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Prevalence of Fetal Alcohol Spectrum Disorder: A Literature Review

Study Authors:

**Linda Burnside, PhD and Don Fuchs, PhD.
University of Manitoba**

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PREVALANCE OF FETAL ALCOHOL SPECTRUM DISORDER: A LITERATURE REVIEW

This review summarizes the research literature to date on the issues and challenges of estimating the prevalence of FASD, and presents the prevalence rates reported in several studies from a wide range of jurisdictions and populations. Estimating the prevalence of FASD is a daunting task, whether one is intent on determining the rate of the condition in the general population or with a specific population known to have a higher risk of FASD. There are arguments as to why both themes are important in understanding the rate of FASD, as one approach lends itself to describing the breadth of occurrence throughout the general population, while the other helps to describe the depth of occurrence as it pertains to vulnerable populations, which will be discussed more extensively in this review. In particular, the review focuses on the need for prevalence rates of FASD in child welfare child-in-care populations. This is a population at high risk for FASD due to the frequency that parental substance abuse brings families to the attention of child welfare systems, estimated to be from 40% to 80% of families involved with child welfare (Besinger, Garland, Litrownik, & Landsverk 1999; Curtis & McCullough, 1993; Department of Health and Human Services, 1999; Dore, Doris, & Wright, 1995; McNichol & Tash, 2001; Semidei, Radel, & Nolan, 2001; Young, Gardner, & Dennis, 1998). Further, children in child welfare care are a most vulnerable group due to the impact of prenatal alcohol exposure on children's functioning and the well documented adversities associated with growing up in care.

INTRODUCTION

Increasingly, prenatal alcohol exposure is being acknowledged as a leading cause of preventable developmental disabilities that have serious detrimental outcomes for individuals throughout the lifespan (Health Canada, 2000; Streissguth, Aase, Clarren, Randels, LaDue & Smith, 1991). Since 2004, Fetal Alcohol Spectrum Disorder (FASD) has been the umbrella term used to describe the range of conditions caused by alcohol-exposed pregnancies (Paley, 2009; Warren, et al., 2004), including Fetal Alcohol Syndrome (FAS), Fetal Alcohol Effects (FAE), partial FAS (pFAS), alcohol-related neurodevelopmental disorders (ARND) and alcohol-related birth defects (ARBD), the diagnostic categories defined by the Institute of Medicine (Hoyme, et al., 2005). Similar diagnostic criteria are employed by the Canadian Guidelines for Diagnosis of Fetal Alcohol Spectrum Disorder (Chudley, Conry, Cook, Loock, Rosales, & LeBlanc, 2005). With its specific dysmorphic anomalies of the face, FAS is often the easiest category of FASD to diagnose, and its manifestation of symptoms is often the most severe. At the opposite end of the continuum, ARND is less uniform in its manifestation and its symptoms are more variable, making it more difficult to diagnose, while diagnoses of ARBD are discouraged in Canada due to even greater variability in diagnostic uniformity (Chudley, et al., 2005).

It is recognized that FASD results in a wide range of impairments on a continuum from mild to severe, with considerable variation in the effect on individual functioning as a result of the complex teratogenic effects of alcohol (Abel & Hannigan, 1995; Barr & Streissguth, 2001; Coles, 1994; Guerri, 1998; O'Leary, 2002; Thomas, Warren & Hewitt, 2010; Uban, Bodnar, Butts, Sliwowska, Comeau, & Weinberg, 2011). Alcohol dose is one factor that plays a significant role in teratogenic effects (Jacobson, Jacobson & Sokol, 1996; O'Leary, 2002; Streissguth et al., 1994); stage of pregnancy is another important variable (O'Leary, 2002). The impact of FASD is also mediated by environmental factors, such as living with an alcoholic parent or being subjected to child abuse or neglect, or other adverse life conditions such as poverty (Streissguth, Bookstein, Barr, Sampson, O'Malley & Young, 2004).

This is a particularly relevant point as it pertains to indigenous populations, who frequently are affected by low socioeconomic status and the drinking patterns that are associated with coping with impoverished living conditions (Abel & Hannigan, 1995; O'Leary, 2003). Importantly, the literature distinguishes that there is no difference in alcohol teratogenesis between Aboriginal and non-Aboriginal populations, but that the differences in prevalence and the issues related to FASD are linked to the social and economic circumstances of oppressed mothers and children, many of whom are Aboriginal (Pacey, 2010).

The body of literature documenting the devastating impact of FASD as a result of the primary impact of alcohol as a teratogen on the developing fetus, and the secondary effects that are influenced by the interaction of environmental factors with the child's developmental disability, is building. Considerable research has examined the effect of FASD on individuals (for example, Brintell, Bailey, Sawhney, Krefting & Bhambhani, 2010; Green, 2007; Malbin, 2004; Zevenbergen & Ferraro, 2001), and on biological, adoptive, and foster families (for example, Brown, Sigvaldason, & Bednar, 2006; Caley, Winkelman, & Mariano, 2009; Jirkowic, Kartin & Olson, 2008; Olson, Oti, Gelo & Beck, 2009). The literature has also begun to document the social and financial costs to society (for example, Fuchs, Burnside, DeRiviere, Brownell, Marchenski, Mudry, & Dahl, 2009; Hutson, 2006; Lupton, Burd & Harwood, 2004; Stade, Ali, Bennett, Campbell, Johnston, Lens, Tran & Koren, 2009; Stade, Ungar, Stevens, Beyenne, & Koren, 2006). It is clear that FASD is a significant public health issue.

What remains clearly obscured, however, is that FASD is an epidemic of unknown proportions (Clarren & Lutke, 2008; Robinson, 1992). Despite growing awareness, it has been difficult to generate a clear picture of the actual prevalence of FASD. The limited number of studies that attempt to estimate the prevalence of FAS are often clouded by variable definitions and differing methodologies, resulting in prevalence rates that are contradictory and confusing, and most certainly an underestimation of its true occurrence (May & Gossage, 2001). Estimating the prevalence of other types of FASD (pFAS, FAE or ARND) is even more challenging due to greater variability in defining diagnostic symptoms, compared to the diagnosis of FAS. As noted by May et al. (2009), "although key diagnostic features of FAS are generally well established, the specific assessment techniques and statistical measurements used to make the definitive diagnosis of FAS and other FASD are still debated" (p. 176).

