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**Continuity of Aspects of Sustained Attention and Impulse Control
During Development in Children Prenatally Exposed to
Cigarettes and Marihuana**

Christy Lynn Chesley

**Thesis presented to the Faculty of Graduate Studies and Research
in partial fulfillment of the requirements for the Degree of Master of Arts, Department of
Psychology, Carleton University, Ottawa, Canada.**

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**"Continuity of Aspects of Sustained Attention
and Impulse Control During Development
in Children Prenatally Exposed to
Cigarettes and Marihuana"**

submitted by

Christy Lynn Chesley

**in Partial fulfillment of the requirements
for the degree of Master of Arts**

Chair

Thesis Supervisor Carleton University

May 2001

Abstract

Aspects of attentional functioning were examined longitudinally in a low-risk sample comprised of children prenatally exposed to cigarettes and marihuana and non-exposed controls. Participants (N=81) were repeatedly assessed at 6, 10-11 and 13-15 years of age using two versions of a continuous performance task (CPT) to evaluate sustained attention and impulse control. Moderate to high stability of performance with respect to CPT-derived measures of sustained attention and impulse control was observed over time for the majority of children in the sample. Differential effects on attentional behavior were observed for cigarettes and marihuana across the three age assessments. Cigarettes were negatively associated with impulse control at age 6 and sustained attention at age 10-11, while marihuana was negatively associated with sustained attention at age 6. These findings suggest that the use of cigarettes and marihuana during pregnancy have long-term consequences for attentional functioning in children born to maternal drug users.

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Continuity of Aspects of Sustained Attention and Impulse Control

During Development in Children Prenatally Exposed to

Cigarettes and Marihuana

Over the past several decades, government agencies and health care professionals have been working to raise awareness regarding the serious consequences of drug use during pregnancy among women who are of child-bearing age. Despite the widespread availability of information, and in the face of a growing body of evidence that drugs taken during pregnancy have measurable harmful effects on the fetus, many women continue to use drugs throughout the course of pregnancy. Cigarettes and marihuana are the two most common non-medicinal drugs taken during pregnancy (Fried, 1993). The current study explores the effects of prenatal exposure to cigarettes and marihuana on aspects of attentional functioning in the offspring of maternal drug users.

In recent years, both cigarettes and marihuana have been investigated in a number of research studies looking at the short and long-term development of cognitive and behavioural functioning in children prenatally exposed to drugs during pregnancy (for reviews, see Olds, 1997; Walker, Rosenberg & Balaban-Gil, 1999). Researchers have looked at a number of outcomes including cognitive development (Fried, Watkinson & Gray, 1998; Day et al., 1994), growth measures (Fried, Watkinson & Gray, 1999) and behaviour problems (Goldschmidt, Day & Richardson, 2000) in the offspring of maternal drug users. Both drugs have been linked to impairments in behavioural regulation in young children born to drug users, with many of these children exhibiting problems with

sustained attention and impulse control (Fried et al., 1992, 1998; Leech, Richardson, Goldschmidt & Day, 1999).

The current study is part of an ongoing longitudinal investigation, the Ottawa Prenatal Prospective Study (OPPS), comprised of a low-risk, predominantly white, middle-class sample of children followed since birth. Since 1978, the OPPS has been examining the consequences of prenatal exposure to a number of drugs commonly used during pregnancy on various aspects of development in the offspring of maternal drug users. The present study will focus on the domain of attention in which the long-term continuity of findings from the OPPS suggest a link between prenatal drug exposure and problems with sustained attention and impulse control in early childhood. The goal of this study is to examine the extent to which early observations of attention deficits in young children exposed prenatally to cigarettes and marijuana translate into continued attentional problems in older children and adolescents.

Sustained attention is defined as the ability to maintain attentional focus over time, and is often assessed in terms of performance on tasks requiring an individual to respond to infrequent events occurring at random intervals over a prolonged period of time (Corkum & Siegel, 1993). Impulse control refers to the ability to refrain from responding inappropriately to an event (Levy, 1980), and is frequently measured using tasks that require an individual to delay or withhold responding (Barkley, 1997). Olson (1989) suggests that two subtypes of impulse control may be present in children by age six. The first subtype is believed to underlie the ability to correctly inhibit responses in structured task situations, and the second subtype underlies the ability to delay

responding in order to gain rewards and comply with social expectations of correct behaviour (i.e., delay of gratification). The present study looks only at the first subtype of impulse control, and assesses children's ability to inhibit responding on a structured laboratory task.

One measurement approach, the use of a continuous performance task (CPT), has been regularly employed to assess both sustained attention and impulse control. The instruments used in the present study (Conners CPT; Gordon Diagnostic Systems) are computerized versions of the CPT that provide among their measures, omission errors (misses), believed to result from problems with sustained attention, commission errors ("false alarms"), considered to reflect deficits in impulse control (Corkum & Siegel, 1993) and measures of changes in performance over time.

Prevalence of Cigarette and Marihuana Use in North America

Results of a recent survey from the U.S. Department of Health and Human Services (2000) estimate that 57.0 million Americans over the age of 12 are current cigarette smokers (corresponding to 25.8% of the population). These figures are generally consistent with the prevalence of smoking in Canada, where an estimated 28.6% of Canadians over the age of 15 reported smoking cigarettes in 1996-97 (Canadian Centre on Substance Abuse, 1999).

An estimated 22 million (22%) American women over the age of 18 are cigarette smokers (Centers for Disease Control and Prevention, 1998). Among American women of child-bearing age, prevalence rates are even higher: 27% of women of reproductive age reported smoking cigarettes in 1992, down from 29% in 1987 (Centers for Disease

Control and Prevention, 1994). Prevalence rates for women of reproductive age in Canada for the same time period are roughly equal to those of their U.S. counterparts, with one third of women reporting regular use of cigarettes (Fried, 1993). The National Institute on Drug Abuse (NIDA) reports that 20% or more of pregnant women continue to smoke during pregnancy (National Institute on Drug Abuse, 1998).

Recently, the National Center for Health Statistics (1998) reported a decline in maternal smoking during pregnancy across all ages (15-49 years) and racial groups from 19.5% in 1989 to 13.6% in 1996. Similar rates of decline were observed among younger women of prime child-bearing age (18-24 years), however, smoking rates for this group were higher by approximately 4% in all years (23.7% in 1989 to 17.5% in 1996) compared to the smoking rates of women aged 25-29 years (19.0% in 1989 to 12.3% in 1996), and by at least 6.5% compared to the smoking rates of women aged 30-49 years (14.2% in 1989 to 10.9% in 1996). This decline can be viewed as a positive indication of a reduction in prevalence rates with respect to prenatal exposure to cigarettes, however it is not yet clear if this decline marks the beginning of a permanent decline in maternal smoking or just a transient reduction in prevalence rates.

A recent Canadian survey monitoring the use of illicit drugs including marihuana reports that 7.0% of women 18 to 65+ years of age living in Ontario used marihuana in 1994 (Addiction Research Foundation, 1994). The percentage of female users of the drug was not substantially different from rates reported in 1987 (6.8%), 1989 (8.2%), and 1991 (6.0%) (Addiction Research Foundation, 1994). Reporting on marihuana users of child-bearing age, 24.1% of female Ontario high school students used marihuana during

the year prior to the survey (Addiction Research Foundation, 1997). These figures are similar to those reported by female Ontario university students (21.7%) surveyed in 1993. Reports estimate that 2.9% of women use marihuana during pregnancy (Fried et al., 1998).

As the prevalence figures indicate, the use of cigarettes and marihuana in North America is not an isolated phenomenon affecting a small segment of the population. The widespread use of these substances among young women of reproductive age has considerable implications in terms of the number of pregnancies potentially affected by prenatal drug exposure. In addition to cases of exposure where a woman knowingly uses drugs during pregnancy, fetal exposure to cigarettes and marihuana can occur inadvertently before a women is aware that she is pregnant, particularly during the first trimester of pregnancy. In North America, at least half of all pregnancies are unplanned (Walker et al., 1999). Given the high prevalence rates of cigarette and marihuana use among women of child-bearing age, the potential for prenatal drug exposure, inadvertent or otherwise, is significant.

As stated previously, several studies have reported connections between maternal smoking and marihuana use during pregnancy and attention deficits in young children (Fried et al., 1992, 1998; Leech et al., 1999). Attention is an aspect of higher cognitive functioning which depends on intact central nervous system (CNS) functioning for optimal efficiency (Mirsky, Fantie & Tatman, 1995). Any disruption to the CNS can therefore interfere with normal operation of attention processes (Mirsky et al., 1995). In order to establish prenatal exposure to cigarettes or marihuana as a possible cause of

disruption to complex cognitive processes like attention, it is important to understand how the drugs, at least in theory, could be expected to exert their effects.

Behavioural teratology is an area of developmental psychology concerned with studying the postnatal behavioural effects associated with prenatal exposure to potentially harmful agents, including drugs, known as *teratogens* (Bee, 1994; Vorhees, 1986).

Teratogens are agents that are capable of adversely affecting development, including, but not limited to, CNS development (Bee, 1994; Vorhees, 1986, Wilson, 1977). The degree of harm to an organism exposed prenatally to a teratogenic substance can be viewed as lying along a dose-dependent continuum from no impairment to death (Wilson, 1977).

The expected differential impact of a teratogen on development is theorized to follow a dose-response curve, with functional (behavioural) teratogenic effects observed at lower doses, followed, as dosage increases, by growth abnormalities, malformations, and finally, at lethal doses, death of the organism (Vorhees, 1986). This model assumes that behavioural indices will be a more sensitive measure of the embryotoxicity of a given agent than growth or malformation indices, provided that the agent under investigation is capable of affecting functional development. Not all teratogens follow the dose-response model, and will not necessarily impact every point along the continuum; it is possible for an agent (e.g., Thalidomide) to cause mutations without affecting functional or growth impairment. Under these circumstances, using behavioural or growth indices to assess the degree of harm associated with such an agent would be inappropriate. However, when considering the effects associated with an agent that is capable of causing

impairment ranging from functional teratogenesis to death, behavioural indices become the most sensitive marker for establishing embryotoxicity (Vorhees, 1986).

With respect to the current study, it is possible that at low doses, cigarettes and marihuana may exert teratogenic affects below the threshold for causing growth impairment or mutations. In the case of these drugs, behavioural indices of impairment such as attention deficits may be among the most sensitive markers for assessing the degree to which these substances are toxic to the developing fetus.

Overview of the Present Study

In the literature review that follows, several broad areas of research are covered. A brief synopsis of the topics presented is provided here. First, the consequences for development in the fetus and newborn resulting from prenatal exposure to cigarettes and marihuana will be described. The emphasis is placed on possible mechanisms through which each drug exerts harmful effects, in order to establish each drug as a teratogenic agent. Second, an introduction to the general area of attention will be presented, followed by a discussion of methodological issues related to the assessment of sustained attention and impulse control (e.g., measurement issues and developmental aspects of attention). Finally, a review of the evidence linking prenatal exposure to cigarettes and marihuana to problems with sustained attention and impulse control will be described.

Cigarettes

In recent years, cigarette use during pregnancy has been the focus of an extensive body of research in the area of behavioural teratology. A great deal is now known about the potential effects of smoking on pregnancy outcome and on early postnatal

development. Cigarettes are a known teratogen, and prenatal exposure is linked to a range of adverse outcomes during pregnancy including increased risk of stillbirth and spontaneous abortion, both possibly caused by placental abnormalities such as abruption (premature separation of the placenta) and placenta previa (abnormally located placenta) which occur in higher-than-normal rates in smokers (Oncken, Hardardottir & Smeltzer, 1998). In the postnatal period, smoking is strongly correlated with lowered birth weight (Fried, 1992; Fried & O'Connell, 1987; Fried et al., 1999) and has been implicated as a contributing factor in sudden infant death syndrome (SIDS) (Slotkin, 1998).

Neurotoxic Effects of Cigarettes on Fetal Development

Cigarette smoke is a complex substance containing more than 4,000 chemicals and constituents (National Institute on Drug Abuse, 1998). Nicotine and carbon monoxide gas (CO) are among the toxins occurring in the highest concentrations (Luck, Nau, Hansen & Steldinger, 1985), and both have been frequently investigated with respect to their effects on fetal and post-natal development. Both substances have been shown to easily cross the placental barrier, and in fact, the fetus is exposed to higher nicotine and CO levels than the smoking mother (Luck et al., 1985; Milberger, Biederman, Faraone, Chen & Jones, 1996; Olds, 1997; Walker et al., 1999). Nicotine and CO are both believed to cause damage to the brain of the developing fetus (Navarro, Seidler, Eylers, et al., 1989; Slotkin, 1998), and have been linked to a broad range of behavioural and cognitive impairments in the offspring of women who smoke during pregnancy (for reviews see Fried, 1992; Olds, 1997; Walker et al., 1999).

One of the problems facing researchers studying the unique effects of nicotine and CO on human fetal development is the issue of concurrent exposure to both toxins that occurs with cigarette smoke. The complex nature of cigarette smoke, and the confounding effect that these substances have on one another, makes it difficult to link impairments arising from prenatal exposure to cigarettes to the effects of any single compound. Due to ethical considerations, controlled studies that isolate the toxins found in cigarettes cannot be conducted with human subjects. Animal studies have thus been employed to allow researchers to examine the unique effects of one substance while controlling for potentially harmful effects associated with the other. The differential effects of nicotine and CO are described below.

Nicotine. Nicotine is the most chemically active agent in cigarette smoke (National Institute on Drug Abuse, 1998) and is a well-documented neural teratogen (Navarro, Seidler, Eylers, et al., 1989; Navarro, Seidler, Schwartz, et al., 1989; Olds, 1997; Slotkin, 1998; Slotkin, Lappi & Seidler, 1992), capable of causing disruption of cellular and synaptic activity in the developing fetus. Nicotine differs from other teratogenic agents in that instead of globally affecting the central nervous system (CNS), it targets a specific population of neurotransmitters that are part of the cholinergic neurotransmitter system (Slotkin, 1998; Navarro, Seidler, Schwartz, et al., 1989). These receptors, termed “nicotinic receptors” are present in human fetal tissue from midgestation (12 weeks gestation) (Cairns & Wonnacott, 1988) and continue to increase rapidly in number throughout the remaining gestational period (Slotkin et al., 1992).

As the fetal brain develops, cells receive stimulation from various neurotransmitters, signaling the start or end of a given stage of cellular development. It is believed that nicotinic receptors play a crucial role in controlling the switch from cell mitosis (replication) to differentiation (Slotkin, 1998). It is via this mechanism that nicotine exerts its toxic effects. During normal development, early in the postnatal period, a spike in cholinergic activity signals the end of cell division and the beginning of differentiation in brain regions with high densities of nicotinic receptors (Navarro, Seidler, Eylers, et al., 1989). These regions include the midbrain, brainstem, and forebrain regions (Slotkin, 1998), with high concentrations found in the basal ganglia and thalamus (Cairns & Wonnacott, 1988). The distribution of nicotinic receptor sites is similar in human fetal brain tissue and in the rat brain, and is consistent with patterns observed in adult human brain tissue (Cairns & Wonnacott, 1988). Studies using rats indicate that prenatal exposure to nicotine disrupts the natural process of cellular maturation. The presence of exogenous nicotine, acting on the nicotinic receptors, prematurely signals the end of cellular mitosis during the prenatal period, resulting in reduced cell numbers and irreversible cellular impairment (Navarro, Seidler, Eylers, et al., 1989; Navarro, Seidler, Schwartz, et al., 1989; Slotkin, 1998).

In addition to directly affecting CNS development, nicotine interferes with normal fetal development by periodically reducing the amount of oxygen and nutrients that reach the fetus. Nicotine is a vasoconstrictor, and by decreasing blood flow to the uterus, disrupts the supply of oxygen and nutrients to the fetus (Bee, 1994; Olds, 1997; Oncken et al., 1998; Walker et al., 1999).

As with most teratogens (Bee, 1994), a critical period of development exists in which nicotine exposure is most damaging to the fetus. Exposure during mid to late gestation, in particular during the third trimester of pregnancy when the brain is undergoing rapid growth and structural organization, appears to be most harmful to the developing fetus (Oncken et al., 1998; Slotkin et al., 1992; Walker et al., 1999). According to the behavioural teratogenic model (Vorhees, 1986), even at low doses, exposure to nicotine during this critical period may result in observable functional impairments in the postnatal period.

Carbon Monoxide. Carbon monoxide (CO) is believed to harm the fetus by producing a state known as hypoxia, characterized by low blood oxygen levels. CO reduces the capacity of the blood to carry oxygen by binding to hemoglobin and displacing oxygen molecules, producing carboxyhemoglobin (COHb). When a woman smokes during pregnancy, COHb inhibits the delivery and release of oxygen to the fetus, resulting in brief, but chronic, intervals where the fetus is exposed to lower than normal levels of oxygen (Olds, 1997; Walker et al., 1999). Some researchers feel that it is chronic, low level exposure to a toxin, particularly one causing hypoxia, that is most harmful to long-term fetal development (Courchesne, Townsend & Chase, 1995; Milberger et al., 1996).

Effects of Prenatal Exposure to Cigarettes on Birth Weight

One way of assessing the teratogenic effects associated with a given substance is through the use of growth measures such as birth weight (Vorhees, 1986; Walker et al., 1999). The rationale behind using growth measures as a marker for teratogenic effects is

that many insults to the developing fetus, such as exposure to certain drugs or malnutrition, result in a condition known as “brain sparing” in which the CNS is protected from harm relative to the rest of the organism (Navarro, Seidler, Schwartz, et al., 1989). In the case of many toxins, growth impairment occurs in the absence of CNS impairment; thus growth measures provide researchers with a means of evaluating the safety threshold of a given substance (Navarro, Seidler, Schwartz, et al., 1989). Cigarette smoking during pregnancy is consistently linked to infant growth impairment, specifically in terms of lowered birth weight (Fried & O’Connell, 1987; Fried et al., 1999; Navarro, Seidler, Eylers, et al., 1989; Navarro, Seidler, Schwartz, et al., 1989). The association is dose dependent; women who are heavy smokers give birth to infants who weigh less than infants born to light or non-smokers (Fried, 1993; National Institute of Drug Abuse, 1998). In the OPPS, smokers who were identified as heavy users (>16 mg nicotine/day; equivalent to a pack or more per day) during the third trimester of pregnancy, gave birth to infants who were on average 181 grams lighter than infants born to non-smokers (Fried & O’Connell, 1987). In comparison, infants born to smokers who reported heavy use during the first trimester only, were on average 134 grams lighter relative to the babies of non-smokers (Fried & O’Connell, 1987). These findings are in agreement with research findings showing that mid to late gestation is a critical period for prenatal exposure to cigarettes (Oncken et al., 1998; Slotkin et al., 1992).

Animal studies investigating the negative effects of maternal cigarette smoking on birth weight outcome have produced striking evidence concerning the harmful effects of nicotine on fetal and infant development. Rat pups exposed to low to moderate doses of

nicotine (equivalent to less than a pack of cigarettes per day in humans) showed neural and cellular abnormalities in the absence of growth impairment, comparable to damage seen at doses of nicotine high enough to cause growth retardation (Navarro, Seidler, Schwartz, et al., 1989). As discussed previously, nicotine is a unique neurotoxin in that it selectively targets the nervous system by acting directly on endogenous nicotinic receptor sites. Brain-sparing does not occur with exposure to nicotine, and CNS damage can occur at levels of exposure that do not impact on growth measures. Given that nicotine exerts negative effects on fetal CNS development below levels at which growth is affected (Slotkin, 1998), researchers need to be cautious when using growth measures to estimate the degree of harm to the infant caused by maternal smoking. Use of standard markers of impairment such as low birth weight may underestimate the harmful effects of maternal cigarette smoking on human fetal and infant development, and as such, may not be appropriate for gauging the degree of damage to the infant resulting from in utero exposure to nicotine. In the case of nicotine, behavioural indices are likely the most sensitive measures for assessing the embryotoxicity associated with the drug (Vorhees, 1986).

