

**BIOPSYCHOSOCIAL PREDICTORS OF CONDUCT PROBLEMS IN ADOLESCENTS
WITH FETAL ALCOHOL SPECTRUM DISORDER**

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ABSTRACT

Research has consistently found an association between prenatal alcohol exposure (PAE) and the development of conduct problems (CP); however, there are numerous biopsychosocial risk factors that often co-occur with PAE and may be important predictors of CP in this high-risk population. The current study applies a biopsychosocial model in order to identify which biological, neuropsychological, family, and social risk factors are most closely associated with the development of CP in adolescents with FASD. This study is part of a larger prospective follow-up study of a cohort of children diagnosed with FASD in the Motherisk Clinic at The Hospital for Sick Children, between 2003 and 2012. Data for predictors were obtained from standardized behavioural questionnaires and life history data gathered during the initial diagnostic assessment. Standardized questionnaires and a semi-structured interview were conducted with caregivers and youth 2 to 7 years post-diagnosis, in order to gather outcome data on the prevalence of various types of CP in adolescence. Multiple linear and logistic regression models were used to identify significant biopsychosocial predictors of CP. After accounting for the effects of a number of key biopsychosocial risk factors, emotional/behavioural regulation ability was found to be a significant and unique predictor of various measures of CP in youth with FASD. Remarkably, this is the first study to investigate the role of these executive processes in the development of CP in individuals with FASD, despite numerous reports of executive functioning deficits in this population. Future intervention efforts aimed at preventing or mitigating CP in individuals with FASD should focus on improving self-regulation skills.

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Introduction

Research has consistently found that children and youth with a history of prenatal alcohol exposure (PAE) or Fetal Alcohol Spectrum Disorder (FASD) exhibit higher rates of externalizing problems and disruptive behaviour disorders compared to non-exposed controls (Day, Helsel, Sonon, & Goldschmidt, 2013; Fagerlund, Autti-Rämö, Hoyme, Mattson, & Korkman, 2011; Fryer, McGee, Matt, Riley, & Mattson, 2007; Larkby, Goldschmidt, Hanusa, & Day, 2011; Mattson & Riley, 2000; Roebuck, Mattson, & Riley, 1999; Sood et al., 2001; Steinhausen, Willms, Metzke, & Spohr, 2003; Walthall, O'Connor, & Paley, 2008). Conduct problems (CP) in children and youth are often associated with adverse consequences, including disruptions in education and involvement in the criminal justice system, which can carry over into adulthood (Advisory Group on Conduct Problems, 2009; Moffitt, 1993). In fact, youth and adults with FASD have been found to be significantly overrepresented in the criminal justice system in North America (Fast, Conry, & Loock, 1999; Moore & Green, 2004; Popova, Lange, Bekmuradov, Mihic, & Rehm, 2011; Roach & Bailey, 2009; Streissguth et al., 1996; Streissguth et al., 2004; Todorow, 2011). Overall, CP in FASD are associated with enormous emotional and financial costs to the individual, their families, and society as a whole. Unfortunately, few studies have focused on investigating the complex constellation of biopsychosocial risk factors for CP in youth with FASD and studies that have been conducted in this area have reported somewhat inconsistent findings. Most importantly, previous studies have largely lacked an underlying theoretical framework to guide study design and predictor selection, and thus, have failed to investigate the influence of a number of potentially important biopsychosocial risk factors that have been linked to the development of CP in the general population. Researchers to date have also failed to uncover biologically plausible mechanisms by which PAE may lead to CP later in

development. Identifying which neuropsychological deficits put those with FASD most at risk for developing CP is particularly critical, as PAE can cause a diverse array of impairments across cognitive and developmental domains. The biopsychosocial theory of the development of CP proposes that CP result from the complex interaction between multiple biological, neuropsychological, family, and social risk factors, at various stages of development. Using the biopsychosocial model of CP to guide our predictor selection, the current study aimed to identify how neuropsychological deficits, as well as co-occurring biological, family, and social risk factors, function together to predict the development of CP in youth with FASD. Moreover, the current study focused on further shedding light on the prevalence of various types of CP in youth with FASD, the unique risk profile for CP in this population, as well as the continuity of CP across middle childhood to adolescence.

Literature Review

Prenatal alcohol exposure (PAE) may disrupt the normal development of a fetus in utero, resulting in a range of physical, cognitive, and behavioural impairments. Fetal Alcohol Spectrum Disorder (FASD) is an umbrella term used to describe the range of impairments caused by exposure to alcohol in utero and encompasses four specific diagnoses: Fetal Alcohol Syndrome (FAS), Partial Fetal Alcohol Syndrome (pFAS), Alcohol-Related Neurodevelopmental Disorder (ARND), and Alcohol-Related Birth Defect (ARBD). A diagnosis of FAS, pFAS, and ARND requires evidence of impairment in three or more central nervous system (CNS) domains manifested by abnormalities in the following: hard and soft neurologic signs, brain structure, cognition, communication, academic achievement, memory, executive functioning (EF), attention, hyperactivity, adaptive behaviour, social skills, and social communication (Chudley et al., 2005). Neuropsychological impairment is the key defining feature of alcohol-related

disorders (Institute of Medicine [IOM], 1996; Chudley et al., 2005), however, the specific profile of neuropsychological impairments seen in individuals with FASD can vary widely (Mattson, Crocker, & Nguyen, 2011).

In addition to the adverse effects of PAE on the development of the CNS, exposure can also disrupt the growth and morphological development of the fetus, resulting in growth deficiency, characteristic facial anomalies (i.e., flattened midface and philtrum, short palpebral fissure length, thin upper lip), and other congenital anomalies. Whether an individual receives a diagnosis of FAS, pFAS, or ARND depends on the presence/absence of characteristic facial anomalies and growth impairment. In order to receive a diagnosis of FAS, the individual must present with all three characteristic facial anomalies and growth impairment. If only two facial anomalies are present, then a diagnosis of pFAS is given. A diagnosis of ARND is provided in cases where an individual presents with only one characteristic facial anomaly or no physical abnormalities, yet exhibits CNS impairment in three or more domains and has confirmed PAE (IOM, 1996; Chudley et al., 2005). Confirmation of PAE is necessary in order to receive a diagnosis under the fetal alcohol spectrum, except in cases where all three facial features and growth impairment are present (i.e., in cases of FAS) (Chudley et al., 2005). The term ARBD is used when an individual displays one or more congenital anomalies that have been observed in individuals with PAE, however, it is recommended that this label be used with caution, as it is unclear whether most of these anomalies are specifically caused by PAE (Chudley et al., 2005). The term Fetal Alcohol Effects (FAE) was used previously to describe individuals with confirmed PAE and neuropsychological impairments who lacked the physical anomalies that are necessary for a diagnosis of FAS (i.e., those classified currently as pFAS and ARND). It is important to note that most individuals with PAE do not present with the full constellation of

physical abnormalities required for a diagnosis of FAS (Rasmussen, Horne, & Witol, 2006; Streissguth & O’Malley, 2000), possibly due to the smaller window of development for facial features during gestation (i.e., between the sixth and ninth weeks of gestation) (May & Gossage, 2011; Sulik, Johnston, & Webb, 1981). In contrast, the CNS, including the brain, develops throughout gestation (for review see Stiles & Jernigan, 2010) and this prolonged window of development increases the likelihood that the CNS will be negatively impacted by maternal alcohol consumption during pregnancy.

Prevalence and Societal Impact of FASD

Although the exact prevalence of FASD is difficult to determine, disorders encompassed under the fetal alcohol spectrum are estimated to affect approximately 1 in 100 births or 1% of the North American population (Sampson et al., 1997), with a significantly higher incidence in certain communities around the world (Petković & Barišić, 2013; May et al., 2006; May et al., 2013; May & Gossage, 2001) and in the child-welfare system (Lange, Shield, Rehm, & Popova, 2013). Moreover, FASD is reported as the leading known cause of cognitive and developmental disabilities in Canada (Canadian Pediatric Society, 2002; Chudley et al., 2005). The cost of FASD to Canada for individuals from birth to 53 years of age was estimated to be 5.3 billion dollars per year (Stade et al., 2009), however, a recent review of the literature in this area suggests that this figure is an underestimate of the true economic impact of FASD because several important cost drivers, such as the cost to the child welfare system, were not accounted for in previous studies (Popova, Stade, Bekmuradov, Lange, & Rehm, 2011).

Individuals with FASD experience high rates of negative long-term outcomes including mental health problems, academic disengagement, trouble with the law, inappropriate sexual behaviour, and problems with substance use (Clarke, Lutke, Minnes, & Ouellette-Kuntz, 2004;

Spohr & Steinhausen, 2008; Spohr, Willms, & Steinhausen, 2007; Streissguth, Barr, Kogan, & Bookstein, 1996; Streissguth et al., 2004; Todorow, 2011). These adverse long-term outcomes result in enormous emotional and financial costs to the individual and their families, as well as society as a whole (Popova et al., 2011; Todorow, 2011). The emotional and financial burden specifically associated with conduct problems (CP) exhibited by this population is of particular concern. CP in children and youth are often associated with a number of negative consequences, including increased caregiver and family stress, physical and emotional harm to the individual and others, loss of interpersonal relationships, disruptions in education, and involvement in the criminal justice system (Advisory Group on Conduct Problems, 2009). As Moffitt (1993) noted, the adverse consequences of CP, including a criminal record and disruption in education, can carry over into adulthood by limiting opportunities and reducing the likelihood of successful adaptation to later developmental challenges (e.g., finding employment). In other words, CP during adolescence can steer youth towards a maladaptive developmental pathway that is defined by fewer opportunities for successful adaption to life challenges and higher risk for adverse long-term outcomes, such as difficulties with employment and dependant living (Moffitt, 1993).

Moreover, CP experienced by individuals with FASD appears to be a significant source of stress for caregivers of affected individuals. One study examining the predictors of stress in biological and adoptive parents of children with FASD found that parent and teacher ratings of externalizing problems were significant and independent predictors of child-related stress, as measured on the Parent Stress Index (Paley, O'Connor, Frankel, & Marquardt, 2006). CP also led to a significant burden on the mental health system, as it is one of the most common reasons for referrals to mental health service agencies for children and youth (Loeber, Burke, Lahey,

Winters, & Zera, 2000). Unfortunately, little progress has been made in terms of identifying the complex constellation of risk factors or predictors of CP in youth with FASD.

Defining Conduct Problems

The term “conduct problems” (CP) is used to describe a wide spectrum of externalizing behaviours, including disobedient, delinquent, antisocial, and aggressive behaviours (Advisory Group on Conduct Problems, 2009; McMahon & Frick, 2005). CP exhibited by children and youth can vary in severity, and specific repetitive patterns of CP constitute criteria for particular mental disorders outlined in the fifth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5), including Conduct Disorder (CD) and Oppositional Defiant Disorder (ODD) (APA, 2013; McMahon & Frick, 2005). CD is described as a “repetitive and persistent pattern of behaviour in which the basic rights of others or major age-appropriate societal norms or rules are violated” (APA, 2013, p. 469). Specific CP associated with CD includes aggression toward people or animals, destruction of property, deceitfulness and theft, and serious violation of rules (APA, 2013). In contrast, ODD is described as a “pattern of angry/irritable mood, argumentative/defiant behaviour or vindictiveness” and often precedes the onset of CD, however the majority of children who are diagnosed with ODD do not go on to develop the more severe behavioural symptoms necessary for a diagnosis of CD (APA, 2013; Lahey & Loeber, 1994; McMahon & Frick, 2005). The term delinquency specifically refers to involvement in acts which are prohibited by law (e.g., theft, assault, drug offences) and is commonly measured using self-reports or official legal records (Murray & Farrington, 2010). Many of the symptoms of CD are however, delinquent acts, and thus, there is much overlap between these two constructs. Research also suggests that CP in youth can be further classified as either overt/confrontational

(e.g., physical aggression, open defiance) or covert/non-confrontational (e.g., theft, substance use, dishonesty) (Fergusson, Horwood, Lynskey, 1994a; Frick et al., 1993).

Conduct Problems in FASD

Research has consistently found that children with PAE exhibit higher rates of externalizing behavioural problems compared to non-exposed controls matched on a number of potential confounders, such as age, gender, ethnicity, social competence, and IQ (Fagerlund, Autti-Rämö, Hoyme, Mattson, & Korkman, 2011; Mattson & Riley, 2000; Roebuck, Mattson, & Riley, 1999; Sood et al., 2001; Steinhausen, Willms, Metzke, & Spohr, 2003; Walthall, O'Connor, & Paley, 2008). Moreover, longitudinal research has shown that PAE is a significant predictor of self-reported externalizing behavioural problems at 22 years of age (Day, Helsel, Sonon, & Goldschmidt, 2013). Another 20-year follow-up study of adults diagnosed with FAS found significantly higher Intrusive and Aggressive Behaviour scores on the Young Adult Behaviour Checklist compared to the normative sample, independent of gender (Spohr, Willms, Steinhausen, 2007).

Consistent with these findings, higher rates of disruptive disorders, including CD and ODD, have been documented in those with PAE compared to controls (Fryer, McGee, Matt, Riley, & Mattson, 2007; Larkby, Goldschmidt, Hanusa, & Day, 2011). Studies utilizing standardized diagnostic interviews to examine the point prevalence of psychiatric disorders in children and youth with FASD or heavy PAE have reported high rates of ODD, with estimates ranging from 18.5 to 38% (Burd, Klug, Martsolf, & Kerbeshian 2003; Fryer et al., 2007). Previous research has also found a strong association between PAE and symptoms of CD or rates of delinquent behaviour in children (D'Onofrio et al., 2007; Fryer et al., 2007; Roebuck et al., 1999) and adolescents (Disney, Iacono, McGue, Tully, & Legrand, 2008; Schonfeld, Mattson, &

Riley, 2005). Fryer et al. (2007) found an 18% point prevalence rate of CD in children and youth with PAE ($n = 39$) using the Computerized Diagnostic Interview Schedule for Children, Version IV, and the Kiddie Schedule for Affective Disorders and Schizophrenia for School-Age Children, Present and Lifetime Version. Schonfeld et al. (2005) used the Conduct Disorder Questionnaire to measure CP in 27 adolescents with heavy PAE and 29 non-exposed controls matched on age, gender, handedness, SES, and ethnicity. These researchers found that 30% of adolescents with heavy PAE met the criteria for probable CD, however none of these individuals exhibited the full constellation of physical abnormalities necessary for a diagnosis of FAS. Moreover, 80% of adolescents who met criteria for probable CD in the total sample had a history of PAE (8/10).

PAE or FASD has also been linked to high rates of involvement in the criminal justice system. A large cross-sectional study conducted in the U.S. found that approximately 60% of adolescents and adults with FASD had experienced “trouble with the law,” operationally defined as being charged, arrested, convicted, or otherwise in trouble with the legal system (Streissguth et al., 1996; Streissguth et al., 2004). In contrast, Spohr, Willms, and Steinhausen (2007) found no increase in major crime or incarceration in a prospectively followed group of 37 young adults with FASD in Germany. A systematic literature review of studies examining the prevalence/incidence of FASD in the criminal justice system in Canada and the U.S. revealed that 10 to 23% of incarcerated individuals are alcohol-affected; however, this study noted that many incarcerated individuals may be affected by PAE, yet may be undiagnosed (Popova, Lange, Bekmuradov, Mihic, & Rehm, 2011). The highest estimate (23%) came from a study in which all youth who were admitted to a forensic inpatient assessment unit in British Columbia for committing a criminal offence were directly assessed for FASD during a one-year period

(Fast, Conry, & Loock, 1999). Interestingly, only 3 of the 67 youth who met criteria for FASD (4.5%) had been given a diagnosis prior to being incarcerated (Fast et al., 1999). Despite issues of under-diagnosis, these findings suggest that youth and adults with FASD are overrepresented in the criminal justice system in North America (Fast et al., 1999; Moore & Green, 2004; Popova et al., 2011; Roach & Bailey, 2009; Todorow, 2011).

Predictors of Conduct Problems in Individuals with PAE/FASD

Predictors of externalizing behaviours in children with PAE.

More recent research has attempted to control for a number of maternal and environmental variables that may partially account for the higher rates of externalizing behaviours documented in children with PAE. Sood et al. (2001) utilized a prospective study design to investigate the dose-response effect of PAE on children's behaviour. The study population included 501 children (6–7 years of age) born predominately to African American mothers (>90%) who were prospectively assessed for alcohol and substance use during pregnancy at a university-based maternity clinic in Michigan. Alcohol, cigarette, and other substance use during pregnancy was prospectively assessed during prenatal visits, and reported alcohol consumption was converted to fluid ounces of absolute alcohol and averaged across visits, in order to obtain a measure of average maternal alcohol intake throughout pregnancy. Child behaviour at follow-up was assessed using the Child Behavior Checklist (CBCL), a standardized parent-report questionnaire (Achenbach & Rescorla, 2001). Data on the following factors known to generally influence child behaviour were collected and explored as potential covariates: prenatal exposure to cigarettes and cocaine, child's gestational age, child's birth weight, child's gender, age of the child at follow-up, whole blood lead levels in the child, maternal age, maternal education, marital status, maternal psychopathology (self-reports on the

Symptom Checklist–90), paternal drinking and use of drugs postnatally, SES, family structure (two-parent family), custody status (with biological mother), home environment (measured using a modified Home Observation for Measurement of the Environment assessment), and child's exposure to violence (caregiver report). Children were divided into non-exposed, low exposure (< 0.3 fluid ounces of alcohol/day) and moderate/heavy exposure (≥ 0.3 fluid ounces of alcohol/day) groups, and the dose-response effect of PAE on child behaviour was examined using ANOVA. Significant between group differences were found among scores on the Total Problems, Delinquent Behavior, and Aggressive Behavior scales. Pairwise comparisons revealed that, when compared to the non-exposed controls, children with moderate/heavy PAE had significantly higher scores on the Delinquent Behaviors, Aggressive Behaviors, and Total Problem scales, while the low PAE group had significantly higher scores on the Aggressive Behavior scale. Most importantly, results from a stepwise regression analysis, which included all of the abovementioned covariates, revealed that PAE, maternal psychopathology, HOME inventory scores, and maternal education were significant and independent predictors of Aggressive Behavior scores. PAE, maternal psychopathology, custody status, levels of lead in the child's blood, and being of male gender were significant and independent predictors of Delinquent Behavior scores. Interestingly, maternal psychopathology was the strongest predictor of child behaviour outcomes. This study was one of the first to examine the effects of PAE on child behaviour while accounting for a variety of potentially relevant genetic and family variables; however, the specificity of the sample (i.e., African American women who received prenatal care) limits the generalizability of the findings (Sood et al., 2001).

D'Onofrio et al. (2007) also set out to examine the association between PAE and the development of externalizing behaviour problems in childhood using a quasi-experimental

design to help account for potential genetic and environmental confounders. The study population included a racially and socioeconomically diverse sample of 4912 mothers with children between 4 and 11 years of age ($N = 8621$), who were initially recruited as part of the National Longitudinal Survey of Youth in the United States. Frequency and quantity of alcohol use during pregnancy were determined via maternal report, primarily within two years postpartum. Mothers rated their children's externalizing behaviour biennially on the Behavior Problem Index, which included 13 items from the CBCL that loaded onto the following three factors: conduct problems, oppositional problems, and attention/impulsivity problems. Mean scores across ages 4 to 11 were calculated for each factor and were used as main outcome variables. Data on the following child, maternal, and familial factors were explored as potential covariates in subsequent analyses; child's gender, prenatal nicotine exposure, maternal intellectual ability, maternal education, maternal age at birth, maternal delinquency (self-report), family income, family ethnicity, blended family (i.e., having two or more children with different fathers), frequency of maternal alcohol use, average quantity of maternal alcohol intake, number of binge drinking episodes in the past month, and maternal alcohol abuse/dependence.

Hierarchical linear regression modeling was used to explore the association between PAE and externalizing problems in children, controlling for the covariates described above. The results of this analysis revealed that PAE, male gender, African American ethnicity, prenatal nicotine exposure, lower maternal education, lower family income, maternal delinquency, lower maternal age at birth, and living in a blended family were all significantly and independently associated with conduct problems in children. A separate hierarchical linear regression model was also constructed, which used contrast codes to compare outcomes in siblings and first-degree cousins who were differentially exposed to prenatal alcohol, in an effort to control for unmeasured

genetic and environmental confounds. This model included sibling and cousin comparisons, as well as the maternal and familial covariates. Results from this more sophisticated statistical model mirrored the results from the previous model and showed that children with higher levels of PAE displayed significantly more conduct problems than relatives in the same family with lower levels of PAE, even after controlling for the effects of the measured covariates (D'Onofrio et al., 2007).

Predictors of externalizing behaviours in children/adolescents with FASD.

Fagerlund, Autti-Rämö, Hoyme, Mattson, and Korkman (2011) set out to investigate how diagnostic and environmental factors functioned to predict behavioural problems in a Finnish cohort of children and youth with FASD ($N = 73$). PAE was confirmed by reviewing birth records and/or via interviews with caregivers or biological parents, and all participants were assessed by a dysmorphologist and provided with a diagnosis according to the revised IOM diagnostic criteria for FASD. The study population was 60% female and ranged in age from 8 to 21 years old ($M = 13$ years, 5 months). Data on diagnostic and environmental risk/protective factors were collected using a structured interview developed by Streissguth and colleagues (1996; 2004), which was conducted with participants' adult caregivers. Risk/protective factors included: age at diagnosis (years), diagnostic category (FAS, pFAS or ARND), dysmorphology score, length of time the individual lived with their biological parents (months), length of time the individual lived with foster/adoptive parents (months), length of time spent in residential care (months), and the number of different home placements by the age of 18. These researchers also investigated the potential protective effects of receiving special education (time spent in years) and having a classroom aid at school (time spent in years). Behavioural problems in children and youth were assessed using the CBCL (completed by adult caregivers) and scores on the

Internalizing Problems, Externalizing Problems, and Total Problems summary scales were used as outcomes variables. Due to the large number of predictors and limited sample size, the unique effect of diagnostic, family/environmental, and educational factors were first investigated using separate multiple linear regression analyses, and significant predictors across domains were subsequently entered into a final multiple linear regression analysis. This procedure was carried out for each of the three outcome variables and all analyses controlled for sex, age at follow-up, and IQ. Results from the separate multiple regression analyses carried out for each category of predictor revealed that lower dysmorphology scores (i.e., a lower number of discernable dysmorphic features) and longer time spent living in residential care were associated with significantly higher scores on the Externalizing Problems scale. Longer time spent in residential care was also associated with increased Internalizing Problems and Total Problems scores. All other predictors, including sex, age, and IQ, were not significant. When dysmorphology scores and time spent in residential care were entered simultaneously into a final regression analysis (controlling for sex, age, and IQ), only time spent in residential care significantly predicted Externalizing Problems scores. The results of this study suggest that the environment in which the child is raised is the strongest predictor of later CP in children and youth with FASD. However, it is also important to consider that children and youth are often placed into residential care *because* of severe externalizing behaviours, such as physical aggression, and thus, the relationship between residential care and externalizing behaviour may be more transactional in nature.

Predictors of conduct disorder in children/adolescents with PAE.

