998). In another study that followed children of depressed and nondepressed mothers for four years after mothers' assessments, it was found that the prevalence of major depressive disorder among the children of depressed parents was significantly higher (26 percent) than in matched children whose parents had no disorder (10 percent) (Beardslee et al., 1993). Other research has found that infants and children of depressed mothers, compared to children of non-depressed mothers, tend to be more fussy, receive lower scores on measures of intellectual and motor development, have more difficult

temperaments and less secure attachments to their mothers, react more negatively to stress, show delayed development of self-regulatory strategies, and exhibit poorer academic performance, fewer social competencies, lower levels of self-esteem and higher levels of behavioural problems (Cummings & Davies, 1994; Goodman & Gotlib, 1999).

Child Adjustment Problems Affect Maternal Health

It is evident that children of depressed mothers are at increased risk of abnormal development and child behavioural problems. Conversely, living with a child with emotional or behavioural disturbance can be immensely stressful to mothers and may increase the risk of (or further exacerbate) maternal depression. Studies of community samples of mothers found increased rates of depressed mood among mothers of clinic-referred disturbed children (Brown, Borden, Clingerman, & Jenkins, 1988). For example, in a community study of 5303 families, Civic and Holt (2000) found that mothers who reported three or more adjustment problems in their children (e.g., temper tantrums, social problems, unhappiness) were 3.6 times more likely than other mothers to show elevated scores (above 15) on the Center for Epidemiological Studies Depression Scale (CES-D; Radloff, 1977). There is further evidence showing that treatment of disruptive behavioural problems in children alleviates stress and symptoms of depression in mothers (Forehand, Wells, & Griest, 1980). Using Webster-Stratton's (1981) Parents and Children Series group-based parenting program for managing disruptive child behaviour, Taylor, Schmidt,

Pepler, and Hodgins (1998) found that, in addition to improved child behaviour, there was a significant reduction in mothers' depressive symptoms as measured by the Beck Depression Inventory (Beck, Ward, Mendelson, Mock, & Erbaugh, 1961).

Childhood behaviour and emotional disorders elicit considerable stress for families and may compromise mothers' perceived competency as a parent (Mash & Johnston, 1990). Studies have found that compared to mothers of normal children, mothers of children with disorders of conduct, attention or hyperactivity experience more life stress (Barkley, Anastopoulos, Guevremont, & Fletcher, 1992; Shelton et al., 1998). Such influences on maternal functioning have even been demonstrated in laboratory settings. Under controlled experimental conditions, Pelham and his colleagues demonstrated how increasingly deviant child behavioural problems can cause increased depressed and anxious feelings, hostility and alcohol consumption in parents (Pelham, Lang, Atkeson, Murphy, Gnagy, Greiner, et al., 1997). Another study by Arnold and O'Leary (1995) found that exposure to negative affect in children (on videotape) caused more depressed feelings and more overreactive discipline in mothers in response to their children's behaviour.

How Much?

While the data show statistically significant associations between depressive symptoms in mothers and adjustment problems in their children, their *clinical significance* is less clear. The magnitudes of reported correlations between

mother and child symptoms are highly variable and vary as a function of the population studied, measures used, and method of assessment. For example, in a community sample of 46 mother-child dyads, Marchand and Hock (1998) used the CES-D to measure maternal depressive symptoms and Achenbach's (1991) Child Behavior Checklist to measure parent-rated child externalising and internalising behaviour. Mothers' symptoms correlated .22 with child externalising behaviour and .29 with child internalising behaviour. In another study that used these same measures with a sample of 194 families of low-income, adolescent mothers and at-risk children, it was found that maternal depression correlated .38 with child externalising behaviour and .32 with child internalising behaviour (Black, Papas, Hussey, Dubowitz, Kotch, & Starr, 2002). The primary difference between the two studies were that a considerable proportion (42 percent) of the children in the second study were maltreated and the mothers were younger ($M = 24$ years) than mothers in the study by Marchand and Hock ($M = 34$ years).

Other studies have shown discrepancies in the magnitude of relations between maternal depressive symptoms and child adjustment problems. Phillips and O'Hara (1991) used the Beck Depression Inventory and Child Behavior Checklist to assess functioning in 70 mother-child dyads and found that maternal depression correlated .20 with child externalising behaviour and .32 with child internalising behaviour. Quite different results were found in a study by Hammen and colleagues. In a sample of 42 families, they found mothers' Beck Depression Inventory scores correlated .39 with child depressive

symptoms measured by the Child Depression Inventory (Kovacs, 1985), .53 with child externalising behaviour measured by the Conners Teacher Rating Scale (Conners, Sitarenios, Parker, & Epstein, 1998), and .54 with total problem scores on the Child Behavior Checklist (Hammen, Adrian, Gordon, Burge, Jaenicke, & Hiroto, 1987). Taken together, such studies do not well inform about the true magnitude of the relation between maternal mood and child adjustment. It is also difficult to glean from the research whether child internalising behaviours are more or less related to maternal depression than child externalising behaviours.

Providing some clarity to these inconsistent findings, Beck (1999) conducted a comprehensive meta-analysis with the objective of deriving a composite effect size from studies that reported relations between maternal depression and child adjustment problems. Beck's analysis utilised 33 correlational studies on a cumulative sample of 4561 mother-child dyads (*N*s ranged from 15 to 1123 dyads). Most studies used the Child Behavior Checklist to assess child symptoms and either the Beck Depression Inventory or CES-D to assess maternal depression. The composite effect size ranged from .29 to .35 (depending on whether calculation was weighted by sample size) which according to Cohen's (1988) operational definitions of effect sizes, indicates a "moderate" relation between maternal depression and child adjustment problems.

Mechanisms of Bidirectional Risk and Resilience

Overlapping occurrence of maternal depressed mood and adjustment problems in children is attributable, in part, to their deleterious mutual influences. The capacity for mutual, transactional relations between maternal and child functioning is evidenced by the mechanisms that mediate their influences. Over the past decade, research has moved beyond the question of whether children of depressed parents are at risk for abnormal development to identify factors that facilitate risk and resilience. To this end, a major challenge lies in identifying the more “active ingredients” in a mix of interrelated mechanisms. The transmission of psychopathology between mother and child could involve genetic transmission, biological neuroregulatory systems, cognitive and interpersonal processes, family functioning or other environmental risk factors (Cummings & Davies, 1994; Silberg & Rutter, 2002). The direction of some influences are from mother to child, whereby impaired functioning in the mother contributes to increased risk to the child (e.g., inherited vulnerability to depression). Other influences are from child to mother, whereby child behaviour affects the mood of the mother (e.g., reduced parental efficacy, environmental stress) and others may be bidirectional (e.g., insecure attachment, exposure to negative behaviours, poor family functioning). Recent empirical reviews have offered theoretical frameworks to help organise the multitude of studies in the area. Cummings and Davies (1994) provided a detailed description of one such a model that showed bidirectional linkages between maternal depression and a range of child development outcomes.

Although their model was transactional, it unfortunately lacked specificity about child outcomes and causal mechanisms. For example, one of the three mechanisms in the model, “parent characteristics,” subsumed “emotional unavailability and all thinking processes of depression” (p. 75).

Goodman and Gotlib (1999) reported on a more specific model of these influences that included several mechanisms (or “theories”) that are relevant to the transmission of risk and resilience between maternal depression and child adjustment problems. The model identified four mechanisms (genetic vulnerabilities, neuroregulatory functioning, exposure to depressive symptoms, and environmental stress) in addition to moderating factors (e.g., father's availability, child intelligence). Though comprehensive and adequately specific, this model describes each mechanism as relevant only to the influence of maternal depression on child adjustment, and not of the influence on child behavioural problems on maternal functioning. Goodman and Gotlib (1999) give only cursory attention to transactional, reciprocal relations shared by maternal mood and child adjustment.

Despite ample evidence of mutual influences on maternal and child outcomes, Goodman and Gotlib's (1999) model did not offer the possibility for such transactional relations. The model shown in Figure 1 is a simplified revision of Goodman and Gotlib's and it depicts bidirectional pathways of risk between maternal and child behavioural problems. The model includes four key mediating mechanisms. Two mechanisms – heritability and neuroendocrine functioning – help to explain how maternal depression affects inherited

vulnerabilities to child behaviour disorders and early emotional regulation and temperament. Two other mechanisms have the capacity for bidirectional transmission of risk. These mechanisms include exposure to negative cognitions and behaviours (including attachment problems, discipline practices, and all mother-child interactions) and family functioning. Beyond these, a fifth mechanism represents shared contextual risk (e.g., poverty) which is relevant because it influences both maternal and child functioning (or moderates their relation) without direct transmission of risk. Absent from the model are interrelations among the mediating factors (e.g., genetic influences on mother-child relations) and moderating factors that protect mothers and children (e.g., paternal involvement; see Goodman & Gotlib [1999]) but because they are not directly relevant to temporal relations in maternal and child functioning, they are beyond the scope of this thesis. The following sections describe the relevant research on each mediating mechanism.

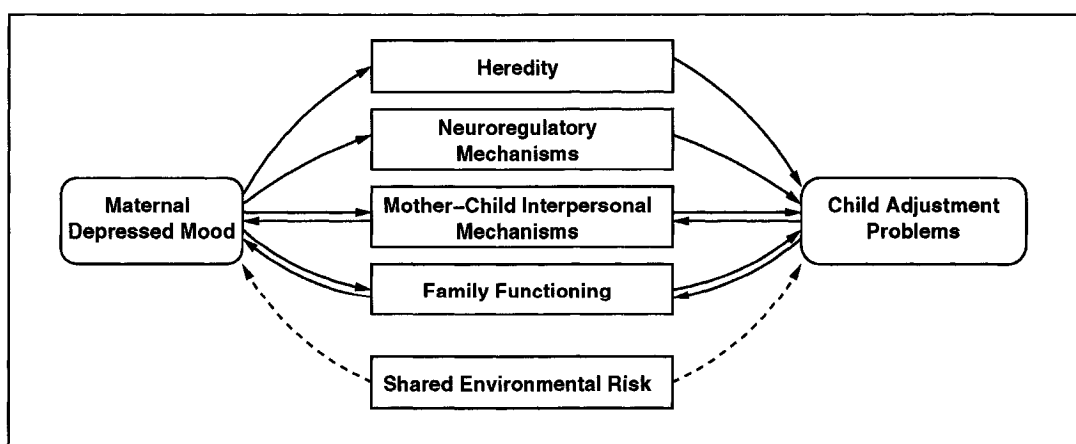


Figure 1. Mechanisms of risk and resilience between maternal mood and child adjustment problems (modified from Goodman and Gotlib [1999] to highlight bidirectional pathways).

Mechanism 1: Heredity

Over the past three decades, a number of twin and adoption studies have provided clear evidence of a genetic component in psychopathology. The risk for an affective disorder in adult first-degree relatives of a patient with unipolar affective disorder is approximately 20 to 25 percent, compared to a general population risk of 7 percent (Tsuang & Faraone, 1990). The design of twin studies rests on the fact that members of monozygotic (MZ) twin pairs share all their genes in common whereas dizygotic (DZ) pairs share half (Eley, 1999). If there are no other differences between MZ and DZ twins in their environments that predispose to the trait or disorder, it may be inferred that any behavioural differences between MZ and DZ pairs can be studied to estimate the extent to which genetic factors are influential (Silberg & Rutter, 2002).

Using this paradigm, twin studies have found that children of depressed mothers are genetically vulnerable to depressive illness (e.g., Cadoret, 1978, Tsuang, 1978). Twin studies that examined age differences have found that genetic influences on depression are weaker during childhood than during adolescence, when prevalence rates for most behavioural problems are higher (Murray & Sines, 1996; Silberg et al., 1999; Thapar & McGuffin, 1994). Twin studies have also found stronger genetic effects on parent-reported symptoms than on child-reported symptoms, but this may be due to lower reliability in child-reported symptoms compared to parent-reported symptoms (Eaves et al., 1997). An implication of these findings is that heritability estimates for psychopathology may be affected by the source of information about child

functioning or by the age of the child. Nonetheless, evidence for genetically-mediated transmission of depression is strong. A meta-analysis of twin studies of depression involving children and adolescents estimated that the genetic effect on major depression accounted for 36 percent (95% CI = 26 to 42 percent) of the variance in child depressive symptoms (Sullivan, Neale, & Kendler, 2000).

Rutter and colleagues have questioned the accuracy of heritability estimates, arguing, quite persuasively, that they do not account for genetic influences on environmental and personality factors that indirectly influence the likelihood of child symptoms (Rutter, Pickles, Murray, & Eaves, 2001). It is unclear, they argue, whether “genetic risk” to children derives from the manifestation of psychological problems or from other factors with which these problems happen to be associated (e.g., life events, coping strategies, or family dysfunction). Solberg and Rutter's (2002) examination of an interaction (or “nature-nurture interplay”) of genetic vulnerabilities with the environmental factors directly questions the validity of twin studies in psychiatric genetic research. They point out that genetic influences tend to correlate with environments; parents who may pass on a genetic vulnerability to their children tend also to create riskier environments than parents who do not pass on such genetic vulnerability. In other words, children may inherit traits (e.g., sensitivity to stress) that evoke environmental events that, in turn, increase the risk of an adjustment problem.

An example of nature-nurture interactions was provided in a study by

O'Connor and colleagues who examined "genetically at-risk" adopted children (O'Connor, McGuire, Reiss, Hetherington, & Plomin, 1998). They found that these children experienced more negative parenting from their adoptive mothers than their adopted siblings as a function of their own disruptive behaviour. Interpreting adoption studies is complicated by the fact that adoptive parents are normally selected on the basis that they will provide safe environments. Nonetheless, there is good evidence to suggest that heritability in mental illness may interact with environmental mechanisms in the transmission of risk from mother to child.

An additional complexity of genetic models is that the increased risk of disorder in children of depressed mothers extends more broadly than depressive illness (Rutter, 1997). There is also evidence to suggest that risk mediation may operate differently for depression than for disruptive behaviour disorders in children of depressed mothers. In a generational study of 90 child-mother-grandmother triads, Warner and colleagues found that depression in parents and depression in grandparents were both associated with increased risk of depression in children (a result consistent with genetic mediation) but that depression in parents but *not* depression in grandparents was associated with increased risk for disruptive behaviour disorder (suggesting the possibility of environmental risk) (Warner, Weissman, Mufson, & Wickramaratne, 1999). The study found that risk for anxiety and substance use disorders in children was mediated similarly to disruptive behaviour disorder, suggesting that the effects of maternal depression on parenting behaviour may increase risk for

several adjustment problems in children but that genetic factors may also play a specific role in child depression (Warner et al., 1999).

Mechanism 2: Neuroendocrine Functioning

An intriguing aspect of the effects of maternal depression on child functioning is the age at which these effects first emerge. Studies have found that just days after birth, infants of depressed mothers, compared to infants of nondepressed mothers, are more likely to have difficult temperaments, exhibited by social unresponsiveness, low activity, negative emotion, irritability and hypersensitivity (Cummings & Davies, 1994; Weissman et al., 1986). These infants also exhibit higher cortisol and norepinephrine levels, poorer orientation, and more abnormal reflexes than infants of healthy mothers (Da Costa, Dritsa, Larouche, & Brender, 2000; Field, 2002; Hedegaard, Henriksen, Sabroe, & Secher, 1993). These behavioural differences indicate that depressive illness in pregnant women has negative effects on the physiology of the developing fetus that are likely mediated by abnormalities in the fetal environment, such as neuroendocrine dysfunction, reduced blood flow to the fetus, or poor health behaviour (Goodman & Gotlieb, 1999). Changes in these systems as a result of depression in the mother are reflected in two areas: brain activity (particularly in the prefrontal cortex) and autonomic reactivity to stress.

First, research by Dawson and colleagues shows that unipolar depression in pregnant women is prospectively linked to reduced left frontal brain activity in the infant during a baseline condition and during playful

interactions with the mother and a familiar adult (controlling for antidepressant medication use; Dawson, Frey, Panagiotides, Yamada, Hessler, & Osterling, 1999). Brain activity in the frontal lobe is thought to play an important role in emotion regulation and expression. In situations designed to elicit negative affect, these infants of depressed mothers also show less distress and greater left frontal EEG activation than infants of nondepressed mothers – even in infants as young as one day old (Jones, Field, Fox, Davalos, Lundy, & Hart, 1998; Jones, Field, Fox, Lundy, & Davolos, 1997). The data indicate that, in addition to genetic influences, maternal depression may affect emotional regulation in children through intrauterine influences on brain functioning.

Second, neurotransmitter and neuroendocrine systems involved in autonomic responses to stress and mood regulation have also been studied in newborns in human studies, as well as in rats and primates. A system of particular interest is neuroendocrine function in the hypothalamic-pituitary-adrenal (HPA) axis that has been linked to adult depression in humans and to attachment disorganisation in human infants (Amsterdam, Maislin, Gold, & Winokur, 1989; Hertzgaard, Gunnar, Erickson, & Nachmais, 1995). These neurological data on brain functioning corroborate behavioural evidence of blunted autonomic functioning. In mother-infant interactions, infants of depressed mothers show elevated heart rate and cortisol levels, lower vagal tone, more gaze aversion, decreased physical activity, and fewer vocalisations than infants of healthy mothers (Field et al., 1988). The pattern of abnormal autonomic functioning appears to endure through childhood – in abnormally

high cortisol secretion in response to mild laboratory stressors at 13 to 15 months of age (Dawson et al., 2001) and adolescence (Birmaher & Heydl, 2001).

Multigenerational studies – both animal and human – have shown that neuroendocrine mechanisms operate independently of heredity. In a cross-fostering design study with newborn rat pups, Francis, Diorio, Liu, and Meaney (1999) found that both the quality of the parent-pup interaction and the pup's associated physiological stress responses mediated by the HPA axis were passed on intergenerationally independent of genetic influence. In another study, Weaver, Grant, and Meaney (2002) found that the rate of hippocampal cell loss in rat pups occurring through apoptosis could be changed by altering the degree of physical contact with the mother. Human studies have also shown that intergenerational transmission of attachment strategies are not genetically determined and can change using intensive parent training (van Ijzendoorn, Juffer, & Duyvesteyn, 1995).

The notion that changes occur in infant neurological and neuroendocrine functioning as a result of depressive illness in the mother adds another dimension to understanding the processes involved in the mother-child transmission of psychopathology or diathesis to psychopathology. Unlike genetic models, the consequences of such changes entail a range of outcomes relating to emotional dysregulation – including internalising and externalising behaviour. The genetic and neuroendocrine mechanisms depicted in Figure 1 would suggest that maternal depression tends to precede child symptoms, but

previous research has also shown that child behaviour can deleteriously affect maternal health. Therefore, to account for mutual influences on maternal and child symptoms, interpersonal and environmental mechanisms must also be involved.

Mechanism 3: Mother-Child Interpersonal Mechanisms

In an early study of infant responses to simulated displays of maternal depression, Cohn and Tronick (1983) found that brief exposures elicited fussiness and distress behaviour in a normal population of infants. When the procedure was later replicated with naturally depressed mothers, infants of depressed mothers responded with flat affect and inattentiveness (Field, 1984). Over the past two decades, many studies since these have shown that an interpersonal mechanism is at play, possibly interacting with inherited diatheses to problems in affective functioning, in the transmission of psychological problems from parents to children. This third mechanism of risk involves exposure to negative behaviours and cognitions – both children's exposure to depressive symptoms in the mother, and mothers' exposure to internalising or externalising behaviours in the child.

Depression may profoundly alter how people think and feel about themselves and other people (American Psychiatric Association, 1994; Beck, 1967; Cicchetti & Toth, 1998). Children of depressed mothers are regularly exposed to such symptoms as dysphoric affect, irritability, confusion, helplessness, and hopelessness (Abraham, Metalsky, & Alloy, 1989) and are

likely to feel the psychological unavailability of their mothers (Downey & Coyne, 1990). Conversely, mothers of children with behavioural problems, depending on the nature of the problem, may be exposed to and feel upset by aggressive, hyperactive, delinquent, or emotionally-disturbed behaviour. Interpersonal relations mediate bidirectional risk in at least three ways: parent-child attachment, child discipline practices, and modelling.

Attachment. Infants normally develop an attachment relationship with the mother during the first year of life. Attachment refers to a mutual emotional bond between the mother and child and it reflects an innate tendency for infants and caregivers to respond to one another in ways that increase the likelihood of survival through proximity in times of stress (Bowlby, 1988). Sensitive and responsive parenting promotes the expectation of care when necessary, which has pervasive influences on child socioemotional development. Bowlby identified the quality of the attachment relationship as a precipitating factor in child behavioural problems that involve distrust or chronic anxiety, stating that unmet needs for security can lead the child to view the world as “comfortless and unpredictable, and they respond either by shrinking from it or doing battle with it” (Bowlby, 1973, p. 208).

Postpartum depression in the mother may disrupt the development of secure mother-child attachment – a potential sequela of her insensitivity to the child’s needs (Cicchetti, Rogosch, & Toth, 1998). Though certainly not an inevitable outcome, insecure attachment relationships tend to be more common

with mothers who suffer from prolonged postpartum depression (Atkinson et al., 2000; Carter, Garrity-Rokous, Chazan-Cohen, Little, & Briggs-Gowan, 2001; Martins & Gaffan, 2000). The manifestation of depressive symptoms affects the mother's emotional availability and sensitivity to the child's needs during critical periods of development as well as her ability to communicate with the child (Stein, Gath, Bucher, Bond, Day, & Cooper, 1991). Studies have found that compared to nondepressed mothers, depressed mothers show fewer positive and animated faces and voices (Raag et al., 1997), more sad and angry faces, fewer expressions of interest (Pickens & Field, 1993), and less accurate matching of happy facial expressions to happy vocal expressions (Lundy, Field, & Pickens, 1996). The effects of these behaviours on children can be immediately reciprocated back to the mother. As discussed above, infants of depressed mothers tend to show behaviours that exacerbate difficult mother-child interactions, showing a "profile of dysregulation" that involves low responsiveness to facial expressions, fussiness and inconsolability, disturbed sleep, and elevated stress hormones (Field, 2002).

Lab studies and naturalistic field studies have found that infants respond negatively to face-to-face interactions with maternal displays of negativity, intrusiveness and withdrawal by showing more anger, reduced activity, and social withdrawal (Field, Healy, Goldstein, & Guthertz, 1990). Studies of these interactions found that nondepressed mothers and their infants have smooth, harmonious interactions, but depressed mothers and their infants have more interactions that are "choppy, uncoordinated, and unpleasant to observe" (Field,

2002, p. 62). In these situations, both the depressed mother and her infant have elevated heart rates and stress hormones, suggesting that they are stressed by their interactions (Field, 2002). Even with infants as young as 3 months, mother-child interaction studies found that depressed mothers, compared to nondepressed mothers, are less responsive and less emotionally available and their infants are less active and expressive (Cicchetti et al., 1998). Furthermore, Cox, Puckering, Pound, and Mills (1987) found that, in comparison to nondepressed mothers, depressed mothers of 2-year-olds exhibited more criticism, disengagement, less warmth, and less responsiveness to the child's cues for attention. Such behaviours, over a period of time, may contribute to insecure mother-child attachments (Bowlby, 1973; Carlson & Sroufe, 1995).

For older children, the long-term consequences of insecure attachment entail problems of emotional dysregulation, heightened sensitivity to stress, and problems in social functioning. Although the link between attachment and psychopathology remains unclear, having few opportunities to refine the skills needed to regulate emotion increases the child's risk of maladjustment in later years (Carlson & Sroufe, 1995). A study by Carter and colleagues found that depression during pregnancy and at 4 months postpartum predicted attachment insecurity when the child was 14 months old and problem behaviours and intellectual competencies when the child was 30 months old (Carter et al., 2001). In another study, 172 children whose mothers were depressed during the first year postpartum were followed at 3 years, 10 months of age (Sharp,

Hay, Pawlby, Schmucker, Allen, & Kumar, 1995). After controlling for birth weight, parental IQ, family functioning, and home environment, the children still scored significantly below normal on standardised tests of intellectual attainment. Other research by Murray (1992) found that maternal depression at 2 months postpartum significantly increased the risk of insecure mother-child attachments 16 months later. A follow-up of the same cohort found that the quality of attachment at 18 months of age mediated the negative relation between postnatal maternal depression at 2 months and child prosocial behaviour at school five years later (Murray et al., 1999). Also, Teti and colleagues found greater levels of insecure attachment – specifically, the Anxious-Depressed classification – among preschoolers of depressed mothers (Teti, Gelfand, Messinger, & Isabella, 1995). The Anxious-Depressed attachment classification resembles some anxious and depressive symptoms (e.g., sad or flat affect, lethargy, panic) that occur upon separation from the caregiver. Disorganised attachment behaviour in infants also appears to be related to the severity and chronicity of maternal depression (Teti et al., 1995).

Despite these findings, there have been studies that showed nonsignificant relations between maternal depression and attachment insecurity. A meta-analysis by van Ijzendoorn, Schuengel, and Bakermans-Kranenberg (1999) included 16 studies on attachment and parental depression. In normal samples, they found a nonsignificant correlation of .06 between child disorganised attachment and parental depression (controlling for socioeconomic status). When only clinical samples were considered, this relation was

significant but still small, .13 or about 1% of shared variance. This estimate may be misleadingly low because the focus of their study was disorganised attachment and not all forms of insecure attachment. Still, other interpersonal mechanisms, in addition to attachment, seem likely to be involved in linking maternal depression to child adjustment problems.

Child Discipline. Maternal depression also influences child behaviour through disruption of discipline practices. A mother who feels depressed is less likely than a mother who feels well to be firm and consistent in her use of discipline and is more likely to “give in” to child tantrums through negative reinforcement. Patterson and colleagues examined the negative effects of ineffective or inconsistent parenting on the development of disruptive behavioural problems (Patterson et al., 1989). In his “coercive family process model,” Patterson (1982) proposed that the effectiveness with which parents manage aggressive or noncompliant behaviours of their children plays a role in the course of those behaviours as the child matures. In the coercive family, as the child's aggression grows more frequent and intense, parents' attempts to curb and manage aggression become increasingly inadequate. A result of inept parenting is the allowance of family interactions in which coercive child behaviours are reinforced (Patterson et al., 1989; Reid & Patterson, 1991). This reinforcement may come in the form of positive regard for the coercive behaviour, such as when a parent laughs at a scene of sibling bully behaviour. When the parent passively allows the child's coercive behaviour to be

reinforced, the likelihood of later repetition and its escalation of its intensity is increased.

