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The Role of Maternal Psychological Distress on Neurobehavioural Disinhibition among Preschoolers Exposed Prenatally to Methamphetamine

Amy Rosso McDonald

A thesis submitted in partial fulfilment of the requirements for the degree of Doctor of Clinical Psychology, The University of Auckland, 2016
Methamphetamine (MA) use in pregnancy has been linked to deficits in fetal growth and newborn neurobehavioural functioning, and later problems in cognitive, psychomotor and behavioural functioning. Beyond the direct biological impact of MA exposure, maternal drug use may affect the child indirectly through the negative lifestyle and psychological factors associated with drug use. Of particular concern is the evidence that links drug dependence and maternal psychological distress with negative parenting behaviours and poor developmental outcomes for children. The specific aim of this investigation was to examine the associations between prenatal MA exposure and indicators of maternal behavioural health (postnatal drug use and psychological functioning) on child neurobehavioural disinhibition measures at 4.5 years. The sample in this longitudinal investigation included 83 children with prenatal MA exposure and 97 matched comparison children. Neurobehavioural disinhibition, involving lack of behavioural control and poor emotion regulation, was assessed using a Gift Delay of Gratification task and maternal report on the Strengths and Difficulties Questionnaire (SDQ) and the Brief Rating Inventory of Executive Functioning - Pre-school Edition (BRIEF-P). Hierarchical linear modelling was used to estimate suspected causal relationships between indicators of maternal behavioural health at four time points across the 4.5 year follow-up, according to self-report on the Substance Use Inventory (SUI) and Brief Symptom Inventory (BSI) scores. Prenatal MA exposure was not associated with any measure of child neurobehavioural disinhibition at 4.5 years. Postnatal drug use and psychological distress were associated with all measures of child neurobehavioural disinhibition. However, when considered within a combined model, only maternal psychological functioning remained significant. In light of the significant role that maternal functioning plays in the development of challenging behaviours, it is clear that treatment that addresses both mental health and substance use behaviours in mothers who use methamphetamine are warranted.
This thesis is dedicated to the mothers, children and whānau of Aotearoa

Take care of what they hear,
Take care of what they see,
Take care of what they feel,
For how the children grow,
So will be the shape of Aotearoa

- Dame Whina Cooper
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Amy Rosso McDonald - April 2016
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CHAPTER ONE: INTRODUCTION

Rationale

Substance abuse among women of childbearing age remains a significant concern internationally. According to the 2013 National Survey of Drug Use and Health (NSDUH) in the United States, 5.2% of pregnant and 9.7% of non-pregnant women between the ages of 15-44 years reported past-month illicit drug use (Substance Abuse and Mental Health Services Administration, 2014). Data on the number of children in New Zealand affected by problematic parental substance use is limited and incomplete. Evidence suggests that between 780,000 and 1.3 million children in the United Kingdom are affected by problematic maternal alcohol and between 250,000 and 350,000 children are affected by problematic maternal drug use (Advisory Council on the Misuse of Drugs, 2003). In Australia, an estimated 10–13% of children are affected by maternal alcohol or other drug use (Battams, Roche, Duvnjak, Trifonoff, & Bywood, 2010) and international studies estimate that 10% of children worldwide are exposed to alcohol and other drug ‘misuse’ (Dawe et al., 2007).

Methamphetamine (MA) use in pregnancy is a complex and increasing public health problem. New Zealand has one of the highest rates of MA use globally, and recent national trends suggest an increase of use among females of reproductive age. Because MA can readily cross the placenta, usage during pregnancy has the potential to cause long-term problems for the developing child. Prenatal MA exposure has been linked to deficits in fetal growth and newborn neurobehavioural patterns of decreased arousal, increased stress, and poor quality of movement. Alterations in neural growth and connectivity and consequent neurobehavioral deficits may increase the risk for later problems in cognitive, emotional and behavioural functioning.

While biological sequelae have received much attention, studies on the possible cognitive, language, motor, emotional, and behavioural functioning of young children prenatally exposed to MA have been less prevalent. A growing consensus suggests that these risks might actually arise from multiple factors that are cumulative in nature (Nair, Schuler, Black, Kettinger, & Harrington, 2003; Tronick & Beeghly, 1999). It is now recognised that searching for a singular effect or the location of effect is misdirected. Most substance users are poly-drug users and maternal addiction occurs in the context of multiple environmental risk factors. Although it is likely that substances have a specific and dose-related effect independent of other factors; it is also feasible to consider the impact of maternal addiction using a systemic, regulatory model of development. Transactional formulations of risk development for this population suggest, indeed, that it is not prenatal exposure to drugs, per se, but also the early child rearing environment that is of significance to children’s developmental outcomes (Hans, 2002; Sameroff & Chandler, 1990).

There is a general understanding that substance use tends to decrease in pregnancy but little prospective data is available on the rates of abstinence and relapse for MA use. More general prenatal substance use studies have found that children who remain in the care of addicted parents are more likely to display behavioural and emotional disturbances than those whose parents quit abusing the
drug. Of particular concern is that mothers who continue to abuse MA after giving birth often suffer from comorbid psychological conditions. These women report having less control over their children and see their children as being more aggressive, which has been linked to negative parenting behaviours and poor developmental outcomes.

One area of behavioural development that warrants attention is neurobehavioural disinhibition, a construct of psychological dysregulation involving lack of behaviour control and poor emotion regulation. It encompasses problems of excessive risk taking, impulsivity, aggression, irritability, difficult temperament, attention deficit-hyperactivity disorder, and impaired executive function. Children and adolescents exhibiting these disinhibitory symptoms can have long-term challenges including academic difficulties, delinquency, mental health problems, and substance abuse. Limited amounts of research have examined aspects of neurobehavioural disinhibition, and with mixed results. These studies indicate that prenatal exposure alone is not sufficient to account for the outcomes of exposed children. They have found it is likely that psychosocial factors related to their environment, such as parental drug dependence, maternal psychological distress, high changes in caregivers, stress and violence all play a significant role in their behavioural outcomes. Despite the clear understanding that co-occurring maternal substance use and psychological distress intensify the behavioural problems associated with prenatal drug exposure, very few studies have looked at these combined effects following pregnancy.

My interest in this research arose from experience working at the World Health Organization (WHO) in the Department of Reproductive Health and Research, after I completed my degree in Masters of Public Health (MPH). During this time I became aware of a more holistic understanding of developmental health issues and developed an understanding of the importance of addressing psychological needs in context. Throughout my clinical training I came across children with complex presentations, often including comorbid neurodevelopmental delays and physical and mental health concerns. This sparked an interest in developmental psychology, infant mental health, and providing specialist psychological support for young children and their families who are at high-risk of or exhibit substantial mental health issues.

The potential effect of methamphetamine use in pregnancy on behavioural problems may have substantial public health implications, because behavioural dysregulation is quite robust and these problems tend to persist over time and predict later psychopathology and criminal behaviour that places tremendous burdens on society. The ability to identify specific neurobehavioural traits in children as early as pre-school age could lead to the development of preventative intervention programs.

Since the prenatal period is a transitional one when old behaviours are abandoned and new ones are established, it is a critical time to intervene (Fleming, Lund, Wilton, Landry, & Scheets, 2008). Understanding this time as one of unique vulnerability may also be critical to improving abstinence, particularly among those with alcohol or drug use problems (Rutherford, Williams, Moy, Mayes, & Johns, 2011). Health professionals who are routinely providing healthcare to women of childbearing age are uniquely positioned to deliver important information about the health risks around the use of alcohol, tobacco and other recreational or psychoactive drugs. Unfortunately, research to date suggests that while a large proportion of health professionals routinely ask about the use of alcohol (78%) and tobacco (88%), a much smaller proportion routinely ask about the use of other psychoactive drugs such as cannabis (52%), opiates (34%), or methamphetamine (33%) (Woulde, 2009). A number of obstacles
may prevent healthcare professionals from discussing substance use with their patients, including: a lack of knowledge about the effects of these substances on the mother and her developing child and insufficient training to adequately assess the risk of using alcohol and/or other drugs (Jones & Kaltenbach, 2013; Wouldes, 2009). Therefore, increasing the awareness of the prevalence and of the effect of methamphetamine use has the potential to improve identification of women who are using methamphetamine in order to provide adequate care and early referral for treatment to prevent more deleterious developmental outcomes for children.

While there has been tremendous progress in our understanding of prenatal methamphetamine exposure and its possible immediate and long-term effects in developmental psychopathology (Papachristou, Frangou, & Reichenberg, 2013), there is still much potential for further advancement. Ultimately, more research needs to be conducted to see if symptoms previously discovered are due to actual drug exposure in utero or to maternal behavioural health in the postnatal environment. Exploring these differences will improve our understanding of the impact of prenatal MA on child outcomes, the psychosocial issues surrounding mothers who continue to use MA beyond their pregnancy, and the unique ways in which comorbidity may increase risk of behavioural problems in children. This could inform risk factor research and designs of targeted interventions to prevent or reduce maternal substance use-related problems and consequences.

The following chapter will present a review of the relevant literature on ways in which prenatal methamphetamine exposure may act in concert with other risk factors in setting the stage for the development of poor neurobehavioural outcomes. First the problem of methamphetamine use among pregnant women will be reviewed to give the reader insight into the complexity of methamphetamine use among this population group. Next the impact of prenatal methamphetamine exposure on child developmental outcomes is considered, including what evidence is currently known on short-term and long-term effects. The importance of psychosocial factors influencing these outcomes is then examined before a review on the shortcomings of current evidence. Finally, an exploration of neurobehavioural disinhibition and the importance of this psychological construct in emotional and behavioural development among pre-schoolers are discussed.

**Methods of Literature Review**

This literature review first provides a brief history of methamphetamine use and harm before examining prenatal drug exposure research and methamphetamine-specific studies to answer several key questions:

1) What is currently known about the likely pattern of substance use among pregnant women or women of childbearing age?
2) What are the factors that contribute to methamphetamine abuse?
3) What are the cellular and molecular pathways by which prenatal drug exposure may influence structural and functional brain development?
4) What current studies are available investigating outcomes of exposed individuals across various areas of functioning (neurobiology, physical growth, cognitive development, behaviour and psychopathology)?
5) What psychosocial and environmental risk factors are deleterious outcomes of prenatal MA exposure?
6) What conceptual models are helpful in understanding the prevalence, development and trajectory of negative developmental and social outcomes, particularly as they relate to substance use and addiction? And
7) What are the current limitations in understanding and potential avenues for future research?
CHAPTER TWO: METHAMPHETAMINE

Methamphetamine Use and Statistics

History & Global Trends

Methamphetamine was first synthesized by a Japanese chemist in 1893 (Suwaki, Fukui, & Konuma, 1997), but did not become widely used until the 1940s. Largely unaware of the risks, it was used by physicians for a diverse number of clinical applications, and also utilised by military personnel during World War II to combat fatigue and increase performance (Karch & Drummer, 2001). Following the end of the war, surplus military stocks flooded the Japanese market, culminating in an epidemic of abuse. It was eventually banned in 1951, following the passage of the stimulant control act. As various pharmaceutical companies withdrew methamphetamine from the domestic market, a worldwide illegal trade began. Global use and production of methamphetamine was generally limited to the West coast of the United States of America until the latter part of the 20th century (Meredith, Jaffe, Ang-Lee, & Saxon, 2005). Since that time, methamphetamine has become one of the most significant drug problems worldwide (Kozel, Douglas, & McKetin, 2007). It is estimated that over 35 million people internationally abuse meth/amphetamines, compared to 18.8 million abusers of cocaine and less than 10 million regular users of opiates (United Nations Office on Drug Control and Crime Prevention, 2015). This figure is considered to be a large underestimate given the stigma and punitive consequences associated with illicit drug use. Historically, higher-use regions have included Asia, the United States, Scandinavia and Oceania.

Methamphetamine Use in New Zealand

Methamphetamine use emerged in New Zealand in the mid-1990s and quickly established a strong foothold. As with other affected nations, the popularity of the drug grew rapidly, moving from the third most common illicit drug in 1998 to second behind marijuana in 2001 (Wilkins, Prasad, Wong, & Rychert, 2014). The first clandestine drug laboratory was discovered in New Zealand in 1996, and annual counts subsequently increased from 5 in 1999 to 211 in 2006 (Newton, 2007). The drug first appeared in New Zealand as speed; a milder version with only about 3-5% purity. These days, methamphetamine normally ranges from 60 to almost 80% pure (Policy Advisory Group, 2015) which makes it highly addictive.

The recent Drug Use in New Zealand survey (Mason, Hewitt, & Stefanogiannis, 2010) estimated the prevalence of having ever used meth/amphetamines was 7.2% (6.2–8.1) among the total population aged 16–64 years, which equates to approximately 189,500 people in New Zealand. The prevalence of having used meth/amphetamines in the past year (2007/8) was 2.1%. This figure is significantly higher than the estimates of other nations: Canada’s annual prevalence of methamphetamine use is 1.5%, in the US it is 1.3%, and in the United Kingdom 1.0 – 1.4% of 16 to 64 year olds had used meth/amphetamines (United Nations Office on Drugs & Crime, 2010).

Community Alcohol and Drug Services (CADs) in Auckland found that the proportion of people receiving a methamphetamine-use disorder diagnosis in 2008/9 was 9-10% and alcohol and drug helplines estimate that three out of every five calls to the helpline are related to concerns regarding
methamphetamine use (Matua Raki, 2010). The Illicit Drug Monitoring System (IDMS) is conducted annually to provide information on the trends in drug use and drug markets in New Zealand. In the latest findings, the current availability of MA was reported to be ‘easy’, and frequent drug users reported increasing availability of MA in the market from 2006 to 2013 (Wilkins et al., 2014).

Of growing concern are the changes observed in population prevalence use, which indicates that the methamphetamine market has shifted to an endemic phase where use is expected to remain high among sub-populations such as victims of physical and sexual abuse, those suffering from mental health concerns, and those from marginalised social and ethnic groups (Wilkins et al., 2014). A particularly vulnerable age group appears to be young adults. The prevalence of meth/amphetamine use in New Zealand peaks among 25–34 year olds for both men and women (Mason et al., 2010).

**Methamphetamine Use in Pregnancy**

Methamphetamine use has largely been associated with young males, but there is growing concern regarding the popularity of methamphetamine among women, particularly of reproductive age. Unlike many other illegal drugs, MA is a drug that appeals equally to men and women. Among treatment samples of MA users, data indicate that nearly as many women enter treatment for MA dependency as men (Brecht, Huang, Evans, & Hser, 2008; Hser, Evans, & Huang, 2005; Rawson et al., 2004) and female youth are more likely to use MA than their male youth counterparts (Gonzales, Ang, McCann, & Rawson, 2008; Gonzales, Mooney, & Rawson, 2010). According to the Department of Health and Human Services Treatment Episode Data Set, women currently make up to 47% of hospital admissions for methamphetamine (Substance Abuse and Mental Health Services Administration, 2007).

The upward trend of methamphetamine use by women worldwide is partly due to the anorectic effect that helps women achieve weight control (Chung, 1998; Hsu, Lin, & Tsay, 2014). The use of methamphetamine as a party drug also contributes to the problem of unplanned pregnancy, particularly in teenagers (Brecht, O’Brien, Von Mayrhauser, & Anglin, 2004). An analysis of 15,214 students in grades 9–12 in the Youth Risk Behavior Survey 2003 found that lifetime methamphetamine use was significantly associated with being sexually active over the past 3 months, having two or more partners, and ever being pregnant or getting someone pregnant. These risks show a dose-response relationship, with increasing frequency of methamphetamine use being significantly associated with increasingly risky sexual encounters (Zapata, Hillis, Marchbanks, Curtis, & Lowry, 2008).

Data regarding methamphetamine use during pregnancy is often hard to capture due to social stigma and judgement associated with drug use. Prevalence estimates of substance use during pregnancy can also vary widely depending on the population sampled, the exposure interval of pregnancy covered and the reference period used in prevalence measures. However, there is general consistency suggesting that prenatal methamphetamine use has increased over the past 20 years, with recent estimates suggesting that 1% of the general population or approximately 19,000 pregnant women use methamphetamine (Substance Abuse and Mental Health Services Administration, 2014). One study within a non-clinical population found that approximately 5.2% of women reported using MA at least once during their pregnancy (Arria et al., 2006). In the United States, data obtained from the Treatment Episode Data Set found that the prevalence of methamphetamine use among pregnant women admitted
for substance abuse treatment rose from 9% in 1994 to 24% in 2004 (Terplan, Smith, Kozloski, & Pollack, 2009). This proportion was higher than methamphetamine admission among both non-pregnant women (12%) and men (7%); making methamphetamine the only illicit drug that does not have a lower use rate for pregnant women than for non-pregnant women.

In New Zealand, the widespread use of methamphetamine in pregnancy has been reflected in the dramatic increase of pregnant women referred to the Alcohol Drug and Pregnancy Team (ADAPT) at National Women’s Hospital. In 2001, 10% (6/60) of the total ADAPT referrals were due to methamphetamine use and associated problems. This escalated to 59% (34/58) in 2003 (Wouldes, LaGasse, Sheridan, & Lester, 2004). Further anecdotal reports from other antenatal departments of hospitals in the Auckland region and community midwives suggest that a much larger number of women are using or have used methamphetamine during their current pregnancy that have not come to the attention of ADAPT. Unfortunately, many pregnant women or women with young children do not enter formal drug treatment services or seek appropriate antenatal care because of the pervasive fear of not being able to take care of or keep their children, and they are afraid of the stigma from health care providers and authorities.

Concern for the impact of substances on their baby leads some women to moderate their use of drugs and alcohol during pregnancy. In a recent prospective study on abstinence and relapse, it was found that among women with substance use prior to pregnancy, 96% of women with heavy drinking, 78% of women with marijuana use, 73% of women with cocaine use, and 32% of cigarette smokers achieved abstinence in pregnancy. These findings are consistent with the National Pregnancy & Health Survey by the National Institute on Drug Abuse in the US, who reported reductions in the prevalence rates of alcohol, cigarette, marijuana, and cocaine use by the second trimester, compared with use in the 3 months before pregnancy (NHSDA, 2009). Analyses from the 1994 to 1995 National Household Surveys on Drug Abuse (NHSDA) also showed lower prevalence rates of past 30-day use of alcohol, cigarettes and any illicit drugs including marijuana among pregnant women (i.e., those who reported being pregnant at the time of the interview) than among non-pregnant counterparts.

A few studies have attempted to examine the pattern of methamphetamine use during pregnancy (Arria et al., 2006; Derauf, Katz, Frank, Grandinetti, & Easa, 2003). The prevalence of methamphetamine use among pregnant women in the population of these studies decreased over the three trimesters of pregnancy (84.3% compared with 56.0% compared with 42.4%), and decreased frequency was observed among users from the first trimester to the third (3.1 compared with 2.4 compared with 1.5 days per week) (Derauf et al., 2003). Overall, however, 55% of the women that used methamphetamine in their first trimester maintained stable use of methamphetamine throughout their pregnancy, whereas only a third decreased their use. Unfortunately, their alcohol intake increased as a measure to self-medicate withdrawal symptoms (Derauf et al., 2003; Wouldes et al., 2013). Despite taking immediate action to remediate the risk of harm, there is evidence to suggest that any amount of methamphetamine use during pregnancy can have negative ramifications, both physically and socially, on the both the mother and child.
Methamphetamine-related Harm to User

Methamphetamine has a unique chemical structure that makes it more potent than its parent compound, amphetamine (Lake & Quirk, 1984). Often compared to another illicit stimulant, cocaine, the ‘high’ produced by methamphetamine is felt sooner and lasts longer (Meredith et al., 2005). Methamphetamine works by increasing the levels of brain chemicals, known as neurotransmitters that include norepinephrine, dopamine and serotonin (Azzaro, Ziance, & Rutledge, 1974; Harris et al., 2006; Taylor & Ho, 1978). Neurons communicate with one another by releasing these neurotransmitters into the synaptic space, essentially ‘activating’ the neighbouring neurons. Methamphetamine floods the brain with neurotransmitters in two ways: first, it acts as a potent dopamine and norepinephrine-releasing agent; second, it inhibits the reuptake of these chemicals into the pre-synaptic neuron once released. This results in an increase in the concentration of these neurotransmitters in the synaptic space. Dopamine and serotonin are thought to be responsible for the euphoric rush methamphetamine users experience immediately after ingestion (Homer et al., 2008). Additionally, dopamine is associated with motor coordination, motivation, hormonal release, and the reward pathway, functions that are consistent with the behavioural changes observed in abusers of methamphetamine (McPherson, 2008). The potential for abuse of methamphetamine is thought to be primarily due to its euphorigenic effects and its psychomotor stimulating properties.

Unfortunately, the associated effects of methamphetamine-altered brain chemistry may be permanent. Chronic methamphetamine use has been shown to cause localized brain damage involving structural changes to both grey and white matter (Homer et al., 2008). The resulting impairment of neurocognitive abilities, including decision-making and social functioning, which in turn affects the abusers interactions with her family, community, and the environment (Meredith et al., 2005).

When taken recreationally, methamphetamine can be smoked, injected, snorted, or swallowed. In New Zealand, the preferred route is smoking (Wilkins et al., 2014). This method has been shown to be more addictive than snorting or swallowing the drug, and produces a unique chemical compound during breakdown, trans-phenylpropene that is thought to be carcinogenic (McKetin, Kelly, & McLaren, 2006). The high is felt in a matter of seconds if smoked or injected, and lasts up to 8 hours (McPherson, 2008). At low doses, the effects are mainly neurologic, including increased alertness, enhanced mood, increased and sustained attention, decreased appetite, increased sexuality, reduced fatigue, and enhanced endurance (Chomchai & Chomchai, 2015). However, the pleasurable effects of methamphetamine diminish with chronic use. Frustrated addicts must resort to taking higher and higher doses to get the same effect. This heightened tolerance occurs rapidly and daily doses have been known to increase from 5 mg to 1000 mg per day within a single year (McPherson, 2008).

Following the administration of methamphetamine, the immediate physiological response includes an increased heart rate, rapid breathing, hypertension, pupil dilation, decreased appetite, and increased body temperature (Gawin & Ellinwood Jr, 1988). High doses of methamphetamine have been known to produce much more severe effects. US hospital-based case studies of acute methamphetamine toxicity events have attributed chest pain, muscle damage, seizures, psychosis, coma, stroke, heart attack, pulmonary oedema, and multi-system organ failure to the drug (Albertson, Derlet, & Van Hoozen, 1999; Derlet, Rice, Horowitz, & Lord, 1989; Gonzales et al., 2010; Hong, Matsuyama, & Nur, 1991; Perez, Arsura, & Strategos, 1999). Chronic use has its own set of risks:
cardiomyopathy, mood disturbances, memory loss, extreme weight loss, and severe dental problems (Darke et al., 2008).

Perhaps the most troubling health effect associated with methamphetamine abuse is psychosis. Delusions and hallucinations resembling those observed in paranoid schizophrenia have been documented in methamphetamine users since the 1950s (Yui, Ikemoto, Ishiguro, & Goto, 2000). The psychosis can take two forms: transient states that subside within a few days, or prolonged episodes in which symptoms can persist for months (Iwanami et al., 1994). Furthermore, methamphetamine users are at risk for relapses of psychotic symptoms that occur months after drug discontinuation. McKetin and colleagues (2006) found Australian methamphetamine addicts were 11 times more likely to suffer from at least one psychotic symptom compared to the general public. Abusers may also experience euphoria; increased energy, alertness, and sexual urges; and decreased fatigue and appetite. They also experience paranoia, depression, irritability, hallucinations, mood swings, and violent behaviour (Maxwell, 2005). Withdrawal too, has its own associated dangers. Discontinuing methamphetamine use may result in severe depression, anxiety, fatigue and suicidal ideation (Gawin & Ellinwood Jr, 1988; Newton, Kalechstein, Duran, Vansluis, & Ling, 2004).
CHAPTER THREE: PRENATAL METHAMPHETAMINE EXPOSURE

Introduction

One of the earliest studies to examine the impact of prenatal methamphetamine exposure on child developmental outcomes was a longitudinal study conducted in Sweden by Billing et al (1988) who followed children from birth to 14 years of age. They found that longer MA exposure was associated with academic and mild physical delays, as well as more aggressive behaviours and poorer peer relationships later in childhood. Despite the advancement in research in the area, this study was beset with methodological problems and potential confounders. They had no formal control group, did not account for exposures to other substances, and only 22% of the children in the sample remained with their biological mothers (Cernerud, Eriksson, Jonsson, Steneroth, & Zetterstrom, 1996). Recent, more reliable findings from the National Institute of Health longitudinal Infant Development Environment and Lifestyle study (IDEAL) took many of the previous study limitations into consideration and accounted for them in a multi-site, longitudinal, prospective analysis of the effects of prenatal MA on children. Most of the findings presented below summarises the literature published to-date from the IDEAL study.

Fetal and Newborn Outcomes

Evidence from preclinical rat models has demonstrated a range of physical, motor, neurotransmitter and behavioural effects in MA exposed offspring. These include increased maternal and offspring fetal mortality, retinal eye defects (Acuff-Smith, George, Lorens, & Vorhees, 1992; Acuff-Smith, Schilling, Fisher, & Vorhees, 1996; Yamamoto, Yamamoto, Fukui, & Kurishita, 1992), cleft palate and rib formations (Yamamoto et al., 1992) decreased rate of physical growth (Acuff-Smith et al., 1996).

Human studies have found that methamphetamine use during pregnancy has been associated with increased incidence of cardiac defects, cleft lip, biliary atresia, stillbirth, cerebral haemorrhage, Mongolian spots, systolic murmur and undescended testes (Plessinger, 1998) as well as adverse somatic growth effects (Oro & Dixon, 1987). These initial reports were limited by reliance on hospital records, retrospective analysis, small sample size, and lack of adjustment for confounding factors. Further studies have found that prenatal MA exposure has been associated with increased prematurity, lower birth weight and being small for gestational age (Nguyen et al., 2010; Smith et al., 2006). Infants exposed to prenatal methamphetamine were also found to be at greater risk for having a smaller head circumference and were shorter in birth length than unexposed infants at birth (Shah et al., 2012). These trends continue through the first three years of life. Exposed children remained significantly shorter than unexposed children (Zabaneh et al., 2012). There is no evidence to suggest the incidence of facial dysmorphism, skeletal, cardiac or respiratory problems after birth associated with prenatal MA (Shah et al., 2012). Furthermore, no central nervous system signs of drug withdrawal have been documented, and no incidence of abnormal head sonograms reported (Smith et al., 2015).

Newborn neurobehavioral effects found in prenatal MA are considered reminiscent of cocaine-exposed infants showing effects on arousal and physiological stress; where there is a dose-response relationship between the amount of amphetamine metabolites in meconium and newborn
neurobehaviour (Smith et al., 2008). Exposed children are more likely to exhibit increased physiological stress than unexposed infants. High dose, but not low dose, exposure of MA in the first half of gestation has also been found to induce delays in behavioural development (McDonnell-Dowling, Donlon, & Kelly, 2014). This is thought to be due to the direct impact of MA in altering N-methyl-D-aspartate (NMDA) receptors in the hippocampus, which underlie changes in behaviour (Šlamberová et al., 2006). The neurochemical changes associated with prenatal MA exposure are linked with learning impairments (Acuff-Smith et al., 1996), behavioural deficits (Weissman & Caldecott-Hazard, 1995), arousal regulation, and attention (Mayes, 1999; Salisbury, Ponder, Padbury, & Lester, 2009), increased motor activity (Acuff-Smith et al., 1992) enhanced conditioned avoidance response (Cho, Lyu, Lee, Kim, & Chin, 1991), postural motor movements (Šlamberová, Pometlová, & Charousová, 2006) and a decreased rate of physical growth and delayed motor development (Cho et al., 1991). Heavy MA use (≥3 days per week) is significantly associated with decreased arousal, increased physiological stress and lethargy (LaGasse et al., 2011; Smith et al., 2008). MA exposed infants are also more likely to demonstrate poorer quality of movement (LaGasse et al., 2011).

Later Developing Child Outcomes

Childhood Behaviours

The evidence investigating more long-term effects on child behaviour is somewhat variable. Heavy methamphetamine use has been found to be associated with increased attention problems and being withdrawn (LaGasse et al., 2012). One study examined the internalising and externalising behaviours at 3 years, and did not find group differences based on MA exposure (Derauf et al., 2011). Instead, the authors found that decreased internalising and externalising behaviours were associated with easy temperaments. Moreover, they found that children living in high-risk environments were more likely to display internalising and externalising behaviours than those living in lower environmental risk. Another study, however, found that methamphetamine exposure was related to increased affective reactions, anxiety and depression problems at age 3 and 5 years, as well as externalizing disorders at the age of 5 (LaGasse et al., 2012; Twomey et al., 2013). Prenatal MA exposure has also been associated with subtle differences in attention processing and a greater risk for developing Attention Deficit and Hyperactivity Disorder (ADHD) (Kiblawi et al., 2013; LaGasse et al., 2012). Furthermore, while not specifically addressing the impact of prenatal MA exposure, there is evidence to suggest that the prevalence of ADHD is higher among children with methamphetamine-dependent parents compared to a control group (Parvaresh, Mazhari, & Nazari, 2015).

The lack of consensus in results when looking at childhood behaviours may be due to the failure to examine moderating and mediating variables (LaGasse et al., 1999; Tronick & Beeghly, 1999). For example, there is evidence that less responsive home environments, caregiver psychological symptoms, and parenting stress experienced by the caregiver are associated with increased child behavioural problems (Twomey et al., 2013). These findings were independent of methamphetamine exposure, highlighting the importance of interventions that address both the child and parental or primary caregiver needs in order to optimise child outcome.
Motor, Cognitive & Language Outcomes

To date, there is no evidence of an effect of MA exposure on receptive and expressive language (Derauf et al., 2011) or development in various mental tasks, including memory, early number concepts, problem-solving, generalisation, and vocalisation (Smith et al., 2011) at three years of age. Heavy MA exposure, however, was associated with decreased grasping scores at ages one and three years. There were no differences in any fine motor skills (Smith et al., 2011). Another study found that MA exposure was associated with delayed gross motor development over the first three years, but not with cognitive development (Woulde, 2014).

Prenatal MA exposure is linked with poor visual motor integration, attention and verbal and spatial memory (Chang et al., 2004; Lu et al., 2009), and spatial performance (Piper et al., 2011). Neuroimaging studies have documented an association between prenatal MA exposure and abnormal brain morphology, including reduced caudate nucleus volumes and cortical thickness in the orbital-frontal cortices, which impacts on cognitive control processes (Derauf et al., 2012b).

Animal studies suggest that the timing of MA exposure during gestation influences outcome. When neonatal rats were exposed to MA in a time period considered to be equivalent to the third trimester in humans, rat offspring had spatial learning and memory deficits (Williams et al., 2003). It has also been found that rats exposed to MA in the second half of gestation demonstrate poor cognitive function than those exposed during the first half of gestation (Hrebícková, Malinová-Sevcíková, Macúchová, Nohejlová, & Slamberová, 2014).

Executive Functioning

Research to date has found that heavy prenatal MA exposure was associated with reduced accuracy on a Stroop-like task among school-aged children, signalling subtle deficits in inhibitory control (Derauf et al., 2012a), a key executive function. The authors found, however, that caregiver psychological distress and child protection service involvement due to physical and/or sexual abuse were also associated with reduced accuracy. Another study looked at the relationship between prenatal MA, early childhood adversity and subsequent childhood neurodevelopment at age 5 and 6.5 years (Abar et al., 2013). Prenatal MA was associated with behavioural and emotional control difficulties at age 5, which was then associated with deficits in executive functioning at age 6.5. The findings also demonstrated that the effects of MA on neurodevelopment functioned primarily through adversity.

Neuroimaging studies of community-derived convenience samples (Chang et al., 2004) have identified alterations in frontal-striatal brain regions thought to be related to specific executive functions such as inhibitory control, working memory, sustained attention, and visual-motor integration (Noland et al., 2005). Of these skills, inhibitory control (the ability to resist a first impulse or to stay on task despite distraction) is considered particularly important for the development of social competence and emotional and cognitive control (Diamond, Barnett, Thomas, & Munro, 2007).

Although the literature on the effect of prenatal MA exposure in this area is sparse, findings from the cocaine literature suggest that subtle neuropsychological difficulties associated with executive functioning, a distinct grouping of higher order cognitive skills, are an important mechanism linking prenatal drug exposure with an increased likelihood of behavioural and emotional regulation problems (Mayes & Fahy, 2001; Richardson, Goldschmidt, Leech, & Willford, 2011; Singer et al., 2004; Singer et
al., 2008) and later adverse outcomes (Bridgett & Mayes, 2011). Prenatal cocaine exposure has been associated with deficits in sustained attention (Accornero et al., 2007; Ackerman, Riggins, & Black, 2010; Bandstra, Morrow, Anthony, Accornero, & Fried, 2001; Noland et al., 2005; Savage, Brodsky, Malmud, Giannetta, & Hurt, 2005), selective attention (Noland et al., 2005), slowed motor and processing speed (Schroder, Snyder, Sielski, & Mayes, 2004), inhibitory control and impulsivity (Accornero et al., 2007; Bendersky, Gambini, Lastella, Bennett, & Lewis, 2003; Carmody, Bennett, & Lewis, 2011; Savage et al., 2005), and poor outcomes on the Stroop (Rose-Jacobs et al., 2009) and Behavior Rating Inventory of Executive Function (BRIEF; Minnes et al., 2014) measures of executive function.

Covariates

From the literature, there are few studies that capture the long-term effects of prenatal methamphetamine use beyond the neonatal period. As was discussed previously, the main reason for this is that there are a variety of other risk factors that co-vary with maternal methamphetamine use, which are also related to poor outcomes. While the large population studies statistically control for many of these prenatal and postnatal confounding variables, there is still variability in what variables are used or in how these variables are defined. In the description below, the variables that are the most commonly controlled for are reviewed.

Prenatal Confounding Variables

Use of legal or illegal substances other than methamphetamine during pregnancy (tobacco, alcohol, marijuana) are commonly controlled. However, drugs included as covariates can show few or no effects (Cornelius et al., 2011) or contradictory effects (Richardson, Goldschmidt, & Willford, 2009). The effects of poly-substance use will be reviewed more thoroughly in Chapter Five.

Postnatal Confounding Variables

In addition to the importance of poly-substance use, the home environment and caregiver stress can significantly influence child outcomes. Independent of methamphetamine exposure, children with more responsive home environments to developmental and emotional needs demonstrated lower risks for internalizing and externalizing behaviour (Smith et al., 2015; Minnes et al., 2014). Further, increased psychological symptoms and parenting stress in the primary caregivers are associated with increased child behavioural problems (Smith et al., 2015). These biopsychosocial factors will be discussed in greater detail in Chapter Five.

Dose-Response

A question often asked is whether the effects of prenatal MA exposure are related to the level of MA use. There are only a few studies in which results of toxicology analysis (e.g., urine or meconium) were reported in relation to prenatal MA exposure (LaGasse et al., 2011). However, studies have used maternal interviews to derive measures of MA use patterns, such as the frequency of use per week (heavy ≥3 days per week). Studies have found that heavy MA use is related to worse outcomes,
including lower arousal and excitability (LaGasse et al., 2011), poor fine motor, attention problems and withdrawn behaviour at ages 3 and 5 years (LaGasse et al., 2012) and poor inhibitory control at 5.5 years (Derauf et al., 2012a).
Pathophysiology of the Drug

The increase of methamphetamine as a frequently abused illicit drug during pregnancy has raised questions regarding its effects on the developing fetus and child. Early in gestation, during the embryonic stage, drugs can have significant teratogenic effects, causing birth defects via a toxic effect on the embryo. As a member of a group of stimulant drugs, methamphetamine can readily pass through the placenta and the blood-brain barrier. After a single dose of methamphetamine was given to pregnant mice, levels of substance in the fetal brain were found to be similar to the levels given in the mother (Won, Bubula, McCoy, & Heller, 2001).

Methamphetamine is believed to impair brain development both directly through its effect on developing neurotransmitter systems critical to neuronal differentiation and brain structure formation, and indirectly through its effect on blood flow to the developing fetal brain. More specifically, methamphetamine has been found to block the reuptake of monoaminergic neurotransmitters, namely dopamine, norepinephrine, and serotonin. This leads to increased concentrations of these neurotransmitters in the synaptic cleft, which has the potential to disrupt the development of neuronal circuitry in the fetus (Thompson, Levitt, & Stanwood, 2009). Furthermore, vasoconstriction of cerebral blood vessels resulting from MA exposure may produce a hypoxic condition in the brain (Salisbury et al., 2009). Prenatal methamphetamine exposure may also affect widespread neuroontogenic processes, such as cell production and migration (Frost & Cadet, 2000), alter the development of the fetal stress response hypothalamic-pituitary-adrenal axis (HPA axis; Lester & Padbury, 2009), and perturb oxidative-, mitochondrial-, and glutamate-associated excitatory pathways, leading to neuronal damage (Tata & Yamamoto, 2007). While not the focus of this study, Figure 1 below provides an illustration summarising how substances, including methamphetamine affect and alter fetal development through a variety of direct and indirect mechanisms.
Figure 1. Mechanisms of action of drug abuse on the developing fetus. Drugs of abuse not only target the developing fetal brain directly, but also can exert effects through a variety of indirect ways with the mothers, including through the uterus, placenta, heart, lungs, and brain (Ross, Graham, Money, & Stanwood, 2015).

Fetal Programming and Brain

Increasing evidence from preclinical, prospective clinical and epidemiological studies suggest that, at least in the case of disease, early development echoes throughout life (Lester & Padbury, 2009). These findings have given rise to the concept of “fetal origins” or “developmental origins” of health and disease. In essence, environmental factors acting early in life “programme” developing systems, altering structure and function and probably behavioural expression. Stress hormones, for example, can alter regulation of the neuroendocrine environment by acting on the HPA axis, which results in an altered set point for physiologic, metabolic and behavioural outcomes.

Brain neurotransmitter systems interact to modulate both behaviour and HPA activity. Disturbances in the HPA regulation and brain monoamine levels have been associated with affective and anxiety disorders in humans. The effects of prolonged exposure to chronic stress, called allostatic load, and concomitantly, prolonged activation of the neuroendocrine stress axes have been related to physical disease and behavioural disorders (Lester & Padbury, 2009). Prenatal drug exposure is thought to be one of those factors that alter the set-points or “hard-wire” physiological systems (Lester & Padbury, 2009).
**Behavioural Dysregulation**

In the fetal programming model, the disruptions in the placental environment following prenatal drug exposure alter HPA set points, resulting in endocrine and behavioural dysregulation in the newborn that can lead to the type of childhood behavioural, emotional and neurocognitive deficits related to prenatal drug exposure described earlier. These deficits may, in turn, have implications for adolescent psychopathology, including early onset of substance use in adolescence (Chapman, Tarter, Kirisci, & Cornelius, 2007). Behavioural dysregulation can begin in utero and is proposed to be a dynamic developmental process as alterations in the quality of the environment (prenatal and postnatal) modify behavioural expression. Behavioural dysregulation is evidenced during infancy as neurobehavioural and neuroendocrine disorganisation (Lester et al., 2009). During childhood, indicators of behavioural dysregulation reflect a deficient capacity to control behaviour and regulate emotion. These phenotypes are important because they appear to be prognostic indicators for substance use. The earlier a drug is used, the greater likelihood of its abuse during adolescence and adulthood. These phenotypes reflect major domains of psychological function, cognition, affect and behaviour and include impulsivity, reactive aggression, sensation seeking, excessive risk taking, irritability, negative affect, difficult temperament, conduct disorder, attention deficit hyperactivity disorder, oppositional defiant disorder, anxiety, depression, and impaired executive functioning (Iacono, Malone, & McGue, 2008). This collection of disturbances in emotion regulation and behaviour control is included in the construct of neurobehavioural disinhibition, and as it thought to reflect disturbances in the prefrontal cortex (Tarter et al., 2003). Some of these disturbances have already been reported in the prenatal methamphetamine literature and prenatal cocaine literature, and may become more visible as the children enter adolescence (Abar et al., 2013; Piper et al., 2011). Disruption of neuroendocrine homeostasis in utero by prenatal drug exposure can be observed at birth and may lead to lasting behavioural dysregulation that increases vulnerability to substance use, resulting in early onset of substance use in adolescents (Tarter, 2002). By identifying these phenotypes in the pre-school population and targeting children who demonstrate these deficits early in life, it may be possible to change their developmental course by preventing an accumulation of deficits and the development of negative interactions with caregivers, peers and teachers (Keenan, 2000).

**Biopsychosocial Effects**

MA children can be viewed as a double jeopardy population given that they are subject to both the teratogenic effects of methamphetamine during pregnancy and are also more likely to be raised in socio-economically disadvantaged families by one or two parents affected by substance abuse disorders. Specifically, in addition to direct methamphetamine exposure during pregnancy, these children often grow up in families characterised by ongoing maternal mental illness and family instability. Such socio-familial risk factors have also been linked to an increased risk of child emotional and behavioural adjustment problems.

The dual hazard of both biological and environmental factors associated with prenatal drug exposure is illustrated in Figure 2 below (Lester & Tronick, 1994). This model illustrates how the complex dynamics seen between prenatal exposure and environmental factors may interact and shape a child’s
emotional and behavioural development. Concerns about the multiple pre- and postnatal risk factors that contribute to poorer child outcomes further highlight the need to consider the effects of both sets of factors on child developmental outcomes.

This transactional model suggests that early vulnerabilities (e.g., prenatal exposure) are influenced by the quality of the relationships or psychosocial factors within the family, as well as by the setting or environment in which these processes take place. It accounts for prenatal and postnatal factors in our understanding of the impact of a teratogen on developmental outcomes. It also suggests that these processes are bi-directional. Parents can impact the behaviour of the child, children can impact on the child-rearing practices or behaviours of the parents, and greater environmental factors can influence either or both.