Hill, Lowers, Locke-Wellman, and Shen (2000) used a prospective longitudinal study design to examine the relative effects of PAE, maternal smoking during pregnancy, and familial

risk for alcohol dependence, on the development of specific mental health disorders in children. The study population included 89 children and adolescents who had a mother and aunt with alcohol dependence (i.e., high familial risk for alcohol dependence) and 61 controls whose first- and second-degree relatives did not have a mental health or substance use disorder (i.e., low familial risk for alcohol dependence). Additional inclusion criteria for children in the high familial risk group included the absence of other Axis I mental health or substance use disorders in first-degree relatives, yet children were not excluded if their parents met the criteria for Antisocial Personality Disorder (APD). The presence or absence of mental health and substance use disorders in parents and first-degree relatives was determined using a structured diagnostic interview or by at least two family-history reports. Mothers were also administered a structured interview in order to determine the number of alcoholic drinks and cigarettes consumed per day during pregnancy. Log-linear and regression analyses of data on PAE were based on a median split of alcohol use for all mothers who reported consumption during pregnancy. Smoking data were categorized dichotomously (yes/no). Both parents and children were administered the Schedule for Affective Disorders and Schizophrenia for School-Age Children (K-SADS) and each participated in an individual unstructured clinical interview. The presence of specific mental health disorders in children was determined by using a "best-estimate" consensus approach considering data from all sources. Separate log-linear regression analyses were used to test the bivariate relationship between each predictor (familial risk, PAE, and smoking) and each specific mental health diagnosis in children (ADHD, CD, ODD, depression, anxiety disorders, and phobia). Both PAE and familial risk for alcohol dependence were associated with a significant increase in rates of CD, ODD, ADHD, depression, and anxiety in children. Smoking was also significantly associated with CD, ODD, and depression in children. A series of logistic

regression analyses using subsets of variables revealed similar results. When the effects of each predictor on child mental health were analyzed adjusting for the effects of the other two predictors using Miettinen's multivariate confounder score approach, the only association that remained significant was the relationship between high familial risk and ODD in children. It is important to note, however, that there is evidence to suggest that Miettinen's statistical approach may notably exaggerate the statistical significance obtained (Pike, Anderson, & Day, 1979). The association between familial risk, PAE, and smoking on the development of CD, ODD, and ADHD in children was further evaluated using three additional logistic regression analyses, which controlled for SES and parental APD. After adjusting for the effects of SES and parental APD, only one association remained significant — the relationship between familial risk and ADHD. Thus, the results of this study indicate no relationship between PAE and child mental health after adjusting for the effects of other genetic and environmental confounding variables, however, several methodological weaknesses limit the conclusions that can be drawn from these results. These include high levels of collinearity among predictors, the use of multiple separate analyses using a subset of predictors, and the use of a non-representative sample of individuals with strong family histories of alcohol dependence (Hill, Lowers, Locke-Wellman, & Shen, 2000).

Disney et al. (2008) investigated the effects of low levels of PAE and smoking on the later development of CD symptoms in adolescence, as well as the potential mediating effects of parental psychopathology. This study included a sample of 626 same-gender twin pairs ($N = 1252$) drawn from the population-based Minnesota Twin Family Study. The large majority of the sample was Caucasian (98%) and lived in a two-parent family (85%). Twin pairs that were later adopted or had severe disabilities were excluded. The number of CD symptoms was assessed in

adolescent twin pairs, at the age of 17, using standardized diagnostic interviews conducted with mothers and youth. CD symptoms were considered present if endorsed by either informant. Data from structured diagnostic interviews administered to both parents were also reviewed to ascertain lifetime alcohol dependence, nicotine dependence, drug abuse/dependence, as well as the presence of parental APD or antisocial behaviour. Parents with "probable" lifetime diagnoses were lumped together with those meeting full diagnostic criteria for a disorder in order to increase sensitivity of lifetime diagnoses. PAE was defined by retrospective maternal reports of one or more alcoholic drinks per week during gestation. Approximately 13% of women reported PAE, with a mean of 2.9 drinks per week. Multiple hierarchical linear regression modeling showed that PAE, male gender, parental alcohol dependence, parental nicotine dependence, and parental drug abuse/dependence all predicted a significant increase in CD symptoms in adolescents. Furthermore, PAE was found to be a significant and independent predictor of CD symptoms in adolescents, even after controlling for parental psychopathology and other birth factors, such as gestational age, birth weight, and monozygosity (Disney et al., 2008).

Ware et al. (2013) designed a unique multisite study to investigate the relationship between PAE and ADHD on the prevalence of specific mental health disorders and problem behaviours in children. The study population included 344 children (8–16 years old) who were divided into four groups: children with PAE who also met the DSM-IV criteria for ADHD ($n = 85$), children with PAE who did not meet the criteria for ADHD ($n = 52$), children who met the criteria for ADHD and had no history of exposure ($n = 74$), and non-exposed children who did not meet criteria for ADHD (controls; $n = 133$). Maternal alcohol consumption was quantified retrospectively using medical/social service records and/or maternal reports. PAE was defined by maternal consumption of more than four drinks on at least one occasion per week during

pregnancy or a minimum of 13 drinks per week throughout pregnancy. Caregivers completed the Computerized Diagnostic Interview Schedule for Children – Fourth Edition and the CBCL, in order to assess the presence of specific mental health diagnoses (including ADHD) and behavioural problems in children. Chi-square tests revealed a significant group difference in the prevalence of generalized anxiety disorder (GAD), major depressive disorder (MDD), ODD, and CD. Pairwise comparisons revealed that significantly more children with PAE and ADHD met the criteria for ODD and CD, compared to children who failed to meet the criteria for ADHD, with and without PAE. The rates of ODD among exposed children with ADHD and non-exposed children with ADHD were comparable, however, children with PAE and ADHD had significantly higher rates of CD than non-exposed children with ADHD. In conclusion, the results of this study indicate that PAE, in combination with an ADHD diagnosis, results in an increased risk of developing CD, above and beyond the risk associated with having either PAE or ADHD alone. Thus, the role of inattention, hyperactivity, and impulsivity on the development of CP in children and youth with FASD warrants further exploration (Ware et al., 2013).

Predictors of delinquency in adolescents with PAE.

Lynch, Coles, Corley, and Falek (2003) set out to investigate the effects of PAE, as well as other important individual, family, and community risk factors, on the development of delinquent behaviour in a community-based sample of predominately African American adolescents. The study included four groups of adolescents: adolescents with PAE and dysmorphology associated with a diagnosis of FAS ($n = 39$), adolescents with PAE without dysmorphology ($n = 77$), adolescents with no history of PAE, but with similar SES and ethnic backgrounds ($n = 48$), and adolescents receiving special education with similar SES and ethnic backgrounds ($n = 84$). Mothers of adolescents included in the PAE groups were initially

recruited during pregnancy from an inner-city hospital in Atlanta and data regarding PAE were typically collected via self-report in the second trimester of pregnancy. PAE was defined as maternal consumption of at least two drinks per week during pregnancy. Follow-up assessments with adolescents occurred at a mean age of 15.1 years and adolescents who did not participate in follow-up were significantly more likely to be “white,” have mothers who were married or separated, and have lower 1-minute Apgar scores at birth. The special education control group was recruited during the follow-up period in an effort to account for the effects of IQ and learning problems on delinquent behaviours. This group was not directly screened for PAE, however, current drinking patterns and liver functioning of the mothers of these youth were similar to that of controls. Unlike other studies, decisions regarding which individual, family, and community risk factors to investigate (in addition to PAE) and how to structure statistical analyses were informed by a developmental and biopsychosocial model of childhood delinquency, proposed by Loeber and Farrington (2000). Thus, the following individual factors were assessed and explored as potential predictors of CP in the entire sample: clinician rated dysmorphology scores, life stress (measured using the Life Events Checklist), drug use, alcohol use (self-reported and dichotomously scored), aggressive behaviour, and attention problems (T-scores on respective subscales of the CBCL). Family factors included parental warmth (adolescent-report on the Parental Warmth/Involvement Scale), parental supervision (adolescent-report on the Parental Strictness/Supervision Scale), consistency of parental discipline (caregiver-report on the Consistency of Discipline scale for the Denver Youth Survey), parental verbal aggression, and parental violence (caregiver-report on respective subscales of the Conflict Tactics Scale). Caregiver substance use was conceptualized as a unique risk factor and included measures of the primary caregiver’s current alcohol use (caregiver-report of alcohol/week) and

drug use (caregiver-report on composite drug index of the Addiction Severity Index). Finally, negative peer influence (caregiver-report on a four-item scale developed for the Denver Youth Survey) and exposure to community violence (caregiver-report on the Screening Survey of Children's Exposure to Community Violence) were explored as potential community risk factors. Adolescent delinquency was assessed at follow-up using the Seattle Self-Report Instrument. This self-report questionnaire produces Last Year Frequency and Ever Variety summary scores, which measure the sum of delinquent acts reported in the past 12 month and the number of different delinquent acts ever committed, respectively. It also produces subscales for Serious Crimes, Delinquency, School and Family Offences, and Official Contacts. ANOVA and MANOVA revealed no significant differences in delinquency scores between groups. Hierarchical regression analyses were then completed to examine the effect of all predictors on adolescent delinquency scores for the entire sample, in which individual factors were entered in Level 1 (excluding PAE), family factors were entered in Level 2, caregiver substance use factors were entered in Level 3, and community factors were entered in Level 4. Results from these analyses revealed that higher levels of adolescent life stress and drug use, and lower levels of parental supervision, were significantly and independently associated with a greater variety of delinquent acts committed by adolescents (i.e., higher Ever Variety Scale scores). Higher levels of adolescent life stress, adolescent drug use, and parental verbal aggression were associated with a greater number of delinquent acts reported in the past 12 months (i.e., higher Last Year Frequency scores). These results suggest that individual and family variables are more strongly related to adolescent delinquency than PAE or community risk factors (Lynch, Coles, Corley, & Falek, 2003). It is important to note, however, that adolescent life stress can also be conceptualized as a comorbid outcome of adolescent delinquency and that drug use can be

conceptualized as a specific type of delinquent behaviour, which poses problems for the interpretation of this study's results. Moreover, this study only included low SES adolescents, who were predominately African American, which limits the generalizability of the findings to other demographically diverse populations. Finally, adolescent delinquency was measured solely using a self-report questionnaire, which may be subject to biases in self-presentation and self-perception (Dodge & Pettit, 2003).

Predictors of criminality in adolescents/adults with FASD.

Streissguth et al. (2004) published a large cross-sectional follow-up study investigating the prevalence of negative life outcomes in an ethnically diverse sample of 415 individuals (6–51 years of age) diagnosed with FAS or FAE at the University of Washington's Fetal Alcohol and Drug Unit. These researchers also selected 10 diagnostic and environmental characteristics (based on their clinical experience) to investigate as potential risk or protective factors for negative life outcomes in a subset of the sample who were 12 years of age or older ($n = 253$). A structured interview was designed by the researchers and conducted with caregivers in order to identify the prevalence of specific negative long-term outcomes and to gather data on the selected risk and protective factors. Data on the following risk/protective factors were gathered from interviews with caregivers, unless otherwise noted: sex (obtained from patient records), age at diagnosis (obtained from patient records), diagnostic category (FAS vs. FAE; obtained from patient records), IQ (WISC-R/WAIS-R), percent of life in a stable/nurturing home, percent of life living with a person who abused alcohol or drugs, percent of life the individual did not have their basic needs met, experience of physical/sexual abuse or domestic violence (yes/no), average number of years spent per household by the age of 18, and quality of the home environment during pre-adolescent years (measured by the Good Quality Home scale for ages 8 to 12 years).

According to caregiver reports, 60% of adolescents and adults had experienced “trouble with the law,” operationally defined as being charged, arrested, convicted, or otherwise in trouble with the legal system. The most commonly reported offences were crimes against persons (e.g., shoplifting/theft, assault, burglary, and domestic violence) and 35% of adolescents and adults had been incarcerated for committing a crime. The selected risk/protective factors were first converted into binary variables and continuous factors (e.g., percent of life in a stable/nurturing home) and were subsequently coded into dichotomous variables by splitting the distribution at the median (e.g., high vs. low percent of life in a stable/nurturing home). Individual odds ratios were then calculated for each negative life outcome investigated, including trouble with the law. Being male, being over the age of 12 at diagnosis, having FAE (opposed to FAS), having a “low” percent of life in a stable/nurturing home, having “fewer” years per household, having experienced abuse or domestic violence, and having a “poorer” Quality Home score all significantly increased the likelihood of experiencing trouble with the law, with unadjusted odds ratios ranging from 1.84 to 2.92. Continuous and dichotomous risk/protective factors were then entered into a logistic regression analysis for each negative life outcome investigated, using the stepwise procedures for “generalized additive models” in the S-PLUS statistical analysis system. Result from the stepwise logistic regression analysis revealed that being male, having a diagnosis of FAE, being diagnosed at an older age (17 vs. 9 years old), and spending a smaller proportion of life in a stable/nurturing home were all associated with a significant increase in the risk of experiencing trouble with the law. Although these results highlight the importance of the family environment in the development of delinquency in youth and adults with FASD, causal inferences cannot be drawn from this cross-sectional study design. Moreover, these researchers failed to use standardized instruments to measure most of the complex risk/protective factors and

outcome variables they investigated, and instead relied on more subjective caregiver judgements, which may reduce the reliability and validity of the data collected (Streissguth, Barr, Kogan, & Bookstein, 1996; Streissguth et al., 2004).

Spoehr, Willms, and Steinhausen (2007) investigated the long-term outcomes of FAS/FAE in a German cohort of young adults followed over 20 years ($N = 37$). Unlike the results of the Streissguth et al. (1996; 2004) study, interviews with caregivers or close relatives failed to reveal an increase in the prevalence of major crime or incarceration. More importantly, these researchers noted that almost all of the protective factors described by Streissguth et al. (2004) were present in this unique cohort (Spoehr, Willms, & Steinhausen, 2007), which may have served to protect against the development of CP in these individuals, and may partially explain the low rates of criminality reported.

Limitations of Previous Research Investigating the Predictors of Conduct Problems in FASD

Although there is a small body of research that has examined the effects of PAE on the development of CP, while controlling for a variety of child, family, and environmental covariates, many of these previous studies have focused on testing the causal relationship between PAE and CP. As a result, the majority of studies did not screen participants for a formal FASD diagnosis, which may limit the generalizability of previous findings to more severely affected individuals who meet criteria for this clinical diagnosis. Outcome measures utilized in previous research have also been limited to standardized questionnaires and structured diagnostic interviews *or* real-life reports of involvement in the legal system, and no study to date has combined these data sources to achieve a more comprehensive evaluation of CP in youth with FASD. Moreover, findings regarding specific risk factors associated with increased CP in

individuals with FASD have been somewhat inconsistent. For example, Fagerlund et al. (2011) found that time spent in residential care significantly predicted externalizing behaviour in children and youth with FASD, which is consistent with Streissguth and colleagues' (1996; 2004) reports that spending less time in a stable/nurturing home is associated with a significantly increased risk of delinquency later in life. Conversely, Fagerlund et al. (2011) failed to find a significant relationship between externalizing behaviour and sex, age, diagnostic category, or specific diagnosis along the FASD-spectrum, which is inconsistent with other researchers' findings (D'Onofrio et al., 2007; Streissguth et al., 1996; 2004).

Researchers have also failed to thoroughly explore biologically plausible mechanisms by which PAE may translate into later CP, such as by causing specific neuropsychological impairments. Most importantly, only one study to date (Lynch et al., 2003) has utilized a biopsychosocial theoretical framework to guide their study design and predictor selection, and thus, most studies have failed to investigate the influence of a number of important individual, family, and social risk factors that have been linked to the development of CP in the larger population. Unfortunately, Lynch et al. (2003) failed to formally screen participants for FASD and included a restricted groups of subjects (predominately African American mother-child dyads), which limits the generalizability of their results to the ethnically diverse Canadian population. Moreover, this study relied solely on self-reports of delinquent behaviour, which may be significantly impacted by social desirability biases. Overall, most studies conducted to date have failed to include the complex constellation of family and social risk factors that often co-occur with maternal alcohol use in pregnancy.

Biopsychosocial Theory of the Development of Conduct Problems

It is generally accepted among researchers that CP arise from a complex interaction between numerous diverse risk factors (e.g., Bassarath, 2001; Burke, Loeber, & Birmaher, 2002; Dodge & Pettit, 2003; Hinshaw & Lee, 2003; McMahon & Frick, 2005; Murray, & Farrington, 2010). A biopsychosocial theory of the development of CP proposes that these issues result from a complex interaction between multiple biological, neuropsychological, family, and social risk factors. The biopsychosocial model of CP prosed by Dodge & Pettit (2003) also emphasizes that CP develop in a non-linear fashion, in which certain individual and environmental factors interact with each other at various stages of development (e.g., early childhood, adolescence) to increase the likelihood of chronic CP. More specifically, these researchers propose that certain life experiences and patterns of information processing tend to mediate the effects of biological and social risk factors on the development of CP (Dodge & Pettit, 2003).

Biological risk factors for conduct problems.

In terms of biological risk factors, results from twin and adoption studies indicate that aggressive and non-aggressive antisocial behaviours generally show a moderate degree of heritability, with aggressive behaviour showing a stronger genetic influence (Burt, 2009; Eley, Lichenstein, & Stevenson 1999; Eley, Lichtenstein , & Moffitt, 2003; Lichtenstein, Tuvblad, Larsson, and Carlström, 2007). Heritability estimates also appear to vary depending on sex and developmental period (Burt, 2009; Tuvblad, Eley, Lichtenstein, 2005; Wang, Niv, Tuvblad, Raine, & Baker, 2013). Interestingly, one large adoption study found that criminality in biological parents significantly predicted criminal convictions in adoptive individuals (over the age of 15), whereas there was no significant association between the criminal history of individuals and their adoptive parents (Mednick, Gabrielli, & Hutchings, 1984). Exposure to certain substances in utero, other than alcohol, have also been associated with higher rates of CP,

including opiates, methadone (de Cubas & Field, 1993), marijuana (Day, Leech, & Goldschmidt, 2011), cigarette smoke (Day, Richardson, Goldschmidt, & Cornelius, 2000; Gerteis et al., 2011) and lead (Dietrich, Ris, Succop, Berger, & Bornschein, 2001; Needleman, Riess, Tobin, Biesecker, & Greenhouse, 1996). It has been suggested, however, that genetic factors and exposure to prenatal toxins may indirectly lead to an increase risk for developing CP by contributing to neuropsychological dysfunction (Dodge & Pettit, 2003). Research has also consistently shown that CD is more prevalent in males than females (Cohen et al., 1993; Feehan, McGee, Raja, & Williams, 1994; Lumley, McNeil, Herschell, & Bahl, 2002; Maughan, Rowe, Messer, Goodman, & Meltzer, 2004; Offord et al., 1987), and more specifically, that males engage in more aggressive behaviours and commit more status and property offences than females (Dodge & Pettit, 2003; Lahey, et al. 2000).

Neuropsychological risk factors for conduct problems.

Self-regulation.

There have been a number of neuropsychological characteristics that have been more consistently associated with the development of CP in children and youth. Research has uncovered an important link between CP and the experience and regulation of emotions, which involve both automatic and effortful processes (Eisenberg & Morris, 2002; Frick & Morris, 2004). In terms of more automatic or involuntary processes, negative emotionality or a propensity to frequently display intense negative emotions (e.g., anger, frustration) has been associated with CP (Singh & Waldman, 2010; Waldman et al., 2011). More importantly, negative emotionality has been shown to predict CP in childhood (Eisenberg et al., 1996, Eisenberg et al. 2005; Eisenberg et al., 2009, Earls & Jung, 1987; McClowry et al., 1994) and young adulthood (Caspi, 2000). Effortful or voluntary executive processes involved in the

regulation of one's emotions and behaviours include attentional control and behavioural inhibition, and have been labeled "effortful control" by some researchers (Eisenberg et al., 1997; Eisenberg et al., 2009; Frick & Morris, 2004; Rothbart & Bates, 1998). Low effortful control (i.e., poor emotional and behavioural regulation) has also been linked to externalizing behaviour problems in childhood (Eisenberg et al., 1996; Eisenberg et al., 2000; Eisenberg et al. 2001; Eisenberg et al. 2005; Eisenberg et al. 2009), CD in adolescence (Dolan & Lennox, 2013), and has been shown to predict convictions for violent offences in early adulthood (Henry, Caspi, Moffit, & Silva, 1996). A more recent study set out to investigate the contributions of specific components of emotional competence and effortful control on disruptive behaviour problems in children. These researchers found that poor emotional regulation and cognitive inflexibility (i.e., difficulties shifting between disparate task demands) were associated with high rates of parent-rated CP, after controlling for ADHD symptoms and IQ; however, there was no significant relationship between CP and the child's inhibitory control or ability to identify/understand emotions (Duncombe, Havighurst, Holland, & Frankling, 2013). In contrast, other researchers have documented a significant link between CP and inhibitory control deficits in children, even after controlling for IQ and problems with attention (Raaijmakers et al., 2008; Riggs, Blair, & Greenberg, 2004; Schoemaker et al., 2012). Moreover, a more recent meta-analysis has documented a significant relationship between inhibitory control and externalizing problems early in development (i.e., preschool aged children), with a medium effect size of 0.24 ($p<0.001$) (Schoemaker, Mulder, Dekovic, & Matthys, 2013).

Interestingly, growing research suggests a U-shaped relationship between emotional reactivity and CP, as both high and low levels of emotional reactivity have been found to be associated with an increased risk of developing CP (Frick et al., 2003; Loney, Frick, Clements,

Ellis, & Kerlin, 2003). More specifically, *low* levels of autonomic arousal (for review see Lahey, Hart, Pliszka, Applegate, & McBurnett, 1993; Lorber, 2004; Matthys, Vanderschuren, & Schutter, 2013) and *low* levels of fear (Frick, Lilienfeld, Ellis, Loney, & Silverthorn, 1999; Shaw, Gilliom, Ingoldsby, & Nagin, 2003) have been documented in a subset of children and youth with CP. Frick and Morris (2004) suggest that children can possess two distinct clusters of temperamental characteristics that can increase the risk of CP in different ways. The first temperament style is defined by high negative emotional reactivity and low effortful control, while the second is defined by low levels of autonomic arousal and low levels of fear (i.e., fearlessness) (Frick & Morris, 2004). Moreover, research suggests that the latter temperament style is also associated with the presence of callous-unemotional traits, or a lack of empathy and guilt (Blair, 1999; Frick et al., 2003; Loney, Frick, Clements, Ellis, & Kerlin, 2003).

Attention and hyperactivity/impulsivity.

Given that problems with attentional and behavioural control have been consistently linked to CP, it is not surprising that children with ADHD have significantly higher rates of CD and ODD than children without ADHD (Ware et al., 2013), and that ADHD is the most common comorbid disorder associated with CP (McMahon & Frick, 2005). Youth with comorbid ADHD and CD are more likely to exhibit CP at a younger age (Moffitt, 1990), and tend to display more severe CP and a wider variety of antisocial behaviours, compared to those with pure CD (Waschbusch, 2002). Overall, there appears to be an interactive effect between the presence of ADHD and the severity and persistence of CP (Jensen, Martin, & Cantwell, 1997). Furthermore, impulsivity (Eisenberg et al. 2001; Eisenberg et al. 2005; Eisenberg et al. 2009; White et al., 1994) and hyperactivity (Herrenkohl, 2000; Klinteberg, Andersson, Magnusson, & Stattin, 1993; Satterfield & Schell, 1997) appear to be more salient risk factors for the development of CP than

inattention (Babinski, 1999; Lahey et al., 1994). One large study of clinically-referred children and youths found that those with the predominantly Hyperactive-Impulsive subtype of ADHD were more likely to demonstrate comorbid CP than individuals with the predominantly Inattentive subtype (Lahey et al., 1994).

Intellectual and academic functioning.

Children and youth with CP have also been shown to have lower IQ scores than controls, and display particularly low verbal reasoning abilities (Culberton, Feral, & Gabby, 1989; Lipsitt, Buka, & Lipsitt, 1990; Schonfeld, Shaffer, O'Connor, & Portnoy, 1988), even after controlling for other confounding variables such as parental IQ, SES, and race (Goodman, Simonoff, & Stevenson, 1995; Hirschi, & Hindelang, 1977; Lynam, Moffitt, & Stouthamer-Loeber, 1993). Conversely, Hogan (1999) found that the majority of studies linking IQ to CP failed to control for ADHD, and noted that the relationship between IQ and CP often failed to reach significance once this variable was accounted for, thus pointing to ADHD as a mediating factor. Consistent with this hypothesis are results from a meta-analytic examination of studies reporting IQ scores for groups of individuals with pure CP, pure problems with hyperactivity/impulsivity and attention (HIA), and comorbid CP and HIA. An analysis of effect sizes from these studies found that the comorbid CP-HIA and pure HIA groups had significantly lower Verbal and Performance IQ scores compared to controls, however, those with pure CP did not significantly differ from controls on either measure of intelligence (Waschbusch, 2002).

