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World Health Organization International Study on the Prevalence of Fetal Alcohol Spectrum Disorder (FASD)

Canadian Component

A report prepared by the Institute for Mental Health Policy Research, Centre for Addiction and Mental Health

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Any errors or omissions in this report are the sole responsibility of the authors.

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Appendices

Abbreviations

ARND: alcohol-related neurodevelopmental disorder

CBCL: Child Behavior Checklist

FAS: fetal alcohol syndrome

FASD: fetal alcohol spectrum disorder

GTA: Greater Toronto Area

pFAS: partial fetal alcohol syndrome

RP: response prevalence

SR: selection rate

Executive Summary

Background and Objectives

Population-based prevalence data on fetal alcohol spectrum disorder (FASD) among the general population of Canada are unavailable. To fill this gap, the objective of this study was to determine the population-based prevalence of FASD among elementary school students in the Greater Toronto Area (GTA) in Ontario, Canada.

Methodology

This screening study used a cross-sectional, observational design utilizing active case ascertainment, along with retrospective collection of prenatal alcohol exposure information. The sampling frame included all students aged 7 to 9 years (grades 2, 3 and 4) attending public school in the GTA from September 2014 to June 2017. Active written consent was sought from the parents/guardians. In addition, all students who received consent had to agree to participate in the study.

Data collection involved two phases. Phase I involved pre-screening, which included taking growth measurements, identifying behavioural and/or learning problems, and performing a dysmorphology examination. Students who were at or below the 10th percentile for height and weight or occipitofrontal circumference; and/or who had behavioural and/or learning problems; and/or who had at least two of the three facial features characteristic of fetal alcohol syndrome (FAS) were selected to proceed to Phase II (screening, along with a random subset of students chosen to proceed as typically developing control children). Biological mothers of students found to have neurodevelopmental deficits (defined as two standard deviations below the mean on a subtest) in a minimum of two domains assessed using a standard neurodevelopmental test battery were invited for an interview. It should be noted that impairment in a minimum of three domains is necessary for a FASD-specific diagnosis; however, the threshold of two impaired domains was set to increase the likelihood that all potential cases were identified. The maternal interview included questions on demographics and living environment, pregnancy history, maternal alcohol use (prior to / following pregnancy recognition, as well as current use), nutrition, tobacco use and other drug use during pregnancy. Additionally, parents/guardians were asked to complete the Child Behavior Checklist (CBCL) to evaluate their child's social competencies and identify any behavioural problems. Final diagnostic screening conclusions were made by consensus by a team of experienced multidisciplinary experts during case conferences, using the Canadian guidelines for FASD diagnosis (Chudley et al., 2005). A group of typically developing control children was randomly selected from a list of all students who completed Phase I and who did not meet criteria for Phase II. These students underwent a complete assessment.

The prevalence of FASD (and of each of the diagnostic categories within the spectrum—FAS, partial fetal alcohol syndrome [pFAS] and alcohol-related neurodevelopmental disorder [ARND]) was estimated, taking into consideration the selection rate, which was used to account for students who dropped out or were lost to follow-up during each phase of data collection. Monte Carlo simulations were employed to derive the confidence interval (CI) for the point estimates.

Results

Five out of the 10 district school boards, representing four out of the five regional municipalities, agreed to participate in the study. In total, 8,209 students were invited to participate. The parents/guardians of 3,854 of these students (46.9%) submitted the completed consent form. Among those who responded, 1,161 (30.1%)

refused to allow their child to participate, and 2,693 (69.9%) allowed their child to participate. In total, 2,555 students participated in Phase I of the study, 793 (31.0%) of whom were eligible to proceed to Phase II. In addition, 87 typically developing control children were randomly selected to undergo a full assessment.

Of those 762 students who underwent a neurodevelopmental assessment (31 students were lost to follow-up, along with three typically developing control children), 323 (42.4%) demonstrated deficits in a minimum of two domains assessed using a standard neurodevelopmental test battery. The biological mothers of these students were invited for an interview. A total of 132 (40.9%) biological mothers agreed to be interviewed (33 declined; 150 were unreachable; and 8 were no longer in the child's life, in which case the child's guardian completed the CBCL). In total, data for 323 potential "cases" (along with data for 84 typically developing control children) were independently reviewed by a panel of experts. Subsequently, 69 identified cases were discussed on a case-by-case basis during multidisciplinary case conferences.

A total of 21 cases of suspected FASD were identified (3 cases of FAS, 2 cases of pFAS and 16 cases of ARND). The estimated prevalence was 1.2 per 1,000 for FAS, 2.0 per 1,000 for pFAS and 15.0 per 1,000 for ARND. Accordingly, the prevalence of FASD was estimated to be 18.1 per 1,000, or about 1.8%. Using a less conservative approach, the prevalence of FASD was estimated to be 29.3 per 1,000, or about 2.9%. Therefore, the population-based prevalence of FASD is likely to range between 2% and 3% among elementary school students (aged 7 to 9 years) in the GTA in Ontario, Canada.

Conclusions

This study provides the first population-based estimate of the prevalence of FASD among elementary school students (aged 7 to 9 years) in Canada. The estimate is approximately double or possibly even triple previous crude estimates. However, it would be beneficial for other provinces and territories to conduct similarly designed active case ascertainment studies to obtain their own population-based prevalence rates in both general and special (high-risk) populations. More effective prevention strategies targeting alcohol use during pregnancy and surveillance of FASD are urgently needed.

1. Introduction

Alcohol is a teratogen that can readily cross the placenta, interfering with the normal progression of the embryo and resulting in damage to the brain and other organs of the developing fetus. Alcohol use during pregnancy has been established as a risk factor for adverse pregnancy outcomes, including stillbirth (Kesmodel et al., 2002), spontaneous abortion (Henriksen et al., 2004), premature birth (Albertsen et al., 2004; Kesmodel et al., 2000; Patra et al., 2011), intrauterine growth retardation (Patra et al., 2011; Yang et al., 2001) and low birth weight (O'Callaghan et al., 2003; Patra et al., 2011). Alcohol use during pregnancy is an established cause of fetal alcohol spectrum disorder (FASD), one of the most disabling potential outcomes of prenatal alcohol exposure. Despite the risk, a significant number of pregnancies are alcohol-exposed; it was recently estimated that in Canada, 10.0% of women consume alcohol while they are pregnant (Popova, Lange, Probst, Gmel et al., 2017).

As outlined in the 2005 Canadian guidelines for diagnosis, FASD includes the following three alcohol-related diagnoses: fetal alcohol syndrome (FAS), partial FAS (pFAS), and alcohol-related neurodevelopmental disorder (ARND) (Chudley et al., 2005). Full FAS is characterized by a triad of signs: 1) prenatal and/or postnatal growth restriction; 2) central nervous system dysfunction demonstrated by intellectual impairment and/or structural abnormalities, microcephaly, developmental delay and complex behavioural problems; and 3) characteristic facial anomalies, including short palpebral fissures, a flat philtrum and thin vermilion border of the upper lip (Astley & Clarren, 2000; Chudley et al., 2005; Cook et al., 2016; Hoyme et al., 2016). Globally, the proportion of FAS cases among all FASD cases was recently estimated to be 18.9%; that is, approximately two out of 10 people with FASD will be diagnosed with FAS (Lange et al., 2017; Popova, Lange, Probst, Gmel et al., 2017).

The main effect of prenatal alcohol exposure is permanent central nervous system damage, which can lead to a myriad of adverse developmental outcomes in exposed children. Developing brain cells and structures can be malformed or have their development interrupted upon exposure to alcohol prenatally. Thus, FASD is associated with a wide range of effects, including permanent brain damage, congenital anomalies, prenatal and/or postnatal growth restriction and characteristic dysmorphic facial features, along with cognitive, behavioural, emotional and adaptive functioning deficits (Chudley et al., 2005; Stratton et al., 1996). The clinical manifestations of FASD may include visual and hearing deficits, mental and behavioural disorders, language disorders, cardiac anomalies, urogenital defects and skeletal abnormalities (Popova, Lange, Shield et al., 2016). A recent study identified over 400 disease conditions associated with FASD (Popova, Lange, Shield et al., 2016). Some of these comorbid conditions (e.g., language, auditory, visual, developmental/cognitive, mental and behavioural problems) are highly prevalent among individuals with FAS, ranging from 50% to 91%, and exceed the rate in the general population (Popova, Lange, Shield et al., 2016). The neurodevelopmental impairments associated with FASD can, later in life, lead to other common adverse outcomes, such as academic failure, substance abuse, mental health problems, contact with law enforcement and an inability to live independently and obtain and maintain employment (Streissguth et al., 1996).

While human-subject research has not been able to delineate the pattern, amount and/or critical period of prenatal alcohol exposure necessary for structural and functional teratogenesis, animal model–based research has demonstrated that the brain is vulnerable to the teratogenic effects of alcohol at virtually every stage of its development, and that the brain is susceptible to prenatal alcohol damage across a wide range of regions (Sulik, 2014). The resulting deficits can range from gross structural abnormalities, such as microcephaly, to subtler damage, including cell death or degeneration in various brain regions (Sulik, 2014). Furthermore, the type and severity of birth defects induced by prenatal alcohol exposure largely depend

on the pattern, the dose and the developmental stage of the embryo at the time of exposure (Jacobson & Jacobson, 1994, 1999; O'Leary-Moore et al., 2011; Sood et al., 2001; Sulik, 2014).

Although animal studies have shown that high blood alcohol concentrations (achieved by consuming a large amount of alcohol over a relatively short period of time—i.e., binge drinking) are the most harmful to a developing fetus (Clarren et al., 1992; Goodlett & Eilers, 1997; Livy et al., 2003), multiple animal models have also shown that even low levels of prenatal alcohol exposure can lead to brain dysfunction, which can in turn lead to behavioural abnormalities (Hamilton et al., 2014). However, beyond the amount of alcohol consumed and the gestational timing of consumption, there are multiple factors that modify fetal susceptibility to the teratogenic effects of ethanol, such as variability in the metabolism and genetic background of both the mother and fetus, environmental influences, maternal age, smoking, nutritional status, stress levels and, possibly, paternal lifestyle (Day et al., 2016; Eberhart & Parnell, 2016; May & Gossage, 2011).

The complexity and chronicity of FASD affects both the individual and their family, and in many cases, people with FASD require lifelong assistance from a wide range of services, including health, community, remedial education and many others. Furthermore, given the high rate of comorbidity among individuals with FASD (Popova, Lange, Shield et al., 2016), it is likely that health care providers from all specialties, along with other service providers, will encounter cases of FASD. Accordingly, FASD is recognized to impart a significant economic burden on society (Lupton et al., 2004; Popova, Lange, Burd et al., 2016). A recent cost-of-illness study (Popova, Lange, Burd et al., 2016) examined the impact of FASD on the material welfare of Canadian society in 2013 by analyzing the direct costs of resources spent on health care, law enforcement, children and youth in care, special education, supportive housing, long-term care, and prevention and research, as well as the indirect costs of productivity losses of individuals with FASD due to increased morbidity and premature mortality. Based on these cost drivers, it was estimated that the annual cost of FASD in Canada is approximately $1.8 billion (from approximately $1.3 billion as the lower estimate, up to $2.3 billion as the upper estimate). The highest contributor to the overall FASD-attributable cost was the cost of productivity losses due to morbidity and premature mortality, which accounted for 41% ($532 million–$1.2 billion) of the overall cost. The second highest contributor to the total cost was the cost to the correctional system, accounting for 29% ($378.3 million). The third highest contributor was the cost of health care, at 10% ($128.5–$226.3 million).

Only a few studies have attempted to estimate the prevalence of FAS or FASD among the general population in Canada (Asante & Nelms-Matzke, 1985; Habbick et al., 1996; Thanh et al., 2014). Based on available data, the prevalence in the general population was recently estimated to be about 1.1 per 1,000 for FAS and 7.9 per 1,000 for FASD (Lange et al., 2017; Popova, Lange, Probst, Gmel et al., 2017). However, the prevalence has been found to be much higher in special populations. For example, the prevalence of FASD in northern communities, based on five studies (Asante & Nelms-Matzke, 1985; Kowlessar, 1997; Robinson et al., 1987; Werk et al., 2013; Williams et al., 1999), was estimated to be 16 times higher than in the general population (Popova, Lange, Probst & Rehm, 2017). The prevalence of FASD among permanent wards in Ontario was reported to be 32.6 per 1,000 (Burge, 2007) and 113 per 1,000 among children in the care of Manitoba's Child Welfare Agency (Fuchs et al., 2005). More recently, Fuchs and Burnside (2014) reported the prevalence of FASD among children and youth in care in Alberta, Manitoba and Ontario to be 103.3 per 1,000, 122.7 per 1,000 and 105.1 per 1,000, respectively; these FASD rates among children in care range from approximately 13.1 to 15.5 times higher compared with the general population of Canada. Furthermore, among adoptees from Eastern Europe in Quebec, the prevalence rates of FAS and FASD have been reported to be 34.5 per 1,000 and 241.4 per 1,000, respectively (Roberts et al., 2009).

Another special population with a suspected high prevalence of FASD is youth and adults in correctional systems. Although data on the prevalence of FASD in correctional systems are absent worldwide, data from Canadian studies are available. Fast et al. (1999) reported a prevalence of 233.5 per 1,000 among youth who were remanded for a forensic psychiatric/psychological assessment in British Columbia and the Yukon. Murphy et al. (2005) reported a prevalence of 116.8 per 1,000 among youth in juvenile detentions centres in British Columbia, and Rojas and Gretton (2007) reported a prevalence of 108.7 per 1,000 among youth in a youth sexual offence treatment program in British Columbia. For the adult correctional population, MacPherson et al. (2011) reported a prevalence of 98.9 per 1,000 among adult male offenders in a medium-security penitentiary in Manitoba, and most recently, McLachlan (2017) reported a prevalence of 175.0 per 1,000 among adults supervised on an active legal order through Yukon Corrections. Based on existing epidemiological data and data from Justice Statistics Canada, it has been estimated that youth with FASD are 19 times more likely to be incarcerated than youth without FASD in any given year (Popova et al., 2011). Thus, FASD is a huge risk factor for problems with the law (as both perpetrators and victims of crimes) and recidivism.

A recent comprehensive literature review revealed that there have been no rigorous population-based epidemiologic studies of FAS or FASD in Canada that used extensive outreach or other methods of active case ascertainment (Popova, Lange, Probst & Rehm, 2017). The few studies that do exist had numerous acknowledged limitations, such as using small samples, being conducted in small communities and excluding individuals who did not meet criteria for a diagnosis of full FAS. The prevalence estimates provided by these studies are not only out of date, but also, as a result of their limitations, are not generalizable to the Canadian population or applicable for decision-making purposes. Moreover, most of these studies used clinic- or record-based systems without active recruitment of participants. Due to such methodological limitations, the prevalence estimates are more likely to be underreported in any population (May & Gossage, 2001). Active case ascertainment methods have been used with school-age children in several countries, including Australia (Elliott et al., 2008); Croatia (Petkovi & Bariši, 2010, 2013); Italy (May et al., 2006, 2011); South Africa (May et al., 2000, 2007, 2013; Olivier et al., 2013; Urban et al., 2008, 2015; Viljoen et al., 2005); and the United States (Burd et al., 1999; Clarren et al., 2001; May et al., 2014, 2015).

Given that FASD has been recognized as the leading known preventable birth defect and cause of developmental delay among Canadians, it is crucial to estimate the prevalence of this disorder. The estimates of the prevalence of FASD are vital for early detection, diagnosis and intervention, as well as for informing policy-makers and politicians of the impact of FASD. In addition, prevalence estimates will help to set priorities for public health policy, public health initiatives funding and health care planning. Updated prevalence estimates are essential to effectively prioritize, plan and deliver health care to high-needs populations such as children, youth and adults with FASD. These estimates are also vital for assessing the population burden of disease and allocating resources for health care and prevention.

2. Objective of the Study

The objective[1] of this study was to determine the population-based prevalence of FASD among elementary school students, aged 7 to 9 years, who attend public schools in the Greater Toronto Area (GTA) in Ontario, Canada.

1 Please note that the objective of this study was broader in scope—to estimate the prevalence of neurodevelopmental disorders (such as fetal alcohol spectrum disorder [FASD], attention-deficit/hyperactivity disorder, autism spectrum disorder and conduct disorder). This report presents findings on the prevalence of FASD only. Prevalence estimates for other neurodevelopmental disorders found during this study are available from the authors upon request.

The objective was a priority of the FASD National Strategic Projects Fund, which was expressed during the National FASD Prevalence Plan Forum held in Manitoba on October 12–14, 2011, and funded by the Public Health Agency of Canada. This project was intended to provide federal, provincial and territorial governments and decision-makers in Canada with evidence-based methods for estimating the population-based prevalence of FASD.

3. Methodology

This study was part of the World Health Organization International Collaborative Research Project on Child Development and Prenatal Risk Factors with a Focus on FASD, which, in addition to Canada, includes countries of Eastern and Central Europe (Belarus, Moldova, Ukraine) and Africa (Namibia, Seychelles). The methodology for determining the prevalence of FASD was developed in consultation with leading international researchers (see "Acknowledgments" section) under the guidance of the World Health Organization, as well as the National Institute on Alcohol Abuse and Alcoholism, and was based on advances in the diagnosis of FASD in Canada (Chudley et al., 2005). The methods were then used to guide the current pilot study, which was intended to test the feasibility and generalizability of the methodology itself.

DESIGN OF THE STUDY

This screening study used a cross-sectional, observational design using active case ascertainment (an epidemiological surveillance strategy in which cases are actively sought for examination and diagnosis), along with retrospective collection of prenatal alcohol exposure information. The study procedures followed a step-wise approach, where only those students meeting predetermined criteria proceeded to the subsequent phase. Figure 1 provides a schematic diagram that depicts the methodology employed.

SAMPLING AND RECRUITMENT

This study was conducted among a sample of students, aged 7 to 9 years, who attended public schools in the GTA from September 2014 to June 2017. Students whose disabilities or behavioural problems were known to be caused by well-characterized genetic factors (e.g., Down's syndrome, Williams syndrome) or by postnatal brain injuries were excluded from the study.

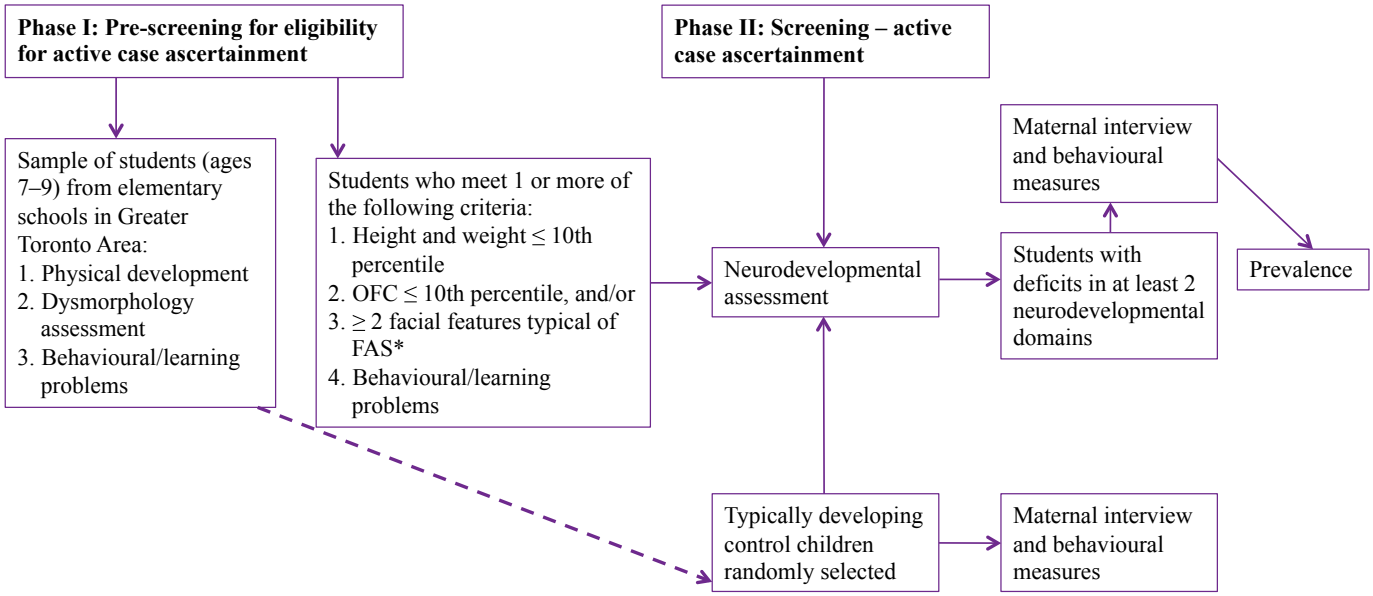
Sampling Frame

The GTA is made up of five regional municipalities: Durham, Halton, Peel, Toronto and York. It is the most populous metropolitan area in Canada, with a total population of 6.42 million in 2016 (Statistics Canada, 2017). The GTA is home to approximately 18.3% of Canada's population (Statistics Canada, 2017) and is representative of the general population of Ontario and Canada with respect to sex, age and drinking patterns (see Appendix A for a comparative analysis of the regional municipalities of the GTA, the GTA, the province of Ontario and Canada).

Public schools in the GTA are administered by 10 district school boards: five secular (non-religious) and five separate (Catholic) boards; there is one secular and one separate board in each region. In 2014–2015, there were 1,514 public elementary schools in the GTA (1,046 secular schools and 468 separate schools), with a total enrolment size of 642,014 (458,359 for secular schools, 183,655 for separate schools) (Government of Ontario, 2014).

FIGURE 1

Study methodology employed



OFC = occipitofrontal circumference.

*Short palpebral fissures, thin upper lip, smooth/flat philtrum.

It should be recognized that the Canadian education system is characterized by inclusion, meaning that all children (including those with intellectual disabilities) attend a "mainstream" school, where they are given equal opportunity and support to study together with their typically developing peers in the same classroom. There are no specialized public schools for children or youth with learning disabilities or developmental delays. The only exception is if the parents/guardians of a child with an intellectual disability have paid for their child to attend a private school.

Sampling Process

Permission to conduct external research was solicited from all 10 school boards. Within the boards that agreed to participate, schools were randomly selected, with preference given to the school with the highest enrolment size. In each of the participating schools, parents of all students in grades 2, 3 and 4 were invited to have their children participate in the study. Active written consent was sought from the parents/guardians.

The informed consent process was as follows: A letter from the principal of each school was sent home with the students, informing their parents/guardians of the study and its purpose, and confirming the school's and board's support of the initiative. The research co-ordinator then went into the school to introduce the study to teachers and students, and to give students consent forms to take home to their parents/guardians. One week later, a second round of consent forms was sent home with students who had not yet returned the completed form. Parents/guardians were given two weeks to return it. All students whose parents/guardians gave consent were then asked by a member of the research team whether they would agree to participate in the study. Those who were interested provided written assent. Only students who agreed took part in the study. All participating students received a small gift as a token of appreciation.

DATA COLLECTION

Data collection involved two phases: Phase I, which involved pre-screening, and Phase II, which involved screening (active case ascertainment, described below; see Appendix B). Data were collected independently by three research groups to minimize selection bias. The first group assessed growth and behavioural and/or learning problems, and conducted dysmorphology assessments; the second group conducted the neurodevelopmental assessments; and the third group conducted maternal interviews. Thus, the information on physical development and dysmorphology was collected and analyzed independent of the neurodevelopmental assessment and the maternal interview to the greatest extent possible.

Phase I: Pre-screening

The purpose of the pre-screening phase was to identify students for a further detailed assessment within the framework of active case ascertainment. The pre-screening phase addressed three aspects of child development relevant to the diagnosis of FASD: 1) growth deficits; 2) facial features characteristic of FAS and pFAS; and 3) behavioural and/or learning difficulties.

Physical Development

To determine the presence of growth deficits, research assistants measured each student's height, weight and occipitofrontal circumference (OFC). Percentiles were calculated using the respective Centers for Disease Control and Prevention (n.d.) clinical growth charts.

Dysmorphology Assessment

In addition to identifying the characteristic facial features that discriminate individuals with and without FAS and pFAS (short palpebral fissures, smooth or flattened philtrum, thin vermilion border of the upper lip), the dysmorphology assessment involved measuring other characteristic features documented to be common among people with FASD, such as ptosis, epicanthal folds, strabismus and "hockey stick" palmar crease (see the physical examination form in Appendix C). The primary method of conducting the dysmorphology assessment was by direct examination of facial and other relevant morphological features. Available normative data were used to compare the palpebral fissure length (Clarren et al., 2010) and inner canthal distance (Hall et al., 1989) measurements to calculate percentile rank. As a secondary method, facial photographs were taken (of students whose parents/guardians provided consent) to calculate computerized measurements of students' facial features. The photographs were also used to validate the direct examination, and were analysed using the FAS Facial Photographic Analysis Software, version 2.0 (Astley, 2012). Parents/guardians were given the opportunity to opt out of having their child photographed.

Behavioural/Learning Problems

Students with behavioural and/or learning difficulties (e.g., maladaptive behaviour, inattention, hyperactivity, learning problems) were identified by teachers and/or parents/guardians. Wherever possible, the school's special education teacher was approached to identify students with suspected behavioural and/or learning difficulties.

Proceeding to Phase II Screening

Students were selected to proceed to Phase II if they met one or more of the following criteria:

a. height and weight at or below the 10th percentile;
b. OFC at or below the 10th percentile;
c. presence of at least two of the three characteristic facial features that discriminate individuals with and without FAS:
    i. short palpebral fissures (2 standard deviations below the mean; at or below the 3rd percentile),
    ii. smooth or flattened philtrum (4 or 5 on the 5-point Likert scale of the lip-philtrum guide), and
    iii. thin vermilion border of the upper lip (4 or 5 on the 5-point Likert scale of the lip-philtrum guide); and
d. existing behavioural and/or learning problems, existing neurodevelopmental disorder (e.g., attention-deficit hyperactivity disorder, conduct disorder, autism spectrum disorder), and/or learning disability.

Typically Developing Control Children

Typically developing control children were randomly selected from a list of all students who completed Phase I and who did not meet any of the criteria to qualify them to proceed to Phase II (see below). These students underwent a complete assessment (i.e., physical, dysmorphological and neurodevelopmental assessments; maternal interviews to collect prenatal alcohol exposure history; and behavioural observations and ratings), as described below, to obtain normative data. Students who were subsequently found (based on maternal interview) to have been prenatally exposed to alcohol at "high risk" levels (see definitions below) or to have a pre-existing neurodevelopmental disorder were excluded from the control group. Following Phase I, all study personnel conducting the assessments were blinded as to which students were selected as controls, and which students were selected because they met the eligibility criteria for Phase II.

Phase II: Screening – Active Case Ascertainment

Phase II included three assessments: 1) neurodevelopmental assessment; 2) maternal interview, which collected information on demographics and living environment, pregnancy history, maternal alcohol use (prior to and following pregnancy recognition, as well as current use), nutrition during pregnancy, and tobacco and drug use during pregnancy; and 3) behavioural observations and ratings by parents/guardians.

Neurodevelopmental Assessment

A team of qualified psychometrists conducted the neurodevelopmental assessments, which included tests of attention, executive function, general cognition, language, processing speed, sensorimotor working memory and visuospatial processing.