The silver lining is that in the same way a chaotic environment has the potential to lead to negative outcomes, a positive or encouraging caregiving environment has the potential to lead to positive outcomes; even when children are faced with a poor start in life (Rutter, 1987). Environmental factors can be regulators, deregulators, stabilisers or destabilisers of child development. In essence, these factors have the potential to exacerbate the child’s developmental vulnerability, or help buffer/protect them from poor outcomes.

Unfortunately, much of the research suggests that the children of substance-using mothers often grow up in environments that are likely to negatively influence development (Lester & LaGasse, 2010). Therefore, in the absence of relevant data, researchers, policymakers, and the general public might therefore assume that children with prenatal exposure and disrupted early environments will manifest the poorest outcomes. However, this might or might not be correct. Alternatively, those concerned with prenatal exposure might overlook or underestimate the effects of environmental adversity, and those focused on environmental adversity might fail to account for the effects of prenatal substance exposure.

Because of this complex double jeopardy issue, the major interest in this thesis concerns how these environmental mechanisms may also be contributing to the outcomes of children born from women who used methamphetamine while pregnant. One such mechanism that has received little attention but may be having an impact on child mental health is maternal behavioural health. As will be
discussed below, MA using women are at a particularly high risk of having comorbid mental health and substance abuse problems. However, few studies have systematically documented the extent and nature of these experiences on MA children’s emotional and behavioural development outcomes. This issue forms the central aim of this thesis.
CHAPTER FIVE: BIO-PSYCHOSOCIAL CHARACTERISTICS OF PARENTING WOMEN WITH SUBSTANCE USE DISORDERS

Introduction

Pregnant and parenting women who suffer from substance use disorders present very complex and difficult challenges to society. At the micro-level, the foremost concern is the safety of the child, whereas at a macro-level, one must understand both the breadth and depth of the issues that need to be considered in order for any necessary improvement to occur (Lester & LaGasse, 2010). Therefore, there is often a tension between systems such as child welfare, who are driven by a mission to protect the child from continued or imminent danger, whereas treatment for parenting women with substance use disorders is driven by the objective to address the myriad of bio-psychosocial factors that impact both her substance use and her parenting abilities in order to ensure she is capable of providing a responsive nurturing environment for her children.

Within recent years, a distinctive biopsychosocial profile has emerged both from the experience of the substance abuse treatment system and from research focused on the characteristics of women in treatment (Lester & LaGasse, 2010). Included in this profile is family history, physical and sexual abuse, psychiatric comorbidity, caregiving environment and parenting attitudes. Certainly not every woman with a substance use disorder fits a composite profile, but the data is overwhelmingly consistent across programmes and research to support some general trends.

Risk of Poly-substance Use and Substance Using Disorders

Poly-substance use is common among women who are drug dependent (DellaGrotta et al., 2010; Lester & Lagasse, 2010; Woudes & Woodward, 2010). Women who use methamphetamine during pregnancy are also likely to be heavy users of tobacco, alcohol and other illicit substances (Arria et al., 2006). In fact, as many as 61% of women using methamphetamine during their pregnancy have tested positive for two or more drugs at the time of delivery (Good, Solt, Acuna, Rotmensch, & Kim, 2010). Each of these substances has the potential to negatively affect development.

Although methodological differences between studies and limited data in the extant literature make generalization of the results for several substances difficult, some summary statements can be made using the current knowledge base regarding the impact of poly-substance use on development. Table 1 illustrates common and unique effects among both legal and illicit drug use on child outcomes (Behnke & Smith, 2013). As is illustrated, legality of a drug does not necessarily correlate with its safety profile. The focus of this review will be on long-term outcomes of poly-substance use associated with poor child behaviour and executive functioning.
Table 1.
Summary of Effects of Prenatal Drug Exposure (adapted from Behnke & Smith, 2013, pp. 1016)

<table>
<thead>
<tr>
<th></th>
<th>Tobacco</th>
<th>Alcohol</th>
<th>Marijuana</th>
<th>Cocaine</th>
<th>Methamphetamine</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Short-term effects</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fetal growth</td>
<td>Effect</td>
<td>Strong effect</td>
<td>No effect</td>
<td>Effect</td>
<td>Effect</td>
</tr>
<tr>
<td>Anomalies</td>
<td>Strong effect</td>
<td>No effect</td>
<td>No effect</td>
<td>No effect</td>
<td>No effect</td>
</tr>
<tr>
<td>Withdrawal</td>
<td>No effect</td>
<td>No effect</td>
<td>No effect</td>
<td>No effect</td>
<td>No effect</td>
</tr>
<tr>
<td>Neurobehaviour</td>
<td>Effect</td>
<td>Effect</td>
<td>Effect</td>
<td>Effect</td>
<td>Effect</td>
</tr>
<tr>
<td><strong>Long-term effects</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Growth</td>
<td>No consensus</td>
<td>Strong effect</td>
<td>No effect</td>
<td>No consensus</td>
<td>Effect</td>
</tr>
<tr>
<td>Behaviour</td>
<td>Effect</td>
<td>Strong effect</td>
<td>Effect</td>
<td>Effect</td>
<td>No consensus</td>
</tr>
<tr>
<td>Cognition/EF</td>
<td>Effect</td>
<td>Strong effect</td>
<td>Effect</td>
<td>Effect</td>
<td>*</td>
</tr>
<tr>
<td>Language</td>
<td>Effect</td>
<td>Effect</td>
<td>No effect</td>
<td>Effect</td>
<td>*</td>
</tr>
<tr>
<td>Achievement</td>
<td>Effect</td>
<td>Strong effect</td>
<td>Effect</td>
<td>No consensus</td>
<td>*</td>
</tr>
<tr>
<td>Drug abuse</td>
<td>Increased Risk</td>
<td>Increased Risk</td>
<td>Increased Risk</td>
<td>*</td>
<td>*</td>
</tr>
</tbody>
</table>

*Note: * indicates limited or no data available.
One of the most extensively studied prenatal exposures is smoking during pregnancy. Tobacco use behaviour among pregnant women remains prevalent, and estimates in New Zealand suggest that around 18.7% of pregnant women reported smoking at the time of antenatal registration (Andrews et al., 2010). Nearly 14% of women continued to smoke throughout their pregnancy (Andrews et al., 2010). Interestingly, compared to other substance use in pregnancy, smokers have the lowest abstinence rates (Forray, Merry, Lin, Ruger, & Yonkers, 2015). It is thought one potential reason for the lower rates of abstinence might be that women with concurrent substance use may substitute smoking for alcohol or illicit substance use while pregnant. Women may perceive illicit substances and alcohol as more harmful and less socially acceptable than cigarettes and thus decide to give up the use of other substances, but not cigarettes (Moore et al., 2010). This is supported by the low smoking abstinence rates in women with concurrent substance use, and findings that smoking status is a predictor of illicit substance use in pregnancy (Forray et al., 2015).

Smoking during pregnancy has been linked to a variety of child behaviour problems, with the majority of studies indicating problems of behavioural dysregulation along the externalising spectrum with some associations extending into adulthood. Smoking during pregnancy has also been linked with disruptive behaviour problems (Wakschlag, Pickett, Kasza, & Loeber, 2006), and increased psychological symptoms for conduct disorder, drug abuse, and depression (Fergusson, Woodward, & Horwood, 1998). Interestingly, children are at the highest risk when mothers smoke during their pregnancy and continue to smoke in the home, suggesting that the mechanism of effect may be behavioural rather than from prenatal exposure per se (Maughan, Taylor, Taylor, Butler, & Bynner, 2001).

The negative impact of prenatal alcohol use is also well established, particularly in regard to fetal alcohol syndrome disorder (FASD), neurodevelopmental outcomes, and central nervous system deficits (Hankin, 2002; Sampson et al., 1997). The number of individuals affected by FASD in New Zealand is largely unknown though anecdotal evidence suggests there are significant numbers of individuals affected with and without a diagnosis (Alcohol Healthwatch, 2007). FASD can be characterised by a range of problems such as intellectual and behavioural deficits, as well as irreversible damage to the brain and body. Children with FASD are likely to be diagnosed as having disorders that fall under the mental health umbrella. These include attachment disorder, oppositional defiant disorder, and conduct disorder. They are also likely to be diagnosed with a neurodevelopmental disorder, including autism spectrum disorder, intellectual disability, and attention deficit hyperactivity disorder. At present, there is no known safe amount of alcohol in pregnancy. Alcohol use in pregnancy, even at low levels, has been associated with increases in child behaviour problems, and a dose-response was also evident, such that greater alcohol exposure was associated with increased risk (Brown et al., 1991; Sood et al., 2001).

Despite a large amount of research and major public health campaigns on the topic, surveys in the US have found that among pregnant women aged 15-44, the prevalence of reported current alcohol and cigarette usage rates have not changed substantially in the last decade. Surveys suggest that around 50% of women believe some alcohol in pregnancy is safe and 20-36% of women continue to consume alcohol during pregnancy (Alcohol Healthwatch, 2007). According to midwives the figure is
closer to 80% for pregnant teenagers (Ministry of Health, 2015). The level of maternal drinking in New Zealand is relatively high. It is estimated that about half of all pregnancies in New Zealand are exposed to alcohol (Ministry of Health, 2015), and around 10% of pregnancies will be exposed to alcohol at high-risk levels (O'Keeffe et al., 2015). Unfortunately, most severe exposure occurs during the vulnerable first trimester, particularly before a pregnancy has been confirmed (Superu, 2015).

The use of marijuana and other drugs during pregnancy has also been associated with maladaptive child outcomes, although less extensively. Two main domains of findings on marijuana use in pregnancy and child outcomes are those related to impulsivity and attention (Leech, Richardson, Goldschmidt, & Day, 1999; Richardson, Ryan, Willford, Day, & Goldschmidt, 2002) and executive functioning (Fried & Smith, 2001; Goldschmidt, Richardson, Willford, & Day, 2008).

The use of cocaine during pregnancy has similarly been associated with a wide array of deleterious child development outcomes. Caregiver reports of child behaviour problems in pre-school-aged (Warner et al., 2006) and elementary school-aged children (Accornero, Anthony, Morrow, Xue, & Bandstra, 2006; Linares et al., 2006) have not been related to cocaine exposure, except in combination with other risk factors (Bendersky et al., 2003; Dennis, Bendersky, Ramsay, & Lewis, 2006; B. G. Sood et al., 2005). However, in longitudinal modelling of caregiver reports at 3, 5, and 7 years of age, the multisite Maternal Lifestyles Study revealed that prenatal cocaine exposure had an independent negative effect on trajectories of behaviour problems (Bada et al., 2007). There have been teacher reports of behaviour problems in prenatally exposed children (Delaney-Black et al., 2000), although again, findings have not been consistent across studies (Richardson, Conroy, & Day, 1996) and some have been moderated by other risks (Bailey et al., 2005). As a stimulant, cocaine affects the neurological arousal systems of children, suggesting that they have more difficulty regulating their arousal, effects that can persist if exposure continues (Mayes, 2002). Similarly, children exposed to cocaine prenatally may also have more difficulty regulating their attention, which can be a risk factor for later development of attention and/or anxiety disorders (Frank, Augustyn, Knight, Pell, & Zuckerman, 2001; Mayes, Grillon, Granger, & Schottenfeld, 1998). Use of cocaine during pregnancy has also been linked to flatter affect and decreased levels of executive functioning in children (Bridgett & Mayes, 2011; Espy, Kaufmann, McDiarmid, & Glisky, 1999). Cocaine exposure was also related to challenging behaviour in boys but not girls in two studies of young children (Delaney-Black et al., 2000; Delaney-Black et al., 2004).

The pervasive maladaptive physical effects associated with substance use during pregnancy highlight the importance of continuing to consider the possible effects on child behaviour. Many of the studies reviewed above that focused on child behavioural outcomes have shown convincing evidence that prenatal exposure to these independent drugs can have long-term outcomes. The majority of studies discussed above attempted to disentangle the effects of prenatal drug use of one kind from exposure to others by using covariates to control for possible confounds, or by looking at group differences based on single drug use, multiple drug use, and no drug use. Although not explicitly discussed, there is also the potential that when used in combination, poly-substance use may have an amplified association with the development of poor developmental outcomes. These symptoms may be associated with developmental or cognitive delays and can significantly interfere with emotional bonding.
between the caregiver and the infant. It is also feasible that this biological vulnerability may be compensated for by sensitive and competent caregiving.

In addition to the direct fetal effects, exposure to parental addiction during childhood can also have multiple indirect consequences for children. Compared to children who do not abuse alcohol or drugs, children of substance-abusing parents are more likely to experience physical, intellectual, social and emotional problems and are often customers of the child welfare system. According to the transactional model of development, one pathway to later developmental problems among drug-exposed infants may be through proximal environmental factors, such as the quality of the mother-child interaction.

Risk of Psychiatric Disorders & Comorbidity

The adverse effects of prenatal substance use are further complicated by the frequency of concurrent substance use and comorbid psychiatric illness (Benningfield et al., 2010; Tuten et al., 2009). Clinicians and researchers have attended to the links between substance abuse and mental illness for several decades (Bukstein, Brent, & Kaminer, 1989; Myers et al., 1984; Regier et al., 1990; Schottenfeld, Carroll, & Rounsaville, 1993). Women with substance use disorders tend to have higher rates of other psychological diagnoses. These connections may arise from several different mechanisms. Substance abuse may potentially trigger or relate to the development of psychiatric symptoms. Conversely, underlying psychopathology may contribute to the abuse of psychoactive substances. Finally, some underlying factor may increase individuals' vulnerability to both substance abuse and mental illness. Regardless of the mechanism driving these connections, these links have been well established. An evaluation by the Center for Substance Abuse Treatment (CSAT), SAMHSA, found that of the 5,110 pregnant women who were receiving services for drug and alcohol abuse treatment, 60% had co-occurring psychiatric disorder (CSAT, 2003).

Dual diagnoses remain a key concern related to substance abuse and mental health. Research studies have documented the extent to which mental illness and substance abuse disorders co-occur and exacerbate one another. In particular, substance abuse disorders have a high rate of occurrence with anxiety and affective disorders (Grant, Hasin, Chou, Stinson, & Dawson, 2004; Kalman, Morissette, & George, 2005; Merikangas et al., 1998; Swendsen & Merikangas, 2000). Indeed, individuals with mental illnesses may be most at risk for developing substance abuse problems (Goodwin et al., 2002). The co-occurrence of mental illness and substance abuse is significant since those with such co-morbid disorders are less likely to be adherent to medication regimens (Drake & Wallach, 1989), have a greater likelihood of psychosis (Dixon, 1999), have higher treatment costs (Drake, Mercer-McFadden, Mueser, McHugo, & Bond, 1998), are more likely to have physical health problems (Dixon, 1999), and are more likely to have contact with the criminal justice system or to be homeless (Osher & Drake, 1996). Additionally, those with dual diagnoses are more likely to relapse after treatment than other substance abusers (Bradizza, Stasiewicz, & Paas, 2006). As such, integrated treatment for individuals with dual diagnoses is more complicated and more intense than treatment for individuals suffering from each of these problems alone (Drake et al., 1998).
Depression is a consistent finding in methamphetamine-using women. Zweben et al. (2004) reported 68% of women seeking outpatient drug treatment reported a history of feeling depressed and 28% reported attempting suicide at some point in their lifetime. The depression frequently reported with MA users may be related to pre-existing depressive symptoms or secondary to MA-induced effects. Long-term MA use has been associated with more severe psychiatric symptoms, a finding possibly attributable to a greater reduction of dopamine transporter density in the brain (Sekine et al., 2001). Given that decreased dopamine has been linked with depression (Dunlop & Nemeroff, 2007), women who use MA during pregnancy, or those recently abstinent, are at increased risk for depressive symptoms.

Pregnancy and the postpartum period are times of significant vulnerability to depression. Bennett et al. (2004) found a 12% prevalence of depression during the second and third trimesters. In addition, the incidence of postpartum depression is reported in 10–22% of women (Burt & Stein, 2002). Pregnancy-related risk factors for postpartum depression include unplanned pregnancy and unemployment (Warner, Appleby, Whitton, & Faragher, 1996), which are often associated with substance abusing women. Pregnant women with substance use disorders have also been found to have a range of emotions regarding their pregnancy. While the pregnancy is often viewed positively as a chance to “start over,” these emotions may be accompanied by feelings of guilt related to their drug use, concern about their baby’s health, general discontent with the state of pregnancy, and negative feelings regarding the circumstances of conception (Comfort & Kaltenbach, 1999; Kissin, Svikis, Morgan, & Haug, 2001). Moreover, as was discussed previously, discontinuing methamphetamine during the pregnancy or afterward could also be associated with a host of mental health concerns, including severe depression, anxiety, fatigue, and suicidal ideation (Gawin & Ellinwood, 1988; Newton et al., 2004).

Maternal mental health is important to child development because it can affect the quality of the parent–child relationship and, in turn, the children’s well being over both the short and the long term. According to the literature, the most prominent predictors of developmental outcome for children and non-exposed children are the psychological resources of the mother (Jeremy & Bernstein, 1984). Furthermore, attachment theorists and researchers have argued that many child behavioural and emotional problems originate in the parent–child relationship, particularly in the parents’ capacity to support the child’s developing behavioural and emotional regulation capacities (Carlson, 2003; Lyons-Ruth, Zoll, Connell, & Grunebaum, 1986; Madigan, Moran, Schuengel, Pederson, & Otten, 2007; Sroufe et al., 2005).

There is now a very strong body of evidence about the influence of maternal mental health on child mental health and behaviour, with a growing number of studies linking mental health disorders in pregnancy and the early postpartum period to poor child and adolescent health outcomes (Giallo, Cooklin, Wade, D’Esposito, & Nicholson, 2014; Kingston, Tough, & Whitfield, 2012; Paschetta et al., 2014; Pearson et al., 2013; Ramchandani, Stein, Evans, & O’Connor, 2005). Co-occurring maternal psychological distress with substance use disorders have been associated with a range of adverse outcomes in pre-school and school-age children, including depression, anxiety, lower social competence, and behavioural problems (Eiden, Colder, Edwards, & Leonard, 2009). In a meta-analytic
review of 193 studies, Goodman et al. (2011) found that maternal depression was associated with significantly higher levels of child internalising behaviour, externalising behaviour and negative affect, with associations moderated by child gender and low family income. Infants as young as 3 months can detect a depressed affect in their mothers and by 18 months maternal depressive symptoms are associated with decreased verbal interaction, increased time playing alone, less competence in object concept tasks, and insecure attachment. These findings at 18 months are noted even though a majority of the women no longer reported depressive symptoms (Weinberg & Tronick, 1998) suggesting children are vulnerable to maternal depressive symptoms during the first 3 months of life and are at risk for long-term developmental delays. Among preteens and adolescents, perinatal depression has been associated with externalizing behaviour (e.g., conduct disorder) on the part of children, school problems, functional impairment (social, cognitive), internalizing symptoms (e.g., depression, anxiety), and alcohol use (Gance-Cleveland, Mays, & Steffen, 2008; Nunes et al., 1998; Nunes et al., 2000).

Less clear is the extent to which maternal mental health disorders occurring after the postnatal period may worsen child outcomes in the longer term and/or independently contribute to poor child emotional and behavioural difficulties. This is because those studies that have collected information on maternal mental health beyond the perinatal period have largely treated maternal depression at later time points as a confounder of the association between perinatal depression and child and adolescent health outcomes, rather than as an exposure in its own right (Brennan et al., 2000; Kiernan & Mensah, 2009; Leis, Heron, Stuart, & Mendelson, 2014; Pearson et al., 2013).

Although much work has been done describing psychiatric comorbidity in cocaine-using persons (e.g., Schottenfeld et al., 1993; Ziedonis et al., 1994) and in the general drug-using population (Bukstein et al., 1989; Conway, Compton, Stinson, & Grant, 2006; Regier et al., 1990; Warner, Kessler, Hughes, Anthony, & Nelson, 1995), less work has been carried out in MA-dependent samples (Glasner-Edwards et al., 2008; Glasner-Edwards et al., 2009; Shoptaw, Peck, Reback, & Rotheram-Fuller, 2003). There is only one study from the larger IDEAL study base which looked at this, and they found that mothers who used MA in pregnancy were 10-12 times more likely to have a substance use disorder (SUD) than that of mothers in the comparison group and were twice as likely to meet the Brief Symptom Inventory (BSI) criteria for a diagnosable psychiatric disorder (Woudes et al., 2013). The authors also found that mothers in NZ were five times more likely to have comorbidity of both substance abuse and psychiatric disorders compared to mothers in the US as a result of increased prenatal alcohol use.

When co-occurring psychiatric disorders are present, they may adversely affect the response to treatment of substance use disorders (see Ries & Goldsmith, 2009 for a review). Published studies have shown that patients who had access to ongoing mental health treatment had better substance abuse outcomes compared to those who did not (Ouimette, Brown, & Najavits, 1998; Ritsher, McKellar, Finney, Ottingham, & Moos, 2002). Thus, the characterization and description of co-occurring disorders is an important first step in the treatment of co-occurring psychiatric disorders in MA abuse. Knowledge about the frequency and characteristics of co-occurring disorders may lead to improved diagnostic efficiency and accuracy. Such information may also guide clinical and programmatic planning for additional mental health services that may be required in the treatment of MA dependence.
Other Psychosocial Risks

Women who use drugs during pregnancy have lifestyles that are burdened by multiple adverse psychosocial circumstances that are likely to impair their ability to parent their new born child (Terplan et al., 2009; Pitzer, Jennen-Steinmetz, Esser, Schmidt, & Laucht, 2011; Rhoades, Greenberg, Lanza, & Blair, 2011; Vohs & Baumeister, 2011; Galarce & Kawachi, 2013), and are also more likely to have multiple psychosocial problems (Woudes et al., 2013). Pregnant substance users face elevated risks of poverty (Hunt, Kuck, & Truitt, 2007), current domestic violence (Derauf et al., 2007); histories of previous trauma, including physical and sexual abuse (Cohen, Greenberg, Uri, Halpin, & Zweben, 2007). One study found that pregnant women who used methamphetamine were more likely than other pregnant women to be younger, live without a partner, have a lower income, have less education, and have received less prenatal care (Smith et al., 2006).

The caustic chemicals and toxic gases associated with parental methamphetamine use also make the home environment particularly dangerous for children. The bulk of the research in this area has focused on the effects of methamphetamine on children's physical health (Hohman, Oliver, & Wright, 2004; Smith et al., 2001) or on the hazardous conditions to which children are exposed, including fires and explosions resulting from methamphetamine production in their homes (Cretzmeyer, Sarrazin, Huber, Block, & Hall, 2003). Children residing in homes where methamphetamine is being used or produced are also at a heightened risk for health harm. Because a child's body is still developing, they are not able to eliminate toxins as efficiently as an adult and are therefore more likely to develop cancer and organ damage (Connell-Carrick, 2007). Furthermore, infants and toddlers are especially vulnerable because of their explorative behaviour—crawling, touching, and putting objects in their mouths. An environmental exposure model set to the current New Zealand remediation standard for methamphetamine residue (≤ 0.5 μg/100 cm²) predicted the daily dose for an infant is 7.6 times that of an adult on a body weight basis (0.00019 mg/kg/day versus 0.000025 mg/kg/day), and a child's dose is 4.4 times greater (0.00011 mg/kg/day) (Hammon & Griffin, 2007; Jones & Kaltenbach, 2013).

The potential for injury from environmental contaminants may be exacerbated by a concurrent lack of parental supervision (Swetlow, 2003). Cases of children presenting to emergency rooms with caustic ingestions resulting in protracted hospital stays and severe debilitating injuries have been subsequently linked to parental use of methamphetamine (Farst et al., 2007). Even asymptomatic children may be at risk for long-term health problems.

Risks for Attachment and Parenting Attitudes

Although for some women, drug use is a social or recreational activity that does not necessarily disrupt their daily caregiving responsibilities (Kearney, Murphy, & Rosenbaum, 1994), for most women, drug abuse is part of a complex and often chaotic lifestyle (Amaro, Fried, Cabral, & Zuckerman, 1990; Anglin & Perrochet, 1998; Barnet, Duggan, Wilson, & Joffe, 1995; Davis, 1990; Hans, Bernstein, & Henson, 1999; Hans, 1999; McGaha & Leoni, 1995; Swartz et al., 1998). When drug use occurs in the context of multiple other risks, it may interfere with the mothers’ ability to care for their children. This can lead to disrupted parental care and early dysfunctional maternal-child dyad interactions that can
compound the negative effects of prenatal drug exposure (Mansoor et al., 2012; Strathearn & Mayes, 2010). There is evidence that the majority of substance-exposed children may be insecurely attached or have disorganised attachment with their primary caregivers (O'Connor, Kogan, & Findlay, 2002; Rodning, Beckwith, & Howard, 1989a; Rodning, Beckwith, & Howard, 1989b).

While there is no literature investigating the quality of mother-infant interactions with prenatal methamphetamine exposure, studies on prenatal cocaine exposure have offered interesting results. Women who used cocaine during pregnancy were found to be less attentive and engaged during a face-to-face interaction with their infants over the first 6 months (Mayes et al., 1997) as well as less enthusiastic and responsive during the first year of the infant’s life than non-using mothers (Burns, Chethik, Burns, & Clark, 1997). Research on toddlers has indicated that these mothers are more likely to be intrusive and hostile and to engage in poorer quality of instruction with their 3 year-olds (Johnson et al., 2002). Maternal poly-drug use has also been found to decrease maternal responsiveness and dyadic reciprocity during feeding interactions (Eiden, 2001). These results indicate a lower quality of early interaction between drug-using parents and their children.

**Stress Effects on Parenting**

In addition to showing less responsiveness, addicted mothers may also be overly negatively reactive to children. Compared to nondrug users, substance abusing women experience higher stress related to parenting, are often more punitive towards their children—frequently associated with their own experience of parental and partner violence (Arellano, 1996; Hans, 1999; Kelley, 1998; Miller, Smyth, & Mudar, 1999; Schuler & Nair, 2001). Clinical observation of “discipline” techniques used by women in substance abuse treatment facilities have been corroborated by research focused on attitudes and behaviours that found that a child’s undesired behaviour was often met with harshly punitive behaviour, expressed by yelling, threatening, and physical punishment. Moreover, the definition that the child’s behaviour was “unacceptable” was often driven by a lack of understanding of basic developmental milestones, thus leading to unrealistic expectations of children’s behaviour (Kaltenbach, 1996; Sowder & Burt, 1980). In a study by Azar and Rohrbeck (1986), drug-using parents frequently expected that their 2-to-3-year-old was capable of engaging in self-care and controlling their impulses (e.g., stopping crying when told to do so). This lack of understanding was also coupled with a low capacity to reflect upon their children’s emotional and cognitive experience. The authors concluded this can impact on how drug-using parents relate to their children.

Child welfare specialists report high levels of neglect, trauma, and abuse found in homes where methamphetamine is used (Anglin, Burke, Perrochet, Stamper, & Dawud-Noursi, 2000). Children may be exposed to drug paraphernalia; adult violence; criminal behaviour related to the purchase of methamphetamine; neglect from parents who may sleep for days following binges or who are preoccupied with obtaining more methamphetamine; and physical, sexual, and emotional abuse from parents and other users who frequent the home (Arellano, 1996; Haight et al., 2005; Nair et al., 2003). Many children of drug abusers also face neglect as their parents fail to care for their basic needs (e.g., food, appropriate housing) due to active substance use or related stressors.
Abusive mothers have been found to respond to their children’s noncompliance with more negative behaviour (Borrego, Gutow, Reicher, & Barker, 2008), which may lead to a cycle of negativity. Bagner et al. (2009) found, in a sample of mothers with a history of prenatal substance use, that parenting stress was associated with more externalizing behaviours in children. Bennet, Bendersky and Lewis (2002) showed that cocaine exposed 5 year-old boys living in high-risk environments were aggressive.

Risks for child abuse and neglect may relate to poor socialisation of emotion within families. The parental anger-intensifying attributional style for especially negative child behaviour (Pidgeon & Sanders, 2009) may cause the children to have a limited ability to feel remorse or empathy. Neglected children who are unable to form secure attachments with their primary caregivers may suffer from many problems in understanding the emotions of others, or forming and maintaining relationships with others (Cicchetti & Toth, 1995; Robinson et al., 2009; Shipman, Edwards, Brown, Swisher, & Jennings, 2005). Following this premise, it is also likely that children are modelling aggressive and punitive behaviours being demonstrated within the home environment, as a way to deal with stress. This may increase a child’s likelihood of developing more severe disruptive behaviours or psychological disorders later in life.

Summary

The existing evidence indicates that although prenatal exposure may confer some degree of developmental disadvantage, it frequently occurs in the inadequate rearing environment and disruptions in emotional parent-child relations or poverty, which may be also strong determinants of the children’s outcome. Taken together, these findings suggest that, as a group, substance-using mothers are at risk of being less responsive to, more negatively reactive to, more likely to abuse or neglect and more likely to model unhelpful behaviours with their children.

The increased life stressors and altered stress response of addicted individuals may also contribute to the pathway from addiction to poor parenting to poor child outcomes. As stated above, drug-using individuals have higher levels of chronic stressors and have the additional physiological stressor of exposure to binging and high levels of drug use, including overstimulation of stress and reward pathways, comorbid psychological conditions, tolerance and adaptation, and withdrawal-related effects. This accumulation of stressors may lead addicted individuals to be overwhelmed, leading them to lack the resources and energy to pay attention to and respond appropriately to their children’s needs and problems. Furthermore, chronic use of substances directly affects brain circuitry involved in inhibition of impulsive behaviour and emotion or stress regulation. This altered stress system could lead them to show heightened (and impulsive or under-regulated) emotional reactions to children’s behaviours in daily interactions. Even relatively minor child behaviours (such as whining) could be stressful to addicted individuals, given their heightened sensitivity to stressors. Heightened sensitivity to stressors in the mother-child relationship, could, in turn, lead addicted parents to become negatively and overly punitive of children - or lead them to shut down and neglect or avoid their children. In addition, drug-using mothers are more likely to have a history of child abuse themselves. This abuse, and its
effects on stress systems, could help explain the greater rates of abuse and neglect by addicted parents as research has documented an inter-generational transmission of abusive behaviours (Oliver, 1993). Research also suggests that when under stress, addicted mothers may be more likely to use substances, and children may be exposed to watching their parents get intoxicated. While under the influence, mothers are then likely to show even greater emotional lability and may be more likely to be abusive. This models drug-using and poor emotional regulation behaviours for their children.

The unresponsive and punitive parenting often observed in drug-using homes has the potential to lead to negative consequences for children's social and emotional development. This type of parenting, particularly in the context of the chaotic family life associated with active drug use and comorbid mental health difficulties, can lead children to have problems with their own emotion regulation and impulsivity, which can lead them to be a greater risk for the development of behaviour problems and, in adolescence and young adulthood, addictive behaviours. Of course, the biological impact of prenatal exposure to substances independently can have deleterious developmental outcomes for children. However, the compromised behavioural health and practices of mothers, combined with increased stress in the family environment, would suggest poor stress-regulation and coping-modelling also play an important role in mediating the effects of prenatal drug use on children's development.

In short, a socio-ecological approach to understanding the effect of prenatal substance exposure in context, suggests that by identifying and treating risks early, there may be a potential to insulate children from MA-induced direct or indirect biological vulnerability. Therefore, high-quality prospective data are urgently needed to better understand the unique and combined effects of these experiences on subsequent outcomes throughout development. Such data would better inform research, prevention/intervention efforts, and public policy in this area.
CHAPTER SIX: NEUROBEHAVIOURAL DISINHIBITION

Introduction

One area of behavioural development that warrants attention among children exposed prenatally to substance use is neurobehavioural disinhibition (ND). ND is a psychological construct developed by Dr. Tarter and his colleagues (2003) that involves describing children’s emotional states and neurological capacities, as well as their behavioural manifestations. It encompasses problems of excessive risk taking, impulsivity, aggression, irritability, difficult temperament, attention deficit-hyperactivity disorder, and impaired executive function. Children and adolescents exhibiting these disinhibitory symptoms often following negative developmental trajectories associated academic difficulties, delinquency, mental health problems, and substance abuse. While somewhat new and novel, the ND profile has been used in the addiction and prenatal substance exposure literature (Chapman et al., 2007; Fisher et al., 2011; Lester et al., 2012). One of the advantages of the conceptualisation of ND is that it includes neurobiological-based functions (e.g. executive processes) and psychological measures of behavioural dysregulation, including emotion regulation and behaviour control. It also considers the equifinality in the etiologic pathways of ND, including genetic vulnerabilities, and various individual (e.g. temperament), familial (e.g. parent psychopathology and parenting-styles), and contextual variables (e.g. early adversity and school environment) (Fisher et al., 2011).

Components of Neurobehavioural Disinhibition

Although neurobehavioural disinhibition has been implicated in numerous outcomes, different sub-disciplines within the field of psychology have approached the study of neurobehavioural disinhibition from diverse frameworks (Bridgett, Oddi, Laake, Murdock, & Bachmann, 2013). Some scientists have attempted to concisely define it by listing functions or underlying operations, while others have focused on neuroanatomical or neuropsychological correlates. Without a unifying construct or definition, investigations of neurobehavioural disinhibition have gone a number of directions; the most prominent main features being: (a) dysregulated emotions, (b) behavioural undercontrol, and (b) deficits in executive functioning.

Emotion Regulation

Emotion development includes changes in emotion expression, understanding, and regulation. Of these, emotion regulation (ER) is particularly likely to be related to neurobehavioural disinhibition. It has been difficult to achieve a consensus on a single definition of ER, however the most widely accepted one is from Saarni (1984) in which ER is referred to as regulating the experience of emotion by monitoring one’s expressive behaviour. At the core of ER is the contribution of modulation (e.g., maintaining, activating, inhibiting) of emotion-related activities in specific situations, but also individual/dispositional differences across situations (e.g., temperament) (Eisenberg & Spinrad, 2004).
ER is an early emerging set of skills that takes a long time to develop, but shows marked improvement over the pre-school period. ER has been linked to several aspects of social functioning in pre-schoolers, and is seen as a vital aspect of social competence and one that determines, in large measure, the crucial social task of pre-school children: positive engagement and self-regulation during social interactions (Carlson & Wang, 2007). By developmental increases in the comprehension and control of emotionality during the pre-schooler years, the child is able to modify their emotional reactions to effectively deal with the increasing complexity and demands of the social world.

Children with neurobehavioural disinhibition are considered low in ER, impulsive and high in emotional intensity; they are easily frustrated and prone to reactive aggression (Hughes & Ensor, 2008). A disinhibited child is irritable and easily thrown off balance and has a harder time than other children returning to a comfortable emotional state after a stressful or arousing experience. His emotions seem to be more intense than those of his peers; leading to irritability, anger and aggressive outbursts. These characteristics commonly provoke negative responses from adults and other children. If so, a vicious cycle can develop, and the child's reactions can become more extreme as time goes on. Poor ER is prominent in developmental psychopathology clinical groups such as children with Autism Spectrum Disorders (Hughes & Russell, 1993; Matchullis, 2012; Robinson, Goddard, Dritschel, Wisley, & Howlin, 2009) and Attention Deficit and Hyperactivity Disorder (Barkley, 1997; Iron, 2012; James, Lai, & Dahl, 2004).

**Behavioural Undercontrol**

Behavioural undercontrol (BU) refers to a wide range of behaviour that is expressed during childhood (and beyond), which reflects inhibited behavioural impulses. Symptoms include impulsivity, aggression, sensation seeking, and psychoticism (Sher & Trull, 1994; Stice, Myers, & Brown, 1998). Individuals high in BU have difficulty delaying gratification and tend to ignore potential long-term negative consequences of their behaviour (Watson & Clark, 1993). Individual differences in BU may result from differences in sensitivity to reward and punishment (Dawe, Gullo, & Loxton, 2004). High BU may reflect greater sensitivity to the reward value of risky behaviours (e.g., substance use) and greater propensity for such behaviour. Under controlled individuals are also seen as less sensitive to punishment, and less likely to inhibit behaviour in order to avoid negative consequences (Gray & Tallman, 1987).

BU is linked to maladaptive behaviours, including alcohol (Doran, Myers, Luczk, Carr, & Wall, 2007; Husson & Chassin, 1994; Slutsk et al., 2002) and tobacco use (Barker, Eriksson, Forsen, & Osmond, 2002; McMahon, 1999), risky sexual behaviours, delinquency, and academic underachievement, particularly in youth (Conway, Kane, Ball, Poling, & Rounsaville, 2003; Cooper, Wood, Orcutt, & Albino, 2003; Jackson, Sher, & Wood, 2000). Adolescent studies frequently assess BU (Erblich & Earleywine, 2003; Rutledge & Sher, 2001) and similar constructs (Devieux et al., 2002; Molina & Pelham, 2003; Soloff, Lynch, & Moss, 2000). Researchers have tested the psychometric properties of measures related to BU. While some have found that the properties of these scales differ by sex or ethnicity (Aggen, Neale, Reysamb, Reichborn-Kjennerud, & Kendler, 2009; Cooke, Kosson, & Michie,
Children with neurobehavioural disinhibition are considered to have high behavioural undercontrol, which manifests as high-rate, risky and impulsive behaviours (Elkins, King, McGue, & Iacono, 2006). These children have a strong need for excitement and are often described as “sensation seekers” or “daredevils.” The tendency toward behavioural control is likely heritable; twin and family studies strong heritability for risk-taking and sensation-seeking behaviour (Iacono, Carlson, Taylor, Elkins, & McGue, 1999; Young, Stallings, Corley, Krauter, & Hewitt, 2000). It is also felt that parents that display behavioural undercontrol (often characterised by SUDs or ADHD) may model these behaviours for their children (King & Chassin, 2004). Indeed, behavioural undercontrol is a strong predictor of later disruptive behaviour problems and substance use.

Executive Functioning

Executive function (EF) broadly refers to higher-level cognitive processes that are necessary for goal-oriented behaviour, including attending, selecting, initiating, implementing, and regulating thought, emotion, behaviour and facets of motor and sensory functioning (Dawson & Guare, 2010; Minnes et al., 2014). In addition to ADHD (Barkley, 1997; Castellanos, Sonuga-Barke, Milham, & Tannock, 2006; Hervey, Epstein, & Curry, 2004; Sergeant, Geurts, Huijbrysts, Scheres, & Oosterlaan, 2003; Willcutt, Doyle, Nigg, Faraone, & Pennington, 2005), impaired executive functioning has been reported in Oppositional Defiant Disorder (ODD), Conduct Disorder (CD) (Henry & Moffitt, 1997; Hill, 2002; Oosterlaan, Scheres, & Sergeant, 2005) and high functioning autism (Verte, Geurts, Roeyers, Oosterlaan, & Sergeant, 2006) as well as in borderline personality disorder (Putnam & Silk, 2005).

Barkley (1997) describes “behavioural inhibition” as the primary executive function in that it helps an individual to be able to think before acting and responding so that other executive functions can then be employed. These other executive functions include: working memory, which allows one to move beyond the “here and now;” the ability to regulate affect and motivation, which impacts the control of one’s behaviours and emotions; the internalization of speech, which leads to metacognitive skills, planning words and concepts, and problem-solving; and reconstitution, the final phase, which allows an individual to recombine behaviours, thoughts, memories, and analyses in order to engage in appropriate problem-solving behaviours (Miyake et al., 2000). Inhibition refers to processes of preventing impulses to take action and/or preventing distracting information from interfering. Failure to inhibit inappropriate responses will lead to stimulus-driven behaviour, where the interaction level with the environment will be high. At a pre-school level, this would include behaviours such as interrupting a conversation, running in to the street without looking and failing to resist temptations are conceived as natural but immature behaviour. When this type of behaviour persists and is no longer age appropriate it is commonly linked with a diagnosis.

Zelazo and Cunningham (2007) have proposed an interactive model in which emotion and their behavioural manifestations correspond to the motivational aspect of cognition in conscious, goal-directed problem solving. On this view, ER is either primary or secondary, but never entirely divorced from the EF of inhibition. According to Zelazo and Cunningham’s model, EF and ER bear a reciprocal
relation, the precise nature of which depends on the motivational significance of the problem and whether the problem itself is emotive (or what is commonly referred to as 'hot') or affectively neutral (or 'cold'). Cool inhibitory functions are elicited by abstract, decontextualized problems. Examples of cold executive functions include working memory, sustained attention, and organization. These executive functions are presumed to be located in the brain's dorsal lateral prefrontal cortex (Chambers, Garavan, & Bellgrove, 2009). Hot inhibitory functions are elicited by problems that involve the quick, automatic processes of affect and motivation, in which rewards and punishments are often present. These involve traits such as impulsivity and response inhibition. It is believed that these executive functions are centred in the orbital and prefrontal cortex of the brain (Aron, Robbins, & Poldrack, 2004; Booth, Charlton, Hughes, & Happe, 2003; Rubia et al., 2001; Vuilleumier et al., 2000).

