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The relationship between maternal depression, adolescent depression, and engagement in health-risk behaviours

by

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Abstract

Maternal depression is a known risk factor for offspring psychopathology; however, the role of maternal depressive symptoms in the adolescent engagement in health-risk behaviours (e.g., substance use and delinquency) needs further study. Using data from 2910 youth in the National Longitudinal Survey of Children and Youth (NLSCY), a nationally representative prospective cohort, the relationship between maternal depressive symptoms and engagement in a variety of health-risk behaviours at age 16-17, as well as time of onset of engagement, was examined. Latent class analysis was used to model trajectories of maternal depressive symptoms from age 4-15, and exploratory factor analysis was used to examine clusters of health-risk behaviours; five trajectories of maternal depressive symptoms during childhood were modeled, and five factors of behaviours were found. Findings indicate that adolescents exposed to high maternal depressive symptoms in mid-childhood engaged in more health-risk behaviours, earlier, than adolescents not exposed to maternal depressive symptoms.

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"While the research and analysis are based on data from Statistics Canada, the opinions expressed do not represent the views of Statistics Canada."

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Chapter 1 – Introduction

1.1 Introduction

Adolescence is a developmental period characterized by multiple physiological, behavioural, and emotional changes. Notably, adolescence marks an increase in the engagement in health-risk behaviours, such as sexual intercourse, substance abuse, violence/delinquency, and suicidal ideation. Furthermore, it is during adolescence in which adult drug-use patterns emerge (Chen & Kandel, 1995), underscoring the potential lifelong impact engagement in health-risk behaviours can have at this age. These behaviours are deemed to be 'health-risk behaviours' as they may entail negative physical and/or psychological health outcomes. For example, cigarettes and alcohol increase risk for the later development of cancer (Bagnardi, Blangiardo, La Vecchia, & Corrao, 2001; Carbone, 1992), and violent behaviours present risk of immediate physical harm. The development of health-risk behaviour engagement is associated with several risk factors, including anxiety and depression (Brooks, Harris, Thrall, & Woods, 2002; Brown et al., 2006; Waller et al., 2006).

During adolescence, there is also substantial increase in the prevalence of depression. Perhaps one of the strongest risk factors for the development of depression is exposure to maternal depression during childhood. The offspring of depressed parents are approximately twice to three times as likely to develop a mood disorder than offspring of non-depressed parents (Kovacs, Devlin, Pollock, Richards, & Mukerji, 1997; Naicker, Wickham, & Colman, 2012; Williamson, Birmaher, Axelson, Ryan, & Dahl, 2004). There are many theorized pathways

through which the intergenerational transmission of depression is thought to occur (Goodman & Gotlib, 1999; Goodman et al., 2011); furthermore, the negative behavioural and psychosocial effects, such as internalizing behaviour, of maternal depression on the child are evident as early as infancy, and persist into adolescence (Civic & Holt, 2000; Feldman et al., 2009; Kim-Cohen, Moffitt, Taylor, Pawlby, & Caspi, 2005; Naicker et al., 2012).While there is evidence to suggest that maternal depression places the child at risk for increased internalizing and externalizing behavioural difficulties, and both externalizing and internalizing difficulties have been linked to the engagement in health-risk behaviours (Brooks et al., 2002; Brown et al., 2006; Goodman, 2010; Waller et al., 2006), little research has investigated the potential association between maternal depression and the adolescent engagement in various health-risk behaviours. Thus, while maternal depression places adolescents at increased risk for psychopathology, and increased psychological difficulties are associated with the engagement in health-risk behaviour, the relationship between maternal depression, adolescent depression/anxiety, and the engagement in health-risk behaviours has not been thoroughly examined to date.

1.2 Health-Risk Behaviours

Adolescence is commonly characterized as a period of experimentation and exploration, especially with respect to behaviours that may confer additional risks to health, such as substance use. Research indicates these behaviours are widespread in adolescence; a 2005 report estimated that 75% of high school students in Michigan had tried alcohol, and 50% had tried an illicit drug

(Johnston, O'Malley, Bachman, & Schulenberg, 2005); in 2011, according to a report by the CDC, 38.7% of high school students had used alcohol in the 30 days prior to the survey, 18% had smoked cigarettes, 23% had used marijuana, 33% had been in a physical fight, and while nearly half the sample had engaged in sexual intercourse, only 60.4% of the students had used a condom during their last sexual intercourse (Centers for Disease Control and Prevention, 2012).

Engagement in health-risk behaviour is widespread in adolescence, and may entail negative outcomes. While not all behaviours confer equal risk, some behaviour patterns adopted during adolescence will be continued through to adulthood and become problematic. Research has demonstrated that alcohol dependence in young adults was predicted by adolescent (age 14-15) alcohol use and antisocial behaviour (Bonomo, Bowes, Coffey, Carlin, & Patton, 2004). Some behaviours, such as substance or tobacco use, may become problematic later in life or may have long-term health implications, and some behaviours confer a more immediate health risk, such as operating a motor vehicle under the influence of substances, substance overdoses, or belonging to a gang. While not all adolescents engage in risky behaviour, and levels of engagement vary between individuals, it is crucial to highlight risk factors for and differences in engagement to fully understand the nature of such engagement and to intervene with groups at higher risk for engagement.

1.2.1 Theoretical Framework of Health-risk Behaviour Engagement

During the transitional phase of adolescence, the primary aspects of normal development generally include increasing independence, autonomy, peer influence and affiliations, self-discovery, and cognitive maturation. Various theories emerged in the 1980s that describe risk-taking as normative behaviours during adolescence; theorists have described these behaviours as instrumental in various aspects of development, including peer acceptance, autonomy, and coping with stress, amongst other developmental processes (Igra & Irwin, 1996). Of these developmental domains, peer influence is a particularly strong risk factor for the development of these behaviours (Hawkins, Catalano, & Miller, 1992; Igra & Irwin, 1996; Lynskey, Fergusson, & Horwood, 1998; Prinstein, Boergers, & Spirito, 2001).

Furthermore, adolescent decision-making processes appear to differ from adult decision-making, which may reflect continuing cognitive development that occurs during this transitional stage. Evidence suggests that adolescents focus on the less, rather than more severe consequences of their behaviours. For example, one study demonstrated that condom use in youth was unrelated to their beliefs regarding STIs or pregnancy, but rather peer acceptance and ease of use (Kegeles, Adler, & Irwin, 1988). However, while these behaviours may contribute to a normal developmental transition into adulthood, engaging in these behaviours may present significant risk to the health and wellbeing of adolescents.

1.2.2 Implications/Importance of Health-risk Behaviour

Injury is frequently cited as one of the leading causes of death and hospitalization in youths aged 15-24, both self-inflicted and accidental; in fact, one study reported that in Canada between 1979 and 2003, 76% of all deaths and 17% of all hospitalizations in 15-19 year olds were injuries, including motor vehicle collisions, firearms, and suicide (Pan et al., 2007). Engagement in risky behaviours, including the use of substances, and violent delinquent behaviours, both present significant risks for serious personal injury, and they may be associated with worse injury severity (Andelic et al., 2010; Muula, Siziya, & Rudatsikira, 2011; Sells & Blum, 1996; Socie, Duffy, & Erskine, 2012; Tremblay, 2002). As such, these behaviours represent a significant risk factor for youth injury and mortality.

Moreover, not only can the consequences of health-risk behaviours themselves be life threatening, both in the short and long term, but early engagement in substance use is associated with increased suicide risk in both males and females (Cho, Hallfors, & Iritani, 2007). Suicide is the second leading cause of death in those aged 10-19 in Canada (Children's Mental Health Ontario, 2004), and suicide rates in Canadian adolescent females are rising (Skinner & McFaull, 2012). Suicide in adolescence is frequently the outcome of the combined risks of mood disorders with substance abuse (Shafii, Steltz-Lenarsky, Derrick, Beckner, & Whittinghill, 1988). Therefore, not only do these behaviours put the individuals' health at risk through the detrimental effects of the behaviour itself, they may also increase an individual's suicide risk. This represents an additional

threat that underlines the importance of understanding the development of health-risk behaviours in adolescence, and their relation to depressed mood. Therefore, these behaviours, such as substance use, unprotected sexual intercourse, and violence/delinquency are responsible for a large portion of mortality and morbidity experienced by adolescents and young adults, underscoring the importance of understanding the etiology of such behaviours, and intervening in those at a higher risk for engagement.

1.2.3 Factors Associated with Health-risk Behaviour

1.2.3.1 Affect Regulation and Stress

Affect regulation (i.e., the management and expression of emotions) has also been linked to health-risk behaviours. Neuroticism, defined as the tendency to experience enduring negative distressing emotions (Costa & McCrae, 1987), has been prospectively associated with engagement in health-risk behaviours (Carrasco & Del Barrio, 2007). Individuals who reported higher levels of maladaptive cognitive emotion regulation (i.e., emotional regulation techniques that are ineffective, and are often distressing), such as rumination, reported greater alcohol consumption than those with lower endorsement of such strategies (Goldstein, 2001). Chinese adolescents high in both neuroticism as well as maladaptive cognitive emotion regulation strategies engaged in a greater number of health-risk behaviours, including violence, self-injury, and substance use, in response to depressive symptoms compared to those with lower endorsement of each of those traits (Auerbach, Claro, Abela, Zhu, & Yao, 2010). The associations

between affect regulation and health-risk behaviours are unsurprising, as both maladaptive cognitive regulation strategies and neuroticism (i.e., persisting negative appraisal and experience of events) are cognitive vulnerabilities associated with depression (Beck, 1987); depression has been robustly associated with health-risk behaviour, detailed below.

The experience of stress has been linked to engagement in health-risk behaviour. Using animal models as a primary guide, stress has been linked to drug abuse and relapse (Ahmed & Koob, 1997; Koob & Le Moal, 1997; Shalev, Erb, & Shaham, 2010; Sinha, Shaham, & Heilig, 2011). Cigarette smoking is well known to be detrimental to one's respiratory health. Nicotine, the active drug in cigarettes, has been demonstrated to assuage the distress of negative affective states such as stress (Edwards, Anda, Gu, Dube, & Felitti, 2007). Furthermore, where stress is implicated in the engagement in substance use, it also perpetuates drug relapses; drug relapse and craving is significantly more likely when presented with stress (Sinha, 2009; Wallace, 1989). With respect to other health-risk behaviours, the experience of stress does not only predict substance use. A recent study of First Nations youth in America revealed that stressful life events were predictive of engagement in both substance use and other risky-behaviours, including measures of delinquency (Baldwin, Brown, Wayment, Nez, & Brelsford, 2011). The relationship between health-risk behaviours, particularly tobacco and alcohol consumption, and stress has also been replicated in the workplace among a sample of adults (Siegrist & Rodel, 2006). While substance use in particular may be encouraged by the occurrence of stressful life events, as

individuals may be seeking to self-medicate to relieve the experience of negative affect, it has also been linked to other health-risk behaviours.

1.2.3.2 Adolescent Depression and Anxiety

The development of health-risk behaviours has been linked to many risk factors, including depressive symptomatology, which has been robustly linked to engagement in a variety of health-risk behaviours. Adolescent depression has been linked to substance use, including tobacco and alcohol, delinquency as well as sexual activity (Brooks et al., 2002; Brown et al., 2006; Chen & Kandel, 1995; Katon et al., 2010; Kofler et al., 2011; Sprott, Doob, & Jenkins, 2001; Waller et al., 2006). The use of substances is often thought of as a coping mechanism for negative emotional states, therefore the association between drug use and depression is instinctive. Compared to youth abstaining from substance use or sexual intercourse, youth who participate in these health-risk behaviours had significantly higher odds of negative mental health outcomes, such as depression or suicidal ideation (Hallfors et al., 2004). Adolescent depression has also been implicated in an increased likelihood of unsafe sexual behaviours, such as nonuse of contraception or an increased number of sexual partners (Brown et al., 2006; Kosunen, Kaltiala-Heino, Rimpela, & Laippala, 2003). In a nationally representative sample of youth in the United States, those who engaged in health-risk behaviours such as violent or aggressive behaviour, substance use, and sexual intercourse had significantly higher odds of reporting depressed mood (Paxton, Valois, Watkins, Huebner, & Drane, 2007). The relationship between depressive symptomatology and unsafe sexual practices presents an additional public health

concern, as sexual intercourse can result in sexually transmitted infections (STIs) or unplanned pregnancy.

While those who experience depressive symptomatology are more likely to engage in health-risk behaviours, the relationship appears to be bidirectional. One study assessing longitudinal associations between substance use in adolescence and mental health in early adulthood found that substance dependence between the ages of 17 – 20 predicted an increase in depression between the ages of 20 – 24 (Marmorstein, Iacono, & Malone, 2010), whereas another study found depressive symptomatology to be predictive of increased substance use (Brooks et al., 2002). However, many studies assessing health-risk behaviours and depressive symptoms in adolescence have been cross-sectional, and therefore do not provide information regarding the temporality of the relationship between depressed mood and health-risk behaviour, perhaps explaining some of the discrepant findings. These results, therefore, do not provide information on whether engaging in health-risk behaviour causes depressed mood, or if depressed mood increases the likelihood of engaging in health-risk behaviours; however, depression and the engagement in health-risk behaviour is undoubtedly linked.

The association between depression and health-risk behaviours is disconcerting; not only are rates of depressed mood estimated to be as high as 10 to 15% in children and adolescents (Angold & Costello, 2001; Kessler & Walters, 1998; Smucker, Craighead, Craighead, & Green, 1986), 20 to 40% of depressed children relapse within 2 years, and 70% will relapse in adulthood (Colman,

Wadsworth, Croudace, & Jones, 2007; Dunn & Goodyer, 2006; Kovacs, Akiskal, Gatsonis, & Parrone, 1994; Lewinsohn, Clarke, Seeley, & Rohde, 1994). Depression in adolescence is evidently of great public health importance.

Anxiety has also been linked to engagement in health-risk behaviour, albeit less robustly. Research has revealed that those who scored in the deviant range of childhood anxiety and depressive symptoms had an increased risk of consuming 3,4-methylenedioxymethamphetamine (MDMA), a recreational amphetamine drug, in early adulthood (Huizink, Ferdinand, van der Ende, & Verhulst, 2006). However, a study in which participants had to make hypothetical decisions about engagement in health-risk behaviour demonstrated that those who were high in trait anxiety chose the safe alternative more often than those lower in trait anxiety (Mitte, 2007). Another study reported that anxiety was related to non-use of contraception at last sexual intercourse (Turner, Latkin, Sonenstein, & Tandon, 2011). The effects of depression and anxiety may exert themselves synergistically; among 'less than daily' adolescent smokers, those with higher levels of both depression and anxiety were 3.3 times more likely to report tobacco dependence in young adulthood (McKenzie, Olsson, Jorm, Romaniuk, & Patton, 2010). Anxiety and depression often co-occur; therefore the effect of one may be difficult to differentiate from another.

1.2.3.3 Parental Factors

Many different parental factors have been studied in the development of health-risk behaviour. A study demonstrated that African-American adolescent

girls were more likely to engage in health-risk behaviours, including risky sexual behaviour, marijuana and alcohol use, violence and antisocial behaviour if they received lower levels of parental monitoring (DiClemente et al., 2001). A study of Colombian youth, where violence and drugs are commonplace, found that in the face of these environmental pressures having a close parent-child bond was protective against the development of delinquent and marijuana use behaviours (Brook, Brook, De La Rosa, Whiteman, & Montoya, 1999); the converse finding is that negative parenting behaviours, including permissive or neglectful parenting, are associated with youth engagement in delinquent behaviours (Hoeve, Dubas, Gerris, van der Laan, & Smeenk, 2011), and research examining data in Canada found that higher levels of delinquent and aggressive behaviour were associated with lower levels of parental nurturance (Sprott et al., 2001). While there is an established relationship between parental attachment and delinquent behaviour, there is some evidence to suggest that this is a bidirectional relationship (Gault-Sherman, 2012). While parental attachment and behaviour predict delinquent behaviour, delinquent behaviour may in turn affect parental attachment and behaviour.

Parental alcohol consumption has been demonstrated to contribute to poorer adolescent outcomes. Parental alcoholism was associated with earlier onset of harmful drinking and alcohol dependence at ages 14-17, an effect that was equal for both female and male offspring (Lieb et al., 2002); a study of Puerto Rican and African American inner city youth replicated the finding that parental alcohol use predicted early alcohol use (Brook et al., 2010). Considering an earlier

age of substance use predicts later substance dependence problems in adulthood (Brook et al., 2004; DeWit, Adlaf, Offord, & Ogborne, 2000; DuRant, Smith, Kreiter, & Krowchuk, 1999; Mason et al., 2010), these results are disconcerting. Another study found that the presence of parental alcohol use and socioeconomic disadvantage together doubled the prevalence of alcohol and cannabis dependence (Melchior, Choquet, Le Strat, Hassler, & Gorwood, 2011). Furthermore, among late adolescent offspring, parental alcohol and drug dependence placed the child at increased risk for externalizing disorders, such as conduct disorder and antisocial behaviour (Marmorstein, Iacono, & McGue, 2009).

1.2.3.4 Gender Differences

Research has revealed some gender differences in the engagement in different types of health-risk behaviours. Males, for example, tend to engage in aggressive health-risk behaviour, such as injury to others (Chun & Mobley, 2010) or delinquency (Nichols, Graber, Brooks-Gunn, & Botvin, 2006), more frequently than females. Males were also found to be more likely to have used marijuana before the age of 13 compared to females (Chen & Kandel, 1995). There is also some recent evidence to suggest that while psychosocial distress influences female sexual risk-taking, it does not contribute to male sexual risk-taking (Brodbeck, Vilen, Bachmann, Znoj, & Alsaker, 2010). Additionally, depression may affect engagement in health-risk behaviour differentially for females and males; light-to-moderate engagement in health-risk behaviour, compared to high-risk activity, showed higher odds for depressive symptomatology for females (Waller et al., 2006). Compared to males, females had higher prevalence of

depression, suicidal ideation with substance use, and suicide attempts in one study (Cho et al., 2007). In that same study, onset of hard drug use was the strongest and most consistent predictor of all suicide risk factors; for females, early onset of smoking predicted suicidal ideation, and early onset of hard drug use predicted suicide attempt (Cho et al., 2007). A recent study of suicide rates amongst adolescents in Canada, examining age-standardized mortality rates between 1998-2008, outlined a trend that suicide rates amongst females are increasing, though they are decreasing for males (Skinner & McFaull, 2012).

However, research on gender differences is not always consistent. While some research purports much greater substance use, including alcohol, in males than females (Windle, 1990), others report little to no gender differences in substance use (Wallace et al., 2003). However, the authors hypothesized that their inconsistent findings may instead reflect a change in female substance use trends over time. However, incongruent findings warrant the need for further research on potential sex differences in the engagement in health-risk behaviour.

1.2.4 Co-occurrence of Health-risk Behaviours

The relationship between the various health-risk behaviours themselves is complex, as these behaviours often co-occur; furthermore, engagement in one type of behaviour may predict engagement in another type of behaviour. For example, research has found alcohol and marijuana use mediates the relationship between depressive symptomatology and having ever had an STI (Shrier, Harris, Sternberg, & Beardslee, 2001). Cannabis use was associated with greater

problems with delinquency, as well as the use of other substances such as alcohol and tobacco (Chen & Kandel, 1995). Illicit drug use at baseline significantly predicted risky sexual behaviour 5 years later in a study of African American and Puerto Rican adolescents (Brook et al., 2004). Another study demonstrated that substance abuse and conduct disorder were significantly associated with the onset of suicidal behaviours (Fergusson, Woodward, & Horwood, 2000); the link between substance use and suicidal ideation and suicide attempt has been replicated by other researchers (Cho et al., 2007; Legleye, Beck, Peretti-Watel, Chau, & Firdion, 2010). One study found that externalizing problems at baseline (ages 11-16) were predictive of all forms of substance use after 3 years of follow-up, and the strongest effect was found for engagement in tobacco use (Goodman, 2010). Despite the complex relationship between the behaviours, many studies only focus on the development of one or two behaviours, or investigate multiple health-risk behaviours cross-sectionally.

1.3 Maternal Depression

Depression is a recurrent psychiatric disorder of which women are more likely to experience depression than men (Kuehner, 2003; Weissman et al., 1996). This may become particularly problematic when the sufferer has a child, as exposure to maternal depression has serious, lasting effects on the offspring. Depression during the first postpartum year has an estimated prevalence ranging from 6% and 30% (Gavin et al., 2005; Goodman, 2007; Horwitz, Briggs-Gowan, Storfer-Isser, & Carter, 2009; Letourneau, Salmani, & Duffett-Leger, 2010), but is more commonly reported to be approximately 15-20% (Horwitz et al., 2009;

Marcus, Flynn, Blow, & Barry, 2003). Estimates of the prevalence of subclinical depressive symptomatology are even higher (Goodman, 2007). Furthermore, mothers who experience depressive symptomatology after birth are at increased risk for recurrent episodes (McMahon, Barnett, Kowalenko, & Tennant, 2005), with one study reporting that depressive symptoms persisted for 1 to 4 years for 56% of women (McMahon, Trapolini, & Barnett, 2008).

1.3.1 Theoretical Framework

1.3.1.1 Public Health Burden of Depression

Depression is frequently cited as a leading cause of disability and significant contributor to the global burden of disease. Economically, the burden of depression is large; with an estimated cost of tens of billions of dollars per year in the United States (Greenberg et al., 2003; Wang, Simon, & Kessler, 2003), occurring mainly through impaired work productivity (e.g., impaired work performance, missed work days) for the depressed individual. Beyond the societal economic burden of depression, the disorder is associated with significant functional impairment and disability for the sufferer. In fact, the World Health Organization estimates that by the year 2020, depression will be the second leading cause of disability experienced by sufferers of the illness, following heart disease (World Health Organization, 1996). However, despite the public health importance and high prevalence of depression, the etiology of the disease is still relatively misunderstood.

1.3.1.2 Etiology of Depression

The leading theory on depression etiology is the Diathesis-Stress model, in which certain individuals tend to have a higher predisposition toward depression, and these vulnerable individuals are more likely to experience depression after the experience of a stressful life event (Nolen-Hoeksema & Hilt, 2008). These vulnerabilities include genetic factors, as well as cognitive predispositions, such as a tendency toward negative automatic thoughts, negative appraisal of events, or having a negative self-view (Beck, 1987). Individuals who demonstrate such cognitive vulnerabilities are more likely to experience depressive symptomatology upon the occurrence of stressful life events (Hankin, 2008a, 2008b), and cognitive ability has a protective effect against depression and anxiety (Hatch et al., 2007; Koenen et al., 2009). The etiology of depression, however, is complex and multifactorial, with various risk and protective factors contributing to the onset of disease.

Maternal depression is a strong predictor of child psychopathology, especially depression, in the affected offspring. Many studies report a 2 to 3-fold increase in the risk of depression for offspring exposed to maternal depression (Hammen & Brennan, 2003; Kovacs et al., 1997; Naicker et al., 2012; Weissman, Wickramaratne, et al., 2006; Williamson et al., 2004). The effects of maternal depression may exert themselves through many pathways (e.g., genetic vulnerability, parenting style), likely working in synergy; however, it is clear that exposure to maternal depression during childhood places the offspring at increased risk for future psychopathology.

1.3.2 Transmission Pathways

It has been postulated that four main pathways encourage the transmission of risk of a mood disorder from a mother to her offspring: heritability of depression, dysfunctional neuro-regulatory mechanisms, negative maternal cognitions and behaviours, and the stressful context of the child's life (Goodman & Gotlib, 1999). Goodman and Gotlib (1999) assert, in their Integrative Model for the Transmission of Risk, that one, or more than one, of these four mechanisms in the risk transmission pathway may characterize the experience of any particular mother-child pair; additionally, while the pathways appear to be discrete categories theoretically, they likely interact with one another on the transmission pathway, and it is unlikely that these occur in isolation.

1.3.2.1 Genetic Mechanisms

Genetically, children may directly inherit their vulnerabilities (e.g., inhibited temperament, negative affectivity) for depression from the depressed mother. Indeed, this has been supported by evidence from familial and twin studies; the risk of a mood disorder is estimated at 25% if a first-degree relative suffers from unipolar depressive disorder (Goodman & Gotlib, 1999). Recent evidence has investigated potential genes that may account for susceptibilities to unipolar depression (Levinson, 2006); further evidence for gene-environment interactions in the development of depression has been documented (Mehta et al., 2012). However, heritability of depression may be affected by both the severity and timing of the mother's illness. Whereas clinical depression is associated with high heritability, subclinical depressive symptomatology is more influenced by

environmental factors (Goodman & Gotlib, 1999; Kendler et al., 1995). While depression is an undoubtedly heritable disorder, the exact role of genetic transmission differs with respect to severity and timing of the mother's illness, as well as the interaction between genetic vulnerabilities and stressful environmental contexts.

1.3.2.2 Neuro-regulatory Mechanisms

The second proposed mechanism is dysfunction in the neuro-regulatory abilities of the offspring. While this is difficult to distinguish from genetic transmission in practice, this mechanism is mainly characterized by an adverse prenatal environment resulting from the women's experience of stress and depression. Depression during pregnancy may result in a fetal environment characterized by abnormal neuroendocrine functioning (e.g., presence of stress hormones); furthermore, the fetal environment may be affected by a decrease in maternal health care behaviour (e.g., smoking, poor nutrition), decreased fetal blood flow, or the exposure to antidepressant therapy in utero (Goodman & Gotlib, 1999). Higher levels of urinary cortisol and norepinephrine, known human stress-response hormones, have been isolated from women who experienced depression during pregnancy (Diego et al., 2004; Field, 1998); furthermore, exposure to maternal depression both pre- and postnatally have been found to increase salivary cortisol levels in infants (Brennan et al., 2008). This association appears to follow a dose-response relationship, as the number of months that a women was depressed during pregnancy predicts cortisol levels at preschool-age (Ashman, Dawson, Panagiotides, Yamada, & Wilkinson, 2002).

Unsurprisingly, exposure to maternal depression in utero affects fetal development; higher levels of cortisol during pregnancy have been linked to slower fetal growth (Trainer, 2002). The prenatal environment may be altered by physiological or behavioural responses to maternal depression, thereby leading to lasting changes in offspring neuroendocrine or neuro-regulatory mechanisms.

1.3.2.3 Interactions with the Depressed Mother

The third mechanism proposed to promote the intergenerational transmission of depression is the child's exposure to maladaptive maternal cognitions, behaviour, and/or affect through interaction (Goodman & Gotlib, 1999). Depression is a disorder characterized by negatively biased self-perception, more internal, global, and stable negative interpretation of events, and increased attention for and interpretation of negative stimuli (Beck, 1987; Espejo, Hammen, & Brennan, 2012; Goodman & Gotlib, 1999; Joormann & Gotlib, 2007). Not only are depressed individuals more likely to engage in conversations with negative content (Gotlib & Robinson, 1982), they may actually elicit feelings of anxiety, depression, and hostility from those with whom they interact (Gotlib & Meltzer, 1987). Therefore, the child's interaction with their depressed mother may elicit negative and reciprocal reactions, and may also model maladaptive cognitions and behaviours for the child during early development.

Another aspect of these interactions that may negatively affect the child occurs through adverse parenting activity. Not surprisingly, depression translates poorly to parenting behaviours; compared to non-depressed women, depressed women are more likely to view their parenting abilities negatively, respond less

sensitively to infants' signals, are less likely to use a warm maternal tone towards their children, make fewer explanations, and ignore their children more (Cox, Puckering, Pound, & Mills, 1987; Foster, Garber, & Durlak, 2008; Lovejoy, Graczyk, O'Hare, & Neuman, 2000; McLearn, Minkovitz, Strobino, Marks, & Hou, 2006; Murray et al., 1999). A meta-analysis of 46 studies found that maternal depression was linked with more hostile and coercive parenting behaviours, less positive behaviour, and disengagement (Lovejoy et al., 2000). Conversely, warm and responsive maternal parenting behaviour has been associated with the child's developing language and cognitive ability (Landry, Smith, Miller-Loncar, & Swank, 1997; Murray & Hornbaker, 1997; Smith, Landry, & Swank, 2006). The parenting styles of depressed mothers may actually represent an early life stressor to the child (Goodman, 2007; Newport, Wilcox, & Stowe, 2002), the effects of which are discussed below. The depressed mother may therefore be unable to meet the developmental needs for the child, thereby limiting or hindering the socio-emotional and/or cognitive development of her offspring.

1.3.2.4. Stressful Environmental Contexts

Finally, the stressful environmental context of the child's life may exacerbate risk transmission of psychopathology from the depressed mother. Children of depressed mothers are not only exposed to the chronic stressor of her depressive symptomatology and the associated negative affect or parenting behaviour, but also to the stressors that may be associated with the development of the mother's depression including, but not limited to, marital discord, poverty,

and stressful life events (Goodman, 2007; Goodman & Gotlib, 1999; Silverstein, Augustyn, Young, & Zuckerman, 2009); in fact, there has been research that suggests these chronic stressors mediate the relationship between maternal depression and behavioural outcomes in children (Brennan, Hammen, Katz, & Le Brocque, 2002; Hammen, Brennan, & Shih, 2004). Research illustrates the notion that the relationship between stress and depression may have some reciprocity. Negative interpersonal styles and psychosocial impairments that are characteristic of depression contribute to stress generation, a process in which the individual contributes to the occurrence of stressors and therefore plays an active role in the generation of stressful events (Hammen, 1991); stress generation in individuals with a history of depression has been documented (Hammen, 2006; Hammen, Hazel, Brennan, & Najman, 2012; Liu & Alloy, 2010). Stressful environmental contexts may promote the development of psychopathology in the offspring of the depressed mother, and may also result in lasting changes in stress response, generation, and management (Hammen et al., 2012); this may be particularly true if the occurrence of depression leads to the generation of more stressful life events, to which the child would most likely also be exposed.

Early life stress, facilitated by the latter two proposed mechanisms of intergenerational depression transmission (i.e., interactions with the depressed mother and exposure to the stressful environmental context), can cause permanent changes in the body's responses to stress (Teicher et al., 2003), making these individuals more susceptible to lasting negative psychological responses to stress; furthermore, allostatic load, the cumulative damage of chronic stress on one's

physiological systems, is also related to many poor health outcomes, one being depression (Brunner & Marmot, 2006). Moreover, both acute and chronic stressors have an established association with depressive symptoms in children and adolescents (Grant et al., 2003; Hammen et al., 2012). Thus, acute and chronic stress both contribute to the development of depressive symptomatology and predict the later experience of acute and chronic stressors, and a past history of depression also predicts the experience of future stress (Hammen et al., 2012); this research provides evidence for the notion of stress generation.

Thus, the developing child may experience lasting changes in stress response resulting from exposure to the stressful context and correlates of maternal depression. In fact, postnatal exposure to maternal depressive symptoms disrupts the hypothalamic-pituitary-adrenal (HPA) axis (Ashman et al., 2002), the hormonal stress response system responsible for the release of cortisol. Adolescents aged 13 years who had been exposed to maternal depression postnatally had elevated basal cortisol levels compared to those not exposed (Halligan, Herbert, Goodyer, & Murray, 2004), suggesting a link between early life stress and hormonal stress response in adolescence. Further research is needed with respect to the role of brain development, especially in light of the growing availability of brain imaging technology. However, frontal lobe activation in 3 year olds was found to be lower in those exposed to chronic maternal depression, and additionally, activation mediated the relationship between maternal depression and the level of behavioural difficulties in the offspring (Dawson et al., 2003).