The following neurodevelopmental test battery was used:

Tests of General Cognition

- The full Wechsler Abbreviated Scales of Intelligence, second edition (WASI-II), which includes the following four subtests:
  - Vocabulary
  - Similarities
  - Block Design
  - Matrix Reasoning

Measures of Attention, Executive Function, Language, Processing Speed, Sensorimotor Working Memory, and Visuospatial Processing

- The following four subtests from the Wechsler Intelligence Scale for Children, fourth edition (WISC-IV):
  - Digit Span (forward and backward)
  - Symbol Search
  - Coding
  - Letter–Number Sequencing
- The following four subtests from the Developmental Neuropsychological Assessment, second edition (NEPSY-II):
  - Auditory Attention and Response Set
  - Fingertip Tapping
  - Arrows
  - Word Generation

Maternal Interview and Behavioural Observations/Ratings

An interview with the biological mother was requested for students who demonstrated deficits (defined as two standard deviations below the mean on a subtest) in a minimum of two domains assessed during the neurodevelopmental assessment. This threshold was set to increase the likelihood that all potential cases were identified, as impairment of a minimum of three domains is necessary for a FASD-specific diagnosis. The 30-minute semi-structured interviews were conducted via telephone. Following the interview, mothers received a gift card as a token of appreciation for their time. During the interview, data were collected on demographics and living environment, pregnancy history, alcohol use (during the past 30 days, lifetime drinking behaviour and drinking behaviour prior to and following pregnancy recognition with the child in the study), nutrition during pregnancy, and tobacco and other drug use prior to and following pregnancy recognition (see Appendix D). Questions regarding alcohol consumption during pregnancy were masked by questions pertaining to mothers' demographics, pregnancy history and nutrition during pregnancy. The definition of a standard drink was provided to each mother to calibrate the amounts consumed, and drink conversion was done whenever necessary using the standard drink conversion chart in Appendix E.

At the end of the interview, the mother was asked to complete the Child Behavior Checklist (CBCL), a well-established standardized parent/caregiver questionnaire used to evaluate social competencies and behavioural problems in children aged 6 to 18 years. It involves a series of open-ended questions and a rating scale of 113 behavioural descriptors.

A minimum of three attempts to contact the biological mother via the method indicated on the consent form—that is, email or telephone—were made. In cases where the biological mother was no longer present in the child's life, alternative sources of information (i.e., birth/medical records, adoption records) regarding

prenatal exposures were not sought (as directed by the Research Ethics Boards). In these cases, the student's biological father or legal guardian was asked to complete the CBCL. All interviews were conducted by experienced interviewers who were fully trained to conduct interviews on sensitive issues such as alcohol consumption during pregnancy.

SCREENING RESULTS: CASE CONFERENCES

The summary findings from the three independent research groups were discussed on a case-by-case basis during multidisciplinary case conferences for all students who proceeded to Phase II and demonstrated deficits in a minimum of two domains assessed during the neurodevelopmental assessment, as well as for the typically developing control children. The selected cases were first reviewed independently by four experts, as well as by the principal investigator and the study co-ordinator (this group included psychologists, geneticists, medical doctors and epidemiologists). They were then discussed during the case conferences. Final diagnostic conclusions were made by consensus. See Appendix F for the final diagnosis form.

The terms "deferred" and "suspected" were used as part of the screening. Deferred cases were those where prenatal alcohol exposure was identified, but where less than three central nervous system domains were considered impaired (thus, the diagnostic criteria for an FASD-specific diagnosis were not met at the time of the assessment). Such students should still undergo a full multidisciplinary diagnostic assessment in the future to determine whether they meet the criteria for a FASD-specific diagnosis at a later time. Suspected cases were those where prenatal alcohol exposure was identified and the diagnostic criteria for an FASD-specific diagnosis were met at the time of the assessment.

It was not possible to confirm any diagnoses, as this was only a screening study and official diagnosis requires a full multidisciplinary diagnostic assessment. The parents/guardians of all students who screened positive for FASD were given their child's assessment results and a written recommendation that they see their family health care provider and receive a full multidisciplinary diagnostic assessment so a proper medical diagnosis could be formally established.

Furthermore, as directed by the Research Ethics Board of Health Canada / Public Health Agency of Canada, the diagnosis of FASD or any other suspected diagnoses were not communicated to the parents/guardians; rather, parents/guardians received the screening results as an independent assessment of the child's strengths and weaknesses in regard to the physical evaluation and neurodevelopmental assessment.

FASD DIAGNOSTIC CATEGORIES

The diagnostic criteria for FAS, pFAS and ARND followed the 2005 Canadian guidelines for diagnosis,[2] which were developed through broad-based consultation among Canadian and American experts in the diagnosis of FASD and its related disabilities (Chudley et al., 2005).

As per the opinion of the multidisciplinary team of experts in FASD diagnosis and in alignment with the revised Canadian FASD diagnostic guidelines (Cook et al., 2016), prenatal alcohol exposure was considered to pose "high risk" if the biological mother reported two or more binge drinking episodes (four or more standard drinks on a single occasion) or seven or more standard drinks within one week. Prenatal alcohol exposure was considered to pose "some risk" if the biological mother reported alcohol consumption, but at lower than high-risk levels. A standard drink is equal to a 341 mL can or bottle of beer, a 142 mL glass of wine, a 85 mL

2 The Canadian FASD diagnostic guidelines were updated in 2016 (Cook et al., 2016).

glass of fortified wine (e.g., sherry, port, vermouth) or a 43 mL shot of liquor (or spirits such as rye, rum, whisky, vodka; see Appendix E for the standard drink conversion chart used during the maternal interview).

FOLLOW-UP PROCEDURES AFTER SCREENING: RECOMMENDATIONS

Parents/guardians received the screening results of students with suspected FASD. They were also given a written recommendation that their child see their family health care provider and undergo a full diagnostic assessment so a formal medical diagnosis could be established. The current report recommends that a needs assessment of these students be conducted following their multidisciplinary assessments and that necessary referrals be made to health care providers, social services, education programs and supports. It also recommends a three-month follow-up with the families of students with suspected FASD to further facilitate the referrals and supports if necessary.

PREVALENCE ESTIMATION

Scenario 1 (Main Analysis)

As described in the Methodology section of this report, the study used a step-wise approach to estimate the prevalence of FASD and each of the diagnostic categories within the spectrum (FAS, pFAS and ARND). For the main scenario, it was assumed that there was no difference in the risk of FASD between those students whose parents/guardians provided consent to participate and those whose parents/guardians did not consent.

For the estimation of FASD, only those students who met predetermined criteria proceeded to the next phase. Specifically, the selection rate (SR) for each diagnostic category was estimated based on the number of students in the sample (n_t) and the number of students who met one or more of the criteria (indicators of FASD) (n_i), as this formula shows:

[Formula 1]

$$SR = \frac{n_i}{n_t}$$

To estimate each prevalence within the spectrum in the general population (PR_G), SRs were used to account for students who dropped out or were lost to follow-up during each phase. As such, the prevalence rates per 1,000 people in the general population (PR_G) were estimated, taking into account the SRs (see Formulas 2 and 3). In the case of FAS, the prevalence (P_{FAS}) did not account for the SR with respect to the maternal interviews (because FAS can be diagnosed without confirmation of prenatal alcohol exposure), whereas for other FASD diagnoses, the prevalence (P_{FASD}) of suspected cases identified among those students for whom maternal interview data were available was estimated. Each prevalence figure was then multiplied by 1,000 (k) to transform the prevalence into population rates (see Formulas 2 and 3).

[Formula 2]

$$PR_{FAS\_G} = SR_{pI} \bullet P_{FAS} \bullet k$$

[Formula 3]

$$PR_{FASD\_G} = SR_{pI} \bullet SR_{pII} \bullet P_{dx} \bullet k$$

SR_{pI} is the selection rate following Phase I; SR_{pII} is the selection rate following Phase II; P_{FAS} is the number of suspected cases of FAS; and P_{dx} is the number of suspected cases of each FASD diagnostic category (including FAS).

Scenario 2 (Sensitivity Analysis; Most Conservative Approach, Lower Estimate)

A sensitivity analysis was performed, where it was assumed that all students who were not selected to proceed to each subsequent phase and all students whose parents/guardians did not provide consent to participate had no risk of having FASD. For these estimations, the response prevalence (RP) following Phase I and II was incorporated into the prevalence estimations (see Formulas 4 and 5).

[Formula 4]

$$PR_{FASD\_G} = SR_{pI} \bullet RP_{pII}\, P_{FAS} \bullet k$$

[Formula 5]

$$PR_{FASD\_G} = SR_{pI} \bullet SR_{pII} \bullet RP_{pI} \bullet RP_{pII} \bullet P_{dx} \bullet k$$

Scenario 3 (Sensitivity Analysis; Least Conservative Approach, Upper Estimate)

A sensitivity analysis was performed to account for the possibility of cases of FAS and other FASD diagnoses among non-selected individuals (i.e., typically developing control children). As such, this scenario includes the one case of suspected ARND found among the typically developing control children. In total, 87 of the 1,762 students who did not meet the criteria to proceed to Phase II were randomly selected to proceed through all study phases. In this analysis, the prevalence of FAS (PR_{FAS_C}) and FASD diagnoses (including FAS; PR_{FASD_C}) among a sample of 41 typically developing control children (i.e., those for whom maternal interview data were available) was incorporated into the above-noted prevalence estimates of FAS and other FASD diagnoses (including FAS) among the general population, using Formulas 6 and 7.

[Formula 6]

$$PR_{FAS\_SENS} = PR_{FAS\_G} + PR_{FAS\_c} \bullet 1 - (SR_{pI}) \bullet k$$

[Formula 7]

$$PR_{FASD\_SENS} = PR_{FASD\_G} + PR_{FASD\_c} \bullet 1 - (SR_{pI} \bullet SR_{pII}) \bullet k$$

Estimation of Confidence Intervals

The corresponding 95% confidence interval (CI) for each respective prevalence estimate was determined using a Monte Carlo–like methodology (Graham & Talay, 2013), by using the 2.5th and 97.5th percentiles of a distribution of prevalences comprised of 100,000 estimates generated by taking sets of samples for the uncertainty distributions of each of the lowest-level parameters.

STATISTICAL ANALYSIS

Demographic characteristics, growth, dysmorphology and neurodevelopmental/behavioural findings, as well as maternal characteristics of students with suspected FASD, were compared with those of typically developing control children, wherever possible. Comparisons were also made across diagnostic groups (FAS/pFAS, ARND and deferred cases). Chi-square was used for analysis of categorical variables. For continuous variables, unpaired Student's *t*-tests for normally distributed data or one-way analysis of variance (ANOVA) were used when comparing two or more groups, respectively. With a statistically significant ANOVA, post-hoc analyses using Tukey's pairwise comparisons of means with equal variance were performed. Significance was set at $\alpha = 0.05$. All statistical analyses were performed using Stata 15 (Stata Corporation, 2017).

ETHICS

The study protocol and all associated materials were reviewed and approved by the Research Ethics Boards at the Centre for Addiction and Mental Health (165/2012) and Health Canada / Public Health Agency of Canada (REB 2012-0052).

Ethical Considerations

The study adhered to the following ethical principles described in the Declaration of Helsinki (World Medical Association, 2013):

a. *Voluntary participation*: participation in this study was voluntary.
b. *Informed consent*: prospective parent/guardian participants were fully informed about the procedures involved in this study and gave written consent for their child and themselves (in the case of the maternal interview) to participate. Furthermore, students were informed about the study purpose and procedures and gave written assent to participate.
c. *Confidentiality and privacy*: information about participants was not available to anyone who was not directly involved in the study. For data collection and entry, participants received a unique ID code, which was used throughout the study. The linked file was stored on a password-protected computer, only accessible to the principal investigator and the study co-ordinator, and was destroyed when the study was completed.
d. *Beneficence and non-maleficence*: benefits to the participants and their families were maximized to the fullest extent possible. Parents of students with suspected FASD were provided with their child's screening results and referred for a full medical examination. No physical harm could be caused to participants by participating in this study. For the benefits of early diagnosis of FASD, see the Discussion section. The potential for psychosocial consequences, including stigmatization resulting from a positive screening result, was properly addressed. For example, no information on apparent differences between students who proceeded to Phase II and those who did not in terms of physical or mental development or other health-related issues was disclosed to students, teachers or other school personnel. Furthermore, all assessors and interviewers were appropriately trained regarding the sensitive nature of the study.

4. Results

SAMPLING AND RECRUITMENT

Five out of the 10 district school boards, representing four out of the five regional municipalities, agreed to participate. Approval was sought from 71 school principals, of whom 40 allowed their school to participate. From those schools that agreed to participate, 8,209 students were invited to participate. A total of 3,854 parents/guardians (46.9%) responded to the request for their child to participate in the study: 1,161 (30.1%) refused to provide consent, and 2,693 (69.9%) gave consent.

On the days of Phase I assessments, 137 students were absent, resulting in 2,556 students available for assessment. Of these, one student did not assent to participating. Therefore, a total of 2,555 students were assessed for growth, dysmorphology, behavioural and/or learning problems. Facial photographs were taken of 1,684 students (65.9%), all of whom had consent from their parents/guardians.

Based on the results of Phase I, 793 (31.0%) students were selected to proceed to Phase II (which included the neurodevelopmental assessment and maternal interview). The following results emerged from Phase I:

- 334 (42.1%) students had growth deficits (height and weight, and/or OFC at or below the 10th percentile) and/or at least two of the three characteristic facial features that discriminate individuals with and without FAS/pFAS.
- 101 (12.7%) students had growth deficits and/or at least two of the three characteristic facial features, along with behavioural and/or learning problems.
- 358 (45.1%) students had behavioural and/or learning problems, but no growth deficits or characteristics facial features.

Of the 793 students eligible for Phase II, 762 (96.1%) completed the neurodevelopmental assessment (22 [2.8%] were lost to follow-up and the parents/guardians of 9 students (1.1%) withdrew from the study prior to the Phase II assessments). Of the 762 students who were assessed, 323 (42.4%) had demonstrated neurodevelopmental deficits in a minimum of two domains assessed using a standard neurodevelopmental test battery. The biological mothers of these students were then invited for an interview, with a total of 132 (40.9%) biological mothers completing the interview. Of the remaining mothers, 33 (10.2%) declined the interview, 150 (46.4%) were unreachable and 8 (2.5%) were no longer in the child's life (in the latter case, the child's guardian completed the CBCL). A total of 136 (42.1%) parents/guardians of students with neurodevelopmental deficits in a minimum of two domains completed the CBCL. A schematic diagram depicting the sampling and recruitment methodology employed is presented in Figure 2.

Typically Developing Control Children

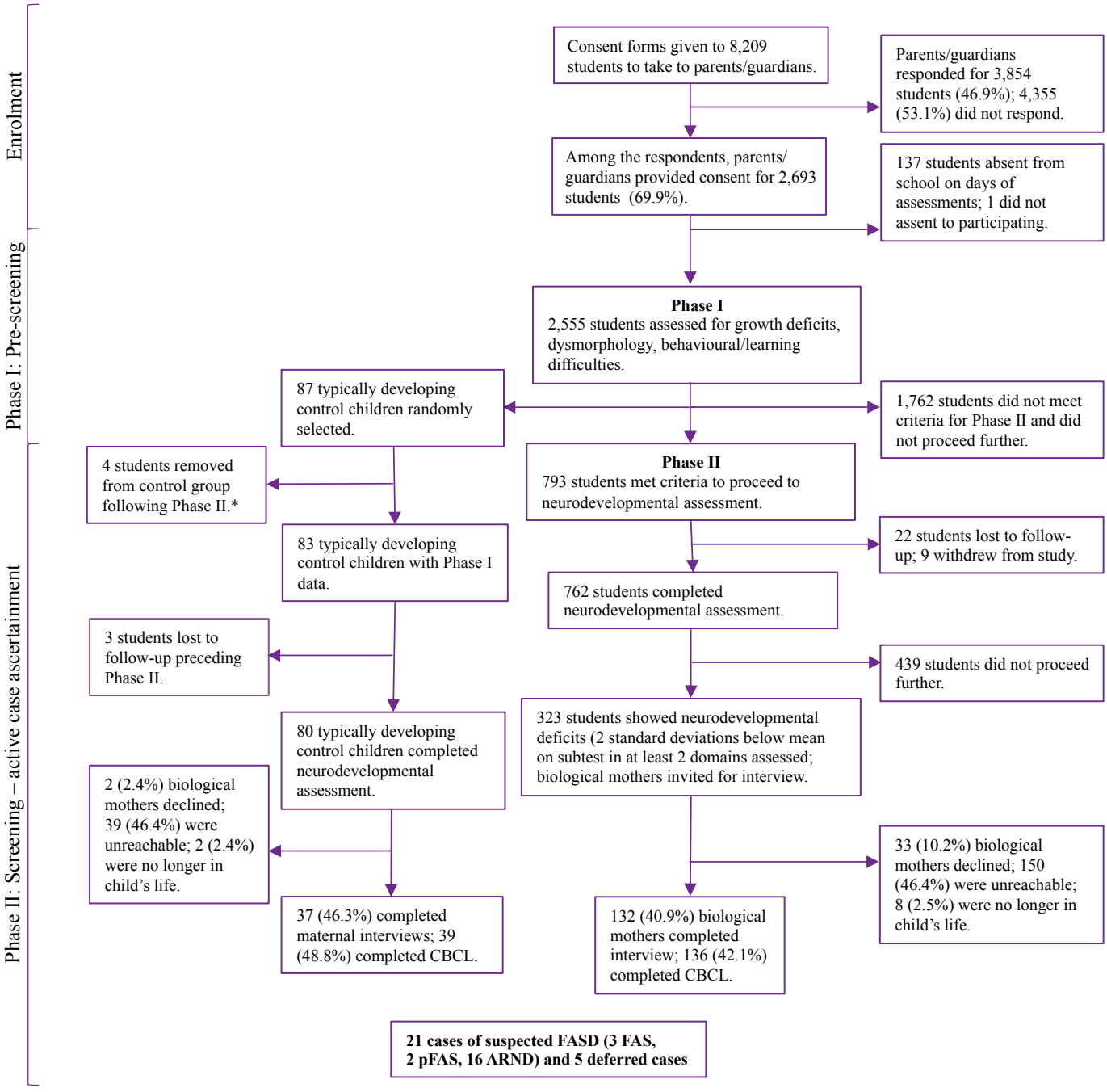
In total, 87 children were randomly selected from the list of students who completed Phase I and who did not meet any of the criteria to proceed to Phase II (as described above). Three of these students (3.4%) were lost to follow-up prior to Phase II. Maternal interview data were obtained for 41 (48.8%) of the remaining 84 typically developing control children. In the case of these remaining students, 2 (2.4%) mothers declined the interview, 39 (46.4%) were unreachable and 2 (2.4%) were no longer in the child's life (in the latter case, the child's guardian completed the CBCL). A total of 43 (51.2%; out of 84) parents/guardians completed the CBCL. Four students were excluded from the group of typically developing control children following Phase II because they were found to have a pre-existing neurodevelopmental disorder (1 had attention-deficit/hyperactivity disorder, 1 had speech delay, 1 was suspected to have ARND and 1 was considered a deferred case). The results are presented in Figure 2.

PHASE I: DEMOGRAPHICS, GROWTH MEASUREMENTS AND DYSMORPHOLOGY

Of the 2,555 students who participated in Phase I, 48.3% were male and had a mean age of 8.7 years (SD = 0.9; age range: 6.4–10.8 years). Students with suspected FASD (n = 21; see p. 27, "Prevalence of FASD") did not differ from typically developing control children (n = 83) in terms of sex, age and ethnicity. Students with suspected FASD were more likely to be at or below the 10th percentile for height and OFC compared with typically developing control children ($p < .001$). The mean height, weight and OFC of students with suspected FASD were 132.8 (SD = 7.7) cm, 31.3 (SD = 9.5) kg and 52.5 (SD = 2.2) cm, respectively. As expected, significantly more students with suspected FASD had shorter PFL (i.e., 2 SD below the mean) compared with typically developing control children ($p < .001$ for right PFL and $p < .01$ for left PFL). A smooth philtrum and narrow vermillion border of the upper lip (lip-philtrum guide scores of 4) were observed among 23.8% and 19.1%, respectively, of students with suspected FASD (see Table 1 for the detailed results of Phase I assessments).

FIGURE 2

Sampling and recruitment methodology



CBCL = Child Behavior Checklist; FASD = fetal alcohol spectrum disorder.

*Four students were removed from the group of typically developing control children due to having a pre-existing neurodevelopmental disorder, including one student with suspected ARND and one student who was considered to be a deferred case.

TABLE 1

Demographic characteristics and growth and dysmorphology measurements of screened students

	Screened in Phase I (*n* = 2,555)	Eligible for Phase II (*n* = 817)	Deficits in 2+ neuro-develop-mental domains (*n* = 323[a])	Selected for case conference review (*n* = 66[b])	Suspected FASD (*n* = 21)	Typically developing control children (*n* = 83)	Statistical test[c]	*p* value
Demographics								
Sex (% male)	48.3	55.2	58.8	50.0	52.4	59.0	$t = 0.547$	.586
Age (years) – mean (*SD*)	8.7 (0.9)	8.6 (0.9)	8.6 (1.0)	8.7 (1.0)	8.9 (0.8)	8.5 (0.8)	$t = 1.859$	.066
Range	6.4–10.8	6.7–10.6	6.9–10.4	6.9–10.3	7.6–10.4	6.5–10.5		.296
Ethnicity – *n* (%)							$X = 7.279$	
Caucasian	605 (23.7)	248 (30.4)	108 (33.4)	28 (42.4)	15 (71.4)	38 (45.8)		
African Canadian / Caribbean	244 (9.6)	73 (8.9)	41 (12.7)	9 (13.6)	1 (4.8)	3 (3.6)		
Eastern European	205 (8.0)	63 (7.7)	16 (5.0)	5 (7.6)	2 (9.5)	7 (8.4)		
Western European	394 (15.4)	124 (15.2)	50 (15.5)	11 (16.7)	3 (14.3)	16 (19.3)		
Chinese/Southeast Asian	313 (12.3)	79 (9.7)	30 (9.3)	3 (4.6)	0 (0.0)	3 (3.6)		
South Asian	353 (13.8)	95 (11.6)	31 (9.6)	4 (6.1)	0 (0.0)	8 (9.6)		
Other	437 (17.1)	135 (16.5)	47 (14.6)	6 (9.1)	0 (0.0)	8 (9.6)		
Growth measurements								
Height (cm) – mean (*SD*)	132.7 (7.9)	130.2 (8.2)	131.0 (8.7)	130.8 (9.1)	132.8 (7.7)	133.9 (7.2)	$t = 0.633$	.528
Height ≤ 10th percentile – *n* (%)	278 (10.9)	202 (24.7)	70 (21.7)	18 (27.3)	5 (23.8)	2 (2.4)	$X = 12.226$	< .001
Weight (kg) – mean (*SD*)	31.1 (8.0)	28.7 (7.8)	29.9 (8.6)	29.8 (9.2)	31.3 (9.5)	32.0 (8.5)	$t = 0.346$	.730
Weight ≤ 10th percentile – *n* (%)	272 (10.7)	202 (24.7)	65 (20.1)	17 (25.8)	4 (19.1)	7 (8.4)	$X = 1.996$	.158
OFC (cm) – mean (*SD*)	53.0 (1.7)	52.31 (1.9)	52.54 (1.9)	52.25 (1.9)	52.5 (2.2)	53.6 (1.4)	$t = 2.942$	.004
OFC ≤ 10th percentile – *n* (%)	256 (10.0)	254 (31.1)	79 (24.5)	20 (30.3)	5 (23.8)	0 (0.0)	$X = 20.760$	< .001
Dysmorphology								
Right PFL (cm) – mean (*SD*)	2.51 (0.18)	2.48 (0.19)	2.50 (0.21)	2.44 (0.17)	2.40 (0.15)	2.51 (0.14)	$t = 3.328$	.001
Right PFL 2 *SD* below mean – *n* (%)	582 (22.8)	281 (34.4)	105 (32.6)	28 (42.4)	10 (47.6)	9 (10.8)	$X = 15.180$	< .001
Left PFL (cm) – mean (*SD*)	2.51 (0.17)	2.48 (0.18)	2.50 (0.20)	2.45 (0.17)	2.41 (0.16)	2.51 (0.13)	$t = 2.658$	.009
Left PFL 2 *SD* below mean – *n* (%)	562 (22.0)	268 (32.8)	96 (29.8)	28 (42.4)	9 (42.9)	11 (13.3)	$X = 9.456$	.002
Inner canthal distance (cm) – mean (*SD*)	2.88 (0.26)	2.82 (0.26)	2.82 (0.28)	2.74 (0.25)	2.84 (0.31)	2.83 (0.22)	$t = 0.253$	.801
Inner canthal distance ≤ 25th percentile – *n* (%)	912 (36.3)	358 (44.6)	148 (46.7)	39 (60.9)	10 (47.6)	38 (46.3)	$X = 4.565$	.335
Philtrum length (cm) – mean (*SD*)	1.18 (0.24)	1.19 (0.28)	1.20 (0.30)	1.24 (0.40)	1.17 (0.18)	1.26 (0.42)	$t = 0.895$	.373
Philtrum score on lip-philtrum guide – *n* (%)							$X = 1.608$	.658
1	175 (6.9)	47 (5.8)	15 (4.6)	2 (3.0)	0 (0.0)	1 (1.2)		
2	907 (35.5)	238 (29.1)	90 (27.9)	15 (22.7)	5 (23.8)	30 (36.1)		
3	1,135 (44.4)	341 (41.7)	149 (46.1)	33 (50.0)	11 (52.4)	38 (45.8)		
4	327 (12.8)	182 (22.3)	65 (20.1)	16 (24.2)	5 (23.8)	14 (16.9)		
5	10 (0.4)	9 (1.1)	4 (1.2)	0 (0.0)	0 (0.0)	0 (0.0)		

cont'd

cont'd

	Screened in Phase I (*n* = 2,555)	Eligible for Phase II (*n* = 817)	Deficits in 2+ neuro-develop-mental domains (*n* = 323[a])	Selected for case conference review (*n* = 66[b])	Suspected FASD (*n* = 21)	Typically developing control children (*n* = 83)	Statistical test[c]	*p* value
Vermillion border score on lip-philtrum guide – *n* (%)							*X* = 1.620	.655
1	300 (12.0)	66 (8.2)	27 (8.5)	3 (4.7)	1 (4.8)	2 (2.4)		
2	1,135 (45.2)	323 (40.3)	134 (42.4)	21 (32.8)	9 (42.9)	35 (42.7)		
3	926 (36.9)	315 (39.3)	124 (39.2)	31 (48.4)	7 (33.3)	36 (43.9)		
4	143 (5.7)	93 (11.6)	29 (9.2)	9 (14.1)	4 (19.1)	9 (11.0)		
5	5 (0.2)	5 (0.6)	2 (0.6)	0 (0.0)	0 (0.0)	0 (0.0)		

FASD = fetal alcohol spectrum disorder; PFL = palpebral fissure length; *SD* = standard deviation.