**Prenatal Exposure and Neurobehavioural Disinhibition**

The growing evidence that stressful events occurring at specific points in time in development exert unique but cumulative effects on the individual's epigenome (Lester et al., 2009) and behavioural adjustment and self-regulation suggests that ND is beneficial way to understand the complexity of prenatal exposure and early adverse social determinants on later development. Evidence to-date suggests that ND in 8–14 year-olds is associated with maternal cocaine (Fisher et al., 2011; Lester et al., 2009) and alcohol (Chapman et al., 2007) use during pregnancy. Neurobehavioral disinhibition in these children also mediated substance use initiation by age 16–19 (Lester et al., 2012). The IDEAL study demonstrated prenatal MA was associated with neurobehavioral disinhibition at 5 and 6.5 years of age (Abar et al., 2013). Early adversity, which was measured from birth to age 3, significantly mediated the relationship between prenatal MA and ND. The authors note that the methodology and measurement of predictors several years antecedent to the executive function outcome made it challenging to disentangle problems caused by prenatal exposure to drugs from those resulting from compromised social environments. These studies did not incorporate multiple informants and only used objective tasks testing executive function. Maternal psychopathology at 1 month postpartum was included in the model as part of the early adversity index score, however there was no specific evaluation of this determinant on ND, nor the impact of severity or comorbidity. In another IDEAL study, prenatal MA, alone and in combination with postnatal drug exposures, was associated with behavioural and executive function deficits at 6.5 years (Himes et al., 2014).

**Importance of the Development of Emotional and Behavioural Regulation**

From a developmental cognitive neuroscience perspective, emotion and cognition are intricately linked and work together to process information and execute action or behaviour (Bell & Wolfe, 2004; Cacioppo & Berntson, 1999). Bi-directional influences are likely: emotions can help organise one’s thinking, learning and action (emotion as regulating), and cognitive processes play in regulating emotions (emotions as regulated).
For a long time, behavioural regulation and associated cognitive processes during early childhood were assumed to be negligible and thus of little or no relevance to understanding pre-school behaviour. However, the recognition of simple behaviours emerging as early as infancy, such as the regulation of eye moments and searching for hidden objects (Diamond, 1990), highlighted the developmental antecedents of more complex higher-order skills. It is now widely recognised that substantial differences between individual children’s early social and academic skills emerge well before formal schooling. Recent efforts to develop new paradigms to assess emotional regulation skills in pre-school have enabled substantial progress in the understanding of the development of the functions that allow for planned, deliberate and flexible behaviour.

In particular, recent years have seen a massive growth in the number of developmentally appropriate tasks available for assessing executive processes in children (Carlson, 2005; Diamond, Prevor, Callender, & Druin, 1997; Espy et al., 1999; Zelazo & Müller, 2002). As a result, our understanding of the development of higher-order cognitive functions and how it is related to emotion regulation and behavioural undercontrol has dramatically improved. For instance, it is now known that emotional, behavioural and executive processes: (1) begin to emerge in the first few years of life (Diamond, 1990); (2) continue to develop through adulthood (Huizinga, Dolan, & van der Molen, Maurits, 2006); (3) are a unitary construct with partially dissociable components (Garon, Bryson, & Smith, 2008); (4) show strong associations with family factors, such as SES and maternal wellbeing (Hughes & Ensor, 2005; Mezzacappa, 2004); (5) show equally robust associations with cognitive characteristics, such as language ability (Blair & Razza, 2007; Hughes, 1998); (6) predict school readiness (Blair & Peters, 2003; Brock, Rimm-Kaufman, Nathanson, & Grimm, 2009) and; (7) can be improved in at-risk samples through pre-school intervention programs (Diamond et al., 2007).

At an individual level, neurological maturation and temperament are critical for the building blocks of executive processes and behavioural regulation. Rapid development of the prefrontal cortex in the years from 3 to 6 suggests that the pre-school period is a crucial time for acquiring skills important for successful outcomes (McClelland et al., 2007). Research has found that elementary forms of the core EF functions are present early during the pre-school period and there are “changes in EF during the latter half of the pre-school period appear to be due to the development of attention and integration of component EFs” (Garon et al., 2008, p. 13). Maturation of these mechanisms is associated with the rapid development of executive attention networks (Rothbart & Rueda, 2005) and skills that help children control, direct and plan their actions.

Most executive processes show a marked improvement between the ages of 3 and 5. In a study by Carlson and colleagues (2005) children aged 3 and 4 were tested with the “less is more” task requiring them to point to a smaller reward in order to receive a larger one. There was significant performance improvement with age and additionally, improvement over trials for the 4-year olds, but not the 3-year-olds, indicating a learning effect for the older children. In Mischel’s (1989) marshmallow test - a widely used measure of delayed gratification – children are asked to choose an immediate reward (e.g. one marshmallow) versus a delayed reward with greater value (e.g. two marshmallows). Prençipe & Zelazo (2005) found that 3 year olds generally choose the immediate reward, whereas 4 year olds show a preference for the delayed reward. This transition reaches its ceiling at 5 years of age; making
the pre-school years an important period in which to address or modify motivational, incentive and emotional processes.

It is clear that the pre-school years are a critical transition period in the development of specific emotional and behavioural processes. However this period is also associated with rapid changes in language ability, symbolic thought, and self-understanding. Therefore, despite our advancement in understanding the components of neurobehavioural disinhibition, there is still little information about the processes by which children move from one level to another, and how all these aspects enable better-regulated and more goal-directed behaviour.

**Measuring Neurobehavioural Disinhibition**

One issue worth noting is that ND has been operationalized in several ways in prior studies. For some, both internalizing and externalizing problems are included (Tarter et al., 2003), whereas for others, the term is only applied to externalizing disorders (Krueger & South, 2009; Krueger et al., 2002). Chapman et al. (2007) and Tarter et al. (2004) treated ND as a unitary construct including measures of executive functioning and behavioural dysregulation. This may be due in part to clinical observations of youths who exhibit ND and for whom problems in the areas tend to co-occur. Other researchers have treated executive functioning as a separate and distinct construct from behavioural dysregulation (Iacono, et al., 2008; Krueger and South, 2009). Models with separate ND component constructs for behavioural dysregulation and executive functioning as opposed to a single-factor solution have been shown to yield excellent fit indices and more substantive findings on the contributions of precursors and their timing on development (Fisher et al., 2011). Therefore, in this study we treat ND as a multicomponent phenomenon.

The work of this thesis focuses mainly on the executive function process of inhibition in specific affect-mediated (‘hot’) situations, as it was previously discussed this component is thought to underlay the different executive functions (Barkley, 1997; Zelazo & Cunningham, 2007) and is most commonly associated with the regulation of expressions of negative affect associated with symptoms of neurobehavioural disinhibition, including impulsivity, hyperactivity, and externalising behaviours (Dolan & Lennox, 2013; Galarce & Kawachi, 2013; Iacono et al., 2008; Willoughby, Kupersmidt, Voegler-Lee, & Bryant, 2011).

This study will combine the emotion regulation and behavioural control processes associated with executive functions to evaluate associated emotional/behavioural outcomes within the same time period (4.5y) (as suggested by Abar et al., 2013) using both parent and examiner-observed measures. For emotion regulation and behavioural undercontrol measures, we selected the maternal-report measures of the Behaviour Rating Inventory of Executive Function – Pre-school Version (BRIEF-P) and the Strengths and Difficulties Questionnaire (SDQ). For inhibition measures, we selected a task in which a child had to selectively suppress dominant motor responses over a temporal delay (Gift Delay, wrap). This task was precisely selected, as ER was considered secondary to the goal. For example, in the Gift Delay task, although emotions might influence on performance, the primary goal was inhibiting peeking...
at the gift while it was being wrapped, rather than controlling one's emotion about receiving the gift. All these measures will be discussed in further detail in the Methods section.
CHAPTER SEVEN: CONCLUDING SUMMARY

Shortcomings of Current Research

The studies examining the direct effects of prenatal MA exposure on emotional and behavioural adjustment are limited and inconsistent, which may be the result of a range of methodological problems. These existing limitations include: cross-sectional analyses, high rates of sample attrition, reliance on maternal self-report of child outcomes and limited consideration of the effects of confounding factors.

First, most studies look at behavioural outcomes at one or two time points, looking for associations between prenatal exposures at birth with outcomes at a desired developmental stage. One study has looked at behavioural outcomes at 3 and 5 years (LaGasse et al., 2012; Twomey et al., 2013) and several others in the later childhood years (Eze et al., 2016; Diaz et al., 2014; Abar et al., 2013) but no studies have looked at the pre-school age of 4.5 years. An investigation at 4.5 years is important because in the previous study conducted by LaGasse et al (2012), no effect of prenatal MA was associated with poor behavioural outcomes at 3 years, however there was an effect at 5 years. The difference may suggest a latent effect or that there is a confounding influence of variability, as these ages are characterised by a substantial acquisition of skills. The preschool years, specifically 4.5 years, is important, as there are rapid changes in language ability, symbolic thought, and self-understanding that enable better regulation and more goal-directed behaviour. The 4.5 year age may also serve as an important time to intervene, particularly in New Zealand, when there is already a formal a nationwide programme offering a free health and development check for 4-year-olds, with the aim to identify and address any health, behavioural, social, or developmental concerns which could affect a child’s ability to get the most benefit from school.

Second, a key reason that many studies do not track patterns over time is that there are high rates of sample attrition, which can cause bias. Adequate sample retention can be difficult to achieve when studying high-risk families over long periods of time, as these families are typically characterised by high levels of environmental instability and often become untraceable. In the IDEAL study, the cohort from the United States had higher attrition rates than the cohort from New Zealand, as many children were taken out of the care of their mother as a result of more punitive social policies for drug use in pregnancy. At the one-month follow-up, 33% of US children were no longer living with their biological mother, compared with 3% of NZ children. When investigating the reason for child protection referrals, nearly 40% of referrals in the US were related to drug-only reasons, compared with 15% in NZ. As a result, the most at-risk children are consequentially often lost to follow-up, further limiting the interpretation of study findings and making it difficult to see the effect of maternal behavioural characteristics over time. Few studies have accounted for the postnatal environment, and those that have usually averaged or aggregated variables as dichotomous variables. Despite the recognition that the postnatal child-rearing environment is an important predictor of child health outcomes, no studies have investigated or tried to characterise the patterns of risk of ongoing maternal behavioural health (ongoing drug use or maternal psychological distress) in the postnatal period.
Third, there are only a few studies that have used direct observation of behaviour or clinical performance (Derauf et al., 2012a; Kiblawi et al., 2013) to measure child outcomes, and these studies did not include triangulation of data by also gathering maternal report. The majority of published studies use maternal self-report as the primary means of assessing child emotional and behavioural adjustment problem. The lack of an objective clinical measure may mean that the results of these studies could be prone to maternal report bias, as maternal reports by mothers with mental health and substance abuse disorders have previously been shown to be unreliable in comparison to reports made by non-disordered mothers (Chi & Hinshaw, 2002; Hennigan, O’Keefe, Noether, Rinehart, & Russell, 2006; Mash & Johnston, 1983).

Fourth, few existing studies have considered how other mechanisms associated with maternal MA dependency might also contribute to child outcome, given that children born from MA-using women are acknowledged as being a double jeopardy population. Follow-up studies relating prenatal methamphetamine and other substance exposures during childhood typically use a behavioural teratology model (Himes et al., 2014; Salisbury et al., 2009). The goal here is to isolate the effects of a teratogen by controlling for effects of potentially confounding variables through study design, such as matching and/or statistics in which confounding variables are covaried (Lester et al., 2009). The variance in outcome explained by the confounding variables is essentially removed from the analysis, and the leftover unexplained variance is attributed to the teratogen. For example, evidence has shown effects of prenatal MA exposure on behaviour problems, independent of the effects of prenatal exposure to alcohol and tobacco, as well as other potentially confounding variables (LaGasse et al., 2012; Abar et al., 2013; Derauf et al., 2012a).

The behavioural teratology model is critically important because it enables researchers to determine not only whether there is a unique drug effect (e.g., drugs affect outcome when confounding factors are controlled) but also the magnitude of the drug effect (e.g., variability in the outcome measure explained by the drug alone). A limitation of the behavioural teratology approach, however, is that it does not lend itself to the study of indirect effects that might be mediating the relationship between teratogenic effects and the developmental outcome. In a developmental model, effects that are removed as confounding variables can be studied as factors that explain more of the variability in developmental outcome in the presence of teratogenic effects. In other words, these factors are included in the model, rather than controlled or removed. In addition, other factors that are hypothesised to be involved in these developmental models, such as postnatal maternal characteristics, can be included. Perhaps the single most difficult methodological issue confronting those in the field of human behavioural teratology is trying to understand causal factors, without being able to implement experimental research designs. Because clinical researchers have no control over mothers’ use of substances during pregnancy and no control of children’s rearing environment after birth, no human study will ever provide definitive evidence about the behavioural teratological effects of a drug. While it would be inappropriate to hold human studies up to the standards of animal research, it is nevertheless desirable for clinical investigators to try to identify the most plausible sources of behavioural problems in children from drug-using families.

Much of the research on drug-exposed infants has made the mistake of assuming that, because mothers in their drug and comparison groups have been matched on a limited number of demographic
variables, any differences between drug-exposed infants and comparison groups are due to teratological factors. However, alternative factors that might explain such differences, including a poor child-rearing environment provided by drug-using families and/or the transmission of behaviour problems found in drug-using parents (Aylward, 1982). The numbers of nonteratological causal explanations for behavioural problems in children of drug-using parents only become greater as children reach school-age. Several techniques are available for illuminating the plausibility of alternative causal factors in human behavioural teratology studies. One such technique is the inclusion of multiple control groups in a study. Possible comparison groups in addition to unexposed infants might be drug-exposed children in adoptive homes or children of drug-using mother who were drug-free during pregnancy. The most important technique for looking at nonteratological causal factors is the inclusion of measures that assess such possible factors. In follow-up work, researchers have chosen to look extensively at sources of variation in the child’s rearing experience, specifically exploring: family stressors, maternal mental health, family interpersonal relationships, child rearing values held by the mother, children’s perception of their parents, and mother’s style of interaction with the child. Child outcome measures need to be carefully analysed not only in relation to maternal drug use, but also to measures of nonteratological factors. It is particularly important to look at the interaction between drug exposure and other factors. For example, one needs to explore questions of whether drug effects occur only in the presence of other risk factors such as impoverished environment, or in children of only one sex. Interactions between drug exposure and environmental factors might lead to theories that drug exposure sensitises children to the effects of poor environments (Hans, 1989; Hans, 1999) and that particular environmental conditions can protect children from the effects of prenatal biological insult environments (Rutter, 1987).

It is also important to note dimensions that have not been well studied. Substance use by the children and more studies of behaviour and psychopathology will undoubtedly be undertaken as the age of children in longitudinal cohort increases. It is somewhat surprising that there is only one study of comorbidity because this is an important focus in the addiction literature, and that there are only a few studies that account for maternal psychological distress. Only a few studies have included postnatal maternal characteristics (Abar et al., 2012; Himes et al., 2014; Derauf et al., 2012) in their analytical models, and in all of these studies, the postnatal variable was dichotomous, as having used (yes/no) a particular substance over a specific time period. No studies have attempted to look describing patterns of maternal substance use and psychopathology over time.

**Concluding Summary**

An important attribute studied in pre-school-age children is emotional-behavioural adjustment. Behavioural and emotional difficulties in young children are relatively common with estimated prevalence rates between 15 and 20% (Egger & Angold, 2006; Skovgaard et al., 2007). Severe and/or persistent early onset problems that co-occur with other child, family or environmental risks may be early markers of psychopathology (Briggs-Gowan, Carter, Bosson-Heenan, Guyer, & Horwitz, 2006; Egger & Angold, 2006; Fanti & Henrich, 2010; Greenberg, Speltz, DeKlyen, & Jones, 2001).
Developmental psychologists have long discussed the idea of disorders and delay in terms of behavioural deficits that are functionally not typical of a chronological age of the child being assessed. A disorder, for example, is defined based on clusters of behaviours not considered typical for that age. Models like ND might be particularly helpful in understanding high-risk children who exhibit a complex disinhibitory psychopathology (Iacono et al., 2008) that has features of disruptive behaviour disorders, affective and anxiety disorders, cognitive impairment and poor self-regulation, all of which are symptoms not well characterised by a single diagnosis.

The construct of neurobehavioural disinhibition underpins a broad range of domains of functioning, including cognitive, social, emotional, motor and behavioural performance; making it critical in mental and physical health and behaviour-related outcomes. Converging evidence supports the assertion that (a) EF and ER undergo dramatic development in the pre-school period; (b) EF and ER have important developmental outcomes in common, particularly emotional-behavioural adjustment, school readiness and social competence; and (c) EF and ER likely draw on common neural substrates (Carlson, 2005). While it is clear that executive processes and behavioural regulation are important for the development of emotional-behavioural adjustment, the association remains unclear (Bridgett et al., 2013; Zhou, Chen, & Main, 2012). There are relatively few empirical tests examining the interrelatedness of these constructs. Studies have not yet simultaneously considered the effects of multiple, interrelated aspects of neurobehavioural disinhibition, which could provide some additional support for models of emotion and behavioural regulation.

Over the past 15 years, much concern has been raised about the potential effects of prenatal methamphetamine exposure on health, psychological and behavioural development (Lester & LaGasse, 2010). Research to date has found that methamphetamine exposed newborns are of a younger gestational age, shorter birth length, smaller head circumference, and smaller birth weight (Smith et al., 2015). There is also research to suggest that the inhibition of monoaminergic neurotransmitter systems of MA use can impact on the neurodevelopmental functioning in children prenatally exposed to MA (McDonnell-Dowling et al., 2014; Wouldes et al., 2012; Smith et al., 2012; Lester et al., 2002). As a result, children exposed prenatally to MA may be compromised in areas related to reactivity, arousal modulation, and attentional regulation (Lester et al., 2002; Lester & LaGasse, 2010). In addition, reduction in placental and fetal blood flow might result in impaired information-processing and problem-solving ability. These findings collectively suggest that prenatal MA exposure may be a risk factor for outcomes related to central nervous system (CNS) development, including emotional and behavioural regulation (Thompson et al., 2009; Lester et al., 2009; Lester et al., 2012).

In addition to MA’s adverse effects on the infant, the home environment and characteristics of mothers who used MA during pregnancy may influence their child’s development (Lester & LaGasse, 2009; Fisher et al., 2011; Lester et al., 2012). Previous research suggests that MA-using mothers are more likely to have ongoing legal difficulties, greater likelihood of developing a substance use disorder, and higher psychological distress or comorbidity at their child’s birth (Lester & LaGasse, 2009). Further, MA-using mothers were more likely to be younger, poorer, single, and less educated than non-MA-using mothers, and are more likely to use other abusive substances (Smith et al., 2006; Smith et al., 2015).
Any one or a combination of these factors has the potential to place children at risk of poor developmental and behavioural outcomes as they grow older (Lester & Tronick, 1994).

Although such factors have increasingly been controlled for in prenatal methamphetamine exposure research, the examination of important maternal characteristics that also may be more common among mothers with a history of substance use has been rare. Maternal characteristics such as low IQ, high levels of psychological distress, and ongoing poly-substance use have generally been found to predict greater emotional-behavioural problems among young children (Brennan et al., 2000; O’Leary, Slep, & Reid, 1999; Wakschlag & Keenan, 2001). Despite this understanding, little is known about the interaction between prenatal drug exposure and postnatal maternal behavioural health (ongoing substance use and psychopathology), with respect to the development of emotional-behavioural and inhibition outcomes (Carlson, 2003; Lester & Lagasse, 2010; Rhoades et al., 2011). This is especially concerning, because these experiences appear to co-occur with great frequency. Therefore, it remains unclear as to whether the adverse developmental effects for children exposed prenatally to methamphetamine are because of the drug, per se, or because of the environment in which these children are raised. Thus, when researchers are examining the effects of prenatal MA exposure on children’s development, the potential effects of other substances, environmental risk factors, and maternal characteristics should be taken into account. The combination of a hazardous environment both prenatally and postnatally creates a perfect storm of health and social complications that increase a child’s risk of mental health problems (Lineberry & Bostwick, 2006). Research findings about stressor-specific effects on underlying neural systems have the potential to yield extremely useful information about the vulnerabilities and types of targeted interventions needed by children in high-risk populations. This is especially true for children with complex conditions, who might not respond to conventional mental health services or improved life circumstances.

The present study is one of the first to examine the effects of methamphetamine exposure emotional-behavioural development while observing the effects of ongoing postnatal substance use, environmental risk, and maternal psychological distress. To our knowledge, no prior studies have systematically described postnatal MA use patterns or psychological distress patterns among substance-abusing mothers. There are two important reasons why the pattern of postnatal MA use is useful to establish. First, from a scientific standpoint, it is important to establish a pattern of use in pregnant MA users, because this information will be crucial to teasing out the impact of prenatal MA exposure and/or ongoing substance use and long-term developmental outcomes. As already suggested in the literature, different patterns of pre- and post-natal MA use might very well be associated with different developmental outcomes. Developing a typology of MA use postnatally can help refine methodology used in future studies of prenatal MA exposure and child developmental outcomes.

Second, from a clinical standpoint, it is very important to understand whether sociodemographic subgroups vary with regard to their MA use pattern. If so, interventions to reduce use during pregnancy could be tailored to have maximum impact. Moreover, it would be useful to understand if various MA use patterns are associated with different profiles of concomitant use of tobacco, alcohol, and other drugs, as well as concurrent comorbid psychological distress. Although some earlier studies have documented maternal MA use in pregnancy and the association with poly-drug use and psychological
comorbidity, little information is available about the changing patterns of this use from pregnancy into the postnatal period. The benefit of the study in New Zealand is that many of the children exposed prenatally to methamphetamine are still living with their biological mother. As a result, we are more closely able to the relationship between mother’s wellbeing and child outcomes as it changes over time.

Taken together, the strong associations between prenatal MA and maternal behaviours and poor child behavioural outcomes, the specific affect-mediated (‘hot’) cognitive deficits associated with problems in emotion regulation and behavioural undercontrol, suggest that it would be important to investigate aspects of hot cognition and associated emotional and behavioural outcomes in children exposed to prenatal methamphetamine. Moreover, the large and steadily growing number of children affected by prenatal exposure to methamphetamine, the potentially additive negative effects of early adversity and maternal comorbidity on the development of neurobehavioural disinhibition, makes this investigation important. This is particularly important while studying high-risk populations, as a series of contextual stressors, such as the potentially additive negative effects of ongoing maternal substance use and psychological distress, also have the potential to impact underlying neural systems and psychological outcomes. In practice, this has the potential to tell us more about how executive processes develop over time and how deficits may manifest behaviourally in pre-schoolers. Understanding potential problems facing MA using families can help inform future screening and early intervention procedures to facilitate healthy family functioning, and prevent more severe developmental outcomes later on in life.

**Thesis Aims and Objectives**

This study will help contribute to the small, but growing literature base on the effects of prenatal methamphetamine exposure on neurobehavioural disinhibition, by aiming to:

**Aim 1:** Chart the course of MA use patterns and concurrent psychological symptoms of distress of women who used MA in pregnancy, and to compare rates of abstinence and relapse up to 4.5 years after delivery.

**Question 1.** What are the MA use patterns of mothers who use methamphetamine during pregnancy, in terms of quantity and frequency changes over the course of pregnancy to 4.5 years postpartum?

**Question 2:** Is continued MA use over the course of pregnancy and up to 4.5 years postpartum indicative of a more severe drug problem, and thus associated with greater use of alcohol, tobacco, and marijuana?

**Question 3.** How do mothers with different patterns of MA use differ with respect to sociodemographic characteristics?
Question 4: What are the psychological distress patterns of mothers who use methamphetamine during pregnancy over the course of 1 month to 4.5 years postpartum compared with a control group?

Question 5: How do mothers with different patterns of MA use differ with respect to their psychological functioning over time?

Aim 2: Examine the associations between prenatal MA exposure and indicators of maternal behavioural health, postnatal drug use and psychological functioning, on child neurobehavioural disinhibition measures at 4.5 years.

Question 1. What is the association between prenatal MA exposure and child neurobehavioural disinhibition at 4.5 years?

Question 2. Are there other infant, maternal or socio-environmental factors that place pre-school children exposed prenatally to MA at increased risk of poor neurobehavioural outcomes? Is this effect independent of the main effect of prenatal exposure?
CHAPTER EIGHT: METHODOLOGY

Overview

Data for this study comes from the New Zealand site of the Infant Development Environment and Lifestyle (IDEAL) Study, a large, multinational, longitudinal study of prenatal methamphetamine exposure on infant and childhood outcomes in the US and NZ being run in collaboration with Professor Barry Lester and Associate Professor Linda LaGasse at the Brown Research Center for the Study of Children at Risk in Providence, Rhode Island. The IDEAL study involves five clinical sites in specific geographic locations known to have MA problems – Los Angeles, CA; Des Moines, IA; Tulsa, OK; Honolulu, HI and Auckland, New Zealand. Data for this study involves only the Auckland, New Zealand site, as this is the only site where data was collected at 4.5 years. The inclusion of the New Zealand site provides an opportunity to determine whether findings generalise to another culture where there is no legal mandate to report mothers to child protective services (CPS) for illicit drug use during pregnancy, where perinatal care is available at no cost and economic assistance provides a variety of monetary benefits for substance dependent mothers (Woudes et al., 2013).

The study protocol and consent forms were approved by the NZ Ministry of Health Regional Ethics Committee, and both Auckland and Waitemata District Health Boards (DHBs) and affiliated Māori ethics committees. Confidentiality regarding maternal substance use was consistent with NZ Ministry of Health Ethics Committee guidelines. Policies did not require mandatory reporting of MA use during pregnancy, however mothers were informed that any evidence of child abuse or neglect required a referral to Child Youth and Family Protective Services.

Study Participants

In NZ, recruitment was conducted through referrals from maternity services at participating hospitals and through independent midwife practices. These 418 referrals were screened prior to birth to determine if the mother met the study criteria (n=308; 79%). Mothers were excluded if they were under 17.5 years of age (13.6%; n=11); displayed low cognitive functioning (3.7%; n=3); were overtly psychotic or had a documented history of psychosis (2.5%; n=2); used opiates, lysergic acid diethylamide, phencyclidine, hallucinogens or cocaine only during pregnancy (19.8%; n=16); or were non-English speaking (1.2%; n=1). An additional 34 mothers (42.0%) were excluded for various other reasons including the mother being incarcerated or institutionalized, plans to move or living a distance from the study site that was prohibitive for follow-up. Exclusion criteria for infants included critical illness or being unlikely to survive (6.2%; n=5); multiple gestations (4.9%; n=4); and a major life-threatening congenital anomaly or documented chromosomal abnormality associated with mental or neurological deficiency (6.2%; n=5).

If the mother agreed, the study staff met with her to explain the study in detail and obtain written consent to participate. NZ study staff met with the mother again post-partum prior to discharge to review the study protocol, affirm consent, collect meconium from all infants and obtain substance use and lifestyle
data consistent with the US protocol. Among the 308 eligible women approached for the study, 239 mother-infant dyads (78%) provided informed consent to participate in the study. To ensure confidentiality, all participating mother-infant dyads were coded using only a numeric identifier.

With informed consent, mothers were interviewed in the hospital to determine the presence or absence of licit and illicit prenatal drug use, information regarding the course of pregnancy, the number of prenatal care visits and sociodemographic information (the Recruitment Lifestyle Interview). Interviewers were trained and certified in the administration of maternal interviews and used scripted introductions to ensure consistency.

Meconium was collected for most infants (n=221; 92%). Meconium samples were collected in the nursery and collection began immediately to obtain the first and/or earliest discharge meconium. The samples were shipped to a central laboratory in the US (United States Drug Testing Laboratory in Des Plaines, Illinois) for analysis of the amphetamine class, cocaine metabolites, cannabinoids, opiates, and cotinine. The specimen was initially screened with an enzyme multiplied immunoassay test (EMIT II: Dade-Behring, Cupertino, California). Positive tests were then analysed using gas chromatography-mass spectrometry to identify the specific drug analyte or metabolite. Information on specific collection procedures and toxicology assays has previously been reported (Smith et al., 2006; Della Grotta et al., 2009). Prenatal exposure to alcohol, tobacco, and marijuana was included in both groups as background variables.

Among the consented, MA use was determined by maternal self-report of any MA use during pregnancy and/or positive meconium toxicology. Of those exposed (n= 108), 105 were identified by self-report only with positive toxicology, and 3 denied use but had a positive toxicology screen. The 131 matched comparison participants denied methamphetamine use during this pregnancy and had a negative meconium screen. The exposed and comparison groups were group matched based on self-identified ethnicity, infant birth weight (<1500 g, 1500-2500 g, >2500g), and maternal education (achievement of 5th form certificate or equivalent according to the National Certificate of Educational Achievement [NCEA] in NZ, or not).

Postnatal follow-up visits were conducted in a research clinic when the child was 1-month, 1 year, 3 years and 4.5 years of age with each visit maintaining a retention rate of 83% or higher (Figure 3). A summary of the data collected at each time point is below in Table 3. At each assessment, mothers were given $40.00 in cash for their participation. Those who drove to the research clinic were provided a $20.00 petrol voucher. A taxi was provided for those who did not drive or did not own an automobile. The children received a colouring book and coloured pencils as part of the Gift Delay task, which they could take with them as a token of appreciation for their participation and hard work. Mothers were also provided a $10.00 gift voucher if they notified us of any change in address or contact details as an incentive to remain in the study. In cases whereby consent for video footage was given, the participants were also gifted a copy of the obtained footage as a memory of the experience of their child on that day.
Table 2. Measures Collected at Each Time Point

<table>
<thead>
<tr>
<th>Measures</th>
<th>Assessment</th>
<th>Prenatal</th>
<th>1m</th>
<th>1y</th>
<th>3y</th>
<th>4.5y</th>
</tr>
</thead>
<tbody>
<tr>
<td>Maternal &amp; Psychosocial Factors</td>
<td>Lifestyle Interview (Appendix E)</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td></td>
<td>Substance Use Inventory (Appendix F)</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td></td>
<td>Brief Symptom Inventory (Appendix G)</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>Child Outcome Measures</td>
<td>Behavior Rating Inventory of Executive Function (BRIEF-P)</td>
<td>X</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Strengths and Difficulties Questionnaire (SDQ)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>X</td>
</tr>
<tr>
<td></td>
<td>Gift Delay (wrap)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>X</td>
</tr>
</tbody>
</table>

Figure 3. Retention percentages (%) at each follow-up visit by age (n=239).

The sample for this study included all children evaluated for neurobehavioural disinhibition at 4.5 Years (n=198; MA-exposed = 96; Comparison = 102). This is a follow-up rate of 83%. It is important to note that the main reason for smaller numbers at the 54 month visit compared with recruitment was not because of attrition or lack of follow-up, but primarily because the IDEAL study did not receive funding until some children were out of window, or over 4.5 Years ± 6 weeks of age. Hence, there were smaller follow-up numbers during this visit. Only a few participants could not be followed-up because they had moved out of country. There was one death because of illness, and one death by suicide.

Since the study was investigating maternal postnatal substance use and psychological distress and the impact on child outcomes, only those children with whom their primary caregiver was their biological mother were included in this analysis (n=180; MA exposed = 83; Comparison = 97). This is a follow-up rate of 75%. No significant differences in maternal and infant characteristics were found between the 180 participants included and the 59 nonparticipants who were not included (Table 2; p > 0.05), except mothers who were included were more likely to have smoked in pregnancy, and mothers who were excluded were more likely to have attended ≤ 4 prenatal visits during their pregnancy. The fact that these women struggled
to attend their prenatal visits may be precisely the reason why they were challenging to follow-up with over the 4.5 Years of the study period, and may suggest that engagement with this particular group of women is challenging.
Table 3.

Comparison of Mother-Infant Dyads Included and Not Included in the Study

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Included (n=180)</th>
<th>Not Included (n=60)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Maternal/Demographic</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ethnicity</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>NZ European</td>
<td>97 53.9%</td>
<td>24 40.0%</td>
<td>.09</td>
</tr>
<tr>
<td>Māori</td>
<td>60 33.3%</td>
<td>24 40.0%</td>
<td></td>
</tr>
<tr>
<td>Pacific Islander</td>
<td>17 9.4%</td>
<td>8 13.0%</td>
<td></td>
</tr>
<tr>
<td>Asian</td>
<td>6 3.3%</td>
<td>3 5.0%</td>
<td></td>
</tr>
<tr>
<td>Annual Household Income &lt; $20,000 (NZ)</td>
<td>43 23.8%</td>
<td>15 25.0%</td>
<td>.81</td>
</tr>
<tr>
<td>Living Alone at Birth</td>
<td>31 17.2%</td>
<td>9 15.0%</td>
<td>.71</td>
</tr>
<tr>
<td>Education &lt; 5th Form Certificate</td>
<td>103 57%</td>
<td>35 58.3%</td>
<td>.81</td>
</tr>
<tr>
<td>Age, Yrs</td>
<td>25.8 6.6</td>
<td>26.1 5.9</td>
<td>.79</td>
</tr>
<tr>
<td>Prenatal Visits, ≤ 4</td>
<td>3 1.6%</td>
<td>6 10.0%</td>
<td>.003</td>
</tr>
<tr>
<td>History of Sexual Abuse</td>
<td>41 2.3%</td>
<td>10 16.7%</td>
<td>.44</td>
</tr>
<tr>
<td>History of Emotional Abuse</td>
<td>122 6.7%</td>
<td>32 53.3%</td>
<td>.17</td>
</tr>
<tr>
<td>History of Physical Abuse</td>
<td>75 41.7%</td>
<td>23 38.3%</td>
<td>.98</td>
</tr>
<tr>
<td>Prenatal Methamphetamine Use</td>
<td>83 46.1%</td>
<td>25 41.7%</td>
<td>.62</td>
</tr>
<tr>
<td>Heavy Methamphetamine Use (≥3 days/wk)</td>
<td>8 4.4%</td>
<td>4 6.7%</td>
<td>.61</td>
</tr>
<tr>
<td>Prenatal Alcohol Use</td>
<td>107 59.4%</td>
<td>33 55.0%</td>
<td>.64</td>
</tr>
<tr>
<td>Heavy Alcohol use (≥5 drinks/session)</td>
<td>38 21.1%</td>
<td>5 8.3%</td>
<td>.28</td>
</tr>
<tr>
<td>Prenatal Marijuana Use</td>
<td>72 40.0%</td>
<td>26 43.3%</td>
<td>.58</td>
</tr>
<tr>
<td>Heavy Marijuana use (≥0.5 points/day)</td>
<td>46 25.6%</td>
<td>16 26.7%</td>
<td>.81</td>
</tr>
<tr>
<td>Prenatal Tobacco Use</td>
<td>130 72.2%</td>
<td>33 55.0%</td>
<td>.02</td>
</tr>
<tr>
<td>Heavy Cigarette use (≥10cigs/day)</td>
<td>50 27.8%</td>
<td>13 21.7%</td>
<td>.37</td>
</tr>
<tr>
<td>Prenatal Other Illicit Drugs</td>
<td>15 8.3%</td>
<td>9 15.0%</td>
<td>.13</td>
</tr>
</tbody>
</table>

Infant Characteristics

<table>
<thead>
<tr>
<th></th>
<th>Included (n=180)</th>
<th>Not Included (n=60)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male gender</td>
<td>98 54.4%</td>
<td>31 51.7%</td>
<td>.79</td>
</tr>
<tr>
<td>Birth weight, g</td>
<td>3413 508</td>
<td>3468 551</td>
<td>.49</td>
</tr>
<tr>
<td>Low birth weight, &lt;2500g</td>
<td>7 3.9%</td>
<td>4 6.7%</td>
<td>.36</td>
</tr>
<tr>
<td>Gestational age, wk</td>
<td>39.4 1.4</td>
<td>39.3 1.5</td>
<td>.48</td>
</tr>
<tr>
<td>Length, cm</td>
<td>51.1 2.4</td>
<td>51.3 2.7</td>
<td>.54</td>
</tr>
<tr>
<td>Head circumference, cm</td>
<td>34.8 1.7</td>
<td>34.9 1.4</td>
<td>.61</td>
</tr>
</tbody>
</table>

Note: Data are presented as n (%) or mean ±SD.

Measures and Procedures

The follow-up cohort was assessed at 1 month, 1 year, 3 years, and 4.5 years. Mothers were contacted to schedule a visit when the child was within window of the approximate age range for that visit. Trained, certified examiners masked to exposure status in a single videotaped laboratory session tested children individually. The 4.5-year assessment averaged about 1 hour and 30 minutes. As is standard practice in individual differences research, measures were presented in a fixed order. While the child was given various assessments, a trained interviewer administered the following subset of measures to the mother:
Maternal Measures

*Lifestyle Interview (LI).* The LI was developed for the Maternal Lifestyle Study, a study that examined information about cocaine-abusing mothers, and has been modified slightly for MA-abusing and New Zealand mothers (Bauer et al., 2002; Lester et al., 2002; Lester et al., 2001). The LI was a structured interview administered by certified interviewers aware of exposure status. Information gathered included the course of pregnancy, household composition, demographics (SES, education, age, ethnicity, and marital status), type of home and environment, services the family receives, changes in residency, and details about child, youth and family services involvement. Participants completed the LI at each visit to update previous information. Administration time was approximately 30 min. The LI, administered at the child’s birth, 1-month, 1-year, 3 years, and 4.5-year visits, was used in this study.

*Substance Use Inventory (SUI).* The SUI (Della Grotta et al., 2010) is a structured interview that elicits retrospective reports of frequency and quantity of drug and alcohol use. The SUI was based on the Maternal Inventory Substance Use (MISU) inventory (Shankaran et al., 2004), a well-validated tool developed on the basis of self-reported tobacco use (Patrick, Cheadle, Thompson, Koepsell, & Kinne, 1994). In this study, the SUI was administered at enrolment and focused on reports of frequency and quantity of drug and alcohol use during each trimester of the pregnancy and the 3 months prior to pregnancy. The questions were designed by using similar language and items regarding prenatal substance use from related studies (Jacobson, Chiodo, Sokol, & Jacobson, 2002; Shankaran et al., 2004; Singer et al., 1997). The mothers were asked to recall which drugs they had used during this pregnancy, how often, how much, and the maximum amount used. Target drugs on the SUI included MA, ecstasy, other amphetamines, cocaine, tobacco, alcohol, marijuana, hashish benzodiazepines/ tranquilizers, barbituates/sedatives, methadone and other opiates. Interviews conducted at the postnatal follow-up assessments asked the same frequency and quantity questions for drug and alcohol use as did the SUI at enrolment, except the reference period at the 1-month follow-up was use over the past one month, and the remaining assessments were use over the past year. Cut-offs for levels of drug use were based on thresholds for detecting effects that have been reported by others (Fried & O’Connell, 1987; Jacobson & Jacobson, 1994) and are consistent with other IDEAL (Derauf et al., 2012a; Smith et al., 2008) studies. Heavy methamphetamine exposure was defined as maternal use ≥ 3 days per week; heavy alcohol use as ≥ 5 drinks/session; heavy marijuana use as ≥0.5 joints/day; and heavy cigarette use as ≥10 cigarettes/day across pregnancy. Reliability over time for the SUI is demonstrated by comparing self-report of prenatal substance use reported on the LI at recruitment versus the self-reported use on the SUI at enrolment. Less than 1% of mothers changed their report of prenatal substance use at that time. Administration time was approximately 15-20 min.

*The Brief Symptom Inventory (BSI).* The BSI (Derogatis, 1993), a 53-item questionnaire used to assess nine patterns of clinically relevant psychological symptoms. The BSI has been used in a variety of clinical settings as a mental health-screening tool. A certified interviewer read out each question to the mothers and asked the participants to rate the extent to which they have been bothered (0= “not at all” to 4= “extremely”) in the past week by various symptoms. The results were then computer-scored to yield measures on the following symptom scales: Somatization (SOM); Obsessive-Compulsive (O-C);
Interpersonal Sensitivity (I-S); Depression (DEP); Anxiety (ANX); Hostility (HOS); Phobic Anxiety (PHOB); Paranoid Ideation (PAR); and Psychoticism (PSY). The two composite scales include: (1) the Global Severity Index (GSI), which measured overall psychological distress level based on the number and severity of symptoms; and (2) the presumptive positive diagnosis of psychiatric disorders. These criteria have been established as a procedure for determining a positive diagnosis for a psychiatric disorder based on adult, non-patient norms: (1) a T-score of 63 or greater on the GSI, or (2) T-scores of 63 or greater on any two primary symptom dimensions (Derogatis, 1993). For easy reference, the composite measure will be referred to as a positive psychiatric diagnosis. According to studies by Aroian & Patsdaughter (1989), and Derogatis (1993) the BSI instrument has good internal reliability showing an average rating between .70 to .85 and high test-retest reliability from .68 to .91. This test is commonly correlated with the Symptom Checklist-90-Revised (SCL-90-R) test, which also has been deemed reliable in assessing functional, psychosocial, and psychological status (Derogatis, 1993). The GSI of the BSI has a test-retest reliability coefficient of .90, thereby providing strong evidence that the BSI represents consistent measurement across time (Derogatis, 1993). For this study, individual scales were used to look at descriptively at patterns of psychological distress over time during the postpartum period. For the main study variable, however, the GSI scores at age 1-month, 1 year, 3 years and 4.5 years, were averaged to obtain an overall score of maternal psychological distress. The Cronbach’s α for individual scales in this study ranged from .77 to .87, and from .94 to .95 for the global scale. Test-retest reliability coefficient on global and individual scales were moderate and varied from .51 to .70.

The Peabody Picture Vocabulary Test, Third Edition (PPVT-III). The PPVT-III (Dunn & Dunn, 1997) was administered at the 30-month home visit. This is a brief screening measure used to assess maternal verbal comprehension. High reliabilities have been reported, with a Cronbach’s α ranging from .73 to .84 and test-retest reliability varying from .76 to .79. The PPVT-III is highly correlated with various IQ scales, as well as maternal education (Bell, Lassiter, Matthews, & Hutchinson, 2001; Jacobson et al., 1991).