The intergenerational transmission of depression risk is undoubtedly complex, with the potential for interaction between risk and protective factors for depression (e.g., cognitive vulnerabilities, neuro-regulatory mechanisms), as well as the child's own experiences and characteristics (e.g., temperament, gender). Important modifiers of the relationship, such as timing of exposure or severity of maternal depression may also play an important role in the child's development of depressive symptomatology.

1.3.3 Timing of Exposure to Maternal Depression

The timing of exposure to maternal depression is an important factor to consider with respect to the development of child or adolescent psychopathology (Goodman & Gotlib, 1999). Along with timing, the effects of depression severity and chronicity have also been investigated. One study found that severity of maternal depression contributed more to depression risk than chronicity, yet that chronicity was associated with more non-depression outcomes in 15 year-old offspring (Hammen & Brennan, 2003). A multitude of research has focused on the first postpartum year as a critical period of exposure to maternal depression. While exposure to maternal depression during childhood has been examined, exposure to maternal depressing during later periods of childhood and into adolescence has been largely under-researched.

The offspring of mothers who were depressed during infancy (age 0-12 months) were more likely to be rated by their kindergarten teacher as having high internalizing and externalizing problems (Essex, Klein, Miech, & Smider, 2001).

Children of mothers who were depressed during the child's first 5 years of life displayed significantly higher levels of antisocial behaviour, and there may be a dose-response relationship between the number of developmental periods during the child's life that the mother was depressed, and the amounts of antisocial behaviour experienced (Kim-Cohen et al., 2005). Exposure to maternal depression during the first post-partum year was associated with child internalizing and externalizing during the early school years; the severity of depression at 4 months was significantly correlated with behavioural problems 7 years later (Fihrer, McMahon, & Taylor, 2009).

While the research outlined above demonstrates that exposure to maternal depression at an early age results in poorer outcomes for the children, the results do not necessarily outline a critical period that exists solely during the first postpartum year. Very few studies have rigorously studied maternal depression to determine if a sensitive period exists at some point during the childhood years. A recent study set out to determine whether a critical period for exposure to maternal depression exists; the results illustrated that a sensitive period for initial exposure to maternal depression exists between the ages of 2-5, with respect to the development of adolescent emotional disorder at age 12-13 (Naicker et al., 2012). Future research into timing of maternal depression is needed, especially with respect to the outcome of health-risk behaviour, rather than emotional disorder, in adolescence.

1.3.4 Effects of Maternal Depression on the Offspring

Despite differences in study methodologies or developmental ages researched, the adverse effects of maternal depression on the child are well documented, and have been reported across childhood through to adulthood.

1.3.4.1 Infancy and Childhood

The effects of maternal depression on the child are evident as early as the child's infancy. Nine-month old infants of depressed mothers show less mature fear regulatory behaviours, are more emotionally negative and have the lowest social engagement compared to controls (Feldman et al., 2009). School-aged children of depressed mothers, compared to children of non-depressed mothers, are more antisocial, have more behavioural problems, such as temper tantrums, interpersonal difficulties, and unhappiness, are more apathetic and withdrawn, and demonstrate features of a cognitive vulnerability for depression, such as negative self-concept, negative attributional style, and negative self-schema (Civic & Holt, 2000; Dawson et al., 2003; Essex, Klein, Cho, & Kraemer, 2003; Essex et al., 2001; Feldman et al., 2009; Fihrer et al., 2009; Gross, Conrad, Fogg, Willis, & Garvey, 1995; Jaenicke et al., 1987; Kim-Cohen et al., 2005).

1.3.4.2 Adolescence

Furthermore, these psychological problems persist through adolescence. The offspring of a depressed parent are at a significantly increased risk of emotional and behavioural problems in childhood and adolescence compared to the offspring of non-depressed parents (Beardslee, Versage, & Gladstone, 1998). After controlling for the effects of the adolescent's own psychopathology, one

study reported that children of depressed mothers attained less education, had less peer support, utilized more mental health services, and had lower life satisfaction (Lewinsohn, Olino, & Klein, 2005). Adolescent children of depressed mothers also report more internalizing and externalizing problems and have elevated depressive symptomatology (Campbell, Morgan-Lopez, Cox, & McLoyd, 2009; Fergusson, Horwood, & Lynskey, 1995; Halligan, Murray, Martins, & Cooper, 2007; Hammen & Brennan, 2003). Exposure to maternal depression at some point by the age of 12 significantly predicted the growth of depressive symptomatology across adolescence (Garber & Cole, 2010); along a similar vein, results of another study indicate that exposure to maternal depression at any point during the child's first 10 years of life was associated with elevated depressive symptomatology in adolescence (Hammen & Brennan, 2003). Furthermore, an estimated 40-70% of those who experience a depressive episode in adolescence will experience a later depressive episode in adulthood (Colman et al., 2007; Dunn & Goodyer, 2006; Kovacs et al., 1994); therefore, the psychological outcomes of exposure to maternal depression are likely to persist throughout adulthood for the majority of children affected. Further longitudinal investigation into various adolescent outcomes associated with maternal depression is needed.

1.3.5 Child Factors Influencing the Outcome of Maternal Depression

While factors associated with the mother's illness, such as timing and severity of her depressive symptoms, may influence the outcome on the child, research has also demonstrated that child-specific factors may also mediate the effect of maternal depression on the child's psychopathology.

1.3.5.1 Gender Differences

The effect of maternal depression on the child may be stronger for female offspring of depressed mothers, who not only are at increased risk for depression (Ensminger, Hanson, Riley, & Juon, 2003), but also are more likely to respond to maternal upset with crying, worrying, or withdrawing, and have increased feelings of responsibility for their mothers' depression (Klimes-Dougan & Bolger, 1998) compared to male offspring of depressed mothers. However, this may just reflect a difference in the effect of exposure to maternal depression on the child; whereas female offspring of depressed mothers may be more susceptible to experiencing depression, male offspring may be at increased risk for conduct problems (Cummings & Davies, 1994). However, research investigating gender differences in response to exposure to maternal depression has found mixed results, with some studies reporting worse outcomes for males, and the converse (Goodman & Gotlib, 1999); recent longitudinal research reported no significant gender differences for the relationship between maternal distress during childhood and adolescence and adult mental health outcomes (Flouri & Malmberg, 2011).

1.3.5.2 Child Psychopathology

The child's own experience of psychopathology may interact with exposure to maternal depression. Depressed adolescents with depressed mothers display significantly more social impairments, negative cognitive styles and interpersonal behaviours than depressed adolescents with non-depressed mothers (Hammen & Brennan, 2001; Hammen, Brennan, & Keenan-Miller, 2008;

Hammen, Shih, & Brennan, 2004). The additional social and functional impairments that are observed in depressed children of depressed mothers may contribute to an earlier onset of depression, higher rates of recurrence and worse clinical features than the depressed adolescents of non-depressed mothers (Hammen & Brennan, 2001; Hammen et al., 2008; Hammen, Shih, Altman, & Brennan, 2003; Rohde, Lewinsohn, Klein, & Seeley, 2005; Weissman, Warner, Wickramaratne, Moreau, & Olfson, 1997; Weissman, Wickramaratne, et al., 2006). Furthermore, offspring of mothers who became depressed during the child's early school-aged years display more passive affective regulation strategies (Silk, Shaw, Skuban, Oland, & Kovacs, 2006), which is a risk factor for both the onset of depression and the engagement in health-risk behaviour. Affect regulation has been found to moderate the relationship between maternal depression and child internalizing problems (Silk, Shaw, Forbes, Lane, & Kovacs, 2006).

1.3.6 Maternal Depression and Health-Risk Behaviours

Various individual factors, including novelty seeking, locus of control, self-esteem, and depression have been linked to engagement in potentially risky behaviours (Igra & Irwin, 1996), however early life and family factors may play a contributing role in propensity toward these behaviours. While there is evidence to suggest that maternal depression is linked to adolescent depression and to a lesser extent, externalizing behaviour, and these have been linked to the engagement in health-risk behaviour, the relationship between maternal depression and adolescent health-risk behaviour has not been thoroughly

researched. There have, however, been a few notable findings. One study demonstrated maternal depression to be associated with alcohol, tobacco, and marijuana use during seventh grade, and this relationship was partially explained by the child's own antisocial behaviour and depressive symptomatology (Cortes, Fleming, Mason, & Catalano, 2009). Additionally, another recent study demonstrated that at age 15, children of mothers who experienced recurrent depression, elevated subclinical symptoms of depression, or stable subclinical depression reported more engagement in health-risk behaviour compared to their peers (Campbell et al., 2009). However, as a still emerging research field, there is little existing evidence examining the relationship between exposure to maternal depression and the engagement in health-risk behaviour.

1.4 Rationale

While research has focused on factors associated with health-risk behaviour, much of this research has focused on characteristics of the individual who is engaging in the behaviours at the time of engagement. There may be early life factors, including social or developmental characteristics during childhood such as exposure to parental mental illness, which may increase an individual's propensity toward such behaviours at a later point in time. The relationship between maternal depression and adolescent engagement in health-risk behaviours is an emerging research topic requiring further examination. A visual representation of this study's rationale is provided below, in Figure 1.

Offspring of depressed mothers are more likely to experience depression, and adolescent depression appears to have a bi-directional relationship with health risk behaviours. Not only are depressed adolescents more likely to engage in health-risk behaviour, engagement in such behaviours may increase the risk for future depression, thus perpetuating the cycle. Maternal depression places the child at an increased risk for internalizing problems, which have their own relationship with health-risk behaviours.

Furthermore, additional outcomes associated with exposure to maternal depression have a relationship with engagement in health-risk behaviours. Maternal depression is linked to poor offspring outcomes, such as increased stress response and maladaptive affect regulation strategies, and some evidence suggests maternal depression is linked to externalizing behaviours. These potential psychological outcomes of maternal depression have been linked to the engagement in health-risk behaviours (Auerbach et al., 2010; Baldwin et al., 2011). Furthermore, parenting style and parental attachment may also foster or protect from the engagement in health-risk behaviour. Depressed mothers are more likely to employ a less engaged parenting style (Cox et al., 1987; Lovejoy et al., 2000; Murray et al., 1999), and the offspring of depressed mothers are less likely to form secure attachment bonds with their mothers (Martins & Gaffan, 2000). This is troubling in light of the fact that a close parental bond may dissuade youth from engaging in substances (Brook et al., 1999). The development of psychopathology in offspring of depressed mothers is complex, with many factors potentially contributing to the relationship. The theoretical link between maternal

depression and the engagement in health-risk behaviour is intuitive. Adolescents who were exposed to maternal depression are at increased odds for depression, which is itself a risk factor for health-risk behaviour engagement. Furthermore, correlates of exposure to maternal depression, such as the experience of stress, maladaptive affect regulation, and withdrawn parenting behaviour are also risk factors for engagement in health-risk behaviour. While the relationship between maternal depression and health-risk behaviour is intuitive, more research is needed to expand the already existent findings in the field and further contribute to the understanding of the potential negative impacts of maternal depression on the child.

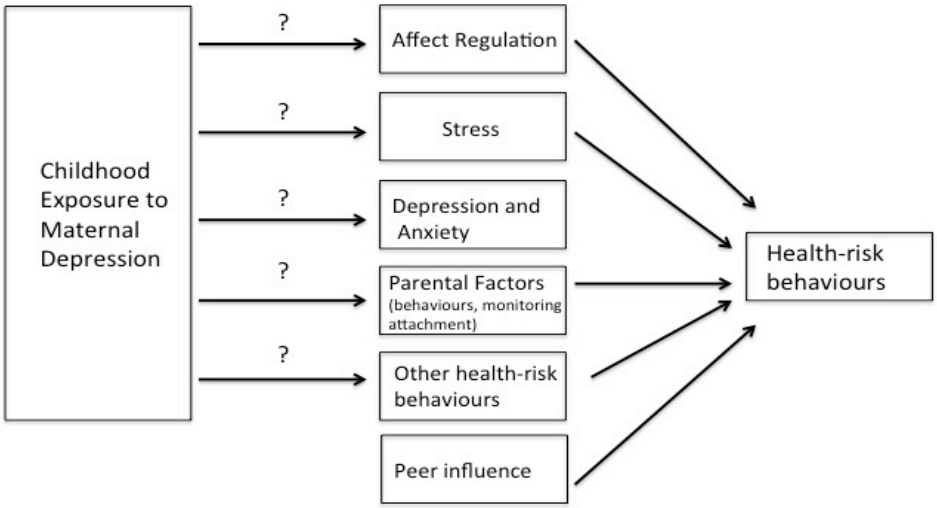

Figure 1 – Conceptual framework of the relationship between maternal depression and health-risk behaviour engagement

1.5 Objective and Hypothesis

Due to the complex interrelation of exposure to maternal depression, adolescent depression, and engagement in multiple health-risk behaviours, this research has multiple objectives.

(1) The primary objective of this study is to investigate whether adolescents with varying experiences with maternal depressive symptomatology throughout childhood, as assessed through the use of trajectory modeling, differ with respect to engagement in groups of similar health-risk behaviour.
(2) This research also aims to identify whether those with varying childhood exposure to maternal depressive symptomatology over time have different odds of engaging in a variety of specific health-risk behaviours.
(3) Finally, this research aims to determine whether adolescent offspring who had differing experiences with maternal depressive symptomatology in childhood have different time-to-onset of health-risk behaviour.

As there is research to suggest that depressed adolescents with depressed mothers fare worse than depressed adolescents without depressed mothers (Hammen & Brennan, 2001), this study will investigate whether adolescent depressive symptomatology modifies the relationship between maternal depression and adolescent health-risk behaviour. Gender will also be treated as a potential effect-modifier, due to potential gender differences in health-risk

behaviour and maternal depression outcomes outlined above. Figure 2 below is a flow chart representing the objective of the hypothesized research, including the adjustment covariates.

It is hypothesized that those with depressed mothers will engage in more health-risk behaviours, earlier, than those without depressed mothers. It is posited that males will participate in more physically aggressive health-risk behaviours (e.g., violence and gang activity) than females; however, research has revealed no gender differences in adolescent smoking (Barnes, Welte, Hoffman, & Tidwell, 2009), and there is contradictory evidence on gender differences in the use of other substances. Therefore, it is hypothesized that there will be no significant gender differences in substance use.

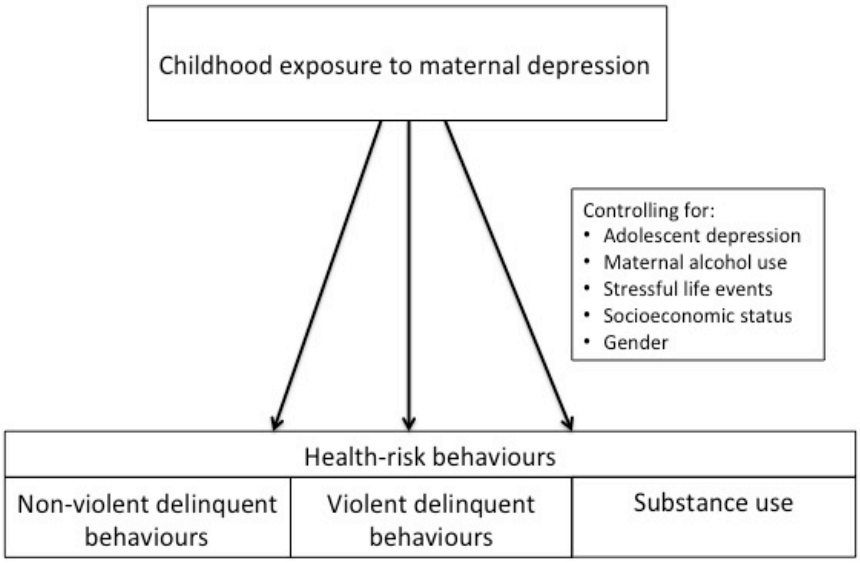

Figure 2 –Flow diagram of the objective of this dissertation

Chapter 2 – Methods

2.1 The National Longitudinal Survey of Children and Youth

This study used data from the National Longitudinal Survey of Children and Youth (NLSCY), a survey designed to measure child wellbeing and development longitudinally in Canada. The NLSCY is a nationally representative population-based cohort of 16,903 Canadian children studied every 2 years beginning in 1994/1995 (Cycle 1) and ending in 2008/2009 (Cycle 8). The first cycle of the NLSCY yielded a responding sample of 13,439 households and 22,831 children (Statistics Canada, 1995).

The first step in data collection was to establish a household roster for each household selected; this comprised basic demographics, information on dwelling conditions, and a relationship grid detailing the relationships of each household member to everyone else in the household (Statistics Canada, 1995). Then, a computer system randomly selected a child (aged 0-11 years) from the household, and the Person Most Knowledgeable (PMK) to the child, primarily the mother, was identified. Up to four children could be selected per household (Statistics Canada, 1997a). The PMK was then asked to complete different questionnaires (the Parent Questionnaire, the General Questionnaire, and the Child's Questionnaire). The Parent Questionnaire was used to collect general health information for the PMK and her spouse/partner, as well as information on the child's social environment, such as PMK mental health, social support, and family functioning. The General Questionnaire collected socio-economic

information, including education, labour force and income. The Children's Questionnaire was completed by the PMK for randomly selected children aged 0-11 years, and included topics such as behaviour, literacy, education, and health. Once the child reached age 10-11, they were asked to complete a self-complete Children's Questionnaire. The PMK first gave permission on behalf of the child, then the child was given the questionnaire and encouraged to complete it in a private setting. Following completion of the questionnaire, it was sealed in an envelope in order to protect confidentiality. The PMK was not permitted to see the child's responses, in hopes of encouraging honest and accurate answers from the children (Statistics Canada, 1995).

2.1.1 Sample

This study used children who were aged 2-5 during 1994/95 (unweighted N=2,910), and followed them longitudinally until they reached the age of 16-17, using data from each available cycle of the NLSCY. This cohort was selected for a few reasons. First, the ages were chosen based on the availability of the data cycles. As engagement in health-risk behaviours tends to increase with age (Brener & Collins, 1998), it is crucial to capture the later adolescent ages of 16-17, when engagement in health-risk behaviours is more likely. Considering that some health-risk behaviours are rare, or underreported, it is crucial to capture an age at which engagement is more likely to be reported.

Additionally, early childhood has been postulated as a critical exposure period to maternal depression (Bagner, Pettit, Lewinsohn, & Seeley, 2010; Fihrer

et al., 2009), and therefore it is crucial to include individuals who were exposed to maternal depression at a young age. Recent evidence suggests a critical period for adolescent depression exists at the age of 4-5 (Naicker et al., 2012) and research on exposure to maternal depression during the later childhood years is limited. Much of the research on timing of maternal depression has focused on the first few post-partum years, largely ignoring the potential impact of exposure to maternal depression at ages later in childhood, which was not the scope of the present study. Therefore, maternal depressive symptomatology was examined between the ages of 4-15 for the children in this specific study. This research was approved by the Health Research Ethics Board at the University of Alberta (Appendix A).

2.2 Materials

2.2.1 Maternal Depression

A shortened form of the Center for Epidemiologic Studies Depression Scale (CES-D), originally 20 questions developed to assess the frequency of depressive symptomatology in the general population (Radloff, 1977), was used to assess maternal depression. In Cycle 1 of the NLSCY, 91.3% of the children's main respondent, the Person Most Knowledgeable (PMK), was the mother (Statistics Canada, 1997a). Therefore, this study was not able to assess parental depression overall, but rather focused on maternal depression. The version of the CES-D used in the NLSCY was shortened to 12 questions by Dr. M. Boyle at Chedoke-McMaster Hospital (Statistics Canada, 1995).

This depression scale includes 12 questions pertaining to how often certain feelings were experienced during the past week. The items were: I felt that I could not shake off the blues even with help from my family or friends; I had trouble keeping my mind on what I was doing; I felt depressed; I felt that everything I did was an effort; I felt hopeful about the future; my sleep was restless; I was happy; I felt lonely; I enjoyed life; I had crying spells; I felt that people disliked me. Responses were assessed on a 4-point scale, ranging from 'rarely or none of the time (less than 1 day)' to 'most or all of the time (5-7 days),' and a final score ranging from 0 to 36 was calculated for each PMK, with higher scores indicating greater depressive symptomatology. The Cronbach alpha coefficient for this scale was 0.82 (Statistics Canada, 1997a), indicating good reliability of the shortened CES-D scale. Chronbach's alpha for the cycle-specific CES-D scores ranged from 0.91 to 0.97, indicating excellent reliability for the scale. Information on the mothers' mental health was gathered every cycle of the NLSCY, every 2 years.

To derive an overall score for maternal depressive symptomatology, the values assigned to each response, ranging from 1 (less than 1 day) to 4 (most or all of the time) were subtracted by one, allowing for a score of zero, then summed, yielding an overall maternal depression score for each cycle of the NLSCY. This variable was continuous, and trajectories of maternal depression throughout childhood were established between child's age 4 and 15 using finite mixture modeling, detailed in the statistical methods section below.

2.2.2 Adolescent Health-Risk Behaviour

Once the child reached 10 years of age, specifically in Cycle 5 (2002/2003), they were asked questions pertaining to risky behaviours. As the children aged, the questions were expanded in order to capture behaviours that become more common as the youth age (Statistics Canada, 2003). The questions included in the 'Risky Behaviour' questionnaire were adapted by the NLSCY Project Team from the National Longitudinal Survey of Youth at Ohio State University, Western Australia Child Health Survey, and from questions provided by Dr. Richard Tremblay from the University of Montreal (Statistics Canada, 2003). There were four possible responses, ranging from 'never' to '5 times or more.' The questions asked are presented in the table below. For this study, engagement in health-risk behaviours was reported during the cycle in which the children reach 16-17, the chosen outcome year for this research. Engagement in these behaviours was rare, and as such the data were skewed. Therefore the two highest response categories (3-4 times, 5 times or more) were combined, yielding 3-reponse categories ('never', 'once or twice', 'three times or more') in order to maintain appropriate category sizes.

Question: "In the past 12 months, about how many times…"
Were you questioned by the police about anything they thought you did?
Have you run away from home?
Have you stayed out all night without permission?
Have you intentionally damaged or destroyed anything that didn't belong to you?
Have you fought with someone to the point where they needed to care for their injuries?
Have you carried a weapon for the purpose of defending yourself or using it in a fight?

Have you sold any drugs?
Have you attempted to touch anyone in a sexual way while knowing that they would probably object to this?
In the past 12 months, were you part of a gang that broke the law by stealing, hurting someone, damaging property, etc? **(Y/N)**
Have you stolen something from a store or school?
Have you attacked someone with the idea of seriously hurting him/her?

The youth were also asked questions to determine whether they engaged in the use of substances, and the extent of such usage. The smoking questions are adapted from the Youth Smoking Survey, the WHO Survey on Health Behaviours in School Children, and the Western Australia Child Health Survey. The questions on alcohol were drawn from the Western Australia Child Health Survey and from questions provided by Dr. Richard Tremblay from the University of Montreal (Statistics Canada, 2003). The questions pertaining to the usage of other drugs and addictive substances were drawn from the Northwest Territories Health Attitudes, Knowledge and Behaviours Study (Statistics Canada, 2003). Finally, questions on driving under the influence or riding as a passenger in a vehicle with an intoxicated driver are included for 16-17 year-olds, and were adapted from the North Carolina Evaluation of School-Based Health Centers (Statistics Canada, 2003). The Smoking, Drinking, and Drugs questions are detailed, with possible responses, in the table below.

Domain	Question	Responses
Smoking		
	Which of the following best describes your experience with smoking cigarettes?	7 responses ranging from 'I have never smoked' to 'about 6-7 days/week'
	If you have smoked one or more cigarettes every day for at least 7 days in a row, how	'I have never done this' or 'I was __ years old'

	old were you when you first did so?	
Drinking		
	How old were you when you first had a drink of alcohol?	'I was __ years old'
	Have you ever been drunk?	Yes/No
	How old were you when you were drunk for the first time?	'I was __ years old'
	Which of the following best describes your experience with drinking alcohol?	9 responses, ranging from 'I have never had a drink of alcohol' to 'about 6-7 days a week'
	In the past 12 months, how often have you been drunk?	6 responses, ranging from 'never' to 'about 6-7 days/week'
Illicit Drugs		
	Which of the following best describes your experience with using marijuana and cannabis products in the past 12 months?	7 responses, ranging from 'I have never done it' to 'about 6-7 days/week'
	Which of the following best describes your experience with the following drugs in the past 12 months? (a. Hallucinogens; b. glue or solvents; c. drugs without a prescription; d. other drugs (e.g., ecstasy, crack, heroin)	6 responses, ranging from 'I have never done it' to '10 times or more'
	How old were you when you did the following drugs for the first time? (a. marijuana, b. hallucinogens, c. glue or solvents, d. drugs without a prescription, e. other drugs)	'I have never done it' or 'I was __ years old'
	In the past 12 months, how many times have you operated a motorized vehicle (e.g., car, motorcycle, boat) after you have been drinking alcohol or doing drugs?	4 responses, ranging from 'never' to '5 times or more'
	In the past 12 months, how many times have you been a passenger in a vehicle when the driver has been drinking or taking drugs?	4 responses, ranging from 'never' to '5 times or more'

The cigarette and alcohol questions contained the most response categories. The responses for the smoking and drinking items were accumulated into 4 categories: never used; infrequent use ('I have not used in the past year,' 'I have had a few', 'not anymore'); near-monthly to monthly use ('a few times per year', '1-2 times per month'), and weekly use (ranging from '1-2 times per week', to '6-7 times per week'). The illicit drug-use questions contained less response categories, and engagement in these behaviours was much more rare in frequency. These items were accumulated into the same 3 categories as the previous scale: never, '1-2 times', or '3 times or more.'

Once the youth reach the age of 12, they were asked additional questions about suicide, particularly whether the youth knows anyone who has committed suicide and whether they have seriously considered or attempted suicide. These questions are adapted from the 1992 British Columbia Adolescent Health survey (Statistics Canada, 2005). The question pertaining to suicidal ideation in the past year was dichotomous, and the question pertaining to suicide attempt in the past year contained 3 response options ('never', 'once', 'more than once'). However, again due to the skewness of the data, the attempted suicide question was recoded to be dichotomous.

Also beginning at the age of 12, the youth are asked questions about dating experiences and frequency of sexual activity, as well as age at first engagement. The question about sexual behaviour on the 12- and 13-year-old questionnaire was adapted from the Youth and AIDS Survey (Statistics Canada, 2005). The NLSCY Project Team designed these questions with consultation from

experts involved in other youth surveys, such as the 1992 British Columbia Adolescent Health Survey and the Minnesota Adolescent Health Survey (Statistics Canada, 2005).

2.3 Additional Covariates

2.3.1 Adolescent Depression

At ages 16-17, youth are asked about feelings of depression, using the same questions asked of the PMK (CES-D), detailed above (Statistics Canada, 1995) . Responses were assessed on a 4-point scale, and summed, yielding a final score ranging from 0 to 36, with higher scores indicating greater depressive symptomatology. The reported Cronbach alpha coefficient for the self-reported youth depression shortened CES-D scale was 0.825, indicating good reliability (Statistics Canada, 2007). The Chronbach's alpha value for the CES-D scores in this specific sample was 0.84 for the older cohort, and 0.99 for the younger cohort, indicating good reliability of the scale. This variable was continuous.

2.3.2 Socioeconomic Status (SES)

Research has identified a link between SES and depression; those with a lower SES tend to experience higher levels of depressive symptomatology (Lorant et al., 2003), and SES has been linked to the engagement in multiple substances (Redonnet, Chollet, Fombonne, Bowes, & Melchior, 2012). Therefore, the regression analyses adjusted for the effect of SES. Five variables, standardized to have a mean of 0 and a standard deviation of 1, were used to derive a SES score for the NLSCY: the level of education of the PMK, the level of education of the

spouse/partner, the prestige of the PMK's occupation, the prestige of the spouse/partner's occupation, and household income (Statistics Canada, 1995). The unweighted average of the five variables was used to derive the SES composite score, which ranged from -2 to 1.75 (Statistics Canada, 1995). This variable was measured at baseline, in 1994/95 (cycle 1).

2.3.3 Stressful Life Events

The association between stressful life events and depression is also well-established (Hammen, 2005), and thus it was controlled for in the analysis. Furthermore, there is an established link between stress and tobacco and substance use (Byrne & Mazanov, 1999), as well as the engagement in other health-risk behaviours (Baldwin et al., 2011). The PMK for children aged 4-15 are asked whether there have been any events in the past 2 years that have caused the child a great amount of worry or unhappiness (Statistics Canada, 1995). The stressful life events included in the NLSCY fall under the following categories: death of parents; death in the family; divorce or separation of parents; moving; death of a pet; hospital stay; stay in a foster home; other separation from parents; illness/injury of child; illness/injury of family member; abuse or fear of abuse; change in household members; alcoholism or mental health disorder in family; or other traumatic events (Statistics Canada, 1995). This variable was a continuous count measure, and was assessed at the latest possible adolescent age, which was collected when the youth was 14-15.

2.3.4 Cohort Indicator Variable

Additionally, due to the wide spread of ages included in the study sample, a cohort indicator variable was included in all analyses. The NLSCY establishes cohorts of 2-year intervals; therefore, included in the age range of 2-5 in 1994-1995, are the sub-cohorts of 2-3, and 4-5 year olds. Thus, in order to control for any potential cohort or age-specific effects, a categorical variable indicating the cohort (0 for 2-3 year olds, 1 for 4-5 year olds) was included in all regression analyses.

2.3.5 Gender

Research suggests that maternal depression may differentially affect male and female offspring; research suggests the impact of maternal depression may be stronger for females (Ensminger et al., 2003; Klimes-Dougan & Bolger, 1998). Furthermore, patterns of engagement in health-risk behaviour appear to be different between female and male adolescents (Brodbeck et al., 2010; Chun & Mobley, 2010; Nichols et al., 2006). Therefore, gender was adjusted for in all analyses, and interactions between gender and maternal depression were explored.

2.3.6 Maternal Alcohol Use

Finally, maternal alcohol use was considered as a potential covariate, as there is evidence that the psychological outcomes observed in children is comparable between mothers who abuse substances and mothers who experience depression and anxiety (Luthar & Sexton, 2007). Parental alcohol use has been associated with earlier onset of drinking behaviour, and problem drinking in

adolescence (Lieb et al., 2002). The mothers were asked questions pertaining to alcohol use, one of which asks the frequency in the past year in which 5 drinks or more are consumed on one occasion (Statistics Canada, 1995). Maternal alcohol abuse was operationalized based on the frequency of binge drinking in the past year, and potential interactions between maternal depression and alcohol use were considered. The most recent available information about maternal alcohol use was reported at age 14-15.

2.4 Statistical Methods

The main exposure measure in this research was maternal depression from the child's ages of 4-15. Due to the longitudinal nature of the NLSCY data, latent class analysis was used to establish trajectories of maternal CES-D scores over time, and the identified trajectories were used as a categorical variable in subsequent regression analyses. The main outcome measure, engagement in different domains of health-risk behaviour, was operationalized in a few different ways. First, to account for the large number of items being studied, and to attempt to determine the underlying structure of engagement in these behaviours, exploratory factor analysis was conducted. Factor scores produced for the identified factors were used in multiple linear regression analysis. To determine different odds of engagement across the maternal depression subgroups, secondary logistic regression analyses were conducted. Finally, using the information collected about age of first engagement, survival analyses were carried out to determine if different subgroups of exposure to maternal depression had different rates of health-risk behaviour engagement.