Learning difficulties appear to be common in children and youth with CP (Broder, Dunivant, Smith, & Sutton, 1981; Fergusson & Lynskey, 1998; Wilgosh & Paitich, 1982), and failing a grade has been shown to predict CP in population-based and longitudinal research (Offord, Boyle, & Racine, 1990; Velez, Johnson, & Cohen, 1989). Conversely, results from one

large longitudinal study utilizing structural equation modeling suggest that the relationship between CP and school achievement in adolescence is non-causal in nature, and can be fully accounted for by the effects of early common predictors of both outcomes, such as IQ and attention problems (Fergusson & Horwood, 1995). Accordingly, Frick et al. (1991) investigated academic underachievement in clinically-referred boys with pure CD, pure ADHD, and comorbid CD and ADHD, and found that only ADHD was associated with academic underachievement after statistically controlling for the co-occurrence of the two disorders.

Family risk factors for conduct problems.

Family structure and stability.

Higher rates of CP in children and youth has been found to be associated with family structure, specifically single parent families, families with cohabiting male partners, parental divorce, and parent-child separation (Ackerman, D'Eramo, Umylny, Schultz, & Izard, 2001; Amato, 2001; Amato & Keith, 1991; Fergusson, Horwood, & Lynskey, 1994b; Velez et al., 1989; Wadsworth, 1979). Some research suggests, however, that the associations between family structure and CP in children generally disappear once differences in quality of the home environment (Van Voorhis, Cullen, Mathers, Garner, & 1988) and parental antisocial behaviour are accounted for (Lahey et al., 1988a). Data from multiple longitudinal studies suggest that disruptions in the family structure, or changes in primary caregivers over time, significantly increase the risk of delinquency in youth (Thornberry, Smith, Rivera, Huizinga, & Stouthamer-Loeber, 1999). Moreover, one study investigating delinquency in a large population of children with a history of maltreatment found that children who were placed in out-of-home care were approximately 2 times more likely to engage in delinquent acts, compared to those who remained in their family home (Ryan & Testa, 2005). This study also found that decreased placement

stability (i.e., more placement changes) was associated with an increased risk for delinquency in males, but not females (Ryan & Testa, 2005).

Maladaptive parenting practices.

Maladaptive parenting practices have been consistently linked to the development and maintenance of CP. Low parental supervision and low parental involvement are two family factors that have been consistently found in the literature to be significantly associated with CP in children and youth (for review see Loeber and Stouthamer-Loeber, 1986; Farrington & Hawkins, 1991; Van Voorhis et al., 1988). Moreover, ongoing parental neglect has been shown to predict recidivism in juvenile offenders (Ryan, Williams, & Courtney, 2013). Nonetheless, more recent research suggests many studies investigating the effects of parental monitoring on CP in childhood have utilized tools that truly measure parental *knowledge* of children's activities, and that parental knowledge of children's activities is actually more closely related to how much information children share with their parent, rather than active parenting behaviours (Eaton, Krueger, Johnson, McGue, & Iacono, 2009; Kerr & Stattin, 2000; Stattin & Kerr, 2000). Moreover, one study found that the significant relationship between variables associated with parental monitoring and child delinquency generally disappear once the child's personality traits are accounted for (Eaton, Krueger, Johnson, McGue, & Iacono, 2009).

Inconsistent, harsh, and physically aggressive discipline practices have been linked to externalizing behaviours in youth (Dodge, Pettit, & Bates, 1994; Farrington & Hawkins, 1991; Glueck & Glueck, 1950; McCord, 1991; Stormshak, Bierman, McMahon, Lengua, & CPPRG, 2000). On the most severe end, research had consistently shown that extreme parenting behaviours or maltreatment, including physical abuse, sexual abuse, and neglect, are associated with a significant increase in the risk of CP in both males and females (Deater-Deckard &

Dodge, 1997; Dodge, Bates, & Pettit, 1990; Dodge, Pettit, Bates, & Valente, 1995; Green, Russo, Navratil, Loeber, & 1999; Ryan & Testa, 2005; Maliglio, 2014; Maxfield & Widom, 1996; Stouthamer-Loeber, Loeber, Homish, & Wei, 2001; Widom, 1989). Utilizing a prospective longitudinal study design, Dodge et al. (1990) found that physical abuse early in life significantly predicted aggressive behaviour in childhood, even after controlling for a number of individual, family, and social factors. Another group of researchers showed that child maltreatment (i.e., physical, sexual, emotional abuse and/or neglect) was more strongly associated with overt (e.g., aggression, violence), rather than covert CP in youth (e.g., property offences) (Stouthamer-Loeber et al., 2001).

Parental psychopathology.

Parental psychopathology, specifically APD and criminality, appears to have a particularly strong influence on the development of CP in children and youth (Biederman, Munir, & Knee, 1987; Farrington, Jolliffe, Loeber, Stouthamer-Loeber, & Kalb, 2001; Frick et al., 1992; Frick & Loney, 2002; Jary & Stewart, 1985; Lahey et al., 1988b; Repo-Tiihonen et al., 2010; Robins, West, & Herjanic, 1975; DeBlois, & Cummings, 1980). Frick and colleagues (1992) found that when parental APD and measures of parenting practices (e.g., maternal supervision, consistent discipline) were simultaneously entered into logit-model analyses, only parental APD significantly predicted CD in children. Moreover, some studies suggest that paternal APD or criminality is more strongly associated with CP in children than is maternal antisocial or delinquent behaviour (Frick et al., 1992; Lahey et al., 1988b; Stewart et al., 1980). Experiencing parental incarceration during childhood also appears to be a strong predictor of CP in males, even after accounting for parental convictions, child risk factors, and other types of parental separation (e.g., loss, death) (Murray, & Farrington, 2005). Children and youth with CP

are also more likely to have parents with substance abuse issues compared to other clinically referred youth (Lahey et al., 1988; Loeber, Green, Keenan, & Lahey, 1995; Stewart et al., 1980) — a finding which is consistent with the well-documented link between PAE and CP. Nevertheless, it is unclear whether the relationship between parental psychopathology and CP in children reflects a biological or social learning process, or both (Bassarath, 2001; Frick et al., 1992), as parents genetically pass on temperamental and neuropsychological traits to their children, and also play a major role in the child's socialization and shaping of the home environment.

Social risk factors for conduct problems.

A number of studies have found a link between the affiliation with deviant peers and CP in youth (Fergusson, Swain, & Horwood, 2002; Kaufmann, Wyman, Forbes-Jones, & Barry, 2007; Keijsers et al., 2012; Patterson & Dishion, 1985), and research shows that youths typically commit delinquent acts in groups rather than alone (Zimring, 1981). Problems with peer relationships and peer rejection have also been directly linked to the development of later CP (Dodge et al., 2003; Kupersmidt & Coie, 1990; Kupersmidt, Burchinal, & Patterson, 1995; Laird, Jordan, Dodge, Pettit, & Bates, 2001; Parker & Asher, 1987, Roff & Wirt, 1984). Path analysis has found support for the hypotheses that peer rejection/isolation actually leads to later association with defiant peers (Rudolph et al., 2014). Moreover, peer rejection seems to play a more important role in the development of CP than does involvement with deviant peers. Results from one study using more sophisticated statistical modeling techniques to control for externalizing behaviour across time, found that early rejection by peers, but not affiliation with antisocial peers, predicted externalizing behaviour in early adolescence (Laird, Jordan, Dodge, Pettit, & Bates, 2001). Moreover, another study found that affiliating with aggressive and

disruptive friends predicted an increase in delinquency in moderately disruptive boys, however, this relationship was not significant in highly disruptive or moderately to highly compliant boys (Vitaro, Tremblay, Kerr, Pagani, & Bukowski, 1997).

Low socioeconomic status (SES) has also been linked to CP in children and adolescents (Dodge, Pettit, & Bates, 1994; Fergusson, Swain-Campbell, & Horwood, 2004; Frick, Lahey, Hartdagen, & Hynd, 1989; Lahey et al., 1995; Loeber, et al., 1995; Velez et al., 1989), however, SES is correlated with a number of other family and community factors that may have a more direct influence on the development of CP. Peeples and Loeber (1994) found that living in a disadvantaged neighbourhood was associated with an increase in the frequency and severity of adolescent delinquency, whereas poverty/welfare was not significantly related to delinquency outcomes (Peeples & Loeber, 1994). Studies utilizing structural equation modeling (SEM) have found that parenting behaviours or family factors largely mediate the relationship between low SES and the development of CP (Dodge, Pettit, & Bates, 1994; Larzelere and Patterson, 1990; McCoy, Frick, Loney, & Ellis, 1999). Accordingly, Fergusson, Swain-Campbell, and Horwood (2004) found that SES failed to remain a significant predictor of youth delinquency after controlling for other important individual, family, school, and peer factors. Other research has found that community factors (e.g., neighbourhood disorder) have only an indirect effect on individual delinquency through parenting behaviours and peer factors (Chung & Steinberg, 2006).

Developmental Trajectory of Conduct Problems

CP can appear as early as preschool (Kim-Cohen et al., 2005), but typically begin to emerge between middle childhood and middle adolescence, and the initial onset of CP is rarely seen after the age of 16 (APA, 2013). Research suggests a general increase in antisocial

behaviour occurs between middle childhood to late adolescence, followed by a steep decrease after the age of 17 (Fergusson & Horwood, 2002; Lahey et al., 2006; Loeber, Stouthamer-Loeber, Van Kammen, & Farrington, 1989). Moffitt (1993) proposed the following two distinct pathways for the development of CP in order to explain age/prevalence trends: life-course-persistent (i.e., individuals who continuously exhibit some form of CP across development periods) and adolescence-limited (i.e., individuals who exhibit CP only during the period of adolescence). Subsequent research supporting Moffitt's theory ultimately led to the delineation of childhood-onset and adolescent-onset types of CD in the DSM-IV (APA, 2000) and DSM-V (APA, 2013). A number of longitudinal studies measuring CP across developmental periods have consistently revealed a distinct group of individuals who exhibit more persistent CP, with an onset in childhood, and a larger group of individuals with adolescent-onset CP, who often remit in adulthood (Barker, Oliver, & Maughan, 2010; Fergusson & Horwood, 2002; Lahey et al., 2006; Odgers et al., 2007; Wiesner, Kim, & Capaldi, 2005). Compared to those with adolescent-onset CP, individuals with childhood-onset CP have been shown to exhibit higher rates of parent- and self-reported CP (Lahey et al., 2006), more severe aggressive or violent behaviour (Lahey et al., 1998; Moffitt, Caspi, Harrington, Milne, 2002), a higher number of self-reported offences during adolescence (Fergusson & Horwood, 2002) and in young adulthood (Moffitt et al., 2002), a higher number of adult convictions (Moffitt et al., 2002), and are more likely to meet the criteria for APD in early adulthood (Moffitt et al., 2002; Wiesner et al., 2005). Further supporting these two distinct pathways to CP are findings that youth with childhood-onset persistent CP are more likely to display a constellation of biological, neuropsychological, family, and social risk factors, than individuals with adolescent-onset CP (Fergusson & Horwood, 2002; Lahey et al., 1998; Lahey et al., 2006; Odgers et al., 2007).

Moffitt (1993) proposed that adolescent-onset CP reflect a misguided attempt to gain a sense of adult status or form an adult identity by breaking societal norms, and predicted that these offenders would gradually desist from delinquency as they emerged into adulthood. More recent research has questioned the transitory nature of CP and resilience in the adolescent-onset population. For example, one study found that individuals with adolescent-onset CP did not differ from those with childhood-onset CP in terms of self-reported CD symptoms at 26 years of age (Odgers et al., 2007), and another study found no difference between groups in terms of self-reported property offences, drug offences, and rule-violations committed in early adulthood (Moffitt et al., 2006). Odgers et al. (2007) also found that, when compared to individuals with low levels of CP across development, individuals with adolescent-onset CP had poorer physical health outcomes and were more likely to have received mental health services, have victimized others, and have substance abuse issues in adulthood. Other studies have found that youth with adolescent-onset CP possess a risk factor profile that is somewhere in between that displayed by childhood-onset youth and non-offending or low CP youth (Fergusson & Horwood, 2002; Odgers et al., 2007). Thus, it appears that more research is needed to track the long-term outcomes of those with adolescent-onset CP.

Longitudinal research has also found evidence to support the further differentiation of pathways or trajectories of CP (e.g., high versus low level offenders, chronic versus decreasing offenders), indicating that Moffitt's childhood- versus adolescent-onset model may be an oversimplification of offending trajectories (Barker et al., 2010; Fergusson & Horwood, 2002; Lahey et al., 2006; Moffitt et al., 2002; Odgers et al., 2007; Wiesner & Capaldi, 2003; Wiesner, Kim, & Capaldi, 2005). Of particular interest, a third distinctive subtype of individuals has been identified. They display high levels of CP in childhood, yet do not continue to display delinquent

behaviour during adolescence (Barker et al., 2010; Moffitt et al., 2002; Odgers et al., 2007). The term childhood-limited CP was proposed to describe this subset of individuals, however, the prognosis of these individuals is still up for debate (Barker et al., 2010; Moffitt et al., 2002; Odgers et al., 2007).

Rationale

Research suggests that a number of biological, neuropsychological, family, and social risk factors likely contribute to the development of CP in youth. Thus, research aimed at determining the predictors of CP in youth with FASD should also take into consideration the relative influence of the above-mentioned risk factors across domains, in addition to the effect of PAE. Identifying which neuropsychological deficits put those with FASD most at risk for developing CP is particularly critical, as PAE can cause a diverse array of impairments across domains (i.e., memory, language, EF) and a clear neuropsychological profile for FASD has yet to be determined (Mattson et al., 2011). Moreover, many of the neuropsychological deficits that have been more consistently documented in children and youth with FASD, such as problems with hyperactivity, behavioural regulation, and social cognition (Mattson et al. 2011), also appear to be strongly associated with the development of CP in the general population. Remarkably, no study to date has investigated the emotional and behavioural regulation abilities of children with FASD as a potential risk factor for CP, despite growing evidence to support the critical role of higher-order executive functions in the development of disruptive behaviour disorders (Frick & Morris, 2004).

Importantly, PAE (and thus FASD) often occurs in the ecological context of other family risk factors, which may also contribute to the development of CP (Malone & Koren, 2012). It is especially critical that the effects of biological and family risk factors be examined, such as

parental psychopathology, disruptions in caregiving, and experience of abuse, because these risk factors often co-occur with PAE or parental alcohol abuse (Astley, 2010; Cannon et al., 2012; Fergusson, Lynskey, & Horwood, 1996; Hans, 1999; Sarkola, Kahila, Gissler, & Halmesmäki, 2007; Sood et al., 2001; Streissguth, Barr, Kogan, & Bookstein, 1996; Streissguth et al., 2004; Werner, 1986) and have also been strongly linked to the development of CP in the general population. A review of the literature on substance-abusing women found that the majority have at least one comorbid mental health disorder, with higher rates of APD compared to samples of non-substance-abusing women (Hans, 1999). A recent population-based study found that mothers of children with FASD ($n = 353$) were significantly more likely to have confirmed alcoholism and a history of mental health problems, compared to mothers of children who were not affected (Cannon et al., 2012). A database review of 1,400 individuals assessed for FASD at one of the seven Washington State FAS Diagnostic & Prevention Network clinics revealed that 70.5% were not living with either of their biological parents at the time of diagnosis (Astley, 2010). Moreover, 50% of children born to a Finnish cohort of mothers seen at a prenatal clinic for women with substance abuse issues ($N = 526$) were taken into care at some point during a 2 to 12-year follow-up period (Sarkola et al., 2007). Sood et al. (2001) compared maternal and family characteristics of mothers who reported abstinence ($n = 117$), low levels of alcohol use ($n = 323$), and moderate to heavy levels of alcohol use ($n = 66$) during pregnancy. Increasing levels of alcohol use during pregnancy were associated with increased maternal age, lower maternal education, higher rates of cigarette and cocaine use during pregnancy, higher levels of postnatal alcohol intake, an increased likelihood of custody changes for the child, a decreased likelihood that the child was in the custody of the biological mother, higher rates of paternal alcohol and drug use, and lower SES and HOME inventory scores. Moreover, children of parents with

substance abuse issues, particularly alcoholism, are at a significantly increased risk of experiencing maltreatment (e.g., physical abuse, sexual abuse, or neglect) (Brown, Cohen, Johnson, & Salzinger, 1998; Chaffin, Kelleher, & Hollenberg, 1996; Egami, Ford, Greenfield, & Crum, 1996; Famularo, Stone, Barnum, & Wharton, 1986). Accordingly, high rates of maltreatment have been reported in cohorts of individuals with FASD (e.g., Streissguth et al., 1996; 2004; Todorow, 2011), however, surprisingly little research has investigated the contribution of child maltreatment on the development of CP in this clinical population.

Finally, only one study to date (Lynch et al., 2003) has examined the development of CP in a population of youth formally diagnosed with FASD using a biopsychosocial and developmental framework. Individual neurocognitive deficits related to PAE, as well as the experience of adverse family and social risk factors, affect the individual as a whole, and thus, their relative effects should be simultaneously examined on development of CP in this population (Malone & Koren, 2012; Rasmussen, Andrew, Zwaigenbaum, & Tough, 2008). Previous studies have examined specific clusters of potentially relevant individual and environmental factors in isolation, which is problematic, as many of these risk factors (particularly biological and family factors) are correlated or frequently co-occur (Frick et al., 1992). Finally, the specific developmental trajectory of CP in youth with FASD has yet to be examined, and thus, it is currently unclear whether groups of youth with FASD can be meaningfully differentiated according to the age-of-onset of CP.

Objectives

The main objective of the current study is to further elucidate the risk factors and pathways that lead to CP in individuals with FASD. More specifically, we aim to provide a more detailed investigation of the effects of specific neuropsychological deficits associated with PAE,

as well as biological, family, and social risk factors that often co-occur with maternal substance use, on the development of CP in a clinical sample of adolescents with FASD. Predictor selection was guided by the biopsychosocial model of CP, and informed by a thorough review of previous research examining risk factors of CP in the general population, as well as in individuals with PAE. This resulted in the careful selection of the following potential biological, neuropsychological, family, and social predictors: male sex (biological), maternal criminality (biological/family), emotional/behavioural regulation skills (neuropsychological), ADHD (neuropsychological), family instability (family), abuse history (family), and problem with peer relations (social). We focused our investigation on specific risk factors that have been shown in previous research to have a strong and unique predictive effect on the development of CP. Finally, age will also be included as a potential predictor, in order to account for the developmental trajectory of CP. Ultimately, we hope this study will shed light on which specific risk factors are most influential, so that they can be targeted in early intervention efforts to reduce the likelihood of the development of significant CP in adolescents and young adults with FASD. Research that directly informs intervention efforts aimed at mitigating CP in this high-risk population is scarce and greatly needed.

Research Questions

- 1) What is the prevalence of various types of CP (i.e., symptoms of ODD, CD, defiance/aggression, school suspensions, and criminality) in a population of Canadian adolescents previously diagnosed with FASD and relative to the general population?
- 2) How prevalent are specific biological, neuropsychological, family, and social risk factors for CP in a population of Canadian adolescents with FASD? Do adolescents with FASD commonly experience multiple risk factors for CP?

- 3) How do biological, neuropsychological, family, and social factors, which have been found to significantly increase the risk for CP in the general population, function together to predict the development of CP in adolescents with FASD?
- 4) What is the continuity of CP in individuals with FASD across the follow-up period (i.e., middle childhood to adolescence)? Do symptoms of ODD and CD measured at diagnosis significantly predict symptoms of ODD and CD measured at follow-up in adolescence? Are there significant sex differences in the continuity of CP in individuals with FASD?

Hypotheses

- 1) The majority of Canadian adolescents with FASD (i.e., over 50%) will exhibit clinically significant symptoms of ODD and CD, and approximately 50% of adolescents will have a history of criminality.
- 2) The majority of Canadian adolescents with FASD will have a diagnosis of ADHD and exhibit clinically significant difficulties with emotional/behavioural regulation. Moreover, Canadian adolescents with FASD will experience high rates of family and social risk factors, including parental criminality, family instability, physical/sexual abuse, and problems with peer relations. Finally, the majority of youth with FASD will have experienced three or more risk factors for CP, which span across domains (e.g., neuropsychological, family).
- 3) Biological and family factors will be the most significant predictors of CP in adolescents with FASD, including male sex, parental criminality, family instability, and history of physical/sexual abuse.
- 4) Symptoms of ODD and CD at diagnosis will have a significant and positive predictive relationship with symptoms of ODD and CD at follow-up, regardless of sex.

Methods

Participants

The current study was conducted as part of a larger prospective follow-up study of a cohort of children diagnosed with FASD in the Motherisk FASD Clinic at The Hospital for Sick Children (HSC), between October 2003 and July 2012. The Motherisk Clinic is one of the leading Canadian diagnostic facilities for FASD, providing specialized diagnostic services to children across Ontario. Individuals were initially brought to the Motherisk Clinic by an array of different child care providers including Children's Aid Society (CAS) workers, adoptive/foster parents, biological relatives, or on occasion, by the biological parents. Diagnostic assessments included a physical, neurological, and dysmorphology evaluation, as well as a comprehensive neuropsychological assessment. Neuropsychological assessments included a clinical interview and the administration of a carefully selected battery of standardized tests, in order to evaluate the presence of impairment in various CNS domains required for an alcohol-related diagnosis according to the Canadian diagnostic guidelines provided by Health Canada (Chudley et al., 2005). Parents, caregivers, and/or legal guardian also completed a structured Case History questionnaire, in order to gather clinically relevant data regarding pregnancy/birth history, developmental history, medical history, mental health history, family history, educational history, and social history.

The potential participant pool for the larger prospective follow-up study included individuals who were assessed and diagnosed with an FASD at the Motherisk Clinic between October 2003 and July 2012, and who were between the ages of 11 and 18 at the time of follow-up ($n = 142$). Recruitment for the follow-up study occurred over a four year period (August 2010 to December 2014) and follow-up assessments were conducted at least two years post-diagnosis.

Of the 142 potential participants, 81 were contacted and agreed to participate in the follow-up study, along with their parents/legal guardians and/or primary caregivers. Although only 57% of eligible participants were included in the follow-up study, the study sample was shown to be representative of the total population of eligible participants diagnosed with an alcohol-related disorder at the Motherisk FASD Clinic with regard to sex, race, guardianship, age of diagnosis, year of diagnosis, diagnostic category, and intellectual ability (Todorow, 2011). Of the 81 adolescents included in the larger prospective follow-up study, 59 met the inclusion and exclusion criteria for the current study (Figure 1). It is important to note, however, that one adolescent had a significant intellectual delay (IQ = 60), as well as receptive language impairments, and thus was excluded from completing the youth-report GAIN-SS, as he did not understand the meaning of many of the questionnaire's items.

Inclusion criteria.

- 1) Participated in a comprehensive diagnostic assessment for FASD at the Motherisk FASD Clinic between October 2003 and July 2012, and
- 2) Received an alcohol-related diagnosis within the spectrum of FASD, according to Canadian diagnostic guidelines, and
- 3) Between 11 and 18 years of age by the period for follow-up (August 2010 to December 2014).

Exclusion criteria.

- 1) History of severe head trauma or a neurological/medical comorbidity post-diagnosis that was not related to prenatal alcohol toxicity (e.g., bacterial meningitis), or
- 2) Failure to obtain signed informed consent, or
- 3) Missing predictor data, or

- 4) Siblings. Full biological sibling pairs were identified through a case file review and only one sibling was randomly selected for inclusion in order to protect against violations of statistical independence.

Recruitment.

Contact information for potential participants, which was collected during their initial diagnostic assessment at the Motherisk Clinic, was accessed for recruitment purposes. The KidCare database at the HSC was also searched in an effort to update contact information. Potential participants (i.e., adolescents and caregivers) were initially contacted by the psychologist for the Motherisk Clinic, Dr. Ellen Fantus, via an introductory research invitation letter, which asked individuals to indicate their interest in the proposed study by responding via telephone. Follow-up telephone calls were made to those who did not respond to the introductory letter within two weeks.

Because many of the potential participants were under the custody and care of a CAS agency at the time of their initial diagnostic assessment, partnerships were established between various CAS agencies in the general Toronto area, in order to gain updated contact information regarding the adolescents' current caseworker. For all adolescents who were under CAS guardianship, a telephone conversation was conducted with the adolescent's caseworker in order to determine the most appropriate informant for the follow-up measures (e.g., foster parent, prime group home worker, caseworker). The study and consent process was then verbally discussed with potential participants over the telephone and adolescents' competency and capacity to consent for themselves was formally assessed. Following verbal consent for inclusion in the follow-up study, appropriate consent/assent forms were mailed to adolescents, as well as parent/legal guardians and/or primary caregivers.