The debate around diagnosis is only one of the key variables contributing to the challenge in establishing prevalence rates. Clarren and Lutke (2008) assert that estimating the true prevalence of FAS/FASD is more complicated than for most other health conditions. Evidence of brain damage is often not evident until the child has reached the school-age years, making early diagnosis difficult, and there are fewer diagnostic resources available for youth and adults, often leaving their functional challenges undiagnosed. Other barriers in determining the occurrence of FASD include the difficulty in confirming maternal alcohol use during pregnancy (Thomas, Warren & Hewitt, 2010), reluctance to diagnose due to the stigmatization of the condition (Nesbit, Philpott, Jeffery & Cahill, 2004), a lack of training in making FASD diagnoses (Clarke, Tough, Hicks, & Clarren, 2005), belief that the condition can be treated effectively without the FASD label (Gardner, 1997) and the lack of diagnostic services in general (Elliott, Payne, Morris, Haan & Bower, 2008).

The challenges around estimating the prevalence of FASD in the population have a profound impact in many realms: program development, service delivery, policy creation, funding projections, staff training, and public awareness. How can a community, a province, a country – any jurisdiction for that matter – determine what this vulnerable population needs without knowing how many people are affected? But most importantly, when we are not able to consistently and accurately measure the

extent of FASD in society, individuals with FASD remain undiagnosed, or misdiagnosed, and either do not receive the interventions that they need or receive interventions that are unfitting for their needs. The psychological and social consequences for these individuals, and subsequently for all of society, are profound.

DIAGNOSIS OF FASD

One of the biggest challenges in establishing prevalence rates of FASD is ensuring that cases of FASD are detected and diagnosed (Warren & Foudin, 2001). The Canadian Guidelines for the Diagnosis of FASD (Chudley et al., 2005) propose diagnostic criteria for the determination of FAS, pFAS, and ARND. The absence of any other diagnosis and evidence of prenatal alcohol exposure are foundational criteria required for the diagnosis of any FASD. FAS is characterized by a) a distinct dysmorphology, including facial features such as a flattened area between the upper lip and nose, thin upper lip, epicanthal folds, and a narrow palpebral fissure (the length of space between the margins of the eyelids); b) a constellation of central nervous system effects; and c) evidence of significant pre- or post-natal growth impairment (less than the 10th percentile). Partial FAS is characterized by the same criteria, with fewer facial anomalies and no growth impairment. ARND is diagnosed on the basis of three central nervous system effects, without the facial dysmorphology or growth impairment. With ARBD covering an even broader range of alcohol-effect conditions than ARND, the Canadian Guidelines suggest using the diagnosis of ARBD with caution due to the challenges in defining clear diagnostic criteria.

The Canadian Guidelines represent a set of diagnostic criteria that is harmonized with the four-digit diagnostic codes developed by Astley and Clarren (2000) to assist clinicians in the United States make more consistent diagnoses of FASD. Each of the four digits corresponds to a specific diagnostic component of the more traditional approach to FASD diagnosis, in order: (1) growth deficiency; (2) FAS dysmorphic features; (3) brain damage/dysfunction; and (4) prenatal alcohol exposure. Each component is also individually ranked according to degree, with “1” representing an absence of that component and “4” representing the classic FAS expression of that component, resulting in a possible 256 codes ranging from 1111 to 4444. One of the unique features of this approach is that in addition to the diagnostic outcome, the code documents how much information about maternal alcohol consumption during pregnancy was available to support the diagnosis. Additionally, the code system documents the presence of prenatal alcohol exposure without judging its causal role, recognizing that there may be other contributing factors (Health Canada, 2000).

However, in identifying that the relationship between the physical manifestation of prenatal alcohol exposure and functionality is unclear (characterized as primary and secondary disabilities), Chasnoff (2011) argues that the 4-digit code model may be too cumbersome for use outside of comprehensive diagnostic clinics. Nanson (2003) previously described the four-digit code as useful to diagnosticians but of limited use to clinicians, who find it necessary to convert the code back into familiar terms like FAS in order to obtain services for children. Further, she notes that the wide range of possible four-digit code outcomes “tends to obfuscate rather than enhance diagnostic clarity” (p. 4). Instead, Chasnoff (2011) proposes an alternative diagnostic approach:

Children with documented prenatal alcohol exposure who meet all physical criteria for growth impairment and facial dysmorphology as well as neurodevelopmental deficits would be assigned a diagnosis of FAS. Children with documented prenatal alcohol exposure who do not meet all growth and/or facial criteria but who meet

criteria for neurodevelopmental deficits would be classified as ARND. Utilizing two diagnoses – FAS and ARND – with strict criteria delineated for all to follow would create a common language and diagnostic schema that would be suitable in the clinical setting while establishing a consistency with ongoing research. (p. 4)

As identified by May et al. (2009) and Nanson (2003), concluding the debate on diagnostic criteria is imperative to establishing a baseline estimation of FASD with some degree of confidence. Without this resolution, prevalence rates will be vulnerable to a degree of ambiguity and limited comparability across studies.

Diagnostic Challenges

Even with standardized diagnostic criteria, it is important to determine who in the population should be assessed for possible FASD. While classic FAS is indicated by distinctive facial features, these features may not be noticeable until after the child has reached the age of two years (Larkby & Day, 1997), and may be less evident once the child has reached adolescence (Clarren & Lutke, 2008; Larkby & Day, 1997; Streissguth, et al., 1991). Additionally, there may be variations in facial features that have cultural origins, with no relationship whatsoever to prenatal alcohol exposure, which complicates accurate diagnosis of FAS (Aase, 1994).

Further, according to Chudley (2008), since children with ARND do not have facial dysmorphism, they are the least visible and therefore the least likely to be identified as having a condition related to prenatal alcohol exposure. Many receive a wide range of different diagnoses, obscuring the prevalence of FASD (Chudley, 2008; Thomas, Warren & Hewitt, 2010). This has a profound impact on determining the prevalence of FASD, as Chudley estimates that “for every child with full blown FAS, there are three or four who have ARND” (p. 721). Conversely, Pacey (2009) cautions that ARND may be subject to over-diagnosis because the effects of ARND are not as specific as FAS.

Another approach in identifying children who have been exposed to alcohol prenatally and may be at risk of having FASD is to screen women during pregnancy to detect those who are using alcohol during pregnancy (Warren & Foudin, 2001). While this strategy may flag those children who require follow-up assessment, it is not a foolproof strategy for confirming the existence of FASD. Abel (1995) estimated that only 4.3% of heavy consumers of alcohol during pregnancy will give birth to a child with FAS, due to the complex interaction of variables that seem to affect its manifestation. How many women will give birth to a child affected by other conditions under the FASD umbrella (e.g. ARND) is not known. Therefore, defining the risk factors that increase the likelihood of giving birth to a child affected by FASD is an important area of study (Warren & Foudin, 2001).