2.4.1 Latent Class Analysis

Latent class analysis was used to establish trajectories of maternal depression throughout childhood. Trajectories of maternal depression from age 4-15 were modeled using the SAS Trajectory Procedure (Jones, Nagin, & Roeder, 2001). PROC TRAJ uses a finite mixture model procedure to estimate multiple groups within the sample; it is designed to describe the trajectory over time in the dependent variable and model unobserved heterogeneity in the population (Arrandale, 2006; Jones et al., 2001). The longitudinal sequence of behaviour is modeled over the time periods measured; however, it is believed that unobserved heterogeneous subpopulations exist that differ with respect to the parameter under observation (Jones et al., 2001). The emphasis of PROC TRAJ is in identifying these distinct subgroups within the sample. PROC TRAJ is capable of modeling up to fourth-order polynomial relationships between age and the observed behaviour (Jones et al., 2001). PROC TRAJ does not have an option for bootstrapping; therefore, normalized population weights were incorporated into the trajectory model. The PROC TRAJ software, applied examples and supporting documentation is available online at http://www.andrew.cmu.edu/user/bjones/index.htm

Model fit is determined using the Bayesian Information Criterion (BIC) values, and improvement in model fit was determined by comparing the BIC values between models (Jones et al., 2001). The change in BIC values between models is an approximation of the log of the Bayes factor (Jones et al., 2001). The

model fit formula for the BIC log Bayes factor approximation, as outlined by Jones, Nagin, & Roeder (2001), is:

$$2\log_e(B_{10}) \approx 2(\Delta BIC)$$

where B_{10} is the Bayes factor and ΔBIC is the difference between the BIC of the more complex model less the BIC of the simpler model (i.e., the model with fewer groups). The strength of the evidence favouring the more complex model is established with the above equation; values of 10 or greater for $2\log_e(B_{10})$, or equivalently $2(\Delta BIC)$, are considered very strong evidence, values between 6 and 10 are deemed to be strong evidence, values between 2 and 6 are considered positive evidence, and values between 0 and 2 are deemed weak evidence for the alternative model (i.e., the model with more groups) (Jones et al., 2001).

Maternal depression (CES-D) scores at child's age 4-5 through age 14-15 were the dependent variable in the mixture model, and corresponding child's age was the independent variable. Starting with a one-group model and an initial fourth-order polynomial, models are fitted with increasing number of subgroups. Following the establishment of the number of groups, the polynomial order of the equations (e.g., linear, quadratic, cubic or quartic) is determined until best model fit between the nested models is established; non-significant orders are lowered, and model fit is again determined by comparing the estimate of the log Bayes factor (Andruff, Carraro, Thompson, & Gaudreau, 2009; Jones et al., 2001). Non-significant quartic, cubic and quadratic orders are lowered; however, non-

significant linear equations should be retained regardless of statistical significance (Andruff et al., 2009).

2.4.2 Exploratory Factor Analysis

Factor analysis, specifically principal components analysis (PCA), was conducted on the multiple health-risk behaviour questionnaires to both reduce the data and to explore the empirical structure of such behaviours. As there was no a priori hypothesis regarding the number of underlying factors, exploratory factor analysis methodology was used. Factors with eigenvalues greater than 1 were retained. As health-risk behaviours tend to co-occur, and therefore cannot be assumed to be uncorrelated, the factor loadings were not be treated as orthogonal, and oblique rotation methods were used. Factor scores were calculated for each determined factor, and these scores will be used in the subsequent multiple linear regression analyses. Factor analysis was conducted using the frequency-based (i.e., 'during the past 12 months how many times have you…') health-risk behaviour questions, including measures of substance use, and delinquency outlined above.

2.4.3 Multiple Linear Regression

To determine whether maternal depression during childhood is related to the engagement in patterns of health-risk behaviour at age 16-17, multiple linear regressions were used; these regressions included maternal depression trajectory group as the main predictor variable, and health-risk behaviour was

operationalized using the factor scores derived from the factor analysis. Factor scores were assessed during the cycle when the child reached age 16-17.

2.4.4 Logistic Regression Analyses

To assess whether the youth in different subgroups of exposure to maternal depression had different odds of engaging in specific types of health-risk behaviours, odds-ratios were calculated from multiple logistic regression analyses. These secondary analyses were separated by domain of health-risk behaviour (e.g., sexual intercourse, drug use, violence). These analyses were conducted using logistic regression for the dichotomous variables (sexual intercourse, suicidal ideation, gang membership), and ordinal logistic regression for the ordinal variables (drug use). Ordinal variables that violated the proportional odds assumption were recoded to be dichotomous (0 indicating no engagement, and 1 indicating some engagement in that behaviour). These behaviours were assessed during the cycle in which the youth reached age 16-17.

2.4.5 Survival Analysis

Cox regression was conducted to compare the rates of engagement in behaviour across the subgroups. These analyses utilized the NLSCY questions pertaining to age of first engagement in the different domains of health-risk behaviours (e.g. “how old were you when you first had a drink of alcohol?” or “how old were you when you tried the following drugs for the first time?”). The age of engagement questions were asked for alcohol and drug use (cigarettes, marijuana, hallucinogens, prescription drugs, and other drugs) as well as sexual

intercourse. Hazard ratios were calculated to compare the time-to-engagement, or age of onset of behaviours, between the distinct maternal depression subgroups. In STATA, the origin was defined as the child's date of birth, and the entry date to the study was defined as November, 1994, when data collection began. The study end date was defined as September, 2009, when the data collection for Cycle 8 had been completed.

2.4.6 Progressive adjustment

Missing data was an obstacle to this research. In order to best preserve sample size in light of missing covariate data, progressive adjustment for covariates was performed. The first adjustment was done for gender, SES, and cohort effects. The second adjustment added current adolescent depression (at age 16-17), and stressful life events in the preceding data cycle (age 14-15). The final adjusted model included the measure of maternal alcohol use in the model. Odds- or hazard-ratios and p-values will be reported for bivariate, and each progressively adjusted model.

2.4.7 Bootstrapping

The sampling variance of the NLSCY is difficult to calculate due to the complex sample design, non-response adjustments, and post-stratification (Statistics Canada, 2005). In order to account for the impact of the complex sampling design on the variance, bootstrap weights should be applied to all analyses (Statistics Canada, 2009). These weights are provided by Statistics Canada. Use of the bootstrapping method improves the accuracy of the variance

estimates by accounting for the components of the complex survey design, and increases the ability for the population-based inference (Statistics Canada, 2009).

Chapter 3 – Results

3.1 Descriptive Statistics

The final sample contained 2,910 children and their mothers, followed from ages 2-5 until age 16-17. All of the mothers in the sample were the biological mother, due to limited statistical power to study adoptive mothers. The children were 50.3% female, and 51.9% of the sample belonged to the older cohort (i.e., aged 4-5 during the first data collection cycle). Baseline SES, derived by Statistics Canada and standardized to have a mean of 0 and a standard deviation of 1, was based on the level of education of the PMK, the level of education of the spouse/partner, the prestige of the PMK's occupation, the prestige of the spouse/partner's occupation, and household income, ranged from -2.56 to 2.82, with a mean of -0.08 and a standard error of 0.73 in this study's sample. The mean count of stressful life events at age 14-15 was 0.36, and the range of reported stressful life events was 0 to 5. The mean (standard error) number of days that mothers reported binge drinking was 1.58 (5.57); responses ranged from 0 days to 104 days of reportedly drinking more than 5 drinks on one occasion. Prevalence of maternal depression, as indicated by a pre-established cut-off score of 9 or greater on the CES-D by researchers using this scale (Somers & Willms, 2002) and prevalence of adolescent depression as determined by a cut-off score of 12 on the 12-item CES-D established by researchers using this scale in adolescents (Poulin, Hand, & Boudreau, 2005), and the mean CES-D scores are reported in Table 1. These cut-off scores were merely used to report the estimated prevalence of depression in this sample;

continuous CES-D scores were used in the latent class analysis for the mothers, and the regression analyses for the youth. Maternal depression prevalence fell within the expected range of 10-20%, and adolescents reported higher depressive scores and had a higher prevalence of depression than mothers, as assessed by the cut-off score on the CES-D. Prevalence of the health-risk behaviour engagement is detailed in Table 2.

Table 1 – Prevalence and mean depression scores for mothers and adolescents

Outcome	Prevalence	Mean CES-D Score (SD)
Maternal depression cycle 1 (n = 2,616)	17.98%	4.81 (4.93)
Maternal depression cycle 2 (n = 2,540)	14.99%	4.34 (4.93)
Maternal depression cycle 3 (n = 2,589)	15.80%	4.38 (5.39)
Maternal depression cycle 4 (n = 2,575)	16.03%	3.92 (5.24)
Maternal depression cycle 5 (n = 2,665)	14.09%	3.68 (4.86)
Maternal depression cycle 6 (n = 2,602)	13.67%	3.55 (4.81)
Maternal depression cycle 7 (n = 1,902)	10.63%	3.29 (4.56)
Adolescent depression at age 16-17(n = 2,210)	20.64%	7.92 (5.64)

Table 2 – Prevalence of health-risk behaviours at age 16-17

Behaviour	Prevalence
Staying out all night without permission	27.01%
Being questioned by police by something they suspect involvement in	20.33%
Stealing something from a store or school	19.83%
Intentionally damaging or destroying something	25.68%
Fought someone to the point where they needed care for their injuries	6.96%
Attacking someone with the intention of causing serious harm	7.84%
Sold drugs	7.87%
Passenger in vehicle driven under the influence (DUI)	29.33%
Driving a vehicle under the influence	11.56%
Cigarette use	Some engagement: 26.17% Once or more per week: 17.79%
Alcohol use	Some engagement: 57.98% Once or more per week: 16.41%
Using alcohol to the point of intoxication	Some engagement: 55.81% Once or more per week: 11.06%
Marijuana use	Some engagement: 35.87% Once or more per week: 10.81%
Hallucinogen use	8.94%
Other drug use	9.05%
Considered Suicide	7.22%
Attempted Suicide	1.74%
Sexual Intercourse	40.75%

Table 3 – Model fit criteria for latent class analysis, fitting the number of trajectories

Number of Trajectories	BIC Value	2(ΔBIC)
1	-41632.64	-
2	-40173.68	2917.92
3	-39740.06	867.24
4	-39570.33	339.46
5	-39473.14	194.38

3.2 Latent Class Analysis

The latent class analysis on maternal depression scores across childhood yielded a 5-trajectory model. The BIC values and the estimates of the log Bayes factors for the 1- to 5-group trajectory models are detailed in table 3. There was strong evidence for a 2-trajectory model over a 1-trajectory model (2(ΔBIC) = 2917.92), and this positive evidence favouring the more complex model was mirrored until 5 trajectories were modeled (2(ΔBIC) = 194.38). Graphs of the 3, 4, and 5-group trajectory models are presented in Figures 1-3. The 3-group trajectory model featured a no symptom, mild symptom and recurrent maternal depressive symptom group. The 4-group trajectory model contained the same maternal CES-D score groups as the 3-trajectory model, but included the trajectory that has increasing mean CES-D scores in mid childhood, peaking around age 8 and dropping off significantly in the adolescent years. Model fit procedure significantly favoured the 5-group model, which is discussed in more detail below.

After establishing the number of trajectories included in the latent class analysis, model fit then focuses on establishing the most parsimonious equation order, or shape of trajectory over time. This is achieved by dropping all non-significant equation orders (e.g. quartic, cubic or quadratic), except for linear equation orders which should remain despite the p-value (Andruff et al., 2009; Arrandale, 2006). In the final model, however, two non-significant quadratic equation orders remained in the model; both group 1 and group 2 had non-significant quadratic orders. However, when these two equation orders were

dropped to linear orders, the model fit was significantly worsened (2(ΔBIC) = -66.54), and thus the non-significant quadratic equations remained in the final trajectory model due to improved overall model fit.

The final model contained 5 maternal depression trajectory groups, and is shown in Figure 3. Group 1, the no symptom reference group, contained 49.46% of the sample, and had a quadratic equation. The mild symptom group (group 2), contained 35.53% of the sample, and had a linear equation. The adolescent exposure group (group 3) contained 9.69% of the sample, and had a cubic equation. The mid-childhood exposure group (group 4) contained 3.68% of the sample, and it had a cubic equation. Finally, the recurrent maternal depression group (group 5) contained 1.64% of the sample and followed a quadratic trajectory over time. Group 3 had a steadily increasing depression trajectory, with the mean CES-D score crossing the suggested cut-off score for the indication of depression by the age of 10. The mothers depressed during mid-childhood (group 4) had increasing symptoms of depression throughout childhood, peaking around child's age 8, then dropping off to mild symptoms by age 14. The recurrently depressed mothers (group 5) reported high symptoms of depression throughout the child's life into adolescence.

3.3 Between-group difference in covariates

The distribution of the covariates (SES, SLEs, gender, maternal alcohol use and adolescent CES-D scores at age 16-17) for the five groups are presented in Table 4. The group with peaking mean maternal CES-D scores in mid-childhood (group 4), had slightly less females than the other groups, there were no

statistically significant differences in gender by group (F = 0.78, p = 0.53). Compared to those in the no maternal depression group (group 1), those in all other maternal depression trajectory groups had significantly lower SES scores at baseline (all *p*-values < 0.004). Compared to the reference group, only those in the mild maternal depressive symptom group (group 2), and the mid-childhood exposure group (group 4) did not have significantly higher count of stressful life events (SLEs) at age 14-15; those in the adolescent exposure group (group 3), with moderate maternal depressive symptoms during adolescence group, (p = 0.003) and the recurrent maternal depressive symptom group (group 5), (p = 0.03) had a significantly higher count of SLEs. Mothers experiencing mild symtpoms (group 2; p < 0.001) and mothers depressed when the youth where in adolescence (group 3; p =0 .03) had a significantly higher count of binge drinking occasions than mothers without symptoms of depression.

Table 4 – Distribution of gender, SES, SLEs, and adolescent CES-D scores in the five latent class groups

Covariate	Group 1	Group 2	Group 3	Group 4	Group 5
Gender (% female)	51.48	48.63	54.41	39.81	50.12
Baseline SES (Mean [SE])	0.11 (0.07)	-0.12 (0.05)	-0.43 (0.08)	-0.32 (0.11)	-0.57 (0.25)
Stressful Life Events (Mean [SE])	0.29 (0.02)	0.40 (0.03)	0.54 (0.08)	0.33 (0.12)	0.74 (0.21)
Maternal Alcohol Use (Mean [SE])	0.81 (0.09)	1.84 (0.23)	2.40 (0.72)	2.05 (0.67)	6.13 (3.20)
Adolescent CES-D (Mean [SE])	7.24 (0.25)	8.26 (0.28)	10.44 (0.93)	8.06 (0.63)	11.53 (1.73)

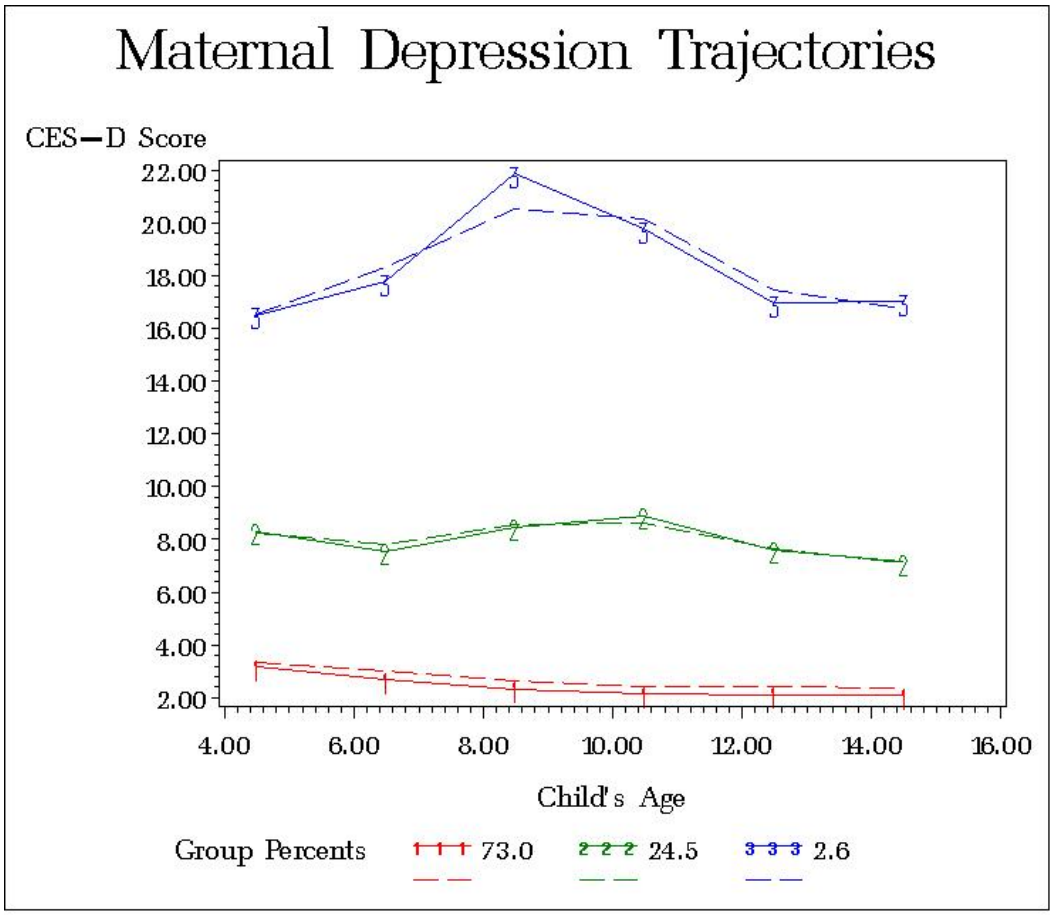

Figure 1: The 3-group trajectory model of maternal CES-D scores by child's age

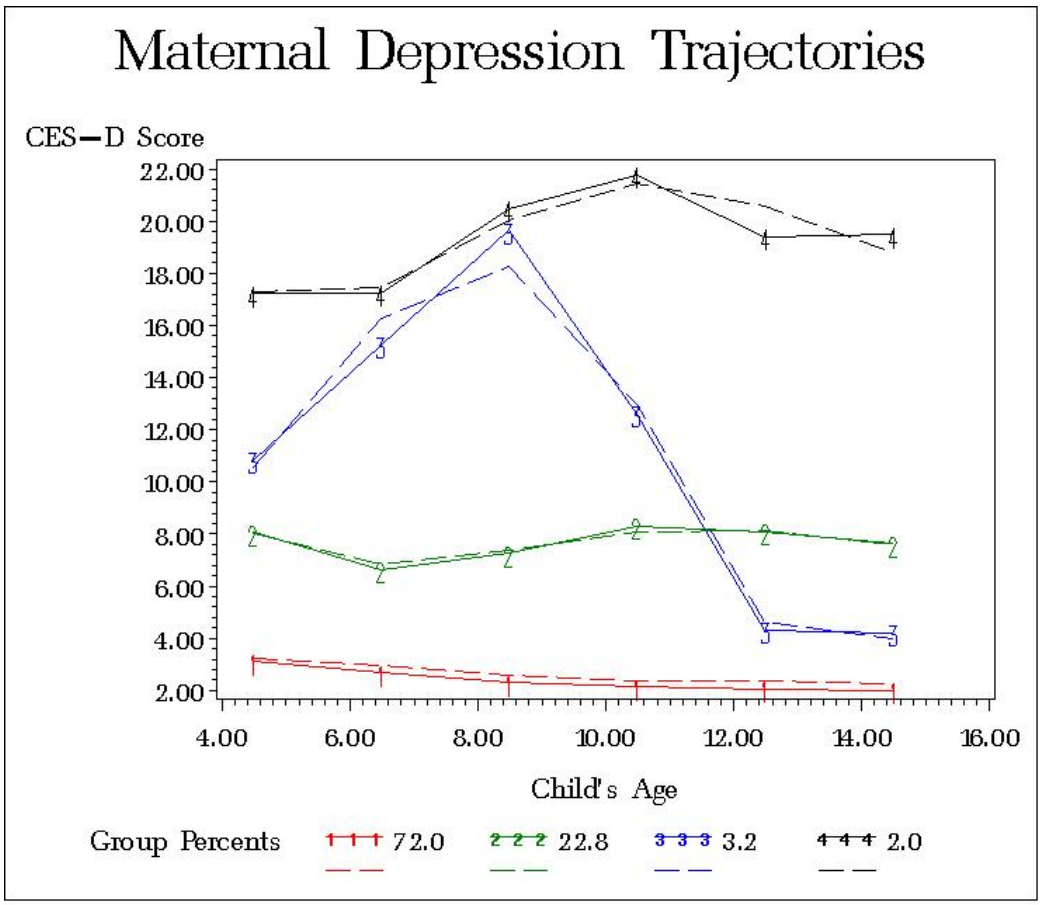

Figure 2: The 4-group trajectory model of maternal CES-D scores by child's age

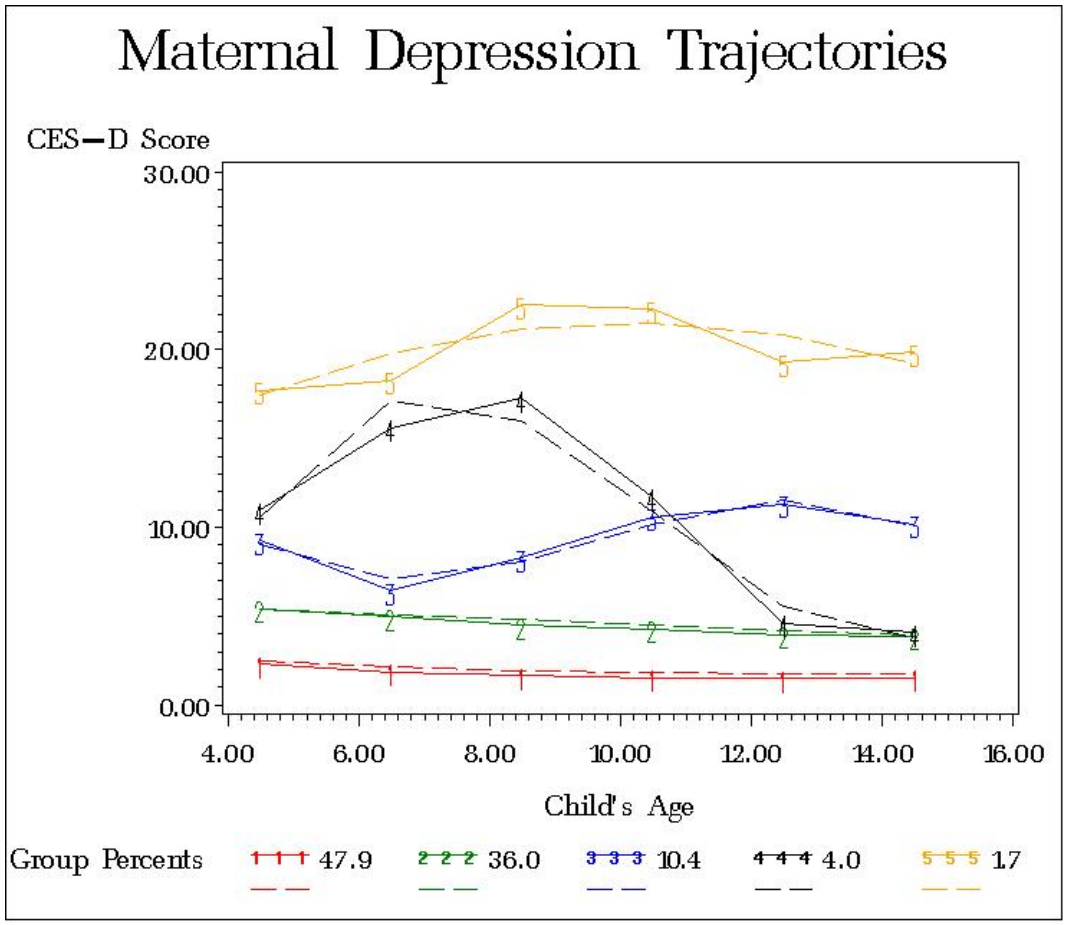

Figure 3: The final 5-group trajectory model of maternal CES-D scores by child's age

3.4 Factor Analysis of Health-Risk Behaviours

The factor analysis yielded five retained factors that had eigenvalues greater than 1, explaining 56% of the variance. Oblique varimax rotation methods were used. The results of the factor analysis and loadings are outlined in Table 5. The eigenvalues for the 5 factors were 5.87, 1.74, 1.36, 1.09, and 1.05, respectively.

Factor 1 contains the more commonly used substances: cigarettes, alcohol and marijuana, and operating/being a passenger in a vehicle driven under the influence. Factor 2 contains the non-violent delinquent variables: stealing, destruction of property, running away and being out all night. Factor 3 contains the less commonly used drugs: hallucinogens and other drugs such as crack/cocaine, as well as selling drugs. Factor 4 contains the violent delinquent behaviours: fighting someone to the point they had to care for their injuries, attacking someone with the intent of seriously hurting them, being questioned by police over something they thought you did, and carrying a weapon. Factor 5 contains the sexual assault variable and the engagement in glue/inhalant use. Factor scores were predicted for each child with complete outcome data.

Table 5 - Factors, variables (factor loadings)

Common Substances (Factor 1)	Non-violent Delinquency (Factor 2)	Illicit Substances (Factor 3)	Violent Delinquency (Factor 4)	Sexual Assault And Inhalants (Factor 5)
Cigarettes (0.54)	Out all night (0.62)	Selling drugs (0.39)	Questioned by police (0.43)	Sexual assault (0.81)
Alcohol (0.92)	Running away (0.80)	Hallucinogens (0.80)	Fought (0.86)	Glue/Inhalants (0.59)
Drunkenness (0.89)	Stealing (0.67)	Prescription Drugs (0.72)	Attack (0.81)	
Marijuana (0.59)	Destroy (0.69)	Other Drugs (0.85)	Weapon (0.49)	
Passenger in vehicle DUI (0.52)				
Driver in vehicle DUI (0.41)				

3.5 Regression Models

3.5.1 Multiple Linear Regression of Adolescent CES-D and Factor Scores

The results of the multiple linear regression models are outlined in Table 6. Compared to adolescents in the no maternal depression group (group 1), adolescents exposed to mild maternal symptoms (group 2), moderate symptoms, in adolescence (group 3), and recurrent maternal depressive symptoms (group 5) had significantly higher CES-D scores at age 16-17 ($p = 0.007$,0 .001, and 0.01, respectively). The youth in the recurrent maternal depression group (group 5) had CES-D scores that were 4.29 points higher than adolescents not exposed to maternal depression; this was the largest mean difference in CES-D scores. This result remained when adjusting for potential gender, SES, and cohort effects, however significance was lost with progressive covariate adjustment. Those exposed to maternal depression during mid-childhood (group 4) did not have significantly higher CES-D scores than the reference group ($p = 0.21$).

With respect to the factor scores, only the mid-childhood exposure group had higher scores than the reference group for common substance use (factor 1; $p = 0.002$). This effect remained upon adjustment of gender, SES, cohort effects, adolescent depression at 16, stressful life events at age 14/15 ($p = 0.005$, and 0.031), but not upon adjustment for maternal alcohol use ($p = 0.09$), though it was marginally significant. Those exposed to symptoms during mid-childhood (group 4) and recurrent maternal depression (group 5) had significantly higher factor scores for the non-violent delinquent behaviours (factor 2) than the reference

group (p = 0.02, and 0.01 respectively). While the same effect was observed when adjustments for gender, SES, and cohort effects were made (p = 0.03, and 0.01, respectively), the significance was lost after adjusting for the additional covariates (SLEs, current adolescent depression, maternal alcohol use; ps > 0.12). Adolescents exposed during mid-childhood (group 4) had significantly higher illicit substance scores (factor 3) than adolescents in the reference group; however, this was only found in bivariate analysis (p = 0.05). These adolescents also had significantly higher violent delinquent behaviour (factor 4) scores than adolescents not exposed to depressive symptoms in both the crude (p = 0.01), and first adjusted model (p = 0.02); however, this effect was only marginally significant when adjusting for current adolescent depression and SLEs (p = 0.06), and was non-significant after adjusting for maternal alcohol use (p = 0.13).

3.5.2 Logistic Regression of Individual Health-risk Behaviours

The following ordinal variables violated the proportional hazards assumption and were recoded to be dichotomous: being out all night, questioned by police, and fighting. Additionally, the following variables violated Statistics Canada data disclosure policy on small cell sizes, and were re-categorized to be dichotomous variables (never vs. ever engagement): stealing, destruction of property, attacking someone with the intention of hurting them, selling drugs, being either passenger/driver in a vehicle DUI, hallucinogen use, and other drug use. Thus, the remaining ordinal variables were: cigarette, alcohol, including alcohol use to intoxication, and marijuana use. The odds-ratios, confidence intervals, and p-values are presented in Table 7.

Compared to adolescents unexposed to maternal depressive symptoms, adolescents exposed to mild symptoms, increased symptoms during adolescence, and recurrent symptoms had significantly higher odds of stealing (*ps* = 0.001, 0.004, and 0.04, respectively); exposure to increased symptoms during mid-childhood was only found to have marginally significant increased odds of reporting stealing (p = 0.08). While this pattern remained upon adjustment for cohort, gender and SES effects, once adjusting for stressful life events, current adolescent depression and maternal alcohol use, only youth exposed to mild maternal depression had significantly increased odds of stealing over the reference group (p = 0.04). Adolescents exposed to recurrent maternal depressive symptoms had significantly higher odds (OR = 4.15, p = 0.005) of destruction of property, and adolescents exposed during mid-childhood (group 4) had marginally significant odds of destruction (OR = 2.09, p = 0.06); exposure to recurrent maternal depression remained marginally significant upon adjustment for all covariates (p = 0.09), until adjustment for maternal alcohol use was done (p = 0.31). Mid-childhood exposure remained marginally significant for higher odds of property destruction in the final adjusted model (OR = 2.45, p = 0.08). Adolescents exposed to increasing maternal depressive symptoms during mid-childhood (group 4) had 10.37 times the odds of reporting attacking someone with the intent of hurting them (p = 0.003), compared to adolescents without exposure to maternal depressive symptoms. This effect remained significant until the final adjustment was performed (OR = 4.84, p = 0.13). Adolescents exposed to increasing maternal depressive symptoms in adolescence (group 3) were 2.32

times as likely to report being a passenger in a vehicle driven under the influence of alcohol or other drugs ($p = 0.03$). While this effect remained marginally significant ($p = 0.07$) when adjusting for cohort, gender, and SES effects, this effect became non-significant after adjusting for other relevant covariates ($p = 0.88$).