Patterson (1982) identified harsh, inconsistent, and physical discipline as characteristic of the coercive family process. Even at a lesser degree, ineffective parents tend to have limited repertoires of discipline strategies of primarily verbal or physical aggression. When mothers' discipline is tied closely to mood or whim, the result is to have a behaviour ignored on one day and punished on the next, and when child aggression is punished with counter-aggression in an unpredictable, erratic fashion, it becomes resistant to change (Patterson et al., 1989).

Patterson's model reflects transactional, mutual influences on maternal mood and child behaviour, whereby externalising behaviours in the child increase as the mother becomes less available or more intrusive as a result of her depression, resulting, ultimately, in poorer parenting, more frustration, and greater family dysfunction. This self-perpetuating cycle of negative behaviour may be driven by low self-esteem and self-efficacy in the mother (Teti & Gelfand, 1991) which, not surprisingly, relates to passive maternal coping orientations (Wells-Parker, Miller, & Topping, 1990) and child behaviour problems (Johnston & Mash, 1989). A reduced sense of efficacy puts mothers at risk for responding hesitantly and maladaptively to child problem behaviours. The influence of maternal depression on rearing practices may also be mediated by personality factors (e.g., perfectionism leading to overprotective behaviours in parents; Enns, Cox, & Larson, 2000) or neglect (e.g., failing to

use child car seats or cover electrical plugs in the home; McLennan & Kotelchuck, 2000). A study of 276 mother-child dyads in inner-city London found that maternal history of depression related to 78% increased risk of child depression but this influence was entirely mediated by child-rated neglect and abuse (Bifulco et al., 2002). There is also evidence that mothers' negative attitudes towards children moderate or worsen the influence of their mood on their children's feelings of global self-worth (Goodman, Adamson, Riniti, & Cole, 1994).

Self-esteem and parental efficacy partially explain why depressed mothers, compared to healthy mothers, tend to be lax, inconsistent, and ineffective in their use of discipline (Cunningham, Bennes, & Seigel, 1988; Downey & Coyne, 1990; Forehand, Lautenschlager, Faust, & Graziano, 1986; Lovejoy et al., 2000; Snyder, 1991). However, conflict that arises from the inconsistent use of discipline also has, in itself, negative effects on child functioning. Depressed mothers and their toddlers and preschool-aged children have been found to engage in patterns of coercive mutual influence, including what are described as retaliation and revenge (Goodman & Gotlieb, 1999). Additionally, conflict between parents and children is prospectively related to child depressive symptoms (Elgar & Arlett, 2002), child externalising behaviours (Elgar, Arlett, et al., 2003), and substance use and delinquency (Elgar, Knight, Worrall, & Sherman, 2003).

Much variability exists between families in how maternal and child behaviours “play off” each another. Some children's responses to maternal

depression manifest in internalising behaviour. Other children (as young as two years of age) engage in comforting behaviours in response to parental distress (Radke-Yarrow, Zahn-Waxler, Richardson, Susman, & Martinez, 1994). Or, as maternal depressive symptoms shift from intrusiveness to withdrawal and sadness, children may actually suppress disruptive behaviour (Hops, Biglan, Sherman, Arthur, Friedman, & Osteen, 1987). A community study found that subclinical levels of dysphoria in the mother reduced the probability that their children and fathers would emit aggressive behaviour, and that children's and fathers' aggressive affect suppressed the mother's dysphoric affect (Hops et al., 1987). An important consideration with regard to mutual influences on maternal and child outcomes is that different influences may lead to similar adjustment problems (equifinality) while similar experiences may contribute to different outcomes (multifinality) (Cicchetti & Rogosch, 1996).

Modelling. Beyond directly influencing each other's behaviour through either attachment insecurity or discipline and conflict, there may also be a social learning component involved in this interpersonal mechanism of risk. As Bandura (1986) described, a child's developing self-schema is ostensibly shaped by the observation, internalisation, and emulation of behaviours exhibited by other people. There is some evidence to support this hypothesis. Breznitz and Sherman (1987) found that in conversations between young children and depressed mothers, children matched the low tone and low rates of speech of their mothers. In support of Bandura's (1986) suggestion that

mothers provide stronger models for girls than for boys, Hops, Sherman, and Biglan (1990) found that 11- to 16-year old daughters of depressed mothers exhibited more dysphoric affect and less happy affect than younger girls (3- to 10-year-old) or boys of either age, and at similar levels to those displayed by their mothers. This result was replicated by Inoff-Germain and colleagues who also found that daughters of depressed mothers were more likely than their sons in the same age group to match their mothers' low mood (Inoff-Germain, Nottelmann, Radke-Yarrow, 1992).

Despite the intuitive appeal of social learning theory, it is difficult to validate in the context of family influences on psychopathology. While it is possible that such behavioural synchrony occurs when children (especially girls) emulate the depressive behaviours modelled by their mothers, these trends are also attributable to post-pubertal sex differences in rates of internalising and externalising behaviours in teenage girls and boys (Offord et al., 1989). Another shortcoming is that social learning theory does not explain why some children respond to maternal depression with externalising behaviour. Consequently, while the data on children's matching of their depressed mothers dysphoric affect seem to support social learning theory, they are also consistent with other explanations.

Mechanism 4: Family Functioning

Depression in mothers and child adjustment problems affect not only one another, but also family functioning and marital relationships. Depressed

mothers have been described as more insensitive towards all family members (Cox et al., 1987) and their parenting style is characterised by hostility, irritability and enmeshment (Parker, Tupling, & Brown, 1979). Such an effect on family functioning constitutes environmental risk (Rutter, 1990) and, thus, is a fourth mechanism through which mothers and children may influence the health of one another (Davies & Windle, 1997; Kelly, 2000).

Marital discord increases the likelihood of ineffective child management practices and influences both depressive symptomatology in mothers (Rutter, 1990) and conduct problems in children (Webster-Stratton & Hammond, 1999). Marital discord may affect children through the arousal and emotional dysregulation caused by witnessing conflict (Cummings & Davies, 1994), negative reinforcement (e.g., acting out to interrupt conflict between parents; Kelly, 2000), or modelling aggressive behaviour seen at home (Bandura, 1973). Children are bystanders to negative interactions between parents and learn to emulate expressions of both overt aggression towards others (Pike & Plomin, 1996; Webster-Stratton & Hammond, 1988) and subtle, vindictive aggression (Webster-Stratton & Hammond, 1999). Conflict between depressed mothers and their partners increases the likelihood of family discord and scapegoating of the children. As Silburg and Rutter (2002) described, children may be blamed for breaking up the family and for a range of associated stresses and adversities, such as widespread interpersonal relationship problems, excessive drinking and drug misuse, and general social disadvantage. Not surprisingly, children's responses to such treatment include more conduct problems,

substance misuse, and delinquent behaviour (Elgar et al., 2002).

Mechanism 5: Shared Environments

Taken together, a substantial body of research indicates that a bidirectional relation shared by maternal depression and child adjustment problems involves multiple, interwoven mechanisms operating within the family context (irrespective of a diagnosis of disorder). Compromised health of either the mother or the child is a risk factor for the other. However, maternal depression and child adjustment problems also tend to coexist with shared environmental risk factors such as low socio-economic status (Dodge, 1990; Glazier, Elgar, Goel, & Holzapfel, 2001; Lipman, Offord, & Boyle, 1994; Lovejoy et al., 2000). Brown and Harris's (1978) classic study of women living in London found that depression was significantly more prevalent among working-class than middle class women. In the study of inner-city Londoners by Bifulco et al. (2002), rates of psychiatric disorder in children of low-income families was fourfold higher than in a comparison sample of average-income families (43 versus 11 percent). The Ontario Child Health Study found that the prevalence rates of emotional and behavioural problems, in the 6 to 11 age group, were about three times higher among families who received income support than among families that did not receive income support (40 versus 13.9 percent; Offord, Boyle, & Jones, 1989). Using data from the Ontario Child Health Study, Curtis and her colleagues reported a significant inverse relation between family income and child behavioural health (Curtis, Dooley, Lipman, &

Fenny, 2001).

The reason for the link between family income and health outcomes is complex. Low-income families tend to be more isolated from communities, to have fewer resources of extra-familial social support (Hashima & Amato, 1994), and to have greater difficulty in accessing child care and mental health services (Dodge, Pettit, & Bates, 1994; Hartley, Bird, & Dempsey, 1999; Hunsley, Aubry, & Lee, 1997). Other marginalised groups, such as refugees and immigrants, also have difficulty accessing mental health services but after controlling for socioeconomic status, do not show poorer health than the general population (Munroe-Blum, Boyle, Offord, & Kates, 1989). Studies of relations between maternal depression and child behavioural problems have not consistently accounted for such potentially confounding variables. This is an important consideration, given that shared environmental risk may help explain some of the association between maternal and child health outcomes, without the direct transmission of risk.

Developmental and Taxonomical Considerations

Important aspects of the mechanisms of risk and resiliency described above are their timing, the context in which they function, and the type and severity of symptoms on which they operate. Certain mechanisms may function differently at various stages of child development. As Goodman and Gotlib (1999) noted, “researchers have tended to study children of depressed mothers either in one developmental period or in such a broad age range that two or more distinct

developmental stages are included (and confounded)” (p. 459). Attachment difficulties may occur during a critical period of infancy, or there may be a delayed “switching on” of a genetic predisposition to an emotional problem during childhood, or inadequate discipline by parents may not emerge until adolescence. Such possibilities bring to question whether the strength of mutual influences on maternal mood and child adjustment might vary across age groups of children.

A second consideration is whether these influences operate only at clinically elevated levels of symptomatology or at the full range of symptoms normally experienced by community samples. Mutual influences on maternal and child adjustment have been shown in clinical studies but inconsistently reported in some community studies (Compas, Howell, Ledoux, Phares, & Williams, 1989; Fergusson, Horwood, & Lynskey, 1995). A community study in New Zealand found significant relations between maternal depressive symptoms and subsequent depressive symptoms in adolescent females but no association between maternal depressive symptoms and depressive symptoms in adolescent males (Fergusson et al., 1995). It is not clear whether the result was attributable to a higher prevalence of clinical levels of internalising symptoms in adolescent females than in adolescent males or to a higher sensitivity in daughters than in sons in response to depressive episodes in mothers. Some have suggested that maternal depression influences only extreme cases of child adjustment problems – an association that is obscured in community samples (e.g., Downey & Coyne, 1990). It may also be that

previous cross-sectional studies precluded adequate examination of mutual influences on maternal and child functioning over time. To address these issues, temporal relations between maternal and child functioning should be studied in healthy and clinical populations using both lengthy time intervals, to capture stable, underlying adjustment problems, and short time intervals, to capture transient, state-like fluctuations in functioning.

Who Distresses Whom?

The research on maternal depression and child behavioural problems, and the mechanisms that mediate them, reveal the capacity for mutual, bidirectional influences. The intergenerational transfer of psychopathology has implications for the etiology, maintenance, assessment, and treatment of psychological problems (Downey & Coyne, 1990). Maternal depressive symptoms may trigger an emotional or behavioural problem in the child, which a depressed mother has difficulty managing, thereby exacerbating the child's problem and increasing or maintaining the mother's depression, and so on. Thus, where maternal depression and child adjustment problems coexist, dyadic intervention may be more effective than treating either the mother or the child alone.

As the model in Figure 1 shows, mental health problems can be passed on from mother to child through genetic or neuroendocrine mechanisms, and the stress and family disruption arising from child behavioural problems can contribute to emotional problems in mothers. It also appears likely that the relations between maternal and child functioning are also attributable to

transactional, bidirectional mechanisms involving insecure attachment, inadequate parenting, conflict, social learning, and family dysfunction. While it is difficult to parse all these mechanisms in a single study to examine their relative mediating influences, consideration of temporal relations between maternal mood and child behavioural problems may help to identify those mechanisms that are most salient. The manifestation of child adjustment problems that occur as a consequence of maternal depression is consistent with theories that claim that such problems arise, in part, from genetic inheritance or impairment of early neuroendocrine functioning. On the other hand, maternal distress that arises as a consequence of child adjustment problems signifies that social and environmental mediation cannot be ruled out as being less significant than genetic and neuroendocrine mediation of risk.

Until recently, few studies have attempted to unravel temporal relations between maternal mood and child adjustment problems despite the intuitive significance of these relations to psychological assessment and treatment (Burbach & Borduin, 1986). Notable exceptions include a study by Forehand and McCombs (1988) in which teacher-rated child behavioural problems (internalising and externalising) in a community sample of 83 adolescents and self-rated depression in their mothers were examined on two occasions one year apart. These researchers found more significant partial correlations between maternal depression at Time 1 and child adjustment at Time 2 (controlling for child adjustment at Time 1) than between child adjustment problems at Time 1 and maternal depression at Time 2 (controlling for maternal

depression at Time 1). Based on differences between partial cross-lagged correlations, it was concluded that maternal depression was the antecedent event in the relation between maternal depression and adolescent functioning. Unfortunately, Forehand and McCombs did not account for the considerable difference in test-retest reliability between their measures of maternal depression ($r = .71$) and child adjustment problems ($r_s = .26$ to $.51$), which made differences between cross-lagged correlations difficult to interpret. In cross-lagged correlations, differences in the reliability of two variables may artificially increase the predictive power of one variable over the other so that the more reliable variable appears to precede the less reliable variable (Cook & Campbell, 1979; Miller, 1997). Adequate sample sizes and consistency in test-retest reliability and cross-sectional correlations are necessary for valid cross-lagged correlation analysis (Kenny, 1975). Replication of the study is needed using a larger sample and similarly reliable mother and child measures.

Another cross-lagged study involved three waves of panel data that were collected over two years from 368 parent-child dyads (Ge, Conger, Lorenz, Shanahan, & Elder, 1995). Unlike the study by Forehand and McCombs (1988), both parents and children provided self-report data on symptoms of depression, anxiety, and hostility. Ge and colleagues found that parent and adolescent distress were reciprocally related over time after earlier symptoms were statistically controlled. However, their study did not show differences between cross-lagged relations over one-year intervals, possibly because their application of structural equation modelling (which does not adequately test

temporal relations; see Rogosa, 1988) included a single latent construct of adolescent distress representing depression, anxiety, and hostility, rather than separate models that may have compared differences across types of child maladjustment. Furthermore, their measure of child symptoms, the Symptom Checklist-90-Revised (Derogatis, 1983), which was designed for adults, may not have provided a sensitive measure of emotional and behavioural problems in children (see McGough & Curry, 1992).

It is inconclusive from previous cross-lagged studies whether antecedent-consequence conditions between maternal mood and child adjustment problems are equivalent across internalising and externalising child behaviours, across child age groups, and between sexes. It is also unclear whether such temporal sequences are consistent across stable symptomatology and more transient, state-like fluctuations in mood and behaviour. To address these issues, two studies were conducted on temporal relations between maternal mood and child adjustment problems. Study 1 involved a national sample of families who were assessed on three occasions over a four-year period. Study 2 involved a smaller sample of mothers who completed daily assessments on themselves and their children over eight consecutive weeks. Although the same question was posed in both studies (who distresses whom?), fundamental differences in research design necessitated different approaches to unravelling temporal relations.

Unravelling Antecedent-Consequence Conditions

Several research designs may be suitable for examining temporal relations between maternal mood and child adjustment problems. First, case histories of mothers currently suffering from depression and children suffering from behavioural problems may be explored for clues about whether adjustment problems in children tend to occur before or after problems in their mothers. Unfortunately, memory errors and biases and unsystematic measures usually render the case history approach inappropriate for quantitative analysis.

An alternative approach may involve treatment studies of maternal depression and child behavioural problems that include outcome measures for other family members. Randomised trials have the significant advantage of inferring causal influences of maternal mood on child behaviour, or child behaviour on maternal mood – a sufficient condition of temporal relations. However, most clinical trials cannot address questions of how such problems originate in healthy populations.

Third, the approach undertaken in this thesis involves panel studies that measure change and mutual influences on maternal and child adjustment over time. Panel studies can be used to infer temporal relations and, unlike case histories and treatment studies, are not limited to clinical populations. Panel studies can be used to determine whether change in maternal emotional functioning tends to precede or follow changes in child behaviour and to examine the magnitude of their mutual influences. Due to the considerable difference in assessment interval (two years in Study 1 versus one day in Study

2), different quasi-experimental approaches were needed to analyse these panel data.

A novel development in psychological research has been the increased availability of large panel survey data sets containing variables of interest to psychological researchers (e.g., National Longitudinal Survey of Children and Youth, National Population Health Survey, Survey of Labour and Income Dynamics) (Statistics Canada, 1996; 1999; 2002). Unfortunately, the potential of these panel studies remains unrealised due somewhat to a lack of accessible guidelines for selecting appropriate analyses. Numerous techniques are available for analysing panel data, including (1) regression with lagged dependent variables, (2) repeated measures analysis of variance (ANOVA), (3) Structural equation modelling (SEM) with reciprocal and lagged effects, (4) log-linear regression, (5) growth curve models, (6) cross-lagged panel correlations, and (7) pooled time-series analysis (Johnson, 1995; Miller, 1997). For the purposes of the present studies, the first five of these seven techniques are not sufficiently adequate for investigating temporal order in continuous variables.

The first two approaches, regression with lagged dependent variables and repeated-measures ANOVAs, are generally appropriate for determining the influence of a fixed effect (e.g., unemployment) across several observations carried out over time (e.g., health outcome). ANOVA analyses can partition between-subjects and within-subjects sources of variance in quasi-experimental studies, but they cannot be applied to complex time-ordered designs involving a large number of observations (Soliday, Moore, & Lande, 2002). Both ANOVAs

and regression analyses carry an assumption of independent observations and are thus inadequate for handling high degrees of serial dependence, as would result in studies involving daily reports. Because observations that are close together in time are likely to be more similar than observations that are far apart in time, both regression and ANOVA would overestimate stability in the data.

With recent advances in software applications, some multivariate regression techniques have been superseded by the third approach, structural equation modelling (SEM, or “path modelling”). SEM is ideal in situations where several measures may be utilised to extract sources of error variance, thus allowing the researcher to study relations between composite “latent variables” (Kline, 1991). SEM may be applied as a hypothesis-testing (confirmatory) approach to the multivariate analysis of a structural theory bearing on some phenomenon that may or may not represent “causal” processes. Parameters between variables are represented by a series of structural regression equations that can be modelled pictorially. The hypothesised model can then be tested statistically in a simultaneous analysis of the entire system of variables to determine the extent to which it is consistent with the data. If goodness of fit is adequate, the model argues for the plausibility of postulated relations. If goodness of fit is inadequate, the tenability of the modeled relations is rejected (Byrne, 1994). SEM is robust in estimating reciprocal influences controlling for biases introduced by measurement errors and autocorrelated errors. However, as demonstrated in the study by Ge et al. (1995), SEM offers no direct test of temporal order aside from visual

comparison of beta weights assigned to cross-lagged pathways or comparison of the goodness of fit (e.g., χ^2 change) of several models to the data. Such approaches are clumsy and unreliable. Rogosa (1988) showed that coefficients produced by SEM models are essentially meaningless to temporal order. In a simulation study, Rogosa demonstrated that beta coefficients could fluctuate from .5 to -.5 depending on the length of time between waves of testing, thus showing that coefficients produced in SEM relate more to the length of time between assessments than to actual data.

Forth, Miller (1997) recommended log-linear models (or square contingency tables) to study temporal order between dichotomous variables. Based on matrix algebra (like SEM), log-linear models may account for change over time and estimate stage-like relationships between variables that inform about their antecedent-consequence conditions. Miller (1997) illustrated the benefits of this approach in a panel study of adolescent marijuana use and friends' marijuana use. However, the use of log-linear models is a crude approach in studies involving just two or three waves of data and, like logistic regression, is inappropriate in situations where it is undesirable to reduce the rich information contained in continuous variables to categorical outcomes.

Fifth, growth curve models can be used to explain change on a continuous outcome and have important advantages in studies where the emphasis is on stable factors that influence patterns of individual change. The strength of growth curve models lies in the capacity to describe individual differences in the trajectory and slope in the outcome variable over time – not

just the direction and magnitude of influences on group outcomes. Growth curve models, however, are incapable of capturing transactional relations between two continuous variables and require many waves of data to perform with adequate statistical power, thus restricting their application to panel studies (Rogosa, 1988).

There is no established convention in statistical methods for describing temporal order in panel data. In previous studies, the type of data collected, number and frequency of assessments, and sample sizes involved have all influenced researchers' choice of design and analysis. After an extensive review of the statistical methods that would validly test temporal order in studies involving both stable (biannual) change and frequent (daily) changes in maternal and child functioning, the sixth and seventh procedures were selected: cross-lagged correlation analysis (Forehand & McCombs, 1988) and pooled time-series analysis (Moore, OsGood, Larzelere, & Chamberlain, 1994). Both methods have important advantages in analysing the type of data used in Studies 1 and 2 of this thesis and, therefore, are described here in some detail.

Procedure 1: Cross-lagged Panel Correlations

Cross-lagged panel correlation analysis is an underutilised and occasionally misapplied technique that provides insight into the temporal order of two or more constructs that are measured repeatedly over time (Cook & Campbell, 1979; Humphreys, 1991). Passive observations obviously do not inform about causality *per se* and it is generally impossible to determine the

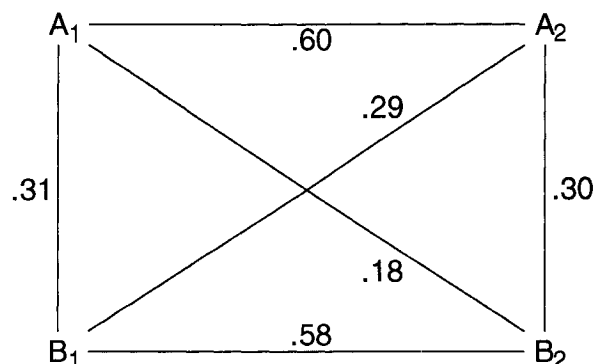


Figure 2. Hypothetical cross-lagged panel correlations between Factors A and B ($N = 500$).

magnitude of causal effects without an experimental design. Confusion between temporal order and causal direction has resulted in negative critiques of cross-lagged panel correlations, with some entirely dismissive of the technique (e.g., Rogosa, 1980; 1988). However, antecedent-consequence conditions – not causal direction, but a necessary condition for causality – can be studied effectively using panel data and cross-lagged panel correlations.

In a hypothetical example shown in Figure 2, two factors, A and B, are measured on 500 participants on two occasions. Differences between cross-lagged correlations may be tested using a r -to- z transformation and, if significant, may indicate temporal order (e.g., $r_{A_1, B_2} < r_{B_1, A_2}$ may suggest that B precedes A). Cross-sectional and test-retest associations provide the interpretative framework for differences between cross-lagged correlations. “Quasi-stationarity” (similarity in test-retest reliability of A and B) and synchronicity (similarity in cross-sectional correlations between A and B) are necessary conditions for inferring temporal sequence from differences between

cross-lagged correlations (Kenny, 1975). That is, $r_{A1,B2} > r_{B1,A2}$ indicates that A precedes B if, and only if, differences between synchronous correlations ($r_{A1,B1}$ and $r_{A2,B2}$) and test-retest correlations (r_{A1A2} and r_{B1B2}) are non-significant (using, for instance, the conservative alpha level of .25 recommended by Kenny [1975]). In the example shown in Figure 2, conditions of quasi-stationarity and synchronicity are met, passing Kenny's "test for spuriousness" (p. 887). Even though the magnitudes of the cross-lagged correlations are small, their difference is significant, $z = 2.26$, $p = .02$, indicating that B tends to precede, more than it follows, A. Again, it is acknowledged that whether B actually causes A is undetermined.

Kenny's (1975) procedure for analysing differences between cross-lagged panel correlation using a modified r -to- z transformation formula, while testing quasi-stationarity and synchronicity, is as follows:

let N be sample size,

$$\begin{aligned}
 k = & (r_{A1,B1} - [r_{B1,B2}][r_{A1,B2}])(r_{A2,B2} - [r_{B1,B2}][r_{B1,A2}]) \\
 & + (r_{A1,A2} - [r_{A1,B1}][r_{B1,A2}])(r_{B1,B2} - [r_{A1,B1}][r_{A1,B2}]) \\
 & + (r_{A1,B1} - [r_{A1,A3}][r_{B1,A2}])(r_{A2,B2} - [r_{A1,A2}][r_{A1,B2}]) \\
 & + (r_{A1,A2} - [r_{A1,A4}][r_{A2,B2}])(r_{B1,B2} - [r_{A2,B2}][r_{B1,A2}])
 \end{aligned}$$

and,

$$z = \frac{\sqrt{N}(r_{A1,B2} - r_{B1,A2})}{\sqrt{(1 - r_{A1,B2}^2)^2 + (1 - r_{B1,A2}^2)^2 - k}}$$

(Hahn [1999] developed a DOS program that performs this calculation). In interpreting the magnitude of cross-lagged correlations, it has also been

recommended that partial correlations be used as an additional control for synchronous associations between A and B (Elgar & Arlett, 2002; Forehand & McCombs, 1988). In a panel study of adolescent conflict and depressive symptoms, Elgar and Arlett (2002) demonstrated that the magnitude of a cross-lagged association may be an artifact of high test-retest reliability and cross-sectional correlations. Additionally, Rogosa (1988) recommended the use of at least three or more cycles of panel data so that the reliability of these differences may be tested. Study 1 of this thesis adhered to each of these recommendations.