Biological Markers of Prenatal Alcohol Exposure

Testing for biological markers that confirm prenatal alcohol exposure has been identified as a possible screening tool for FASD, one that is especially important when maternal drinking patterns prenatally may not be known (Thomas, Warren & Hewitt, 2010). At birth, meconium (the infant’s first stool) has been found to contain the biological record in fatty acid ethyl esters of the total quantity of prenatal alcohol exposure in the last half of pregnancy (Bearer, Lee, Salvator, Minnes, Swick, Singer, et al., 1999; Gareri, Lynn, Handley, Rao, & Koren, 2008; Goh, Chudley, Clarren, Koren, Orrbines, Rosales & Rosenbaum, 2008). Administered as an anonymous universal screening tool, it facilitates inclusion of all children born in a particular testing facility or region and is considered to be more accurate at identifying

alcohol-exposed children than maternal self-report screening methods (discussed below). In the study conducted by Gareri et al., (2008), a prevalence rate of alcohol-exposed births of 2.5% - 3.5% was reported. However, further diagnostic testing of the children themselves would be required to determine an actual diagnosis of FASD and aid in establishing an accurate prevalence rate. Additionally, children who are identified through this screening approach should be offered early intervention services to mitigate the detrimental effects of prenatal alcohol exposure.

As noted earlier, classic FAS is indicated by specific facial abnormalities, features which have been documented in the literature for some time (Clarren, 1981). One of the most distinctive features of FAS is a shortened palpebral fissure, defined in general terms as the horizontal length of the eye opening; in specific clinical terms, “the horizontal distance between the endocanthion and the exocanthion” (Clarren, Chudley, Wong, Friesen, & Brant, 2010, p. e68). While various studies have demonstrated that palpebral fissure length does vary across cultures, an emerging approach to assessing biological markers is through measurement of palpebral fissures (Clarren, et al., 2010). Research by these authors aimed to establish normative values for palpebral fissure length by measuring the palpebral fissures of 2097 Canadian children of diverse cultural heritage in Vancouver and Winnipeg. Consistent with previous studies, they found that palpebral fissures continue to grow with age until later adolescence, that boys have slightly larger palpebral fissures at all ages, and that differences in palpebral fissure length attributable to culture were consistently close enough to establish normative data to be used as the single standard for all school age children in Canada, and potentially elsewhere.

Universal Screening

Identification of FASD may also be achieved through the screening of women who are consuming alcohol during pregnancy (Thomas, Warren & Hewitt, 2010). Universal screening methods for FASD generally focus on screening when members of the population access normative services, such as health care services where physicians can detect and deter alcohol use by pregnant women and identify affected children at an early age (Hicks, Sauve, Lyon, Clarke, & Tough, 2003). Since indicators of FASD are rarely present in newborn children, self-report screening tools administered with pregnant women during prenatal care services and after delivery have often been utilized (Barr & Streissguth, 2001). While screening is not the same as diagnosis (Goh, et al., 2008), screening can identify children who may have been exposed to alcohol prenatally who may require follow-up in the future for assessment of possible FASD should they show other indicators, which may contribute to establishing a prevalence rate over time (Schoellhorn & Podvin, 2002). Screening tools for FASD are non-invasive and can result in earlier intervention for children who diagnosed (Goh, et al., 2008).

However, universal screening of pregnant women may underestimate the rate of FASD, as women who are most at risk for having an alcohol-exposed child often do not access prenatal care (Williams, Odaibo, & McGee, 1999). Research has also suggested that many health care providers are uncomfortable asking women about their alcohol use (Whaley & O'Connor, 1999). Finally, screening tools need to be “universal” – standardized across jurisdictions and become part of the normal course of care in order to be truly effective in identifying individuals at risk (Hicks et al., 2003). Burd, Klug, Martsof, Martsof, Deal, & Kerbeshian (2006) propose a staged screening protocol, beginning with the identification of prenatal alcohol exposure, pursuing a more detailed exposure assessment for risk stratification, and an examination of maternal risk factors, toward this end.

Screening with High Risk Populations

Research on the efficacy of screening tools has often focused on populations where risky substance misuse is a known issue. Consequently, higher rates of concerning alcohol use during pregnancy are expected. For example, Elliott and Hanson (2006) report that studies of screening tools with American Indian women in South Dakota at their first prenatal care appointment have found up to 56% of respondents reporting that they had consumed alcohol while pregnant. While this information can point to the need for intensive prevention strategies with this population, the authors note that focusing screening tools on high risk populations can perpetuate the myth that FAS is a problem affecting indigenous populations only.

However, screening with high risk populations can be helpful in understanding the extent and depth of the issue with a population that is susceptible to the condition's manifestation due to drinking patterns that escalate the risk of FASD's occurrence. As noted briefly in the introduction to this literature review, children in care may be considered a high risk population due to the frequency that parental substance abuse brings families to the attention of child welfare. Given the immense responsibility child welfare agencies have for children in care, understanding the prevalence of FASD affecting this vulnerable population is a necessity in order to meet their needs most appropriately.

Screening in Specific Settings

While not necessarily intended as a strategy for gathering evidence of the prevalence of FASD, there is increasing recognition that individuals who may have FASD (particularly children) may be identified through their involvement in normative social systems such as the education system. Of course, the main purpose for identifying children with signs of FASD in school is the opportunity for early intervention to ameliorate its deleterious effects as much as possible. Many educators have endorsed efforts to raise awareness of FASD with teachers, providing them with training to recognize indicators of FASD, refer children for diagnostic assessment, and adapt teaching approaches to best meet the learning needs of affected children (Blackburn & Whitehurst, 2010; Olson, Jirikowic, Kartin & Astley, 2007). Given that indicators of FASD may not be present until children reach school-age, schools are an important setting to include in screening strategies.

There is ample evidence in the literature that a disproportionate number of youth and adults with FASD are involved with the criminal justice system (Fast, Conry, & Loock, 1999; Goh et al., 2008). Screening of youth who are involved with the youth criminal justice system may aid in identifying youth who had not previously been diagnosed with FASD. Strategies such as the FASD Youth Justice Project in Manitoba conduct screening of youth age 12 – 18 who are undergoing pre-sentencing, with those who have confirmed prenatal alcohol exposure referred for a diagnostic assessment. A similar screening tactic is used in Vancouver with the Asante Centre Fetal Alcohol Syndrome Probation Officer Screening & Referral Form for all youth with probation orders who are suspected of having FASD. In their study of youth offenders screened after being remanded to a psychiatric and psychological assessment unit, Fast, Conry and Loock (1999) found that 1% of the youth had FAS and 23% had FASD (including FAS).