With respect to the substance use variables, adolescents exposed to increasing symptoms in mid-childhood had higher odds of cigarette use, drunkenness, marijuana use, and hallucinogen use than the reference group. In the crude analysis, these adolescents were 4.31 times more likely to report cigarette use ($p < 0.001$); in the final adjustment model, the youth still had 2.37 times the odds of cigarette use at age 16-17 ($p = 0.03$). In the crude analysis, adolescents exposed during mid-childhood had 2.5 times the odds of drinking to intoxication than adolescents in group 1 ($p = 0.002$), and this effect remained significant upon adjustment for gender, SES, cohort ($p = 0.01$), current depression and stressful life events ($p =0.04$). However, upon final adjustment, these adolescents had 2.23 times the odds of the reference adolescents of drunkenness, but this effect was no longer statistically significant ($p = 0.10$). In the crude analysis, these adolescents had only marginally significantly higher odds of reporting marijuana use (OR = 2.19, $p = 0.07$); however, in the final model this effect was statistically significant (OR = 3.16, $p = 0.03$). The finding that adolescents exposed to maternal depression during mid-childhood had significantly higher odds of reporting hallucinogen use was consistent upon all levels of covariate adjustment; the final model found the highest odds and level of statistical significance (OR = 7.66, p =

0.006). Finally, no statistically significant associations were found for exposure to maternal depression and sexual intercourse, considering suicide, selling drugs, driving a vehicle under the influence, being out all night or being questioned by police.

Table 6 – Associations between maternal depression trajectory group and adolescent depression at 16, factor 1-5 scores

Outcome	Group°	Crude Regression Coefficient (95% CI)	Adjusted[1] Regression Coefficient (95% CI)	Adjusted[2] Regression Coefficient (95% CI)	Adjusted[3] Regression Coefficient (95% CI)
Depression at 16 (CES-D Score)	Group 2	1.02** (0.28 - 1.75)	0.93* (0.20 - 1.66)	n/a	n/a
	Group 3	3.20** (1.28 - 5.11)	2.98** (0.94 - 5.02)		
	Group 4	0.82 (-0.47 - 2.12)	0.94 (-0.44 - 2.32)		
	Group 5	4.29* (0.86 - 7.72)	3.80* (0.47 - 7.12)		
Common Substance (Factor 1) Scores	Group 2	0.02 (-0.16 - 0.20)	-0.002 (-0.21 - 0.20)	-0.02 (-0.23 - 0.02)	-0.04 (-0.25 -0.18)
	Group 3	0.19 (-0.16 - 0.57)	0.14 (-0.30 - 0.57)	0.05 (-0.37 - 0.48)	0.05 (-0.49 -0.59)
	Group 4	0.48** (0.17 - 0.80)	0.44 ** (0.14 – 0.75)	0.35 * (0.03 – 0.66)	0.40 (-0.06 -0.85)
	Group 5	0.16 (-0.42 - 0.74)	0.04 (-0.57 - 0.65)	-0.38 (-1.06 – 0.3)	-0.5 (-1.43 –0.43)
Non-violent Delinquent (Factor 2) Scores	Group 2	0.16 (-0.04 - 0.36)	0.12 (-0.09 - 0.33)	0.04 (-0.15 - 0.24)	0.01 (-0.21 –0.23)
	Group 3	0.24 (-0.08 - 0.56)	0.26 (-0.10 - 0.62)	0.06 (-0.25 - 0.36)	-0.03 (-0.41 –0.35)
	Group 4	0.45 * (0.08 - 0.82)	0.41 * (0.03 – 0.79)	0.21 (-0.14 - 0.57)	0.26 (-0.20 -0.72)
	Group 5	1.05 * (0.22-1.89)	1.01 * (0.20 – 1.81)	0.66 (-0.25 - 1.56)	0.81 (-0.21 –1.82)
Illicit Substance (Factor 3) Scores	Group 2	0.09 (-0.07 - 0.25)	0.07 (-0.10 - 0.24)	0.06 (-0.11 - 0.23)	0.02 (-0.15 –0.21)
	Group 3	0.17 (-0.10 - 0.46)	0.10 (-0.21 - 0.40)	0.002 (-0.29 - 0.30)	-0.09 (-0.46 -0.27)
	Group 4	0.49 * (-0.01 - 0.99)	0.45 (-0.09 - 0.99)	0.41 (-0.17 - 1.00)	0.69 (0.12 – 1.25)
	Group 5	0.15 (-0.24 - 0.54)	0.06 (-0.34 - 0.47)	-0.12 (-0.69 - 0.45)	-0.25 (-0.96 –0.45)
Violent Delinquent (Factor 4) Scores	Group 2	0.24 (-0.01 - 0.48)	0.22 (-0.06 - 0.49)	0.18 (-0.09 - 0.44)	0.11 (-0.15 -0.37)
	Group 3	-0.04 (-0.29 - 0.21)	-0.001 (-0.25 - 0.25)	-0.07 (-0.32 - 0.19)	-0.06 (-0.37 –0.26)
	Group 4	0.64 * (0.16 – 1.13)	0.55 * (0.09 – 1.00)	0.44 (-0.02 - 0.89)	0.45 (-0.13 –1.03)
	Group 5	0.48 (-0.14 - 1.09)	0.44 (-0.13 - 1.01)	0.35 (-0.43 - 1.12)	0.45 (-0.48 –1.38)
Sexual	Group 2	0.12 (-0.01 - 0.25)	0.10 (-0.05 - 0.24)	0.09 (-0.06 - 0.24)	0.08 (-0.08 –0.25)

Assault (Factor 5) Scores	Group 3	0.07 (-0.15 - 0.28)	0.03 (-0.21 - 0.26)	-0.08 (-0.32 - 0.15)	-0.09 (-0.39 –0.22)
	Group 4	-0.014 (-0.39 - 0.36)	-0.05 (-0.45 - 0.34)	-.11 (-0.54 - 0.32)	-0.08 (-0.36 –0.20)
	Group 5	-0.003 (-0.30 - 0.29)	-0.04 (-0.37 - 0.29)	-0.24 (-0.68 - 0.20)	-0.36 (-0.96 –0.24)

[1] Adjusted for gender, SES, cohort

[2] Adjusted for gender, SES, cohort, current adolescent depression, stressful life events (SLEs)

[3] Adjusted for gender, SES, cohort, current adolescent depression, SLEs, maternal alcohol use

* $p < 0.05$

**$p < 0.005$

° Note: Group 2 – mild maternal depressive symptoms; Group 3 –maternal depressive symptoms during adolescence; Group 4 –maternal depressive symptoms during mid-childhood; Group 5 – recurrent maternal depressive symptoms

Table 7 – Associations between maternal depression trajectory group and various health-risk behaviours

Outcome	Group°	Crude Odds-ratio (95% CI)	Adjusted[1] Odds-ratio (95% CI)	Adjusted[2] Odds-ratio (95% CI)	Adjusted[3] Odds-ratio (95% CI)
Being out all night	Group 2	1.13 (0.78-1.63)	1.04 (0.70 – 1.54)	1.03 (0.67 – 1.59)	0.98 (0.61 – 1.58)
	Group 3	0.80 (0.40 - 1.60)	0.69 (0.31 – 1.56)	0.64 (0.27 – 1.48)	0.81 (0.33 - 2.04)
	Group 4	2.29 (0.85 - 6.16)	2.18 (0.77 – 6.13)	1.90 (0.59 – 6.17)	1.36 (0.33 – 5.71)
	Group 5	1.52 (0.42-5.39)	1.41 (0.39 – 5.16)	0.65 (0.13 – 3.25)	0.86 (0.14 – 5.24)
Questioned by police	Group 2	1.21 (0.8 - 1.84)	1.19 (0.71 – 1.97)	1.20 (0.69 – 2.08)	1.14 (0.63 – 2.08)
	Group 3	0.94 (0.45-1.98)	0.96 (0.39 – 2.29)	0.92 (0.40 – 2.12)	0.88 (0.31 – 2.46)
	Group 4	1.42 (0.58-3.49)	1.34 (0.44 – 4.08)	1.16 (0.35 – 3.90)	1.35 (0.40 – 4.57)
	Group 5	1.44 (0.35 - 5.90)	1.47 (0.34 – 6.25)	0.74 (0.18 – 3.05)	0.95 (0.20 – 4.57)
Stealing from a store or school	Group 2	2.24 ** (1.43 – 3.51)	2.22 ** (1.34 – 3.66)	1.92 * (1.14-3.23)	1.79 * (1.03 – 3.13)
	Group 3	3.60 ** (1.49 – 8.68)	4.27 ** (1.72 –10.57)	1.82 (0.79 – 4.20)	1.46 (0.50 – 4.36)
	Group 4	3.68 (0.86 – 15.8)	3.92 * (1.01 –15.27)	3.39 (0.75 –15.40)	1.91 (0.5 – 7.73)
	Group 5	3.40 * (1.07 –10.84)	3.74 * (1.13 –12.40)	2.14 (0.67 – 6.79)	2.71 (0.56 -13.21)
Destruction of property	Group 2	1.13 (0.77 – 1.67)	1.10 (0.71 – 1.70)	1.02 (0.64 – 1.63)	0.91 (0.53 – 1.54)
	Group 3	1.62 (0.76 – 3.43)	1.76 (0.84 – 3.67)	0.97 (0.51 – 1.84)	0.82 (0.38 – 1.79)
	Group 4	2.09 (0.96 – 4.54)	1.94 (0.79 – 4.80)	1.57 (0.58 – 4.20)	2.45 (0.91 – 6.64)
	Group 5	4.15 ** (1.54 –11.16)	3.91 * (1.35 –11.33)	3.07 (0.83 –11.35)	2.13 (0.5 – 9.14)
Fighting	Group 2	1.92 * (1.03 - 3.58)	1.70 (0.83 – 3.48)	1.66 (0.80 – 3.42)	1.48 (0.68 – 3.22)
	Group 3	0.7 (0.24 - 2.01)	0.57 (0.17 - 1.98)	0.57 (0.15 – 2.2)	0.62 (0.14 – 2.71)
	Group 4	1.25 (0.4 - 3.92)	0.88 (0.24 – 3.19)	0.83 (0.21 – 3.33)	1.15 (0.30 – 4.44)
	Group 5	2.62 (0.46-15.09)	1.94 (0.32 –11.94)	1.47 (0.14 –15.12)	1.27 (0.08 -21.14)
Attacking	Group 2	1.94 * (1.09 – 3.48)	1.67 (0.87 – 3.21)	1.57 (0.77 – 3.20)	1.43 (0.65 – 3.16)
	Group 3	1.01 (0.19 – 5.45)	0.87 (0.09 – 8.30)	0.77 (0.14 – 4.32)	0.77 (0.08 – 7.62)

	Group 4	10.37 ** (2.19 –49.03)	8.40 ** (1.87 –37.65)	8.28 * (1.39 –49.19)	4.84 (0.63 -36.99)
	Group 5	4.51 (0.50 –43.47)	3.35 (0.33 –33.82)	2.48 (0.36 –16.99)	2.32 (0.18 -30.64)
Selling Drugs	Group 2	0.95 (0.46 – 1.95)	1.01 (0.44 – 2.29)	0.88 (0.38 – 2.03)	0.71 (0.29 – 1.72)
	Group 3	1.17 (0.36 – 3.75)	1.37 (0.37 – 5.05)	1.03 (0.24 – 4.46)	0.83 (0.15 – 4.80)
	Group 4	2.36 (0.80 – 6.98)	2.40 (0.67 – 8.54)	1.89 (0.42 – 8.49)	2.54 (0.43 -15.07)
	Group 5	2.46 (0.67 – 9.05)	2.60 (0.66 –10.28)	1.42 (0.24 – 8.25)	1.28 (0.15 -10.66)
Passenger in a vehicle driven under the influence	Group 2	1.13 (0.79 – 1.60)	1.06 (0.76 – 1.50)	1.05 (0.73 – 1.52)	0.99 (0.66 – 1.50)
	Group 3	2.32 * (1.11 – 4.83)	2.09 (0.93 – 4.70)	1.18 (0.61 – 2.28)	1.06 (0.51 – 2.21)
	Group 4	1.12 (0.51 – 2.45)	1.11 (0.47 – 2.61)	0.95 (0.37 – 2.43)	1.55 (0.49 – 4.87)
	Group 5	2.20 (0.83 – 5.81)	1.87 (0.66 – 5.27)	1.09 (0.34 – 3.50)	1.40 (0.33 – 5.98)
Driver in a vehicle driven under the influence	Group 2	1.49 (0.94 – 2.36)	1.23 (0.74 – 2.05)	1.18 (0.69 – 2.00)	1.08 (0.62 – 1.87)
	Group 3	1.28 (0.53 – 3.08)	1.00 (0.35 – 2.80)	0.81 (0.30 – 2.44)	0.79 (0.23 – 2.70)
	Group 4	1.08 (0.42 – 2.82)	0.95 (0.33 – 2.72)	0.71 (0.22 – 2.29)	0.73 (0.21 – 2.50)
	Group 5	0.84 (0.18 – 3.82)	0.65 (0.14 – 2.98)	0.28 (0.04 – 1.86)	0.26 (0.03 – 2.22)
Cigarette use	Group 2	1.24 (0.92 – 1.68)	1.13 (0.81 – 1.57)	1.08 (0.78 - 1.51)	1.03 (0.72 – 1.47)
	Group 3	1.51 (0.74 – 3.08)	1.21 (0.54 – 2.71)	1.22 (0.58 – 2.55)	0.92 (0.37 – 2.29)
	Group 4	4.31 ** (2.06 – 9.06)	4.47 ** (2.04 – 9.83)	4.01 ** (1.71-9.40)	2.37 * (1.11 – 5.07)
	Group 5	1.22 (0.5 - 3.01)	0.90 (0.34 – 2.35)	0.40 (0.10-1.54)	0.51 (0.1 - 2.77)
Alcohol use	Group 2	1.24 (0.92 – 1.69)	1.16 (0.80 – 1.68)	1.15 (0.78 – 1.71)	1.09 (0.73 – 1.64)
	Group 3	1.05 (0.57 – 1.93)	0.98 (0.46 – 2.08)	1.22 (0.72 – 2.05)	1.06 (0.56 – 2.02)
	Group 4	1.62 (0.89 – 2.95)	1.65 (0.90 – 2.99)	1.56 (0.83 – 2.93)	1.66 (0.72 – 3.82)
	Group 5	0.87 (0.31 – 2.38)	0.73 (0.25 – 2.12)	0.49 (0.15 – 1.64)	0.51 (0.12 – 2.23)
Alcohol use to intoxication	Group 2	1.04 (0.75 – 1.43)	0.99 (0.68 – 1.44)	0.93 (0.62 – 1.39)	0.94 (0.61 – 1.46)
	Group 3	0.85 (0.48 – 1.49)	0.73 (0.35 – 1.51)	0.63 (0.29 – 1.35)	0.66 (0.25 – 1.71)
	Group 4	2.5 ** (1.41 – 4.44)	2.14 * (1.18 – 3.89)	1.97 * (1.03 – 3.76)	2.23 (0.85 – 5.86)

	Group 5	1.38 (0.52 – 3.66)	1.23 (0.44 – 3.45)	0.81 (0.20 – 3.37)	0.95 (0.18 – 5.10)
Marijuana use	Group 2	1.21 (0.91 – 1.61)	1.21 (0.88 – 1.65)	1.16 (0.83 -1.62)	1.11 (0.78 – 1.59)
	Group 3	1.18 (0.59-2.36)	1.13 (0.51 – 2.50)	1.28 (0.65 – 2.52)	1.04 (0.45 – 2.40)
	Group 4	2.19 (0.95 – 5.02)	2.33 (0.98 – 5.56)	2.01 (0.80 – 5.07)	3.16 * (1.16 – 8.62)
	Group 5	1.33 (0.59 – 2.98)	1.29 (0.55 – 3.02)	0.78 (0.27 – 2.17)	0.95 (0.29 – 3.13)
Hallucino-gen use	Group 2	1.25 (0.71 – 2.23)	1.20 (0.63 – 2.29)	1.29 (0.63 – 2.62)	1.20 (0.56 – 2.56)
	Group 3	1.08 (0.53 – 2.20)	0.65 (0.23 – 1.81)	0.64 (0.21 – 1.94)	0.37 (0.10 – 1.36)
	Group 4	4.36 * (1.42 –13.36)	3.96 * (1.14 –13.68)	4.67 * (1.29 –16.86)	7.66 ** (1.80 -32.54)
	Group 5	1.10 (0.32 – 3.83)	1.01 (0.26 – 3.89)	0.89 (0.20 – 4.00)	0.54 (0.07 – 3.86)
Other drug (e.g. crack/ cocaine) use	Group 2	1.71 (0.98 – 2.99)	1.48 (0.82 – 2.66)	1.46 (0.79 – 2.70)	1.45 (0.74 – 2.83)
	Group 3	1.92 (0.78 – 4.72)	1.13 (0.37 – 3.45)	0.84 (0.27 – 2.62)	0.79 (0.22 – 2.82)
	Group 4	2.25 (0.75 – 6.70)	1.87 (0.60 – 5.87)	1.52 (0.45 – 5.14)	2.05 (0.49 – 8.59)
	Group 5	2.06 (0.56 – 7.55)	1.06 (0.26 – 4.30)	0.75 (0.16 – 3.57)	0.65 (0.09 – 4.73)
Considered Suicide	Group 2	1.47 (0.77 – 2.80)	1.61 (0.83 – 3.12)	1.55 (0.69 – 3.48)	1.31 (0.51 – 3.36)
	Group 3	1.61 (0.70-3.71)	1.62 (0.64 – 4.16)	0.84 (0.30 – 2.50)	0.62 (0.17 – 2.23)
	Group 4	0.68 (0.17 – 2.77)	0.77 (0.18 – 3.25)	0.59 (0.10 – 3.50)	0.43 (0.08 – 2.31)
	Group 5	1.72 (0.43 – 6.40)	1.91 (0.49 – 7.42)	0.59 (0.11-3.17)	0.78 (0.10 – 5.84)
Sexual intercourse	Group 2	1.36 (0.99 – 1.89)	1.21 (0.85 – 1.73)	1.20 (0.83 – 1.72)	1.15 (0.79 – 1.67)
	Group 3	1.16 (0.67 – 2.01)	0.84 (0.45 – 1.57)	0.80 (0.40 – 1.59)	0.75 (0.34 – 1.65)
	Group 4	1.31 (0.54 – 3.19)	1.18 (0.45 – 3.09)	1.03 (0.37 – 2.82)	1.12 (0.4 – 3.12)
	Group 5	2.03 (0.72 – 5.69)	1.31 (0.41 – 4.16)	0.86 (0.24 – 3.07)	1.14 (0.25 – 5.21)

[1] Adjusted for gender, SES, cohort

[2] Adjusted for gender, SES, cohort, current adolescent depression, stressful life events (SLEs)

[3] Adjusted for gender, SES, cohort, current adolescent depression, SLEs, maternal alcohol use

* $p < 0.05$, **$p < 0.005$

° Note: Group 2 – mild maternal depressive symptoms; Group 3 –maternal depressive symptoms during adolescence; Group 4 –maternal depressive symptoms during mid-childhood; Group 5 – recurrent maternal depressive symptoms

3.5.3 Survival Analysis of Substance Use and Sexual Intercourse

The proportional hazard assumption of Cox regression models was tested, and was not violated. Hazard ratios, confidence intervals, and p-values from the Cox regression models are displayed in Table 8. Survival graphs for each survival analysis outcome are presented in Figures 4-10.

Adolescents exposed to mild depressive symptoms (group 2: HR = 1.27, p = 0.03) and increasing symptoms during mid-childhood then decreased symptoms in adolescence, (group 4: HR = 2.18, $p < 0.001$) were significantly more likely to engage in cigarette use earlier than adolescents in the reference group in the crude analysis. The effect remained significant only for adolescents exposed during mid-childhood, who were 2.33 times more likely to engage in cigarette use earlier compared to adolescents in the reference group in the final adjustment model (p = 0.003). A similar pattern was observed with respect to alcohol use, with adolescents exposed to maternal depression during mid-childhood, who were 1.70 times more likely to engage in alcohol use earlier than the reference adolescents in the fully adjusted model (p = 0.02). No significant associations were found for alcohol use to the point of intoxication in the Cox regression models. These adolescents were also 2.58 times more likely to engage in marijuana use earlier than adolescents in the reference group (p = 0.003) in the fully adjusted model, and significant associations were found for all levels of adjustment. The same pattern was found for hallucinogen use; adolescents with depressed mothers during mid-childhood had significantly higher hazard ratios compared to adolescents without depressed mothers at all levels of covariate adjustment (ps =

0.01, .02, .02, .006, respectively). In the final model, these adolescents were 5.56 times more likely to engage in hallucinogen use earlier than those in the reference group, after adjusting for the effect of gender, cohort, SES, SLEs, current depression, and maternal alcohol use. Finally, while significant associations were found for the mild maternal depressive symptom group (group 2; HR = 1.28, p = 0.04) and the recurrent, high maternal depressive symptom group (group 5; 1.98, p = 0.04) for sexual intercourse in the bivariate analysis, this significant effect was lost upon adjustment for relevant covariates.

Kaplan-Meier survival graphs for each survival analysis outcome are presented in Figures 4-10. While the curves do not illustrate dramatic group differences in time-to-onset of the various behaviours, the mid-childhood exposure group, illustrated by the yellow line, is generally the group with the lowest survival estimates. Thus, the graphs follow the overall trend of the Cox regression models, where adolescents exposed to increased levels of maternal depressive symptoms during childhood and decreased levels of maternal depressive symptoms during early adolescence engage in the use of various substances earlier than adolescents not exposed to maternal depressive symptoms.

Table 8 - Cox regression model associations between maternal depression trajectory group and time of onset of health-risk behaviours

Outcome	Group°	Crude Hazard-ratio (95% CI)	Adjusted[1] Hazard-ratio (95% CI)	Adjusted[2] Hazard-ratio (95% CI)	Adjusted[3] Hazard-ratio (95% CI)
Cigarette use	Group 2	1.27 * (1.02 – 1.57)	1.25 * (1.00 -1.56)	1.13 (0.91 – 1.42)	1.10 (0.86 – 1.39)
	Group 3	1.17 (0.77 – 1.77)	1.01 (0.63 – 1.62)	1.04 (0.64 – 1.67)	0.89 (0.50 – 1.59)
	Group 4	2.18 ** (1.52 – 3.13)	2.15 ** (1.48 – 3.12)	2.27 ** (1.52 – 3.41)	2.33 ** (1.33 – 4.08)
	Group 5	1.15 (0.80 – 1.67)	0.92 (0.44 – 1.92)	0.43 (0.16 – 1.13)	0.54 (0.18 – 1.59)
Alcohol use	Group 2	1.15 * (1.02 – 1.31)	1.14 (0.99 – 1.31)	1.08 (0.93 – 1.27)	1.08 (0.92 – 1.27)
	Group 3	0.96 (0.70 – 1.31)	0.95 (0.68 – 1.34)	1.15 (0.89-1.48)	1.17 (0.87 – 1.59)
	Group 4	1.37 * (1.05 – 1.80)	1.43 * (1.05 – 1.96)	1.44 * (1.00 – 2.08)	1.70 * (1.07 – 2.71)
	Group 5	1.16 (0.80 – 1.67)	1.31 (0.87 – 1.98)	0.92 (0.58 – 1.47)	0.86 (0.47 – 1.55)
Drunken-ness	Group 2	1.12 (0.93 – 1.36)	1.03 (0.89-1.20)	0.97 (0.81 – 1.16)	0.96 (0.78 – 1.17)
	Group 3	0.85 (0.57 – 1.26)	0.90 (0.62 – 1.29)	0.89 (0.59 – 1.35)	1.01 (0.63 – 1.61)
	Group 4	1.14 (0.7 – 1.88)	1.28 (0.82 – 2.01)	1.52 (0.80 – 2.91)	2.17 (0.96 – 4.89)
	Group 5	1.14 (0.67 – 1.94)	1.04 (0.59 – 1.87)	0.97 (0.45 – 2.09)	1.30 (0.66 – 2.57)
Marijuana use	Group 2	1.28 * (1.05 – 1.57)	1.24 * (1.01 – 1.51)	1.09 (0.88 – 1.37)	1.06 (0.84 – 1.33)
	Group 3	1.05 (0.72 – 1.53)	0.99 (0.65 – 1.51)	1.01 (0.67 – 1.53)	0.90 (0.55-1.47)
	Group 4	1.78 * (1.10 – 2.86)	1.91 * (1.15 – 3.16)	1.93 * (1.07 – 3.49)	2.58 ** (1.42 – 4.69)
	Group 5	1.28 (0.71 – 2.32)	1.33 (0.72 – 2.47)	0.78 (0.37 – 1.62)	0.89 (0.39 – 2.02)
Hallucino-gen use	Group 2	1.48 (0.93 – 2.38)	1.52 (0.91 – 2.53)	1.39 (0.79 – 2.45)	1.26 (0.69 – 2.30)
	Group 3	1.33 (0.70 – 2.53)	1.04 (0.43 – 2.50)	0.86 (0.33 – 2.22)	0.64 (0.20 – 2.05)
	Group 4	3.52 * (1.32 – 9.38)	3.51 * (1.26 – 9.77)	3.67 * (1.24 –10.83)	5.56 * (1.66 -18.65)
	Group 5	1.11 (0.01 - 238.74)	1.12 (0.01- 218.22)	0.80 (0.002 – 250.86)	0.57 (.0001 – 5616.7)
Other drugs	Group 2	1.95 **	1.77 *	1.54	1.53

(e.g. crack/ cocaine) use		(1.25 – 3.03)	(1.13 – 2.77)	(0.95 – 2.49)	(0.90 – 2.59)
	Group 3	1.86 (0.94 – 3.69)	1.23 (0.56 – 2.70)	0.92 (0.39 – 2.17)	1.01 (0.4 – 2.53)
	Group 4	1.59 (0.63 – 4.01)	1.43 (0.59 – 3.50)	1.19 (0.40 – 3.52)	1.43 (0.05-40.49)
	Group 5	2.51 (0.09 – 69.53)	1.59 (0.04 –62.34)	1.11 (0.03 –40.90)	1.26 (0.001 – 1636.6)
Sexual intercourse	Group 2	1.28 * (1.01 – 1.62)	1.26 (0.98 – 1.62)	1.13 (0.88 – 1.45)	1.12 (0.87 – 1.43)
	Group 3	1.15 (0.78 – 1.69)	1.00 (0.65 – 1.54)	0.85 (0.52 – 1.37)	0.83 (0.49 – 1.41)
	Group 4	1.20 (0.69 – 2.08)	1.28 (0.74 – 2.24)	1.09 (0.58 – 2.06)	1.12 (0.62 – 2.01)
	Group 5	1.98 * (1.04 – 3.79)	1.86 (0.89 – 3.86)	1.02 (0.43 – 2.46)	1.25 (0.50-3.12)

[1] Adjusted for gender, SES, cohort

[2] Adjusted for gender, SES, cohort, current adolescent depression, stressful life events (SLEs)

[3] Adjusted for gender, SES, cohort, current adolescent depression, SLEs, maternal alcohol use

* $p < 0.05$

**$p < 0.005$

° Note: Group 2 – mild maternal depressive symptoms; Group 3 –maternal depressive symptoms during adolescence; Group 4 –maternal depressive symptoms during mid-childhood; Group 5 – recurrent maternal depressive symptoms

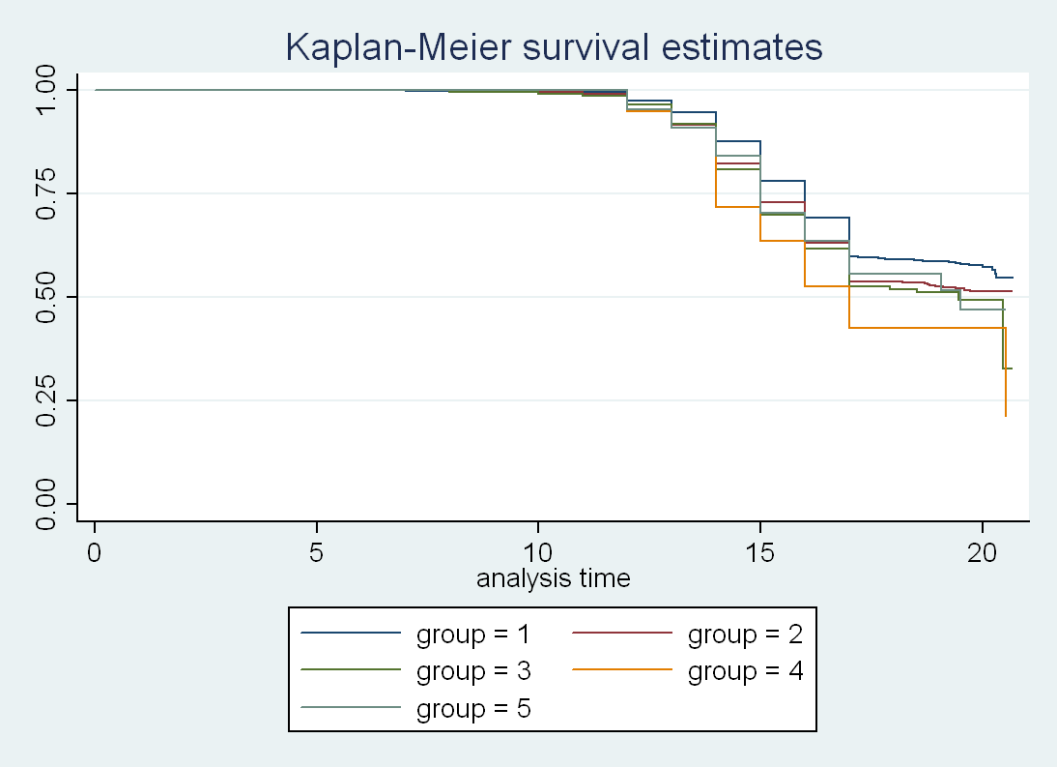

Figure 4: Kaplan-Meier survival estimates for engagement in cigarette use by maternal depression trajectory group

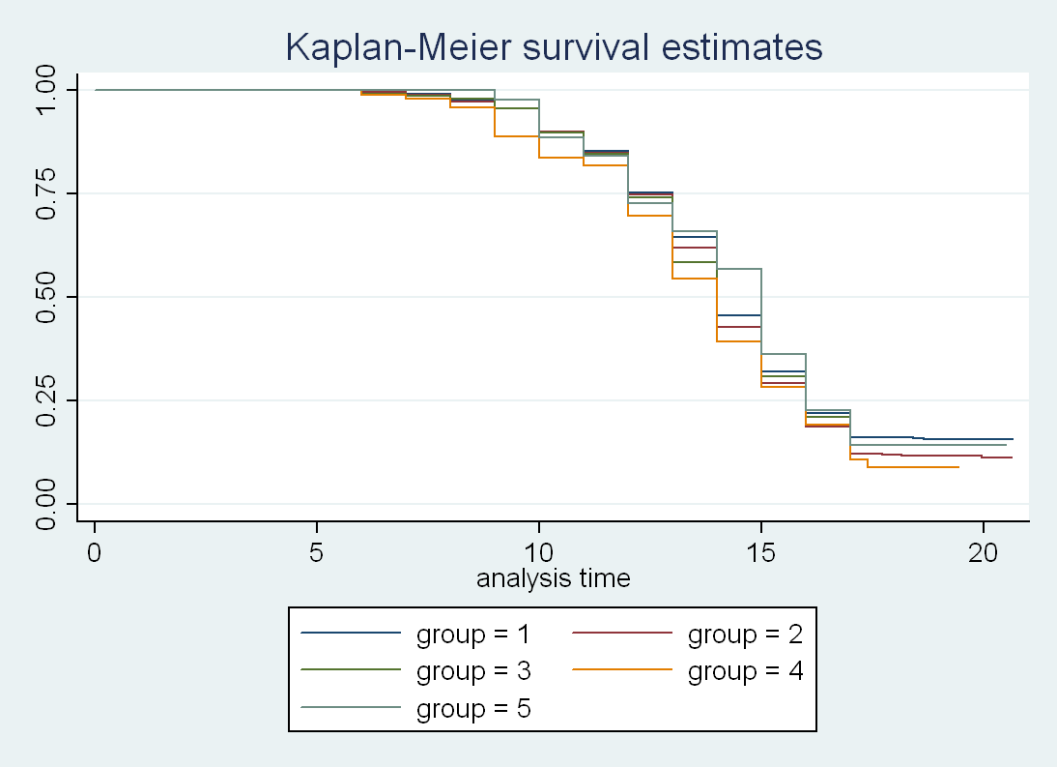

Figure 5: Kaplan-Meier survival estimates for engagement in alcohol use by maternal depression trajectory group