Effects of Prenatal Exposure to Cigarettes on Behavioural Functioning in Newborns and Infants

In contrast to the extensive body of literature reviewed previously that documents the teratogenic effects of nicotine seen with respect to birth weight, fewer studies report on teratogenicity in terms of functional (behavioural) impairment that can be attributed to prenatal exposure to nicotine. Research findings suggest that prenatal exposure to

nicotine may result in a number of teratogenic effects with respect to altered behavioural functioning in human newborns and infants (for reviews, see Olds, 1997; Walker et al., 1999). In a study conducted using the OPPS sample, Fried and Makin (1987) found that prenatal exposure to nicotine was associated with decreased auditory habituation (assessed at 3-6 days of age) and increased tremors (assessed at 9 days of age) in newborn offspring of maternal drug users compared to newborn controls. At 30 days of age, infants exposed prenatally to nicotine continued to display increased tremors, and demonstrated increased muscle tone, motor reflexivity, and a higher incidence of startles compared to non-exposed infants (Fried & Makin, 1987). At one year of age, infants in the OPPS sample who were prenatally exposed to nicotine continued to show impairments on aspects of auditory functioning (Fried & Watkinson, 1988), a finding which suggests that the teratogenic effects of nicotine on functional development are not transient, and may have long-term consequences for the offspring of maternal smokers.

Marihuana

The evidence concerning teratogenic effects in offspring of women who use marihuana during pregnancy is sparse. Animal research has shown that Δ^9 -tetrahydrocannabinol (THC), the active ingredient in marihuana, crosses the placental barrier following maternal consumption of the drug (Hutchings & Fried, 1999) and thus has the ability to act directly upon the fetus, but relatively little research is available that addresses the role marihuana plays in human pregnancy outcome. In contrast to findings from animal studies that link chronic prenatal exposure to moderate to high doses of marihuana (in combination with a low protein diet) to adverse outcomes such as

increased stillbirths and postnatal deaths, and low birth weight (Charlebois & Fried, 1980), human studies do not show strong links between maternal use of marihuana during pregnancy and higher incidence of adverse pregnancy outcomes, and low birth weight in the postnatal period.

Findings from the OPPS, the sample upon which the present study is based, showed no differences in terms of pregnancy outcome between babies born to marihuana users and babies born to comparison subjects matched on a number of control variables (Fried & O'Connell, 1987). However, the OPPS is a low-risk sample and findings must be interpreted with this point in mind, as it is believed that lifestyle factors (i.e., diet, general health, education, etc.) characteristic of low-risk groups play an important role in protecting the offspring of marihuana users from potential harmful effects of the drug (Fried, 1989).

Fried (1993) has suggested that the effects of impaired development in children prenatally exposed to marihuana may be subtle in nature, and not easily assessed with global testing procedures typically used to measure behaviour in infancy. He postulates that differences between drug exposed and comparison children may not be discernable until brain development reaches a certain stage of maturation in early childhood when specific, rather than global tests of competency can be employed. This hypothesis is based on studies from the OPPS that failed to detect any differences between children prenatally exposed to marihuana and comparison children prior to the age of four. At four years of age, differences were observed between these groups, with marihuana-exposed children showing deficits in verbal ability and memory (Fried, 1995). Follow-up

testing with the OPPS sample showed impaired performance among the marihuana-exposed children at age six years on a test measuring sustained attention (Fried et al., 1992).

Day et al. (1994) have reported similar findings in a high-risk sample of three year old children exposed prenatally to marihuana. After controlling for the effects of various confounding variables including prenatal exposure to alcohol, tobacco, and cocaine, marihuana exposure during pregnancy was significantly associated with negative performance on a test of cognitive development (Stanford-Binet Intelligence Scale: Fourth Edition, SBIS). Interestingly, these negative findings were attenuated among white children by preschool/day-care attendance. However, for African-American children, preschool/day-care attendance did not moderate the negative effects of marihuana. These findings lend weight to the hypothesis discussed previously that improving lifestyle factors in a child's environment (i.e., diet, general health, education, etc.) may, for some children, protect against the potential harmful effects of marihuana exposure in utero.

In a study conducted on a high-risk sample of three-year-old offspring born to women who used marihuana, alcohol, and opiates during pregnancy, Griffith, Azuma and Chasnoff (1994) found that marihuana exposure was associated with lower subscale scores on abstract/visual reasoning on a test of cognitive development (Stanford-Binet Intelligence Scale: Fourth Edition; SBIS). No differences were observed between drug-exposed and non-drug exposed controls in terms of overall cognitive ability measured by the SBIS. The authors point out that the majority of drug-exposed children displayed

normal cognitive development, and attribute these findings to early therapy interventions designed to reduce the effect of lifestyle factors (i.e., poor diet, health, lower parental education and income, etc.) associated with high-risk populations.

Summary

Very little research exists that addresses the degree to which prenatal exposure to marihuana in humans is harmful to the developing fetus. Marihuana is an illegal drug, and studies concerning its use are limited by serious methodological problems including concurrent use of other drugs, self-report, lack of ability to quantify dosage, etc. (Hutchings & Fried, 1999).

Findings from animal studies suggest that drug exposure in combination with poor environmental conditions (i.e. low protein diet) can increase the probability of producing negative effects such as stillbirth, and postnatal death in the offspring (Charlebois & Fried, 1980). The offspring of rats that were fed a high protein diet and exposed to marihuana while pregnant did not show the adverse outcomes of rat pups born to rats fed a low protein diet, suggesting that the effects of marihuana are most likely to be harmful under high risk environmental conditions (Fried, 1989). Early evidence to date using human subjects is suggestive that cognitive and behavioural deficits are present in children exposed prenatally to marihuana (Day et al., 1994; Fried, 1992, 1995; Griffith et al., 1994). However, more research, particularly longitudinal studies that follow children's development over time, will need to be conducted before a clear picture concerning the range of teratogenic effects of marihuana in the offspring of maternal drug users will begin to emerge.

General Attention Literature

Models of Attention

In recent years, theorists studying human have proposed that the construct loosely referred to in the literature as *attention*, should not be viewed as a unified concept, but rather as a system linking a number of disparate elements within the larger domain of cognitive functioning (Barkley, 1997; Mirsky, Anthony, Duncan, Ahearn & Kellam, 1991; Mirsky et al., 1995). This multi-component view of attention stems in part from research showing that different aspects of attention rely on the coordination of a range of cognitive functions that vary across tasks and situations (Mirsky et al., 1991). Mirsky and colleagues (Mirsky, 1996; Mirsky et al., 1995; Rebok et al., 1997) have proposed a five-factor model of attention based on research conducted using non-clinical adult and children populations. The model organizes the global construct of attention into five distinct elements – focus/execute, sustain, shift, encode and stabilize- each relying on different cognitive functions for its execution.

Focus/execute involves the ability to scan an array and locate a target, and execute a motor response; sustain represents the ability to remain focused on-task for a prolonged period of time; shift is concerned with the ability to disengage focus; encode is the ability to hold information in working memory while actively using such information, and stabilize assesses the maintenance of effort as a function of time. Within the framework of this model, both the ability to sustain attention and control impulsive responding are proposed to fall under the domain of the “sustain” element of attention. Impulse control is also considered as part of the “stabilize” element (Mirsky, 1996). The researchers

further postulate that each element of attention is localized in a specific region of the brain, and that together, these individual components coordinate their actions to form a unified attentional system (Mirsky et al., 1991; Rebok et al., 1997).

In developing the five-factor model of attention, Mirsky et al. (1995) developed a large test battery to examine differential components of the attentional system, and as part of an overall evaluation of different aspects of attention, used outcome measures obtained from a continuous performance test to assess both sustained attention and impulse control.

Barkley (1997) has proposed a model of executive function that views behavioural inhibition, also referred to as response inhibition and impulse control, as a key component involved in the efficient operation of specific components of attention. Barkley suggests that impairments typically associated with attention deficit hyperactivity disorder (ADHD), specifically problems with impulse control and sustained attention, are due to dysregulation in the area of executive function, a component of cognitive function linked to the domain of attention. Within this framework, behavioural inhibition refers to the ability to control self-directed responses by: (a) inhibiting a prepotent response to an event, (b) stopping a response once it has been initiated, and (c) keeping other events from encroaching on the delay period (the period of time between response inhibition and response execution) (Barkley, 1997). Barkley (1997) posits that impaired behavioural inhibition has a negative effect on several aspects of higher cognitive functioning that rely on intact inhibitory control for their efficient operation. These executive functions are involved in the efficient execution of goal-directed

behaviour, and include working memory, self-regulation of affect/motivation/arousal, internalization of speech and reconstitution. According to Barkley's model, these executive functions control self-regulatory aspects of motor behaviour, and if these functions are disrupted as a result of deficits in behavioural inhibition, the outcome is behaviour characterized by poor impulse control and response inhibition. Deficits in sustained attention are viewed in terms of this model as arising from the dysregulation of goal-directed behaviour, and are secondary to problems observed with impulse control.

In summary, Barkley views deficits in sustained attention and impulse control as stemming from problems with behavioural inhibition. Impairments in terms of behavioural inhibition in turn impact on executive functions that control among other things, self-regulatory motor responses which play a role in inhibiting inappropriate responding, and goal-directed behaviour which impacts on the ability to sustain attention.

Tasks Used to Assess Attention

Many studies that have looked at performance measures of sustained attention and impulse control have used a version of the continuous performance task (CPT) developed by Rosvold, Mirsky, Sarason, Bransome and Beck, in 1956 (as cited in Corkum & Siegel, 1993). This test has gained popularity in recent years as an assessment device used to differentiate between hyperactive and comparison children (Barkley, 1997; Corkum & Siegel, 1993; Sykes, Douglas & Morgenstern, 1973). As mentioned previously, the CPT provides performance measures for both sustained attention (errors of omission) and impulse control (errors of commission), as well as providing information regarding stability of performance over time.

This tool is widely used in both clinical and non-clinical settings, but several caveats need to be kept in mind when interpreting the results of studies that have used this test to look at deficits in attentional performance. Many versions exist that differ on task variables such as length of task, task difficulty, and modality (e.g., auditory and visual). Inter-test reliability is poor and studies can differ significantly in terms of findings because of the lack of standardization on task variables across studies (Corkum & Siegel, 1993). Mirsky et al. (1995) suggest that for a tool to be a good assessment instrument it must meet a number of conditions including: a) it must measure different aspects of attention, b) must have established reliability and validity, and c) must have valid test norms for different ages and populations.

A debate currently exists in the literature concerning the types of errors that the different types of CPT errors (omission and commission) are purported to measure. Most researchers are in agreement that omission errors reflect a deficit in sustained attention (Corkum & Siegel, 1993; Halperin et al., 1988). With respect to commission errors, although some researchers feel that these errors reflect problems with impulse control (Corkum & Siegel, 1993), others (Conners, 1995; Halperin et al., 1988; Mirsky, 1996) point to recent evidence that suggests that several sub-types of commission errors exist, and that only certain sub-types of errors are consistent with problems with impulsive responding. Specifically, on CPT tasks using an AX paradigm, where a participant is “primed” to respond to a target stimulus “X” only when preceded by an alerting stimulus “A”, only errors reflecting an “A-not-X” response where the individual responds to an

“A” that is not followed by an “X”, in combination with fast response times, are felt to reflect problems with impulse control.

Normative Development of Sustained Attention and Impulse Control

A key issue in the study of pathological development is defining what constitutes abnormal . When looking at pathological behaviour in terms of development, it is often the timing and context in which the behaviour occurs that makes it appear pathological. For example, poor impulse control is normal and expected behaviour in very young children, but this type of behaviour would perhaps be considered unusual in an older child. Pathological behaviour is that which persists beyond the period in which normal development is expected to be complete. For example, a sustained attention deficit is identified as a failure to perform at age-appropriate levels on tasks measuring sustained attention. In order to identify children with attentional deficits, researchers must be knowledgeable with respect to the normal developmental trajectory of a given aspect of attention because the normal trajectory is a baseline for establishing what qualifies as abnormal behaviour (Olson, Schilling, & Bates, 1999).

In terms of the normal development of sustained attention and impulse control, it is possible that they will not share a common developmental trajectory. Some theorists have proposed a stage-like theory of development, with various brain regions and cognitive/al functions developing at different times (Anastopoulos & Barkley, 1988; Barkley, 1997; Mirsky et al., 1995). Under this view of differential development, care must be taken to accurately determine what is considered normal behaviour at a given age, or one runs the risk of classifying behaviour as pathological, when in fact, normal

developmental processes are still at work. Prior to the completion of normative development, the purpose for testing for differences on measures of attention is not for classifying attention deficits, but to identify children who may be at risk for attentional problems later on (Gordon, 1984).

Sustained Attention. The results of several normative studies carried out on non-referred children indicate that sustained attention develops rapidly throughout early childhood, and does not show stability until children are between the ages of 8 to 10 years, with adult-level performance perhaps not being reached until early adolescence, around the age of 13 years (Corkum & Siegel, 1993; Lin, Hsiao, & Chen, 1999; Greenberg & Waldman, 1993; Rebok, et al., 1997). Based on these findings, it is not meaningful to talk of deficits in sustained attention in young children under 8 years of age, as normal development is still progressing and maturing into middle to late childhood.

Impulse Control. A number of studies have looked at the normative development of impulse control using normative samples. The results of two longitudinal studies conducted on normal children, suggest that impulse control develops earlier than sustained attention, with measures showing stability between the ages of 6 and 8 years (Olson, 1989; Olson et al., 1999). However, other studies have shown a later developmental trend, showing rapid change in the ability to inhibit responding between the ages of 6-8 and 10-12. Levin et al. (1991) (as cited in Barkley, 1997) found impulse control continued to develop in normal children between 7-8 and 9-12 years of age. Similar findings are reported by Greenberg & Waldman (1993), who found impulse

control began to stabilize in late childhood between the ages of 10-13. It is not clear why researchers are not in agreement regarding the normative development of impulse control, but it is possible that some of the disagreement comes from lack of standardization across studies with respect to the CPT versions used to assess impulse control. For example, Greenberg & Waldman (1993) used a version of the CPT that incorporated a very difficult task that was shown to dramatically disrupt impulse control, particularly among younger children. It is probably reasonable to postulate that impulse control is testable in children between the ages of 6 and 8, provided that the CPT version used has appropriate age norms for comparison.

Prenatal Drug Exposure and Problems with Sustained Attention and Impulse Control

The following section reviews findings relating prenatal exposure to cigarettes and marihuana to problems with sustained attention and impulse control. The findings related to cigarettes and marihuana will be discussed separately.

Cigarettes

Several studies have shown a link between prenatal exposure to cigarettes and problems with sustained attention and impulse control. Fried et al. (1992) report that prenatal exposure to cigarettes was associated with poor impulse control in six year old offspring of maternal drug users. As part of an ongoing longitudinal study, participants in the OPPS (N=126) were assessed with respect to performance on a continuous performance task (CPT) measuring sustained attention and impulse control. Children of heavy smokers performed poorly on direct tests of delayed responding (measuring impulse control) and sustained attention (vigilance) compared to the children of non-

smokers. Significant differences were found between these groups in terms of lower efficiency ratios (rewarded responses/total responses) on the delay task and an increased number of commission errors on the test of sustained attention. Measures indicative of problems with sustained attention, namely errors of omission on the vigilance task, did not distinguish among the offspring of smokers and non-smokers. These results suggest that maternal smoking during pregnancy may play a role in the development of poor impulse control in offspring exposed to cigarettes prenatally.

In another study conducted with the OPPS sample, Kristjansson, Fried & Watkinson (1989) found that prenatal exposure to cigarettes was associated with a significant number of commission errors on an auditory task and also to higher levels of activity in 4-7 year old children (N=76-79). These findings are reflective of problems with impulsive responding, and are consistent with results obtained on six year-olds in the same sample (Fried et al., 1992).

Results of a recent follow-up study with the OPPS sample indicated that in adolescents aged 13 to 16 years, prenatal exposure to cigarettes was not associated with problems with impulse control (Fried & Watkinson, in press). These findings suggest that problems with impulse control previously observed in younger children ranging from 4 to 7 years of age (Fried et al., 1992; Kristjansson et al., 1989) had resolved by the time the children in the OPPS had reached their teens.

In a study looking at the effects of prenatal exposure to cigarettes, marihuana, alcohol, and cocaine on sustained attention and impulse control in six year old offspring (N=608) born to maternal drug users, Leech et al. (1999) found that exposure to nicotine

during the second and third trimester of pregnancy was significantly associated with errors of omission on a CPT task. In contrast to studies that have found a link between nicotine and errors of commission on a CPT task (Fried et al., 1992; Kristjansson et al., 1989), results such as those reported by Leech et al. (1999) suggest a connection between prenatal exposure to nicotine and problems with sustained attention.

Several methodological problems with this study (Leech et al., 1999) are apparent that impact on the validity of the findings. When analyzing the results of the CPT task, scores were averaged over blocks. This presents a problem because when assessing sustained attention, it is important to look at changes in performance over time, particularly for trials that occur at the end of the task where performance decrements (increased errors) may be most evident. Another point concerns the CPT task employed in this study. Validity and reliability data was not provided for the CPT version used by Leech et al. (1999). As discussed previously, studies using different tasks can differ substantially in terms of findings, and without validity and reliability measures, this limits the cross-study validity of these findings. In addition, it is not clear if the researchers controlled for the potentially confounding influence of other drugs, in this case, marihuana, alcohol and cocaine.

Streissguth and colleagues (Streissguth, Martin, Barr, Sandman, Kirchner & Darby, 1984) found prenatal exposure to nicotine was significantly related to both errors of omission and errors of commission on vigilance task. These findings suggest a link between nicotine and problems with both sustained attention and impulse control. Participants were four year old children (N=452) born to women who had used primarily

alcohol and nicotine during pregnancy. In terms of this study, meaningful interpretation of findings relating to measures of sustained attention and impulse control may not be possible because in children as young as four years of age, normal developmental milestones have yet to be reached. Results reported using such a young sample, well in advance of the expected completion of normal development must be interpreted with caution, and differences observed between children exposed to cigarettes and comparison children should be interpreted as indicative of potential future risk only.

Milberger et al. (1996) found that cigarette smoking during pregnancy was linked to a higher incidence of ADHD in the male offspring of heavy smokers in comparison to children of women who were classified as non-smokers during pregnancy. Milberger et al. (1996) compared rates of maternal smoking during pregnancy in 140 boys diagnosed with ADHD and 120 normal comparison boys ranging in age from 6 to 17 years, and found significant differences between the groups. Twenty-two percent of children with ADHD had a history of prenatal exposure to cigarettes, while only 8% of comparison children had mothers who smoked during pregnancy.