Study Procedure

Data for predictors were obtained from a structured Case History questionnaire, as well as a number of standardized behavioural questionnaires, completed by parents/legal guardians and/or primary caregivers as part of the initial diagnostic assessment. Adolescents' abuse history, family instability, and ADHD diagnosis were updated according to information provided by caregivers at follow-up, via a semi-structured interview. CP in adolescents with FASD was evaluated at follow-up using standardized parent- and self-report questionnaires, as well as information obtained from semi-structured interviews completed with parents/legal guardians and/or primary caregivers (see below for details). The semi-structured interview was developed as part of the larger observation follow-up study in order to obtain data on specific long-term outcomes of interest. The "Case History Interview" was conducted via telephone by Ms. Todorow with the adolescent's parent, legal guardian or primary caregiver. The Case History Interview took approximately one hour to complete and was comprised of 110 questions, the majority being close-ended questions utilizing various response formats, including dichotomous, multiple choice, free choice and numerical rating scales. The interview was conducted with the individual who had the greatest overall knowledge of the adolescent's current and past behaviours (e.g., biological parent, grandparent, adoptive parent, foster parent, prime group home worker, social worker).

The SES of families was also measured at follow-up using the Hollingshead Four Factor Index of Social Status. The Hollingshead provides an index of social class which was calculated using the parents', legal guardians' or primary caregivers' highest level of education and classification of employment (Hollingshead, 1975). All standardized questionnaires were sent by mail to be completed by the appropriate informant, along with stamped, return-addressed

envelopes. Adolescents with reported difficulties with reading or reading comprehension were provided the opportunity to complete the self-report questionnaires over the telephone, in an interview format. Parent-report questionnaires were completed by the adult caregiver determined to have the greatest knowledge of the adolescent's current behaviour. All study procedures were previously approved by the Research Ethics Board at the HSC and Human Participants Review Sub-Committee at York University.

Predictor Measures

Parental criminality.

Parents, legal guardians and/or primary caregiver completed a structured Case History questionnaire, as part of their child's initial diagnostic assessment for FASD, which included yes/no questions pertaining to the criminal history of the child's biological parents. More specifically, informants indicated whether or not each of the child's biological parents had a criminal record or a history of incarceration. Unfortunately, the child's biological father's history was unknown in approximately one third of cases, and thus, only maternal criminality could be accurately reported on. Therefore, individuals with a biological mother who was reported to have either a criminal record or history of incarceration was coded as having a positive history of maternal criminality. Maternal criminal history was selected as a measure of parental antisocial behaviour, as involvement with the legal system and incarceration are overt outcomes that are more likely to be known to the child's alternative legal guardian or primary caregivers, as opposed to specific mental health diagnoses such as APD. Moreover, antisocial attitudes and behaviours could not be directly assessed in biological parents, as the majority of the children assessed at the Motherisk FASD Clinic were estranged from their biological parents.

Emotional/behavioural regulation skills.

A parent/legal guardian or primary caregiver completed the Parent Form of the Behaviour Rating Inventory of Executive Function (BRIEF) (Gioia, Isquith, Guy, & Kenworthy, 2000) as part of their child's initial diagnostic assessment for FASD. The BRIEF is a norm-referenced questionnaire used to measure EF behaviours in individuals between five and 18 years of age. The BRIEF yields eight theoretically and empirically derived subscales, reflecting features of EF including: Inhibit, Shift, Emotional Control, Initiate, Working Memory, Plan/Organize, Organization of Materials and Monitor. The Inhibit, Shift, and Emotional Control subscales comprise the Behavioural Regulation Index, while the remaining subscales comprise the Metacognition Index. Raw scores for each subscale and index are transformed into *T*-scores ($M = 50$, $SD = 10$), providing a similar metric for all subscales and indexes, as well as information about an individual's scores relative to the scores of individuals in the normative sample. *T*-scores of 65 and above (i.e., 94th percentile and above) are considered clinically significant. *T*-scores on the Behavioural Regulation Index were used as a measure of individuals' emotional and behavioural regulation abilities at the time of diagnosis, as the subscales (and corresponding items) that comprise this index tap into an individual's ability to resist impulses, inhibit behaviours when needed, shift cognitive resources and behaviours depending on situational and task demands, and effectively modulate or control emotional responses. Test-retest correlations for the Behavioural Regulation Index on the Parent Form (measured after an average interval of two and three weeks) ranged from .80 to .84. Factor analysis supported a two-factor model, with subscales consistently loading onto either the Behavioural Regulation or Metacognition factor. Evidence for convergent validity comes from the fact that measures on the BRIEF correlated in an expected fashion with other rating scales' measures of attention and behavioural functioning

(e.g. CBCL, Conners Rating Scale, Behaviour Assessment System for Children) (Gioia et al., 2000).

Attention-deficit/hyperactivity disorder.

Whether the adolescent had been previously diagnosed with ADHD by a family physician, psychiatrist, or psychologist was ascertained by cross-referencing information provided by parents, legal guardians and/or primary caregivers via the Case History questionnaire and semi-structured interview, conducted at diagnosis and follow-up, respectively. More specifically, the Case History questionnaire asked caregivers to indicate whether their child had previously been diagnosed with ADHD, and during the semi-structured interviews, caregivers were asked if their child had ever been diagnosed with a mental disorder defined by the DSM-IV and to specify the given diagnosis/diagnoses. Moreover, each participant's comprehensive psychodiagnostic reports were reviewed to determine whether a comorbid diagnosis of ADHD was given or confirmed at the time of the initial diagnostic assessment for FASD. All sources of information were considered together in order to make a dichotomous rating of whether or not the adolescent had been diagnosed with ADHD (regardless of subtype) and any inconsistency between reports of ADHD diagnostic status was clarified with caregivers. In some cases, an ADHD diagnosis had been provided following additional psychological/psychiatric assessments conducted during the period between FASD diagnosis and follow-up.

Social problems.

A parent/legal guardian or primary caregiver also completed the Conners' Parent Rating Scales-Revised: Long Form (CPRS-R:L) (Conners, 1997) as part of the initial diagnostic assessment. The CPRS-R:L is a norm-referenced parent-report questionnaire, which specializes in assessing the symptoms of ADHD and related problems in children and youth (ages 3 to 17).

Endorsements on the CPRS-R:L load onto the following 14 subscales: Oppositional, Cognitive Problems/ Inattention, Hyperactivity, Anxious-Shy, Perfectionism, Social Problems, Psychosomatic, ADHD Index, Conners' Global Index: Restless-Impulsive, Conners' Global Index: Emotional Liability, Conners' Global Index: Total, DSM-IV Symptoms: Inattentive, DSM-IV Symptoms: Hyperactive-Impulsive, and DSM-IV Symptoms: Total. Raw scores are converted into *T*-scores ($M = 50$, $SD = 10$) and *T*-scores of 65 and above (i.e., 94th percentile and above) are considered to represent a clinically significant problem. *T*-scores for the Social Problems subscale of the CPRS-R:L were used to quantify problems with peer relations at the time of diagnosis. Test-retest reliability coefficients (after a six to eight week interval) for the Social Problems subscales of the CPRS-R:L was .82. Convergent validity is supported by moderate correlations between identical scales on parent- and teacher-report forms. Discriminant validity is supported by the ability of the CPRS-R:L to accurately distinguish between individuals with a diagnosis of ADHD, individuals with "emotional problems" determined by a mental health professional, and those without attentional or emotional concerns (Conners, 1997).

Family instability.

A measure of family stability was created by dividing the total number of placements with different caregivers by the child's age at follow-up. Therefore, higher numbers reflect greater family instability experienced by the individual across his/her lifespan. As part of the semi-structured interview completed at follow-up, parents/legal guardians or primary caregivers reported the total number of different homes with different caregivers experienced by adolescents (i.e., total number of changes in primary caregiver). If the adolescent had returned back to a previous caregiver at one point in their lives, this was not counted as an additional placement. For example, if an individual was originally in the care of a biological parent and

then was placed with a foster family, yet was ultimately returned to the care of their biological parent, this was coded as two total placements with different caregivers.

Child abuse.

Adolescent's history of physical and sexual abuse was ascertained by cross-referencing information provided by parents, legal guardians and/or primary caregivers via the Case History questionnaire and semi-structured interview, conducted at diagnosis and follow-up, respectively. At both time points, caregivers were asked to indicate whether their child had a confirmed history of physical abuse and sexual abuse. History of abuse of those under the care of a CAS agency was also confirmed with the social worker assigned to the individual's case, via a review of official social service records, whenever possible. All sources of information were considered together in order to make a dichotomous rating of a positive or negative history of physical or sexual abuse. Any discrepancies between reports of each type of maltreatment were resolved by reviewing all data with the supervising clinical psychologist, Dr. Ellen Fantus, and coming to a consensus decision, with stronger weight given to data collected at follow-up, in order to account for any maltreatment that occurred during the period between diagnosis and follow-up.

Outcome Measures

Various forms of CP in adolescents with FASD were measured using a variety of methods and multiple informants. The use of a variety of assessment methods (i.e., interviews with caregivers, various behavioural rating scales) and multiple informants (i.e., youth, caregivers) has been deemed critical by experts in the field in order to gather an accurate, reliable, and comprehensive representation of CP in youth (for review see McMahon & Frick, 2005).

Caregiver-reported continuous measures of conduct problems.

A parent/legal guardian and/or primary caregiver of individuals diagnosed with FASD completed the Child Behaviour Checklist for Ages 6-18 (CBCL) at diagnosis and at follow-up. The CBCL is a norm-referenced, parent-report questionnaire, which assesses behavioural and emotional problems, as well as competency in social relationships, activities, and school performance. The CBCL yields *T*-scores ($M = 50$, $SD = 10$) for eight Syndrome scales including: Anxious/Depressed, Withdrawn/Depressed, Somatic Complaints, Social Problems, Thought Problems, Attention Problems, Rule-Breaking Behaviour and Aggressive Behaviour. The CBCL also produces total *T*-scores for Internalizing and Externalizing syndromes, as well as a Total Problems. Furthermore, the CBCL generates *T*-scores for scales reflecting criteria for DSM-IV mental health diagnoses, including Affective Problems, Anxiety Problems, Somatic Problems, Attention Deficit/Hyperactivity Problems, Oppositional Defiant Problems and Conduct Problems. For the Syndrome and DSM-Oriented scales, *T*-scores between 65 and 69 (93rd to 97th percentile) are considered to be in the borderline clinical range, while *T*-scores greater or equal to 70 (98th percentile and above) are in the clinical range. Research shows that scores in either the borderline or clinical range significantly differentiate between clinically-referred children/youth for behavioural and/or emotional problems and non-referred children/youth that are similar in terms of demographic variables (Achenbach & Rescorla, 2001).

T-scores on the DSM-Oriented Oppositional Defiant Problems and Conduct Problems scales of the CBCL completed at follow-up were used as continuous outcomes measures of CP in adolescence. The DSM-Oriented Oppositional Defiant and Conduct Problems scales were selected over the empirically derived Rule-Breaking and Aggressive Behaviour Syndrome scales because they map directly onto the diagnostic criteria for specific disruptive behaviour disorders

outlined in the DSM-IV (ODD and CD, respectively), which facilitates the interpretation of the data. These scales also correspond to the recently released DSM-V criteria for ODD and CD, as the criteria for these two disorders have largely remained unchanged (i.e., the same number and description of behaviours/symptoms are used to make a diagnosis) (APA, 2000, 2013). In comparison to the empirically derived scales, when creating the DSM-Oriented scales, clinical judgement was used to remove items measuring more developmentally normative behaviours/characteristics or items that were weaker indicators of psychopathology in childhood, and thus, scores on the DSM-Oriented scales represent a more focused measure of psychopathology (Lacalle, Ezpeleta, & Doménech, 2012). Most importantly, the use of the DSM-Oriented scales allows for a more direct comparison of this study's findings to the results from previous research on CP and FASD, as most studies have used a clinical-diagnostic approach to measuring CP (e.g., the presence of an ODD/CD diagnosis, count of ODD/CD symptoms endorsed on standardized diagnostic interviews). Finally, research suggests that the clinically derived DSM-Oriented scales and the empirically derived Syndrome scales of the CBCL have similar psychometric properties (Achenbach, Dumenci, & Rescorla, 2003; Ebetsutani et al., 2010; Nakamura, Ebetsutani, Bernstein, & Chorpita, 2009).

T-scores on the Oppositional Defiant Problems and Conduct Problems scales of the CBCL completed by caregivers at diagnosis were also examined as potential predictors of *T*-scores on the same scales at follow-up in a separate analysis, in order to evaluate the continuity or continuity of CP across the follow-up period (middle childhood to adolescence).

Test-retest reliability coefficients (after a mean of eight days) for the Oppositional Defiant and Conduct Problems scales of the CBCL were .85 and .93, respectively. The content validity of the CBCL items has been supported by four decades of research and revision, as well

as by the findings that all items significantly discriminate between referred and non-referred children, matched on demographic factors such as, age, gender, socioeconomic status and ethnicity (Achenbach & Rescorla, 2001).

Parents/legal guardians and/or primary caregivers also completed the Conners Rating Scales 3rd Edition – Short Form (Conners 3-P(S)) as part of the follow-up assessment. The Conners 3-P(S) is a norm-referenced tool which specializes in assessing symptoms of ADHD, and common co-occurring problems in children and adolescents, including CP (Conners, 2008). The Conners 3-P(S) can be used to assess individuals between the ages of six and 18, and includes a subset of items from the full-length Conners 3. This measure includes a subset of items that comprise the five Content Scales on the full-length version, plus a complete replication of the Positive and Negative Impression validity scales. The Conners 3-P(S) produces *T*-scores for the following Content Scales: Inattention, Hyperactivity/Impulsivity, Learning Problems, Executive Functioning, Defiance/Aggression, and Peer Relations. The Conners 3-P(S) uses a 5-level classification system to group *T*-scores into clinically relevant categories: Very Elevated, Elevated, High Average, Average and Low. *T*-scores ≥ 70 (i.e., above the 97th percentile) are in the Very Elevated range and indicate an area of significant concern, while *T*-scores between 65 and 69 (i.e., 93rd to 97th percentile) are considered in the Elevated range and also indicate significant concerns (Conners, 2008).

T-scores on the Defiance/Aggression scale of the Conners 3-P(S) completed at follow-up were used as an additional continuous outcomes measure of CP in adolescence. The test-retest reliability coefficient for the Defiance/Aggression scale of the Conners 3-P(S) after a two- to four-week interval is .70 (adjusted $r = .92$). The Conners 3-P(S) is strongly associated with the full-length version, with correlation coefficients for all subscales above .90. Factor analyses have

confirmed the factor structure of the Conners 3-Short forms (Conners, 2008). Convergent validity is supported by moderate to high correlations between scales on the Conners 3-P(S) and scales which assess similar constructs on other related measures such as the Conners' Rating Scale-Revised, the Behaviour Assessment System for Children, Second Edition, the CBCL/4-18, and the BRIEF (Marocco & Rzepa, 2008). Discriminant validity is strongly supported by the ability of the Conners 3-P(S) to accurately distinguish between clinical and non-clinical groups, as well as differentiate between various clinical populations such as children with Disruptive Behaviour Disorders, Learning Disorders and ADHD (Conners, 2008).

Youth-reported continuous measure of conduct problems.

Adolescents completed the Global Appraisal of Individual Needs – Short Screener (GAIN-SS), which is a widely used screening tool designed to identify adolescents and adults who likely have substance abuse disorders, internalizing disorders, externalizing disorders, and problems related to crime and violence, and thus, may need treatment in one or more of these areas (Dennis, Feeney, Stevens, & Bedoya, 2008). The GAIN-SS takes approximately five minutes to complete, is designed for self-administration, and has been utilized with adolescents as young as 10 years old. A modified version of the GAIN-SS has been created by a team of researchers specializing in adolescent substance abuse from The Hospital for Sick Children and The Center for Addictions and Mental Health. The modified version of the GAIN-SS is identical to the original GAIN-SS, except that it includes six additional screening items which cover additional behavioural health problems, however, no clinical cut-off points have been established for these items. The GAIN-SS produces four subscales: Internalizing Disorder Screener, Externalizing Disorder Screener, Substance Disorder Screener and Crime/Violence Screener. For each item, a response is given in relation to the recency of the problem outlined in the item (e.g.,

3 = past month; 2 = 2 to 12 months ago; 1 = more than 1 year ago; 0 = never). Raw scores on the Crime/Violence Screener of the GAIN-SS were used as a measure of self-reported CP in adolescence. The items that comprise this subscale assess current and past prevalence of a variety of delinquent acts including aggressive behaviour towards other, theft, vandalism, making and distributing illegal substances, and driving while under the influence (Dennis et al., 2008). Good internal consistency has been found for both adolescents and adults on the Total Disorder Screener and the four subscreeners (Cronbach's alpha = .87 on the Total Disorder Screener). The Total Disorder Screener and the four subscreeners were highly correlated ($r = .84$ to $.94$) with their respective scales on the full-length version of the GAIN, which provides evidence for convergent validity. Confirmatory factor analysis suggests that each of the subscreeners has good discriminant validity and that the overall structure of the tool is consistent with the model used in the full-length version of the GAIN (Dennis, Chan, & Funk, 2006; Dennis et al., 2008).

Caregiver-reported categorical measures of conduct problems.

A measure of delinquency was also obtained from the semi-structured Case History Interview conducted with parents, legal guardians, and/or primary caregivers at follow-up. This Case History Interview (designed as part of the larger follow-up study) was based on the Case History form currently used by the Motherisk FASD Clinic and the Life History Interview developed by Streissguth and colleagues (1996; 2004). It includes specific questions pertaining to the adolescent's legal history. More specifically, informants were asked whether the adolescent had ever been in contact with the law for committing a criminal offence (i.e., brought into a police station, arrested, charged, convicted, or incarcerated). Adolescents who have ever

been in contact with the law for committing a criminal offence were coded as having a positive history of trouble with the law.

A measure of CP within the school environment was obtained from the semi-structured Case History Interview conducted with parents, legal guardians, and/or primary caregivers at follow-up. More specifically, informants were asked whether the adolescent had ever been formally suspended from a school and those who had been formally suspended were coded as having a positive history of school suspension.

Statistical Analysis

Statistical analyses were conducted using RStudio (Version 0.98.501). Descriptive statistics were used to evaluate the prevalence of the selected biopsychosocial risk factors and various types of CP investigated in youth. Next, Pearson's product-moment correlations, Student's *t*-tests, and Pearson's Chi-squared tests with Yates' continuity correction were used to examine the relationship between each of the selected biopsychosocial predictors and each of the outcome variables. Following exploring bivariate relationships, separate simultaneous multiple linear regression models were constructed to examine the effects of the selected biopsychosocial predictors (including age and sex) on CBCL Oppositional Defiant Problems *T*-scores, CBCL Conduct Problems *T*-scores, Conners 3 Defiance/Aggression *T*-Scores, and GAIN Crime/Violence Screener raw scores. Regression diagnostics were used to test assumptions of linearity, normality, and homogeneity of variance. More specifically, each model's residuals were plotted against each predictor in order to assess whether the partial relationships were linear. Histograms of the Studentized residuals of each model were created in order to assess that the errors were normally distributed. Studentized residuals of each model were then plotted by predicted values in order to evaluate whether the residuals had homoscedasticity. Moreover,

Studentized residuals were plotted against each predictor separately to further assess for homogeneity of variance. A variance inflation factor (VIF) for each predictor was calculated in order to assess for multicollinearity. Finally, in order to assess for the possibility of influential observations, an index plot of the hat values, Studentized residuals, and Cook's distances for each model were created. Multiple logistic regression analyses were also performed on the data to obtain the odds of having a history of trouble with the law and a history of school suspension in association with each of the biopsychosocial predictors (including age and sex). All predictors were simultaneously entered into the multiple regression models for each continuous and categorical outcome variable, as the biopsychosocial model of CP purposes that individual differences in CP can be explained by the influence of several biological, neuropsychological, family, and social factors, acting concurrently on an individual. Finally, CBCL Oppositional Defiant and Conduct Problems *T*-scores at follow-up were regressed on CBCL Oppositional Defiant and Conduct Problems *T*-scores at diagnosis, respectively, controlling for sex and time between baseline and follow-up (months), in order to evaluate the continuity of CP in this population.

Results

Participants

Demographic statistics describing the study population are shown in Table 1. The majority of study participants were male (71%) and given a diagnosis of ARND (97%). Two adolescents were diagnosed with pFAS and none with FAS. The study sample was racially, ethnically, and socioeconomically diverse; however, a valid measure of socioeconomic status (SES) could not be obtained for 12% ($n = 7$) of individuals, as they were under the care of CAS and living in a group home or residential treatment facility, or were living in a semi-independent housing situation, and thus, had no parental figures. Moreover, two caregivers failed to complete the Hollingshead Four Factor Index of Social Status. The sample's mean Full Scale IQ score fell within the Low Average ($M = 86.86$, $SD = 11.09$, range = 60–115). Age at diagnosis ranged from 4.78 to 16.05 years old ($M = 10.58$ years, $SD = 2.71$) and the length of time from diagnosis to follow-up ranged from 1.87 to 6.84 years ($M = 3.99$ years, $SD = 1.38$ years). Participants were between 11.00 and 18.83 years of age at follow-up, with a mean age of 14.56 years ($SD = 2.23$). Adolescents were living in a variety of different home environments at the time of follow-up and the majority of adolescents were not in the care of a biological parent (92%) (Table 1).

Prevalence of Conduct Problems in Adolescents with FASD

Caregivers' endorsements on the CBCL revealed high levels of conduct problems (mean T -score = 67.73, $SD = 9.23$) and oppositional defiant problems (mean T -score = 65.58, $SD = 8.58$) in Canadian adolescents with FASD, with mean T -scores falling in the Borderline Clinical range (T -score = 65–69). More specifically, 42% ($n = 25$) of adolescents had scores on the CBCL Conduct Problem scale that fell within the Clinical range (T -score ≥ 70) and another 22% ($n = 13$) had scores that fell within the Borderline Clinical range. Oppositional defiant problems

were slightly less prevalent, as 36% ($n = 21$) of scores fell within the Clinical range and 20% ($n = 12$) fell within the Borderline Clinical range. Finally, 27% of adolescents with FASD ($n = 16$) had scores on both the Oppositional Defiant Problems and Conduct Problems scales that fell within normal limits.

A similar pattern of results were seen with caregivers' endorsements on the Conners 3-P(S). The sample's mean score of the Defiance/Aggression scale on the Conners 3-P(S) fell at the cusp of the Borderline Clinical and Clinical range (mean T -score = 69.51, $SD = 16.89$). Moreover, 46% ($n = 27$) of adolescents had scores on the Defiance/Aggression scale that fell within the Very Elevated range (T -score ≥ 70) and another 14% ($n = 8$) had scores that fell within the Elevated range (T -score = 65–69).

With regard to self-reported delinquency on the GAIN-SS, 47% ($n = 27$) of adolescents who completed the modified GAIN-SS obtained scores on the Crime/Violence Screener that were classified in the Moderate risk range and 7% ($n = 4$) of scores fell within the High risk range. Taken together, these results indicate that just over half of our sample of adolescents with FASD (53%) may require intervention in the areas of interpersonal violence, drug-related crimes and property crimes.

Qualitative and quantitative data regarding adolescents' criminal history was also obtained from the semi-structured Case History Interview conducted with parents, legal guardians, and/or primary caregivers at follow-up. According to respondents, 32% ($n = 19$) of adolescents had been in contact with the law for committing a criminal offence (i.e., brought into a police station, arrested, charged, convicted, or incarcerated). Of those individuals, 74% ($n = 14$) had been formally arrested, 42% ($n = 8$) were formally charged with an offence, 21% ($n = 4$) were convicted, and one individual had been incarcerated overnight. In terms of categories of

criminal offences, 17% ($n = 10$) of the sample had been in contact with the law on at least one occasion for theft, 14% ($n = 8$) for assault, 7% ($n = 4$) for mischief (e.g., breaking windows of cars with rocks, damaging property), 3% ($n = 2$) for robbery, 3% ($n = 2$) for other weapons-related charges (e.g., threatening assault with a weapon), 2% ($n = 1$) for disorderly conduct (engaged in indecent act/exposure of genitals), 2% ($n = 1$) for a drug-related offence (caught with a marijuana pipe), and 2% ($n = 1$) for breach of curfew.

