Early primary school outcomes associated with **maternal use of alcohol and tobacco during pregnancy** and with **exposure to parent alcohol** and **tobacco use postnatally**






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Better Beginnings, Better Futures Longitudinal Study

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Executive Summary

Objective

The main purpose of the present study was to analyze longitudinal data from the "Better Beginnings, Better Futures" (BBBF) prospective study to examine relationships between prenatal exposure to alcohol and tobacco, separately and in combination, on developmental outcomes in young children from disadvantaged Ontario communities over the first four years of primary school (i.e. from 4 to 8 years of age). We also examined the effects of postnatal exposure to maternal drinking and smoking.

Four hypotheses were explored:

1. Children with higher-risk drinking mothers would show poorer developmental outcomes than those with lower-risk drinking mothers.
2. Children whose mothers smoked during pregnancy would show poorer developmental outcomes than those whose mothers did not smoke.
3. Children whose mothers were both high-risk drinkers and smokers during pregnancy would show the greatest developmental problems during primary school.
4. Maternal drinking and smoking during pregnancy would be more predictive of children's primary school problem behaviours than postnatal exposure to parental drinking and smoking behaviour during the preschool years.

Methodology

Two sets of statistical analyses were used. First, analysis of covariance (ANCOVA) allowed us to determine whether prenatal exposure to alcohol and/or tobacco may have differential effects on various aspects of children's functioning during the early primary school years. This first analysis was designed as a "proof-of-concept" or exploratory model. Measures in five domains of child development outcomes were analyzed in the ANCOVA analysis: general development, cognitive development/academic performance, social/emotional functioning, behaviour problems, and physical health.

Second, based on results from the ANCOVA, a more complex statistical technique (structural equation modelling [SEM]) was used to examine the pathways from prenatal and postnatal exposure to alcohol and tobacco, to parent and teacher reports of children's behaviour problems at age 8 (Grade 3). In this first-path analysis of this large and complex dataset, we focused on children's externalizing (misbehaviour and problem behaviour) and internalizing (distress and emotion) behaviour problems in particular, because the latent trait structure of these behaviour problems was well enough documented in the research literature to use confirmatory techniques.

Both of the above-mentioned sets of analyses were carried out on the BBBF longitudinal dataset made up of over 400 children. These children and their families were recruited from disadvantaged Ontario communities at birth, and were followed prospectively at 33 and 48 months, and again at age 8. Thus, it was also possible to measure postnatal exposure to alcohol (i.e. maternal drinking) and tobacco (i.e. second-hand or environmental smoke), and to examine whether any negative effects of

prenatal exposure to alcohol and tobacco on children's developmental outcomes increased or decreased over a four-year period between 4 and 8 years of age. Maternal alcohol use was assessed using the CAGE questionnaire (Ewing, 1984), while maternal tobacco use was assessed with questions from the National Longitudinal Survey of Children and Youth (NLSCY) and other population surveys. For all analyses, a comprehensive set of family socio-economic, cultural and demographic variables listed in Appendix 2 were employed as covariates in order to eliminate confounding effects of these variables.

Results

In the first ANCOVA analysis, children whose mothers reported higher-risk alcohol consumption during pregnancy showed long-term negative outcomes in measures of school performance and behaviour problems. These problems were accentuated in children whose mothers reported both alcohol and tobacco use during the pregnancy. However, negative outcomes were not evident in mothers who used only tobacco during pregnancy.

Further, the negative effects were more apparent at some times than at others: when children were 4 years of age, and faced with the challenges of formal school entry (i.e. poor school readiness) and again at 8 years of age, when individual differences in conceptual thinking may have been particularly salient to teachers. The percentage of measures demonstrating the disadvantage of children exposed to prenatal alcohol and tobacco increased from 37% at age 4 to 47% at age 8.

Second, results of the SEM suggest that the effects of the prenatal drinking and smoking were evident even when drinking and smoking behaviour at 33 months was taken into account. Although parental smoking behaviour (at age 33 months) predicted teacher reports of internalizing behaviour, prenatal maternal smoking accounted for both parent and teacher reports of externalizing problems and prenatal maternal drinking predicted teacher reports of both internalizing and externalizing problems.

Conclusions

Maternal drinking and tobacco use during pregnancy predicted that a child will have problems in elementary school, even when taking into account later smoking and drinking behaviour by the child's parents. If these effects have endured for eight years, it seems unlikely that such effects will dissipate. If the trends are maintained as we expect, children's academic and social behaviour may continue to be compromised into early adolescence. That is, prenatal exposure to maternal drinking and smoking may be linked to problems in or negative effects associated with cognitive and social development at critical periods in children's development, with lifelong consequences.

1. Introduction

This secondary data analysis was commissioned by the Fetal Alcohol Spectrum Disorder Initiative, Public Health Agency of Canada. The data analyses used the Better Beginnings, Better Futures (BBBF) longitudinal database. The BBBF longitudinal study began in 1993. Over 500 children born in 1994 in six disadvantaged neighbourhoods across Ontario were recruited for the longitudinal study at birth; over 400 remained in the cohort at 8 years of age (Grade 3).

Children who are prenatally exposed to alcohol and tobacco have been found to be at risk for a range of adverse health and developmental outcomes from infancy into adulthood (Huizink & Mulder, 2006; Richter & Richter, 2001). The main purpose of the present study was to examine relationships between prenatal and postnatal exposure to alcohol and tobacco separately and in combination on developmental outcomes in young children over the first four years of primary school.

Four hypotheses were explored in this study:

1. Children with higher-risk drinking mothers would show poorer developmental outcomes than those with lower-risk drinking mothers.
2. Children whose mothers smoked during pregnancy would show poorer developmental outcomes than those whose mothers did not smoke.
3. Children whose mothers were both higher-risk drinkers and smokers during pregnancy would show the greatest developmental problems during primary school.
4. These prenatal effects would be evident even when taking into account more recent (i.e. postnatal) data on parental drinking and smoking collected when the child was 33 months old.

- Measures in five domains of child development outcomes were analyzed, including general development, cognitive development/academic performance, social/emotional functioning, physical health, and externalizing and internalizing behaviour problems.

Two sets of analyses were used. First, analysis of covariance (ANCOVA) was used to determine whether prenatal exposure to alcohol and/or tobacco may have differential effects on these various aspects of children's functioning during the early primary school years. Based on results from the ANCOVA, more complex statistical techniques (structural equation modelling [SEM]) were used to examine the pathways from prenatal and postnatal exposure to alcohol and tobacco smoke, to parent and teacher reports of children's behaviour problems at age 8 (Grade 3). In this path analysis, we focused on externalizing and internalizing behaviour problems. Given the large array of variables available and the complexity of SEM, we needed to reduce the scope to make SEM feasible. We decided to begin with an array of 12 variables that allowed us to use existing research literature to develop a confirmatory model of the behaviour problem measures.

The BBBF longitudinal dataset was made up of over 400 children. These children and their families were recruited from disadvantaged Ontario communities at birth, and were followed prospectively at 33 and 48 months, and again at age 8. Thus, it was also possible to measure postnatal exposure to alcohol (i.e. maternal drinking) and tobacco (i.e. second-hand or environmental smoke), and to examine whether any negative effects of prenatal exposure to alcohol and tobacco on children's developmental outcomes increased or decreased over a four-year period between 4 and 8 years of age.

Although previous studies have found evidence for an association between prenatal substance exposure and adverse effects on children's development and functioning, many other studies have used clinical samples with very high prenatal substance exposure. In contrast, this is a large community-based sample of children who were followed prospectively from 3 months to 8 years of age. A strength of the prospective longitudinal study design is the ability to control for many familial and demographic factors.

Maternal alcohol use was assessed using the CAGE questionnaire (Ewing, 1984), while maternal tobacco use was assessed with questions from the National Longitudinal Survey of Children and Youth (NLSCY) and other population surveys.

Because associations between smoking during pregnancy and child outcomes may be due to more than just nicotine, for consistency, we use the term "prenatal tobacco exposure" in this report to refer to the effects on offspring of maternal smoking during pregnancy.

In the following section, we review some of the current research literature on the effects of prenatal exposure to alcohol and tobacco on children's health.

2. Literature Review

Exposure to Prenatal and Postnatal Alcohol and Tobacco

2.1 Methodological Considerations

In discussing the associations between maternal substance use and children's health, it is important to note at the outset several important caveats, summarized eloquently by Richter and Richter (2001), as well as Huizink and Mulder (2006). In this area of research, "gold standard" experimental designs (randomized controlled trials) are precluded because of obvious ethical concerns. Thus, the ability to draw causal conclusions is limited. Most research studies are based on cross-sectional designs with clinical samples – highly exposed children (sometimes termed "cases") who are selected and compared to non-exposed children. In these types of studies, information on alcohol and smoking during pregnancy is collected retrospectively, up to 12 months or more after birth of the child. Use of a retrospective design increases the possibility of recall bias, wherein the mother is hesitant to admit substance use, or forgets the amount she consumed. A prospective longitudinal design is preferable, since the women are typically recruited during pregnancy and their child is followed for a long period (e.g. from 3 months to 8 years of age in the current BBBF longitudinal study).

A number of confounding factors can mediate the demonstrated associations between prenatal substance exposure and effects on children. These confounds may include, for example, socio-economic status (SES) and other demographic variables such as maternal education; prenatal nutrition, caffeine, drug use and psychological stress; prenatal medical care; and the postnatal environment such as exposure to second-hand smoke, quality of the parent–child interactions, and other familial risk factors. Two studies indicate that the association between prenatal tobacco exposure and effects on children may be influenced more by confounders than the association between prenatal alcohol exposure and effects on children (D'Onofrio et al., 2008; Sen & Swaminathan, 2007). Finally, it should be noted that the methods of assessment, specific outcome measures and level of substance use vary greatly among studies. Results from different studies, therefore, are not always directly comparable (Huizink & Mulder, 2006).

> Nevertheless, the preponderance of evidence clearly points to strong associations between prenatal alcohol and tobacco exposure and adverse consequences on children's physical, cognitive, social/emotional and behavioural development (Richter & Richter, 2001).

Although a relatively large body of literature has examined effects of prenatal exposure to alcohol and tobacco in newborns and infants, there are fewer studies on older children. Thus, in the sections below, we focus on effects on preschool and primary school-aged children.

- It is important to assess developmental outcomes in childhood since these outcomes predict health and well-being into adolescence and adulthood (Pihlakoski et al., 2006).

2.2 Alcohol Exposure in Prenatal and Postnatal Periods

In Canada, the rate of mothers who report drinking any alcohol during pregnancy is approximately 10.5%, according to the 2005 Canadian Community Health Survey (CCHS). In this nationally representative survey, women who had given birth in the previous five years were asked about their alcohol use during pregnancy. In the 2005 survey, the rate of alcohol use was slightly lower than in the previous two CCHS surveys: 12.4% of women in 2003 and 12.2% of women in 2000–01 reported drinking alcohol during pregnancy (Public Health Agency of Canada, 2008). In the CCHS, women over the age of 35 or between 15 and 19 years were generally more likely to report alcohol consumption than mothers between the ages of 20 and 34. Regionally, Quebec had the highest rate of maternal drinking during pregnancy in 2005 at 17.7%, and Newfoundland and Labrador had the lowest rate at 4.1%.

Slightly higher rates of maternal alcohol use during pregnancy were reported in the National Longitudinal Survey of Children and Youth (NLSCY) – in 2002–03, 15.6% of mothers reported consuming alcohol during their pregnancy and in 2000–01, 13.9% did so (Government of Canada, 2007).

A more in-depth analysis of the 2000–01 CCHS data from Alberta indicates that younger mothers (under 20 years) were more likely to binge drink (i.e. drink 5 or more drinks on one occasion) than mothers over 26 years of age. These analyses also indicate that higher-income pregnant women in Alberta were more likely to be drinkers; however, when they did drink, lower-income pregnant women were more likely to binge drink once per month or more.

These data are supported by the 2006–07 Maternity Experiences Survey results, which indicated that approximately 10.5% of women reported drinking during pregnancy. The Maternity Experiences Survey is a project of the Public Health Agency of Canada's Canadian Perinatal Surveillance System and was conducted by Statistics Canada. The study surveyed women 15 years of age and older who had had a singleton birth in the three months prior to the 2006 Census (Public Health Agency of Canada, 2009).

These figures indicate that a substantial number of Canadian children will continue to be exposed to alcohol in the prenatal stage unless there are dramatic changes in maternal behaviour.

2.2.1 Heavy Drinking During Pregnancy and Fetal Alcohol Spectrum Disorder

Alcohol is established as a significant teratogen, and results in a host of cognitive, social and behavioural deficits such as impairments in general intellectual functioning, language and academic achievement; developmental delays; and problems with learning, memory, adaptive functioning, attention, inhibition, and state regulation (Bailey et al., 2004; Mattson, Riley, Gramling, Delis & Jones, 1998; Streissguth & O'Malley, 2000). The consequences of alcohol use in pregnancy range from subtle problems to the unique cluster of abnormalities that constitutes Fetal Alcohol Syndrome (FAS) – the most severe of the four conditions that comprise Fetal Alcohol Spectrum Disorder (FASD) (Chudley et al., 2005; Jacobson & Jacobson, 2002). FAS was first described in 1973 by Jones and Smith, and is caused by heavy drinking during pregnancy. A diagnosis of FAS requires evidence of four main features (growth deficiency, facial malformation, central nervous system damage and confirmed (or unconfirmed) prenatal alcohol exposure), although substantial developmental disabilities are also evident in children without facial malformation (Chudley et al., 2005). The term "FASD" refers collectively to a number of disabilities associated with prenatal exposure to alcohol. The three conditions in the spectrum, all permanent and preventable, include FAS, partial-FAS (pFAS), and alcohol-related neurodevelopmental disorder (ARND). The latter two terms are applied to children who have confirmed prenatal alcohol exposure and who exhibit some, but not all, of the FAS features (Chudley et al., 2005). In Canada, it is estimated that 9 out of 1,000 babies each year are born with FASD (Public Health Agency of Canada, 2007).

The majority of studies to date have focused on binge drinking during pregnancy and the associations with FAS among exposed children (Huizink & Mulder, 2006). Substantial evidence indicates that binge-like drinking patterns, in which the fetus is exposed to high blood alcohol concentrations over relatively short periods of time, are particularly harmful for offspring, and place the fetus at the highest risk of FASD (Maier & West, 2001). Binge drinking is often defined as 5 or more drinks on one occasion; one standard drink is equivalent to 0.5 oz. of absolute alcohol (AA) (Streissguth, Barr & Sampson, 1990). For example, Streissguth, Barr and Sampson (1990) reported that children whose mothers reported *any* binge drinking in the period prior to pregnancy recognition demonstrated poorer academic performance on reading and arithmetic at age 7 than children whose mothers abstained or did not binge drink during pregnancy.

Notwithstanding the profound negative effects of heavy drinking during pregnancy, a growing body of literature has documented adverse effects on children's functioning at low to moderate levels of prenatal alcohol exposure (e.g. Jacobson & Jacobson, 1994; Sayal, Heron, Golding & Emond, 2007; Sood et al., 2001).

2.2.2 Moderate and Low Levels of Drinking During Pregnancy: Cognitive and Behavioural Outcomes

Recently, the effects of low levels of prenatal alcohol consumption have come under particular scrutiny. Controversial results from the U.K. Millennium Cohort study published online late in 2008 indicated that children born to mothers who drank up to 1 to 2 drinks per week or per occasion during pregnancy were not at increased risk of clinically relevant behavioural difficulties or cognitive deficits compared with children of abstinent mothers. The odds ratios in this study actually indicated *lower* risks of these problems at age 3 among children of light drinkers, even after controlling for possible confounds including socio-economic

factors, current drinking, mother's mental health, and child–parent relationship (Kelly et al., 2009). This study has received much media attention and prompted several commentaries and debates among researchers and clinicians, some of whom listed numerous methodological limitations of Kelly and colleagues' study (e.g. Gijsen, Fulga, Garcia-Bourmissen & Koren, 2008; Nathanson, Jayesinghe & Roycroft, 2007; Sayal, 2009).

Indeed, Kelly and colleagues' (2009) results were surprising in light of a growing body of literature that has documented the adverse effects of low and moderate levels of prenatal alcohol on behaviour, IQ, learning, and other educational outcomes among early school-aged children (Jacobson & Jacobson, 1994; Jacobson, Chiodo, Sokol & Jacobson, 2002; Sayal, Heron, Golding & Emond, 2007; Sood et al., 2001). For example, Sayal and colleagues (2007) reported an increased risk of behavioural and emotional problems (composite score of these problems) among girls whose mothers drank less than 1 drink per week during pregnancy. These effects were observed for parent ratings at age 47 months and 81 months, and were confirmed by later teacher ratings between 7 and 9 years of age. Similarly, in a prospective study of 501 mother-child pairs, Sood et al. (2001) reported that children with *any* prenatal exposure to alcohol were 3.2 times more likely to have delinquent behaviour scores in the clinical range. Other behavioural outcomes related to prenatal alcohol exposure include psychosocial deficits and problem behaviours, which have been found in FAS children and in children who were prenatally exposed to moderate levels of alcohol. These children were at increased risk of psychiatric disorders (Streisguth, Barr, Kogan & Bookstein, 1996) and were more likely to be rated as hyperactive, disruptive, impulsive or delinquent (Roebuck, Mattson & Riley, 1999).

The level of cognitive deficits among children with low–moderate prenatal alcohol exposure has not been studied extensively. Several studies, however, indicate that moderate alcohol exposure is associated with cognitive deficits in primary school-aged children, including IQ decrements, learning and memory problems and deficits in information-processing speed (Carmichael-Olson et al., 1997; Streissguth, 2007; Streissguth, Barr & Sampson, 1990; Wilford, Leech & Day, 2006). Streissguth, Barr and Sampson (1990), for example, found that moderate alcohol exposure (defined in this study as 2 or more drinks/day) was related to a 6-point decrease in IQ and lower reading and arithmetic achievement test scores at age 7, after adjustment for 15 covariates including prenatal tobacco exposure. In a prospective study of 636 mother-child pairs, Wilford, Leech and Day (2006) reported that moderate alcohol exposure (approximately 1 drink per day) during the first and second trimesters was related to decreases in composite IQ score as well as verbal, abstract/visual, and quantitative subscales at age 10 among African American children.