In panel studies involving a small number of observations and a large number of participants, cross-lagged panel correlations provide a simple, valid method for unravelling antecedent-consequence conditions. However, the number of comparisons involved can become untenable with many observations. A two-wave study requires just two comparisons of standard and partial cross-lagged panel correlations, but a four-wave study requires 12 sets of comparisons and a five-wave study requires 20 comparisons. Clearly, in a study involving dozens of daily reports collected over weeks and months, cross-lagged panel correlations have two serious shortcomings – cumulative Type I error and lacking capacity to separate serial dependence (autocorrelation) from estimates of quasi-stationarity.

Procedure 2: Pooled Time-Series Analysis

The procedure that is best suited to examination of cycles, trends, and

drifts in daily report data is time series analysis. A time series is a sequence of measurements over equal periods of time, such as days, months, or years. In time series analysis, time-dependent regularities in the data are described using a mathematical model that statistically removes their influence before assessing the possible effects of other variables (Cook & Campbell, 1979). Variables that are believed to affect the process are called the exogenous or independent variables. For example, if change in maternal mood is fixed as an exogenous variable, then its influence on daily reported child behaviour can be estimated. The variance explained in such a model can then be compared to its converse; i.e., child behavioural problems held constant to predict change in maternal mood. Such tests can also be used to study delayed effects by varying the time interval between independent and dependent variables. The approach is conceptually analogous to cross-sectional panel correlations but the statistical analyses are quite different.

Despite the fact that much clinical research involves data that are collected from many points in time, relatively few psychological methods take full advantage of the richness of time-ordered data (Johnson, 1995; Moore et al., 1994; Soliday et al., 2002). One reason why time series modelling is underutilised in behavioural research could be that ordinary time series assume more observations than are typically available in clinical data. Cook and Campbell (1979) recommend at least 50 data points. Another reason is that ordinary time series analysis is limited to a single case and thus provides no basis for generalisation to a larger population. Third, visual analysis of time

series data is cumbersome when large individual differences exist in the data – a common scenario in psychological research.

Pooled time series analysis avoids these limitations by exploiting many observations from relatively small samples, thereby requiring fewer observations per subject than are needed in ordinary time series. Pooled time-series models have the power advantage over ordinary time-series models by using regression to perform within-subject analysis and it is far more tolerant of missing data than ANOVA. A pooled time-series model is essentially a “replicated time-series” which Cook and Campbell (1979) describe as one of the strongest quasi-experimental designs available because it allows for the assessment of change while controlling for selection, maturation, and history. Previous applications of pooled time-series analysis of daily clinical events include studies of foster children's behavioural disturbances as a function of the number of children residing in the foster home (Moore et al., 1994), spousal interactions in intact marriages as a function of marital duration and number of children in the family (Johnson, 1995), and behavioural side effects of steroid medications used to treat steroid-sensitive nephrotic syndrome in paediatric settings (Soliday et al., 2002).

Pooled times-series analysis involves a regression-based approach to examining trends over time utilising one of two model types: a fixed effect model, which specifies differences among individuals in separate intercept terms, or a random effects model, which allows differences among individuals by including an additional error component (Jaccard & Wan, 1993). Results are

communicated similarly to regression analysis using beta coefficients (β) and proportions of predicted variance (R^2). For this coefficient to be valid, however, the analysis must correct for violations of the assumption of independence between observations. In pooled time-series models, this violation takes two forms: between-subject variance (greater similarity among all observations for the same subject) and serial correlation, or “autocorrelation” (greater similarity among observations closer together in time relative to those further apart). Thus, analyses require two data transformations.

First, individual differences are controlled so that only within-subject variation is examined. In fixed effects models, dummy variables can be used to remove all between-subjects variance from the pooled time-series analyses (Dielman, 1989). The number of dummy variables needed equals the number of participants in the sample minus one. This approach is equivalent to subtracting individuals' scores from their own mean and performing a regression on the differences. Because dummy variables absorb all differences across participants, the remaining analysis is based strictly on within-subject variation over time.

Second, because autocorrelation violates the standard statistical assumption of independent residuals, it is addressed using a version of generalised least squares in which data are transformed to remove the lack of independence (Moore et al., 1994). The procedure involves (1) pooling the data and estimating the regression model of interest, (2) estimating the degree of correlation among the residuals that are adjacent in time, (3) transforming the

dependent variables to remove that correlation, (4) conducting the regression analysis once again on the transformed variables, and (5) examining the correlations among residuals from this second analysis to ensure that the transformation was successful. This procedure, illustrated by Soliday et al. (2002), utilises the Durbin-Watson statistic (available in SPSS regression analysis), which can be transformed to an autocorrelation estimate, r :

$$r = \frac{(2 - dw)}{2}$$

where “dw” refers to the Durbin-Watson value. Dependent variables are then transformed using the formula:

$$X^* t = X_t - r(X_{t-1})$$

where $X^* t$ is the new version of the variable at time t , t is the original version, r is the autocorrelation estimate from the Durbin-Watson statistic, and $t - 1$ is the time period previous to t . Each newly transformed value becomes the difference between the old value and the product of r and the value from the previous assessment. According to Ostrom (1990), one or two iterations of the procedure is normally sufficient to correct for autocorrelation, using a threshold of $r = -.15$. The procedure is tedious, but failure to correct for autocorrelation can lead to overestimates of the statistical significance of time series relations.

With these corrections, regression may be performed on the pooled data

with dummy variables controlling for between-subject variance and transformed data controlling for autocorrelation. Consideration should also be paid to effects that cannot be attributed to ongoing trends (e.g. maturation or clinical improvement during the course of treatment). Fortunately, pooled time-series analysis controls for history by exploiting a key feature of the replicated time-series design. In ordinary time-series analysis, investigators typically remove systematic time trend when correcting for serial correlation, which is a major reason for the complexity of these corrections. Autoregressive integrated moving averages (ARIMA) models are helpful when the goal is forecasting future trends or assessing the impact of a single event, but ARIMA models are not generally useful in psychological studies where the investigator may be inclined to use polynomial regression to control for ongoing time trends while exploring linear relations. More relevant to the objective of this thesis is the capability of pooled time-series analysis to test the significance of influences between continuous variables that are measured over many days on a relatively small sample of participants. This analytical approach was utilised in Study 2.

Objectives and Research Hypotheses

To reiterate, the general objective of Studies 1 and 2 was to delineate temporal influences between maternal mood and child adjustment problems across varying time intervals. The question at issue was whether the influence that maternal depression has upon child functioning is greater than or less than the influence that child behavioural problems have on maternal functioning. In

Study 1, antecedent-consequence conditions were studied in a cross-lagged study of maternal mood and child adjustment problems using data from a three-wave four-year panel study. In Study 2, similar relations were studied in a pooled time-series study using daily report data. It was hypothesised that symptoms of maternal and child maladjustment would be positively correlated and stable over time and that the presence of maternal depression would increase the risk of child behavioural problems in the future, and vice versa. It was expected that differences between cross-lagged correlations and pooled time-series models would indicate whether maternal depression is more of an antecedent or a consequence of behavioural problems in children and whether such temporal relations differ between transient and stable components of such symptomatology.

Secondary questions addressed in these studies are whether internalising and externalising child behaviours are equally related to maternal mood (Study 1) and whether dimensions of maternal negative mood (e.g., anxiety, fatigue, sadness) are differentially related to disruptive child behaviour (Study 2). With consideration of the interpersonal mechanisms that transmit bidirectional risk between maternal and child functioning, it was hypothesised that externalising behaviour in the child (e.g., aggression and hyperactivity) would relate more to maternal depression than child internalising behaviours (e.g., anxiety, low mood). It was also hypothesised that mothers' symptoms of fatigue, anger, and depression, compared to mothers' symptoms of anxiety or confusion, would relate more strongly to disruptive child behaviours.

Questions pertaining to intergenerational transfer of psychological problems in a community population have implications for how maternal depression and child behaviour problems develop and interact over time. Their clinical relevance lies in understanding how undetected or untreated emotional problems in mothers contribute to child adjustment problems or impede interventions for the child that rely on parent coaching and consistent implementation of parent-facilitated behavioural interventions. These questions are also relevant to understanding how undetected or untreated behavioural problems in children contribute to maternal distress or impede the treatment of depression in mothers. Whilst it is well established that transactional relations between family members exist in adjustment problems, a gap exists in the research literature about their mutual influences and temporal properties. Determining *when* maternal mood is most influential on child functioning and *when* child behaviour is most influential on maternal functioning may help to identify the more salient mechanisms of bidirectional risk and resiliency for maternal depression and child behavioural problems. Such knowledge also translates to public health policy that promotes healthy development in children of depressed mothers and that helps parents of children with adjustment problems create supportive, healthy home environments.

**STUDY 1: FOUR-YEAR CROSS-LAGGED STUDY OF MATERNAL MOOD
AND CHILD ADJUSTMENT PROBLEMS**

Objectives

Despite the considerable body of research on the mutual influences of maternal mood and child adjustment problems, the antecedent-consequence conditions of these constructs remain unclear. Furthermore, previous cross-lagged studies, such as Forehand and McCoombs (1988) and Ge et al. (1995), have not clearly demonstrated whether such temporal relations are equivalent across internalising and externalising child behaviours, across child age groups, and between sexes. To address these issues, cross-lagged correlations and regressions were used to examine temporal relations between maternal depressive symptoms and three domains of child functioning – hyperactivity, aggression, and emotional problems. The objectives of Study 1 were to determine whether the influence of maternal depressive symptoms on child adjustment problems is less than or greater than the influence of child adjustment problems on maternal mood, and to examine the magnitude of these mutual influences over a four-year period, independent of prior symptoms and shared risk factors. It was expected that maternal and child symptoms would be related and stable and would show mutual influences over time.

Although previous studies did not lend well to firm hypotheses about temporal relations between maternal mood and child adjustment problems, the

study builds upon previous research in several respects. As noted in the previous section, applications of cross-lagged panel correlations to infer temporal order from panel data were criticised because differences between cross-lagged correlations may result from factors unrelated to causation, such as differences in reliability of the two variables under study or differences in synchronous associations (Miller, 1997; Rogosa, 1980). The present study utilised both Kenny's (1975) test for consistency in test-retest correlations (quasi-stationarity) and cross-sectional correlations (synchronicity) and partial cross-lagged correlations to additionally control for within-time associations. To also study the reliability of temporal relations based on cross-lagged correlations, three cycles of panel data were used (Rogosa, 1988).

Methods

Participants

Data were utilised from the National Longitudinal Survey of Children and Youth (NLSCY), a national study administered by the Government of Canada that was designed to provide information on the development and well-being of children as they grow from infancy to adulthood (Statistics Canada, 1996). At the time of the present study, the first three NLSCY data cycles were available for secondary analysis. The sampling procedure used by Statistics Canada identified dwellings with eligible children (0 to 14 years) in all Canadian provinces. Sampling frames were adopted from the Labour Force Survey (Statistics Canada, 2002) and National Population Health Survey (Statistics

Canada, 1999) to ensure that the NLSCY sample represented all socioeconomic conditions in Canada. Eligible participants were randomly selected from each frame and 86.3% of those contacted agreed to participate.

From each household, data were provided by the adult most knowledgeable about the target child (in most cases, the mother). If there were more than one eligible child in the family, the target child was randomly selected. Eligibility criteria for the secondary data analyses in the present study were that this adult was the child's mother (birth, step, adoptive, or foster) and that the child was between 4 and 11 years of age, resulting in a sample of 20,849 mother-child dyads in Cycle 1, 18,334 in Cycle 2 and 16,581 in Cycle 3. This age criterion was chosen so that all children in the selected sample were assessed using the same measures. Demographic characteristics of the sample are provided in Table 1. Based on Cycle 1 data, participants who left the study at Cycles 2 and 3 were not significantly different than participants who remained in the study in terms of maternal depressive symptoms and child adjustment. As well, sample attrition over the course of the study was equivalent across categories of income adequacy, mother's relationship with the child, and mother's age group.

Measures

Maternal depressive symptoms were self-reported using a modified version of the CES-D (M. Boyle, personal communication, 1994). Using data from the Ontario Child Health Study (Offord et al., 1987), Statistics Canada

Table 1

Demographic Characteristics of the Sample in Study 1 at Cycle 1

<i>N</i>	20,849
Mother's relationship to child	
Birth mother (%)	20,520 (98.42)
Adoptive mother (%)	206 (0.99)
Stepmother (%)	90 (0.43)
Foster mother (%)	33 (0.16)
Number of children (SD)	2.36 (1.07)
Mean age of mother (SD)	33.04 (5.86)
Mean age of mother at child's birth (SD)	27.54 (4.89)
Age of mother	
15 - 24 years (%)	1,443 (6.92)
25 - 29 years (%)	4,076 (19.55)
30 - 34 years (%)	7,272 (34.88)
35 - 39 years (%)	5,420 (26.00)
40+ years (%)	2,687 (12.89)
Teenage mother at time of child's birth (%)	940 (4.51)
Single-parent family (%)	480 (14.50)
Highest education of mother	
Some trade, technical or vocational schooling (%)	3,190 (15.30)
Some college (%)	4,316 (20.70)
Some undergraduate (%)	1,689 (8.10)
Trade, technical or vocational certificate (%)	3,541 (16.98)
College diploma (%)	3,421 (16.41)
Undergraduate degree (%)	3,920 (18.80)
Postgraduate degree (%)	411 (1.98)
Income adequacy	
Lowest (%)	786 (3.48)
Lower middle (%)	3,996 (17.53)
Middle (%)	7,882 (34.52)
Upper middle (%)	7,507 (32.97)
Highest (%)	2,660 (11.70)

removed eight items from Radloff's (1977) original scale, including one item from the "positive affect" factor, two items from the "somatic and retarded activity" factor, one item from the "interpersonal factor," and four items that did not load on any factor. The "depressed affect" factor was left intact. The remaining 12 items that were used in the NLSCY are shown in Appendix A. Scores ranged from 0 to 36 with higher scores indicating the presence of more depressive symptoms. Items included nine negative statements (e.g., "I felt lonely") and three reverse-scored positive statements (e.g., "I enjoyed life"). Respondents indicated how well each statement describes their mood over the previous two weeks using a 3-point scale (0 = not true, 1 = somewhat true, 2 = very true). This modified version of the CES-D showed good internal consistency in the present study ($\alpha = .82$).

The Ontario Child Health Study (OCHS) behaviour scale was used to measure mothers' ratings of three areas of child functioning: hyperactivity, aggression, and emotional problems (Boyle et al., 1987). This scale was developed initially to screen for child psychiatric disorder in the OCHS according to DSM-III-R (American Psychiatric Association, 1987) diagnostic criteria. Its items were interspersed throughout the NLSCY survey with other measures of children's health, academic, and social functioning. Each item of the scale described a problem behaviour to which the mother indicated its frequency on a three-point scale (0 = never, 1 = sometimes, 2 = often). Eight questions measured hyperactivity (e.g., "how often would you say your child can't sit still, is restless or hyperactive?"), six questions measured aggression

(e.g., “how often would you say your child kicks, bites, or hits other children?”), and eight questions measured child emotional problems (e.g., “how often would you say your child is nervous, high strung, or tense?”). Scores range from 0 to 16 on hyperactivity and emotional problems scales and 0 to 12 on the aggression scale with higher scores reflecting more symptoms. Brunshaw and Szatmari (1988) found that hyperactivity, aggression and emotional disorder scales had adequate predictive validity in relation to psychiatric diagnoses of hyperactivity disorder (69% sensitivity, 82% specificity), conduct disorder (74% sensitivity, 75% specificity) and depression (52% sensitivity, 93% specificity), respectively. In the present study, the OCHS behaviour scale showed adequate to good internal consistency ($\alpha = .82$ for hyperactivity, $\alpha = .77$ for aggression, and $\alpha = .84$ for emotional problems).

Socio-demographic information was also collected on the family characteristics such as household income, the number of persons in the family, and the mother’s age and highest level of education. Household income was adjusted for the number of persons in the family using a 5-point ranking of income adequacy (Lowest [$< \$10,000$ for one to four persons or $< \$15,000$ for five or more persons], Lower middle [$\$10,000 - \$14,999$ for one or two persons, $\$10,000 - \$19,999$ for three or four persons, or $\$15,000 - \$29,999$ for 5 or more persons], Middle [$\$15,000 - \$29,999$ for one or two persons, $\$20,000 - \$39,999$ for three or four persons, or $\$30,000 - \$59,999$ for five or more persons], Upper middle [$\$30,000 - \$59,999$ for one or two persons, $\$40,000 - \$79,999$ for three or four persons, or $\$60,000 - \$79,999$ for five or more

persons], and Highest [$> \$60,000$ for one or two persons or $> \$80,000$ for three or more persons].

Procedure

In the data utilised in the present study, mothers provided all information on behalf of the child and the family to questions on demographic characteristics, health, family functioning, and education. Interviews were conducted either in person or by telephone by Statistics Canada interviewers between December 1994 and April 1995 (Cycle 1), between December 1996 and April 1997 (Cycle 2), and between October 1998 and June 1999 (Cycle 3). Participants were not paid.

Data Analysis

Unweighted data were used in all analyses. Correlations were used to examine the stability in, and relations between, maternal depression and each domain of child adjustment. Cross-lagged panel correlations were used to study antecedent-consequence conditions in maternal and child variables over time lags of two and four years (Cook & Campbell, 1979). Differences between cross-lagged correlations were tested using Kenny's (1975) *r*-to-*z* transformation and, if significant, were used to infer a temporal order in maternal and child outcomes (e.g., $r_{A1,B2} > r_{B1,A2}$ indicates that A precedes B). Logistic hierarchical regression analysis was used to estimate the risk of future child adjustment problems attributed to maternal depression (controlling for present child

functioning) and the risk of future maternal depression attributed to child adjustment problems (controlling for present maternal functioning).

Dichotomous variables were based on cutoffs identifying the upper 10% of the sample on each measure (corresponding to an epidemiologically-defined level of “deviance” but not necessarily representative of true prevalence of disorder). Regression models included four-year time lags and blocks of variables entered hierarchically to control for the influences of shared risk factors (mother’s age, income adequacy, education, and family size) and prior levels of functioning. All analyses were performed at a secure research facility at a Statistics Canada Regional Office in Halifax, Nova Scotia, Canada.

Results

Maternal depressive symptoms and child adjustment problems both moderately correlated with socio-demographic variables. As shown in Table 2, mothers who reported more symptoms tended to be younger and to have less education, lower income, and smaller families. Associations were also found between these socio-demographic variables and mother reports of child hyperactivity, aggression, and emotional problems. These correlations were small, explaining just 1 to 4% of shared variance, but indicated that both maternal mood and child adjustment were associated to some extent with demographic indicators.

Correlations between maternal depressive symptoms and child

Table 2

Correlations Between Demographic Characteristics and Maternal Depression and Child Adjustment Problems (Cycle 1)

<i>Measure</i>	<i>Mother's age[‡]</i>	<i>Mother's education[§]</i>	<i>Income adequacy[§]</i>	<i>Family size[‡]</i>
Maternal depression	-.12**	-.11**	-.20**	-.08**
Child hyperactivity	-.12**	-.09**	-.09**	-.11**
Child aggression	-.11**	-.04**	-.08**	.04**
Child emotional disorder	-.04**	-.02	-.03*	-.09**

[‡] Pearson Correlation.

[§] Spearman Correlation.

*p < .01. **p < .001.

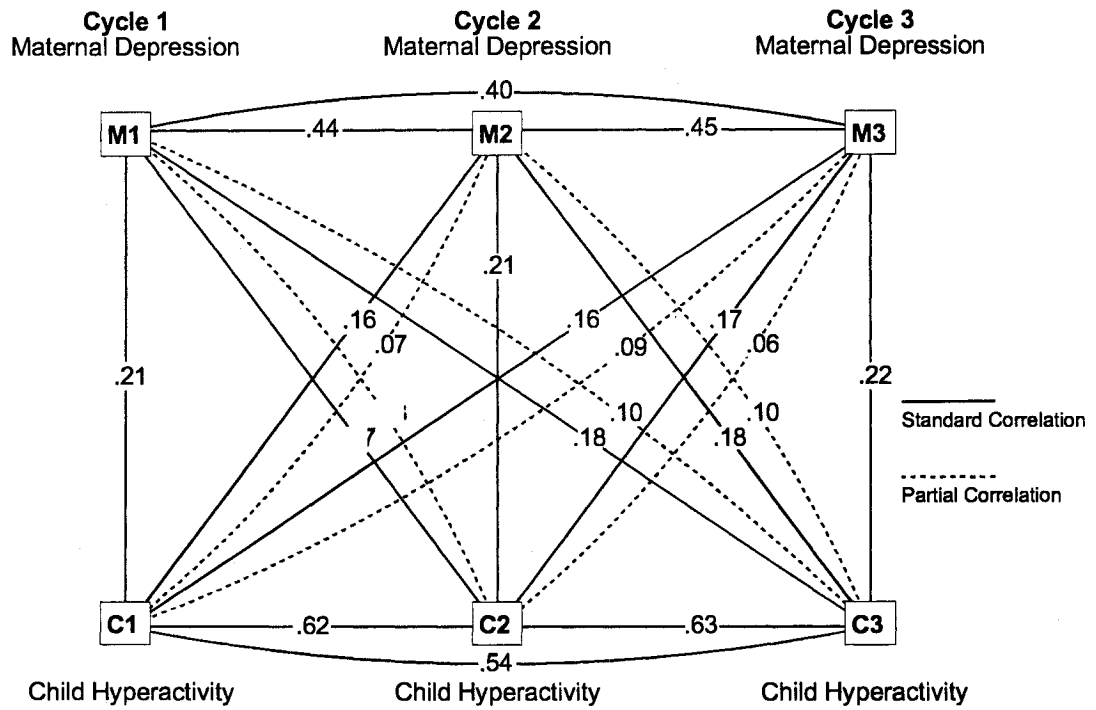


Figure 1. Cross-sectional, test-retest, and cross-lagged correlations between maternal depression and child hyperactivity across Cycles 1, 2, and 3 (all paths significant, $p < .01$).

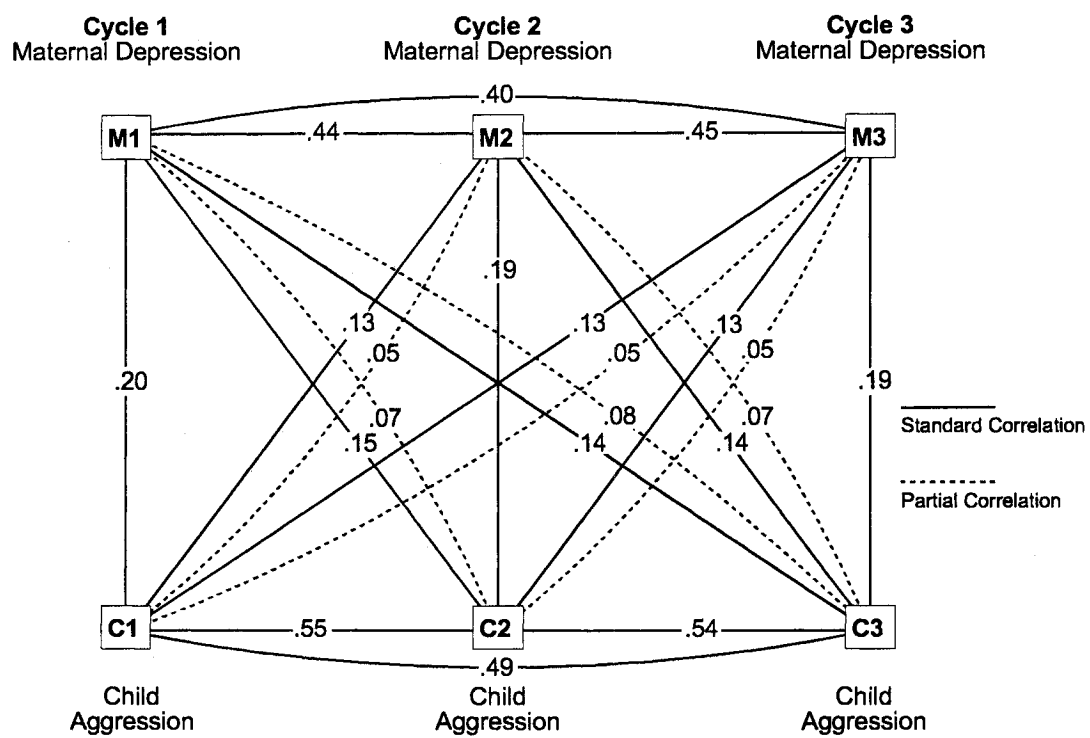


Figure 2. Cross-sectional, test-retest, and cross-lagged correlations between maternal depression and child aggression across Cycles 1, 2, and 3 (all paths significant, $p < .01$).