A serious methodological problem with this study concerns classification of the smoking groups. The researchers used a dichotomous classification system in which women were identified as either smokers or non-smokers. Smokers were defined on the basis of smoking at least one pack of cigarettes per day for at least three months during pregnancy. All other participants were classified as non-smokers. Under this classification, a woman who smoked $\frac{3}{4}$ of a pack of cigarettes per day for the entire pregnancy would be considered a non-smoker. It is possible that some of the cases of

ADHD in the non-smoking group could be due in part to smoking, as some women who were light to moderate smokers may have been present in this group. The results of this study need to be interpreted carefully with the classification system in mind.

In a large-scale, longitudinal, prospective study referred to as the Collaborative Perinatal Project (NCP), maternal smoking during pregnancy was a highly significant predictor of problems with hyperkinetic-impulsive behaviour in 7-year olds (N=2,339) as compared to asymptomatic controls (N=12,511) (Nichols & Chen, 1981).

Summary. The results of several studies show that exposure to cigarette smoke during pregnancy is associated with problems with impulse control and sustained attention in the offspring of maternal drug users. The majority of the data applies to children under the age of six. It is important to continue assessing these children longitudinally in order to determine the extent to which these early findings are continuous over time. It will be interesting to see whether these findings remain significant over time, possibly indicating a developmental delay or even a more permanent deficit for which continued central nervous system development is unable to compensate.

Marihuana

As part of an ongoing, longitudinal prospective study, Fried et al. (1992) found that prenatal exposure to marihuana was associated at age six with increased errors of omission on a continuous performance task measuring sustained attention.

As discussed in previous sections, errors of omission are thought to be indicative of problems with sustained attention. It is important to note that data used in this study

was collected from the OPPS sample when the children were six years old. In terms of assessment of sustained attention, the age of the participants calls into question the validity of results showing differences between marihuana and control groups on errors of omission. Based on normative data from non-clinical populations (Corkum & Siegel, 1993; Lin et al., 1999; Greenberg & Waldman, 1993; Rebok, et al., 1997), it is believed that sustained attention does not begin to develop until children are between the ages of 8 and 10. Therefore, in terms of this study, meaningful interpretation of findings relating to measures of sustained attention may not be possible because in six year-olds, normal developmental milestones have not yet been attained. As Gordon (1984) points out, the goal when testing young children is not to determine whether performance is abnormal, but rather to identify individuals who may be "at risk" for problems with sustained attention and impulse control later in their development. Results such as those reported by Fried et al. (1992) demonstrating differences on tests of sustained attention prior to the expected completion of normal development must be interpreted with caution, and differences observed between the marihuana exposed children and comparison children interpreted as indicative of potential risk in the future.

In another study conducted on the OPPS sample, Fried et al. (1998) found that prenatal exposure to marihuana compared to no exposure in 9 to 12 year olds (N=131), was linked to problems with executive functioning believed to be involved with impulse control behaviours. After controlling for potentially confounding variables however, marihuana was no longer significantly associated with executive function variables.

Follow-up study of the OPPS sample conducted when participants were between the ages of 13 and 16 years, revealed prenatal exposure to marihuana to be associated with increased errors of omission, consistent with problems with sustained attention (Fried & Watkinson, in press).

In a recent study conducted on a high-risk sample, Leech et al. (1999) found increased errors of commission on CPT tests measuring impulse control in six-year-olds exposed to marihuana during the second trimester of pregnancy. Cigarettes were not found to be associated with errors of commission on the CPT.

There are several methodological problems that need consideration before generalizing these findings to other samples. Validity and reliability data was not provided for the CPT version employed in this study, thus limiting the cross-study validity of these findings. It is also not clear if Leech et al. (1999) controlled for the potentially confounding influence of other drugs, in this case, cigarettes, alcohol and cocaine.

Finally, the results in this study are based on a high-risk sample. As discussed previously, some of the negative effects associated with prenatal exposure to marihuana are attenuated in otherwise low-risk environments. It is possible that in this study, the children were not afforded protection against the teratogenic effect of the drug due to the presence of other factors (e.g., presence of other drugs, low socioeconomic status) in their environment. These findings should be interpreted as representative of only other high-risk samples.

In another study using the same high-risk sample as Leech et al. (1999), Goldschmidt et al., (2000) found that third trimester use of marihuana was significantly associated with measures of hyperactivity and poor impulse control in a sample of 10 year olds (N=636). Attention deficits were assessed using the Swanson, Noland, and Pelham (SNAP) assessment, a questionnaire completed by the mothers rating behaviours characteristic of ADHD symptomology. This study suffers from some of the same methodological drawbacks as the study by Leech et al. (1999). It is not certain to what extent the researchers controlled for the presence of other drugs taken during pregnancy by the mothers in this study, but it should be noted that women who used marihuana throughout pregnancy were more likely to drink and smoke cigarettes than abstainers of marihuana. As mentioned previously, this is a high-risk sample, and results should be interpreted with this in mind.

Summary. Results of studies looking at the effects of prenatal exposure to marihuana on aspects of attentional functioning are far from consistent, and not without methodological problems that make interpretation difficult. Fried et al. (1992) found that in 6-year olds marihuana was associated with increased errors of omission, thought to be reflective of problems with sustained attention. Work with the same sample revealed marihuana to be linked to problems with executive function in children 9-12 years of age (Fried et al., 1998). Two studies based on a high-risk sample found higher numbers of commission errors indicative of problems with impulse control (Leech et al., 1999) and higher parent ratings of behaviours typical of ADHD, namely impulsivity and hyperactivity (Goldschmidt et al., 2000).

Present Study

Maternal cigarette smoking has been clearly linked to early problems relating to fetal and infant development (e.g., stillbirth, miscarriage, low birth weight, SIDS).

Evidence for the teratogenic effects of marijuana is not as strong, but with continued research in the area of fetal and infant development, the picture will undoubtedly become clearer. The long-term effects of prenatal drug exposure to cigarettes and marijuana on cognitive and behavioural development in older children and adolescents is less evident, as most of the work carried out to date has looked at infants and young children. A key issue for long-term studies looking at prenatal drug exposure is determining to what extent early signs of abnormal development consistent with putative teratogenic effects of a drug persist and continue to impact on long-term development.

Recent cross-sectional reports have produced findings that indicate prenatal exposure to cigarettes and marijuana may exert negative effects on aspects of attentional function in children. The topic under investigation in this thesis is a study of continuity looking at the long-term development of children prenatally exposed to cigarettes and marijuana with respect to findings related to attention. Specifically this study examined sustained attention and impulse control, both of which have been identified as problem areas for prenatally drug exposed children in the OPPS sample. Findings from previous studies looking at the performance of children exposed to cigarettes (Fried et al., 1992; Kristjansson et al., 1989; Leech et al., 1999; Streissguth et al., 1984)) and marijuana (Fried et al., 1992, 1998; Goldschmidt et al., 2000; Leech et al., 1999) are suggestive of problems with sustained attention and impulse control relative to non-exposed control

children, however, these studies are cross-sectional and limit the extent to which specific hypotheses concerning the continuity of these aspects of attention can be formulated.

The goal of the present study was to investigate of two areas of interest relating to attentional functioning in a sample of children participating in the OPPS who were followed longitudinally from age 6 to between the ages of 13 and 15 years. The research focused first on assessing the stability of attentional performance over time, followed by an examination of whether children prenatally exposed to nicotine and marihuana differed in the direction (i.e., improved or declined over time) and level (i.e., good or poor) of their performance relative to non-exposed controls on tests measuring sustained attention and impulse control. The following three questions were developed in order to examine longitudinal stability of attentional performance, and differences in performance over time across drug groups:

- 1) How stable is attentional performance over time? Further, do children prenatally exposed to nicotine and marihuana differ in their degree of stability from non-exposed control children, and if so, at what age(s) are these differences observed? For the purposes of the present study, stability is defined as the degree to which an individual maintains his or her quartile rank position in a distribution of scores on a given measure assessed repeatedly over time. Under this definition, a stable individual would be one who maintains the same rank order on a given measure across all three testing sessions.
- 2) Are there differences in terms of direction and level of performance over time on measures of sustained attention and impulse control between children prenatally

- exposed to nicotine and marihuana and non-exposed controls? Do children prenatally exposed to nicotine and marihuana differ in the percentage of individuals who improve or decline over time in terms of performance on tasks of attention from non-exposed controls, and if so, at what age(s) are these differences observed? Do the drug groups differ with respect to the distribution of good and poor performers, and if so, at what age(s) are differences observed?
- 3) Do the drug groups differ in terms of attentional performance over time after controlling for the effects of other influences that may impact on attentional behaviour?

METHOD

Participants

Children and their families had been participants since birth as part of an ongoing, longitudinal investigation initiated in 1978 in Ottawa, Canada. Subjects in the Ottawa Prenatal Prospective Study (OPPS) have been the focus of a number of reports examining the neurocognitive development of offspring born to mothers who used cigarettes, marihuana and alcohol during pregnancy. The OPPS is comprised of a low-risk, predominantly white, middle-class sample. Six hundred and ninety-eight women initially volunteered to participate in the OPPS after learning of the study through their doctors, or through notices posted in hospitals and clinics in the Ottawa area. The women were interviewed throughout their pregnancies, once during each trimester of pregnancy remaining following entrance to the study. The same female interviewer conducted all interviews, both initially and at follow-up, to establish rapport and to enhance reliability

of self-report data. Information collected during interviews included age, education and family income data, as well as information pertaining to pattern and quantity of drug use (cigarette, marihuana and alcohol), general health (both current and prior to pregnancy), height and weight information, diet, exposure to secondhand smoke, and history of previous pregnancy.

Subsequent to extensive testing of all offspring born to the initial sample of 698 women during the neonatal and infancy period, a cohort of 190 children was selected for follow-up after 1980. In order to accommodate statistical considerations such as sufficient sample size and power, over-sampling of children exposed prenatally to cigarettes, marihuana and alcohol was carried out during the final selection process. One hundred and forty children born to women who reported any use of marihuana during pregnancy, and/or who drank in excess of a daily average of 0.85 ounces of absolute alcohol per day, and/or who consumed on average, at least 16 mg of nicotine (equivalent to one package of cigarettes of average strength) per day were included in the follow-up sample. For comparison purposes, 50 children born to non-users of marihuana, who abstained or drank little alcohol during pregnancy, and who were non-smokers, were randomly selected to participate in follow-up studies.

Standardized laboratory testing of sustained attention and impulse control was conducted at three separate testing sessions during the follow-up period spanning early childhood to adolescence. A trained female assessor who was blind with respect to maternal drug history carried out all testing in controlled conditions in the laboratory at Carleton University. The first assessment took place when the children were 6 years of

age. The second assessment was given as part of a test battery administered once when the children were between 9 and 12 years of age, and a third assessment, again part of a test battery, was administered once during adolescence, when participants were between 12 and 15 years of age. At each testing session, maternal ratings of behaviour related to aspects of attention were also collected.

Sample selection and exclusion criteria. Of the 190 children initially selected for follow-up, 157 children were available to participate in the present study. The primary focus of the study relates to continuity of performance over time; therefore only children who had successfully completed all three testing sessions were selected for the present study (n=96). Children who were clinically identified as having ADHD or who were taking medication (i.e., Ritalin) for attention problems at any of the three testing sessions were excluded from the present study (n=4).

Additional exclusion of subjects was necessary because suitable age norms were not available for one of the tests used in the present study. Age norms for the Gordon Diagnostic System (GDS) Vigilance Task (Gordon, 1984) were only available in terms of percentile ranking, and as such, were not appropriate for use in comparing across the different tests employed to look at sustained attention and impulse control. Both tests used to assess attention in the present study, the GDS Vigilance Task and the Conners' Continuous Performance Test (CPT; Conners, 1995), considered children between the ages of 10 and 11 years to be homogeneous with respect to age. To create homogeneous age groups, children who were either nine years of age (n=1) or 12 years of age (n=9) at the time of the second assessment were excluded, resulting in a group comprised of 10 to

11 year olds. At the third assessment, children who were tested prior to 13 years of age were also excluded (n=1). The final sample used in the present study was comprised of 81 children (46 boys, 35 girls).

Instruments and Procedures

Participants in the OPPS were administered a number of tests purporting to measure various aspects of attention, and prior to the final test selection, an assessment of each instrument was carried out to determine its suitability for inclusion in the present study. After carefully considering methodological issues (e.g., does a test provide the necessary outcome variables to assess sustained attention and/or impulse control) and the design of the proposed study, the GDS Vigilance Task, the Conners' CPT, and the Conners' Parent Rating Scale-48 (CPRS-48, Conners, 1989) were selected for use in the present study. To take into account the possibility of ceiling effects occurring with simpler tasks used to assess attention in younger children, the GDS Vigilance Task was replaced by the Conners' CPT, a related, but more difficult task, administered when the participants were between 13 and 15 years of age.

First Assessment: Age 6 Years

Sustained Attention and Impulse Control. The children were assessed using the GDS Vigilance Task, a 9-minute computerized task that runs on a self-contained, one-button, solid-state console. This task is a well-established standard version of the CPT, and meets Mirsky et al.'s (1995) criteria of a good measure of attention, as it assesses both sustained attention and impulse control, has established reliability and validity data, and appropriate test norms (see Appendix A for psychometric properties).

The Vigilance Task uses a “1/9” paradigm which requires the participant to press a button in response to the number “9” (target stimulus) when preceded by the number “1” (alerting stimulus). A fixed sequence of single-digit numbers is presented, with 15 “1/9” presentations occurring in each of three 3-minute time blocks. Each number is presented for 200 milliseconds (ms), with an interstimulus interval of 800 ms occurring between presentations. Three blocks of 180 trials are presented, for a total of 540 trials and 45 “1/9” presentations over the entire 9-minute task.

Two measures of interest were taken from the GDS Vigilance Task summary data at 6 years of age for use in the present study: the number of errors of omission (a measure of the ability to sustain attention), where the child failed to respond to the target stimulus, and the number of errors of commission (a measure of the ability to inhibit impulsive responding), where the child pushed the button in response to non-target stimuli. Scores on each measure were computed for each 3-minute block, and for the entire 9-minute task.

Each child was given standardized instructions on how to perform the task. The assessor remained in the room with the child for the duration of the assessment to ensure that the child understood all instructions, and complied with the demands of the task.

Conners’ Parent Rating Scale-48. At the time of testing, each mother completed a behavioural symptom checklist, the Conners’ Parent Rating Scale-48 (CPRS-48; Conners, 1989). The instrument is a 48-item observational assessment tool that enables parents to rate their child’s behaviour across six domains using a four-point scale. Four items from the Impulsive-Hyperactive Scale were used in the current study: “excitable,

impulsive”; “restless or ‘squirmy’”; “wants to run things”; and “restless, always on the go” (see Appendix A for psychometric properties).

Second Assessment: Age 10-11 Years

During the follow-up assessment given between 10 and 11 years of age, each child was tested again as part of a large neuropsychological test battery. Participants ranged in age from 10 to 11 years; for the purposes of reporting results, the age of participants at the second assessment will hereby be reported as 10 years of age. All children were re-assessed using the GDS Vigilance Task. As with the first assessment when the children were 6 years of age, the mothers completed the CPRS-48. All testing procedures were carried out in a manner consistent with procedures employed when the children were tested initially at age 6 years. Omission errors and commission errors were the two variables of interest from the GDS data collected at 10 years of age for use in the present study.

Third Assessment: Age 13-15 Years

Participants at the third assessment ranged in age from 13 to 15 years; for the purposes of reporting results, the age of participants at the third assessment will hereby be reported as 13 years of age.

Sustained Attention and Impulse Control. The Conners’ CPT, a 14-minute computerized task (DOS version) running on a 66MHz Packard Bell, IBM compatible computer running Windows 3.1, was used to assess both sustained attention and impulse control in the 13 year old participants. This task is a well-established standard version of the CPT, and meets Mirsky et al.’s (1995) criteria of a good measure of attention, as it

assesses both sustained attention and impulse control, has established reliability and validity data, and appropriate test norms (see Appendix A for psychometric properties).

The standard version of the task (Conners, 1995), which uses letters as stimuli, requires the participant to press a button in response to all letters (target stimuli) *except* the letter “X” (non-target stimulus), and to withhold responding when “X” appears. A sequence of target letters is presented, with 2 “X” presentations occurring per 20-trial sub-block. Each letter is presented for 250 milliseconds (ms), with a variable interstimulus interval of either 1, 2 or 4 seconds occurring between presentations. Six blocks, each comprised of 3 sub-blocks of 20 trials, are presented, for a total of 360 trials and 36 “X” presentations over the entire 14-minute task.

Two measures of interest were taken from the Conners’ CPT summary data at 13 years of age for use in the present study: the number of errors of omission (a measure of the ability to sustain attention), where the child failed to respond to the target stimulus, and the number of errors of commission (a measure of the ability to inhibit impulsive responding), where the child pushed the button in response to a non-target stimulus (“X”). Scores on each measure were computed for each of the 6 blocks, and for the entire 14-minute task.

Each child was given standardized instructions on how to perform the task. As was the case during previous testing sessions, the assessor remained in the room during testing to ensure that all task instructions were understood, and compliance with task demands was met.

Mothers once again completed the CPRS-48 at the time of their child's assessment.

In addition to the data collected on the attention variables, information regarding the child's history of postnatal exposure to cigarette smoke was gathered at each assessment to be considered as a possible confounding influence with respect to attentional performance. Postnatal exposure to cigarette smoke was measured via a questionnaire given to the mothers at each testing session which asked mothers to report for each year of a child's life to date, whether or not a child had been exposed to cigarette smoke on a regular basis (i.e., at least 2 hours per day). For each child in the present study (n=81) a score was computed that represented the proportion of lifetime daily exposure to postnatal exposure to cigarette smoke. Postnatal exposure included sources of cigarette smoke both inside and outside the child's home environment.

Statistical Analysis

All analyses were carried out using archival data collected as part of the OPPS protocol began in 1978. Data analyses reflect the goals of the present study; to examine longitudinal stability of aspects of attentional functioning in the OPPS cohort, and to determine if non-exposed control children and drug-exposed children differ with respect to the direction and level of their attentional performance over time. Sustained attention and impulse control were analyzed separately for both nicotine and marihuana. Omission errors from the two CPT tasks were used as the dependent variable of interest in analyses assessing sustained attention, and commission errors from the two CPT tasks were the primary dependent variable in analyses assessing impulse control. In terms of the present

study, the CPT tasks that were administered to the OPPS sample unfortunately did not allow for an evaluation of the different sub-types of commission errors. Although an AX paradigm (Gordon, 1984) was used during two of the three testing sessions, measures of response time are not available for these data in order to permit an evaluation of the different sub-types of commission errors. The CPT used for the third testing session (Conners, 1995) did not employ an AX paradigm. The pattern of responding is different for this task, and does not allow for an examination of the different types of commission errors. With the preceding considerations in mind, for the purpose of the current study, commission errors were employed as the dependent measure in relevant analyses assessing impulse control.

The CPRS-48 Impulsive-Hyperactive scale was employed as a second dependent variable for analyses assessing impulse control. Parent ratings of impulsivity have been shown to correlate with objective, laboratory testing of impulse control (Conners, 1989), and this variable was included as a secondary measure of impulsive behaviour.

For all statistical analyses, maternal smoking during pregnancy was categorized into 3 groups: non-smoking; light-moderate (0.1-15.9 mg nicotine/day), and heavy (≥ 16 mg nicotine/day). Maternal marihuana use during pregnancy was categorized in a similar fashion into 3 groups: no/infrequent use, moderate use (>1 and <6 joints/week), and heavy use (≥ 6 joints/week). In the case of cigarettes, the control group was comprised of non-smokers, and for marihuana, the control group included members of the no/infrequent use category.