It appears that there may be dose–response effect of alcohol on child outcomes, wherein the heavier the level of maternal drinking during pregnancy, the greater the magnitude of negative effects on the exposed child (Goldschmidt, Richardson, Stoffer, Geva & Day, 1996; Jacobson and Jacobson, 2002; Jacobson, Jacobson, Sokol, Chiodo & Corobana, 2004; Sood et al., 2001). For example, Jacobson, Jacobson, Sokol, Chiodo and Corobana (2004) reported that each additional ounce of absolute alcohol (AA) per day (approximately 2 standard drinks) during pregnancy was related to a 2.9 point decrease in overall IQ at age 7.

In the section below, we examine the effects on externalizing and internalizing behaviour problems in particular.

2.2.3 Externalizing and Internalizing Behaviour Problems

An emerging literature has begun to document the associations between prenatal alcohol exposure and externalizing behaviour problems in school-aged children. Specifically, researchers have documented higher rates of inattentive, hyperactive, aggressive and antisocial behaviour in alcohol-exposed children compared with children with no exposure to alcohol (Brown et al., 1991; Mattson & Riley, 2000; Nanson & Hiscock, 1990; Sood et al., 2001).

In the aforementioned study by Sood et al. (2001), low levels of prenatal alcohol exposure (i.e. 1 alcoholic drink per week) were significantly associated with higher externalizing (aggressive and delinquent), internalizing (anxious/depressed and withdrawn), and other behaviour problems at 6 to 7 years of age. These results persisted even after careful control for confounding factors, including prenatal tobacco exposure, maternal age, education, marital status, SES and the home environment. Similarly, in a smaller sample of 88 Caucasian children 6 to 13 years old, heavy prenatal alcohol exposure (mothers were known to abuse alcohol, but children did not have diagnosis of FAS) was related to greater externalizing (attention, aggression, delinquency), internalizing (total score) and total behaviour problem scores (Mattson & Riley, 2000). In one retrospective study of children with FAS or fetal alcohol effects (FAE) (now part of the FASD) or children with attention deficit hyperactivity disorder (ADHD) and controls (Nanson and Hiscock, 1990), parents rated both groups of children as being more hyperactive and more inattentive than the children with no FAS and no ADHD.

Few studies have compared parent and teacher rating of externalizing problems. However, Brown et al. (1991) , did compare these two groups of informants. Although teacher reports reflected more social competence problems, depression and externalizing behaviours in 5-year-old children whose mothers continued to drink during pregnancy compared with those whose mothers stopped drinking or who never drank, parent reports revealed no such differences (Brown et al., 1991).

Recently, researchers have also turned their attention to the associations between prenatal alcohol exposure and children's internalizing problems such as depression and anxiety. O'Connor and colleagues have published a series of reports indicating associations between prenatal alcohol and childhood-onset depression (O'Connor & Kasari, 2000; O'Connor & Paley, 2006). For example, O'Connor and Paley (2006) used SEM to investigate the pathways from prenatal alcohol exposure to child depressive symptoms and the mediating effects of maternal and child characteristics, in a small sample of children aged 4 to 5 years. Results indicated that prenatal alcohol exposure was associated with more negative child affect. In turn, mothers of more negative children were less emotionally connected to their children, and those children had higher levels of depressive symptomatology. Interestingly, these results could not be explained by current maternal drinking patterns (O'Connor & Paley, 2006). Similarly, analyses from a large prospective sample of children prenatally exposed to moderate levels of alcohol indicated an association between higher rates of internalizing problems at age 10 and greater prenatal alcohol exposure, after controlling for significant covariates that also predicted problem behaviours (Day & Richardson, 2000).

2.2.4 Postnatal Alcohol Exposure

Both prenatal and postnatal alcohol exposure appear to shape children's developmental trajectories (O'Connor & Paley, 2006). It is of importance, then, that prenatal alcohol use is strongly correlated with postnatal use (Carmichael-Olson, O'Connor & Fitzgerald, 2001). Such an association raises the possibility that there is some aspect of postnatal drinking that could account for effects attributed to prenatal drinking, and deserves to be addressed.

A large body of literature has examined the adverse effects of children of alcoholics (termed COAs). These studies suggest that children of alcoholics are at higher risk for a variety of emotional, behavioural and other developmental problems (Fitzgerald, Davies & Zucker, 2002).

Despite the well-documented adverse effects of postnatal exposure to alcohol, several researchers have noted that the effects of maternal current drinking do not have much of an effect on the strong association between prenatal alcohol and internalizing behaviour problems (O'Connor & Paley, 2006), aggressive behaviour, or social competence of school-aged children (Brown et al., 1991). It is possible that prenatal alcohol exposure may have effects on behaviour problems and socio-emotional functioning that are independent of current maternal drinking, or that postnatal alcohol use must be at a relatively high level to significantly contribute to adverse effects on children (O'Connor & Paley, 2006).

2.3 Tobacco Exposure in the Prenatal and Postnatal Periods

In the 2005 Canadian Community Health Survey (CCHS), 13.4% of women reported smoking cigarettes during pregnancy, and 14.1% of women reported being exposed to second-hand (environmental) tobacco smoke during their pregnancy (Public Health Agency of Canada, 2008). These rates have decreased since the 2000–01 CCHS, when 17.7% of women reported smoking during pregnancy and 22.4% reported being exposed to second-hand smoke. Similar rates of smoking during pregnancy were observed in the National Longitudinal Survey of Children and Youth (NLSCY); in 2002/03, 15.9% of women reported smoking during pregnancy, and earlier in 2000/01, 18.5% reported this behaviour (Government of Canada, 2007). In the 2005 CCHS survey, younger mothers, and mothers with less than a high school education were more likely to report this behaviour. Regionally, in the 2005 CCHS, British Columbia and Ontario had the lowest rates of maternal smoking during pregnancy (9.7% and 10.3%, respectively); Nunavut and Northwest Territories had the highest rates (59.5% and 32.8%, respectively). Data from the NLSCY indicate that 35% of women who reported smoking during pregnancy smoked 10 or more cigarettes a day.

Unfortunately, it appears that the majority of smokers will continue this behaviour throughout their pregnancy. In the U.S. National Pregnancy and Health Study, approximately two-thirds of women who smoked prior to their pregnancy continued smoking into the last trimester (National Institute on Drug Abuse, 1996).

2.3.1 Level of Prenatal Tobacco Exposure Associated with Adverse Outcomes

- Research indicates a dose–response gradient, wherein the adverse effects on children exposed prenatally to tobacco (and its numerous by-products) is dependent on the frequency and quantity of maternal smoking during the gestation period (Richter & Richter, 2001).

The greater the exposure, the more likely the child is to suffer. For example, birth weight decreases in direct proportion to the number of cigarettes smoked (Cornelius & Day, 2007).

- The timing of exposure also affects the outcomes in the exposed child, with the most pronounced effects of smoking on birth weight, for example, occurring during the third trimester (Richter & Richter, 2001).

Dose–response relationships have also been documented with other childhood outcomes, including cognitive and behavioural functioning (Huizink & Mulder, 2006; Martin, Dombrowski, Mullis, Wisenbaker & Huttunen, 2006). In a prospective longitudinal study of 676 Finnish children, Martin and colleagues (2006) classified maternal tobacco use during pregnancy as none, light (1–5 cigarettes/day) and heavy (6 or more cigarettes per day). At 12 years of age, children of light smokers exhibited levels of behaviour problems and academic achievement that were intermediate between those reported for non-smokers and for heavy smokers.

2.3.2 Effects on Growth, Cognitive and Behavioural Outcomes

A substantial body of literature has documented the adverse effects of maternal smoking during pregnancy on birth weight and infant growth (Cornelius & Day, 2007; Richter & Richter, 2001). Children born to mothers who smoke are also at risk of health conditions such as cleft palate, decreased lung function and middle ear disease; these effects are independent of the adverse health effects of environmental tobacco smoke (Richter & Richter, 2001).

A smaller literature base is available for effects of prenatal tobacco exposure beyond the neonatal and infant period. The available research does indicate relationships between prenatal tobacco exposure and childhood cognitive and behavioural developmental deficits, such as lower scores in general intellectual functioning, reduced verbal ability, increased activity, inattention and impulsivity, and higher rates of conduct disorder and other behaviour problems (Cornelius & Day, 2007; Huizink & Mulder, 2006; Richter & Richter, 2001).

In terms of cognitive outcomes, in the Ottawa Prenatal Prospective Study for example, tobacco exposure was significantly related to lower cognitive functioning and poorer language development at 2, 3 and 4 years of age (Fried & Watkinson, 1990; Fried, O'Connell & Watkinson, 1992). When those children were 9 to 12 years old, prenatal tobacco exposure was negatively associated with language and reading abilities. Similar results on cognitive functioning were reported by Milberger, Biederman, Faraone, Chen & Jones (1996) and Olds, Henderson and Tatelbaum (1994). In these three studies, associations between prenatal tobacco exposure and cognitive deficits remained significant after adjustment for confounds such as SES, education, marital status and parental IQ. However, none of the studies controlled for ongoing exposure to environmental tobacco smoke. Some researchers assert that associations between prenatal

tobacco exposure and cognitive development can be explained by differences in genetics or the home environment, such as postnatal exposure to second-hand smoke; this area is discussed in section 2.3.4 (D'Onofrio et al., 2008; Eskenazi & Trupin, 1995).

Although prenatal exposure to tobacco appears to influence cognitive functioning, a stronger association is apparent with children's behaviour problems (D'Onofrio et al., 2008). For example, one 10-year longitudinal study reported that mothers who smoked frequently while pregnant were more than four times as likely as less frequent smokers or non-smokers to have sons who developed a conduct disorder, and were more than five times as likely to have daughters who became dependent on drugs (Weissman, Warner, Wickramaratne & Kandel, 1999). It appears that there are clear long-term adverse effects of prenatal tobacco exposure on behaviour, according to results from a New Zealand birth cohort study (Fergusson, Woodward & Horwood 1998). Fergusson and colleagues reported that children exposed, compared with those not exposed to maternal smoking during pregnancy, had higher symptom rates of chronic disease, substance abuse, and depression at 16 to 18 years of age. The effects remained after the authors controlled for socio-economic disadvantage, impaired child-rearing behaviour, and parental and family problems. The bulk of the literature on behavioural outcomes has focused on Attention Deficit Hyperactivity Disorder (ADHD) and other externalizing behaviours. We review some of these studies in the next section.

2.3.3 Externalizing and Internalizing Behaviour Problems

Smoking during pregnancy has been consistently linked with externalizing problems in childhood, especially in boys (e.g. Ashford, van Lier, Timmermans, Cuijpers & Koot, 2008; Martin, Dombrowski, Mullis, Wisenbaker & Huttenen, 2006; Wakschlag, Pickett, Cook, Benowitz & Leventhal, 2002; Williams et al., 1998). For example, prenatal exposure to tobacco (mother smoked 1 or more cigarette(s)/day) was related to significantly higher parent-rated activity levels at age 5 in a sample of 676 children from the Helsinki Longitudinal Project in Finland (Martin, Dombrowski, Mullis, Wisenbaker & Huttenen, 2006). Among the same sample at age 12, children who were prenatally exposed to tobacco were rated by their teachers as being more distractible and less mature than children who had no prenatal exposure to tobacco. Martin and colleagues controlled for a range of possible confounds, including SES, maternal age and maternal psychological distress but did not control for postnatal or environmental tobacco smoke exposure. In a population-based cohort of 1,452 twin pairs aged between 5 and 16 years from the Greater Manchester Twin Register, maternal prenatal smoking was found to have a statistically significant relationship with both parent and teacher ratings of ADHD, even after control for two sets of potential confounds – genetic factors and familial/environmental influences (Thapar et al., 2003). Linnet et al. (2003) found consistent evidence of independent effects of smoking on a variety of symptoms related to ADHD in 4- to 7-year-old children, after statistical control for factors known to confound the relationships with ADHD (e.g. familial psychopathology). In a sample of 4,879 children from an Australian longitudinal study, Williams et al. (1998) found a dose–response relationship between externalizing behaviour problems and maternal smoking during pregnancy at 5 years of age. Weaker relationships were evident for internalizing behaviour problems. The associations appeared to be independent of a wide range of possible confounds, such as SES, education, social class, marital status and mental health. Williams and colleagues concluded that these results are strongly suggestive of a causal relationship.

Unfortunately, none of the four studies described above appeared to statistically adjust for the effects of postnatal or environmental exposure to tobacco smoke. There remains disagreement in the literature about the importance of confounding factors on the relationship between prenatal tobacco exposure and child behaviour problems. For example, although Williams et al. (1998) concluded that the relationship is causal, Maughan, Taylor, Caspi and Moffitt (2004) asserted that the association between prenatal tobacco and conduct disorder may be better accounted for by confounds, including antisocial behaviour in both parents, depression in mother and family environment. D'Onofrio et al. (2008) agreed, suggesting that environmental and genetic factors account for the associations between prenatal tobacco exposure and externalizing problems.

Compared with the knowledge base for externalizing outcomes, the relationship between prenatal tobacco exposure and internalizing behaviours is less well documented. Results for these studies have been mixed. Weitzman, Gormaker and Sobol (1992) employed a sample of 2,256 children aged 4 to 11 years from the U.S. National Longitudinal Survey of Youth (NLSY). Three groups of children were compared: those whose mothers smoked both during and after pregnancy; those whose mothers smoked only during pregnancy; and those whose mothers smoked only after pregnancy. Weitzman et al. did not include a direct comparison with mothers who did not smoke at all, so results are less clear with respect to the unique influence of prenatal smoking. However, it was clear that children whose mothers smoked both during and after pregnancy had significantly increased levels of depression and anxiety compared with children whose mothers smoked only after or only during pregnancy. This association remained after adjusting for the child's sex, birth weight, and various demographic and maternal characteristics. More recently, Ashford, van Lier, Timmermans, Cuijpers and Koot (2008) also used a longitudinal sample, and reported that prenatal tobacco exposure was a predictor of internalizing (and externalizing) behaviour problems in 396 children over the period of ages 5 to 18 years.

However, two studies have found that the effect of prenatal smoking on internalizing problems diminished after controlling for potentially confounding variables. For instance, the increased risk of internalizing problems among exposed children was found to disappear after controlling for variables such as socio-demographic factors, maternal anxiety and depression, birth weight, and pre- and perinatal complications (Williams et al., 1998) or after adjusting for socio-economic disadvantage, impaired child-rearing behaviours, and parental and family problems (Fergusson, Woodward & Horwood, 1998).

2.3.4 Postnatal Tobacco Exposure

Many women who smoke cigarettes during pregnancy continue to smoke after the pregnancy (Cornelius & Day, 2007). Children born to mothers who smoked during pregnancy are thus likely to continue to be exposed after the pregnancy. The most often cited consequence of postnatal exposure to environmental tobacco smoke (ETS) is an increased risk of sudden infant death syndrome (SIDS) (Cornelius & Day, 2007). Behavioural and cognitive outcomes, however, are also affected by postnatal exposure to ETS; however, results are mixed. For example, Cornelius, Goldschmidt, DeGenna and Day (2007) reported that environmental tobacco smoke was not a significant predictor of child behaviour at age 6 when prenatal tobacco exposure was considered. Weitzman, Gormaker and Sobol (1992), in contrast, reported a significant relationship between both prenatal and postnatal exposure and children's behaviour problems, even after controlling for confounds such as child's age, sex, family structure and income.

2.4 Effects of the Combination of Prenatal Alcohol and Tobacco

It is widely acknowledged that alcohol and tobacco use during pregnancy typically occur in combination (Cornelius & Day, 2007; Sen & Swaminathan, 2007). Specifically, research indicates that between 40% and 76% of women who report smoking during the first trimester of their pregnancy report concurrent alcohol use (Cornelius, Taylor, Geva & Day, 1995; Day, Cornelius & Goldschmidt, 1992; Streissguth, Barr & Sampson, 1990). Despite these statistics, few studies have assessed the effects of the interaction of both substances on the exposed child.

In the Ottawa Prenatal Prospective Study, among children aged 3 and 4 years, heavier maternal use of both alcohol and tobacco during pregnancy was related to statistically lower average scores for child comprehension and motor skills compared with groups reporting lighter use of the two substances (Fried, O'Connell & Watkinson, 1992; Fried & Watkinson, 1990). This effect was not evident at ages 5 and 6. Other reports have indicated that prenatal alcohol exposure has greater effects than prenatal tobacco. In analyses from the U.S. NLSY, Sen and Swaminathan (2007) examined the effects of both substances on children's behaviour problems between 4 and 10 years of age. Results indicated that whereas prenatal alcohol exposure continued to have effects on behaviour problems after controlling for confounds, prenatal smoking largely ceased to have any significant effects after controlling for maternal mental health and background, and postnatal smoking and drinking.

Based on this review of the literature, the present study was designed to examine whether prenatal and postnatal exposure to alcohol and tobacco, separately or in combination via mothers' reports of drinking and smoking during pregnancy, had any lasting association with a wide range of children's developmental outcomes over the first four years of primary school. **Although other studies have documented the effects of maternal substance use on child health, there are several strengths of the present study:**

- **First**, the prospective longitudinal study design (the BBBF Project) allowed us to determine the effects of both prenatal and postnatal substance exposure, and to assess developmental outcomes in the same children at several time-points.
- **Second**, the considerable sample size and diversity of participating families may increase the generalizability of results to other disadvantaged children in Canada.
- **Third**, we assessed effects on a wide range of child outcomes in the cognitive/academic, social/emotional, behavioural and health domains.
- **Finally**, the separate and combined effects of prenatal exposure to alcohol and tobacco were assessed.

3. Method

3.1 Data Source

This report used data from the BBBF Longitudinal Study database for analyses of the effects of prenatal and postnatal alcohol and tobacco on children's health and developmental outcomes during early primary school.