[a]Out of 786 students who completed the neurodevelopmental assessment in Phase II. [b]Selected out of 323 students who demonstrated deficits in a minimum of two neurodevelopmental domains, along with 84 typically developing control children (total 407 cases). [c]Comparing students with suspected FASD with typically developing control children.

There were no statistically significant differences between students with suspected FASD and typically developing control children in terms of inner canthal distance, philtrum length, and frequency of hypoplastic midface, railroad track configuration of ear, strabismus, ptosis, epicanthal fold, anteverted nares, clinodactyly, camptodactyly, difficulties with pronation/supinaton of elbow and hockey stick upper palmar crease (not presented in Table 1; available from the authors upon request).

NEURODEVELOPMENTAL AND BEHAVIOURAL ASSESSMENT

Neurodevelopmental assessment data revealed that compared with typically developing control students, students with suspected FASD were characterized by lower scores on IQ ($p < .001$), verbal comprehension ($p < .001$), perceptual reasoning ($p = .002$), working memory ($p < .001$) and processing speed ($p < .001$), as per the composite scores of the WASI-II and WISC-IV (Table 2 and Figure 3). Furthermore, the standard scores on all but one of the subtests (NEPSY-II: Word Generation, Semantic, which measures language) were statistically significantly lower among students with suspected FASD compared with typically developing control children (Table 2 and Figure 4).

As depicted in Figure 5, students with suspected FASD were more likely than typically developing control children to have composite scores on the WASI-II and WISC-IV that were 1 to 2 standard deviations below the mean. Overall, significantly more students with suspected FASD had scores 1.5 standard deviations below the mean or lower on the Verbal Comprehension Index, Perceptual Reasoning Index, Full-Scale IQ-4, Working Memory Index and Processing Speed Index, compared with typically developing control children. Significantly more typically developing control children had scores at least 1.5 standard deviations above the mean on these indices, compared with students with suspected FASD.

TABLE 2

Mean standard scores on neurodevelopmental tests of students with suspected FASD and typically developing control children

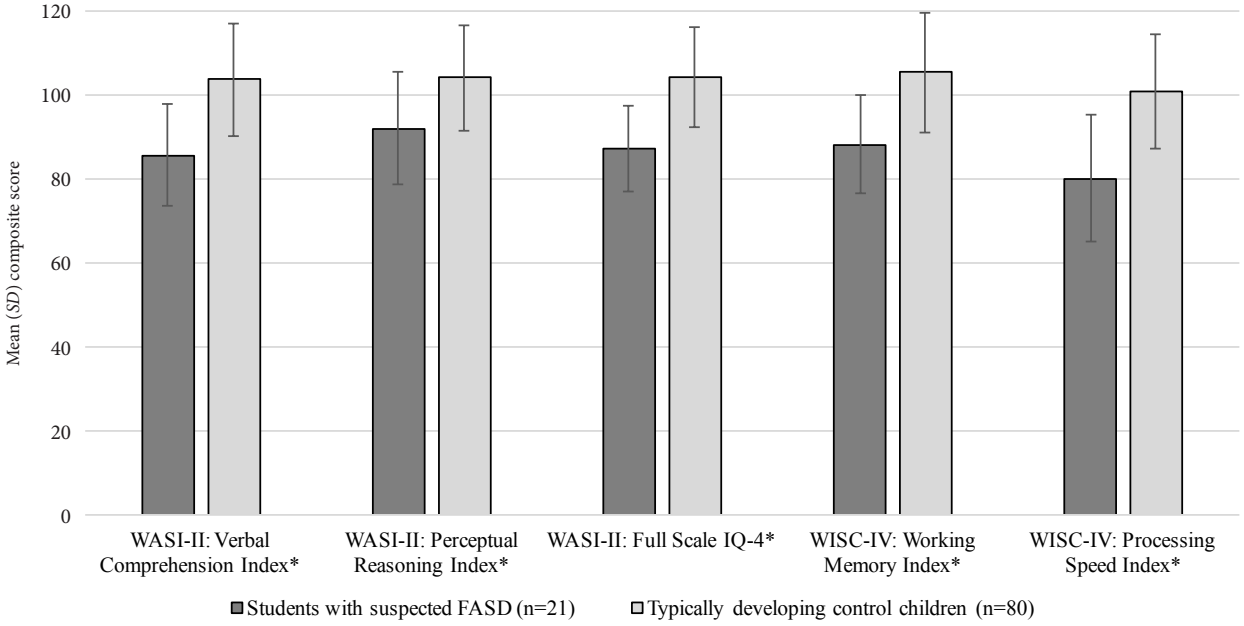
Neurodevelopmental test	Students with suspected FASD (n = 21)	Typically developing control children (n = 80)	Statistical test[a]	p value
WASI-II				
Verbal Comprehension Index (composite score) – mean (*SD*)	85.5 (12.0)	103.5 (13.5)	t = 5.559	< .001
Vocabulary (scaled score) – mean (*SD*)	7.6 (3.3)	11.3 (3.1)	t = 4.832	< .001
Similarities (scaled score) – mean (*SD*)	7.2 (2.2)	10.3 (2.7)	t = 4.875	< .001
Perceptual Reasoning Index (composite score) – mean (*SD*)	91.9 (13.5)	103.9 (12.6)	t = 3.837	< .001
Block Design (scaled score) – mean (*SD*)	8.6 (2.6)	11.5 (3.3)	t = 3.741	< .001
Matrix Reasoning (scaled score) – mean (*SD*)	8.9 (3.0)	10.4 (2.9)	t = 2.058	.042
Full-Scale IQ-4 (composite score) – mean (*SD*)	87.2 (10.2)	104.2 (11.9)	t = 5.969	< .001
WISC-IV				
Working Memory Index (composite score) – mean (*SD*)	88.0 (11.6)	105.2 (14.2)	t = 5.125	< .001
Digit Span (scaled score) – mean (*SD*)	9.3 (2.4)	11.1 (3.0)	t = 2.516	.014
Letter–Number Sequences (scaled score) – mean (*SD*)	6.6 (3.0)	11.3 (5.9)	t = 3.568	< .001
Processing Speed Index (composite score) – mean (*SD*)	80.0 (15.2)	100.7 (13.7)	t = 6.010	< .001
Coding (scaled score) – mean (*SD*)	5.9 (3.6)	9.5 (2.7)	t = 5.233	< .001
Symbol Search (scaled score) – mean (*SD*)	6.9 (2.8)	11.2 (5.8)	t = 3.322	.001
NEPSY-II				
Auditory/Executive Function				
Auditory Attention (scaled score) – mean (*SD*)	8.6 (3.9)	10.3 (2.8)	t = 2.247	.027
Response Set (scaled score) – mean (*SD*)	7.2 (3.8)	11.2 (3.0)	t = 5.135	< .001
Sensorimotor Processing				
Fingertip Tapping, Repetitions (combined scaled score) – mean (*SD*)	8.8 (3.5)	10.9 (2.9)	t = 2.834	.006
Fingertip Tapping, Sequences (combined scaled score) – mean (*SD*)	8.6 (3.3)	10.6 (2.5)	t = 3.027	.003
Fingertip Tapping, Dominant (combined scaled score) – mean (*SD*)	8.7 (3.5)	10.3 (2.5)	t = 2.398	.018
Fingertip Tapping, Non-dominant (combined scaled score) – mean (*SD*)	8.8 (3.6)	10.6 (2.3)	t = 2.910	.005
Visuospatial Processing				
Arrows (scaled score) – mean (*SD*)	7.1 (3.5)	10.7 (2.5)	t = 5.438	< .001
Language				
Word Generation, Semantic (scaled score) – mean (*SD*)	11.1 (2.7)	12.0 (2.5)	t = 1.668	.099
Word Generation, Letter (scaled score) – mean (*SD*)	7.6 (3.1)	10.1 (2.5)	t = 3.934	< .001

WASI-II = Wechsler Abbreviated Scales of Intelligence, 2nd edition; WISC-IV = Wechsler Intelligence Scale for Children, 4th edition; NEPSY-II = A Developmental Neuropsychological Assessment, 2nd edition.

[a]Comparing students with suspected FASD with typically developing control children.

FIGURE 3

Mean (*SD*) composite scores on the WASI-II and WISC-IV among students with suspected FASD and typically developing control children

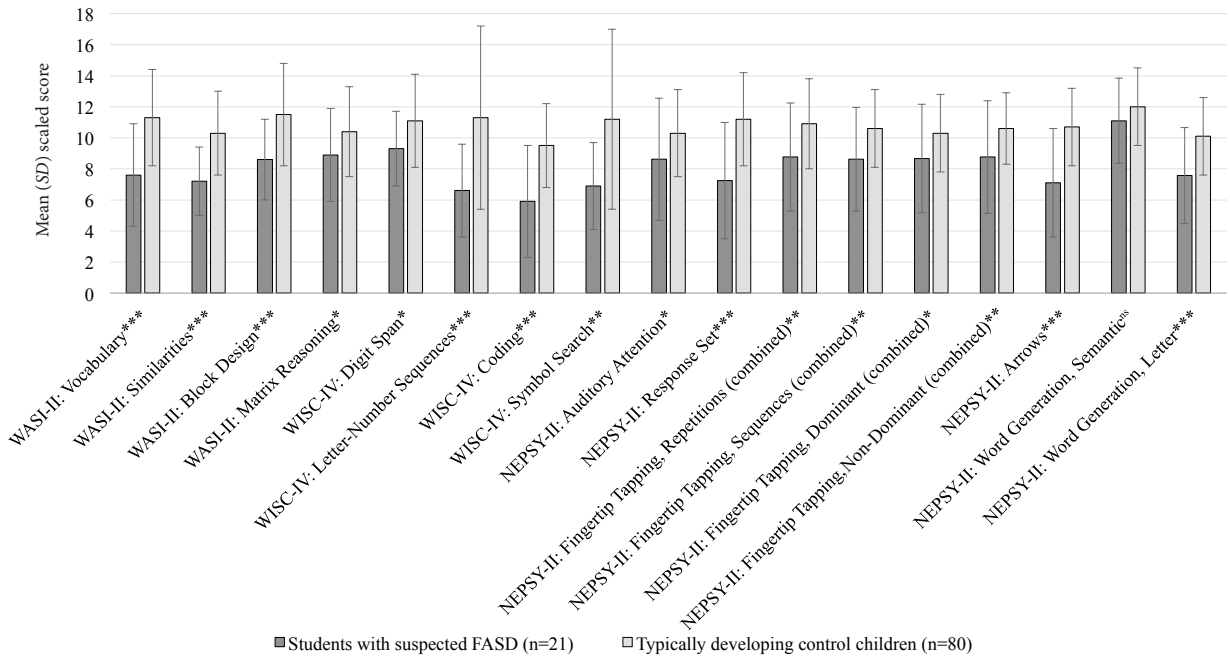


FASD = fetal alcohol spectrum disorder; WASI-II = Wechsler Abbreviated Scales of Intelligence, 2nd edition; WISC-IV = Wechsler Intelligence Scale for Children, 4th edition.

*$p < .001$.

FIGURE 4

Mean (*SD*) scaled scores on the subtests of the WASI-II, WISC-IV and NEPSY-II among students with suspected FASD and typically developing control children

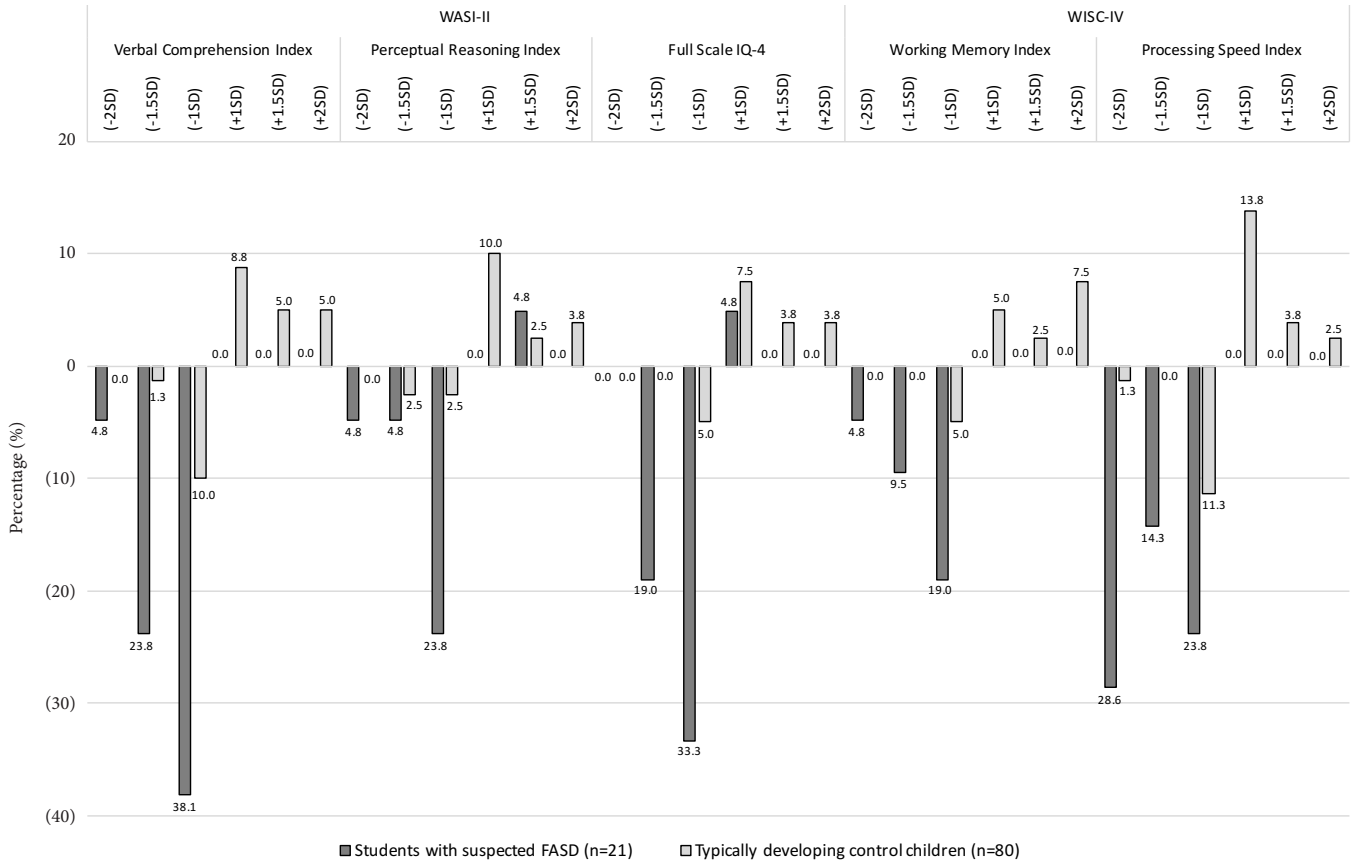


FASD = fetal alcohol spectrum disorder; WASI-II = Wechsler Abbreviated Scales of Intelligence, 2nd edition; WISC-IV = Wechsler Intelligence Scale for Children, 4th edition; NEPSY-II = A Developmental Neuropsychological Assessment, 2nd edition; ns = non-significant.

*$p < .05$. **$p < .01$. ***$p < .001$.

FIGURE 5

Percentage of students with suspected FASD and typically developing control children who scored 2, 1.5 and 1 *SD* below and above the mean on the WASI-II and WISC-IV composited scores



Note: The percentages for each composite score do not add up to 100% because the remaining students fell within the mean; that is, less than 1 *SD* above and more than 1 *SD* below the mean.

FASD = fetal alcohol spectrum disorder; *SD* = standard deviations; WASI-II = Wechsler Abbreviated Scales of Intelligence, 2nd edition; WISC-IV = Wechsler Intelligence Scale for Children, 4th edition.

With respect to the CBCL, students with suspected FASD scored significantly higher than typically developing control children on the Social Problems (p = .010), Thought Problems (p =.012), Attention Problems (p < .001) and Rule-Breaking Behavior (p = .002) Syndrome scales; Total Problems Syndrome Summary scales (p =.006); and Attention Deficit/Hyperactivity Problems (p = .001) and Conduct Problems (p = .009) DSM-Oriented scales (Table 3 and Figure 6). Typically developing control children scored significantly higher than students with suspected FASD on all Competence scales (Activities [p = .001], Social [p = .034], School [p < .001] and Total Competence [p < .001]) (Table 3 and Figure 6).

Furthermore, students with suspected FASD scored higher than typically developing control children (although not at statistically significant levels) on the Anxious/Depressed, Withdrawn/Depressed, Aggressive Behavior and Somatic Complaints Syndrome scales; Internalizing Problems and Externalizing Problems Syndrome Summary scales; Affective Problems, Anxiety Problems and Somatic Problems DSM-Oriented scales; as well as Sluggish Cognitive Tempo, Obsessive-Compulsive Problems and Post-Traumatic Stress Problems on the School-Age scales (Table 3 and Figure 6).

TABLE 3

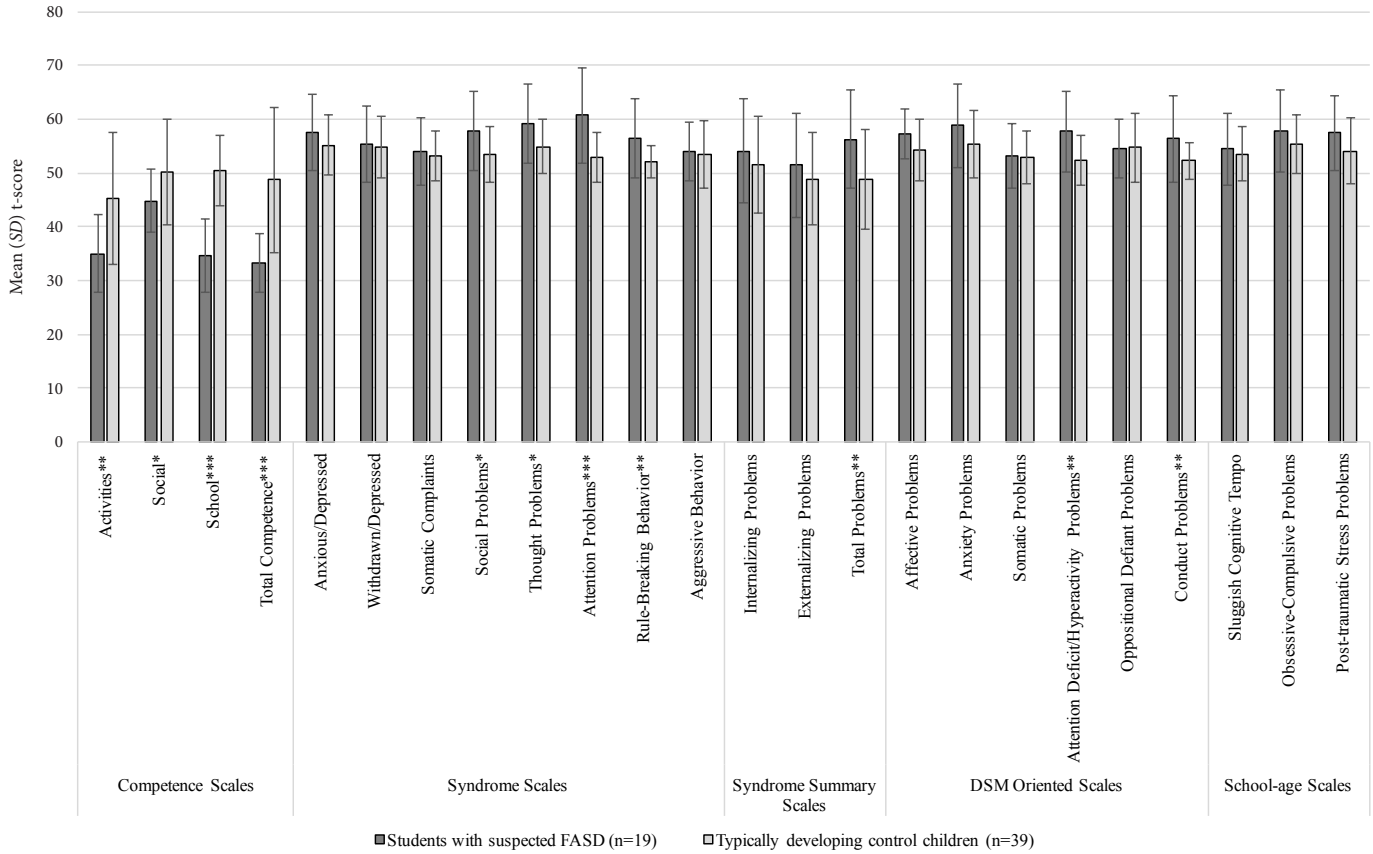
Mean scores on the Child Behavior Checklist (CBCL) of students with suspected FASD and typically developing control children

CBCL scale	Students with suspected FASD ($n = 19$)[a]	Typically developing control children ($n = 39$)	Statistical test[b]	p value
Competence scales				
Activities (*t*-score) – mean (*SD*)	35.0 (7.2)	45.3 (12.3)	$t = 3.371$	.001
Social (*t*-score) – mean (*SD*)	44.8 (5.8)	50.2 (9.8)	$t = 2.173$	.034
School (*t*-score) – mean (*SD*)	34.6 (6.8)	50.4 (6.5)	$t = 8.531$	< .001
Total Competence (*t*-score) – mean (*SD*)	33.3 (5.5)	48.7 (13.5)	$t = 4.618$	< .001
Anxious/Depressed (*t*-score) – mean (*SD*)	57.5 (7.1)	55.2 (5.6)	$t = 1.311$	.512
Withdrawn/Depressed (*t*-score) – mean (*SD*)	55.3 (7.1)	54.7 (5.7)	$t = 0.302$	.764
Somatic Complaints (*t*-score) – mean (*SD*)	54.0 (6.3)	53.2 (4.6)	$t = 0.582$	.563
Social Problems (*t*-score) – mean (*SD*)	57.8 (7.3)	53.4 (5.2)	$t = 2.652$	.010
Thought Problems (*t*-score) – mean (*SD*)	59.2 (7.4)	54.9 (5.0)	$t = 2.598$	.012
Attention Problems (*t*-score) – mean (*SD*)	60.7 (8.8)	52.9 (4.6)	$t = 4.449$	< .001
Rule-Breaking Behavior (*t*-score) – mean (*SD*)	56.4 (7.3)	52.1 (3.0)	$t = 3.191$	.002
Aggressive Behavior (*t*-score) – mean (*SD*)	53.9 (5.5)	53.4 (6.2)	$t = 0.287$	.775
Syndrome Summary scales				
Internalizing Problems (*t*-score) – mean (*SD*)	54.1 (9.6)	51.5 (9.0)	$t = 1.009$	.317
Externalizing Problems (*t*-score) – mean (*SD*)	51.4 (9.8)	48.9 (8.6)	$t = 1.004$	.320
Total Problems (*t*-score) – mean (*SD*)	56.2 (9.1)	48.8 (9.2)	$t = 2.864$	.006
DSM-Oriented scales				
Affective Problems (*t*-score) – mean (*SD*)	57.3 (4.7)	54.3 (5.8)	$t = 1.937$	.058
Anxiety Problems (*t*-score) – mean (*SD*)	58.8 (7.8)	55.4 (6.3)	$t = 1.794$	.078
Somatic Problems (*t*-score) – mean (*SD*)	53.1 (6.0)	52.9 (4.8)	$t = 0.143$	.887
Attention Deficit/Hyperactivity Problems (*t*-score) – mean (*SD*)	57.7 (7.4)	52.4 (4.6)	$t = 3.372$	.001
Oppositional Defiant Problems (*t*-score) – mean (*SD*)	54.6 (5.5)	54.7 (6.5)	$t = 0.081$	.936
Conduct Problems (*t*-score) – mean (*SD*)	56.3 (8.0)	52.3 (3.4)	$t = 2.729$	.009
School-Age scales				
Sluggish Cognitive Tempo (*t*-score) – mean (*SD*)	54.5 (6.7)	53.5 (5.0)	$t = 0.595$	.555
Obsessive-Compulsive Problems (*t*-score) – mean (*SD*)	57.9 (7.6)	55.3 (5.4)	$t = 1.490$	.142
Post-traumatic Stress Problems (*t*-score) – mean (*SD*)	57.4 (7.0)	54.1 (6.1)	$t = 1.851$	.070

[a]CBCL data are not available for two students with suspected FAS for whom a maternal interview was not obtained. [b]Comparing students with suspected FASD with typically developing control children.

FIGURE 6

Mean *(SD)* t-score on Child Behavior Checklist scales among students with suspected FASD and typically developing control children



FASD = fetal alcohol spectrum disorder.

*$p < .05$. **$p < .01$. ***$p < .001$.

MATERNAL CHARACTERISTICS

Total Sample of Interviewed Mothers

The 173 biological mothers interviewed had a mean age of 40.9 years (*SD* = 5.0; age range: 26–56 years). Almost all mothers were married or living with their partners at the time of their pregnancy (96.0%), were employed in the 12 months leading up to their pregnancy (81.5%), had achieved a post-secondary education (i.e., college diploma, university degree or graduate degree; 83.3%) at the time of their pregnancy, and had planned their pregnancy (72.8%). In regard to paternal characteristics of all interviewed mothers (*n* = 173), 93.6% were reportedly employed 12 months leading up to their partner's pregnancy, and the majority had achieved a post-secondary education at the time of their partner's pregnancy (65.3%).

Of 173 interviewed mothers, 74.6% reported consuming alcohol (any amount, at any frequency) prior to pregnancy recognition (11.0% reported "high-risk" levels and 63.6% reported "some risk" levels). Only 6.4% of mothers reported alcohol consumption at some-risk levels following pregnancy recognition. Overall, 34.1% (of 173) of mothers had smoked cigarettes prior to pregnancy recognition: 24.3% daily and 9.8% occasionally. Following pregnancy recognition, 86.4% of mothers who smoked before pregnancy recognition

quit smoking. Those mothers who continued to smoke during pregnancy (4.6%) did so daily, rather than occasionally. Furthermore, 28.6% of interviewed mothers reported using marijuana or hashish, 4.1% reported using club drugs, 0.6% reported using crack/cocaine and 6.4% reported using hallucinogens prior to pregnancy recognition. No one reported any drug use following pregnancy recognition.

Mothers of Students with Suspected FASD Compared with Mothers of Typically Developing Control Children

The mothers of students with suspected FASD did not differ significantly from mothers of typically developing control children with respect to age, ethnicity, marital status and employment status at the time of pregnancy with the child who participated in the study. However, mothers of students with suspected FASD had lower levels of education than mothers of typically developing control children at the time of pregnancy ($p < .01$). A total of 15.8% of mothers of students with suspected FASD reported receiving financial support (which was at least half of their income) from the child's grandmother or grandfather (10.5%) and/or from the child's father (5.3%) during pregnancy. Only 5.4% of mothers of typically developing control children reported receiving financial support, and the amount was less than half of their income ($p < .05$).

In terms of paternal characteristics, there were no statistically significant differences in employment status between fathers of students with suspected FASD and fathers of typically developing control children. However, a higher proportion of fathers of typically developing control children had achieved a post-secondary education at the time of their partner's pregnancy (86.4%) compared with fathers of students with suspected FASD (52.7%).