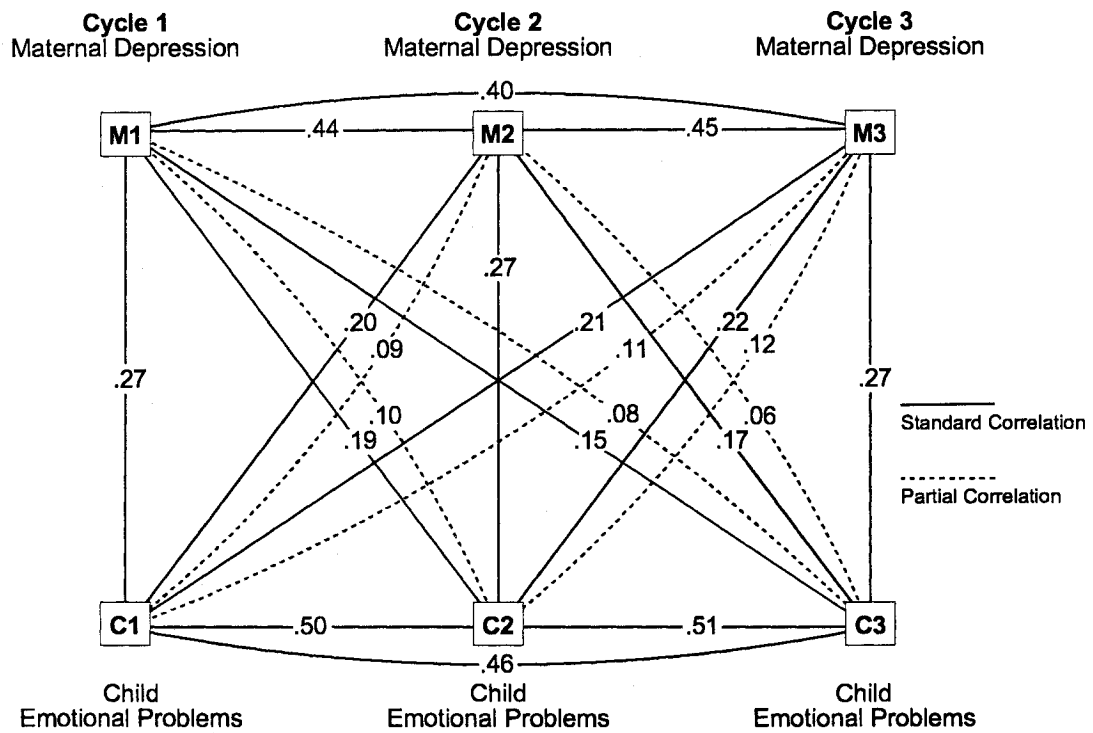


Figure 3 Cross-sectional, test-retest, and cross-lagged correlations between maternal depression and child emotional problems across Cycles 1, 2, and 3 (all paths significant, $p < .01$).

adjustment problems across Cycles 1, 2 and 3 are shown in Figure 3 (maternal depression and child hyperactivity), Figure 4 (maternal depression and child aggression), and Figure 5 (maternal depression and child emotional problems). Positive correlations were found between maternal depression and each domain of child adjustment. In Cycle 1, maternal depressive symptoms correlated with child hyperactivity, $r(12,477) = .21, p < .001$, child aggression, $r(12,462) = .20, p < .001$, and child emotional problems, $r(12,489) = .27, p < .001$. These relations did not change significantly in Cycles 2 and 3. Also, correlations between subscales of the OCHS behaviour scale from Cycles 1, 2, and 3 showed that child adjustment problems were interrelated: between hyperactivity and aggression ($r_s = .41$ to $.46, p < .001$), between hyperactivity and emotional problems ($r_s = .41$ to $.45, p < .001$), and between aggression and emotional problems ($r_s = .36$ to $.45, p < .001$).

Between Cycles 1 and 3, the four-year test-retest reliability in maternal depressive symptoms was $r(12,683) = .40, p < .001$ and in child adjustment problems was $r(3,666) = .54, p < .001$ (hyperactivity), $r(3,657) = .49, p < .001$ (aggression), and $r(3,673) = .46, p < .001$ (emotional problems). Unfortunately, because test-retest reliability was higher in child hyperactivity than maternal mood ($r_{C1,C3} > r_{M1,M3}$ and $r_{C1,C3} > r_{M1,M3}$), the quasi-stationarity assumption was not met in some comparisons of their cross-lagged correlations. As well, because test-retest reliability in child aggression was higher than maternal mood ($r_{C1,C2} > r_{M1,M2}$, $r_{C1,C3} > r_{M1,M3}$ and $r_{C1,C3} > r_{M1,M3}$), the quasi-stationarity assumption was not met in their cross-lagged correlations.

Cross-lagged correlations between maternal depression and child adjustment problems between cycles were positive and significant, but dropped substantially after within-cycle associations were statistically controlled. For instance, as shown in Figure 3, maternal depressive symptoms in Cycle 1 correlated with child hyperactivity in Cycle 2, $r_{M1,C2}(8,577) = .17, p < .001$, but the corresponding partial correlation, controlling for child hyperactivity at Cycle 1, was smaller but still statistically significant, $r_{M1,C2}(8,432) = .08, p < .001$. Similar changes between correlations and partial correlations were observed in relations between maternal depression and each child problem area over Cycles 1 and 2, Cycles 2 and 3, and Cycles 1 and 3, indicating that uncontrolled cross-lagged correlations were inflated by either the stability of mother and child symptoms, their cross-sectional correlations, or both. Therefore, both standard and partial correlations were used in cross-lagged correlation analyses (Rogosa, 1980).

Table 3 shows the results of cross-lagged correlation comparisons between maternal mood and child hyperactivity, aggression and emotional problems. Cross-lagged correlations involving maternal depression and child hyperactivity were not statistically different between Cycles 1 and 2 ($r_{M1,C2} \approx r_{M2,C1}$), but were different between Cycles 1 and 3 ($r_{M1,C3} > r_{M3,C1}$) and between Cycles 2 and 3 ($r_{M2,C3} > r_{M3,C2}$). As shown in Figure 1, partial correlations between maternal depression and future child hyperactivity were greater than partial correlations between child hyperactivity and future maternal depression. Cross-lagged correlations between maternal depression and child aggression

Table 3

Differences Between Cross-Lagged Panel Correlations

Comparison	Z-score	
	Standard Correlations	Partial Correlations
Maternal depression and child hyperactivity[†]		
$r_{(M1, C2)} \approx r_{(C1, M2)}$	1.21	1.19
$r_{(M2, C3)} > r_{(C2, M3)}$	0.86 [†]	3.40 ^{**†}
$r_{(M1, C3)} > r_{(C1, M3)}$	1.65 ^{*†}	0.81 [†]
Maternal depression and child aggression[§]		
$r_{(M1, C2)} \geq r_{(C1, M2)}$	2.34 ^{**†}	2.33 ^{**†}
$r_{(M2, C3)} > r_{(C2, M3)}$	0.83 [†]	1.65 ^{*†}
$r_{(M1, C3)} > r_{(C1, M3)}$	0.80 [†]	2.40 ^{**†}
Maternal depression and child emotional problems[¶]		
$r_{(M1, C2)} \approx r_{(C1, M2)}$	-1.18	-1.04
$r_{(M2, C3)} < r_{(C2, M3)}$	-4.19 ^{**}	-4.99 ^{**}
$r_{(M1, C3)} < r_{(C1, M3)}$	-4.49 ^{**}	-2.43 ^{**}

*p < .05.

**p < .01.

† Assumption of quasi-stationarity was not met ($\alpha > 25\%$).

‡ See Figure 3.

§ See Figure 4.

¶ See Figure 5.

followed a similar pattern. Cross-lagged (partial) correlations between maternal depression and future child aggression were greater than between child aggression and future maternal depression ($r_{M1,C2} > r_{M2C1}$, $r_{M2,C3} > r_{M3,C2}$, and $r_{M1C3} > r_{M3,C1}$). However, an inverted pattern of results was found in cross-lagged correlations involving maternal depression and child emotional problems. As shown in Figure 3, two of three cross-lagged correlations between maternal depression and future child emotional problems were significantly lower than between child emotional problems and future maternal depression ($r_{M2,C3} < r_{M3,C2}$ and $r_{M1,C3} < r_{M3,C1}$). In these comparisons, differences were significant in both standard and partial correlations and the quasi-stationarity assumption was held.

In separate cross-lagged correlation analyses for male and female children and younger and older children (using a median split of children's ages), no sex or age differences were found in either the stability of measures, their synchronous relations, or their cross-lagged correlations over 2- and 4-year time lags.

Logistic regression models were used to better quantify the risk of maternal depression attributable to prior child adjustment problems and the risk of child adjustment problems attributable to prior maternal depression. These models utilized data from Cycles 1 and 3, thereby setting a four-year time lag between predictor and criterion variables. Using 90th percentile cutoffs, it was found that the prevalence of child behavioural problems in Cycle 1 was 2.0 to 2.4 times higher when maternal depressive symptoms were also above the

Table 4

Number of Children with Adjustment Problems in Families with Depressed and Non-depressed Mothers (Cycle 1) (%)

Child adjustment problem	Non-depressed mothers	Depressed mothers	χ^2
	18,159 (88.8)	2,279 (11.2)	
Hyperactivity 1,861 (14.7)	1,497 (13.3)	369 (26.7)	177.05*
Aggression 1,753 (13.8)	1,337 (12.4)	359 (26.1)	190.31*
Emotional disorder 1,751 (13.8)	1,336 (12.0)	398 (28.8)	289.11*

Note: Cutoffs on point scales of the OCHS identified slightly more than the upper 10%. Row Ns do not sum due to missing data.

* $p < .001$.

Table 5

Hierarchical (Logistic) Regression Analysis of Child Adjustment Problems in Cycle 1 Predicting the Risk of Depression in Mothers in Cycle 3.

<i>Variable</i>	β	<i>SE</i> (β)	<i>OR</i> [95% <i>CI</i>]
<i>(a) Presence of child hyperactivity predicting risk of maternal depression:</i>			
Block 1			
Mother's age	0.00	.01	1.00 [0.98 - 1.02]
Income adequacy	-0.36***	.06	0.70 [0.62 - 0.78]
Mother's education	-0.10**	.03	0.91 [0.85 - 0.96]
Family size	-0.21***	.05	0.81 [0.73 - 0.90]
Block 2			
Maternal depression (Cycle 1)	1.68***	.13	5.39 [4.20 - 6.91]
Block 3			
Child hyperactivity (Cycle 1)	0.24	.14	1.27 [0.96 - 1.69]
Model $R^2 = .06$. $\Delta R^2 = .02$ for Block 1; $\Delta R^2 = .03$ for Block 2; $\Delta R^2 = .01$ for Block 3.			
<i>(b) Presence of child aggression predicting risk of maternal depression:</i>			
Block 1			
Mother's age	0.00	.01	1.00 [0.98 - 1.02]
Income adequacy	-0.37	.06	0.69 [0.62 - 0.77]
Mother's education	-0.10**	.03	0.90 [0.85 - 0.96]
Family size	-0.21***	.05	0.81 [0.73 - 0.90]
Block 2			
Maternal depression (Cycle 1)	1.70***	.13	5.45 [4.25 - 6.99]
Block 3			
Child aggression (Cycle 1)	0.46**	.14	1.59 [1.21 - 2.08]
Model $R^2 = .06$. $\Delta R^2 = .02$ for Block 1; $\Delta R^2 = .03$ for Block 2; $\Delta R^2 = .01$ for Block 3.			
<i>(c) Presence of child emotional problems predicting risk of maternal depression:</i>			
Block 1			
Mother's age	0.00	.01	1.00 [0.98 - 1.02]
Income adequacy	-0.36***	.06	0.70 [0.62 - 0.78]
Mother's education	-0.10**	.03	0.90 [0.85 - 0.96]
Family size	-0.21***	.05	0.81 [0.73 - 0.90]
Block 2			
Maternal depression (Cycle 1)	1.69***	.13	5.42 [4.23 - 6.95]
Block 3			
Child emotional problems (Cycle 1)	0.69***	.13	2.00 [1.54 - 2.58]
Model $R^2 = .06$. $\Delta R^2 = .02$ for Block 1; $\Delta R^2 = .03$ for Block 2; $\Delta R^2 = .01$ for Block 3.			
* $p < .05$. ** $p < .01$. *** $p < .001$.			

Table 6

*Hierarchical (Logistic) Regression Analysis of Depression in Mothers in Cycle 1
Predicting Child Adjustment Problems in Cycle 3*

<i>Variable</i>	β	<i>SE</i> (β)	<i>OR</i> [95% CI]
<i>(a) Presence of maternal depression predicting risk of child hyperactivity:</i>			
Block 1			
Mother's age	0.00	.01	1.00 [0.97 - 1.02]
Income adequacy	-0.29***	.07	0.75 [0.65 - 0.86]
Education	-0.07*	.04	0.93 [0.86 - 1.00]
Family size	-0.19***	.06	0.83 [0.73 - 0.94]
Block 2			
Child hyperactivity (Cycle 1)	2.06***	.14	7.83 [5.93 - 10.34]
Block 3			
Maternal depression (Cycle 1)	0.40*	.21	1.49 [0.98 - 2.25]
Model $R^2 = .11$. $\Delta R^2 = .02$ for Block 1; $\Delta R^2 = .08$ for Block 2; $\Delta R^2 = .01$ for Block 3.			
<i>(b) Presence of maternal depression predicting risk of child aggression:</i>			
Block 1			
Mother's age	-0.05***	.01	0.95 [0.92 - 0.98]
Income adequacy	-0.23**	.08	0.80 [0.69 - 0.92]
Education	-0.02	.04	0.98 [0.91 - 1.06]
Family size	0.14*	.06	1.15 [1.03 - 1.30]
Block 2			
Child aggression (Cycle 1)	1.99***	.15	7.33 [5.50 - 9.76]
Block 3			
Maternal depression (Cycle 1)	0.24	.22	1.28 [0.83 - 1.97]
Model $R^2 = .09$. $\Delta R^2 = .01$ for Block 1; $\Delta R^2 = .08$ for Block 2; $\Delta R^2 = .00$ for Block 3.			
<i>(c) Presence of maternal depression predicting risk of child emotional problems:</i>			
Block 1			
Mother's age	-0.05***	.01	0.95 [0.92 - 0.97]
Income	-0.14*	.07	0.87 [0.77 - 0.99]
Education	0.01	.04	1.01 [0.94 - 1.08]
Family size	-0.06	.06	0.94 [0.83 - 1.06]
Block 2			
Child emotional problems (Cycle 1)	1.78***	.15	5.92 [4.42 - 7.92]
Block 3			
Maternal depression (Cycle 1)	0.75***	.18	2.11 [1.47 - 3.02]
Model $R^2 = .08$. $\Delta R^2 = .01$ for Block 1; $\Delta R^2 = .06$ for Block 2; $\Delta R^2 = .01$ for Block 3.			
* $p < .05$. ** $p < .01$. *** $p < .001$.			

cutoff (see Table 4). Results of the regression analyses are shown in Tables 5 and 6. After controlling for family size, income adequacy, and mothers' age and education, and the presence of maternal depression at Cycle 1, the presence of child hyperactivity, aggression and emotional problems at Cycle 1 increased the risk of maternal depression at Cycle 3 by 27% (n.s.), 59% ($p < .001$), and 100% ($p < .001$) respectively. Conversely, after controlling for demographics and the presence of child hyperactivity at Cycle 1, the presence of maternal depression at Cycle 1 increased the risk of child hyperactivity in Cycle 3 by 49% ($p < .05$). In analogous regressions, maternal depression in Cycle 1 increased the risk of child aggression and emotional problems by 28% (n.s.) and 111% ($p < .001$) respectively.

Interpretation

The primary goal of Study 1 was to examine temporal relations between depressive symptoms in mothers and adjustment problems in their children. Maternal and child outcomes shared significant associations within cycles, and as shown in regression analyses, mutual risk was also significant. In fact, the presence of maternal depression more than doubled the rates of each child problem area, which is consistent with previous studies (Beardslee et al., 1998; Weissman et al., 1984). More importantly, antecedent-consequence conditions in maternal and child distress were found to change as a function of whether the child's symptoms were internalising (e.g., low mood, anxiety) or externalising (e.g., aggression, hyperactivity). Maternal and child symptoms

were largely synchronous, but differences between cross-lagged correlations showed that maternal depression tended to *precede* child hyperactivity and aggression and *follow* child emotional problems.

These temporal relations are unlike those reported by Forehand and McCombs (1988), who found that maternal depression preceded both internalising and externalising child behavioural problems. This difference may be attributable to the methods used in the two studies. The present study used maternal reports of child behaviour whereas Forehand and McCombs relied on teacher reports. Cross-informant studies of child behavioural problems have found low to moderate agreement between parent and teacher ratings – due either to differences in data reliability or to the fact that children behave differently at home and at school (Briggs-Gowan, Carter, & Schwab-Stone, 1996). With a nationally representative sample of Canadian families, the present study may have simply yielded more accurate results than were generated from the small sample in Forehand and McCombs's study. Furthermore, replication of differences in cross-lagged correlations across three data cycles indicates that these temporal relations are more reliable than those based on Forehand and McCombs's two data cycles (Rogosa, 1988).

Synchronicity and quasi-stationarity in cross-lagged correlations were taken into account in the present study and were found to affect the interpretation of antecedent-consequence conditions. Violation of the quasi-stationarity assumption is known to bias differences between cross-lagged correlations so that the more reliably measured variable appears to precede the

less reliably measured variable (Cook & Campbell, 1979; Miller, 1997), as was the found in maternal depression to child hyperactivity and child aggression. For this reason, replication of these results is needed using equally reliable measures of maternal and child symptoms.

Regression analyses showed evidence of bidirectional risk at “clinically significant” levels of maternal depression and child adjustment problems. By converting continuous variables to dichotomous variables and controlling for the influence of shared contextual risk, it was found that the presence of child emotional problems increased the risk of maternal depression four years later more than the presence of child hyperactivity or conduct disorder. Also, the presence of maternal depression increased the risk of child emotional problems four years later more than child hyperactivity or conduct disorder. This interpretation assumes equal sensitivity in the modified CES-D and OCHS behaviour scale, but indicates that at high levels of distress there was greater mutual influence between mothers' and children's depressive symptoms than between maternal depression and externalising behaviour in children.

No age or sex differences were found in relations between child behaviour and maternal mood. This was unexpected given the variation normally found between boys and girls and younger and older children in the prevalence and expression of adjustment problems (Offord et al., 1987). Indeed, the influence of maternal depression on child functioning is attributable to multiple factors, not all of which operate equally or simultaneously. Emotional unavailability (Downey & Coyne, 1990), attachment difficulties

(Carlson & Sroufe, 1995), and ineffective discipline (Cunningham et al., 1988) all influence child functioning to varying degrees over time. Conversely, adjustment problems at different stages of child development may evoke different emotional responses in mothers. Although it is perilous to generalise conclusions drawn from children in one developmental stage to other periods of children's lives, these data indicate that in children between the age 4 and 11, the temporal sequencing of maternal depression and child adjustment problems are consistent. Nonetheless, because mutual influences on maternal and child problems may have delayed or additive effects over time (Barkley et al., 1992), further studies are needed that consider their stability and time of onset. Certain risk mechanisms may be most influential during stages in which there is considerable plasticity in child development, but once affected they may continue to impact further development (Goodman & Gotlib, 1999). The absence of sex differences suggests that despite how common externalising or internalising behaviours are in boys and girls, their influence on maternal depression, and vice versa, were similar. It may be that in mothers' appraisals of child behaviour, there are implicit comparisons made with other boys or girls of similar age groups resulting in relative ratings rather than absolute ratings. In any case, more cross-lagged studies are needed using data from older age groups to explore whether the direction of such influences alters in boys and girls during mid- to late-adolescence.

The contribution of Study 1 was a consistent trend shown in temporal relations between maternal depression and child adjustment problems that

differs for internalising and externalising child behaviours. Clearly, the magnitude of some partial cross-lagged correlations was too small to be interpreted. As is often the case in large epidemiological studies, a small but statistically significant correlation does not necessarily reflect a meaningful relation between two variables. However, the pattern of differences between cross-lagged correlations shown in Table 2 does contribute to an understanding of the antecedent-consequence conditions in maternal and child functioning. The sample size and time frame afforded advantages over previous cross-lagged studies on mother-child relations in psychological functioning. The transactional nature of mother and child functioning requires large samples to delineate temporal associations with acceptable statistical power. Still, despite the size and scope of the NLSCY, its biannual assessments did not inform about relations in more frequent, transient changes in maternal and child functioning. For this reason, it remains unclear whether similar temporal relations between depressed mood in mothers and behavioural disturbances in children may emerge had these symptoms been assessed more frequently. To address this issue, a second study was undertaken to study similar antecedent-consequence conditions using daily assessments of maternal mood and child behaviour.

**STUDY 2: POOLED TIME SERIES STUDY OF MATERNAL MOOD
AND CHILD BEHAVIOURAL PROBLEMS**

Objectives

It is possible that some of the mechanisms that transmit risk between maternal and child health outcomes (Figure 1) operate within shorter time frames than can be captured using biannual assessments (e.g., mother-child conflict arising from low mood in the mother and/or oppositional behaviour in the child).

Therefore, using daily reports collected from mother-child dyads over a period of eight consecutive weeks, temporal relations were studied between dimensions of maternal negative mood (e.g, depression, fatigue, tension) and disruptive child behaviour (inattentive/impulsive behaviour and oppositional/defiant behaviour).

The first objective of Study 2 was to determine whether temporal relations in daily changes in maternal and child outcomes are similar to those that emerged in Study 1 with data drawn from biannual assessments. The second objective was to explore which of six discrete dimensions of maternal mood (i.e., tension-anxiety, depression-dejection, anger-hostility, fatigue-inertia, vigour-activity, and confusion-bewilderment) are more strongly related, retrospectively and prospectively, to disruptive child behaviour. The third objective was to study whether the antecedent-consequence conditions of mothers' and children's health outcomes vary as a function of dimensions of

maternal mood and the severity and type of externalising child behaviour (i.e., inattention/impulsiveness and opposition/defiance).

With regard to interpersonal mechanisms of risk involved in mutual influences on maternal mood and child adjustment problems and in light of the results of Study 1 in which maternal mood problems were found to be more of a precursor to, than a consequence of, child externalising behaviour, it was expected that child inattention/impulsiveness and opposition/defiance problems would be greater following low mood in mothers (particularly feelings of fatigue, depression, and tension) than on the day before such child behaviour occurred. It was also hypothesised that maternal mood and child externalising behaviour would share significant synchronous associations and that the magnitude of transactional relations between maternal and child functioning would be greater among children who were diagnosed with disorders of attention (i.e., Attention Deficit Hyperactivity Disorder) and conduct (i.e., Oppositional Defiant Disorder and Conduct Disorder) as compared to a group of healthy children without such disorders.

Methods

Families were recruited from a summer treatment program (STP) for children with Attention Deficit Hyperactivity Disorder and other disruptive behavioural problems (cf. Pelham et al., 2002). The STP was an 8-week day camp and clinical research program that provided cost-free treatment to children. The program enrolled children with and without Attention Deficit Hyperactivity

Disorder or other behavioural disorders. Families participated in the STP and participated in research studies in exchange for free treatment. Children attended the program from 8:00 AM until 5:00 PM, Monday through Friday, and participated in academic, art, and recreational activities led by a staff of teachers and aides. A behavioural point system was in effect throughout the day, in which children earned points for appropriate behaviour and lost points for inappropriate behaviour. Staff members gave continual behavioural feedback and children exchanged points for reinforcers and honours. Parents met in a weekly support group to receive training on the use of daily report cards, time-out procedures, token systems, and effective discipline. A more extensive description of the STP is available elsewhere (Pelham et al., 2002).

Participants

Families volunteered to enrol in the STP through advertisements in local newspapers throughout the community. All 36 families that enrolled in the Summer of 2002 were asked to participate in the present study. Inclusion criteria were that the child was between 6 and 12 years of age and the mother self-reported sufficient fluency in English to provide informed consent and to independently complete daily assessments. The consent form used in the study is provided in Appendix B. The study procedures were approved by the Research Ethics Board of the IWK Health Centre and the Dalhousie University Health Sciences Research Ethics Board.

Thirty of the 36 (83.33%) families enrolled in the STP agreed to

participate. Participating and non-participating families were not significantly different in terms of the age, sex, and number of siblings of the child, and the age, education, income, and marital status of the mother.

Measures

Intake measures. Family intake assessments were comprised of a brief demographic measure that parents completed on the family, the Beck Depression Inventory-II that mothers completed on themselves, the Child Behavior Checklist that mothers completed on the children, and the National Institutes of Mental Health Diagnostic Interview Schedule for Children (DISC) that either mothers or fathers completed on their children. Parents completed other assessments as well but their data were not used in the present study.

The Beck Depression Inventory is a well-established adult self-report assessment of the severity of depressive symptoms (Beck et al., 1961). Its latest revision, the Beck Depression Inventory-II (Beck, Steer, & Brown, 1996), matches diagnostic criteria for major depressive episodes as described in the fourth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM; American Psychiatric Association, 1994). The Inventory includes 21 items, each consisting of four self-evaluative statements, scored 0 to 3, describing a depressive symptom to varying magnitudes. Total scores range from 0 to 63 with higher scores indicating greater depression severity. Using a cutoff score of 14, the Beck Depression Inventory-II has been found to have a sensitivity of 84% and a specificity of 81% in identification of major depressive

disorder in community populations (Beck, Steer & Brown, 1996). The Inventory also has high predictive validity, test-retest reliability and internal consistency, showing a coefficient alpha of .91 in a sample of depressed outpatients (Beck, Steer, & Brown, 1996). Its internal consistency in the present study was also high ($\alpha = .94$).

The Child Behavior Checklist is a 118-item parent-rated measure of child behaviour that assesses, in a standardised format, the social competencies and emotional and behavioural problems of children and adolescents age 6 to 18 years (Achenbach, 1991). The Checklist assesses problems that span eight areas of functioning – four internalising problems (anxious/depressed symptoms, somatic complaints, thought problems, withdrawn/depressive) and four externalising problems (aggressive behaviour, attention problems, rule-breaking behaviour, social problems). Because normative cutoffs for internalising and externalising problems differ for males and females, standardised t-scores are computed using procedures described by Achenbach (1991). Results are stable over time and accurately screen for six DSM-oriented problem areas: affective problems; anxiety problems; somatic problems; attention deficit/hyperactivity problems; oppositional defiant problems; and conduct problems. In the present study, internal consistency of the Checklist was $\alpha_s = .80$ (fathers) and $.85$ (mothers) for internalising problems and $\alpha_s = .76$ (fathers) and $.77$ (mothers) for externalising problems.

The DISC is a structured interview designed to assess over 31 psychiatric disorders in children and adolescents age 6 to 17 years as

described in the DSM and the International Statistical Classification of Diseases and Related Health Problems (ICD-10; World Health Organisation, 1992) (Shaffer, Fisher, Lucas, & the NIMH DISC Editorial Board, 1998). A computerised version allows the parent interview to be conducted in about one hour. DISC items are organised by diagnosis and parents are asked dichotomous questions whether the child had specific symptoms during the past year and the previous four weeks. The criterion validity, acceptability, inter-rater reliability, and test-retest reliability of the DISC for use in children 9 years and older have been well established (Piacentini, Shaffer, Fisher, Schwab-Stone, Davies, & Gioia, 1993; Schwab-Stone, Fisher, Piacentini, Shaffer, Davies, & Briggs, 1993; Shaffer et al., 1993; 1996).