Separate statistical analyses looking at differential effects of gender on sustained attention and impulse control were not undertaken as part of the present study due to concerns regarding small overall sample size.

Analytic Plan

A number of descriptive and statistical analyses were employed in the present study to answer questions pertaining to two aspects of attentional functioning over time: stability of performance and direction and level of performance. With respect to stability, the goal was to examine the degree to which children maintained the same relative level of attentional performance across age intervals, or transitions, from age 6 to age 10, and from age 10 to age 13. A second goal related to the issue of stability was to determine if significant differences in stability existed between drug-exposed children and non-exposed controls at one or both of the age transitions that were possibly reflective of differences in developmental processes across groups.

The study of stability is limited to exploring the degree to which performance changes or remains the same over time. Stability analyses reveal nothing about the direction of performance changes over time (i.e., whether performance improved or declined) and nothing about the level of performance, either good or poor. To investigate group differences in direction of performance over time, analyses were carried out to determine if children prenatally exposed to nicotine and marijuana differ from non-exposed controls in terms of the percentage of children who improve and decline in performance at each age interval. To look at group differences in level of performance over time, analyses were conducted to test whether the drug groups differed with respect

to distribution of good and poor performers. Analyses were performed for each age group individually, and additionally, across each age interval, based on average performance over time. Finally, differences in performance over time among the smoking and marihuana groups were examined while controlling for the effects of potentially confounding variables.

In all cases, separate analyses were carried out for cigarettes and marihuana, for both omission errors and commission errors.

Stability of Attentional Performance: Description of Relevant Analyses. To examine the stability of attentional performance over time, the data for both omission and commission errors was analyzed using change scores computed across age intervals. Change scores were measures of stability computed on differences in performance from age 6 years to age 10 years (transition 1), from age 10 years to age 13 years (transition 2), and across all three assessments from age 6 years to age 13 years. Raw scores for each participant for both omission errors and commission errors at each of the three testing sessions were transformed into standardized z-scores to adjust for differences between the GDS Vigilance Task and the Conners' CPT. Each distribution of error scores was ranked from best to worst performance, and divided into quartiles. Standardized error scores falling in the first quartile were representative of the lowest number of errors (best performance), and were assigned a quartile rank of "1". Standardized error scores in the second quartile were assigned a rank of "2", scores in the third quartile were assigned a rank of "3", and standardized error scores falling in the fourth quartile, representative of the highest number of errors (worst performance) were assigned a quartile rank of "4".

Quartile ranks were recorded for each participant according to the quartile rank associated with their standardized z-score, for each type of error, at each of the three testing sessions, for a total of six quartile rank scores per individual. Appendix B presents the quartile ranks obtained by each subject at age 6, 10, and 13, categorized by drug group, for both omission and commission errors. Appendix C presents the percentage of cases within each drug group having quartile ranks from 1 to 4, at each of the ages tested, for both omission and commission errors.

For each participant, two change scores were calculated that represented the absolute difference in quartile rank position from the first assessment at age 6 years to the second assessment at age 10 years (transition 1), and the absolute difference in rank position from the second assessment to the third assessment at age 13 years (transition 2). Change scores represent the total “distance traveled” by an individual in quartile units from one assessment to another, within the distribution of scores. Change scores ranged from “0” (no change in rank position from one assessment to the second assessment) to “3” (e.g., rank position of “1” at one assessment and a rank position of “4” at the second assessment). Change scores do not take into account the direction of the change in quartile rank, rather, they measure the absolute magnitude, irrespective of direction, of stability of performance over time.

Stability was previously defined as the degree to which an individual maintains his or her rank position on a given outcome measure relative to the rest of the sample over time. For the purposes of the present study, attentional performance was considered highly stable at each transition if an individual obtained a change score of “0”. Change

scores of “1”, “2” and “3” reflect the magnitude of stability exhibited by an individual at each transition, with a score of “1” defined as moderate stability, a score of “2” defined as low stability, and a change score of “3” defined as unstable performance. Stability was assessed separately at both transition 1 and transition 2. An overall change score was also calculated for each participant; scores at transition 1 and transition 2 were added to yield an overall measure of stability from age 6 years to age 13 years (ranging from 0 to 6). Overall change scores of “0” through “6” reflect the magnitude of stability exhibited by an individual from age 6 to age 13. A score of “0” was defined as highly stable performance, a score ranging from “1-2” was defined as moderate stability, a score ranging from “3-4” was defined as low stability, and a change score in the range of “5-6” was defined as unstable attentional performance over time.

To answer the question “How stable is attentional performance over time?”, descriptive analyses were used to report on the percentage of children who exhibited varying degrees of stable performance over time, at each transition, and overall from age 6 to age 13. At each transition, stability was assessed by looking at the percentage of children who exhibited change scores equal to 0 (highly stable performance), 1 (moderate stability), 2 (low stability) and 3 (unstable performance). Stability from age 6 to age 13 was assessed by looking at the percentage of children who exhibited change scores equal to 0 (highly stable performance), 1-2 (moderate stability), 3-4 (low stability), and 5-6 (unstable performance) across all three assessments.

To assess whether children prenatally exposed to nicotine and marihuana differ in degree of stability from non-exposed controls, group differences in stability at transition

1 (age 6 to age 10) and transition 2 (age 10 to age 13) were tested using separate chi-square analyses for nicotine and marihuana, and for omission errors and commission errors.

Direction and Level of Attentional Performance: Description of Relevant Analyses. To examine group differences with respect to the direction (improvement vs. decline) and level (good vs. poor performance) of attentional performance over time, the data for omission and commission errors was analyzed using various chi-square analyses, nonparametric procedures and repeated measures analysis of covariance.

Using quartile ranks, two direction scores were calculated for each participant that reflected the direction of rank order changes at transition 1 (age 6 to age 10) and transition 2 (age 10 to age 13). For example, the direction score at transition 1 was computed by subtracting an individual's quartile rank at age 10 from their quartile rank at age 6. Negative scores reflected a decline in performance, while positive scores indicated improvement in terms of quartile rank order position in the distribution from previous assessments. Tests for differences among the different drug groups in terms of the direction of changes in performance at each transition were carried out using chi-square tests of independence.

Chi-square analyses and k-sample median tests (Conover, 1999; Glass & Hopkins, 1996) were employed to test for differences in the distribution of good and poor performers across groups at each assessment (i.e., age 6, age 10, and age 13), each transition (i.e., age 6 to age 10, and age 10 to age 13), and overall, from age 6 to age 13. At each assessment, using chi-square analyses, groups were tested for differences in the

distribution of good and poor performers based on quartile ranks within each drug group. Additional analyses were conducted using median tests, on the basis that differences in population medians could be interpreted as differences in performance with respect to omission and commission errors. Tests at each transition and overall were based on average performance, measured in terms of an average quartile rank score, across the ages being assessed. Group differences over time were assessed using median tests. At each transition, quartile ranks at both ages were summed for each child (ranging from 2-8) and divided by two, to yield an average quartile rank score (ranging from 1-4) across the two ages. Higher scores indicated poorer average performance over time; similarly, lower scores indicated good average performance over the ages tested. Similarly, overall group differences in the distribution of good and poor performers were tested based on an overall average quartile rank score (ranging from 1-4) calculated for each participant across all three assessments from 6 to 13 years.

Univariate repeated measures mixed-design analyses of covariance (ANCOVA) were used to assess group differences in performance over time on each of the attention variables while controlling for the effects of potentially confounding variables. Each attention variable (omission errors, commission errors and the subscale score on the CPRS-48 Impulsive-Hyperactive scale) was analyzed separately as the dependent variable with separate analyses conducted for both nicotine and marihuana as independent variables. Covariates selected for inclusion in the ANCOVAs varied across the different analyses depending on their association with nicotine and marihuana, as well as their association with the various attention variables.

Confounding Variables

A number of variables were examined as potential confounding variables prior to conducting statistical analyses. Variables that were considered for the above analyses included family income during pregnancy, average level of parent's education and the proportion of lifetime daily postnatal exposure to cigarette smoke. Birth order was also considered as a potential confound because studies have shown that this variable is significantly correlated with the ability to sustain attention (Streissguth et al., 1984). Finally, mother's age at time of child's birth was considered because women who smoke during pregnancy tend to be younger than non-smokers (Fried, 1992).

Using one-way analyses of variance (ANOVA), each of the potentially confounding variables was tested for association with nicotine and marihuana respectively using an alpha level equal to 0.10. Variables that were significantly related to either drug at an alpha level of 0.10 or less were considered as possible confounding variables in subsequent analyses for the drug with which a given variable was associated. Each of the possible confounding variables was further examined for association with each of the attention variables, and if significantly related at an alpha level of 0.05 or less, was included as a covariate in the relevant repeated measures ANCOVA. Maternal alcohol use and maternal use during pregnancy of the drug that was not considered to be of primary interest, for example, in the case of cigarettes, use of marihuana, were included as covariates in all statistical analyses.

Prior to conducting the statistical analyses using ANCOVA procedures, the distributions of scores for omission errors and commission errors at each of the three

testing sessions were examined with respect to outliers and screened for violations of normality and linearity. All distributions of omission error scores were found to be positively skewed, and were normalized using logarithm transformations. Similarly, two of the three commission error scores were positively skewed (commission errors at age 6 and age 10 years), and were normalized using logarithm transformations. The distribution of commission errors at age 13 years did not require transformation prior to the analyses. Following transformations, all omission and commission error scores were transformed into standardized z-scores to allow for comparisons between error scores on the GDS Vigilance Task and the Conners' CPT. Unless otherwise noted, in all subsequent ANOVA and ANCOVA analyses using the transformed variables, assumptions regarding homogeneity of variance, lack of factor by covariate interaction, and sphericity (for repeated measures analyses) were met.

In analyses where drug variables were used as covariates, nicotine, marihuana, and alcohol were expressed as continuous variables. All distributions of drug scores were positively skewed; to correct for non-normal distributions, nicotine and marihuana were normalized using logarithm transformations, while alcohol required an inverse transformation to achieve normality.

RESULTS

Demographic characteristics of the present sample for both smoking and marihuana groups are presented in Table 1. Annual family income and mother's age at time of birth are similar to those of women who gave birth in the Ottawa region at the time that participants in the OPPS sample were recruited for study (Fried & Watkinson,

Table 1

Demographic Characteristics and Maternal Drug Usage for Nicotine and Marihuana

	Nicotine (mg/day)			Marihuana (joints/week)		
	0	>0 & <16	≥16	≤1	>1 & <6	≥6
N	31	31	19	57	11	13
Annual Income (Can.\$)	32,900	31,989	29,105	34,676	22,364	26,308 ^b
Mother's Age (years)	28.7	27.8	26.7	29.0	26.6	24.4 ^{c†}
Ave. Parent Education*	2.9	2.6	2.2 ^a	2.8	2.5	2.1 ^{b†}
Parity	2.1	1.8	2.1	2.1	1.7	1.6
Prop. Postnatal Passive Nicotine Exp. (Lifetime)	.25	.50	.88 ^{d†}	.46	.42	.73
Nicotine (mg/day)	0.0	3.4	23.6 ^{d†}	2.8	4.0	1.8
Marihuana (joints/week)	1.2	0.75	0.92	.05	2.5	17.0 ^{d†}
Alcohol (oz AA/day)	.18	.15	.11	.14	.25	.14

*Education coded as: 1=did not finish high school; 2=graduated high school; 3=graduated college or university; 4=post-graduate degree.

Annual Income, Mother's Age and Average Parent Education are based on figures obtained at birth.

Nicotine and Marihuana were log transformed prior to statistical analyses; Alcohol was normalized using inverse transformation prior to statistical analyses.

Note: Statistical tests were one-way between-subjects ANOVAs with Bonferroni's criteria ($\alpha=.05$ over all post hoc comparisons) for continuous data and Chi-square analysis for frequency data. ^a $p \leq 0.10$, ^b $p \leq 0.05$, ^c $p \leq 0.01$, ^d $p \leq 0.001$.

†All pairwise comparisons differ. ‡ Heavy group differs from Control.

1988). Most demographic variables did not differ significantly across the drug groups, however, a few differences were apparent for the marihuana groups. Significant differences were observed between heavy users and controls on measures of mother's age and average parent education; in both cases, lower scores were reported for the heavy user group relative to controls.

Cigarettes

Omission Errors

All tests of significance for omission errors were evaluated using $\alpha=.05$ because of concerns regarding low power due to relatively small sample size.

Transition 1 (Age 6 to Age 10)

Group Differences in Stability. Significant overall differences across smoking groups in terms of stability for omission errors were observed at transition 1; Pearson $\chi^2(6, N=81) = 13.27, p<.05$. Pairwise testing of groups using chi-square analyses revealed significant differences between the heavy smoking group and the non-smoking group with respect to the percentage of cases who showed no change in quartile rank position (change score = 0) at transition 1; Pearson $\chi^2(1, N=50) = 9.22, p<.01$. In the heavy smoking group, 53% of cases showed no change in quartile rank position compared to 13% of cases in the non-smoking group, indicating significantly higher stability for the heavy smoking group between the ages of 6 and 10 for omission errors. Within the heavy smoking group, 67% of children who showed no change in quartile rank at transition 1 (comprising 32% of total cases in the heavy smoking group) had

quartile ranks equal to 4 at both assessments. No other group differences were observed with respect to stability at transition 1.

Stability of Attentional Performance Over Time. Group differences in stability for omission errors were reported between the ages of 6 and 10 (transition 1), therefore descriptive analyses for stability of attention are presented separately for each smoking group at this transition. Table 2 presents the percentage of cases at transition 1 at each change score (ranging from 0 to 3), for omission errors for each smoking group.

Direction of Performance. Chi-square analyses were performed to test for differences across groups in the percentage of cases that improved and/or declined in performance with respect to omission errors between the ages of 6 and 10. Table 2 presents the percentage of cases that improved and declined in performance for each change score for each smoking group. No significant differences were observed across smoking groups with respect to the percentage of cases that improved (Pearson $\chi^2(2, N=81) = 4.33, n.s.$), or showed a decline (Pearson $\chi^2(2, N=81) = 1.95, n.s.$) in attentional performance. Across all groups, 36% of cases improved and 36% of cases declined in performance at transition 1.

Level of Performance: Distribution of Good and Poor Performers. At age 6, group differences in terms of quartile rank were not observed; Pearson $\chi^2(6, N=81) = 10.32, n.s.$. A median test revealed no differences among smoking groups at age 6 with respect to the percentage of cases exceeding the overall median for the groups (grand median = 3); Pearson $\chi^2(2, N=81) = 5.86, n.s.$. At age 6, the distribution of good and poor performers for the sample was not significantly related to group membership.

Table 2

Cigarettes and Omission Errors:Percentage of Cases Within Change Scores at Transitions 1 and 2

	Transition 1 (6 to 10 years) Change Scores*				Transition 2 (10 to 13 years) Change Scores			
	0 (n/a)	1 (+/-)	2 (+/-)	3 (+/-)	0 (n/a)	1 (+/-)	2 (+/-)	3 (+/-)
Non-Smoking	12.9 (n/a)	51.6 (44/56)	22.6 (43/57)	12.9 (75/25)	32.3 (n/a)	32.3 (50/50)	2.3 (50/50)	3.2 (100/1)
Light-Moderate	29.0 (n/a)	45.2 (57/43)	22.6 (71/29)	3.2 (0/100)	29.0 (n/a)	48.4 (47/53)	16.1 (40/60)	6.5 (50/50)
Heavy	52.6 ^{a‡} (n/a)	21.1 (50/50)	26.3 (20/80)	0 (0/0)	42.1 (n/a)	26.3 (40/60)	21.1 (75/25)	10.5 (100/0)

*Change scores defined as: 0, stable performance; 1, moderate stability; 2, low stability; 3, unstable performance

(+/-) represent the percentage of cases that moved from a higher quartile rank to a lower quartile rank (+) or moved from a lower quartile rank to a higher quartile rank (-) within each change category, with high quartile ranks (i.e., 4) indicating poor performance and low quartile ranks (i.e., 1) indicating good performance with respect to omission errors

^ap 0.01; [‡] Heavy group differs from Non-smoking

No differences among groups were observed with respect to quartile ranks at age 10; Pearson $\chi^2(6, N=81) = 10.71$, *n.s.*. At age 10, a median test revealed significant overall differences among smoking groups with respect to median quartile rank for omission errors (grand median = 3); Pearson $\chi^2(2, N=81) = 6.95$, $p < .05$. Pairwise multiple comparisons using 2-group median tests revealed significant differences between the heavy smoking group and the non-smoking group (grand median = 3); Pearson $\chi^2(1, N=50) = 5.70$, $p < .05$, and between the heavy smoking group and the light-moderate group (grand median = 3); Pearson $\chi^2(1, N=50) = 4.40$, $p < .05$, with respect to median quartile rank. In both cases, the heavy smoking group had a significantly higher percentage of cases (47%) exceeding the grand median relative to the other two groups (16% in the non-smoking group, and 19% in the light-moderate group). These results indicate that a significantly higher percentage of poor performers were members of the heavy smoking group at age 10 compared to the non-smoking and light-moderate groups.

To test for differences among groups at transition 1, an average quartile rank was computed for each child (ranging from 1-4). At transition 1, a median test revealed significant overall differences among smoking groups with respect to median quartile rank for omission errors (grand median = 2.5); Pearson $\chi^2(2, N=81) = 9.55$, $p < .01$. Pairwise multiple comparisons revealed significant differences between the heavy smoking group and the non-smoking group (grand median = 2.5); Pearson $\chi^2(1, N=50) = 7.42$, $p < .01$, and between the heavy smoking group and the light-moderate group (grand median = 2.5); Pearson $\chi^2(1, N=50) = 7.42$, $p < .01$, with respect to median quartile rank. In both instances, the heavy smoking group had a significantly higher percentage of cases

(68%) exceeding the grand median relative to the other two groups (29% in both the non-smoking and light-moderate groups). Compared to the non-smoking and light-moderate groups, a significantly higher percentage of individuals in the heavy smoking group exhibited poorer average performance between the ages of 6 and 10 years.

Transition 2 (Age 10 to Age 13)

Group Differences in Stability. No significant differences across smoking groups in terms of stability were observed at transition 2; Pearson $\chi^2(6, N=81) = 5.28, n.s.$

Stability of Attentional Performance Over Time. Table 2 presents the percentage of cases at transition 2 at each change score (ranging from 0 to 3), for omission errors for each smoking group. Group differences in stability for omission errors were not observed between age 10 and age 13, therefore descriptive analyses looking at stability individually for each smoking group were not warranted. Descriptive analyses for stability for the entire sample, collapsing over groups are as follows: 33% of children between the ages of 10 and 13 exhibited highly stable performance, 37% exhibited moderate stability, 24% exhibited low stability, and 6% exhibited unstable performance at transition 2.

Direction of Performance. Table 2 presents the percentage of cases that improved and declined in performance for each change score for each smoking group at transition 2. No significant differences were observed across smoking groups with respect to the percentage of cases that improved (Pearson $\chi^2(2, N=81) = .13, n.s.$), or showed a decline (Pearson $\chi^2(2, N=81) = 1.69, n.s.$) in performance. Across all groups, 35% of cases improved and 32% of cases declined in performance at transition 2.

Level of Performance: Distribution of Good and Poor Performers. The results of chi-square analyses and median tests for groups at age 10 were reported in previous sections.