Conclusion

It is likely that multiple screening strategies are the most comprehensive approach to identifying individuals who may have been exposed to alcohol prenatally, as no single screening tool is necessarily suitable for all ages, groups or cultures (Goh, et al., 2008). With comprehensive screening, individuals

who may be affected by FASD can be identified and referred for assessment and diagnosis, and although good screening will screen in some individuals who will be determined not to have FASD, it is an approach that can contribute to early diagnosis and intervention and lead to more accurate prevalence rates over time.

However, even with comprehensive screening, the diagnosis of FASD may also present barriers of a practical nature. Not every community in Canada is near a diagnostic clinic, so even when children are suspected of having functional challenges related to prenatal alcohol exposure as a result of screening strategies, there may not be services available to assess for FASD, particularly in rural and remote regions of Canada (Clarren & Lutke, 2008). Some provinces, such as Saskatchewan, have endeavoured to overcome this barrier by introducing travelling FASD diagnostic teams consisting of a pediatrician, psychologist, social worker, physiotherapist, occupational therapist, and speech therapist (Health Canada, 2000). The challenges of screening and diagnosing adults who are affected by FASD are even greater than for identifying children at risk of FASD, challenges which are exacerbated in rural and remote communities.

Consequently, a more practical approach may be to focus screening in high risk communities, where high alcohol consumption is a known concern, and with high risk populations who, often as a result of FASD are involved with intervention systems such as child welfare or the criminal justice system. As described above, there are disadvantages to this approach, especially in creating the perception that only certain groups are vulnerable to prenatal alcohol exposure disabilities. The costs and social advantages of universal screening should therefore be compared with the costs and benefits of screening identified high risk groups to determine the most efficient and effective approach (Goh et al., 2008).

PREVALENCE

Prevalence refers to the rate of a condition (in this case, FASD) within a population, capturing both new and existing cases during a particular time period, across all age ranges (May & Gossage, 2001). Generally, only children with the most severe expression of FASD (FAS and partial FAS) are the focus of most prevalence studies, as these are the conditions that are easiest to diagnose with some degree of consistency in diagnostic criteria. Despite the growing body of literature on FASD as a condition that merits greater understanding and appropriate intervention by schools, health care systems, and social services, most of the professional literature acknowledges that the actual prevalence of FASD is still unknown. Reported rates of FAS/FASD in the literature vary greatly:

- 0.58 per 1,000 prevalence of FAS in a retrospective study of an Aboriginal population in Saskatchewan (Habbick, Nanson, Snyder, Casey & Schulman, 1996);
- 9.1 per 1,000 prevalence of FASD, based on data from a Seattle population of children (Sampson, et al., 1997);
- 1 -3 per 1,000 live births in Canada for FAS, with estimated prevalence of FASD approximately 10 times higher (Health Canada, 2000);
- 46 per 1,000 prevalence of FASD of Aboriginal children in the Yukon, and 25 per 1,000 for children in northern British Columbia (Asante & Nelms-Matzke, 1985).

It is accepted that FASD is under-diagnosed around the world. The prevalence of FASD worldwide is estimated at 0.33 per 1,000 (Nesbit, Philpott, Jeffery, & Cahill, 2004), also viewed to be an under-estimation. Although current prevalence statistics are considered to be conservative, there are no prevalence benchmarks currently set that serve as a valid starting point. As noted above, one of the key factors behind under-diagnosis is the difficulty in making a comprehensive and accurate diagnosis, usually because of the inability to confirm maternal substance misuse (Aase, 1994). Under-reporting of FASD may also occur, often because practitioners want to protect patients from the stigmatization of the condition (Smith & Rosales, 1998) since the diagnosis offers no prescribed treatment to address its effects (Nesbit, et al., 2004). Retrospective studies, such as the one by Habbick et al. (1996) cited above that found no change in FAS rates in Saskatchewan after 20 years, may not have been stringent enough in its earlier assessment of the facial anomalies consistent with the presentation of FAS (O'Leary, 2002).

Prevalence Rates Around the World

Although studies of the prevalence of FAS around the world vary greatly in their methodology (among other limitations affecting comparability, according to May, et al., 2009), they do provide information about the growing international concern about FASD and the importance of understanding its prevalence. Examples of studies include:

- Australia:** Bower, Silva & Henderson (2000) found an incidence rate of 0.02 per 1,000 live births in non-indigenous children, and 2.76 per 1,000 live births in indigenous children in Western Australia. A prevalence study in the Northern Territory by Harris and Bucens (2003) reported 1.7 per 1,000 indigenous live births for FAS. In 2007, a study in Queensland found a prevalence of 1.5 per 1,000 among aboriginal children (Rothstein, Heazlewood, & Fraser).
- Japan:** Based on the finding that one-third of Japanese children of women with alcohol addiction had been exposed to alcohol prenatally, Suzuki, Morita, Muraoka, and Niimi (2005) estimate that 10% had suspected FASD abnormalities.
- Norway:** Elgen, Bruaroy and Laegreid found in their 2007 survey of all Norwegian pediatric and child psychiatry clinics a national prevalence rate of 0.3 per 1,000.
- Sweden:** A community study examining the causes of mild mental retardation in schools in a Swedish city reported an FAS prevalence rate of 0.45 per 1,000 (Hagberg, Hagberg, Lewerth, & Lindberg, 1981).
- South Africa:** A 2007 report by May, et al. reported high rates of FAS and pFAS combined at 72.1 per 1,000 in a third review of a sample of first grade children.
- Italy:** A similar study of first grade students in Italy reported a prevalence rate of 3.7 – 7.4 per 1,000 for FAS (May, et al., 2006).
- Russia:** Riley, et al. (2003) found in screening children in special boarding schools for individuals with mental retardation a prevalence rate of FAS of 7.0 per 1000.
- France:** In a comparison of prevalence rates of FAS between the United States and France by Sampson et al. (1997), the rate of severe FAS was 1.3 per 1,000 live births and 4.8 per 1,000 for all types of FASD combined.

United States - Alaska: Egeland, Perham-Hester, Gessner, Ingle, Bemer and Middaugh (1998) recruited cases that met the FAS diagnostic criteria from a range of health care services from 1977 to 1992, reporting a prevalence rate of 3.0 per 1,000 for the indigenous population and 0.2 per 1,000 for the non-indigenous population.

Canada: Prevalence studies in Canada are few in number and limited in scope (Tough & Jack, 2011). Where provincial prevalence rates are reported, they are dated (all prior to 1994) or based on estimates. Only provinces in western and central Canada have published statistics on prevalence.