- **The BBBF Longitudinal Study is one of the most ambitious research projects on the long-term impacts of early childhood prevention programming for disadvantaged children in Canada. The diversity of the participating communities (francophone, Aboriginal, recent immigrants, and multicultural) increases the likelihood that findings will be applicable to children across Canada.**

The longitudinal study began in 1993 and is following two groups of children and their families who experienced up to four years of BBBF prevention programming. One group received Better Beginning programs from birth to age 4 (the younger group), and a second group received the programs from ages 4 to 8 (the older group). Also included in the longitudinal research is a comparison group of children and their families from several demographically matched communities that did not receive BBBF funding. (See bbbf.queensu.ca/research for a complete description of the research design and analyses.)

Data from the younger children only are included in the present study, as these children were involved in the study from 3 months of age. From 1993 to 2003, data were collected on approximately 600 children when the children were 3 months, 18 months, 33 months and 48 months, and in Grades 1 (age 6 years) and 3 (age 8 years). Data were collected by trained researchers in each community via a parent interview, direct child measures and, beginning at 48 months, from the child's teacher using a teacher report form. Over 100 outcome measures have been gathered at each data collection point, covering a wide range of child, parent/family and neighbourhood characteristics.

One of the unique features of the current study is the number of potentially confounding variables that were statistically controlled in all analyses. The measures used as covariates in all analyses were those that might bias the results due to factors other than smoking or drinking during pregnancy. By including these measures in the analyses, statistical controls were employed to remove any bias these variables may have had on the differences between groups. A complete list of the measures used as covariates in the analyses appears in Appendix 2, and includes measures of family income, maternal education, immigrant status, home language and single-parent status. Also included in this list of control variables is whether or not children resided in a BBBF or comparison community. Thus, any outcome differences resulting from Better Beginning program effects have been statistically eliminated from the following analyses.

3.2 Better Beginnings, Better Futures Study Characteristics

The BBBF study has generated the most extensive and intensive longitudinal database involving disadvantaged children and families in Canada. The BBBF longitudinal study contains more information about early child development and parent behaviour in disadvantaged neighbourhoods than the National Longitudinal Survey of Children and Youth (NLSCY; Statistics Canada & Human Resources Development Canada, 1995), the Ontario Child Health Study (OCHS; Statistics Canada, 2004) and the Montreal Longitudinal Study (MLS; Tremblay, Mâsse, Kurtz & Vitaro, 1996). The NLSCY longitudinal samples are selected to match the general Canadian population in terms of socio-economic and other demographic variables. Hence in these longitudinal samples, there are relatively few children living in disadvantaged families. This is also true of the OCHS sample in Ontario. Further, since the OCHS and MLS began studying children longitudinally at ages 4 and 6, respectively, no data were collected in these two studies from mothers or children at or immediately following the children's birth. Finally, neither the OCHS nor MLS collected as wide a variety of child outcome measures as the BBBF longitudinal study.

3.3 Measures of Maternal Alcohol and Tobacco Use

As part of the first parent interview, when their child was 3 months old, mothers in the BBBF study were asked a series of questions concerning, among other things, their use of alcohol and tobacco when they were pregnant with this child. These questions are similar to those used in the NLSCY and other population surveys and are presented in Table 1. The questions concerned mothers' reports of alcohol use and cigarette smoking during their pregnancy, as well as indications of high-risk problem drinking using the four questions from the CAGE questionnaire (Ewing, 1984), and are described below.

Responses to the alcohol-use questions were categorized as "never drank," "drank less than once per month," "drank more than once per month." Responses to the questions concerning cigarette smoking during pregnancy were categorized as "never smoked," "smoked less than ½ pack per day," "smoked more than ½ pack per day."

Table 1

Questions in Parent Interview Concerning Maternal Alcohol Use and Smoking

ALCOHOL

1. Did you drink alcohol during your pregnancy?
a. If yes, did you change the amount you drank while you were pregnant?
b. How often did you drink alcohol during this pregnancy?
c. When you drank alcohol during this pregnancy, how many drinks would you have, on average, each time?
CAGE 1. Did you ever feel that you ought to cut down on your drinking?
CAGE 2. Did people annoy you by criticizing your drinking?
CAGE 3. Did you ever feel bad or guilty about your drinking?
CAGE 4. Did you ever have a drink first thing in the morning to steady your nerves or get rid of a hangover?

SMOKING

1. Did you smoke cigarettes during your pregnancy?
a. If yes, how many cigarettes did you smoke on a typical day during this pregnancy?
b. Did you change your smoking pattern during this pregnancy?
c. If you stopped smoking, in which month of pregnancy did you stop?
2. Did any of the other people living in your household smoke cigarettes during your pregnancy?
a. If yes, how many?

3.4 Sample Size

The size of the longitudinal sample at the various data collection points (i.e. child ages 33 months, 48 months, Grade 1 and Grade 3) for which prenatal alcohol and tobacco use responses were available appear in Table 2 for alcohol use and Table 3 for tobacco use. The attrition in the longitudinal sample was approximately 19% from 48 months to Grade 3. Analyses of differences between families that were maintained in the dataset compared with those that dropped out yielded no indication of bias resulting from sample attrition (see Peters et al., 2000 for a thorough discussion of these attrition analyses). More specifically, with regard to the data analyzed for the present study, there were no differences between the retained sample and those lost in terms of mothers' reports of smoking or drinking patterns during pregnancy.

Table 2

Longitudinal Sample Sizes: Alcohol Use During Pregnancy

Mother's reported drinking during pregnancy		Child age at time of longitudinal data collection			
		33 Mos.	48 Mos.	Gr. 1	Gr. 3
Never	*N* (%)	414 (77.4)	399 (76.9)	343 (77.1)	330 (78.4)
< once/mo.	*N* (%)	89 (16.6)	88 (17.0)	75 (16.9)	67 (15.9)
> once/mo.	*N* (%)	32 (6.0)	32 (6.2)	27 (6.1)	24 (5.7)
Total	*N* (%)	535 (100)	519 (100)	445 (100)	421 (100)

Table 3

Longitudinal Sample Sizes: Tobacco Use During Pregnancy

Mother's reported smoking during pregnancy (# cig./day)		Child age at time of longitudinal data collection			
		33 Mos.	48 Mos.	Gr. 1	Gr. 3
None	*N* (%)	352 (68.2)	345 (68.9)	290 (66.4)	275 (67.6)
< ½ pack	*N* (%)	118 (22.9)	110 (22.0)	101 (23.1)	88 (21.6)
> ½ pack	*N* (%)	46 (8.9)	46 (9.2)	46 (10.5)	44 (10.8)
Total	*N* (%)	516 (100)	501 (100)	437 (100)	407 (100)

As shown in Tables 2 and 3, the sample size when the children were 48 months of age is approximately 500; the sample was reduced to 407 by Grade 3. Over 20% of the sample at each point in time consisted of mothers who reported some prenatal alcohol consumption; over 30% reported some prenatal tobacco exposure. Approximately 6% of these mothers reported using alcohol more than once per month during pregnancy, and 9% reported smoking more than ½ pack of cigarettes per day. These rates are higher than those reported in the Canadian Community Health Survey (see Section 2).

Approximately 99% of the children were residing with their biological mother at age 3 months, and this decreased slightly over time to 97% at age 33 months and 96% at Grade 1 and Grade 3. Due to the small number of children living with a foster parent or guardian at Grade 1 (N = 12) and Grade 3 (N = 9), it was not possible to analyze the data to see if those living with non-biological parents differed in exposure to prenatal alcohol or tobacco when compared with those living with a biological parent. Thus, the results of analyses reported here apply almost exclusively to children who were living with at least one biological parent from birth to Grade 3.

3.5 Sample Definition

3.5.1 Tobacco Use

Due to the relatively imprecise data on the number of cigarettes smoked daily reported by the mothers, two categories of prenatal smoking were formed: those mothers that reported *any* smoking during pregnancy and those that reported *no* smoking. Thus, the smoking sample includes women who reported smoking less than a half pack per day (about two-thirds of the mothers) as well as heavier smokers.

3.5.2 Alcohol Use

For prenatal alcohol consumption, several ways of categorizing the mothers' reports of alcohol use were explored in conjunction with several of the child outcome measures. The most sensitive measure was whether the mother answered "Yes" to one or more of the four CAGE questions (see Table 1). If she did, she was considered a higher-risk drinker (MHRD) during pregnancy. If not, she was considered lower risk. The decision to use this method of identifying children who were exposed to higher versus lower risk of prenatal alcohol was based on several studies that indicated the use of scores of 1 or greater on the CAGE as being the most sensitive to problem drinking in women while scores of 2 or greater on the four CAGE questions have been found to be most sensitive to higher-risk drinking in men (Bradley, Boyd-Wickizer, Powell & Burman, 1998; Midanik, Zahnd & Klein, 1998; Moraes, Viellas & Reichenheim, 2005).

Table 4

Sample Sizes for the Four Groups of Mothers Regarding Drinking and Smoking During Pregnancy

Data cycles	Smoking during pregnancy	Drinking during pregnancy (CAGE)		
		Lower-risk drinker	Higher-risk drinker	Total
33 Months	Didn't smoke	302	22	324
	Smoked	122	24	146
	Total	424	46	470
	Chi-sq test (Exact test): χ^2 (1) = 10.6, p<0.001			
48 Months	Didn't smoke	298	21	319
	Smoked	115	24	139
	Total	413	45	458
	Chi-sq test (Exact test): χ^2 (1) = 12.5, p<0.001			
Grade 1	Didn't smoke	249	16	265
	Smoked	107	23	130
	Total	356	39	395
	Chi-sq test (Exact test): χ^2 (1) = 13.3, p<0.001			
Grade 3	Didn't smoke	241	13	254
	Smoked	97	21	118
	Total	338	34	372
	Chi-sq test (Exact test): χ^2 (1) = 15.6, p<0.001			

Note that this is a behavioural definition of higher risk based on reported feelings of guilt, annoyance, sober second thought ("I ought to cut back") and hangover avoidance by morning drinking. The guilt, annoyance and second-thought criteria are likely to have captured a substantial number of cases where the amount of drinking was moderate as well as the behaviour of very heavy drinkers. We did not try to quantify the amount of alcohol consumed in any of the analyses reported here.

There is emerging evidence that the most severe harmful effects of prenatal alcohol exposure result from mothers' binge drinking rather than from more regular or more frequent light or moderate consumption. Although more research is needed on more subtle outcomes resulting from prenatal exposure to lower concentrations of alcohol, the higher-risk versus lower-risk dichotomy of mothers' prenatal alcohol consumption based on a CAGE score of 1 or more was adopted for analyses of children's prenatal exposure to alcohol in this study.

3.5.3 Prevalance of Alcohol and Tobacco Use During Pregnancy

This strategy divided mothers into four groups regarding alcohol use and smoking during pregnancy: 1) higher-risk drinking, smoking; 2) higher-risk drinking, non-smoking; 3) lower-risk drinking, smoking; 4) lower-risk drinking, non-smoking (see Table 4 for sample sizes).

The chi-square statistical test results reported at each age reflect a highly statistically positive relationship between mothers' reports of prenatal smoking and their reports of high-risk drinking.

We decided that the BBBF longitudinal dataset contained enough detailed information on mothers' alcohol and tobacco use during pregnancy to warrant further analyses concerning relationships with children's development, school readiness and functioning during early primary school. The sample sizes were considered to be adequate to allow analyses of the independent and combined association between prenatal exposure to alcohol and to tobacco, with a wide range of measures of child development.

3.6 Measures of Child Development

The BBBF dataset has measures of five major domains of children's development.

1. Children's general development
2. Cognitive development/ academic performance,
3. Social/emotional functioning
4. Behaviour problems
5. Child health

These domains correspond closely with the five domains of school readiness currently employed in Canada (Janus & Offord, 2000). A total of 79 child outcome measures were selected for preliminary analysis. Most of these measures were collected when the children were 48 months, 6 years (Grade1) and 8 years (Grade 3) of age. Three of the measures had been collected when the children were 33 months old. The specific child outcome measures selected for analysis are listed in Appendix 1 for each of the five domains of child development.

4. Data Analysis

4.1 Data Analysis Part 1: ANCOVA

Analyses of the 79 child outcome measures were carried out using Analyses of Covariance (ANCOVA) with the mother's drinking alcohol and smoking during pregnancy as the two independent variables. The general idea of ANCOVA is to use statistical methods to create a level playing field for the comparisons of groups. The measures used as covariates in the ANCOVA analyses were those that might bias the results due to factors other than smoking or drinking during pregnancy. By including these measures in the analyses, statistical controls were used to remove any bias these variables may have had on the differences between groups. A complete list of the measures used as covariates in the analyses appears in Appendix 2, and includes measures of family income, maternal education, immigrant status, home language and single-parent status.

With the four prenatal smoking and drinking groups statistically equated on the covariate variables, we could then compare the average results for different groups with increased confidence. We compared the averages in a statistical manner so that we were less tempted to seize on a false result that favours our hypotheses.

4.1.1 Statistical Significance

It is standard practice to report the results of statistical analyses in terms of significance levels or *p* values. In the current analyses, the significance level (*p* value) is the probability that the difference between groups on any given measure is due to chance factors alone. If the *p* value is low (i.e. .01 or less), we can conclude that differences between groups are likely to be due to differences in whether or not mothers drank or smoked during pregnancy rather than being due to chance. If the *p* value is .01 or less, we conclude that the group differences are statistically significant. Statistically significant results allow us to say something similar to the phrase used in consumer polls; we will be right at least 99 times out of 100 whenever we say that the averages of two groups are in fact different, and not due to chance. We chose to use a conservative *p* value of .01 because of the large number of tests we were reporting.

4.1.2 Effect Size

The effect size reflects how large average differences are across different variables in a standardized manner. One of the problems with using many different measures is that the numbers used mean different things from one measure to another. A difference of 10 points means one thing in a depression score and another in an IQ score. In an effort to produce numbers that mean the same thing from measure to measure, we calculated a statistic called an effect size (more specifically a d statistic). When we compare two groups of children, the d statistic allows us to express the difference between the two groups in units determined by the variability of the children within their groups. This gives a common metric across measures and effectively allows us to compare "apples to oranges."

In social and health science research, it is convention to consider effect size (E.S.) indices as small if the value is between .2 and .5; medium if between .5 and .8; and large if the E.S. is .8 or greater. We report effect sizes for our all analyses where the data are available (including statistically significant and non-significant results). Note that for non-significant effect sizes, we have no confidence that the observed value of the effect size is dependably greater than 0.0.

4.1.3 Analysis Process

Each of the child outcome measures was analyzed three ways. First, outcomes for children of the smoking mothers were compared with children of the non-smoking mothers. This allowed for a comparison of children exposed to any prenatal tobacco to those exposed to none. A **second** analysis compared outcomes for children of the high-risk drinking mothers to those of low-risk drinking mothers. A **third** analysis compared children of mothers who were both high-risk drinkers and smokers to those who were low-risk drinkers and non-smokers. This comparison allowed for an assessment of the outcomes of children exposed to both prenatal tobacco and high levels of alcohol.

4.2 Data Analysis Part 2: Structural Equation Modelling

For the Structural Equation Modelling (SEM) analysis of the relationship between measures of maternal tobacco and alcohol consumption and later internalizing and externalizing behaviours, we limited the analysis to the four measures of smoking and drinking behaviour (and two arithmetic products of those measures), four measures of externalizing behaviours and two measures of internalizing behaviours collected from the teachers of the children and the same from the parents of the children.

The earliest measures were the tobacco use and alcohol use measures collected from mothers when the children were 3 months old. These are the same measures described in Table 1, and were coded dichotomously (i.e. higher-risk drinking versus no high-risk drinking; any smoking during pregnancy versus no smoking). The product of the two measures was used as a third variable, sensitive to an interaction between tobacco and alcohol use. Note that the product gave the non-drinkers and non-smokers the same value as the non-drinking smokers and the non-smoking drinkers. Thus, these "one substance only" cases were included in the SEM analysis while they were omitted from the ANCOVA analyses described above.

When the children were 33 months old, we also collected data on maternal alcohol use and smoking in the home (as an indication of exposure to second-hand smoke). These were coded dichotomously and the product computed. This gave us a total of six measures of smoking and drinking behaviour, three collected at each of two times.

When the child was in Grade 3, we collected a large array of measures of child behaviour and social and emotional functioning, as described above. From that list of measures, we chose six parent report measures and six teacher measures that would allow us to estimate externalizing behaviour and internalizing behaviour. The measures are listed below.

4.2.1 Parent Measures

Internalizing measures **(distress and emotion)**

- OCHS parent-rated depression scale
- NLSCY parent-rated emotional disorder scale

Externalizing measures **(misbehaviour and problem behaviour)**

- OCHS parent-rated oppositional defiant scale
- NLSCY parent-rated indirect aggression scale
- NLSCY parent-rated hyperactive scale
- NLSCY parent-rated physical aggression scale

4.2.2 Teacher Measures

Internalizing measures **(distress and emotion)**

- OCHS teacher-rated passive victimization scale
- NLSCY teacher-rated emotional disorder scale

Externalizing measures **(misbehaviour and problem behaviour)**

- NLSCY teacher-rated delinquency scale
- NLSCY teacher-rated indirect aggression scale
- NLSCY teacher-rated hyperactivity scale
- NLSCY teacher-rated physical aggression scale

4.2.3 Procedure

We controlled for the same covariates described above by computing the covariate adjusted residuals for our Grade 3 parent and teacher variables (12 measures).

The data were analyzed using AMOS 17.0 (SPSS; Levesque 2007). Although AMOS does not have an option for selecting list-wise/pair-wise deletion, it can handle missing cases using a method called "Full Information Maximum Likelihood" (FIML, also known as "Raw Maximum Likelihood"), which is the technique that we used to deal with missing cases. This technique leads to indefinite sample sizes because while 502 people contributed data, only 177 have complete data for all 18 variables. Classical list-wise deletion would have limited the analysis to the information provided by the 177 subjects with complete data. The FIML procedure uses all the information available from the 502 subjects while assessing for bias imposed by the procedure. Given that the missing data were randomly missing, this technique is more efficient.