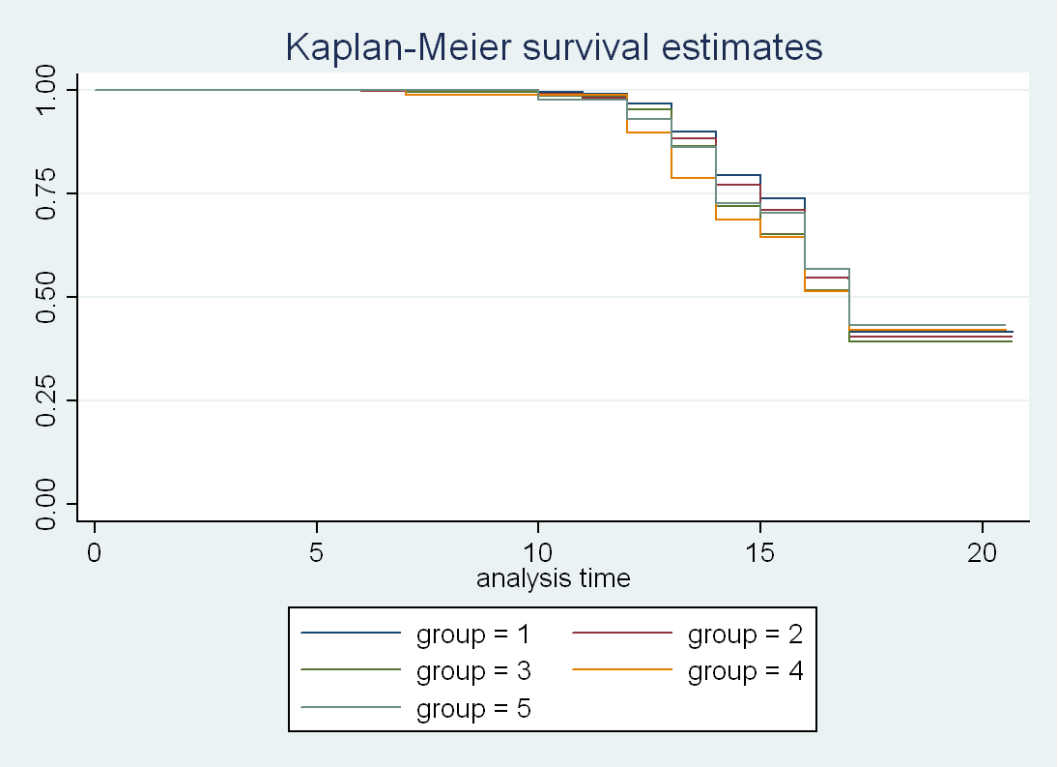

Figure 6: Kaplan-Meier survival estimates for engagement in alcohol use to the point of intoxication by maternal depression trajectory group

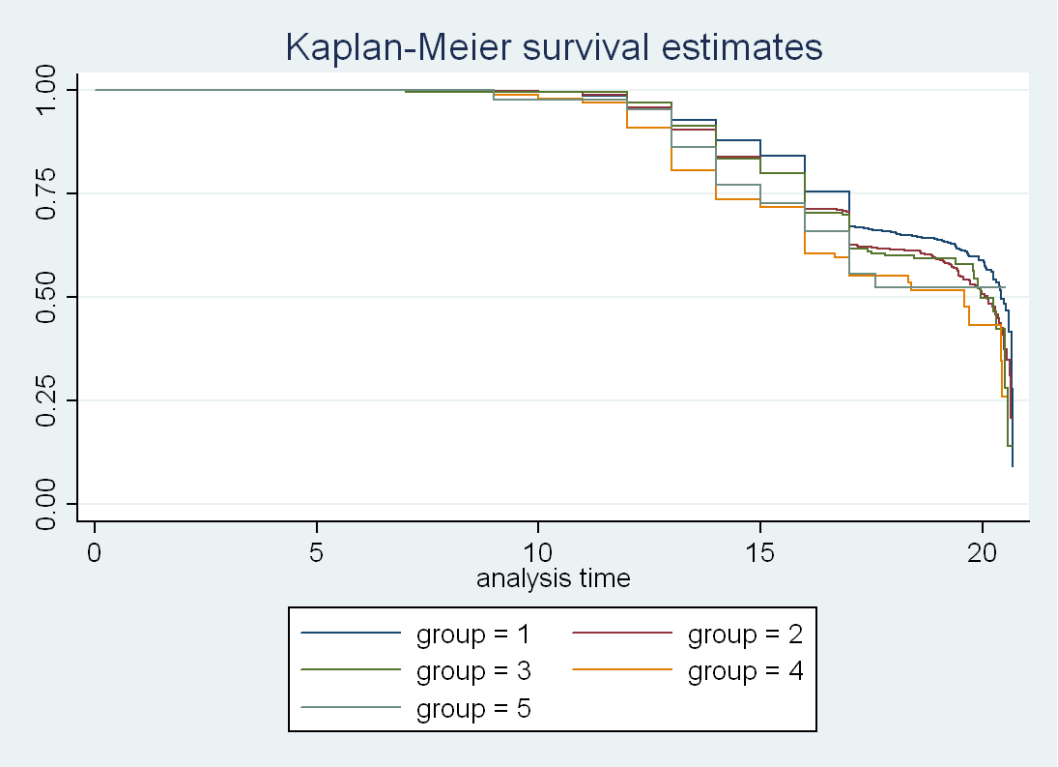

Figure 7: Kaplan-Meier survival estimates for engagement in marijuana use by maternal depression trajectory group

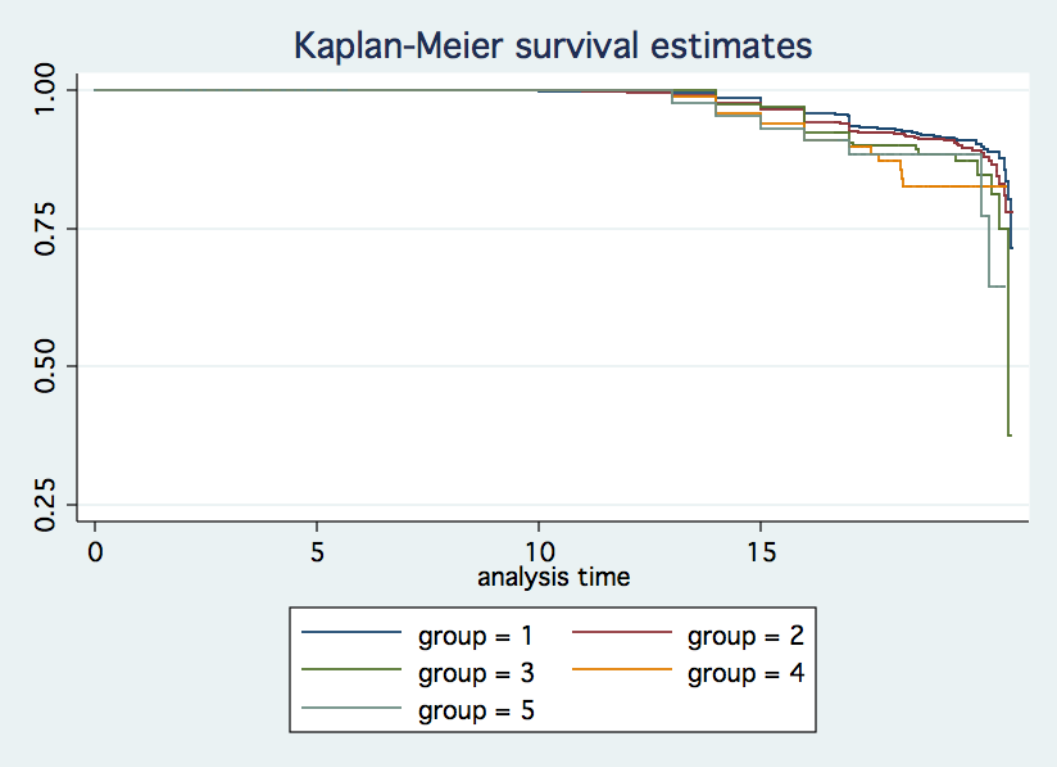

Figure 8: Kaplan-Meier survival estimates for engagement in hallucinogen use by maternal depression trajectory group

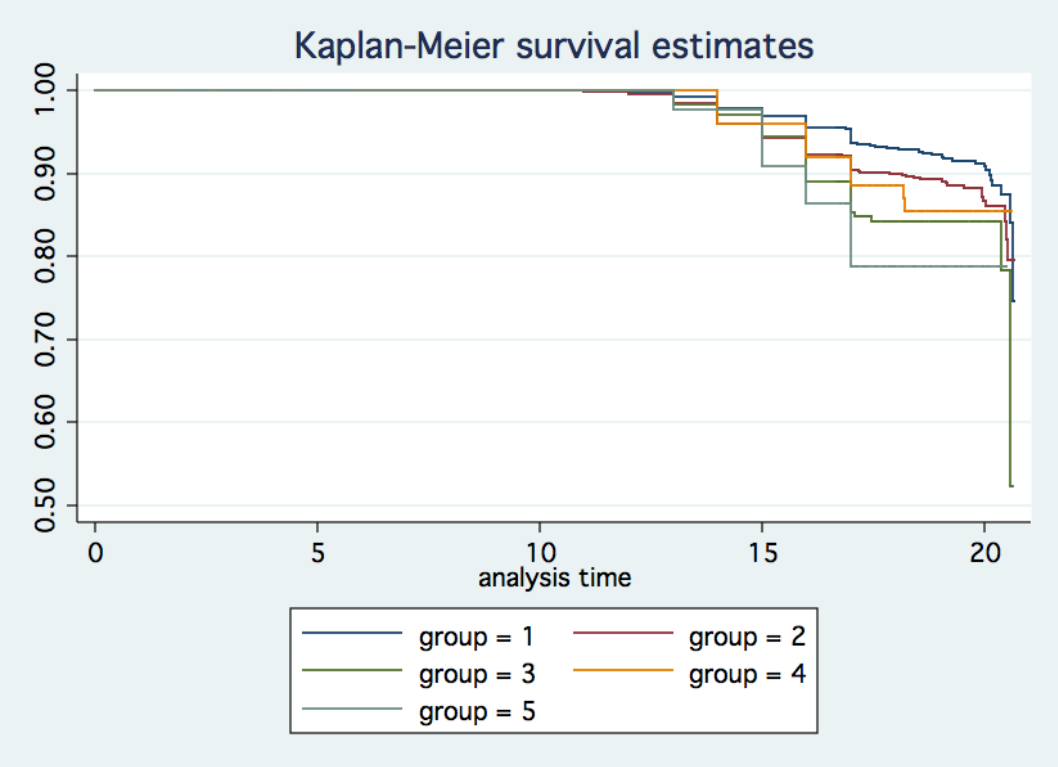

Figure 9: Kaplan-Meier survival estimates for engagement in other drug use (eg. crack/cocaine) by maternal depression trajectory group

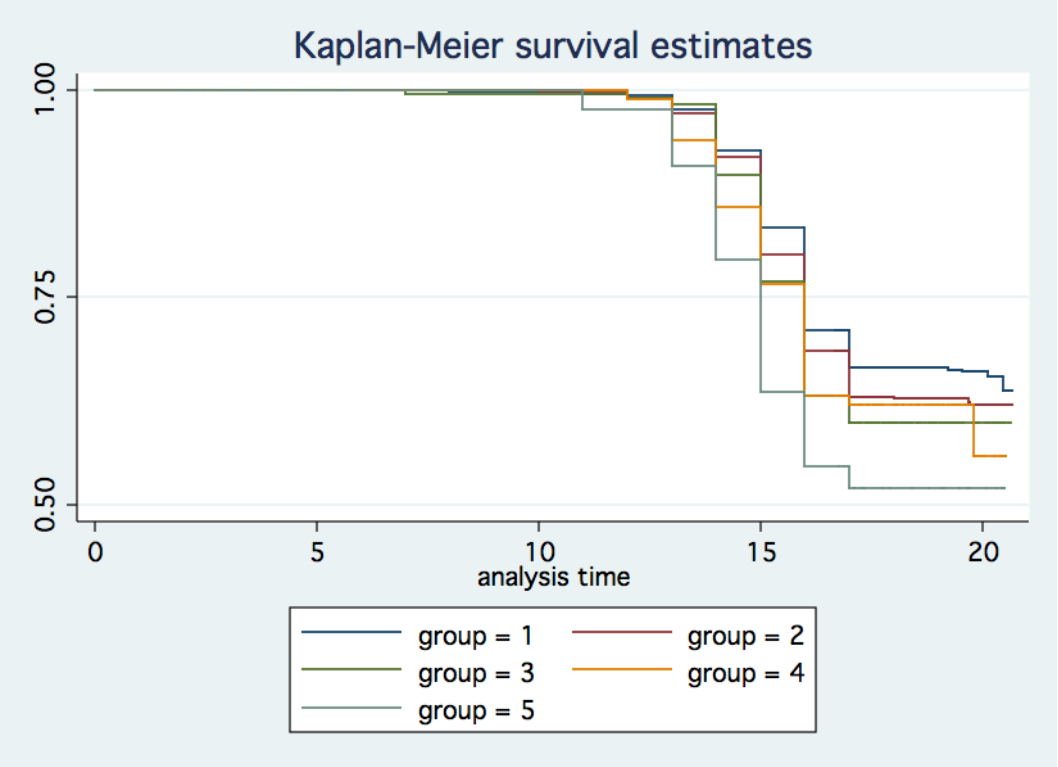

Figure 10: Kaplan-Meier survival estimates for engagement in sexual intercourse by maternal depression trajectory group

3.5.4 Interactions

Interactions were tested for maternal depression group with gender, depression, stressful life events and maternal alcohol use. No significant interactions with stressful life events, current adolescent depression, or maternal alcohol use were found. Significant maternal depression trajectory group by gender interactions were found for inhalant use and sexual assault (factor 5) scores ($p = 0.03$), and marginally significant interactions by gender were found for current adolescent depression ($p = 0.07$), and non-violent delinquency (factor 2) scores ($p = 0.06$). The results, stratified by gender, are presented in Table 9. Females in the recurrent maternal depression group (group 5) had significantly higher CES-D scores ($p = 0.01$) than males. Males in the recurrently depression maternal depression trajectory (group 5) had significantly higher non-violent delinquency scores ($p = 0.02$) than females. Finally, compared to males, females in the mid-childhood exposure group (group 4) had significantly lower sexual assault/inhalant use scores ($p = 0.04$), and females in group 5 had marginally significantly lower sexual assault/inhalant use scores ($p = 0.08$). Finally, compared to females, males exposed to maternal depression during adolesence had significantly lower sexual assault/inhalant use scores ($p = 0.05$).

3.6 Missing Data

Missing outcome data was an obstacle for this research. 43.44% of the sample had incomplete health-risk behaviour data, which affected the sample size of the factor analysis (unweighted N=1632). However, incomplete outcome data

was not significantly associated with maternal depression group ($F = 1.34$, $p = 0.25$), baseline SES ($t = -1.37$, $p = 0.17$), stressful life events ($t = -0.47$, $p = 0.64$), gender ($F = 2.84$, $p = 0.09$), or maternal alcohol use ($t = -0.99$, $p = 0.32$). Incomplete outcome data was significantly associated with being in the older cohort ($F = 16.65$, $p < 0.001$), and depression at 16 ($t = -3.47$, $p = 0.001$); however, counter intuitively, those with missing data had significantly lower CES-D scores than those with complete outcome data.

3.7 Attrition

Attrition was another issue in this research. While 7600 children were surveyed in cycle 1, 2358 dropped out after cycle 1, and did not respond to any other data cycles. However, a large portion of this was largely due to budgetary restrictions to Statistics Canada (Statistics Canada, 1997b). Furthermore, the bootstrap weights used in subsequent analyses account for this attrition. A further 2375 had dropped out by the outcome years, in Cycle 7 and 8, leaving the remaining 2910 in the study. The weighted prevalence indicated that the attrition rate for this study was 37.47%. Attrition from the study was not associated with maternal depression at any cycle, except for depression during 1996/97 (cycle 2); those that dropped out had significantly higher CES-D scores (4.85), than those who remained (4.23), $t = 2.29$, $p = 0.02$. Attrition was not associated with child's gender ($F = 2.66$, $p = 0.10$), cohort ($F = 2.55$, $p = 0.11$), or maternal alcohol use at child's age 14 ($t = -1.18$, $p = 0.24$). However, drop out from the study was significantly associated with the report of a stressful life event at baseline in 1994/95 ($F = 2.48$, $p = 0.05$), stressful life events reported at age 14-15 ($t = 11.87$,

$p < 0.001$), maternal alcohol use in 1994/95 ($t = 2.48$, $p = 0.01$), and a significantly lower baseline SES ($t = -2.04$, $p = 0.04$).

Table 9 – Multiple linear regression interactions results

Variable	Group°	Girls (Mean[95%CI])	Boys (Mean[95%CI])
Depression at 16 (CES-D Score)[1]	Group 2	0.64 (-0.65 – 1.93)	0.21 (-0.78 – 1.19)
	Group 3	2.76 (-1.22 – 6.73)	2.36 (-1.14 – 5.86)
	Group 4	1.08 (-1.21 – 3.37)	-1.06 (-2.51 – 0.38)
	Group 5	10.56 * (2.58 – 18.55)	1.91 (-3.27 – 7.10)
Non-violent Delinquent (Factor 2) Score[2]	Group 2	-0.06 (-0.32 – 0.19)	0.08 (-0.28 – 0.45)
	Group 3	-0.08 (-0.67 – 0.51)	-0.02 (-0.53 – 0.49)
	Group 4	-0.07 (-0.50 – 0.35)	0.56 (-0.26 – 1.38)
	Group 5	-0.32 (-1.64 – 1.01)	1.30 * (0.22 – 2.38)
Sexual Assault and Inhalants (Factor 5) Score[2]	Group 2	0.02 (-0.11 – 0.16)	0.12 (-0.18 – 0.42)
	Group 3	0.17 (-.22 - .56)	-0.45 * (-0.89 - -0.01)
	Group 4	-0.24 (-0.46 – -0.1)	0.11 (-0.34 – 0.56)
	Group 5	-0.43 (-.91 – 0.05)	-0.30 (-1.21 – 0.61)

[1] Analysis adjusted for gender, SES, cohort, SLEs, and maternal alcohol use
[2] Analysis adjusted for gender, SES, cohort, SLEs, current adolescent depression, and maternal alcohol use
* $p < 0.05$
° Note: Group 2 – mild maternal depressive symptoms; Group 3 –maternal depressive symptoms during adolescence; Group 4 –maternal depressive symptoms during mid-childhood; Group 5 – recurrent maternal depressive symptoms

Chapter 4 – Discussion and Conclusion

A preponderance of research has reported the outcomes that may arise after exposure to maternal depression, occurring during childhood through to adolescence and young adulthood (Goodman & Gotlib, 1999; Goodman et al., 2011). The results of this study are congruent with the hypothesis that exposure to maternal depression during childhood is associated with internalizing and externalizing, as well as substance use, behaviours in adolescence. A summary of the main results, ordered by type of statistical analysis, is outlined below.

4.1 Summary of Main Results

Some of the descriptive findings in this study warrant further discussion. The estimated prevalence of maternal depression in the sample fell within the expected range of 10-20% (Horwitz et al., 2009; Marcus et al., 2003), and the prevalence of maternal depression decreased as the children aged. Also, prevalence of depression in the adolescents (21%) exceeded the prevalence of depression in mothers; this is to be expected, as adolescents tend to experience higher levels of depressive symptomatology (Angold & Costello, 2001), and an estimated 25% of youth were found to be depressed using the same scale (Poulin et al., 2005). However, these prevalence estimates are based on established cut-off points of a symptomatology scale, and are not based on a clinician's diagnosis, and thus may not accurately represent the true prevalence of depression.

With respect to the health-risk behaviours, some were reported more frequently than others. Nearly half the sample had engaged in sexual intercourse by age 16-17, 30% were passengers in vehicles driven under the influence, 27%

reported staying out all night, and a quarter of the sample reported property destruction. Certain behaviours were much less common; less than 10% reported fighting, attacking someone, considering suicide or selling drugs, and less than 2% reported attempting suicide. With respect to substance use, some substances were much more commonly reported, such as cigarette use, where 26% of youth reported some use, and almost 20% smoked at least 1-2 times per week. Almost 60% had tried alcohol and reported being drunk, and 16% of the sample used alcohol at least 1-2 times per week. A third of the adolescents had tried marijuana, and 10% reported weekly use. Some substances, as expected, were much less commonly used, such as hallucinogens (9%), and other drugs such as crack or cocaine (9%). Therefore, engagement in health-risk behaviours in adolescence is widespread, yet variable, with certain behaviours being much more common than others.

4.1.1 Trajectories of Maternal Depression Throughout Childhood

The latent class analysis model yielded 5 distinct trajectories of maternal depressive symptoms from age 4-15 of the children. These trajectories were then used as a categorical variable in further regression analyses. Children in the no depressive symptom group, were exposed to very low CES-D scores in childhood. The majority of the sample fell within this category. The next group of children were exposed to mild, slightly elevated mean CES-D scores. Whereas the cut-off to indicate depression with this scale is a score of 9 (Letourneau et al., 2010; Naicker et al., 2012; Somers & Willms, 2002), the mean maternal CES-D score hovered around 5 across childhood. The adolescents in the third group were

exposed to increasing mean CES-D throughout later childhood into the adolescent years. Mean maternal CES-D scores for those in the fourth group started relatively high, at a score of approximately 11, then rose from age 4 to age 8, after which they steeply plummeted to similar mean CES-D scores as those experienced by mothers in group 2. Finally, mothers who were included in the final group experienced high and recurrent depressive symptoms.

It is important to note that while using trajectory modeling as a categorical variable is useful in describing longitudinal data, these discrete trajectories may not fully represent real-world phenomenon, and caution must be taken in interpreting the findings with respect to membership to distinct maternal depression groups. However, the use of latent class analysis allows clusters of individuals who follow similar behavioural trajectories over time to be identified, and used in further statistical prediction and inference (Jones et al., 2001). Thus, modeling trajectories is a useful tool for the repeated measurement of depressive symptoms; the use of trajectories with respect to childhood exposure to maternal depressive symptoms allows group membership to be inferred from the longitudinal scores on the CES-D. The recurrent maternal depression group was small, however the model fit criterion was significantly improved in the 5-trajectory model, compared to the 4-trajectory model. Thus, while the small group size may have become problematic when combined with missing data, discussed further in this chapter in the limitations section, the selected model from the latent class analysis best represented the data on maternal depressive symptomatology, thereby improving our estimates of childhood exposure to maternal depression.

4.1.2 Maternal Depression and Adolescent Depression

As expected, maternal depression significantly predicted adolescent CES-D scores; additionally, this appeared to follow a dose-response relationship, with children in the recurrent maternal depression group (group 5) reporting the highest mean CES-D scores of all the youth, followed by adolescents in the adolescent exposure to maternal depression group, and finally those whose mothers reported mild symptoms. However, adolescents exposed during mid-childhood did not have significantly higher CES-D scores than adolescents in the reference group. Adolescents in the mid-childhood exposure group, however, were exposed to higher maternal CES-D scores in mid-to-late childhood, with CES-D scores decreasing and dropping below the suggested cut-off score to indicate the presence of maternal depression (Somers & Willms, 2002) in adolescence. Therefore, perhaps this effect is explained by the proximal effects of being exposed to maternal depression during adolescence. It is possible that those adolescents who were more recently exposed to higher levels of maternal depression may be more likely to experience or report their own depressive symptoms. Adolescents in the adolescent exposure group and recurrent exposure group, for example, were both being exposed to higher mean maternal CES-D scores in the later years (age 14-15) than adolescents in the other trajectory groups. Indeed, there is an established relationship with current maternal depression and adolescent psychopathology (Naicker et al., 2012; Pilowsky et al., 2006).

When examining potential interactions, the results indicated that females in the recurrent maternal depression group had significantly higher CES-D scores than males, congruent with findings that female offspring of depressed mothers may have more internalizing difficulties than male offspring (Ensminger et al., 2003; Lewis, Rice, Harold, Collishaw, & Thapar, 2011).

4.1.3 Maternal Depression and Groups of Similar Behaviours

The factor analysis yielded 5 factors with high face validity. The highly loading (loadings of greater than 0.4) variables on factor 1 were the more common substances (cigarettes, alcohol, drunkenness, marijuana) as well as driving or being a passenger in vehicles driven under the influence. Factor 2 included non-violent delinquent behaviours, such as being out all night, running away, stealing, and destruction of property. Factor 3 had high loadings with the less common substances (hallucinogens, prescription drugs, and other drugs) as well as being involved in the sale of illicit substances. The variables that had high loadings on factor 4 were the violent delinquent behaviours, such as fighting, attacking someone with the intention of seriously hurting them, carrying a weapon, and being questioned by the police about something they suspected the youth's involvement in. Factor 5, however, contained two seemingly unrelated variables: inhalant use, and sexual assault. Missing outcome data, which is discussed further in the limitations section of this chapter, impacted the sample size used in the factor analysis.

Examination of the relationship between maternal depression and the factor scores for each of the 5 factors revealed a different pattern than the relationship between maternal depression and adolescent depression. Adolescents exposed to increasing mean maternal depressive scores in mid-childhood, then low levels of maternal depressive symptoms in early adolescence, engaged in more common substance use, non-violent delinquency, and violent delinquency behaviours than adolescents not exposed to maternal depression; additionally, these adolescents had significantly higher illicit substance use scores in the crude analysis, though this effect was lost upon final covariate adjustment. Children exposed to high mean maternal CES-D scores in mid-childhood appeared to have the poorest adolescent outcomes, including substance use, non-violent and violent delinquency.

These results may reflect the early development of a lasting behavioural pattern as a result of earlier exposure to maternal depression. Research indicates that during infancy, while most children engage in the use of physical aggression, those that do not learn to regulate its use during childhood are the most likely to continue the use of aggressive behaviour during adolescence (Tremblay et al., 2004). Furthermore, correlates of maternal depression are involved in the development of childhood aggression. For example, hostile parenting in childhood is associated with higher levels of physical aggression (Cote, Vaillancourt, Barker, Nagin, & Tremblay, 2007); hostile and negative parenting behaviours have been linked to maternal depression (Lovejoy et al., 2000). Pre-school children who were exposed to higher levels of maternal stress had more severe

externalizing problems at 3 different time points of follow-up: pre-school, first, and second grade (Baker, Heller, & Henker, 2000), indicating that maternal stress, which has its own relationship with depression, may be associated with the development of longitudinal externalizing behavioural patterns. Children of depressed mothers have demonstrated more externalizing behaviours (Civic & Holt, 2000; Essex et al., 2003; Essex et al., 2001), and those demonstrating higher levels of childhood externalizing behaviours were more likely to engage in persistent physical aggression, into high school and adulthood (Brame, Nagin, & Tremblay, 2001; Moffitt, Caspi, Harrington, & Milne, 2002; Nagin & Tremblay, 2001). Thus, those who become unable to regulate behavioural problems, perhaps through exposure to maladaptive parenting behaviour or maternal distress, may develop externalizing behavioural patterns that continue into adolescence. This notion of behavioural continuity is supported by research that found that high levels of externalizing behaviours, including aggression, at earlier ages predicts the later engagement in problem behaviours, including substance use (Goodman, 2010; Harachi et al., 2006; Moffitt et al., 2002; Rogosch, Oshri, & Cicchetti, 2010; Thompson et al., 2011); another study found that of childhood externalizing behaviours, physical aggression was the best predictor of later engagement in health-risk behaviours (Timmermans, Van Lier, & Koot, 2008). One study found that conduct problems during mid-childhood (age 7-9) were predictive of later substance use, abuse and dependence (Fergusson, Horwood, & Ridder, 2007). Therefore, perhaps exposure to higher levels of maternal depressive

symptomatology during mid-childhood has lasting detrimental effects, not only on delinquent behaviours, but also on the likelihood to use substances.

Parents are instrumental in the development of social, emotional, and cognitive abilities, and maternal depression has been implicated in cognitive, social, and emotional development during infancy and early toddlerhood (Cicchetti, Rogosch, Toth, & Spagnola, 1997; Gravener et al., 2012; Lyons-Ruth, Connell, Grunebaum, & Botein, 1990; Murray et al., 1999). This is unsurprising, as during childhood, the mother is able to foster such development through responsive parenting behaviours (Landry et al., 1997; Smith et al., 2006); pathways through which maternal depression exerts effects on the children is through exposure to maladaptive maternal cognitions, affect, and behaviours, as well as exposure to the stressful environmental context associated with maternal depression (Goodman & Gotlib, 1999; Goodman et al., 2011).

Mid-childhood, however, is a period of increasing cognitive, social and emotional development; during this age period, for example, children begin school, start to develop linguistic (e.g., reading and writing) skills, and begin to engage in social relationships with peers. Cognitive, social, and emotional development may be particularly relevant to the understanding of the development of both internalizing and externalizing behavioural patterns. In fact, IQ, a crude measure of cognitive ability, has been inversely linked to the development of both depression (Koenen et al., 2009; Leech, Larkby, Day, & Day, 2006) and delinquency (Moffitt, Gabrielli, Mednick, & Schulsinger, 1981; White, Moffitt, & Silva, 1989), indicating that lower cognitive capacity may be

linked to the development of internalizing and externalizing difficulties. Research has also illustrated that social-cognitive competence may improve a child's ability to regulate behavioural problems, including aggression, during mid-childhood (Hoglund, Lalonde, & Leadbeater, 2008). Whereas positive and responsive maternal behaviours, such as shared reading activity, play, or even sustaining basic positive social interactions, can foster the child's social and cognitive development during this period, many studies report decreased engagement in these behaviours, and increased negative behaviours by mothers with depression (Cox et al., 1987; Foster, Garber, et al., 2008; Lovejoy et al., 2000; McLearn et al., 2006; Murray et al., 1999).

Exposure to maternal depression, and the associated negative parenting behaviours, in the mid-childhood period, during which the child is entering a stage of increased cognitive development, may result in lasting developmental deficits, which may predispose the child to future health-risk behaviour in adolescence. Another study found that parenting competence mediated the relationship between maternal depression and child behaviour problems in 3-6 year olds; however, since this research was cross-sectional, the long-term adolescent effects of exposure at this age were not described (Gartstein & Sheeber, 2004). It is known that child behaviour problems predict future behavioural problems, including delinquency and substance use (Goodman, 2010; Harachi et al., 2006; Thompson et al., 2011), and decreased maternal engagement during this period of increased potential for cognitive growth is at least cross-sectionally associated with externalizing and internalizing difficulties (Gartstein

& Sheeber, 2004). This indicates that during this age window, children may be more sensitive to the lessened maternal engagement characteristic of maternal depression, resulting in lasting effects for adolescents.