Daily report measures. To minimise burden, missing data and sample attrition, mothers' daily reports were made as brief as possible by formatting two measures into booklets. These measures were the short version of Profile of Mood States (Shacham, 1983) which mothers completed on themselves and the Inattention/Overactivity With Aggression Conners Scale (IOWA; Loney & Milich, 1982) which mothers completed on their children. Assessment order of the Profile of Mood States and IOWA alternated each day throughout the study to control for possible transfer effects between instruments.

The Profile of Mood States is a measure of transient, distinct mood states. Respondents indicate how well a set of mood adjectives (e.g., tense, angry, worn-out) describes their current mood using a 5-point scale ranging

from “not at all” to “extremely.” The Profile includes six factor analytically-derived subscales: tension-anxiety, depression-dejection, anger-hostility, fatigue-inertia, vigour-activity, and confusion-bewilderment. “Total mood disturbance” may be computed by reverse-scoring items on the vigour-activity subscale and summing all the items. Using the original 65-item scale (McNair, Lorr, & Droppleman, 1981), Shacham (1983) used the internal consistency of each subscale and face validity of each item to shorten the instrument from 65 to 37 items. Shacham (1983) found that factor scores of the shortened scale correlated highly with the original ($r_s = .95$ to $.98$) and had similar internal consistency ($\alpha_s = .78$ to $.91$). The internal consistency of the Profile's scales in the present study was $\alpha_s = .84$ to $.87$ (tension-anxiety), $.80$ to $.88$ (depression-dejection), $.85$ to $.90$ (anger-hostility), $.81$ to $.86$ (fatigue-inertia), $.85$ to $.91$ (vigour-activity), and $.78$ to $.83$ (confusion-bewilderment). Although the Profile is not a clinical assessment tool, in a sample of 438 cancer patients, Baker and colleagues found that the depression-dejection subscale correlated significantly with the CES-D ($r = .65$), indicating convergent validity with an established clinical measure of depressive symptoms (Baker, Denniston, Zabora, Pollard, & Dudley, 2002).

The IOWA is a 10-item measure designed for parents or teachers that assesses disruptive behavioural problems in children age 6 to 17 years (Loney & Milich, 1982). Using core DSM symptoms to screen or monitor symptoms of Attention Deficit/Hyperactivity Disorder and Oppositional Defiant Disorder, items include a behaviour description and a 4-point scale ranging from “not at all” to

“very much.” The IOWA has two symptom clusters, the inattention/impulsiveness factor and the opposition/defiance factor. The five items comprising the inattention/impulsiveness factor are (1) fidgeting, (2) hums and makes other odd noises, (3) excitable/impulsive, (4) inattentive/easily distracted, and (5) fails to finish things he/she starts. Five items of the opposition/defiance factor are (1) quarrelsome, (2) acts "smart", (3) temper outburst (explosive, unpredictable behaviour), (4) defiant, and (5) uncooperative. The IOWA measure takes minimal time to complete and has been found to have good reliability, predictive validity, and sensitivity to behaviour change (Pelham, Milich, Murphy, & Murphy, 1989). The internal consistency of the IOWA in the present study was α s = .72 to .82 (inattention/impulsiveness) and .64 to .72 (opposition/defiance).

Procedure

At intake, 2 to 3 months prior to the STP, trained staff interviewed parents and carried out DISC assessments of Attention Deficit Disorder, Conduct Disorder, Oppositional Defiant Disorder, and other psychiatric disorders. Mothers' and fathers' ratings of child symptoms were combined on a symptom-by-symptom basis and symptoms were scored if either the mother or father reported it. Cross-informant DISC assessments have been found to yield more accurate results than using single informants (Piacentini, Cohen, & Cohen, 1992) and was deemed the most reliable method of identifying non-clinical and clinical groups of children for comparison purposes. Additional data

were collected at intake using the Beck Depression Inventory-II to measure maternal depressive symptoms and Child Behavior Checklist to measure mother-rated child behavioural problems.

Mothers were supplied with booklets for each week of the STP that contained the daily assessments (i.e., Profile of Mood States and IOWA). Because the STP operated from Mondays to Fridays, there were five assessments per week, except for four assessments on the first and last weeks, yielding a total of 38 data cycles. Mothers completed assessments at home in the evenings and, at the end of each week, submitted completed assessments to a drop-off location. STP staff was in regular contact with mothers to encourage compliance. Participants were compensated \$40 at the end of data collection.

Results

Diagnoses based on cross-informant DISC assessments with mothers and fathers were assigned to 20 of the 30 children in the sample. These diagnoses included Attention Deficit Hyperactivity Disorder (Predominantly Inattentive Type or Combined Type) ($N = 19$), Oppositional Defiant Disorder ($N = 8$), and Conduct Disorder ($N = 8$). The sample was first grouped into non-clinical and clinical groups on the basis of whether the child was assigned a DISC diagnosis of any kind. A profile of the demographic characteristics and baseline functioning of non-clinical and clinical groups is shown in Table 7. Children in the clinical group, compared to the non-clinical group, were more likely to be

male than female and showed significantly more internalising, externalising, and total problems as measured on the Child Behavior Checklist. There were trends suggesting that mothers in the clinical group were older and reported more depressive symptoms than mothers of the non-clinical group, but a small sample size and large error variance appeared to preclude statistically significant differences in these areas. A cross-tabulation showed that of the ten (33.33%) mothers who scored above the cutoff of 14 on the Beck Depression Inventory-II, their children were no more likely to have received a DISC diagnosis compared to children of mothers who scored below the cutoff. However, with regard to scores on the Child Behavior Checklist, children of mothers who scored above the depression cutoff, compared to children of mothers who scored below the depression cutoff, scored marginally more internalising problems ($M = 63.50$ vs. $M = 51.80$, $F(1, 28) = 4.07$, $p = .05$) and significantly more externalising problems ($M = 62.00$ vs. $M = 52.95$, $F(1,28) = 5.53$, $p < .05$) but not more total problems ($M = 60.33$ vs. $M = 50.00$, $F(1,28) = 2.05$, $p = .16$).

Of a possible 1170 recordings across 38 data cycles, mothers provided a total of 623 recordings on the daily assessments, indicating 54.56% compliance ($M = 21.07$, $SD = 10.63$, Minimum = 1.00, Maximum = 35.00). Four hundred and twelve recordings were from mothers of children who were assigned a clinical diagnosis – 378 from mothers of children diagnosed with Attention Deficit Hyperactivity Disorder, 323 from mothers of children diagnosed with either Oppositional Defiant Disorder or Conduct Disorder (70.14% overlap).

Table 7

Descriptive Statistics on Maternal and Child Functioning in Non-Clinical and Clinical Samples.[†]

Variable	Non-Clinical (N = 10)	Clinical (N = 20)	Comparison
<i>Child:</i>			
Male (%)	5 (50.00)	17 (85.00)	$\chi^2 (df=1) = 4.18, p=.04$
Female (%)	5 (50.00)	3 (15.00)	
Age (SD)	9.40 (1.36)	9.40 (1.88)	$F(1,28) = 0.01, p=.99$
No. siblings (SD)	2.00 (.94)	1.45 (1.28)	$F(1,28) = 1.45, p=.24$
2+ siblings (%)	4 (40.00)	4 (20.00)	$\chi^2 (df=1) = 1.36, p=.24$
Lives with father (%)	8 (100.00)	14 (87.50)	$\chi^2 (df=1) = 1.09, p=.30$
Internalising Behaviour (SD)	41.30 (7.21)	59.47 (11.62)	$F(1,27) = 20.17, p<.001$
Externalising Behaviour (SD)	40.30 (8.43)	63.40 (8.39)	$F(1,27) = 50.39, p<.001$
Total Problems (SD)	37.70 (7.96)	65.26 (9.57)	$F(1,27) = 60.54, p<.001$
<i>Mother:</i>			
Age (SD)	38.20 (4.10)	41.00 (3.61)	$F(1,25) = 3.43, p=.08$
Education (SD) [‡]	1.00 (1.12)	1.12 (1.05)	$F(1,25) = .07, p=.79$
Family income (SD) [§]	4.70 (2.67)	4.45 (2.98)	$F(1,28) = .05, p=.83$
Marital status			
Single/separated (%)	2 (20.00)	8 (40.00)	$\chi^2 (df=2) = 1.20, p=.27$
Married/common-law (%)	8 (80.00)	12 (60.00)	
Depressive symptoms (SD)	7.00 (8.34)	12.45 (14.43)	$F(1,28) = 1.21, p=.28.$
Depressed (%) [¶]	3 (30.00)	7 (35.00)	$\chi^2 (df=1) = .75, p=.78.$

[†] "Clinical" refers to DISC diagnosis of any disruptive behaviour disorder.

[‡] Scale: 1=no high school, 2=high school, 3=technical school, 4=graduated from college or university (bachelor's degree), 5=postgraduate degree.

[§] Scale: 1= < \$10,000, 2=\$10,000-19,999, 3=\$20,000-29,999, 4=\$30,000-39,999, 5=\$40,000-49,999, 6=\$50,000-59,999, 7=\$60,000-69,999, 8=\$70,000-79,999, 9=\$80,000-89,999, 10=\$90,000-99,999, 11=\$100,000-125,000, 12= >\$125,000.

[¶] Beck Depression Inventory-II scores above the cutoff of 14.

Two hundred and twelve recordings were provided by mothers whose children did not meet diagnostic criteria for a behavioural disorder.

Visual inspection of the data provided some indication that child inattention/impulsivity and opposition/defiance may have been likely to occur on days when there was also disturbance in mothers' mood. Sample data from two participants are shown in Figures 6 and 7. In Figure 6, total mood disturbance in Participant A can be seen to vary significantly from day to day and to modestly decrease over the eight-week duration of the study. Figure 6 also suggests that this mother's mood may have changed, in part, as a function of her child's behavioural disturbance. Similarly, in Figure 7, maternal mood disturbance in Participant B appears to have coincided with child behavioural problems. While these data suggest some support for the research hypotheses, they more clearly demonstrate the untenability of exploring temporal relations by visual analysis.

Preliminary examination of the pooled data lent some support for the hypothesis that low mood in mothers coincided with disruptive child behaviour. Mood disturbance correlated positively with child inattention/impulsiveness, $r_s = .22$ (depression-dejection), $-.22$ (vigour-activity), $.28$ (anger-hostility), $.40$, (tension-anxiety), $.24$ (confusion-bewilderment), and $.22$ (fatigue-inertia), all $ps < .001$, and all but vigour-activity correlated with child opposition/defiance, $r_s = .32$ (depression-dejection), $.36$ (anger-hostility), $.42$ (tension-anxiety), $.32$ (confusion-bewilderment), and $.34$ (fatigue-inertia), all $ps < .001$.

While encouraging, these results were not entirely justified because the

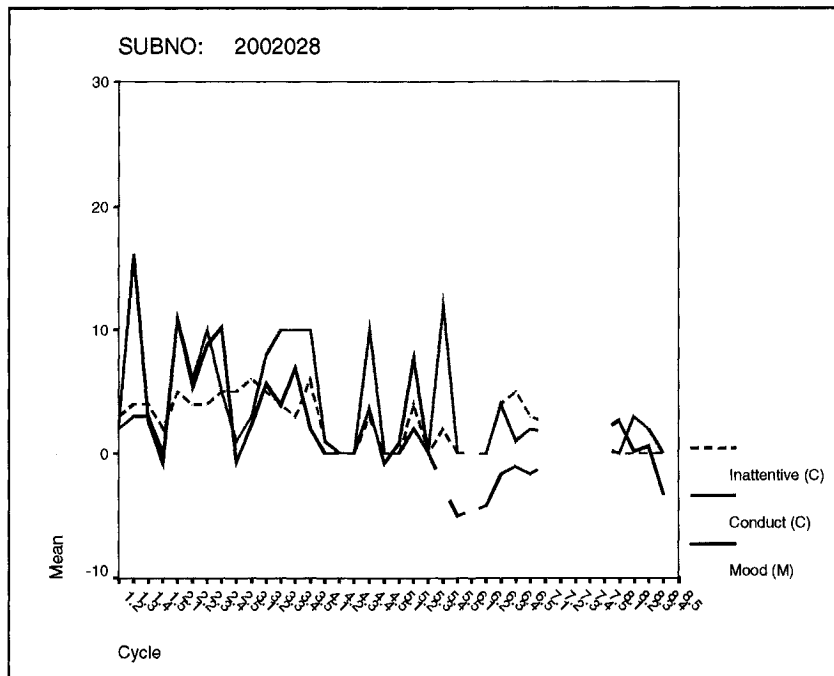


Figure 6. Maternal mood and child behaviour in Participant A.

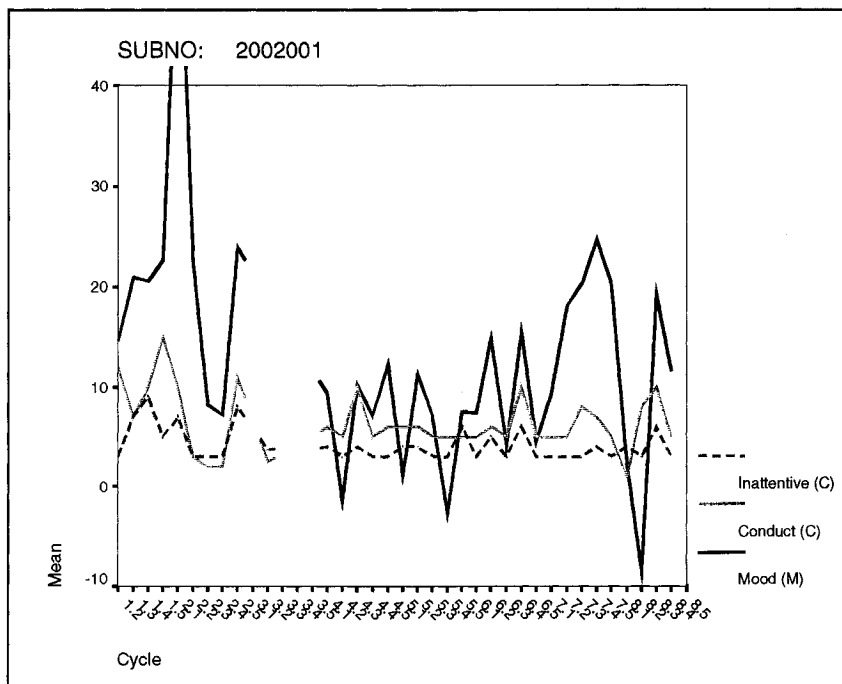


Figure 7. Maternal mood and child behaviour in Participant B.

data did not meet the statistical assumption of independent observations. Thus, pooled time-series analysis was used to correct for the lack of independent observations, individual differences, and ongoing time trends (e.g., decline in child symptoms resulting from treatment). As described above, pooled time-series analysis has the form of a linear model that treats each observation for each subject as a separate case. Its power advantage over more common analyses is that it was used to test relations between two constructs that were measured on a relatively small sample over large number of observations.

Between-subject variance

Dichotomous dummy variables representing each of the 30 participants were used to control for individual differences in the data. This statistical control is a basic technique of a fixed effects pooled time-series analysis (Dielman, 1989). Each dummy variable was coded as 1 on all the observations for one subject and 0 on all other observations. The number of dummy variables required to identify all participants was one less than the sample size.

The degree of between-subject variance absorbed by these dummy variables was high, accounting for 78% of the variance in child inattention/impulsiveness and 36% of the variance in child opposition/defiance. Between-subject variance in maternal mood accounted for 79% of the variance in depression-dejection, 74% in confusion-bewilderment, 66% in tension-anxiety, 64% in vigour-activity, 62% in anger-hostility, 47% in fatigue-inertia, and 70% in

total mood disturbance. The fixed effects model was deemed more reliable for handling such large individual differences than an error components or “random effects” model (according to Dielman [1989], the error components model in pooled time-series provides less control for individual differences because it reduces between-subject variance to a level consistent with within-subject variance rather than eliminate it altogether).

Serial dependence

Estimates of serial dependence (autocorrelation) were performed using multiple regressions on maternal and child outcomes with the following predictors: maternal age, maternal education, family income, and number of children (Block 1), dummy variables to control for individual differences (Block 2), and pooled observations of either maternal mood or a child behavioural outcome (Block 3). Block 1 controlled for sociodemographic factors that may have constituted shared environmental risk for mothers and children. Block 1 predicted 3% of the variance in child inattention/impulsiveness, 2% of the variance in child opposition/defiance, and 2 to 12% of the variance in maternal mood (2% [vigour-activity], 5% [fatigue-inertia], 10% [tension-anxiety], 11% [confusion-bewilderment], 11% [anger-hostility], 12% [depression-dejection], and 12% [total mood disturbance]).

The Durbin-Watson statistic was derived to estimate autocorrelation and transform the data using formulas described by Soliday et al. (2002) (see pp. 49-50). After transforming the variables (and y-intercept), one iteration of the

procedure successfully reduced autocorrelation estimates to under .15, reflecting the high within-subject variability in maternal mood and child behaviour illustrated in Figures 7 and 8. Autocorrelation statistics for original and transformed data in maternal mood (predicted by child behaviour) and child behaviour (predicted by maternal mood) are shown in Table 8. Transformed data were used in all further analyses.

Cross-sectional relations

Pooled time-series models included dummy variables to control for between-subject variance, transformed data to control for serial dependence, and hierarchical regression models, analogous to those described above, to control for shared sociodemographic influences on maternal and child symptoms. Using this method, cross-sectional relations were analysed between maternal mood and child inattention/impulsiveness (Table 9) and between maternal mood and child opposition/defiance (Table 10). In these and all other analyses, child symptoms were positively related to five dimensions of emotional disturbance (depression-dejection, anger-hostility, tension-anxiety, confusion-bewilderment, and fatigue-inertia) and negatively related to vigour-activity.

In children with and without diagnoses of Attention Deficit Hyperactivity Disorder (using cross-informant DISC assessments), significant cross-sectional relations were found between child inattention/impulsivity and maternal anger-hostility and significant inverse relations were found between child

Table 8

Durbin-Watson Statistics (dw) and Serial Dependence (r) in Original and Transformed Data.

<i>Dependent Variable</i>	<i>Original</i>		<i>Transformed</i>	
	<i>dw</i>	<i>r</i>	<i>dw</i>	<i>r</i>
<i>Maternal:</i>				
depression-dejection	1.695	.153	2.040	-.020
vigour-activity	1.764	.118	1.948	.026
anger-hostility	1.702	.149	1.964	.018
tension-anxiety	1.602	.199	1.973	.135
confusion-bewilderment	1.636	.182	1.762	.119
fatigue-inertia	1.419	.291	2.113	-.057
total mood disturbance	1.601	.198	2.048	-.024
<i>Child:</i>				
inattention/impulsiveness	1.752	.124	2.035	-.018
opposition/defiance	1.858	.071	2.028	.014

inattention/impulsivity and maternal vigour-activity (Table 9, centre column). Also, a significant cross-sectional relation emerged between child inattention/impulsivity and maternal depression-dejection, but only in the non-clinical group of children without diagnoses of Attention Deficit Hyperactivity Disorder.

Quite different results emerged in cross-sectional relations between child opposition/defiance and maternal mood (Table 10, centre column). Interestingly, child opposition/defiance related to maternal tension-anxiety, but only in children whose behaviour met diagnostic criteria of either Oppositional Defiant Disorder or Conduct Disorder. No other cross-sectional relations were found between child opposition/defiance and maternal mood.

Maternal mood following child behaviour

Tables 9 and 10 also show the results of time-lagged pooled time-series analyses in which the criterion (child behaviour) alternated from one day prior to when maternal mood was measured to one day subsequent to maternal mood. Some data were censored in these analyses to prevent time-lags from extending over a weekend.

Child Inattention/impulsiveness was significantly related to maternal fatigue-inertia on the following day – irrespective of whether the child's behaviour met diagnostic criteria for Attention Deficit Hyperactivity Disorder (Table 9, left column). Child inattention/impulsiveness was also significantly

Table 9

Pooled Time Series Analysis of Child Inattention/Impulsiveness on the Day Previous, Same Day as, and Day After Maternal Mood.

<i>Predictor</i>	<i>Previous Day</i>		<i>Same Day</i>		<i>Next Day</i>	
	<i>B (SE)</i>	β	<i>B (SE)</i>	β	<i>B (SE)</i>	β
Non-clinical ($N = 11$; 244 pooled observations). $R^2 = .065$ (previous day), .112 (same day), .105 (next day)						
<i>Maternal:</i>						
depression-dejection	.04 (.09)	.09	.16 (.06)	.35**	.17 (.06)	.34**
vigour-activity	-.08 (.02)	-.06	-.03 (.01)	-.21*	-.03 (.02)	-.22*
anger-hostility	.19 (.07)	.39**	.22 (.04)	.44**	.09 (.04)	.20*
tension-anxiety	.09 (.07)	.23	.02 (.04)	.03	.16 (.04)	.47**
confusion-bewilderment	.09 (.06)	.18	.05 (.04)	.10	.03 (.04)	.06
fatigue-inertia	.03 (.02)	.24*	.02 (.01)	.09	.01 (.02)	.00
Clinical [†] ($N = 19$; 378 pooled observations). $R^2 = .023$ (previous day), .079 (same day) .034 (next day)						
<i>Maternal:</i>						
depression-dejection	.11 (.13)	.08	.09 (.10)	.06	.12 (.05)	.21*
vigour-activity	-.05 (.06)	.09	-.11 (.05)	-.18*	-.07 (.12)	.05
anger-hostility	.05 (.11)	.04	.32 (.08)	.26**	.20 (.09)	.15*
tension-anxiety	.19 (.11)	.15	.14 (.09)	.10	.20 (.08)	.17*
confusion-bewilderment	.08 (.11)	.01	.05 (.08)	.04	.08 (.09)	.06
fatigue-inertia	.13 (.05)	.19**	.04 (.04)	.05	.02 (.04)	.01

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

[†] "Clinical" refers to diagnosis of Attention Deficit/Hyperactivity Disorder (either Predominantly Inattentive or Combined Type).

Note. Controlled for between-subject variance, maternal age, education, marital status, and household income. Time-lagged analyses were based on four recordings per week per subject.

Table 10

Pooled Time Series Analysis of Child Opposition/Defiance on the Day Previous, Same Day as, and Day After Maternal Mood.

Predictor	Previous Day		Same Day		Next Day	
	B (SE)	β	B (SE)	β	B (SE)	β
Non-clinical (N = 14; 250 pooled observations). $R^2 = .018$ (previous day), .026 (same day), .035 (next day)						
<i>Maternal:</i>						
depression-dejection	.32 (.31)	.11	.06 (.23)	.02	.03 (.23)	.01
vigour-activity	.05 (.05)	.06	-.06 (.05)	.06	.08 (.05)	.10
anger-hostility	.18 (.19)	.07	.30 (.16)	.11	.27 (.15)	.11
tension-anxiety	.08 (.19)	.08	.24 (.13)	.13	.24 (.11)	.15*
confusion-bewilderment	.48 (.19)	.18*	.25 (.14)	.10	.22 (.15)	.10
fatigue-inertia	.01 (.05)	.00	.07 (.05)	.08	.08 (.06)	.08
Clinical [†] (N = 16; 373 pooled observations). $R^2 = .058$ (previous day), .144 (same day), .067 (next day)						
<i>Maternal:</i>						
depression-dejection	.18 (.20)	.11	.11 (.15)	.07	.30 (.19)	.19
vigour-activity	-.10 (.10)	.15	-.10 (.08)	-.14	-.01 (.09)	.02
anger-hostility	.17 (.18)	.11	.28 (.13)	.18	.02 (.14)	.01
tension-anxiety	.05 (.18)	.04	.45 (.13)	.32**	.43 (.14)	.30**
confusion-bewilderment	.49 (.17)	.31**	.10 (.13)	.06	.29 (.14)	.19*
fatigue-inertia	.01 (.08)	-.01	.03 (.06)	.03	.02 (.06)	.02

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

[†] "Clinical" refers to diagnoses of either Oppositional Defiant Disorder or Conduct Disorder.

Note. Controlled for between-subject variance, maternal age, education, marital status, and household income. Time-lagged analyses were based on four recordings per week.

related to subsequent maternal anger-hostility, but only in the non-clinical group and not the clinical group.

As shown in Table 10 (left column), child opposition/defiance was related to maternal confusion-bewilderment on the following day – irrespective of whether the child's behaviour met diagnostic criteria for Oppositional Defiant Disorder or Conduct Disorder. However, child opposition/defiance was not significantly related to any other subsequent maternal mood.

Maternal mood preceding child behaviour

Tables 9 and 10 also show relations between maternal mood and subsequent child behaviour on the following day. Child inattention/impulsivity was related to maternal depression-dejection, tension-anxiety, and anger-hostility on the previous day (Table 9, right column). These relations were found consistently in both non-clinical and clinical samples (children with and without diagnoses of Attention Deficit Hyperactivity Disorder). Additionally, child inattention/impulsivity was negatively related to maternal vigour-activity on the previous day, but only in the non-clinical sample and not in the clinical sample.

Finally, child opposition/defiance was found to be related to maternal tension-anxiety on the previous day – in children with and without diagnoses of either Oppositional Defiant Disorder or Conduct Disorder. Children in the clinical group also showed opposition/defiance as a consequence of maternal confusion-bewilderment.

Antecedent-consequence conditions

To summarise, pooled time-series analysis revealed significant relations, concurrently and over time, between maternal mood and child behaviour. The significance of these relations varied as a function of mood dimensions in the mother, the type of externalising behaviour in the child, and whether the child was diagnosed with a behavioural disorder. Therefore, antecedent-consequence conditions in maternal mood and child behaviour changed according to different moods and behaviours.

In both non-clinical and clinical samples, maternal fatigue-inertia was related to prior child inattention/impulsiveness but not subsequent inattention/impulsiveness, indicating that this mood in mothers changed as a consequence of child inattention/impulsiveness. However, changes to maternal depression-dejection, tension-anxiety, and, in the non-clinical group, vigour-activity appeared to be more of an antecedent than a consequence of child inattention/impulsiveness. Maternal anger-hostility was independently related to previous, concurrent, and subsequent child inattention/impulsiveness, possibly reflecting a transactional relation between maternal and child symptoms.