Significant overall differences in quartile rank at age 13 were observed among the smoking groups; Pearson $\chi^2(6, N=81) = 14.22, p<.05$. Pairwise testing of groups revealed significant differences between the moderate smoking group and the non-smoking group with respect to the distribution of quartile ranks at age 13; Pearson $\chi^2(3, N=62) = 10.48, p<.05$. A significantly higher percentage of cases in the moderate smoking group (48%) had quartile ranks equal to 2, compared to the non-smoking group (19%). As well, a significantly lower percentage of cases in the moderate smoking group (3%) had quartile ranks equal to 3, compared to the non-smoking group (26%). No other significant group differences in quartile rank were observed at age 13 for omission errors. Median tests revealed no group differences at age 13 with respect to the percentage of cases exceeding the overall median for the groups (grand median = 2); Pearson $\chi^2(2, N=81) = 3.12, n.s.$

A median test at transition 2 revealed significant overall differences among smoking groups with respect to median quartile rank for omission errors (grand median = 2.5); Pearson $\chi^2(2, N=81) = 10.55, p<.01$. Pairwise multiple comparisons revealed significant differences between the heavy smoking group and the non-smoking group (grand median = 2.5); Pearson $\chi^2(1, N=50) = 7.42, p<.01$, and between the heavy smoking group and the light-moderate group (grand median = 2.5); Pearson $\chi^2(1, N=50) = 8.78, p<.01$, with respect to median quartile rank. In both instances, the heavy smoking

group had a significantly higher percentage of cases (68%) exceeding the grand median relative to the other two groups (29% in the non-smoking group, and 26% in the light-moderate group), indicating that a higher percentage of individuals in the heavy smoking group exhibited poorer average performance relative to the other smoking groups between the ages of 10 and 13.

Overall Analyses (Age 6 to Age 13)

Group Differences in Stability. No significant differences across smoking groups in terms stability were observed from age 6 to age 13; Pearson $\chi^2(12, N=81) = 15.69, n.s.$

Stability of Attentional Performance Over Time. Group differences in stability for omission errors were not observed between age 6 and age 13, therefore descriptive analyses looking at stability for each smoking group individually were not warranted. Descriptive analyses for stability for the entire sample, collapsing over groups are as follows: from age 6 to age 13, 10% of children exhibited highly stable performance (change score = 0), 53% exhibited moderate stability (change score range = 1-2), 35% exhibited low stability (change score range = 3-4), and 2% exhibited unstable performance (change score range = 5-6).

Direction of Performance. To test for differences in direction of performance across groups from age 6 to age 13, an overall performance change score was computed for each participant based on a combination of direction of quartile rank change at transition 1 (improved, declined, no change) and similarly at transition 2 (improved, declined, no change). A chi-square analysis was used to test for differences among smoking groups on each of the nine combinations of performance change scores (e.g.,

improved transition 1-improved transition 2, declined transition 1-declined transition 2, etc.). No differences were observed with respect to direction of performance from age 6 to 13 for any of the groups, (Pearson $\chi^2(16, N=81) = 22.20, n.s.$), however, these results should be interpreted with caution because more than 25% of cells had a low expected count (<5).

Level of Performance: Distribution of Good and Poor Performers. The results of chi-square analyses and median tests for the individual age groups were reported in previous sections. To test for overall differences in the distribution of good and poor performers from age 6 to age 13, an average quartile rank across each of the three ages tested was computed for each child (ranging from 1-4). A median test revealed significant overall differences among smoking groups with respect to median quartile rank for omission errors (grand median = 2.33) overall; Pearson $\chi^2(2, N=81) = 9.69, p<.01$. Pairwise multiple comparisons using 2-group median tests revealed significant differences between the heavy smoking group and the non-smoking group (grand median = 2.67); Pearson $\chi^2(1, N=50) = 5.15, p<.05$, and between the heavy smoking group and the light-moderate group (grand median = 2.67); Pearson $\chi^2(1, N=50) = 9.44, p<.01$, with respect to median quartile rank. In both instances, the heavy smoking group had a significantly higher percentage of cases (79%) exceeding the grand median relative to the other two groups (42% in the non-smoking group, and 36% in the light-moderate group). Individuals in the heavy smoking group had a higher median quartile rank from age 6 to age 13, indicating poorer average performance for this group across all ages, compared to the other smoking groups.

Repeated Measures ANCOVA

The data for omission errors for smoking groups were analyzed with a 3 x 3 (Smoking Group x Age) mixed-design analysis of covariance, with age serving as the repeated measure. Two variables were identified as confounds for omission errors. Average parent education and proportion of postnatal passive nicotine exposure were significantly related to both maternal smoking and omission errors, and were therefore included as covariates with marijuana and alcohol in the repeated measures ANCOVA. The effects of smoking group and age were examined with all covariates included in the analyses. Table 3 presents standard scores taken from the ANCOVA analyses, for omission errors, both unadjusted and adjusted for covariates, for the three smoking groups.

No significant main effects were found for age $F(2,148) = .19, n.s.$, or smoking group $F(2,74) = .43, n.s.$. There was no significant age by smoking group interaction $F(4,148) = .53, n.s.$. The lack of a significant main effect of age is likely due to the use of standardized z-scores in the place of raw scores for omission errors to allow for comparisons between the GDS Vigilance Task and the Conners' CPT at the third assessment. The three smoking groups did not differ in their overall performance with respect to omission errors at any of the assessments.

Table 3

Omission Errors for Smoking Groups: Means for Raw and Adjusted Standard Scores

	Unadjusted [z-score (SD)]			Adjusted for Covariates [z-score (SE)]		
	None 0	Light-Mod >0 & <16	Heavy ≥16	None 0	Light-Mod >0 & <16	Heavy ≥16
N	31	31	19	31	31	19
GDS age 6 Omissions	-.11 (.78)	-.04 (1.01)	.24 (1.28)	.03 (.20)	-.03 (.18)	.01 (.27)
GDS age 10-11 Omissions	-.13 (.88)	-.19 (1.07)	.53 (.93)	.05 (.19)	-.22 (.17)	.26 (.26)
Conners' CPT Omissions	-.14 (.99)	.01 (.99)	.21 (1.04)	-.05 (.21)	.01 (.18)	.07 (.28)

SD = standard deviation; SE = standard error

Commission ErrorsTransition 1 (Age 6 to Age 10)

Group Differences in Stability. Using chi-square analysis, no significant differences across smoking groups in terms of stability for commission errors were observed at transition 1; Pearson $\chi^2(6, N=81) = 9.20, n.s.$

Stability of Attentional Performance Over Time. Table 4 presents the percentage of cases at transition 1 at each change score for commission errors for each smoking group. Group differences in stability were not observed between age 6 and age 10, therefore descriptive analyses looking at stability for each smoking group independently were not warranted. Descriptive analyses for stability for the entire sample, collapsing over groups are as follows: 37% exhibited highly stable performance, 40% exhibited moderate stability, 17% exhibited low stability, and 6% exhibited unstable performance at transition 1.

Direction of Performance. Table 4 presents the percentage of cases that improved and declined in performance for each change score for each smoking group at transition 1. No significant differences were observed across smoking groups with respect to the percentage of cases that improved (Pearson $\chi^2(2, N=81) = .42, n.s.$), or declined (Pearson $\chi^2(2, N=81) = 1.69, n.s.$) in attentional performance. Across all groups, 31% of cases improved and 32% of cases declined in performance at transition 1.

Level of Performance: Distribution of Good and Poor Performers. No significant group differences in quartile rank were revealed at age 6 for commission errors; Pearson $\chi^2(6, N=81) = 12.52, n.s.$ Median tests looking at each age group individually revealed

Table 4

Cigarettes and Commission Errors:Percentage of Cases Within Change Scores at Transitions 1 and 2

	Transition 1 (6 to 10 years) Change Scores*				Transition 2 (10 to 13 years) Change Scores			
	0 (n/a)	1 (+/-)	2 (+/-)	3 (+/-)	0 (n/a)	1 (+/-)	2 (+/-)	3 (+/-)
Non-Smoking	32.3 (n/a)	45.2 (43/57)	22.6 (43/57)	0 (0/0)	35.5 (n/a)	35.5 (64/36)	25.8 (50/50)	3.2 (0/100)
Light-Moderate	38.7 (n/a)	41.9 (46/54)	6.5 (50/50)	12.9 (50/50)	41.9 (n/a)	22.6 (29/71)	29.0 (56/44)	6.5 (0/100)
Heavy	42.1 (n/a)	26.3 (60/40)	26.3 (60/40)	5.3 (100/0)	31.6 (n/a)	31.6 (67/33)	31.6 (67/33)	5.3 (0/100)

*Change scores defined as: 0, stable performance; 1, moderate stability; 2, low stability; 3, unstable performance

(+/-) represent the percentage of cases that moved from a higher quartile rank to a lower quartile rank (+) or moved from a lower quartile rank to a higher quartile rank (-) within each change category, with high quartile ranks (i.e., 4) indicating poor performance and low quartile ranks (i.e., 1) indicating good performance with respect to commission errors

significant overall differences at age 6 with respect to median quartile rank for commission errors (grand median = 3); Pearson $\chi^2(2, N=81) = 10.77, p<.01$. Pairwise multiple comparisons revealed significant differences between the heavy smoking group and the non-smoking group (grand median = 3); Pearson $\chi^2(1, N=50) = 9.22, p<.01$, and between the heavy smoking group and the light-moderate group (grand median = 3); Pearson $\chi^2(1, N=50) = 5.96, p<.05$, with respect to median quartile rank. In both instances, the heavy smoking group had a significantly higher percentage of cases (53%) exceeding the grand median relative to the other two groups (13% in the non-smoking group; 19% in the light-moderate group). A significantly higher percentage of individuals exhibiting poor performance at age 6 were members of the heavy smoking group than were members of either the non-smoking or light-moderate group.

At age 10, chi-square analyses revealed no differences among groups in terms of quartile ranks; Pearson $\chi^2(6, N=81) = 3.94, n.s.$. No differences were observed at age 10 with respect to the percentage of cases exceeding the overall median for groups (grand median = 3); Pearson $\chi^2(2, N=81) = 2.06, n.s.$. At age 10, the distribution of good and poor performers for the sample was not significantly related to group membership.

No significant differences were observed among the smoking groups in the percentage of cases exceeding the overall median for the groups at transition 1 (grand median = 2.5); Pearson $\chi^2(2, N=81) = 4.09, n.s.$. Based on average quartile ranks obtained between the ages of 6 and 10, the distribution of good and poor performers for the sample was not significantly related to group membership.

Transition 2 (Age 10 to Age 13)

Group Differences in Stability. No significant differences across smoking groups in terms of stability for commission errors were observed at transition 2; Pearson $\chi^2(6, N=81) = 1.75, n.s.$

Stability of Attentional Performance Over Time. Table 4 presents the percentage of cases at transition 2 at each change score for commission errors for each smoking group. Group differences in stability were not observed between age 10 and age 13, therefore descriptive analyses looking at stability for each smoking group independently were not warranted. Descriptive analyses for stability for the entire sample, collapsing over groups, are as follows: 37% of children exhibited highly stable performance, 30% exhibited moderate stability, 28% exhibited low stability, and 5% exhibited unstable performance at transition 2.

Direction of Performance. Table 4 presents the percentage of cases that improved and declined in performance for each change score for each smoking group at transition 2. No significant differences were observed across smoking groups with respect to the percentage of cases that improved (Pearson $\chi^2(2, N=81) = 2.32, n.s.$), or declined (Pearson $\chi^2(2, N=81) = .54, n.s.$) in attentional performance. Across all groups, 32% of cases improved and 31% of cases declined in performance at transition 2.

Level of Performance: Distribution of Good and Poor Performers. The results of chi-square analyses and median tests for groups at age 10 were reported in previous sections.

No significant differences among groups were reported with respect to quartile rank at age 13; Pearson $\chi^2(6, N=81) = .74, n.s.$ No differences were observed at age 13

with respect to the percentage of cases exceeding the overall median for smoking groups (grand median = 3); Pearson $\chi^2(2, N=81) = .00, n.s.$. At both age 10 and age 13, the distribution of good and poor performers for the sample was not significantly related to group membership.

At transition 2, no significant differences were observed among the smoking groups with respect to the percentage of cases exceeding the overall median for the groups (grand median = 2.5); Pearson $\chi^2(2, N=81) = .27, n.s.$. At transition 2, the distribution of good and poor performers for the sample was not significantly related to group membership.

Overall Analyses (Age 6 to Age 13)

Group Differences in Stability. No significant differences across smoking groups in terms stability were observed from age 6 to age 13; Pearson $\chi^2(12, N=81) = 13.39, n.s.$.

Stability of Attentional Performance Over Time. Group differences in stability for commission errors were not observed between age 6 and age 13, therefore descriptive analyses looking at stability for each smoking group independently were not warranted. Descriptive analyses for stability for the entire sample, collapsing over groups, are as follows: 15% of children exhibited highly stable performance (change score = 0), 51% exhibited moderate stability (change score range = 1-2), 31% exhibited low stability (change score range = 3-4), and 4% exhibited unstable performance (change score range = 5-6) from age 6 to age 13.

Direction of Performance. No differences were observed with respect to direction of performance from age 6 to 13 for any of the groups (Pearson $\chi^2(16, N=81) =$

17.56, *n.s.*), however, these results should be interpreted with caution because more than 25% of cells had a low expected count (<5).

Level of Performance: Distribution of Good and Poor Performers. The results of chi-square analyses and median tests for the individual age groups were reported in previous sections. Over all ages, no significant differences were observed among the smoking groups in the percentage of cases exceeding the overall median for groups (grand median = 7); Pearson $\chi^2(2, N=81) = 1.27, n.s.$. Across all ages, the distribution of good and poor performers for the sample was not significantly related to group membership.

Repeated Measures ANCOVA

As was the case for omission errors, the data for commission errors were analyzed with a 3 x 3 (Smoking Group x Age) mixed-design analysis of covariance, with age serving as the repeated measure. For these analyses, prenatal marijuana and alcohol consumption were used as covariates in analyses carried out on commission errors across the three smoking groups. Table 5 presents standard scores taken from the ANCOVA analyses, for commission errors, both unadjusted and adjusted for covariates, for the three smoking groups.

No significant main effect of age $F(2,152) = .15, n.s.$, or smoking group $F(2,76) = .43, n.s.$, was observed. There was no significant age by smoking group interaction $F(4,152) = .53, n.s.$. These results indicate that the three groups did not differ significantly in terms of their overall performance with respect to commission errors over the three test sessions.

Table 5
Commission Errors for Smoking Groups: Means for Raw and Adjusted Standard Scores

	Unadjusted [z-score (SD)]			Adjusted for Covariates [z-score (SE)]		
	None 0	Light-Mod >0 &<16	Heavy ≥16	None 0	Light-Mod >0 &<16	Heavy ≥16
N	31	31	19	31	31	19
GDS age 6 Commissions	-.02 (.76)	-.15 (1.00)	.26 (1.31)	-.00 (.18)	-.14 (.18)	.23 (.23)
GDS age 10-11 Commissions	.00 (1.09)	-.14 (.92)	.22 (.99)	.03 (.18)	-.14 (.18)	.17 (.23)
Conners' CPT Commissions	-.04 (1.05)	.06 (1.02)	-.03 (.92)	-.02 (.18)	.05 (.18)	-.05 (.24)

SD = standard deviation; SE = standard error

CPRS-48.

Maternal ratings of impulsive/hyperactive behaviour were analyzed with a 3 x 3 (Smoking Group x Age) mixed-design analysis of covariance, with age serving as the repeated measure. Covariates included in the analyses for the CPRS-48 and smoking were prenatal marihuana and alcohol consumption. Because sphericity was not assumed for these analyses, degrees of freedom were adjusted using Huynh-Feldt correction (Stevens, 1996). Table 6 presents standard scores taken from the ANCOVA analyses, for the CPRS-48, both unadjusted and adjusted for covariates, for the three smoking groups.

No significant main effects were found for age $F(1.961,134) = .01, n.s.$, or smoking group $F(2,67) = .40, n.s.$. There was no significant age by smoking group interaction $F(3.921,134) = .29, n.s.$. Maternal ratings of children's behaviour as impulsive/hyperactive did not differ significantly across the three assessments or the smoking groups.

Marihuana**Omission Errors**

All tests of significance were evaluated using $\alpha=.05$ because of concerns regarding low power due to relatively small sample size.

Transition 1 (Age 6 to Age 10)

Group Differences in Stability. No significant differences across marihuana groups in terms of stability were observed at transition 1; Pearson $\chi^2(6, N=81) = 6.01, n.s.$

Table 6

CPRS-48 Scores* for Smoking Groups: Means for Raw and Adjusted Scores

	Unadjusted (SD)			Adjusted for Covariates (SE)		
	None 0	Light-Mod >0 & <16	Heavy ≥16	None 0	Light-Mod >0 & <16	Heavy ≥16
N	31	31	19	31	31	19
6 years	.99 (.71)	.96 (.55)	.92 (.51)	.99 (.11)	.99 (.11)	.89 (.15)
10-11 years	.94 (.59)	.86 (.69)	.80 (.69)	.94 (.12)	.88 (.12)	.76 (.16)
13-15 years	.75 (.61)	.84 (.67)	.72 (.39)	.74 (.11)	.86 (.11)	.69 (.15)

*Note: Scores for the CPRS-48 are presented in original units, not standard scores

SD = standard deviation; SE = standard error

Stability of Attentional Performance Over Time. Table 7 presents the percentage of cases at each change score for omission errors for each marihuana group at transition 1. Group differences in stability were not observed between age 6 and age 10, therefore descriptive analyses looking at stability for each marihuana group independently were not warranted. Descriptive analyses for stability for the entire sample, collapsing over groups, are as follows: 28% of children exhibited highly stable performance, 42% exhibited moderate stability, 24% exhibited low stability, and 6% exhibited unstable performance at transition 1.

Direction of Performance. Table 7 presents the percentage of cases that improved and declined in performance for each change score for each marihuana group. No significant differences were observed across groups with respect to the percentage of cases that improved (Pearson $\chi^2(2, N=81) = 3.17, n.s.$) at transition 1. In the no/infrequent use group, 30% of cases improved, compared to 46% in the moderate group and 54% in the heavy marihuana group.