Beyond the contributions of cognitive development during this age period, the ability of the child to properly regulate their emotions may play a role in the longitudinal development of behavioural problems. As previously outlined, most children engage in aggressive behaviour during childhood; however, those that do not learn the skills to regulate it during the pre-school years, are likely to continue along an aggressive trajectory into adolescence (Tremblay et al., 2004). Maladaptive emotional regulation strategies are a known risk factor for future externalizing and internalizing psychological disorders (Southam-Gerow & Kendall, 2002). As maladaptive emotional regulation is linked to future internalizing and externalizing difficulties (Southam-Gerow & Kendall, 2002), as well as health-risk behaviour engagement (Auerbach et al., 2010; Goldstein, 2001), perhaps exposure to maternal depression during mid-childhood affects the development of adaptive emotional regulation strategies. Considering the instrumental role of parents in social and emotional development (Bariola, Gullone, & Hughes, 2011), it is intuitive that the parental maladaptive emotional regulation strategies, including those characteristic of depression, are associated with poorer emotional development in children (Dix, 1991; Feldman, Eidelman, & Rotenberg, 2004). In fact, in children aged 4-9, low emotional regulation was associated with increased externalizing behaviours (Eisenberg, Cumberland, et al., 2001). Maternal displays of emotion also contribute to the development of

emotion regulation in children; while children of mothers who display more positive emotions tend to exhibit better emotional regulation, children of parents who express more negative emotions demonstrate poorer emotional regulation strategies (Eisenberg, Gershoff, et al., 2001; Garner & Power, 1996). Considering this evidence, it is unsurprising that infants of depressed mothers demonstrate worse emotional regulation strategies, placing them at increased risk for future internalizing and externalizing difficulties (Cicchetti et al., 1997; Feldman et al., 2009; Gravener et al., 2012; Maughan, Cicchetti, Toth, & Rogosch, 2007; Silk, Shaw, Skuban, et al., 2006). However, despite the high levels of cognitive, social, and emotional transitions from middle childhood through adolescence, there has been limited research on parental contributions to childhood emotional regulation development beyond infancy and early childhood (Bariola et al., 2011); as such, little is known about the parental role in emotional development beyond the first few years of life. Therefore, it is possible that exposure to maternal depressive symptoms, and the associated emotions, places the child at risk for maladaptive emotion regulation strategies, which has a negative life-long impact.

Furthermore, exposure to maternal depression, and the associated negative affect and behaviours, during childhood may itself be a stressor to the child (Goodman, 2007; Newport et al., 2002). The experience of stress and anxiety during childhood is a normal process, as the child must learn emotional regulation strategies to cope with future exposure to natural stressors; however, difficulties in stress management in school-aged children arise when the child has developed maladaptive emotional responses to stress, such as externalizing (e.g., aggression

or acting out) or internalizing (e.g., depression, withdrawal) behaviours (Siemon, 1978). Considering the poor emotional regulation abilities children develop in response to negative parental emotions, as well as maternal depression (Eisenberg, Gershoff, et al., 2001; Feldman et al., 2004; Feldman et al., 2009; Silk, Shaw, Forbes, et al., 2006; Silk, Shaw, Skuban, et al., 2006), it would follow that these children would respond less adaptively to the experience of stress. Thus, while these children are more likely to experience stress, they may be less equipped to properly manage it, leading to increased internalizing and externalizing behaviours in response to stress.

Longitudinal research has described the association between poor self-control in childhood (aged 3-11) and a host of adult difficulties, including poorer physical health, financial difficulties, criminal convictions, and substance dependence (Moffitt et al., 2011). Therefore, this age window may be especially crucial to learning proper self-regulation techniques, and through exposure to maternal maladaptive emotional regulation and negative parenting behaviours characteristic of depression likely leads to poorer emotional development of the child. In line with this hypothesis, one study found that current maternal depressive symptoms, when the child was 8-9, were associated with lower adaptive functioning and social competence in the child (Luoma et al., 2001). Additionally, if the proper emotional management of stress is not learned during childhood, adolescents may be more likely to engage in health-risk behaviours in response to stressful stimuli, which have a well-documented relationship with

stress (Baldwin et al., 2011; Galambos & Tilton-Weaver, 1998; Siegrist & Rodel, 2006; Sinha, 2009).

Therefore, it is likely that exposure to maternal depression, including the maladaptive parenting behaviours and associated stressors, during this period of rapidly developing cognitive, social, and emotional skills has lasting behavioural effects, which then affect the child's propensity toward externalizing and later risk-taking behaviour. Perhaps the adolescentswho were exposed to worsening maternal depressive symptoms during the early school years, and who would have been exposed to the associated negative maternal behaviour, affect, and cognitions, as well as the associated stressful context of parental depression, develop an externalizing behavioural pattern in response to social, emotional, and/or cognitive deficits. This may in turn led to a long-term trajectory of problem behaviours, including the engagement in health-risk behaviours, such as delinquency and substance use.

Considering the null findings of increased engagement in health-risk behaviour for adolescents exposed to increasing symptoms during adolescence (group 3), the results of this present study suggest a sensitive period for exposure to maternal depressive symptoms for adolescent health-risk behaviour engagement occurs during mid-childhood, before the onset of adolescence. Temporality of more recent exposure to maternal depressive symptoms during early adolescence (group 3) was not associated with increased engagement in health-risk behaviours, and thus the results are suggestive of the existence of a

sensitive period for exposure to maternal depression, with respect to delinquency and substance use.

Some recent brain imaging research may shed some light on the lack of association between adolescent exposure to maternal depression and risky behaviour engagement. Research has illustrated that during adolescence, cognitive development shifts mainly to the improvements of existing abilities, rather than the development of new cognitive capacities (Luna, Padmanabhan, & O'Hearn, 2010; Luna & Sweeney, 2004). In fact additional research found that on a task of inhibitory control, an executive cognitive function in which the individual must inhibit a dominant response, older children (aged 9-12) significantly outperformed children aged 6-8 (Williams, Ponesse, Schachar, Logan, & Tannock, 1999), suggesting that mid-childhood may be a key developmental window in the development of cognitive inhibitory executive functioning processes, which may be relevant to future tendency toward risk-behaviours.

Surprisingly, however, while adolescents in the recurrent maternal depression group only had significantly higher non-violent delinquency scores than adolescents not exposed to maternal depression; adolescents exposed to recurrent maternal depression throughout childhood did not have significantly higher factor scores on the remaining behaviours compared to adolescents not exposed to maternal depression during childhood. While this may reflect inadequate sample size in the recurrent depression group (1.64% of the sample fell into group 5), it may also reflect differences in adolescent outcomes associated with varied timing of exposure to or severity of maternal depressive

symptomatology. The adolescents who were exposed to high levels of depression in mid to late childhood, and then maternal CES-D scores lowered in adolescence, whereas the adolescents with recurrently depressed mothers were exposed to stable high mean maternal depressive symptoms. While these results illustrate some externalizing behaviour difficulties, such as non-violent delinquency, associated with recurrent maternal depression, perhaps recurrent maternal depression is more predictive of internalizing than externalizing difficulties, and may reflect slight co-occurrence of internalizing and externalizing behavioural problems during adolescence. Furthermore, the children exposed to recurrent maternal depression were also exposed to more severe depressive symptomatology, which may have had different adolescent effects than mid-childhood exposure followed by remittance (group 4). Therefore, the different significant associations with adolescent outcomes may reflect differential outcomes associated with varying experiences with maternal depression throughout childhood and early adolescence.

Finally, a few significant interactions between maternal depression group and child's gender were found. Males in the recurrently depression maternal depression trajectory (group 5) had significantly higher non-violent delinquent scores than females. Compared to males, females exposed to maternal depressive symptoms in mid-childhood had significantly lower inhalant use/sexual assault scores, and females with recurrently depressed mothers had marginally significantly lower sexual assault/inahalant use scores. These results are consistent with the literature that reports the increased likelihood for males to

engage in delinquent or externalizing behaviours than females (Chun & Mobley, 2010; Nichols et al., 2006).

4.1.4 Maternal Depression and Individual Health-risk Behaviours

The results from the logistic regressions examining the association between maternal depression trajectory group and a variety of health-risk behaviours indicated that those exposed to maternal depression were more likely to engage in certain health-risk behaviours. While these associations between exposure to maternal depressive symptoms and engagement in health-risk behaviours were intuitive with respect to previous research on maternal depression and childhood externalizing behaviour (Brennan et al., 2002; Campbell et al., 2009; Essex et al., 2003), there are a few surprising findings. Compared to adolescents without exposure to maternal depression in childhood, those exposed to high levels of maternal depressive symptomatology in mid childhood (group 4) had significantly higher odds of stealing, attacking someone with the intent of hurting them, cigarette use, drunkenness, marijuana use and hallucinogen use. These adolescents are not only more likely to engage in delinquency, but also the use of various potentially harmful substances. Adolescents exposed to increasing levels of maternal depression, peaking during late childhood again demonstrated the poorest outcomes in the logistic regression analyses. They had significantly increased odds of delinquent behaviours, both violent and not, as well as increased odds of engagement in use of substances such as cigarettes, alcohol, drinking to intoxication, marijuana and hallucinogens. As previously discussed, the adolescents in this trajectory group, exposed to increasing maternal CES-D

scores in mid-childhood, may be more likely to develop patterns of externalizing and substance use behaviour. As discussed extensively in the previous section, these childhood ages may represent a sensitive period, during which exposure to maternal depression has lasting impacts on the future engagement in substance use, and delinquency into adolescence. Furthermore, externalizing problems has been show to predict future substance use (Goodman, 2010; Harachi et al., 2006; Thompson et al., 2011), which again may indicate the development of a behavioural pattern in response to maternal depression exposure early in childhood that persists into adolescence.

Adolescents exposed to recurrent maternal depression had significantly higher odds of stealing, and destruction of property compared to adolescents not exposed to maternal depression. Thus, these adolescents had more depressive symptoms, as discussed with respect to adolescent CES-D scores, but also certain, non-violent, delinquent behaviours, including stealing and property destruction. Perhaps these children are responding to the stress associated to exposure to maternal depressive symptomatology, as well as the experience of their own internalizing problems, by acting out with externalizing delinquent behaviours. It is also possible that, as a result to the lifelong exposure to recurrent maternal depression, the attachment quality of the mother-child relationship is poor (Beardslee et al., 1998; Lyons-Ruth et al., 1990; Martins & Gaffan, 2000), which is associated with adolescent externalizing behaviour, including engagement in delinquent behaviours (Allen, Porter, McFarland, McElhaney, & Marsh, 2007; Brook et al., 1999; Gault-Sherman, 2012).

Those exposed to recurrent and higher mean levels of maternal depressive symptoms throughout childhood did not have significantly higher odds of most health-risk behaviours. Whereas most research reports that those exposed to higher recurrence/chronicity and severity of maternal depression generally demonstrate worse outcomes (Brennan et al., 2000; Campbell et al., 2009; Hammen & Brennan, 2003), it is surprising that very few associations with the various health-risk behaviours were found. These null findings were unexpected, as previous research reports those exposed to maternal depression are more likely to engage in substance use (Campbell et al., 2009) and/or externalizing behaviours (Brennan et al., 2002; Hammen & Brennan, 2003); furthermore, there is an established link between adolescent depression and health-risk behaviours activity (Brooks et al., 2002; Brown et al., 2006; Sprott et al., 2001; Waller et al., 2006). As reported by the multiple linear regression analyses, adolescents exposed to recurrent maternal depression had significantly higher mean CES-D scores compared to those not exposed to maternal depressive symptoms; furthermore, the adolescents in the recurrent maternal depressive symptomatology group had the highest mean CES-D scores (mean [SE] = 11.53 [1.74]).

Again, this may reflect an issue of limited statistical power, as this group contained only 1.64% of the sample. This limitation, coupled with decreasing sample sizes associated with missing data, discussed below in the limitations section of this chapter, may account for the non-significant results. This notion is supported by the large, yet non-significant, odds-ratios for some of the behaviours. For example, though in the fully-adjusted model for stealing, these

adolescents did not have significantly higher odds than the reference group, the odds-ratio was 2.71, indicating that these adolescents are almost 3 times as likely to steal than those not exposed to maternal depression in childhood; yet, this effect was not statistically significant. This pattern was mirrored for attacking someone with intent to cause bodily harm (OR = 2.32, p = 0.52) and destruction of property (OR = 2.13, p = 0.31). This indicates that perhaps recurrent maternal depression is associated with violent and non-violent delinquent behaviours, however, this trajectory group may have been underpowered to detect statistical significance, especially in light of missing covariate data in the progressively adjusted models, discussed further in the limitations section of this chapter. It is possible, however, that recurrent maternal depression throughout childhood is not associated with the engagement in health-risk behaviour, though the large value of the observed odds-ratios indicate it is more likely an issue of small group size.

Finally, compared to those not exposed to maternal depressive symptoms in childhood, adolescents exposed to moderate and rising levels of maternal depressive symptomatology throughout childhood (group 3) had significantly higher odds of being a passenger in a vehicle operated under the influence of alcohol and drugs, however, this significant effect was only found for the crude analysis, and adjustment for relevant covariates resulted in a loss of statistical significance. Thus, adolescents who were exposed to higher levels of maternal depression during early adolescence did not have increased odds of any health-risk behaviour beyond the crude association with being a passenger in a motor vehicle operated under the influence of drugs or alcohol. However, it is unlikely

that this reflects an issue of power, as this group contained almost twice the number of children than the mid-childhood exposure group, the group that had the most statistically significant associations with health-risk behaviours. This again suggests the existence of a critical period for exposure to maternal depression with respect to delinquent or substance use behaviour. Adolescents who were exposed in mid-childhood were more likely to engage in several health-risk behaviours than adolescents exposed to maternal depressive symptomatology in adolescence; however, the adolescents with more recent exposure to higher levels of maternal depressive symptoms (group 3) did have significantly higher depressive symptomatology themselves, and those exposed during mid-childhood (group 4), did not. Therefore, the results of this study suggest that there may be a specific age window occurring in mid-childhood, a period of increased cognitive, social and emotional development, during which exposure to maternal depressive symptoms has longitudinal implications for engagement in health-risk behaviour. This may reflect the impact of lasting cognitive or emotional regulation deficits associated with exposure to maladaptive maternal affect, behaviours, and cognitions during this age period. Furthermore, proximity to maternal depressive symptoms appears to have an effect on adolescent internalizing symptoms, but not engagement in health-risk behaviours.

4.1.5 Maternal Depression and Time-to-onset of Health-risk Behaviours

The results from the Cox regression models indicated that compared to adolescents not exposed to maternal depressive symptoms in childhood (group 1), those exposed to high levels of maternal depressive symptomatology during mid

to late childhood (group 4) were significantly more likely to engage in cigarette use, alcohol use, marijuana use, and hallucinogen use earlier. Therefore, not only are adolescents who were exposed to high maternal depressive scores in mid-childhood more likely to engage in these behaviours, they also engaged in them earlier than adolescents not exposed to maternal depression. Again, this may suggest the early development of lasting behavioural patterns, as childhood externalizing behaviours have not only been associated with increased engagement in substance use, they are also linked to earlier engagement (Thompson et al., 2011).

Adolescents exposed to rising maternal depressive symptoms during adolescence (group 3) were not significantly more likely to engage in any of the health-risk behaviours earlier than adolescents not exposed to maternal depressive symptoms. Hazard ratios for the adolescents exposed to maternal depression during adolescence remained around the null value of 1 for all adjustments and all behaviours. This indicates that earlier exposure to maternal depression, during the childhood years, is critical to the earlier engagement in health-risk behaviour, including the use of substances and engagement in sexual intercourse.

Surprisingly, adolescents exposed to recurrent maternal depressive symptomatology (group 5) were not more likely to engage in any substance use earlier than adolescents not exposed to maternal depressive symptoms during childhood. While these adolescents were almost twice as likely to engage in sexual intercourse earlier than adolescents in the reference group, this significant effect was only found in the bivariate analysis. However, the bivariate analysis

supports the association between sexual risk behaviour, in this case early age of sexual debut, and maternal depressive symptomatology. There is a large body of research reporting the association between increased depressive symptomatology and sexual risk behaviours, including earlier debut, more partners, concurrent partners, and non-use of contraception (Brooks et al., 2002; Chen, Wu, Yi, Huang, & Wong, 2008; Kosunen et al., 2003; Ramrakha, Caspi, Dickson, Moffitt, & Paul, 2000; Shrier et al., 2001; Turner et al., 2011). As there is are established links between recurrent maternal depression and adolescent depressive symptoms, the results of this present study included, and depressive symptomatology and sexual health risk, the findings that youth exposed to recurrent maternal depressive symptoms throughout childhood were twice as likely to engage in sexual intercourse earlier than youth not exposed to maternal depressive symptomatology were expected. This effect was lost upon progressive adjustment for relevant covariates; however, this may again reflect issues with statistical power and missing data, discussed further in the limitations section of this chapter.

It was unexpected, however, that youth exposed to recurrent maternal depression did not engage in substance use earlier than youth not exposed to maternal depressive symptoms. In fact, the adolescents exposed to recurrent depressive symptoms during childhood were generally less likely to use substances earlier than the reference adolescents, with hazard ratios often below the null value of 1. This effect was not statistically significant, however. These findings were not anticipated, as there is an established association between adolescent psychopathology, including internalizing disorders such as depression,

and an earlier onset of substance use (Armstrong & Costello, 2002), and the adolescents in this maternal depression trajectory group had the highest depressive symptoms in the sample. This may suggest that recurrent maternal depression is not associated with earlier engagement in substance use, or again may reflect issues with statistical power of the smallest trajectory group.

4.2 Implications and Public Health Importance

The overall results of this study indicate that exposure to high levels of maternal depressive symptoms during childhood is associated with increased engagement in health-risk behaviours, including violent and non-violent delinquency and substance use; additionally, the results are suggestive of a sensitive period of exposure to maternal depressive symptoms in the development of adolescent health-risk behaviour. Furthermore, adolescents who were exposed to the highest levels of depressive symptomatology during mid to late childhood were more likely to engage in the use several substances earlier than adolescents who were not exposed to maternal depressive symptomatology. Exposure to maternal depressive symptoms in adolescence, as well as exposure to recurrent maternal depressive symptoms throughout childhood was associated with increased adolescent depressive symptomatology at age 16-17.

These results underscore the public health importance and highlight the longitudinal consequences of childhood exposure to maternal depressive symptomatology. Increased engagement in such behaviour can present significant health risk to the adolescent. Some of these behaviours, such as fighting or

attacking someone, present immediate mortality or injury risks. Conversely, some of the health and mortality risks associated with these behaviours are more insidious, such as sexually transmitted infections from sexual risk-taking behaviour, or cancer risk from the long-term increased use of tobacco or alcohol. Teenage substance use may in turn lead to the development of adult dependence problems; research examining predictors of adult alcohol dependence found that recreational teenage alcohol consumption was the clearest predictor of later dependence (Bonomo et al., 2004). Adolescent externalizing behaviours are also associated with negative long-term outcomes, including, but not limited to, poorer adult mental health, increased financial difficulties, dissatisfaction with family life in adulthood, and future engagement in various health-risk behaviours (Colman et al., 2009; Mason et al., 2010).

Beyond the risks associated with the increased engagement in health-risk behaviour are the risks associated with earlier age of debut of these behaviours. The results of this study indicate that exposure to increased maternal depressive symptoms during mid to late childhood was associated with earlier engagement in cigarette, alcohol, marijuana, and hallucinogen use. It is a robust finding that time to debut of drug use predicts later substance abuse problem behaviours as well as future engagement in additional health-risk behaviours (Brook et al., 2004; DeWit et al., 2000; DuRant et al., 1999; Mason et al., 2010). Further hazards of early substance use may be reflected in increased suicide risk (Cho et al., 2007), which represents a significant portion of young adult and adolescent mortality (Skinner & McFaull, 2012). One study reported that in both males and females, early

substance use was associated with suicide risk factors; for males, suicidal risk factors were associated with earlier onset of illicit drug use, and in females suicidal risk was predicted by earlier onset of cigarettes, drinking to intoxication, and illicit drug use (Cho et al., 2007). Early engagement in substance use clearly contributes to additional health problems extending through to adulthood.

These behaviours are undoubtedly prevalent during adolescence; almost 20% of the sample reported weekly cigarette use, and almost 60% had drank alcohol to the point of intoxication and 10% were doing so weekly, despite the legal drinking age in Canada ranging from 18-19 across the provinces. Considering the widespread engagement in these behaviours and the associated potential long-term consequences, there is a clear need for intervention to curb the development of delinquent and substance use behaviour. Intervention efforts should focus on early identification and treatment of depressed parents in order to curb the lasting impact exposure to this illness can present (Goodman, 2007). Pregnant women, who ideally have an established rapport with a health-care practitioner, should be screened for depression or risk of future depressive episodes when seeking pre or post-natal care. It is worth noting that adequate sensitivity (0.83) and specificity (0.89) can be achieved with symptomatology scales such as the Beck Depression Inventory (Holcomb Jr, Stone, Lustman, Gavard, & Mostello, 1996), which are relatively simple and quick to administer at routine prenatal and post-natal check-ups. Considering that women who experience post-partum depression are likely to experience a recurrent depressive episode (McMahon et al., 2005; McMahon et al., 2008), it is disconcerting that

research examining a large sample of pregnant women seeking prenatal care found that while 20% of women met the depression cut-off criteria on the CES-D, less than 15% of these women were being treated for depression (Marcus et al., 2003). This reflects room for improvement in the screening and treatment of maternal depression. Furthermore, children of affectively ill parents should be screened for indicators of predisposition toward the development of psychopathology, such as measures of affect regulation or stress reactivity (Goodman, 2007).

Considering the wealth of resources illustrating the damaging effects of exposure to maternal depression during childhood on the offspring across various stages of life, attention must be focused on the development of effective interventions for families in which parental mental illness is a problem. It has been suggested that a universal approach to prevention is not the most efficacious with respect to the treatment of depression, and that prevention efforts should be focused on those at high risk for the development of psychopathology (Goodman, 2007; Goodman et al., 2011; Vinokur, Price, & Schul, 1995). The KOPP program in the Netherlands is a family-based early intervention program, focusing on the children of depressed parents, a high-risk group for the development of depression. Their results indicate that an early intervention using multiple components, including psycho-education about depressive illness, early screening, treatment and support for depressed mothers, as well as early screening of children, significantly favoured children and parents receiving the intervention over wait-list controls (Van Doesum, Riksen-Walraven, Hosman, & Hoefnagels,

2008). Additional research has reported offspring improvements during the remission of maternal depression (Foster, Webster, et al., 2008; Weissman, Pilowsky, et al., 2006).

Some research has investigated the role of therapy in the treatment of substance use and delinquency in adolescence. First, contrary to the interventions suggested to target those at higher risk to develop depression, it has been suggested that population-wide strategies may be the best intervention approach for externalizing and substance use problems (Goodman, 2010). A recent meta-analysis of 24 family-therapy interventions for delinquency and substance use found a modest but significant effect favouring the family therapy compared to treatment as usual or alternative therapies (Baldwin, Christian, Berkeljon, & Shadish, 2012). The effectiveness of family-based therapy is unsurprising, considering the bidirectional relationship between parenting and the development of health-risk behaviour patterns; parent behaviours and the parent-child relationship have been associated with externalizing behaviours, and externalizing behaviours in turn affect parenting behaviours and the quality of the relationship (DiClemente et al., 2001; Gault-Sherman, 2012; Hoeve et al., 2011; Sprott et al., 2001; Willoughby & Hamza, 2011). Furthermore, one study found that between 1999 and 2003 the availability of internal (school-based) and external (psychological services) substance-use counseling for high-school students significantly decreased over time despite stable drug-use prevalence, and inverse associations were found between use of external counseling and drug use (Terry-McElrath, Johnston, O'Malley, & Yamaguchi, 2005). The authors reflect that the

decreased availability of internal counseling represents a missed opportunity; in-school counselors could provide invaluable psychological services to students, and may be an under-used resource in reducing the engagement in health-risk behaviours. Access to such therapies, as outlined above, should be broadened in attempts to curb the intergenerational transmission of mental illness from the parents to the children, and to assuage the negative outcome of increased and earlier engagement in health-risk behaviours including delinquency and substance use.

4.3 Limitations

There were some limitations worth noting in this study. The main limitations of this research were using self-reported scales to assess depression, self-reports of health-risk behaviour engagement, study attrition, and missing data on both measures of the covariates and the outcomes.

4.3.1 Self-reported scales

First, the NLSCY uses self-reported questionnaires to determine symptoms of depression and anxiety, in lieu of a clinical diagnosis. Therefore, while this study is limited in inferential ability with respect to the clinical definition of depression, it is able to make assertions based on sub-threshold depressive symptomatology, which may more closely mirror what is observed in the general population. The depression scale used, the CES-D, was developed based on symptomatology criteria for the clinical diagnosis of depression (Radloff, 1977), and the scale demonstrated good reliability in this sample, in

both mothers and adolescents. Recent research suggests that sub-threshold depressive symptoms, which are more common in the general population than clinical depression, are related to significantly decreased health when compared to the absence of depressive symptoms (Ayuso-Mateos, Nuevo, Verdes, Naidoo, & Chatterji, 2010) as well as the development of future psychological disorder (Fergusson, Horwood, Ridder, & Beautrais, 2005) Furthermore, evidence from a meta-analyses of various studies investigating the effects of maternal depression on the offspring demonstrated no difference in effect sizes between studies using mothers clinically diagnosed with depression, and studies relying on symptomatology scales (Goodman et al., 2011). Thus, while this is a limitation with respect to inferences specific to major depressive disorder as an illness, including those with sub-threshold symptoms of depression in the sample may actually increase the generalizability of these findings, as more of the population experiences sub-threshold symptoms of depression, and these symptoms are also associated with lower overall indices of mental and physical health.

Additionally, the main outcome measure, health-risk behaviour, was self-reported by the youth and is therefore subject to potential biases, such as social desirability bias, in which responses are biased by what is perceived to be an acceptable response on a survey. However, it is more likely that these behaviours are under-reported than over-reported; since most of these behaviours are illegal, the youth may be hesitant to report illicit activity due to social desirability bias, or fear of stigmatization, judgment or punishment. Therefore the estimates of such behaviours may not be accurate, but are likely underreported, and thus the effect

of maternal depression on these behaviours in this study is likely underestimated. However, the effect may not have been underestimated if exposure to maternal depression has an effect on social desirability bias, and honest reporting on the survey is differential between the exposure groups. While the effect of maternal depression exposure on social desirability bias is unknown, it may have had an effect on the results of this research. As such, it is unclear what effect underreporting of behaviours had on the results.

In attempts to increase honest reporting of such behaviours by the youth, they were informed that their answers would remain entirely confidential, after completing the self-report questionnaires, the youth sealed their own responses in an envelope to establish a sense of confidentiality (Statistics Canada, 1995). Therefore, while the self-report of the health-risk behaviours was an obstacle in this study, it is likely that engagement was under-reported, and therefore the overall effect of maternal depression on the engagement in health-risk behaviour is likely underestimated by these findings.

4.3.2 Attrition

Another limitation of this research is the attrition rates from the NLSCY. While the sample size is large, there are large losses to follow-up that occur between Cycle 1 and Cycle 8. High rates of attrition can introduce selection bias; there is a possibility that those who did not continue the study are somehow different than those who remained in the sample. Due to budgetary constraints, Statistics Canada reduced the sample size between Cycle 1 and 2 of the NLSCY,

halting the follow-up of a total of 5,928 children in the original sample eligble for the NLSCY (Statistics Canada, 1997b); thus, the raw numbers of how many dropped out of this study makes attrition appear to be a more serious issue than it was in this study. The weighted estimate of attrition in this sample was 37.47%; attrition was significantly related to cycle 2 maternal depressive scores ($p = 0.02$), the report of stressful life events at baseline ($p = 0.05$), maternal alcohol use at baseline ($p=.01$), and baseline SES ($p = 0.04$). This may have impacted the results, as those with higher depressive scores, higher number of SLEs and alcohol use, and lower SES dropped out from the study and the adolescent engagement in health-risk behaviour for this high-risk group was not monitored. However, as those youth who were have higher risk factors for internalizing and externalizing behaviours dropped out of the study before the outcome years, the attrition rate suggests that the true association between maternal depression and health-risk behaviour engagement may be stronger than reported in this research.

4.3.3 Missing outcome data

The strength of associations between maternal depression and the engagement in health-risk behaviour may have been underestimated due to missing outcome data in the sample. Different than sample drop-out, missing outcome data relates to those who remained in the sample over the course of follow-up, but who had missing or incomplete data on the engagement in health-risk behaviours. Compared to those with complete outcome data, those with missing data were more likely to be in the older cohort (aged 4-5 in 1994; $p = 0.001$), and an inverse relationship was observed between current depression and

missing data ($p = 0.001$); those with missing outcome data reported significantly lower CES-D scores than those with complete data. However, while this effect was statistically significant, the mean adolescent CES-D score for those with complete data was 8.40 and those with incomplete data was 7.03. Thus, while this was a statistically significant difference, it only relates to a mean difference of approximately one point on the CES-D scale, on which scores ranges from 0 to 36. This may have had an effect on the reported findings, however, especially the factor analysis which was impacted by missing outcome data. Perhaps the effect was overestimated, due to those with complete data reporting higher levels of depressive symptoms. No significant associations were observed between maternal depression group, gender, SES, or SLEs and incomplete outcome data.

Missing outcome data largely impacted the sample size of the factor analysis, as factor scores could only be provided for 1632 youth, those with complete outcome data (56.56%). Secondary logistic regression analyses were performed in attempts to overcome this limitation, as regressions using item-level responses as the outcome were inclusive of more youth than the regressions utilizing factor scores. Only those with complete data were able to have a calculated factor score; therefore, large numbers of potential respondents were excluded from the multiple linear regression analyses. Examination of each type of behaviour in logistic regression models allowed more participants to be included in the analyses.

4.3.4 Missing covariate data

The observed associations between childhood exposure to maternal depressive symptomatology and the engagement in health-risk behaviours may have also been under-estimated by the presence of missing covariate data. The progressive adjustment of covariates was conducted in attempts to describe these associations in light of missing covariate data. Many observed significant observations were lost as progressive covariate adjustment was performed. While this may reflect that these covariates, such as adolescent depression or maternal alcohol use, explained the previously significant associations between childhood exposure to maternal depression and the engagement in health-risk behaviour, it is also possible that this reflects a loss of power as the number of youth included in the models dwindled. For example, in the factor analysis regression models, the initial bivariate associations contained only 1632 due to incompleteness of outcome data. In the regression models for the final adjustment including all covariates, only 1161 youth were included; missing covariate data dwindled the available sample for the final adjustment models. It is unclear what effect this had on the results, but it is possible that the loss of significance may have occurred due to missing covariate data or by adjustment for variables that potentially confounded the relationship between maternal depression and health-risk behaviours.