The Infant/Toddler Home Observation for Measurement of the Environment (HOME). The HOME (Caldwell & Bradley, 2003) was used at 30 months to assess the quality and quantity of stimulation and support available to a young child (from birth to 3 years of age) in the home environment, computed as an overall score. The inventory contains 45 items representing the following six scales: emotional and verbal responsibility of the mother, avoidance of restriction and punishment, organisation of the environment, provision of appropriate play materials, maternal involvement with the child, and opportunities for variety in the daily routine. Items were scored on the basis of information obtained from the answers to the questions of the semi-structured interview and from direct observation of the family home environment by a trained assessor. Higher total HOME scores indicate more enriched home environments. Internal consistency coefficients for the different subscales ranged from .44 to .89, with an internal consistency coefficient for the total scale of .80 (Bradley, 1994). The empirical validity of this measure has been widely explored, with results supporting the ability of the inventory to distinguish among environments varying in terms of several indices of quality (Totsika & Sylva, 2004). Several studies have found that the HOME can

**Neurobehavioural Disinhibition Measures at 4.5 Years**

*The Gift Delay (wrap).* The Gift Delay (wrap) task (Kochanska, Murray, Jacques, Koenig, & Vandengeest, 1996) is a measure of delay of gratification for children aged 22 months and above. In this EF task, children are required to suppress a dominant response for a particular period of time. The Gift Delay (wrap) task is therefore categorised as a simple response inhibition task, measuring inhibitory control in isolation, placing no working memory demand on the participants. Children were told they had done a great job with the previous tasks they had completed, and were informed they were going to receive a prize. However, the experimenter “forgot” to wrap it. The experimenter asked the child to turn around in their seat, so that he or she could wrap the present “so it will be a big surprise” and reminded the child not to peek. The experimenter then pretended to wrap a small gift noisily in a standardised manner (rifling through a paper bag, cutting wrapping paper with scissors, rustling the wrapping paper and tearing off tape) for 60 seconds. Latency to peek (in seconds) was recorded. A higher score indicated that the child was less likely to peek, indicating better inhibition.

*The Behavior Rating Inventory of Executive Functioning—Pre-school Version (BRIEF-P).* The BRIEF-P (Gioia, Andrews, & Isquith, 1996) is a parent informant report of children’s (aged 2- to 5- years) everyday self-regulatory functioning in their real-world environment. It contains 63 items, which are summed to form five scales: Inhibit (16 items), Shift (10 items), Emotional Control (10 items), Working Memory (17 items), and Plan/Organize (10 items). These scales yield three composite indices: Inhibitory Self-Control Index (ISCI; Inhibit + Emotional Control), Flexibility Index (FI; Shift + Emotional Control), and Emergent Metacognition Index (EMI; Working Memory + Plan/Organize). The overall composite index is the Global Executive Composite (GEC). BRIEF-P scales and indices yield T scores (with population $M = 50$, $SD = 10$) based on age and gender. Higher scores indicate more EF problems. The nationally representative standardization sample of 2- to 5- year-old children ($N = 460$) was 54% male. The BRIEF-P has adequate reliability and validity (Sherman & Brooks, 2010). Each of the scales demonstrated strong internal consistency; Cronbach’s alphas were high ($\alpha = .80–.90$). Test–retest reliability coefficients were generally high (.80–.89) to very high (.90 +) for an average four-week interval, indicating high temporal stability (Gioia et al., 2003). Pre-school-age children diagnosed with ADHD had significantly higher BRIEF-P scores than typically developing children, but BRIEF-P scores had low, non-significant correlations with performance-based measures of working memory and inhibitory control (Mahone & Hoffman, 2007). Hence the BRIEF-P measures used in this study were analysed separately from the performance-based Gift Delay (wrap) task. The Emotional Control and Inhibit scales were the only subscales included in this study, as they relate most closely with the neurobehavioural disinhibition construct. For this study, the BRIEF-P was administered via interview to the mother at the 4.5-year visit with an internal consistency of .82.

*The Strengths and Difficulties Questionnaire (SDQ).* The SDQ (Goodman, 1997) was completed by the child’s mother at the 4.5-year follow-up assessment. The SDQ is an internationally applied and
validated screening questionnaire. It assesses mental and behavioural difficulties and strengths of 3 to 16 year old children along important domains of child psychopathology, including emotional symptoms, conduct problems, hyperactivity-inattention and peer problems, as well as personal strengths (e.g., prosocial behaviour). Each scale consists of five items, each rated on a three-point scale (“not true,” “somewhat true,” “certainly true”). Higher scores indicate more problems and/or more serious problems. Reliability is generally satisfactory, whether judged by internal consistency (α = .73) or test-retest reliability (.62) (Goodman, 2001). The SDQ correlates substantially with other indexes of psychopathology such as the Rutter and Achenbach questionnaires (Goodman, 1999; Goodman, Ford, Simmons, Gatward, & Meltzer, 2000; Lengua, Sadowski, Friedrich, & Fisher, 2001). Moreover, the SDQ discriminates well between children with and without psychopathological symptoms (Goodman et al., 1999) and there is evidence to indicate that it can be employed as an effective screen for child psychiatric disorders in community samples (Goodman, Renfrew, & Mullick, 2000; Goodman, 2001). This study used only the conduct and hyperactivity-inattention problem subscales from the SDQ, as these relate to the components of emotion dysregulation and behavioural undercontrol of neurobehavioural disinhibition. For this study, the SDQ was administered via interview to the mother at the 54-month visit, with an internal consistency of .73.

**Covariates**

Covariates were selected based on previous research on prenatal drug exposure and later outcomes (e.g., see Lester & Lagasse, 2010 for a review). Child gender (0=female, 1=male) was included as a covariate, as was child ethnicity (Māori vs. Non-Māori), low socioeconomic status (<$20,000NZD), maternal education (<5th Form Certificate), living alone at birth (yes/no), HOME, PPVT, maternal age at birth, gestational age, and birth growth parameters (birth weight, length and head circumference). Data on prenatal use of tobacco, alcohol and marijuana while pregnant was obtained from the SUI at enrolment. Child general cognitive functioning (IQ) at 4.5 years ± 6 weeks, was also included as a covariate, as low IQ can influence behavioural outcomes associated with neurobehavioural inhibition, and there is potential for confounding influence. This was measured using *The Wechsler Preschool and Primary Scale of Intelligence – Third Edition* (WPPSI-III; The Psychological Corporation, 2002). The WPPSI-III subscales of verbal abilities, processing speed, and performance abilities were standardized and summed to produce a composite IQ score. The Full Scale IQ (FSIQ) is considered the most representative estimate of global intellectual functioning. The WPPSI-III has extensive standardisation data and satisfactory psychometric properties. These covariates represent extraneous factors chosen to help isolate unique associations between prenatal MA exposure and maternal behavioural health (postnatal drug use and psychological distress) on neurobehavioural disinhibition.

**Statistical Analysis**

*Exploratory Data Analysis*

Prior to analysis, all data was examined through various statistical and graphical measures for accuracy of data, missing values, fit between their distributions and violation of the assumptions underlying the statistical techniques used. The variables were examined separately for the MA exposed women and
the comparison group. A power analysis was conducted earlier as part of the larger longitudinal nature of the IDEAL study, based on retaining 80% of the sample.

For most variables, few missing values were observed (≤ 3.3% of cases). However, the BSI at 1 month and 1 year had missing values on more than 5% of cases. This is consistent with some of the time points at which there were the lowest rates of follow-up (89.1% - refer to Figure 3). These variables were considered important to the analysis, so were not deleted. Furthermore, no pattern of missing values was apparent (Little’s MCAR, EM; p ≥ 0.05). As a result, it was determined safe to exclude cases listwise in subsequent analyses.

Any identified outliers were first checked against original scoring sheets to protect against any data entry errors. Several remaining outliers were identified on the BSI and BRIEF-P measures. There are various procedures to address influential outliers. The data may be transformed, or the outlier can be eliminated or adjusted so that it is one unit larger than the next largest score or one unit smaller than the next smallest score in the data for that variable (Tabachnick & Fidell, 2013). It was acknowledged that outliers are not uncommon in data sets derived from high-risk populations, and cases with extreme scores were considered to be a legitimate part of the sample, and therefore remained in the analysis. However, these variables were transformed in order to reduce their impact.

Normality was assessed through an examination of skewness and kurtosis values, means, medians, histograms and box-plots. The majority of the data deviated from normality. This was particularly the case on the BSI and BRIEF-P variables, where means and medians were significantly different, and skewness and kurtosis was greater than the acceptable ± 1.00 (Tabachnick & Fidell, 2013). These measures were positively skewed and had kurtosis values well above zero, indicating a distribution that is too peaked, with short, thick tails. While deviations from normality are not uncommon in research involving samples from high-risk population and normality is not always required for analysis, it was felt that the degree of deviation from normality would make a substantive difference in the analysis. The use of logarithmic, square root, and inverse transformations were explored (Tabachnick & Fidell, 2013). Given that the data differed substantially and was positively skewed, the logarithmic transformation was most successful in normalising these variables and also helped to serve as a remedy for outliers.

Data Analysis

Maternal and infant demographic characteristics were examined using independent samples t-tests for continuous measures or Chi-square for categorical measures. A one-way analysis of variance (ANOVA) was used to compare more than two groups. Hierarchical linear models and SPSS (version 22.0, IBM Corp, Armonk, NY) examined the effects of prenatal MA exposure and maternal behavioural health (postnatal drug use and psychological distress symptoms) on child neurobehavioural disinhibition measures at 4.5 years of age. The models were specified to address the primary research questions while considering potential covariates. On conceptual grounds, child gender and ethnicity were included on all models. Covariates were selected based on conceptual reasons, published literature, and characteristics from Table 3 that differed among methamphetamine exposure groups (p < .05) and if not highly correlated (r < .7) with other covariates. To provide uniformity across analyses, covariates that met criteria for one outcome were retained in all models. The final covariate set included child gender; child ethnicity (Māori vs. Non-Māori); child FSIQ; prenatal exposure to alcohol, tobacco and marijuana (yes/no); low income
(<$20,000 NZD) at birth; mother’s age and mother’s education (≤5th form certificate). In all analyses, the focus was on the estimation of the magnitude of hypothesised relationships (β co-efficient) and the 95% confidence intervals, with statistical significance levels reported to aid interpretation.
CHAPTER NINE: RESULTS

Maternal and Infant Characteristics at Birth

The total sample size was 180, with 83 MA-exposed mother-child dyads, and 97 comparison dyads. Table 4 provides descriptive information about the maternal and infant characteristics at birth by prenatal MA exposure in pregnancy. Overall, demographically the cohort was 53.9% New Zealand European, 33.3% Māori, 9.4% Pacific Islander and 2.8% Other. No significant differences were found between the two groups in terms of ethnicity ($p > .05$), infant birth weight ($p > .05$), or maternal education ($p > .05$). This confirms that the two study groups were matched on the variables intended.

Methamphetamine exposed infants did not differ from the comparison group on any of the infant characteristic variables. Mothers in the prenatal exposed MA group were more likely to be of low SES (annual household income <$20,000 NZ; $\chi^2 (1, n=174) = 8.93, p < .001, \phi = .24$), living without a partner at birth ($\chi^2 (1, n=179) = 8.37, p < .001, \phi = .23$), and have histories of psychopathology ($\chi^2 (1, n=174) = 4.61, p = .03, \phi = .18$), and sexual ($\chi^2 (1, n=171) = 3.98, p = .046, \phi = .17$), emotional ($\chi^2 (1, n=173) = 10.74, p < .001, \phi = .26$), and physical abuse ($\chi^2 (1, n=172) = 13.83, p < .001, \phi = .29$), compared to the comparison group. Mothers that used MA during pregnancy were also more likely to use marijuana ($\chi^2 (1, n=180) = 31.19, p < .001, \phi = .43$), tobacco ($\chi^2 (1, n=180) = 26.97, p < .001, \phi = .39$) while they were pregnant compared with the comparison group. Not surprisingly, heavy marijuana use ($\chi^2 (1, n=177) = 24.71, p < .001, \phi = .39$), and heavy tobacco use ($\chi^2 (1, n=179) = 16.93, p < .001, \phi = .32$), were also associated with using MA during pregnancy. This shows that MA-using mothers are faced with greater psychosocial stressors including poverty, social isolation, traumatic life histories, and poly-drug use, compared with mothers who do not use MA during pregnancy. It also shows that children exposed to methamphetamine had also been exposed to a range of different drugs. In the MA exposed group, 91.6% were exposed to tobacco, 62.7% to alcohol, and 62.7% to marijuana. Only 1.1% of this group reported using MA alone; the other 98.9% reported using MA in conjunction with 1 to 5 other licit and illicit drugs.
Maternal and Infant Characteristics at Birth by Prenatal Methamphetamine Exposure

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>MA Exposed (n= 83)</th>
<th>Comparison (n=97)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ethnicity</td>
<td></td>
<td></td>
<td>.53</td>
</tr>
<tr>
<td>NZ European</td>
<td>50 60.2%</td>
<td>47 48.5%</td>
<td></td>
</tr>
<tr>
<td>Māori</td>
<td>24 28.9%</td>
<td>36 37.1%</td>
<td></td>
</tr>
<tr>
<td>Pacific Islander</td>
<td>7 8.4%</td>
<td>10 10.3%</td>
<td></td>
</tr>
<tr>
<td>Other</td>
<td>2 2.4%</td>
<td>3 3.1%</td>
<td></td>
</tr>
<tr>
<td>Annual Household Income &lt; $20,000 (NZ)</td>
<td>29 34.9%</td>
<td>14 14.4%</td>
<td>.003</td>
</tr>
<tr>
<td>Living Without A Partner</td>
<td>22 26.5%</td>
<td>9 9.3%</td>
<td>.004</td>
</tr>
<tr>
<td>Education &lt; 5th Form Certificate</td>
<td>52 62.7%</td>
<td>51 52.6%</td>
<td>.19</td>
</tr>
<tr>
<td>Age, Yrs</td>
<td>26.4 6.2</td>
<td>25.34 6.8</td>
<td>.28</td>
</tr>
<tr>
<td>Prenatal Visits, ≤ 4</td>
<td>81 97.6%</td>
<td>95 97.9%</td>
<td>.66</td>
</tr>
<tr>
<td>History of Sexual Abuse</td>
<td>25 30.1%</td>
<td>16 16.5%</td>
<td>.046</td>
</tr>
<tr>
<td>History of Emotional Abuse</td>
<td>66 79.5%</td>
<td>56 57.7%</td>
<td>.001</td>
</tr>
<tr>
<td>History of Physical Abuse</td>
<td>47 56.6%</td>
<td>28 28.9%</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>History of Psychopathology</td>
<td>57 68.7%</td>
<td>51 53.6%</td>
<td>.032</td>
</tr>
<tr>
<td>Prenatal Methamphetamine Use*</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Heavy Methamphetamine Use (&gt;3 days/wk)</td>
<td>8 9.6%</td>
<td>0 0.0%</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Prenatal Alcohol Use</td>
<td>52 62.7%</td>
<td>55 56.7%</td>
<td>.51</td>
</tr>
<tr>
<td>Heavy Alcohol use (≥5 drinks/session)</td>
<td>21 25.3%</td>
<td>17 17.5%</td>
<td>.28</td>
</tr>
<tr>
<td>Prenatal Marijuana Use</td>
<td>52 62.7%</td>
<td>20 20.6%</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Heavy Marijuana use (≥0.5 joints/day)</td>
<td>36 43.4%</td>
<td>10 10.3%</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Prenatal Tobacco Use</td>
<td>76 91.6%</td>
<td>54 55.7%</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Heavy Cigarette use (≥10/day)</td>
<td>36 43.4%</td>
<td>14 14.4%</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Prenatal Other Illicit Drugs</td>
<td>15 18.1%</td>
<td>0 0.0%</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Male gender</td>
<td>45 54.2%</td>
<td>53 54.6%</td>
<td>.96</td>
</tr>
<tr>
<td>Birth weight, g</td>
<td>3340 456</td>
<td>3476 543</td>
<td>.07</td>
</tr>
<tr>
<td>Low birth weight, &lt;2500g</td>
<td>3 3.6%</td>
<td>4 4.1%</td>
<td>.86</td>
</tr>
<tr>
<td>Gestational age, wk</td>
<td>39.23 1.5</td>
<td>39.63 1.3</td>
<td>.06</td>
</tr>
<tr>
<td>Length, cm</td>
<td>50.91 2.4</td>
<td>51.16 2.4</td>
<td>.49</td>
</tr>
<tr>
<td>Head circumference, cm</td>
<td>34.53 1.7</td>
<td>34.9 1.7</td>
<td>.09</td>
</tr>
</tbody>
</table>

Note: Data are presented as n (%) or mean ±SD. *Two cases do not have data on prenatal methamphetamine exposure because of identification by exposure of meconium only.

Trends in Methamphetamine Use Over Time

Question 1. What are the MA use patterns of mothers who use methamphetamine during pregnancy, in terms of quantity and frequency changes over the course of pregnancy to 4.5 years postpartum?

As was outlined in the first aim of the thesis, a more thorough investigation of women that used methamphetamine during pregnancy was undertaken in order to understand the trends in the nature of the drug used during and after pregnancy. The Cochran-Armitage Trend Test (one-sided) was used to test trends of MA use across pregnancy, and then from pregnancy to 4.5 years postpartum. Table 5 shows the extent to which MA use declined from pregnancy to 1 month postpartum, and then increased from 1 month to 4.5 Years postpartum. Specifically, 19.3% of women that were using MA in pregnancy were still using MA at 1 month, which increased to 36.1% at 1 year and then plateaued at 41.0% at 3 years and 39.8% at 4.5 years.
The mean number of grams used per day also declined among users over the three trimesters from 0.38g ($SD$ 0.44) in trimester one to 0.33g ($SD$ 0.58) by trimester 3. By 1 month postpartum, the mean number of grams used per day had declined to 0.04 ($SD$ 0.12). However, further trends indicate that the mean number of grams used per day increased to 0.27g ($SD$ = 0.16) by 4.5 years, which is nearly equivalent to the number of grams being used in trimester three ($M$= 0.33g, $SD$ 0.58). Maximum quantity used per day also showed a decrease from 0.59g in the first trimester to 0.64g in the second to 0.54g in the third. There was a large increase in maximum quantity from 0.10g in 1 month postpartum to 0.31g by 1 year, and by 4.5 years was 0.55g. The sample reported the highest mean (0.54g, $SD$ 0.82) and maximum grams used per day ($M$= 1.10, $SD$ 1.69) in the three months prior to pregnancy. Overall, this suggests that women using MA before pregnancy generally decreased their use over the course of pregnancy. Women that continued to use after pregnancy did so in smaller amounts, however this tended to increase over the first year postpartum.

The range over time varied to a large degree, and there was no consistent trend. Of particular note, however, is that the largest ranges reported were over the pre-pregnancy and pregnancy time periods, 0.5-10.0g and 0.03-8.0g respectively. Closer investigation revealed that only one or two participants were using such large amounts, where more common quantities of use were closer to the means. We chose not to analyse trimester effects in addition to reported analyses due to pattern of declining use and quitting during the second and third trimester.
Table 5.

Frequency and Quantity of Methamphetamine (MA) Use Among MA-Using Mothers, Pre-pregnancy to 4.5 Years Postpartum \((n = 83)\)

<table>
<thead>
<tr>
<th></th>
<th>Prior 3 months</th>
<th>First Trimester</th>
<th>Second Trimester</th>
<th>Third Trimester</th>
<th>1 Month</th>
<th>12 Months</th>
<th>36 Months</th>
<th>54 Months</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n</td>
<td>%</td>
<td>n</td>
<td>%</td>
<td>n</td>
<td>%</td>
<td>n</td>
<td>%</td>
</tr>
<tr>
<td>No Use</td>
<td>5</td>
<td>6.0</td>
<td>5</td>
<td>6.0</td>
<td>44</td>
<td>53.0</td>
<td>50</td>
<td>60.2</td>
</tr>
<tr>
<td>Use</td>
<td>78</td>
<td>94.0</td>
<td>78</td>
<td>94.0</td>
<td>39</td>
<td>47.0</td>
<td>33</td>
<td>39.8</td>
</tr>
<tr>
<td>Quantity of MA</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Avg g/day</td>
<td>0.54</td>
<td>0.82</td>
<td>0.38</td>
<td>0.44</td>
<td>0.39</td>
<td>0.60</td>
<td>0.33</td>
<td>0.58</td>
</tr>
<tr>
<td>Maximum (g/day)</td>
<td>1.10</td>
<td>1.69</td>
<td>0.59</td>
<td>0.79</td>
<td>0.64</td>
<td>1.34</td>
<td>0.54</td>
<td>1.40</td>
</tr>
<tr>
<td>Range</td>
<td>0.05</td>
<td>10.00</td>
<td>0.10</td>
<td>4.00</td>
<td>0.10</td>
<td>8.00</td>
<td>0.03</td>
<td>8.00</td>
</tr>
</tbody>
</table>
Patterns of Poly-drug Use Over Time

Question 2: Is continued MA use over the course of pregnancy and up to 4.5 years postpartum indicative of a more severe drug problem, and thus associated with greater use of alcohol, tobacco, and marijuana?

As earlier results indicated that women using methamphetamine at birth were also using a range of other substances, a further investigation of postnatal poly-drug use from 1 month to 4.5 years postpartum was undertaken. Figure 4 below shows the percentage of self-reported mothers using methamphetamine (yes/no), heavy alcohol (≥5 drinks per session; yes/no), heavy tobacco use (≥10 cigarettes in a day; yes/no), and heavy marijuana use (≥0.5 joints per day; yes/no) during pregnancy, and at 1 month, 1 year, 3 years and 4.5 years postpartum. The figure is split into patterns of poly-drug use for mothers who used methamphetamine during pregnancy compared to the comparison group.

Compared with drug use during pregnancy, there was a substantial reduction in the use of most substances from the birth of the child to 1 month postpartum. The exception was heavy alcohol use, which only declined from 25.3% to 19.8% in the methamphetamine group and 17.5% to 16.0% in the comparison group. The rates then increase again substantially from 1 month postpartum to 4.5 years postpartum. In the methamphetamine group, heavy alcohol increased from 19.8% at 1 month to 59.8% at 3 years, and then declined slightly to 42.2% at 4.5 years. In the comparison group, heavy alcohol use rose from 16.0% at 1 month to 42.3% at 4.5 years.
A steady increase for other licit and illicit drug use was observed after the birth of the baby, particularly in the first year postpartum. These trends were tested using the Cochran's Armitage test with Bonferroni adjustments on the p-value as multiple comparisons were performed. Methamphetamine use, heavy alcohol use and heavy marijuana use demonstrated a significant increase \( (p < 0.01) \) over time among the women who used methamphetamine during pregnancy. Using the McNemar’s test with Bonferroni adjustments on the p-value \( (p < 0.01) \) for multiple comparisons, the most significant jumps were found to be between 1 month and 1 year postpartum. Heavy tobacco use remained relatively stable from pregnancy to 4.5 years postpartum among women who used methamphetamine during pregnancy. Among the comparison group, heavy tobacco and heavy alcohol use demonstrated a significant increase from pregnancy to 4.5 years postpartum. The most significant jumps, according to the McNemar’s test with Bonferroni adjustments on the p-value \( (p < .001) \) were again between 1 month and 1 year.

There was substantial heterogeneity with respect to individual MA use patterns over time among women in the sample. Approximately 20.5\% (n=17) of mothers who used MA during pregnancy were classified as consistently high or “chronic” users across time. These are women that used MA at least three of the four follow-up time points. Of these, there were 6.0\% (n=5) of mothers who used MA during pregnancy that continued to use at all time follow-up time points. There was a larger proportion of women (45.8\%, n=38) who were classified as “pausers”, as they picked up using MA again at least at one time point in the follow-up period. The remaining 33.7\% (n=28) were “abstainers” or women that gave up use of MA after pregnancy. There were also 12.4\% (n=12) of “postpartum initiators” or women from the comparison group that used at least once after pregnancy. 11.3\% (n=11) of these women used at one time point and 1.0\% (n=1) used at three time points during the postpartum period. None of the comparison women that started using in the postnatal period reported using methamphetamine in the 3 months prior to pregnancy.

Demographic and Psychosocial Characteristics of Postnatal Drug Use Groups

Question 3. How do mothers with different patterns of MA use differ with respect to sociodemographic characteristics?

After finding the various groups, it was important to see how these groups differed with respect to maternal health and social background factors. Table 6 displays the characteristics of mothers at the birth of their child by their postnatal drug use pattern determined from earlier analyses. Women who were using methamphetamine during pregnancy were divided into three groups: chronic users, pausers and abstainers. Women who were not using methamphetamine during pregnancy were divided into two groups: postpartum initiators and never used. General linear models were applied to the postnatal drug use patterns to compare groups.

Chronic users, pausers, abstainers, and postpartum initiators were more likely to have used tobacco and marijuana in pregnancy compared with the never used group. No statistical differences between groups were found on alcohol use, both on any or heavy use in pregnancy. Chronic users were more likely to live alone at birth compared with those that never used methamphetamine or heavy licit or illicit drugs postnatally. Some use in the postpartum period by mothers that used methamphetamine in pregnancy (“pausers”) was also associated with living alone at birth compared with never used, postpartum
initiators, and abstainers. Mothers in the pauser, abstainer and postpartum initiator groups were also more likely to come from low socioeconomic backgrounds compared with those women that never used. Mothers in the postpartum initiator and pausers groups were also more likely to have reported a history of emotional abuse compared with those that never used in the postpartum period. The pausers group was more likely to report a history of physical abuse compared with those that never used or abstained use after pregnancy. As expected, the never used and postpartum initiators group were less likely to have used any alcohol/drug treatment support over the postnatal study period. Also, the abstainers group were less likely to have entered formal alcohol/drug treatment programmes or services compared with the pausers group. Naturally, this suggests that mothers whom are continuing to use methamphetamine and alcohol, tobacco, and marijuana after pregnancy in heavy doses are more likely to be engaged with services to get support compared with those that stop their use or were not using in pregnancy. It is interesting, however, that only 1 mother out of 12 among those that started using methamphetamine and other heavy drug use in the postnatal period sought alcohol/drug treatment input. There is clearly a need for these women to access services, however, they did not do so.
Table 6.

Demographic and Psychosocial Characteristics by Postnatal Drug Group Patterns

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>MA Exposed</th>
<th>Comparison</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Chronic (n=17)</td>
<td>Pausers (n=38)</td>
<td>Abstainers (n=28)</td>
</tr>
<tr>
<td>Prenatal Tobacco (y/n)</td>
<td>17 100.0%</td>
<td>36 94.7%</td>
<td>5 17.9%</td>
</tr>
<tr>
<td>Heavy Tobacco (≥10 cig/day)</td>
<td>12 70.5%</td>
<td>18 47.4%</td>
<td>6 21.4%</td>
</tr>
<tr>
<td>Prenatal Alcohol (y/n)</td>
<td>8 47.1%</td>
<td>27 71.1%</td>
<td>17 60.7%</td>
</tr>
<tr>
<td>Heavy Alcohol (≥5 drinks/session)</td>
<td>2 11.8%</td>
<td>14 36.8%</td>
<td>5 17.9%</td>
</tr>
<tr>
<td>Prenatal Marijuana (y/n)</td>
<td>11 64.7%</td>
<td>27 71.1%</td>
<td>14 50.0%</td>
</tr>
<tr>
<td>Heavy Marijuana (≥0.5 joints/day)</td>
<td>7 41.2%</td>
<td>19 50.0%</td>
<td>10 35.7%</td>
</tr>
<tr>
<td>Heavy Methamphetamine (&gt;3 days/wk)</td>
<td>2 11.8%</td>
<td>4 10.5%</td>
<td>2 7.1%</td>
</tr>
<tr>
<td>Ethnicity</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>NZ European</td>
<td>12 70.6%</td>
<td>20 52.6%</td>
<td>18 64.3%</td>
</tr>
<tr>
<td>Māori</td>
<td>5 29.4%</td>
<td>13 34.2%</td>
<td>6 21.4%</td>
</tr>
<tr>
<td>Pacific Islander</td>
<td>0 -</td>
<td>4 10.5%</td>
<td>3 10.7%</td>
</tr>
<tr>
<td>Other</td>
<td>0 -</td>
<td>1 2.6%</td>
<td>1 3.6%</td>
</tr>
<tr>
<td>Living Alone at Birth</td>
<td>5 29.1%</td>
<td>14 36.8%</td>
<td>3 10.7%</td>
</tr>
<tr>
<td>Prenatal visits, ≤4</td>
<td>0 -</td>
<td>0 -</td>
<td>1 3.6%</td>
</tr>
<tr>
<td>Low SES (income &lt;$20,000)</td>
<td>4 23.5%</td>
<td>16 42.1%</td>
<td>9 32.1%</td>
</tr>
<tr>
<td>Education (&lt;5th Form Cert)</td>
<td>12 70.6%</td>
<td>26 68.4%</td>
<td>14 50.0%</td>
</tr>
<tr>
<td>HOME (30 months)</td>
<td>33.69 5.86</td>
<td>32.53 6.14</td>
<td>35.35 5.25</td>
</tr>
<tr>
<td>PPVT (30 months)</td>
<td>95.42 6.92</td>
<td>92.81 7.59</td>
<td>92.79 11.64</td>
</tr>
<tr>
<td>History of Emotional Abuse</td>
<td>13 76.5%</td>
<td>33 86.8%</td>
<td>20 71.4%</td>
</tr>
<tr>
<td>History of Physical Abuse</td>
<td>8 47.1%</td>
<td>26 68.4%</td>
<td>13 46.4%</td>
</tr>
<tr>
<td>History of Sexual Abuse</td>
<td>7 41.2%</td>
<td>9 23.7%</td>
<td>9 32.1%</td>
</tr>
<tr>
<td>History of Psychopathology</td>
<td>10 58.8%</td>
<td>28 73.7%</td>
<td>19 67.9%</td>
</tr>
<tr>
<td>Postnatal Alcohol/Drug Treatment</td>
<td>9 52.9%</td>
<td>23 60.5%</td>
<td>9 32.1%</td>
</tr>
</tbody>
</table>
Maternal Psychological Distress Over Time

Question 4: What are the psychological distress patterns of mothers who use methamphetamine during pregnancy over the course of 1 month to 4.5 years postpartum compared with a control group?

With respect to maternal mental health, Table 7 displays the psychological symptoms of all mothers at 1 month, 1 year, 3 years and 4.5 years following the birth of their child. An independent samples t-test was conducted at each time point to compare levels of psychological distress scores for women who used methamphetamine in pregnancy compared with those that did not, as measured by the subscales and overall General Severity Index of the Brief Symptom Inventory (BSI).

At 1 month, mothers in the methamphetamine group reported higher obsessive-compulsive, hostility, phobic anxiety, paranoid ideation and psychoticism ($p < 0.05$) symptoms than comparison mothers. At 1 year, mothers in the methamphetamine group reported higher paranoid ideation. At 3 years, mothers who were using MA at birth reported higher psychoticism. At 4.5 years, mothers in the methamphetamine group reported higher levels of interpersonal sensitivity and hostility.

A mixed between-within subjects analysis of variance (MANOVA) was conducted to assess the association of MA use in pregnancy on GSI scores across four time periods (1 month, 1 year, 3 years, 4.5 years). There were no significant interactions between MA exposure and time Wilks' Lambda = .96, $F (3,128) = 1.62$, $p = .19$, partial eta squared = .04. There was a substantial main effect for time, Wilks' Lambda = .89, $F (3,128) = 5.00$, $p < .001$, partial eta squared = .11, with both groups showing an increase in psychological distress from 1 month to 3 years, followed by a slight reduction in psychological distress at 4.5 years (see Table 7). The main effect comparing the groups was significant, $F (1, 130) = 6.86$, $p = .01$, partial eta squared = .50, suggesting that the two groups differed in their overall mental health. An investigation of the means, suggests that the women who used methamphetamine in pregnancy reported higher levels of overall psychological distress at each time point compared with women who had not used methamphetamine in pregnancy. These differences were significant ($p < .05$) particularly at 1 month and 3 years postpartum, indicating that overall, women who used MA during pregnancy reported higher levels of psychological distress at 1 month and 3 years postpartum compared with mothers that did not use MA during pregnancy. A significantly higher proportion of MA using women met the criteria for a positive psychiatric diagnosis than comparison mothers at 1 month ($\chi^2 (1, n=170) = 8.69$, $p = .002$, phi = .24) and 4.5 years ($\chi^2 (1,n=169) = 4.05$, $p = .03$, phi = .17).
Table 7.

Mean Brief Symptom Inventory (BSI) Scores for the MA Exposed and Comparison Groups From 1 Month to 4.5 Years Postpartum

<table>
<thead>
<tr>
<th></th>
<th>MA Exposed</th>
<th></th>
<th>Comparison</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>1 Month</td>
<td>1 Year</td>
<td>3 Years</td>
</tr>
<tr>
<td></td>
<td>(n=78)</td>
<td>(n=76)</td>
<td>(n=79)</td>
</tr>
<tr>
<td>Somatisation</td>
<td>-0.33 (0.35)</td>
<td>-0.29 (0.39)</td>
<td>-0.27 (0.38)</td>
</tr>
<tr>
<td></td>
<td>Obsessive Compulsive</td>
<td>-0.06* (0.33)</td>
<td>-0.11 (0.39)</td>
</tr>
<tr>
<td>Interpersonal Sensitivity</td>
<td>-0.15 (0.38)</td>
<td>-0.14 (0.38)</td>
<td>-0.11 (0.36)</td>
</tr>
<tr>
<td>Depression</td>
<td>-0.29 (0.40)</td>
<td>-0.24 (0.37)</td>
<td>-0.20 (0.38)</td>
</tr>
<tr>
<td>Anxiety</td>
<td>-0.35 (0.38)</td>
<td>-0.31 (0.36)</td>
<td>-0.22 (0.37)</td>
</tr>
<tr>
<td>Hostility</td>
<td>-0.26* (0.37)</td>
<td>-0.15 (0.40)</td>
<td>-0.18 (0.38)</td>
</tr>
<tr>
<td>Phobic Anxiety</td>
<td>-0.23* (0.38)</td>
<td>-0.26 (0.42)</td>
<td>-0.21 (0.41)</td>
</tr>
<tr>
<td>Paranoid Ideation</td>
<td>-0.10* (0.37)</td>
<td>-0.09* (0.35)</td>
<td>-0.09 (0.38)</td>
</tr>
<tr>
<td>Psychoticism</td>
<td>-0.27* (0.35)</td>
<td>-0.26 (0.36)</td>
<td>-0.19* (0.39)</td>
</tr>
<tr>
<td>GSI</td>
<td>-0.45* (0.52)</td>
<td>-0.41 (0.49)</td>
<td>-0.31* (0.42)</td>
</tr>
<tr>
<td>Positive Diagnosis</td>
<td>43* 55.1%</td>
<td>42   55.2%</td>
<td>45   56.9%</td>
</tr>
</tbody>
</table>

*Note: * indicates significance at $p < .05$. 
Maternal Psychological Functioning and Postnatal Substance Use

Question 5: How do mothers with different patterns of MA use differ with respect to their psychological functioning over time?

After identifying higher psychological distress across time among women who used methamphetamine in pregnancy, it was important to investigate how psychological distress varied according to their pattern of postnatal drug use. As no patterns of psychopathology were identified in the individual subscales earlier, postnatal psychological distress was therefore calculated as the aggregate mean of GSI scores across all time points (1 month, 1 year, 3 years and 4.5 years).

A one-way between-groups analysis of variance was conducted to explore the association of postnatal substance use patterns with levels of psychological distress, as measured by an average GSI score on the BSI. Table 8 shows the results, and means are presented diagrammatically in Figure 5. Participants were divided into five groups according to their patterns of substance use over the postnatal period established in earlier results (Group 1: "never used"; Group 2: "postpartum initiators"; Group 3: "abstainers"; Group 4: "pausers"; Group 5: "chronic users"). There was a statistically significant difference at the $p < .05$ level in average GSI scores for the five groups: $F (4, 175) = 5.56$, $p < .001$. Despite reaching statistical significance, the actual difference in mean scores between the groups was quite small. The effect size, calculated using eta squared, was 0.11. Post-hoc comparisons using the Tukey HSD test indicated that the mean score for the "never used" group ($M = -0.63$, $SD = 0.39$) was significantly different from the "pausers" group ($M = -0.28$, $SD = 0.31$). The "abstainers" group ($M = -0.57$, $SD = 0.49$) was also significantly different from the "pausers" group ($M = -0.28$, $SD = 0.31$). The "postpartum initiators" ($M = -0.37$, $SD = 0.39$) and the "chronic users" group ($M = -0.47$, $SD = 0.39$) did not differ significantly from any of the other groups. These results indicate that the women with the highest levels of psychological distress were those in the "pausers" group, or the women that used methamphetamine during pregnancy, and then used the drug again at least once in the postpartum period. Overall, the results suggest that those women that "never used" methamphetamine or that gave up their use after pregnancy ("abstainers") generally report lower levels of psychological distress compared with women that continued to use ("pausers", "chronic users") or started using after pregnancy ("postpartum initiators").
Table 8.

*Aggregated Postnatal Mental Health Outcomes (1m, 1y, 3y, 4.5y) by Postnatal Drug Group Patterns*

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>MA Exposed</th>
<th>Comparison</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Chronic (n=17)</td>
<td>Pausers (n=38)</td>
<td>Abstainers (n=28)</td>
</tr>
<tr>
<td>Psychological Functioning (GSI), M (SD)</td>
<td>-0.47 (0.39)</td>
<td>-0.28 (0.31)</td>
<td>-0.57 (0.49)</td>
</tr>
<tr>
<td>Positive Psych Diagnosis (n, %)</td>
<td>13 76.5%</td>
<td>36 94.7%</td>
<td>21 75.0%</td>
</tr>
<tr>
<td>Postnatal Mental Health Input (n, %)</td>
<td>6 35.3%</td>
<td>25 65.8%</td>
<td>17 60.7%</td>
</tr>
</tbody>
</table>

Note: GSI refers to the mean Global Severity Index (GSI) score of the Brief Symptom Inventory (BSI) aggregated from the 1m, 1y, 3y and 4.5y follow-up assessments. A Positive Psych Diagnosis (Y/N) was established by the participant endorsing either (1) a T-score of 63 or greater on the GSI, or (2) T-scores of 63 or greater on any two primary symptom dimensions (Derogatis, 1993) at any one time point during follow-up. Postnatal Mental Health Input (Y/N) refers to a participant receiving any form of mental health input (e.g., self-help, group or individual therapy) at any time point during follow-up.

*Figure 5.* Mean Global Severity Index (Log 10) of the Brief Symptom Inventory score by pattern of postnatal substance use from 1 month to 4.5 years.

*Note: Global Severity Index (GSI) scores closer to zero indicate greater mental health concerns*
Neurobehavioural Disinhibition

Question 1. What is the association between prenatal MA exposure and child neurobehavioral disinhibition at 4.5 years?

The second aim of the thesis was to examine the impact of prenatal methamphetamine exposure and maternal behavioural health (postnatal drug use and psychological functioning) on child neurobehavioural disinhibition at 4.5 years. Table 9 describes the neurobehavioural disinhibition measures of all study children at 4.5 years by prenatal methamphetamine exposure. Outcomes examined included the Inhibit and Emotional control scales from the BRIEF-P, the conduct problems and hyperactivity scores from the SDQ, and latency to peek on the lab-based Gift Delay (wrap) task. Child Full Scale IQ (FSIQ) from the WPPSI-III was included as a covariate, given its potential confounding influence on outcomes. In this analysis, there were no group differences on performance on any of these measures.

Table 9.

Child Neurobehavioural Disinhibition Outcome Measures at 4.5 years by Prenatal Methamphetamine Exposure

<table>
<thead>
<tr>
<th>Measures</th>
<th>MA Exposed (n=83)</th>
<th>Comparison (n=97)</th>
<th>p</th>
</tr>
</thead>
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<tr>
<td></td>
<td>M     SD</td>
<td>M     SD</td>
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<tr>
<td>Child Outcome Measures</td>
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<tr>
<td>BRIEF-P Inhibit</td>
<td>55.52 11.96</td>
<td>54.73 10.66</td>
<td>.64</td>
</tr>
<tr>
<td>SDQ Conduct Problems</td>
<td>2.41   2.05</td>
<td>2.19  1.87</td>
<td>.47</td>
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<tr>
<td>SDQ Hyperactivity</td>
<td>4.05   4.05</td>
<td>4.10  2.08</td>
<td>.88</td>
</tr>
<tr>
<td>Gift Delay (Wrap)</td>
<td>45.78 20.13</td>
<td>44.43 21.46</td>
<td>.67</td>
</tr>
<tr>
<td>Child Covariates</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>WPPSI-III FSIQ</td>
<td>94.02 13.02</td>
<td>97.15 13.03</td>
<td>.11</td>
</tr>
</tbody>
</table>

Prenatal Methamphetamine Exposure and Postnatal Characteristics of the Environment

Table 10 shows a summary of postnatal characteristics of the environment by prenatal methamphetamine exposure. In the postnatal period through 4.5 years, the MA exposed group were more likely to report higher levels of psychological symptoms and postnatal drug use (illicit drug use and heavy licit drug use) (p < .05) than the comparison group. Women that had used methamphetamine during pregnancy were also significantly more likely to have attended parent training, alcohol/drug treatment programmes, or mental health treatment programmes by 4.5 years (p < .05) compared with the comparison group. No differences were found between groups on the HOME inventory or maternal vocabulary PPVT measures at 30 months.
Bivariate analyses with Pearson product-moment correlation coefficients were used to explore correlational relationships between the study variables. As displayed in Table 11, a number of factors were significantly associated with the child neurobehavioural outcome measures. There were strong, positive correlations between the BRIEF-P Inhibit and BRIEF-P Emotional Control ($r=.70, p < .01$), SDQ Conduct Problems ($r=.71, p < .01$) and Hyperactivity ($r=.70, p < .01$). The BRIEF-P Emotional Control scale was also highly correlated with the SDQ Conduct scale ($r=.66, p < .01$). The SDQ Hyperactivity scale was highly correlated with the SDQ Conduct problems scale ($r=.58, p < .01$). As for the predictor variables, a child’s ethnicity as being Māori was negatively correlated with the HOME inventory ($r=-.41, p < .01$), suggesting that being Māori was associated with less enriched home environments. The HOME inventory was also modestly negatively correlated with maternal education being lower than 5th form certification ($r=-.32, p <.01$), where more enriched home environments were associated with mothers having had an education beyond 5th form certification. There was a modest, positive correlation between child FSIQ and the HOME inventory ($r=.39, p < .01$), with higher IQ scores associated with more enriched home environments. Child FSIQ was also negatively correlated with child ethnicity being Māori ($r=-.31, p < .01$), with higher IQ scores associated with not being Māori. Consistent with earlier analyses, the Pearson product-moment correlation coefficient found that prenatal methamphetamine use was modestly associated with prenatal tobacco use ($r=.48, p < .01$) and prenatal marijuana use ($r=.48, p < .01$). Prenatal marijuana use was also associated with prenatal tobacco use ($r=.41, p < .01$). Again, suggesting that mothers using one drug, are often using another, and that it is likely that the children in this study were exposed to more than one substance in utero. Maternal psychological distress was associated with low socioeconomic status (income <$20,000 NZD; $r=.31, p <.01$) and less enriched home environments on the HOME inventory ($r=-.35, p < .01$). Since many of these predictor variables were significant, the covariates selected in this study included child gender, ethnicity, prenatal alcohol exposure, prenatal tobacco exposure, prenatal marijuana exposure, child FSIQ, low SES (income <$20,000 NZD), HOME inventory, mother’s age at birth, and mother’s education. These variables were included in order to investigate potential confounding variables.
Table 11.