The SEM analysis was broken into segments to simplify the process. One segment modelled the relationships among the alcohol and tobacco measures, another tackled the internalizing and externalizing measures.

5. Results

5.1 Results of the ANCOVA

ANCOVA analyses were carried out on a total of 79 child outcome measures collected as part of the BBBF longitudinal study when children were 33 months, 48 months, 6 years (Grade 1) and 8 years (Grade 3) of age. Each measure was independently analyzed using Analysis of Covariance (ANCOVA) procedures. For each measure, children exposed to tobacco during pregnancy were compared with those not exposed, yielding the Prenatal Exposure to Tobacco, or PET effect. Also, children whose mothers were considered higher risk for problem drinking during pregnancy based on a CAGE score of 1 or higher were compared with children whose mothers were considered lower risk, the Maternal Higher-Risk Drinker, or MHRD effect. Finally, for each measure, the PET and MHRD group was compared with the non-PET and non-MHRD group. This comparison was designed to determine the effects of the combination of prenatal exposure to tobacco plus maternal higher-risk drinking during pregnancy. For all analyses, the set of family socio-economic, cultural and demographic variables listed in Appendix 2 were employed as covariates to eliminate confounding effects of these variables associated with maternal prenatal tobacco use or higher-risk drinking. Appendix 1 presents a summary of the results of the three statistical comparisons of all 79 child outcome measures.

Since these three hypotheses are all directional in nature, one-tailed statistical tests of significance were used for all analyses. Also, the results in Appendix 1 are presented in terms of whether each analysis yielded a difference in means in the hypothesized direction (i.e. poorer performance represented by a negative sign (–), or a difference in means in the opposite direction from that hypothesized, represented by a plus sign (+)).

In this section, all statistically significant results are presented and described (the non-significant (NS) results are reported in Appendix 1). For each statistically significant effect (p <.01), the effect size (E.S.) is presented where the E.S. reflects how large the mean difference is in standard deviation units.

The three major hypotheses tested in the ANCOVA analyses included:

1. Children with high-risk drinking mothers would show poorer developmental outcomes than those with low-risk-drinking mothers.
2. Children whose mothers smoked during pregnancy would show poorer developmental outcomes than those whose mothers did not smoke.
3. Children whose mothers were both high-risk drinkers and smokers during pregnancy would show the greatest developmental problems during primary school.

▸ In social and health science research, an E.S. of .2 to .5 is considered small, .5 to .8 moderate, and >.8 is considered large (Cohen, 1977).

The results are presented separately according to the domain of child functioning reflected by the measures. Group means, standard errors and sample sizes for the analyses of all variables are presented in Appendix 3.

5.1.1 Child General Development

As summarized in Table 5, statistically significant results occurred for all three measures of children's general development: the Developmental Inventory for Screening Children (DISC) Overall score at 33 and 48 months of age and the ABC School Readiness. **Children exposed to tobacco and higher-risk alcohol use by their mothers during pregnancy showed significantly poorer outcomes on the three measures of general development compared with the group of children who were not exposed to either tobacco or high-risk maternal drinking during pregnancy** (i.e. the PET and MHRD comparison).

> The effect sizes for these group differences ranged from –.50 to –.65, differences that are considered to be moderate in size.

There were no statistically significant effects of exposure to tobacco alone, or exposure to higher-risk maternal alcohol use alone.

Table 5

General Child Development: Effect Sizes for Statistically Significant (p < .01, 1-tailed) Measures

Measure and Age of Child	Prenatal Exposure to Tobacco (PET)	Mother Higher risk Drinker (MHRD)	PET and MHRD
At 33 months of age (1 measure)			
Developmental Inventory for Screening Children (DISC) Overall Development Quotient (E= measure collected directly from the child by a trained researcher)	N.S.	N.S.	-.57
At 48 months (2 measures)			
DISC overall Developmental Quotient (E)	N.S.	N.S.	-.50
ABC School Readiness (T=Teacher rating)	N.S.	N.S.	-.65
Summary (3 measures)			
# significant / # of tests	0/3	0/3	3/3
% significant	0%	0%	100%

E = measure collected directly from trained researcher
T = measure collected from teacher

5.1.2 Children's Cognitive Development and Academic Performance Measures

There were a total of 23 measures analyzed in the domain of children's cognitive development and academic performance, and the results of these analyses are summarized in Table 6. Of the 23 measures, 7 showed statistically significant differences, with the children exposed to both tobacco and mother's higher-risk drinking during pregnancy showing poorer performance when compared with the group that was exposed to neither. **Six of these group differences yielded effect sizes in the moderate range (.52–.69), but the measure of teacher ratings of the child's school preparedness in Grade 3 yielded a much larger difference, with an effect size of 1.03**. (Note: E = directly

Table 6

Cognitive Development and Academic Achievement: Effect Sizes for Statistically Significant ($p < .01$, 1- tailed) Measures

Measure and Age of Child	Prenatal Exposure to Tobacco (PET)	Mother Higher risk Drinker (MHRD)	PET and MHRD
33 months of age (1 measure)			
None Significant			
48 months (3 measures)			
DISC Auditory of Memory Scale (E)	N.S.	–.44	–.63
Grade 1 (8 measures)			
None significant			
Grade 3 (11 measures)			
School preparedness (T)	N.S.	–.69	–1.03
Attitudes toward academics (T)	N.S.	–.53	–.69
Academic functioning (T)	N.S.	–.58	–.65
Adaptive functioning (T)	N.S.	N.S.	–.62
Suspended from school (P = Parent rating)	N.S.	N.S.	–.56
Special ed. services (T)	N.S.	N.S.	–.64
Summary (23 Measures)			
# significant/# of tests	0/23	4/23	7/23
% significant	0%	17%	30%

T = teacher-rated measure
P = parent-rated measure

from trained researcher, T = teacher, and P = parent indicate the source of each measure). Of the 7 significant effects, 6 were on measures collected when the children were in Grade 3, and 5 of these 6 were based on ratings by the child's teacher.

There were also 4 significant negative effects of children exposed to higher-risk mother's drinking during pregnancy. There were no differences on any of the 23 measures associated with exposure to tobacco during pregnancy.

Table 7

Social/Emotional Functioning: Effect Sizes for Statistically Significant ($p < .01$, 1-tailed) Measures

Measure and Age of Child	Prenatal Exposure to Tobacco (PET)	Mother Higher risk Drinker (MHRD)	PET and MHRD
33 months (1 measure)			
None significant			
48 months (6 measures)			
None significant			
Grade 1 (8 measures)			
None significant			
Grade 3 (N=8)			
Emotional disorder (T)	N.S.	–.65	–.80
Conflict management (T)	N.S.	N.S.	–.60
Summary			
# significant/# of tests	0/23	1/23	2/23
% significant	0%	4%	9%
T = teacher-rated measure			

5.1.3 Children's Social/Emotional Functioning Measures

A total of 23 measures of various aspects of children's social and emotional functioning were analyzed and the results are summarized in Table 7. **Only 2 measures yielded statistically significant differences – teachers' ratings of children's emotional problems and their ability to manage conflict with peers at Grade 3.** Children exposed to both tobacco and higher-risk maternal drinking during pregnancy showed higher levels of emotional problems and poorer conflict management, as rated by their teachers, than those children exposed to neither. Also, children exposed to higher-risk maternal drinking showed higher levels of emotional problems in Grade 3 than children not exposed to higher-risk maternal drinking. Again, there was no indication of compromised social or emotional functioning associated with children being exposed to tobacco during pregnancy.

Table 8

Child Health Measures, Statistically Significant ($p < .01$, 1-tailed) Measures

Measure and Age of Child	Prenatal Exposure to Tobacco (PET)	Mother Higher risk Drinker (MHRD)	PET and MHRD
48 months (3 measures)			
None significant			
Grade 1 (3 measures)			
Child exposed to second-hand smoke (P)	–.79	N.S.	–.83
Grade 3 (3 measures)			
Child exposed to second-hand smoke (P)	–.64	N.S.	–.49
Summary			
# significant/# of tests	2/9	0/9	2/9
% significant	22%	0%	22%
P = parent-rated measure			

Table 9

Child Behaviour Problems: Effect Sizes for Statistically Significant ($p < .01$, 1-tailed) Measures

Measure and Age of Child	Prenatal Exposure to Tobacco (PET)	Mother Higher risk Drinker (MHRD)	PET and MHRD
33 months (1 measure)			
None significant			
48 months (5 measures)			
Disruptive behaviour (T)	N.S.	–.52	–.83
Hyperactivity (T)	N.S.	–.52	–.88
Indirect aggression (T)	N.S.	–.61	–.84
Physical aggression (T)	N.S.	N.S.	–.85
Grade 1 (8 measures)			
Delinquency (T)	N.S.	–.69	N.S.
Grade 3 (8 measures)			
Physical aggression (P)	N.S.	N.S.	–.59
Hyperactivity (T)	N.S.	–.56	–.77
Indirect aggression (T)	N.S.	N.S.	–.86
Physical aggression (T)	N.S.	–.49	–.76
Delinquency (T)	N.S.	–.66	–.98
Summary			
# significant/# of tests	0/22	6/22	10/22
% significant	0%	27%	45%

T = teacher-rated measure

P = parent-rated measure

5.1.4 Children's Health Measures

Of the 9 measures reflecting children's health at various ages, only the measure of children being exposed to second-hand smoke yielded statistically significant effects in Grades 1 and 3. This is not, strictly speaking, a child outcome measure; instead, it reflects the fact that children whose mothers reported smoking during pregnancy as well as those who reported smoking and higher-risk drinking during pregnancy also indicated that their children were exposed to more second-hand smoke when in Grades 1 and again in Grade 3. No other measure of child health showed any indication of negative effects associated with either smoking or higher-risk drinking during pregnancy.

5.1.5 Children's Behaviour Problems Measures

There were 22 measures of children's behaviour problems. As summarized in Table 9, children who were exposed to both tobacco and higher-risk maternal drinking during pregnancy showed significantly higher levels of several types of behaviour problems than children exposed to neither. **Of 22 measures analyzed, 10 were statistically significant, and 9 of the 10 significant effects were on ratings by the child's teacher, primarily at 4 years of age and again in Grade 3. Further, most of these differences were quite large, yielding effect sizes near or above .80.**

Six of the 22 behaviour problem measures were also significantly higher for children who were exposed to higher-risk maternal drinking during pregnancy compared with children not exposed. All 6 of these significant effects were on ratings by the children's teacher, 3 when the children were 4 years old and 3 when they were in Grade 3. (Note: This is covered in some detail in the discussion section.) Again, there was no indication of an association between children's prenatal exposure to tobacco and later ratings of behaviour problems.

5.1.6 Summary of Significant Child Outcomes

In this final section of results, we attempt to summarize the main findings of the analyses just described. The first summary is presented in Table 10. Here we show the number and percentages of outcome measures in each child domain that yielded statistically significant results.

The first column presents the results of comparisons between children who were exposed to some tobacco prenatally and those exposed to no tobacco, the PET effect. Out of the 79 measures analyzed, only 2 were statistically significant. Those were the measures of exposure to second-hand smoke at Grades 1 and 3, a finding that indicates the children of mothers who smoked during pregnancy were exposed to more second-hand smoke at ages 6 and 8 than children of mothers who did not smoke during pregnancy. There were no other significant outcomes in any of the other 79 measures associated with smoking versus no smoking during pregnancy. As discussed previously, although exposure to second-hand smoke is

Table 10

Summary of Statistically Significant ($p < .01$, 1-tailed) Child Outcome Effects by Domain

Child Domain Measured	Prenatal Exposure to Tobacco (PET)	Mother Higher risk Drinker (MHRD)	PET and MHRD
Child Development (3 measures)			
# significant/# of tests	0/3	0/3	3/3
% significant	0%	0%	100%
Average of significant effects			–.57
Average of all effect sizes	–.29	–.28	–.57
Cognitive Development/Academic Performance (23 measures)			
# significant/# of tests	0/23	4/23	7/23
% significant	0%	17%	30%
Average of significant effects		–.56	–.69
Average of all effect sizes	–.18	–.29	–.40
Social/Emotional Functioning (22 measures)			
# significant/# of tests	0/22	1/22	2/22
% significant	0%	4%	9%
Average of significant effects		–.65	–.70
Average of all effect sizes	–.13	–.18	–.27

Table 10 (cont'd)

Summary of Statistically Significant ($p < .01$, 1-tailed) Child Outcome Effects by Domain

Child Domain Measured	Prenatal Exposure to Tobacco (PET)	Mother Higher risk Drinker (MHRD)	PET and MHRD
Child Health (9 measures)			
# significant/# of tests	2/9	0/9	2/9
% significant	22%	0%	22%
Average of significant effects	–.72		–.66
Average of all effect sizes	–.23	–.07	–.20
Behaviour Problems (22 measures)			
# significant/# of tests	0/22	6/22	10/22
% significant	0%	27%	45%
Average of significant effects		–.56	–.81
Average of all effect sizes	–.25	–.28	–.50
Summary (Total of 79 measures)			
# significant/# of tests	2/79	11/79	24/79
% significant	2%	14%	30%
Average of significant effects	–.72	–.59	–.72
Average of all effect sizes	–.20	–.23	–.27

unhealthy, it is not truly a child health outcome such as asthma or physical illness, so this effect needs to be viewed with some caution.

Column 2 summarizes the results of comparisons between children whose mothers engaged in higher-risk drinking during pregnancy versus children of mothers who were not higher-risk drinkers, the MHRD effect. Eleven of the 79 analyses (14%) were statistically significant. Six of these 11 effects involved high levels of behaviour problems for children of higher-risk drinking mothers, and 4 involved poorer cognitive development for this group. Thus, the negative effects associated with children of mothers who engaged in higher-risk drinking during pregnancy are manifested primarily in poorer cognitive and academic functioning and also greater manifestation of behaviour problems such as aggression and hyperactivity.

Column 3 of Table 10 (PET and MHRD) summarizes the results of the comparison between the children who were exposed to both maternal smoking and higher-risk drinking

Table 11

Summary of Statistically Significant ($p < .01$, 1-tailed) Child Outcome Effects by Child's Age

Age of Child	Prenatal Exposure to Tobacco (PET)	Mother Higher risk Drinker (MHRD)	PET and MHRD
33 Months (3 measures)			
# significant/# of tests	0/3	0/3	1/3
% significant	0%	0%	33%
Mean significant effect size			–.57
48 Months (19 measures)			
# significant/# of tests	0/19	4/19	7/19
% significant	0%	21%	37%
Mean significant effect size		–.52	–.74
6 years (Grade 1) (27 measures)			
# significant/# of tests	1/27	0/27	2/27
% significant	4%	0%	9%
Mean significant effect size	–.79		–.76
8 years (Grade 3) (30 measures)			
# significant/# of tests	1/30	7/30	14/30
% significant	3%	20%	47%
Mean significant effect size	–.64	–.59	–.72
Summary (Total of 79 measures)			
# significant/# of tests	2/79	11/79	24/79
% significant	2%	14%	30%
Mean significant effect size	–.72	–.59	–.72

during pregnancy versus children who were not exposed to either smoking or higher-risk drinking. Here 24 of 79 or 30% of the statistical comparisons were significant and, as for the MHRD comparisons, the differences were most pronounced in poorer cognitive and academic functioning as well as higher levels of behaviour problems.

In Table 11, the results of the analyses are reorganized according to the age of the child when the statistically significant effects occurred. The picture that emerges from the results in Table 11 is clear. The poorer performance of children exposed to higher-risk maternal drinking during pregnancy, either alone or in combination with prenatal tobacco exposure, occurred predominantly on measures collected when children were 4 years of age or 8 years of age, with very few effects noted at age 6 (Grade 1).

The final way in which we summarized the significant findings is in terms of the three data collection sources. Some measures were collected directly from the child by trained researchers in each neighbourhood. These measures included standardized cognitive and language lists such as the Peabody Picture Vocabulary Test (PPVT); the Wechsler Block Design Test; the Developmental Inventory for Screening Children (DISC), and height, weight, EQAO reading, math and writing scores from school records at Grade 3.

Many of the measures were collected from parents through a lengthy in-home interview.

- Of the 79 measures analyzed for this report, 11 were collected by the local site researchers, directly or indirectly from the child.

- Of the 79 child outcome measures, 31 were based on parents' reports, while 37 were provided by the child's teachers via a teacher report form that they completed on each child in the longitudinal research sample when the children were in junior kindergarten (age 4), Grade 1 (age 6) and Grade 3 (age 8) (See Discussion section.)

Table 12

Summary of Statistically Significant ($p < .01$, 1-tailed) Child Outcome Effects by Data Collection Source

Child Domain Measured	Prenatal Exposure to Tobacco (PET)	Mother Higher risk Drinker (MHRD)	PET and MHRD
Child Development (3 measures)			
Researcher collected (2)			2
Parent rated (0)			
Teacher rated (1)			1
Cognitive Development/Academic Performance (23 measures)			
Researcher collected (7)		1	1
Parent rated (4)			1
Teacher rated (12)		3	5
Social/Emotional Functioning (22 measures)			
Researcher collected (0)			
Parent rated (10)			
Teacher rated (12)		1	2
Child Health (9 measures)			
Researcher collected (2)	2		2
Parent rated (7)			
Teacher rated (0)			
Behaviour Problems (22 measures)			
Researcher collected (0)			
Parent rated (10)			1
Teacher rated (12)		6	9

Table 12 (cont'd)

Summary of Statistically Significant ($p < .01$, 1-tailed) Child Outcome Effects by Data Collection Source

Child Domain Measured	Prenatal Exposure to Tobacco (PET)	Mother Higher risk Drinker (MHRD)	PET and MHRD
Summary (Total of 79 measures)			
Researcher collected (11 measures)			
# significant/# of tests		1/11	3/11
% significant		9%	27%
Mean significant effect size		-.44	–.57
Parent rated (31 measures)			
# significant/# of tests	2/31	0/31	4/31
% significant	6%	0%	13%
Mean significant effect size	–.72		–.62
Teacher rated (37 measures)			
# significant/# of tests	0/37	10/37	17/37
% significant	0%	27%	46%
Mean significant effect size		–.58	–.77

The significant outcome results for each of these three data sources are presented in Table 12 separately for each of the five child domains. **As in previous summaries, Table 12 highlights the fact that most significant outcomes occurred on measures in the two domains of Cognitive/Academic Performance and Behaviour Problems associated with mother's higher-risk drinking during pregnancy either alone (MHRD) or also including smoking during pregnancy (MHRD and PET).**

Of the 11 statistically significant effects in the domains of cognitive/academic performance, 8 resulted from measures provided by teachers. In the domain for behaviour problems, 15 of the 16 significant effects were based on ratings provided by teachers. Overall, in all five domains of measures, there were 37 significant outcomes and 27 of these were based on teacher-provided data.