Significant overall differences across groups were observed in the percentage of cases that declined in performance at transition 1 (Pearson $\chi^2(2, N=81) = 8.16, p<.05$). Pairwise testing of groups revealed significant differences between the moderate marihuana group and the no/infrequent use group; Pearson $\chi^2(1, N=68) = 5.14, p<.05$, and the heavy marihuana group and the no/infrequent use group; Pearson $\chi^2(1, N=70) = 4.03, p<.05$. A significantly lower percentage of cases in both the moderate group (10%) and the heavy group (15%) declined in performance between the ages of 6 and 10, compared to the percentage of children that declined in the no/infrequent use group

Table 7

Marihuana and Omission Errors:**Percentage of Cases Within Change Scores at Transitions 1 and 2**

	Transition 1 (6 to 10 years)				Transition 2 (10 to 13 years)			
	Change Scores*				Change Scores			
	0 (n/a)	1 (+/-)	2 (+/-)	3 (+/-)	0 (n/a)	1 (+/-)	2 (+/-)	3 (+/-)
No/Infrequent Use	24.6 (n/a)	47.4 (40/60)	22.8 (31/69)	5.3 (66/33)	29.8 (n/a)	35.1 (40/60)	28.1 (63/37)	7.0 (75/25)
Moderate Use	45.5 (n/a)	36.4 (100/0)	18.2 (50/50)	0 (0/0)	27.3 (n/a)	63.6 (57/43)	0 (0/0)	9.1 (100/0)
Heavy Use	30.8 (n/a)	23.1 (67/33)	30.8 (100/0)	15.4 (50/50)	53.8 (n/a)	23.1 (67/33)	23.1 (0/100)	0 (0/0)

*Change scores defined as: 0, stable performance; 1, moderate stability; 2, low stability; 3, unstable performance

(+/-) represent the percentage of cases that moved from a higher quartile rank to a lower quartile rank (+) or moved from a lower quartile rank to a higher quartile rank (-) within each change category, with high quartile ranks (i.e., 4) indicating poor performance and low quartile ranks (i.e., 1) indicating good performance with respect to omission errors

(46%). No significant differences were observed between the moderate and the heavy group with respect to the percentage of cases that declined in performance; Pearson $\chi^2(1, N=24) = .22, n.s.$. Closer examination of these results using a median test revealed significant overall differences across groups in the percentage of cases that exceeded the overall median for groups (grand median = 3) at age 6; Pearson $\chi^2(2, N=81) = 6.79, p<.05$. At age 6, 55% of cases in the moderate group had quartile ranks equal to 4, and in the heavy group, 39% of children had quartile ranks equal to 4 at age 6. In the no/infrequent use group, only 19% of children had quartile ranks of 4 at age 6 years. The high number of cases with quartile ranks equal to 4 (poor performance) at age 6 in the moderate and heavy marihuana groups restricted the number of children in these groups whose quartile rank scores at age 10 were able to reflect a decline in performance from age 6. This may account for why significantly fewer cases in both these groups showed a decline in performance between the ages of 6 and 10 years.

Level of Performance: Distribution of Good and Poor Performers. No differences were observed across the marihuana groups in terms of quartile rank at age 6; Pearson $\chi^2(6, N=81) = 8.82, n.s.$. As discussed previously, a median test revealed significant overall differences among marihuana groups at age 6 with respect to median quartile rank for omission errors (grand median = 3); Pearson $\chi^2(2, N=81) = 6.79, p<.05$. Pairwise multiple comparisons revealed significant differences between the moderate marihuana group and the no/infrequent use group (grand median = 2.5); Pearson $\chi^2(1, N=68) = 5.31, p<.05$. The moderate marihuana group had a significantly higher percentage of cases (55%) exceeding the median relative to the no/infrequent use group (19%), indicating

that at age 6, with respect to omission errors, a significantly higher percentage of poor performers were members of the moderate marihuana group at age 6 compared to the no/infrequent use group.

No differences among groups were observed with respect to quartile ranks at age 10; Pearson $\chi^2(6, N=81) = 6.12, n.s.$ No differences were observed at age 10 with respect to the percentage of cases exceeding the overall median for the groups (grand median = 3); Pearson $\chi^2(2, N=81) = .06, n.s.$ At age 10, the distribution of good and poor performers for the sample was not significantly related to group membership.

At transition 1, no significant differences were observed among the marihuana groups in the percentage of cases exceeding the overall median for groups (grand median = 2.5); Pearson $\chi^2(2, N=81) = 3.58, n.s.$ At transition 1, the distribution of good and poor performers for the sample was not significantly related to group membership.

Transition 2 (Age 10 to Age 13)

Group Differences in Stability. No significant differences across marihuana groups in terms of stability were observed at transition 2; Pearson $\chi^2(6, N=81) = 8.94, n.s.$

Stability of Attentional Performance Over Time. Table 7 presents the percentage of cases at each change score for omission errors for each marihuana group. Group differences in stability were not observed between age 10 and age 13, therefore descriptive analyses looking at stability individually for each marihuana group were not warranted. Descriptive analyses for stability were reported for the entire sample, collapsing over drug groups. Stability analyses at transition 2 for the marihuana groups

are the same as previously reported for omission errors across smoking groups at transition 2.

Direction of Performance. Table 7 presents the percentage of cases that improved and declined in performance for each change score for each marihuana group at transition 2. No significant differences were observed across groups with respect to the percentage of cases that improved (Pearson $\chi^2(2, N=81) = 2.82, n.s.$), or declined (Pearson $\chi^2(2, N=81) = .17, n.s.$) in attentional performance. Across all groups, 35% of cases improved and 32% of cases declined in performance at transition 2.

Level of Performance: Distribution of Good and Poor Performers. The results of chi-square analyses and median tests for groups at age 10 were reported in previous sections.

No significant group differences in quartile rank were observed at age 13 among the marihuana groups; Pearson $\chi^2(6, N=81) = 1.79, n.s.$. No differences were observed at age 13 with respect to the percentage of cases exceeding the overall median for groups (grand median = 2); Pearson $\chi^2(2, N=81) = .25, n.s.$. At age 13, the distribution of good and poor performers for the sample was not significantly related to group membership.

At transition 2, no significant differences were observed among the marihuana groups in the percentage of cases exceeding the overall median for groups (grand median = 2.5); Pearson $\chi^2(2, N=81) = .01, n.s.$. At transition 2, the distribution of good and poor performers for the sample was not significantly related to group membership.

Overall Analyses (Age 6 to Age 10)

Group Differences in Stability. No significant differences across marihuana groups in terms stability were observed from age 6 to age 13; Pearson $\chi^2(12, N=81) = 5.41, n.s.$

Stability of Attentional Performance Over Time. Group differences in stability for omission errors were not observed between age 6 and age 13, therefore descriptive analyses looking at stability for each marihuana group individually were not warranted. Descriptive analyses for stability were reported for the entire sample, collapsing over drug groups. Overall stability analyses for the marihuana groups are the same as previously reported for smoking groups for omission errors.

Direction of Performance. No differences were observed with respect to direction of performance from age 6 to 13 for any of the groups (Pearson $\chi^2(16, N=81) = 19.08, n.s.$), however, these results should be interpreted with caution because more than 25% of cells had a low expected count (<5).

Level of Performance: Distribution of Good and Poor Performers. The results of chi-square analyses and median tests for the individual age groups were reported in previous sections. No significant differences were observed among the marihuana groups in the percentage of cases exceeding the overall median for groups (grand median = 2.33); Pearson $\chi^2(2, N=81) = 1.65, n.s.$ Across all ages, the distribution of good and poor performers for the sample was not significantly related to group membership.

Repeated Measures ANCOVA

As was the case for cigarettes, the data for omission errors for marihuana groups were analyzed with a 3 x 3 (Marihuana Group x Age) mixed-design analysis of

covariance, with age serving as the repeated measure. For these analyses, one variable was identified as a confound for omission errors. Average parent education was significantly related to both maternal marihuana use and omission errors, and was therefore included as a covariate with nicotine and alcohol in the repeated measures ANCOVA. The effects of age and marihuana group were examined with all covariates included in the analyses. Table 8 presents standard scores taken from the ANCOVA analyses, for omission errors, both unadjusted and adjusted for covariates, for the three marihuana groups.

No significant main effects were found for age $F(2,150) = .14, n.s.$, or marihuana group $F(2,75) = .84, n.s.$. There was no significant age by marihuana group interaction $F(4,150) = 1.78, n.s.$. As was the case with the smoking analyses, the lack of a significant main effect of age is likely due to the use of standardized z-scores to allow for comparisons between the GDS Vigilance Task and the Conners' CPT at the third assessment. The three marihuana groups did not differ in their overall performance with respect to omission errors at any of the assessments.

Commission Errors

Transition 1 (Age 6 to Age 10)

Group Differences in Stability. No significant differences across marihuana groups in terms of stability were observed at transition 1; Pearson $\chi^2(6, N=81) = 6.40, n.s.$

Stability of Attentional Performance Over Time. Table 9 presents the percentage of cases at each change score for commission errors for each marihuana group at

Table 8

Omission Errors for Marihuana Groups: Means for Raw and Adjusted Standard Scores

	Unadjusted [z-score (SD)]			Adjusted for Covariates [z-score (SE)]		
	No/Infrequent ≤1	Moderate >1 & <6	Heavy ≥6	No/Infrequent ≤1	Moderate >1 & <6	Heavy ≥6
N	57	11	13	57	11	13
GDS age 6 Omissions	-.10 (.94)	.15 (1.27)	.31 (1.02)	-.08 (.14)	.20 (.31)	.17 (.30)
GDS age 10-11 Omissions	.03 (.99)	.32 (1.03)	-.40 (.95)	.08 (.13)	.38 (.30)	-.66 (.29)
Conners' CPT Omissions	.02 (1.06)	.00 (.58)	-.07 (1.08)	.04 (.14)	-.05 (.32)	-.13 (.31)

SD = standard deviation; SE = standard error

Table 9

Marihuana and Commission Errors:**Percentage of Cases Within Change Scores at Transitions 1 and 2**

	Transition 1 (6 to 10 years)				Transition 2 (10 to 13 years)			
	Change Scores*				Change Scores			
	0 (n/a)	1 (+/-)	2 (+/-)	3 (+/-)	0 (n/a)	1 (+/-)	2 (+/-)	3 (+/-)
No/Infrequent Use	6.8 (n/a)	33.3 (47/53)	22.8 (46/54)	7.0 (50/50)	40.4 (n/a)	28.1 (50/50)	26.3 (53/47)	5.3 (0/100)
Moderate Use	36.4 (n/a)	54.5 (50/50)	9.1 (100/0)	0 (0/0)	18.2 (n/a)	36.4 (50/50)	36.4 (75/25)	9.1 (0/100)
Heavy Use	38.5 (n/a)	53.8 (43/57)	0 (0/0)	7.7 (100/0)	38.5 (n/a)	30.8 (75/25)	30.8 (50/50)	0 (0/0)

*Change scores defined as: 0, stable performance; 1, moderate stability; 2, low stability; 3, unstable performance

(+/-) represent the percentage of cases that moved from a higher quartile rank to a lower quartile rank (+) or moved from a lower quartile rank to a higher quartile rank (-) within each change category, with high quartile ranks (i.e., 4) indicating poor performance and low quartile ranks (i.e., 1) indicating good performance with respect to commission errors

transition 1. Group differences in stability were not observed between age 6 and age 10, therefore descriptive analyses looking at stability for each marihuana group independently were not warranted. Descriptive analyses for stability were reported for the entire sample, collapsing over drug groups. Stability analyses at transition 1 for the marihuana groups are the same as previously reported for commission errors across smoking groups at transition 1.

Direction of Performance. Table 9 presents the percentage of cases that improved and declined in performance for each change score for each marihuana group at transition 1. No significant differences were observed across groups with respect to the percentage of cases that improved (Pearson $\chi^2(2, N=81) = .19, n.s.$), or declined (Pearson $\chi^2(2, N=81) = .17, n.s.$) in attentional performance. Across all groups, 31% of cases improved and 32% of cases declined in performance at transition 1.

Level of Performance: Distribution of Good and Poor Performers. Chi-square analyses looking at group differences in quartile rank at age 6 revealed no differences among groups; Pearson $\chi^2(6, N=81) = 6.89, n.s.$. Median tests looking at each age group individually revealed no significant differences at age 6 with respect to the percentage of cases exceeding the overall median for groups (grand median = 3); Pearson $\chi^2(2, N=81) = 4.15, n.s.$.

At age 10, no significant group differences in quartile ranks were observed; Pearson $\chi^2(6, N=81) = 3.12, n.s.$. A median test revealed no differences at age 10 with respect to the percentage of cases exceeding the overall median for groups (grand median

= 3); Pearson $\chi^2(2, N=81) = .41, n.s.$. At both age 6 and age 10, the distribution of good and poor performers for the sample was not significantly related to group membership.

At transition 1, no significant differences were observed among the marihuana groups in the percentage of cases exceeding the overall median for groups (grand median = 2.5); Pearson $\chi^2(2, N=81) = 3.19, n.s.$. The distribution of good and poor performers for the sample was not significantly related to group membership.

Transition 2 (Age 10 to Age 13)

Group Differences in Stability. No significant differences across marihuana groups in terms of stability were observed at transition 2; Pearson $\chi^2(6, N=81) = 3.63, n.s.$.

Stability of Attentional Performance Over Time. Table 9 presents the percentage of cases at each change score for commission errors for each marihuana group. Group differences in stability for commission errors were not observed between age 10 and age 13, therefore descriptive analyses looking at stability for each marihuana group independently were not warranted. Descriptive analyses for stability were reported for the entire sample, collapsing over groups. Stability analyses at transition 2 for the marihuana groups are the same as previously reported for commission errors across smoking groups for transition 2.

Direction of Performance. Table 9 presents the percentage of cases that improved and declined in performance for each change score for each marihuana group at transition 2. No significant differences were observed across groups with respect to the percentage of cases that improved (Pearson $\chi^2(2, N=81) = 1.57, n.s.$), or declined (Pearson $\chi^2(2,$

$N=81$) = .54, *n.s.*) in attentional performance. Across all groups, 32% of cases improved and 31% of cases declined in performance at transition 2.

Level of Performance: Distribution of Good and Poor Performers. The results of chi-square analyses and median tests for groups at age 10 were reported in previous sections.

Chi-square analyses looking at group differences in quartile rank at age 13 revealed no significant differences among groups; Pearson $\chi^2(6, N=81) = 1.92$, *n.s.*. Using a median test, no differences were observed at age 13 with respect to the percentage of cases exceeding the overall median for groups (grand median = 3); Pearson $\chi^2(2, N=81) = .07$, *n.s.*. At both age 10 and age 13, the distribution of good and poor performers for the sample was not significantly related to group membership.

At transition 2, no significant differences were observed among the marihuana groups in the percentage of cases exceeding the overall median for groups (grand median = 2.5); Pearson $\chi^2(2, N=81) = 2.45$, *n.s.*. At transition 2, the distribution of the good and poor performers for the sample was not significantly related to group membership.

Overall Analyses (Age 6 to Age 13)

Group Differences in Stability. No significant differences across marihuana groups in terms stability were observed from age 6 to age 13; Pearson $\chi^2(12, N=81) = 10.92$, *n.s.*.

Stability of Attentional Performance Over Time. Group differences in stability for commission errors were not observed between age 6 and age 13, therefore descriptive analyses looking at stability for each marihuana group independently were not warranted.

Descriptive analyses for stability were reported for the entire sample, collapsing over groups. Overall stability analyses for the marihuana groups are the same as previously reported for commission errors across smoking groups from age 6 to 13.

Direction of Performance. No differences were observed for any of the groups with respect to direction of performance from age 6 to 13, (Pearson $\chi^2(16, N=81) = 14.21, n.s.$), however, these results should be interpreted with caution because more than 25% of cells had a low expected count (<5).

Level of Performance: Distribution of Good and Poor Performers. No significant differences were observed among the marihuana groups in the percentage of cases exceeding the overall median for groups (grand median = 7); Pearson $\chi^2(2, N=81) = 1.90, n.s.$ Across all ages, the distribution of good and poor performers for the sample was not significantly related to group membership.

Repeated Measures ANCOVA

The data for commission errors were analyzed with a 3 x 3 (Marihuana Group x Age) mixed-design analysis of covariance, with age serving as the repeated measure. For these analyses, prenatal nicotine and alcohol consumption were used as covariates in analyses carried out on commission errors across the three marihuana groups. Table 10 presents standard scores taken from the ANCOVA analyses for commission errors, both unadjusted and adjusted for covariates, for the three marihuana groups.

No significant main effect of age $F(2,152) = .41, n.s.$, or marihuana group $F(2,76) = .46, n.s.$, was observed. There was no significant age by marihuana group interaction $F(4,152) = 1.27, n.s.$ These results indicate that the three groups did not differ

Table 10
Commission Errors for Marihuana Groups: Means for Raw and Adjusted Standard Scores

	Unadjusted [z-score (SD)]			Adjusted for Covariates [z-score (SE)]		
	No/Infrequent ≤1	Moderate >1 &<6	Heavy ≥6	No/Infrequent ≤1	Moderate >1 &<6	Heavy ≥6
N	57	11	13	57	11	13
GDS age 6 Commissions	-.05 (1.03)	.26 (.83)	-.01 (1.05)	-.06 (.13)	.33 (.31)	.01 (.28)
GDS age 10-11 Commissions	.00 (1.01)	.29 (.82)	-.25 (1.11)	-.02 (.13)	.42 (.31)	-.28 (.28)
Conners' CPT Commissions	.08 (1.02)	-.30 (.69)	-.10 (1.12)	.07 (.14)	-.26 (.32)	-.11 (.28)

SD = standard deviation; SE = standard error

significantly in terms of their overall performance with respect to commission errors over the three test sessions.

CPRS-48

Maternal ratings of impulsive/hyperactive behaviour for marihuana groups were analyzed with a 3 x 3 (Marihuana Group x Age) mixed-design analysis of covariance, with age serving as the repeated measure. Covariates included in analyses for the CPRS-48 and marihuana were annual family income, and prenatal nicotine and alcohol consumption. Table 11 presents standard scores taken from the ANCOVA analyses, for the CPRS-48, both unadjusted and adjusted for covariates, for the three marihuana groups.

No significant main effect was revealed for age $F(2,132) = .15, n.s.$, or marihuana group $F(2,66) = 2.85, n.s.$. There was no significant age by marihuana group interaction $F(4,132) = .01, n.s.$. Maternal ratings of children's behaviour as impulsive/hyperactive did not differ significantly across the three assessments or the marihuana groups.

DISCUSSION

The present longitudinal study, spanning development from age 6 to early adolescence, was undertaken to examine aspects of attentional functioning in children who were prenatally exposed to cigarettes and marihuana. The study focused on sustained attention and impulse control, both of which have been identified as problem areas for prenatally drug exposed children in the OPPS sample. Specifically, the aim was to examine the degree to which children exhibited stability of attentional performance over time, and to determine whether differences in stability were present across drug

Table 11

CPRS-48 Scores* for Marihuana Groups: Means for Raw and Adjusted Scores

	Unadjusted (SD)			Adjusted for Covariates (SE)		
	No/Infrequent ≤1	Moderate >1 & <6	Heavy ≥6	No/Infrequent ≤1	Moderate >1 & <6	Heavy ≥6
N	57	11	13	57	11	13
6 years	.90 (.59)	.90 (.39)	1.29 (.74)	.89 (.08)	1.02 (.21)	1.25 (.17)
10-11 years	.82 (.60)	.78 (.58)	1.19 (.79)	.81 (.09)	.90 (.22)	1.16 (.19)
13-15 years	.73 (.54)	.67 (.48)	1.02 (.83)	.69 (.08)	.82 (.19)	1.06 (.16)

*Note: Scores for the CPRS-48 are presented in original units, not standard scores

SD = standard deviation; SE = standard error

groups. An additional goal was to look at aspects of attentional performance across drug groups relating to direction of performance (i.e., improve or decline) over time, and the distribution of good and poor performers across groups. Also of interest was to determine whether the drug groups differed with respect to performance after controlling for the effects of other influences that may impact on attentional behaviour. Sustained attention and impulse control were assessed using two versions of a continuous performance task, the GDS Vigilance task and the Conners' CPT.

Results from the present study suggest differential effects for prenatal exposure to cigarettes and marihuana with respect to sustained attention and impulse control. Such findings are consistent with multidimensional models of attention (Barkley, 1997; Mirsky et al., 1991; Mirsky et al., 1995) that view sustained attention and impulse control as elements of an attentional system that function within a larger cognitive framework.