Pearson’s Product Moment Correlations between Predictor and Outcome Variables

<table>
<thead>
<tr>
<th>Variables</th>
<th>1</th>
<th>2</th>
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<tr>
<td>5. Low SES (Income&lt;$20,000)</td>
<td>.240**</td>
<td>.145</td>
<td>.101</td>
<td>.162**</td>
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<td>6. Maternal Education (&lt;5th Form)</td>
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<td>.138</td>
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<td>-.066</td>
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<td>-.088</td>
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<td>10. Psychological Functioning</td>
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<td>.242**</td>
<td>.128</td>
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<tr>
<td>11. Postnatal Drug Use</td>
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<td>.489**</td>
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<td>.542**</td>
<td>.166**</td>
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<td>12. Child ethnicity, Māori</td>
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<td>15. BRIEF-P Inhibit</td>
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<td>.164**</td>
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<td>.123</td>
<td>.321**</td>
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<td>17. SDQ Conduct Problems</td>
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<td>.188</td>
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<td>.351**</td>
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<td>18. SDQ Hyperactivity</td>
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<td>.700**</td>
<td>.488**</td>
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<td>19. Gift Delay (wrap)</td>
<td>.032</td>
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<td>.107</td>
<td>-.029</td>
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<td>-.156**</td>
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<td>-.128</td>
<td>-.264**</td>
<td>-.381**</td>
</tr>
</tbody>
</table>

Note. Coefficients represent correlations of variables for total sample (n = 180).

** Correlation is significant at the 0.01 levels (2-tailed)

* Correlation is significant at the 0.05 levels (2-tailed)
Neurobehavioural Outcomes by Prenatal Methamphetamine Exposure and Maternal Behavioural Health

Question 2. Are there other infant, maternal or socio-environmental factors that place pre-school children exposed prenatally to MA at increased risk of poor neurobehavioural outcomes? Is this effect independent of the main effect of prenatal exposure?

Hierarchical linear regression was used to assess how prenatal methamphetamine exposure, postnatal drug use, and maternal psychological distress predict scores of child neurobehavioural disinhibition, after controlling for the influence of child ethnicity and gender. Full models for each measure are presented in Tables 12-14 below; specifically BRIEF-P Inhibit and Emotional Control outcomes in Table 12; SDQ Conduct and Hyperactivity in Table 13 and Gift Delay (wrap) in Table 14.

Model 1

In the first model, the magnitudes of the relationship between prenatal methamphetamine exposure and child neurobehavioural disinhibition outcomes at 4.5 Years were investigated. Prenatal methamphetamine exposure was defined dichotomously (yes/no) based on combined self-report and toxicology data. Child neurobehavioural disinhibition was measured by BRIEF-P Inhibit and Emotional Control subscale T scores, SDQ Conduct Problems and Hyperactivity subscale mean scores, and Gift Delay (Wrap) latency to peek in seconds. Statistical adjustment for child’s gender and ethnicity was made in this analysis, as well as in subsequent models, to protect against the possibility that the scores based on general population reference samples did not adequately capture ethnicity and gender-related variation in scores. As shown in Table 12,13, and 14, prenatal methamphetamine exposure was not related to any neurobehavioural disinhibition measure. Accordingly, no additional analyses were run to evaluate mediating pathways between prenatal methamphetamine exposure and child neurobehavioural disinhibition.

Model 2

Model 2 estimated the relationship between postnatal maternal drug use and the child’s levels of neurobehavioural disinhibition at 4.5 years. Postnatal drug use was measured as a sum of having reported using methamphetamine, heavy alcohol, heavy tobacco, heavy marijuana, or other illicit drug use (yes/no) at 1 month, 1 year, 3 years and 4.5 years. Greater endorsement meant greater use of these substances over time. Within this model framework, the neurobehavioural disinhibition scores were regressed simultaneously with the summed postnatal maternal drug use score. Estimates reveal a statistically significant association between postnatal drug use and mother’s report of child emotional control and behaviour on the BRIEF-P Inhibit and Emotional Control measures and the SDQ Conduct measure under this model. Each standard deviation increase in level of postnatal maternal drug use was associated with a estimated .01 point increase in the level of child inhibition difficulty ($B = .01; 95\% CI .00,.01, p = .03$, an estimated .01 point increase in child emotion control difficulties ($B = .01; 95\% CI .00,.02, p = .002$), and an estimated .12 increase in the level of child conduct behaviour problems ($B =
0.12; 95% CI .05,.21, \( p = .002 \). No association between the SDQ Hyperactivity or Gift Delay (wrap) measures and maternal postnatal drug use were found.

**Model 3**

In the next step, the model was elaborated to include a mean GSI Brief Symptom Inventory score over 1 month, 1 year, 3 years and 4.5 years as an indication of maternal psychological functioning. Postnatal maternal drug use and the psychological distress composite score were modestly correlated \( (r = .43) \). Maternal psychological functioning was associated with all maternal report and lab-based measures of neurobehavioural disinhibition \( (p < .001) \). For each unit of increase in maternal psychological distress, there was an estimated .10 point increase in inhibitory control problems \( (95\% CI .07,.13) \), a .09 point increase in emotional control problems \( (95\% CI .05,.13) \), a 1.82 point increase in conduct problems \( (95\% CI 1.07,2.57) \), a 1.28 point increase in hyperactivity problems \( (95\% CI 1.38,3.18) \) and a 17.15 point decrease in delay of gratification \( (95\% CI -25.74,-8.25) \). There was a reduction in the magnitude of the estimated relationship between postnatal drug use and all levels of neurobehavioural disinhibition, once statistical adjustment for psychological distress was included in the model.
Table 12.

Estimated Effect of Prenatal Methamphetamine Exposure and Postnatal Maternal Functioning on BRIEF-P Inhibit and Emotional Control Measures

<table>
<thead>
<tr>
<th>Model</th>
<th>BRIEF-P Inhibit</th>
<th>BRIEF-P Emotional Control</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>B (SE)</td>
<td>β</td>
</tr>
<tr>
<td>Model 1 (child ethnicity and gender)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Prenatal MA use</td>
<td>0.98 (.69)</td>
<td>.04</td>
</tr>
<tr>
<td>Model 2 (child ethnicity and gender)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Postnatal drug use</td>
<td>0.01 (.00)</td>
<td>.22</td>
</tr>
<tr>
<td>Model 3 (model 2 with mother’s psychological functioning)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Postnatal drug use</td>
<td>.00 (.00)</td>
<td>.03</td>
</tr>
<tr>
<td>Psychological distress</td>
<td>.10 (.02)</td>
<td>.49</td>
</tr>
<tr>
<td>Model 4 (model 3 with prenatal methamphetamine exposure and covariates: prenatal alcohol, tobacco, marijuana; child FSIQ; HOME; income &lt;$20,000; mother’s age; mother’s education)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Postnatal drug use</td>
<td>.00 (.00)</td>
<td>-.02</td>
</tr>
<tr>
<td>Psychological distress</td>
<td>.09 (.02)</td>
<td>.44</td>
</tr>
</tbody>
</table>

Note: Child’s ethnicity was related to Inhibit scores (p<0.05) in Models 1-2 and with Emotional Control scores in Model 1. Child’s gender was related to the Emotional Control score ( p <0.05) in Models 1-4. The HOME score was associated with inhibit scores in Model 5 (p<0.05). Mother’s education level (<5th form certificate) was associated with emotional control in Model 4 (p<0.05).
Table 13.

*Estimated Effect of Prenatal Methamphetamine Exposure and Postnatal Maternal Functioning on SDQ Conduct Problems and Hyperactivity Measures*

<table>
<thead>
<tr>
<th>Model</th>
<th>SDQ- Conduct Problems</th>
<th>SDQ- Hyperactivity</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>B (SE) β 95% CI</td>
<td>p</td>
</tr>
<tr>
<td>Model 1 (child ethnicity and gender)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Prenatal MA use</td>
<td>0.20 (0.29) 0.05 -0.38, 0.78 0.498</td>
<td>0.20 (0.29) 0.05 -0.38, 0.78 0.498</td>
</tr>
<tr>
<td>Model 2 (child ethnicity and gender)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Postnatal drug use</td>
<td>0.12 (0.04) 0.27 .05, .21 .002</td>
<td>0.12 (0.04) 0.27 .05, .21 .002</td>
</tr>
<tr>
<td>Model 3 (model 2 with mother’s psychological functioning)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Postnatal drug use</td>
<td>.06 (.04) .12 -0.03, .14 .18</td>
<td>.06 (.04) .12 -0.03, .14 .18</td>
</tr>
<tr>
<td>Psychological distress</td>
<td>1.82 (0.38) .39 1.07, 2.57 &lt;.001</td>
<td>1.82 (0.38) .39 1.07, 2.57 &lt;.001</td>
</tr>
<tr>
<td>Model 4 (model 3 with prenatal methamphetamine exposure and covariates: prenatal alcohol, tobacco, marijuana; child FSIQ; income &lt;$20,000; mother’s age; mother’s education)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Postnatal drug use</td>
<td>.04 (.04) .07 -0.04, 0.12 .38</td>
<td>.04 (.04) .07 -0.04, 0.12 .38</td>
</tr>
<tr>
<td>Psychological distress</td>
<td>1.67 (.37) .37 1.93, 2.41 &lt;.001</td>
<td>1.67 (.37) .37 1.93, 2.41 &lt;.001</td>
</tr>
</tbody>
</table>

*Note:* Child’s ethnicity was related to conduct problem scores (p<0.05) in Model 1. Mother’s education level (<5th form) was related to conduct problem and hyperactivity scores (p<0.05) in Model 4.
Table 14.

*Estimated Effect of Prenatal Methamphetamine Exposure and Postnatal Maternal Functioning on Gift Delay (wrap)*

<table>
<thead>
<tr>
<th>Model</th>
<th>Gift Delay (Wrap)</th>
<th>B (SE)</th>
<th>β</th>
<th>95% CI</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Model 1 (child ethnicity and gender)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Prenatal MA use</td>
<td></td>
<td>1.15 (3.21)</td>
<td>.03</td>
<td>-5.18, 7.49</td>
<td>.72</td>
</tr>
<tr>
<td>Model 2 (child ethnicity and gender)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Postnatal drug use</td>
<td></td>
<td>-.06 (.46)</td>
<td>.89</td>
<td>-.97, .85</td>
<td>.89</td>
</tr>
<tr>
<td>Model 3 (model 2 with mother’s psychological functioning)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Postnatal drug use</td>
<td></td>
<td>0.63 (.47)</td>
<td>.12</td>
<td>-.30, 1.57</td>
<td>.18</td>
</tr>
<tr>
<td>Psychological distress</td>
<td></td>
<td>-16.96 (4.41)</td>
<td>-.35</td>
<td>-25.69, -8.24</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Model 4 (model 3 with prenatal methamphetamine exposure and covariates: prenatal alcohol, tobacco, marijuana; child FSIQ; income &lt;$20,000; mother’s age; mother’s education)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Postnatal drug use</td>
<td></td>
<td>.770 (0.47)</td>
<td>.15</td>
<td>-.16, 1.69</td>
<td>.104</td>
</tr>
<tr>
<td>Psychological distress</td>
<td></td>
<td>-17.15 (4.34)</td>
<td>-.35</td>
<td>-25.74, -8.56</td>
<td>&lt;.001</td>
</tr>
</tbody>
</table>

*Note:* Child’s FSIQ was associated with gift delay performance ($p=.02$) in Model 4.
Model 4

In model 4, the dichotomous term for prenatal methamphetamine exposure was reintroduced to investigate the relationship between prenatal methamphetamine use and subsequent maternal postnatal drug use and psychological functioning at the 4.5-year follow-up visit. Additional covariates were included to assess potential confounding. Each covariates indicated in the Statistical Analyses description was evaluated and included in the final model if it was related to the neurobehavioural disinhibition scores or met the “shift in estimate” criterion specific for confounding ($\pm 1SD$). Prenatal tobacco, alcohol and marijuana use, as well as child’s FSIQ, low income (<$20,000 NZD), maternal age and low maternal education (<5th form certificate) were significantly related to the neurobehavioural disinhibition scores (see correlation matrix, Table 11) and were included along with child gender and ethnicity. No covariates met the shift in estimate criteria for confounding. Within Model 4, prenatal methamphetamine exposure was not related to postnatal maternal drug use or psychological functioning. As presented in Tables 12-14, the relationship linking maternal behavioural health to child neurobehavioural disinhibition measures did not change appreciably with inclusion of prenatal methamphetamine use and other covariates. A closer investigation of each neurobehavioural measure is included below.

Inhibit.

Ethnicity and gender were entered at step 1, explaining 6.5% of the variance in inhibition. After entry of postnatal substance use at step 2, the variance explained by the model was 11.0%. When psychological functioning was added at step 3, the total variance explained by the model as a whole was 30.0%, $F(4,169) = 14.69, p < .001$. Psychological functioning explained an additional 19.0% of the variance in inhibition after controlling for child ethnicity and gender. The addition of the other covariates in the final model only explained a further 4.2% in the variance, with the total model as a whole explaining 34.3% of the variance $F(5,169), 14.18, p < .001$. In the final model, only psychological functioning and HOME scores were statistically significant, with maternal psychological stress recording a higher beta value (.44, $p < .001$) than the HOME score (.24, $p = .004$)

Emotional Control.

Ethnicity and gender entered at step 1 explained 9.9% of the variance in emotional control. After entry of postnatal substance use at step 2, the variance explained by the model was 16.1%. When psychological functioning was added at step 3, the total variance explained by the model as a whole was 26.8%, $F(4,169) = 12.70, p < .001$. Psychological functioning explained an additional 10.7% of the variance (R square change) in emotional control scores after controlling for child ethnicity and gender. The addition of the other covariates in the final model only explained a further 5.0% in the variance, with the total model as a whole explaining 31.8% of the variance $F(5,169), 12.70, p < .001$. In the final model, psychological functioning, mother’s education (below 5th form certificate) and child gender were statistically significant, with maternal psychological stress recording a higher beta value (.33, $p < .001$) than mother’s education (.24, $p = .002$) or boy gender (.24, $p = .001$).
Conduct Problems.
Ethnicity and gender entered at step 1 explained 4.9% of the variance in conduct problems. After entry of postnatal substance use at step 2, the variance explained by the model was 11.5%. When psychological functioning was added at step 3, the total variance explained by the model as a whole was 24.4%, $F(4,167) = 10.87, p < .001$. Psychological functioning explained an additional 12.9% of the variance (R square change) in conduct problem scores after controlling for child ethnicity and gender. The addition of prenatal methamphetamine exposure and the other covariates in the final model only explained a further 4.5% in the variance, with the total model as a whole explaining 28.8% of the variance $F(5,167), 10.84, p < .001$. In the final model, only maternal psychological functioning and education (below 5th form certificate) were statistically significant, with maternal psychological stress recording a higher beta value (.37, $p < .001$) than the mother’s education (.22, $p = .004$).

Hyperactivity Problems.
Ethnicity and gender entered at step 1 explained 2.2% of the variance in hyperactivity problems. After entry of postnatal substance use at step 2, the variance explained by the model was 4.0%. When psychological functioning was added at step 3, the total variance explained by the model as a whole was 19.1%, $F(4,167) = 7.95, p < .001$. Psychological functioning explained an additional 15.1% of the variance (R square change) in hyperactivity scores after controlling for child ethnicity and gender. The addition of prenatal methamphetamine exposure and the other covariates in the final model only explained a further 4.1% in the variance, with the total model as a whole explaining 23.2% of the variance $F(5,167), 8.07, p < .001$. In the final model, only maternal psychological functioning and education (below 5th form certificate) were again statistically significant, with maternal psychological stress recording a higher beta value (.39, $p < .001$) than mother’s education (.21, $p = .009$).

Delay of Gratification (Gift Delay, wrap)
Ethnicity and gender entered at step 1 explained 0.6% of the variance in simple inhibition. No change in variance was made after entry of postnatal substance use at step 2. When psychological functioning was added at step 3, the total variance explained by the model as a whole was 10.5%, $F(4,166) = 3.94, p = .005$. Psychological functioning explained 9.9% of that variance (R square change) of scores after controlling for child ethnicity and gender. The addition of prenatal methamphetamine exposure and the other covariates in the final model only explained a further 3.5% in the variance, with the total model as a whole explaining 14.0% of the variance $F(5,166), 4.32, p < .001$. In the final model, maternal psychological functioning and child FSIQ were significant. Maternal psychological distress recorded a higher beta value (-.35, $p < .001$) than child FSIQ (.21, $p = .02$). This suggests that while child FSIQ, and maternal psychological functioning may all be associated with simple inhibition, that maternal psychological function may have more of an influence. Higher levels of maternal psychological distress were correlated with a greater difficulty inhibiting, or waiting to peek ($r = -.26$, Table 11).
CHAPTER TEN: DISCUSSION

Review of the Current Study

Methamphetamine use in New Zealand is a serious public health concern, and the use of this drug has been associated with a host of negative consequences (Baskin-Sommers & Sommers, 2006; Chang, Alicata, Ernst, & Volkow, 2007; Ernst, Chang, Leonido-Yee, & Speck, 2000). Methamphetamine use among pregnant women is particularly concerning, as there are negative consequences experienced by both the mother and the developing fetus (Piper et al., 2011; Sowell et al., 2010; Zabaneh et al., 2012). There is also emerging evidence to suggest that prenatal methamphetamine exposure may have subtle effects on child behavioural and cognitive outcomes (Abar et al., 2013; Lester & LaGasse, 2010). Negative consequences include poor behaviour and executive function difficulties (Smith et al., 2011), and problems with externalising behaviours, aggression, inhibitory control and attention. This collection of adverse behavioural and cognitive effects is described as neurobehavioural disinhibition.

The current study sought to add to a limited body of research on prenatal methamphetamine exposure by prospectively examining the association between prenatal methamphetamine and preschool neurobehavioural disinhibition and the extent to which postnatal maternal behavioural functioning, including ongoing substance use and psychological functioning, might also influence these high-risk outcomes. Also examined were the patterns of ongoing drug abuse and the mental health profiles of these mothers following from birth to age 4.5 years. Findings relating to each of the study aims are discussed below.

Patterns of Substance Use and Maternal Psychological Distress Over Time

The first aim of this study was to chart the course of MA use patterns among mothers who used MA in pregnancy, and to compare rates of abstinence and relapse up to 4.5 years after delivery. On average, in this sample of 87 MA-using pregnant women, MA use declined from the first trimester of pregnancy through the last trimester. Importantly, 66.3% of mothers who used MA in pregnancy continued to use the drug after the birth of their child. Mothers that continued to use after pregnancy did so in smaller amounts early on and then increased their use significantly over the first year postpartum, which continued to increase up to 4.5 years after birth. These findings speak to the nature of methamphetamine as a highly addictive stimulant that is difficult to overcome. The initial decrease in substance use during pregnancy, followed by an abrupt increase at one-year post-delivery, suggests that women who stop drug use in pregnancy only pause their use and that this may be a compelling missed prevention opportunity.

Poly-substance Use

In terms of poly-substance use, MA-using women were more likely to be using marijuana, tobacco or other illicit drugs in pregnancy compared with the control group. This is consistent with
studies that have found that MA-users tend to be using the drug in combination with a range of other substances (Arria et al., 2006; Brecht et al., 2004). Mothers using MA during pregnancy did not significantly decrease their tobacco use across pregnancy, or over the postpartum period up to 4.5 years. Women that continued using MA in the postpartum period were significantly more likely to report heavy tobacco use at 1, 3 and 4.5 years postpartum compared to those women that gave up MA use, or had never used MA. Heavy marijuana use, however decreased substantially from pregnancy to 1 month postpartum, yet increased again significantly over the following 12 months. Women that continued to use MA in the postpartum period were significantly more likely to report heavy marijuana use at 1 year and 3 years compared with those that never used. These results support the vast literature suggesting that methamphetamine is frequently used in conjunction with other drugs and substances of abuse (Arria et al., 2006; Derauf et al., 2007; Della Grotta et al., 2010; Lester & Lagasse, 2010; Wouldes & Woodward, 2010). Interestingly, both groups reported relatively similar proportions of heavy alcohol use in pregnancy and at 4.5 years postpartum. The findings from this study and these trends over time signal the need for intensified education and prevention efforts about the health-related consequences of neonatal alcohol, marijuana and tobacco exposure. Given that a high proportion of these women frequently received prenatal care, the efficacy of providing educational messages via the obstetrician’s office or midwifery care should be explored (El-Mohandes et al., 2003).

Patterns of MA Use in Postnatal Period

Looking more closely at individual patterns of MA use, this study identified three patterns of maternal postnatal drug use from 1 month postpartum through the first 4.5 years of a child’s life: a chronic (20.5%) group who continued to use across the majority of the postpartum period; a “pausers” (45.8%) group who had stopped using briefly after pregnancy but then used intermittently over the postpartum period, and; an “abstainers” (33.7%) group who gave up their use after pregnancy. While by no means definitive, the use patterns developed in this study could serve as a starting point for future research investigating the relationship between quantity, frequency, and persistence of MA use and possible long-term developmental outcomes among children of addicted mothers.

Notably, the women who “abstained” their MA use were more likely to use alcohol than women who were “chronic” high users of MA from pregnancy through to 4.5 years postpartum. This finding is difficult to interpret, but we can speculate that one reason for it is that women might think alcohol is less harmful than methamphetamine. While all women should be advised and supported to eliminate their MA use during pregnancy and into the postpartum period, clinicians should also remain vigilant regarding concomitant or increased alcohol use. Another explanation for the finding is that that a reduction in MA use triggers withdrawal symptoms that could lead to self-medication with alcohol. If confirmed, this is a potentially serious and compensatory correlate of decreasing MA use during pregnancy, given the known adverse neonatal effects of alcohol. More intervention including retention in treatment may be needed to support decreasing or quitting MA.

Surprisingly, we found that some women in our control group, who denied using methamphetamine during pregnancy and had a negative meconium confirmation, started using methamphetamine in the postpartum period (“postpartum initiators”; 12.4%). However, the majority of
control women, “never used” (87.6%). It is uncertain whether the control women who started using MA after pregnancy really were not using in pregnancy, or if they were missed by the methodological procedures. There is some evidence to suggest that accurate identification of prenatal drug exposure is improved with analysis by immunoassay with gas chromatography/mass spectrometry (GC/MS) confirmation and maternal self-report from in-hospital interviews (Chasnoff, Landress, & Barrett, 1990; Gawin & Ellinwood, 1988; Gray et al., 2009; Ostrea, Brady, Gause, Raymundo, & Stevens, 1992; Zuckerman et al., 1989). However, there is also evidence that the meconium assay, at best, can only provide a record of drug use during the second half of pregnancy (Lester et al., 2001). Meconium begins to form in the 12th week of gestation and collects endogenous and exogenous waste from the second trimester (Smith et al., 2015). If methamphetamine use was stopped in the first trimester, as was the case for many women, meconium results would be negative. Therefore, it is possible that these women used methamphetamine during the first trimester of their pregnancy, and have been misclassified. Alternatively, it could suggest that they are in high-risk environments for substance use and dependence, and did in fact only start using methamphetamine after the birth of their baby. Some research suggests that adjustment to life with a newborn can be stressful, and use of drugs can be one way women try to cope with these changing life demands (Davis, 1990). Future research may benefit from supporting maternal self-report measures with a toxicology screening process (e.g., hair analysis, urine, drug in blood, etc.) at each time point in order to accurately classify women based on their use.

While more robust, this type of methodology, however, can be costly, and may also have the potential to infringe on recruitment and retention rates, as some women may view this process as invasive.

Women in the “pausers” group (94.7%) and the “postpartum initiators” (91.7%) groups were also more likely to have a positive psychiatric diagnosis at 1 month compared with the other groups. Interestingly, these were also the women that were more likely to have had some mental health input at some point over the 54 month follow-up period (65.8% and 58.3% respectively). The “abstainers” also had higher rates of service input (60.7%), which may have contributed to their ability to abstain from substances and have lower levels of psychological distress after pregnancy. Unfortunately, the results in this study do not have the capacity to draw these causal conclusions. However, it offers an interesting point of interest for further studies to consider. Comparatively, mothers in the “chronic” and “never used” groups had lower rates of mental health support in the postnatal period (35.3% and 29.4% respectively). For those that have “never used” methamphetamine, earlier results suggest that these are women that have low rates of use of all substances, and are less likely to come from backgrounds of high psychosocial stress. Coupled with lower rates of psychological distress, these women are likely to be those that do not need formal input from mental health services, or do not meet criteria for mental health support. For “chronic users”, the lower rates of mental health support input suggest that these are the women that have a high need for mental health and/or alcohol/drug treatment support, yet are the ones that have the lowest rates of identification or engagement.
Psychological & Socioeconomic Correlates of Ongoing Substance Use

We demonstrated that these groups differed on maternal sociodemographic variables, as well as on levels of psychological distress. Notably, we found that women who continued to use methamphetamine after pregnancy were more likely to live alone, come from low socioeconomic backgrounds, report a history of emotional and physical abuse, report higher psychological distress and met the criteria for a positive psychiatric diagnosis based on the Brief Symptom Inventory (BSI). These findings support the large body of evidence that families affected by substance abuse and/or addiction are more likely to be affected by other adversities such as mental health difficulties, single-parent status, social isolation and financial and other stressors associated with living in poverty (Derauf et al., 2007; Oyserman, Mowbray, Meares, & Firminger, 2000). The observations are also consistent with previous findings that MA-using adults tend to have a higher incidence of comorbid mental health difficulties than non MA-using adults (Salo et al., 2011; Terplan et al., 2009; T. A. Wouldes et al., 2013; Zweben et al., 2004). MA use alters neurotransmitters in the brain that are associated with mood and emotional states. Prolonged use of MA leads to damaged neurotransmitter receptors and presynaptic reuptake mechanisms, and is theorised to be associated with persistent psychological symptoms, even after abstinence (Bauer et al., 2002).

Other factors that may have contributed to a high incidence of psychological distress in the MA group compared with the control group include lower SES and higher rates of marijuana and smoking. Numerous investigators have reported that low SES is associated with psychopathology (Davidson, 2000; Lester et al., 2002). Additionally, smoking tobacco and marijuana use has been associated with maternal psychological symptoms (Della Grotta et al., 2010). Thus, numerous factors may contribute to the increased rate of psychological symptoms in the MA-using mothers.

The main symptoms of psychological distress in MA-using mothers included obsessive-compulsive, interpersonal sensitivity, hostility, phobic anxiety, paranoid ideation and psychotism. This is consistent with reports among MA users that MA use is associated with symptoms of depressed mood, paranoia, irritability, mood swings, aggressive behaviours, anxiety and psychosis, even long after abstinence (Maxwell, 2005; McKetin et al., 2006; Newton et al., 2004). We did not observe a notable pattern of distinguishable psychological symptoms between the MA-using group and the control group over time. The lack of a clear pattern may suggest that there is little specificity regarding the association between unique types of distress and ongoing substance use. However, the associations across heterogeneous types of distress may also reflect comorbidity of mental health problems (e.g., stress, anxiety, and depression) that is common in pregnant and postpartum women (Wisner et al., 2013). Notably, there were not sizable differences with depressive symptoms between the groups at any time point. This was surprising, as methamphetamine addiction is highly associated with depressive symptoms (Zweben et al., 2004). Alcohol use, however, is also highly associated with depressive symptoms, so it is likely that if the control group was using alcohol in high amounts, that they too were suffering from increased depressive symptoms similar to the MA-using group.

Moreover, despite a significant difference in the incidence of psychological distress in our MA-using mothers, the control mothers also had a high incidence of psychological distress. In our sample, 32% of control mothers met the criteria for a positive psychiatric diagnosis at 1 month postpartum, where
the incidence typically reported is approximately 10% (Evans, Heron, Francomb, Oke, & Golding, 2001; Gotlib, Whiffen, Mount, Milne, & Cordy, 1989). One explanation is that both maternal groups had high rates of heavy alcohol use during pregnancy (MA-using mothers: 25.3% and control mothers: 17.5%). It has been found that alcohol use during pregnancy is correlated with 2.75-increased odds of having a positive psychiatric diagnosis among women using methamphetamine during pregnancy (Wouldes et al., 2013). Therefore, the high level of psychological distress in the control groups could also be related to the high incidence of alcohol use.

For both groups, psychological symptoms of distress were highest at 3 years postpartum. This is consistent with studies that have found the toddler period to be more stressful for parents relative to other periods of early childhood (Shaw, Bell, & Gilliom, 2000). A toddlers’ growing desire to assert their independence (e.g., the onset of the ‘terrible twos’) can increase parent-child conflict during this period, and mothers with mental health and/or addiction difficulties, may be less able to provide the gentle guidance and the setting of appropriate limits necessary to successfully negotiate these conflicts (Gelfand & Teti, 1990; Teti, Kim, Crosby, Mayer, & Towe-Goodman, 2012). Consistent with this notion, there is evidence of higher levels of parental distress and decreases in parental satisfaction relative to the infancy period, particularly during the ‘terrible twos’ when frustrations associated with rearing a physically mobile but cognitively unsophisticated toddler (Fagot & Kavanagh, 1993; Shaw & Bell, 1993). Some mothers experiencing psychological distress may be more likely to avoid confrontation with their toddlers, expressing fears over their child’s wilful behaviour and their inability to assert appropriate authority (Teti et al., 2012). Other mothers may resort to harsh discipline, showing greater hostility towards their children and utilising more physical punishment than their non-psychologically distressed counterparts (Gelfand & Teti, 1990; McLoyd, 1998). Maternal feelings of helplessness and lack of control over their children’s behaviour may increase the likelihood they will employ coercive or punitive tactics in disciplinary encounters (Bugental & Happaney, 2004). In fact, maternal psychological distress is considered a risk factor for physical abuse and maltreatment of young children (Arnow et al., 2011). In either case, the ineffective socialisation techniques employed by mothers that are psychologically distressed are often met with dysfunctional behaviour on the part of the toddler. In some cases, children of psychologically distressed mothers show more frequent defiance, hostility, aggression and externalising behaviour. Alternatively, the toddlers of psychological distressed mothers may show more depressed affect and withdrawal themselves, as well as helplessness in the face of challenges. Notably, the behaviour of these toddlers’ often matches that of their mother, such that the affect and symptoms of the mother are mirrored or modelled in her child’s actions (Gelfand & Teti, 1990). Consistent with the notion that the toddler period is more stressful for parents relative to other periods of early childhood, symptom levels of maternal distress have been found to decrease in early childhood after peaking at age 2 to 3 (Shaw et al., 2000). Findings from this study also support this trend, with symptoms decreasing from 3 years to 4.5 years.

While it was helpful to identify patterns of ongoing substance use and postnatal psychological distress among women who used methamphetamine during pregnancy over time, it may be important for future studies to model substance use alongside psychological symptoms at each time point to investigate the influence of comorbidity, and try to attempt to answer what is driving these associations.
As of now, it is unclear if mental health difficulties are causing ongoing substance use, or if ongoing substance use is causing higher rates of psychological distress. This might be done using a latent growth curve model format or structural equation modelling using path analysis.

Maternal Behavioural Health and Neurobehavioural Disinhibition Outcomes

The second aim of the study was to examine the associations between prenatal MA exposure and indicators of postnatal maternal behavioural health, including drug use and psychological functioning, on child neurobehavioural disinhibition measures at 4.5 years. The main findings were: (1) maternal prenatal methamphetamine use during pregnancy predicted self-reported maternal drug use 4.5 years postpartum, but did not predict child neurobehavioural disinhibition, as measured by the BRIEF-P, SDQ and Gift Delay (wrap); (2) postnatal maternal drug use and psychological distress were associated with child neurobehavioural disinhibition; (3) maternal psychological distress was related, independent of postnatal drug use, to child neurobehavioural disinhibition; (4) the relationship between postnatal maternal drug use and neurobehavioural disinhibition was attenuated once maternal psychological distress was taken into account, and finally; (5) the inclusion of possible confounding covariates in the model did not influence the interpretation of study results. These findings are particularly meaningful, given that prenatal exposure to teratogenic substances (methamphetamine, tobacco, alcohol, and marijuana) were controlled for, highlighting a potentially unique association between maternal psychological distress and later child development among drug-using mothers.

The observed lack of association between prenatal methamphetamine exposure and elements of child neurobehavioural disinhibition in this study differs from previous IDEAL findings of behavioural and executive functioning difficulties (Abar et al., 2013; Himes et al., 2014; Piper et al., 2011). This outcome is also somewhat unexpected, considering evidence of abnormalities in the monoaminergic system as a consequence of in-utero methamphetamine exposure (Behnke et al., 2013; Thompson et al., 2009) as well as other published reports of increased externalising difficulties and decreased inhibitory control in methamphetamine-exposed children (Derauf et al., 2012a; Diaz et al., 2014; Eze et al., 2016; LaGasse et al., 2012).

In comparison with the other studies, this study focused on children who remained in the care of their biological mothers. The exclusion of children residing with alternate caregivers may have resulted in the elimination of children whose mothers had more severe drug use during pregnancy. Sample restrictions such as these provide increased covariate control but may have diminished the prenatal methamphetamine exposure. Alternatively, this study could have captured more robust measures of comorbid psychological distress over time among mothers who used methamphetamine in pregnancy and the postpartum period, which therefore had a greater effect on child developmental outcomes as opposed to the initial drug exposure. Moreover, the earliest study of neurobehavioural disinhibition among children exposed prenatally to substances was 5 years of age (Abar et al., 2013). It is possible that observed differences in the exposed group between these age groups suggests that 4.5 years to detect differences may be too early, and the direct effects come out at an older age. General evidence suggests that pre-school age may be a good time to start identifying emotional-behavioural...
difficulties in children, however age 4.5 may be just slightly too early to measure methamphetamine-related impairments in emotional regulation, behavioural under control and inhibition. It may be that such deficits may manifest later in childhood, as academic and social demands increase and prefrontal brain structures mature. It is also possible the BRIEF-P, SDQ and Gift Delay (wrap) task as measures of neurobehavioural disinhibition may not have been sufficiently sensitive to detect subtle deficits. These measures were selected primarily on previous research suggesting their ability to measure various aspects of behavioural regulation, emotional control and inhibition, particularly among pre-school-aged children (Carlson, 2005; Goodman et al., 2000; Hughes & Ensor, 2008; Willoughby, Blair, Wirth, & Greenberg, 2010). However, the finding of no difference between groups was consistent across all measures, including both maternal self-report and lab-based measures. It was also hypothesised that the fact that some of the control women were using methamphetamine postnatally may have had an impact on the results. However, after running a separate analysis to check out this hypothesis, still no differences were found.

Alternatively, it is more likely that the effects are largely mediated or driven by the environmental factors observed in this study. For example, while previous studies have found that prenatal methamphetamine did have a direct effect on neurobehavioural disinhibition factors, starting from emotional reactivity and early indicators of attention difficulties at 3 years up to high levels of aggression and externalising behaviours at 8 years to executive function problems at 9 years, early adversity significantly mediated this relationship (Abar et al., 2013; Billing, Eriksson, Jonsson, Steneroth, & Zetterström, 1994; Eze et al., 2016; LaGasse et al., 2012). Further findings from the cocaine literature support this. Fisher et al. (2011) for example, found that early adversity, measured by postnatal drug exposure, unstable home environment, low SES, caregiver experiences of abuse and psychopathology, mediated the effects of prenatal exposure of cocaine to later deficits in behavioural dysregulation and later deficits in executive functioning. Unfortunately, all of these studies included an aggregate measure, so there was no way to differentiate the associate effects of psychological distress versus postnatal drug use versus low SES. Only one study looked at the effect of methamphetamine exposure alone and postnatal drug exposure on behaviour and executive function deficits at 6.5 years, measured by the CBCL, the Conners’ and Children’s Memory Scale (Himes et al., 2014). The authors found that prenatal methamphetamine exposure alone, and in combination with postnatal drug exposure, were associated with greater behavioural problems and executive function deficits. They found no group differences between caregiver BSI responses, however these scores were only averaged through 3 years of age. Postnatal substance use included use of methamphetamine and tobacco use only. Therefore, measurement differences could be contributing to the different findings, as well as increased alcohol use known to be a key mediating factor for psychological distress in New Zealand (Wouldes et al., 2013). It is important to consider parental psychosocial characteristics during early childhood as these may result in maladaptive outcomes for children.

The findings in this study that maternal psychological distress affects neurobehavioural development at 4.5 years is in keeping with previous work linking maternal psychological distress with decreased neurodevelopment as early as 1 month (Smith et al., 2012), impaired cognitive development (Grace, Evindar, & Stewart, 2003), and child behavioural problems in boys up to age 5 years (Sinclair
& Murray, 1998). Many studies have documented an association between parental psychopathology or psychological symptoms and child behaviour problems, including inhibitory control and neurobehavioural disinhibition. In general, these studies suggest that a child of parents who experience psychological distress may show a number of difficulties including greater externalising problems and social incompetence (Rutter & Quinton, 1984; Zahn-Waxler et al., 1988). The findings of this study are also in keeping with prior research showing that current environmental factors are generally better predictors of developmental outcomes than are early biological risk factors such as perinatal health problems (Adams, Hillman, & Gaydos, 1994; Escalona, 1982; Werner, Bierman, & French, 1971), low birth weight (Brooks-Gunn, Klebanov, & Duncan, 1996; Ornstein, Ohlsson, Edmonds, & Asztalos, 1991), prenatal substance exposure (Hurt et al., 1995; Hurt, Malmud, Betancourt, Brodsky, & Giannetta, 2001), and other biological risk factors (Sameroff, Chandler, & Horowitz, 1975). Such findings are consistent with the general notion that current context is more important than past events in influencing current behaviour (Lewis, 1998).

It is possible that the findings of increased neurobehavioural disinhibition in children exposed to higher levels of maternal psychological distress might also reflect the postnatal effects of cumulative adversity conditions (e.g., low SES, household violence, maternal education, ethnic disadvantage) on the developmental of childhood mental health and cognitive and psychosocial impairments (Biederman et al., 1995; Rutter, Cox, Tupling, Berger, & Yule, 1975). In this study, we found that the mother’s education level being below 5th form certificate, less enriched home environments on the HOME inventory, and a child’s ethnicity being Māori, were associated with poorer developmental outcomes, independently of maternal psychological distress. The emotional control scale of the SDQ was also associated with being male. Our findings are consistent with others that found behaviour problems related to caregiver psychological symptoms and poor-quality homes related to increased attention problems, aggression, ADHD problems and neurobehavioural disinhibition. For example, Abar et al (2013) found that prenatal MA use was associated with later behavioural and emotional control at 5 years, which was associated with later deficits in executive function at 6.5 years. The authors concluded that the effects of prenatal MA on neurobehavioural disinhibition largely functioned through early adversity, defined by postnatal substance use, extreme poverty, primary caregiver changes, caregiver sexual/physical abuse, psychological diagnosis, depression, home environment, community violence and social position from birth up to 3 years of age. Gender effects have not been reported at this age in studies of prenatal methamphetamine or prenatal cocaine exposure. However, one study using the CBCL found that there were gender effects on scales of externalising behaviour problems (LaGasse et al., 2012).

One of the unanticipated findings in this study was how few additional predictors of pre-schooler neurobehavioural disinhibition were significant once maternal mental health was considered in statistical models. We did not find consistent associations with other factors that have engendered a great deal of attention with respect to child development, namely socioeconomic status and substance abuse. One possible explanation is that these factors were “overshadowed” by the influence of maternal psychological distress, which may have more of a direct impact than these factors. For example, it is plausible that some of these risk factors are mediators of the relationship between maternal
psychological distress and child development, where maternal psychological distress represents a key point of intervention for interrupting the chain of risk. Finally, the lack of consistent effect of socioeconomic status may be related to a delayed effect of disadvantage on child development. For example, it may be that the effect of disadvantage is more apparent in later developmental stages. Therefore, it is important to reflect on these concerns within context. Several studies have documented the relationship between psychosocial stressors and adversity with increased mental health difficulties (Fisher et al., 2011; Kieling et al., 2011; Murali & Oyebode, 2004), suggesting that treating these women includes addressing the social risks these women are facing.