5.1.7 Summary of Major Findings of ANVOCA Analysis

The major findings from the ANCOVA analysis can be summarized as follows:

- Higher-risk drinking, as defined by scores on the CAGE screening test for alcoholism, was associated with poorer child cognitive/academic performance and more child behaviour problems during early primary school.
- The negative effects of problem drinking during pregnancy on children's academic performance and behaviour problems were exacerbated if the mothers also reported smoking cigarettes during the pregnancy.
- There was little indication of any long-term negative effects on children's behaviour associated with their mother's smoking during pregnancy. The only negative effects associated with smoking during pregnancy were greater child exposure to second-hand smoke reported by parents when the children were 6 and 8 years of age. Although this is an undesirable outcome for children, there was no indication of poorer general health, more asthma or reduced growth during primary school in children exposed to tobacco prenatally.
- There were a total of 37 statistically significant outcome effects, all of which indicated a negative relationship between prenatal higher-risk drinking alone or in conjunction with smoking. Of these 37 effects, 33 or 90% occurred on measures collected at junior kindergarten (age 4) or Grade 3 (age 8). There were virtually no negative effects noted on measures collected when the children were in Grade 1 (age 6). This is especially true if the measures of second-hand smoke are discounted.
- As noted above, there were 33 significant effects indicating a negative association between prenatal higher-risk drinking and behaviour problems in junior kindergarten and Grade 3. Of these 33 effects, 27 or 82% were based on teacher ratings of the children's academic performance and behaviour.

5.2 Results of the Structural Equation Modelling

5.2.1 Drinking and Smoking

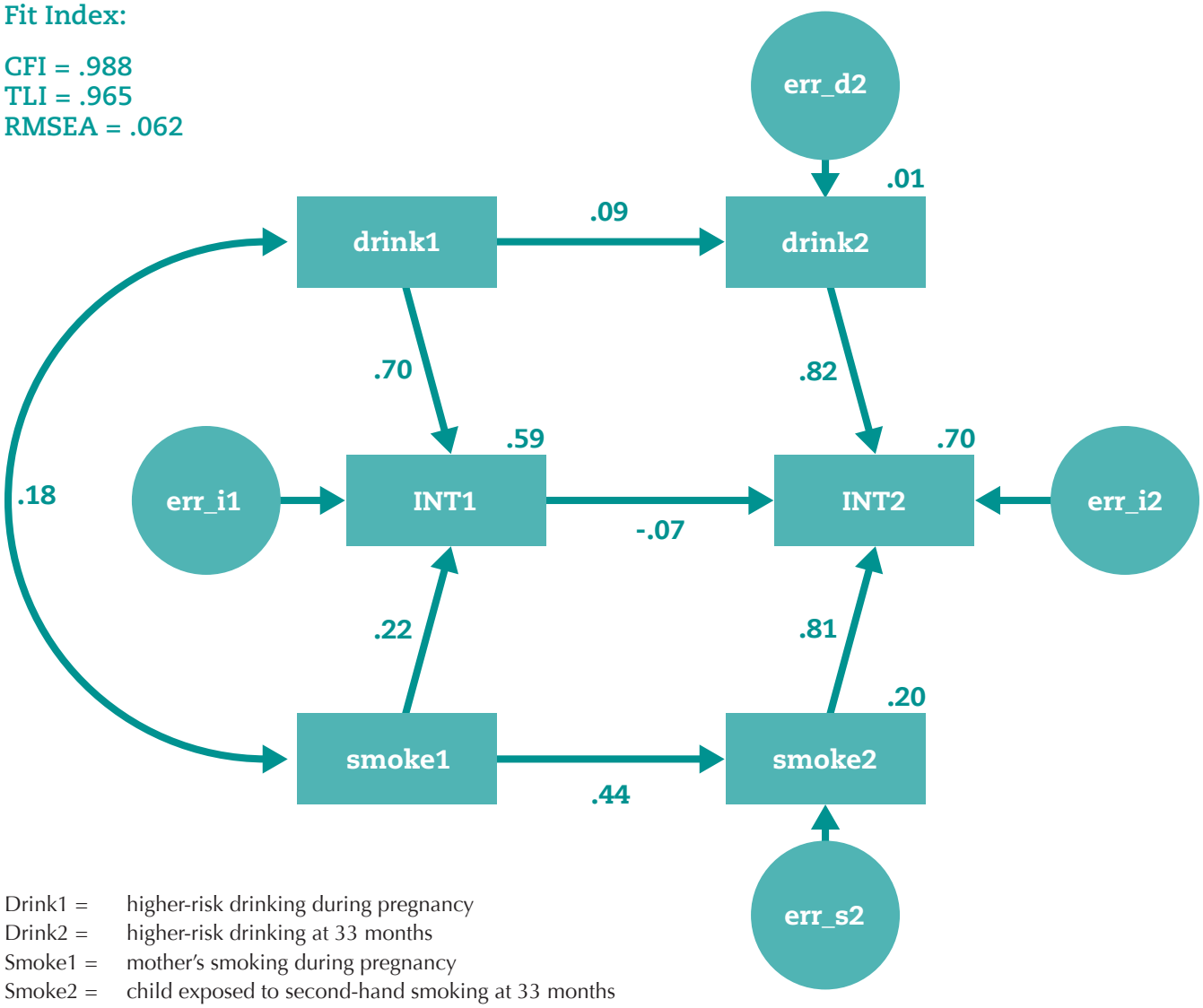
Although the modelling of the smoking and drinking measures was an exploratory analysis, we were able to use both order effects and structural relationships to simplify the model (we assumed that measures taken at 33 months did not have a causal effect on measures taken at 3 months). We placed the measures of smoking and drinking behaviour prior to the product of those two variables. All variables in this model were manifest variables (i.e. they were measured directly and included the six drinking and smoking variables listed in Figure 1). We chose to limit paths to those between measures of the same behaviour at different times and to measures of different behaviour at the same time. Figure 1 (Drinking and Smoking Structural Equation Model) shows the results of the modelling. With a Root Mean Square Error of Approximation (RMSEA) of .062 and Comparative Fit Index (CFI) and Tucker-Lewis Index (TLI) of .988 and .965, respectively, the model is a good enough fit to the data.

Figure 1

Drinking and Smoking Structural Equation Model

Fit Index:

CFI = .988
TLI = .965
RMSEA = .062



Drink1 =	higher-risk drinking during pregnancy
Drink2 =	higher-risk drinking at 33 months
Smoke1 =	mother's smoking during pregnancy
Smoke2 =	child exposed to second-hand smoking at 33 months
INT1 =	Smoke1 + Drink1
INT2 =	Smoke2 + Drink2
err =	error component. These reflect the portion of the measure that is not measuring the construct of interest, but rather some unknown or random phenomenon – hence error or disturbance.

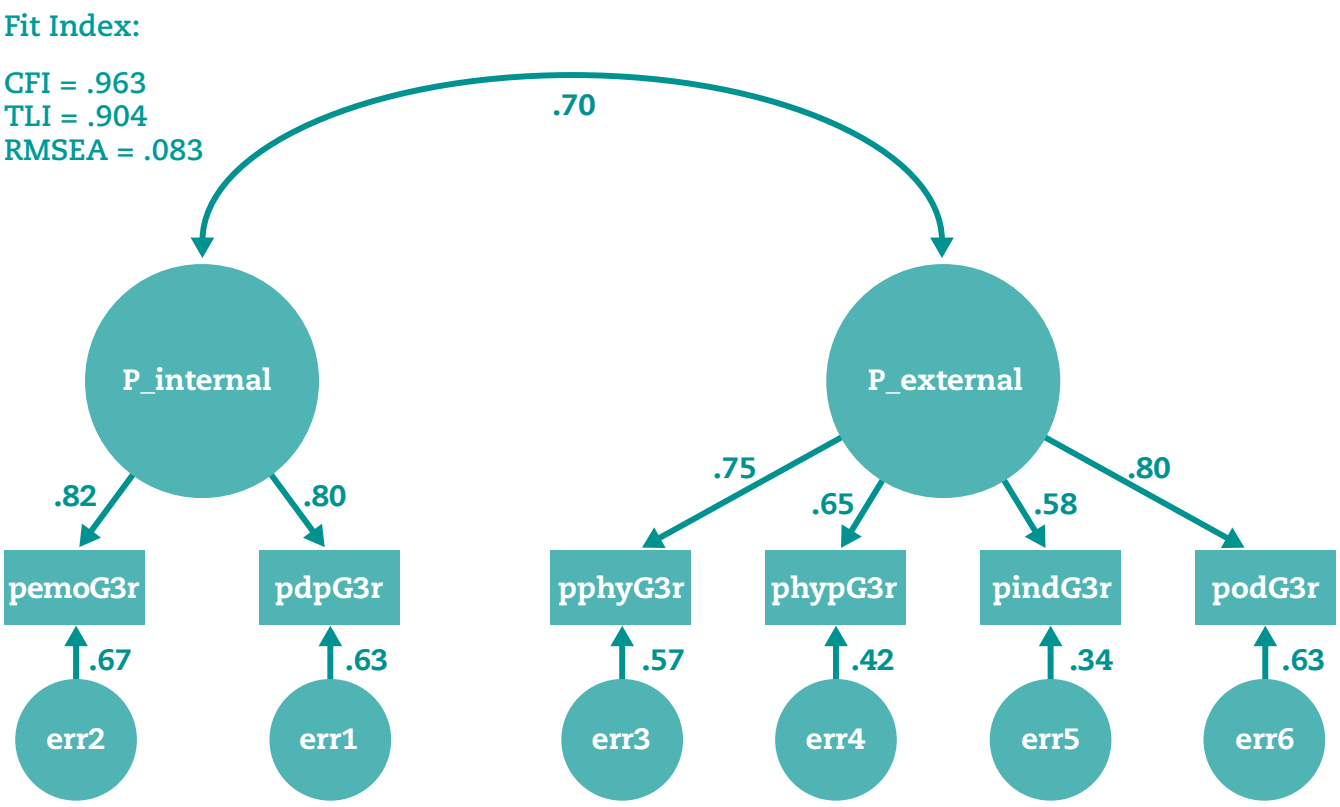
5.2.2 Parent and Teacher Ratings of Internalizing and Externalizing Behaviour

This analysis is a confirmatory factor analysis of the six measures rated by teachers and the six measures rated by parents. The internalizing/externalizing split is a well-established relationship closely associated with, but not limited to, the Achenbach measures (e.g. Achenbach & Rescoria, 2001). Our preliminary attempts at fitting models that incorporated both teacher and parent data produced either trivial or ill-fit models, so we split the models into a teacher model and a parent model, as presented below in Figure 2 (Parent Confirmatory Factor Analysis) and Figure 3 (Teacher Confirmatory Factor Analysis).

Both models have adequate, but not great goodness of fit (Teacher RMSEA = .091, CFI = .944, TLI = .852; Parent RMSEA = .083, CFI = .963, TLI = .904) with the parent fit appearing to be somewhat better than the teacher fit.

Figure 2

Parent Confirmatory Factor Analysis

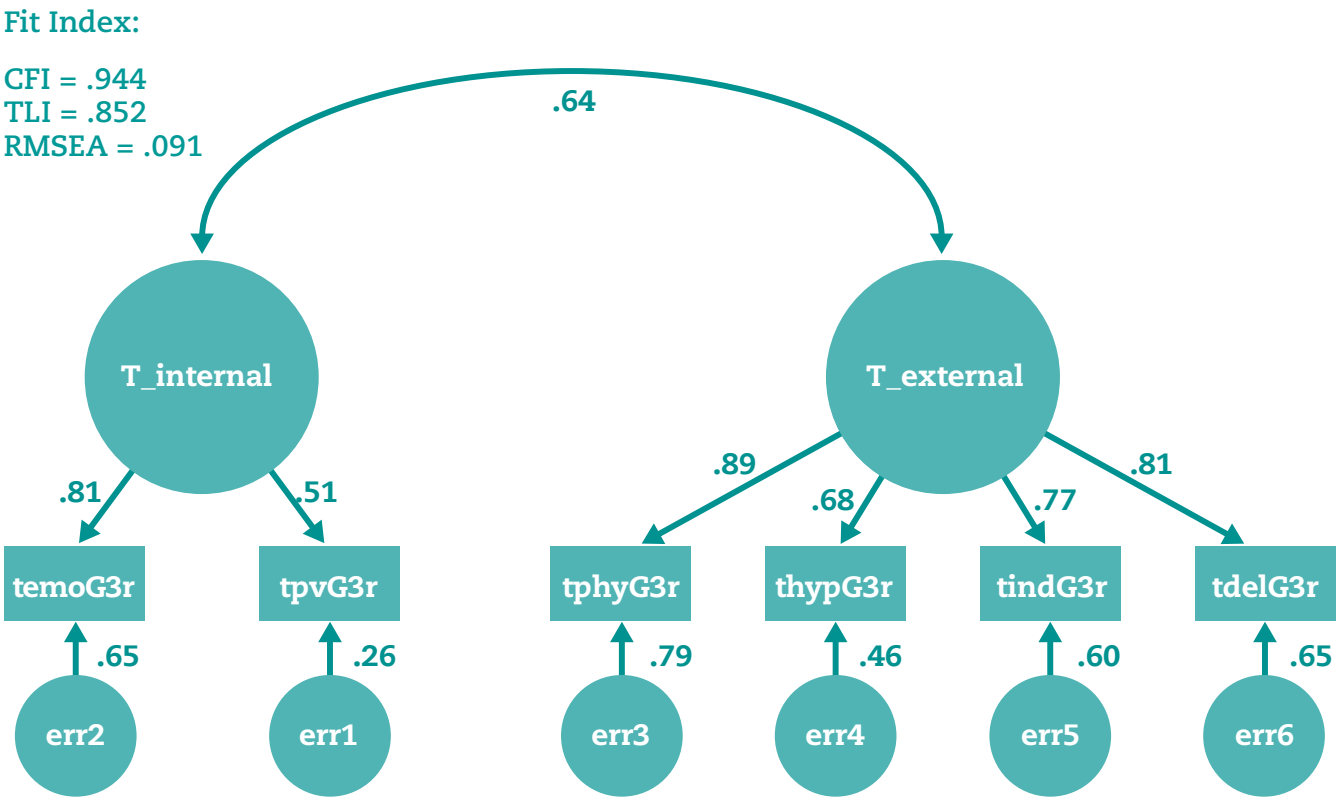


P_internal = parent ratings of internalizing behaviour problems
pemoG3r = parent ratings of emotional disorder at Grade 3
pdpG3r = parent ratings of depression scale at Grade 3
P_external = parent ratings of externalizing behaviour problems
pphyG3r = parent ratings of physical aggression at Grade 3
phypG3r = parent ratings of hyperactivity at Grade 3
pindG3r = parent ratings of indirect aggression at Grade 3
podG3r = parent ratings of oppositional defiant behaviour at Grade 3
err = error component. These reflect the portion of the measure that is not measuring the construct of interest, but rather some unknown or random phenomenon – hence error or disturbance.

Next, we linked the smoking/drinking model with the teacher model and then with the parent model to estimate paths from smoking and drinking measures to latent traits of externalizing and internalizing behaviours (the latent traits are the factors – teacher internalizing, teacher externalizing, parent internalizing and parent externalizing. They are latent in that they are not measured directly; rather, they are inferred from the behaviour of other variables). The unreduced model had 12 paths from the six predictor manifest variables to the two latent trait outcome variables. Using a reverse stepwise technique, we deleted the smallest path with p value greater than .2 until every remaining path on the diagram from the tobacco/alcohol variables to the internalizing/externalizing variables had a p value of .2 or smaller. These results are presented in Figure 4 (Parent-Reduced Model) and Figure 5 (Teacher-Reduced Model).