Among mothers of students with suspected FASD, only 63.2% of pregnancies were planned compared with 83.8% among mothers of typically developing control children (although the difference was not statistically significant). Compared with mothers of typically developing control children, the mean number of pregnancies was higher among mothers of students with suspected FASD (2.7 [$SD = 1.2$] vs. 3.5 [$SD = 2.3$], respectively); more children were born prematurely (10.8% vs. 15.8%, respectively); and more children were born with a birth defect (5.4% vs. 21.1%, respectively; none of these differences were statistically significant). Interestingly, mothers of students with suspected FASD had a mean point of pregnancy recognition that was approximately one week earlier than that of mothers of typically developing control children (4.4 [$SD = 1.2$] vs. 4.9 [$SD = 1.8$], respectively; however, this difference was not statistically significant).

None of the mothers reported having a current drinking problem or ever having sought help for a drinking problem. All mothers of students with suspected FASD reported alcohol consumption prior to pregnancy recognition (high-risk levels: 63.2%, and some risk levels: 36.8%). Only 10.5% mothers of students with suspected FASD reported alcohol consumption following pregnancy recognition (some-risk levels only).

Significantly more mothers of students with suspected FASD reported ever having smoked tobacco in their lifetime (73.7%) compared with mothers of typically developing control children (46.0%; $p < .05$). Moreover, significantly more mothers of students with suspected FASD reported smoking tobacco prior to pregnancy recognition compared with mothers of typically developing control children (68.4% vs. 18.9%, respectively; $p < .001$). In addition, the proportion of daily smokers was significantly higher among mothers of students with suspected FASD compared with mothers of typically developing control (57.9% vs. 8.1%, respectively; $p < .001$).

With respect to substance use prior to pregnancy recognition, there were no significant differences between mothers of students with suspected FASD and mothers of typically developing control, with the exception of marijuana or hashish. The proportion of mothers of students with suspected FASD who used marijuana or hashish was more than double the proportion of mothers of typically developing control (68.4 % vs. 27.0%, respectively; $p < .01$). Notably, none of the mothers reported any drug use following pregnancy recognition.

The maternal demographic characteristics and rates of substance use during pregnancy among all interviewed mothers, mothers of students with suspected FASD and mothers of typically developing control children are presented in Table 4.

TABLE 4

Maternal characteristics and substance use during pregnancy

	All interviewed mothers ($n = 173$)	Mothers of students with suspected FASD ($n = 19$)	Mothers of students considered deferred cases ($n = 5$)	Mothers of typically developing control children ($n = 37$)	Statistical test[a]	p value
Demographics						
Current age (years) – mean (*SD*)	40.9 (5.0)	41.7 (6.0)	42.6 (5.0)	41.4 (4.9)	$t = 0.188$	.851
Range	26–56	32–49	38–51	30–56		
Ethnicity – *n* (%)					$X = 7.933$	.160
Caucasian	85 (49.1)	14 (73.7)	2 (40.0)	29 (78.4)		
Aboriginal	1 (0.6)	0 (0.0)	0 (0.0)	0 (0.0)		
African Canadian / Caribbean	11 (6.4)	1 (5.3)	0 (0.0)	0 (0.0)		
Eastern European	12 (6.9)	0 (0.0)	1 (20.0)	3 (8.1)		
Western European	23 (13.3)	4 (21.1)	1 (20.0)	2 (5.4)		
Chinese / Southeast Asian	16 (9.3)	0 (0.0)	1 (20.0)	2 (5.4)		
South Asian	8 (4.6)	0 (0.0)	0 (0.0)	0 (0.0)		
Other	16 (9.3)	0 (0.0)	0 (0.0)	1 (2.7)		
Marital status when pregnant – *n* (%)					$X = 0.482$	.786
Single	6 (4.5)	1 (5.3)	0 (0.0)	1 (2.7)		
Married, living with husband	137 (79.2)	14 (73.7)	4 (80.0)	30 (81.1)		
Not married, but living with partner	29 (16.8)	4 (21.1)	1 (20.0)	6 (16.2)		
Separated from spouse	1 (0.6)	0 (0.0)	0 (0.0)	0 (0.0)		
Employment status 12 months before pregnancy – *n* (%)					$X = 0.090$	.764
Employed	141 (81.5)	17 (89.5)	5 (100.0)	34 (91.9)		
Unemployed	32 (18.5)	2 (10.5)	0 (0.0)	3 (8.1)		

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	All interviewed mothers (*n* = 173)	Mothers of students with suspected FASD (*n* = 19)	Mothers of students considered deferred cases (*n* = 5)	Mothers of typically developing control children (*n* = 37)	Statistical test[a]	*p* value
Highest level of education completed by pregnancy – *n* (%)					*X* = 15.220	.004
Less than 9 years	3 (1.7)	1 (5.3)	0 (0.0)	0 (0.0)		
Uncompleted high school diploma	3 (1.7)	0 (0.0)	0 (0.0)	0 (0.0)		
High school diploma	22 (12.7)	5 (26.3)	0 (0.0)	0 (0.0)		
College diploma	56 (32.4)	6 (31.6)	1 (20.0)	9 (24.3)		
University degree	77 (44.5)	6 (31.6)	4 (80.0)	21 (56.8)		
Graduate degree	11 (6.4)	1 (5.3)	0 (0.0)	7 (18.9)		
Paternal characteristics						
Employment status 12 months before partner's pregnancy – *n* (%)					*X* = 0.496	.780
Employed	162 (93.6)	17 (89.5)	5 (100.0)	35 (94.6)		
Unemployed	5 (2.9)	1 (5.3)	0 (0.0)	1 (2.7)		
Highest level of education completed at time of partner's pregnancy – *n* (%)						
Less than 9 years	1 (1.2)	0 (0.0)	0 (0.0)	0 (0.0)	*X* = 9.043	.107
Uncompleted high school diploma	10 (5.8)	3 (15.8)	0 (0.0)	2 (5.4)		
High school diploma	40 (23.1)	4 (21.1)	2 (40.0)	2 (5.4)		
College diploma	46 (26.6)	6 (31.6)	2 (40.0)	12 (32.4)		
University degree	58 (33.5)	3 (15.8)	0 (0.0)	16 (43.2)		
Graduate degree	9 (5.2)	1 (5.3)	1 (20.0)	4 (10.8)		
Pregnancy-related characteristics						
Received financial support during pregnancy from relative and/or non-relative	21 (12.1)	3 (15.8)	2 (40.0)	2 (5.4)	*X* = 3.801	.149
Received financial support was at least half of respondent's income	14 (8.1)	3 (15.8)	2 (40.0)	0 (0.0)	*X* = 6.187	.045
Financial supported provided by:						
Child's grandmother or grandfather	7 (4.1)	2 (10.5)	0 (0.0)	0 (0.0)	*X* = 6.587	.086
Child's father	6 (3.5)	1 (5.3)	2 (40.0)	0 (0.0)		
Other relative	3 (1.7)	0 (0.0)	0 (0.0)	1 (2.7)		
Other non-relative	1 (0.6)	0 (0.0)	0 (0.0)	0 (0.0)		
Planned pregnancy with participating child – *n* (%)	126 (72.8)	12 (63.2)	5 (100.0)	31 (83.8)	*X* = 5.242	.155
Number of pregnancies – mean (*SD*)	2.8 (1.3)	3.5 (2.3)	2.8 (1.3)	2.7 (1.2)	*X* = 1.809	.076
Range	1–11	1–11	1–4	1–7		
Number of live births – mean (*SD*)	2.3 (0.9)	2.5 (1.6)	2.0 (0.7)	2.2 (0.6)	*t* = 0.873	.387
Range	1–8	1–8	1–3	1–4		
Any children born prematurely (yes) – *n* (%)	28 (16.2)	3 (15.8)	0 (0.0)	4 (10.8)	*X* = 0.285	.594
Any children with a birth defect (yes) – *n* (%)	20 (11.6)	4 (21.1)	0 (0.0)	2 (5.4)	*X* = 3.213	.073
Point of pregnancy recognition (weeks) – mean (*SD*)	4.6 (2.3)	4.1 (1.2)	4.4 (2.6)	4.9 (1.8)	*t* = 1.717	.092
Range	1–20	1–6	1–8	2–9		

	All interviewed mothers (n = 173)	Mothers of students with suspected FASD (n = 19)	Mothers of students considered deferred cases (n = 5)	Mothers of typically developing control children (n = 37)	Statistical test[a]	p value
Alcohol use						
Lifetime abstainer – n (%)	17 (9.8)	0 (0.0)	0 (0.0)	0 (0.0)		
Age of first drink (years) – mean (*SD*)	17.7 (3.0)	16.6 (2.0)	18 (1.4)	17.0 (2.0)	t = 0.692	.492
Age when began to drink regularly (years) – mean (*SD*)	20.7 (5.3)	18.2 (1.7)	20.3 (2.5)	19.4 (3.3)	t = 1.381	.174
Current drinking problem – n (%)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)		
Ever sought help for a drinking problem – n (%)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)		
Ever felt they should cut down their drinking – n (%)	4 (2.3)	0 (0.0)	0 (0.0)	1 (2.7)	X = 1.065	.587
Alcohol use prior to pregnancy recognition – n (%)					X = 31.605	< .001
High risk	19 (11.0)	12 (63.2)	4 (80.0)	0 (0.0)		
Some risk	110 (63.6)	7 (36.8)	1 (20.0)	25 (67.6)		
No risk (no use)	44 (25.4)	0 (0.0)	0 (0.0)	12 (32.4)		
Beverage preference of mothers who used alcohol prior to pregnancy recognition – n (%)					X = 8.509	.075
Beer	28 (16.2)	5 (26.3)	1 (20.0)	5 (13.5)		
Wine	76 (43.9)	11 (57.9)	4 (80.0)	15 (40.5)		
Wine coolers or champagne	13 (7.5)	2 (10.5)	0 (0.0)	2 (5.4)		
Liquor/cocktails	13 (7.5)	1 (5.3)	0 (0.0)	4 (10.8)		
Alcohol use following pregnancy recognition – n (%)					X = 0.496	.481
High risk	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)		
Some risk	11 (6.4)	2 (10.5)	1 (20.0)	2 (5.4)		
No risk (no use)	162 (93.6)	17 (89.5)	4 (80.0)	35 (94.6)		
Beverage preference of mothers who used alcohol following pregnancy recognition – n (%)					X = 2.469	.291
Beer	3 (1.7)	0 (0.0)	0 (0.0)	2 (5.4)		
Wine	8 (4.6)	2 (10.5)	1 (20.0)	0 (0.0)		
Wine coolers or champagne	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)		
Liquor/cocktails	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)		
Tobacco use						
Ever smoked in their lifetime – n (%)	81 (47.1)	14 (73.7)	3 (60.0)	17 (46.0)	X = 6.525	.038
Current smoker – n (%)						
Daily	21 (12.1)	4 (21.1)	0 (0.0)	2 (5.4)	X = 0.278	.598
Occasionally	6 (3.5)	2 (10.5)	1 (20.0)	2 (5.4)		
Does not smoke	146 (84.4)	13 (68.4)	4 (80.0)	33 (89.2)		

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	All interviewed mothers (*n* = 173)	Mothers of students with suspected FASD (*n* = 19)	Mothers of students considered deferred cases (*n* = 5)	Mothers of typically developing control children (*n* = 37)	Statistical test[a]	*p* value
Tobacco use prior to pregnancy recognition – *n* (%)					$X = 17.233$	< .001
Daily	42 (24.3)	11 (57.9)	2 (40.0)	3 (8.1)		
Occasionally	17 (9.8)	2 (10.5)	1 (20.0)	4 (10.8)		
Did not smoke	114 (65.9)	6 (31.6)	2 (40.0)	30 (81.1)		
Number of cigarettes smoked per day prior to pregnancy recognition (daily smokers) – mean (*SD*)	6.9 (4.5)	8.1 (6.4)	4.5 (0.7)	4.4 (3.2)	$t = 1.207$	.248
Range	1–25	1–25	4–5	1–8		
Tobacco use following pregnancy recognition – *n* (%)					$X = 0.496$	.481
Daily	8 (4.6)	2 (10.5)	0 (0.0)	2 (5.4)		
Occasionally	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)		
Did not smoke	165 (95.4)	17 (89.5)	5 (100.0)	35 (94.6)		
Number of cigarettes smoked per day following pregnancy recognition (daily smokers) – mean (*SD*)	4.5 (3.6)	2.5 (0.7)	0 (0.0)	3.0 (1.4)	$t = 0.447$	.699
Range	1–12	2–3		2–4		
Drug use						
Drug use during pregnancy (prior to pregnancy recognition) – *n* (%)						
Anabolic steroids	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)		
Club drugs (ecstasy, GHB, rohypnol)	7 (4.1)	1 (5.3)	0 (0.0)	2 (5.4)	$X = 0.001$	.982
Crack/cocaine	1 (0.6)	1 (5.3)	0 (0.0)	0 (0.0)	$X = 1.983$	.159
Dissociative drugs (PCP, ketamine, salvia, DXM)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)		
Hallucinogens (LSD, mushrooms, peyote)	11 (6.4)	3 (15.8)	0 (0.0)	3 (8.1)	$X = 0.774$	.379
Heroin or opium	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)		
Marijuana or hashish	48 (28.6)	13 (68.4)	2 (40.0)	10 (27.0)	$X = 8.887$	.003
Methamphetamines/amphetamines	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)		
Non-medical inhalants (gasoline, paint thinners, glue, nitrous oxide, whippets, poppers)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)		
Prescription drugs (valium, Xanax, codeine, morphine, Vicodin, Lortab, Percocet)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)		
Other drugs or substances	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)		

	All interviewed mothers (*n* = 173)	Mothers of students with suspected FASD (*n* = 19)	Mothers of students considered deferred cases (*n* = 5)	Mothers of typically developing control children (*n* = 37)	Statistical test[a]	*p* value
Drug use during pregnancy (following pregnancy recognition) – *n* (%)						
Anabolic steroids	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)		
Club drugs (ecstasy, GHB, rohypnol)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)		
Crack/cocaine	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)		
Dissociative drugs (PCP, ketamine, salvia, DXM)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)		
Hallucinogens (LSD, mushrooms, peyote)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)		
Heroin or opium	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)		
Marijuana or hashish	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)		
Methamphetamines/amphetamines	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)		
Non-medical inhalants (gasoline, paint thinners, glue, nitrous oxide, whippets, poppers)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)		
Prescription drugs (valium, Xanax, codeine, morphine, Vicodin, Lortab, Percocet)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)		
Other drugs or substances	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)		

[a]Comparing students with suspected FASD with typically developing control children.

PREVALENCE OF FASD

Data from 323 potential "cases" (along with that from 84 typically developing control children) were independently reviewed by a panel of experts. Subsequently, 69 cases, identified by the experts, were discussed on a case-by-case basis during multidisciplinary case conferences. Final screening results revealed that 21 students met the criteria outlined in the 2005 Canadian guidelines for FASD diagnosis (Chudley et al., 2005): 3 students had suspected FAS, 2 students had suspected pFAS and 16 students had suspected ARND. Growth impairments were present in 10 students with suspected FASD; 7 students with suspected FASD had one facial feature characteristic of FAS; 2 students had two characteristic facial features; and 3 students had all three facial features characteristic of FAS. Central nervous system impairments in at least three domains were recognized in all suspected FASD cases; and 7 students had prenatal alcohol exposure within the level of some risk; 12 students had prenatal alcohol exposure within the level of high risk; and 2 students had unconfirmed levels of prenatal alcohol exposure (i.e., 2 suspected FAS).

In addition to the 21 students with suspected FASD, 5 students were considered to be deferred cases (i.e., prenatal alcohol exposure was identified, but fewer than three central nervous system domains were considered impaired). The final FASD screening results are summarized in Table 5.

TABLE 5

Final FASD screening results

Student	Growth impairments present	Number of characteristic facial anomalies	Impairment in 3+ central nervous system domains	Prenatal alcohol exposure risk level	Screening conclusion
Suspected cases					
1	No	0	Yes	Some	Suspected ARND
2	No	1	Yes	High	Suspected ARND
3	No	0	Yes	High	Suspected ARND
4	No	0	Yes	High	Suspected ARND
5	Yes	0	Yes	High	Suspected ARND
6	Yes	1	Yes	Some	Suspected ARND
7	No	0	Yes	High	Suspected ARND
8	Yes	1	Yes	High	Suspected ARND
9	Yes	1	Yes	High	Suspected ARND
10	No	0	Yes	Some	Suspected ARND
11	No	0	Yes	Some	Suspected ARND
12	Yes	1	Yes	High	Suspected ARND
13	Yes	1	Yes	Some	Suspected ARND
14	No	1	Yes	Some	Suspected ARND
15	No	0	Yes	High	Suspected ARND
16	No	0	Yes	High	Suspected ARND
17	Yes	3	Yes	Unconfirmed	Suspected FAS
18	Yes	3	Yes	Unconfirmed	Suspected FAS
19	Yes	3	Yes	High	Suspected FAS
20	Yes	2	Yes	High	Suspected pFAS
21	No	2	Yes	Some	Suspected pFAS
Deferred cases					
1	No	0	2	Some	Deferred
2	No	0	1	High	Deferred
3	No	1	2	High	Deferred
4	No	2	2	High	Deferred
5	No	0	2	High	Deferred

ARND = alcohol-related neurodevelopmental disorder; FAS = fetal alcohol syndrome; pFAS = partial fetal alcohol syndrome.

The FASD prevalence estimates obtained in all three scenarios took into consideration the selection rate, which accounted for the students and their mothers who dropped out or were lost to follow-up during each respective phase of data collection.

Scenario 1 (Main Analysis)

As per the main analysis, which assumed that there was no difference in the risk of FASD between students whose parents/guardians provided consent and students whose parents/guardians did not provide consent (meaning that the level of maternal alcohol consumption during pregnancy was the same in both groups of participants), the prevalence of suspected FAS was estimated to be 1.2 per 1,000 (95% CI: 0.0–2.8 per 1,000), pFAS 2.0 per 1,000 (95% CI: 0.0–5.1 per 1,000) and ARND 15.0 per 1,000 (95% CI: 8.1–22.7 per 1,000). The overall FASD prevalence was estimated to be 18.1 per 1,000 (95% CI: 10.8–26.3 per 1,000) or 1.8% (Table 6).

TABLE 6

Prevalence of FASD among elementary school students in Greater Toronto Area, Ontario, Canada

FASD diagnostic categories	Number of suspected cases	Main analysis			Sensitivity analysis					
		Scenario 1			Scenario 2 (lower estimate)			Scenario 3 (upper estimate)		
		Prevalence	95% CI		Prevalence	95% CI		Prevalence	95% CI	
			LE	UE		LE	UE		LE	UE
Suspected FAS	3	1.2	0.0	2.8	1.2	0.0	2.7	1.2	0.0	2.8
Suspected pFAS	2	2.0	0.0	5.1	0.8	0.0	2.1	2.0	0.0	5.1
Suspected ARND	16	15.0	8.1	22.7	5.9	3.3	9.3	26.1	9.6	52.8
Suspected FASD	21	18.1	10.8	26.3	7.8	4.8	11.7	29.3	12.4	56.2

ARND = alcohol-related neurodevelopmental disorder; CI = confidence interval; FAS = fetal alcohol syndrome; FASD = fetal alcohol spectrum disorder; LE = lower estimate; pFAS = partial fetal alcohol syndrome; UE = upper estimate.

Sensitivity Analyses

Scenario 2 (Most Conservative Approach, Lower Estimate)

Following the most conservative approach to estimating prevalence, which assumed that all students who were not selected to proceed to each subsequent phase and that all students whose parents/guardians did not provide consent had no risk of having FASD (assuming that their mothers did not consume alcohol during pregnancy), the prevalence of suspected FAS was estimated to be 1.2 per 1,000 (95% CI: 0.0–2.7 per 1,000), pFAS 0.8 per 1,000 (95% CI: 0.0–2.1 per 1,000) and ARND 5.9 per 1,000 (95% CI: 3.3–9.3 per 1,000). The overall FASD prevalence was estimated to be 7.8 per 1,000 (95% CI: 4.8–11.7 per 1,000) or 0.8%.

Scenario 3 (Least Conservative Approach, Upper Estimate)

Following the least conservative approach to estimating prevalence, which assumed that there was no difference in FASD risk between students whose parents/guardians provided consent and those whose parents/guardians did not and that there was a possibility of FAS and other FASD diagnoses among non-selected individuals (i.e., typically developing control children), the prevalence of suspected FAS was estimated to be 1.2 per 1,000 (95% CI: 0.0–2.8 per 1,000), of pFAS 2.0 per 1,000 (95% CI: 0.0–5.1 per 1,000) and of ARND 26.1 per 1,000 (95% CI: 9.6–52.8 per 1,000). The overall FASD prevalence was estimated to be 29.3 per 1,000 (95% CI: 12.4–56.2 per 1,000) or 2.9%.

Given that the main assumption of the least conservative approach (scenario 2) is unrealistic, the population-based prevalence of FASD is likely to range between approximately 2% and 3% among elementary school children, aged 7 to 9 years, in the GTA in Ontario, Canada.

COMPARISON OF FASD DIAGNOSTIC GROUPS WITH TYPICALLY DEVELOPING CONTROL CHILDREN

There were no statistically significant differences in demographic characteristics between FASD diagnostic groups, students considered deferred cases and typically developing control children. However, students with suspected FAS/pFAS had a smaller mean OFC ($p < .05$) compared with both students with suspected ARND and typically developing control children, and also had shorter palpebral fissures ($p < .05$ for right PFL and $p < .01$ for left PFL) compared with typically developing control children. Students with suspected ARND were more likely to have lower composite scores on the WASI-II and WISC-IV compared with typically developing control children: IQ ($p < .05$), verbal comprehension ($p < .01$), perceptual reasoning ($p = .001$), working memory ($p < .001$) and processing speed ($p < .05$). In addition, students with suspected FAS/pFAS were more likely to have lower composite scores for IQ ($p < .05$), verbal comprehension ($p < .01$) and processing speed ($p < .05$) compared with typically developing control children. Table 7 presents a comparison of demographic characteristics, growth measurements, dysmorphology and neurodevelopmental assessment summary results across FASD diagnostic groups, deferred cases and typically developing control children.

5. Discussion

This study provides the first population-based estimate of the prevalence of FASD among elementary school students (aged 7 to 9 years). The prevalence is likely to range between approximately 2% and 3%. This estimate is roughly double or possibly even triple previous crude estimates: 10 per 1,000 or 1% (adopted for Canada from the Unites States; Roberts & Nanson, 2001) and 7.9 per 1,000 or about 0.8%, based on statistical modelling using country-specific indicators (Lange et al., 2017).

These estimates may reflect the overall prevalence of FASD in those under 10 years of age in Canada; however, confirming this prevalence rate would require the representation of all other provinces and territories of Canada.

Regardless, these current findings are in line with recent estimates in the United States, where the prevalence of FASD among the general population was estimated to be between 2% and 5% (May et al., 2014). However, current estimates are lower than recent estimates for some European countries and South Africa, due to higher rates of alcohol consumption overall, as well as among pregnant women. For example, the prevalence estimate for Croatia is 4%–7% (Petković& Barišić, 2010, 2013); for Italy 4%–5% (May et al., 2006, 2011); and for South Africa 6%–21% (May et al., 2013; Urban et al., 2015).

TABLE 7

Comparison of demographic characteristics, growth and dysmorphology measurements and neurodevelopmental results across FASD diagnostic groups, deferred cases and typically developing control children

	Students with suspected FAS/ pFAS (*n* = 5)	Students with suspected ARND (*n* = 16)	Students considered deferred cases (*n* = 5)	Typically developing control children (*n* = 83)	Statistical test	*p* value
Demographics						
Sex (% male)	3 (60.0)	8 (50.0)	1 (20.0)	49 (59.0)	$X = 3.206$	.361
Age (years) – mean (*SD*)	8.6 (0.9)	9.0 (0.8)	9.1 (1.1)	8.5 (0.8)	$F = 2.03$	.114
Range	7.6–9.9	7.8–10.4	7.5–10.2	6.5–10.5		
Ethnicity – *n* (%)					$X = 12.272$	.833
Caucasian	5 (100.0)	10 (62.5)	3 (60.0)	38 (45.8)		
African Canadian/Caribbean	0 (0.0)	1 (6.3)	0 (0.0)	3 (3.6)		
Eastern European	0 (0.0)	2 (12.5)	0 (0.0)	7 (8.4)		
Western European	0 (0.0)	3 (18.8)	1 (20.0)	16 (19.3)		
Chinese/Southeast Asian	0 (0.0)	0 (0.0)	0 (0.0)	3 (3.6)		
South Asian	0 (0.0)	0 (0.0)	0 (0.0)	8 (9.6)		
Other	0 (0.0)	0 (0.0)	1 (20.0)	8 (9.6)		
Growth measurements						
Height (cm) – mean (*SD*)	128.6 (8.4)	134.1 (7.2)	133.8 (7.8)	133.9 (7.2)	$F = 0.85$	.470
Height ≤ 10th percentile – *n* (%)	2 (40.0)	3 (18.8)	0 (0.0)	2 (2.4)	$X = 15.994$	.001
Weight (kg) – mean (*SD*)	27.0 (2.6)	32.6 (10.5)	31.1 (6.8)	32.0 (8.5)	$F = 0.60$	.614
Weight ≤ 10th percentile – *n* (%)	2 (40.0)	2 (12.5)	0 (0.0)	7 (8.4)	$X = 5.844$	.119
OFC (cm) – mean (*SD*)	51.5 (1.5)	52.8 (2.3)	54.6 (1.9)	53.6 (1.4)	$F = 4.73$	< .05[b,c]
OFC ≤ 10th percentile – *n* (%)	2 (40.0)	3 (18.8)	0 (0.0)	0 (0.0)	$X = 25.890$	< .001
Dysmorphology						
Right PFL (cm) – mean (*SD*)	2.32 (0.13)	2.43 (0.16)	2.48 (0.23)	2.51 (0.14)	$F = 4.19$	< .05[b]
Right PFL 2SD below mean – *n* (%)	3 (60.0)	7 (43.8)	2 (40.0)	9 (10.8)	$X = 16.668$	.001
Left PFL (cm) – mean (*SD*)	2.28 (0.12)	2.46 (0.15)	2.48 (0.16)	2.50 (0.13)	$F = 4.54$	< .01[b]
Left PFL 2SD below mean – *n* (%)	4 (80.0)	5 (31.3)	2 (40.0)	11 (13.3)	$X = 16.015$	.001
Inner canthal distance (cm) – mean (*SD*)	2.88 (0.31)	2.83 (0.32)	2.80 (0.19)	2.83 (0.22)	$F = 0.10$	.959
Inner canthal distance ≤ 25th percentile – *n* (%)	2 (40.0)	8 (50.0)	1 (20.0)	38 (46.3)	$X = 16.942$	.152
Philtrum length (cm) – mean (*SD*)	1.34 (0.17)	1.13 (0.15)	1.24 (0.24)	1.26 (0.42)	$F = 0.69$	.562
Philtrum score on the lip-philtrum guide – *n* (%)					$X = 27.897$	.001
1	0 (0.0)	0 (0.0)	1 (20.0)	1 (1.2)		
2	1 (20.0)	4 (25.0)	0 (0.0)	30 (36.1)		
3	0 (0.0)	11 (68.8)	3 (60.0)	38 (45.8)		
4	4 (80.0)	1 (6.3)	1 (20.0)	14 (16.9)		
5	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)		

	Students with suspected FAS/ pFAS (*n* = 5)	Students with suspected ARND (*n* = 16)	Students considered deferred cases (*n* = 5)	Typically developing control children (*n* = 83)	Statistical test	*p* value
Vermillion border score on the lip-philtrum guide – *n* (%)					*X* = 26.114	.002
1	0 (0.0)	1 (6.3)	0 (0.0)	2 (2.4)		
2	1 (20.0)	8 (50.0)	3 (60.0)	35 (42.7)		
3	0 (0.0)	7 (43.8)	2 (40.0)	36 (43.9)		
4	4 (80.0)	0 (0.0)	0 (0.0)	9 (11.0)		
5	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)		
Neurodevelopmental data[a]						
WASI-II: Verbal Comprehension Index	82.0 (6.4)	86.6 (13.2)	96.8 (8.5)	103.5 (13.5)	F = 10.72	< .01[b,c]
WASI-II: Perceptual Reasoning Index	96.2 (10.0)	90.5 (14.5)	94.4 (5.3)	103.9 (12.6)	F = 5.81	.001[d]
WASI-II: Full-Scale IQ-4	87.4 (6.3)	87.2 (11.3)	95.0 (7.1)	104.2 (11.9)	F = 12.44	< .05[a,d]
WISC-IV: Working Memory Index	93.0 (8.8)	86.4 (12.2)	95.6 (8.1)	105.2 (14.2)	F = 9.59	< .001[d]
WISC-IV: Processing Speed Index	82.8 (14.0)	79.1 (15.9)	86.6 (12.4)	100.7 (13.7)	F = 12.92	< .05[a,d]

Note: Other comparison included deferred vs. typically developing control children, deferred vs. FAS/pFAS and deferred vs. ARND. There were no significant differences found for these comparisons. FASD = fetal alcohol spectrum disorder; PFL = palpebral fissure length; *SD* = standard deviation.