4.3.5 Causality

As with any observational research, causality cannot be determined from our findings. While these results suggest that exposure to maternal depressive

symptoms during childhood is associated with engagement in health-risk behaviours, it is possible that maternal depression may be a marker for another significant risk factor; for example, perhaps hostile parenting style is the main risk factor for health-risk behaviour engagement, which is associated with maternal depression but may not be a direct result of exposure to depression itself. Furthermore, the results observed could be confounded by the presence of an unmeasured factor, such as the adolescent's peer group. While these results suggest a preliminary association between maternal depression and adolescent health-risk behaviour engagement, further research will be needed to confirm and explore the observed results.

4.4 Strengths

While there were limitations to note, this research had notable methodological strength over some past research. These strengths include the use of a nationally representative cohort with a large sample size, the prospective collection of maternal depressive symptomatology, and the longitudinal nature of the data.

4.4.1 Nationally-representative prospective cohort design

A main strength of this research is the sample; the NLSCY is a nationally representative cohort including various communities across Canada, and this specific sample size was large, containing 2910 children with varying levels of exposure to maternal depressive symptoms throughout childhood. The bootstrap weights used in the regression analyses ensure the national representativeness of the sample. Thus, not only are the found associations representative of this

specific large sample, they are also generalizable to the various experiences of youth across Canada. Furthermore, the prospective collection of maternal depressive information every 2 years was another methodological strength of this research. As the information was collected prospectively, the possibility of recall bias is greatly reduced. For example, one study looking into the effect of past vs. current maternal depression on the child relied on reported history of maternal depression (Foster, Garber, et al., 2008), and while childhood exposure to maternal depression "at some point during childhood" was found to have an effect, there may be associated recall bias in being asked to remember past history. Psychiatric events, including depressive episodes, are indeed subject to recall bias (Colman & Jones, 2004); for example, one study demonstrated that regardless of age, individuals were significantly more likely to report their first depressive episode occurred within the past 5 years, illustrating that there may be difficulties recalling depressive symptomatology that occurred in the past (Simon et al., 1995). Furthermore, in this study, information was systematically collected every 2 years, allowing for comparisons of the effects of timing of exposure, and the valid establishment of trajectories of maternal depression throughout various childhood ages, which is another strength over research reporting the effects of exposure to maternal depression at some point during childhood (Foster, Garber, et al., 2008; Hammen & Brennan, 2003)

4.4.2 Longitudinal design

The longitudinal nature of this study's design allows for increased temporal inference with respect to cause and effect. While cross-sectional studies

have been able to detect an association between maternal depressive symptoms and childhood/adolescent psychopathology, they are unable to determine the temporality of the association; that is, if the maternal depression preceded the childhood/adolescent psychopathology or if the childhood/adolescent psychopathology preceded the maternal depression. Maternal depressive symptoms were prospectively collected every 2 years across the span of the sample's childhood years, and as such, we can ascertain that the experience of maternal depressive symptomatology did precede the development of health-risk behaviours in adolescence, and we are able to make assertions with respect to timing of exposure to maternal depressive symptoms. Thus, this research has increased temporal inferential ability over the cross-sectional research on maternal depression. Additionally, the longitudinal design coupled with the prospectively assessed maternal depression information allowed for the development of valid age-specific trajectory modeling of childhood exposure to maternal depressive symptoms. As discussed above with respect to recall bias, this methodological strength allows us to further investigate the impact of childhood exposure to maternal depression, without relying on reported histories, as well as increased inferential ability on age-specific effects related to childhood exposure. The establishment of maternal depressive symptom trajectories allows for the evaluation of potential sensitive periods, during which exposure to maternal depressive symptomatology may have different adolescent outcomes. Furthermore, research investigating the longitudinal associations of early-childhood exposure to maternal depression and late adolescent outcomes is

somewhat scant; therefore, this research adds to the body of longitudinal research, and supports the notion that childhood exposure to maternal depression is associated with poorer adolescent internalizing and externalizing outcomes.

4.5 Future Research

Future research should confirm and validate the associations between exposure to maternal depressive symptomatology in childhood and the engagement in various health-risk behaviours in different samples or using different adolescent ages, in attempts to confirm the existence of a sensitive period of exposure during mid-childhood for the future engagement in health-risk behaviour. Further focus on the role of maternal depressive symptoms and the role on cognitive and socio-emotional development in the sensitive age group would also be beneficial to the body of literature. As research in this field is still relatively rare, especially longitudinal research into adolescence, the findings of this study should be further investigated in other samples. Research could also longitudinally investigate whether these effects persist, or perhaps worsen, in adulthood, and could further examine the potential development and continuation of externalizing behavioural patterns from mid-childhood through adolescence. Future researchers could also examine clinical substance abuse disorders, or replicate this study design using maternal histories of clinical diagnoses of depression rather than use of self-reported symptomatology scales.

A preponderance of research has demonstrated the compelling and lasting effects of being exposed to maternal depression during childhood; some researchers have described this as a selective focus on the effects of maternal

psychopathology on the child over the contributions of the father to childhood development (Connell & Goodman, 2002). There has been a lack of focus on the exposure to paternal psychopathology, with a few studies reporting poor behavioural outcomes for children exposed to paternal depression (Dave, Sherr, Senior, & Nazareth, 2008; Fletcher, Feeman, Garfield, & Vimpani, 2011). While this study was unable to include measures of father psychopathology, in Cycle 1 of the NLSCY, 91.3% of the survey respondents were the mother and thus there was limited information about the fathers in this sample, future research should investigate the role of the father in the development of psychopathology and the engagement in health-risk behaviours in adolescence. A meta-analysis examining and comparing the effects of maternal vs. paternal depressive symptoms on the child found that while the effect of maternal psychopathology was greater on child's internalizing symptoms, the effect was equal for maternal and paternal psychology on child externalizing behaviour (Connell & Goodman, 2002). Thus, paternal mental health may have crucial, yet largely undiscovered, effects on adolescent engagement in health-risk behaviour, and future research should investigate this potential association.

Furthermore, treatments specific to children of parents suffering from mental illness need to be broadened and investigated further in the context of engagement in health-risk behaviours. Ideally, the effect of early childhood interventions, such as the aforementioned KOPP program in the Netherlands, needs to be investigated longitudinally to determine whether early intervention for affectively ill parents has lasting effect into the adolescent years or adulthood.

Furthermore, as discussed earlier, interventions led by school counselors or family-based therapy could be used in attempts to curb adolescent engagement in substance use and delinquency, and again, the long-term outcomes of such interventions on the engagement in health-risk behaviour could be documented.

4.6 Conclusion

Exposure to parental mental illness in childhood is a significant risk factor for both internalizing and externalizing difficulties throughout childhood into adolescence (Bagner et al., 2010; Beck, 1999; Brennan et al., 2000; Campbell et al., 2009; Essex et al., 2001; Fergusson et al., 1995; Fihrer et al., 2009); furthermore, the results of this study suggest that exposure to maternal depressive symptoms throughout childhood is associated with increased and earlier engagement in health-risk behaviours, including substance use, and both violent and non-violent delinquent behaviours. The results of this study suggest that a sensitive period exists during mid-childhood, during which exposure to maternal depressive symptomatology appears to have the strongest effects on adolescent engagement in delinquent and substance use behaviours. Engagement in such behaviours, which are increasingly prevalent during the adolescent years, may be associated with severe immediate or future morbidity and mortality risk, including cancer from alcohol and tobacco use (Bagnardi et al., 2001; Carbone, 1992), later psychiatric or substance abuse disorders (Armstrong & Costello, 2002; Goodman, 2010; Marmorstein & Iacono, 2003; Marmorstein et al., 2010; Windle, 1990), as well as personal harm or death, from consequences of the behaviour itself (e.g., a

vehicle collision caused by impaired driving), or from increased future suicide risk (Cho et al., 2007; Hallfors et al., 2004; Legleye et al., 2010).

Considering maternal depression is related to increased odds of engagement in health-risk behaviour, as well as earlier engagement in substance use, both of which present future risk of health problems, early identification and treatment of depressed mothers may have an impact in reducing the engagement in health-risk behaviours in adolescence, as well as assuaging the associated mortality and morbidity risks with these behaviours. Through interventions targeting children of depressed parents as a high-risk group, the public health burden associated with exposure to maternal depression, such as the negative implications of adolescent engagement in health-risk behaviours or the intergenerational transmission of psychopathology, could be significantly reduced for future generations.

References

Ahmed, S. H., & Koob, G. F. (1997). Cocaine- but not food-seeking behavior is reinstated by stress after extinction. *Psychopharmacology (Berl), 132*(3), 289-295.

Allen, J. P., Porter, M., McFarland, C., McElhaney, K. B., & Marsh, P. (2007). The Relation of Attachment Security to Adolescents' Paternal and Peer Relationships, Depression, and Externalizing Behavior. *Child Development, 78*(4), 1222-1239.

Andelic, N., Jerstad, T., Sigurdardottir, S., Schanke, A. K., Sandvik, L., & Roe, C. (2010). Effects of acute substance use and pre-injury substance abuse on traumatic brain injury severity in adults admitted to a trauma centre. *J Trauma Manag Outcomes, 4*, 6.

Andruff, H., Carraro, N., Thompson, A., & Gaudreau, P. (2009). Latent Class Growth Modelling: A tutorial. *Tutorials in Quantitative Methods for Psychology, 5*(1), 11-24.

Angold, A., & Costello, E. J. (2001). The epidemiology of depression in children and adolescents. In I. M. Goodyer (Ed.), *The depressed child and adolescent* (2 ed.). Cambridge: Cambridge University Press.

Armstrong, T. D., & Costello, E. J. (2002). Community studies on adolescent substance use, abuse, or dependence and psychiatric comorbidity. *J Consult Clin Psychol, 70*(6), 1224-1239.

Arrandale, V. H. (2006). *An evaluation of two existing methods for analyzing longitudinal respiratory symptom data.* Master of Science, University of British Columbia, Vancouver.

Ashman, S. B., Dawson, G., Panagiotides, H., Yamada, E., & Wilkinson, C. W. (2002). Stress hormone levels of children of depressed mothers. *Dev Psychopathol, 14*(2), 333-349.

Auerbach, R. P., Claro, A., Abela, J. R., Zhu, X., & Yao, S. (2010). Understanding risky behaviour engagement amongst Chinese adolescents. *Cognitive Therapy Research, 34*, 159-167.

Ayuso-Mateos, J. L., Nuevo, R., Verdes, E., Naidoo, N., & Chatterji, S. (2010). From depressive symptoms to depressive disorders: the relevance of thresholds. *Br J Psychiatry, 196*(5), 365-371.

Bagnardi, V., Blangiardo, M., La Vecchia, C., & Corrao, G. (2001). A meta-analysis of alcohol drinking and cancer risk. *Br J Cancer, 85*(11), 1700-1705.

Bagner, D. M., Pettit, J. W., Lewinsohn, P. M., & Seeley, J. R. (2010). Effect of maternal depression on child behavior: a sensitive period? *J Am Acad Child Adolesc Psychiatry, 49*(7), 699-707.

Baker, B. L., Heller, T. L., & Henker, B. (2000). Expressed emotion, parenting stress, and adjustment in mothers of young children with behavior problems. *J Child Psychol Psychiatry, 41*(7), 907-915.

Baldwin, J. A., Brown, B. G., Wayment, H. A., Nez, R. A., & Brelsford, K. M. (2011). Culture and context: buffering the relationship between stressful life events and risky behaviors in American Indian youth. *Subst Use Misuse, 46*(11), 1380-1394.

Baldwin, S. A., Christian, S., Berkeljon, A., & Shadish, W. R. (2012). The effects of family therapies for adolescent delinquency and substance abuse: a meta-analysis. *J Marital Fam Ther, 38*(1), 281-304.

Bariola, E., Gullone, E., & Hughes, E. K. (2011). Child and adolescent emotion regulation: the role of parental emotion regulation and expression. *Clin Child Fam Psychol Rev, 14*(2), 198-212.

Barnes, G. M., Welte, J. W., Hoffman, J. H., & Tidwell, M. C. (2009). Gambling, alcohol, and other substance use among youth in the United States. *J Stud Alcohol Drugs, 70*(1), 134-142.

Beardslee, W. R., Versage, E. M., & Gladstone, T. R. (1998). Children of affectively ill parents: a review of the past 10 years. *J Am Acad Child Adolesc Psychiatry, 37*(11), 1134-1141.

Beck, A. T. (1987). Cognitive models of depression. *Journal of Cognitive Psychotherapy: An International Quarterly, 1*, 5-37.

Beck, C. T. (1999). Maternal depression and child behaviour problems: a meta-analysis. *J Adv Nurs, 29*(3), 623-629.

Bonomo, Y. A., Bowes, G., Coffey, C., Carlin, J. B., & Patton, G. C. (2004). Teenage drinking and the onset of alcohol dependence: a cohort study over seven years. *Addiction, 99*(12), 1520-1528.

Brame, B., Nagin, D. S., & Tremblay, R. E. (2001). Developmental trajectories of physical aggression from school entry to late adolescence. *J Child Psychol Psychiatry, 42*(4), 503-512.

Brener, N. D., & Collins, J. L. (1998). Co-occurrence of health-risk behaviors among adolescents in the United States. *J Adolesc Health, 22*(3), 209-213.

Brennan, P. A., Hammen, C., Andersen, M. J., Bor, W., Najman, J. M., & Williams, G. M. (2000). Chronicity, severity, and timing of maternal depressive symptoms: relationships with child outcomes at age 5. *Dev Psychol, 36*(6), 759-766.

Brennan, P. A., Hammen, C., Katz, A. R., & Le Brocque, R. M. (2002). Maternal depression, paternal psychopathology, and adolescent diagnostic outcomes. *J Consult Clin Psychol, 70*(5), 1075-1085.

Brennan, P. A., Pargas, R., Walker, E. F., Green, P., Newport, D. J., & Stowe, Z. (2008). Maternal depression and infant cortisol: influences of timing, comorbidity and treatment. *J Child Psychol Psychiatry, 49*(10), 1099-1107.

Brodbeck, J., Vilen, U. L., Bachmann, M., Znoj, H., & Alsaker, F. D. (2010). Sexual risk behavior in emerging adults: gender-specific effects of hedonism, psychosocial distress, and sociocognitive variables in a 5-year longitudinal study. *AIDS Educ Prev, 22*(2), 148-159.

Brook, J. S., Adams, R. E., Balka, E. B., Whiteman, M., Zhang, C., & Sugerman, R. (2004). Illicit drug use and risky sexual behavior among African American and Puerto Rican urban adolescents: the longitudinal links. *J Genet Psychol, 165*(2), 203-220.

Brook, J. S., Balka, E. B., Crossman, A. M., Dermatis, H., Galanter, M., & Brook, D. W. (2010). The Relationship between parental alcohol use, early and

late adolescent alcohol use, and young adult psychological symptoms: a longitudinal study. *Am J Addict, 19*(6), 534-542.

Brook, J. S., Brook, D. W., De La Rosa, M., Whiteman, M., & Montoya, I. D. (1999). The role of parents in protecting Colombian adolescents from delinquency and marijuana use. *Arch Pediatr Adolesc Med, 153*(5), 457-464.

Brooks, T. L., Harris, S. K., Thrall, J. S., & Woods, E. R. (2002). Association of adolescent risk behaviors with mental health symptoms in high school students. *J Adolesc Health, 31*(3), 240-246.

Brown, A., Yung, A., Cosgrave, E., Killackey, E., Buckby, J., Stanford, C., . . . McGorry, P. (2006). Depressed mood as a risk factor for unprotected sex in young people. *Australas Psychiatry, 14*(3), 310-312.

Brunner, E., & Marmot, M. (2006). Social organization, stress and health. . In M. Marmot & R. G. Wilkinson (Eds.), *Social Determinants of Health, 2nd Ed.* (pp. 6-30). Oxford: Oxford University Press.

Byrne, D. G., & Mazanov, J. (1999). Sources of adolescent stress, smoking and the use of other drugs. *Stress Medicine, 15*, 215-227.

Campbell, S. B., Morgan-Lopez, A. A., Cox, M. J., & McLoyd, V. C. (2009). A latent class analysis of maternal depressive symptoms over 12 years and offspring adjustment in adolescence. *J Abnorm Psychol, 118*(3), 479-493.

Carbone, D. (1992). Smoking and cancer. *The American Journal of Medicine, 93*(1, Supplement 1), S13-S17.

Carrasco, M. A., & Del Barrio, M. V. (2007). Five factor model of personality as predictor of aggressive behavior in children and adolescents. *Revista de Psicopatología y Psicología Clínica, 12*, 23-32.

Centers for Disease Control and Prevention. (2012). Youth Risk Behavior Surveillance. *Morbidity and Mortality Weekly Report, 61*(4), 1-162.

Chen, K., & Kandel, D. B. (1995). The natural history of drug use from adolescence to the mid-thirties in a general population sample. *American Journal of Public Health, 85*, 41-47.

Chen, Y., Wu, J., Yi, Q., Huang, G., & Wong, T. (2008). Depression associated with sexually transmitted infection in Canada. *Sexually Transmitted Infections, 84*(7), 535-540.

Cho, H., Hallfors, D. D., & Iritani, B. J. (2007). Early initiation of substance use and subsequent risk factors related to suicide among urban high school students. *Addict Behav, 32*(8), 1628-1639.

Chun, H., & Mobley, M. (2010). Gender and grade-level comparisons in the structure of problem behaviors among adolescents. *J Adolesc, 33*(1), 197-207.

Cicchetti, D., Rogosch, F. A., Toth, S. L., & Spagnola, M. (1997). Affect, cognition, and the emergence of self-knowledge in the toddler offspring of depressed mothers. *J Exp Child Psychol, 67*(3), 338-362.

Civic, D., & Holt, V. L. (2000). Maternal depressive symptoms and child behavior problems in a nationally representative normal birthweight sample. *Matern Child Health J, 4*(4), 215-221.

Colman, I., & Jones, P. B. (2004). Birth cohort studies in psychiatry: beginning at the beginning. *Psychol Med, 34*(8), 1375-1383.

Colman, I., Murray, J., Abbott, R. A., Maughan, B., Kuh, D., Croudace, T. J., & Jones, P. B. (2009). Outcomes of conduct problems in adolescence: 40 year follow-up of national cohort. *BMJ, 338*, a2981.

Colman, I., Wadsworth, M. E., Croudace, T. J., & Jones, P. B. (2007). Forty-year psychiatric outcomes following assessment for internalizing disorder in adolescence. *Am J Psychiatry, 164*(1), 126-133.

Connell, A. M., & Goodman, S. H. (2002). The association between psychopathology in fathers versus mothers and children's internalizing and externalizing behavior problems: a meta-analysis. *Psychol Bull, 128*(5), 746-773.

Cortes, R. C., Fleming, C. B., Mason, W. A., & Catalano, R. F. (2009). Risk Factors Linking Maternal Depressed Mood to Growth in Adolescent Substance Use. *J Emot Behav Disord, 17*(1), 49-64.

Costa, P. T., & McCrae, R. R. (1987). Neuroticism, somatic complaints, and disease: Is the bark worse than the bite? *Journal of Personality, 55*, 299-316.

Cote, S. M., Vaillancourt, T., Barker, E. D., Nagin, D., & Tremblay, R. E. (2007). The joint development of physical and indirect aggression: Predictors of continuity and change during childhood. *Dev Psychopathol, 19*(1), 37-55.

Cox, A. D., Puckering, C., Pound, A., & Mills, M. (1987). The Impact of Maternal Depression in Young Children. *Journal of Child Psychology and Psychiatry, 28*(6), 917-928.

Cummings, E. M., & Davies, P. T. (1994). Maternal depression and child development. *J Child Psychol Psychiatry, 35*(1), 73-112.

Dave, S., Sherr, L., Senior, R., & Nazareth, I. (2008). Associations between paternal depression and behaviour problems in children of 4-6 years. *Eur Child Adolesc Psychiatry, 17*(5), 306-315.

Dawson, G., Ashman, S. B., Panagiotides, H., Hessl, D., Self, J., Yamada, E., & Embry, L. (2003). Preschool outcomes of children of depressed mothers: role of maternal behavior, contextual risk, and children's brain activity. *Child Dev, 74*(4), 1158-1175.

DeWit, D. J., Adlaf, E. M., Offord, D. R., & Ogborne, A. C. (2000). Age at first alcohol use: a risk factor for the development of alcohol disorders. *Am J Psychiatry, 157*(5), 745-750.

DiClemente, R. J., Wingood, G. M., Crosby, R., Sionean, C., Cobb, B. K., Harrington, K., . . . Oh, M. K. (2001). Parental monitoring: association with adolescents' risk behaviors. *Pediatrics, 107*(6), 1363-1368.

Diego, M. A., Field, T., Hernandez-Reif, M., Cullen, C., Schanberg, S., & Kuhn, C. (2004). Prepartum, postpartum, and chronic depression effects on newborns. *Psychiatry, 67*(1), 63-80.

Dix, T. (1991). The affective organization of parenting: adaptive and maladaptive processes. *Psychol Bull, 110*(1), 3-25.

Dunn, V., & Goodyer, I. M. (2006). Longitudinal investigation into childhood- and adolescence-onset depression: psychiatric outcome in early adulthood. *British Journal of Psychiatry, 188*, 216-222.

DuRant, R. H., Smith, J. A., Kreiter, S. R., & Krowchuk, D. P. (1999). The relationship between early age of onset of initial substance use and engaging in multiple health risk behaviors among young adolescents. *Arch Pediatr Adolesc Med, 153*(3), 286-291.

Edwards, V. J., Anda, R. F., Gu, D., Dube, S. R., & Felitti, V. J. (2007). Adverse childhood experiences and smoking persistence in adults with smoking-related symptoms and illness. *Perm J, 11*(2), 5-13.

Eisenberg, N., Cumberland, A., Spinrad, T. L., Fabes, R. A., Shepard, S. A., Reiser, M., . . . Guthrie, I. K. (2001). The Relations of Regulation and Emotionality to Children's Externalizing and Internalizing Problem Behavior. *Child Development, 72*(4), 1112-1134.

Eisenberg, N., Gershoff, E. T., Fabes, R. A., Shepard, S. A., Cumberland, A. J., Losoya, S. H., . . . Murphy, B. C. (2001). Mothers' emotional expressivity and children's behavior problems and social competence: mediation through children's regulation. *Dev Psychol, 37*(4), 475-490.

Ensminger, M. E., Hanson, S. G., Riley, A. W., & Juon, H. S. (2003). Maternal psychological distress: adult sons' and daughters' mental health and educational attainment. *J Am Acad Child Adolesc Psychiatry, 42*(9), 1108-1115.

Espejo, E. P., Hammen, C., & Brennan, P. A. (2012). Elevated appraisals of the negative impact of naturally occurring life events: a risk factor for depressive and anxiety disorders. *J Abnorm Child Psychol, 40*(2), 303-315.

Essex, M. J., Klein, M. H., Cho, E., & Kraemer, H. C. (2003). Exposure to maternal depression and marital conflict: gender differences in children's later mental health symptoms. *J Am Acad Child Adolesc Psychiatry, 42*(6), 728-737.

Essex, M. J., Klein, M. H., Miech, R., & Smider, N. A. (2001). Timing of initial exposure to maternal major depression and children's mental health symptoms in kindergarten. *Br J Psychiatry, 179*, 151-156.

Feldman, R., Eidelman, A. I., & Rotenberg, N. (2004). Parenting stress, infant emotion regulation, maternal sensitivity, and the cognitive development of triplets: a model for parent and child influences in a unique ecology. *Child Dev, 75*(6), 1774-1791.

Feldman, R., Granat, A., Pariente, C., Kanety, H., Kuint, J., & Gilboa-Schechtman, E. (2009). Maternal depression and anxiety across the postpartum year and infant social engagement, fear regulation, and stress reactivity. *J Am Acad Child Adolesc Psychiatry, 48*(9), 919-927.

Fergusson, D. M., Horwood, L. J., & Lynskey, M. T. (1995). Maternal depressive symptoms and depressive symptoms in adolescents. *J Child Psychol Psychiatry, 36*(7), 1161-1178.

Fergusson, D. M., Horwood, L. J., & Ridder, E. M. (2007). Conduct and attentional problems in childhood and adolescence and later substance use,

abuse and dependence: results of a 25-year longitudinal study. *Drug Alcohol Depend, 88 Suppl 1*, S14-26.

Fergusson, D. M., Horwood, L. J., Ridder, E. M., & Beautrais, A. L. (2005). Subthreshold depression in adolescence and mental health outcomes in adulthood. *Arch Gen Psychiatry, 62*(1), 66-72.

Fergusson, D. M., Woodward, L. J., & Horwood, L. J. (2000). Risk factors and life processes associated with the onset of suicidal behaviour during adolescence and early adulthood. *Psychol Med, 30*(1), 23-39.

Field, T. (1998). Maternal depression effects on infants and early interventions. *Prev Med, 27*(2), 200-203.

Fihrer, I., McMahon, C. A., & Taylor, A. J. (2009). The impact of postnatal and concurrent maternal depression on child behaviour during the early school years. *J Affect Disord, 119*(1-3), 116-123.

Fletcher, R. J., Feeman, E., Garfield, C., & Vimpani, G. (2011). The effects of early paternal depression on children's development. *Med J Aust, 195*(11-12), 685-689.

Flouri, E., & Malmberg, L. E. (2011). Gender differences in the effects of childhood psychopathology and maternal distress on mental health in adult life. *Soc Psychiatry Psychiatr Epidemiol, 46*(7), 533-542.

Foster, C. E., Webster, M. C., Weissman, M. M., Pilowsky, D. J., Wickramaratne, P. J., Talati, A., . . . King, C. A. (2008). Remission of maternal depression: relations to family functioning and youth internalizing and externalizing symptoms. *J Clin Child Adolesc Psychol, 37*(4), 714-724.

Foster, C. J., Garber, J., & Durlak, J. A. (2008). Current and past maternal depression, maternal interaction behaviors, and children's externalizing and internalizing symptoms. *J Abnorm Child Psychol, 36*(4), 527-537.

Galambos, N. L., & Tilton-Weaver, L. C. (1998). Multiple-risk behaviour in adolescents and young adults. *Health Rep, 10*(2), 9-20 (Eng); 29-21 (Fre).

Garber, J., & Cole, D. A. (2010). Intergenerational transmission of depression: a launch and grow model of change across adolescence. *Dev Psychopathol, 22*(4), 819-830.

Garner, P. W., & Power, T. G. (1996). Preschoolers' emotional control in the disappointment paradigm and its relation to temperament, emotional knowledge, and family expressiveness. *Child Dev, 67*(4), 1406-1419.

Gartstein, M. A., & Sheeber, L. (2004). Child behavior problems and maternal symptoms of depression: a mediational model. *J Child Adolesc Psychiatr Nurs, 17*(4), 141-150.

Gault-Sherman, M. (2012). It's a two-way street: the bidirectional relationship between parenting and delinquency. *J Youth Adolesc, 41*(2), 121-145.

Gavin, N. I., Gaynes, B. N., Lohr, K. N., Meltzer-Brody, S., Gartlehner, G., & Swinson, T. (2005). Perinatal depression: a systematic review of prevalence and incidence. *Obstet Gynecol, 106*(5 Pt 1), 1071-1083.

Goldstein, B. I. (2001). Coping style and attributional style as mediators of alcohol use and depression among young adults. . *Dissertation Abstracts International: Section B: The Sciences and Engineering., 62*, 2057.

Goodman, A. (2010). Substance use and common child mental health problems: examining longitudinal associations in a British sample. *Addiction, 105*(8), 1484-1496.

Goodman, S. H. (2007). Depression in mothers. *Annu Rev Clin Psychol, 3*, 107-135.

Goodman, S. H., & Gotlib, I. H. (1999). Risk for psychopathology in the children of depressed mothers: a developmental model for understanding mechanisms of transmission. *Psychological Review, 106*(3), 458-490.

Goodman, S. H., Rouse, M. H., Connell, A. M., Broth, M. R., Hall, C. M., & Heyward, D. (2011). Maternal depression and child psychopathology: a meta-analytic review. *Clin Child Fam Psychol Rev, 14*(1), 1-27.

Gotlib, I. H., & Meltzer, S. J. (1987). Depression and the Perception of Social Skill in Dyadic Interaction. *Cognitive Therapy and Research, 11*(1), 41-53.

Gotlib, I. H., & Robinson, L. A. (1982). Responses to depressed individuals: discrepancies between self-report and observer-rated behavior. *J Abnorm Psychol, 91*(4), 231-240.

Grant, K. E., Compas, B. E., Stuhlmacher, A. F., Thurm, A. E., McMahon, S. D., & Halpert, J. A. (2003). Stressors and child and adolescent psychopathology: moving from markers to mechanisms of risk. *Psychol Bull, 129*(3), 447-466.

Gravener, J. A., Rogosch, F. A., Oshri, A., Narayan, A. J., Cicchetti, D., & Toth, S. L. (2012). The Relations among Maternal Depressive Disorder, Maternal Expressed Emotion, and Toddler Behavior Problems and Attachment. *J Abnorm Child Psychol, 40*(5), 803-813.

Greenberg, P. E., Kessler, R. C., Birnbaum, H. G., Leong, S. A., Lowe, S. W., Berglund, P. A., & Corey-Lisle, P. K. (2003). The economic burden of depression in the United States: How did it change between 1990 and 2000? [Article]. *Journal of Clinical Psychiatry, 64*(12), 1465-1475.

Gross, D., Conrad, B., Fogg, L., Willis, L., & Garvey, C. (1995). A longitudinal study of maternal depression and preschool children's mental health. *Nurs Res, 44*(2), 96-101.

Hallfors, D. D., Waller, M. W., Ford, C. A., Halpern, C. T., Brodish, P. H., & Iritani, B. (2004). Adolescent depression and suicide risk: association with sex and drug behavior. *Am J Prev Med, 27*(3), 224-231.

Halligan, S. L., Herbert, J., Goodyer, I. M., & Murray, L. (2004). Exposure to postnatal depression predicts elevated cortisol in adolescent offspring. *Biol Psychiatry, 55*(4), 376-381.

Halligan, S. L., Murray, L., Martins, C., & Cooper, P. J. (2007). Maternal depression and psychiatric outcomes in adolescent offspring: a 13-year longitudinal study. *J Affect Disord, 97*(1-3), 145-154.

Hammen, C. (1991). Generation of stress in the course of unipolar depression. *J Abnorm Psychol, 100*(4), 555-561.

Hammen, C. (2005). Stress and depression. *Annu Rev Clin Psychol, 1*, 293-319.

Hammen, C. (2006). Stress generation in depression: reflections on origins, research, and future directions. *J Clin Psychol, 62*(9), 1065-1082.

Hammen, C., & Brennan, P. A. (2001). Depressed adolescents of depressed and nondepressed mothers: tests of an interpersonal impairment hypothesis. *J Consult Clin Psychol, 69*(2), 284-294.

Hammen, C., & Brennan, P. A. (2003). Severity, chronicity, and timing of maternal depression and risk for adolescent offspring diagnoses in a community sample. *Archives of General Psychiatry, 60*(3), 253-258.

Hammen, C., Brennan, P. A., & Keenan-Miller, D. (2008). Patterns of adolescent depression to age 20: the role of maternal depression and youth interpersonal dysfunction. *J Abnorm Child Psychol, 36*(8), 1189-1198.

Hammen, C., Brennan, P. A., & Shih, J. H. (2004). Family discord and stress predictors of depression and other disorders in adolescent children of depressed and nondepressed women. *J Am Acad Child Adolesc Psychiatry, 43*(8), 994-1002.