Finally, with regard to CP within the school environment, 53% ($n = 31$) of adolescents had been suspended from a school on at least one occasion and the majority of these youth had been suspended on multiple occasions ($n = 23$). Unfortunately, we could not calculate a mean number of school suspensions for the sample, as some caregivers were not able to give an accurate estimate of the number of suspensions given to the adolescent over his/her school career, often because this was a regular occurrence for some adolescents. Accordingly, the number of reported suspensions ranged from one to more than 50 (Mdn for those suspended = 3).

Continuity of Conduct Problems in Adolescents with FASD

CBCL Oppositional Defiant and Conduct Problems T -scores at follow-up were regressed on CBCL Oppositional Defiant and Conduct Problems T -scores at diagnosis, respectively, controlling for sex and time between baseline and follow-up, in order to assess the continuity of CP in youth with FASD across the follow-up period (mean length of time from diagnosis = 3.99 years, $SD = 1.38$, range = 1.87–6.84 years). As expected, T -scores on the CBCL Oppositional Defiant scale at diagnosis significantly predicted T -scores on the CBCL Oppositional Defiant scale at follow-up ($p = .004$). Specifically, a one-point increase on the Oppositional Defiant scale at diagnosis was associated with an increase of .35 on the Oppositional Defiant Problems scale at follow-up, after controlling for sex and time between diagnosis and follow-up. Moreover, the

model including sex, time between diagnosis and follow-up, and CBCL Oppositional Defiant *T*-scores at diagnosis explained a significant proportion of variance in the CBCL Oppositional Defiant *T*-scores at follow-up, $R^2 = 0.16$, $F(3, 55) = 3.44$, $p = .023$. Similarly, controlling for sex and time between diagnosis and follow-up, *T*-scores on the CBCL Conduct Problems scale at diagnosis significantly predicted *T*-scores on the CBCL Conduct Problems scale at follow-up ($p < .001$), with a one-point increase at diagnosis corresponding with an increase of .51 at follow-up. This model including sex, time between diagnosis and follow-up, and CBCL Conduct Problem *T*-scores at diagnosis explained 28% of variance in adolescents' CBCL Conduct Problems *T*-scores at follow-up, $F(3, 55) = 7.102$, $p < .001$. Finally, sex was not a significant predictor of CBCL Oppositional Defiant *T*-scores ($p = .190$) or Conduct Problems *T*-Scores at follow-up ($p = .420$). Similarly, time between diagnosis and follow-up was not a significant predictor of CBCL Oppositional Defiant ($p = .879$) or Conduct Problems *T*-Scores at follow-up ($p = .864$).

Prevalence of Biopsychosocial Risk Factors for Conduct Problems in FASD

Biological and family risk.

As predicted, high rates of biological and family risk factors for CP were documented in our Canadian sample of adolescents with FASD. More specifically, 47% ($n = 28$) of adolescents' biological mothers had a reported history of criminality (i.e., had either a criminal record or had been incarcerated for committing a criminal offense). Unfortunately, further data regarding the types and frequency of offences committed were unavailable. Moreover, although adolescents' biological fathers' histories were unknown in 32% ($n = 19$) of cases, 60% of fathers known to respondents also had a reported history of criminality (24/40). Alarmingly high rates of maltreatment were also reported, with 44% ($n = 26$) of caregivers reporting a confirmed history

of either physical or sexual abuse experienced by the adolescent. Disruptions in the family structure and changes in home placements were also a frequent occurrence in our sample. Ninety-two percent of adolescents with FASD ($n = 54$) had experienced at least one separation from a parental figure or a change in primary caregiver. Furthermore, the majority of the sample experienced multiple caregiver disruptions, as only 8% ($n = 5$) of adolescents had lived with the same caregiver for their whole lives and the mean number of different home placements was 3.56 ($SD = 2.15$, range = 1–12).

Neuropsychological and social risk.

As expected, the majority of adolescents with FASD also displayed specific neuropsychological weaknesses and problems with peer relations that put them further at risk for the development of CP. Specifically, 80% ($n = 47$) of adolescents had been given a formal diagnosis of ADHD, which corresponds to significant difficulties with inattention and/or hyperactivity/impulsivity that impairs functioning in at least two settings (e.g., home, work, school). Adolescents with FASD also exhibited significant difficulties with emotional/behavioural regulation (an executive process also termed effortful control), as the sample's mean *T*-score on the BRIEF Behavioural Regulation Index fell within the clinically significant range (T -score ≥ 65). Moreover, 80% ($n = 47$) of caregivers endorsed clinically significant scores on the Behavioural Regulation Index of the BRIEF. With regard to endorsements on the individual scales that comprise this Index, 76% ($n = 45$) of the study population had clinically elevated scores on the Inhibit scale and 69% ($n = 41$) had clinically elevated scores on the Shift and Emotional Control scales, respectively (Table 2). Finally, problems with peer relations were also common in this sample, with 58% ($n = 34$) of caregivers endorsing clinically significant

concerns (T -score ≥ 65) on the Social Problems subscale of the CPRS-R:L (mean T -score = 67.76 $SD = 14.72$).

Accumulative risk.

In order to quantitatively assess the accumulation of risk factors for CP in the current sample, each of the continuous predictors (other than age) were transformed into clinically meaningful dichotomous variables representing the presence or absence of risk. Specifically, family instability was coded as positive if the adolescent had 3 or more changes in primary caregiver and home placement throughout his/her life. Moreover, adolescents with clinically significant T -scores (T -score ≥ 65) on the Behavioural Regulation Index of the BRIEF and Social Problems subscale of the CPRS-R:L were coded positive for poor emotional/behavioural regulation skills and social problems, respectively. Figure 2 depicts the percentage of adolescents with zero to six of the selected biopsychosocial risk factors for CP. The presence of multiple risk factors was common in this sample, as 90% of adolescents were coded positive for two or more risk factors ($n = 53$). Moreover, the median for the sample was four risk factors, spanning multiple domains.

Bivariate Analyses of Biopsychosocial Predictors and Outcome Variables

Pearson's product-moment correlation analysis was conducted for each of the continuous biopsychosocial predictors (age, emotional/behavioural regulation skills, family instability, and social problems) and the continuous outcome measures of CP (CBCL Oppositional Defiant Problems T -scores, CBCL Conduct Problems T -scores, Conners 3 Defiance/Aggression T -Scores, and GAIN Crime/Violence Screener raw scores). Multiple Student's t -tests were conducted for each of the categorical biopsychosocial predictors (sex, maternal criminality, abuse history, ADHD diagnosis) and the continuous outcome measures of CP, as well as for each

of the continuous biopsychosocial predictors and the categorical outcome measures of CP (trouble with the law, school suspension). Finally, Pearson's Chi-squared tests with Yates' continuity correction were used to examine the relationship between categorical predictors and outcome measures.

Results revealed that significantly more males with FASD had a history of trouble with the law ($\chi^2 (1, N = 58) = 5.98, p = 0.014$) and school suspensions ($\chi^2 (1, N = 58) = 13.71 p < 0.001$) than females. Those who had been suspended from a school were also significantly older ($M = 181.26$ months) than those who had no history of school suspensions ($M = 167.39$ months), $t(57) = -2.04, p = 0.046$. The opposite pattern was seen for the relationship between age and oppositional defiant behaviour, as *T*-scores on the CBCL Oppositional Defiant Problems scale decreased with increasing age ($r(57) = -.26, p = 0.051$). Youth with biological mothers who had a reported criminal history or history of incarceration had significantly higher self-reported raw scores on the GAIN-SS Crime/Violence Screener ($M = 3.85$) than those who were coded negative for maternal criminality ($M = 2.23$), $t(56) = -2.45, p = 0.017$. Those with a positive maternal history of criminality also had elevated *T*-scores on the parent-reported Conners 3-P(S) Defiance/ Aggression scale ($M = 74.46$) when compared to those coded negative for maternal criminality ($M = 65.03$), $t(57) = -2.21, p = 0.031$. Adolescents with a positive history of physical and/or sexual abuse had significantly higher self-reported raw scores on the GAIN-SS Crime/Violence Screener ($M = 3.73$) than adolescents who had no history of abuse ($M = 2.38$), $t(56) = -2.01, p = 0.0496$. Family instability was significantly correlated with self-reported raw scores on the GAIN-SS Crime/Violence Screener, with adolescents who had experienced higher family instability (i.e., more home placements with different caregivers throughout life) reporting more delinquent behaviours on the GAIN-SS ($r(56) = .29, p = 0.027$). The group of adolescents

with FASD who had been in contact with the law for committing a criminal offense also had a significantly higher mean family instability rating ($M = 2.54$) than the group of adolescents with FASD who had no legal history ($M = 1.83$), $t(57) = -2.26, p = 0.028$. With regard to individual neuropsychological predictors, adolescents with FASD and ADHD had significantly higher T -scores on the Conners 3-P(S) Defiance/Aggression scale ($M = 71.89$) than adolescents with FASD who did not meet criteria for ADHD ($M = 60.17$), $t(57) = -2.22, p = 0.031$. Moreover, the mean CBCL Oppositional Defiant Problems T -score for the FASD plus ADHD group ($M = 66.66$) was higher than the mean T -score for the group of youth with FASD and no diagnosis of ADHD ($M = 61.33$); however, this did not meet the cut-off for significance ($p = 0.054$). Adolescents who had been in trouble with the law had significantly lower mean T -scores on the Social Problems subscale of the CPRS-R:L ($M = 62.16$) than adolescents with no legal history ($M = 70.43$), $t(57) = 2.07, p = 0.043$. In other words, youth with FASD who had been in trouble with the law had fewer parent-reported social problems than youth with FASD who had not been in contact with the law. Finally, T -scores on the BRIEF Behavioural Regulation Index were significantly correlated with CBCL Oppositional Defiant Problems T -scores ($r(57) = .31, p = 0.016$), Conners 3-P(S) Defiance/Aggression T -scores ($r(57) = .44, p < 0.001$), and GAIN-SS Crime/Violence Screener raw scores ($r(56) = .41, p = 0.001$). Moreover, BRIEF Behavioural Regulation Index T -scores were positively correlated with CBCL Conduct Problems T -scores, however, this relationship did not reach significance ($r(57) = .25, p = 0.061$).

Regression Diagnostics

An examination of the regression diagnostics for all the linear regression models that regressed continuous measure of CP (e.g., CBCL Oppositional Defiant Problems T -scores, CBCL Conduct Problems T -scores, Conners 3-P(S) Defiance/Aggression T -scores, GAIN-SS

Crime/Violence Screener raw scores) on the selected biopsychosocial predictors revealed residuals that appeared approximately normally distributed and did not seriously violate linearity or homogeneity. An examination of leverage, discrepancy, and influence revealed that none of the cases in the data set had extreme leverage, were excessively outlying, or were excessively influential. An examination of the variance inflation factor (VIF) statistic for each predictor in the models did not reveal any issues with multicollinearity (VIF range = 1.19–1.84). Moreover, no major violations of normality or homogeneity were revealed for the linear regression models that regressed CBCL Oppositional Defiant and Conduct Problems *T*-scores at follow-up on CBCL Oppositional Defiant and Conduct Problems *T*-scores at diagnosis, respectively. Accordingly, none of the cases in the data set had extreme leverage, were excessively outlying, or were excessively influential.

Predictors of Caregiver-Reported Continuous Measures of Conduct Problems

Table 3 illustrates the parameters of the first multiple regression model for the prediction of Oppositional Defiant Problems *T*-scores from age, sex, maternal criminality, abuse history, family stability, ADHD diagnosis, BRIEF Behavioural Regulation Index *T*-scores, and CPRS-R:L Social Problems *T*-scores. Youth's *T*-scores on the Behavioural Regulation Index of the BRIEF at diagnosis was the only significant predictor of youth's Oppositional Defiant Problems *T*-scores on the CBCL at follow-up ($p = .037$). Specifically, a one-point increase on the Behavioural Regulation Index of the BRIEF was associated with an increase of .23 on the CBCL Oppositional Defiant Problems scale, after accounting for the effects of age, sex, maternal criminality, abuse history, family instability, ADHD, and social problems. The model including the selected biopsychosocial predictors explained 23% of variance in adolescents' CBCL

Oppositional Defiant Problems *T*-scores at follow-up, however, the multiple R^2 was not significant, $F(8, 50) = 1.88, p = .085$.

The parameters of the multiple regression model for the prediction of CBCL Conduct Problems *T*-scores from age, sex, maternal criminality, abuse history, family stability, ADHD diagnosis, BRIEF Behavioural Regulation Index *T*-scores, and CPRS-R:L Social Problems *T*-scores are shown in Table 4. No significant predictors were identified and the selected biopsychosocial predictors failed to explain a significant proportion of variance in youth's Conduct Problems *T*-scores on the CBCL at follow-up, $R^2 = 0.13, F(8, 50) = 0.94, p = .491$.

Table 5 displays the parameters of the multiple regression model for the prediction of Conners 3 Defiance/Aggression *T*-Scores from the selected biopsychosocial risk factors in adolescents with FASD. Once more, *T*-scores on the BRIEF Behavioural Regulation Index was the only significant predictor of adolescents' Defiance/Aggression *T*-Scores on the Conners 3 ($p = .018$), such that a one-point increase on the Behavioural Regulation Index was associated with a half-point increase on Conners 3 Defiance/Aggression *T*-Scores after accounting for the effects of age, sex, maternal criminality, abuse history, family instability, ADHD, and social problems. The model including the abovementioned biopsychosocial predictors explained a significant proportion of variance in the Conners 3 Defiance/Aggression *T*-Scores of youth with FASD, $R^2 = 0.26, F(8, 50) = 2.16, p = .047$.

Predictors of Youth-Reported Continuous Measure of Conduct Problems

Table 6 illustrates the parameters of the first multiple regression model for the prediction of GAIN-SS Crime/Violence Screener raw scores from age, sex, maternal criminality, abuse history, family stability, ADHD diagnosis, BRIEF Behavioural Regulation Index *T*-scores, and CPRS-R:L Social Problems *T*-scores. Once again, *T*-scores on the Behavioural Regulation Index

of the BRIEF at diagnosis was the only significant predictor of self-reported raw scores on the Crime/Violence Screener of the GAIN-SS at follow-up ($p = .048$). Specifically, a one-point increase on the Behavioural Regulation Index of the BRIEF was associated with a raw score increase of 0.07 on the GAIN-SS Crime/Violence Screener after accounting for the effects of age, sex, maternal criminality, abuse history, family instability, ADHD, and social problems. Although the multiple R^2 for the model including the selected biopsychosocial predictors only approached significance, it explained 25% of variance in self-reported raw scores on the GAIN-SS Crime/Violence Screener, $F(8, 49) = 2.09, p = .055$.

Predictors of Caregiver-Reported Categorical Measures of Conduct Problems

Two separate logistic regression analyses were conducted to predict coming in contact with the law for committing a criminal offence and being suspended from school (Tables 7 and 9, respectively) using age, sex, maternal criminality, abuse history, family stability, ADHD diagnosis, BRIEF Behavioural Regulation Index T -scores, and CPRS-R:L Social Problems T -scores. Sex ($p = .046$), family instability ($p = .035$), and ADHD diagnosis ($p = .035$) were significant and independent predictors of youth with FASD having been in trouble with the law. Specifically, holding all other biopsychosocial predictors constant, males with FASD were approximately 13 times more likely to have been in contact with the law for committing a criminal offence than females with FASD ($OR = 13.28$). These results also showed that having a formal ADHD diagnosis significantly lowered the odds of getting in trouble with the law for youth with FASD ($OR = 0.12$) when all other biopsychosocial predictors were held constant. Specifically, individuals with FASD who had not been formally diagnosed with ADHD were approximately 8 times more likely to have been in contact with the law for committing a criminal offence than those who had been given a diagnosis of both FASD and ADHD ($OR = 8.14$).

Finally, these analyses revealed that for every one-unit increase in family instability ([total number of placements with different caregivers/adolescent's age in months at follow-up] x 100) the odds of getting in trouble with the law increased by a factor of 2.96. For ease of interpretation, this logistic regression model was subsequently re-run with "total number of home placements" in place of the "family instability" variable (Table 8). The results of this model were comparable to the original model outlined in Table 7, with sex ($p = .046$), number of homes ($p = .035$), and ADHD diagnosis ($p = .033$) being the only significant and independent predictors of trouble with the law. This model revealed that the odds of getting in trouble with the law roughly double with each additional home placement ($OR = 1.82$).

Finally, the logistic regression analysis for the prediction of school suspension from the selected biopsychosocial risk factors revealed the following significant and independent predictors: sex ($p = .003$), T -scores on the Behavioural Regulation Index of the BRIEF ($p = .047$), and T -scores on the Social Problems scale of the Conners' Parent Rating Scales-Revised: Long Form ($p = .025$) (Table 9). One again, males with FASD were approximately 20 times more likely to have been suspended from a school than females with FASD ($OR = 19.68$). The results of this model also revealed that a one-point increase on the Behavioural Regulation Index of the BRIEF was associated with an 8% increase in the odds of being suspended from a school, holding all other biopsychosocial predictors constant ($OR = 1.08$). Finally, holding all other variables in the model constant, lower T -scores on the Social Problems Scale of the Conners Revised (i.e., lower levels of reported social problems) was shown to increase the risk of school suspension, with an 8% increase in the odds of getting suspended for every one-point *decrease* on the Social Problems scale of the Conners' Parent Rating Scales-Revised: Long Form.

Bivariate Relationships Between Subscales of the BRIEF Behavioural Regulation Index and Measures of Conduct Problems

In order to further explore the relationship between specific aspects of self-regulation and CP in youth with FASD, Pearson product-moment correlations were performed between *T*-scores on the three subscales that comprise Behavioural Regulation Index of the BRIEF (Inhibit, Shift, and Emotional Control) and all continuous outcome measures of CP at follow-up (Table 10). Multiple Student's t-tests were also conducted for each of the Behavioural Regulation Index subscales and the two categorical outcome measures of CP (i.e., trouble with the law and school suspension). These results revealed that *T*- scores on the Inhibit subscale of the BRIEF were significantly positively correlated with all continuous measures of CP, with coefficients ranging from small to medium ($r = .26\text{--}.34$). We also found significant moderate positive correlations ($r = .32\text{--}.41$) between *T*-scores on the Emotional Regulation subscale of the BRIEF and scores on three of the four continuous measures of CP (CBCL Oppositional Defiant Problems *T*-scores, Conners 3 Defiance/Aggression *T*-Scores, and GAIN Crime/Violence Screener raw scores). Finally, *T*-scores on the Shift subscale were significantly positively correlated with *T*-scores on the Conners 3 Defiance/Aggression scale ($r = .37$) and raw scores on the GAIN Crime/Violence Screener ($r = .33$). In contrast, there were no significant differences in mean BRIEF subscale scores between adolescents with FASD who had been in contact with the law for committing a criminal offense and those with FASD who had no legal history (Table 11). Similarly, no significant relationship was found between mean *T*-scores on the Inhibit, Shift, or Emotional Control subscales and having been suspended from school (Table 12).

Discussion

Research has consistently linked PAE to the later development of CP, yet little research has investigated the complex constellation of biopsychosocial risk factors that may be associated with CP in youth with FASD. Moreover, previous researchers have failed to utilize contemporary theoretical frameworks of CP to guide their study design and predictor selection. The current study was informed by the biopsychosocial model of CP and supporting research in the general population, as well as in individuals with PAE. Extending the findings of a larger prospective follow-up study of Canadian adolescents with FASD, the current study aimed to 1) identify the prevalence of various types of CP in this adolescent cohort; 2) identify the prevalence of potential biological, neuropsychological, family, and social risk factors for CP; 3) further elucidate how common neuropsychological deficits associated with PAE, as well as co-occurring biological, family, and social risk factors, function together to predict the development of CP in this population; and 4) assess the continuity of CP across middle childhood to adolescence.

Prevalence and Continuity of Conduct Problems in Adolescents with FASD

As expected, high rates of CP were found in this cohort of Canadian adolescents with FASD across multiple assessment methods. Specifically, just over half of the sample had scores that fell in the Clinical/Borderline range on the CBCL DSM-Oriented Oppositional Defiant Problems (56%) and Conduct Problems scales (64%), as well as the Conners 3-P(S) Defiance/Aggression scale (59%). Accordingly, a similar proportion of adolescents' endorsements on the GAIN-SS Crime/Violence Screener fell within the High/Moderate risk range (53%). The high rate of clinically significant symptoms of ODD and CD found in this sample is consistent with the large body of previously outlined research documenting a strong

link between PAE and the development of disruptive behaviour disorders (Burd et al., 2003; Disney et al., 2008; D'Onofrio et al., 2007; Fryer et al., 2007; Larkby et al., 2011; Roebuck et al., 1999; Schonfeld et al., 2005). This study, however, is unique in that it has confirmed high rates of CP using a variety of assessment methods (i.e., interviews with caregivers, standardized behavioural rating scales) and across multiple informants (i.e., caregivers and youth).

As expected, we also found that *T*-scores on the DSM-Oriented Oppositional Defiant Problems and Conduct Problems scales of the CBCL at diagnosis significantly predicted *T*-scores of each of these scales, respectively, at follow-up (on average 4 years post-diagnosis), after controlling for sex. Unfortunately, the only comparable longitudinal study investigating psychopathology and behaviour in individuals with PAE across middle childhood and adolescence only included a restricted sample of those diagnosed with full-blown FAS (Steinhausen & Spohr, 1998). However, similar to our findings, these researchers found no significant change in *T*-scores on the CBCL Delinquent Problems and Aggressive Behaviour scales between the first assessment period (mean age = 9.8 years) and follow-up (mean age = 13.1 years), and average *T*-scores for both assessment periods fell within the non-clinical range. In contrast, this sample did display an elevated prevalence of conduct disorders on structured psychiatric interviews and the rate of conduct disorders remained relatively constant across the follow-up period, with some variability at the individual level (i.e., some symptoms remitted, while other new cases met criteria over time). Lower rates of CP in individuals with FAS compared to those with ARND/pFAS have been documented in other studies (Schonfeld et al., 2005; Streissguth et al., 1996, 2004) but further analysis of this trend is beyond the scope of the current discussion. Findings from cross-sectional studies that included a broader age range of individuals with FASD also provide support for the persistence of CP in this population across

development (Fagerlund et al., 2011; Streissguth et al., 2004). More specifically, Fagerlund et al. (2011) found that scores on the CBCL Externalizing Problems scale did not differ significantly between the following three age grouping of individuals with FASD: 8–12 years old, 13–16 years old, and 17–20 years old. Similarly, Streissguth et al., (2004) found comparable rates of trouble with the law in individuals with FASD who were 12–20.9 years old and those 21 years old and above (61% vs. 58%); however, as expected, rates of formal involvement with the criminal justice system were significantly lower in children (14%). Nevertheless, our results suggest that CP symptoms displayed by individuals with FASD remained relatively stable across middle childhood to adolescence. It also appears that receiving a formal FASD diagnosis alone does not have a significant impact on symptoms of ODD and CD demonstrated by this population and more focused interventions are necessary to significantly mitigate these persistent difficulties.

Approximately one third of adolescents in this sample had been in contact with the law for committing a criminal offence, which is approximately seven times higher than the similarly defined overall youth crime rate in Canada reported for 2013 by Statistics Canada (Boyce, Cotter, & Perreault, 2014). This estimate included youth (12 to 17 years of age) who had been accused of a police-reported crime and were either charged or diverted from the criminal justice system (i.e., provided with a warning, caution, or referral to a supportive community-based program) (Boyce et al., 2014). This alarming finding regarding youth crime is consistent with previous research documenting an overrepresentation of FASD in the criminal justice system in North America (Fast et al., 1999; Popova et al., 2011) and highlights the importance of allocating resources to the prevention and mitigation of CP earlier in development for this high-risk population. On a more encouraging note, the prevalence of “trouble with the law” in our

Canadian cohort (32%) is significantly less than the prevalence of this outcome reported in the American cohort followed in the landmark Seattle Longitudinal Study, published almost two decades ago (61%) (Streissguth et al., 1996). The lower rates of involvement in the criminal justice system documented in the current study may be due to improved diagnosis and intervention for those affected by FASD over the past 20 years and/or better overall identification and support of individuals with disabilities in Canada compared to the United States. Moreover, although identical operational definitions for “trouble with the law” were used in both studies, the age range of individuals included in Streissguth et al.’s (1996) adolescent cohort was 12 to 20.9 years ($M = 15.8$), compared to 11 and 18.8 years in the current study ($M = 14.56$). Thus, it is possible that the inclusion of young adults (aged 19 to 20.9) in the American cohort in combination with the inclusion of younger teens (aged 11 to 11.9) in the current study could also partially explain this discrepancy.