Perhaps as the antecedent to future FASD prevalence research, many countries are examining their rates of alcohol consumption by pregnant women, recognizing the risks associated between prenatal alcohol use and the occurrence of FASD. They include studies from Sweden (Nilsen, Holmqvist, Hultgren, Bendtsen & Cedergren, 2008), Finland (Autti-Ramo, Fagerlund, Ervalahti, Loimu, Korkman & Hoyme, 2005), Korea (Kim & Park, 2011), and Taiwan (Yen, Yang, Lai, Chen, Yeh & Wang, 2011). In Canada, alcohol consumption rates during pregnancy are available for each province and territory (Public Health Agency of Canada, 2008; Tough & Jack, 2011). Conversely, a recent study by Miller, et al. (2006) in Russia assessed children for the facial dysmorphology of FAS, reporting that 70% of children with high scores for the facial features were assessed as moderately or severely delayed, but they were unable to obtain reliable histories of maternal alcohol use during pregnancy, despite recognition that alcohol use in Russia is among the highest in the world.

The Context of Prevalence

Various studies have examined drinking patterns, particularly around young women of child-bearing age, to discern which groups might pose a higher risk of producing alcohol-exposed children. Such strategies help to inform the development of prevention strategies, particularly public awareness campaigns that aim to inform women about the dangers of drinking alcohol during pregnancy (Health Canada 2000). With increasing data to suggest that young women are drinking more alcohol than in previous generations, particularly engaging in binge drinking (for example, Autti-Ramo, Fagerlund, Ervalahti, Loimu, Korkman & Hoyme, 2005; Goransson, Magnusson, Bergman, Rydberg, & Heilig, 2003; Health Canada, 2000), and that at least half of all pregnancies are unplanned (Elliott, et al., 2008; Nanson, 1997), understanding alcohol consumption rates can be an important component of understanding prevalence rates.

However, May and Gossage (2001) note that there is great variability in drinking patterns around the world, with per capita consumption of alcohol much higher in many European countries than in the United States. Paradoxically, the reported rate of FAS in the United States is much higher than that reported in European studies, a reflection of the American approach to prevalence research (focusing on high risk populations) and the fact that the United States has both more abstainers and more heavy drinkers than many other countries. They state:

Therefore, it is not the prevalence of all drinkers or the amounts that they drink over a long period of time in European countries, but rather the proportion of drinkers who consume substantially large quantities in a short period of time that elevates the frequency of occurrence of the major and most severe FAS symptoms, which make up the diagnosis of FAS. (p. 163)

As it pertains to Canada in particular, Philp (2000) speculates that “there are pockets in the country – destitute neighbourhoods and remote alcohol-plagued native reserves – where estimates peg the incidence at nearly one in five” (p. A16). Reported statistics of FASD in First Nations populations include 10.9 per 1,000 in the Yukon and British Columbia (Sandor, Smith, MacLeod, Tredwell, Wood & Newman, 1981), 190 per 1,000 on a British Columbia reserve (Robinson, Conroy, & Conroy, 1987), and 7.2 per 1,000 incidence rate in Thompson, Manitoba (Williams, Odaibo & McGee, 1999). Prevalence statistics stemming from First Nations communities have perpetuated the myth that FASD is an Aboriginal problem (Van Bibber, 1993).

However, the viewpoint that the prevalence of FASD is connected to a set of social circumstances, particularly poverty and high alcohol consumption such as binge drinking (conditions which plague too many First Nations reserves) is gaining recognition. Larkby and Day (1997) assert that women with alcohol addiction often experience additional problems which exacerbate substance misuse (such as unstable marriages, partners with substance abuse, and the challenges of parenting as a single parent). Women at high risk of substance use during pregnancy also appear to have more children than other women, which can affect the FASD prevalence rate when deriving statistics from a small population where FASD has already been found (Abel, 1995).

Other studies have focused on the identification of other maternal risk factors that appear to have an influence on the occurrence of FAS due to their association with binge drinking. These factors include use of other drugs including tobacco, a transient lifestyle, unemployment, early age onset of regular drinking, alcohol misuse in the family, tenuous marital status, and community tolerance for heavy drinking (Coyne, De Costa, Heazlewood & Newman, 2008; Elliott & Hanson, 2006; Gladstone, Levy, Nulman & Koren, 1997; May et al., 2008). Jacobson, Jacobson and Sokol (1996) found that maternal age (in particular, being older than age 25) appears to be a risk factor. More recently, May (2011) summarized what is known about cofactors that affect the prevalence and severity of FASD: maternal nutrition, drinking patterns that vary due to differences across cultural groups, maternal older age, having more pregnancies, and genetic factors. As discussed earlier in this paper, universal screening programs are recommended as a means to identify maternal alcohol use during pregnancy as well as children to follow for diagnosis and early intervention (Hicks, Sauve, Lyon, Clarke & Tough, 2003); recognition of maternal risk factors can assist in determining where to focus screening efforts and what factors to include in screening tools.

A further challenge in identifying children who should be screened for FASD assessment is that some children who have been alcohol-exposed may not present with noticeable impairments in their functioning. May et al. (2009) found that some children who had been diagnosed as a result of thorough screening processes did not present with the severe physical, behavioural and intellectual deficits that most often lead to detection and referral for assessment. While some children who are alcohol-affected may function well enough to avoid detection, the authors argue that undetected and undiagnosed children may have significant learning disabilities or diminished functioning in other academic or social areas and, without intervention, may not reach their full potential. May et al. (2009) argue that population-level prevalence studies are necessary to provide a more complete picture of FASD, including the wide breadth of symptoms experienced by individuals throughout the spectrum.

Efforts to Estimate Prevalence

In their examination of prevalence studies, May and Gossage (2001) describe three main approaches: passive surveillance studies, clinic-based studies, and active case/population-based studies.

Varying methodologies make it difficult to compare prevalence rates across studies or even determine which study might approximate the actual occurrence of FAS (May & Gossage, 2001). Further, estimating prevalence is compromised by inconsistent diagnostic criteria, definitions, and diagnostic assessment processes used across studies, affecting which cases were included or excluded in a given prevalence query (Pacey, 2009).

Even with consistent diagnostic criteria, confirming maternal alcohol consumption during pregnancy can be difficult. Due to the significant stigmatization of women who drink during pregnancy, mothers may not readily acknowledge exposing their children to substances prenatally. Consequently, FASD is prone to under-counting (Pacey, 2009). Prevalence rates may also vary with age (Elliott et al., 2008), given that the capacity for FASD diagnosis is relatively recent and has focused mainly on children and adolescents; the extent of FASD in the adult population is more unknown.