Findings from this study suggest that influence of ongoing maternal drug use on child neurobehavioural disinhibition should be interpreted within the context of maternal psychological functioning. When maternal drug use and psychological difficulties coexist, it may be difficult to ascertain their relative influence on child neurobehavioural disinhibition. In this study, self-reported drug use, and psychological symptoms were slightly correlated, and both were related to child neurobehavioural disinhibition. When considered together in statistical models, however, the relationship between postnatal maternal drug use and child neurobehavioural disinhibition was somewhat dampened. Other studies have shown an association between continued maternal drug use and child behaviour in cocaine-exposed children (Chasnoff et al., 1998; Griffith, Azuma, & Chasnoff, 1994). There is also one study that found prenatal cocaine exposure was not related to child behaviour problems at age 5, and recent maternal drug use and psychological functioning had relationships with internalising and externalising behaviour scores on the CBCL (Accornero, Morrow, Bandstra, Johnson, & Anthony, 2002). However, the authors also found that within a combined model, only maternal psychological functioning remained significant. The co-occurrence of maternal drug use and psychological distress may further compromise overall maternal behavioural health and parenting behaviour. Accordingly, drug-using mothers who also suffer from psychological distress may have a more negative influence on child neurobehavioural inhibition outcomes than drug-using mothers who are not experiencing psychological distress. This would explain the attenuated relationship observed between postnatal drug use and child neurobehavioural disinhibition, once maternal psychological functioning was included in the statistical model.

These findings are in support of a growing body of evidence linking early adversity, both with and without preceding prenatal drug exposure, with inhibitory control deficits (Pears, Fisher, Bruce, Kim, & Yoerger, 2010; Valiente, Lemery-Chalfant, & Swanson, 2010) and with later appearance in adolescence of a more complex disinhibitory phenotype (Fisher et al., 2011). However, because caregiver psychological distress was an aggregate measure of time points including those antecedent to the inhibitory control outcome, it may be unmeasured concurrent exposure to caregiver distress rather than earlier or cumulative exposure that is driving this association.

Within this context, the association between child development and maternal distress found underscores its importance as an early life risk factor. Future research regarding the relationship between prenatal and postnatal distress and child development should address two key questions: (1) what protective influences and interventions lessen the impact of maternal psychological distress on poor child outcomes; and (2) what influences and interventions minimise or reverse the adverse
neurological sequelae of a sub-optimal intrauterine environment (e.g., due to prenatal substance exposure)? Notably, most research to date largely explores intrauterine risk factors, without also examining potential protective factors that occur during the prenatal and postnatal periods.

Very few reviews have synthesized evidence relating prenatal and postpartum maternal psychological distress to infant development. Of these, most address a single form of maternal distress during the prenatal or postpartum period; and, the majority focus on older children (Kingston et al., 2012; Kingston & Tough, 2014). Of importance, this study examined the influence of chronicity of psychological symptoms over time on behavioural development. Notably, the findings indicate that approximately 76% of children in the study were living with mothers with a positive mental health diagnosis across the early childhood period. 21% of these mothers met the criteria for a positive diagnosis at all time points in the study (1 month, 1 year, 3 years, and 4.5 years), indicating high levels of chronic psychological distress. It is likely that some of these women may have experienced mental health difficulties prior to pregnancy, and that over time their symptoms worsened as they made the transition to parenthood and experienced the demands of early caregiving. Neurobehavioural disinhibition difficulties were significantly higher for children whose mothers had higher and a more persistent pattern of psychological distress compared with children whose mothers never met the criteria for a positive psychiatric diagnosis or met it at only one time point. These findings are consistent with previous studies reporting poor outcomes for children of mothers with chronic postnatal mental health difficulties (Grace et al., 2003; Kingston et al., 2012; Kingston & Tough, 2014; Sohr-Preston & Scaramella, 2006) notably, that they are at increased risk of emotional and behavioural difficulties. One study has found that proportions of infants with sub-optimal cognitive development were similar in women with brief postpartum depression (symptoms at 4 months, not at 12-15 months) and no depression, whereas infants of mothers with chronic depression (postpartum depression at 4, 12-15 months), were over 3 times more likely to have poor cognitive development (Cornish et al., 2005).

Researchers have found depressed mothers are less responsive (Cox, Puckering, Pound, & Mills, 1987) and emotionally unavailable (Malphurs et al., 1996) to their children compared to non-depressed mothers, and that infants of depressed mothers are more likely to establish an avoidant attachment style (Lyons-Ruth et al., 1986) and poor emotion regulation (Cicchetti & Toth, 1991). This lack of contact and insecure attachment style can impede the activation and growth of neurotransmitters, possibly accounting for the delays in infant neurodevelopment.

Given that maternal psychological distress (e.g., stress, anxiety, depression) in pregnancy is common (Priest, Austin, Barnett, & Buist, 2008; Woods, Melville, Guo, Fan, & Gavin, 2010) and a substantial proportion of women who experience distress in pregnancy or during the postpartum period continue to have symptoms into their child’s early years (Austin, Tully, & Parker, 2007; Beeghly et al., 2002; Horwitz, Briggs-Gowan, Storfer-Isser, & Carter, 2009), maternal psychological distress represents a prevalent, enduring and modifiable influence that may significantly impact fetal and child development. Interest in the effect of maternal psychological distress on infant outcomes and its underlying mechanisms has surged over the past two decades. While much of the early research in this field focused on studying the impact of postpartum depression on outcomes such as maternal-infant interaction and infant temperament, more recent studies have explored the effects of different forms of
maternal psychological distress and their timing on a broader array of infant outcomes, including infant development. Some key findings of this body of evidence suggest that: (a) deficits in infancy are associated with deficits in later childhood stages (Auerbach, Atzaba-Poria, Berger, & Landau, 2004), (b) predictors of developmental deficits can be detected during the first 10 months of life (Skovgaard et al., 2008), (c) infant developmental deficits and their causes are amenable to early intervention targeted at the infant and its family (Bergman, Sarkar, Glover, & O'Connor, 2010), and (d) intervention aimed at reducing maternal psychological distress can lower the risk of adverse developmental outcomes (Poobalan et al., 2007). Further research is needed to disentangle prenatal and postnatal risk and protective factors on toddler cognitive development, the mechanisms that underlie the association, and the mitigating influence of the postnatal environment on prenatal risk.

**Clinical and Research Implications**

Maternal substance use is consistently reported in the literature as having adverse effects on child outcomes (Ministry of Social Development, 2011). Children who have a parent with mental health and/or addiction difficulties are at an increased risk of a number of poor developmental outcomes, including developing mental health and/or addiction issues themselves. They experience higher rates of suicidal ideation and interpersonal and behaviour problems (Fraser, James, Anderson, Lloyd, & Judd, 2006). Parental substance misuse can affect children’s emotional and psychological development, commonly resulting in challenges with attachment and family functioning, increasing the risk of violence and abuse.

The current study has shown how maternal behavioural health (postnatal drug use and psychological functioning) and early exposure to social risk might be shaping neurobehavioural disinhibition among pre-school-aged children exposed prenatally to methamphetamine. Through highlighting the effects of pre- and postnatal factors, the current study has provided support to the double jeopardy hypothesis, which postulates that children born to methamphetamine-using mothers have the potential to be compromised by both the prenatal teratogenic effects of methamphetamine and by being born into adverse socio-familial environments associated with maternal methamphetamine use. Specifically, the current study has supported this perspective by showing that:

1. Variation in neurobehavioural disinhibition scores was not wholly explained by infant characteristics or prenatal exposure to methamphetamine and other substances in pregnancy. Rather, maternal psychological distress was the key mechanism predicting neurobehavioural disinhibition outcomes, independent of prenatal exposure to methamphetamine and other substances in pregnancy and in the postpartum period.

2. Other social risks also had an effect on outcomes, including living alone, postnatal poly-substance use, less enriched home environments, less maternal educational achievement and lower SES.
Taken together in the context of previously published literature, these results suggest that while prenatal exposure to methamphetamine may be having some effect on child development outcomes, it appears that maternal behavioural health and socio-familial risk significantly contributes to the longer-term outcomes of pre-school children born to mothers using methamphetamine in pregnancy. The role of maternal clinical and social risk factors as contributors to child mental health outcomes has been well documented in previous studies. For example, Luthar et al. (2003) reported that highly depressed drug-dependent mothers were likely to have children experiencing both internalising and externalising problems, as maternal caregiving was compromised by both the substance abuse disorder and mental illness. Therefore, determining the contributing factors to the mental health outcomes of children born to mothers characterised by substance dependency, mental health difficulties and socio-environmental adversity may be highly complex, given that confounding factors such as maternal mental health have been shown to explain the association between prenatal methamphetamine exposure and child developmental outcomes.

The subtle effects argument, discussed by Crea et al. (2008) and Savage et al. (2005), proposes that the effects of exposure to adverse biological and environmental mechanisms compound over time, and that poorer outcomes are more likely to be observed in children’s later development as opposed to early development. This is largely due to disadvantaged children’s difficulties becoming more pronounced in the face of increasing cognitive and behavioural demands set by increasingly structured environments. One factor thought to contribute to this process is ongoing substance use and mental health difficulties. While the current study saw little observable effect of ongoing postnatal substance use on pre-school children’s behavioural regulation outcomes, possible effects may yet emerge over time as these children encounter differing socio-familial experiences as the addiction and/or mental health concerns persist. As a result, the emerging between-groups differences currently detected between MA exposed children with and without experiences of concurrent maternal comorbidity may continue to widen in response to cumulative or chronic exposure.

Figure 6 below highlights the possible pathways to risk identified by the findings of the current study. Maternal risk factors relating to maternal methamphetamine use primarily predict the wellbeing of children by 4.5-years. Following this, children may arrive at poor outcomes via the continued exposure to possible risks, leading to high levels of environmental instability and disruption of early attachment relationships. This model also suggests that a relationship between prenatal exposure and poor outcome might continue to emerge over time as the risks associated with continued substance use and mental health difficulties compounds those resulting from prenatal exposure, as per the subtle effects argument.
The finding that maternal psychological distress was consistently related to pre-schooler neurobehavioural disinhibition provides compelling evidence for an upstream, preventative approach to child developmental problems. Maternal mental health is a modifiable risk factor. Emerging evidence suggests that early intervention aimed at reducing maternal mental health problems in the prenatal and postnatal periods is effective at improving maternal mental health, maternal functioning, parenting, child cognitive development, and maternal-child interaction (Milgrom, Schembri, Ericksen, Ross, & Gemmill, 2011; Phipps, Raker, Ware, & Zlotnick, 2013; Poobalan et al., 2007). The need for maternal mental health screening as a component of routine well-child visits has been acknowledged by the American Academy of Paediatrics (Earls & Committee on Psychosocial Aspects of Child and Family Health, American Academy of Pediatrics, 2010), the Canadian Paediatric Society (Bernard-Bonnin, 2004), New Zealand’s Ministry of Health (2002) and international organisations (Austin & Priest, 2005; Austin & Marcé Society Position Statement Advisory Committee, 2014; Austin & Hightet, 2011) organizations. However, maternal mental health problems and addiction problems remain severely under detected and undermanaged in both paediatric (Horwitz et al., 2007) and obstetrical care (Coates, Schaefer, & Alexander, 2004; Wouldes, 2009). Without standardised screening and treatment, over 50% of pregnant women with depression and anxiety (K. Grant, McMahon, & Austin, 2008) will experience chronic symptoms through their child’s early years (Horwitz et al., 2009).

Given that women see an obstetrical care provider on average fourteen times during pregnancy and postpartum and a paediatrician or family physician several times through well-child visits thereafter, one of the earliest and potentially most impactful approaches to risk reduction of child cognitive delay may be to embed effective, feasible, and sustainable approaches to maternal screening, referral, and treatment as components of routine obstetrical and well-child care (Olson, Dietrich, Prazar, & Hurley, 2006). However, despite these recommendations, universal psychosocial assessment has not been widely implemented as part of routine prenatal care. As a result, mental health and/or addiction concerns frequently go undetected in the course of routine prenatal care. This is particularly problematic because during the perinatal period many women do not discuss their mental health and/or addiction concerns with others or seek professional assistance, and often these issues frequently continue into the

Figure 6. Pathways to risk: prenatal and postnatal maternal factors and child factors on child neurobehavioural disinhibition.
postpartum period. Moreover, service utilisation rates of children with developmental and mental health issues are typically low (<50%), particularly among children under 7 years of age. Depending on the nature of the developmental problem, preventative and treatment services are frequently not available or easily accessible. As Waddell et al. (2007) note with respect to child mental health, “it is increasingly evident that treatment services alone cannot reduce the burden of illness. As well, the understanding that many mental disorders arise during childhood has encouraged a shift toward considering prevention” (p. 174).

Finally, maternal psychological distress may represent one of the most modifiable and feasible strategies for reducing risk factors for child developmental delays. Hertzman and Boyce (2010) conceptualised developmental risk factors as those that are most proximal to the child (e.g., family-based factors), those in the meso-environment (e.g., neighbourhood; school), and those belonging to the macro-environment (social/economic/political). It is plausible that addressing maternal psychological distress may, in turn, affect a mother’s relationship with her child and the overall family functioning with benefits that extend to the meso-environment. This is not to underestimate the impact of other risks external to the family; rather, these discussion points are intended to highlight the importance of addressing maternal psychological distress as one of the most upstream factors in a child’s life.

While genetic and teratogenic environmental mechanisms are important in linking mothers’ substance use and/or mental health issues to children’s difficulties, these risks are considered to be malleable, meaning that measures can be taken to reduce them. For example, there is good evidence that the quality of parenting and family interaction is a key mediating variable (Ramchandani & Stein, 2003). The interventions that improve the quality of parenting and family and whānau interactions are likely to lessen the risk of harm and adverse outcomes for children. This study provides a strong argument for ensuring that adult mental health and addiction services proactively work to intervene early to support service users in their role as parents, both directly and by linking them to appropriate parenting supports in the community.

Clear evidence now points to the critical importance of caregiver relationships in the first three years of life and their impact on physical and mental health outcomes. Infants are at a key developmental period in terms of their physical, social and emotional development. Virtually all aspects of early human development, including the architecture of the brain, are affected by the caregiving environment during the prenatal and infancy period (Phillips & Shonkoff, 2000). Responsive, warm and attuned caregiving can enhance the structural development and chemistry of the brain. Adverse circumstances and problematic or unresponsive caregiving relationships in early life have been shown to increase the risk of a range of emotional, behavioural and health problems in both the short term and the longer term into adulthood. For these reasons, supporting mothers who have mental health and/or addiction concerns to develop positive responsive relationships with their children from an early stage (prenatally and postnatally) is critical to ensuring positive outcomes for both the children and the wider family and whānau.

In recent years, New Zealand has made some progress towards addressing the needs of children of parents with mental health and/or addiction difficulties. However, too often these needs are overlooked within existing service provision. The adult mental health and addiction sector in New
Zealand has traditionally been based around services for individuals, without routinely identifying or considering the needs of children. Consequently, despite the evidence showing the vulnerability of this group, and the increasing evidence on the effectiveness of interventions for them, children of parents with mental health and/or addiction difficulties often remain an “invisible population” within services and communities.

It must be noted that the resilience of children and families can be considerable, and many children of parents with mental health and/or addiction concerns grow up without adverse outcomes. A focus on families and whānau in which parents have mental health and/or addiction difficulties is not intended to imply that parents are negligent or uncaring or are to blame for their children’s difficulties. It does, however, recognise that many of them will at times require particular support and assistance.

There is significant potential for early intervention to build on the strengths and resiliency of children who may experience difficulties as a result of their parent, family or whānau’s circumstances to intervene before potential problems arise. It is clear that many mental health, emotional and behavioural problems can be prevented before they begin if we are able to intervene early in the lives of children who may be at risk (Beardslee, Chien, & Bell, 2011). Compelling evidence now demonstrates that interventions focused on families and whānau in which parents are facing mental health and/or addiction concerns can help to prevent later mental health issues and reduce the prevalence and burden of mental illness and addiction for future generations (Beardslee, Chien and Bell, 2011). These approaches target known risk factors, and enhance protective factors. In a systematic review and meta-analysis, Seigenthaler et al. (2012) identified that interventions with children of parents with mental health and/or addiction difficulties decreased the risk of new diagnoses of mental health problems by 40 percent.

Children of parents with mental health and/or addiction difficulties (COPMIA) services is one preventative strategy aimed to support children’s healthy social and emotional development and prevent the development of mental health and addiction by addressing risk and protective factors; promoting psychosocial resilience in children; improving parent-child interactions; reducing stigma; and promoting social network support (Saxena, Jané Llopis, & Hosman, 2006). Interventions used to achieve these aims include an awareness of the need for adult mental health and addiction services to take a ‘whole of family and whānau’ approach. Individual treatment of parental mental health difficulties in isolation tends to not achieve good outcomes for the child, parent or whānau, especially compared with a family-focused approach. A whole-family, strengths-based approach that is informed by the service user and involves well-integrated services appears to be the most effective form of intervention. Key to this approach is that adult services have systems in place to identify parents of dependent children, and to provide psychoeducation and support services that address the needs of the children, parents and wider family system and that give them access to advice and support from infant, child and adolescent mental health (ICAMH) and alcohol and other drug (AOD) services and to more specialised therapeutic programmes when needed. Conversations about parenting should be an essential part of practice in adult services. The aim is to make discussions about parenting a normal part of the parent-mental health/addictions worker partnership and to empower parents to support the needs of their child. This is best accomplished by providing practitioners and organisations with tools that support a paradigm shift.
in the way they think about and support the needs of parents and their children and that promote a view of the parent as the 'expert' on their child, using a strengths-based approach.

Families and whānau experiencing mental health and addiction related problems are more likely to live in economic deprivation, be unemployed, have housing difficulties, and live in isolation. Taking a holistic approach to these complexities is likely to improve outcomes for disenfranchised individuals and whānau. Developmental assessments form a routine part of well-child visits. However, Tough et al. demonstrated that a substantial proportion of children are not identified as being at risk by primary care providers, or referred for appropriate follow-up services.

Currently, there are few existing social policies that enable drug-dependent women to access professional child wellbeing services as part of their current treatment programme. The two existing methods for accessing clinical help for child difficulty involve referral by a general practitioner (GP) or by investigation from Child, Youth and Family (CYF) only after a community complaint against the mother has been laid. These two options are problematic, as firstly, drug-dependent women are hesitant to approach GPs for fear that it may lead to further investigation or prosecution; and secondly, CYF investigation is likely to reinforce this fear rather than facilitating accessible and ongoing intervention and support (Lester & Padbury, 2009; Ornoy, Michailevskaya, Lukashov, Bar-Hamburger, & Harel, 1996). Given that the current study has shown that chronic users with high levels of maternal psychological distress are not widely seeking service support, developing protocols for other more supportive and readily available services need to be developed. If MA mothers were able to access funded support services earlier, MA mothers of children with subclinical behavioural problems might be more inclined to seek our professional sources of support. This may, as a result, effectively ameliorate any adverse emotional and behavioural risks observed among the current MA sample.

Strengths

The present study has many strengths and noteworthy methodological advantages. It is the first of its kind to explore patterns of MA use during the postnatal period, it has a relatively large sample size, it uses structured interviews administered by trained research personnel, it uses a detailed questionnaire to obtain substance use history during each time point for individual drugs and the levels of use for each drug. In addition, a study such as this opens the door for new investigations into the pattern of MA use among mothers.

Methodological strengths of this study included the detailed measure of maternal substance, a demographically similar comparison group and the examination of a wide range of infant and maternal clinical and social factors. In addition, the prospective longitudinal design allowed for detailed information to be collected regarding maternal and children’s family circumstances from pregnancy to 4.5years. Another novel feature of this study was the two-tiered approach to assess developmental outcomes across both groups. This approach consisted of maternal report standardised screening measures (BRIEF-P, SDQ) followed by a lab-based task by an external examiner (Gift Delay, wrap) to assess different aspects of the neurobehavioural construct. By incorporating an objective clinical measure, the current study reduced that likelihood of parent-report biases, given that mood-disorder
and/or substance-dependent women either over or under-report the significance of their child’s difficulty (Chi and Hinshaw, 2002; Mash and Johnston, 1983), leading to some bias in the interpretation of the results.

Problems noted on the SDQ and BRIEF-P tend to persist over time and predict later psychopathology and substance use behaviours (Espy, Sheffield, Wiebe, Clark, & Moehr, 2011; Fontaine, McCrory, Boivin, Moffitt, & Viding, 2011; Goodman et al., 2000). The fact that this study found strong effects of maternal psychological distress on these measures may have substantial public health implications. The ability to identify specific behavioural syndromes in children as early as pre-school age could lead to the development of preventive intervention programmes.

In addition, this study was the first to examine postnatal substance use trends in greater detail to determine patterns of use in conjunction with maternal mental health symptoms among women who use methamphetamine in pregnancy. It therefore provided more information on addiction and MA use among pregnant women over the postpartum period, and how these patterns have an impact on child developmental outcomes.

The current sample of MA children retained to age 4.5 years in the care of their biological mothers was much larger than sample size retained in other studies. For example, by their 3-year follow-up, Smith et al. (2015) had a sample retention rate of 70.4%, and it was recognised that the level of sample drop-out resulted in a loss of valuable data concerning very high-risk and difficult to track children who are most in need of psychosocial support. In contrast, the current study has been able to report the outcomes of high-risk MA children in New Zealand due to the multiple tracking strategies employed by the research team, consequentially resulting in low rates of attrition. Lastly, where other studies have only controlled for the effects of confounding variables such as maternal social background or poly-drug use, the current study employed a hierarchical linear regression analyses to examine the role of such extraneous variables. The current study considered how both maternal psychosocial characteristics and poly-substance use during the postnatal period might alternatively explain MA children’s neurobehavioural inhibition scores at follow-up, thereby examining a wide range of mechanisms shaping the wellbeing of pre-school aged children.

Limitations

Although efforts were made to remedy many of the methodological difficulties characteristic of previous MA studies, the current study is not without its own limitations and these should be considered alongside the interpretation of the findings. The most noteworthy limitation is the reliance on mother’s report of both maternal and child functioning. Underreporting may have occurred due to the mother’s desire to represent the child and herself in the most positive light or as a reflection of increased tolerance for problem behaviours. Alternatively, mothers experiencing psychological difficulties or under the influence of substances may have over reported problem behaviours due to an excessively negative perception of the child’s behaviours. Additionally, the mother may have underreported or inaccurately perceived her own functioning. Self-report of drug use is widely used in research, primarily because of its availability, low cost, and limited intrusiveness compared to biological assays. However, the
limitations of self-report data due to under-reporting are well recognised (Anthony, Neumark, & Van Etten, 2000). Procedures to encourage valid report from mothers were included in data collection (e.g., using experienced interviewers, building strong relationships with these mothers over time). Still, the maternal self-report of postnatal drug use in this research may underestimate to some degree actual drug use, particularly with regard to illicit drugs, such as methamphetamine, ecstasy and other amphetamines. Another potential limitation of the study is that women may not have been able to recall the timing and amount of MA use during their pregnancy and in the postnatal period; however other research supports the use of the calendar method to overcome recall bias (Jacobson et al., 2002). In addition, Jacobson and colleagues (2002) demonstrate that the correlation between antenatal and retrospective reports of cocaine use during pregnancy was higher than any of the other drugs used during pregnancy.

Although this study measured many relevant variables, the conceptual model was not exhaustive and may have excluded other caregiving variables that might be important in understanding the neurobehavioural outcomes of children exposed prenatally to methamphetamine. Additional predictors may be valuable to include in future research. Child temperament, for example, may predict future emotional–behavioural problems (Shaw, Owens, Vondra, Keenan, & Winslow, 1996) and intellectual functioning (Dixon & Smith, 2000). Father involvement and paternal substance abuse also has been found to predict children's developmental outcomes (Coley, 2001). Supportive parenting in early childhood has been found to predict children's adjustment even after the negative effects of harsh discipline are controlled for (Pettit, Bates, & Dodge, 1997). Therefore, it may be that exposure to violence, attachment style, parenting style, paternal executive functioning and attention-related problems would also be appropriate variables to consider as additional predictors.

We were unable to determine if the children were exposed to prenatal psychological distress, such as depression, in utero, as the BSI did not specifically ask about psychological symptoms during pregnancy. We only had information as to whether the mother had ever previously been diagnosed with a mental health condition, anytime prior to 1 month postpartum. This could have been in pregnancy, or several years prior. There is some evidence to suggest that significant mental health difficulties, such as depression, in the first 30 days after delivery, is likely to indicate that the depression started prior to or during pregnancy. However, we cannot be sure if the mental health effects found in this study are due solely to postnatal psychological distress or if exposure to prenatal psychological distress contributes to the effects. Furthermore, the high rates of physical and emotional abuse found in this study among women using methamphetamine during pregnancy suggests that many women may be dealing with several adverse events of the past that are impacting on their mental health and wellbeing. As a result, it would be important to consider including these factors as potential predictors of substance use and psychological distress.

The assessment of mental health concerns or psychological functioning was not performed with a diagnostic tool, however, the BSI instrument used is an established and validated instrument that can determine whether a subject's scores are elevated to the point of clinical concern (Derogatis & Melisaratos, 1983). Although they did not have a formal clinical diagnosis, these women reported significant levels of psychological distress at a clinical level. Moreover, substance use comorbidity was
defined by quality, frequency and pattern of use, and not by a formal clinical diagnosis of problematic use.

The sample included mother-infant dyads drawn from urban neighbourhoods in the Auckland region. Although these inclusion/exclusion criteria were established to control for potential confounders and improve the ability to detect methamphetamine effects, such restriction limits the ability to generalise results beyond the population under study. Moreover, as we included only biological mothers, the sample size was limited to those infants who remained in the custody of their biological mothers from pregnancy to the 4.5-year visit. While in our analyses we did not find large differences in the characteristics or outcomes between those mother-infant dyads included and not included in this study, there is the potential that women that no longer have their children may have the greatest risks.

Suggestions for Future Research

This study provides some of the first evidence that postnatal drug use and maternal psychological distress contribute independently toward high-risk trajectories that might have an underlying neurodevelopmental basis and that might follow a distinct developmental pathway from prenatal substance exposure and to the onset and growth in behavioural dysregulation to changes in neurological deficits. However, before these findings can be expected to influence policy, further research is needed: replication studies and studies with expanded and alternative theoretical scopes that explore similar outcomes among children with prenatal exposure and early adversity across early and late adolescence. To the extent that converging evidence from multiple studies of this population are found, it might be possible to leverage this information for policy changes that address the needs of these individuals. More comparative and experimental findings are also required to better inform policy. It is important to note the advantages of the present prospective data and the time-ordered theoretical sequencing for model specification. At the same time, causation cannot be inferred without rigorous experimental designs. In the interim, it is important that researchers studying populations with early adversity begin to collect high quality measures of substance use whenever possible. Similarly, researchers conducting longitudinal studies of prenatally exposed children must begin to treat the early environment as more than a set of control variables. Integrating these two disciplines will help to develop an evidence base upon which intervention and policy work can be based.

More information is critically needed about the effects of not only maternal drug use, but also the effects of other adverse life circumstances that often occur in tandem with illicit drugs on long-term development of children, family functioning and quality of life. This study underscores the fact that clinicians who care for pregnant women should be comprehensively assessing patients for maternal drug use, and in areas affected by MA, allocation of resources should be planned for intervention with MA-using pregnant women. A priority of future research should be to describe the pathways between maternal psychological distress and child development problems in order to inform intervention approaches and the timing. Future research should also consider whether pathways to child development are similar across various forms of developmental problems (e.g., internalising versus externalising, etc.).
Although maternal drug use and psychological distress seem to be detrimental to parent-child interactions and the development of attachment, this association is not simply unidirectional; it is a transactional system that evolves between the mothers and children over time. Considering and delineating the transactional effects between parental interactions and child characteristics which may act as buffering or vulnerability factors is especially important. Child characteristics and behaviours affect responses from caregivers and vice versa, and therefore influence the future pattern of mother-child interactions. For example, difficult temperaments, which are frequently observed in children of drug-users, have been linked with poor parenting practices (Young Mun, Fitzgerald, Von Eye, Puttler, & Zucker, 2001). Future studies would benefit from accounting for temperament characteristics as a predictor of behavioural outcomes.

Importantly, the effect of prenatal methamphetamine exposure on child neurobehavioural disinhibition at 4.5 years may have been under-represented, due to the previously discussed sample restrictions that may have excluded more severely methamphetamine-exposed children. Future research would benefit from continued study of children exposed prenatally to methamphetamine into the school age and adolescent years to determine whether neurobehavioural disinhibition emerges as children mature. In addition, more consideration should be given to the influence of the postnatal environment, including factors such as parenting beliefs, behaviours and attachment that may mediate the relationship between maternal functioning and child behaviours. Within this line of research, inclusion of multiple indicators of the neurobehavioural disinhibition construct drawn from varying sources such as teachers and more examiner-based measures may yield more reliable and valid measurement of study variables. Alternate conceptual models should also be evaluated, including other relevant environmental factors, such as parental social support and familial exposure to violence.

Conclusion

In summary, this study adds to our understanding of the influence of prenatal methamphetamine exposure on child developmental outcomes, and, in particular, emphasises the importance of the child’s postnatal caregiving environment. Surprisingly, there was no teratogenic effect of prenatal methamphetamine exposure on neurobehavioural disinhibition among pre-schoolers. However, maternal behavioural health, including postnatal drug use and psychological distress did have an effect. The encouraging output of this is that maternal behaviours are much more responsive to interventions, and suggests that early intervention and the right kind of treatment programme targeting both maternal mental health and/or addiction in a family-focused framework can start to ameliorate the potential adverse outcomes of children of this age group.

The findings of this review have particular utility for clinicians involved in the care of women and children. The results support an early approach to prevention and intervention of child developmental problems where maternal psychological health and/or addiction represent an early, modifiable influence. As such, routine prenatal care should encompass psychosocial assessment of mental health and/or addiction concerns and referral or intervention as required. This process should continue throughout the postpartum period, either in the care of a perinatal clinician, a paediatrician or a mental health worker.
Children who have a parent with mental health and/or addiction concerns are at an increase risk of a number of poor developmental outcomes, including poor neurobehavioral disinhibition, which has been linked with developing mental health and/or addiction difficulties themselves. However, these risks are considered malleable and expanding international evidence indicates a number of interventions are effective in improving short- and longer-term outcomes for this group of children. While some progress has been made in recent years, children of parents with mental health and/or addiction difficulties generally remain an “invisible population” within New Zealand. All mental health and addiction services have a role in recognising, supporting and protecting these children by working proactively to intervene early, support strengths, and address vulnerabilities.

Substance use and psychological distress among pregnant and postpartum mothers is a leading preventable cause of mental, physical, and psychological problems in infants and children. The results of this study indicated that maternal psychological distress plays a large role in behavioural outcomes among pre-school-aged children. Children exposed to high levels of maternal psychological distress face unique and greater risks for poor outcomes later in life. Independent of methamphetamine exposure, children with more responsive home environments to developmental and emotional needs demonstrated lower risks for neurobehavioral disinhibition. Further, increased maternal psychological symptoms were associated with increased neurobehavioral disinhibition. These findings highlight the importance of interventions that address both the child and parent needs in order to optimise the child outcome. Identifying these children in the context of early screening in infancy and pre-school has the potential to lead to reduced costs and improved outcomes. Priority should be given to development of effective screening and intervention efforts to assist pregnant and postpartum women in seeking mental health and/or addiction treatment, evaluation of the effectiveness of current treatment programs, and investigation of barriers to treatment of women with comorbid conditions.


Hrebícková, I., Malinová-Sevcíková, M., Macúchová, E., Nohejlová, K., & Slamberová, R. (2014). Exposure to methamphetamine during first and second half of prenatal period and its

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APPENDICES

Appendix A. Participant Information Sheet - Recruitment

Clinical Division - Health Psychology

SCHOOL OF MEDICINE
The University of Auckland Private Bag 92019
Auckland
New Zealand.
Level 12
Auckland Hospital Support Building
Grafton, Auckland
www.health.auckland.ac.nz
Telephone: 64 9 373 7599 ext 86221
Facsimile: 64 9 373 7013
Email: t.wouldes@auckland.ac.nz

Infant Development, Environment And Lifestyle Study

“IDEAL STUDY”

Participant Information

You are invited to participate with your baby in a study being carried out under the direction of Dr Trecia Wouldes, a Developmental Psychologist from the University of Auckland.

WHAT IS THE STUDY?

The purpose of the study is to learn more about the growth and development of children born to mothers who have used any methamphetamines during their pregnancy. This includes drugs that are often referred to as Xstasy, “P”, Pure, Ice, Crystal Meths, or Speed. At present we don’t know very much about the effect these drugs may have on a baby or young child’s development. Therefore, we are joining four other research groups that are located in the United States to look at the physical, emotional and behavioural development of children exposed to methamphetamines prenatally.

The study will involve two groups of babies and their mothers. These groups include: a group of 120 mothers who have reported they have used methamphetamines during their current pregnancy and their babies and a group of 120 mothers who have reported they have not used methamphetamines during their current pregnancy and their babies. All English speaking mothers living in the Auckland area who report that they have used methamphetamines during their pregnancy will be eligible for inclusion in the study. However, some mothers and infants may need to be excluded due to mental and physical health problems. Mothers that will be excluded are those who have a history of psychosis or mental illness or demonstrate obvious signs of psychotic behaviour. Infants that will be excluded are infants that are seriously ill at birth. Recruitment for this study will be through three hospitals, National Women’s, North Shore and Waitakere.

WHAT DOES THE STUDY INVOLVE?

If you agree to take part, we will interview you periodically over the next 3 years and ask you to bring your child to our IDEAL child development centre at the University of Auckland where we will measure your child’s physical, emotional and behavioural development. The IDEAL development centre is located across from the Starship Children’s Hospital in Grafton Road. If you do not have transport, we will arrange a taxi for each visit.
The following is an outline of what will be included in the interviews and the measures we will be using to look at your child’s development.

**WITHIN TWO DAYS AFTER YOUR CHILD’S BIRTH:**

1. We will ask you to complete a short interview that will ask you about your current pregnancy, whether you used any drugs in the three months prior to becoming pregnant and during your pregnancy. We will also ask you about how your pregnancy went and whether you had any complications. At this time we will also review the hospital records of you and your newborn for medical information, such as medications you had during labour and delivery and your baby’s birth weight. This interview will take approximately 15-20 minutes and is usually done before you leave hospital. If you leave hospital before this interview and the following assessments of your infant, we will ask your permission to visit you at your home.

2. At this time we will also assess your infant’s early behavioural capabilities. This assessment will consist of a detailed physical examination of your baby and an evaluation of your baby’s ability to change their behaviour in response to different situations such as being unwrapped or cuddled. We will look for signs that your baby is healthy such as how well s/he can control their behaviour and feelings, communicate their needs by crying, and whether s/he shows any symptoms of withdrawal or neurological distress. At this time we will “flick” the bottom of your infant’s foot with a probe. This usually startles the infant and makes him/her cry. This is done so that we can test a particular reflex as well as obtain an audiotape of your infant’s cry. This assessment will take about a 1/2-hour.

3. During the time your infant is in hospital we will want to collect a sample of your infant’s first bowel movement. This is called “meconium”. This sample of meconium will be sent to the United States where it will be tested to see if your infant has been exposed to methamphetamines or other stimulants such as cocaine during the last half of gestation. After these samples have been tested they will be destroyed.

4. We will also be asking about 15 mothers who have used methamphetamines during their pregnancy to be audio taped talking about their experience of using methamphetamines during their pregnancy, what effect they think using this drug may have for the development of their infant and why they chose to use this drug. After these interviews are transcribed you will have an opportunity to read the transcript and make any changes you wish. These audiotapes can also be returned to you.

**AT APPROXIMATELY 1 MONTH AFTER YOUR BABY IS BORN:**

1. At approximately one-month after your infant is born we will ask you to bring your infant to the IDEAL child development centre. At this time we will interview you again. Included in this interview are general questions about your education, your background and your current and past history of drug use. The interviewer will also ask about any symptoms of emotional distress such as levels of depression.

2. At this time we will repeat the assessment of your child’s behavioural capabilities that we did at birth and tape record your child’s cry for later analysis as we described in number 2. above. The interview and infant assessment will take approximately 1 hour.

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IDEAL STUDY, VERSION 2, 27/02/2004 2/5
WHEN YOUR INFANT IS 6 MONTHS OLD:

1. We will ask you about how you ha ve been feeling over the past 6-months and whether you have experienced any signs or symptoms of depression. We will also videotape you playing with your infant and ask you about your child’s behaviour in the past 6-months. This will take approximately 45 minutes. Using these videotapes we will measure the emotional communication between you and your child. After these tapes have been reviewed you can have them returned to you if you wish.

2. At this time we will test your child’s ability to attend to and remember photographs of people and shapes. This will take approximately 10 minutes.

WHEN YOUR CHILD IS 12, 24 and 36 MONTHS OLD:

1. At each of these yearly assessments you will be interviewed about your lifestyle including your drug use, your living conditions and your relationship with your child. At 24-months we will ask you questions about your personality and we will ask you to take a vocabulary test. Yearly meetings at 12 and 36 months will last approximately 2 hours, the 24-month visit will last approximately 3 hours. Tea or coffee and a snack for your child will be arranged.

2. At each yearly visit, your child’s emotional, behavioural and physical development will be measured to determine whether he/she is meeting developmental milestones for his/her age group. In addition, at 24 months your child will be videotaped while he/she is carrying out a number of tasks that will test his/her problem solving abilities. In addition, at the 24-month visit we will ask if we can collect two salivary cortisol samples from your infant. One will be taken at the beginning of the assessment and one after your child has completed the developmental tests. Cortisol can be measured in saliva. Collection of salivary cortisol involves taking a cotton swab and collecting a little saliva from your infant’s mouth. The change in cortisol can tell us about individual differences in how children react physically to challenging situations such as being in a strange environment, or learning new tasks. These samples will be sent to the United States to examine the changes in cortisol. After these tests are carried out the samples will be destroyed.

WHEN YOUR CHILD IS 30 MONTHS OLD:

1. We will make a home visit to see how your child interacts within his/her own environment. At this time we will interview you about the resources that you have available to you to help you take care of your child and other family members. We will also ask you about your neighbourhood and your relationships with neighbours and other family members. This interview will take approximately 1 ½ hours.

BENEFITS

You and your child will receive no direct benefit from this study. Your child’s development will be monitored closely by study personnel. If you or members of our team have concerns about your child’s development, appropriate referrals will be made. In addition, the results of this study will help us understand how lifestyle and environment during and after pregnancy affects child development.
INCONVENIENCES OR RISKS

The assessment of your baby’s cry that will take place during the hospital examination and 1 month home visit necessarily requires us to make your baby cry. The probe used to cause a startle followed by a cry may cause your baby a slight discomfort that quickly disappears. Some of the questions we ask are of a personal and sensitive nature. You may feel uncomfortable when answering these questions and may choose not to answer any of the questions we ask.

There are no other discomforts or dangers to you or your child.

PARTICIPATION

Your participation in this study is entirely voluntary (your choice), you do not have to take part if you do not wish. If you agree to take part in the study you are free to withdraw at any time without giving a reason and this will not affect your hospital care or support you may be receiving through other social services now or in the future.

If for some reason, you are unable to come to our development centre, we are asking your permission to allow us to ask questions of whoever is the primary person who is caring for your child at the time.

If you have any complaints about your or your child’s participation in this study, or would like more information about the rules for research studies, or the rights of people who take part in such studies, you may wish to contact the Health Advocates Trust on telephone 0800 555 050 Franklin to Northland.

For Maori health support, or to discuss any concerns or issues regarding this study, please contact Mata Forbes RN(ON), Maori Health Services Co-ordinator/Advisor, 5th Level, GM Suite, Auckland City Hospital. Tel 307 4949 extn. 23939 or Mobile 021 348 432.

COMPENSATION

In the unlikely event of a physical injury as a result of your participation in this study, you may be covered by ACC under the Injury Prevention, Rehabilitation and Compensation Act. ACC cover is not automatic and your case will need to be assessed by ACC according to the provisions of the 2002 Injury Prevention Rehabilitation and Compensation Act. If your claim is accepted by ACC, you still might not get any compensation. This depends on a number of factors such as whether you are an earner or non-earner. ACC usually provides only partial reimbursement of costs and expenses and there may be no lump sum compensation payable. There is no cover for mental injury unless it is a result of physical injury. If you have ACC cover, generally this will affect your right to sue the investigators.

CONFIDENTIALITY

All information you give us will be treated in the strictest confidence. No material which could personally identify you will be used in any reports on this study. All interviews, audiotapes and videotapes will be assigned a code number. Interviewers will only know your infant’s date of birth and your first names. They will not have access to any other identifying information. The developmental psychologists that assess your infant will not know whether you have used drugs during pregnancy and will also only be able to identify you through your code number, your
infant’s date of birth and your first names. These codes and the names associated with them will be stored separately on external drives and locked in separate cabinets.

No information collected in this study will be placed in your or your baby’s hospital or medical records. The study will have a comprehensive security system, with all information you provide being stored anonymously on computer files. Access to these files will be confined to study investigators.

Although we will make every attempt to protect your privacy, if one of the researchers should find that your child is at risk physically or developmentally we will refer you to the study paediatrician or appropriate social services.