[a]Neurodevelopmental data were available for 80 typically developing control children. [b]FAS/pFAS vs. typically developing control children. [c]FAS/pFAS vs. ARND. [d]ARND vs. typically developing control children.

Interestingly, in the current study, students with suspected FASD did not differ from typically developing control children in terms of demographic characteristics, specifically sex, age and ethnicity; however, as was expected, they were shorter, had smaller occipitofrontal circumferences and had certain dysmorphological features that discriminate children with and without FAS/pFAS. Again, not unexpectedly, students with suspected FASD had lower composite scores for IQ, verbal comprehension, perceptual reasoning, working memory and processing speed compared with typically developing control children. Furthermore, students with suspected FASD had significantly higher scores on the Social Problems, Thought Problems, Attention Problems and Rule-Breaking Behavior Syndrome scales, and on the Attention Deficit/Hyperactivity Problems and Conduct Problems DSM-Oriented scales of the CBCL. This finding is consistent with Mattson and Riley (2000), who found that externalizing behavioural problems are elevated in children with FASD compared with IQ-matched and non-alcohol-exposed peers.

Even though students with suspected FASD were significantly different from typically developing control children on the majority of neurodevelopmental indicators, as a group, their mean scores did not demonstrate profound deficits. Unfortunately, this finding is misleadingly optimistic. When looking at the individual performances of students with suspected FASD, 28.6%, 9.6% and 19.0% of them had scores 1.5 standard deviations below the mean or lower on the WASI-II verbal comprehension, perceptual reasoning and IQ indices, and 14.3% and 42.9% had scores 1.5 standard deviations below the mean or lower on the WISC-IV working memory and processing speed indices. Although 2 standard deviations below the mean is used as the cut-point in the 2005 Canadian guidelines, 1.5 standard deviations below the mean is considered to be clinically significant, as it places the individual in the borderline range of intellect and is highly significant with respect to daily functioning. By comparison, the vast majority of typically developing control children had scores at least 1 standard deviation above the mean on these composite scales.

These data clearly indicate a need for timely interventions and support for children with FASD. The findings also show that the effects of alcohol are broad and that alcohol affects people differently due to various factors (including variability in the metabolism and genetic background of both the mother and fetus, environmental influences, maternal smoking behaviour, nutritional status, stress levels [Eberhart & Parnell, 2016; May & Gossage, 2011], and possibly paternal lifestyle [Day et al., 2016]).

In the current study, mothers of students with suspected FASD were more likely to have achieved a lower level of education at the time of their pregnancy compared with mothers of typically developing control children. They were also more likely than mothers of typically developing control children to have smoked tobacco and used marijuana or hashish prior to pregnancy recognition. This finding reflects an increased likelihood of partaking in risky behaviour. Importantly, all mothers of students with suspected FASD reported alcohol consumption *before* pregnancy recognition, but only 10.5% reported consuming alcohol *after* pregnancy recognition. Based on this information, which was obtained via maternal self-reports, it can be concluded that the negative effects of alcohol consumption on the developing fetus occurred even before the mothers knew they were pregnant. Exemplary work by Sulik (2005) has shown that the characteristic facial dysmorphology of FAS and pFAS can be produced in a mouse model following exposure to high dosages of alcohol on the 7th to 9th day of gestation, which corresponds to the 3rd and 4th week of pregnancy in humans—that is, when most women are unaware that they are pregnant. Another study of mice found that specific facial phenotypes were predictive of unique patterns of brain abnormalities with respect to brain volume and shape (Lipinski et al., 2012). Thus, it can be concluded that consuming alcohol during this critical period, particularly large amounts, can cause significant harm to the developing fetal brain. However, the risks to a developing fetus are not limited to heavy alcohol use. A recent systematic review and meta-analysis reported that although evidence of the effects of light prenatal alcohol consumption, defined as drinking ≤ 32 g (about two standard drinks) of alcohol per week during pregnancy, is limited, there was some evidence that it is associated with smaller gestational age and preterm delivery (Mamluk et al., 2017).

Nonetheless, the current study indicates that FASD is a relatively prevalent alcohol-related developmental disability. However, it is a largely preventable condition. Moreover, given that the current study estimated the prevalence of FASD among a diverse sample of elementary school students in the GTA, the findings emphasize that FASD is not restricted to disadvantaged groups, but rather, that it occurs throughout society, regardless of socioeconomic status, age or ethnicity.

This study used the most reliable approach to estimating FASD prevalence—active case ascertainment. It has primary advantages over other approaches, namely, representativeness of data by studying an entire community/population, a high chance of accurate diagnosis of FASD by clinical specialists, and elimination of self-selection biases (May & Gossage, 2001). Given these advantages, active case ascertainment is known to produce the most accurate FASD prevalence estimates (May & Gossage, 2001).

These advantages also indicate that active case ascertainment is an appropriate methodology for use in other jurisdictions and settings, such as child protection services and the criminal justice system, and with specific populations, such as Aboriginal people and people with psychiatric disorders, where the prevalence of FASD is expected to be higher (Lange et al., 2017; Popova, Lange, Probst & Rehm, 2017).

However, it should be acknowledged that the prevalence found in the current study is likely still underestimated for a number of reasons. First, the participation rate was lower than desired. Although two rounds of consent forms were distributed, it was not possible to ensure that all parents/guardians received the forms because they were given to students to take home rather than being mailed directly. As a result, it is not known whether parents/guardians who did not respond actually received the form or whether they were "soft" refusals, meaning they received the form but did not wish to participate in the study.

Second, because this study was highly sensitive in nature, not all parents/guardians would be willing to allow their child to participate due to fear of stigmatization and the possibility of causing conflict within the family.

Third, the information letter and consent form were provided to parents/guardians in English only. Since the GTA is multicultural, it is possible that non-English-speaking parents/guardians could not understand the consent form and therefore were unable to provide consent.

Despite being lower than desired, the participation rate among parents/guardians who responded (69.9%) was higher than in similar studies that used active consent protocols in schools. For example, only 50% of parents/guardians gave consent in an FASD prevalence study in Italy (May et al., 2006) and only 25% gave consent in a study conducted in the state of Washington in the United States (Clarren et al., 2001).

There is a fourth reason the prevalence rate of FASD found in the current study is likely underestimated. Identifying students with suspected behavioural and/or learning difficulties involved teacher and/or parent/guardian referrals. However, in some cases, the teachers were not available to provide referrals, and it is also possible that some parents/guardians were not willing to identify behavioural and/or learning difficulties in their children due to social desirability bias. As a result, some cases of pFAS and ARND might have been missed.

Fifth, the participation rate of the biological mothers was low and using self-reports limited the accuracy of the information these mothers provided. Despite all efforts to recruit biological mothers (i.e., attempting at least three times via email and telephone to make contact and schedule an interview, offering a gift card), the participation rate was only 40.9%. Moreover, among mothers of students with suspected FASD, only 10.5% reported alcohol consumption following pregnancy recognition. However, because prenatal alcohol exposure is a highly sensitive issue, using self-reports to obtain information on this topic limits accuracy due to social desirability and recall bias (Lange et al., 2014). Lange et al. (2014) found that the prevalence of prenatal alcohol exposure was four times higher when measured by meconium testing compared with maternal self-reports. Thus, it is reasonable to suspect that alcohol use during pregnancy was underreported in the current study and, as a result, that some cases of pFAS and ARND were missed. The same problems were reported by other studies conducted in the United States and Europe (see, for example, May et al., 2014; Ortega-García et al., 2012).

Sixth, the deferred cases and cases of suspected FASD were identified among only those students with available maternal reports. However, some students met the diagnostic criteria for pFAS/ARND (impairments in three or more brain domains), but maternal reports were not available for these students, which meant a diagnosis could not be inferred.

Seventh, when a maternal interview was not possible, alternative sources of information about maternal alcohol use were not sought; this was a stipulation of the Research Ethics Boards. However, it is common practice in clinical settings and is recommended in recent FASD diagnostic guidelines to seek reliable collateral sources (e.g., family member, social service agency, medical record, adoption records) and/or documentation of positive testing with established alcohol-exposure biomarkers during pregnancy or at birth (Hoyme et al., 2016).

Finally, it was brought to the team's attention that two students had pre-diagnosed FAS, as indicated by their school records. However, since the parents/guardians of these students did not provide consent, they were not included in the study. It is possible that there were additional pre-existing cases among non-consented students that were not divulged to the study team. This possibility is strengthened by the finding that there was one student with suspected ARND and another student considered a deferred case among the randomly selected typically developing control children.

It is important to note that this was a screening study, meaning that students who were most in need of a full diagnostic assessment were identified and it is not necessarily the case that diagnoses of all suspected and deferred cases will be confirmed after a full multidisciplinary clinical assessment. In addition, although the current study followed the 2005 Canadian guidelines for FASD diagnosis (Chudley et al., 2005), modifications were made to enhance the feasibility of using the guidelines in a large-scale study. First, students were identified as having postnatal growth deficits if *both* their height and weight were at or below the 10th percentile, whereas the Canadian guidelines indicate that deficits in *either* height or weight are sufficient evidence of postnatal growth impairment. Second, although the guidelines consider birth parameters (weight and length at or below the 10th percentile) as evidence of prenatal growth impairment, such information was not available for the current study.

In general, estimating the prevalence of FASD in Canada, or elsewhere, is difficult. Reasons include the challenges of diagnosing FASD, the fact that FAS is the only FASD-specific diagnosis included in the *International Classification of Diseases and Related Health Problems* (World Health Organization, 2004), inadequate or unavailable diagnostic capacity across the country (Clarren et al., 2011) and lack of a universal diagnostic approach and definitions (Coles et al., 2016). The challenges of diagnosing FASD are exacerbated by a lack of uniformly accepted diagnostic criteria (Chudley et al., 2017). It was recently reported that existing FASD diagnostic guidelines lack convergent validity and are limited in their concordance with respect to the specific diagnostic categories (Coles et al., 2016). Although the current criteria overlap considerably with one another, clinicians have limited guidance in selecting the optimal criteria because there is a general lack of studies comparing the specificity, sensitivity and accuracy of the various criteria (Burd et al., 2010). Ultimately, the absence of a standardized set of criteria can lead to diagnostic misclassification (Astley & Clarren, 2000), which itself has a number of consequences. Specifically, it can result in inaccurate prevalence estimates, inappropriate treatments and interventions for patients, and, from a clinical research point of view, the inability to detect a clinically meaningful difference between two groups (Astley & Clarren, 2000). Alarmingly, children and adolescents who have been affected by prenatal alcohol exposure are often undiagnosed or misdiagnosed, even in clinical settings where FASD is an important area of emphasis (Chasnoff et al., 2015).

However, identifying a child who was exposed to alcohol in utero is crucial because it can prompt close monitoring of the child's development, facilitate early diagnosis and, if necessary, lead to the implementation of timely interventions, which are key to improving the quality of life of individuals with FASD, potentially preventing other common adverse outcomes, including school failure and dropout, addiction, mental health problems, sexually deviant behaviour, dependent living, involvement with the law and incarceration (Streissguth et al., 1996). Thus, early identification and diagnosis have the ability to alter the developmental trajectory of the affected individual. Early diagnosis is also important for parents/guardians because it helps to explain the behavioural problems often exhibited by children with FASD and can improve their way of parenting by increasing their understanding. Furthermore, early diagnosis could prevent subsequent alcohol-exposed births by providing appropriate interventions, treatment, counselling and support to the birth mother (Astley et al., 2000).

Although FASD is considered a leading cause of non-genetic developmental disabilities, another major concern is that while most children with FASD (75%–80%) have a low-average IQ or higher (Mattson et al., 2011), they struggle with everyday living skills (Streissguth et al., 1996). This makes accessing developmental disability services extremely difficult. In terms of schooling, children have different needs, strengths and weaknesses, but some children, such as those with FASD, need learning activities that are carefully structured and reinforced, along with environmental modifications (Green, 2007).

Academic achievement is lower among students with FASD compared with other students (Chudley et al., 2005). The difficulties that children with FASD experience include grasping mathematical concepts, such as gauging time and handling money; thinking things through and learning from experience; and understanding the consequences of their actions and "cause and effect" relationships. They also have difficulty with social skills, storing and retrieving information, following instructions and impulsivity and distractibility (Millar et al., 2017). Functional skills (i.e., adaptive behaviour) are often severely compromised in relation to both chronological age and intellectual ability (American Psychiatric Association, 2013). Unfortunately, teachers commonly report lacking knowledge of FASD and how to plan for affected children (Koren et al., 2010).

Students with FASD often display a number of inappropriate or "challenging" behaviours. Compared with students who do not have FASD, students with FASD have poor non-verbal reasoning and academic achievement, as well as teacher-rated problem behaviours (Aragón et al., 2008). A significant problem is often a marked discrepancy between seemingly high verbal skills and an inability to communicate effectively (Mattson et al., 2011). The combination of poor self-control and inadequate communication skills creates social problems that can leave teachers, parents and students feeling frustrated and helpless.

Given the high prevalence of FASD among students aged 7 to 9 years and their complex and unique learning characteristics, there is a clear need to increase the capacity of education systems to recognize, document and develop FASD-specific education strategies (Millar et al., 2017). Most education systems do not recognize FASD as a distinct handicapping condition or as a separate funding category. Students with FASD typically are categorized as having mild, moderate or severe developmental delay, or as suffering from an emotional or behavioural disability. However, generic categories do little to define students' individual needs or to identify appropriate interventions (Millar et al., 2017).

Implementing this project was challenging. First, it was difficult and time-consuming to obtain approval from the Research Ethics Boards due to the sensitive nature of the study in general, the risk of stigmatizing positively screened students and their families, and the request to take facial photographs of study participants. However, all possible measures were taken to protect participants (see "Ethical considerations" section). Obtaining permission from school boards in the GTA was another challenge. It was an extremely lengthy process and only five out of 10 school boards agreed to participate. A large portion of principals (43.7% of those contacted) refused to allow their school to participate. Moreover, as stipulated by the school boards, teachers could only be passively involved in the consent process (i.e., they handed out the second round of consent forms to students who had not returned the form after the first week and collected the returned consent forms), which meant that the consent forms had to be given to students to bring home to their parents/guardians. This process limited the ability to determine whether parents/guardians actually received the consent form, and might have contributed to the lower than expected response rate.

Despite the challenges, this study provides the first population-based estimate of FASD among elementary school students in Canada. Determining prevalence is vital for informing policy-makers and politicians about the impact of FASD. Accurate estimates are necessary to generate policy and funding support for the numerous services required by those affected by FASD, thereby alleviating some of the economic burden associated with FASD in Canada. The data obtained in this study will enable effective monitoring of the prevalence of FASD and raise awareness of FASD and alcohol use during pregnancy.

The results of the current study clearly show that FASD must be considered a serious public health problem in Canada. They support the need to improve prevention initiatives around alcohol use among not only pregnant women, but among all women of childbearing age, as well as the need to provide support to affected individuals and their families.

References

Albertsen, K., Andersen, A.N., Olsen, J. & Gronbaek, M. (2004). Alcohol consumption during pregnancy and the risk of preterm delivery. *American Journal of Epidemiology, 159*, 155–161.

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Arlington, VA: Author.

Aragón, A.S., Coriale, G., Fiorentino, D., Kalberg, W.O., Buckley, D., Gossage, J.P., . . . May, P.A. (2008). Neuropsychological characteristics of Italian children with fetal alcohol spectrum disorders. *Alcoholism, Clinical and Experimental Research, 32*, 1909–1919.

Asante, K.O. & Nelms-Maztke, J. (1985). *Report on the survey of children with chronic handicaps and fetal alcohol syndrome in the Yukon and Northwest British Columbia*. Whitehorse, YT: Council for Yukon Indians.

Astley, S.J. (2012). FAS facial photographic analysis software. Version 2.0.0. Seattle, WA: FAS Diagnostic and Prevention Network, University of Washington.

Astley, S.J., Bailey, D., Talbot, C. & Clarren, S.K. (2000). Fetal alcohol syndrome (FAS) primary prevention through FAS diagnosis: II. A comprehensive profile of 80 birth mothers of children with FAS. *Alcohol and Alcoholism, 35*, 509–519.

Astley, S.J. & Clarren, S.K. (2000). Diagnosing the full spectrum of fetal alcohol exposed individuals: Introducing the 4-digit diagnostic code. *Alcohol and Alcoholism, 35*, 400–410.

Burd, L., Cox, C., Poitra, B., Wentz, T., Ebertowski, M., Martsolf, J.T., . . . Klug, M.G. (1999). The FAS Screen: A rapid screening tool for fetal alcohol syndrome. *Addiction Biology, 4*, 329–336.

Burd, L., Klug, M.G., Li, Q., Kerbeshian, J. & Martsolf, J.T. (2010). Diagnosis of fetal alcohol spectrum disorders: A validity study of the fetal alcohol syndrome checklist. *Alcohol, 44*, 605–614.

Burge, P. (2007). Prevalence of mental disorders and associated service variables among Ontario children who are permanent wards. *Canadian Journal of Psychiatry, 52*, 305–314.

Centers for Disease Control and Prevention. (n.d.). Clinical growth charts. Atlanta, GA: Author. Retrieved from https://www.cdc.gov/growthcharts/clinical_charts.htm

Chasnoff, I.J., Wells, A.M. & King, L. (2015). Misdiagnosis and missed diagnoses in foster and adopted children with prenatal alcohol exposure. *Pediatrics, 135*, 264–270.

Chudley, A.E., Conry, J., Cook, J.L., Loock, C., Rosales, T., LeBlanc, N. & National Advisory Committee on Fetal Alcohol Spectrum Disorder. (2005). Fetal alcohol spectrum disorder: Canadian guidelines for diagnosis. *Canadian Medical Association Journal, 172*(Suppl. 5), S1–S21.

Chudley, A. (2017). Fetal alcohol spectrum disorder diagnosis: Current practices and future considerations. *Biochemistry and Cell Biology.* [Epub ahead of print]

Clarren, S.K., Astley, S.J., Gunderson, V.M. & Spellman, D. (1992). Cognitive and behavioral deficits in nonhuman primates associated with very early embryonic binge exposures to ethanol. *Journal of Pediatrics, 121*, 789–796.

Clarren, S.K., Chudley, A.E., Wong, L., Friesen, J. & Brant, R. (2010). Normal distribution of palpebral fissure lengths in Canadian school age children. *Canadian Journal of Clinical Pharmacology, 17*, e67–e78.

Clarren, S.K., Lutke, J. & Sherbuck, M. (2011). The Canadian guidelines and the interdisciplinary clinical capacity of Canada to diagnose fetal alcohol spectrum disorder. *Journal of Population Therapeutics and Clinical Pharmacology, 18*, e494–e499.

Clarren, S.K., Randels, S.P., Sanderson, M. & Fineman, R.M. (2001). Screening for fetal alcohol syndrome in primary schools: A feasibility study. *Teratology, 63*, 3–10.

Coles, C.D., Gailey, A.R., Mulle, J.G., Kable, J.A., Lynch, M.E. & Jones, K.L. (2016). A comparison among 5 methods for the clinical diagnosis of fetal alcohol spectrum disorders. *Alcoholism, Clinical and Experimental Research, 40*, 1000–1009.

Cook, J.L., Green, C.R., Lilley, C.M., Anderson, S.M., Baldwin, M.E., Chudley, A.E., . . . Rosales, T. (2016). Fetal alcohol spectrum disorder: A guideline for diagnosis across the lifespan. *Canadian Medical Association Journal, 188*, 191–197.

Day, J., Savani, S., Krempley, B.D., Nguyen, M. & Kitlinska, J.B. (2016). Influence of paternal preconception exposures on their offspring: Through epigenetics to phenotype. *American Journal of Stem Cells, 5*, 11–18.

Eberhart, J.K. & Parnell, S.E. (2016). The genetics of fetal alcohol spectrum disorders. *Alcoholism, Clinical and Experimental Research, 40*, 1154–1165.

Elliott, E., Payne, J.M., Morris, A., Haan, E. & Bower, C. (2008). Fetal alcohol syndrome: A prospective national surveillance study. *Archives of Disease in Childhood, 93*, 732–737.

Fast, D.K., Conry, J. & Loock, C.A. (1999). Identifying fetal alcohol syndrome among youth in the criminal justice system. *Journal of Developmental and Behavioral Pediatrics, 20*, 370–372.

Fuchs, D. & Burnside, L. (2014). *Study on the prevalence of FASD in Canadian child welfare settings: Final report*. Ottawa, ON: Centre of Excellence for Child Welfare.

Fuchs, D., Burnside, L., Marchenski, S. & Murdy, A. (2005). *Children with disabilities receiving services from child welfare agencies in Manitoba*. Ottawa, ON: Centre of Excellence for Child Welfare.

Goodlett, C.R. & Eilers, A.T. (1997). Alcohol-induced Purkinje cell loss with a single binge exposure in neonatal rats: A stereological study of temporal windows of vulnerability. *Alcoholism, Clinical and Experimental Research, 21*, 738–744.

Government of Ontario. (2014). *Ontario public schools enrolment: Enrolment in publicly funded elementary and secondary schools in Ontario*. Ottawa, ON: Author. Retrieved from https://www.ontario.ca/data/ontario-public-schools-enrolment

Graham, C. & Talay, D. (2013). *Stochastic simulation and Monte Carlo methods: Mathematical foundations of stochastic simulation*. Berlin, Germany: Springer.

Green, J.H. (2007). Fetal alcohol spectrum disorders: Understanding the effects of prenatal alcohol exposure and supporting students. *Journal of School Health, 77*, 103–108.

Habbick, B.F., Nanson, J.L., Snyder, R.E., Casey, R.E. & Schulman, A.L. (1996). Fetal alcohol syndrome in Saskatchewan: Unchanged incidence in a 20-year period. *Canadian Journal of Public Health, 87*, 204–207.

Hall, J.G., Froster-Iskenius, U.G. & Allanson, J.E. (1989). *Handbook of normal physical measurements*. New York, NY: Oxford University Press.

Hamilton, D.A., Barto, D., Rodriguez, C.I., Magcalas, C.M., Fink, B.C., Rice, J.P., . . . Savage, D.D. (2014). Effects of moderate prenatal alcohol exposure and age on social behavior, spatial response perseveration errors and motor behavior. *Behavioural Brain Research, 269*, 44–54.

Henriksen, T.B., Hjollund, N.H., Jensen, T.K., Bonde, J.P., Andersson, A.M., Kolstad, H., . . . Olsen, J. (2004). Alcohol consumption at the time of conception and spontaneous abortion. *American Journal of Epidemiology, 160*, 661–667.

Hoyme, H.E., Kalberg, W.O., Elliott, A.J., Blankenship, J., Buckley, D., Marais, A.S., . . . May, P.A. (2016). Updated clinical guidelines for diagnosing fetal alcohol spectrum disorders. *Pediatrics, 138*, e20154256.

Jacobson, J.L. & Jacobson, S.W. (1994). Prenatal alcohol exposure and neurobehavioral development: Where is the threshold? *Alcohol Health & Research World, 18*, 30–36.

Jacobson, J.L. & Jacobson, S.W. (1999). Drinking moderately and pregnancy. Effects on child development. *Alcohol Research & Health, 23*, 25–30.

Kesmodel, U., Olsen, S.F. & Secher, N.J. (2000). Does alcohol increase the risk of preterm delivery? *Epidemiology, 11*, 512–518.

Kesmodel, U., Wisborg, K., Olsen, S.F., Henriksen, T.B. & Secher, N.J. (2002). Moderate alcohol intake during pregnancy and the risk of stillbirth and death in the first year of life. *American Journal of Epidemiology, 155*, 305–312.

Koren, G.I., Fantus, E. & Nulman, I. (2010). Managing fetal alcohol spectrum disorder in the public school system: A needs assessment pilot. *Canadian Journal of Clinical Pharmacology, 17*, e79–e89.

Kowlessar, D.L. (1997). *An examination of the effects of prenatal alcohol exposure on school-age children in a Manitoba First Nation community: A study of fetal alcohol syndrome prevalence and dysmorphologies* (Unpublished master's thesis). University of Manitoba, Winnipeg.

Lange, S., Probst, C., Gmel, G., Rehm, J. & Popova, S. (2017). Global prevalence of fetal alcohol spectrum disorder among children and youth: A systematic review and meta-analysis. *JAMA Pediatrics, 171*, 948–956.

Lange, S., Shield, K., Koren, G., Rehm, J. & Popova, S. (2014). A comparison of the prevalence of prenatal alcohol exposure obtained via maternal self-reports versus meconium testing: A systematic literature review and meta-analysis. *BMC Pregnancy and Childbirth, 14*, 127.

Lipinski, R.J., Hammond, P., O'Leary-Moore, S.K., Ament, J.J., Pecevich, S.J., Jiang, Y., . . . Sulik, K.K. (2012). Ethanol-induced face-brain dysmorphology patterns are correlative and exposure-stage dependent. *PLoS One, 7*, e43067.

Livy, D.J., Miller, E.K., Maier, S.E. & Andwest, J.R. (2003). Fetal alcohol exposure and temporal vulnerability: Effects of binge-like alcohol exposure on the developing rat hippocampus. *Neurotoxicology andTeratology, 25*, 447–458.

Lupton, C., Burd, L. & Harwood, R. (2004). Cost of fetal alcohol spectrum disorders. *American Journal of Medical Genetics. Part C, Seminars in Medical Genetics, 127C*, 42–50.