Figure 3

Teacher Confirmatory Factor Analysis



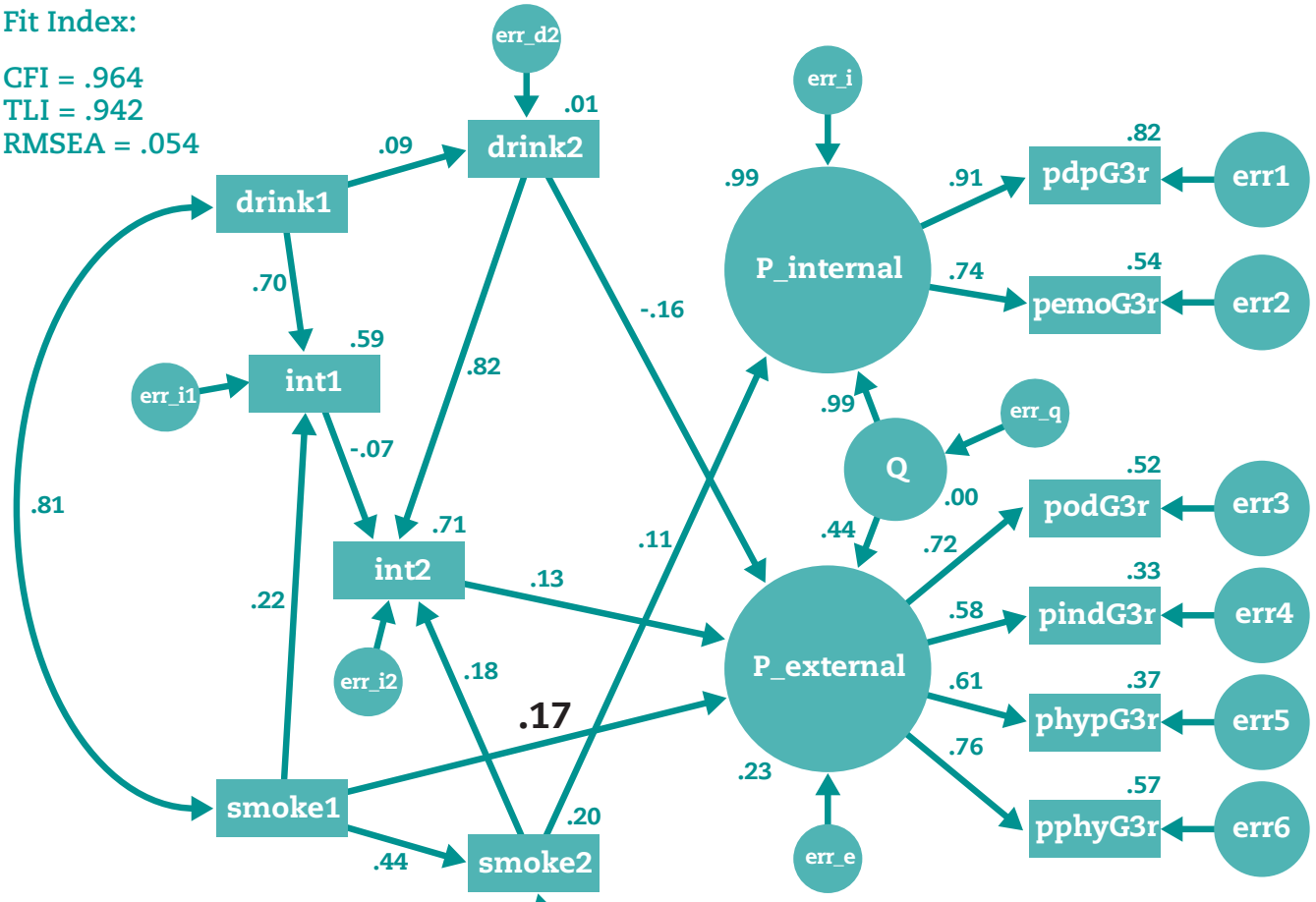
T_internal = teacher ratings of internalizing behaviour problems
temoG3r = teacher ratings of emotional disorder at Grade 3
tpvG3r = teacher ratings of passive victimization scale at Grade 3
T_external = teacher ratings of externalizing behaviour problems
tphyG3r = teacher ratings of physical aggression at Grade 3
thypG3r = teacher ratings of hyperactivity at Grade 3
tindG3r = teacher ratings of indirect aggression at Grade 3
tdelG3r = teacher ratings of delinquency at Grade 3
err = error component. These reflect the portion of the measure that is not measuring the construct of interest, but rather some unknown or random phenomenon – hence error or disturbance.

Figure 4

Parent-Reduced Model

Fit Index:

CFI = .964
TLI = .942
RMSEA = .054



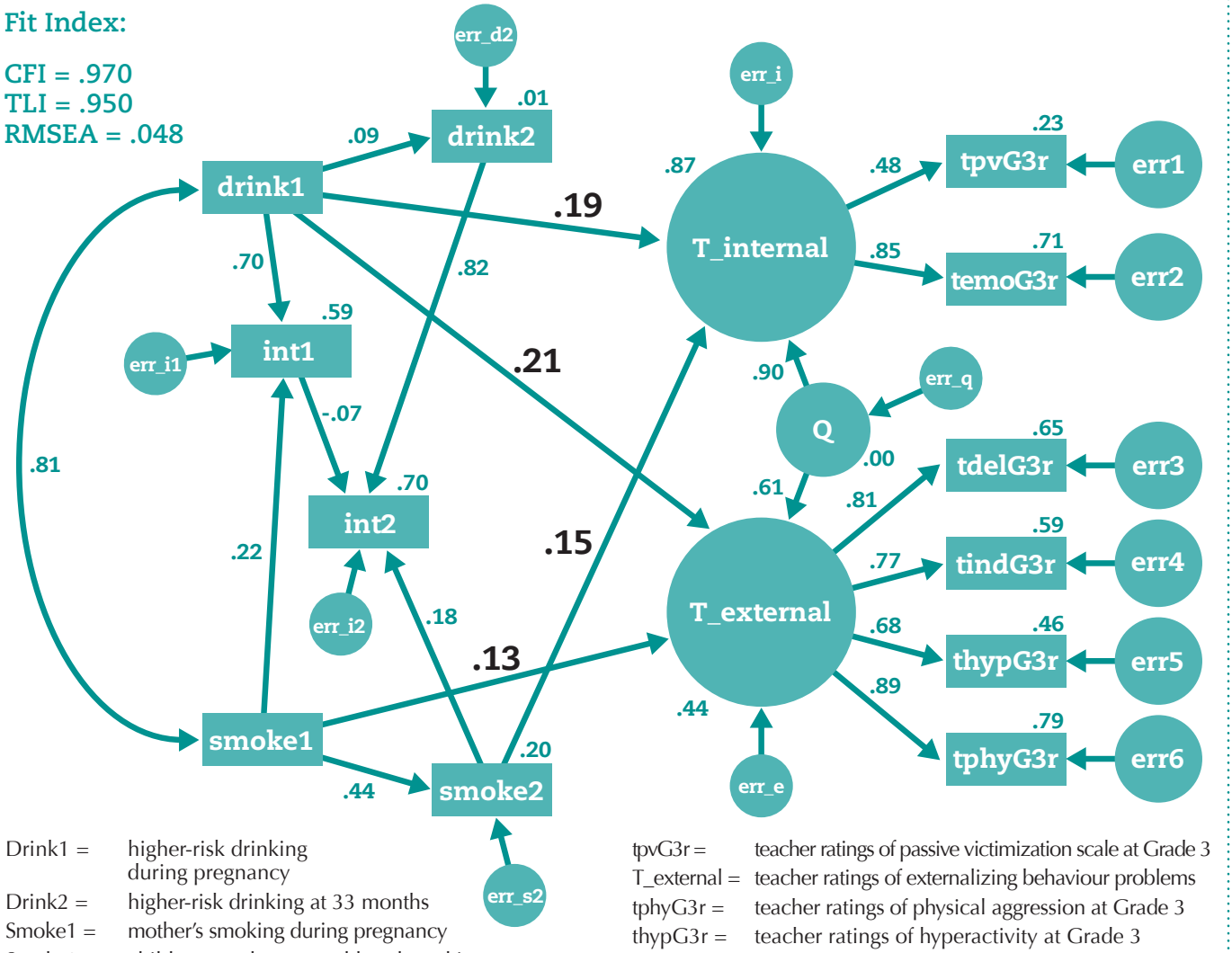
Drink1 = higher-risk drinking during pregnancy
Drink2 = higher-risk drinking at 33 months
Smoke1 = mother's smoking during pregnancy
Smoke2 = child exposed to second-hand smoking at 33 months
Int1 = Smoke1 + Drink1
Int2 = Smoke2 + Drink2
err = error component. These reflect the portion of the measure that is not measuring the construct of interest, but rather some unknown or random phenomenon – hence error or disturbance.
P_internal = parent ratings of internalizing behaviour problems
pemoG3r = parent ratings of emotional disorder at Grade 3
pdpG3r = parent ratings of depression scale at Grade 3

P_external = parent ratings of externalizing behaviour problems
pphyG3r = parent ratings of physical aggression at Grade 3
phypG3r = parent ratings of hyperactivity at Grade 3
pindG3r = parent ratings of indirect aggression at Grade 3
podG3r = parent ratings of oppositional defiant behaviour at Grade 3
Q = the portion of the relationship between the internalizing and internalizing factors that is not accounted for by the smoking and drinking measures. It is unknown. It allows the internalizing and externalizing factors to be correlated without requiring that we account for the correlation between them using our measures. The magnitude of the relationship is quite large, and smoking and drinking cannot account for all that is going on there.

Both models have good fit (Teacher RMSEA = .048, CFI = .970, TLI = .950, Parent RMSEA = .054, CFI = .964, TLI = .942), with the parent fit appearing to be somewhat better than the teacher fit. Because this is an exploratory analysis, the parent model retains three non-significant paths with $p < .2$, to facilitate replication. All of the retained paths (path coefficients) in the teacher model are significantly greater than 0.

Figure 5

Teacher-Reduced Model



Drink1 = higher-risk drinking during pregnancy
Drink2 = higher-risk drinking at 33 months
Smoke1 = mother's smoking during pregnancy
Smoke2 = child exposed to second-hand smoking at 33 months
Int1 = Smoke1 + Drink1
Int2 = Smoke2 + Drink2
err = error component. These reflect the portion of the measure that is not measuring the construct of interest, but rather some unknown or random phenomenon – hence error or disturbance.
T_internal = teacher ratings of internalizing behaviour problems
temoG3r = teacher ratings of emotional disorder at Grade 3
tpvG3r = teacher ratings of passive victimization scale at Grade 3
T_external = teacher ratings of externalizing behaviour problems
tphyG3r = teacher ratings of physical aggression at Grade 3
thypG3r = teacher ratings of hyperactivity at Grade 3
tindG3r = teacher ratings of indirect aggression at Grade 3
tdelG3r = teacher ratings of delinquency at Grade 3
Q = the portion of the relationship between the internalizing and internalizing factors that is not accounted for by the smoking and drinking measures. It is unknown. It allows the internalizing and externalizing factors to be correlated without requiring that we account for the correlation between them using our measures. The magnitude of the relationship is quite large and smoking and drinking cannot account for all that is going on there.

6. Discussion

Alcohol is well established as a teratogenic substance (Streissguth, Landesman-Dwyer, Martin & Smith, 1980). Experimental animal studies have manipulated alcohol use to cause malformations among offspring; cross-sectional or correlational studies with humans have correlated the presence of similar malformations among infants to retrospectively measured maternal alcohol use during pregnancy; and longitudinal studies with prospective measures of alcohol use in pregnancy have confirmed those same malformations.

Quite sensibly, the early human research literature has been focused on large doses of alcohol and striking malformations in the faces of children (Huizink & Mulder, 2006; Jacobson & Jacobson, 2002; Richter & Richter, 2001). More recent research has focused on the less visible teratogenic effects of alcohol in humans. Problems with executive function – notably attention, impulsive behaviour and hyperactivity – have been demonstrated in correlational studies, as have other behaviour problems including antisocial and delinquent behaviour. Prenatal exposure to alcohol has been associated with internalizing behaviour problem, such as depression and anxiety. Deficits in cognitive functioning and learning are also evident among children with prenatal alcohol exposure, including memory and information-processing difficulties, poor problem-solving skills, impaired planning and response inhibition, lower IQ scores and problems with linguistic, perceptual and motor development.

Recent debate has been focused on dose effects. From a public health perspective, the question of how much alcohol, if any, is safe to drink during pregnancy has sparked substantial debate (Gijsen, Fulga, Garcia-Bourmessen & Koren, 2008; Kelly et al., 2009; Sayal, 2009).

The results of the first set of analyses (ANCOVA) presented above indicate that the children of mothers who report higher-risk drinking during pregnancy manifest a range of compromised developmental outcomes in early primary school compared with the children of mothers who reported lower-risk or no drinking during pregnancy. Out of a total of 79 different measures, 11 (14%) were significant at the 1% level. These negative outcomes occurred most frequently in the domain of children's behaviour problems (6 of 22 measures), more specifically in higher ratings of aggressive and hyperactive behaviours by Junior Kindergarten (JK) teachers when the children were 4 years old, and again by Grade 3 teachers when the children were 8 years old. In contrast, parent ratings did not indicate significant negative effects of prenatal alcohol exposure on children's behaviour problems.

The second domain of children's functioning in which negative associations with prenatal alcohol exposure were evident was cognitive development/academic performance (4 of 23 measures). Statistically significant negative effects were found in auditory and memory performance on the DISC developmental task administered by trained researchers at 4 years of age, and poorer ratings by Grade 3 teachers on measures of children's school preparedness, attitudes toward academics, and general academic functioning.

Animal studies have confirmed that the alcohol, tobacco has teratogenic effects on the nervous system of the fetus. Although the effects of prenatal tobacco exposure of birth weight and infant growth are well established, the effects on cognitive, behavioural and social/emotional functioning into childhood are less well documented (Cornelius & Day, 2007; Huizink & Mulder, 2006; Richter & Richter, 2001). In our findings, maternal

reports of smoking during pregnancy, collected when a child was 3 months old, were predictive of measurable problems in only one of five broad domains: child health (2 of 9 measures). Out of a total of 79 different measures, only 2 (2%) were significant at the 1% level. Since we would expect about 1% of the tests to be significant by chance alone; finding 2% of the tests to be significant is marginal evidence of an effect. Thus, for this approach to the analysis of data and given the large number of tests, we do not have conclusive evidence of smoking effects. A different statistical approach may give different results.

We also compared the children of women who reported both smoking and drinking during pregnancy with women who did neither (i.e. we left out the women who only smoked or only drank). The combined smoking and drinking was predictive in all five broad domains: general development (3 of 3 measures), cognitive development/academic performance (7 of 23 measures), social and emotional functioning (2 of 22 measures), behaviour problems (10 of 22 measures) and child health (2 of 9 measures). Out of a total of 79 different measures, 24 (30%) were significant at the 1% level. The apparent additive effect of the smoking and the drinking is intriguing, but must be interpreted cautiously given the possibility of selection bias. For example, women who smoke and drink during pregnancy may drink more than women who drink but do not smoke, or their nutritional status may be poorer, their body may already be coping with oxidative stress from smoking, or they may be living with a higher level of stress in their lives. In addition, people tend to smoke more when they are drinking.

Note that our use of statistical control techniques for 15 covariates would not have been sufficient to deal with such potential confounds.

The finding that the combination of prenatal exposure to both alcohol and tobacco predicted the most negative long-term effects on children in primary school is, however, consistent with the research literature that has reported that the negative effects of prenatal exposure to alcohol are increased when combined with other potentially harmful substances, including tobacco or non-prescription drugs (Fried, O'Connell & Watkinson, 1992; Fried & Watkinson, 1990).

- These findings also point to the importance of any future research to collect information about the use of multiple substances during pregnancy, in order to avoid inappropriate conclusions about the effects of one substance if no information on other substances is collected.

For example, if a study on maternal smoking during pregnancy does not collect information about the mothers' drinking during pregnancy, and many of the smoking mothers also engaged in high-risk drinking, negative child outcomes may be attributed to prenatal exposure to tobacco when, in fact, they may be more strongly related to exposure to alcohol or the combination of the two substances.

Note also that the amounts of alcohol and tobacco use during pregnancy need not be large. Our criterion for smoking was "any smoking." Our criterion for higher-risk drinking was a score of at least 1 on the CAGE scale. A score of 1 can be obtained by someone who feels badly about their drinking behaviour, who feels the need to cut back, or who has been criticized by others about their drinking.

Few studies of either prenatal alcohol use or smoking collect information on the use of both substances prenatally. For example,

Martin, Dombrowski, Mullis, Wisenbaker and Huttunen (2006) recently reported results from the Helsinki Longitudinal Project indicating that smoking during pregnancy was associated with several negative effects on children's development at ages 5 and 12 years of age. The authors acknowledged in their conclusions that, "Smoking, drinking and other forms of drug use are correlated, and some of the resulting effects may have been related to maternal drug use during pregnancy. This study was unable to control for pregnancy drug and alcohol use, which is a clear limitation" (p. 499). The study of either prenatal smoking or alcohol use that does not include information on the use of both substances runs the risk of forming conclusions on the effect of one substance while the effects may result either from the use of the other substance, or, as in the present study, the negative effects of using both substances prenatally (see, for example, O'Connor & Paley, 2006).

Further, few studies control for other parent or family variables such as parent education, single-parent status or family income. Several studies have reported that both prenatal alcohol use and prenatal smoking are strongly related to these variables, so if they are not controlled in analyses, the subsequent child outcomes may be more a function of the child's socio-economic environment after birth, than the smoking or alcohol use prenatally. Consistent with this concern are the findings of several studies that have reported no or much-reduced effects on children's development of maternal prenatal smoking when socio-demographic factors and postnatal environment were controlled in the analyses (D'Onofrio et al., 2008; McGee and Stanton, 1994).

Several of the negative outcomes of prenatal alcohol abuse and smoking on children's cognitive development at 33 and 48 months of

age were based on results of a standardized test of development administered to children individually by trained researchers. Nearly all of the other negative effects associated with maternal alcohol abuse and smoking during pregnancy occurred in ratings by the child's teacher at 4 and 8 years of age. The fact that teachers would not have been aware of the mothers' smoking or drinking behaviour prenatally or when the (now about 8 years old) child was 33 months old strengthens the confidence one can place on these results. The ratings collected from the children's parents, on the other hand, showed virtually no association between the mother's prenatal drinking or smoking and children's later behaviour in the ANCOVA analyses.

The finding of larger effects in teacher report data than in parent report data is consistent with the literature (Brown et al., 1991). Several interpretations of the different results between teacher and parent ratings are possible. One of many is that teacher ratings of children's behaviour and academic performance are generally considered to be more valid than those of parents, because teachers have extensive experience observing many children whereas parents' experience is typically much more limited in this regard. Parents are not able to compare their child's behaviour with those of many other children, while teachers are constantly making such comparisons. Also, since many of the child behaviour problems rated by both parents and teachers involve difficulties in relationships with peers, teachers would have more opportunity to observe a child's peer interactions than parents. Finally, the negative outcomes in the area of academic performance were based on ratings of the child's behaviour in the classroom setting, ratings that can be collected only from teachers since parents have no or extremely limited opportunity to observe such behaviours.

The fact that significant effects were present in the teachers' rating when children were age 4 (Junior Kindergarten) and age 8 (Grade 3) but not age 6 (Grade 1) may reflect the fact that children face major developmental transitions at ages 4 and 8. At 4 and 5 years of age, individual differences in children's school readiness are viewed as resulting from different levels of maturity in cognitive and social development. The challenges of formal school entry at this age accentuate individual differences in children's social and cognitive maturity. The finding that children whose mothers reported higher-risk drinking during pregnancy, and particularly if they also reported smoking, showed compromised cognitive development and elevated levels of hyperactive and aggressive behaviours may reflect their delayed social and cognitive development and difficulty in adapting successfully to the challenges of formal school entry at age 4.