Only maternal confusion-bewilderment emerged as a consequence to child opposition/defiance. In the clinical group, maternal confusion-bewilderment also emerged as a precursor to future child opposition/defiance, again indicative of a transactional relation. Maternal tension-anxiety emerged as another significant precursor to future child opposition/defiance.

Interpretation

The main objective of Study 2 was to examine cross-sectional and longitudinal relations between daily fluctuations in maternal mood and disruptive child behaviour. Temporal relations did emerge, with child behaviour influencing maternal mood and maternal mood influencing child behaviour over one-day intervals after cross-sectional associations were statistically controlled. These relations accounted for 2.3 to 14.4 percent of the variance observed in child behaviour. However, the direction of these relations changed as a function of dimensions of maternal mood, whether the child exhibited problems of inattention and hyperactivity or oppositional tendencies, and whether the child's behaviour met diagnostic criteria for disruptive behaviour disorder.

In terms of how maternal mood was influenced by child behaviour, the data indicate that mothers experienced more anger and fatigue following child problems of inattention and hyperactivity than before such child problems occurred. This finding may be indicative of the strain associated with child hyperactivity, regardless of whether the child is diagnosed with a disorder. The data also show that maternal functioning was subsequently affected by child oppositional behaviour. However, mothers' feelings of confusion were both antecedents and consequences to child oppositional behaviour. Such transactional influences may be a reflection of mothers' lack of confidence in their use of discipline strategies resulting, ultimately, in further reinforcement of their children's difficult behaviour.

Such reactions to disruptive child behaviour may reflect the initial

emotional consequences to maternal functioning, but child functioning was found to be influenced by maternal mood as well. Though it has been suggested that maternal depression plays an influential role in only serious forms of child behavioural problems – an association that may be obscured in community populations (e.g., Downey & Coyne, 1990) – the present study suggests otherwise. That is, in both clinical and non-clinical groups, child problems of inattention, hyperactivity and conduct were significantly influenced by mothers' feelings of depression, anxiety, and anger. The data indicated that children were differentially sensitive to certain mood states in mothers. For instance, child inattention and hyperactivity were more likely occur after the mothers reported feelings of depression, anxiety, tension, and low energy than before such feelings. Children also exhibited more oppositional behaviour on the day following mothers' reported feelings of anxiety than the day before such feelings. Such temporal relations suggest that compared to mothers who feel contented with, and responsive in, their interactions with children, mothers who feel sad, anxious, hostile or lethargic may have greater difficulty communicating with children and utilising effective discipline strategies that may curb disruptive behaviours in their children (Cunningham et al., 1988). These data are also consistent with Patterson's (1982) coercive family process model that suggests that one of the motivations in disruptive child behaviour is to elicit attention from a mother who is withdrawn and unresponsive to the child's needs.

The mutual influences found in the present study are consistent with the theoretical model of transactional influences between maternal mood and child

behaviour shown Figure 1, and with previous studies that found child adjustment problems to have a negative influence on maternal emotional functioning (e.g., Pelham et al., 1997; Study 1) which, in turn, influences further adjustment problems in the child (Goodman & Gotlib, 1999). These results are unique in that they are clearly inconsistent with an assumption of equivalent temporal relations across dimensions of maternal mood and externalising child behaviour. By delineating distinct mood states in mothers, these findings illuminate specific areas of intervention that may facilitate treatments for children. For example, helping mothers feel more energetic and less sad, worried, and angry, could potentially result in a 3.4 to 10.5 percent reduction in child hyperactivity. Such results could not have been found in studies that utilised generalised (and therefore confounded) screening instruments of maternal depression such as the CES-D (e.g., McCombs & Forehand, 1988).

The important advantage of the pooled time-series design in this study was its ability to test the significance of bidirectional influences while controlling for the autocorrelation in daily report data that would plague any other statistical analysis. Another advantage of the design was that the possibility of spurious time-lagged relations was diminished by exploiting hundreds of observations. The pooled time-series design offered excellent control for historical effects and yielded high statistical power from a relatively small sample. However, while the internal validity of the study was strong, its external validity was still limited by the small size and unique features of the sample. Participants were self-selected in that they responded to advertisements by volunteering to enroll in a

treatment program rather than consent to participate in a study after being randomly selected from the population. Families who seek help are likely to be importantly different from families with similar problems who do not seek help. Therefore, some caution is warranted in generalising the conclusions of the study to the general population. Still, the results are unique to the research literature and may stimulate further research on the temporal relations in daily-reported maternal mood and child adjustment problems.

GENERAL DISCUSSION

The main objective of Studies 1 and 2 was to attempt to unravel antecedent-consequence conditions in maternal mood disturbances and emotional and behavioural problems in children, thereby elucidating mechanisms of risk and resiliency involved in the intergenerational transfer of psychological problems. In Study 1, it was found that over extended periods of time, maternal depressive symptoms tended to emerge more as a precursor to, than a consequence of, externalising behaviours in children, and that the inverse temporal relation existed between maternal mood and internalising behaviours in children. Study 2 provided convergent findings and more detailed account of mutual influences on maternal mood and child externalising behaviour on a daily basis and revealed properties of their temporal relations that depended on dimensions of maternal mood and on the type and severity of child behaviour. These studies provide new information about how maternal and child behaviour influence one another with different methodological approaches and levels of analysis.

This general discussion expands upon the general implications of both studies in terms of what they revealed about the mechanisms of risk and resilience through which mutual influences operate on maternal and child functioning. Also discussed are methodological issues that influenced the interpretation of the research, such as the timing of assessments and the exclusive reliance on mother reports, and a research agenda is presented with

directions for further studies. Finally, the practical implications of the results are explored for clinical assessment and treatment as well as public health policy that affects the health and well-being of children and families.

Mechanisms of risk and resilience

One conclusion that was borne out in these studies is that mothers' emotional functioning is significantly compromised by disruptive child behaviour. Whether the outcome manifested in mothers' symptoms of depression, anxiety, fatigue, or confusion, the consequences of child hyperactivity and aggression in terms of maternal mental health were significant and consistently negative. In Study 1, child aggression was found to increase the maternal depression after four years by 46 percent. In Study 2, time-lagged pooled time-series models showed that several mood states in mothers (particularly fatigue, anger, and confusion) changed for the worse as a consequence of child hyperactivity and aggression. These influences are indicative of the stress, frustration, and hopelessness elicited by problems of child inattention, hyperactivity, defiance, or aggression. Mediating mechanisms may include interpersonal stress (Pelham et al., 1997), low self-esteem and low parental efficacy (Teti & Gelfand, 1991), passive coping (Wells-Parker et al., 1990), and the mother's relationships with her partner and other family members (Kelly, 2000). It could be inferred, therefore, that mothers who are able to deal with disruptive child behaviour, with active coping, high self-esteem, high parental efficacy, and positive family and marital relationships, may show better functioning than mothers without these characteristics.

A second conclusion is that emotional distress in mothers has negative consequences for child adjustment. In Study 1, cross-lagged panel correlations revealed that child hyperactivity and aggression were more likely to follow, than precede, depressive symptoms in their mothers. Regression analyses showed that over a four-year period, maternal depression increased the risk of child hyperactivity by 40 percent and increased the risk of child emotional disorder by 75 percent. In Study 2, it was found that children reacted to maternal mood – particularly anxiety, depression, and anger – in terms of their disruptive behaviours one day later. The influence of maternal depression on child adjustment is well explained by the many mechanisms that mediate risk from the depressed mother to the child, including emotional dysregulation stemming from either prenatal influences on neuroendocrine functioning (Field, 2002) or attachment insecurity (Carlson & Sroufe, 1995), parent-child conflict (Elgar & Arlett, 2002), marital problems (Kelly, 2000), or family dysfunction (Downey & Coyne, 1990). The influence is also consistent with either a genetically-mediated vulnerability to emotional problems (Cadoret, 1978), a delayed “switching on” of a genetic predisposition to emotional disorder in mid-adolescence (Goodman & Godlib, 1999), a constitutional diathesis that interacts with environmental factors to increase the risk of emotional problems (Silberg & Rutter, 2002), or a social learning mechanism whereby the child emulates the mother's depressive symptoms and inadequate coping behaviour (Bandura, 1973). Unfortunately, without measures of any of these potential mediating mechanisms, it remains unclear which most actively transmit risk for child

psychopathology. However, because child behaviour also influenced future maternal emotional functioning, as determined in logistic regression results in Study 1, it is clear that transactional interpersonal and contextual mechanisms play an active role in addition to genetic and neuroendocrine mechanisms of risk and resilience, as portrayed in Figure 1.

A third and perhaps the most practical conclusion to draw from this research is that symptoms of psychological distress shown by mothers and children are dynamically interrelated, exhibiting mutual influences over time after prior levels of distress are statistically controlled. As many frustrated mothers well know, disruptive child behaviour elicits stress (Barkley et al., 1992; Pelham et al., 1997; Shelton et al., 1998) and can compromise mothers' satisfaction with parenting and perceived competencies in being good parents (Mash & Johnston, 1990; Teti & Gelfand, 1991). Consequently, parenting behaviour is disrupted, including discipline practices that, in turn, exacerbates children's disruptive behaviour and further perpetuates the cycle. This pattern of mutual influence was evident in both Studies 1 and 2 and is consistent with Patterson's "coercive family process model" (Patterson et al., 1989). As the child's disruptive behaviour becomes more frequent and intense, mothers become increasingly frustrated, fatigued, and anxious while their attempts to manage the child's behaviour turn less effective (Cunningham et al., 1988). As Patterson described in the context of child aggression, inconsistent, ineffective discipline by parents may permit family interactions that positively reinforce coercive child behaviour and negatively reinforce parents' poor discipline (Patterson et al.,

1989). The implication is that mothers who experience emotional distress (at clinical or subclinical levels) but also effectively self-monitor and regulate their responses to coercive child behaviour may be better able to utilise effective behavioural management techniques, thereby interrupting the coercive cycle and helping the child and the family to function normally.

Dimensions of mood and behaviour

A secondary objective of Studies 1 and 2 was to explore change in temporal relations between maternal mood and child adjustment problems as a function of whether the child's behaviour is internalising or externalising and as a function of which mood state is most salient in the mother. In Study 1, antecedent-consequence conditions in maternal mood and child adjustment shifted depending on the type of child behaviour exhibited, with child hyperactivity and aggression emerging in response to maternal depressed mood and maternal depressed mood emerging in response to child emotional problems. This dichotomy in temporal relations revealed an important characteristic of family influences of externalising problems in children: that maternal depressed mood, left undetected, untreated, or poorly managed, poses a long-term health risk to children. Minor adjustment problems may, without intervention, evolve to more serious problems as the parent becomes increasingly withdrawn and inattentive to the child's needs, ineffective in discipline practices, and detached from the child's social and academic challenges (Patterson et al., 1989). The influence of child emotional problems

on maternal emotional problems is a scarcely reported finding in a research literature dominated by studies on effects of maternal depression on child outcomes (e.g., Cummings & Davies, 1994; Goodman & Gotlib, 1999). Yet, because the direction of such an influence is counter to genetic and biological processes, it further emphasises the salience of interpersonal influences on the mental health of mothers and children.

Another contribution of Study 2 was a demonstration that mutual influences on maternal and child outcomes existed not only in stable symptomatology, but also in day-to-day fluctuations in functioning. It was shown that several domains of maternal mood, some extending beyond symptoms of depression (e.g., tension-anxiety), share significant, negative influences on child adjustment. Dimensions of mood expressed by mothers and the types of emotional and behavioural problems exhibited by children are relevant to their temporal relations. Thus, conclusions made about their temporal sequence should avoid broad generalisations about antecedent-consequence conditions in maternal mood problems and child adjustment problems. In essence, mothers and children are differentially sensitive to different types of behaviour and mood.

Methodological Issues

While the Studies 1 and 2 helped to illuminate family influences in behavioural health conditions in women and children, at least four methodological issues affect their interpretation and, ultimately, their implications for clinical contexts

and public health policy. These caveats include the conceptualisation of “risk,” the potential significance of unmeasured variables, the frequency of assessments, and the exclusive reliance on mother reports of child behaviour. Careful consideration of each of these issues is warranted in designing follow-up studies.

Risk factors and temporal effects. Whereas temporal relations suggest the possibility of risk, it is acknowledged the type historical data needed to properly quantify this risk was lacking in this thesis. Kraemer, Kazdin, Offord, Kessler, Jensen, and Kupfer (1997) described basic criteria of risk estimation studies, emphasising that the prediction of dimensional outcomes using correlations or linear regressions is distinct from a valid, quantifiable estimation of risk. They define a risk factor as a specific, dichotomous characterisation (e.g., child with a disruptive behaviour disorder) that is clearly shown to *precede* the outcome and that can be used to divide the population into groups high-risk and low-risk groups – “a special type of correlate that requires documentation of precedence” (Kraemer et al., 1997, p. 340). In Studies 1 and 2, continuous variables were required by the analyses that were used to examine temporal relations. As a result, maternal and child outcomes showed constant fluctuation and high degrees of comorbidity. Without diagnostic histories, the actual *precedence* of antecedent conditions could not be demonstrated. If it were shown that a clinically significant symptoms were present in either mother or child before such symptoms were shown in the other, then it may be possible to

identify a "variable risk factor" (Kraemer et al., 1997). Therefore, antecedent-consequence conditions shown here should be interpreted in light of the fact that maternal depression and child adjustment problems tend to co-occur, and that symptoms that tend to precede, more than they follow, other symptoms do not sufficiently indicate risk of a disorder.

Unmeasured Variables. While it was possible to utilise repeated measures of psychological functioning, it was not possible to investigate linkages with factors that previous research has shown moderate and mediate relations between maternal and child outcomes. Unravelling their temporal relations contributes to understanding these mechanisms, but they remain somewhat of a "black box" in that only input and output were observed. Both Studies 1 and 2 may have been enriched by the inclusion of measures of mediating factors (e.g., child discipline, parental efficacy), moderating factors (e.g., father involvement, child intelligence), and common coexisting problems (e.g., maternal anxiety, child academic problems). The potential pitfall is that by not acknowledging the role of unmeasured "third" variables, one may erroneously conclude that direct, unbuffered, and unmediated relations exist between maternal mood and child adjustment. Were there an attempt to identify characteristics of mothers and children who are most at risk to each another, as well as variables that moderate (or "buffer") the risk to children and to mothers, it is possible that a more robust exploration of their mutual influences and temporal relations would have been achieved.

Assessment Intervals. The panel data used in Studies 1 and 2 were collected using biannual and daily assessment intervals, respectively, thus enabling the investigation of a research question across quite different time parameters. A potential shortcoming of this approach is that such a large difference in assessment intervals may have precluded an accurate account of changes in maternal and child functioning occurring over weeks and months – more common time frames discussed in the clinical literature. This issue speaks to a general methodological challenge in developmental psychopathology research regarding the optimal number and timing of occasions of measurement in prospective studies. If measurement occasions occur too close in time, there may be insufficient time for any substantively significant change in behaviour to have occurred. If measurement occasions occur too far apart, important processes underlying key mechanisms of change may be inadvertently excluded, thus yielding findings suspect with regard to the underlying mechanisms or processes contributing to the changes in behaviour. This was demonstrated by Sher and Wood's (1997) panel study of child alcohol-related behaviour, that involved four data cycles collected with one-year intervals between cycles. Differences between cross-lagged correlations across one-year intervals were unlike those across the entire four-year interval (using the first and last cycles), leading to very different conclusions based on their data depending only on which assessment interval they used.

Currently, not enough is known about the underlying mechanisms in intergenerational influences on mental health and development to definitely state

what the optimal measurement intervals should be in prospective research designs. In accordance with the objectives of this thesis, Studies 1 and 2 did successfully address mutual influences in maternal and child functioning in stable underlying symptomatology and frequent, subclinical change in functioning. Nonetheless, an appropriate follow-up investigation that seeks to replicate these findings may utilise different combinations of assessment intervals, such as weekly or monthly assessments.

Depressive Bias? A third consideration is the potential peril of examining how maternal mood and child behaviour influence one another with an exclusive reliance on mother reports as a source of data. Beyond their true association, there is an alternative theory of the significant mutual relations found in Studies 1 and 2. That is, mothers who reported feeling depressed had a greater tendency to distort or exaggerate the severity of their children's problem behaviour. This "depressive bias" hypothesis has intuitive appeal but limited evidence. Studies in which maternal reports were compared with behavioural observations and teacher-reports as gold standards have not consistently found that depressed mothers are any less accurate in assessing their child's behaviour than nondepressed mothers (see a critical review of by Richters [1992]). In fact, there is even evidence to suggest that memory is *facilitated* by depressed mood in mothers' assessments of child behaviour, which would make mothers the most appropriate data source on how child adjustment problems may influence their own well-being (McKendree-Smith & Scogin, 2000).

The question regarding depressive bias was demonstrated in a study by Modell and colleagues that examined the influence of maternal depression and its treatment on maternal reports of child behaviour (Modell, Modell, Wallander, Hodgens, Duke, & Wisely, 2001). In this study, antidepressant treatment was administered to a sample of 24 depressed mothers and treatment effects were examined using mothers' self-reported depressive symptoms on the Beck Depression Inventory and mothers' ratings of their child's behaviour on the Conners Parent Rating Scale. As a result of treatment, maternal depressive symptoms declined by 53 percent, child behavioural problems declined by 20 percent, and these improvements were significantly correlated. Despite the fact that child behaviour was not measured by independent raters, nor was there a control group, the authors still concluded that antidepressant treatment only corrected a "depressive bias" in maternal reports of child behaviour. The authors did not acknowledge the possibility that improved child behaviour may have occurred through an improvement in mother-child interactions.

Similar confusion regarding depressive biases has plagued a number of studies. Maternal reports of child behaviour are often the sole criteria by which child symptoms are assessed and diagnosed even though it remains unclear whether maternal distress or psychopathology may influence the accuracy of these reports. Typically, however, studies have shown that mothers – depressed or not – provide reliable and accurate assessments of child psychopathology and that depressed mothers are not significantly biased to report child symptoms that had not occurred (Faraone, Biederman, & Milberger,

1995; Querido, Eyeberg, & Boggs, 2001; Sawyer, Streiner, & Baghurst, 1998).

In consideration of the limited evidence for a “depressive bias” in maternal reports of child behaviour, the findings from Studies 1 and 2 may have still been affected by other sources of method variance. For instance, relations between maternal mood and child adjustment outcomes may have been affected by a social desirability bias. Mothers who were reluctant to report child symptoms may have been equally reluctant to self-report their own mood states. As well, although no outlying data points were found in either study, a tendency exaggerate the severity of both child symptoms and mood (even in just a few participants) may have inflated the size of a correlation or beta coefficient. The ideal way to circumvent this threat to internal validity is to utilise multiple sources of information. Replication of these studies that include supplemental data from either behavioural observations of mother-child interactions, child self-reports, child assessments of maternal mood, paternal reports of maternal mood and child behaviour, or peer-, teacher-, or sibling-reports, may contribute to more a reliable and valid assessments of maternal and child functioning as well as a test the “depressive bias” hypothesis.

Future Research

The relative contributions of mechanisms of risk and resilience involved in coercive mutual influences between emotional distress in mothers and adjustment problems in children are best elucidated by clinical and laboratory studies. As demonstrated here, however, panel studies still provide insight into

the direction and magnitude of such influences and they generate testable hypotheses regarding the mechanisms of risk and resilience to be studied in further research. An agenda for future studies is offered here with the objective of refining the integrative model of risk mechanisms depicted in Figure 1. Such research may further our understanding of family influences on maternal and child mental health:

1. To better quantify the mutual effects on maternal and child functioning, experimental data are needed from clinical trials of psychosocial interventions and large field experiments of mental health promotion programs that include outcome measures for children, fathers, and other family members;
2. More research is needed on the consistency of temporal relations between maternal and child outcomes using panel studies with varying assessment intervals and with community and clinical populations – two key factors that were confounded between Studies 1 and 2 of this thesis;
3. Studies have suggested that child psychological symptoms may be related more strongly to those of the father than to those of the mother and that differences may exist in how sons and daughters are influenced by parental distress (e.g., Banez & Compas, 1990; Compas, Howell, Ledoux, et al., 1989; Compas, Howell, Phares, Williams, & Giunta, 1989;

Forehand & Smith, 1986; Holahan & Moos, 1987). While no such child sex differences were found in Studies 1 and 2, further research is warranted of the influence of paternal distress and paternal involvement on child and maternal functioning;

4. Additional longitudinal studies are needed to determine whether child behavioural reactions to specific dimensions of maternal mood and parents' behavioural reactions to internalising and externalising child behaviour differ across stages of child and adolescent development;
5. To address the methodological weaknesses in Studies 1 and 2, replication studies involving measures of key mediating constructs, varying assessment intervals, and multiple data sources would contribute to testing the validity of these results.

Practical Implications

Mental health problems in mothers and children are common, burdensome, and intrinsically intertwined. Clinicians who work with families with emotional and behavioural problems should be aware that interventions for maternal depression or childhood disorders may indirectly enhance functioning by way of influences on any of the mediating factors described here (e.g., poor parenting practices, difficult parent-child relations) thereby lending health benefits to all family members. The practical implications of such linkages are explored here

for clinical assessment and treatments, and public health policy.

Clinical Assessment and Treatment

Treatments for maternal depression may help reduce child morbidity, and treatments for child adjustment problems may help reduce maternal morbidity. From a clinical standpoint, it has long been recognised that intergenerational transmission of psychopathology may be interrupted with dyadic assessment and treatments for parents and children (Rutter, 1990). This principle has yet to be fully integrated into common psychological treatments. Some treatment models (e.g., psychostimulant medication for child hyperactivity) focus almost exclusively on individual symptoms and do not always account for the contribution of family dynamics to illness and treatment outcomes. When parents are the facilitators of treatments for children, it is likely that their thinking will influence the success of the interventions (Hoza et al., 2000) – especially when the intervention is self-administered by the parent with little or no contact with a therapist (Elgar & McGrath, 2003). Parent training programs for managing most forms of disruptive behaviour in children are premised on the notion that change in parent behaviour leads to change in child behaviour. Alternatively, behavioural treatments for adult depression emphasise the response contingencies of family members and peers. The results of the present studies underscore the potential for enhancing unilateral treatment models such as these by considering latent but sometimes salient influences of maternal adjustment on child health and child adjustment on maternal health.

Illustrating the negative consequences of such family influences, the Multimodal Treatment Study of Children with Attention Deficit Hyperactivity Disorder found that mothers' self-reported use of dysfunctional discipline, low self-esteem, and low parenting efficacy, each predicted worse child outcome as a result of treatment for the Disorder (Hoza et al., 2000). In another study, maternal stress, hostility, and depressive symptoms each predicted whether or not children who were referred for treatment for conduct problems would actually start treatment, suggesting that some of the most distressed families that are referred to specialist care may not even show to treatment sessions (Calam, Bolton, & Roberts, 2002). On the other hand, the positive consequences of such family influences were demonstrated in a three-year controlled trial by Cicchetti, Rogosch, and Toth (2000). They found that when toddlers participated in a treatment for maternal depression ("toddler-parent psychotherapy"), mothers became significantly better compared to a non-treatment group and at a three-year follow up, the children showed higher IQ than children whose mothers received individual psychotherapy. Other studies have shown similar success using attachment-centred and cognitive-behavioural interventions for mothers and children (Sanders & McFarland, 2000; Sexson, Glanville, & Kaslow, 2001).

The data converge on an important conclusion. Concomitant or prior treatment of maternal depression by medication or psychotherapy may enhance the treatment of child behaviour problems, and concomitant or prior treatment of child behaviour problems may enhance efficacy of depression treatments in

mothers. Just as there is evidence that untreated problems in children or mothers can deleteriously influence the health of the other, there is also evidence that treating families as an enmeshed system may extend the benefits of treatment from the individual to the family. Family factors should not be considered secondary in clinical practice and treatment planning (Hoza et al., 2000).

Public health policy

In the general population, depression afflicts approximately one in five mothers and emotional and behavioural problems afflict approximately one in four children. Worse still, only a minority of those with these conditions will receive adequate assessment and treatment. Public health initiatives that are intended to help promote the health and well-being of children or mothers should have beneficial side effects analogous to those observed in clinical settings. Epidemiological studies suggest that primary prevention (i.e., risk reduction) community health interventions that address problems of poverty, child neglect, access to good quality child care and health care, or maternal education may help to reduce the incidence of maternal depression and child adjustment problems (Curtis et al., 2001; Rutter, 1989; Lipman, Boyle, Dooley, & Offord, 2002). Secondary prevention initiatives that focus on screening and early intervention mental health initiatives for high-risk populations may promote resilience in child and maternal well-being. There are several potential areas of policy impact but, unfortunately, supporting data from large field studies are

lacking and sorely needed.

Conclusion

This research addressed an understudied question of whether mood disturbance in mothers is normally an antecedent or a consequence of adjustment problems in children. According to the data, the answer to this question depends somewhat on the type and severity of symptoms and the time parameters involved. However, transactional relations between maternal and child outcomes were consistently found in two independent studies. The implications of these findings were explored for developmental psychopathology as well as clinical practice and policy. While it remains for future research to clarify the involvement of important mediating and moderating mechanisms, the findings were consistent with a transactional-developmental model of maternal mood and child adjustment that was described at the outset of this thesis. Simply stated, psychological functioning in the child or the mother plays an influential role in the aetiology, maintenance, treatment, and prevention of emotional distress in mothers and child maladjustment. In a research literature that is dominated by negative, even pathological, language used to describe the deleterious consequences of symptoms in mothers or children, it is worthwhile to also appreciate the benefits attributable to having a mother or a child who is healthy. Unravelling antecedent-consequence conditions of maternal and child health outcomes illuminates aspects of both risk and resilience, offering new directions for further investigation.