The principal findings related to cigarettes suggest negative associations with impulse control at age 6, and sustained attention between the ages of 10 and 11 years. With respect to stability of attentional performance, from age 6 to age 13, the majority of children across all three smoking groups displayed moderate to high stability, with change scores ranging from zero to two for both sustained attention and impulse control. Only a small number of children were unstable across all ages, from 6 to 13 years. Between age 6 and 10 years, children born to heavy smokers showed higher stability of performance on tests assessing sustained attention compared to children whose mothers did not smoke cigarettes during pregnancy. In terms of level of performance observed across groups, at age 6, prenatal exposure to cigarettes was associated with higher

commission errors, and at age 10, with higher omission errors in children born to heavy smokers relative to children in the non-smoking and light-moderate smoking groups. No systematic trends towards improvement or decline in performance were observed across groups in terms of the ability to sustain attention or inhibit impulsive responding.

Unlike prenatal exposure to cigarettes which showed links to both sustained attention and impulse control, an association between attentional functioning and prenatal exposure to marihuana was found only for sustained attention at age 6. As was the case with cigarettes, the majority of children displayed moderate to high stability from age 6 to age 13 with respect to performance on tests measuring sustained attention and impulse control. Stability figures were the same for both cigarettes and marihuana for impulse control. In terms of level of performance, at age 6, prenatal exposure to marihuana was associated with higher errors of omission for children born to moderate and heavy users of marihuana. Prenatal use of marihuana was not related to problems with impulse control for any group, across the ages tested.

Cigarettes

Sustained Attention. In the present study, performance on tests assessing sustained attention differentiated between children born to heavy smokers and the rest of the sample at age 10. The heavy smoking group contained a significantly higher percentage of poor performers compared to the other smoking groups, and over one-third of the children in the heavy smoking group had omission error scores in the 4th quartile at both age 6 and age 10. This level of poor performance resulted in higher stability between ages 6 and 10 for the heavy smoking group relative to the non-smoking group,

and is possibly reflective of differences in maturational rate across groups with respect to sustained attention.

Researchers looking at the normative development of attentional processes have found that the ability to sustain attention does not develop in normal children until approximately 8-10 years of age, when cognitive processes governing this aspect of attention reach maturity (Corkum & Siegel, 1993; Greenberg & Waldman, 1993; Rebok et al., 1997). Based on the findings of normative studies, it is reasonable to posit that the ability to sustain attention had begun to develop in children in the non-smoking group by the second assessment at age 10-11. Poorer attentional performance for the heavy smoking group at this age may indicate that for these children, maturation of cognitive systems responsible for sustained attention developed later than non-exposed children, resulting in a developmental delay with respect to the ability to sustain attention.

By age 13, differences in performance that were observed across the groups at age 10 were no longer significant, suggesting that if the ability to sustain attention develops later in the children of heavy smokers, these maturational differences may have resolved by age 13.

The present study is the first to date to look at the relationship between prenatal exposure to cigarettes and attentional functioning in children between the ages of 10 and 11 years. These findings suggest that differences observed across smoking groups at age 10 do not represent permanent deficits for the children of heavy smokers. Rather, the picture appears to be consistent with a delay of maturation of function that was overcome

in time, given that the groups do not differ with respect performance on tests of sustained attention at age 13.

Impulse Control. At age 6, the performance of children born to heavy smokers was reflective of higher commission errors, compared to the performance of children born to non-smokers and light-moderate smokers. These findings replicate results reported in previous studies using the OPPS sample (Kristjansson et al., 1989; Fried et al., 1992). Work with other samples (Streissguth et al., 1984) has revealed similar findings that suggest an association between prenatal exposure to cigarettes and impulse control in young children between the ages of 4 to 7 years.

Impulse control begins to mature in early childhood, between the ages of 6 and 8 years, with development continuing throughout middle and late childhood (Olson et al., 1999; Levin et al., 1991; Greenberg & Waldman, 1993). As with sustained attention, considered within the context of normative development of impulse control, poorer performance observed at age 6 for children born to heavy smokers may indicate a delay in onset of maturation of cognitive systems governing the ability to inhibit impulsive responding.

Differences across smoking groups with respect to impulse control were not observed at age 10 or age 13. These findings are supported and extended by a recent study with the OPPS sample (Fried & Watkinson, in press) in which it was shown that significant differences with respect to impulse control observed in younger children at age 6 were not associated with prenatal exposure to cigarettes in children ranging in age from 13 to 16 years. This suggests that if group differences in performance at age 6 are

the result of maturational differences, these differences may have been overcome by age 10 in the children born to heavy smokers.

In summary, findings from the present study relating to cigarettes demonstrate that although cigarettes are shown to impact both sustained attention and impulse control, the differential timing of this impact is consistent with multi-component models of attention that view attention as a system comprised of a number of disparate elements within a larger cognitive framework (Barkley, 1997; Mirsky et al., 1991; Mirsky et al., 1995). The concept of localized elements of attention, each governing specific aspects of attentional functioning, is supported by normative studies of sustained attention and impulse control that propose different developmental trajectories for each of these aspects of attention (Greenberg & Waldman, 1993; Rebok et al., 1997), each possibly relying on different areas of the brain for their function. The evidence suggests that for children born to heavy smokers, the attentional system is vulnerable to the effects of prenatal exposure to cigarettes, with differential impacts for sustained attention and impulse control. The impact on each element of attention appears to be most significant at the point where normative development is taking place. It is encouraging that in the present study, children born to heavy smokers appear to “catch up” to other children with respect to attentional functioning, and do not continue to display compromised performance relative to children born to non-smokers and light-moderate smokers.

The evidence suggests that children exposed prenatally to heavy doses of cigarette smoke are disadvantaged to some degree during development relative to non-exposed children with respect to attentional performance, but recover over time to reach levels of

performance that are not significantly different from performance observed in normal comparison children. These findings are intriguing, and raise questions regarding the mechanisms behind such recovery of attentional function. Findings based on animal studies indicate that irreversible cellular changes occur with prenatal exposure to nicotine (Navarro, Seidler, Eylers, et al., 1989; Slotkin, 1998). It may be the case that for children exposed to nicotine prenatally, differences at the cellular level relative to non-exposed children may result in divergent developmental pathways sharing a common end-point for these groups with respect to attentional functioning. It is also possible that the “catch-up” observed in children born to heavy smokers is the result of moderating influences such as school attendance or aspects of the home environment that compensate for negative effects of cigarette exposure during pregnancy. The low-risk, middle class environment characteristic of the OPPS sample is one in which such compensatory mechanisms are likely to be present, and it may be the case that it is precisely the benign background experienced by participants in the OPPS that enables recovery of function to take place in the children of heavy smokers.

These findings, although somewhat tempered by the possibility of Type I error, to be discussed in detail in later sections, suggest that prenatal exposure to cigarettes may be associated with developmental delays for both sustained attention and impulse control. If in fact prenatal exposure to cigarettes impacts on cognitive development in such a way that the timing of normal developmental processes is altered, this has implications for children that extend beyond the laboratory setting. In the classroom, where children must be able to pay attention and refrain from impulsive responding, behaviour consistent with

poor sustained attention and impulse control (i.e. distractibility, inability to remain on-task, excessive talking, etc.) could place children of heavy smokers at a social and academic disadvantage compared to other children.

Barkley (1998) states that children with ADHD may suffer from a host of associated emotional, behavioural, social and academic difficulties in addition to problems with attentional functioning. Academically, children with ADHD typically perform at levels that are below expected achievement based on known ability derived from IQ and achievement tests. Poor school performance is theorized to result from deficits in the ability to sustain attention and inhibit impulsive behaviour (Barkley, 1998). Children with ADHD frequently have dysfunctional relationships with family members, teachers, and other children (Barkley, 1998). Pope, Bierman, and Mumma (as cited in Barkley, 1998) found that hyperactive children are seen as more aggressive, intrusive and noisy in their interactions with peers. Children with ADHD are also viewed as more defiant, less compliant, and talkative in their interactions with their parents (Danforth, Barkley, and Stokes as cited in Barkley, 1998). Barkley, Fischer, Edelbrock, and Smallish (as cited in Barkley, 1998) report that these behaviours exhibited by children with ADHD impact negatively on the way in which others respond to these children, and are predictive of future conflictual interactions with family members. It should be emphasized that the OPPS is not a clinical sample, and that children participating in the OPPS who were clinically diagnosed with ADHD were excluded from the current study. Children in the present sample are not known to have ADHD and therefore any similarities between children suffering from clinical disorders with sustained attention

and impulse control and the children in this study who exhibited problems on laboratory tasks with respect to these aspects of attentional functioning should be interpreted with caution.

Longitudinal Findings. On the basis of repeated measures ANCOVAs, after statistically controlling for the effects of parental education, passive postnatal nicotine exposure, use of alcohol and marihuana for sustained attention and use of alcohol and marihuana for impulse control, no significant differences in performance were observed across smoking groups related to either aspect of attention. These results were somewhat surprising given that a number of cross-sectional studies carried out using both the OPPS and other samples, have shown differences in performance in children prenatally exposed to cigarettes for both sustained attention (Leech et al., 1999; Streissguth et al., 1984) and impulse control (Fried et al., 1992; Kristjansson et al., 1989; Streissguth et al., 1984). One possible explanation for the lack of significant findings using a repeated measures ANCOVA approach is that because differences at each age across the drug groups are small, and these differences do not show consistency over time (i.e., the same deficits in performance are not observed across all ages for one group), these subtle differences may become lost when groups are tested using a repeated measures design – resulting in a kind of “smoothing” effect.

A further issue concerning the use of ANCOVA for these data relates to the use of standardized z-scores in the place of raw omission and commission error scores at each assessment. The use of standard scores was necessary in order to compare across different versions of the CPTs employed in the present study, but the use of z-scores

made it unlikely that age-related changes in performance over time would be observed, a finding reported by others employing longitudinal designs to study attentional performance (Rebok et al., 1997; Fischer, Barkley, Fletcher, & Smallish, 1993). To test for age-related changes in attentional performance (main effect of age), the means for errors (both omission and commission) across age categories were compared to assess for significant differences after collapsing across drug groups (Glass & Hopkins, 1996). The use of standard z-scores resulted in a situation where the means were the same (equal to zero) at each age category tested, and as such, would not result in an observed significant main effect of age for the present study.

It seems unlikely that the lack of significant findings are the result of statistical over-control due to the use of covariates in the present study. Cross-sectional studies routinely control for the same covariates used in the present study, and associations between attention variables remain significant with covariates included in the analyses (Leech et al., 1999; Fried et al., 1992; Fried & Watkinson, in press).

Maternal ratings of children's impulsive/hyperactive behaviour did not differ across the three smoking groups after controlling for potentially confounding variables. It should be noted that mother's ratings are subjective appraisals of behaviours exhibited by their children, and are not objective measures of attentional function. Fried et al. (1992) reported similar findings in a study looking at the attentional behaviour of 6-year-old participants in the OPPS.

Mother's ratings of impulsive/hyperactive behaviour that failed to differentiate across the different smoking groups were inconsistent with CPT outcome measures that

revealed higher commission errors at age 6 for children born to heavy smokers relative to children in the other two groups. Women who reported heavy use of cigarettes during pregnancy did not rate their children as significantly more impulsive/hyperactive than did mothers who reported light-moderate or no use of cigarettes while pregnant. Maternal ratings were not assessed cross-sectionally in the present study, and it is possible that mother's ratings may have differed at one or more of the ages tested, but as discussed previously, if differences were present and were not consistent over time, these differences could have become lost as part of the repeated measures analysis.

Marihuana

Sustained Attention. At age 6, significantly more children in the moderate marihuana group had omission error scores reflective of poor performance compared to the children in the no/infrequent use group. Between the ages of 6 and 10 years, the observed percentage of children in the no/infrequent use group that showed a decline in performance did not differ from the percentage that were expected to decline. In contrast, fewer children than expected in both the moderate and heavy marihuana groups declined in performance relative to children in the no/infrequent use group. The difference in the number of cases that declined in performance seems to stem from a relatively high percentage of cases in both the moderate and heavy marihuana groups with omission error scores falling in the 4th quartile of the distribution at 6 years of age. For these children, direction scores at age 10 (obtained by subtracting an individual's quartile rank at age 10 from their quartile rank at age 6) would either reflect stable performance, or improved performance. These children were limited in terms of demonstrating a decline

in performance because their position in the distribution could not get any lower at age 10 than a quartile rank of 4.

The results from the present study are in partial agreement with the existing literature looking at the relationship between marihuana and sustained attention. The present study replicates the results of previous work looking at 6 year old children from the OPPS that suggests an association between sustained attention and prenatal exposure to marihuana (Fried et al., 1992). In a recent study looking at attentional behaviour in adolescents aged 13 to 16 years (Fried & Watkinson, in press), the relationship between sustained attention and prenatal marihuana exposure continued to be significant for individuals exposed to high levels of marihuana *in utero*. In the present study, differences in performance across marihuana groups on tasks assessing sustained attention were only observed at age 6, and do not support recent findings relating to attentional functioning in adolescents. However, the present study was carried out using fewer subjects compared to the study by Fried and Watkinson (in press), and findings suggesting no effect of marihuana on sustained attention at age 13 for the present study could be due to low power to detect significant results commonly found with small sample sizes (Stevens, 1996).

Impulse Control. In the present study, prenatal exposure to marihuana was not associated with impulse control for any marihuana group at any age assessment. The lack of significant findings linking prenatal exposure to marihuana and problems with impulse control is consistent with previous studies carried out with the OPPS sample that

showed no association between commission errors and marihuana in 6 year olds (Fried et al., 1992) or adolescents aged 13-16 years (Fried & Watkinson, in press).

In contrast, Leech et al. (1999) found marihuana to be linked to problems with impulse control in a high-risk sample of 6-year-olds characterized by low socioeconomic status. Findings from the animal literature (Charlebois & Fried, 1980) and from human studies (Day et al., 1994; Griffith et al., 1994) suggest that prenatal exposure to marihuana is most likely to be harmful in combination with lifestyle factors (i.e., poor diet, health, lower parental education and income, etc) characteristic of high-risk populations. Children in the present study are considered low-risk, and may have been afforded protection by other factors in their environment (e.g., good diet, education, etc.) against possible long-term teratogenic effects associated with marihuana (Fried, 1989).

These results must be interpreted with caution as they are corroborated only by findings from the same low-risk sample (OPPS) and need to be replicated in other low-risk samples before drawing strong conclusions. However, findings such as those reported in the present study are suggestive that for low-risk samples, prenatal exposure to marihuana may have minimal impact on cognitive systems governing impulse control.

In summary, as was the case with cigarettes, marihuana appears to act in a differential fashion on sustained attention and impulse control, and such findings are consistent with models of attention that propose a multidimensional system approach to attentional functioning (Barkley, 1997; Mirsky et al., 1991; Mirsky et al., 1995).

Longitudinal Findings. No differences were observed for marihuana groups with respect to the repeated measures ANCOVAs for the sustained attention and impulse

control. As was discussed earlier with respect to cigarettes, these results may reflect the fact that group differences that are observed inconsistently over time can be lost when groups are tested using a repeated measures design.

Maternal ratings of children's impulsive/hyperactive behaviour did not differ across the three marihuana groups after controlling for potentially confounding variables. As was previously noted, mother's ratings are subjective appraisals of behaviours exhibited by their children, and are not objective measures of attentional function. With respect to the present findings, the fact that maternal ratings of impulsive/hyperactive behaviour did not differ across marihuana groups is in agreement with objective measures of impulse control that showed no significant differences in performance across drug groups. In contrast to these findings, previous studies looking at maternal ratings of impulsive/hyperactive behaviour found that compared to controls, mothers who were heavy marihuana users during pregnancy were more likely to rate their children's behaviour at age 6 as impulsive, despite objective evidence that found no association between marihuana and impulse control (Fried et al., 1992). In another study using a high-risk sample of 10-year-olds, Goldschmidt et al. (2000) found that marihuana use during pregnancy was predictive of maternal ratings of impulsive and hyperactive behaviour. It is interesting that previous studies have found higher maternal ratings of impulsive/hyperactive behaviour associated with marihuana use during pregnancy, while the present study found no difference in maternal ratings of behaviour. One possible explanation for these apparently discrepant findings is that the use of a repeated measures ANCOVA to investigate group differences on the CPRS-48 may have resulted in a

situation similar to that which occurred with omission and commission errors. Small differences in maternal ratings observed with cross-sectional studies did not remain significant across groups when analyzed longitudinally. Maternal ratings were not examined separately at each age to assess for group differences in the present study, so it is possible that had these analyses been carried out, findings similar to those reported in other cross-sectional studies with the same sample (Fried et al., 1992) would have emerged.

The findings in the present study suggest a link between prenatal exposure to cigarettes and marihuana and aspects of attentional functioning. However, a number of alternative explanations for these findings must also be considered. In the case of both cigarettes and marihuana, behavioural effects observed in the offspring of maternal drug users are likely not due solely to prenatal drug exposure, but rather result from a combination of drug exposure and a host of other influences in the child's environment. Children are exposed to a number of factors throughout development that may have an impact on attentional functioning. Barkley (1998), in a review of the literature investigating possible etiologies for ADHD, states that problems with attentional functioning can arise from a number of factors including CNS damage, complications during pregnancy, exposure to teratogens, and hereditary factors. In addition to prenatal influences, postnatal environmental factors such as demographic and socioeconomic status differences, social environment, lifestyle factors, and school attendance, to name a few, could individually or collectively impact on child development, and are difficult to control for in an experimental sense.

A considerable body of research exists that reports on links between ADHD and the quality of social relationships experienced by children with clinical attention problems. Some researchers have suggested that negative caretaking behaviours in early childhood, such as overcontrol of a child's behaviour (Silverman & Ragusa, 1991), and intrusive behaviours on the part of the primary caretaker (Jacobvitz & Sroufe, 1987) are associated with later development of ADHD symptomatology in children. In contrast, Barkley (1998) reports that negative parent-child interactions are more often the result of dysregulated behaviours on the part of the child, and do not stem from the way in which parents behave towards the child. Barkley, Fischer, Edelbrock, and Smallish (as cited in Barkley, 1998) suggest that factors in the child's social environment are not the primary cause of attentional dysfunction in children, but can serve to exacerbate existing difficulties experienced by a child. It should be stressed that these studies were carried out with the intent of investigating clinical problems with attention (i.e., ADHD) and the attentional difficulties experienced by the children in the present study represent non-clinically diagnosed problems with sustained attention and impulse control. The social environment experienced by children diagnosed with clinical attention deficits should not be generalized to children in the OPPS who are members of a non-clinical population. In general, it would appear that caretaking variables may account for some of the attentional difficulties in children with diagnosed attention problems, but caution is needed when generalizing from clinical populations to non-clinical samples such as the OPPS.

The OPPS is a low-risk sample, and many of the postnatal influences are similar across the different drug groups, which for this sample, provides a measure of control

over the impact of environmental and lifestyle factors when exploring subsequent behavioural differences in the offspring of maternal drug users.