Common Approaches to Estimating the Prevalence of FASD

May and Gossage (2001) originally provided a critique of the most common approaches used to estimate the prevalence of FASD: passive surveillance systems, clinic-based studies, and active case ascertainment methods. Their review was updated in 2009 (May, Gossage, Kalberg, Robinson, Buckley, Manning & Hoyme, 2009). They found that the highest rates of FAS were reported by clinic-based and active case ascertainment approaches, especially the latter which has tended to study high risk populations. May et al. (2009, p. 180) provide an excellent summary in a table format of key prevalence studies conducted via one of these three methods.

1) Passive Surveillance Systems

This approach utilizes existing records in a particular geographic area or with a particular population. Typical records reviewed under this approach include birth certificates, registries of children with disabilities or birth defects, and medical records from diagnostic clinics or hospitals. Assuming that a common definition and diagnostic approach to the confirmation of FASD can be established, this approach can be advantageous, as it accesses existing records, making data collection relatively inexpensive and time efficient.

However, many databases were not set up to track the occurrence of FASD, especially historical records dating back 10 or more years. Depending on the nature of the service, individuals with FASD may appear on more than one type of database (resulting in multiple counts of the same person, inflating the prevalence rate). Conversely, individuals with FASD may not get captured at all if they did not use a particular service whose database is reviewed (Clarren & Lutke, 2008). FASD is a complex condition to diagnose, requiring multiple indicators which are often not identifiable at birth. When FASD co-occurs with another condition, it is often the condition that is easier to diagnose (e.g. Down syndrome) that is recorded as the primary disability; secondary disabilities may not be noted at all. Finally, passive systems are vulnerable to the limitations of the original database, such as incomplete or inconsistent records, and data entry errors.

Examples of prevalence rates of FASD obtained from passive surveillance methods include:

- The Centers for Disease Control and Prevention (CDC) published three prevalence studies of birth anomalies in the United States, including FAS, using the Birth Defect Monitoring Program (BDMP) that tracks all hospital discharges after birth (May et al., 2009). Rates have

increased at each of the three report periods, although this may be due to better reporting. Still, the rate range from 0.09 – 2.9 per 1,000 from 1981 – 1986 is lower than the rates produced by any other method.

- A frequently cited report by Burd, Martsolf and Klug (1996) of birth records in North Dakota identified a prevalence rate of FAS as 1.1 – 2.0 per 1,000.

2) Clinic –Based Studies

Clinic-based studies on the prevalence of FASD are conducted in health care facilities (e.g. hospitals, prenatal clinics) and focus on collecting information about mothers' prenatal health and maternal behaviours (e.g. substance use during pregnancy) using screening questionnaires. The most common method of estimating FASD prevalence, a key advantage of this approach is the opportunity to gather detailed information from women throughout their pregnancies and/or after birth in a standardized way. It is also possible to identify control or comparison groups to analyze differences between jurisdictions or within a clinic's own population.

When the subjects of the research (pregnant women) all attend the same kind of service, clinic-based studies can allow for a great degree of consistency in gathering data. However, the women at highest risk for giving birth to a child with FASD are the least likely to access prenatal services and therefore may be absent from prevalence rates using this method. Additionally, although FAS may be diagnosed at birth (only possible with the most severe symptomology), the alcohol-exposure conditions under the FASD umbrella are often not identified until the child is between 3 and 12 years of age when deficits in functioning may be first recognized.

Well known examples of clinic-based prevalence studies include a series of studies led by Abel and Sokol (1987; 1991) and Abel (1995) and an often cited study by Sampson et al. (1997):

- Abel and Sokol reviewed 18 clinic-based studies in 1987, and 20 in 1991 (including some that were part of their original review). Abel continued this work in 1995 with 35 clinic-based studies from 40 different sites in the Western world. Prevalence rates differed due to the fact that many of the clinics outside of North America tracked populations that were mainly middle class and Caucasian (May et al., 2009), reporting few FAS cases resulting in a low prevalence rate (0.97 per 1,000 for the Western world and 1.95 per 1,000 for the US). Abel's analysis (1995) concluded that low socioeconomic status was a distinguishing feature between the European and American populations, reporting that FAS occurred at a rate of 4.3 per 1,000 when the population was heavy drinkers.
- A longitudinal study by Sampson et al. (1997) based on the clinical data gathered in Seattle (Streissguth, Aase, Clarren, Randels, LaDue & Smith, 1991; Streissguth et al., 1994) estimated the rate of FAS and ARND combined to be 9.1 per 1,000, a figure that is often quoted and still considered to be a conservative rate.

3) Active Case Ascertainment Methods

Active case ascertainment models focus on small, specific populations or groups, often those who are considered at high risk for prenatal alcohol exposure, and assess for characteristics of FASD. This approach tends to yield the highest number of cases and rates of FAS for a particular population.

High risk groups are often those most affected by poverty and adverse life circumstances, often resulting in a higher representation of indigenous populations in FAS prevalence reports.

However, active case ascertainment approaches are time and labour intensive and, as a result, can be costly. Engagement of sample populations is often sensitive and time-consuming, due to the vulnerability of the high risk populations that are usually sought in active case ascertainment methods. Cooperation from many sectors (families, service providers, diagnosticians, community members, etc.) are required in order to encourage participation in the research, and are more difficult to ascertain with a highly stigmatized topic like FASD and maternal drinking.

Examples of this kind study include:

- Robinson, Conry and Conry (1987) examined every child living in an isolated British Columbia reserve (102 children), and found a prevalence rate of 190 per 1,000.
- May (1991) examined the rates of FAS in three Native American groups (Navajo, Pueblo, and Southwest Plains) from 1969-1977 and from 1978 – 1982, reporting high variation in prevalence rates across the populations (1.0, 1.3 and 17.5 per 1,000 respectively).

However, active ascertainment models focusing on high risk populations with high alcohol consumption are prone to finding higher levels of FASD (which may not be applicable to lower risk groups where alcohol consumption is not as high). The high prevalence rates associated with a particular population can also be misinterpreted to mean that FASD is specific to that cultural group, rather than recognizing the characteristics of poverty, oppression, adverse life experiences, and high alcohol use as the main factors.