**IF YOU WANT TO KNOW MORE**

If you want to know more about the study (either now or at a later date) please feel free to contact:

**Dr Trecia Wouldes**, Developmental Psychologist, Department of Psychological Medicine, University of Auckland, Ph: 373 7599, extension 86621, or

**Dr Carl Kuschel**, Neonatal Specialist, National Women’s Health, Ph: 638 9919, extension 4082 or

**Associate Professor Janie Sheridan**, School of Pharmacy, University of Auckland, Ph: 373-7599, extension 85427

*We are committed to treating all our study participants in a fair and ethical manner. This study has received ethical approval from the Auckland Ethics Committee. Finally, we would like to thank you for considering assisting us with this research.*
CONSENT FORM

Infant Development, Environment and Lifestyle - “IDEAL STUDY”

REQUEST FOR INTERPRETER

<table>
<thead>
<tr>
<th>Language</th>
<th>Request</th>
<th>Yes</th>
<th>No</th>
</tr>
</thead>
<tbody>
<tr>
<td>English</td>
<td>I wish to have an interpreter.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Maori</td>
<td>E hiahia ana ahau ki tetahi kaiwhakamaori/kaiwhaka pakeha korero.</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>Samoan</td>
<td>Out e mana’o ia I ai se fa’amatala upu.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tongan</td>
<td>Oku ou fiema’u ha fakatoniuea.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cook Island</td>
<td>K a inangaro au I tetai tangata uri reo.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nieuau</td>
<td>Fia manako au ke fakanoga e taha tagata fakahokohoko kupu</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

I have read and I understand the attached information sheet that invites me and my child to take part in the IDEAL STUDY that is designed to follow the development of my child until he/she is 3 years old. YES NO

I have had the opportunity to use whanau support or a friend to help me ask questions and understand the study. YES NO

I understand that taking part in this study is voluntary (my choice) and that I may withdraw from the study at any time and that this will in no way affect my or my child’s continuing health care. YES NO

I understand that my participation in this study is confidential and that no material which could identify me or my child will be used in any reports on this study. YES NO

I understand the compensation provisions for this study. YES NO

I have had adequate time to consider whether to take part. YES NO

I know who to contact if I have any questions about the study. YES NO
I consent to a sample of my baby’s first bowel movement being sent to the United States so that it can be tested for tobacco as well as illegal drugs such as methamphetamine, cocaine and opiates. YES NO

I consent to a sample of my child’s saliva being sent to the United States so that it can be tested for a hormone called “cortisol” which is a measure of stress. YES NO

I understand that if I allow the bowel movement sample and the saliva sample to be sent to the United States for testing that this consent does not allow the researchers or laboratories to store these samples for further use or to do genetic testing on these samples. YES NO

I consent to my interview being audio-taped YES NO

I consent to my child’s cry being audio-taped YES NO

I consent to myself and my child being videotaped playing together with toys. YES NO

I wish to receive a copy of the results of this study. YES NO

I ___________________________ hereby consent to take part in this study.

Date: __________________ Signature: ______________________________

I ___________________________ hereby consent for my child to take part in this study.

Date: __________________ Signature: ______________________________

CONTACT DETAILS FOR RESEARCHERS:

Dr Trecia Woulde Phone: 373-7599 extension 86221
Dr Carl Kuchel Phone: 638-9919 extension 4082

Dr Janie Sheridan Phone: 373-7599 extension 85427

Project explained by: ________________________________

Project Role: ________________________________

Date: __________________ Signature: ________________________________
Appendix C. Participant Information Sheet – 4.5 Years

Participant Information

Clinical Division - Health Psychology

Infant Development, Environment And Lifestyle Study

“IDEAL STUDY”

Participant Information

You are invited to continue your participation with your child in our Infant Development, Environment And Lifestyle (IDEAL) Study being carried out under the direction of Dr Trecia Woudes, a developmental psychologist in the Department of Psychological Medicine at the University of Auckland.

WHAT IS THE STUDY?

The purpose of the study is to learn more about the growth and development of children born to mothers who have used any methamphetamine during their pregnancy. Dr. Trecia Woudes would like to extend the NZ IDEAL Study by collecting further data at 4½ and 5½ years of age to determine whether children exposed to drugs that are often referred to as Xstasy, “P”, Pure, Ice, Crystal Meths, or Speed, as well as alcohol or other drugs are prepared for formal schooling. Indicators for “school readiness” are likely to include good health or not meeting developmental milestones that originate during early childhood. Therefore the IDEAL Team would like to welcome you back to attend further developmental follow-ups.

WHAT DOES THE STUDY INVOLVE?

If you agree to take part, we will ask you to bring your child to our research centre for two further visits when your child is 4½ and 5½ years of age. At these visits we will interview you and observe your child completing further tests to determine their readiness for school. Our research centre is located at the University of Auckland across from the Starship Children’s Hospital on Grafton Road. If you do not have transport, we will arrange and pay for a taxi for each visit or provide you with petrol vouchers of $20.00 for each visit.

The following is an outline of what will be included in the interviews and the measures we will be using to look at your child’s development.
WHEN YOUR CHILD IS 4 ½ AND 5½ YEARS OF AGE:

1. At the 4½ and 5½ year visit, you will be interviewed about your lifestyle including your drug use, your living conditions and your relationship with your child. We will also ask you questions about what resources are available to you to help you take care of your child and other family members. Additionally, health and nutrition issues related to your child will be investigated. Each visit will take approximately 2-3 hours. Tea or coffee and a snack for you and your child will be arranged.

2. Your child’s emotional, behavioural, learning skills and physical development will be measured to determine whether he/she is prepared for formal school entry at 4½ and how he/she is settling into the school environment at 5½ years. In addition, your child will be videotaped while he/she is carrying out a number of tasks that will test his/her problem solving abilities. Similar to the visit at 24-months, at the 4½ year visit we will again ask if we can collect two saliva samples from your child. One will be taken at the beginning of the assessment and one after your child has completed the developmental tests. At 4½ years we would also like to seek permission to collect a sample of your saliva as well. These samples will be tested for cortisol, a substance in your body that has been associated with stress. You may remember, that the collection procedure involves taking a cotton swab and collecting a little saliva from you and your child’s mouths. The change in cortisol can tell us about individual differences in how adults and children react physically to challenging situations such as being in a strange environment, or learning new tasks. These samples will be sent to the United States. After these tests are carried out the samples will be destroyed.

3. At the 4½ year visit we will also be asking if we can use a further cotton swab of saliva from you and your child’s mouth to collect genetic material. Research has shown that stressful experiences in life can affect our behaviour and health through turning on and turning off certain genes. When this occurs there may be markers left in specific genes. Determining which genes may be affected by life experiences may help us in developing treatments to counteract these effects. We will test the saliva we collect to determine whether certain genes have been affected by different life experiences such as stress. These tests will be carried out in NZ. When they are complete the samples will be destroyed. The samples of saliva we collect will only have a code number associated with them so that they can never be associated with you or your child.

4. A vision test will also be included at 4½ years. The vision testing for this study will last approximately 30 minutes during which time we will test your child’s ability to see objects in the distance, to use both eyes together as a team and to judge the direction of movements of dots on a screen using a special computer programme. We will also be videotaping your child’s eye movements to help us judge how well they are performing on these tests. Frequent breaks will be offered to your child as necessary.

5. When your child is 5½ years of age we will also ask you if we can write to your child’s teacher to ask him/her to fill out two questionnaires about your child’s learning and behaviour in the classroom. We will not tell the teacher anything about your child, other than he/she is involved in a lifestyle study investigating the growth and development of NZ children exposed to a variety of risk factors that may or may not include things like low income, restricted resources, family mental illness, and tobacco, alcohol and/or drug use.
BENEFITS

You and your child will receive no direct benefit from this study. Your child’s development will be monitored closely by study personnel. If you or members of our team have concerns about your child’s development, appropriate referrals will be made. In addition, the results of this study will help us understand how lifestyle and environment during and after pregnancy affects school readiness and health. However, should we find any problems with your child’s vision you will receive a referral for further eye testing and information regarding financial support to correct your child’s vision.

INCONVENIENCES OR RISKS

Some of the questions we ask are of a personal and sensitive nature. You may feel uncomfortable when answering these questions and may choose not to answer any of the questions we ask.

There are no other discomforts or dangers to you or your child.

PARTICIPATION

Your participation in this study is entirely voluntary (your choice), you do not have to take part if you do not wish. If you agree to take part in the study you are free to withdraw at any time without giving a reason and this will not affect your health care or support you may be receiving through other social services now or in the future.

If for some reason, you are unable to come to our development centre, we are asking your permission to allow us to ask questions of whoever is the primary person who is caring for your child at the time.

If you have any queries or concerns regarding your rights as a participant in this research study, you can contact an independent Health and Disability Advocate. This is a free service provided under the Health and Disability Commissioner Act:

Telephone (NZ wide): 0800 555 050
Free Fax (NZ wide): 0800 2787 7678 (0800 2 SUPPORT)
Email: Advocacy@hdc.org.nz

COMPENSATION

In the unlikely event of a physical injury as a result of your participation in this study, you may be covered by ACC under the Injury Prevention, Rehabilitation and Compensation Act. ACC cover is not automatic and your case will need to be assessed by ACC according to the provisions of the 2002 Injury Prevention Rehabilitation and Compensation Act. If your claim is accepted by ACC, you still might not get any compensation. This depends on a number of factors such as whether you are an earner or non-earner. ACC usually provides only partial reimbursement of costs and expenses and there may be no lump sum compensation payable. There is no cover for mental injury unless it is a result of physical injury. If you have ACC cover, generally this will affect your right to sue the investigators.
CONFIDENTIALITY

All information you give us will be treated in the strictest confidence. No material which could personally identify you will be used in any reports on this study. All interviews, audiotapes and videotapes will be assigned a code number. The developmental psychologists that assess your infant will not know whether or not you have used drugs during pregnancy and will also only be able to identify you through your code number, your infant’s date of birth and your first names. These codes and the names associated with them will be stored separately on external drives and locked in separate cabinets.

No information collected in this study will be placed in your or your baby’s hospital or medical records. The study will have a comprehensive security system, with all information you provide being stored anonymously on computer files. Access to these files will be confined to study investigators.

Although we will make every effort to protect your privacy, if one of our researchers should find that your child is at risk physically or developmentally we will refer you to the study psychologist, your General Practitioner or appropriate social services and this will be discussed with you prior to referral.

IF YOU WANT TO KNOW MORE

If you want to know more about the study (either now or at a later date) please feel free to contact:

Dr Trecia Woudes, Developmental Psychologist, Department of Psychological Medicine, University of Auckland, Direct Dial 923 6221.

Dr Nicola Astice, Optometrist, Department of Optometry and Vision Science, University of Auckland, Direct Dial 923 2956.

Dr Ben Thompson, Optometrist, Department of Optometry and Vision Science, University of Auckland, Direct Dial: 923 6020.

We are committed to treating all our study participants in a fair and ethical manner. This study has received ethical approval from the Northern X Regional Ethics Committee.

Finally, we would like to thank you for considering assisting us with this research.
Appendix D. Consent Form – 4.5 Years

Mother and Child Consent Form

Clinical Division - Health Psychology

MOTHER AND CHILD CONSENT FORM

Infant Development, Environment and Lifestyle - “IDEAL STUDY”

REQUEST FOR INTERPRETER

<table>
<thead>
<tr>
<th>Language</th>
<th>Request Language</th>
<th>English</th>
<th>Maori</th>
<th>Samoan</th>
<th>Tongan</th>
<th>Cook Island</th>
<th>Niuean</th>
</tr>
</thead>
<tbody>
<tr>
<td>I wish to have an interpreter.</td>
<td>E hiahia ana ahau ki tetahi kaiwhakamaori/kaiwhaka pakeha korero.</td>
<td>Yes</td>
<td>No</td>
<td>Yes</td>
<td>No</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>Out e mana’o ia I ai se fa’amatala upu.</td>
<td>Io</td>
<td>Leai</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Oku ou fiema’u ha fakatomulea.</td>
<td>Io</td>
<td>Ikai</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>K a inangaro au I tetai tangata uri reo.</td>
<td>Yes</td>
<td>No</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fia manako au ke fakaaoaga e taha tagata fakahoko ho kupu</td>
<td>E</td>
<td>Nakai</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

I have read and I understand the attached information sheet dated March 2011 that invites me and my child to take part in the IDEAL STUDY that is designed to follow the development of my child at 4 ½ and 5 ½ years of age. YES NO

I have had the opportunity to use whanau support or a friend to help me ask questions and understand the study. YES NO

I understand that taking part in this study is voluntary (my choice) and that I may withdraw from the study at any time and that this will in no way affect my or my child’s continuing health care. YES NO

I understand that my participation in this study is confidential and that no material which could identify me or my child will be used in any reports on this study. YES NO

I understand the compensation provisions for this study. YES NO

I have had adequate time to consider whether to take part. YES NO
Mother and Child Consent Form

I know who to contact if I have any questions about the study.  
YES  NO

I consent to a sample of my child’s saliva being collected so that it can be tested for a substance called “cortisol” which is a measure of stress.  
YES  NO

I consent to a sample of my saliva being collected so that it can be tested for a substance called “cortisol” which is a measure of stress.  
YES  NO

I consent to a sample of my child’s saliva being collected so that it can be tested to see whether some genes are associated with stress.  
YES  NO

I consent to a sample of my saliva being collected so that it can be tested to see whether some genes are associated with early childhood stress.  
YES  NO

I consent to myself and my child being videotaped playing together with toys.  
YES  NO

I consent to my child being videotaped for eye movements during the vision testing.  
YES  NO

I wish to receive a copy of the results of this study.  
YES  NO

I  hereby consent to take part in this study.

Date:  Signature:  

I  hereby consent for my child to take part in this study.

Date:  Signature:  

CONTACT DETAILS FOR RESEARCHERS:

Dr Trecia Woulde  
Phone:  DD: 923 6221

Dr Nicola Anstice  
Direct Dial: 923 2956.

Dr Ben Thompson  
Direct Dial: 923 6020.

Project explained by:  

Project Role:  

Date:  Signature:  

IDEAL STUDY, VERSION 5, March 2011
Appendix E. Lifestyle Interview

Form 423—NZ Lifestyle Interview - 12/24/36/54/66

<table>
<thead>
<tr>
<th>StudyNumber:</th>
<th>Node:</th>
<th>Site:</th>
</tr>
</thead>
</table>

<table>
<thead>
<tr>
<th>ID #:</th>
<th>Name Code:</th>
<th>Visit #:</th>
<th>Date of Assessment:</th>
<th>(MM/DD/YYYY)</th>
<th>Evaluator #</th>
</tr>
</thead>
</table>

A. IDENTIFICATION

Instructions: Items 1-3 are to be completed by the interviewer. The determination of primary caretaker sheet is used to determine primary caretaker (Item 1).

1. Primary caretaker:
   (Use relationship codes)
   a. Interview respondent:
      (Use relationship codes)
   b. Is the respondent?
      - 1-Primary caretaker
      - 2-Parent
      - 3-Alternate caretaker
      - 4-Informed person

2. Date of last visit:
   / / / 

3. Child’s corrected age:

4. Reference date: (One year prior to date of this interview)
   / / / 

Opening script: “This interview asks questions about who lives in the house where ______ (study child) lives and what services ______ (study child) and you (primary caretaker) have received. We will also spend a few minutes discussing the neighborhood in which ______ (study child) lives.”

5. Is the biological mother currently in the home?
   - 1-Yes
   - 2-Deceased

The word “you” refers to the primary caretaker who is living in the same household as the study child. If the respondent is not the primary caretaker, substitute the primary caretaker’s name for the word “you” in the following questions.

6. What is your [primary caretaker’s] marital status?
   - 1-Married
   - 2-Separated
   - 3-Living together/De Facto
   - 4-Widowed
   - 5-Divorced
   - 6-Never married (skip to A7)
   a. How long have you been in this status? (Ask for anniversary date)
      / / Years / / Months

   Draft
A. IDENTIFICATION (continued)

7. Please list all of the people who currently live in the household with (study child): If a sibling has the same biological mother and same biological father as the study child, skip adopted sibling column.

Do not include the study child in this list. Ask all applicable questions before going on to the next person.

<table>
<thead>
<tr>
<th>Relationship to child</th>
<th>Relationship code</th>
<th>Same biological mother as child</th>
<th>Same biological father as child</th>
<th>Adopted sibling</th>
<th>Age at last b/day</th>
</tr>
</thead>
<tbody>
<tr>
<td>a.</td>
<td></td>
<td>Yes</td>
<td>No</td>
<td>N/A</td>
<td>O 1</td>
</tr>
<tr>
<td>b.</td>
<td></td>
<td>Yes</td>
<td>No</td>
<td>N/A</td>
<td>O 1</td>
</tr>
<tr>
<td>c.</td>
<td></td>
<td>Yes</td>
<td>No</td>
<td>N/A</td>
<td>O 1</td>
</tr>
<tr>
<td>d.</td>
<td></td>
<td>Yes</td>
<td>No</td>
<td>N/A</td>
<td>O 1</td>
</tr>
<tr>
<td>e.</td>
<td></td>
<td>Yes</td>
<td>No</td>
<td>N/A</td>
<td>O 1</td>
</tr>
<tr>
<td>f.</td>
<td></td>
<td>Yes</td>
<td>No</td>
<td>N/A</td>
<td>O 1</td>
</tr>
<tr>
<td>g.</td>
<td></td>
<td>Yes</td>
<td>No</td>
<td>N/A</td>
<td>O 1</td>
</tr>
</tbody>
</table>

8. Interviewer: Choose the one situation that best describes the study child's current living situation. (Disregard siblings in present living situation.) Do not ask this question. Code from information in A7.

- O 1-Both biological parents
- O 2-Biological mother only
- O 3-Biological father only
- O 4-Both biological parents in extended family
- O 5-Biological mom in extended family
- O 6-Biological dad in extended family
- O 7-Maternal grandparent(s)
- O 8-Paternal grandparent(s)
- O 9-Other non-adoptive relative(s)
- O 10-Relative adoptive parent

   a. Were the same people living in the household on _________ (reference date)?
      O 1-Yes    O 0-No (If no, make note in margin how household was different)
B. MOTHER INVOLVEMENT

If mother is deceased, ask, "How many living biological children currently under 18 did the mother have in total, including this child?" B.2 will = Yes, and B.2a will = B1 minus 1.

1. How many living biological children under 18 does the mother have in total including this child?
   ○ 99-Unknown

2. Does the mother have any biological children under 18 who are not living with her? (exclude study child)
   ○ 1-Yes (if Yes, complete 2a & 2b)
   ○ 2-No
   ○ 3-Unknown

   a. How many biological children under 18 years of age are not living with the mother? (exclude study child)
      ○ 99-Unknown

   b. Please tell me the gender and ages of (study child) brothers and sisters who are under 18 and are not living with him/her, but have the same biological mother as (study child). USE CODE FOR RELATIONSHIP.

<table>
<thead>
<tr>
<th>Comments</th>
<th>Age at last birthday</th>
<th>Sex</th>
<th>Same biological father as child</th>
<th>With whom is sibling living?</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td></td>
<td>O 1</td>
<td>Yes</td>
<td>O 1</td>
</tr>
<tr>
<td>2.</td>
<td></td>
<td>O 2</td>
<td>No</td>
<td>O 2</td>
</tr>
<tr>
<td>3.</td>
<td></td>
<td>O 1</td>
<td>Yes</td>
<td>O 1</td>
</tr>
<tr>
<td>4.</td>
<td></td>
<td>O 2</td>
<td>No</td>
<td>O 2</td>
</tr>
</tbody>
</table>

* Use relationship code system. This question should be coded to reflect adult's relationship to the sibling, not the study child (e.g., If the sibling is living with his/her father who is not the study child's father, this would be coded as 002 because the relationship code is determined by the adult's relationship to the sibling, not the study child).

If A4 pg. 1 (biological mother in home) = "Yes" or biological mom is deceased, skip to section D1.

3. With whom or where does the mother live?
   ○ 1-Alone
   ○ 2-With child's father (child lives elsewhere)
   ○ 3-With relative(s)
   ○ 4-Without non-relative(s)
   ○ 5-Incarcerated
   ○ 6-Hospital or institution
   ○ 7-Military
   ○ 8-Transient, no stable living situation
   ○ 9-Unknown
NZ Lifestyle Interview - 12/24/36/54/66 (continued)

B. MOTHER INVOLVEMENT (continued)

4. How often does the mother see the child?
   ○ 1-Every day
   ○ 2-Once or twice a week
   ○ 3-Once or twice a month
   ○ 4-Less than once a month
   ○ 5-No contact
   ○ 6-Unknown

5. What best describes the mother's current plans in relationship to her child?
   ○ 1-Separation is long-term
   ○ 2-Future plans uncertain
   ○ 3-Plans to reunite with child
   ○ 4-Unknown (no information available)

6. Has the study child's last name changed since the last visit?
   ○ 1-Yes
   ○ 0-No
   a. If yes, is the child's last name the same as the child's father's?
      ○ 1-Yes
      ○ 0-No

C. BIOLOGICAL MOTHER'S CURRENT PARTNER

Interviewer: This section is only administered to the biological mother. If the respondent is not the biological mother, skip to D1. Ask the following questions as they relate to the biological mother's current partner, regardless of whether or not he is the biological father. Please refer to the current partner/father of the baby by name throughout this section.

Script: "Now we want to ask you some questions about your current partner."

1. Do you currently have a partner?
   ○ 1-Yes
   ○ 0-No (skip C3)

2. Is your current partner the father of [study child]?
   ○ 1-Yes
   ○ 0-No

Interviewer: Code the following item based on the response to item C2

3. Identity of the partner to be discussed in this section
   ○ 1-Father of the study child
   ○ 2-Current partner (not father of the study child)

Script: "For the next few questions, let's talk about _________ (current partner's name)."

4. How long have you and [current partner] been together?
   [ ] Years [ ] Months

(Note: Inquire what is the date, so months can be calculated accurately and record in margin)
**NZ Lifestyle Interview - 12/24/36/54/66 (continued)**

<table>
<thead>
<tr>
<th>ID #:</th>
<th>Name Code:</th>
<th>Date of Assessment:</th>
<th>Site:</th>
</tr>
</thead>
</table>

### C. BIOLOGICAL MOTHER’S CURRENT PARTNER (continued)

5. Does (current partner) currently smoke cigarettes?
   - 1-Yes
   - 0-No

6. Does (current partner) currently drink alcohol?
   - 1-Yes
   - 0-No

7. Does (current partner) currently use drugs?
   - 1-Yes
   - 0-No
   - a. if yes, which drugs does he use? (select all that apply)
     - 1-Marijuana/Hashish
     - 1-Methamphetamine
     - 1-Ecstasy
     - 1-Amphetamines
     - 1-Cocaine / Crack
     - 1-Benzodiazepines / Tranquilizers
     - 1-Berbiturates / Sedatives
     - 1-Heroin / Methadone
     - 1-Opiates
     - 1-LSD / Hallucinogens
     - 1-MSTI
     - 1-Homebake

8. Since (reference date), has (current partner) been in any type of drug or alcohol treatment or support program?
   - 1-Yes
   - 0-No
   - 2-Unknown

**Interviewer:** If the biological mother’s current partner is the biological father of the study child, skip to D1

9. Is biological father deceased?  
   - 1-Yes (skip to D1)  
   - 0-No

**Script:** "The remainder of this section is about (study child’s) biological father."

10. With whom or where does the child’s biological father live?
    - 1-Alone
    - 4-With non-relative(s)
    - 7-Military
    - 2-With child’s mother (child lives elsewhere)
    - 5-Incarcerated
    - 8-Transient, no stable living situation
    - 3-With relative(s)
    - 6-Hospital or institution
    - 9-Unknown

11. How often is the child’s biological father in contact with the mother?
    - 1-Every day
    - 2-Once or twice a week
    - 3-Once or twice a month
    - 4-Less than once a month
    - 5-No contact
    - 6-Unknown
C. BIOLOGICAL MOTHER'S CURRENT PARTNER (continued)

12. How often does the biological father see the child?
   - 1-Every day
   - 2-Once or twice a week
   - 3-Once or twice a month
   - 4-Less than once a month
   - 5-No contact
   - 6-Unknown

13. What best describes the biological father's current plans in relationship to his child?
   - 1-Separation is long-term
   - 2-Future plans uncertain
   - 3-Plans to reunite with child
   - 4-Unknown (no information available)

14. Does (child's biological father) currently smoke cigarettes?
   - 1-Yes
   - 0-No
   - 2-Unknown

15. Does (child's biological father) currently drink alcohol?
   - 1-Yes
   - 0-No
   - 2-Unknown

16. Does (child's biological father) currently use drugs?
   - 1-Yes
   - 0-No
   - 2-Unknown
   a. If yes, which drugs does he use? (select all that apply)
      - 1-Marijuana/hashish
      - 1-Methamphetamine
      - 1-Ecstasy
      - 1-Amphetamines
      - 1-Cocaine/Crack
      - 1-Benzodiazepines/Tranquilizers
      - 1-Barbiturates/Sedatives
      - 1-Heroin/Methadone
      - 1-Opiates
      - 1-LSD/Hallucinogens
      - 1-MSTI
      - 1-Homework

17. Since (reference date), has (child's biological father) been in any type of drug or alcohol treatment or support program?
   - 1-Yes
   - 0-No
   - 2-Unknown
D. PERSONAL HISTORY

Interviewer: Count listings from A7 - do not ask this question.

1. How many people live in the child's household now? (DO NOT include study child)

2. Do you support or contribute money to the household?
   - ○ 1-Yes
   - ○ 0-No

Does support or contribute money to the household?
(De-identified: Individual need not be living in household. N/A = No such person)

   a. Child's father who is not the child's caretaker
      - ○ 1-Yes
      - ○ 0-No
      - ○ 2-Not known
      - ○ 3-N/A

   b. Primary caretaker's partner who is not the child's father
      - ○ 1-Yes
      - ○ 0-No
      - ○ 2-Not known
      - ○ 3-N/A

   c. Other adult (if more than one, choose one making largest contribution)
      - ○ 1-Yes
      - ○ 0-No
      - ○ 2-Not known
      - ○ 3-N/A

3. Apart from the primary caretaker, who contributes the most money to the child's household?
   - ○ 1-Child's father
   - ○ 2-Caretaker's partner
   - ○ 3-Other adult
   - ○ 4-No other contributors

4. What is the total income in the child's household from all sources over the last year? (Note: Show calculations in margin)
   - ○ <$5,000
   - ○ $5,000-9,999
   - ○ $10,000-19,999
   - ○ $20,000-29,999
   - ○ $30,000-39,999
   - ○ $40,000-49,999
   - ○ $50,000 or more
   - ○ Unknown
D. PERSONAL HISTORY (continued)

Complete questions 5-8 for the primary caretaker and the adult contributing the most money to the household (see D3). If no other contributing adults, complete only for the primary caretaker.

<table>
<thead>
<tr>
<th>Highest grade completed</th>
<th>Occupational scale</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 = 1st – 6th year</td>
<td>0 = Homemaker / Housewife</td>
</tr>
<tr>
<td>2 = Form 1</td>
<td>1 = Benefit recipient; unemployed; farm laborer; menial service worker</td>
</tr>
<tr>
<td>3 = Form 2</td>
<td>2 = Unskilled worker</td>
</tr>
<tr>
<td>4 = Form 3</td>
<td>3 = Machine operator; semi-skilled worker</td>
</tr>
<tr>
<td>5 = Form 4</td>
<td>4 = Skilled manual worker; craftsman; tenant farmer</td>
</tr>
<tr>
<td>6 = Form 5</td>
<td>5 = Clerical, sales worker; small farm owner; small business owner (up to 2 employees)</td>
</tr>
<tr>
<td>7 = Form 6</td>
<td>6 = Technician; semi-professional; small business owner (up to 5 employees)</td>
</tr>
<tr>
<td>8 = Form 7</td>
<td>7 = Medium-small business owner (up to 20 employees); farm owner; manager; minor professional</td>
</tr>
<tr>
<td>9 = 1 Year University</td>
<td>8 = Administrator; lesser professional; proprietor of medium-sized business; graduate student</td>
</tr>
<tr>
<td>10 = 2 Years University</td>
<td>9 = Higher executive; proprietor of large business; major professional</td>
</tr>
<tr>
<td>11 = 3 Years University</td>
<td></td>
</tr>
<tr>
<td>12 = 4 Years University</td>
<td></td>
</tr>
<tr>
<td>13 = BASc/BCom/LLB</td>
<td></td>
</tr>
<tr>
<td>14 = MA/MSc/MChB/MMD</td>
<td></td>
</tr>
<tr>
<td>15 = PhD</td>
<td></td>
</tr>
</tbody>
</table>

5a. What is the highest grade primary caretaker has completed?

<table>
<thead>
<tr>
<th>Highest grade</th>
<th>Educational scale</th>
</tr>
</thead>
</table>

5b. What is the highest grade contributing adult has completed?

<table>
<thead>
<tr>
<th>Highest grade</th>
<th>Educational scale</th>
</tr>
</thead>
</table>

6a. What is primary caretaker’s usual occupation?

<table>
<thead>
<tr>
<th>Occupational scale</th>
</tr>
</thead>
</table>

6b. What is contributing adult’s usual occupation?

<table>
<thead>
<tr>
<th>Occupational scale</th>
</tr>
</thead>
</table>

7a. Is primary caretaker currently working or employed?

- ○ 1-Yes
- ○ 0-No

7b. Is contributing adult currently working or employed?

- ○ 1-Yes
- ○ 0-No

8a. Is primary caretaker currently in school?

- ○ 1-Yes
- ○ 0-No

8b. Is contributing adult currently in school?

- ○ 1-Yes
- ○ 0-No
NZ Lifestyle Interview - 12/24/36/54/66
(continued)

E. HOUSING

1. How many times has (study child) changed addresses since (reference date)?  (Note: Show changes in margin)

2. What best describes the kind of housing the study child currently lives in?
   - 1-Owner-occupied house or condominium
   - 2-Rented apartment or house (if rented, go to E2a)
   - 3-Hotel/Motel
   - 4-Congregate care / Social service facility
   - 5-No stable residence
     a. If rented, is it:
        - 1-Section 8
        - 2-State housing
        - 3-Private rental
        - 4-Unknown

3. How many rooms are in (study child’s) home? (Note: Count LR, DR, BR, KIT, FR, and write out rooms. Code based on household referenced in question E2)

F. CHILD AND FAMILY SERVICES

Script: “Now I would like to ask you about what kind of services you (primary caretaker) and (study child) have received. I am going to read you a list of services that children may receive. Please tell me if (study child) has received any of the services since (reference date).”

1. Since (reference date), did he/she receive or is he/she currently receiving

<table>
<thead>
<tr>
<th>Service</th>
<th>If yes, are you (primary caretaker only) satisfied with the service?</th>
</tr>
</thead>
<tbody>
<tr>
<td>a. Visiting nurse/Home health care</td>
<td>1-Yes</td>
</tr>
<tr>
<td>b. Health screening/specialty care</td>
<td>1-Yes</td>
</tr>
<tr>
<td>c. Psychological/Developmental assessment</td>
<td>1-Yes</td>
</tr>
<tr>
<td>d. Crisis nursery (to protect child)</td>
<td>1-Yes</td>
</tr>
<tr>
<td>e. Therapeutic day care/nursery</td>
<td>1-Yes</td>
</tr>
<tr>
<td>f. Physical therapy/Occupational therapy</td>
<td>1-Yes</td>
</tr>
<tr>
<td>g. Early intervention program (infant stimulation)</td>
<td>1-Yes</td>
</tr>
</tbody>
</table>
NZ Lifestyle Interview - 12/24/36/54/66
(continued)

F. CHILD AND FAMILY SERVICES (continued)

Script: "I am going to read a list of services that families may receive. Please tell me if you (substitute primary caretaker's name if you are interviewing a parent, alternate caretaker or informed person) received any of these services since (reference date)."

2. Did you (primary caretaker or family) receive: (Note: Indicate details of the service received in margin)

<table>
<thead>
<tr>
<th>Services</th>
<th>Received</th>
<th>If yes, are you (primary caretaker only) satisfied with the service?</th>
</tr>
</thead>
<tbody>
<tr>
<td>a. Family planning/Birth control</td>
<td>○ 1-Yes</td>
<td>○ 0-No ○ 2-Unknown</td>
</tr>
<tr>
<td>b. Temporary housing / Temporary shelter</td>
<td>○ 1-Yes</td>
<td>○ 0-No ○ 2-Unknown</td>
</tr>
<tr>
<td>c. Nutritional support</td>
<td>○ 1-Yes</td>
<td>○ 0-No ○ 2-Unknown</td>
</tr>
<tr>
<td>d. Parent training</td>
<td>○ 1-Yes</td>
<td>○ 0-No ○ 2-Unknown</td>
</tr>
<tr>
<td>e. Respite care (for primary caretaker)</td>
<td>○ 1-Yes</td>
<td>○ 0-No ○ 2-Unknown</td>
</tr>
<tr>
<td>f. Shelter for abused women</td>
<td>○ 1-Yes</td>
<td>○ 0-No ○ 2-Unknown</td>
</tr>
<tr>
<td>g. CYFS social worker for family</td>
<td>○ 1-Yes</td>
<td>○ 0-No ○ 2-Unknown</td>
</tr>
<tr>
<td>h. Tangible support (e.g. clothing, appliances, furniture, toys)</td>
<td>○ 1-Yes</td>
<td>○ 0-No ○ 2-Unknown</td>
</tr>
<tr>
<td>i. Transportation</td>
<td>○ 1-Yes</td>
<td>○ 0-No ○ 2-Unknown</td>
</tr>
<tr>
<td>j. Health care (doctor visits - including hospitalization)</td>
<td>○ 1-Yes</td>
<td>○ 0-No ○ 2-Unknown</td>
</tr>
<tr>
<td>k. Mental health - counseling, outpatient</td>
<td>○ 1-Yes</td>
<td>○ 0-No ○ 2-Unknown</td>
</tr>
<tr>
<td>l. Mental health - inpatient</td>
<td>○ 1-Yes</td>
<td>○ 0-No ○ 2-Unknown</td>
</tr>
<tr>
<td>m. Mental health - medication only / medication monitoring</td>
<td>○ 1-Yes</td>
<td>○ 0-No ○ 2-Unknown</td>
</tr>
<tr>
<td>n. Mental health - self-help (e.g., support groups)</td>
<td>○ 1-Yes</td>
<td>○ 0-No ○ 2-Unknown</td>
</tr>
<tr>
<td>o. Alcohol / Drug treatment-inpatient</td>
<td>○ 1-Yes</td>
<td>○ 0-No ○ 2-Unknown</td>
</tr>
<tr>
<td>p. Alcohol / Drug treatment-outpatient</td>
<td>○ 1-Yes</td>
<td>○ 0-No ○ 2-Unknown</td>
</tr>
<tr>
<td>q. Alcohol / Drug-self help (12 step, e.g. AA, NA)</td>
<td>○ 1-Yes</td>
<td>○ 0-No ○ 2-Unknown</td>
</tr>
</tbody>
</table>

Draft
### G. FINANCIAL CAPITAL

1. Since (reference date), have you (primary caretaker) been working for pay?
   - 1-Yes
   - 0-No
   - 2-Unknown
   
   **If yes:**
   
   a. Have you (primary caretaker) been required to work as a condition of receiving a government support check?
      - 1-Yes
      - 0-No
      - 2-Unknown
   
   b. Have you (primary caretaker) been working part-time or full-time?
      - 1-Part-time
      - 2-Full-time
      - 3-Unknown

2. Since (reference date), have you (primary caretaker) attended school or job training?
   - 1-Yes (if yes, indicate in margin what school or job training)
   - 0-No (skip to G3)
   - 2-Unknown
   
   **If yes:**
   
   a. Have you (primary caretaker) been required to attend school or job training as condition of receiving a government support check?
      - 1-Yes
      - 0-No
      - 2-Unknown

*If biological father is deceased or child currently lives with a *non-relative* - adoptive parent*, skip to G5. If the biological father is the primary caretaker, skip to G5.

3. Since (reference date), has the child's biological father attended school or job training?
   - 1-Yes (if yes, indicate in margin what school or job training)
   - 0-No
   - 2-Unknown

4. Since (reference date), has the biological father been working for pay?
   - 1-Yes
   - 0-No
   - 2-Unknown
   
   a. If yes, has the biological father been working part-time or full-time?
      - 1-Part-time
      - 2-Full-time
      - 3-Unknown
5. Is mother receiving child support from child's biological father?
   - 1: Yes
   - 0: No
   - 2: Unknown

6. Since (reference date), have you (primary caretaker) or (study child) received any of the following government benefits?
   - **Income / Subsistence**
     - a. Sickness benefits
     - b. Widows/Oorphaned benefits
     - c. NZ superannuation
   - d. Food stamps
   - e. Other government support (transitional retirement)
   - f. Domestic purposes benefit
   - g. Invalid benefit
   - h. Unemployment benefit

7. Since (reference date), have you (primary caretaker) or (study child) received any of the following medical benefits?
   (See manual for appropriate probes.)
H. HOUSEHOLD ENVIRONMENT

Script: "Now I would like to discuss with you alcohol, tobacco and other drugs used in (study child's) home."

1. Does anyone who lives with (study child) currently smoke cigarettes?
   - 1-Yes
   - 0-No
   - 2-Unknown

2. Does anyone who lives with (study child) currently drink alcohol?
   - 1-Yes
   - 0-No
   - 2-Unknown

3. Does anyone who lives with (study child) currently use drugs?
   - 1-Yes
   - 0-No
   - 2-Unknown
   
a. If yes, which drugs are used? (select all that apply)
   - 1-Marijuana/hashish
   - 1-Methamphetamine
   - 1-Ecstasy
   - 1-Amphetamines
   - 1-Cocaine / Crack
   - 1-Benzodiazepines / Tranquilizers
   - 1-Barbiturates / Sedatives
   - 1-Heroin / Methadone
   - 1-Opiates
   - 1-LSD / Hallucinogens
   - 1-MSTI
   - 1-Homoeake

4. Do any of your friends or family use methamphetamine, ecstasy, or other amphetamines?
   - 1-Yes
   - 0-No
   - 2-Unknown

5. Do any of your friends or family use other drugs such as marijuana, cocaine, crack or heroin?
   - 1-Yes
   - 0-No
   - 2-Unknown
NZ Lifestyle Interview - 12/24/36/54/66
(continued)

I. CHILD PROTECTIVE SERVICES

Script: "This next section will be asking about Child Youth and Family Service involvement, which here in (state) is called (local agency that serves as child protective service).

Interviewer: Use the name of the Local Agency in place of CYFS throughout this section.

1. Has Child Youth and Family Service (CYFS)/Local Agency been involved with (study child) since ____________ (reference date)?
   ○ 1-Yes
   ○ 0-No (skip to J1)

2. Was there a report/referral to CYFS made on behalf of the study child that was open at the last visit?
   ○ 1-Yes
   ○ 0-No

   a. If yes, what were the reasons for the report/referral to CYFS/Local agency? (select all that apply)
      ○ 1-Mother abandoned child
      ○ 1-Mother thought to be incapable to care for child
      ○ 1-Evidence of neglect
      ○ 1-Evidence of physical abuse
      ○ 1-Evidence of sexual abuse
      ○ 1-Maternal drug or alcohol use
      ○ 1-Mother's social or economic circumstances
      ○ 1-Mother's physical or mental condition
      ○ 1-Mother is already known to CYFS
      ○ 1-Mother incarcerated
      ○ 1-Mother deceased
      ○ 1-Unknown reason for referral

   b. What actions were taken as a result of CYFS/Local agency report/referral? (select all that apply)
      ○ 1-The case was opened / accepted for further CYFS services
      ○ 1-Parental rights terminated (complete dates below)
      ○ 1-The child is under CYFS supervision with mother
      ○ 1-The child is under CYFS supervision with other relative
      ○ 1-The child has been placed in out-of-home care
      ○ 1-Unknown status
      ○ 1-The mother indicated a wish to voluntarily relinquish the child for adoption

   If parental rights were terminated:

   Date maternal rights terminated
   [ ] / [ ] / [ ]

   Date paternal rights terminated
   [ ] / [ ] / [ ]
I. CHILD PROTECTIVE SERVICES (continued)

3. Has a report/referral to CYFS been made on behalf of (study child) since the last visit?
   ○ 1-Yes
   ○ 0-No
   a. If yes, How many reports/referrals have been made on behalf of the study child since the last visit?
      
   b. What were the reasons for the report/referral to CYFS/Local agency? (select all that apply)
      ○ 1-Mother abandoned child
      ○ 1-Mother thought to be incapable to care for child
      ○ 1-Evidence of neglect
      ○ 1-Evidence of physical abuse
      ○ 1-Evidence of sexual abuse
      ○ 1-Maternal drug or alcohol use
      ○ 1-Mother's social or economic circumstances
      ○ 1-Mother's physical or mental condition
      ○ 1-Mother is already known to CYFS
      ○ 1-Mother incarcerated
      ○ 1-Mother deceased
      ○ 1-Unknown reason for referral

c. What actions were taken as a result of CYFS/Local agency report/referral? (select all that apply)
   ○ 1-The case was opened / accepted for further CYFS services
   ○ 1-The child is under CYFS supervision with mother
   ○ 1-The child is under CYFS supervision with other relative
   ○ 1-The child has been placed in out-of-home care
   ○ 1-The child indicated a wish to voluntarily relinquish the child for adoption
   ○ 1-Parental rights terminated (complete dates below)

   If parental rights were terminated:
   Date maternal rights terminated
   / / / / / /
   Date paternal rights terminated
   / / / / / /

   If study child has never been discharged from the hospital STOP HERE
NZ Lifestyle Interview - 12/24/36/54/66
(continued)

<table>
<thead>
<tr>
<th>ID #:</th>
<th>Name Code:</th>
<th>Date of Assessment:</th>
<th>Site:</th>
</tr>
</thead>
</table>

J. FOSTER CARE PLACEMENT

1. Is (study child) currently in an out-of-home placement (e.g. foster family care, group home, or residential care)?
   - 1-Yes
   - 0-No
   If no:

   a. Has a report/referral to Child Youth and Family Service (CYFS) been made on any other children in (study child's) current household?
      - 1-Yes
      - 0-No

   If question J1 = "no", answer J1a, then skip to K1. If question J1a = "yes", continue answering questions J1b-f.

   b. How many times has (study child) been removed from the care of his/her mother since (reference date)?
      (Note: Indicate removals in margin)

   c. How many changes in placement has (study child) had since (reference date)? (include changes from one caretaker to another, including his/her mother.)
      (Note: Indicate changes in margin)

   d. What was the date (study child) was placed in current foster care setting?
      (Month) / (Year)

   e. What is the (study child's) current placement setting?
      - 1-Pre-adoptive home
      - 2-Foster family home-relative/licensed
      - 3-Foster family home-relative/not licensed
      - 4-Foster family home-non-relative
      - 5-Group home
      - 6-Institution

   f. (Study child's) most recent case plan goal is best described as:
      - 1-Reunify with parent(s)
      - 2-Live with other relative(s)
      - 3-Adoption
      - 4-Guardianship
      - 5-Case plan goal not yet established
      - 6-Unknown
K. PROBABILITY OF SUCCESS

Script: "I'm going to ask you some questions about (study child's) neighborhood. The questions are divided into three short parts, and for each part I will give you a different scale card to use in answering the questions. In all three parts we are interested in what you think about (study child's) current neighborhood.