Hammen, C., Hazel, N. A., Brennan, P. A., & Najman, J. (2012). Intergenerational transmission and continuity of stress and depression: depressed women and their offspring in 20 years of follow-up. *Psychol Med, 42*(5), 931-942.

Hammen, C., Shih, J., Altman, T., & Brennan, P. A. (2003). Interpersonal impairment and the prediction of depressive symptoms in adolescent children of depressed and nondepressed mothers. *J Am Acad Child Adolesc Psychiatry, 42*(5), 571-577.

Hammen, C., Shih, J. H., & Brennan, P. A. (2004). Intergenerational transmission of depression: test of an interpersonal stress model in a community sample. *J Consult Clin Psychol, 72*(3), 511-522.

Hankin, B. L. (2008a). Cognitive vulnerability-stress model of depression during adolescence: investigating depressive symptom specificity in a multi-wave prospective study. *J Abnorm Child Psychol, 36*(7), 999-1014.

Hankin, B. L. (2008b). Stability of cognitive vulnerabilities to depression: a short-term prospective multiwave study. *J Abnorm Psychol, 117*(2), 324-333.

Harachi, T. W., Fleming, C. B., White, H. R., Ensminger, M. E., Abbott, R. D., Catalano, R. F., & Haggerty, K. P. (2006). Aggressive behavior among girls and boys during middle childhood: predictors and sequelae of trajectory group membership. *Aggressive Behavior, 32*(4), 279-293.

Hatch, S. L., Jones, P. B., Kuh, D., Hardy, R., Wadsworth, M. E., & Richards, M. (2007). Childhood cognitive ability and adult mental health in the British 1946 birth cohort. *Soc Sci Med, 64*(11), 2285-2296.

Hawkins, J. D., Catalano, R. F., & Miller, J. Y. (1992). Risk and protective factors for alcohol and other drug problems in adolescence and early adulthood: implications for substance abuse prevention. *Psychol Bull, 112*(1), 64-105.

Hoeve, M., Dubas, J. S., Gerris, J. R., van der Laan, P. H., & Smeenk, W. (2011). Maternal and paternal parenting styles: unique and combined links to adolescent and early adult delinquency. *J Adolesc, 34*(5), 813-827.

Hoglund, W. L. G., Lalonde, C. E., & Leadbeater, B. J. (2008). Social-cognitive Competence, Peer Rejection and Neglect, and Behavioral and Emotional Problems in Middle Childhood. *Social Development, 17*(3), 528-553.

Holcomb Jr, W. L., Stone, L. S., Lustman, P. J., Gavard, J. A., & Mostello, D. J. (1996). Screening for depression in pregnancy: Characteristics of the Beck Depression Inventory. *Obstetrics & Gynecology, 88*(6), 1021-1025.

Horwitz, S. M., Briggs-Gowan, M. J., Storfer-Isser, A., & Carter, A. S. (2009). Persistence of Maternal Depressive Symptoms throughout the Early Years of Childhood. *J Womens Health (Larchmt), 18*(5), 637-645.

Huizink, A. C., Ferdinand, R. F., van der Ende, J., & Verhulst, F. C. (2006). Symptoms of anxiety and depression in childhood and use of MDMA: prospective, population based study. *BMJ, 332*(7545), 825-828.

Igra, V., & Irwin, C. E. (1996). Theories of Adolescent Risk-Taking Behavior. In R. J. DiClemente, W. B. Hansen & L. E. Ponton (Eds.), *Handbook of Adolescent Health Risk Behavior*. New York: Plenum Press.

Jaenicke, C., Hammen, C., Zupan, B., Hiroto, D., Gordon, D., Adrian, C., & Burge, D. (1987). Cognitive vulnerability in children at risk for depression. *J Abnorm Child Psychol, 15*(4), 559-572.

Johnston, L. D., O'Malley, P. M., Bachman, J. G., & Schulenberg, J. E. (2005). Teen drug use down but progress halts among youngest teens. *Journal of Human Behavior in the Social Environment, 38*, 1-27.

Jones, B. L., Nagin, D. S., & Roeder, K. (2001). A SAS procedure based on mixture models for estimating developmental trajectories. *Sociological Methods & Research, 29*(3), 374-393.

Joormann, J., & Gotlib, I. H. (2007). Selective attention to emotional faces following recovery from depression. *J Abnorm Psychol, 116*(1), 80-85.

Katon, W., Richardson, L., Russo, J., McCarty, C. A., Rockhill, C., McCauley, E., . . . Grossman, D. C. (2010). Depressive symptoms in adolescence: the association with multiple health risk behaviors. *Gen Hosp Psychiatry, 32*(3), 233-239.

Kegeles, S. M., Adler, N. E., & Irwin, C. E., Jr. (1988). Sexually active adolescents and condoms: changes over one year in knowledge, attitudes and use. *Am J Public Health, 78*(4), 460-461.

Kendler, K. S., Kessler, R. C., Walters, E. E., Maclean, C., Neale, M. C., Heath, A. C., & Eaves, L. J. (1995). Stressful Life Events, Genetic Liability, and Onset of an Episode of Major Depression in Women. *American Journal of Psychiatry, 152*(6), 833-842.

Kessler, R. C., & Walters, E. E. (1998). Epidemiology of DSM-III-R major depression and minor depression among adolescents and young adults in the National Comorbidity Survey. *Depress Anxiety, 7*(1), 3-14.

Kim-Cohen, J., Moffitt, T. E., Taylor, A., Pawlby, S. J., & Caspi, A. (2005). Maternal depression and children's antisocial behavior: nature and nurture effects. *Archives of General Psychiatry, 62*(2), 173-181.

Klimes-Dougan, B., & Bolger, A. (1998). Coping with maternal depressed affect and depression: adolescent children of depressed and well mothers. *Journal of Youth and Adolescence, 27*(1), 1-15.

Koenen, K. C., Moffitt, T. E., Roberts, A. L., Martin, L. T., Kubzansky, L., Harrington, H., . . . Caspi, A. (2009). Childhood IQ and Adult Mental

Disorders: A Test of the Cognitive Reserve Hypothesis. *American Journal of Psychiatry, 166*(1), 50-57

138.

Kofler, M. J., McCart, M. R., Zajac, K., Ruggiero, K. J., Saunders, B. E., & Kilpatrick, D. G. (2011). Depression and delinquency covariation in an accelerated longitudinal sample of adolescents. *J Consult Clin Psychol, 79*(4), 458-469.

Koob, G. F., & Le Moal, M. (1997). Drug abuse: hedonic homeostatic dysregulation. *Science, 278*(5335), 52-58.

Kosunen, E., Kaltiala-Heino, R., Rimpela, M., & Laippala, P. (2003). Risk-taking sexual behaviour and self-reported depression in middle adolescence--a school-based survey. *Child Care Health Dev, 29*(5), 337-344.

Kovacs, M., Akiskal, H. S., Gatsonis, C., & Parrone, P. L. (1994). Childhood-onset dysthymic disorder. Clinical features and prospective naturalistic outcome. *Arch Gen Psychiatry, 51*(5), 365-374.

Kovacs, M., Devlin, B., Pollock, M., Richards, C., & Mukerji, P. (1997). A controlled family history study of childhood-onset depressive disorder. *Arch Gen Psychiatry, 54*(7), 613-623.

Kuehner, C. (2003). Gender differences in unipolar depression: an update of epidemiological findings and possible explanations. *Acta Psychiatr Scand, 108*(3), 163-174.

Landry, S. H., Smith, K. E., Miller-Loncar, C. L., & Swank, P. R. (1997). Predicting cognitive-language and social growth curves from early maternal behaviors in children at varying degrees of biological risk. *Dev Psychol, 33*(6), 1040-1053.

Leech, S. L., Larkby, C. A., Day, R., & Day, N. L. (2006). Predictors and Correlates of High Levels of Depression and Anxiety Symptoms Among Children at Age 10. *J Am Acad Child Adolesc Psychiatry, 45*(2), 223-230.

Legleye, S., Beck, F., Peretti-Watel, P., Chau, N., & Firdion, J. M. (2010). Suicidal ideation among young French adults: association with occupation, family, sexual activity, personal background and drug use. *J Affect Disord, 123*(1-3), 108-115.

Letourneau, N., Salmani, M., & Duffett-Leger, L. (2010). Maternal depressive symptoms and parenting of children from birth to 12 years. *West J Nurs Res, 32*(5), 662-685.

Levinson, D. F. (2006). The genetics of depression: a review. *Biol Psychiatry, 60*(2), 84-92.

Lewinsohn, P. M., Clarke, G. N., Seeley, J. R., & Rohde, P. (1994). Major depression in community adolescents: age at onset, episode duration, and time to recurrence. *J Am Acad Child Adolesc Psychiatry, 33*(6), 809-818.

Lewinsohn, P. M., Olino, T. M., & Klein, D. N. (2005). Psychosocial impairment in offspring of depressed parents. *Psychol Med, 35*(10), 1493-1503.

Lewis, G., Rice, F., Harold, G. T., Collishaw, S., & Thapar, A. (2011). Investigating environmental links between parent depression and child depressive/anxiety symptoms using an assisted conception design. *J Am Acad Child Adolesc Psychiatry, 50*(5), 451-459 e451.

Lieb, R., Merikangas, K. R., Hofler, M., Pfister, H., Isensee, B., & Wittchen, H. U. (2002). Parental alcohol use disorders and alcohol use and disorders in offspring: a community study. *Psychol Med, 32*(1), 63-78.

Liu, R. T., & Alloy, L. B. (2010). Stress generation in depression: A systematic review of the empirical literature and recommendations for future study. *Clin Psychol Rev, 30*(5), 582-593.

Lorant, V., Deliege, D., Eaton, W., Robert, A., Philippot, P., & Ansseau, M. (2003). Socioeconomic inequalities in depression: a meta-analysis. *Am J Epidemiol, 157*(2), 98-112.

Lovejoy, M. C., Graczyk, P. A., O'Hare, E., & Neuman, G. (2000). Maternal depression and parenting behavior: a meta-analytic review. *Clin Psychol Rev, 20*(5), 561-592.

Luna, B., Padmanabhan, A., & O'Hearn, K. (2010). What has fMRI told us about the development of cognitive control through adolescence? *Brain Cogn, 72*(1), 101-113.

Luna, B., & Sweeney, J. A. (2004). The emergence of collaborative brain function: FMRI studies of the development of response inhibition. *Ann N Y Acad Sci, 1021*, 296-309.

Luoma, I., Tamminen, T., Kaukonen, P., Laippala, P., Puura, K., Salmelin, R., & Almqvist, F. (2001). Longitudinal study of maternal depressive symptoms and child well-being. *J Am Acad Child Adolesc Psychiatry, 40*(12), 1367-1374.

Luthar, S. S., & Sexton, C. C. (2007). Maternal drug abuse versus maternal depression: vulnerability and resilience among school-age and adolescent offspring. *Dev Psychopathol, 19*(1), 205-225.

Lynskey, M. T., Fergusson, D. M., & Horwood, L. J. (1998). The origins of the correlations between tobacco, alcohol, and cannabis use during adolescence. *J Child Psychol Psychiatry, 39*(7), 995-1005.

Lyons-Ruth, K., Connell, D. B., Grunebaum, H. U., & Botein, S. (1990). Infants at Social Risk - Maternal Depression and Family Support Services as Mediators of Infant Development and Security of Attachment. *Child Development, 61*(1), 85-98.

Marcus, S. M., Flynn, H. A., Blow, F. C., & Barry, K. L. (2003). Depressive symptoms among pregnant women screened in obstetrics settings. *J Womens Health (Larchmt), 12*(4), 373-380.

Marmorstein, N. R., & Iacono, W. G. (2003). Major depression and conduct disorder in a twin sample: Gender, functioning, and risk for future psychopathology. *Journal of the American Academy of Child and Adolescent Psychiatry, 42*(2), 225-233.

Marmorstein, N. R., Iacono, W. G., & Malone, S. M. (2010). Longitudinal associations between depression and substance dependence from adolescence through early adulthood. *Drug Alcohol Depend, 107*(2-3), 154-160.

Marmorstein, N. R., Iacono, W. G., & McGue, M. (2009). Alcohol and illicit drug dependence among parents: associations with offspring externalizing disorders. *Psychol Med, 39*(1), 149-155.

Martins, C., & Gaffan, E. A. (2000). Effects of early maternal depression on patterns of infant-mother attachment: a meta-analytic investigation. *J Child Psychol Psychiatry, 41*(6), 737-746.

Mason, W. A., Hitch, J. E., Kosterman, R., McCarty, C. A., Herrenkohl, T. I., & Hawkins, J. D. (2010). Growth in adolescent delinquency and alcohol use in relation to young adult crime, alcohol use disorders, and risky sex: a comparison of youth from low- versus middle-income backgrounds. *J Child Psychol Psychiatry, 51*(12), 1377-1385.

Maughan, A., Cicchetti, D., Toth, S. L., & Rogosch, F. A. (2007). Early-occurring maternal depression and maternal negativity in predicting young children's emotion regulation and socioemotional difficulties. *J Abnorm Child Psychol, 35*(5), 685-703.

McKenzie, M., Olsson, C. A., Jorm, A. F., Romaniuk, H., & Patton, G. C. (2010). Association of adolescent symptoms of depression and anxiety with daily smoking and nicotine dependence in young adulthood: findings from a 10-year longitudinal study. *Addiction, 105*(9), 1652-1659.

McLearn, K. T., Minkovitz, C. S., Strobino, D. M., Marks, E., & Hou, W. (2006). Maternal depressive symptoms at 2 to 4 months post partum and early parenting practices. *Arch Pediatr Adolesc Med, 160*(3), 279-284.

McMahon, C., Barnett, B., Kowalenko, N., & Tennant, C. (2005). Psychological factors associated with persistent postnatal depression: past and current relationships, defence styles and the mediating role of insecure attachment style. *J Affect Disord, 84*(1), 15-24.

McMahon, C., Trapolini, T., & Barnett, B. (2008). Maternal state of mind regarding attachment predicts persistence of postnatal depression in the preschool years. *J Affect Disord, 107*(1-3), 199-203.

Mehta, D., Quast, C., Fasching, P. A., Seifert, A., Voigt, F., Beckmann, M. W., . . . Goecke, T. W. (2012). The 5-HTTLPR polymorphism modulates the influence on environmental stressors on peripartum depression symptoms. *J Affect Disord, 136*(3), 1192-1197.

Melchior, M., Choquet, M., Le Strat, Y., Hassler, C., & Gorwood, P. (2011). Parental alcohol dependence, socioeconomic disadvantage and alcohol and cannabis dependence among young adults in the community. *Eur Psychiatry, 26*(1), 13-17.

Mitte, K. (2007). Anxiety and risky decision-making: The role of subjective probability and subjective costs of negative events. *Personality and Individual Differences, 43*, 243-253.

Moffitt, T. E., Arseneault, L., Belsky, D., Dickson, N., Hancox, R. J., Harrington, H., . . . Caspi, A. (2011). A gradient of childhood self-control predicts health, wealth, and public safety. *Proc Natl Acad Sci U S A, 108*(7), 2693-2698.

Moffitt, T. E., Caspi, A., Harrington, H., & Milne, B. J. (2002). Males on the life-course-persistent and adolescence-limited antisocial pathways: follow-up at age 26 years. *Dev Psychopathol, 14*(1), 179-207.

Moffitt, T. E., Gabrielli, W. F., Mednick, S. A., & Schulsinger, F. (1981). Socioeconomic status, IQ, and delinquency. *J Abnorm Psychol, 90*(2), 152-156.

Murray, A. D., & Hornbaker, A. V. (1997). Maternal directive and facilitative interaction styles: associations with language and cognitive development of low risk and high risk toddlers. *Dev Psychopathol, 9*(3), 507-516.

Murray, L., Sinclair, D., Cooper, P., Ducournau, P., Turner, P., & Stein, A. (1999). The socioemotional development of 5-year-old children of postnatally depressed mothers. *J Child Psychol Psychiatry, 40*(8), 1259-1271.

Muula, A. S., Siziya, S., & Rudatsikira, E. (2011). Prevalence and socio-demographic correlates for serious injury among adolescents participating in the Djibouti 2007 Global School-based Health Survey. *BMC Res Notes, 4*, 372.

Nagin, D. S., & Tremblay, R. E. (2001). Parental and early childhood predictors of persistent physical aggression in boys from kindergarten to high school. *Arch Gen Psychiatry, 58*(4), 389-394.

Naicker, K., Wickham, M., & Colman, I. (2012). Timing of First Exposure to Maternal Depression and Adolescent Emotional Disorder in a National Canadian Cohort. . *PLoS One, 7*(3).

Newport, D. J., Wilcox, M. M., & Stowe, Z. N. (2002). Maternal depression: a child's first adverse life event. *Semin Clin Neuropsychiatry, 7*(2), 113-119.

Nichols, T. R., Graber, J. A., Brooks-Gunn, J., & Botvin, G. J. (2006). Sex differences in overt aggression and deliquency among urban minority middle school students. *Journal of Applied Developmental Psychology, 27*(1), 78-91.

Nolen-Hoeksema, S., & Hilt, L. (2008). *Handbook of Depression in Adolescents*. New York: Routledge.

Pan, S. Y., Desmeules, M., Morrison, H., Semenciw, R., Ugnat, A. M., Thompson, W., & Mao, Y. (2007). Adolescent injury deaths and hospitalization in Canada: magnitude and temporal trends (1979-2003). *J Adolesc Health, 41*(1), 84-92.

Paxton, R. J., Valois, R. F., Watkins, K. W., Huebner, E. S., & Drane, J. W. (2007). Associations between depressed mood and clusters of health risk behaviors. *Am J Health Behav, 31*(3), 272-283.

Pilowsky, D. J., Wickramaratne, P. J., Rush, A. J., Hughes, C. W., Garber, J., Malloy, E., . . . Weissman, M. M. (2006). Children of currently depressed mothers: a STAR*D ancillary study. *J Clin Psychiatry, 67*(1), 126-136.

Poulin, C., Hand, D., & Boudreau, B. (2005). Validity of a 12-item version of the CES-D used in the National Longitudinal Study of Children and Youth. *Chronic Dis Can, 26*(2-3), 65-72.

Prinstein, M. J., Boergers, J., & Spirito, A. (2001). Adolescents' and their friends' health-risk behavior: factors that alter or add to peer influence. *J Pediatr Psychol, 26*(5), 287-298.

Radloff, L. S. (1977). The CES-D Scale: a self-report depression scale for research in the general population. *Journal of Applied Psychological Measurement 1*, 385-401.

Ramrakha, S., Caspi, A., Dickson, N., Moffitt, T. E., & Paul, C. (2000). Psychiatric disorders and risky sexual behaviour in young adulthood: cross sectional study in birth cohort. *BMJ, 321*(7256), 263-266.

Redonnet, B., Chollet, A., Fombonne, E., Bowes, L., & Melchior, M. (2012). Tobacco, alcohol, cannabis and other illegal drug use among young adults: the socioeconomic context. *Drug Alcohol Depend, 121*(3), 231-239.

Rogosch, F. A., Oshri, A., & Cicchetti, D. (2010). From child maltreatment to adolescent cannabis abuse and dependence: a developmental cascade model. *Dev Psychopathol, 22*(4), 883-897.

Rohde, P., Lewinsohn, P. M., Klein, D. N., & Seeley, J. R. (2005). Association of parental depression with psychiatric course from adolescence to young adulthood among formerly depressed individuals. *J Abnorm Psychol, 114*(3), 409-420.

Sells, C. W., & Blum, R. W. (1996). Morbidity and mortality among US adolescents: An overview of data and trends. *Am J Public Health, 86*(4), 513-519.

Shafii, M., Steltz-Lenarsky, J., Derrick, A. M., Beckner, C., & Whittinghill, J. R. (1988). Comorbidity of mental disorders in the post-mortem diagnosis of completed suicide in children and adolescents. *J Affect Disord, 15*(3), 227-233.

Shalev, U., Erb, S., & Shaham, Y. (2010). Role of CRF and other neuropeptides in stress-induced reinstatement of drug seeking. *Brain Res, 1314*, 15-28.

Shrier, L. A., Harris, S. K., Sternberg, M., & Beardslee, W. R. (2001). Associations of depression, self-esteem, and substance use with sexual risk among adolescents. *Preventive Medicine, 33*(3), 179-189.

Siegrist, J., & Rodel, A. (2006). Work stress and health risk behavior. *Scand J Work Environ Health, 32*(6), 473-481.

Siemon, M. (1978). Mental health in school-aged children. *MCN Am J Matern Child Nurs, 3*(4), 211-217.

Silk, J. S., Shaw, D. S., Forbes, E. E., Lane, T. L., & Kovacs, M. (2006). Maternal depression and child internalizing: the moderating role of child emotion regulation. *J Clin Child Adolesc Psychol, 35*(1), 116-126.

Silk, J. S., Shaw, D. S., Skuban, E. M., Oland, A. A., & Kovacs, M. (2006). Emotion regulation strategies in offspring of childhood-onset depressed mothers. *J Child Psychol Psychiatry, 47*(1), 69-78.

Silverstein, M., Augustyn, M., Young, R., & Zuckerman, B. (2009). The relationship between maternal depression, in-home violence and use of physical punishment: what is the role of child behaviour? *Arch Dis Child, 94*(2), 138-143.

Simon, G. E., VonKorff, M., Ustun, T. B., Gater, R., Gureje, O., & Sartorius, N. (1995). Is the lifetime risk of depression actually increasing? *J Clin Epidemiol, 48*(9), 1109-1118.

Sinha, R. (2009). Modeling stress and drug craving in the laboratory: implications for addiction treatment development. *Addict Biol, 14*(1), 84-98.

Sinha, R., Shaham, Y., & Heilig, M. (2011). Translational and reverse translational research on the role of stress in drug craving and relapse. *Psychopharmacology (Berl), 218*(1), 69-82.

Skinner, R., & McFaull, S. (2012). Suicide among children and adolescents in Canada: trends and sex differences, 1980-2008. *Canadian Medical Association Journal*.

Smith, K. E., Landry, S. H., & Swank, P. R. (2006). The role of early maternal responsiveness in supporting school-aged cognitive development for children who vary in birth status. *Pediatrics, 117*(5), 1608-1617.

Smucker, M. R., Craighead, W. E., Craighead, L. W., & Green, B. J. (1986). Normative and reliability data for the Children's Depression Inventory. . *Journal of Abnormal Child Psychology, 14*, 25-40.

Socie, E., Duffy, R. E., & Erskine, T. (2012). Subtance use and type and severity of injury among hospitalized trauma cases: Ohio, 2004-2007. *Journal of Studies on Alcohol & Drugs, 73*(2), 260-267.

Somers, M. A., & Willms, J. D. (2002). Maternal Depression and Childhood Vulnerability. In J. D. Willms (Ed.), *Vulnerable Children: Findings from Canada's national longitudinal survey of children and youth* (pp. 211-228). Edmonton: University of Alberta Press and Human Resources.

Southam-Gerow, M. A., & Kendall, P. C. (2002). Emotion regulation and understanding: implications for child psychopathology and therapy. *Clin Psychol Rev, 22*(2), 189-222.

Sprott, J. B., Doob, A. N., & Jenkins, J. M. (2001). Problem Behaviour and Delinquency in Children and Youth. *Statisitcs Canada Catalogue no. 85-002-XPE, 21*(4), 1-13.

Statistics Canada. (1995). *National Longitudinal Survey of Children and Youth: Microdata User Guide - Cycle 1*. Ottawa.

Statistics Canada. (1997a). *National Longitudinal Survey of Children and Youth: Microdata User Guide - Cycle 2.* Ottawa.

Statistics Canada. (1997b). *National Longitudinal Survey of Children and Youth: Overview of Survey Instruments for 1996-1997 Data Collection - Cycle 2.* Ottawa, Ontario.

Statistics Canada. (2003). *National Longitudinal Survey of Children and Youth: Microdata User Guide - Cycle 5.* Ottawa.

Statistics Canada. (2005). *National Longitudinal Survey of Children and Youth: Microdata User Guide - Cycle 6.* Ottawa.

Statistics Canada. (2007). *National Longitudinal Survey of Children and Youth: Microdata User Guide - Cycle 7.* Ottawa.

Statistics Canada. (2009). *National Longitudinal Survey of Children and Youth: Microdata User Guide- Cycle 8.* Ottawa.

Teicher, M. H., Andersen, S. L., Polcari, A., Anderson, C. M., Navalta, C. P., & Kim, D. M. (2003). The neurobiological consequences of early stress and childhood maltreatment. *Neurosci Biobehav Rev, 27*(1-2), 33-44.

Terry-McElrath, Y. M., Johnston, L. D., O'Malley, P. M., & Yamaguchi, R. (2005). Substance abuse counseling services in secondary schools: a national study of schools and students, 1999-2003. *J Sch Health, 75*(9), 334-341.

Thompson, R., Jiyoung Kim Tabone, Litrownik, A. J., Briggs, E. C., Hussey, J. M., English, D. J., & Dubowitz, H. (2011). Early Adolescent Risk Behavior Outcomes of Childhood Externalizing Behavioral Trajectories. *The Journal of Early Adolescence, 31*(2), 234-257.

Timmermans, M., Van Lier, P. A. C., & Koot, H. M. (2008). Which forms of child/adolescent externalizing behaviors account for late adolescent risky sexual behavior and substance use? *Journal of Child Psychology and Psychiatry, 49*(4), 386-394.

Trainer, P. J. (2002). Corticosteroids and pregnancy. *Semin Reprod Med, 20*(4), 375-380.

Tremblay, R. E. (2002). Prevention of injury by early socialization of aggressive behavior. *Inj Prev, 8 Suppl 4*, IV17-21.

Tremblay, R. E., Nagin, D. S., Seguin, J. R., Zoccolillo, M., Zelazo, P. D., Boivin, M., . . . Japel, C. (2004). Physical aggression during early childhood: trajectories and predictors. *Pediatrics, 114*(1), e43-50.

Turner, A. K., Latkin, C., Sonenstein, F., & Tandon, S. D. (2011). Psychiatric disorder symptoms, substance use, and sexual risk behavior among African-American out of school youth. *Drug Alcohol Depend, 115*(1-2), 67-73.

Van Doesum, K. T. M., Riksen-Walraven, J. M., Hosman, C. M. H., & Hoefnagels, C. (2008). A Randomized Controlled Trial of a Home-Visiting Intervention Aimed at Preventing Relationship Problems in Depressed Mothers and Their Infants. *Child Development, 79*(3), 547-561.

Vinokur, A., Price, R., & Schul, Y. (1995). Impact of the JOBS intervention on unemployed workers varying in risk for depression. *American Journal of Community Psychology, 23*(1), 39-74.

Wallace, B. C. (1989). Psychological and environmental determinants of relapse in crack cocaine smokers. *J Subst Abuse Treat, 6*(2), 95-106.

Wallace, J. M., Bachman, J. G., O'Malley, P. M., Schulenberg, J. E., Cooper, S. M., & Johnston, L. D. (2003). Gender and ethnic differences in smoking, drinking and illicit drug use among American 8th, 10th and 12th grade students, 1976-2000. *Addiction, 98*(2), 225-234.

Waller, M. W., Hallfors, D. D., Halpern, C. T., Iritani, B. J., Ford, C. A., & Guo, G. (2006). Gender differences in associations between depressive symptoms and patterns of substance use and risky sexual behavior among a nationally representative sample of U.S. adolescents. *Arch Womens Ment Health, 9*(3), 139-150.

Wang, P. S., Simon, G., & Kessler, R. C. (2003). The economic burden of depression and the cost-effectiveness of treatment. *International Journal of Methods in Psychiatric Research, 12*(1), 22-33.

Weissman, M. M., Bland, R. C., Canino, G. J., Faravelli, C., Greenwald, S., Hwu, H. G., . . . Yeh, E. K. (1996). Cross-national epidemiology of major depression and bipolar disorder. *JAMA, 276*(4), 293-299.

Weissman, M. M., Pilowsky, D. J., Wickramaratne, P. J., Talati, A., Wisniewski, S. R., Fava, M., . . . for the STAR*D-Child Team. (2006). Remissions in Maternal Depression and Child Psychopathology: A STAR*D-Child Report. *JAMA, 295*(12), 1389-1398.

Weissman, M. M., Warner, V., Wickramaratne, P., Moreau, D., & Olfson, M. (1997). Offspring of depressed parents. 10 Years later. *Arch Gen Psychiatry, 54*(10), 932-940.

Weissman, M. M., Wickramaratne, P., Nomura, Y., Warner, V., Pilowsky, D., & Verdeli, H. (2006). Offspring of Depressed Parents: 20 Years Later. *American Journal of Psychiatry, 163*(6), 1001-1008.

White, J. L., Moffitt, T. E., & Silva, P. A. (1989). A prospective replication of the protective effects of IQ in subjects at high risk for juvenile delinquency. *J Consult Clin Psychol, 57*(6), 719-724.

Williams, B. R., Ponesse, J. S., Schachar, R. J., Logan, G. D., & Tannock, R. (1999). Development of inhibitory control across the life span. *Dev Psychol, 35*(1), 205-213.

Williamson, D. E., Birmaher, B., Axelson, D. A., Ryan, N. D., & Dahl, R. E. (2004). First episode of depression in children at low and high familial risk for depression. *Journal of the American Academy of Child and Adolescent Psychiatry, 43*(3), 291-297.

Willoughby, T., & Hamza, C. A. (2011). A longitudinal examination of the bidirectional associations among perceived parenting behaviors, adolescent disclosure and problem behavior across the high school years. *J Youth Adolesc, 40*(4), 463-478.

Windle, M. (1990). A longitudinal study of antisocial behaviors in early adolescence as predictors of late adolescent substance use: gender and ethnic group differences. *J Abnorm Psychol, 99*(1), 86-91.

World Health Organization. (1996). *The global burden of disease : a comprehensive assessment of mortality and disability from diseases, injuries, and risk factors in 1990 and projected to 2020*. [Cambridge, Mass.] :: Published by the Harvard School of Public Health on behalf of the World Health Organization and the World Bank ;.

Appendix A – Ethics Approval

Approval Form

Date: December 8, 2011
Principal Investigator:
Thomas Wild

Study ID: Pro00026162
Study Title:
The relationship between maternal depression, adolescent depression, and the engagement in health-risk behaviours

Sponsor/Funding Agency:
9/27/2011 9/27/2011 ID00003751 Canadian Institutes of Health Research

Thank you for submitting the above study to the Health Research Ethics Board - Health Panel. Your application has been reviewed and approved on behalf of the committee.
This study uses data from the National Longitudinal Survey of Children and Youth (NLSCY), facilitated by Statistics Canada. The primary data collection did not involve identifiers.
A renewal report must be submitted next year prior to the expiry of this approval if your study still requires ethics approval. If you do not renew on or before the renewal expiry date, you will have to re-submit an ethics application.
Approval by the Health Research Ethics Board does not encompass authorization to access the patients, staff or resources of Alberta Health Services or other local health care institutions for the purposes of the research. Enquiries regarding Alberta Health Services administrative approval, and operational approval for areas impacted by the research, should be directed to the Alberta Health Services Regional Research Administration office, #1800 College Plaza, phone (780) 407-6041.

Sincerely,
Colleen Norris, Ph.D.
Associate Chair, Health Research Ethics Board - Health Panel

Note: This correspondence includes an electronic signature (validation and approval via an online system).