Appendix A

*Modified version of the CES-D used to assess
maternal depression in Study 1*

CES-D*Response Scale:*

0 = rarely or none of the time (less than one day)

1 = some or a little of the time (1-2 days)

2 = occasionally or a moderate amount of the time (3-4 days)

3 = most or all of the time (5-7 days)

During the past week:

1. I did not feel like eating; my appetite was poor.	0	1	2	3
2. I felt that I could not shake off the blues even with help from my family or friends.	0	1	2	3
3. I had trouble keeping my mind on what I was doing.	0	1	2	3
4. I felt depressed.	0	1	2	3
5. I felt that everything I did was an effort.	0	1	2	3
6. I felt hopeful about the future.	0	1	2	3
7. My sleep was restless.	0	1	2	3
8. I was happy.	0	1	2	3
9. I felt lonely.	0	1	2	3
10. I enjoyed life.	0	1	2	3
11. I had crying spells.	0	1	2	3
12. I felt that people disliked me.	0	1	2	3

Appendix B

Informed Consent Form used in Study 2



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CONSENT FORM

Title Page

Title: Diary Study of Maternal Mood and Child Behavioural Problems

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If you have concerns or questions at any time during or after the study, contact:

***Frank Elgar
Department of Psychology
Dalhousie University, Halifax, NS
Tel: 494-1938 or 429-6917
E-mail: fjelgar@is2.dal.ca***

Introduction

We invite you to take part in a research study conducted under the auspices of Dalhousie University. Taking part in this study is voluntary and you may withdraw from the study at any time. The quality of your care and your child's care at the Summer Treatment Program will not be affected by whether you participate or not. If you are a student or employee of Dalhousie University, your performance evaluation will not be affected by your desire not to participate. The study is described below. This description tells you about the risks, inconvenience, or discomfort which you might experience. Participating in the study might not benefit you, but we might learn things that will benefit others. You should discuss any questions you have about this study with the people who explain it to you.

Purpose of the study

The purpose of this study is to find out whether changes in mothers' mood tend to occur before or after changes in their children's behaviour (e.g., hyperactivity, acting out).

Who can participate in the study

You may participate in this study if you are a mother of a child age 6 to 17 years and are fluent in English.

Who will be conducting the research

The study will be conducted by Mr. Frank Elgar, a doctoral student in psychology at Dalhousie University. The study will be supervised by Dr. Patrick McGrath, a professor of psychology at Dalhousie University

What you will be asked to do

You will be given a diary in which you will answer questions about your mood and your child's behaviour. This involves reading a list of 37 emotions (e.g., sad, cheerful, confused) and indicating on a 5-point scale how well each one describes your mood that day (from "not at all" to "extremely"). You will also read 10 descriptions of disruptive child behaviour (e.g., hyperactive, inattentive, acts out) and indicate on a 4-point scale how well each describes your child's behaviour that day (from "not at all" to "very much"). These questions will take about 5 to 10 minutes to answer and you will answer these questions in the evening (6-9 pm) every day for a period of 21 days.

You will complete these questionnaires with Mr. Elgar at the beginning of the study and at the end of each week of the study. There are four interviews in all. You and Mr. Elgar will share contact information and arrange interview

times that are convenient for you. At the end of the study, Mr. Elgar will arrange to pick up your diary.

Possible Risks and Discomforts

There is minimal potential risk posed to the participants of this study. Inadvertent disclosure of your information to your child or other family members (e.g., from seeing the contents of your diary or overhearing telephone interviews) may cause unforeseen difficulties. You will be advised of ways to reduce the risk of this from occurring. You may discontinue the study for any reason at any time.

Potential Benefits

There is evidence to suggest that exercises in identifying changes in one's mood and sources of stress may be beneficial to individuals who are prone to mild depressive episodes. It is possible – but cannot be guaranteed – that keeping a “daily mood diary” may help interrupt negative thoughts that are an important part of depression.

Compensation

You will be paid \$40 for your participation in this study. This will be paid to you after the study whether or not you are able to complete the entire study.

Confidentiality

Appropriate safeguards will be used to ensure your information is stored securely and kept strictly confidential. Once all your information is collected, it will be entered into a computer database and labelled with an anonymous code number. All information will be stored anonymously at a secure research facility at Dalhousie University and will be accessible only by the Study Investigator and Supervisor. A key sheet linking your name and phone number to your code number will be securely stored in a separate location at Dalhousie University.

You will not be identified in any reports or publications. Any papers arising from this study will describe only group trends and not individual data.

Questions

If you have any questions or concerns during the course of the study, contact Mr. Elgar at 494-1938 (e-mail: fjelgar@is2.dal.ca). If your question is urgent, you may call after regular office hours at 429-6917.

Summary and Follow-up

Over a period of 21 days, you will conduct a brief (3-5 minute) diary assessment each day and participate in four 10-15 minute telephone-based assessments. At the end of the study, Mr. Elgar will inform you whether your data reflect a clinically significant level of depressive symptoms and whether your child's data reflect significant problems of conduct or attention. If either is the case, Mr. Elgar will inform you of local mental health services.

Termination

You may discontinue the study at any time. Your decision to discontinue the study will not affect your treatment or your child's treatment at the Summer Treatment Program or your evaluation or treatment at Dalhousie University (if applicable).

Problems or Concerns

In the event that you have any difficulties with, or wish to voice concern about, any aspect of your participation in this study, you may contact Human Research Ethics / Integrity Coordinator at Dalhousie University's Office of Human Research Ethics and Integrity for assistance: (902) 494-1462

Signatures

I have read the explanation about his study. I have been given the opportunity to discuss it and my questions have been answered to my satisfaction. I hereby consent to take part in this study. However I realize that my participation is voluntary and that I am free to withdraw from the study at any time.

Participant:

(Name)

(Date)

Person who has obtained consent:

(Name)

(Date)

REFERENCES

- Achenbach, T. M. (1991). *Manual for the Child Behavior Checklist/4-18 and 1991 Profile*. Burlington, VT: University of Vermont, Department of Psychiatry.
- American Psychiatric Association (1987). *Diagnostic and statistical manual of mental disorders (3rd ed. Revised)*. Washington, DC: Author.
- American Psychiatric Association (1994). *Diagnostic and statistical manual of mental disorders (4th ed.)*. Washington, DC: Author.
- Amsterdam, J., Maislin, G., Gold, P., & Winokur, A. (1989). The assessment of abnormalities in hormonal responsiveness at multiple levels of the hypothalamic-pituitary-adrenocortical axis in depressive illness. *Psychoneuroendocrinology, 14*, 43-63.
- Arnold, E. H., & O'Leary, S. G. (1995). The effect of child negative affect on maternal discipline behavior. *Journal of Abnormal Child Psychology, 23*, 585-595.
- Ashman, S. B., & Dawson, G. (2002). Maternal depression, infant psychobiological development, and risk for depression. In S. H. Goodman, & I. H. Gotlib (Eds.), *Children of depressed parents: Mechanisms of risk and implications for treatment* (pp. 37-58). Washington, DC: American Psychological Association,.
- Atkinson, L. P., Paglia, A., Coolbear, J., Niccols, A., Parker, K. C. H., & Guger,

- S. (2000). Attachment security: A meta-analysis of maternal mental health correlates. *Clinical Psychology Review, 20*, 1019-1040.
- Baker, F., Denniston, M., Zabora, J., Pollard, A., & Dudley, W. N. (2002). A POMS short form for cancer patients: Psychometric and structural evaluation. *Psychooncology, 11*, 273-281.
- Bandura, A. (1973). *Aggression: A social learning approach*. Englewood Cliffs, NJ: Prentice-Hall.
- Bandura, A. (1986). *Social foundations of thought and action: A social cognitive theory*. Englewood Cliffs, NJ: Prentice Hall.
- Banez, G. A., & Compas, B. E. (1990). Children's and parent's daily stressful events and psychological symptoms. *Journal of Abnormal Child Psychology, 18*, 591-605.
- Barkley, R. A., Anastopoulos, A. D., Guevremont, D. C., & Fletcher, K. E. (1992). Adolescents with attention deficit hyperactivity disorder: Mother-adolescent interactions, family beliefs and conflicts, and maternal psychopathology. *Journal of Abnormal Child Psychology, 20*, 263-288.
- Beardslee, W. R., Keller, M. B., Lavori, P. W., Staley, J., & Sacks, N. (1993). The impact of parental affective disorder on depression in offspring: A longitudinal follow-up in a nonreferred sample. *Journal of the American Academy of Child and Adolescent Psychiatry, 32*, 723-730.
- Beardslee, W. R., Schultz, L. H., & Selman, R. L. (1987). Level of social-cognitive development, adaptive functioning, and DSM-III diagnoses in adolescent offspring of parents with affective disorders: Implications for

- the development of the capacity for mutuality. *Developmental Psychology*, 23, 807-815.
- Beardslee, W. R., Versage, E. M., & Gladstone, T. R. G. (1998). Children of affectively ill parents: A review of the past 10 years. *Journal of the American Academy of Child and Adolescent Psychiatry*, 37, 1134-1141.
- Beck, A. T. (1967). *Depression: Clinical, experimental, and theoretical aspects*. New York: Harper & Row.
- Beck, A. T. (1970). *Depression: Causes and treatment*. Philadelphia, PA: University of Pennsylvania Press.
- Beck, A. T., Steer, R. A., & Brown, G. K. (1996). *Beck Depression Inventory Manual (2nd ed)*. San Antonio, TX: The Psychological Corporation.
- Beck, A. T., Ward, C. H., Mendelson, M., Mock, J., & Erbaugh, J. (1961). An inventory for measuring depression. *Archives of General Psychiatry*, 4, 53-63
- Beck, C. T. (2002). Maternal depression and child behaviour problems. *Journal of Advanced Nursing*, 29, 623-629.
- Belsher, G., & Costello, G. C. (1988). Relapse after recovery from unipolar depression: A critical review. *Psychological Bulletin*, 104, 84-96.
- Bifulco, A., Moran, P. M., Ball, C., Jacobs, C., Bains, R. Bunn, A., & Cavagin, J. (2002). Child adversity, parental vulnerability and disorder: Examination inter-generational transmission of risk. *Journal of Child Psychology and Psychiatry and Allied Disciplines*, 43, 1075-1086.
- Billings, A. G., & Moos, R. H. (1986). Children of parents with unipolar

- depression: A controlled 1-year follow-up. *Journal of Abnormal Child Psychology*, 14, 149-166.
- Birmaher, B., & Heydl, P. (2001). Biological studies in depressed children and adolescents. *International Journal of Neuropsychopharmacology*, 4, 149-157.
- Black, M. M., Papas, M. A., Hussey, J. M., Dubowitz, H., Kotch, J. B., & Starr, R. H. (2002). Behavior problems among preschool children born to adolescent mothers: Effects of maternal depression and perceptions of partner relationships. *Journal of Clinical Child and Adolescent Psychology*, 31, 16-26.
- Bowlby, J. (1973). *Attachment and loss. Vol. 2. Separation*. New York: Basic Books.
- Bowlby, J. (1988). *A secure base: Parent-child attachment and healthy human development*. New York: Basic Books.
- Boyle, M. H., Offord, D. R., Hofmann, H. G., Catlin, G. P., Byles, J. A., Cadman, D. T., Crawford, J. W., Links, P. S., Rae-Grant, N. I., & Szatmari, P. (1987). Ontario child health study: I. Methodology. *Archives of General Psychiatry*, 44, 826-831.
- Breton, J. J., Bergeron, L., Valla, J. P., Berthiaume, C., Gaudet, N., & Lambert, J. (1999). Quebec child mental health survey: Prevalence of DSM-III-R mental health disorders. *Journal of Child Psychology and Psychiatry and Allied Disciplines*, 40, 375-384.
- Breznitz, Z., & Sherman, T. (1987). Speech patterning of natural discourse of

well and depressed mothers and their young children. *Child Development, 58*, 395-400.

Briggs-Gowan, M. J., Carter, A. S., Schwab-Stone, M. (1996). Discrepancies among mother, child, and teacher reports: Examining the contributions of maternal depression and anxiety. *Journal of Abnormal Child Psychology, 24*, 749-765.

Brown, R. T., Borden, K. A., Clingerman, S. R., & Jenkins, P. (1988).

Depression in attention deficit-disordered and normal children and their parents. *Child Psychiatry and Human Development, 18*, 119-132.

Brown, G. W., & Harris, T. (1978). *Social origins of depression*. New York: Free Press.

Brunshaw, J. M., & Szatmari, P. (1988). The agreement between behaviour checklists and structured psychiatric interviews for children. *Canadian Journal of Psychiatry, 33*, 474-481.

Burbach, D. J., & Borduin, C. M. (1986). Parent-child relations and the etiology of depression: A review of methods and findings. *Clinical Psychology Review, 6*, 133-153.

Byrne, B.M. (1994). *Structural equation modelling with EQS and EQS/Windows: Basic concepts, applications, and programming*. Thousand Oaks, CA: Sage.

Cadore, R. J. (1978). Evidence for genetic inheritance of primary affective disorder in adoptees. *American Journal of Psychiatry, 135*, 463-466.

Calam, R., Bolton, C., & Roberts, J. (2002). Maternal expressed emotion,

- attributions and depression and entry into therapy for children with behaviour problems. *British Journal of Clinical Psychology*, *41*, 213-216.
- Carlson, E. A., Sroufe, L. A. (1995). Contribution of attachment theory to developmental psychopathology. In D. Cicchetti & D. J. Cohen (Eds.). *Developmental psychopathology, Vol. 1: Theory and methods* (pp. 581-617). New York: John Wiley & Sons.
- Carter, A. S., Garrity-Rokous, F. E., Chazan-Cohen, R., Little, C., & Briggs-Gowan, M. J. (2001). Maternal depression and comorbidity: Predicting early parenting, attachment security, and toddler social-emotional problems and competencies. *Journal of the American Academy of Child and Adolescent Psychiatry*, *40*, 18-26.
- Cohen, J. (1988). *Statistical power analysis for the behavioral sciences* (2nd ed.). Hillsdale, NJ: Erlbaum.
- Cox, A. D., Puckering, C., Pound, A., & Mills, M. (1987). The impact of maternal depression in young people. *Journal of Child Psychology and Psychiatry*, *28*, 917-928.
- Cicchetti, D., & Rogosch, F. A. (1996). Equifinality and multifinality in developmental psychopathology. *Development and Psychopathology*, *8*, 597-600.
- Cicchetti, D., Rogosch, F. A., & Toth, S. L. (1998). Maternal depressive disorder and contextual risk: Contributions to the development of attachment insecurity and behaviour problems in toddlerhood. *Developmental Psychopathology*, *10*, 283-300.

- Cicchetti, D., Rogosch, F. A., & Toth, S. L. (1998). Maternal depressive disorder and contextual risk: Contributions to the development of attachment insecurity and behaviour problems in toddlerhood. *Developmental Psychopathology, 10*, 283-300.
- Cicchetti, D., Rogosch, F. A., & Toth, S. L. (2000). The efficacy of toddler-parent psychotherapy for fostering cognitive development in offspring of depressed mothers. *Journal of Abnormal Child Psychology, 28*, 135-148.
- Cicchetti, D., & Toth, S. L. (1998). The development of depression in children and adolescents. *American Psychologist, 53*, 221-241.
- Civic, D., & Holt, V. L. (2000). Maternal depressive symptoms and child behaviour problems in a nationally representative normal birthweight sample. *Maternal and Child Health Journal, 4*, 215-221.
- Clayton, P. J. (1987). Anxious depression: A reemerging subtype of depression. In G. Racagni & E. Smeraldi (Eds.), *Anxious Depression: Assessment and Treatment* (pp. 1- 6). New York: Raven Press.
- Cohn, J. E., & Tronick, E. Z. (1983). Three-month-old infants' reaction to simulated maternal depression. *Child Depression, 54*, 185-193.
- Compas, B. E., Howell, D. C., Ledoux, N., Phares, V., & Williams, R. A. (1989). Parent and child stress and symptoms: An integrative analysis. *Developmental Psychology, 25*, 550-559.
- Compas, B. E., Howell, D. C., Phares, V., Williams, R. A., & Giunta, C. T. (1989). Risk factors for emotional/behavioural problems in young

- adolescents: A prospective analysis of adolescent and parental stress and symptoms. *Journal of Consulting and Clinical Psychology, 57*, 732-740.
- Conners, C.K., Sitarenios, G., Parker, J.D.A., & Epstein, J.N. (1998). Revision and restandardization of the Conners Teacher Rating Scale (CTRS-R): Factor structure, reliability, and criterion validity. *Journal of Abnormal Child Psychology, 26*, 279-291.
- Cook, T. D., & Campbell, D. T. (1979). *Quasi-experimentation: Design and analysis issues for field settings*. Chicago, IL: Rand McNally.
- Craddock, N., Jones, I. (1999). Genetics of bipolar disorder. *Journal of Medical Genetics, 36*, 585-594.
- Cummings, E. M., & Davies, P. T. (1994). Maternal depression and child development. *Journal of Child Psychology, Psychiatry, and Allied Disciplines, 35*, 73-112.
- Cunningham, C. E., Bennes, B. B., & Siegel, L. S. (1988). Family functioning, time allocation, and parental depression in the families of normal and ADHD children. *Journal of Clinical Child Psychology, 17*, 160-177.
- Curtis, L. J., Dooley, M. D., Lipman, E. L., & Feeny, D. J. (2001). The role of permanent income and family structure in the determination of child health in Canada. *Health Economics, 10*, 287-302.
- Da Costa, D., Dritsa, M., Larouche, J., & Brender W. (2000). Psychosocial predictors of labor/delivery complications and infant birth weight: A prospective multivariate study. *Journal of Psychosomatic Obstetrics and Gynaecology, 21*, 137-148.

- Davies, P. T., & Windle, M. (1997). Gender-specific pathways between maternal depressive symptoms, family discord, and adolescent adjustment. *Developmental Psychology, 33*, 657-668.
- Dawson, G., Ashman, S. B., Hessler, D., Spieker, S., Frey, K., Panagiotides, H., & Embry, L. (2001). Autonomic and brain electrical activity in securely- and insecurely-attached infants of depressed mothers. *Infant Behavior and Development, 24*, 135-149.
- Dawson, G., Frey, K., Panagiotides, H., Yamada, E., Hessler, D., & Osterling, J. (1999). Infants of depressed mothers exhibit atypical frontal electrical brain activity during interactions with mother and with a familiar, nondepressed adult. *Child Development, 70*, 1058-1066.
- Derogatis, L. R. (1983). *SCL-R-90 administration, scoring, and procedures manual II*. Townsen, MD: Clinical Psychometric Research
- Dielman, T. E., (1989). *Pooled cross-sectional and time series data analysis*. New York: Marcel Dekker.
- Dodge, K. A. (1990). Developmental psychopathology in children of depressed mothers. *Developmental Psychology, 26*, 3-6.
- Dodge, K. A., Pettit, G. S., & Bates, J. E. (1994). Socialization mediators of the relation between socioeconomic status and child conduct problems. *Child Development, 65*, 649-665.
- Downey, G., & Coyne, J. C. (1990). Children of depressed parents: An integrative review. *Psychological Bulletin, 108*, 50-76.
- Duggan, C., Sham, P., Minne, C., Lee, A., & Murray, R (1998). The family

- history as a predictor of poor long-term outcome in depression. *British Journal of Psychiatry*, *173*, 527-530.
- Eaves, L. J., Silberg, J. L., Meyer, J. M., Maes, H. H., Simonoff, E., Pickles, A., Rutter, M., Neale, M. C., Reynolds, C. A., Erikson, M. T., Heath, A. C., Loeber, R., Truett, K. R., & Hewitt, J. K. (1997). Genetics and developmental psychopathology: II. The main effects of genes and environment on behavioral problems in the Virginia Twin Study of Adolescent Behavioral Development. *Journal of Child Psychology, Psychiatry, and Allied Disciplines*, *38*, 965-980.
- Elgar, F. J., & Arlett, C. (2002). Perceived social inadequacy and depressive symptoms in adolescents. *Journal of Adolescence*, *25*, 306-310.
- Elgar, F. J., Arlett, C., & Groves, R. (in press). Stress, coping and behavioural problems in rural and urban adolescents. *Journal of Adolescence*.
- Elgar, F. J., Knight, J., Worrall, G. J., & Sherman, G. (2003). Attachment characteristics and behavioural problems in rural and urban juvenile delinquents. *Child Psychiatry and Human Development*, *34*, 16-30.
- Elgar, F. J., & McGrath, P. J. (2003). Self-administered psychosocial treatments for children and families. *Journal of Clinical Psychology*, *59*, 255-274.
- Eley, T. (1999). Behavioural genetics as a tool for developmental psychology: Anxiety and depression in children and adolescents. *Clinical Child and Family Psychology Review*, *2*, 21-36.
- Enns, M. W., Cox, B. J., & Larsen, D. K. (2000). Perceptions of parental bonding and symptom severity in adults with depression: Mediation by

- personality dimensions. *Canadian Journal of Psychiatry*, 45, 263-268.
- Fergusson, D. M., Horwood, L. J., & Lynskey, M. T. (1995). Maternal depressive symptoms and depressive symptoms in adolescents. *Journal of Child Psychology and Psychiatry and Allied Disciplines*, 36, 1161-1178.
- Field, T. (1984). Early interactions between infants and their postpartum depressed mothers. *Infant Behaviour and Development*, 7, 537-540.
- Field, T. (2002). Prenatal effects of maternal depression. In S. H. Goodman, & I. H. Gotlib (Eds.), *Children of depressed parents: Mechanisms of risk and implications for treatment (pp 59-88)*. Washington, DC: American Psychological Association.
- Field, T., Healy, B., Goldstein, S., & Guthertz, M. (1990). Behaviour-state matching and synchrony in mother-child interactions of nondepressed vs. depressed dyads. *Developmental Psychology*, 26, 7-14.
- Field, T., Healy, B., Goldstein, S., Perry, S., Bendell, D., Schanberg, S., Zimmerman, E. A., & Kuhn, C. (1988). Infants of depressed mothers show "depressed" behaviour even with non-depressed adults. *Child Development*, 59, 1569-1579.
- Forehand, R., Lautenschlager, G. J., Faust, J., & Graziano, W. G. (1986). Parent perceptions and parent-child interactions in clinic-referred children: A preliminary investigation of the effects of maternal depressive moods. *Behavior Research and Therapy*, 24, 73-75.
- Forehand, R., & McCombs, A. (1988). Unravelling the antecedent-consequence conditions in maternal depression and adolescent functioning. *Behavior*

Research and Therapy, 26, 399-405.

- Forehand, R., McCoombs, A., & Brody, G. H. (1987). The advances between parental depressive mood states and child functioning. *Advances in Behaviour Research and Therapy, 9, 1-20.*
- Forehand, R., & Smith, K. A. (1986). Who depresses whom? A look at the relationship of adolescent mood to maternal and paternal mood. *Child Study Journal, 16, 19-23.*
- Forehand, R., Wells, K. C. & Griest, D. L. (1980) An examination of the social validity of a parent training program. *Behavior Therapy, 11, 488-502.*
- Francis, D., Diorio, J., Liu, D., & Meaney, M. (1999). Nongeomic transmission across generations of maternal behaviour and stress responses in the rat. *Science, 286, 1155-1158.*
- Ge, X., Conger, R. D., Lorenz, F. O., Shanahan, M., & Elder, G. H. Jr. (1995). Mutual influences in parent and adolescent psychological distress. *Developmental Psychology, 31, 406-419.*
- Goodman, S. H., Adamson, L. B., Riniti, J., & Cole, S. (1994). Mothers' expressed attitudes: Associations with maternal depression and children's self-esteem and psychopathology. *Journal of the American Academy of Child and Adolescent Psychiatry, 33, 1265-1274.*
- Goodman, S. H., Brogan, D., Lynch, M. E., & Fielding, B. (1993). Social and emotional competence in children of depressed mothers. *Child Development, 64, 516-531.*
- Goodman, S. J., & Gotlib, I. H. (1999). Risk for psychopathology in the children

- of depressed mothers: A developmental model for understanding mechanisms of transmission. *Psychological Review*, 106, 458-490.
- Hahn, A. (1999, May 11). Computer Programm für "cross-lagged" Korrelationen. Retrieved from the World Wide Web on January 21, 2003 at <http://userpage.zedat.fu-berlin.de/~ahahn/cross.htm>.
- Hammen, C., Adrian, C., Gordon, D., Burge, D., Jaenicke, C., & Hiroto, D. (1987). Children of depressed mothers: Maternal strain and symptom predictors of dysfunction. *Journal of Abnormal Psychology*, 96, 190-198.
- Hammen, C., Burge, D., & Stansbury, K. (1990). Relationship of mother and child variables to child outcomes in a high risk sample: A causal modelling analysis. *Developmental Psychology*, 26, 24-30.
- Hartley, D., Bird, D., & Dempsey, P. (1999). Mental health and substance abuse. In Ricketts, T. (Ed.), *Rural Health in the United States* (pp. 159-178). New York: Oxford University Press.
- Hashima, P. Y., & Amato, P. R. (1994). Poverty, social support, and parental behaviour. *Child Development*, 65, 394-403.
- Healy, C. M., & McKay, M. F. (2000). Nursing stress: The effects of coping strategies and job satisfaction in a sample of Australian nurses. *Journal of Advanced Nursing*, 31, 681-688.
- Hedegaard, M., Henriksen, T. B., Sabroe, S., & Secher, N. J. (1993). Psychological distress in pregnancy and preterm delivery. *British Medical Journal*, 24, 307, 234-239.
- Hertzgaard, L., Gunnar, M., Erickson, M., & Nachmais, M. (1995).