Not surprisingly, we also found that approximately half of adolescents with FASD in our cohort had been suspended from a school on at least one occasion (53%) and that many had a history of multiple school suspensions ($n = 23$). The prevalence of school suspension found in this Canadian sample is comparable to that reported in the American cohort by Streissguth et al. (1996) ($\approx 53\%$), suggesting that youth with FASD are not only exhibiting significant CP in the home and community, but also within the school environment. High rates of suspension in this population may indicate a poor understanding and/or ability to meet the unique behavioural and emotional needs of children and youth with FASD within the school system in Ontario. CP and their current consequences within the school setting likely negatively impact learning, and in combination with the neurocognitive impairments related to PAE, put this population at greater

risk for poor academic achievement, poor attitudes towards school, and ultimately, school disengagement.

Prevalence of Biopsychosocial Risk Factors for Conduct Problems in FASD

As predicted, many Canadian youth with FASD had complex family histories and high rates of biological and family risk factors that have been found to be associated with CP in the general population, including parental criminality, family instability, and a history of maltreatment. As mentioned previously, PAE often occurs in the context of social and family adversity and is only one piece of a very complex clinical picture (for review see Malone & Koren, 2012). In the current study, 47% of adolescents' biological mothers had a reported history of criminality. Although this is the first study, to our knowledge, which has reported on rates of criminality in biological mothers of individuals with FASD, this finding is consistent with significantly higher rates of APD found in substance-abusing women when compared to controls (Hans, 1999). It is important to note, however, that we did not gather information regarding the types of criminal behaviour in which these women engaged that ultimately resulted in a criminal record or incarceration (e.g., substance-related, property-related, violent, etc.) or the context in which the delinquent behaviour occurred.

Family instability was also frequently reported, as 92% of adolescents had experienced at least one change in primary caregiver (mean number of home placements = 3.56) and only 8% of the sample were still in the care of a biological parent. In line with this finding, Sood et al. (2001) showed that higher levels of alcohol use during pregnancy were associated with an increased likelihood of custody changes for the child and a decreased likelihood that the child was in the custody of his/her biological mother at age 6 to 7 years, among other maternal and family risk factors. Moreover, Streissguth et al. (1991) reported an average of five different

principal home placements in an American sample of adolescents ($n = 43$) and adults ($n = 18$) with FAS/FAE. A database review of the first 1,400 patients diagnosed with FASD through the Washington State Fetal Alcohol Syndrome Diagnostic & Prevention Network (WA FAS DPN) also documented high rates of separation from biological parents at the time of FASD diagnosis (70.5%); however, close to one third of children were still living with one of their biological parents in this American sample (Astley, 2010). Differences with regard to clinic referral policies (i.e., physician referral versus self-referral), regional child protection standards, and/or adoption trends may contribute to the overall lower rate of adolescents reported to be living with a biological parent in this cohort. Alternatively, our study documented home placement two to seven years post-diagnosis ($M = 3.99$ years), and thus, it is possible that the risk of biological parents losing or relinquishing custody may increase with time and/or as the child develops and his/her disabilities become more pronounced. Consistent with this theory, Streissguth et al. (1991) found that only 9% of a sample, consisting primarily of adolescents with FASD between the ages of 12 and 17, were still living with both biological parents.

One of the most alarming findings is that nearly half of Canadian adolescents with FASD had experienced either physical or sexual abuse during childhood and/or adolescence (44%). As stated previously, a number of studies have documented a significant association between parental substance abuse and child maltreatment (Brown, Cohen, Johnson, & Salzinger, 1998; Chaffin, Kelleher, & Hollenberg, 1996; Egami, Ford, Greenfield, & Crum, 1996; Famularo, Kinscherff, & Fenton, 1992; Famularo, Stone, Barnum, & Wharton, 1986), however, this link is likely complex and other critical factors such as parental personality characteristics, family functioning, parental supervision, and social support, may play a mediating role (Ammerman, Kolko, Kirisci, Blackson, & Dawes, 1991; Hans, 1999; Magura & Laudet, 1996; Wolock &

Magura, 1996). Streissguth et al., (2004) found that 70.8% of individuals with FASD aged 12 to 20.9 years had been a victim of physical abuse, sexual abuse, or domestic violence; however, this higher maltreatment estimate may due to the inclusion of the experience of domestic violence, which was not measured in the current study. Unfortunately, it appears that the risk of victimization for individuals with FASD only increases across the lifespan, as a cross-sectional study of Canadian adults with FASD (Mean age = 22 years) found that 77% had a history of physical and/or sexual abuse (Clarke et al., 2004).

Although we only assessed a limited number of potential family risk factors for CP in the current study, these findings help to elucidate the early life adversity commonly experienced by individuals with FASD and highlight the necessity for the consideration of family factors when conducting research and clinical work with this high-risk population.

The large majority of adolescents with FASD also displayed neuropsychological deficits that have been previous linked to the development of CP, particularly significant difficulties with inattention and hyperactivity/impulsivity (80% were diagnosed with ADHD), and emotional/behavioural regulation (80% of *T*-scores on the BRIEF Behavioural Regulation Index were clinically elevated). The high prevalence of ADHD found in this study is consistent with the large body of literature linking FASD or heavy PAE to problems with attention using both direct testing (Burden, Jacobson, Sokol, & Jacobson, J. L 2005; Infante et al., 2015; Kooistra, Crawford, Gibbard, Kaplan, & Fan, 2011; Mattson, Calarco, & Lang, 2006; Streissguth et al., 1986) and standardized parent- and teacher-report measures (Astley, Olson, et al. 2009; Brown et al., 1991; Mattson, & Riley, 2000; Olson, Sampson, Barr, Streissguth, & Bookstein, 1992). There is also growing evidence that problems with EF, including difficulties with emotion regulation, inhibitory control, and cognitive shifting, are core deficits in FASD (Haley, Handmaker, &

Lowe, 2006; Khoury, Milligan, & Girard, 2015; Kodituwakku, 2010; Nash et al., 2015; Nguyen et al. 2014). In fact, a more recent study has found that children and youth with heavy PAE who also meet the criteria for ADHD had significantly higher scores than non-exposed controls with ADHD on all scales of the BRIEF and that scores on the BRIEF could distinguish those with PAE and ADHD from those with idiopathic ADHD. These results indicate that PAE is associated with high levels of executive dysfunction in the real world, which is above and beyond what can be accounted for by the presence of a comorbid ADHD diagnosis (Nguyen et al. 2014). More recently, the existing Canadian diagnostic guidelines for FASD (Chudley et al., 2005) were updated by a steering committee of experts in the field and “affect regulation” was added as a new neurodevelopmental domain to be specifically evaluated as part of a formal FASD diagnostic assessment (Cook et al., 2016). In other words, in addition to “executive function,” which includes impulse control, emotion regulation is now recognized as a unique area of impairment in this population that can be used as evidence for pervasive brain dysfunction resulting from PAE, and therefore, may contribute to the FASD diagnosis (Cook et al., 2016). Similarly, in the DSM-5, “impaired self-regulation” (as defined by deficits in attention, affect/ behavioural regulation, and/or impulse control) has been included as one of the essential criteria for the proposed diagnosis of Neurobehavioral Disorder Associated With Prenatal Alcohol Exposure, which is included in the Conditions for Further Study section (APA, 2013). In sum, more recent diagnostic guidelines for alcohol-related disorders have put a stronger emphasis on deficits in emotional/behavioural regulation, which is consistent with our findings of high levels of impairment in this specific neuropsychological domain, as measured by the BRIEF. Deficits in social skills have been documented across the lifespan in individuals with PAE (for review see Kully-Martens, Denys, Treit, Tamana, & Rasmussen, 2012), so it is not

surprising that many individuals in this cohort also had clinically significant social problems at follow-up (58% of *T*-scores on the CPRS-R:L Social Problems subscale were clinically elevated).

Finally, our analysis revealed that the majority of adolescents with FASD in this sample experienced an accumulation of biopsychosocial risk factors for CP throughout development. Specifically, 90% of adolescents had experienced two or more of the selected biopsychosocial risk factors and 64% had experienced four or more risk factors, spanning multiple domains. The additive risk model for the development of CP proposes that the number of risk factors present is the strongest predictor of CP. In other words, it is the accumulation of risk factors experienced by an individual that increases the likelihood of the development of CP, more so than the presence of any specific risk factor (Dodge & Pettit, 2003). Findings from a number of empirical studies support the basic tenets of this model (Ackerman, Schoff, Levinson, Youngstrom, & Izard, 1999; Deater-Deckard, Dodge, Bates, & Pettit, 1998; Flouri & Tzavidis, 2008; Stouthamer-Loeber, Loeber, Wei, Farrington, & Wikström, 2002). Moreover, results from one longitudinal study utilizing a large community-based sample suggest that different domains of risk factors (e.g., individual, family, sociocultural) each explain a unique proportion of the variance in CP, indicating increased risk associated with the presence of diverse risk factors that span domains (Deater-Deckard et al., 1998). Consistent with the additive model, the current study revealed a high degree of risk, often across multiple domains, experienced by youth with FASD, paired with a high prevalence of CP during adolescence. Unfortunately, we did not have the power to determine whether an additive, interactive, or transactional model of risk best fits the development of CP in FASD; however, future studies should further explore this line of inquiry (Burke et al., 2002).

Predictors of Continuous Measures of Conduct Problems

Biological variables (i.e., male sex, maternal criminality) and family factors (i.e., family instability, abuse history) were hypothesized to be the most significant predictors of CP in adolescents with FASD. Bivariate analyses revealed that most of the biopsychosocial risk factors investigated in the current study were significantly associated with at least one of the continuous measures of adolescent CP in the expected direction, including male sex, maternal criminality, abuse history, family instability, ADHD, and emotional/behavioural regulation. However, counter to our initial hypothesis, when all predictors were simultaneously entered into multiple regression models, emotional/behavioural regulation ability was the only significant and unique predictor of various measures of CP in youth with FASD. More specifically, caregiver ratings on the BRIEF Behavioural Regulation Index at diagnosis significantly predicted self-reported delinquency on the GAIN-SS Crime/Violence Screener, caregiver ratings on the CBCL Oppositional Defiant Problems scale and Conners 3 Defiance/Aggression scale, and a positive history of school suspensions at follow-up, after accounting for the effects of age, sex, maternal criminality, abuse history, family stability, ADHD, and social problems. Remarkably, this is the first study to investigate the role of emotional/behavioural regulation in the development of CP in individuals with FASD, despite the growing body of literature suggesting that EF deficits are a central feature of FASD (Haley, Handmaker, & Lowe, 2006, Khoury, Milligan, & Girard, 2015; Kodituwakku, 2010; Nash et al., 2015; Nguyen et al. 2014).

Interestingly, none of the biopsychosocial factors in the current study significantly predicted CBCL Conduct Problems *T*-scores in youth with FASD and the overall model failed to account for a significant amount of variance in these scores. The DSM-Oriented Conduct Problems scale on the CBCL is composed of 17 items that were rated as “very consistent” with

the DSM-IV diagnostic criteria for Conduct Disorder by the majority of a group of 22 experienced child psychiatrists and psychologists (Achenbach & Rescorla, 2001). Conduct Disorder, however, is a heterogeneous construct (Blair, Leibenluft, & Pine, 2014), and thus, this scale includes items that tap into various forms of behaviour that violate the basic rights of others or societal norms, such as acting aggressively towards others, lack of guilt, destroying property, setting fires, deceitfulness, theft, truancy, running away from home, and other serious rule violations (APA, 2000). It is possible the heterogeneity with regards to the types of externalizing behaviours assessed by this scale has partly contributed to the fact that our model was not able to account for a significant amount of variance in these scores. Moreover, there is both theoretical and empirical evidence to support the distinction between overt and covert forms of conduct problems (Loeber & Schmaling, 1985). Many of the items on the DSM-Oriented Conduct Problems scale tap into covert externalizing problems (e.g., theft, lying/cheating, threatening others, setting fires), which Frick and Morris (2004) maintain are less likely to be associated with deficits in self-regulation and more likely to be associated with callous/unemotional traits or low levels of fear inhibition. It is also important to highlight here that our sample had a mean age of 14 years old (Grade 8/9), making it a relatively young adolescent sample. The emergence of the more severe antisocial behaviours that are captured by this scale by mid-adolescence may be dependent on other factors at the individual, family, peer, and neighbourhood levels that could not be directly assessed in the current study. At the individual level, callous/unemotional traits have been shown to predict earlier onset, more severe, and more persistent CP in youth (Dandreaux & Frick, 2009; Frick & Dickens, 2006; Kroneman, Hipwell, Loeber, Koot, & Pardini, 2011; Rowe et al., 2010), and thus, these traits may be an important predictor of scores on the CBCL Conduct Problems scale in our sample population of younger adolescents. Within

the family and peer context, greater levels of maladaptive parenting (e.g., low parental involvement, poor monitoring/supervision) and delinquent peer affiliation has also been linked to the development of earlier onset and more severe CP in youth (Dandreaux & Frick, 2009). Although abuse history and a more general measure of social problems were included as predictors in the current study, data on specific parenting practices and youths' association with deviant peers was not available in the current study. Affiliation with deviant peers may be one factor that could account for a significant proportion of variance found on the DSM-Oriented Conduct Disorder scale in this specific population, as multiple studies have shown that youth are significantly more likely to exhibit various antisocial behaviours when within delinquent peer groups (Fergusson, Swain, Horwood, 2002; Henry, Tolan, & Gorman-Smith, 2001; Lahey, Gordon, Loeber, Stouthamer-Loeber, & Farrington, 1999). More distal factors, such as residing in a disadvantaged neighbourhood, may also play an important role in predicting youths' engagement in the various antisocial behaviours captured on the DSM-Oriented Conduct Problem scale, particularly by increasing the likelihood of affiliating with deviant peers and exposure to deviant peer group norms (Brody et al., 2001; Ge, Brody, Conger, Simons, & Murry, 2002).

Relationship between BRIEF subscales and measures of conduct problems.

In order to further explore the relationship between emotional/behavioural regulation and CP in FASD, post-hoc correlations were performed between scores on the three subscales that comprise the Behavioural Regulation Index of the BRIEF (Inhibit, Shift, and Emotional Control) and scores on all continuous measures of CP completed at follow-up. *T*-scores on the Inhibit and Emotional Control subscale at diagnosis were found to have significant positive correlations with scores on nearly all continuous measures of CP at follow-up, suggesting that behavioural

inhibition and the ability to regulate one's emotions may be particularly important in the prediction of CP in youth with FASD. Nevertheless, significant positive moderate correlations were also found between *T*-scores on the Shift scale and two continuous outcomes measures of CP (GAIN-SS Crime/Violence Screener and Conners 3 Defiance/Aggression scale), and thus, difficulties with cognitive and behavioural shifting may also play a partial role in the development of CP in this population.

The individual items that comprise the Inhibit subscale on the BRIEF are intended to assess difficulties with inhibitory control and impulsivity in everyday life (e.g., talks at the wrong times, gets out of control more than friends, has trouble putting the brakes on his/her actions, gets out of seat at the wrong times) and scores on the Inhibit scale have been shown to differentiate between children with ADHD, Combined Type from those with ADHD, Predominantly Inattentive Type, as well as controls (Gioia et al., 2000). Inhibitory control or behavioural inhibition has also been deemed a critical component of effortful control, or the active/voluntary aspect of self-regulation (Eisenberg et al., 1997; Frick & Morris, 2004). On the other hand, items on the Shift scale are designed to assess difficulties regarding the ability to flexibly shift one's attention, thoughts, and/or behaviours in real-life situations, and thus, adapt to differing situations and task-demands (e.g., becomes upset with new situations, resists changes of routine, food, places, etc., tries the same approach to a problem over and over even when it does not work, thinks too much about the same topic). Attentional control, specifically the ability to orient one's attention and thoughts away from distressing stimuli, has also been deemed an essential aspect of effortful control (Eisenberg et al., 1997; Frick & Morris, 2004); however, it is not clear whether the Shift subscale on the BRIEF adequately taps into this specific cognitive ability. Finally, the items that comprise the Emotional Control subscale on the BRIEF were

designed to tap into an individual's ability to modulate or regulate his/her emotions, as a lack of emotional self-regulation can manifest as extreme emotional reactions to stimuli or emotional liability (e.g., reacts more strongly to situations than other children, has explosive, angry outburst, becomes upset too easily, mood is easily influenced by the situation) (Gioia et al., 2000). As the name indicates, high scores on the Emotional Control subscale reflect poor emotional control (Gioia et al., 2000), however, it is also important to note that endorsements of the items that comprise this subscale could also suggest negative emotionality or an automatic tendency to react to situations with intense negative emotions (Rothbart, Ahadi, & Hershey, 1994). Unfortunately, it is quite difficult to clearly differentiate effortful control or "top-down" self-regulation from emotional/behavioural reactivity or "bottom-up" self-regulation and the specific subscales that comprised the Behavioral Regulation Index of the BRIEF were not developed to tease apart this difference (Cole, Martin, & Dennis, 2004; Bridgett, Burt, Edwards, & Deater-Deckard, 2015). Nevertheless, it appears that high scores on the Behavioral Regulation Index likely reflect real-world manifestations of difficulties with *both* top-down and bottom-up components of self-regulation.

Role of self-regulation and related executive functions in the development of conduct problems.

Although researchers from distinct, yet related disciplines (e.g., social psychology, cognitive psychology, developmental psychopathology, neuropsychology) have used various terms to describe finer-grained components of self-regulation (Bridgett et al., 2015), in the broadest sense, self-regulation can be defined as one's control over their attention, cognitions, emotions, and behaviours (Bandura, 1991; Karoly, 1993; Masten & Coatsworth, 1998). Linking the social and personality perspective of self-regulation with more recent work in the fields of

cognitive sciences and neuropsychology, Hofmann, Schmeichel, and Baddeley (2012) have highlighted the intricate connection between self-regulation and specific executive functions, including working memory, inhibitory control, and cognitive switching. Findings from decades of research suggest that the ability to self-regulate is critical to the development of competence across domains of life and its impairment can contribute to a wide range of adverse outcomes (for review see Bridgett et al., 2015; Eisenberg, Spinrad, & Eggum, 2010; Masten & Coatsworth, 1998; Moffitt et al., 2011). Specifically, there is a growing body of literature, across subdisciplines of psychology, linking specific aspects of self-regulation or EF to the development of CP. As stated previously, poor effortful control and negative emotionality have been consistently linked to the development of childhood CP in the general population (Earls & Jung, 1987; Eisenberg et al., 1996; Eisenberg et al., 2000; Eisenberg et al. 2001; Eisenberg et al. 2005; Eisenberg et al., 2009; McClowry et al., 1994). Accordingly, findings from a number of meta-analytic studies also suggest a robust and statistically significant link between CP and poor performance on measures of EF, however, the strength of this relationship varied significantly depending on the specific type of EF task utilized and how CP were operationalized (Morgan & Lilienfeld; Ogilvie, Stewart, Chan, & Shum, 2011; Schoemaker et al., 2013). Of particular interest from a developmental standpoint, longitudinal studies utilizing performance-based measures of EF (e.g., inhibitory control, working memory, shifting) have found that baseline proficiency and greater gains in EF across early childhood predict lower levels of parent- and teacher-rated externalizing problems (Hughes & Ensor, 2011; Riggs, Blair, & Greenberg, 2004; Utendale & Hastings, 2011). There is also some evidence to suggest that certain executive functions or aspects of self-regulation, including emotional reactivity, speed of inhibitory control processes, and risky decision making, are significantly and independently associated with CP,

after controlling for ADHD (Graziano, McNamara, Geffken, & Reid, 2013; Hobson, Scott, & Rubia, 2011). Interestingly, a more recent study found impaired inhibition and switching ability in the presence of distressing emotional stimuli in aggressive adolescents with CD compared to controls, suggesting that executive functions in this population may be negatively impacted or depleted by emotional distress (Euler, Sterzer, & Stadler, 2014). Moreover, results from a series of well-designed experiments conducted with undergraduate students suggest that the underlying neuropsychological resources necessary for self-regulation are finite, and that depleting these resources can increase the likelihood of aggressive behaviour in response to external provocation, however, this relationship was moderated by trait self-control (Dewall, Baumeister, Stillman, & Gailliot, 2007).

From a theoretical standpoint, Frick and Morris (2004) postulate that deficits with self-regulation can have both a direct and indirect influence on the development of CP in children and youth. A child who is more emotionally reactive or more likely to respond to new stimuli with intense negative emotions, and who also has difficulty inhibiting their behaviour and flexibly controlling their attention/cognitions, is less able to quickly and successfully adapt to changing situations and may be more prone to defiant/aggressive reactions in everyday life (Frick and Morris, 2000; Hubbard et al., 2002). As stated previously, individuals prone to developing CP may have less cognitive and behavioural control when exposed to emotionally distressing situations (Euler, Sterzer, & Stadler, 2014), which in turn, may increase the likelihood of aggressive responding (Dewall, Baumeister, Stillman, & Gailliot, 2007). Conversely, emotional dysregulation paired with impaired behavioural and attentional control can also negatively affect social information processing, which then increases the likelihood of maladaptive responses in social situations (e.g., verbal or physical aggression) (Crick & Dodge, 1996; Dodge & Pettit,

2003; Frick & Morris, 2004). In support of this hypothesis, a more recent longitudinal study found that better executive control predicted more adaptive social information processing in preschool children ($N = 316$) (Denham, et al., 2014). Moreover, Fischer, Greitemeyer, & Frey (2008) found that depleting an individual's self-regulation resources significantly increases the likelihood of engaging in confirmatory information processing. There is also some evidence to support the hypothesis that problems with emotion regulation, particularly negative emotional reactivity, can undermine the ability to process and internalize social norms or rules of conduct typically learned following a disciplinary encounter (Kochanska 1993; 1995; 1997). Although social information processing was not directly assessed in the current study, it is possible that the link between poor self-regulation and CP in youth with FASD may be partially mediated by maladaptive patterns of information processing (Dodge & Pettit, 2003). Greenbaum, Stevens, Nash, Koren, & Rovet (2009) found that children with FASD performed more poorly on certain measures of social cognition and facial emotion processing compared to children with ADHD and controls. However, bivariate correlations between scores on the social cognition/emotion processing tasks and overall levels of externalizing behaviours in the FASD group were not significant (Greenbaum et al., 2009).

Linking PAE to conduct problems through brain-based impairments in self-regulation.

Our findings that caregiver ratings of emotional/behavioural control at diagnosis significantly predict later CP in adolescents with FASD, even after controlling for ADHD and a number of other biopsychosocial risk factors, lends further support for the critical role of self-regulation in the general development of CP. That said, Eisenberg et al. (2010) pointed out that there are likely multiple pathways leading to the development of CP and that poor self-regulation

may be a particularly critical risk factor in some pathways, but may play a lesser role in others.

The results of the current study suggest that deficits in emotional/behavioural regulation are a particularly salient risk factor for CP in youth with FASD and it is hypothesized that PAE-induced damage to brain areas underlying these executive processes may partially explain the particularly high rate of CP documented in this clinical population.