For this first part, I'm going to ask you three questions about the chances for kids in (study child's) neighborhood. The answer scale goes from 1 to 5; 1 is very high, 2 is high, 3 is in the middle, neither high nor low, 4 is low, and 5 is very low. Please answer with the number that comes the closest to what you think are the chances for these things happening in (study child's) neighborhood."

Script: In (study child's) neighborhood, what do you think are a teenager's chance of:

<table>
<thead>
<tr>
<th>Activity</th>
<th>Very high</th>
<th>High</th>
<th>In the middle</th>
<th>Low</th>
<th>Very low</th>
</tr>
</thead>
<tbody>
<tr>
<td>Completing Form 5</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>Completing university</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>Landing a stable, well-paying job when adult</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
</tbody>
</table>

L. NEIGHBORHOOD PROBLEMS

Script: "The next part has 18 questions about possible problems in (study child's) neighborhood. The answer scale goes from 1 to 3; 1 is a big problem, 2 is somewhat of a problem, and 3 is not a problem. Please answer with the number that comes closest to how much of a problem you think these things are in (study child's) neighborhood."

Script: In (study child's) neighborhood, how much of a problem do you think the following are:

<table>
<thead>
<tr>
<th>Activity</th>
<th>A big problem</th>
<th>Somewhat of a problem</th>
<th>Not a problem</th>
</tr>
</thead>
<tbody>
<tr>
<td>High unemployment</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>Vandalism</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>Little respect for laws</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>Prostitution</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>Abandoned houses</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>Sexual assaults or rapes</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>Illegal gambling</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>Run down houses</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>AIDS</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>Assaults and muggings</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>Delinquent gangs or drug gangs</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>Homelessness</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
</tbody>
</table>
NZ Lifestyle Interview - 12/24/36/54/66
(continued)

L. NEIGHBORHOOD PROBLEMS (continued)

<table>
<thead>
<tr>
<th></th>
<th>A big problem</th>
<th>Somewhat of a problem</th>
<th>Not a problem</th>
</tr>
</thead>
<tbody>
<tr>
<td>13. Drug use or drug dealing</td>
<td>O 1</td>
<td>O 2</td>
<td>O 3</td>
</tr>
<tr>
<td>14. City officials ignoring problems</td>
<td>O 1</td>
<td>O 2</td>
<td>O 3</td>
</tr>
<tr>
<td>15. Unsupervised children</td>
<td>O 1</td>
<td>O 2</td>
<td>O 3</td>
</tr>
<tr>
<td>16. Teenage mothers</td>
<td>O 1</td>
<td>O 2</td>
<td>O 3</td>
</tr>
<tr>
<td>17. Teenagers hanging out</td>
<td>O 1</td>
<td>O 2</td>
<td>O 3</td>
</tr>
<tr>
<td>18. Police not caring about your problems</td>
<td>O 1</td>
<td>O 2</td>
<td>O 3</td>
</tr>
</tbody>
</table>

M. PEOPLE AND SERVICES IN THE NEIGHBORHOOD

Script: "This part has eight questions about the people and services in (study child’s) neighborhood. The answer scale goes from 1 to 5; 1 means you strongly agree, 2 means you agree, 3 means you are neutral, neither agree nor disagree, 4 means you disagree, and 5 means you strongly disagree. Please answer with the number that comes closest to how you feel about the people and services in (study child’s) neighborhood."

<table>
<thead>
<tr>
<th></th>
<th>Strongly agree</th>
<th>Agree</th>
<th>Neither agree or disagree</th>
<th>Disagree</th>
<th>Strongly disagree</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Your neighbors have similar views about how to raise children.</td>
<td>O 1</td>
<td>O 2</td>
<td>O 3</td>
<td>O 4</td>
<td>O 5</td>
</tr>
<tr>
<td>2. This is a close-knit neighborhood.</td>
<td>O 1</td>
<td>O 2</td>
<td>O 3</td>
<td>O 4</td>
<td>O 5</td>
</tr>
<tr>
<td>3. There are a lot of adults around here that your children can look up to.</td>
<td>O 1</td>
<td>O 2</td>
<td>O 3</td>
<td>O 4</td>
<td>O 5</td>
</tr>
<tr>
<td>4. You can count on people in the neighborhood to let you know about opportunities for your kids.</td>
<td>O 1</td>
<td>O 2</td>
<td>O 3</td>
<td>O 4</td>
<td>O 5</td>
</tr>
<tr>
<td>5. Schools are so bad around here, you can’t blame teenagers for not going to classes.</td>
<td>O 1</td>
<td>O 2</td>
<td>O 3</td>
<td>O 4</td>
<td>O 5</td>
</tr>
<tr>
<td>6. Unless you know the right people, you can’t get services in this neighborhood.</td>
<td>O 1</td>
<td>O 2</td>
<td>O 3</td>
<td>O 4</td>
<td>O 5</td>
</tr>
<tr>
<td>7. If you want good health and social services for your Children, you can’t find them around here.</td>
<td>O 1</td>
<td>O 2</td>
<td>O 3</td>
<td>O 4</td>
<td>O 5</td>
</tr>
<tr>
<td>8. Getting help when your children need it always takes more time and energy than you seem to have.</td>
<td>O 1</td>
<td>O 2</td>
<td>O 3</td>
<td>O 4</td>
<td>O 5</td>
</tr>
</tbody>
</table>
### Appendix F. Substance Use Inventory

#### Form 427—NZ Substance Use Inventory - 12/24/36/54/66 (SUI-12/24/36/54/66)

<table>
<thead>
<tr>
<th>Study Number</th>
<th>Node</th>
<th>Site</th>
</tr>
</thead>
</table>

<table>
<thead>
<tr>
<th>ID #</th>
<th>Name Code</th>
<th>Visit #</th>
<th>Date of Assessment</th>
<th>(MM/DD/YYYY)</th>
<th>Evaluator #</th>
</tr>
</thead>
</table>

**PART II. SUBSTANCE USE HISTORY**

**A. TOBACCO (20 cigarettes = 1 pack)**

1. On the days that you smoked, how much did you smoke?
   - □ □ \# of cigarettes or cigars
   - □ □ other (describe)

2. On average, how many days in one week did you smoke this amount?
   - □ □ Every day
   - □ □ Almost every day
   - □ □ 3-4 times a week
   - □ □ 1-2 times a week
   - □ □ 2-3 times a month
   - □ □ Once a month
   - □ □ Less than once a month

3. What was the most you ever smoked in one day?
   - □ □ \# of cigarettes or cigars
   - □ □ other (describe)

4. Which brand did you smoke?

5. Date quit? (Do not record date of last use)
   - □ □ / □ □ / □ □

**B. ALCOHOL**

1. On the days that you drank, what did you drink and how much? (\# drinks)
   (Note: Ask about size of bottle/glass)
   - Beer □ □
   - Wine □ □
   - Liquor □ □

2. On average, how many days in one week did you drink this amount?
   - □ □ Beer
   - □ □ Wine
   - □ □ Liquor
   - □ □ Every day
   - □ □ Almost every day
   - □ □ 3-4 times a week
   - □ □ 1-2 times a week
   - □ □ 2-3 times a month
   - □ □ Once a month
   - □ □ Less than once a month

3. Overall, what was the maximum number of drinks you ever drank in one day?

4. Did you ever drink more than 5 drinks at any one time?
   - □ 1-Yes
   - □ 0-No

5. How often did this happen since the (reference date)?
   - □ □ \# of times

6. Date quit? (Do not record date of last use)
   - □ □ / □ □ / □ □
## C. MARIJUANA

1. On the days that you smoked marijuana, how much did you smoke?
   - [ ] [ ] # of joints?
   - [ ] [ ] other (describe)

2. On average, how many days in one week did you smoke this amount?
   - [ ] [ ] Every day
   - [ ] [ ] Almost every day
   - [ ] [ ] 3-4 times a week
   - [ ] [ ] 1-2 times a week
   - [ ] [ ] 2-3 times a month
   - [ ] [ ] Once a month
   - [ ] [ ] Less than once a month

3. Did you share the joints with other people?
   - [ ] 1-Yes
   - [ ] 0-No

4. How many other people did you share the joints with?

5. What was the most you ever smoked in one day?
   - [ ] [ ] # of joints?
   - [ ] [ ] other (describe)

6. Did you share the joints with other people?
   - [ ] 1-Yes
   - [ ] 0-No

7. How many other people did you share the joints with?

## D. HASHISH

1. Specify

2. On the days that you used, how much did you use?
   - [ ] [ ] grams
   - [ ] [ ] other (describe)

3. On average, how many days in one week did you use this amount?
   - [ ] [ ] Every day
   - [ ] [ ] Almost every day
   - [ ] [ ] 3-4 times a week
   - [ ] [ ] 1-2 times a week
   - [ ] [ ] 2-3 times a month
   - [ ] [ ] Once a month
   - [ ] [ ] Less than once a month

4. What was the most you ever used in one day?
   - [ ] [ ] grams
   - [ ] [ ] other (describe)

5. Date quit? (Do not record date of last use)
   - [ ] [ ] / [ ] [ ] / [ ] [ ]
### NZ Substance Use Inventory - 12/24/36/54/66 (SUI-12/24/36/54/66)

(continued)

<table>
<thead>
<tr>
<th>ID #:</th>
<th>Name/Code:</th>
<th>Date of Assessment</th>
<th>(MM/DD/YYYY)</th>
<th>Site:</th>
</tr>
</thead>
</table>

#### E. METHAMPHETAMINE

1. On the days that you used, how much did you use?
   - [ ] grams
   - [ ] # of pills
   - [ ] balls
   - [ ] bowls
   - [ ] lines
   - [ ] rocks
   - [ ] other (describe)

2. On average, how many days in one week did you use this amount?
   - Every day [ ]
   - Almost every day [ ]
   - 3-4 times a week [ ]
   - 1-2 times a week [ ]
   - 2-3 times a month [ ]
   - Once a month [ ]
   - Less than once a month [ ]

3. How much did it cost?
   - [ ] per day
   - [ ] per week
   - [ ] per month
   - [ ] other (describe)

4. What was the most you ever used in one day?
   - [ ] grams
   - [ ] # of pills
   - [ ] balls
   - [ ] bowls
   - [ ] lines
   - [ ] rocks
   - [ ] other (describe)

5. How old were you when you started using?

6. How do you use Meth? (select all that apply)
   - Sniff/Snort [ ]
   - Ingest [ ]
   - Smoke [ ]
   - Inject/IV [ ]

7. Date quit? (Do not record date of last use)
   - [ ] / [ ] / [ ]

UNKOWN [ ]
### NZ Substance Use Inventory - 12/24/36/54/66 (SUI-12/24/36/54/66) (continued)

<table>
<thead>
<tr>
<th>ID #</th>
<th>Name Code</th>
<th>Date of Assessment</th>
<th>(MM/DD/YYYY) Site</th>
</tr>
</thead>
</table>

**F. ECSTASY**

1. On the days that you used Ecstasy, how much did you use?
   - Doses
   - # of pills
   - Other (describe)

2. On average, how many days in one week did you use this amount?
   - Every day  ○ 1
   - Almost every day  ○ 2
   - 3-4 times a week  ○ 3
   - 1-2 times a week  ○ 4
   - 2-3 times a month  ○ 5
   - Once a month  ○ 6
   - Less than once a month  ○ 7

3. How much did it cost?
   - Per day
   - Per week
   - Per month
   - Other (describe)

4. What was the most you ever used in one day?
   - Doses
   - # of pills
   - Other (describe)

5. How old were you when you started using?

6. Date quit? (Do not record date of last use)

**UNKOWN ○ 1**
### G. AMPHETAMINES

1. Specify

2. On the days that you used amphetamines, how much did you use?
   - grams
   - # of pills
   - other (describe)

3. On average, how many days in one week did you use this amount?
   - Every day  ○ 1
   - Almost every day  ○ 2
   - 3-4 times a week  ○ 3
   - 1-2 times a week  ○ 4
   - 2-3 times a month  ○ 5
   - Once a month  ○ 6
   - Less than once a month  ○ 7

4. How much did it cost?
   - per day
   - per week
   - per month
   - other (describe)

   UNKNOWN ○

5. What was the most you ever used in one day?
   - grams
   - # of pills
   - other (describe)

6. How old were you when you started using?

7. Date quit? (Do not record date of last use)

---

Please PRINT CLEARLY

1 2 3 4 5 6 7 8 9 0
<table>
<thead>
<tr>
<th>H. BENZODIAZEPINES / TRANQUILIZERS</th>
<th>I. BARBITURATES / SEDATIVES</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Specify</td>
<td>1. Specify</td>
</tr>
<tr>
<td>2. On the days that you used, how much did you use?</td>
<td>2. On the days that you used, how much did you use?</td>
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<tr>
<td>- .    grams</td>
<td>- .    grams</td>
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<tr>
<td>- .    # of pills</td>
<td>- .    # of pills</td>
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<tr>
<td>- .    other (describe)</td>
<td>- .    other (describe)</td>
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<tr>
<td>3. On average, how many days in one week did you use this amount?</td>
<td>3. On average, how many days in one week did you use this amount?</td>
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<tr>
<td>Every day</td>
<td>Every day</td>
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<td>- 1</td>
<td>- 1</td>
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<td>Almost every day</td>
<td>Almost every day</td>
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<tr>
<td>- 2</td>
<td>- 2</td>
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<td>3-4 times a week</td>
<td>3-4 times a week</td>
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<td>- 3</td>
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<td>1-2 times a week</td>
<td>1-2 times a week</td>
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<td>- 4</td>
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<td>2-3 times a month</td>
<td>2-3 times a month</td>
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<td>- 5</td>
<td>- 5</td>
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<tr>
<td>Once a month</td>
<td>Once a month</td>
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<tr>
<td>- 6</td>
<td>- 6</td>
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<tr>
<td>Less than once a month</td>
<td>Less than once a month</td>
</tr>
<tr>
<td>4. What was the most you ever used in one day?</td>
<td>4. What was the most you ever used in one day?</td>
</tr>
<tr>
<td>- .    grams</td>
<td>- .    grams</td>
</tr>
<tr>
<td>- .    # of pills</td>
<td>- .    # of pills</td>
</tr>
<tr>
<td>- .    other (describe)</td>
<td>- .    other (describe)</td>
</tr>
<tr>
<td>5. Date quit? (Do not record date of last use)</td>
<td>5. Date quit? (Do not record date of last use)</td>
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<tr>
<td>-     /    /</td>
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</tr>
</tbody>
</table>

Please PRINT CLEARLY
### J. COCAINE/Crack

1. Specify

2. On the days that you used, how much did you use?
   - ,  grams
   - ,   other (describe)

3. On average, how many days in one week did you use this amount?
   - Every day 1
   - Almost every day 2
   - 3-4 times a week 3
   - 1-2 times a week 4
   - 2-3 times a month 5
   - Once a month 6
   - Less than once a month 7

4. What was the most you ever used in one day?
   - ,  grams
   - ,   other (describe)

5. Date quit? (Do not record date of last use)
   - / / 

### K. HEROIN

1. Specify

2. On the days that you used, how much did you use?
   - ,  grams
   - ,   other (describe)

3. On average, how many days in one week did you use this amount?
   - Every day 1
   - Almost every day 2
   - 3-4 times a week 3
   - 1-2 times a week 4
   - 2-3 times a month 5
   - Once a month 6
   - Less than once a month 7

4. What was the most you ever used in one day?
   - ,  grams
   - ,   other (describe)

5. Date quit? (Do not record date of last use)
   - / / 

Draft
### L. METHADONE

1. Specify

2. On the days that you used, how much did you use?
   - grams
   - other (describe)

3. On average, how many days in one week did you use this amount?
   - Every day
   - Almost every day
   - 3-4 times a week
   - 1-2 times a week
   - 2-3 times a month
   - Once a month
   - Less than once a month

4. What was the most you ever used in one day?
   - grams
   - other (describe)

5. Date quit? (Do not record date of last use)
   - / / / 

### M. "HOMEBAKE"

1. Specify

2. On the days that you used, how much did you use?
   - grams
   - other (describe)

3. On average, how many days in one week did you use this amount?
   - Every day
   - Almost every day
   - 3-4 times a week
   - 1-2 times a week
   - 2-3 times a month
   - Once a month
   - Less than once a month

4. What was the most you ever used in one day?
   - grams
   - other (describe)

5. Date quit? (Do not record date of last use)
   - / / / 

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_Draft_
N. MSTI

1. Specify

2. On the days that you used, how much did you use?
   • grams
   • other (describe)

3. On average, how many days in one week did you use this amount?
   - Every day
   - Almost every day
   - 3-4 times a week
   - 1-2 times a week
   - 2-3 times a month
   - Once a month
   - Less than once a month

4. What was the most you ever used in one day?
   • grams
   • other (describe)

5. Date quit? (Do not record date of last use)
   / / /}

O. OXYCONTIN

1. On the days that you used, how much did you use?
   • grams
   • # of pills
   • other (describe)

2. On average, how many days in one week did you use this amount?
   - Every day
   - Almost every day
   - 3-4 times a week
   - 1-2 times a week
   - 2-3 times a month
   - Once a month
   - Less than once a month

3. What was the most you ever used in one day?
   • grams
   • # of pills
   • other (describe)

4. Date quit? (Do not record date of last use)
   / / /
NZ Substance Use Inventory - 12/24/36/54/66 (SUI-12/24/36/54/66)
(continued)

P. OTHER OPIATES

1. Specify
   
2. On the days that you used, how much did you use?
   - [ ] grams
   - [ ] # of pills
   - [ ] other (describe)

3. On average, how many days in one week did you use this amount?
   - Every day  [ ]
   - Almost every day  [ ]
   - 3-4 times a week  [ ]
   - 1-2 times a week  [ ]
   - 2-3 times a month  [ ]
   - Once a month  [ ]
   - Less than once a month  [ ]

4. What was the most you ever used in one day?
   - [ ] grams
   - [ ] # of pills
   - [ ] other (describe)

5. Date quit? (Do not record date of last use)
   [ ] / [ ] / [ ]

Q. INHALANTS

1. Specify

2. On the days that you used, how much did you use?
   - [ ] # of hits
   - [ ] other (describe)

3. On average, how many days in one week did you use this amount?
   - Every day  [ ]
   - Almost every day  [ ]
   - 3-4 times a week  [ ]
   - 1-2 times a week  [ ]
   - 2-3 times a month  [ ]
   - Once a month  [ ]
   - Less than once a month  [ ]

4. What was the most you ever used in one day?
   - [ ] # of hits
   - [ ] other (describe)

5. Date quit? (Do not record date of last use)
   [ ] / [ ] / [ ]
### NZ Substance Use Inventory - 12/24/36/54/66 (SUI-12/24/36/54/66) (continued)

<table>
<thead>
<tr>
<th>ID #:</th>
<th>Name Code:</th>
<th>Date of Assessment:</th>
<th>(MM/DD/YYYY) Site:</th>
</tr>
</thead>
</table>

#### R. LSD/HALLUCINOGENS

1. Specify

2. On the days that you used, how much did you use?
   - [ ] [ ] # of pills
   - [ ] [ ] grams
   - [ ] [ ] other (describe)

3. On average, how many days in one week did you use this amount?
   - [ ] [ ] Every day
   - [ ] [ ] Almost every day
   - [ ] [ ] 3-4 times a week
   - [ ] [ ] 1-2 times a week
   - [ ] [ ] 2-3 times a month
   - [ ] [ ] Once a month
   - [ ] [ ] Less than once a month

4. What was the most you ever used in one day?
   - [ ] [ ] # of pills
   - [ ] [ ] grams
   - [ ] [ ] other (describe)

5. Date quit? (Do not record date of last use)
   - [ ] [ ] / [ ] [ ] / [ ] [ ]

#### S. PCP/ANGEL DUST

1. Specify

2. On the days that you used, how much did you use?
   - [ ] [ ] grams
   - [ ] [ ] other (describe)

3. On average, how many days in one week did you use this amount?
   - [ ] [ ] Every day
   - [ ] [ ] Almost every day
   - [ ] [ ] 3-4 times a week
   - [ ] [ ] 1-2 times a week
   - [ ] [ ] 2-3 times a month
   - [ ] [ ] Once a month
   - [ ] [ ] Less than once a month

4. What was the most you ever used in one day?
   - [ ] [ ] grams
   - [ ] [ ] other (describe)

5. Date quit? (Do not record date of last use)
   - [ ] [ ] / [ ] [ ] / [ ] [ ]
T. OTHER MEDICATIONS OR SUBSTANCES

1. Specify
   
2. On the days that you used, how much did you use?
   - [ ] grams
   - [ ] other (describe)
   
3. On average, how many days in one week did you use this amount?
   - Every day [ ]
   - Almost every day [ ]
   - 3-4 times a week [ ]
   - 1-2 times a week [ ]
   - 2-3 times a month [ ]
   - Once a month [ ]
   - Less than once a month [ ]

4. What was the most you ever used in one day?
   - [ ] grams
   - [ ] other (describe)
   
5. Date quit? (Do not record date of last use)
   
Date form completed: [ ] / [ ] / [ ]
Form completed by: (evaluator #) [ ]
NZ Substance Use Inventory - 12/24/36/54/66 (SUI-12/24/36/54/66)  
(continued)

If this is the 36-Month follow-up interview, please continue on to the next section, Personal Safety. If this is either the 12 or 24-Month follow-up, please leave the Personal Safety section BLANK, fill in the form completion date and evaluator number on the final page, and fax all pages to the DMC.

PART III. PERSONAL SAFETY

A. PERSONAL SAFETY

1. Since the reference date, have you been mentally or emotionally abused or mistreated?  
   - 1-Yes   0-No
   
   a. If yes, do you want help with this problem?  
     - 1-Yes   0-No

   We can talk about this at the end of the session.

2. Have you been physically abused since the reference date?  
   - 1-Yes   0-No

   If yes,
   
   a. Who is the person who did this to you? Tell me their relationship to you (mark all that apply)
      - 1-Father of child   0-Parent   0-Friend/Acquaintance
      - 1-Partner   0-Sibling or other family member   0-Don't know

   b. How many times did this happen since the reference date?  
      - 1-Daily   2-About once a week   3-About once a month   4-Once

   c. On what part of your body were you hurt? (mark all that apply)
      - 1-Head   0-Arms   0-Back
      - 1-Neck   0-Legs   0-Other (specify below)
      - 1-Chest   0-Stomach/Abdomen

   d. Were you hospitalized overnight for any of these incidents?  
      - 1-Yes   0-No

   e. Did you seek help?  
      - 1-Yes   0-No

   1) If yes, from which sources (mark all that apply)?  
      - 1-Family/Friends   0-Medical treatment   0-Shelter
      - 1-Police   0-Mental Health treatment   0-Other (specify below)

   f. Do you want help with this problem?  
      - 1-Yes   0-No

   We can talk about this at the end of the session.
3. Since the reference date, have you been sexually abused?
   ○ 1-Yes ○ 0-No
   
   a. Who is the person who did this to you? Tell me their relationship to you. (mark all that apply)
      ○ 1-Father of child ○ 1-Parent ○ 1-Friend/Acquaintance
      ○ 1-Other partner ○ 1-Sibling or other family member ○ 1-Don't know
   
   b. How many times did this happen since the reference date?
      ○ 1-Daily ○ 2-About once a week ○ 3-About once a month ○ 4-Once
   
   c. Were you hospitalized overnight for any of these incidents?
      ○ 1-Yes ○ 0-No
   
   d. Did you seek help?
      ○ 1-Yes ○ 0-No
      
      1) If yes, from which sources (mark all that apply)?
         ○ 1-Family/Friends ○ 1-Medical treatment ○ 1-Shelter
         ○ 1-Police ○ 1-Mental Health treatment ○ 1-Other (specify below)
   
   e. Do you want help with this problem?
      ○ 1-Yes ○ 0-No

   We can talk about this at the end of the session.

4. Are you currently receiving help for any form of abuse (emotional, physical, sexual)?
   ○ 1-Yes ○ 0-No
   
   a. If yes, from which sources (mark all that apply)?
      ○ 1-Family/Friends ○ 1-Medical treatment ○ 1-Shelter
      ○ 1-Police ○ 1-Mental Health treatment ○ 1-Other (specify below)

5. Are you currently in any drug treatment program?
   ○ 1-Yes ○ 0-No

   We can discuss referral for treatment at the end of the session.

   Date form completed:  
   / /  
   
   Form completed by: (evaluator #)  
   
   175
Appendix G. Brief Symptom Inventory

Form 405—NZ Brief Symptom Inventory (BSI®) -12/36

Opening script: In this questionnaire, I will read to you a list of problems and complaints that people sometimes have. I am going to have you select a response from the response card where the possible answers are numbered 0 to 4. "0" means "Not at all", "1" means "A little bit", "2" means "Moderately", "3" means "Quite a bit", "4" means "Extremely". Choose the number on the response card that best describes how much of a problem this has been for you during the past 7 days, including today.

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<td>Name Code:</td>
<td>Visit #:</td>
<td>Date of Assessment:</td>
<td>(MM/DD/YYYY)</td>
<td>Evaluator #:</td>
</tr>
</tbody>
</table>

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<thead>
<tr>
<th></th>
<th>Not at all</th>
<th>A little bit</th>
<th>Moderately</th>
<th>Quite a bit</th>
<th>Extremely</th>
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<tbody>
<tr>
<td>1. Nervousness or shakiness inside</td>
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<td>2. Faintness or dizziness</td>
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<td>3. The idea that someone else can control your thoughts</td>
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<td>4. Feeling others are to blame for most of your troubles</td>
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<td>5. Trouble remembering things</td>
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<td>6. Feeling easily annoyed or irritated</td>
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<td>7. Pains in heart or chest</td>
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<td>8. Feeling afraid in open spaces or on the streets</td>
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<td>9. Thoughts of ending your life</td>
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<td>10. Feeling that most people cannot be trusted</td>
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<td>11. Poor appetite</td>
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<td>12. Suddenly scared for no reason</td>
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<td>13. Temper outbursts that you could not control</td>
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<td>14. Feeling lonely even when you are with people</td>
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<td>15. Feeling blocked in getting things done</td>
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<td>16. Feeling lonely</td>
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<td>17. Feeling blue</td>
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<td>18. Feeling no interest in things</td>
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<td>19. Feeling fearful</td>
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<td>20. Your feelings being easily hurt</td>
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<td>21. Feeling that people are unfriendly or dislike you</td>
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<table>
<thead>
<tr>
<th>ID #</th>
<th>Name Code</th>
<th>Date of Assessment (MM/DD/YYYY)</th>
<th>Site</th>
<th>Not at all</th>
<th>A little bit</th>
<th>Moderately</th>
<th>Quite a bit</th>
<th>Extremely</th>
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<td>22.</td>
<td>Feeling inferior to others</td>
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<td>23.</td>
<td>Nausea or upset stomach</td>
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<td>24.</td>
<td>Feeling that you are watched or talked about by others</td>
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<td>25.</td>
<td>Trouble falling asleep</td>
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<td>26.</td>
<td>Having to check and double-check what you do</td>
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<td>27.</td>
<td>Difficulty making decisions</td>
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<td>28.</td>
<td>Feeling afraid to travel on buses, subways, or trains</td>
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<td>29.</td>
<td>Trouble getting your breath</td>
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<td>30.</td>
<td>Hot or cold spells</td>
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<td>31.</td>
<td>Having to avoid certain things, places or activities because they frighten you</td>
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<td>32.</td>
<td>Your mind going blank</td>
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<td>33.</td>
<td>Numbness or tingling in parts of your body</td>
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<td>34.</td>
<td>The idea that you should be punished for your sins</td>
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<td>35.</td>
<td>Feeling hopeless about the future</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>36.</td>
<td>Trouble concentrating</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>37.</td>
<td>Feeling weak in parts of your body</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>38.</td>
<td>Feeling tense or keyed up</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>39.</td>
<td>Thoughts of death or dying</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>40.</td>
<td>Having urges to beat, injure, or harm someone</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>41.</td>
<td>Having urges to break or smash things</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>42.</td>
<td>Feeling very self-conscious with others</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ID #</td>
<td>Name Code</td>
<td>Date of Assessment (MM/DD/YYYY)</td>
<td>Site</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th></th>
<th>Not at all</th>
<th>A little bit</th>
<th>Moderately</th>
<th>Quite a bit</th>
<th>Extremely</th>
</tr>
</thead>
<tbody>
<tr>
<td>43. Feeling uneasy in crowds, such as shopping or at a movie</td>
<td>○</td>
<td>○</td>
<td>○</td>
<td>○</td>
<td>○</td>
</tr>
<tr>
<td>44. Never feeling close to another person</td>
<td>○</td>
<td>○</td>
<td>○</td>
<td>○</td>
<td>○</td>
</tr>
<tr>
<td>45. Spells of terror or panic</td>
<td>○</td>
<td>○</td>
<td>○</td>
<td>○</td>
<td>○</td>
</tr>
<tr>
<td>46. Getting into frequent arguments</td>
<td>○</td>
<td>○</td>
<td>○</td>
<td>○</td>
<td>○</td>
</tr>
<tr>
<td>47. Feeling nervous when you are left alone</td>
<td>○</td>
<td>○</td>
<td>○</td>
<td>○</td>
<td>○</td>
</tr>
<tr>
<td>48. Others not giving you proper credit for your achievements</td>
<td>○</td>
<td>○</td>
<td>○</td>
<td>○</td>
<td>○</td>
</tr>
<tr>
<td>49. Feeling so restless you couldn’t sit still</td>
<td>○</td>
<td>○</td>
<td>○</td>
<td>○</td>
<td>○</td>
</tr>
<tr>
<td>50. Feelings of worthlessness</td>
<td>○</td>
<td>○</td>
<td>○</td>
<td>○</td>
<td>○</td>
</tr>
<tr>
<td>51. Feeling that people will take advantage of you if you let them</td>
<td>○</td>
<td>○</td>
<td>○</td>
<td>○</td>
<td>○</td>
</tr>
<tr>
<td>52. Feelings of guilt</td>
<td>○</td>
<td>○</td>
<td>○</td>
<td>○</td>
<td>○</td>
</tr>
<tr>
<td>53. The idea that something is wrong with your mind</td>
<td>○</td>
<td>○</td>
<td>○</td>
<td>○</td>
<td>○</td>
</tr>
</tbody>
</table>

Date Form Completed: [ ] / [ ] / [ ]
Form Completed By: (Evaluator #) [ ]
### NZ Strengths and Difficulties Questionnaire

For each item, please mark the box for NOT TRUE, SOMETHING TRUE or CERTAINLY TRUE. It would help us if you answered all items as best as you can even if you are not absolutely certain. Please give your answers on the basis of your child’s behaviour over the last six months.

<table>
<thead>
<tr>
<th></th>
<th>Not True</th>
<th>Somewhat True</th>
<th>Certainly True</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Considerate of other people’s feelings</td>
<td>☐</td>
<td>☐</td>
</tr>
<tr>
<td>2.</td>
<td>Restless, overactive, cannot stay still for long</td>
<td>☐</td>
<td>☒</td>
</tr>
<tr>
<td>3.</td>
<td>Often complains of headaches, stomach-aches or sickness</td>
<td>☐</td>
<td>☒</td>
</tr>
<tr>
<td>4.</td>
<td>Shares readily with other children, for example toys, treats, pencils</td>
<td>☐</td>
<td>☐</td>
</tr>
<tr>
<td>5.</td>
<td>Often loses temper</td>
<td>☐</td>
<td>☒</td>
</tr>
<tr>
<td>6.</td>
<td>Rather solitary, prefers to play alone</td>
<td>☐</td>
<td>☒</td>
</tr>
<tr>
<td>7.</td>
<td>Generally well behaved, usually does what adults request</td>
<td>☐</td>
<td>☒</td>
</tr>
<tr>
<td>8.</td>
<td>Many worries or often seems worried</td>
<td>☐</td>
<td>☒</td>
</tr>
<tr>
<td>9.</td>
<td>Helpful if someone is hurt, upset or feeling ill</td>
<td>☐</td>
<td>☒</td>
</tr>
<tr>
<td>10.</td>
<td>Constantly fidgeting or squirming</td>
<td>☐</td>
<td>☒</td>
</tr>
<tr>
<td>11.</td>
<td>Has at least one good friend</td>
<td>☐</td>
<td>☒</td>
</tr>
<tr>
<td>12.</td>
<td>Often fights with other children or bullies them</td>
<td>☐</td>
<td>☒</td>
</tr>
<tr>
<td>13.</td>
<td>Often unhappy, depressed or tearful</td>
<td>☐</td>
<td>☒</td>
</tr>
<tr>
<td>14.</td>
<td>Generally liked by other children</td>
<td>☐</td>
<td>☒</td>
</tr>
<tr>
<td>15.</td>
<td>Easily distracted, concentration wanders</td>
<td>☐</td>
<td>☒</td>
</tr>
<tr>
<td>16.</td>
<td>Nervous or clingy in new situations, easily loses confidence</td>
<td>☐</td>
<td>☒</td>
</tr>
<tr>
<td>17.</td>
<td>Kind to younger children</td>
<td>☐</td>
<td>☒</td>
</tr>
<tr>
<td>18.</td>
<td>Often lies or cheats</td>
<td>☐</td>
<td>☒</td>
</tr>
<tr>
<td>19.</td>
<td>Picked on or bullied by other children</td>
<td>☐</td>
<td>☒</td>
</tr>
<tr>
<td>20.</td>
<td>Often volunteers to help others (parents, teachers, other children)</td>
<td>☐</td>
<td>☒</td>
</tr>
<tr>
<td>21.</td>
<td>Thinks things out before acting</td>
<td>☐</td>
<td>☒</td>
</tr>
<tr>
<td>22.</td>
<td>Steals from home, school or elsewhere</td>
<td>☐</td>
<td>☒</td>
</tr>
<tr>
<td>23.</td>
<td>Gets along better with adults than with other children</td>
<td>☐</td>
<td>☒</td>
</tr>
<tr>
<td>24.</td>
<td>Many fears, easily scared</td>
<td>☐</td>
<td>☒</td>
</tr>
<tr>
<td>25.</td>
<td>Good attention span, sees chores or homework through the end</td>
<td>☐</td>
<td>☒</td>
</tr>
</tbody>
</table>

Do you have any other comments or concerns?
NZ Strengths and Difficulties Questionnaire

B. Overall, do you think that your child has difficulties in one or more of the following areas:

Emotions, concentration, behaviour or being able to get on with other people?

<table>
<thead>
<tr>
<th></th>
<th>No</th>
<th>Yes – minor difficulties</th>
<th>Yes – definite difficulties</th>
<th>Yes – severe difficulties</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
</tbody>
</table>

If you have answered “Yes”, please answer the following questions about these difficulties:

1. How long have these difficulties been present?

<table>
<thead>
<tr>
<th></th>
<th>Less than a month</th>
<th>1-5 months</th>
<th>6-12 months</th>
<th>Over a year</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th></th>
<th>Not at all</th>
<th>Only a little</th>
<th>Quite a lot</th>
<th>A great deal</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
</tbody>
</table>

Do the difficulties upset or distress your child?

<table>
<thead>
<tr>
<th></th>
<th>HOME LIFE</th>
<th>FRIENDSHIPS</th>
<th>CLASSROOM LEARNING</th>
<th>LEISURE ACTIVITIES</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

2. Do the difficulties interfere with your child’s everyday life in the following areas?

<table>
<thead>
<tr>
<th></th>
<th>No</th>
<th>Yes – minor difficulties</th>
<th>Yes – definite difficulties</th>
<th>Yes – severe difficulties</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
</tbody>
</table>

3. Do the difficulties put a burden on you or the family as a whole?

<table>
<thead>
<tr>
<th></th>
<th>Not at all</th>
<th>Only a little</th>
<th>Quite a lot</th>
<th>A great deal</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
</tbody>
</table>

Date form completed: / / From completed by: (evaluator #)
NZ SDQ Summary Sheet

A. RECORD OF SYMPTOM SCORES

<table>
<thead>
<tr>
<th>Raw scores</th>
<th>Total scores</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Emotional Symptom</td>
<td></td>
</tr>
<tr>
<td>2. Conduct Problems</td>
<td></td>
</tr>
<tr>
<td>3. Hyperactivity</td>
<td></td>
</tr>
<tr>
<td>4. Peer Problems</td>
<td></td>
</tr>
<tr>
<td>5. Prosocial</td>
<td></td>
</tr>
</tbody>
</table>

6. Total Difficulties Score

B. RECORD OF IMPACT SCORES

1. Does the parent think that his/her child has difficulties in one or more of the following areas: Emotions, concentration, behaviour or being able to get on with other people?
   - 1:Yes
   - 0:No  (skip the following scoring and date the form of completion)

   a. Difficulties upset or distress child

   b. Interfere with HOME LIFE

   c. Interfere with FRIENDSHIPS

   d. Interfere with CLASSROOM LEARNING

   e. Interfere with LEISURE ACTIVITIES

6. Total Impact Score

Data form completed: / / From completed by: (evaluator #)
Appendix I. Gift Delay (Wrap) Scoring Sheet

GIFT WRAP TASK

ID #: ___________ Name Code: ___________ Visit: ___________ Date of Assessment: ___________ Evaluator #: ___________

Trial Error
- Examiner error during trial makes trial unscorable: ☐ (97)
- Child refused to do whole task – Behaviour problem: ☐ (98)
- Child didn’t understand task – Language: ☐ (99)

Introduction to Task

Now I have a present for you to say thank you for all the games you’ve been playing with us today. You’ve worked so hard and done really well.

Oops! I forgot to wrap it up. I’m going to wrap it now, so let’s turn your chair around so you can’t see me do it, as I don’t want to spoil the surprise. *Now, don’t look until I say you can.*

Task

Child sits in chair with his/her back to the table. Assessor PRETENDS to wrap the gift noisily over 60 seconds (scrunch paper to emulate wrapping). Once wrapping has begun, no prompts or reminders are provided for the child. If he/she turns around do not remind or instruct the child to stop peaking or to go back to seat.

*Timing of the child’s resistance to peaking begins once assessor has finished speaking the final part of the introduction to task (see text underline and in bold above).

NOTE: WRAP PRESENT PRIOR TO EXECUTIVE FUNCTION TASK SO THAT YOU ARE FREE TO TIME PEEKS. YOU CAN SCRUNCH A PIECE OF COPY PAPER TO EMULATE SOUND OF WRAPPING.

<table>
<thead>
<tr>
<th>PEAKS</th>
<th>DURATION OF PEAKS IN SECONDS</th>
</tr>
</thead>
<tbody>
<tr>
<td>LATENCY TO FIRST Peek 1 ☐</td>
<td>= ___________ SECONDS</td>
</tr>
<tr>
<td>Peek 2 ☐</td>
<td></td>
</tr>
<tr>
<td>Peek 3 ☐</td>
<td></td>
</tr>
<tr>
<td>Peek 4 ☐</td>
<td></td>
</tr>
<tr>
<td>Peek 5 ☐</td>
<td></td>
</tr>
<tr>
<td>Peek 6 ☐</td>
<td></td>
</tr>
<tr>
<td>Peek 7 ☐</td>
<td></td>
</tr>
<tr>
<td>Peek 8 ☐</td>
<td></td>
</tr>
<tr>
<td>Peek 9 ☐</td>
<td></td>
</tr>
<tr>
<td>Peek 10 ☐</td>
<td></td>
</tr>
<tr>
<td>Peek 11 ☐</td>
<td></td>
</tr>
<tr>
<td>Peek 12 ☐</td>
<td></td>
</tr>
</tbody>
</table>

| LOWER SCORE OPTIMAL TOTAL NUMBER OF PEAKS FOR 60 SECONDS |
| LOWER SCORE OPTIMAL TOTAL SECONDS PEEKING |
| LOWER SCORE OPTIMAL OVERALL PEAK RESISTANCE SCORE |

2 = Mostly full torso turns in order to peek, getting out of chair to come and look, standing on chair to look over assessor etc.
1 = Peeking occurred but it was mostly only over the shoulder head turns and quite subtle movements.
0 = No peaking occurred at all.

Date form completed: ___________ / ___________ / ___________

From completed by: (evaluator #: ___________)

y