MacPherson, P.H., Chudley, A.E. & Grant, B.A. (2011). *Fetal alcohol spectrum disorder (FASD) in a correctional population: Prevalence, screening and characteristics*. Ottawa, ON: Correctional Service Canada.

Mamluk, L., Edwards, H.B., Savović, J., Leach, V., Jones, T., Moore, T.H.M., . . . Zuccolo, L. (2017). Low alcohol consumption and pregnancy and childhood outcomes: Time to change guidelines indicating apparently "safe" levels of alcohol during pregnancy? A systematic review and meta-analyses. *BMJ Open, 7*, e015410.

Mattson, S.N., Crocker, N. & Nguyen, T.T. (2011). Fetal alcohol spectrum disorders: Neuropsychological and behavioral features. *Neuropsychology Review, 21*, 81–101.

Mattson, S.N. & Riley, E.P. (2000). Parent ratings of behavior in children with heavy prenatal alcohol exposure and IQ-matched controls. *Alcoholism, Clinical and Experimental Research, 24*, 226–231.

May, P.A., Baete, A., Russo, J., Elliott, A.J., Blankenship, J., Kalberg, W.O., . . . Hoyme, H.E. (2014). Prevalence and characteristics of fetal alcohol spectrum disorders. *Pediatrics, 134*, 855–866.

May, P.A., Blankenship, J., Marais, A.S., Gossage, J.P., Kalberg, W.O., Barnard, R., . . . Seedat, S. (2013). Approaching the prevalence of the full spectrum of fetal alcohol spectrum disorders in a South African population-based study. *Alcoholism, Clinical and Experimental Research, 37*, 818–830.

May, P.A., Brooke, L., Gossage, J.P., Croxford, J., Adnams, C., Jones, K.L., . . . Viljoen, D. (2000). Epidemiology of fetal alcohol syndrome in a South African community in the Western Cape province. *American Journal of Public Health, 90*, 1905–1912.

May, P.A., Fiorentino, D., Coriale, G., Kalberg, W.O., Hoyme, H.E., Aragón, A.S., . . . Ceccanti, M. (2011). Prevalence of children with severe fetal alcohol spectrum disorders in communities near Rome, Italy: New estimated rates are higher than previous estimates. *International Journal of Environmental Research and Public Health, 8*, 2331–2351.

May, P.A., Fiorentino, D., Gossage, J.P., Kalberg, W.O., Hoyme, H.E., Robinson, L.K., . . . Ceccanti, M. (2006). The epidemiology of FASD in a province in Italy: Prevalence and characteristics of children in a random sample of schools. *Alcoholism, Clinical and Experimental Research, 30*, 1562–1575.

May, P.A. & Gossage, J.P. (2001). Estimating the prevalence of fetal alcohol syndrome: A summary. *Alcohol Research and Health, 25*, 159–167.

May, P.A. & Gossage, J.P. (2011). Maternal risk factors for fetal alcohol spectrum disorders: Not as simple as it might seem. *Alcohol Research & Health, 34*, 15–26.

May, P.A., Gossage, J.P., Marais, A.S., Adnams, C.M., Hoyme, H.E., Jones, K.L., . . . Viljoen, D.L. (2007). The epidemiology of fetal alcohol syndrome and partial FAS in a South African community. *Drug and Alcohol Dependence, 88*, 259–271.

May, P.A., Keaster, C., Bozeman, R., Goodover, J., Blakenship, J., Kalberg, W.O., . . . Hoyme, H.E. (2015). Prevalence and characteristics of fetal alcohol syndrome and partial fetal alcohol syndrome in a Rocky Mountain region city. *Drug and Alcohol Dependence, 155*, 118–127.

McLachlan, K. (2017). *FASD study: Prevalence in corrections*. Whitehorse, YK: Government of Yukon, Department of Justice. Retrieved from www.justice.gov.yk.ca/FASDStudy.html

Millar, J.A., Thompson, J., Schwab, D., Hanlon-Dearman, A., Goodman, D., Koren, G. & Masotti, P. (2017). Educating students with FASD: Linking policy, research and practice. *Journal of Research in Special Education, 17*, 3–17.

Murphy, A., Chittenden, M. & McCreary Centre Society. (2005). *Time out II: A profile of BC youth in custody*. Vancouver, BC: McCreary Centre Society. Retrieved from www.mcs.bc.ca/pdf/time_out_2.pdf

O'Callaghan, F.V., O'Callaghan, M., Najman, J.M., Williams, G.M. & Bor, W. (2003). Maternal alcohol consumption during pregnancy and physical outcomes up to 5 years of age: A longitudinal study. *Early Human Development, 71*, 137–148.

O'Leary-Moore, S.K., Parnell, S.E., Lipinski, R.J. & Sulik, K.K. (2011). Magnetic resonance–based imaging in animal models of fetal alcohol spectrum disorder. *Neuropsychology Review, 21*, 167–185.

Olivier, L., Urban, M., Chersich, M., Temmerman, M. & Viljoen, D. (2013). Burden of fetal alcohol syndrome in a rural West Coast area of South Africa. *South African Medical Journal, 103*, 402–405.

Ortega-García, J.A., Gutierrez-Churango, J.E., Sánchez-Sauco, M.F., Martínez-Aroca, M., Delgado-Marín, J.L., Sánchez-Solis, M., . . . Martínez-Lage, J.F. (2012). Head circumference at birth and exposure to tobacco, alcohol and illegal drugs during early pregnancy. *Child's Nervous System, 28*, 433–439.

Patra, J., Bakker, R., Irving, H., Jaddoe, V.W.V., Malini, S. & Rehm, J. (2011). Dose-response relationship between alcohol consumption before and during pregnancy and risk of low birth weight, preterm birth and small for gestational age (SGA): A systematic review and meta-analyses. *BJOG, 118*, 1411–1421.

Petković G. & Barišić I. (2010). FAS prevalence in a sample of urban schoolchildren in Croatia. *Reproductive Toxicology, 29*, 237–241.

Petković G. & Barišić I. (2013). Prevalence of fetal alcohol syndrome and maternal characteristics in a sample of schoolchildren from a rural province of Croatia. *International Journal of Environmental Research and Public Health, 10*, 1547–1561.

Popova, S., Lange, S., Burd, L. & Rehm, J. (2016). Economic burden of fetal alcohol spectrum disorder in Canada in 2013. *Alcohol and Alcoholism, 51*, 367–375.

Popova, S., Lange, S., Mihic, A., Bekmuradov, D. & Rehm, J. (2011). Prevalence of fetal alcohol spectrum disorder in correctional systems: A systematic literature review. *Canadian Journal of Public Health, 102*, 336–340.

Popova, S., Lange, S., Probst, C., Gmel, G. & Rehm, J. (2017). Estimation of national, regional, and global prevalence of alcohol use during pregnancy and fetal alcohol syndrome: a systematic review and meta-analysis. *Lancet Global Health, 5*, e290–e299.

Popova, S., Lange, S., Probst, C. & Rehm, J. (2017). Prevalence of alcohol consumption during pregnancy and fetal alcohol spectrum disorders among the general and Aboriginal populations in Canada and the United States. *European Journal of Medical Genetics, 60*, 32–48.

Popova, S., Lange, S., Shield, K., Mihic, A., Chudley, A.E., Mukherjee, R.A.S., . . . Rehm, J. (2016). Comorbidity of fetal alcohol spectrum disorder: A systematic review and meta-analysis. *Lancet, 387*, 978–987.

Public Health Agency of Canada. (2003). *Fetal alcohol spectrum disorder (FASD): A framework for action*. Ottawa, ON: Author.

Robert, M., Carceller, A., Domken, V., Ramos, F., Dobrescu, O., Simard, M.N. & Gosselin, J. (2009). Physical and neurodevelopmental evaluation of children adopted from Eastern Europe. *Canadian Journal of Clinical Pharmacology, 16*, e432–e440.

Roberts, G. & Nanson, J. (2001). *Best practices. Fetal alcohol syndrome / fetal alcohol effects and the effects of other substance use during pregnancy*. Ottawa, ON: Minister of Public Works and Government Services Canada. Retrieved from http://publications.gc.ca/collections/Collection/H49-156-1-2001E.pdf

Robinson, G.C., Conry, J.L. & Conry, R.F. (1987). Clinical profile and prevalence of fetal alcohol syndrome in an isolated community in British Columbia. *Canadian Medical Association Journal, 137*, 203–207.

Rojas, E.Y. & Gretton, H.M. (2007). Background, offence characteristics, and criminal outcomes of Aboriginal youth who sexually offend: A closer look at Aboriginal youth intervention needs. *Sexual Abuse, 19*, 257–283.

Sood, B., Delaney-Black, V., Covington, C., Nordstrom-Klee, B., Ager, J., Templin, T., . . . Sokol, R.J. (2001). Prenatal alcohol exposure and childhood behavior at age 6 to 7 years: I. Dose-response effect. *Pediatrics, 108*, E34.

Stata Corporation. (2017). Statistical software: Release 15. College Station, TX: Author.

Statistics Canada. (2017). Population and dwelling counts, for Canada, provinces and territories, and census divisions, 2016 and 2011 censuses – 100% data [Table]. Ottawa, ON: Author. Retrieved from www12.statcan.gc.ca/census-recensement/2016/dp-pd/hlt-fst/pd-pl/Table.cfm?Lang=Eng&T=701&S=3

Stratton, K., Howe, C. & Battaglia, F. (Eds). (1996). *Fetal alcohol syndrome: Diagnosis, epidemiology, prevention, and treatment*. Washington, DC: National Academies Press.

Streissguth, A.P., Barr, H.M., Kogan, J. & Bookstein, F.L. (1996). *Understanding the occurrence of secondary disabilities in clients with fetal alcohol syndrome (FAS) and fetal alcohol effects (FAE)*. Seattle, WA: University of Washington.

Sulik, K.K. (2005). Genesis of alcohol-induced craniofacial dysmorphism. *Experimental Biology and Medicine, 230*, 366–375.

Sulik, K.K. (2014). Fetal alcohol spectrum disorder: Pathogenesis and mechanisms. *Handbook of Clinical Neurology, 125*, 463–475.

Thanh, N.X., Jonsson, E., Salmon, A. & Sebastianski, M. (2014). Incidence and prevalence of fetal alcohol spectrum disorder by sex and age group in Alberta, Canada. *Journal of Population Therapeutics and Clinical Pharmacology, 21*, e395–e404.

Urban, M., Chersich, M.F., Fourie, L.A., Chetty, C., Olivier, L. & Viljoen, D. (2008). Fetal alcohol syndrome among grade 1 schoolchildren in Northern Cape province: Prevalence and risk factors. *South African Medical Journal, 98*, 877–882.

Urban, M.F., Olivier, L., Viljoen, D., Lombard, C., Louw, J.G., Drotsky, L.M., . . . Chersich, M.F. (2015). Prevalence of fetal alcohol syndrome in a South African city with a predominantly black African population. *Alcoholism, Clinical and Experimental Research, 39*, 1016–1026.

Viljoen, D.L., Gossage, J.P., Brooke, L., Adnams, C.M., Jones, K.J., Robinson, L.K., . . . May, P.A. (2005). Fetal alcohol syndrome epidemiology in a South African community: A second study of a very high prevalence area. *Journal of Studies on Alcohol, 66*, 593–604.

Werk, C.M., Cui, X. & Tough, S. (2013). Fetal alcohol spectrum disorder among Aboriginal children under six years of age and living off reserve. *First Peoples Child & Family Review, 8*, 7e16.

Williams, R.J., Odaibo, F.S. & McGee, J.M. (1999). Incidence of fetal alcohol syndrome in northeastern Manitoba. *Canadian Journal of Public Health, 90*, 192–194.

World Health Organization. (2004). *International classification of diseases and related health problems* (10th rev.). Geneva, Switzerland: Author.

World Medical Association. (2013). *Declaration of Helsinki: Ethical principles for medical research involving human subjects.* Retrieved from https://www.wma.net/policies-post/wma-declaration-of-helsinki-ethical-principles-for-medical-research-involving-human-subjects/

Yang, Q., Witkiewicz, B.B., Olney, R.S., Liu, Y., Davis, M., Khoury, M.J., . . . Erickson, J.D. (2001). A case-control study of maternal alcohol consumption and intrauterine growth retardation. *Annals of Epidemiology, 11*, 497–503.

Appendix A

Comparative Analysis of Regional Municipalities of the Greater Toronto Area (GTA), the GTA, Ontario and Canada

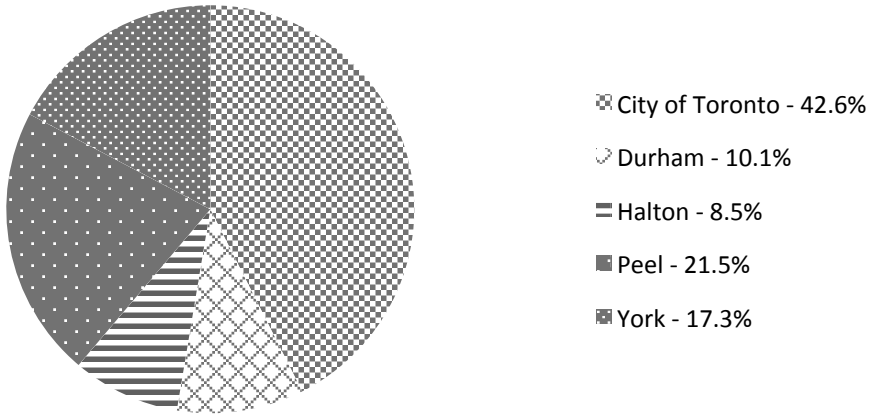
Size and Population

The GTA is made up of the City of Toronto and four neighboring municipal areas: Durham, Halton, Peel and York. The GTA covers an area of 7,124.15 km² (2,751 mi²) (Country Digest, 2017).

In 2016, the GTA population (6,417,516) made up 18.3% of Canada's total population (35,151,728): City of Toronto (2,731,571; 42.6%), Durham (645,862; 10.1%), Halton (548,435; 8.5%), Peel (1,381,739; 21.5%) and York (1,109,909; 17.3%) (Statistics Canada, 2017a; Figure A1). The GTA population density in 2016 was 849 people per km² (2,199 mi²): City of Toronto (4,149.5/km² [10,747/mi²]), Durham (241.0/km² [624/mi²]), Halton (520.4/km² [1,348/mi²]), Peel (1,040.0/km² [2,694/mi²]) and York (585.9/km² [1,517/mi²] (Statistics Canada, 2017a).

FIGURE A1

Population distribution in Greater Toronto Area by regional municipality, 2016



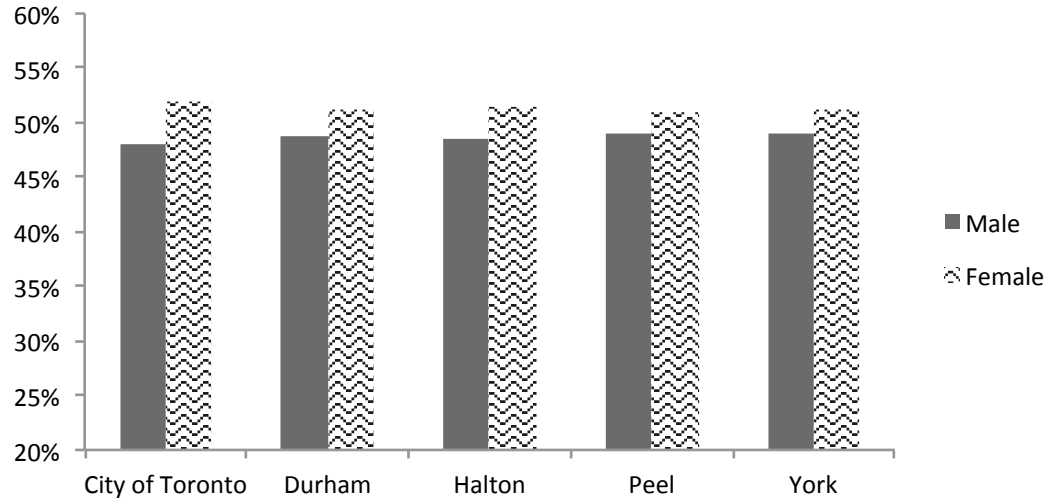
Source: Statistics Canada (2017a).

Demographics

The sex composition of the GTA population in 2011 (the latest available year) was 48.0% (2,937,500) male: City of Toronto (1,255,585; 48.0%), Durham (296,310; 48.7%), Halton (243,735; 48.6%), Peel (637,180; 49.1%) and York (504,690; 48.9%; Figure A2). This demographic is comparable to the national average of 49.0% (Statistics Canada, 2012; Figure A3).

FIGURE A2

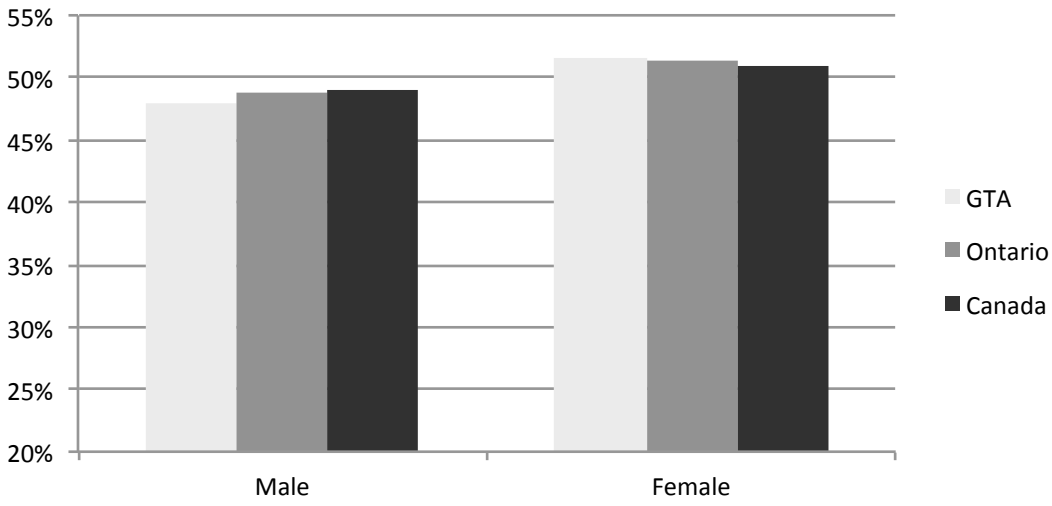
Population composition by sex in Greater Toronto Area by regional municipality, 2011



Source: Statistics Canada (2012).

FIGURE A3

Population composition by sex in Greater Toronto Area compared with Ontario and Canada, 2011 and 2016

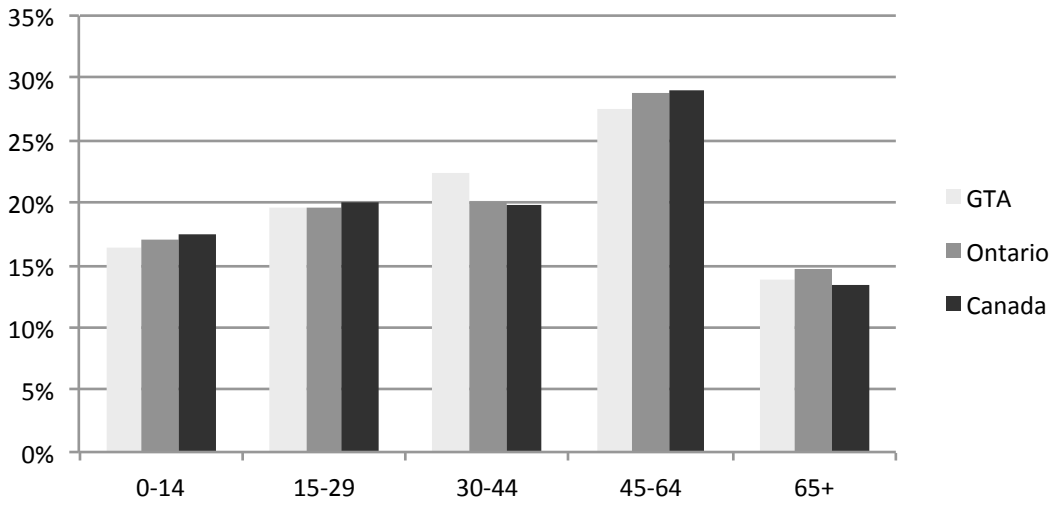


Source: Statistics Canada (2012, 2017b).

The median population age in the GTA in 2011 was 39.2 years, 1.4 years below the national median of 40.6 years (Statistics Canada, 2012; Figure A4): 39.2 years was the median age in the City of Toronto (38.2: males, 40.1: females), 39.2 years in Durham (males: 38.2, females: 40.1), 39.3 years in Halton (males: 38.5, females: 40.1), 38.9 years in Peel (males: 36.1, females: 37.6) and 39.3 years in York (males: 38.3, females: 40.0). The national average by sex in Canada was 39.6 years for males and 41.5 years for females (Statistics Canada, 2017b). The age group 15–64 made up 69.4% in the GTA in 2016, and is projected to decline over the period 2016–2041. Growth in the other regions is projected to be significantly faster than the Ontario average, with the addition of over 1.8 million people to the suburbs of the GTA (Ontario Ministry of Finance, 2017).

FIGURE A4

Population composition by age group in Greater Toronto Area compared with Ontario and Canada, 2011

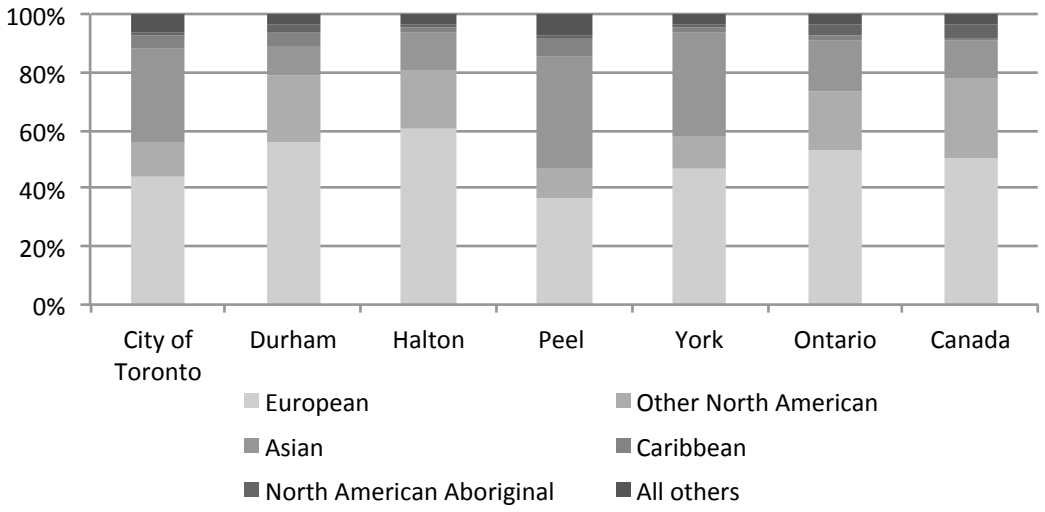


Source: Statistics Canada (2012).

The 2011 Census (Statistics Canada, 2013a; Figure A5) identified the three most common ethnicities by region. City of Toronto: English (777,115; 12.9%), Chinese (594,735; 12.0%) and Canadian (728,740; 11.3%); Durham: European (424,495; 70.6%), North American (176,865; 29.4%) and Asian (77,145; 12.8%); Halton: European (369,410; 74.6%), North American (122,145; 24.7%) and Asian (77,145; 15.0%); Peel: East Indian (268,865; 20.9%), Canadian (155,560; 12.1%) and English (143,750; 11.2%); and York: European (545,890; 53.3%), Asian (415,715; 40.6%) and North American (135,445; 13.2%). Although 53.3% (545,890) of York residents reported European ethnicities, similar to those of Durham and Halton, a higher proportion reported Asian ethnicities compared with these two regions (40.6%). The proportion of European ethnicities was also higher in Durham (70.6%) than it was in the GTA (53.0%). Peel residents accounted for 27.6% (732,805) of the GTA's 2,654,140 visible minority residents, who reported on average 3.6 ethnicities per person. Across Canada, 32.6% (10,563,805) of people noted Canadian as their ethnic origin, followed by English (6,509,500; 19.8%), French (5,065,690; 15.4%), Chinese (1,487,580; 4.5%) and First Nations (1,369,115; 4.2%).

FIGURE A5

Ethnic composition in Greater Toronto Area by regional municipality compared with Ontario and Canada, 2011

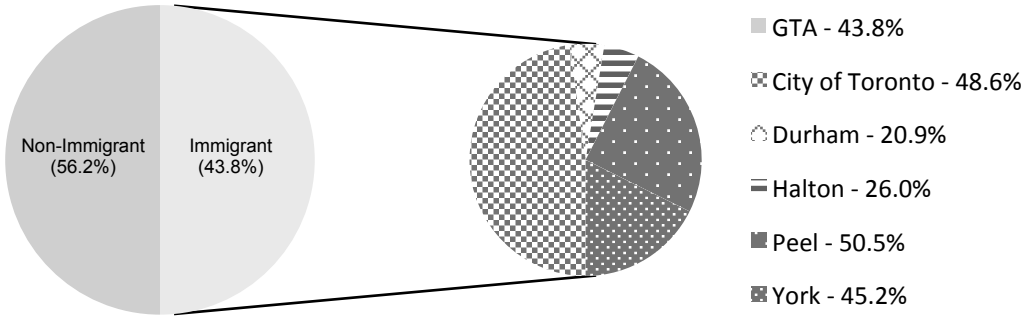


Source: Statistics Canada (2013a).

The proportion of immigrants in the GTA in 2011 represented 43.8% (2,620,455) of the population: City of Toronto (1,252,210; 48.6%), Durham (125,845; 20.9%), Halton (128,740; 26.0%), Peel (650,530; 50.5%) and York (463,120; 45.2%) (Statistics Canada, 2013b; Figure A6). The 2011 National Household Survey indicates that during this time, Canada had a total of 6,775,800 immigrants, representing 20.6% of the country's population (Statistics Canada, 2013b; Figure A7).

FIGURE A6

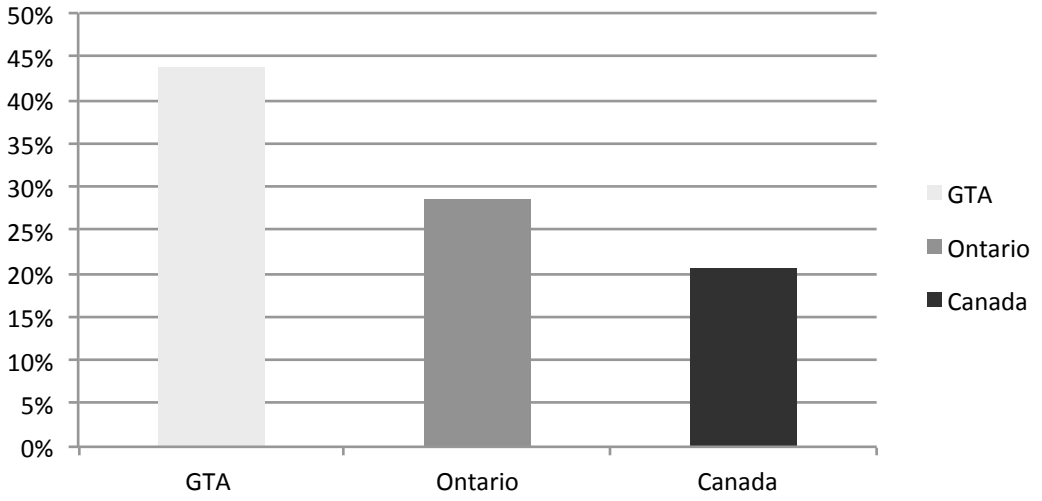
Proportion of immigrants in Greater Toronto Area by regional municipality, 2011



Source: Statistics Canada (2013b).