At age 7, another major transition begins in normal cognitive development, namely the transition to conceptual thinking or, in Piagetian terminology, concrete operational thought (Piaget, 1964). Delays in cognitive development at this age mean that children cannot successfully adapt to academic tasks requiring the use of concepts in mathematics and reading, resulting in poor school performance and possible frustration and conflict with more mature peers. Consequently, the negative effects on children's cognitive and social development of prenatal exposure to alcohol and tobacco may be particularly noticeable by teachers at this age.

In the second set of analyses, we used a structural equation modelling technique to address some of the issues not dealt with in the more coarse-grained ANCOVA analyses reported above. We selected a subset of variables measured in Grade 3 that are related to problems in social and emotional function for dependent variables, and we included measures of drinking and smoking exposure at two times in the children's development – in utero and when the child was 33 months old. The use of the intermediate measures of smoking and drinking behaviour act as a general control for the

"third variable problem." If something that we have not measured is related to both our predictor and our outcome measure, we can get a spurious relationship mediated by that unseen variable. However, if such a variable exists, the spurious effect ought to be more powerful when the smoking or drinking was measured at 33 months of age than when measured at 3 months of age. The more recent measurement ought to "carry" the third variable effect more strongly than the more distant measure.

When we examine the measures of child behaviour collected from teachers, there is clear support for the hypothesis that drinking during pregnancy leads to problems in social and emotional functioning in elementary school, with significant paths from reported drinking during pregnancy and both internalizing behaviours (r = .19) and externalizing behaviours (r = .21). The more proximal measure of maternal drinking when the child was 33 months old does not predict either externalizing or internalizing behaviour. Smoking during pregnancy does predict externalizing behaviour problems (r = .13) and exposure to second-hand smoke at 33 months does predict internalizing behaviour (r = .15). If we interpret this at face value, it suggests that the mother's smoking behaviour during pregnancy can have effects that are evident 8 years later in a child's classroom behaviour and that those effects are over and above the effects of more recent (albeit 4 or 5 years ago) exposure to second-hand smoke. Given that the effects of paths are additive, the effect of smoking and drinking combines for an essentially doubled effect on teacher-rated externalizing behaviour.

As with the first set of analyses (ANCOVA), the same analysis using measures of child behaviour collected from parents shows fewer effects. Only smoking during pregnancy is a significant predictor of externalizing behaviour (r = .17).

Structural equation modelling is a correlational technique. While it is a truism that correlation does not prove causation, the findings of our two models interact with the existing literature in a powerful manner. Drinking and smoking during pregnancy are significant predictors of problems in externalizing behaviour noted 8 years later by both teachers and parents and of internalizing behaviour noted by teachers. These predictors are significant even when "competing" for covariance with related measures collected much closer in time to the behaviour data collection. In the context of recent animal and human findings, the most responsible interpretation of these findings is that smoking and drinking during pregnancy cause some problems in Grade 3 and the predictive relationships observed in the SEM is due to the causal impact of tobacco and alcohol use during pregnancy.

How big is the effect we are looking at? Is it merely statistical, or is it of a magnitude that people would notice?

One way to approach this problem is to look at comparative effect sizes. Meyer et al. (2001) presented an array of effect sizes from meta-analyses that allow a researcher to fit a finding onto a scale. In the table below, our own findings have been embedded among other effect size findings, using the r statistic to make them more compatible with the results of SEM. For example, when people with allergic reactions use antihistamines for runny nose and sneezing, the effect size averages 0.11. Prenatal smoking has an effect of .13 on externalizing ratings. If our data are accurate, then abstinence from smoking and drinking during pregnancy ought to have an effect on internalizing behaviour about as powerful as taking non-steroidal anti-inflammatory drugs (NSAIDS) for pain or taking anti-histamines for allergies. Given that the effects of prenatal smoking and drinking are both evident in teacher reports, the effect of the double abstinence may be twice as large. Moreover, there may be another independent relationship with second-hand

smoke. Keep in mind that causal inference from a SEM of this type must be cautious in the absence of manipulation of the independent variable.

r	Treatment effect
0.03	Anti-hypertensive medication on reduced risk of stroke
0.08	Bypass in stable heart disease on 5-year survival
0.11	Anti-histamine on runny nose and sneezing
0.13	**Prenatal smoking on teacher externalizing ratings**
0.14	NSAIDs on pain
0.15	**Second-hand smoke on teacher internalizing ratings**
0.17	**Prenatal smoking on parent externalizing ratings**
0.19	**Prenatal drinking on teacher internalizing ratings**
0.21	**Prenatal drinking on teacher externalizing ratings**
0.38	Viagra on male sexual function

Data (effect sizes) from the first statistical analysis of this report (i.e. the ANCOVA results) are reported in terms of the d statistic rather than the r statistic. We looked for meta-analytic studies not included in Meyer et al.'s (2001) article that would expand the range of comparators for our results.

Bhutta, Cleves, Casey, Cradock and Anand (2002) reported a meta-analysis of the cognitive and behavioural outcomes of children who were born preterm. When we interpolate from their measure of weighted mean difference (WMD) by dividing by the theoretical standard deviation of the ability test scores, they demonstrate a mean d of about 0.72 for cognitive measures. Thus, the preterm children in the studies they found were about 11 IQ points or .7 standard deviations below the comparator children born at term. This effect is proportional to gestational age (r = .71) , showing an increase in the WMD of roughly .67 points or in the d statistic of about .044 per week of prematurity. Our observed mean d statistics for measures of general child development (see Appendix 1) were –.29 for prenatal maternal tobacco use, –.28 for prenatal maternal alcohol use and –.57 for the difference between children of mothers who used both substances and mothers who used neither. Thus our observed d statistics correspond to those observed in Bhutta, Cleves, Casey, Cradock and Anand's (2002) study at roughly 6,6 and 13 weeks of prematurity for tobacco, alcohol and joint exposure, respectively. In rough terms, smoking and drinking during pregnancy even at the relatively low levels found in our sample seem to be the equivalent of 6 weeks of prematurity – per substance used.

Schachter, Pham, King, Langford and Moher (2001) conducted a meta-analysis of the effects of Ritalin on children and adolescents with ADHD. In general, they found that the medication was effective based on an array of behavioural measures, but noted that the effects as reported by teachers were stronger than those reported by parents. These researchers reported a mean effect size of .78 for teacher reports and .54 for parent reports. Our results for behavioural problem reports showed effect sizes for teachers of .29, .40 and .70 and for parents of .21, .12 and .25 for prenatal tobacco exposure, alcohol exposure and the combined exposure, respectively.

Paolucci and Violato (2004) reported a meta-analysis on the effects of child sexual abuse. These authors reported a variety of weighted mean effect sizes for diagnoses ranging from

.16 to .44. The effect size for academic performance was .19. This compares with our effect sizes for all measures of academic and cognitive performance of –.09, –.26 and –.37 for prenatal tobacco exposure, alcohol use and the combined use, respectively. (See Appendix 1.)

Kitzmann, Gaylord, Holt and Kenny (2003) reported a meta-analysis of the effects on children of witnessing domestic violence. On a variety of problems (internalizing, externalizing, social, academic and other), they reported a mean effect size of .40 when comparing children who witnessed violence with those who did not. Our mean d statistics for measures of general development (see Appendix 1) were –.29 for prenatal maternal tobacco use, –.28 for prenatal maternal alcohol use and –.57 for the difference between children of mothers who used both substances and mothers who used neither.

These findings help to put the measured effects of smoking and drinking into perspective. When we compare the effects we found with those observed with three predictors of worse performance (prematurity, witnessing domestic violence and child sexual abuse), there was a strong overlap. When we compare with intervention for ADHD with Ritalin, the effects of combined prenatal substance use are roughly comparable (but reversed in sign) to the impact of Ritalin on behavioural measures. In broad terms, the impact of the substances appears to be comparable to moderate prematurity (6 weeks for each substance, 12 for both), child sexual abuse (either substance, or twice the magnitude if both were used), or witnessing domestic violence (less impact for one substance, greater impact for combined). The impact of ADHD that is reversible by using Ritalin appears to be roughly comparable to use of both substances.

While these comparisons to meta-analytic findings must be considered approximate, they do give a sense of scale. In our sample, the impact of smoking and drinking during pregnancy is of a magnitude that compares with prematurity, sexual abuse, witnessing domestic violence and the Ritalin-reversible effects of ADHD. It is also important to note that these are aggregate findings, and it is hard to imagine that every child would show the same magnitude of effects – there will be lots of variation.

In conclusion, the results of the present study suggest that children whose mothers report higher-risk alcohol consumption during pregnancy show long-term negative outcomes in measures of school performance and behaviour problems compared with mothers who report lower-risk drinking. These problems are accentuated in children whose mothers also report smoking during the pregnancy. Further, the negative effects are most apparent when children are 4 years of age, and faced with the challenges of formal school entry (i.e. poor school readiness), and again at age 8, when individual differences in conceptual thinking may be particularly salient to teachers. The percentage of measures reflecting the disadvantage of children exposed to prenatal alcohol and tobacco increased from 37% at age 4 to 47% at age 8. If this pattern continues, the negative effects on children's academic and social behaviour may continue to be compromised as they enter early adolescence; that is, prenatal exposure to maternal high-risk drinking and smoking may be linked to disrupted cognitive and social development at critical periods in children's development, with lifelong consequences.

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Appendix 1

Effects of Alcohol and Tobacco During Pregnancy: Summary Results (p-values and effect sizes)

Note

A negative sign (–) indicates poorer outcomes on the measure associated with prenatal exposure to tobacco and/or high-risk drinking; a positive sign indicates a better outcome on the measure associated with prenatal exposure to tobacco and/or high-risk drinking.

*Statistically significant results are shown in **bold**.*

	Outcome Variables	Sample size (n)	Prenatal Exposure to Tobacco (PET): 0 = not exposed, 1 = exposed	Mother High-Risk Drinker (MHRD): 0 = no, 1 = yes	Interaction: not exposed, not high-risk drinker vs. exposed, high-risk drinker
General Child Development					
1	DISC – overall development quotient, 33 months	404	–0.22	–0.34	**–0.57**
2	DISC – overall development quotient, 48 months	427	–0.19	–0.31	**–0.50**
3	ABC – maturity/school-readiness scale, 48 months	273	–0.47	–0.18	**–0.65**
Mean effect sizes			–0.29	–0.28	–0.57
Cognitive Development/Academic Performance					
4	DISC – auditory and memory development quotient, 33 months	404	–0.19	–0.34	–0.53
5	DISC – auditory and memory development quotient, 48 months	427	–0.19	**–0.44**	**–0.63**
6	PPVT W-ability, 48 months	426	–0.12	–0.33	–0.45
7	WPPSI Block Design – Standardized Score, 48 months	424	–0.36	–0.11	–0.47
8	Teacher rated: student-preparedness scale, Grade 1	282	–0.15	–0.25	–0.41
9	Child's attitudes toward academics scale, Grade 1	285	–0.02	–0.23	–0.25
10	Teacher rated: academic functioning scale, Grade 1	285	–0.12	–0.15	–0.27
11	Teacher rated: adaptive functioning scale, Grade 1	285	+0.02	–0.42	–0.41

	Outcome Variables	Sample size (n)	Prenatal Exposure to Tobacco (PET): 0 = not exposed, 1 = exposed	Mother High-Risk Drinker (MHRD): 0 = no, 1 = yes	Interaction: not exposed, not high-risk drinker vs. exposed, high-risk drinker
12	Parent reported: child repeated a grade, Grade 1	345	+0.05	+0.19	+0.25
13	Parent reported: child suspended from school, Grade 1	345	–0.09	–0.13	–0.11
14	Teacher reported: received special education/services, Grade 1	286	+0.10	–0.15	–0.21
15	Teacher reported: child limited by learning disability, Grade 1	286	–0.05	+0.01	–0.11
16	Teacher rated: student-preparedness scale, Grade 3	239	–0.35	**–0.69**	**–1.03**
17	Child's attitudes toward academics scale, Grade 3	244	–0.16	**–0.53**	**–0.69**
18	Teacher rated: academic functioning scale, Grade 3	245	–0.07	**–0.58**	**–0.65**
19	Teacher rated: adaptive functioning scale, Grade 3	245	–0.25	–0.37	**–0.62**
20	Parent reported: child repeated a grade, Grade 3	325	+0.01	+0.06	+0.01
21	Parent reported: child suspended from school, Grade 3	326	–0.46	–0.37	**–0.56**
22	Teacher reported: received special education/services, Grade 3	246	–0.23	–0.54	**–0.64**
23	Teacher reported: child limited by learning disability, Grade 3	246	+0.26	–0.37	–0.11
24	EQAO – math, Grade 3	150	+0.15	–0.11	–0.04
25	EQAO – reading, Grade 3	142	+0.43	–0.14	–0.29
26	EQAO – writing, Grade 3	148	–0.30	–0.07	–0.37
Mean effect sizes			–0.09	–0.26	–0.37

	Outcome Variables	Sample size (n)	Prenatal Exposure to Tobacco (PET): 0 = not exposed, 1 = exposed	Mother High-Risk Drinker (MHRD): 0 = no, 1 = yes	Interaction: not exposed, not high-risk drinker vs. exposed, high-risk drinker
	Social/Emotional Functioning				
27	Parent rated: PSBQ prosocial scale, 48 months	383	–0.11	–0.07	–0.18
28	Parent rated: PSBQ anxious scale, 48 months	429	+0.06	–0.00	+0.06
29	Teacher rated: PSBQ prosocial scale, 48 months	268	–0.09	–0.18	–0.28
30	Teacher rated: PSBQ anxious scale, 48 months	274	–0.08	–0.24	–0.33
31	Teacher rated: NLSCY emotional disorder scale, 48 months	272	–0.23	–0.31	–0.55
32	Teacher rated: NLSCY prosocial scale, 48 months	268	–0.08	–0.21	–0.28
33	Parent rated: OCHS depression scale, Grade 1	340	–0.05	–0.02	–0.07
34	Teacher rated: OCHS passive victimization scale, Grade 1	269	–0.08	–0.10	–0.18
35	Parent rated: NLSCY emotional disorder scale, Grade 1	346	–0.08	+0.06	–0.02
36	Parent rated: NLSCY prosocial scale, Grade 1	345	+0.04	–0.14	–0.10
37	Teacher rated: NLSCY emotional disorder scale, Grade 1	283	+0.15	–0.30	–0.15
38	Teacher rated: NLSCY prosocial scale, Grade 1	272	+0.14	–0.09	–0.04
39	Teacher rated: SSRS conflict management scale, Grade 1	285	+0.05	–0.20	–0.15
40	Ability to get along with other scale, Grade 1	259	+0.16	–0.27	–0.11
41	Parent rated: NLSCY emotional disorder scale, Grade 3	327	–0.20	–0.07	–0.27
42	Parent rated: OCHS depression scale, Grade 3	319	–0.26	–0.01	–0.27
43	Teacher rated: OCHS passive victimization scale, Grade 3	231	–0.35	+0.04	–0.32
44	Parent rated: NLSCY prosocial scale, Grade 3	325	–0.06	–0.19	–0.25
45	Teacher rated: NLSCY emotional disorder scale, Grade 3	242	–0.14	**–0.65**	**–0.80**
46	Teacher rated: NLSCY prosocial scale, Grade 3	235	–0.25	–0.24	–0.49
47	Teacher rated: SSRS conflict management scale, Grade 3	246	–0.18	–0.42	**–0.60**
48	Ability to get along with other scale, Grade 3	271	–0.08	–0.25	–0.33
Mean effect sizes			–0.08	–0.18	–0.26

	Outcome Variables	Sample size (n)	Prenatal Exposure to Tobacco (PET): 0 = not exposed, 1 = exposed	Mother High-Risk Drinker (MHRD): 0 = no, 1 = yes	Interaction: not exposed, not high-risk drinker vs. exposed, high-risk drinker
	Behaviour Problems				
49	Bates Temperament scale, 33 months	407	+.04	–.11	–.07
50	Parent rated: PSBQ disruptiveness scale, 48 months	402	–0.12	+0.14	+0.02
51	Teacher rated: PSBQ disruptiveness scale, 48 months	269	–0.32	**–0.52**	**–0.83**
52	Teacher rated: NLSCY hyperactivity scale, 48 months	273	–0.36	**–0.52**	**–0.88**
53	Teacher rated: NLSCY indirect aggression scale, 48 months	272	–0.23	**–0.61**	**–0.84**
54	Teacher rated: NLSCY physical aggression scale, 48 months	272	–0.41	–0.44	**–0.85**
55	Parent rated: OCHS oppositional defiant scale, Grade 1	384	–0.02	–0.08	–0.09
56	Parent rated: NLSCY hyperactivity scale, Grade 1	348	–0.03	–0.36	–0.39
57	Parent rated: NLSCY indirect aggression scale, Grade 1	323	–0.14	+0.05	–0.09
58	Parent rated: NLSCY physical aggression scale, Grade 1	348	–0.15	–0.13	–0.27
59	Teacher rated: NLSCY hyperactivity scale, Grade 1	286	+0.01	–0.18	–0.17
60	Teacher rated: NLSCY indirect aggression scale, Grade 1	245	–0.27	+0.01	–0.27
61	Teacher rated: NLSCY physical aggression scale, Grade 1	282	–0.38	–0.07	–0.45
62	Teacher rated: NLSCY delinquency scale, Grade 1	268	–0.39	–0.30	**–0.69**
63	Parent rated: OCHS oppositional defiant scale, Grade 3	326	–0.35	+0.05	–0.30
64	Parent rated: NLSCY hyperactivity scale, Grade 3	327	–0.39	+0.07	–0.32
65	Parent rated: NLSCY indirect aggression scale, Grade 3	316	–0.52	+0.06	–0.46
66	Parent rated: NLSCY physical aggression scale, Grade 3	327	–0.33	–0.26	**–0.59**
67	Teacher rated: NLSCY hyperactivity scale, Grade 3	243	–0.21	**–0.56**	**–0.77**
68	Teacher rated: NLSCY indirect aggression scale, Grade 3	210	–0.33	–0.53	**–0.86**
69	Teacher rated: NLSCY physical aggression scale, Grade 3	243	–0.27	**–0.49**	**–0.76**
70	Teacher rated: NLSCY delinquency scale, Grade 3	228	–0.33	**–0.66**	**–0.98**
Mean effect sizes			–0.25	–0.25	–0.50