With increased awareness of FASD and technological reporting advances, active case ascertainment studies have attempted to identify new cases of FASD within a broader general population. The establishment of the International Network of Paediatric Surveillance Units in 1998, involving 14 countries, has led to a number of national active surveillance studies by Paediatric Surveillance Units (PSUs) on a wide range of children's health conditions, including studies on the prevalence of FAS in Australia and New Zealand (Grenier, et al., 2006). In this model, a report card listing health conditions under surveillance is sent to practicing pediatricians each month, to be returned (or entered online) for each child for whom a specific diagnosis has been made. Prevalence rates are calculated from these responses.

An example of a recent prospective study using a larger population surveillance sample:

- Examining data of newly diagnosed cases of FAS from the Australian PSU, Elliott, Payne, Morris, & Haan (2008) found a prevalence rate of 0.58 for children under age 15. A higher prevalence rate of 1.14 was found for children under age 5. Considering this to be an under-representation of actual prevalence, the authors note that many physicians fail to routinely ask about alcohol use in pregnancy, which is necessary for FASD diagnosis, and that people living in remote, rural areas of Australia (especially Indigenous people) may not have access to a pediatrician where diagnoses might be made and reported.

Some active case ascertainment studies are also reported in the grey literature. For example, Cox (2007) assessed the prevalence of FASD in a Maritimes First Nation community by having classroom teachers use a Medicine Wheel Index Screening Tool to identify children who should be evaluated for possible FASD. Children who were considered by their teachers to have significant difficulties in more than one domain (cognitive, social, emotional and physical) and whose parents were identified in semi-structured interviews about their children's developmental history as well as about adverse life experiences (losses, abuse, and substance use by parents), were referred to a local diagnostic clinic. Children were tested between January 1998 and June 1999, resulting in a total of 36 children being diagnosed with a prenatal alcohol-exposure disability. The prevalence rate for this sample population was 37 per 1,000 for FAS, and 193 per 1,000 for FAS and other conditions under the FASD umbrella.

Conclusion

Each approach to estimating prevalence discussed above has its strengths and limitations, which must be understood to appreciate the interpretation of reported prevalence rates. To date, no universally accepted methodology has been identified that would help to determine the actual rate of FAS. May and Gossage (2001) acknowledge that simply calculating an average across all the various prevalence studies is a tempting but flawed way to estimate the true rate of prevalence. With current studies, researchers must assess the demographic and social characteristics of each population under study to estimate how representative the findings may be to the general population.

To estimate the prevalence of the broader range of prenatal alcohol-exposure disabilities (all that fall under the FASD umbrella, not just the more narrowly defined FAS) is even more challenging. Based on the many studies reviewed by May and Gossage (2001), and in recognition of the various limitations of each study, they estimate that FAS in the general population of the United States to be between 0.5 and 2 per 1,000 births. The prevalence of FAS and other conditions under the spectrum is "likely to be at least 10 per 1,000, or 1 % of all births. This rate is too high for any population to accept" (p. 166).

FASD Prevalence and Special Populations

FASD and First Nations Populations

Pacey (2009) is particularly critical of group-specific active case ascertainment studies, citing a number of cautions that can lead to erroneous assumptions about actual prevalence. In general, population-based studies conducted with small groups are prone to fluctuating prevalence rates with a small number of cases contained within a small sample. He notes that even one new case of FASD in a small sample can have a significant effect on the prevalence rate, and when rates are reported out of the context of their sample size, misinterpretations as to their true implications may occur.

In particular, Pacey (2009) is concerned about the studies that have been conducted in First Nations reserves in Canada that tend to report high prevalence rates of FASD. Application of these rates to the general Aboriginal population does not take into account the variations across reserves in alcohol consumption practices and socioeconomic status which are often associated with maternal alcohol use during pregnancy. There may also be regional differences that have not yet been explored: Pacey notes that published prevalence research has to date focused on Aboriginal communities in western Canada

and the prairie provinces, which may not be representative of the experiences of reserves in the eastern part of the country. Importantly, Pacey states:

In absolute terms, even if prevalence is potentially higher among Aboriginal people in Canada, the proportional difference in population size means that the numeric ‘burden’ of FAS or FAS/ARND is greater in the non-Aboriginal population. (p. 20)

To illustrate this point, Pacey summarizes the prevalence findings reported by Sampson et al. (1997), as shown in Table 1 below (Pacey, 2009, p. 20).

Table 1

Populations

	Total	Aboriginal	North American Indian	Metis	Inuit	Non-Aboriginal
	29,639,030	976,305	608,850	292,305	45,070	28,662,725
FAS Prevalence						
2.8/1,000	82,989	2,734	1,705	818	126	80,256
4.8/1,000	142,267	4,686	2,922	1,403	216	137,581
Combined FAS and ARND						
9.1/1,000	269,715	8,884	5,541	2,660	410	260,831

Finally, Pacey notes that in high risk communities, such as reserves facing significant economic constraints and high volume drinking patterns, more than one child with FAS may be born to a single mother. The resulting prevalence rate does not account for the number of women who may give birth to an alcohol-exposed child, only the number of children with the condition. Patterns of recurrence in same birth mothers have been found in other studies, such as by Harris and Bucens (2003) in Australia.

Despite the tendency for prevalence studies to discern statistics from marginalized populations where there is high risk for substance misuse, resulting in an over-representation of Aboriginal people in FASD prevalence reports, it is still true that FASD is a serious problem in First Nations communities (Nesbit, et al., 2004; Streissguth, et al., 1994). While explanations of high prevalence rates to describe the impact of social variables on First Nations peoples are warranted, the risk of over-representation in prevalence rates should not deter researchers from studies to understand the depth of the problem of FASD as it affects high risk groups such as some First Nations communities.

Prevalence of FASD Affecting Children in Care

Studies such as those conducted by Steinhausen, Willms, and Spohr (1993) have described the family backgrounds of alcohol-exposed children, noting that family life for many such children is marked by a high degree of upheaval and crisis. Unsurprisingly, the nature of alcohol addiction and adverse life circumstances associated with maternal binge drinking (and therefore the occurrence of FASD) are also strongly associated with children needing child welfare intervention. Spohr, Willms and Steinhausen (1994) report that numerous alcohol-exposed children are not raised by their biological mothers and often spend their lives growing up in child welfare care. Besharov (1994) estimated that between 65%

and 80% of children with FASD were raised by someone other than their birth parents. In Alberta, up to 29% of children in care were estimated to have been diagnosed with FASD (Alberta Health and Wellness, 2000).