FIGURE A7

Proportion of immigrants in Greater Toronto Area compared with Ontario and Canada, 2011



Source: Statistics Canada (2013b).

In 2011, 78.5% of people in the GTA were affiliated with a religious group: 57.6% with Christianity, 7.2% with Islam and 5.5% with Hinduism. No religious affiliation was reported for 21.5% of GTA residents (Statistics Canada, 2013c). In the City of Toronto, 74.7% of people affiliated with a religious group: Christianity was the largest single religious group (54.1%), followed by those who reported no religion (24.2%) (Statistics Canada, 2013c). The percentage of people affiliated with a religious group in Durham was 74.7% in 2011, with 68.2% identifying as Christian (Statistics Canada, 2013c). Secularism is becoming more prevalent in Durham, with 25.3% of residents reporting no religious affiliation in 2011 (Statistics Canada, 2013c). Compared with the GTA, Halton and York had similar proportions with respect to religious affiliation, with 77.4% and 77.1%, respectively. Christianity was the largest group in both regions, with 69.4% in Halton and 55.5% in York, followed by those who reported no affiliation (22.6% and 22.9%, respectively). In 2011, 87.0% of Peel's population indicated being affiliated with a specific religion (the highest rate in the GTA): 56.9% identified as Christian, followed by 9.5% Sikh and 9.4% Muslim; 13.0% of Peel residents had no religious affiliation (Statistics Canada, 2013c). As evident from the data, the dominant religion across Canada is Christianity (67.3%) (Statistics Canada, 2013d; Figure A8). This is split mainly between those who are Catholic (38.7%) and Protestant (17.2%) (Statistics Canada, 2013d). A further 7.2% of Canadians reported that they follow other religions, identifying as Muslim (3.2%), Hindu (1.5%), Sikh (1.4%), Buddhist (1.1%) and Jewish (1.0%) (Statistics Canada, 2013d).

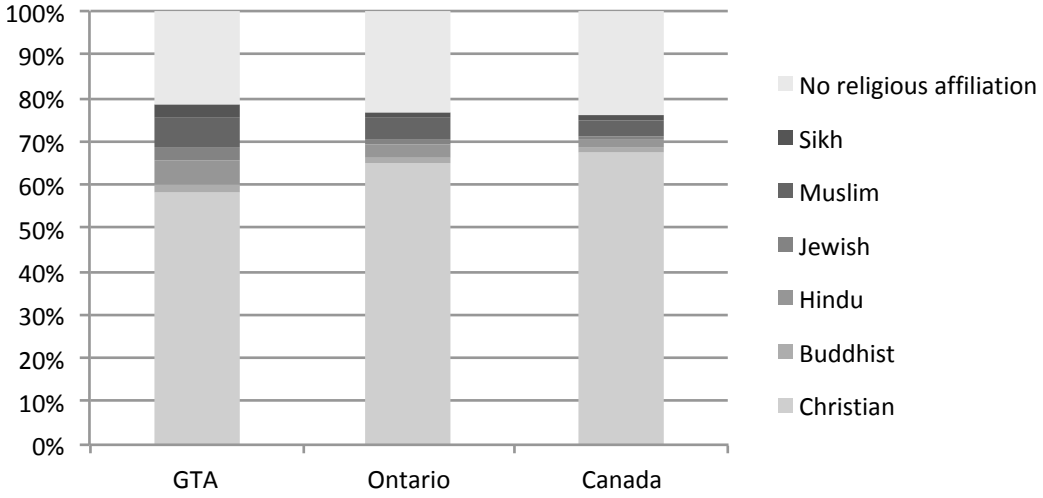
The median household income in the GTA was $71,554 (Canada: $84,593): City of Toronto ($58,381), Durham ($81,119), Halton ($91,955), Peel ($77,588) and York ($89,100) (Statistics Canada, 2013e; Figure A9).

Alcohol Consumption

Regional data from the Centre for Addiction and Mental Health (2017) indicate that the proportion of binge drinking (5+ drinks per occasion) was similar (about 6%) in the GTA and the rest of Ontario (Figure A10 and Figure A11). The proportion of lifetime drinkers was slightly lower in the GTA compared with the rest of Ontario (90.3% vs 95.3%, $p < .001$; Figure A12 and Figure A13).

FIGURE A8

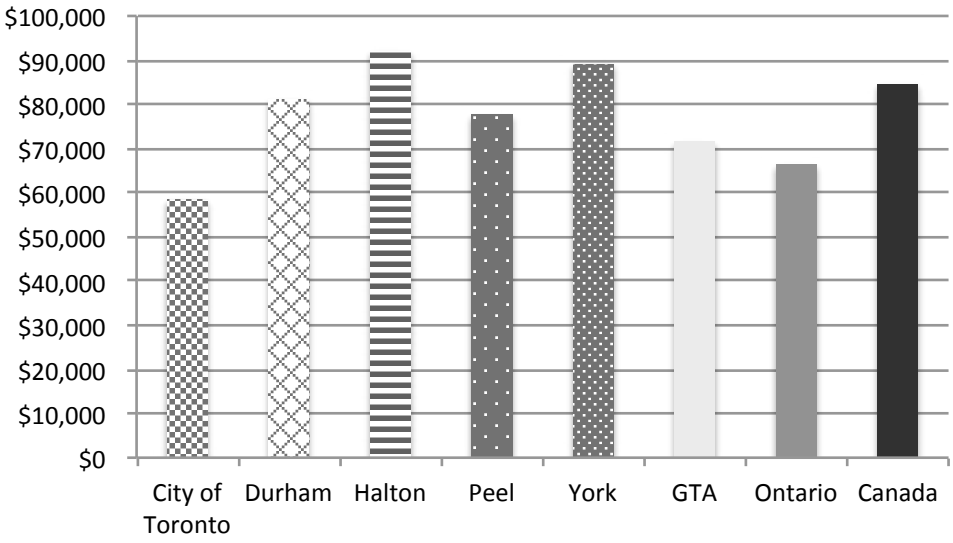
Composition of religious affiliation in Greater Toronto Area compared with Ontario and Canada, 2011



Source: Statistics Canada (2013c, 2013d).

FIGURE A9

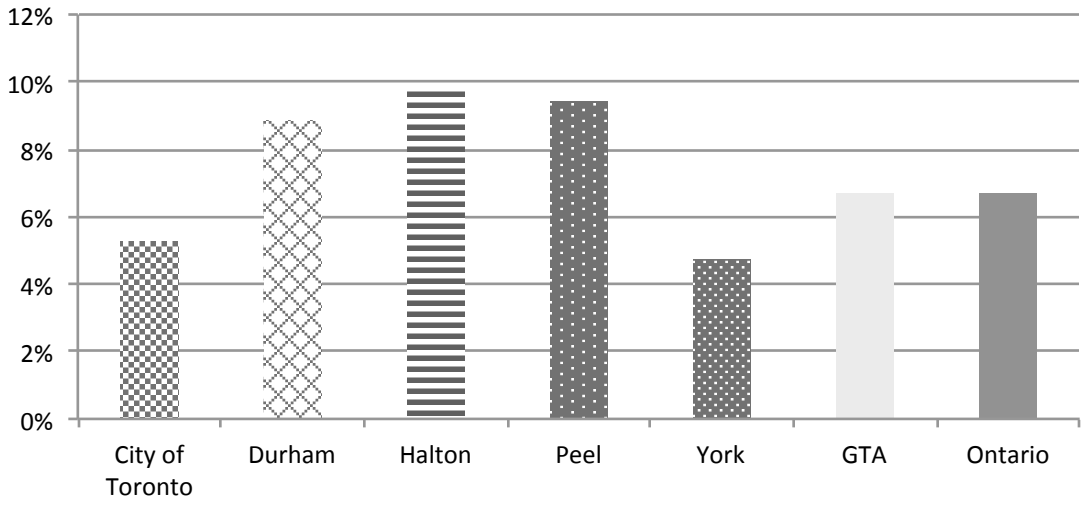
Median household income in Greater Toronto Area by regional municipality compared with Ontario and Canada, 2011



Source: Statistics Canada (2013e).

FIGURE A10

Binge drinking[a] (weekly) in Greater Toronto Area by regional municipality compared with Ontario, 2014–2016

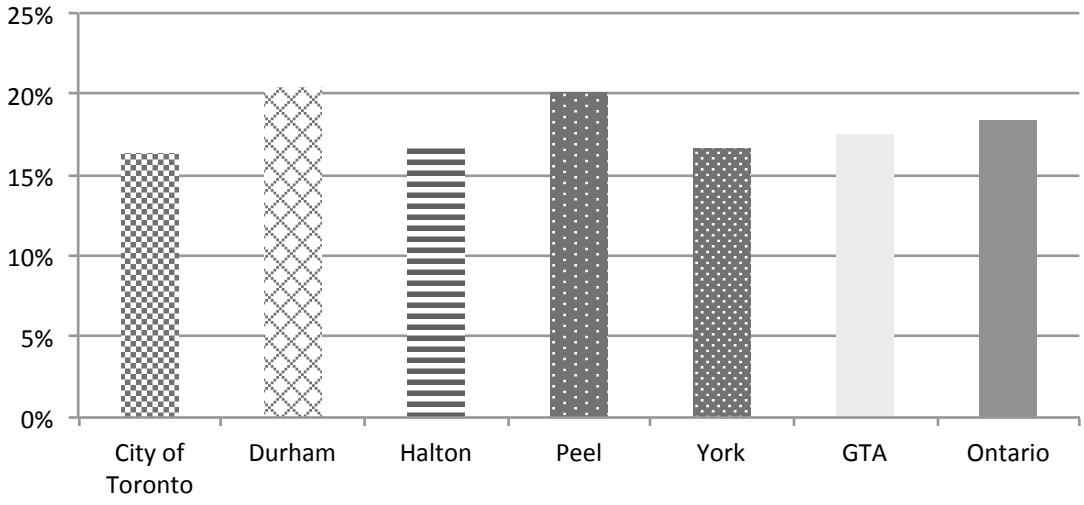


Note: Data obtained directly from the Centre for Addiction and Mental Health Monitor.

[a]Defined by CAMH Monitor as the percentage of people reporting drinking five or more alcoholic drinks on a single occasion on a weekly basis during the 12 months before the survey (available for 1996–2016).

FIGURE A11

Binge drinking[a] (monthly) in Greater Toronto Area by regional municipality compared with Ontario, 2014–2016

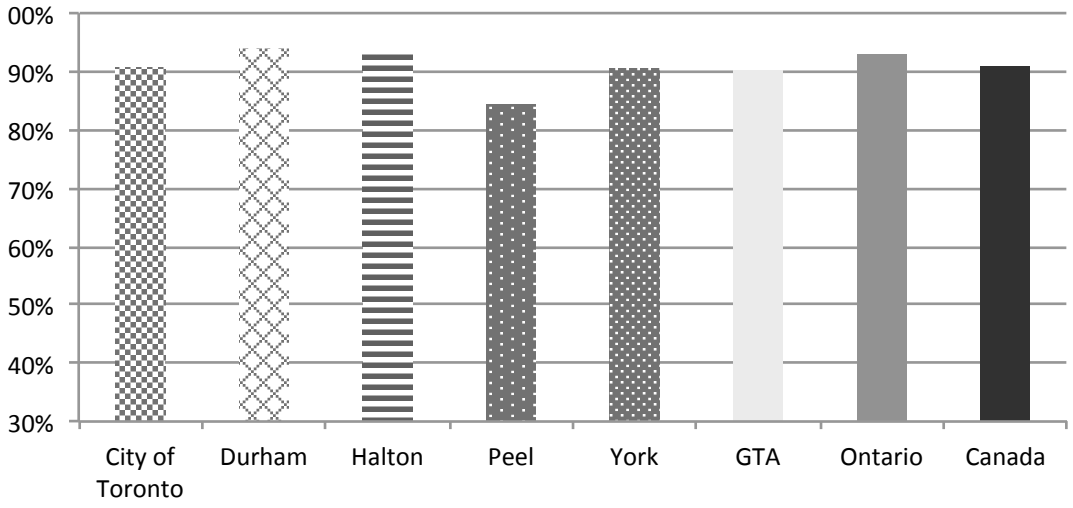


Note: Data obtained directly from the Centre for Addiction and Mental Health Monitor.

[a]Defined by CAMH Monitor as the percentage of people reporting drinking five or more alcoholic drinks on a single occasion on a monthly basis during the 12 months before the survey (available for 1996–2016).

FIGURE A12

Lifetime drinkers[a] in Greater Toronto Area by regional municipality compared with Ontario and Canada



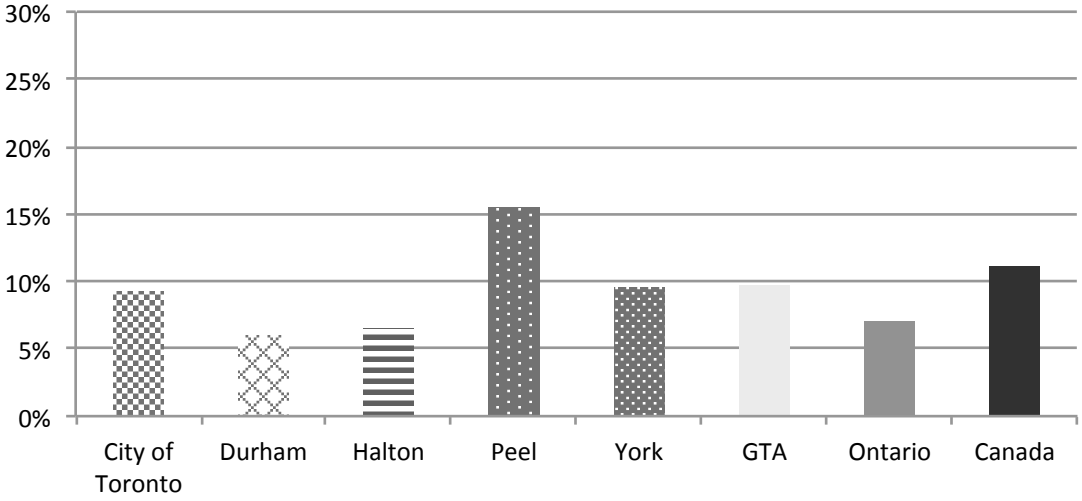
Note: Data for regions, GTA and Ontario obtained directly from the Centre for Addiction and Mental Health Monitor.

[a]Defined by CAMH Monitor as *current drinkers* (those reporting drinking alcohol in past 12 months) plus *former drinkers* (those drinking alcohol in their lifetime, but not in past 12 months).

Source: Canadian Tobacco, Alcohol and Drugs Survey (2015).

FIGURE A13

Lifetime abstainers[a] in Greater Toronto Area by regional municipality compared with Ontario and Canada, 2014–2016



Note: Data for regions, GTA and Ontario obtained directly from the Centre for Addiction and Mental Health Monitor.

[a]Defined by CAMH Monitor as those never drinking alcohol in their lifetime.

Source: World Health Organization (2014).

References

Country Digest. (2017). Toronto population 2017. Retrieved from countrydigest.org/toronto-population/

Ontario Ministry of Finance. (2017). *Ontario population projections update, 2016–2041. Greater Toronto Area and its census divisions, population by five-year age group, 2016–2041 – reference scenario* [Table]. Retrieved from https://www.fin.gov.on.ca/en/economy/demographics/projections/table10.html (accessed September 15, 2017).

Statistics Canada. (2012). *Age groups (25) and sex (3) for the population of Canada, provinces, territories, census divisions and census subdivisions, 2011 census* [Table]. *Census profile. 2011 Census* (Catalogue no. 98-311-XCB2011026). Ottawa, ON: Author. Retrieved from http://bit.ly/2mfx80H (accessed September 15, 2017).

Statistics Canada. (2013a). *Regional municipality, Ontario* [Table]. *NHS profile, 2011. National Household Survey* (Catalogue no. 99-004-XWE). Ottawa, ON: Author. Retrieved from www12.statcan.gc.ca/nhs-enm/2011/dp-pd/prof/index.cfm?Lang=E (accessed September 15, 2017).

Statistics Canada. (2013b). *Canada* [Table]. *NHS profile, 2011. National Household Survey* (Catalogue no. 99-004-XWE). Ottawa, ON: Author. Retrieved from www12.statcan.gc.ca/nhs-enm/2011/dp-pd/prof/index.cfm?Lang=E (accessed September 15, 2017).

Statistics Canada. (2013c). *Regional municipality, Ontario* [Table]. *NHS profile, 2011. National Household Survey* (Catalogue no. 99-004-XWE). Ottawa, ON: Author. Retrieved from www12.statcan.gc.ca/nhs-enm/2011/dp-pd/prof/index.cfm?Lang=E (accessed September 15, 2017).

Statistics Canada. (2013d). *Canada* [Table]. *NHS profile, 2011. National Household Survey* (Catalogue no. 99-004-XWE). Ottawa, ON: Author. Retrieved from www12.statcan.gc.ca/nhs-enm/2011/dp-pd/prof/index.cfm?Lang=E (accessed September 15, 2017).

Statistics Canada. (2013e). *Regional municipality, Ontario* [Table]. *NHS profile, 2011. National Household Survey* (Catalogue no. 99-004-XWE). Ottawa, ON: Author. Retrieved from www12.statcan.gc.ca/nhs-enm/2011/dp-pd/prof/index.cfm?Lang=E (accessed September 15, 2017).

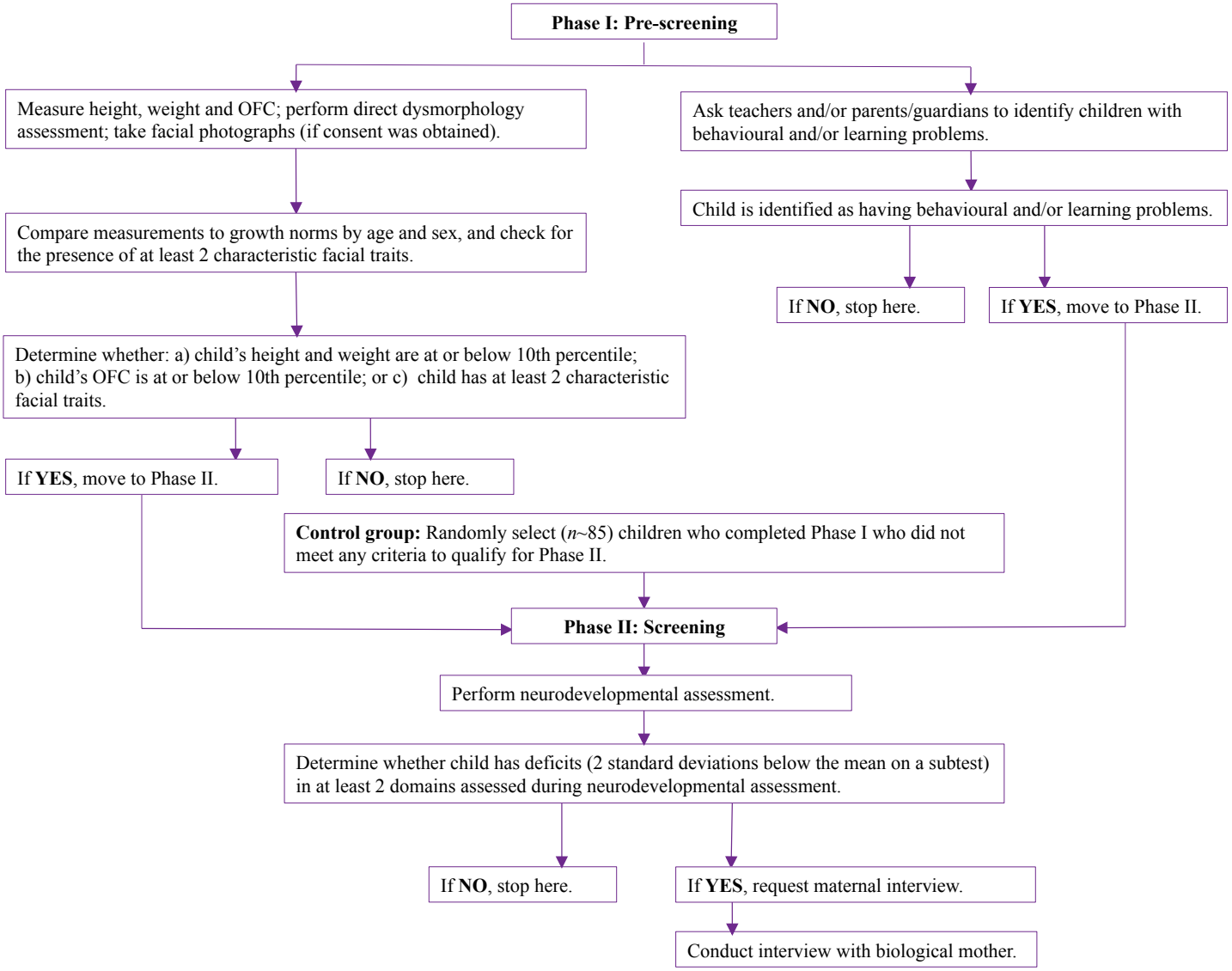
Statistics Canada. (2017a). *Regional municipality (Census division), Ontario and Ontario (Province)* [Table]. *Census profile, 2016 Census* (Catalogue no. 98-316-X2016001). Ottawa, ON: Author. Retrieved from www12.statcan.gc.ca/census-recensement/2016/dp-pd/prof/index.cfm?Lang=E (accessed September 15, 2017).

Statistics Canada. (2017b). *Canada (Country) and Canada (Country)* [Table]. *Census profile, 2016 Census* (Catalogue no. 98-316-X2016001). Ottawa, ON: Author. Retrieved from www12.statcan.gc.ca/census-recensement/2016/dp-pd/prof/index.cfm?Lang=E (accessed September 15, 2017).

World Health Organization. (2014). *Global status report on alcohol and health*. Geneva, Switzerland: Author. Retrieved from www.who.int/substance_abuse/publications/global_alcohol_report

Appendix B

Study Methodology



Case conference: Independently collected data are discussed on case-by-case basis by an expert panel that includes psychologists, geneticists, medical doctors, epidemiologists and study co-ordinator.

Disclosure of results: Provide parents/guardians with screening results as independent assessment of the child's strengths and weaknesses in regard to the physical evaluation and neurodevelopmental assessment.

Dissemination of study findings: Provide all participating school boards with summary of study results on aggregate level.

GLOBAL ID NUMBER

Appendix C

Physical Examination Form[1]

PHASE 1: Pre-screening

Child's gender: ☐ Male ☐ Female

Child's date of birth: _____/_____/_____ dd/mm/yy

Child's current age: _____/_____ yy/mm

Child's ethnic origin:

☐ Caucasian

☐ Aboriginal

☐ African Canadian / Jamaican

☐ Eastern European (e.g., Poland, Hungary, Croatia, Romania, Ukraine, Russia)

☐ Western European (e.g., Ireland, Italy, Netherlands, Denmark, United Kingdom, Germany)

☐ Chinese/Southeast Asian (e.g., Philippines, Thailand, Vietnam, Cambodia)

☐ South Asian (e.g., Afghanistan, Bangladesh, India, Pakistan, Sri Lanka)

☐ Other (please specify): _____

(HINT: When trying to determine a child's ethnic origin, try asking "Where were your parents born or which country did your parents come from?" or "Where were your grandparents born or which country did your grandparents come from?")

Date of examination: _____/_____/_____ dd/mm/yy

Photographs taken: ☐ Yes ☐ No

1 This form was adapted from the Collaborative Initiative on Fetal Alcohol Spectrum Disorders (CIFASD) consortium.

GLOBAL ID NUMBER

GROWTH

Height

1. Height (cm): _____

1a. Height percentile: _____

1b. Height percentile ≤ 10th percentile: ☐ Yes ☐ No

Weight

2. Weight (kg): _____

2a. Weight percentile: _____

2b. Weight percentile ≤ 10th percentile: ☐ Yes ☐ No

HEAD/FACE

Occipitofrontal Circumference (OFC)

3. OFC (cm): _____

3a. OFC percentile: _____

3b. OFC percentile ≤ 10th percentile: ☐ Yes ☐ No

Palpebral Fissure Length (PFL)

4. Left PFL (cm): _____

4a. Left PFL relative to mean (+1 *SD*, +2 *SD*, Mean, -1 *SD*, -2 *SD*): _____

4b. Left PFL ≤ 2 *SD* below the mean (≤ 3rd percentile): ☐ Yes ☐ No

5. Right PFL (cm): _____

5a. Right PFL relative to mean (+1 *SD*, +2 *SD*, Mean, -1 *SD*, -2 *SD*): _____

5b. Right PFL ≤ 2 *SD* below the mean (≤ 3rd percentile): ☐ Yes ☐ No

Inner Canthal Distance (ICD)

6. ICD (cm): _____

6a. ICD percentile: _____

Philtrum

7. Philtrum length (cm): _____

8. Philtrum score on the lip-philtrum guide (smoothness): _____

8a. Smooth philtrum (4 or 5 on the lip-philtrum guide): ☐ Yes ☐ No

Vermilion Border

9. Vermillion border (upper lip) score on the lip-philtrum guide: _____

9a. Thin vermillion border (4 or 5 on the lip-philtrum guide): ☐ Yes ☐ No

Hypoplastic

10. Hypoplastic midface: ☐ Yes ☐ No

Railroad Track Ears

11. Railroad track configuration of ears: ☐ Yes ☐ No

Strabismus

12. Strabismus: ☐ Yes ☐ No

12a. **If yes**: ☐ Unilateral ☐ Bilateral

Ptosis

13. Ptosis: ☐ Yes ☐ No

Epicanthal Folds

14. Epicanthal folds: ☐ Yes ☐ No

Anteverted Nares

15. Anteverted nares: ☐ Yes ☐ No

JOINTS

Clinodactyly

16. Clinodactyly 5th fingers: ☐ Yes ☐ No

16a. **If yes**: ☐ Unilateral ☐ Bilateral

Camptodactyly

17. Camptodactyly: ☐ Yes ☐ No

17a. **If yes**: ☐ Unilateral ☐ Bilateral

Pronation/Supination of Elbow

18. Difficulty pronation/supination elbows: ☐ Yes ☐ No

HANDS

Palmar Crease

19. Hockey stick upper palmar crease: ☐ Yes ☐ No

19a. **If yes**: ☐ Unilateral ☐ Bilateral

20. Other altered palmar creases: ☐ Yes ☐ No

20a. **If yes**: ☐ Unilateral ☐ Bilateral

20b. Single crease: ☐ Yes ☐ No
Hypoplastic thenar crease: ☐ Yes ☐ No
Other: _____

21. Name of examiner (conducting dysmorphology assessment): _____

22. Did participant receive a learning and/or behavioural referral? ☐ Yes ☐ No

Screening Results

22. Participant should proceed to Phase II (based on height, weight, OFC, PFL, philtrum and vermillion border ratings, and question 22): ☐ Yes ☐ No

Children should be referred to the second phase of the study—the neurodevelopmental assessment—if they are found to: 1) have growth deficits (height and weight at or below the 10th percentile; and/or OFC at or below the 10th percentile); 2) and/or have at least two of the three characteristic facial features (short palpebral fissures, smooth or flattened philtrum, thin vermilion border of the upper lip); and/or 3) have been previously identified as having learning and/or behavioural problems.