- Adrenocortical response to the strange situation in infants with disorganised/disoriented attachment relationships. *Child Development*, 66, 1100-1106.
- Hofstra, M. B., Van der Ende, J., & Verhulst, F. C. (2000). Continuity and change of psychopathology from childhood into adulthood: A 14-year follow-up study. *Journal of the American Academy of Child and Adolescent Psychiatry*, 39, 850-858.
- Hops, H., Biglan, A., Sherman, L., Arthur, J., Friedman, L., & Osteen, V. (1987). Home observations of family interactions of depressed women. *Journal of Consulting and Clinical Psychology*, 55, 341-346.
- Hops, H., Sherman, L., & Biglan, A. (1990). Maternal depression, marital discord, and children's behaviour: A developmental perspective. In G. R. Patterson (Ed.), *Depression and aggression in family interactions* (pp. 185-208). Hillsdale, NJ: Erlbaum.
- Holahan, C. J., & Moos, R. H. (1987). Risk, resistance, and psychological distress: A longitudinal analysis with adults and children. *Journal of Abnormal Psychology*, 96, 3-13.
- Hoza, B., Owens, J. S., Pehlam, W. E. Jr., Swanson, J. M., Conners, C. K., Hinshaw, S. P., Arnold, L. E., & Kraemer, H. C. (2000). Parent cognitions as predictors of child treatment response in Attention-Deficit/Hyperactivity Disorder. *Journal of Abnormal Child Psychology*, 28, 569-583.
- Humphreys, L. G. (1991). Causal inference from observational data: Use of a redesigned cross-lagged methodology. *Intelligence*, 15, 151-156.

- Hunsley, J., Aubry, T., & Lee, C. (1997). *A profile of Canadian consumers of psychological services*. Ottawa, Canada: Canadian Psychological Association.
- Inoff-Germain, G., Nottelmann, E. D., & Radke-Yarrow, M. (1992). Evaluative communications between affectively ill and well mothers and their children. *Journal of Abnormal Child Psychology*, *20*, 189-212.
- Jaccard, J., & Wan C. K. (1993). Statistical analysis of temporal data with many observations: Issues for behavioral medicine data. *Annals of Behavioral Medicine*, *15*, 41-50.
- Johnson, D. (1995). Alternative methods for quantitative analysis of panel data in family research: Pooled time-series models. *Journal of Marriage and the Family*, *57*, 1065-1077.
- Johnston, C., & Mash, E. J. (1989). A measure of parenting satisfaction and efficacy. *Journal of Clinical Child Psychology*, *18*, 167-175.
- Jones, N. A., Field, T., Fox, N. A., Davalos, M., Lundy, B., & Hart, S. (1998). Newborns of mothers with depressive symptoms are physiologically less developed. *Infant Behavior and Development*, *21*, 537-541.
- Jones, N. A., Field, T., Fox, N. A., Lundy, B., & Davalos, M. (1997). EEG activation in one-month-old infants of depressed mothers. *Development and Psychopathology*, *9*, 491-505.
- Judd, L. L., Akiskal, H. S., & Paulus, M. P. (1997). The role and clinical significance of subsyndromal depressive symptoms (SDD) in unipolar major depressive disorder. *Journal of Affective Disorders*, *45*, 5-18.

- Katon, W., & Sullivan, M. D. (1990). Depression and chronic mental illness. *Journal of Clinical Psychiatry, 51*, 3-14.
- Keller, M. B., Lavori, P. W., Mueller, T. I., Endicott, J., Coryell, W., Hirschfeld, R., & Shea, T. (1992). Time to recovery, chronicity, and levels of psychopathology in major depression: A 5-year prospective follow-up of 431 subjects. *Archives of General Psychiatry, 49*, 809-816.
- Keller, M. B., & Shapiro, R. W. (1981). Major depressive disorder: Initial results from a one-year prospective naturalistic follow-up study. *Journal of Nervous and Mental Disorders, 169*, 761-768.
- Kelly, J. B. (2000). Children's adjustment in conflicted marriage and divorce: A decade review of research. *Journal of the American Academy of Child and Adolescent Psychiatry, 39*, 963-973.
- Kessler, R. C., McGonagle, K. A., Zhao, S., Nelson, C. B., Hughes, M., Eshleman, S., Wittchen, H-U., & Kendler, K. S. (1994). Lifetime and 12-month prevalence of DSM-III-R psychiatric disorders in the United States: Results from the National Comorbidity Survey. *Archives of General Psychiatry, 51*, 8-19.
- Kendler, K. S., & Prescott, C. A. (1999). A population-based twin study of lifetime major depression in men and women. *Archives of General Psychiatry, 50*, 789-796.
- Kenny, D. A. (1975). Cross-lagged panel correlation: A test for spuriousness. *Psychological Bulletin, 82*, 887-903.
- Kline, R. (1991). Latent variable path analysis in clinical research: A beginner's

- tour. *Journal of Clinical Psychology*, 47, 471-484.
- Kovacs, M. (1985). The Children's Depression Inventory. *Psychopharmacology Bulletin*, 21, 995-998.
- Kraemer, H. C., Kazdin, A. E., Offord, D. R., Kessler, R. C., & Jensen, P. S. (1997). Coming to terms with the terms of risk. *Archives of General Psychiatry*, 54, 337-343.
- Kringlen, E., Torgersen, S., & Cramer, V. (2001). A Norwegian psychiatric epidemiological study. *American Journal of Psychiatry*, 158, 1091-1098.
- Kurstjens, S., & Wolke, D. (2001). Effects of maternal depression on cognitive development of children over the first 7 years of life. *Journal of Child Psychology, Psychiatry and Allied Disciplines*, 42, 623-636.
- Lipman, E. L., Offord, D. R., & Boyle, M. H. (1994). Relation between economic disadvantage and psychosocial morbidity in children. *Canadian Medical Association Journal*, 151, 431-437.
- Lipman, E. L., Boyle, M. H., Dooley, M. D., & Offord, D. R. (2002). Child well-being in single-mother families. *Journal of the American Academy of Child and Adolescent Psychiatry*, 41, 75-82.
- Loney, J., & Milich, R. (1982). Hyperactivity, inattention, and aggression in clinical practice. In M. Wolraich & D. K. Rough (Eds.), *Advances in development and behavioral paediatrics* (pp. 113-147). Greenwich, CT: JAI Press.
- Lovejoy, M. C., Craczyk, P. A., O'Hare, E., & Neuman, G. (2000). Maternal depression and parenting behaviour: A meta-analytic review. *Clinical*

Psychology Review, 20, 561-592.

- Lundy, B., L., Field, T., & Pickens, J. (1996). Newborns of mothers with depressive symptoms are less expressive. *Infant Behavior and Development*, 19, 419-424.
- Lundy, B. L., Jones, N. A., Field, T., Nearing, G., Davalos, M., Pietro, P. A., Schanberg, S., & Kuhn, G. (1999). Prenatal depression effects on neonates. *Infant Behaviour and Development*, 22, 119-129.
- Lyons-Ruth, K., Lyubchik, A., Wolfe, R., & Bronfman, E. (2002). Parental depression and child attachment: Hostile and helpless profiles of parent and child behaviour among families at risk. In S. H. Goodman & I. H. Gotlib (Eds.), *Children of depressed parents: Mechanisms of risk and implications for treatment* (pp. 72-96). Washington, DC: American Psychological Association.
- Lytton, H. (1990). Child and parent effects in boys' conduct disorder: A reinterpretation. *Developmental Psychology*, 26, 683-697.
- Marchand, J. F., & Hock, E. (1998). The relation of problem behaviours in preschool children to depressive symptoms in mothers and fathers. *Journal of Genetic Psychology*, 159, 353-366.
- Martins, C., & Gaffa, E. A. (2000). Effects of early maternal depression on patterns of infant-mother attachment: A meta-analytic investigation. *Journal of Child Psychology, Psychiatry and Allied Disciplines*, 41, 737-746.
- Mash, E. J., & Johnston, C. (1990). Determinants of parenting stress:

- Illustrations from families of hyperactive children and families of physically abused children. *Journal of Clinical Child Psychology*, 19, 313-328.
- McNair, D.M., Lorr, M., & Droppleman, L.F. (1982). *Manual for the Profile of Mood States*. San Diego, CA: Educational and Industrial Testing Service.
- McGough, J., & Curry, J. F. (1992). Utility of the SCL-90-R with depressed and conduct-disordered adolescent inpatients. *Journal of Personality Assessment*, 59, 552-63.
- McGrath, E., Keita, G. P., Stickland, B. R., & Russo, N. F. (1990). *Women and depression: Risk factors and treatment issues*. Washington, DC: American Psychological Association.
- McKendree-Smith, N., & Scogin, F. (2000). Depressive realism: Effects of depression severity and interpretation time. *Journal of Clinical Psychology*, 56, 1601-1608.
- McLennan, J. D., & Kotelchuck, M. (2000). Parental prevention practices for young children in the context of maternal depression. *Pediatrics*, 105, 1090-1095.
- Miller, T. Q. (1997). Statistical methods for describing temporal order in longitudinal research. *Journal of Clinical Epidemiology*, 50, 1155-1168.
- Modell, J. D., Modell, P. J. G., Wallander, J., Hodgens, P. B., Duke, P. L., & Wisely, P. D. (2001). Maternal ratings of child behaviour improve with treatment of maternal depression. *Family Medicine*, 33, 691-695.
- Monck, E., Graham, P., Richman, N., & Dobbs, R. (1994). Adolescent girls: II.

- Background factors in anxiety and depressive states. *British Journal of Psychiatry*, 165, 770-780.
- Moore, K. J., Osgood, D. W., Larzelere, R. E., & Chamberlain, P. (1994). Use of pooled time series in the study of naturally occurring clinical events and problem behaviour in a foster care setting. *Journal of Consulting and Clinical Psychology*, 62, 718-728.
- Murray, C. J. L., & Lopez, A. D. (1996). *The global burden of disease: A comprehensive assessment of mortality and disability from diseases, injuries and risk factors in 1990 and projected to 2020*. Geneva, Switzerland: World Health Organisation.
- Murray, K. T., & Sines, J. O. (1996). Parsing the genetic and nongenetic variance in children's depressive disorder. *Journal of Affective Disorders*, 38, 23-34.
- Murray, L. (1992). The impact of postnatal depression on infant development. *Journal of Child Psychology and Psychiatry*, 33, 543-561.
- Murray, L., Sinclair, D., Cooper, P., Ducournau, P., Turner, P., & Stein, P. (1999). The socioemotional development of 5-year-old children of postnatally depressed mothers. *Journal of Child Psychology and Psychiatry and Allied Disciplines*, 40, 1259-1271.
- O'Connor, T. G., McGuire, S., Reiss, D., Hetherington, E. M., & Plomin, R. (1998). Co-occurrence of depressive symptoms and antisocial behaviour in adolescence: A common genetic liability. *Journal of Abnormal Psychology*, 107, 27-37.

- Offord, D. R. (1995). Child psychiatric epidemiology: Current status and future prospects. *Canadian Journal of Psychiatry, 40*, 284-288.
- Offord, D. R., Boyle, M. H., Fleming, J. E., Blum, H. M., & Grant, N. I. (1989). Ontario Child Health Study. Summary of selected results. *Canadian Journal of Psychiatry, 34*, 483-491.
- Offord, D. R., Boyle, M. H., & Jones, B. R. (1987). Psychiatric disorder and poor school performance among welfare children in Ontario. *Canadian Journal of Psychiatry, 32*, 518-525.
- Offord, D. R., Boyle, M. H., Szatmari, P., Rae-Grant, N. I., Links, P. S., Cadman, D. T., Byles, J. A., Crawford, J. W., Blum, H. M., Byrne, C., Thomas, H., & Woodward, C. A. (1987). Six-month prevalence of disorder and rates of service utilization. *Archives of General Psychiatry, 44*, 832-836.
- Ostrom, C. W. Jr. (1990). *Time series analysis: Regression techniques (2nd ed.: Series no. 07-009)*. Newbury Park, CA: Sage.
- Munroe-Blum, H., Boyle, M. H., Offord, D. R., & Kates, N. (1989). Immigrant children: Psychiatric disorder, school performance, and service utilization. *American Journal of Orthopsychiatry, 59*, 510-519.
- Parker, G., Tupling, H., & Brown, L. B. (1979). A parental bonding instrument. *British Journal of Medical Psychology, 52*, 1-10.
- Patterson, G. R. (1982). *Coercive family process*. Eugene, OR: Castalia Publishing Company.
- Patterson, G. R., Reid, J. B., & Dishion, T. J. (1992). *Antisocial boys*. Eugene,

OR: Castalia Publishing Company.

- Patterson, G. R., DeBayshe, B. D., & Ramsey, E. (1989). A developmental perspective on antisocial behaviour. *American Psychologist, 44*, 329-335.
- Pelham, W. E., Aronoff, H. R., Midlam, J. K., Shapiro, C. J., Gnagy, E. M., Chronis, A. M., Onyango, A. N., Forehand, G., Nguyen, A., & Wasmonskey, J. (1999). A comparison of Ritalin and Adderall: Efficacy and time-course in children with attention-deficit/hyperactivity disorder. *Pediatrics, 103*, 1-14.
- Pelham, W. E., Carlson, C., Sams, S. E., Vallano, G., Dixon, M. J., & Hoza, B. (1993). Seperate and combined effects of methylphenidate and behaviour modification on boys with attention deficit-hyperactivity disorder in the classroom. *Journal of Consulting and Clinical Psychology, 61*, 506-515.
- Pelham, W. E., Hoza, B., Pillow, D. R., Gnagy, E. M., Kipp, H. L., Greiner, A. R., Waschbusch, D. A., Trane, S. T., Greenhouse, J., Wolfson, L., & Fitzpatrick, E. (2002). Effects of methylphenidate and expectancy on children with ADHD: Behavior, academic performance, and attributions in a Summer Treatment Program and regular classroom setting. *Journal of Consulting and Clinical Psychology, 70*, 320-335.
- Pelham, W. E., Lang, A. R., Atkeson, B., Murphy, D. A., Gnagy, E. M., Greiner, A. R., Vodde-Hamilton, M., & Greenslade, K. E. (1997). Effects of deviant child behaviour on parental distress and alcohol consumption in laboratory interactions. *Journal of Abnormal Child Psychology, 25*, 413-424.

- Pelham, W. E., Milich, R., Murphy, D. A., & Murphy, H. A. (1989). Normative data on the IOWA Conners Teacher Rating Scale. *Journal of Consulting and Clinical Psychology, 18*, 259-262.
- Phillips, L.H.C., & O'Hara, M. W. (1991). Prospective study of postpartum depression: 4½-year follow-up of women and children. *Journal of Abnormal Psychology, 100*, 151-155.
- Piacentini, J. C., Cohen, P., & Cohen, J. (1992). Combining discrepant diagnostic information from multiple sources: Are complex algorithms better than simple ones? *Journal of Abnormal Child Psychology, 20*, 51-63.
- Piacentini, J., Shaffer, D., Fisher, P., Schwab-Stone, J., Davies, M., & Gioia, P. (1993). The Diagnostic Interview Schedule for Children-Revised Version (DISC-R), III: Concurrent criterion validity. *Journal of the American Academy of Child and Adolescent Psychiatry, 32*, 658-665.
- Pike, A., & Plomin, R. (1996). Importance of nonshared environmental factors for childhood and adolescent psychopathology. *Journal of the American Academy of Child and Adolescent Psychiatry, 35*, 560-570.
- Querido, J. G., Eyeberg, S. M., & Boggs, S. R. (2001). Revisiting the accuracy hypothesis in families in young children with conduct problems. *Journal of Clinical Child Psychology, 30*, 253-261.
- Radke-Yarrow, M. (1998). *Children of depressed mothers*. New York: Cambridge University Press.
- Radke-Yarrow, M., Zahn-Waxler, C., Richardson, D. T., Susman, A., &

- Martinez, P. (1994). Caring behaviour in children of clinically depressed and well mothers. *Child Development, 65*, 1405-1414.
- Radloff, L. S. (1977). The CES-D scale: A self-report depression scale for research in the general population. *Applied Psychological Measurement, 1*, 385-401.
- Reid, J. B., & Patterson, G. R. (1991). Early prevention and intervention with conduct problems: A social interaction model for the integration of research and practice. In G. Stoner, M. R. Shinn, & H. M. Walker (Eds.), *Interventions for achievement and behaviour problems* (pp. 715-739). Silver Spring, MD: National Association of School Psychologists.
- Richters, J. E. (1992). Depressed mothers as informants about their children: A critical review of the evidence for distortion. *Psychological Bulletin, 112*, 485-499.
- Rogosa, D. (1980). A critique of cross-lagged correlation. *Psychological Bulletin, 88*, 245-258.
- Rogosa, D. (1988). Myths about longitudinal research. In M. Gottman (Ed.), *The analysis of change* (pp. 3-66). Mahwah, NJ: Lawrence Erlbaum.
- Rutter, M. (1990). Commentary: Some focus and process considerations regarding effects of parental depression on children. *Developmental Psychology, 26*, 60-67.
- Rutter, M. (1997). Implications of genetic research for child psychiatry. *Canadian Journal of Psychiatry, 42*, 561-562.
- Rutter, M. (1989). Isle of Wight revisited: Twenty-five years of child psychiatric

- epidemiology. *Journal of the American Academy of Child and Adolescent Psychiatry*, 28, 633-653.
- Rutter, M., Cox, A., Tupling, C., Berger, M., & Yule, W. (1975). Attainment and adjustment in two geographical areas: I. The prevalence of psychiatric disorder. *British Journal of Psychiatry*, 126, 493-509.
- Rutter, M., Pickles, A., Murray, R., & Eaves, L. (2001). Testing hypotheses on specific environmental causal effects on behaviour. *Psychological Bulletin*, 127, 291-324.
- Rutter, M., & Quinton, D. (1984). Parental psychiatric disorder: Effects on children. *Psychological Medicine*, 14, 853-880.
- Rutter, M., Tizard, J., Yule, W., Graham, P., & Whitmore, K., (1976). Isle of Wright Studies 1964-1974. *Psychological Medicine* 6, 313-32.
- Sawyer, M. G., Streiner, D. L., & Baghurst, P. (1998). The influence of distress on mothers' and fathers' reports of childhood emotional and behavioral problems. *Journal of Abnormal Child Psychology*, 26, 407-414.
- Shacham, S. (1983). A shortened version of the Profile of Mood States. *Journal of Personality Assessment*, 47, 305-306.
- Schwab-Stone, M., Fisher, P., Piacentini, J., Shaffer, D., Davies, M., & Briggs, M. (1993). The Diagnostic Interview Schedule for Children-Revised Version (DISC-R), II: Test-re-test reliability. *Journal of the American Academy of Child and Adolescent Psychiatry*, 32, 651-657.
- Shaffer, D., Fisher, P., Dulcan, M. K., Davies, M., Piacentini, J., Schwab-Stone, M. E., Lahey, B. B., Bourdon, K., Jensen, P., Bird, H. R., Canino, G., &

- Regier, D. A. (1996). The NIMH Diagnostic Interview Schedule for Children Version 2.3 (DISC-2.3): Description, acceptability, prevalence rates, and performance in the MECA study. *Journal of the American Academy of Child and Adolescent Psychiatry, 35*, 865-877.
- Shaffer, D., Fisher, P., Lucas, C., & the NIMH DISC Editorial Board (1998). *Diagnostic Interview Schedule for Children, Version IV*. New York: Columbia University, Division of Psychiatry.
- Shaffer, D., Schwab-Stone, M., Fisher, P., Cohen, P., Piacentini, J., Davies, M., Conners, C. K., & Regier, D. (1993), The Diagnostic Interview Schedule for Children-Revised Version (DISC-R), I: Preparation, field testing, interrater reliability, and acceptability. *Journal of the American Academy of Child and Adolescent Psychiatry, 32*, 643-650.
- Sexson, S. B., Glanville, D. N., & Kaslow, N. J. (2001). Attachment and depression: Implications for family therapy. *Child and Adolescent Psychiatric Clinics of North America, 10*, 465-471.
- Sharp, D., Hay, D. F., Pawlby, S., Schmucker, G., Allen, H., & Kumar, R. (1995). The impact of postnatal depression on boys' intellectual development. *Journal of Child Psychology and Psychiatry, 36*, 1315-1336.
- Shelton, T. L., Barkley, R. A., Crosswait, C., Moorehouse, M., Fletcher, K., Barrett, S., Jenkins, L., & Metevia, L. (1998). Psychiatric and psychological morbidity as a function of adaptive disability in preschool children with aggressive and hyperactive-impulsive inattentive behaviour.

Journal of Abnormal Child Psychology, 26, 475-494.

- Sher, K. J., & Wood, P. K. (1997). Methodological issues in conducting prospective research on alcohol-related behaviour: A report from the field. In K. J. Bryant, M. Windle, & S. G. West (Eds.), *The science of prevention: Methodological advances from alcohol and substance abuse research* (pp. 3-41). Washington, DC: American Psychological Association Press.
- Silberg, J., Pickles, A., Rutter, M., Hewitt, J., Simonoff, E., Maes, H., Carbonneau, R., Murrele, L., Foley, D., & Eaves, L. (1999). The influence of genetic factors and life stress on depression among adolescent girls. *Archives of General Psychiatry*, 56, 225-232.
- Silberg, J., & Rutter, M. (2002). Nature-nurture interplay in the risk associated with parental depression. In S. H. Goodman & I. H. Gotlib (Eds.), *Children of depressed parents: Mechanisms of risk and implications for treatment* (pp. 13-36). Washington, DC: American Psychological Association.
- Silverstein, B. (2002). Gender differences in the prevalence of somatic versus pure depression: A replication. *American Journal of Psychiatry*, 159, 1051-1052.
- Soliday, E., Moore, K. J., & Lande, M. B. (2002). Daily reports and pooled time series analysis: Pediatric psychology applications. *Journal of Pediatric Psychology*, 27, 67-76.
- Snyder, J. (1991). Discipline as a mediator of the impact of maternal stress and

mood on child conduct problems. *Development and Psychopathology*, 3, 263-276.

Statistics Canada (1996). *National Longitudinal Survey of Children and Youth: User's handbook and microdata guide*. Microdata documentation: 89M0015GPE. Ottawa, Canada: Special Surveys Division, Statistics Canada.

Statistics Canada (1999). *Information about the National Population Health Survey*. Microdata documentation: 82F0068XIE. Ottawa, Canada: Author.

Statistics Canada (2002). *Guide to the Labour Force Survey*. Catalogue No. 71-543-GIE. Ottawa, Canada: Author.

Stein, A., Gath, D. H., Bucher, J., Bond, A., Day, A., & Cooper, P. J. (1991). The relationship between postnatal depression and mother-child interaction. *British Journal of Psychiatry*, 158, 46-52.

Sullivan, P. F., Neale, M. C., & Kendler, K. S. (2000). Genetic epidemiology of major depression: Review and meta-analysis. *American Journal of Psychiatry*, 157, 1552-1562.

Taylor, T. K., Schmidt, F., Pepler, D., & Hodgins, C. (1998). A comparison of eclectic treatment with Webster-Stratton's parents and children series in a children's mental health center: A randomized controlled trial. *Behavior Therapy*, 29, 221-240.

Teti, D. M., & Gelfand, D. M. (1991). Behavioral competence among mothers of infants in the first year: The mediational role of maternal self-efficacy. *Child Development*, 62, 918-929.

- Teti, D. M., Gelfand, D. M., Messinger, D. S., & Isabella, R. (1995). Maternal depression and the quality of early attachment: An examination of infants, preschoolers, and their mothers. *Developmental Psychology, 31*, 364-376.
- Thapar, A., & McGuffin, P. (1994). A twin study of depressive symptoms in childhood. *British Journal of Psychiatry, 165*, 259-265.
- Tsuang, M. T. (1978). Genetic counseling for psychiatric patients and their families. *American Journal of Psychiatry, 135*, 1465-1475.
- Tsuang, M., T., & Faraone, S. V. (1990). *The genetics of mood disorders*. Baltimore, MD: Johns Hopkins University Press.
- van Ijzendoorn, M. H., Juffer, F., & Duyvesteyn, M. G. (1995). Breaking the intergenerational cycle of insecure attachment: A review of the effects of attachment-based interventions on maternal sensitivity and infant security. *Journal of Child Psychology and Psychiatry, 36*, 225-248.
- van Ijzendoorn, M. H., Schuengel, C., & Bakermans-Kranenburg, M. J. (1999). Disorganized attachment in early childhood: Meta-analysis of precursors, concomitants, and sequelae. *Development and Psychopathology, 11*, 225-249.
- Warner, V., Weissman, M. M., Mufson, L., & Wickramaratne, P. J. (1999). Grandparents, parents and grandchildren at high risk for depression: A three-generation study. *Journal of the American Academy of Child and Adolescent Psychiatry, 38*, 289-296.
- Webster-Stratton, C. (1981). Modification of mothers' behaviours and attitudes

- through videotape modeling group discussion program. *Behavior Therapy*, 12, 634-642.
- Webster-Stratton, C., & Hammond, M. (1988). Maternal depression and its relationship to life stress, perceptions of child behaviour problems, parenting behaviours, and child conduct problems. *Journal of Abnormal Child Psychology*, 16, 299-315.
- Webster-Stratton, C., & Hammond, M. (1999). Marital conflict management skills, parenting style, and early-onset conduct problems: Processes and pathways. *Journal of Child Psychology and Psychiatry*, 40, 917-927.
- Weissman, M. M., John, K., Merikangas, K. R., Prusoff, B. A., Wickramaratne, P., Gammon, D., Angold, A., & Warner, V. (1986). Depressed parents and their children: General health, social, and psychiatric problems. *American Journal of Diseases of Childhood*, 140, 801-805.
- Weissman, M. M., Leaf, P. J., Tischler, G. L., Blazer, D. G., Karno, M., Bruce, M. L., & Florio, L. P. (1988). Affective disorders in five United States communities. *Psychological Medicine*, 18, 141-153.
- Weissman, M. M., Prusoff, B. A., Gammon, G. D., Merikangas, K. R., Leckman, J. F., & Kidd, K. K. (1984). Psychopathology in the children (ages 6-18) of depressed and normal parents. *Journal of the American Academy of Child Psychiatry*, 23, 78-84.
- Wells-Parker, E., Miller, D. I., & Topping, S. (1990). Development of control of outcome scales and self-efficacy scales for women in four life roles. *Journal of Personality Assessment*, 54, 564-575.

- World Health Organisation. (1992). *International Statistical Classification of Diseases and Related Health Problems* (10th ed.). Geneva, Switzerland: Author.
- Zubrick, S. R., Silburn, S. R., Burton, P., & Blair, E. (1995). Mental health disorders in children and young people: Scope, cause and prevention. *Australian and New Zealand Journal of Psychiatry*, 34, 570-578.