In previous studies with the OPPS, prenatal exposure to cigarettes and marihuana accounted for 1 to 5 percent of the variance associated with cognitive function after controlling for a number of demographic, socioeconomic and lifestyle variables (Fried & Watkinson, 1998). In contrast, factors other than prenatal drug use accounted for 13% to 35% of the variance associated with cognitive functioning (Fried & Watkinson, 1988). While it is important to consider alternative explanations for differences observed in children exposed to cigarettes and marihuana relative to non-exposed controls, it is equally important not to trivialize findings associated with prenatal drug exposure simply because they account for small differences over and above lifestyle and demographic factors. In contrast to other samples (Leech et al., 1999; Goldschmidt et al., 2000), the demographic background of the OPPS sample is fairly benign, suggesting that findings based on a low-risk sample may present a conservative estimate of the degree of harm attributable to prenatal exposure to cigarettes and nicotine.

In addition to various demographic, socioeconomic and lifestyle variables that may account for some of the results reported in the present study, various statistical and methodological limitations may have impacted on the validity of the present findings, and any interpretation of the results needs to be considered with the following factors in mind.

Sample Size Considerations

Sample size for the present study was small due to (a) limited availability of subjects who had used either cigarettes or marihuana or both during pregnancy, and (b) various exclusion criteria employed to control for several methodological issues. Small sample size raises a number of concerns relating to power and control over Type I error. The relatively small sample used in this study resulted in low power to detect significant results, thus increasing the likelihood of Type II error. This problem was further compounded by the use of nonparametric statistics for some analyses, as these tests are generally less powerful than parametric statistical methods. An additional concern was control over Type I error, which was of particular concern in the present study given the use of multiple statistical tests. Due to concerns that adequate control over Type I error would cause power to be reduced to unacceptably low levels, statistical control over Type I error (e.g., using a more stringent alpha level than .05) was not undertaken in the present study. It is possible that some of the significant findings in the present study are due to chance. It is unlikely however, that all the findings in the present study are reflective of Type I error, given that the results, in a general sense, parallel the findings of others (Fried et al., 1992; Fried & Watkinson, in press; Kristjansson et al., 1989; Streissguth et al., 1994) looking at sustained attention and impulse control in children prenatally exposed to cigarettes and marihuana.

Control Over Effects of Confounding Variables

The use of nonparametric statistics for a number of analyses resulted in a situation where it was not possible to control for the effects of confounding variables, including the use of the drug that was not considered to be of primary interest. Except for results

based on ANCOVA procedures, an interpretive problem exists with respect to the degree to which findings can be attributed to the drug of primary interest. It is possible that significant findings associated with cigarettes for example, are influenced by marihuana, or by the concurrent presence of both drugs in the analyses. However, this seems unlikely given that the drugs had differential effects observed at different ages on sustained attention and impulse control. Cigarettes were associated with impulse control at age 6 and sustained attention at age 10, while marihuana was linked to sustained attention at age 6, and showed no association with impulse control. It is also possible that significant findings are attributable to some other variable that could not be controlled for in the analyses.

Concerns regarding lack of statistical control over possible confounding effects of the drug that was not considered to be of primary interest were alleviated in part by a chi-square analysis looking at the distribution of marihuana groups over smoking groups. No significant differences across smoking groups with respect to the different levels of marihuana use were observed, indicating that for all nonparametric analyses the effects of the drug not considered to be of primary interest were evenly distributed across all levels of the drug under investigation.

Generalizability

As previously discussed, the OPPS is a low-risk sample, and the results of findings in the present study can be generalized only to other low-risk samples. This is particularly important in the case of marihuana because characteristics that define a low-risk sample, for example, diet, education and higher socioeconomic status, may afford

protection for the offspring of maternal users of marihuana against some of the negative effects of the drug.

CPT Measures Used to Assess Sustained Attention and Impulse Control

In this study, children were tested repeatedly over a wide age range, necessitating the use of two different versions of the CPT to ensure an appropriate level of task difficulty at each of the ages assessed. One drawback to using two different versions of the CPT was that in order to compare performance across the two tests, omission and commission error scores had to be converted to standard z-scores. In doing so, it was not possible to look at age-related changes in performance over time, and to examine the developmental trajectories of sustained attention and impulse control across the different smoking and marihuana groups.

The CPTs employed in this study differed greatly in terms of the outcome measures they provided, and this limited the types of analyses that were possible with respect to the study of sustained attention and impulse control.

The use of a single version of the CPT administered repeatedly across ages would enable researchers to study developmental changes over time. It is recommended that in the future, researchers who employ CPTs to assess impulse control use instruments that employ an AX paradigm and include measures of reaction time to allow and evaluation of the different sub-types of commission errors. It is further recommended that future research investigating sustained attention and impulse control using a CPT consider utilizing measures of perceptual sensitivity (d') to identify children who have difficulty

with target discrimination, and response bias (*Beta*; β) to differentiate between children who choose to respond with caution and those who are “risk takers”.

Investigation of these issues was beyond the scope of the present study because the CPT versions employed were not consistent in the types of outcome measures that they provided. The present study is a good preliminary investigation looking at aspects of sustained attention and impulse control over time. It does however, emphasize the need for well-designed longitudinal studies that use instruments that can be administered to children at different ages, and that include a variety of measures to allow for a more detailed examination of sustained attention and impulse control than this study was able to provide.

Summary and Conclusions

The findings from the present study suggest an association between prenatal exposure to cigarettes and marihuana and attentional functioning in the offspring of maternal users of these drugs. The results suggest that cigarette smoking during pregnancy is associated with impulse control in young children at age 6, and sustained attention in middle childhood, between the ages of 10 and 11 years. Associations with aspects of attention and prenatal exposure to marihuana were not as strong. Marihuana exposure was associated with sustained attention at age 6 only, and was not associated with problems with impulse control at any age tested. However, these findings should not be taken to mean that prenatal exposure does not place a child at risk for future problems, rather these results need to be considered within the framework of a low-risk sample.

It is important to bear in mind that although differences were observed across drug groups with respect to performance on tasks assessing sustained attention and impulse control, these findings do not necessarily imply clinical deficits in performance for the offspring of maternal users of cigarettes and marihuana. Appropriate age-normed data was not available to compare the performance of children in the present sample to levels of performance observed in normative samples. It is possible that in the present study, the level of performance observed in children showing poor sustained attention and impulse control may still fall within the range of normal attentional functioning in the general population.

These results suggest that prenatal exposure to cigarettes and marihuana may be associated with long-term differences in attentional functioning in the offspring of maternal drug users relative to non-exposed control children. Although far from conclusive, there is sufficient evidence to suggest that use of cigarettes and marihuana during pregnancy may compromise the attentional resources of children exposed to these substances. Although the differences observed in the OPPS sample are subtle, given that they represent preventable risks to children, women should be cautioned against the use of cigarettes and marihuana during pregnancy.

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Appendix A

Psychometric Properties of the GDS Vigilance Task, Conners' CPT, and the CPRS-48

Unless otherwise indicated, all psychometric data comes from the user's manuals provided by the test developers for each of the following instruments:

GDS Vigilance Task

Test-retest Reliability. In a report assessing the reliability of the GDS Vigilance Task (Gordon & Mettelman, 1988) moderate to high test-retest correlations at 30-45 days (r ranging from .72 to .85) and 1 year (r ranging from .68 to .94) were observed in a sample of non-referred school-aged children.

Inter-rater Reliability. Performance on GDS measures correlate with teacher and parent ratings of hyperactive behaviour (Gordon, McClure & Post, 1983; from the Interpretive Guide to the Gordon Diagnostic System).

Conners' CPT

Classification Error Rates. Conners (1995) reports low classification error rates for the Conners' CPT. Mean false positive rates (mean = 10.8%) and false negative rates (mean = 11.8%) were found with a sample of 720 clinical and general population cases.

CPRS-48

Inter-rater Reliability. Mothers' and fathers' ratings show moderate subscale correlations ranging from .46 to .57.

Internal Consistency. Alpha reliability coefficients are found in the low to moderate range for the CPRS-48, ranging from .13 to .65.

Appendix B

Quartile Ranks for Subjects at 6, 10 and 13 Years for Omission andCommission Errors Categorized by Drug Group

Sub ID	Smoking Group	Marihuana Group	Omission Errors			Commission Errors		
			Age 6	Age 10	Age 13	Age 6	Age 10	Age 13
1552	Non-Smoking	No/Infrequent Use	4	1	3	2	1	1
2142	Non-Smoking	No/Infrequent Use	1	2	4	1	3	4
2881	Non-Smoking	No/Infrequent Use	3	2	2	2	2	1
3421	Non-Smoking	No/Infrequent Use	3	4	1	3	1	3
3801	Non-Smoking	No/Infrequent Use	1	2	3	3	1	3
3882	Non-Smoking	No/Infrequent Use	2	1	1	2	1	1
4012	Non-Smoking	No/Infrequent Use	3	4	4	3	4	4
4891	Non-Smoking	No/Infrequent Use	1	1	3	2	1	1
5271	Non-Smoking	No/Infrequent Use	2	3	1	2	3	1
5421	Non-Smoking	No/Infrequent Use	4	1	1	1	3	1
5691	Non-Smoking	No/Infrequent Use	3	3	2	2	2	2
5692	Non-Smoking	No/Infrequent Use	1	2	2	1	3	2
5733	Non-Smoking	No/Infrequent Use	1	3	1	1	1	1
5821	Non-Smoking	No/Infrequent Use	3	2	3	3	4	2
5901	Non-Smoking	No/Infrequent Use	2	3	1	1	1	3
5941	Non-Smoking	No/Infrequent Use	3	2	3	2	1	1
5991	Non-Smoking	No/Infrequent Use	3	3	4	3	3	2
5992	Non-Smoking	No/Infrequent Use	1	3	3	1	2	3
6031	Non-Smoking	No/Infrequent Use	4	3	1	4	2	2
6171	Non-Smoking	No/Infrequent Use	2	4	2	2	4	3
6391	Non-Smoking	No/Infrequent Use	2	3	2	3	3	2
4331	Non-Smoking	Moderate	3	3	4	2	1	4
5882	Non-Smoking	Moderate	1	3	2	3	3	4
6001	Non-Smoking	Moderate	3	2	1	3	4	4
1012	Non-Smoking	Heavy	4	1	1	4	4	3
5731	Non-Smoking	Heavy	3	1	1	2	3	2
5931	Non-Smoking	Heavy	3	4	4	3	4	4
6152	Non-Smoking	Heavy	4	2	4	4	4	2
6341	Non-Smoking	Heavy	2	1	3	3	3	3
6631	Non-Smoking	Heavy	1	4	3	4	3	4
6661	Non-Smoking	Heavy	3	1	1	1	2	4
712	Light-Moderate	No/Infrequent Use	4	2	4	3	2	4
2272	Light-Moderate	No/Infrequent Use	1	1	2	1	1	2
2561	Light-Moderate	No/Infrequent Use	3	2	2	2	2	2
2671	Light-Moderate	No/Infrequent Use	2	3	2	1	4	2
3461	Light-Moderate	No/Infrequent Use	3	1	2	4	1	1
3462	Light-Moderate	No/Infrequent Use	4	4	4	4	4	4
3542	Light-Moderate	No/Infrequent Use	2	1	2	4	3	3
3912	Light-Moderate	No/Infrequent Use	2	1	3	2	3	3
4011	Light-Moderate	No/Infrequent Use	2	3	2	2	3	1
4391	Light-Moderate	No/Infrequent Use	2	3	1	3	3	3

Sub ID	Smoking Group	Marihuana Group	Omission Errors			Commission Errors		
			Age 6	Age 10	Age 13	Age 6	Age 10	Age 13
4632	Light-Moderate	No/Infrequent Use	2	1	1	3	1	2
5101	Light-Moderate	No/Infrequent Use	1	3	4	3	4	4
5241	Light-Moderate	No/Infrequent Use	3	1	1	1	1	3
5531	Light-Moderate	No/Infrequent Use	2	1	1	2	1	1
5532	Light-Moderate	No/Infrequent Use	3	4	4	3	3	4
6041	Light-Moderate	No/Infrequent Use	1	4	1	1	4	2
6331	Light-Moderate	No/Infrequent Use	2	1	4	2	1	4
6401	Light-Moderate	No/Infrequent Use	1	2	1	1	1	2
6411	Light-Moderate	No/Infrequent Use	4	4	4	4	4	4
6601	Light-Moderate	No/Infrequent Use	1	1	2	4	1	4
6711	Light-Moderate	No/Infrequent Use	4	4	2	3	3	3
6771	Light-Moderate	No/Infrequent Use	1	3	2	1	2	4
6781	Light-Moderate	No/Infrequent Use	1	2	1	1	3	1
782	Light-Moderate	Moderate	4	3	2	3	2	3
5732	Light-Moderate	Moderate	4	4	4	2	3	2
5881	Light-Moderate	Moderate	1	1	2	1	2	2
6011	Light-Moderate	Moderate	3	1	2	2	2	1
783	Light-Moderate	Heavy	4	2	2	4	3	3
2931	Light-Moderate	Heavy	1	1	2	1	1	1
6051	Light-Moderate	Heavy	2	2	4	3	4	2
6551	Light-Moderate	Heavy	4	3	2	1	1	3
2932	Heavy	No/Infrequent Use	2	4	2	4	4	4
3381	Heavy	No/Infrequent Use	4	4	2	1	3	1
3541	Heavy	No/Infrequent Use	1	3	4	1	2	3
3891	Heavy	No/Infrequent Use	1	1	1	1	1	1
5351	Heavy	No/Infrequent Use	4	4	1	3	4	3
5352	Heavy	No/Infrequent Use	4	4	4	2	4	4
5721	Heavy	No/Infrequent Use	4	2	1	4	2	1
6491	Heavy	No/Infrequent Use	1	3	4	2	2	4
6511	Heavy	No/Infrequent Use	3	4	2	3	2	2
6621	Heavy	No/Infrequent Use	3	4	4	4	4	3
6671	Heavy	No/Infrequent Use	1	3	3	1	1	4
6741	Heavy	No/Infrequent Use	3	3	4	4	4	4
6791	Heavy	No/Infrequent Use	2	2	4	4	2	3
6071	Heavy	Moderate	4	3	2	3	1	3
6451	Heavy	Moderate	4	4	1	4	4	2
6461	Heavy	Moderate	4	4	4	4	4	2
6801	Heavy	Moderate	4	3	3	4	3	1
2771	Heavy	Heavy	4	4	4	4	3	2
6052	Heavy	Heavy	1	1	1	4	1	1

Appendix C

Percentage of Cases with Quartile Ranks 1 Through 4 at 6, 10, and 13 YearOmission and Commission Errors Categorized by Drug GroupOmission Errors for Smoking Groups

% within Smoking Group

		Smoking Group			Total
		Non-Smoking	Light-Moderate	Heavy	
Omissions	1	25.8%	29.0%	26.3%	27.2%
Quartile	2	19.4%	29.0%	10.5%	21.0%
Rank Age	3	38.7%	16.1%	15.8%	24.7%
6	4	16.1%	25.8%	47.4%	27.2%
Total		100.0%	100.0%	100.0%	100.0%

% within Smoking Group

		Smoking Group			Total
		Non-Smoking	Light-Moderate	Heavy	
Omissions	1	25.8%	38.7%	10.5%	27.2%
Quartile	2	25.8%	19.4%	10.5%	19.8%
Rank Age	3	32.3%	22.6%	31.6%	28.4%
10-11	4	16.1%	19.4%	47.4%	24.7%
Total		100.0%	100.0%	100.0%	100.0%

% within Smoking Group

		Smoking Group			Total
		Non-Smoking	Light-Moderate	Heavy	
Omissions	1	35.5%	22.6%	26.3%	28.4%
Quartile	2	19.4%	48.4%	21.1%	30.9%
Rank Age	3	25.8%	3.2%	10.5%	13.6%
13-15	4	19.4%	25.8%	42.1%	27.2%
Total		100.0%	100.0%	100.0%	100.0%

Commission Errors for Smoking Groups

% within Smoking Group

		Smoking Group			Total
		Non-Smoking	Light-Moderate	Heavy	
Commissions	1	22.6%	32.3%	21.1%	25.9%
Quartile Rank	2	32.3%	22.6%	10.5%	23.5%
Age 6	3	32.3%	25.8%	15.8%	25.9%
	4	12.9%	19.4%	52.6%	24.7%
Total		100.0%	100.0%	100.0%	100.0%

% within Smoking Group

		Smoking Group			Total
		Non-Smoking	Light-Moderate	Heavy	
Commissions	1	29.0%	32.3%	21.1%	28.4%
Quartile Rank	2	16.1%	19.4%	26.3%	19.8%
Age 10-11	3	32.3%	29.0%	15.8%	27.2%
	4	22.6%	19.4%	36.8%	24.7%
Total		100.0%	100.0%	100.0%	100.0%

% within Smoking Group

		Smoking Group			Total
		Non-Smoking	Light-Moderate	Heavy	
Commissions	1	25.8%	19.4%	26.3%	23.5%
Quartile Rank	2	25.8%	29.0%	21.1%	25.9%
Age 13-15	3	22.6%	25.8%	26.3%	24.7%
	4	25.8%	25.8%	26.3%	25.9%
Total		100.0%	100.0%	100.0%	100.0%

Omission Errors for Marihuana Groups

% within Marihuana Group

		Marihuana Group			Total
		No/Infrequent	Moderate	Heavy	
Omissions	1	29.8%	18.2%	23.1%	27.2%
Quartile	2	26.3%		15.4%	21.0%
Rank Age	3	24.6%	27.3%	23.1%	24.7%
6	4	19.3%	54.5%	38.5%	27.2%
Total		100.0%	100.0%	100.0%	100.0%

% within Marihuana Group

		Marihuana Group			Total
		No/Infrequent	Moderate	Heavy	
Omissions	1	24.6%	18.2%	46.2%	27.2%
Quartile	2	21.1%	9.1%	23.1%	19.8%
Rank Age	3	29.8%	45.5%	7.7%	28.4%
10-11	4	24.6%	27.3%	23.1%	24.7%
Total		100.0%	100.0%	100.0%	100.0%

% within Marihuana Group

		Marihuana Group			Total
		No/Infrequent	Moderate	Heavy	
Omissions	1	29.8%	18.2%	30.8%	28.4%
Quartile	2	29.8%	45.5%	23.1%	30.9%
Rank Age	3	14.0%	9.1%	15.4%	13.6%
13-15	4	26.3%	27.3%	30.8%	27.2%
Total		100.0%	100.0%	100.0%	100.0%

Commission Errors for Marihuana Groups**% within Marihuana Group**

		Marihuana Group			Total
		No/Infrequent	Moderate	Heavy	
Commissions	1	29.8%	9.1%	23.1%	25.9%
Quartile Rank	2	26.3%	27.3%	7.7%	23.5%
Age 6	3	24.6%	36.4%	23.1%	25.9%
	4	19.3%	27.3%	46.2%	24.7%
Total		100.0%	100.0%	100.0%	100.0%

% within Marihuana Group

		Marihuana Group			Total
		No/Infrequent	Moderate	Heavy	
Commissions	1	31.6%	18.2%	23.1%	28.4%
Quartile Rank	2	21.1%	27.3%	7.7%	19.8%
Age 10-11	3	24.6%	27.3%	38.5%	27.2%
	4	22.8%	27.3%	30.8%	24.7%
Total		100.0%	100.0%	100.0%	100.0%

% within Marihuana Group

		Marihuana Group			Total
		No/Infrequent	Moderate	Heavy	
Commissions	1	26.3%	18.2%	15.4%	23.5%
Quartile Rank	2	22.8%	36.4%	30.8%	25.9%
Age 13-15	3	24.6%	18.2%	30.8%	24.7%
	4	26.3%	27.3%	23.1%	25.9%
Total		100.0%	100.0%	100.0%	100.0%