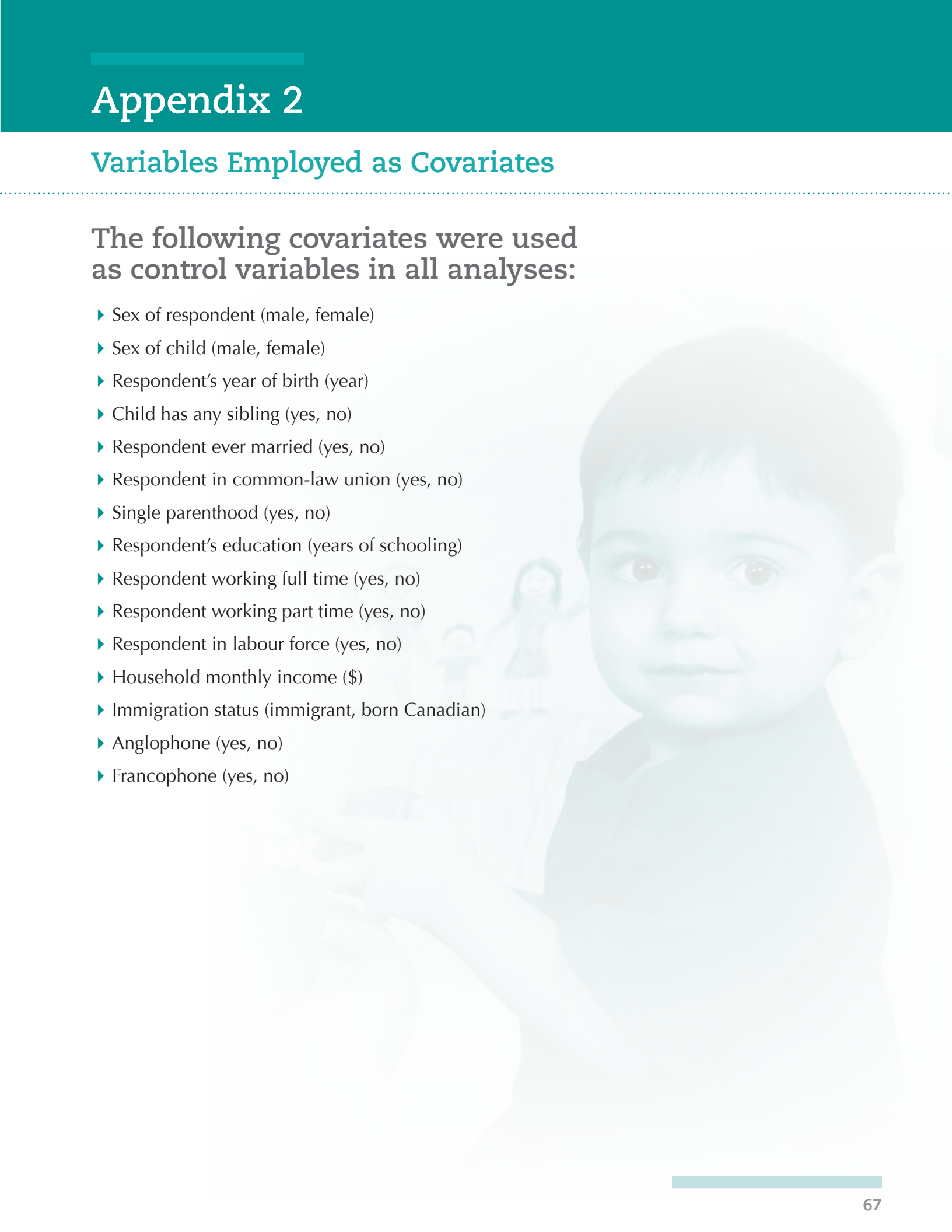
Outcome Variables		Sample size (n)	Prenatal Exposure to Tobacco (PET): 0 = not exposed, 1 = exposed	Mother High-Risk Drinker (MHRD): 0 = no, 1 = yes	Interaction: not exposed, not high-risk drinker vs. exposed, high-risk drinker
Child Health					
71	Parent-reported child's health, 48 months	438	–0.15	+0.01	–0.14
72	Child's height, 48 months	411	+0.07	+0.01	+0.08
73	Child's weight, 48 months	414	+0.02	+0.07	+0.09
74	Parent-reported child's health, Grade 1	348	–0.09	–0.00	–0.09
75	Child's asthma, Grade 1	348	+0.07	–0.07	+0.01
76	Child's exposure to second-hand smoke, Grade 1	345	**–0.79**	–0.04	**–0.83**
77	Parent reported child's health, Grade 3	327	+0.16	–0.13	+0.02
78	Child's asthma, Grade 3	327	+0.08	–0.13	–0.05
79	Child's exposure to second-hand smoke, Grade 3	324	**–0.64**	+0.15	**–0.49**
Mean effect sizes			–0.14	–0.01	–0.16
Mean of all effect sizes in Appendix 1			–0.15	–0.21	–0.36

Appendix 2

Variables Employed as Covariates

The following covariates were used as control variables in all analyses:

- Sex of respondent (male, female)
- Sex of child (male, female)
- Respondent's year of birth (year)
- Child has any sibling (yes, no)
- Respondent ever married (yes, no)
- Respondent in common-law union (yes, no)
- Single parenthood (yes, no)
- Respondent's education (years of schooling)
- Respondent working full time (yes, no)
- Respondent working part time (yes, no)
- Respondent in labour force (yes, no)
- Household monthly income ($)
- Immigration status (immigrant, born Canadian)
- Anglophone (yes, no)
- Francophone (yes, no)



Appendix 3

Group Means, Standard Errors and Sample Sizes for All Measures

CHILDREN'S GENERAL DEVELOPMENT

At 33 months of age

Developmental Inventory for Screening Children (DISC): Overall Development Quotient

Group		Means	Std. Error	N
Prenatal Exposure to Tobacco (PET)	Not exposed	100.24	1.36	278
	Exposed	97.66	1.39	126
Mother Higher-Risk Drinker (MHRD)	No	100.94	0.62	368
	Yes	96.96	1.80	36
PET and MHRD	Not exposed, not higher-risk drinker	100.97	0.68	261
	Exposed, higher-risk drinker	94.41	2.52	19

At 48 months of age

DISC Overall Development Quotient

Group		Means	Std. Error	N
Prenatal Exposure to Tobacco (PET)	Not exposed	98.80	1.39	293
	Exposed	96.32	1.37	134
Mother Higher-Risk Drinker (MHRD)	No	99.54	0.66	384
	Yes	95.57	1.80	43
PET and MHRD	Not exposed, not higher-risk drinker	98.35	0.72	274
	Exposed, higher-risk drinker	91.89	2.44	24

ABC School Readiness

Group		Means	Std. Error	N
Prenatal Exposure to Tobacco (PET)	Not exposed	26.35	1.58	186
	Exposed	21.61	1.44	87
Mother Higher-Risk Drinker (MHRD)	No	24.90	0.67	248
	Yes	23.06	1.97	25
PET and MHRD	Not exposed, not higher-risk drinker	25.59	0.74	176
	Exposed, higher-risk drinker	19.02	2.57	15

CHILDREN'S COGNITIVE DEVELOPMENT/ACADEMIC PERFORMANCE

At 48 months of age

DISC Auditory and Memory Development Quotient

Group		Means	Std. Error	N
Prenatal Exposure to Tobacco (PET)	Not exposed	96.42	1.74	293
	Exposed	93.62	1.71	34
Mother Higher-Risk Drinker (MHRD)	No	98.35	0.83	384
	Yes	91.69	2.24	43
PET and MHRD	Not exposed, not higher-risk drinker	98.15	0.90	274
	Exposed, higher-risk drinker	88.69	3.05	24

At Grade 3

Teacher-Rated Student Preparedness Scale

Group		Means	Std. Error	N
Prenatal Exposure to Tobacco (PET)	Not exposed	11.56	0.71	153
	Exposed	13.03	0.53	86
Mother Higher-Risk Drinker (MHRD)	No	10.83	0.28	213
	Yes	13.76	0.82	26
PET and MHRD	Not exposed, not higher-risk drinker	10.21	0.32	145
	Exposed, higher-risk drinker	14.62	0.93	18

Teacher-Rated Child's Attitudes Toward Academics Scale

Group		Means	Std. Error	N
Prenatal Exposure to Tobacco (PET)	Not exposed	13.64	0.63	156
	Exposed	13.06	0.47	88
Mother Higher-Risk Drinker (MHRD)	No	14.30	0.25	218
	Yes	12.40	0.73	26
PET and MHRD	Not exposed, not higher-risk drinker	14.58	0.29	148
	Exposed, higher-risk drinker	12.09	0.84	18

Teacher-Rated Child's Academic Functioning Scale

Group		Means	Std. Error	N
Prenatal Exposure to Tobacco (PET)	Not exposed	9.67	0.63	156
	Exposed	9.42	0.47	89
Mother Higher-Risk Drinker (MHRD)	No	10.60	0.25	219
	Yes	8.50	0.73	26
PET and MHRD	Not exposed, not higher-risk drinker	10.60	0.29	148
	Exposed, higher-risk drinker	8.25	0.84	18

Teacher-Rated Child's Adaptive Functioning Scale

Group		Means	Std. Error	N
Prenatal Exposure to Tobacco (PET)	Not exposed	15.44	0.86	157
	Exposed	14.15	0.65	89
Mother Higher-Risk Drinker (MHRD)	No	15.74	0.34	220
	Yes	13.85	1.00	26
PET and MHRD	Not exposed, not higher-risk drinker	16.14	0.39	149
	Exposed, higher-risk drinker	12.96	1.14	18

Parent-Reported Child Suspended from School (%)

Group		Means	Std. Error	N
Prenatal Exposure to Tobacco (PET)	Not exposed	8.68	0.015	220
	Exposed	24.44	0.042	106
Mother Higher-Risk Drinker (MHRD)	No	12.56	0.018	294
	Yes	25.23	0.077	32
PET and MHRD	Not exposed, not higher-risk drinker	8.02	0.015	209
	Exposed, higher-risk drinker	27.29	0.100	21

Teacher-Reported Child Received Special Education/Services (%)

Group		Means	Std. Error	N
Prenatal Exposure to Tobacco (PET)	Not exposed	37.38	0.037	157
	Exposed	48.67	0.052	89
Mother Higher-Risk Drinker (MHRD)	No	38.66	0.032	220
	Yes	65.19	0.088	26
PET and MHRD	Not exposed, not higher-risk drinker	36.22	0.038	149
	Exposed, higher-risk drinker	67.95	0.105	18

CHILDREN'S SOCIAL/EMOTIONAL FUNCTIONING MEASURES

At Grade 3

Teacher-Rated Emotional Disorder Scale

Group		Means	Std. Error	N
Prenatal Exposure to Tobacco (PET)	Not exposed	4.78	0.66	155
	Exposed	5.31	0.50	87
Mother Higher-Risk Drinker (MHRD)	No	3.86	0.26	216
	Yes	6.23	0.77	26
PET and MHRD	Not exposed, not higher-risk drinker	3.73	0.30	147
	Exposed, higher-risk drinker	6.62	0.88	18

Teacher-Rated Conflict Management Scale

Group		Means	Std. Error	N
Prenatal Exposure to Tobacco (PET)	Not exposed	14.61	0.53	157
	Exposed	14.08	0.39	89
Mother Higher-Risk Drinker (MHRD)	No	14.97	0.20	220
	Yes	13.71	0.61	26
PET and MHRD	Not exposed, not higher-risk drinker	15.40	0.24	149
	Exposed, higher-risk drinker	13.62	0.70	18

CHILDREN'S HEALTH MEASURES

At Grade 1

Child Exposed to Second-Hand Smoke

Group		Means	Std. Error	N
Prenatal Exposure to Tobacco (PET)	Not exposed	5.45	1.70	231
	Exposed	16.67	1.52	114
Mother Higher-Risk Drinker (MHRD)	No	10.77	0.77	309
	Yes	11.35	2.12	36
PET and MHRD	Not exposed, not higher-risk drinker	4.43	0.86	217
	Exposed, higher-risk drinker	16.22	2.69	22

At Grade 3

Child Exposed to Second-Hand Smoke

Group		Means	Std. Error	N
Prenatal Exposure to Tobacco (PET)	Not exposed	4.11	1.86	219
	Exposed	12.74	1.50	105
Mother Higher-Risk Drinker (MHRD)	No	9.44	0.77	292
	Yes	7.41	2.24	32
PET and MHRD	Not exposed, not higher-risk drinker	4.21	0.84	208
	Exposed, higher-risk drinker	10.81	2.64	21

CHILDREN'S BEHAVIOUR PROBLEMS MEASURES

At 48 months of age

Teacher-Rated Disruptiveness Scale

Group		Means	Std. Error	N
Prenatal Exposure to Nicotine (PEN)	Not exposed	6.78	0.92	185
	Exposed	8.70	0.89	84
Mother High-Risk Drinker (MHRD)	No	6.17	0.42	243
	Yes	9.31	1.17	26
PEN and MHRD	Not exposed, not high-risk drinker	5.71	0.46	174
	Exposed, high-risk drinker	10.78	1.58	15

Teacher-Rated Hyperactivity Scale

Group		Means	Std. Error	N
Prenatal Exposure to Nicotine (PEN)	Not exposed	4.65	0.57	187
	Exposed	6.05	0.55	86
Mother High-Risk Drinker (MHRD)	No	4.33	0.26	247
	Yes	6.37	0.73	26
PEN and MHRD	Not exposed, not high-risk drinker	3.99	0.28	176
	Exposed, high-risk drinker	7.44	0.98	15

Teacher-Rated Indirect Aggression Scale

Group		Means	Std. Error	N
Prenatal Exposure to Nicotine (PEN)	Not exposed	1.40	0.30	186
	Exposed	1.83	0.29	86
Mother High-Risk Drinker (MHRD)	No	1.04	0.13	246
	Yes	2.19	0.38	26
PEN and MHRD	Not exposed, not high-risk drinker	0.91	0.15	175
	Exposed, high-risk drinker	2.49	0.51	15

Teacher-Rated Physical Aggression Scale

Group		Means	Std. Error	N
Prenatal Exposure to Nicotine (PEN)	Not exposed	1.69	0.40	185
	Exposed	2.78	0.39	87
Mother High-Risk Drinker (MHRD)	No	1.66	0.18	246
	Yes	2.81	0.51	26
PEN and MHRD	Not exposed, not high-risk drinker	1.41	0.20	174
	Exposed, high-risk drinker	3.65	0.69	15

At Grade 1

Teacher-Rated Delinquency Scale

Group		Means	Std. Error	N
Prenatal Exposure to Nicotine (PEN)	Not exposed	0.93	0.24	186
	Exposed	1.54	0.20	82
Mother High-Risk Drinker (MHRD)	No	1.00	0.11	240
	Yes	1.47	0.29	28
PEN and MHRD	Not exposed, not high-risk drinker	0.59	0.11	176
	Exposed, high-risk drinker	1.66	0.35	18

At Grade 3

Parent-Rated Physical Aggression Scale

Group		Means	Std. Error	N
Prenatal Exposure to Nicotine (PEN)	Not exposed	1.82	0.33	221
	Exposed	2.54	0.27	106
Mother High-Risk Drinker (MHRD)	No	1.91	0.14	295
	Yes	2.46	0.40	32
PEN and MHRD	Not exposed, not high-risk drinker	1.61	0.15	210
	Exposed, high-risk drinker	2.88	0.47	21

Teacher-Rated Hyperactivity Scale

Group		Means	Std. Error	N
Prenatal Exposure to Nicotine (PEN)	Not exposed	5.33	0.76	156
	Exposed	6.28	0.57	87
Mother High-Risk Drinker (MHRD)	No	4.55	0.30	217
	Yes	7.06	0.89	26
PEN and MHRD	Not exposed, not high-risk drinker	4.14	0.35	148
	Exposed, high-risk drinker	7.60	1.01	18

Teacher-Rated Indirect Aggression Scale

Group		Means	Std. Error	N
Prenatal Exposure to Nicotine (PEN)	Not exposed	1.83	0.45	133
	Exposed	2.55	0.32	77
Mother High-Risk Drinker (MHRD)	No	1.61	0.16	189
	Yes	2.77	0.52	21
PEN and MHRD	Not exposed, not high-risk drinker	1.54	0.19	127
	Exposed, high-risk drinker	3.42	0.57	15

Teacher-Rated Physical Aggression Scale

Group		Means	Std. Error	N
Prenatal Exposure to Nicotine (PEN)	Not exposed	2.43	0.52	154
	Exposed	3.24	0.39	89
Mother High-Risk Drinker (MHRD)	No	2.10	0.20	217
	Yes	3.58	0.60	26
PEN and MHRD	Not exposed, not high-risk drinker	1.57	0.24	146
	Exposed, high-risk drinker	3.85	0.68	18

Teacher-Rated Delinquency Scale

Group		Means	Std. Error	N
Prenatal Exposure to Nicotine (PEN)	Not exposed	1.12	0.31	148
	Exposed	1.68	0.24	80
Mother High-Risk Drinker (MHRD)	No	0.84	0.20	205
	Yes	1.96	0.36	23
PEN and MHRD	Not exposed, not high-risk drinker	0.78	0.14	141
	Exposed, high-risk drinker	2.47	0.42	16

Early primary school outcomes

associated with **maternal use of alcohol and tobacco during pregnancy** and with **exposure to parent alcohol** and **tobacco use postnatally**