The high proportion of children with FASD in child welfare care has been described in Manitoba (Fuchs, Burnside, Marchenski, & Mudry, 2005). In a study to determine the nature and scope of disabilities experienced by all children in care in Manitoba in 2004 (sample size approximately 5500 children in care), 11% had been diagnosed with FASD and a further 6% were in the process of being tested for the condition, considered to be an underestimation of the actual prevalence within this population due to the stringent criteria used to confirm diagnosis in this study. Further examination of a group of children with FASD (Fuchs, Burnside, Marchenski, & Mudry, 2007) revealed that children with FASD came into care at a younger age (2.5 years) than other children in care, became permanent wards more quickly because of the young age at which they were apprehended, and consequently spent the majority of their lives in care. Importantly, it was noted that the reasons children came into care mainly had to do with the struggles of their parents, as opposed to conditions originating in the child, such as their condition of FASD. This is in contrast to the findings of Habbick et al. (1996) who found that, although Saskatchewan children who were alcohol-exposed also entered care on average at age two, they were more likely to be placed because of parents' inability to handle an alcohol-affected child, as well as deal with their own substance abuse. However, similar findings were reported by Ernst et al. (1999) in reviewing the experiences of women in the Seattle Birth to Three Program, where many of the women's children were in care after three years, either at her request or as a result of child welfare concerns.

Understanding the impact of FASD on the lives of children and adolescents in care is essential for child welfare agencies, given the responsibilities agencies have for children in their care. A body of literature is developing with regard to the specific needs and experiences of children in care with FASD. For example, Streissguth et al. (1996) emphasized the importance of early, long-term placement stability, reporting that multiple placements increased the risk of adverse outcomes for children with FASD. Weiner and Morse (1994) drew the same conclusion from their study of early interventions for children with FAS. In a national survey of child welfare agencies in the United States, 87% of agencies reported that prenatally alcohol-exposed children were more likely to experience multiple placements and stayed in care longer than other children in care (Curtis & McCullough, 1993).

In Manitoba, a series of studies have examined the needs and experiences of children and youth with FASD, describing their life trajectories in care (Fuchs, et al., 2007), their need for placement stability in adolescence when foster homes tend to breakdown as behavioural challenges increase (Fuchs, Burnside, Marchenski, & Mudry, 2008), the higher costs incurred to provide for their care (Fuchs, Burnside Marchenski, Mudry, & DeRiviere, 2008), their higher utilization and costs of health care services and prescription medications (Fuchs et al., 2009), and their needs as they reach adulthood and are transitioning from child welfare care (Fuchs, Burnside, Reinink, & Marchenski, 2010).

With the expanded capacity to diagnose FASD in Manitoba, more children and youth who exhibit functional indicators of FASD can be referred for a multi-disciplinary team assessment, and diagnostic capacity is growing in other jurisdictions in Canada as well. However, Aronson (2000) observes that information confirming maternal alcohol use in pregnancy (which is necessary for an FASD diagnosis) is often not well documented for children in care, particularly those who have become permanent wards and where no contact occurs between biological family and the child welfare agency to make inquiries about the child's prenatal history. Additionally, Nanson (2003) and Chasnoff (2011)

each caution that it can be difficult to isolate FASD as the cause of the many issues facing children in care, given the extensive literature on the effects of out-of-home placement on children and the consequences of adverse life experiences, such as abuse, that may have precipitated the child's admission to care.

As with other high risk populations involved with social service systems, it is not surprising to find a high concentration of children with FASD in child welfare care. Specific research into the prevalence of FASD affecting the child in care population is at a beginning stage, and should be supplemented with comparable research to ascertain the prevalence of FASD affecting children in the general population. Information of this nature will certainly facilitate early identification of children in care in need of assessment for possible diagnosis of FASD, as well as appropriate supports and services. More importantly, prevalence rates for children in care help sensitize child welfare agencies to the critical role they play as the guardians of children with FASD in ensuring that their care is suited to their high needs.

Conclusion

Around the world, countries are recognizing that current national and international data on the prevalence of FASD is critical to planning for prevention and diagnostic services. The benefits of early diagnosis and intervention are well documented, and children should have access to multi-disciplinary clinics where their needs can be assessed and addressed in a timely way (Elliott et al., 2008). Further, "the common statement that FASD is preventable will only come to pass if we increase our efforts for early detection and intervention in a variety of settings" (Burd, et al., 2006, p. 93).

To date, many prevalence studies are conducted on a pilot basis with limited resources and budgets, preventing the true prevalence of FASD in the population from being known. While current studies point to the likelihood that reported prevalence rates are an underestimation of actual prevalence, we don't yet know with certainty how far current reported rates are from the true prevalence rate. Further, current studies around the world highlight the growing awareness and concern about the impact of FASD in many countries, but due to differences in definitions and research methodologies, it is not yet possible to make comparisons about FASD prevalence across studies with full confidence. The work on establishing a baseline FASD prevalence rate is emerging, but not yet realized.

Related to establishing prevalence rates is confirming consistent diagnostic and definitional criteria for all types of FASD (May, et al. 2009; Nanson, 2003). This standardization will aid in ensuring that prevalence rates are measuring the same phenomenon across (and within) jurisdictions.

Many researchers make a compelling argument for the importance of population level prevalence rates (for example, Hoyme, et al., 2005; May et al., 2009; Stratton et al., 1996). MacMillan, MacMillan, Offord and Dingle (1996) provide specific advocacy for prevalence studies in the broader population that will help to dispel the perception that FAS occurs more frequently among Aboriginal populations, an argument that is applicable to other marginalized populations around the world:

Because there is insufficient information about the prevalence of FAS in the non-native population, it is impossible to conclude that there is a higher prevalence among native people. (p. 1576)

Pacey (2009) agrees that more work needs to be done to establish baseline prevalence rates in the general Canadian population, which could then be used to compare rates of prevalence within particular populations. However, he also acknowledges that it is unwise to ignore the possibility that, due to their histories of oppression and compromised socioeconomic status, FASD may have higher prevalence rates in Aboriginal communities. To that end, Kowalsky, Thurston, Verhoef, & Rutherford (1996) recommend the development of guidelines for researchers when working with Aboriginal communities on FASD research that recognize the sensitivity of this issue to Aboriginal people and aims to reduce stigmatization and stereotyping.

And given the immense responsibility child welfare agencies have for children in care with FASD (Fuchs, et al., 2007; Fuchs, et al., 2010), child welfare agencies also need to understand the prevalence of FASD affecting its child in care population. It has been demonstrated that children in care are at high risk for having FASD as a disability (Fuchs, et al, 2005), and the guardians of these children, the child welfare agencies, need to have a solid understanding of the impact of FASD and the needs of children with the condition throughout the lifespan. Without knowing the extent of FASD as it affects the child-in-care population, child welfare agencies will be compromised in their ability to anticipate, plan for, and provide for the needs of this vulnerable group of children.

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