Please make sure the child's identification number (global ID number) is written at the top of each sheet. If you have questions about how an item should be completed, you can phone the study coordinator at: (XXX) XXX-XXXX ext. XXXX or e-mail XXXXXXXXX.

GLOBAL ID NUMBER

Appendix D

Interview of Biological Mother[1]

Date of interview: ____/____/____ (dd/mm/yy)

Start time: _____ am/pm (circle one)

Interviewer's name: _____

**All questions need to be asked unless specified otherwise. All comments for interviewers are written in italics, and appear after an asterisk.*

Section A: Demographics and Living Environment

** "First off, I'm going to ask you some questions about your living situation, education, and work history when you were pregnant with your child."*

1. How old are you? _____ (years)

2a. What best describes your nationality?

- ☐ Caucasian
- ☐ Aboriginal origins
- ☐ African Canadian / Jamaican
- ☐ Eastern European (e.g., Poland, Hungary, Croatia, Romania, Ukraine, Russia)
- ☐ Western European (e.g., Ireland, Italy, Netherlands, Denmark, UK, Germany)
- ☐ Chinese/Southeast Asian (e.g., Philippines, Thailand, Vietnam, Cambodia)
- ☐ South Asian (e.g., Afghanistan, Bangladesh, India, Pakistan, Sri Lanka)
- ☐ Other

2b. If "Other," please specify: _____

1 Some sections of this questionnaire were adapted from the Collaborative Initiative on Fetal Alcohol Spectrum Disorders (CIFASD) consortium and from Dr. Philip May's studies.

3. What was your marital status when you were pregnant with your child?

- ☐ Single
- ☐ Married, living with husband
- ☐ Not married but living with partner
- ☐ Separated from spouse
- ☐ Divorced
- ☐ Widowed

4. Were you unemployed within the 12-month period leading up to your pregnancy?

- ☐ Yes **Go to question 5a.*
- ☐ No

4a. What did you do in the 12 months leading up to your pregnancy? (i.e., what was your job/ occupation?) _____

5a. How many full-time years of school had you completed by the time you were pregnant?
**(Starting point: Grade 1; e.g., if completed Grade 9, enter "9")* _____

5b. What was the highest level of education that you had completed by the time you were pregnant?

- ☐ No formal schooling or < Grade 5
- ☐ Less than 9 years
- ☐ 9 years (uncompleted high school diploma)
- ☐ High school diploma / vocational or trade school
- ☐ College degree or unfinished university education
- ☐ University graduate
- ☐ Scientific degree (master's degree or doctorate)

If the mother is single, separated, divorced or widowed, skip questions 6 and 7 and go to question 8a.

6. Was your spouse/partner unemployed during the 12-month period leading up to your pregnancy?

- ☐ Yes *Go to question 7a.*
- ☐ No

6a. What did he do within the 12 months before your pregnancy? (i.e., what was his job/occupation?)

_____

7a. How many full-time years of school had he completed by the time you were pregnant?
(Starting point: Grade 1; e.g., if completed Grade 9, enter "9") _____

7b. What was the highest level of education he had completed by the time you were pregnant?

- ☐ No formal schooling or < Grade 5
- ☐ Less than 9 years
- ☐ 9 years (uncompleted high school diploma)
- ☐ High school diploma /vocational or trade school
- ☐ College degree or unfinished university education
- ☐ University graduate
- ☐ Scientific degree (master's degree or doctorate)

8a. Did anyone else help to support you financially when you became pregnant?

☐ Yes ☐ No

8b. Did s/he provide at least half of your financial support?

- ☐ Yes *Go to question 8c.*
- ☐ No *Stop here, and go to Section B.*

8c. Who provided this support?

- ☐ Child's grandmother and/or grandfather
- ☐ Child's father
- ☐ Other relative
- ☐ Other non-relative

Section B: Pregnancy-Related Questions

**"Now I'm going to ask you about your pregnancy with (name of the child) and any previous times you may have been pregnant."*

9. Did you plan to get pregnant with this child?

   ☐ Yes

   ☐ No, not at that time

   ☐ No, not at any time

10. How many times have you been pregnant? _____

11. How many live-born children have you had? _____
    **For live-born children only (specify this to the participant):*

12. Were any of them born premature (< 37 weeks' gestation)?

    ☐ Yes ☐ No

13a. Did any of them have any birth defects?

    ☐ Yes ☐ No

13b. Did the child participating in this study have any birth defects?

    ☐ Yes

    ☐ No **Stop here and go to Section C.*

13c. Please specify what birth defect(s) **Check yes or no for each defect.*

    Down syndrome: ☐ Yes ☐ No

    Cleft lip: ☐ Yes ☐ No

    Neural tube defect: ☐ Yes ☐ No

    Cystic fibrosis: ☐ Yes ☐ No

    Heart defect: ☐ Yes ☐ No

    If yes to heart defect, please specify: _____

    Other: ☐ Yes ☐ No

    If yes, please specify: _____

14a. Do any of your children have fetal alcohol syndrome (FAS) or fetal alcohol spectrum disorder (FASD)?

☐ Yes **If yes, go to question 14b.*

☐ No **If no, go to question 15.*

☐ Do not know what FAS or FASD are

14b. Was the child participating in this study diagnosed with FAS or FASD?

☐ Yes ☐ No

15. If you had any other pregnancy complications, please specify: _____

_____

_____

Section C: Maternal Alcohol Use

**"The next set of questions is about your use of alcoholic beverages at different times in your life. Please answer these questions to the best of your ability. We are asking these questions because we want to accurately reflect the information for the purposes of our research. The information is completely confidential and we are not here to judge you, nor should you judge yourself."*

Lifetime Drinking Behaviour

16. Have you consumed alcohol ever in your lifetime?

☐ Yes

☐ No **Stop here and proceed to Section D.*

17. How old were you the first time you drank alcohol? DO NOT include childhood sips you might have had from an older person's drink.

_____ years old (or best guess)

☐ Refused to answer

☐ Cannot remember

18. How old were you when you first began to drink alcohol regularly, meaning once a month or more often?

_____ years (or best guess)

☐ Refused to answer

☐ Cannot remember

19. If you are currently NOT drinking alcoholic beverages, when did you have your last drink?

_____ days ago, or

_____ weeks ago, or

_____ months ago, or

_____ years ago

20. If you do NOT drink, are you a recovering drinker? ☐ Yes ☐ No

Remarks: _____

_____

_____

21. Have you ever stopped drinking alcohol completely? **Check only one answer.*

☐ Yes, currently not drinking

☐ No

☐ Yes, stopped for a while one or more times in the past

22. If you have stopped drinking alcohol at any time in the past, why did you stop? _____

_____

_____

23. What keeps or kept you abstinent (i.e., sober)? _____

_____

_____

24. In the past, have you started drinking alcohol again after stopping for a period of time? Meaning, for example, did you stop drinking for a week, or a month, or a year or for several years, and then start drinking again? Check only ONE answer.

☐ Yes, once

☐ No

☐ Yes, more than once

25. What was the longest period of time you were abstinent (did not drink)? How many days, weeks, months or years?

_____ days, or

_____ weeks, or

_____ months, or

_____ years

26. Do you currently have a drinking problem?

☐ Yes ☐ No

If yes, why do you think so? _____

27. Have you ever gone to anyone for help with a drinking problem?

☐ Yes ☐ No

28. Have you ever had any health problems as a result of drinking alcoholic beverages?

☐ Yes **Ask the woman to explain. Use the lines below.*

☐ No

_____

_____

29. Have you ever felt that you ought to cut down on your drinking?

☐ Yes ☐ No

Drinking Behaviour during the Past 30 Days

30. As you think back over the past 30 days, on the days that you drank alcohol, how many drinks did you usually drink?

_____ drinks (prompt: 1–2 drinks, 3–4, 5-7, 8–10, more than 10)

☐ Refused to answer

☐ Cannot remember

31. If you do drink, on the average, how often did you drink this amount?

☐ Every day	☐ Almost every day	☐ 3–4 times a week	☐ 1–2 times a week	☐ 2–3 times a month	☐ Once a month
☐ 1–2 times in 3 months	☐ Less than 1 time in 3 months	☐ Refused to answer	☐ Cannot remember	☐ NA; not greater than 0	

32. On how many different days during the past 30 days did you have one or more drinks of beer, wine, or liquor?

_____ days

33. On how many days did you have three (3) or more drinks of beer, wine, or liquor, on the same occasion during the past 30 days?

_____ days

34. What is the most you had to drink on any one day that you drank beer, wine, or liquor during the past 30 days?

_____ drinks

35. How many days did you have this number of drinks of beer, wine, or liquor in the past 30 days?

_____ days

Drinking Behaviour before Pregnancy with Index Child

*"*Now, I would like to ask you a few questions about your drinking behavior during your pregnancy, but before you knew/recognized that you were pregnant with (name of child)."*

36. Before you knew you were pregnant with (name of child), on the days you drank alcohol, how many drinks did you usually drink?

_____ drinks (prompt: 1–2 drinks, 3–4, 5–7, 8–10, more than 10)

☐ Refused to answer

☐ Cannot remember

37. If you do drink, on the average, how often did you drink this amount?

☐ Every day	☐ Almost every day	☐ 3–4 times a week	☐ 1–2 times a week	☐ 2–3 times a month	☐ Once a month
☐ 1–2 times in 3 months	☐ Less than 1 time in 3 months	☐ Refused to answer	☐ Cannot remember	☐ NA; didn't drink more on some days	

38. Did you have days when you drank more than _____ drinks?

☐ Yes ☐ No

39. If yes, how many did you usually drink then?

_____ drinks (prompt: 1–2 drinks, 3–4, 5–7, 8–10, more than 10)

☐ Refused to answer

☐ Cannot remember

☐ NA; did not drink more on other days

40. On the average, how often did you drink this amount?

☐ Every day	☐ Almost every day	☐ 3–4 times a week	☐ 1–2 times a week	☐ 2–3 times a month	☐ Once a month
☐ 1–2 times in 3 months	☐ Less than 1 time in 3 months	☐ Refused to answer	☐ Cannot remember	☐ NA; didn't drink more	

41. What beverage did you usually drink?

- ☐ Beer
- ☐ Wine
- ☐ Wine coolers or champagne
- ☐ Liquor/cocktails
- ☐ Any homemade alcoholic beverage. Describe: _____
- ☐ Other: _____
- ☐ NA; does not drink

42. How far along were you when you found out you were pregnant?

_____ weeks

☐ Cannot remember

Drinking Behaviour with Index Child

43. Once you knew you were pregnant, on the days that you drank alcohol, how many drinks did you usually drink?

_____ drinks (prompt: 1–2 drinks, 3–4, 5–7, 8–10, more than 10)

- ☐ Refused to answer
- ☐ Cannot remember
- ☐ NA; woman didn't drink during pregnancy

44. If you do drink, on the average, how often did you drink this amount?

☐ Every day	☐ Almost every day	☐ 3–4 times a week	☐ 1–2 times a week	☐ 2–3 times a month	☐ Once a month
☐ 1–2 times in 3 months	☐ Less than 1 time in 3 months	☐ Refused to answer	☐ Cannot remember	☐ NA; didn't drink	

45. Did you have days when you drank more than _____ drinks?

- ☐ Yes
- ☐ No
- ☐ NA; woman didn't drink during pregnancy

46. If yes, how many did you usually drink then?

_____ drinks (prompt: 1–2 drinks, 3–4, 5–7, 8–10, more than 10)

- ☐ Refused to answer
- ☐ Cannot remember
- ☐ NA; woman didn't drink during pregnancy

47. On the average, how often did you drink this amount?

☐ Every day	☐ Almost every day	☐ 3–4 times a week	☐ 1–2 times a week	☐ 2–3 times a month	☐ Once a month
☐ 1–2 times in 3 months	☐ Less than 1 time in 3 months	☐ Refused to answer	☐ Cannot remember	☐ NA; didn't drink more	

48. What beverage did you usually drink?

- ☐ Beer
- ☐ Wine
- ☐ Wine coolers or champagne
- ☐ Liquor/cocktails
- ☐ Any homemade alcoholic beverage. Describe: _____
- ☐ Other: _____
- ☐ NA; did not drink

49. During the *first three months* of your pregnancy, on the days that you drank alcohol, how many drinks did you usually drink?

_____ drinks (prompt: 1–2 drinks, 3–4, 5–7, 8–10, more than 10)

☐ Refused to answer

☐ Cannot remember

☐ NA; woman didn't drink during 1st trimester

50. If you did drink, on the average, how often did you drink this amount?

☐ Every day	☐ Almost every day	☐ 3–4 times a week	☐ 1–2 times a week	☐ 2–3 times a month	☐ Once a month
☐ 1–2 times in 3 months	☐ Less than 1 time in 3 months	☐ Refused to answer	☐ Cannot remember	☐ NA; didn't drink	

51. During the *middle three months* of your pregnancy, on the days that you drank alcohol, how many drinks did you usually drink?

_____ drinks (prompt: 1–2 drinks, 3–4, 5–7, 8–10, more than 10)

☐ Refused to answer

☐ Cannot remember

☐ NA; woman didn't drink during 2nd trimester

52. If you did drink, on the average, how often did you drink this amount?

☐ Every day	☐ Almost every day	☐ 3–4 times a week	☐ 1–2 times a week	☐ 2–3 times a month	☐ Once a month
☐ 1–2 times in 3 months	☐ Less than 1 time in 3 months	☐ Refused to answer	☐ Cannot remember	☐ NA; didn't drink	

53. During the *last three months* of your pregnancy, on the days that you drank alcohol, how many drinks did you usually drink?

_____ drinks (prompt: 1–2 drinks, 3–4, 5–7, 8–10, more than 10)

☐ Refused to answer

☐ Cannot remember

☐ NA; woman didn't drink during 3rd trimester

54. If you did drink, on the average, how often did you drink this amount?

☐ Every day	☐ Almost every day	☐ 3–4 times a week	☐ 1–2 times a week	☐ 2-3 times a month	☐ Once a month
☐ 1–2 times in 3 months	☐ Less than 1 time in 3 months	☐ Refused to answer	☐ Cannot remember	☐ NA; didn't drink more on some days	

Section D: Nutrition during Pregnancy[2]

**"Now I'm going to ask you some questions about your nutrition immediately before and during your pregnancy with (name of child)."*

(Select one response per question unless otherwise specified.)

55. Before you became pregnant did you take any vitamins or supplements?

☐ Yes

☐ No

☐ Don't know

☐ Refused to answer

If yes, specify what: _____

_____

2 This section was adapted from the CIFASD study.

56. While you were pregnant did you take any vitamins or supplements?

- ☐ Yes
- ☐ No
- ☐ Don't know
- ☐ Refused to answer

If yes, specify what and when (by trimester): _____

_____

*"*Disclaimer: I want to let you know that I will repeat the same answer choices for the following questions. This may sound repetitive, but it is to ensure that we capture the most specific and accurate answer possible. Feel free to stop me when an answer choice sounds most correct to you. The following questions are about your nutrition **during** your pregnancy with (name of child)."*

57. During your pregnancy, how often did you eat at least 3 or more servings of whole-grain products or high-fibre starches a day? A serving is 1 slice of 100% whole-grain bread, 1 whole-wheat tortilla, 1 cup of whole-grain cereal like shredded wheat, grape nuts, high-fibre cereals or oatmeal; 3–4 whole-grain crackers; 1/2 cup of brown rice or whole-wheat pasta; boiled or baked potatoes, yucca, yams or plantain.

- ☐ Refused to answer
- ☐ Every day
- ☐ 1–3 times/month
- ☐ 5–6 times/week
- ☐ Less than 1 time/month
- ☐ 2–4 times/week
- ☐ Never
- ☐ 1 time/week

58. How often did you eat at least 2–3 servings of fruit a day? A serving is 1/2 cup or 1 medium fruit (the size of a tennis ball) or 3/4 cup of 100% fruit juice.

- ☐ Refused to answer
- ☐ Every day
- ☐ 1–3 times/month
- ☐ 5–6 times/week
- ☐ Less than 1 time/month
- ☐ 2–4 times/week
- ☐ Never
- ☐ 1 time/week

59. How often did you eat at least 3–4 servings of vegetables a day? A serving is 1/2 cup of vegetables (1 cup is about the size of your fist) or 1 cup of leafy raw vegetables.

- ☐ Refused to answer
- ☐ Every day
- ☐ 1–3 times/month
- ☐ 5–6 times/week
- ☐ Less than 1 time/month
- ☐ 2–4 times/week
- ☐ Never
- ☐ 1 time/week

60. How often did you consume at least 2–3 servings of milk, yogurt, cheese or cottage cheese a day? A serving is 1 cup of milk or yogurt, or 1 1/2–2 ounces of cheese (1 ounce is about the size of 4 stacked dice.)

- ☐ Refused to answer
- ☐ Every day
- ☐ 1–3 times/month
- ☐ 5–6 times/week
- ☐ Less than 1 time/month
- ☐ 2–4 times/week
- ☐ Never
- ☐ 1 time/week

61. How often did you eat 5 ounces of meat, chicken, turkey, fish, eggs or beans? 3 ounces of meat or chicken is the size of a deck of cards OR 1 regular hamburger, 1 chicken breast or leg or one pork chop; and one egg or 1/2 cup of beans is 2 ounces.

- ☐ Refused to answer
- ☐ More than 1 time/day
- ☐ 1 time/week
- ☐ 1 time/day
- ☐ 1–3 times/month
- ☐ 5–6 times/week
- ☐ Less than 1 time/month
- ☐ 2–4 times/week
- ☐ Never

62. How often did you eat regular processed meats (like bologna, salami, hotdogs, sausage or bacon), not including low-fat processed meats (like roast beef, turkey, lean ham, low-fat cold cuts / hotdogs)?

- ☐ Refused to answer
- ☐ More than 1 time/day
- ☐ 1 time/week
- ☐ 1 time/day
- ☐ 1–3 times/month
- ☐ 5–6 times/week
- ☐ Less than 1 time/month
- ☐ 2–4 times/week
- ☐ Never

63. For how many meals did you eat fried foods such as fried chicken, fried fish, french fries, fried plantains or fried yucca?

- ☐ Refused to answer
- ☐ More than 1 meal/day
- ☐ 1 meal/week
- ☐ 1 meal/day
- ☐ 1–3 meals/month
- ☐ 5–6 meals/week
- ☐ Less than 1 meal/month
- ☐ 2–4 meals/week
- ☐ Never

64. How often did you eat sweets such as a slice of cake, 2 cookies, a pastry, a donut, a muffin or a candy bar more than 2 times per day?

- ☐ Refused to answer
- ☐ Every day
- ☐ 1–3 times/month
- ☐ 5–6 times/week
- ☐ Less than 1 time/month
- ☐ 2–4 times/week
- ☐ Never
- ☐ 1 time/week

65. How often did you drink 12 ounces or more of non-diet soda, fruit drink/punch or Kool-Aid? 1 can of soda is 12 ounces.

- ☐ Refused to answer
- ☐ More than 1 time/day
- ☐ 1 time/week
- ☐ 1 time/day
- ☐ 1–3 times/month
- ☐ 5–6 times/week
- ☐ Less than 1 time/month
- ☐ 2–4 times/week
- ☐ Never

66. How often did you get at least 30 total minutes of physical activity? (e.g., walking briskly, gardening, golf, jogging, swimming, biking, dancing)

- ☐ Refused to answer
- ☐ Every day
- ☐ 1–3 times/month
- ☐ 5–6 times/week
- ☐ Less than 1 time/month
- ☐ 2–4 times/week
- ☐ Never
- ☐ 1 time/week

Section E: Tobacco and Drug Use during Pregnancy[3]

**"Next, I'd like to ask you a few questions about your experiences with tobacco and drugs that were not prescribed for you or not taken as prescribed. Again, all your answers are confidential and we'd like you to be as honest as possible."*

(Select one response per question unless otherwise specified.)

67a. Have you ever smoked a cigarette or used other tobacco products?

- ☐ Yes, in the past
- ☐ Yes, currently **Go to question 67b*
- ☐ No, never **Go to question 70*

67b. How often do you currently smoke cigarettes or use other tobacco products?

- ☐ Every day
- ☐ Occasionally

3 This section was adapted from the Collaborative Initiative on Fetal Alcohol Spectrum Disorders (CIFASD) study.

68. Before you knew you were pregnant with (child's name), did you use cigarettes or other tobacco products?

- ☐ Every day*
- ☐ 2–3 times/months
- ☐ 5–6 times/week
- ☐ 1 time/month
- ☐ 3–4 times/week
- ☐ < 1 time/month
- ☐ 1–2 times/week
- ☐ Never
- ☐ Refused to answer

* Specify average number of cigarettes/day: _____

69. After you found out you were pregnant with (child's name), did you use cigarettes or other tobacco products?

- ☐ Refused to answer
- ☐ Every day*
- ☐ 2–3 times/month
- ☐ 5–6 times/week
- ☐ 1 time/month
- ☐ 3–4 times/week
- ☐ <1 time/month
- ☐ 1–2 times/week
- ☐ Never

* Specify average number of cigarettes/day: _____

70. Have you ever used any drugs (either legal or illegal) for recreational purposes?

- ☐ Yes **Go to question 71.*
- ☐ No **Stop here, proceed to end of questionnaire.*

71. Before you knew you were pregnant with (child's name), did you use...? **Read categories below:*

	Don't know	No	Yes	If yes, how often? Don't know	Every day	5–6 times/ week	3–4 times/ week	1–2 times/ week	2–3 times/ month	1 time/ month	< 1 time/ month
Marijuana or hashish	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Heroin or opium	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Crack/cocaine	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Methamphetamine/ amphetamine	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Club drugs (e.g., ecstasy, GHB, rohypnol)	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Dissociative drugs (e.g., PCP, ketamine, salvia, DXM [used in cough and cold medications])	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Hallucinogens (e.g., LSD, mushrooms, peyote [mescaline])	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Non-medical inhalants (e.g., gasoline, paint thinners, glue, nitrous oxide, whippets, poppers)	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Abuse prescription drugs (e.g., valium, Xanax, codeine, morphine, Vicodin, Lortab, Percocet)	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Anabolic steroids	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Any other drug or substance [Specify]:_____	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐

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72. After you found out you were pregnant with (child's name), did you use...? **Read categories below:*

				If yes, how often?							
	Don't know	No	Yes	Don't know	Every day	5–6 times/ week	3–4 times/ week	1–2 times/ week	2–3 times/ month	1 time/ month	< 1 time/ month
Marijuana or hashish	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Heroin or opium	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Crack/cocaine	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Methamphetamine/ amphetamine	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Club drugs (e.g., ecstasy, GHB, rohypnol)	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Dissociative drugs (e.g., PCP, ketamine, salvia, DXM [used in cough and cold medications])	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Hallucinogens (e.g., LSD, mushrooms, peyote [mescaline])	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Non-medical inhalants (e.g., gasoline, paint thinners, glue, nitrous oxide, whippets, poppers)	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Abuse prescription drugs (e.g., Valium, Xanax, codeine, morphine, Vicodin, Lortab, Percocet)	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Anabolic steroids	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Any other drug or substance [Specify]:_____	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐

*"This is the end of the interview. Do you have any questions that you would like to ask me, or any comments about this experience?"_____

_____

_____

*"Thank you so much for your assistance with this portion of the study. We greatly appreciate your co-operation."

End time: _____ a.m./p.m. (circle one)

Do Not Ask!

Interviewer Observations of the Participant

How confident do you feel about the validity of this participant's answers?

☐ Completely confident

☐ Some doubts

☐ No confidence

Were you able to complete the questionnaire?

☐ No ☐ Yes

Interviewer comments (If "No confidence" on above questions, please specify why. Also add any other comments on this interview.) _____

_____

_____

_____

Appendix E

Standard Drink Conversion Chart

One standard drink is equal to:

341 mL beer[1] (5% alcohol)	=	142 mL = wine[2] (12% alcohol)	=	85 mL fortified wine[3] (16%–18% alcohol)	=	43 mL liquor[4] (40% alcohol)

[1] Regular beers have an average alcohol content of 5%, but some have as much as 6% or 7%, making them stronger than a "standard" drink. "Light" beers have an average alcohol content of 4%.

[2] One bottle of wine (750 mL) contains approximately 5 standard drinks of alcohol.

[3] Such as sherry, port or vermouth.

[4] One bottle of liquor (500 mL) contains approximately 11 standard drinks of alcohol.

Appendix F

Final Diagnosis Form[1]

Based on the available information, please fill out the following criteria for diagnoses of:

- fetal alcohol syndrome (FAS) or
- partial fetal alcohol syndrome (pFAS) or
- alcohol-related neurodevelopmental disorder (ARND)

Criteria for the diagnosis of FAS, after excluding other diagnoses:

A. Evidence of prenatal or postnatal growth impairment in at least *one* of the following:

a.	Birth weight or birth length at or below the 10th percentile for gestational age	☐ Yes	☐ No	☐ Not available
b.	Height or weight at or below the 10th percentile for age	☐ Yes	☐ No	☐ Not available
c.	Disproportionately low weight-to-height ratio (equal to 10th percentile)	☐ Yes	☐ No	☐ Not available

Criteria for the diagnosis of FAS or pFAS after excluding other diagnoses:

B. Simultaneous presentation of all *three for FAS* or *two for pFAS* of the following facial anomalies at any age:

a.	Short palpebral fissure length (2 or more standard deviations below the mean)	☐ Yes	☐ No	☐ Not available
b.	Smooth or flattened philtrum (rank 4 or 5 on the lip-philtrum guide)	☐ Yes	☐ No	☐ Not available
c.	Thin upper lip (rank 4 or 5 on the lip-philtrum guide)	☐ Yes	☐ No	☐ Not available

1 Based on Chudley et al. (2005).

Criteria for the diagnosis of FAS, pFAS and ARND after excluding other diagnoses:

C. Evidence of impairment in *three or more* (non-overlapping) central nervous system domains:

☐ Yes (check off all that apply below) ☐ No ☐ Not available

☐ Hard and soft neurologic signs

☐ Brain structure

☐ Cognition

☐ Communication

☐ Academic achievement

☐ Memory

☐ Executive functioning and abstract reasoning

☐ Attention deficit/hyperactivity

☐ Adaptive behaviour, social skills, social communication

Criteria for the diagnosis of FAS (optional), pFAS and ARND after excluding other diagnoses:

D. Confirmed maternal alcohol exposure:

☐ Yes

☐ No

☐ Not available

State the final diagnosis of the child (place a check mark in the respective box) based on the available medical record and/or on your assessment.

FAS ☐ Yes ☐ No ☐ Deferred

pFAS ☐ Yes ☐ No ☐ Deferred

ARND ☐ Yes ☐ No ☐ Deferred

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