Individuals with FASD exhibit structural abnormalities in the frontal lobe, particularly in the orbitofrontal cortex (Sowell et al., 2002) and frontal lobe gray matter (Astley, Aylward, et al., 2009). Furthermore, microstructural abnormalities have been documented in white matter pathways innervating the frontal cortex (Fryer et al., 2009; Lebel et al., 2008; Sowell et al., 2008). Research suggested that the orbitofrontal cortex plays a role in emotion-related executive functions, motivational states, decision-making, and behavioural inhibition (Horn, Dolan, Elliott, Deakin, & Woodruff, 2003; Jollant et al., 2010; Rolls, 2004; Rudebeck & Murray, 2014; Tekin & Cummings, 2002; Winstanley, 2007). Individuals with lesions in the orbitofrontal cortex region tend to exhibit impulsive and disinhibited behaviour, as well as emotional lability and poor decision-making (Blumenfeld, 2010; Tekin & Cummings, 2002). More recently, the orbitofrontal cortex has been linked to forecasting the outcomes of choices or actions and appraising the value of such outcomes in the moment (Rudebeck & Murray, 2014). In addition to the orbitofrontal cortex, research suggests that other regions of the limbic cortex may be particularly sensitive to alcohol teratogenicity, specifically the cingulate gyrus (Bjorkquist, Fryer, Reiss, Mattson, & Riley, 2010) and parahippocampal gyrus (Coles et al., 2001). Compared to controls, individuals with histories of heavy PAE have significantly reduced cingulate white matter, even after controlling for total cranial white matter volume (Bjorkquist et al., 2010). Coles et al. (2011) found that individuals with FASD had significantly reduced parahippocampal

volumes relative to controls, although these researchers did not investigate regional volume loss relative to total brain volume. The anterior cingulate has been linked to executive attention/control (Posner & Rothbart, 1998), affective processing and regulation (Devinsky, Morrell, & Vogt, 1995), conflict monitoring, and decision-making (Botvinick, 2007). Interestingly, results from a recent meta-analytic study suggest that CP in the general population are also associated with structural and functional reductions in frontal lobe structures, specifically the orbitofrontal cortex, anterior cingulate cortex, and dorsolateral prefrontal cortex. These findings are consistent regardless of age, gender, psychiatric comorbidity, or type of antisocial population studied (e.g., violent vs. non-violent, residing in an institution vs. the community) (Yang & Raine, 2009). Thus, it appears that there is significant overlap between brain abnormalities found in those with FASD/PAE and individuals with CP in the general population, and more importantly, these brain areas (e.g., orbitofrontal cortex, anterior cingulate) appear to play a significant role in the executive processes that underlie self-regulation.

PAE also results in damage to white matter tracts in the brain, most notably the corpus callosum (e.g., Astley, Aylward, et al., 2009; Bookstein, Sampson, Streissguth, & Connor, 2001; Fryer et al., 2009; Lebel et al., 2008; Riley et al., 1995; Sowell et al., 2001, 2008). Recent conceptualizations suggest that beyond its role in motor and sensory function, white matter is critical for high-order cognitive processing (Wozniak & Muetzel, 2011). Evidence from studies of brain-based disorders affecting white matter, such as multiple sclerosis, suggest that white matter integrity is critical for normal EF, attention, processing speed, working memory and motor skills (Chanraud, Zahr, Sullivan, & Pfefferbaum, 2010; Pfefferbaum et al., 2000; Rao, 1995; Sullivan & Pfefferbaum, 2011; Wilde et al., 2006; Wozniak et al., 2007). In line with this conceptualization, a longitudinal study of a population-based cohort of children in the

Netherlands ($N = 784$) found that a smaller corpus callosum length in infancy significantly predicted poorer emotional control and behavioural inhibition on the BRIEF – Preschool Version at four years of age, even after adjusting for age, gender, gestational age at birth, head circumference, maternal age, maternal education, and maternal smoking history (Ghassabian et al., 2013). Thus, it appears that PAE-induced damage to the specific brain areas, particularly the corpus callosum, orbitofrontal cortex, and anterior cingulate, may contribute to the significant impairments in emotional/behavioural regulation found in this population, and furthermore, this link provides a biologically plausible mechanism by which PAE increases the risk of CP later in development.

Effects of family environment on self-regulation and related executive functions

It is important to consider that, consistent with our findings, research suggests that PAE often co-occurs with the experience of early life adversity (Astley, 2010; Cannon et al., 2012; Fergusson, Lynskey, & Horwood, 1996; Hans, 1999; Sarkola, Kahila, Gissler, & Halmesmäki, 2007; Sood et al., 2001; Streissguth, Barr, Kogan, & Bookstein, 1996; Streissguth et al., 2004; Werner, 1986), which may also negatively impact the development of executive functions and self-regulatory skills. Specifically, a number of studies have found a significant association between childhood maltreatment and deficits in specific executive functions involved in self-regulation (Hofmann et al., 2012), including working memory, inhibitory control (Cowell, Cicchetti, Rogosch, & Toth, 2015; DePrince, Weinzierl, & Combs, 2009; Masson, Bussières, East-Richard, R-Mercier, & Cellard, 2015; Mezzacappa, Kindlon, & Earls, 2001; Sonuga-Barke, & Rubia, 2008), and emotional regulation (Alink, Cicchetti, Kim, & Rogosch, 2009; Tottenham et al., 2010). Moreover, a review of the findings from functional and structural imaging studies suggest that childhood maltreatment predominantly affects brain areas comprising fronto-limbic

and fronto- cortical networks, including the orbitofrontal cortex and anterior cingulate, which mediate behavioural and emotional regulation (Hart & Rubia, 2012). Finally, results from more recent studies examining the link between childhood maltreatment, emotion regulation, and behavioural problems suggest that poor emotion regulation partially mediates the relationship between child maltreatment and externalizing behaviours (Kim & Cicchetti, 2010; Langevin, Hébert, Cossette, 2015), with complete mediation effects found in children with insecure attachment styles (Alink, Cicchetti, Kim, & Rogosch, 2009). In the current study, we found that abuse history failed to remain a significant predictor of various measures of CP in youth with FASD once emotional/behavioural regulation was accounted for. Thus, it is possible that maltreatment experienced by children with FASD may primarily increase the risk of CP indirectly by further disrupting the development of brain regions critical to self-regulation.

Similarly, parenting practices early in development predict later EF (Bernier, Carlson, & Whipple, 2010; Hammond, Muller, Carpendale, Bibok, & Liebermann-Finstone, 2012) and effortful control (Kochanska, Murray, & Harlan, 2000; Lengua, Honorado, & Bush, 2007). Moreover, there is some evidence to suggest that the relationship between parenting practices and effortful control may be bidirectional in nature across time (Eisenberg, Taylor, Widaman, & Spinrad, 2015). Furthermore, EF and effortful control has been shown to mediate the link between parenting behaviours and child externalizing behaviours in the general population (Belsky, Pasco Fearon, & Bell, 2007; Chang, Olson, Sameroff, & Sexton, 2011; Eisenberg et al., 2005; Reuben, et al, 2015; Sulik et al., 2015; Valiente, et al., 2006), however, one study found that this relationship only held true for boys (Chang, Olson, Sameroff, & Sexton, 2011). Although parenting practices (other than abuse) were not directly assessed in the current study, there is evidence to suggest that parental alcohol abuse is associated with more maladaptive

parenting practices in biological parents, including inappropriate parental expectations of children, lack of empathy for children's needs, greater value of physical punishment, more punitive discipline practices, and more parent-child role reversal (Gallant, Gorey, Gallant, Perry, & Ryan, 1998; Miller, Smyth, & Mudar, 1999), which then can negatively impact the development of the child's self-regulatory capacities. Research on parents with cocaine abuse issues has found that maternal harshness accounted for the relationship between prenatal cocaine exposure and young children's effortful control. The association between maternal harshness and effortful control was then further moderated by the child's autonomic reactivity (Eiden, Godleski, Schuetze, & Colder, 2015). Eiden, Edwards, and Leonard (2007) found that paternal alcohol problems predicted both lower maternal and paternal warmth/sensitivity early in the child's life (i.e., at 2 years). A longitudinal relationship was then found between lower maternal warmth/sensitivity and poorer self-regulation skills in children at 3 years, which then went on to predict higher externalizing problems later in childhood (Eiden, Edwards, & Leonard, 2007).

Despite the lack of literature focusing on how the postnatal environment, and specifically parenting practices, may moderate the teratogenic effects of alcohol on child development, it is reasonable to postulate that the lack of sensitive parenting likely experienced by many individuals with FASD early in life may have a further negative impact on the development of EF and self-regulation. In support of this theory, Lynch et al. (2003) found that family variables, specifically higher levels of parental verbal aggression and lower levels of parental supervision, were more strongly related to adolescent delinquency than PAE in a community-based sample of predominantly African American families with low SES. Along this line of reasoning, there may be a protective effect of being adopted or placed into a stable and nurturing home earlier on in life, on the later development of EF and self-regulation skills in individuals with PAE. However,

as stated previously, we were not able to directly assess the quality of any of the home environments lived in by our cohort of youth with FASD. Future studies should focus on investigating whether abuse history, parenting practices, and family stability moderate the effect of behavioural/emotional regulation on CP. Future research efforts should also focus on coordinating multi-centred studies so that larger sample sizes can enable the utilization of more sophisticated statistical techniques, such as structural equation modeling, to parcel out the interaction of or mediation effects of specific biopsychosocial risk factors for CP across development.

Predictors of Categorical Measures of Conduct Problems

The current study found a number of unique predictors for categorical measures of CP that represent adverse real-world outcomes (i.e., trouble with the law, school suspensions). Increased placement instability and male sex were found to be important predictors of the negative social consequences of CP. Moreover, difficulties with emotional/behavioural regulation were associated with an increased risk of school suspension (discussed in detail above). Unexpectedly, having an ADHD diagnosis and increased problems with peer relations were found to significantly reduce the likelihood of experiencing trouble with the law and being suspended from school, respectively.

Sex differences in the social consequences of conduct problems.

Compared to females with FASD, males were found to be 13 and 20 times more likely to have come in contact with the law and have been suspended from a school, respectively. Being male was also found to significantly increase the odds of experiencing trouble with the law and having a disrupted school experience (i.e., ever suspended, expelled, or dropped out) in the cross-sectional study of American youth and young adults with FASD conducted by Streissguth

and colleagues (2004); however, these researchers found only a 2-fold increase in these outcomes associated with sex. Our findings are more in line with results from research in the general population, which suggest that males are 10 to 15 times more likely to meet criteria for life-course-persistent CP than females (Moffitt, 2003, 2006; Moffitt, Caspi, Rutter, & Silva, 2001).

Interestingly, our bivariate analyses revealed that mean *T*-scores on continuous measures of CP (e.g., CBCL Oppositional Defiant Problems scale, GAIN Crime/Violence Screener) did not significantly differ between males and females. Moreover, sex was not a significant predictor of any of the continuous measures of CP following multiple linear regression analysis. Thus, it is possible that the males and females with FASD in our cohort demonstrate similar degrees of CP, but that males with FASD may engage in more overt forms of CP, such as destruction of property or physical aggression, which are more likely to be noticed, and in turn, lead to serious consequences such as trouble with the law or school suspension. In support of this hypothesis, one study utilizing differential item functioning (DIF) found that when the level of the latent CP was controlled, boys in kindergarten were rated by teachers as exhibiting more overt CP (e.g., fights, breaks things), while girls in kindergarten were rated as exhibiting more covert or nonphysical CP (e.g., stubborn, takes property) (Wu et al., 2012). Furthermore, CP in males may be more likely to be viewed primarily as a behavioural issue and targeted with behavioural strategies, such as negative consequences. Whereas, in females with FASD, significant CP may be more commonly conceptualized as an indicator or consequence of underlying emotional or mental health issues (opposed to a core behavioural issue), and thus, the presence of CP in females with FASD may be the impetus for adults to seek therapeutic services, such as individual psychotherapy or residential treatment. It will be important to further investigate how sex and

sex/gender impact the ways in which caregivers, teachers, and society as a whole, responds to CP in youth with FASD.

Family instability and trouble with the law.

We also found that family instability significantly increased the odds of coming into contact with the law for committing a criminal offence, with each additional home placement associated with approximately a 2-fold increase in risk ($OR = 1.82$). Consistent with our findings, Streissguth et al. (2004) found that, when average years per household by age 18 was converted into a dichotomous variable by dividing the distribution at the median, “fewer” years per household significantly increased the odds of trouble with the law ($OR = 2.10$) and confinement (i.e., being confined to a juvenile detention centre, jail/prison, psychiatric hospital, or inpatient substance-abuse treatment setting) ($OR = 2.27$). Results from their logistic regression models also revealed that average years per household substantially increased the odds of confinement ($OR = 7.35$), yet failed to significantly predict trouble with the law, after adjusting for the effects of the other environmental and diagnostic factors of interest. However, the variable “average years per household” was included as a nonlinear term in the regression model, suggesting a more complex relationship with the outcome variable.

Streissguth and colleagues (2004) also found that a lower percentage of life spent in a stable/nurturing home significantly predicted trouble with the law using logistic regression analysis, suggesting that the overall quality of the home environments in which an individual with FASD is placed may be a better predictor of CP than the frequency of placements changes. In line with this theory, Fagerlund et al. (2011) found that, after adjusting for a number of diagnostic and demographic variables, time spent in residential care was significantly and positively associated with externalizing problems on the CBCL in children and youth with

FASD, whereas the total number of home placements did not have a significant relationship with this outcome. Unfortunately, despite government regulations, residential care is often characterized by fewer opportunities for individualized caregiving/parenting and more frequent changes in “family” composition (i.e., high turnover for caregivers and children) than more traditional family environments (Roy, Rutter, & Pickles, 2000). As mentioned previously, however, the relationship between residential care and externalizing problems found in this study is not necessarily causal in nature, as children with FASD with severe externalizing problems are often hard to place and may end up in residential care because of their behavioural issues (Fagerlund et al., 2011). Regrettably, we were not able to assess the quality of the home environments in which our cohort lived, and thus, future research is necessary in order to further elucidate the specific role of the quality of the home environment in the development of CP in youth with FASD.

Inverse relationships between ADHD/social functioning and the social consequences of conduct problems.

Surprisingly, in our sample of youth with FASD, we found that having a formal ADHD diagnosis decreased the likelihood of having been in trouble with the law. It is unlikely that this unexpected relationship represents a true inverse association between underlying symptoms of ADHD and CP in FASD, given that decades of research has linked ADHD, and specifically, hyperactivity and impulsivity to the development of CP (e.g., Klinteberg, Andersson, Magnusson, & Stattin, 1993; Ware et al., 2013; Waschbusch, 2002; White, Moffitt, Caspi, Bartusch, Needles, & Stouthamer-Loeber, 1994). Alternatively, it is possible that, for some of the individuals with FASD coded as “no ADHD” in our study, their problems with attention and/or hyperactivity/impulsivity failed to reach the clinical threshold to warrant a formal

diagnosis, and thus, treatment with a stimulant medication was not explored. In fact, 96% youth in our sample with a comorbid ADHD diagnosis had a reported history of stimulant use (45/47), while only 33% of those without a formal diagnosis of ADHD had ever been treated with an attention medication (4/12). Stimulants have been shown to result in significant improvements in hyperactivity and impulsivity in children with FASD (Doig, McLennan, & Gibbard, 2008; Oesterheld et al., 1998; O’Malley, Koplin, & Dohner, 2000; Snyder, Nanson, Snyder, & Block, 1997), and thus, it is possible that the high rate of treatment with these medications in youth with a formal diagnosis of ADHD in our sample is what is underlying the protective effect found against trouble with the law. It is also possible that youth with FASD who received a formal diagnosis of ADHD also gained access to other interventions and supports (e.g., behaviour supports in school, IEP) earlier in life, compared to those who were not formally identified as having ADHD, which may have also helped to reduce the likelihood of experiencing trouble with the law later in life.

Finally, counter to our predictions, we found that lower *T*-scores on the Social Problems scale of the CPRS-R:L at diagnosis (i.e., fewer social problems endorsed) significantly increased the likelihood of having a history of school suspension. The Social Problems scale of the CPRS-R:L taps into difficulties making and keeping friends, peer rejection, and low self-esteem (e.g., does not get invited over to friends’ houses, loses friends quickly, feels inferior to others). In other words, adolescents with FASD with the lower *T*-scores on the Social Problems scale are likely relatively more socially competent and have more friends than those in the sample with higher *T*-scores on this scale. That said, mean *T*-scores on the Social Problems scale of the CPRS-R:L still fell within the Borderline Clinical range for this sample (mean *T*-score = 67.76 SD = 14.72), indicating relatively high levels of social problems in the sample as a whole.

However, the subset of adolescents with FASD that are socially competent enough to develop interpersonal relationships and maintain friendships, may actually end up doing so with more delinquent peers (Fast & Conry; 2009; Thomas et al., 1998). This pattern of affiliation may be due a variety of factors, including a strong desire for social acceptance, the tendency to be rejected by higher-functioning, prosocial peers, an increased likelihood of being accepted by peers with similar adverse life experiences, and poorer social decision-making skills (Fast & Conry; 2009; Greenbaum, Stevens, Nash, Koren, & Rovet, 2009). Thus, it is possible that an affiliation with a delinquent peer group may mediate the relationship between higher social functioning and increased odds of school suspension. Within the school environment, deviant peers may support, encourage, and even manipulate youth with FASD into committing antisocial acts, which ultimately result in an increased risk of school suspensions (Dishion & Dodge, 2005; Greenspan & Driscoll, 2014; Fast & Conry; 2009). It is also possible that some of the school suspensions incurred by youth with FASD in this sample may have been a result of truancy. It can also be hypothesized that youth with FASD may be more likely to skip school if they have other friends to do so with. Nevertheless, an important next step will be replicating these findings in a larger cohort of youth and further exploring how belonging to a delinquent peer group may play into the development of specific types of CP in this socially vulnerable population.

Interventions Targeting Self-Regulation to Mitigate Conduct Problems in FASD

Our results suggest that poor emotional/behavioural regulation is a particularly salient risk factor for CP in youth with FASD, and thus, future intervention efforts aimed at mitigating or preventing CP in individuals with FASD should focus on improving self-regulation skills. Although the research is limited, there are a number of intervention programs that have been shown to improve self-regulation and reduce problem behaviours in children, adolescents, and/or

emerging adults, including Dialectical Behaviour Therapy (DBT) (Geddes, Dziurawiec, & Lee, 2013; MacPherson, Cheavens, & Fristad, 2013; Rizvi & Steffel, 2014), Tool of the Mind (Barnett et al., 2008), the Promoting Alternative Thinking Strategies (PATHS) Curriculum (Kam, Greenberg, & Kusche, 2004; Riggs, Greenberg, Kusché, & Pentz, 2006), and the Alert Program for Self-Regulation® (Barnes, Vogel, Beck, Schoenfeld, & Owen, 2008; Nash et al., 2015; Wells, Chasnoff, Schmidt, Telford, & Schwartz, 2012). In line with our findings, a study examining the underlying mechanism by which the PATHS Curriculum affects child behaviour found that improvements in inhibitory control post-intervention significantly mediated the relationship between the treatment condition and teacher-reported externalizing problems at one-year follow-up (Riggs, Greenberg, Kusché, & Pentz, 2006). Even more exciting are results from a treatment study investigating a multi-pronged intervention that included the PATHS curriculum in formerly homeless families, which found that changes in children's EF fully mediated intervention-related changes in CP (Piehler et al., 2014). Moreover, of particular interest, two studies have documented improvements in self-regulation in children with FASD following the 12-week Alert Program for Self-Regulation (Nash et al., 2015; Wells, Chasnoff, Schmidt, Telford, & Schwartz, 2012). In the most recent study, Nash and colleagues found that children with FASD who received the 12-week Alert Program for Self-Regulation demonstrated significant improvements in behavioural inhibition and emotional control compared to wait-list controls, with improvements in inhibitory control maintained up to six months post-intervention. Moreover, children in the treatment group were found to have fewer parent-reported externalizing behaviour problems post-intervention, however, this finding only approached significance ($p = .08$) (Nash et al., 2015). Furthermore, this team of researchers found increased gray matter changes in brain regions critical for emotional/behavioural regulation in children

with FASD following treatment with the Alert Program, specifically in the left middle frontal gyrus, right frontal pole, and right anterior cingulate. These results, however, should be interpreted with caution, as they failed to remain significant once a correction for multiple comparisons was applied. Within the treatment group, significant positive correlations were also found between gray matter volumes in the left medial and right inferior frontal gyrus and scores on the BRIEF Emotion Regulation Index. A similar association was found between increased grey matter volumes of the bilateral superior and right middle frontal gyrus and improved performance on the Inhibition subtest of the NEPSY-II. These results suggest that the Alert Program may lead to positive neuroanatomical changes in brain areas that underlie emotional/behavioural regulation in children with FASD, providing further support for the efficacy of this intervention in improving self-regulation (Soh et al., 2015).

There is also evidence to suggest that benefits of improved self-regulation and related executive functions may generalize to other areas of life. Specifically, there is some evidence that poor EF significantly contributes to the real-life deficits in social and adaptive functioning commonly observed in individuals with FASD (Kully-Martens, Denys, Treit, Tamana, & Rasmussen, 2012; McGee, Fryer, Bjorkquist, Mattson, & Riley, 2008; Schonfeld, Paley, Frankel, & O'Connor, 2006; Ware et al., 2012). Thus, strengthening these higher-order functions may also lead to increased competency in these important domains of life. Finally, we recommend that interventions aimed at bolstering self-regulation and underlying EF should be implemented during early childhood and again in adolescence, as these periods of development are associated with increased brain plasticity, providing optimal opportunity for neurocognitive change and development (Bradshaw, Goldweber, Fishbein, & Greenberg, 2012).

Study Limitations and Future Directions

The current study had a number of noteworthy limitations regarding aspects of sampling, study design, and the statistical approaches utilized, which should be considered when interpreting the main findings. Firstly, our study population was composed of adolescents who were previously clinically referred to The Hospital for Sick Children for a formal FASD diagnostic assessment relatively early in life (mean age at diagnosis = 10.58 years). PAE can affect many neurodevelopmental domains; however, those with overt behavioural difficulties may be more likely to be identified as “at-risk” and referred for a formal FASD assessment earlier in development. Research has suggested that the majority of referrals to outpatient child and adolescent mental health clinics are due to CP, specifically ODD and CD (Loeber, Burke, Lahey, Winters, & Zera, 2000), and thus, it is possible that our clinical sample may have had higher rates of externalizing behaviours than other individuals with FASD who are diagnosed later in life (i.e., early adulthood).

Moreover, 97% of the study population were diagnosed with ARND and no participant met the criteria for a diagnosis of FAS. As stated previously, most individuals with PAE do not present with the full constellation of physical abnormalities required for a diagnosis of FAS (Rasmussen, Horne, & Witol, 2006; Streissguth & O’Malley, 2000). Furthermore, it is possible that those exhibiting the visible facial dysmorphology associated with PAE were more likely to have been labeled by medical professionals as having FAS or pFAS early in development, and therefore, may not have been referred for a multidisciplinary assessment at The Hospital for Sick Children during childhood or adolescence. In fact, the diagnostic criteria for FAS and pFAS set out by the Institute of Medicine in 1996 did not require evidence of behavioural or cognitive abnormalities, thereby enabling medical professionals to provide these diagnoses without the

necessity of a formal cognitive or neuropsychological assessment (Stratton, Howe, & Battaglia, 1996). Interestingly, Streissguth et al. (1996) found that adolescents and adults with FAE (i.e., ARND or pFAS) had higher rates of trouble with the law than individuals diagnosed with FAS (Streissguth et al., 2004). Moreover, Schonfeld et al. (2005) found that, while 30% of the total group of adolescents with heavy PAE met criteria for probable CD, none of the adolescents within the FAS subgroup did (Schonfeld et al., 2005). The finding of fewer externalizing problems documented in those with FAS may be the result of earlier identification and increased access to interventions and services for this visual subpopulation, due to their observable physical dysmorphology (Streissguth et al., 2004). Overall, the results of this follow-up study may overestimate the prevalence of CP in adolescents across the FASD spectrum and should not be directly generalized to the smaller proportion of individuals displaying the observable physical dysmorphology associated with an FAS or pFAS diagnosis. On the other hand, a significant strength of our study is that our follow-up sample appears to be representative of the individuals who were diagnosed with FASD at The Hospital for Sick Children across Ontario during the study years, specifically with regard to sex, race, guardianship, age of diagnosis, year of diagnosis, diagnostic category, and intellectual ability. In other words, our prospective follow-up study does not seem to be at risk for bias from differential loss to follow-up.

Due to the prospective study design, we were only able to gather a crude measure of maternal criminality (e.g., whether or not the adolescent's mother had a criminal record or history of incarceration) and often from a third party (e.g., adoptive parent, family case worker), which likely affected the accuracy of this data. Potential errors in measurement of this variable may have led to an underestimate of the association between maternal criminality and CP in youth with FASD. More detailed information about the criminal activity in which mothers

engaged, specifically whether or not they had a history of violent or aggressive offending, may have improved the predictive power of this variable, as research has suggested that aggressive behaviour is particularly heritable (Burt, 2009; Eley, Lichtenstein, & Stevenson 1999; Eley, Lichtenstein, & Moffitt, 2003; Lichtenstein, Tuvblad, Larsson, and Carlström, 2007). Moreover, we were not able to gather data regarding the criminal history of approximately one third of the biological fathers in this sample, and thus, could not create an overall estimate of parental criminality. It is possible that paternal or overall parental criminality may have been a better estimate of genetic risk, as some studies suggest that paternal APD or criminality is more strongly associated with CP in children than is maternal antisocial or delinquent behaviour (Frick et al., 1992; Lahey et al., 1988b; Stewart et al., 1980).

Another noteworthy limitation of the current study mentioned previously is that we were not able to assess the quality of the adolescent's current home environment or that of the adolescent's longest home placement, which may be an important mitigating or protective factor for the development of CP. Similarly, parental supervision and involvement could not be directly assessed in the current study, although previous research has consistently found these specific elements of parenting to be significantly associated with CP in the general population (for review see Loeber and Stouthamer-Loeber, 1986; Farrington & Hawkins, 1991; Van Voorhis et al., 1988). It is possible that children with multiple biological and neuropsychological risks factors for CP may be placed into a stable and nurturing family environment, and are subsequently provided with warm and responsive parenting, along with developmentally appropriate supervision and limit setting, which may serve to buffer the impact of other individual risk factors for the development of CP. As stated previously, future longitudinal research is necessary in order to further elucidate the specific role the family environment and parenting factors play in

the development of CP in youth with FASD, particularly given the high prevalence of early caregiving disruptions, abuse, and neglect.

The current study relied heavily on caregiver-reports to assess potential predictors and later expression of CP. Future studies should attempt to replicate our findings using performance-based measures of neuropsychological functioning, specifically attention and executive functions underlying self-regulation. Moreover, outcome measures should be expanded to include teacher- and clinician-rated measures of CP, as well as more detailed information around criminal history (e.g., number/type of charges). Finally, we also lacked a measure of callous/unemotional traits in our cohort, which have been proposed to play a significant role in covert and proactive externalizing problems (Frick & Morris, 2004).

The current study may be underpowered to detect smaller, yet important effects of the selective biopsychosocial predictors on measures of CP. Moreover, our modest sample size did not allow for the use of more sophisticated statistical analyses necessary to further tease apart more complex relationships between our predictors and outcome measures. Larger, multi-site studies are likely necessary to enable the utilization of more sophisticated statistical techniques, such as structural equation modeling, in order to parcel out the possible mediation and/or moderation effects of specific biopsychosocial risk factors on CP in FASD. Of specific interest, future studies should investigate whether abuse history or other early environmental risk factors partially account for (i.e., mediate) the relationship between emotional/behavioural regulation and CP in this high-risk population.

Finally, future research should also aim to uncover the neuroanatomical or brain-based correlates of CP in FASD using various imaging technologies (e.g., MRI, DTI, fMRI). Uncovering potential relationships between CP and structural/functional abnormalities in brain

areas linked to executive functions, specifically emotional/behavioural regulation skills, is a critical next step in further substantiating the link being PAE and CP.

Conclusion

Poorer self-regulation at diagnosis significantly predicted self-reported delinquency, caregiver ratings of defiance and aggression, as well as a history of school suspensions in youth with FASD at follow-up, even after accounting for the effects of other important biopsychosocial predictors. These results suggest that deficits in self-regulation are a particularly salient risk factor for CP in youth with FASD. Remarkably, this is the first study to investigate the role of self-regulation in the development of CP in this population. Our findings, however, are consistent with the growing body of literature linking CP in the general population to specific deficits in self-regulation and related executive functions. Thus, future intervention efforts aimed at preventing or mitigating CP in individuals with FASD should focus on improving self-regulation skills through direct training. Targeted interventions for this high-risk population are long overdue and will likely lead to substantial benefits for individuals, their families, and society as a whole.

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Table 1

Demographic Characteristics of Adolescent Participants

Demographic/Family Characteristics	Percentage	n
Gender		
Male	71.19	42
Female	28.81	17
Ethnicity		
Caucasian/European	54.24	32
Aboriginal	8.47	5
African/West Indian	3.39	2
Hispanic	3.39	2
Caucasian/European & African/West Indian	18.64	11
Caucasian/European & Aboriginal	6.78	4
Other Mixed Ethnicities	5.08	3
Home Placement at Follow-Up		
Biological Parent(s)	8.47	5
Adoptive Parent(s)	25.42	15
Foster Parent(s)	33.90	20
Biological Grandparent(s)	16.95	10
Other Biological Relative(s)	3.39	2
Group Home	5.08	3
Residential Treatment Facility	3.39	2
Semi-independent/Assisted Living	3.39	2
SES		

High	3.39	2
Medium-High	23.73	14
Medium	27.12	16
Medium-Low	16.95	10
Low	13.56	8
Not Applicable ^a	11.86	7
Unavailable ^b	3.39	2

^a A valid measure of SES could not be obtained for those under the care of CAS and living in a group home, residential treatment facility or semi-independent housing facility, and thus had no parental figures. ^b Missing data.

Table 2

Prevalence of Scores in the Elevated Range, Mean T-Scores and Standard Deviations for the Behavioural Regulation Index and Sub-Scales on the BRIEF

Index/Scale	% Participants Elevated ^a (n)	M	SD
Index			
Behavioural Regulation	79.66 (47)	72.78	12.69
Scales			
Inhibit	76.27 (45)	72.64	12.08
Shift	69.49 (41)	69.03	13.41
Emotional Control	69.49 (41)	67.90	12.98

^aT-scores ≥ 65 (i.e., ≥ approximately 90th percentile, as scales are not normally distributed)

Table 3

*Multiple Linear Regression Model for the Prediction of CBCL Oppositional Defiance Problems**T-Scores from Selected Biopsychosocial Risk Factors in Adolescents with FASD (n = 59)*

Predictor	B	SE(B)	p	95% CI
Age	-0.08	0.05	.085	[-0.18, 0.01]
Male	-3.26	2.61	.218	[-8.50, 1.99]
Maternal Criminality	-1.01	2.87	.725	[-6.78, 4.75]
Abuse History	0.15	2.34	.949	[-4.55, 4.86]
Family Instability ^a	-0.62	1.03	.545	[-2.68, 1.43]
ADHD Diagnosis	4.34	2.86	.136	[-1.41, 10.09]
BRIEF Behavioural Regulation ^b	0.23	0.11	.037*	[0.01, 0.44]
CPRS-R:L Social Problems ^b	-0.03	0.08	.717	[-0.20, 0.14]

Note. $R^2 = 0.23$, $F(8, 50) = 1.88$, $p = .085$. BRIEF = Behaviour Rating Inventory of Executive Function; CPRS-R:L = Conners' Parent Rating Scales-Revised: Long Form; ^a[total number of placements with different caregivers/adolescent's age in months at follow-up] x 100; ^bT-Score.
* $p < 0.05$

Table 4

Multiple Linear Regression Model for the Prediction of CBCL Conduct Problems T-Scores from Selected Biopsychosocial Risk Factors in Adolescents with FASD (n = 59)

Predictor	B	SE(B)	p	95% CI
Age	-0.08	0.05	.156	[-0.19, 0.03]
Male	0.68	2.98	.820	[-5.31, 6.68]
Maternal Criminality	0.97	3.28	.769	[-5.62, 7.56]
Abuse History	-1.23	2.68	.647	[-6.61, 4.14]
Family Instability ^a	-0.14	1.17	.906	[-2.49, 2.21]
ADHD Diagnosis	-0.08	3.27	.981	[-6.65, 6.49]
BRIEF Behavioural Regulation ^b	0.15	0.12	.236	[-0.10, 0.39]
CPRS-R:L Social Problems ^b	0.05	0.09	.591	[-0.14, 0.24]

Note. $R^2 = 0.13$, $F(8, 50) = 0.94$, $p = .491$. BRIEF = Behaviour Rating Inventory of Executive Function; CPRS-R:L = Conners' Parent Rating Scales-Revised: Long Form; ^a[total number of placements with different caregivers/adolescent's age in months at follow-up] x 100; ^bT-Score.
^{*} $p < 0.05$

Table 5

*Multiple Linear Regression Model for the Prediction of Conners 3 Defiance/Aggression T-**Scores from Selected Biopsychosocial Risk Factors in Adolescents with FASD (n = 59)*

Predictor	B	SE(B)	p	95% CI
Age	-0.07	0.09	.449	[-0.26, 0.11]
Male	-4.34	5.05	.395	[-14.49, 5.82]
Maternal Criminality	2.79	5.55	.617	[-8.36, 13.95]
Abuse History	0.33	4.53	.942	[-8.77, 9.44]
Family Instability ^a	-0.71	1.98	.720	[-4.70, 3.27]
ADHD Diagnosis	7.53	5.54	.180	[-3.60, 18.66]
BRIEF Behavioural Regulation ^b	0.51	0.21	.018*	[0.09, 0.93]
CPRS-R:L Social Problems ^b	-0.01	0.16	.939	[-0.33, 0.31]

Note. $R^2 = 0.26$, $F(8, 50) = 2.16$, $p = .047^*$. BRIEF = Behaviour Rating Inventory of Executive Function; CPRS-R:L = Conners' Parent Rating Scales-Revised: Long Form; ^a[total number of placements with different caregivers/adolescent's age in months at follow-up] x 100; ^bT-Score.
^{*} $p < 0.05$

Table 6

Multiple Linear Regression Model for the Prediction of GAIN-SS Crime/Violence Screener Raw Scores from Selected Biopsychosocial Risk Factors in Adolescents with FASD (n = 58)

Predictor	B	SE(B)	p	95% CI
Age	0.01	0.01	.484	[-0.02, 0.04]
Male	-0.32	0.79	.691	[-1.91, 1.28]
Maternal Criminality	0.82	0.87	.348	[-0.92, 2.56]
Abuse History	0.66	0.71	.357	[-0.77, 2.10]
Family Instability ^a	0.34	0.31	.276	[-0.28, 0.97]
ADHD Diagnosis	0.16	0.87	.854	[-1.58, 1.90]
BRIEF Behavioural Regulation ^b	0.07	0.03	.048*	[0.00, 0.13]
CPRS-R:L Social Problems ^b	-0.02	0.02	.442	[-0.07, 0.03]

Note. $R^2 = 0.25$, $F(8, 49) = 2.09$, $p = .055$. BRIEF = Behaviour Rating Inventory of Executive Function; CPRS-R:L = Conners' Parent Rating Scales-Revised: Long Form; ^a[total number of placements with different caregivers/adolescent's age in months at follow-up] x 100; ^bT-Score.
* $p < 0.05$

Table 7

Multiple Logistic Regression Model for the Prediction of Contact with the Law for Committing a Criminal Offence from Selected Biopsychosocial Risk Factors in Adolescents with FASD (n = 59)

Predictor	B	SE(B)	p	OR (e^B)	95% CI (e^B)
Age	0.03	0.02	.105	1.03	[1.00, 1.07]
Male	2.59	1.30	.046*	13.28	[1.58, 350.98]
Maternal Criminality	0.35	1.02	.730	1.42	[0.19, 11.38]
Abuse History	-0.63	0.95	.506	0.53	[0.07, 3.13]
Family Instability ^a	1.09	0.52	.035*	2.96	[1.30, 9.70]
ADHD Diagnosis	-2.10	0.99	.035*	0.12	[0.01, 0.77]
BRIEF Behavioural Regulation ^b	-0.02	0.04	.638	0.98	[0.91, 1.06]
CPRS-R:L Social Problems ^b	-0.06	0.03	.066	0.94	[0.88, 1.00]

Note. OR = odds ratio; CI = confidence ratio; e^B = exponentiated B; BRIEF = Behaviour Rating Inventory of Executive Function; CPRS-R:L = Conners' Parent Rating Scales-Revised: Long Form; ^a[total number of placements with different caregivers/adolescent's age in months at follow-up] x 100; ^bT-Score.

* $p < 0.05$.

Table 8

Multiple Logistic Regression Model for the Prediction of Contact with the Law for Committing a Criminal Offence from Age, Sex, Maternal Criminality, Abuse History, Number of Home Placements, ADHD Diagnosis, Emotional/Behavioural Regulation, and Social Problem (n = 59)

Predictor	B	SE(B)	p	OR (e^B)	95% CI (e^B)
Age	0.02	0.02	.309	1.02	[0.98, 1.05]
Male	2.57	1.29	.046*	13.11	[1.56, 341.88]
Maternal Criminality	0.33	1.02	.745	1.39	[0.19, 11.12]
Abuse History	-0.59	0.94	.528	0.55	[0.08, 3.23]
Home Placements ^a	0.60	0.28	.035*	1.82	[1.16, 3.48]
ADHD Diagnosis	-2.11	0.99	.033*	0.12	[0.01, 0.75]
BRIEF Behavioural Regulation ^b	-0.02	0.04	.634	0.98	[0.91, 1.06]
CPRS-R:L Social Problems ^b	-0.05	0.03	.082	0.95	[0.89, 1.00]

Note. OR = odds ratio; CI = confidence ratio; e^B = exponentiated B; BRIEF = Behaviour Rating Inventory of Executive Function; CPRS-R:L = Conners' Parent Rating Scales-Revised: Long Form; ^atotal number of different home placements with different caregivers; ^bT-Score.

* $p < 0.05$.

Table 9

Multiple Logistic Regression Model for the Prediction of a History of School Suspension from Selected Biopsychosocial Risk Factors in Adolescents with FASD (n = 59)

Predictor	B	SE(B)	p	OR (e^B)	95% CI (e^B)
Age	0.02	0.02	.301	1.02	[0.99, 1.06]
Male	2.98	1.00	.003**	19.68	[3.38, 194.43]
Maternal Criminality	-1.55	1.03	.131	0.21	[0.02, 1.43]
Abuse History	-1.18	0.91	.196	0.31	[0.05, 1.71]
Family Instability ^a	1.01	0.53	.058	2.74	[1.09, 8.67]
ADHD Diagnosis	-0.84	1.05	.421	0.43	[0.04, 3.04]
BRIEF Behavioural Regulation ^b	0.08	0.04	.047*	1.08	[1.01, 1.18]
CPRS-R:L Social Problems ^b	-0.08	0.03	.025*	0.93	[0.86, 0.98]

Note. OR = odds ratio; CI = confidence ratio; e^B = exponentiated B; BRIEF = Behaviour Rating Inventory of Executive Function; CPRS-R:L = Conners' Parent Rating Scales-Revised: Long Form; ^a[total number of placements with different caregivers/adolescent's age at follow-up] x 100; ^bT-Score.

* $p < 0.05$. ** $p < 0.01$.

Table 10

Correlations Between T-scores on Subscales of the BRIEF Behavioural Regulation Index and Various Continuous Measures of Conduct Problems in Adolescents with FASD

	BRIEF Behavioural Regulation Index Subscales		
	Inhibit	Shift	Emotional Control
CBCL Oppositional Defiance Problems	.26*	.23	.32*
CBCL Conduct Problems	.28*	.19	.21
Conners 3-P(S) Defiance/Aggression scale	.33*	.37**	.41**
GAIN-SS Crime/Violence Screener ^a	.34**	.33*	.39**

Note. All correlations are between T-scores unless otherwise indicated. ^araw scores. BRIEF = Behaviour Rating Inventory of Executive Function; CBCL = Child Behaviour Checklist for Ages 6–18. Conners 3-P(S) = Conners Rating Scales 3rd Edition – Short Form; GAIN-SS = Global Appraisal of Individual Needs – Short Screener.

* $p < 0.05$. ** $p < 0.01$.

Table 11

T-tests and Descriptive Statistics for Subscales of the BRIEF Behavioural Regulation Index by Trouble with the Law

BRIEF Subscale	Group				95% CI ^a	<i>t</i>	<i>p</i>	<i>df</i>				
	TWL		No TWL									
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>								
Inhibit	72.58	10.40	72.68	12.92	[-6.70, 6.89]	0.03	.98	57				
Shift	66.58	14.58	70.20	12.85	[-3.87, 11.11]	0.97	.34	57				
Emotional Control	66.37	15.79	68.63	11.57	[-5.03, 9.54]	0.62	.54	57				

Note. ^a95% confidence interval for mean difference. BRIEF = Behaviour Rating Inventory of Executive Function; TWL = trouble with the law.

Table 12

T-tests and Descriptive Statistics for Subscales of the BRIEF Behavioural Regulation Index by History of School Suspension

BRIEF Subscale	Group				95% CI ^a	<i>t</i>	<i>p</i>	<i>df</i>				
	School		No School									
	Suspension	Suspension	Suspension	Suspension								
M	SD	M	SD									
Inhibit	75.29	8.40	69.71	14.76	[-11.76, 0.61]	-1.81	.08	57				
Shift	71.26	13.30	66.57	13.35	[-11.64, 2.27]	-1.35	.18	57				
Emotional Control	68.77	12.72	66.93	13.43	[-8.67, 4.97]	-0.54	.59	57				

Note. ^a95% confidence interval for mean difference. BRIEF = Behaviour Rating Inventory of Executive Function.

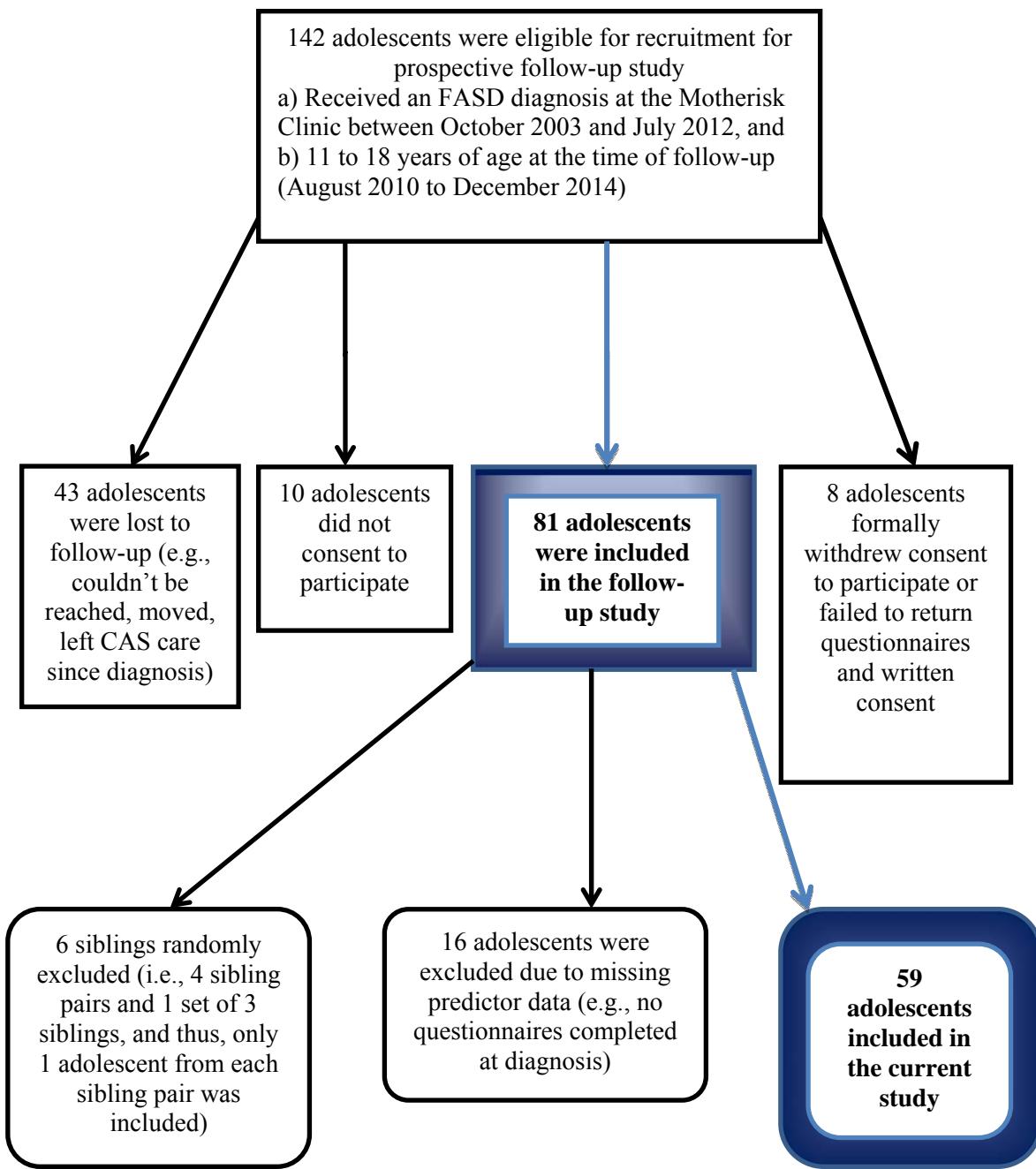


Figure 1. Flowchart of participant recruitment process.

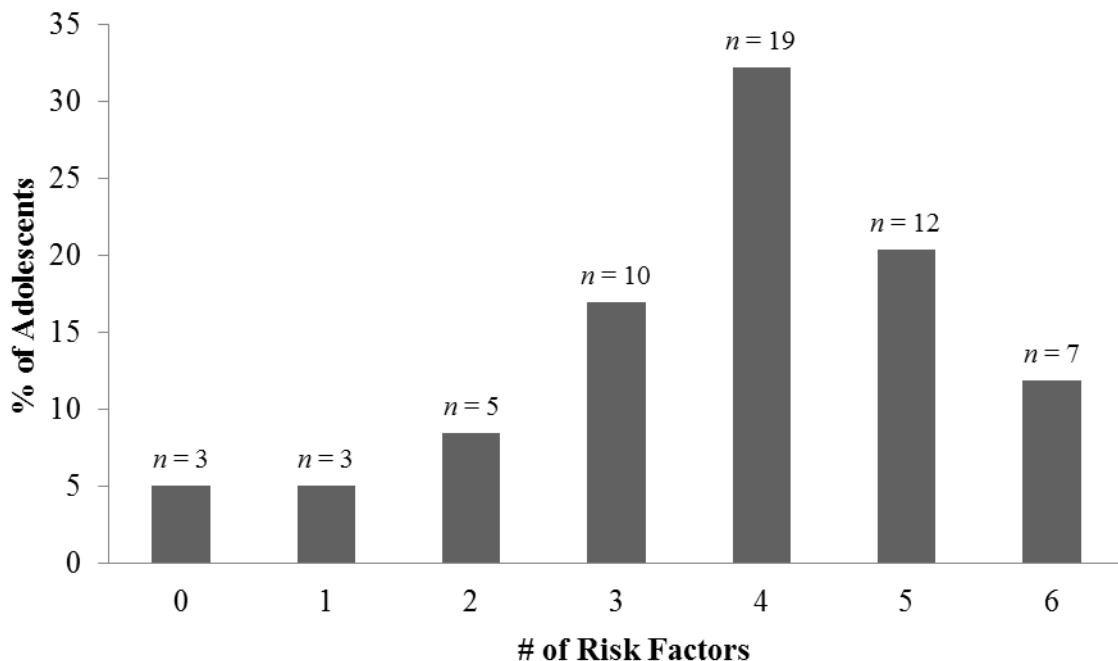


Figure 2. Percentage of adolescents with FASD who had experienced zero to six of the following dichotomous biopsychosocial risk factors for conduct problems: maternal criminality (biological mother had a reported criminal record or history of incarceration), family instability (3 or more home placements with different caregivers), history of physical and/or sexual abuse, ADHD, poor emotional/behavioural regulation skills (clinically elevated *T*-score on the Behavioural Regulation Index of the BRIEF), and social problems (clinically elevated *T*-score on the Social Problems subscale of the CPRS-R:L).